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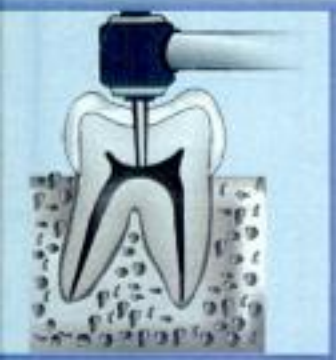
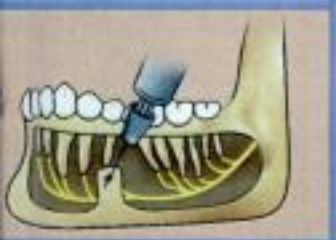
Textbook of ENDODONTICS

Nisha Garg
Amit Garg





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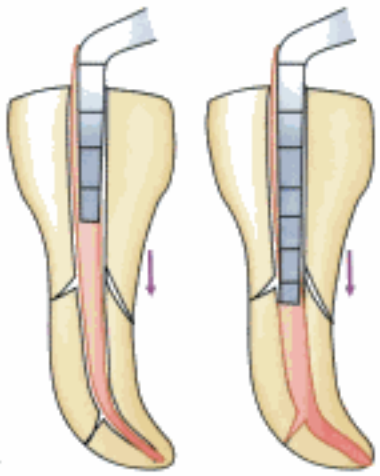

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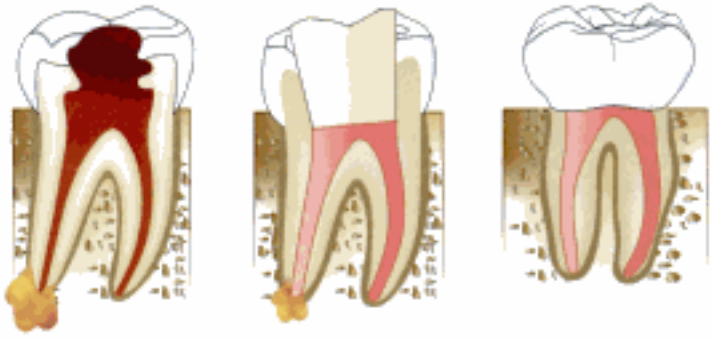

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Preface

The amount of literature available in dentistry today is vast. Endodontics being no exception. However during both our graduation as well as postgraduation we always felt the need for a book which would help us to revise and update our knowledge. When we were doing undergraduation, there were no Indian authored books on Endodontics. We were thus motivated to frame a specialized, precise, concise, easy to read and remember yet, up-to-date Textbook of Endodontics.

The line diagrams are in an expressive interpretation of endodontic procedures, which are worked upon and simplified to render them more comprehensive and comparable with real photographs. These illustrations (around 1200) are easy to remember and reproduce during examinations.

Emphasis is laid upon the language which is simple, understandable and exclusively designed for undergraduates, postgraduates, general practitioners and teachers in the field.

It took us more than three years to accomplish the arduous task of writing this book. This thrust for knowledge led us to link everywhere, where we could medline journals, books and more.

Nevertheless, a never ending approach and internal craving of mind and soul finally resulted in publication of this book. God perhaps gave us some ability and showered His light on us, guiding us for this task.

Till the last week before the publication of this book, we were frantically looking for loopholes, missing information and any important updates we might have missed out. To the best of our knowledge we did everything we could. But for knowledge one life is not enough. The sky is the limit.

We await the response of this first edition, which would improve us in the next editions to come.

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Acknowledgements

For past many years we always dreamt of writing a *Textbook of Endodontics* for the students. First and foremost we bow in gratitude to Almighty God for giving us courage to venture out this project. Without His blessings, we could not have completed the book.

Since textbooks are the products of invigorating collaborations and never ending efforts we thank our friends, colleagues, seniors, juniors, teachers and students. We shall like to express our gratitude to all the contributors, who have contributed their share of knowledge and experience in successful completion of the task.

We thank Dr Vivek Hegde (Consultant Endodontist) and Dr Gurkeerat Singh (Consultant Orthodontist) for providing procedures on RCT.

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We would also like to thank Mr Arun (DTP Operator) and Mr Manoj (Graphic Designer).

We are thankful to members of our family who co-operated with us throughout these years with their sacrifices and constant support.

Contents

1 Introduction and Scope of Endodontics

- Introduction
- Modern Endodontics
- Single Visit Endodontics
- Patient Education

INTRODUCTION

Endo is a Greek word for "Inside" and Odont is Greek for "Tooth". Endodontic treatment treats inside of the tooth.

Endodontics is the branch of clinical dentistry associated with the prevention, diagnosis and treatment of the pathosis of the dental pulp and their sequelae.

That is, the *main aim of the endodontic therapy involves to*:

i. Maintain vitality of the pulp.
ii. Preserve and store the tooth with damaged and necrotic pulp.
iii. Preserve and restore the teeth which have failed to the previous endodontic therapy, to allow the tooth to remain functional in the dental arch.

Thus we can say that the primary goal of endodontic therapy is to create an environment within the root canal system which allows the healing and continued maintenance of the health of the periradicular tissue.

Since nothing is as good as the natural teeth, one should take care of them. The endodontic therapy is a necessary treatment to cure a damaged or diseased tooth.

Endodontics has been defined as art as well as science of clinical dentistry because in spite of all the factual scientific foundation on which the endodontics is based, to provide an ideal endodontic treatment is an art in itself.

Before understanding what is root canal therapy, how and when it is performed and other facts regarding endodontic therapy, we should be familiar with the *history of endodontics*.

Endodontics has been practiced as early as second or third century BC. The history of endodontics begins in 17th century and since then many advances, developments and research work has been proceeded continuously.

Though advances in endodontics have been made continuously, but especially after Pierre Fauchard (1678-1761) [Founder of modern dentistry] in his textbook "Le Chirugien Dentiste" described the pulp very precisely.

Latter in 1725, Lazare Rivere introduced the use of clove oil as sedative and in 1746, Pierre Fauchard demonstrated the removal of pulp tissue. Dr Grossman, the pioneer of endodontics divided the evolution of endodontics in four eras from 1776 to 1976, each consisting of 50 years.

Pre science : 1776 to 1826
Age of discovery : 1826 to 1876
Dark age : 1876 to 1926
The renaissance : 1926 to 1976
Innovation Era : 1977 till date

Pre science (1776 to 1826): In this era, endodontic therapy mainly consisted of crude modalities like abscesses were being treated with poultices or leeches and pulps were being extirpated using hot cautery.

Age of discovery (1826 to 1876): In this era there occurred the development of anesthesia, gutta-percha and barbed broaches. Also the medications were created for treating pulpal infections and the cements and pastes were discovered to fill them.

The dark age (1876 to 1926): In spite of introduction of X-rays and general anesthesia, extractions was the choice of treatment than endodontics in most of the cases of damaged teeth because theory of the focal infection was main concern at that time.

The renaissance (1926 to 1976): In this era, endodontics was established as science and therapy, forming its golden era. It showed the improvement in anesthesia and radiographs for better treatment results. The theory of focal infection was also fading out, resulting in more of endodontics being practiced and in 1943, because of growing interest in endodontics, the AAE, that is, the American Association of Endodontists was found.

Innovation era: It is the period from 1977 onwards in which tremendous advancements at very fast rate are being introduced in the endodontics. The better vision, better techniques of biomechanical preparations, and obturation are being developed resulting in the simpler, easier and faster endodontics with more of the successful results.

Also the concept of single visit endodontics is now globally accepted in contrast to multiple visits.

MODERN ENDODONTICS

As we have seen, over the years, there has been a great improvement in the field of endodontics. Many researches have been conducted and papers are being presented regarding the advances, modifications and change in attitude regarding endodontic therapy. In the past two decades, extensive studies have been done on microbial flora of pulp and the periapical tissue. The biological changes, role of innate and acquired immunological factors are being investigated in dental pulp after it gets infected, healing of the periapical tissue after undergoing root canal therapy is also being investigated.

Various ways to reduce the levels of microbial infection viz. chemical, mechanical and their combination have led to development of newer antimicrobial agents and techniques of biomechanical preparation for optimal cleaning and shaping of the root canals.

To increase the efficiency of root canal instrumentation, introduction of engine driven rotary instruments is made. Introduction of Nickel Titanium multitapered instruments with different types of cutting tips have allowed the better, easier and efficient cleaning and shaping of the root canals.

The advent of endomicroscope in the field of endodontics has opened the great opportunities for an endodontist. It is used in every phase of the treatment, i.e. from access opening till the obturation of root canals. It makes the images both magnified and illuminated, thus helps in making the treatment more predictable and eliminating the guess work.

Introduction of newer obturation systems like system B Touch and heat have made it possible to fill the canal three dimensionally. Material like MTA (Mineral Trioxide Aggregate), a root canal repair material has made the procedures like apexification, perforation repair to be done under moist field. Since endodontics is based on the principles of inflammation, pulp and periapical disease processes and the treatment available, future of endodontics lies to redefine the rationale of endodontic therapy using newer modalities and to meet the set of standards of excellence in the future.

SINGLE VISIT ENDODONTICS (SVE)

Single visit endodontics is gaining popularity these days as compared to multiple visits. SVE implies to cleaning, shaping and disinfection of a root canal system followed by obturation of the root canal at the same appointment. The concept of single visit endodontics started at least 100 years back. Initiating and completing an endodontic treatment in one appointment has always been surrounded by controversy. In 1982 a survey revealed that 87 percent of endodontists did not trust that most necrotic teeth could be treated successfully in one visit. In addition, the majority of endodontists thought that performing treatment in this manner would cause more postoperative pain than if performed in multiple appointments. But now many studies have shown that completing the treatment in single sitting show no difference in quality of the treatment, success rate and incidence of post-operative complications. However, a growing number dentists are practicing more and more single visit endodontics.

Though the concept of SVE is gaining the recognition, but it is still surrounded by controversies regarding post operative pain, flare-ups and the healing rate followed by root canal therapy.

The *most common factors which appear to be responsible for not performing SVE are as follows*:

i. Doubt of postoperative pain
ii. Fear of failure of the endodontic therapy.
iii. Discomfort to patient because he/she has to keep the mouth open for a long period of time.
iv. Lack of time.
v. Lack of experience and equipment.

Advantages of SVE

- ***Convenience***: Patient doesn't have to endure the discomfort of repetitive local anesthesia, treatment procedure and postoperative recovery.
- ***Efficiency***: The clinicians doesn't have to refamiliarize himself/herself to patient's particular anatomy or landmarks.
- ***Patient comfort***: Because of reduced number of visits and injections.
- ***Reduced intra-appointment pain***: Mostly the mid treatment flare-ups are caused by leakage of the temporary cements. This figure has seen to be reduced in SVE cases.
- ***Economics***: Extra cost of multiple visits, use of fewer materials and comparatively less chair side time all increase the economics to both patient as well as doctor.
- ***Minimizes the fear and anxiety***: Specially beneficial for patients who have psychological trauma and fera of dentist.
- ***Reduces incomplete treatment***: Some patients don't return to complete the root canal therapy, SVE reduces this risk.
- ***Lesser errors in working length***: In multiple visits, the reference point could be lost because of fracture or unwanted grinding in case of flare-ups leading to loss of actual working length. These errors are avoided in SVE.
- ***Restorative consideration***: In SVE, immediate place-ment of coronal restoration (post and core placements) ensure effective coronal seal and esthetics.

Disadvantages of SVE

- It is tiring for patients to keep their mouth open for long durations
- If mid treatment flare ups happen to occur, it is easier to establish drainage in a tooth which is not obturated
- Clinician may lack the proficiency to properly treat a case in single visit
  - SVE cannot be performed in all cases.
  - If hemorrhage or exudation occurs, it becomes difficult for the clinician to control and complete the case in same visit.
  - Difficult cases with very fine, curved, calcified, multiple canals may not be treatable in single visit.

Criteria of Case Selection as given by Oliet Include

- Positive patient acceptance
- Absence of acute symptoms
- Absence of continuous hemorrhage or exudation
- Absence of anatomical interferences like presence of fine, curved or calcified canals
- Availability of sufficient time to complete the case
- Absence of procedural difficulties like canal blockage, ledge formation or perforations.

Conditions where Single Visit Endodontics cannot be Performed

- Teeth with anatomic anomalies such as calcified and curved canals
- Asymptomatic non vital teeth with periapical pathology and no sinus tract
- Acute alveolar abscess cases with frank pus discharge
- Patients with acute apical periodontitis
- Symptomatic non vital teeth and no sinus tract
- Retreatment cases
- Patients with allergies or previous flare-ups
- Teeth with limited access
- Patients who are unable to keep mouth open for long durations such as patients with TMJ disorders.

Indications of Single Visit Endodontics

- Vital teeth
- Fractured anteriors where esthetics is the concern
- Patients who require sedation every time
- Non vital teeth with sinus tract
- Medically compromised patients who require antibiotics prophylaxis
- Physically compromised patients who cannot come to dental clinics frequently.

In conclusion, single-visit endodontics has been shown to be an effective treatment modality, which compared to multiple-visit therapy, is more beneficial to patients and dentists in many ways provided there is careful case selection and adherence to standard endodontic principles. The prevention and elimination of apical periodontitis are the goals of a successful endodontics therapy, once the way to accomplish these goals is determined, the decision to provide treatment in multiple visits or single visit will follow itself.

PATIENT EDUCATION

Most of the patient who are given endodontic treatment, are often curious and interested in their treatment. For such patients following information should be transferred to the patient in anticipation of frequently asked questions.

Who Performs an Endodontic Therapy?

Generally, all dentists receive basic education in endodontic treatment but an endodontist is preferred for endodontic therapy. General dentists often refer patients needing endodontic treatment to endodontists.

Who is an Endodontist?

An endodontist is a dentist who undergoes a special training in diagnosing and treating the problems associated with inside of the tooth. To become specialists, they complete dental school and an additional two or more years of advanced training in endodontics. They perform routine as well as difficult and very complex endodontic procedures, including retreatment of previous root canals that have not healed completely, as well as endodontic surgery.

What is Endodontics?

Endodontics is the diagnosis and treatment of inflamed and damaged pulps. Teeth are composed of protective hard covering (enamel, dentin and cementum) encasing a soft living tissue called pulp (Fig. 1.1). Pulp contains blood vessels, nerves, fibres and connective tissue. The pulp extends from the crown of the tooth to the tip of the roots where it connects to the tissues surrounding the root. The pulp is important during a tooth's growth and development. However, once a tooth is fully mature it can survive without the pulp, because the tooth continues to be nourished by the tissues surrounding it.

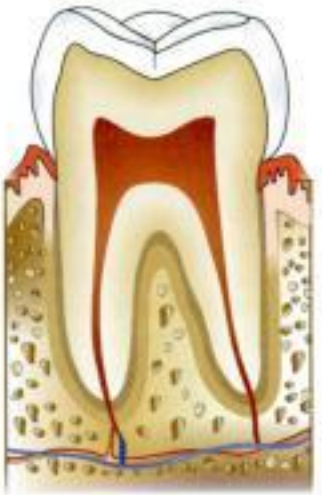


Fig. 1.1: Normal anatomy of a tooth showing enamel, dentin, cementum and pulp

How does Pulp Become Damaged?

Number of ways which can damage the pulp include tooth decay, gum diseases, injury to the tooth by accident (Fig. 1.2).

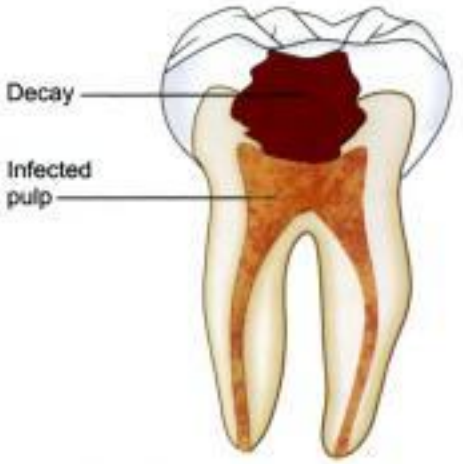


Fig. 1.2: Tooth decay causing damage to pulp

Why do I Feel Pain?

When pulp becomes infected, it causes increased blood flow and cellular activity, and pressure cannot be relieved from inside the tooth. This causes pain. Pulp can even die without causing significant pain.

How can You Tell if Pulp is Infected?

When pulp gets inflamed, it may cause toothache on taking hot or cold, spontaneous pain, pain on biting or on lying down. on occasion a damaged pulp is noticed by drainage, swelling, and abscess at the root end (Fig. 1.3). Sometimes, however, there are no symptoms.

Why do I Need Root Canal Therapy?

Because tooth will not heal by itself, the infection may spread around the tissues causing destruction of bone and supporting tissue. This may cause tooth to fall out. Root canal treatment is done to save the damaged pulp by thorough cleaning and shaping of the root canal system and

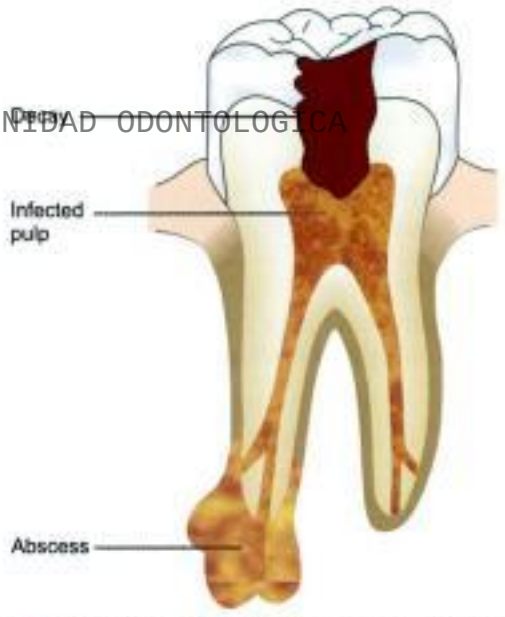


Fig. 1.3: Tooth with infected pulp and abscess formation

then filling it with gutta-percha (rubber like) material to prevent recontamination of the tooth. Tooth is permanently restored with crown with or without post.

What are Alternatives to Root Canal Therapy?

If tooth is seriously damaged and its support is compromised, then extraction is only alternative.

What is Root Canal Procedure?

Once the endodontic therapy is recommended, your endodontist will numb the area by injecting local anesthetic. After this a rubber sheet is placed around the tooth to isolate it. Then the opening is made in the crown of the tooth and very small sized instruments are used to clean the pulp from pulp chamber and root canals (Fig. 1.4). After thorough cleaning and shaping of root canals (Fig. 1.5), they are filled with rubber like material called gutta-percha, which will prevent the bacteria from entering this space again (Fig. 1.6).

After completion of endodontic therapy, the endodontist places the crown or other restoration so as to restore the tooth to pull function (Fig. 1.7).

What are Risks and Complications?

It has been seen that more than 95 percent cases of endodontic therapy are successful. However sometimes because of unnoticed canal malformations, instrument errors a root canal therapy may fail.

Fig. 1.4: Cleaning and shaping of root canal system

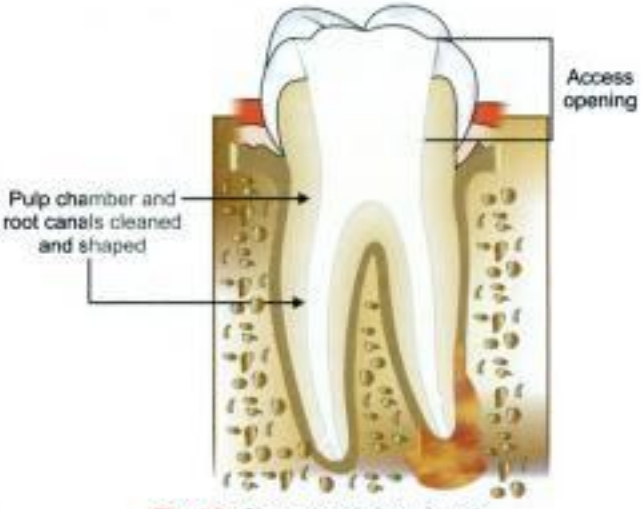


Fig. 1.5: Cleaned and shaped tooth

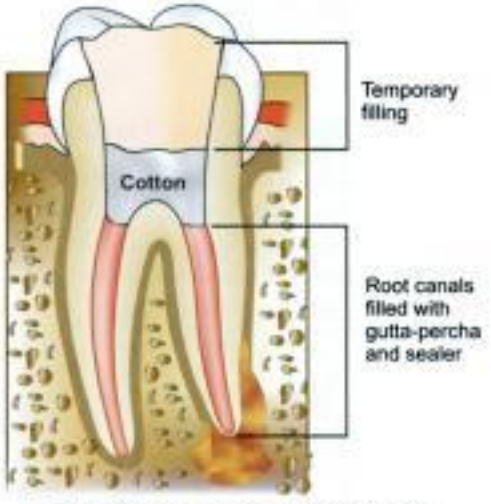


Fig. 1.6: Obturation of root canal system

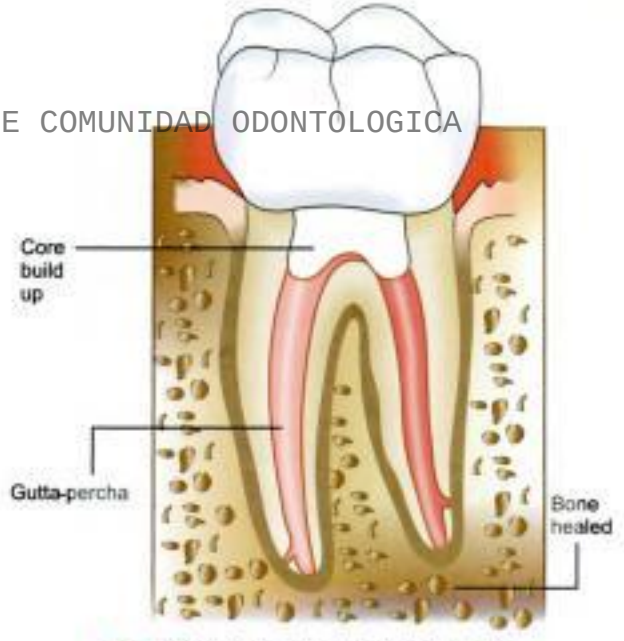


Fig. 1.7: Complete restoration of tooth with crown placed over the restored tooth

Does the tooth need any special care after endodontic therapy?

Since unrestored tooth is more prone to fracture so you should not chew hard until it has been completely restored, otherwise you should continue your regular oral hygiene routine including brushing, flossing and regular check-up.

How many Visits will it Take to Complete this Treatment?

Nowadays most of the treatment can be completed in 1-2 visits. But treatment time can vary according to condition of the tooth.

Will I Feel Pain during or after Treatment?

Nowadays with better techniques, and better understanding of anesthesia most of the patients feel comfortable during the treatment. But for first few days after therapy, you might feel sensation especially if pain and infection was present prior to the procedure. This pain can be relieved by medication. If continuous severe pain or pressure remains, consult your endodontist.

Will I have a Dead Tooth after Root Canal Therapy?

No, since tooth is supplied by blood vessels present in periodontal ligament. It continues to receive the nutrition and remains healthy.

Will the Tooth Need Any Special Care or Additional Treatment after Endodontic Treatment?

You should not chew or bite on the treated tooth until you have had it restored by your dentist. The unrestored tooth is susceptible to fracture, so you should visit your dentist for a full restoration as soon as possible. Otherwise, you need only practice good oral hygiene, including brushing, flossing, and regular check-ups and cleanings.

Most endodontically treated teeth last as long as other natural teeth. In a few cases, a tooth that has undergone endodontic treatment does not heal or the pain continues. Occasionally, the tooth may become painful or diseased months or even years after successful treatment. Often when this occurs, redoing the endodontic procedure can save.

Can All Teeth be Treated Endodontically?

Most of the teeth can be treated endodontically. But sometimes when root canals are not accessible, root is severely fractured, tooth cannot be restored or tooth doesn't have sufficient bone support, it becomes difficult to treat the tooth endodontically. However, advances in endodontics are making it possible to save the teeth that even a few years ago would have been lost.

Newer researches, techniques and materials have helped us to perform the endodontic therapy in better way with more efficiency. Since introduction of rotary instruments and other technologies reduce the treatment time, the concept of single visit is gaining popularity nowadays. It has been shown that success of endodontic therapy depends on the quality of root canal treatment and not the number of visits. In the modern world single visit endodontics is becoming quite popular.

2 Pulp and Periradicular Tissue

- Introduction
- Development of Dental Pulp
- Histology of Dental Pulp
- Anatomy of Dental Pulp
- Supportive Element
- Pulpal Response to Inflammation
- Innervation of Pulp
- Functions of Pulp
- Age Changes in Pulp
- Pulp Stones
- Calcific Metamorphosis
- Periradicular Tissues

INTRODUCTION

The dental pulp is soft tissue of mesenchymal origin located in the centre of the tooth. It consists of specialized cells, odontoblasts arranged peripherally in direct contact with dentin matrix. This close relationship between odontoblasts and dentin is known as 'Pulp – dentin complex" (Fig. 2.1).

Fig. 2.1: Pulp dentin complex

The pulp is connective tissue system composed of cells, ground substances, fibers, interstitial fluid, odontoblasts, fibroblasts and other cellular components. Pulp is actually a microcirculatory system consists of arterioles and venules as the largest vascular component. Due to lack of true collateral circulation pulp is dependent upon few arterioles entering through the foramen. Due to presence of the specialized cells, i.e. odontoblasts as well as other cells which can differentiate into hard tissue secreting cells; the pulp retains its ability to form dentin throughout the life. This enables the vital pulp to partially compensate for loss of enamel or dentin occurring with age. The injury to pulp may cause discomfort and the disease. Consequently the health of pulp is important to successful completion of the restorative procedures. Because the symptoms as well as radiographic and clinical signs of pulp diseases are not always differentiated from sign and symptoms of other dental diseases, knowledge of biology of pulp is essential for development of rational treatment plan. In this chapter, we

would discuss the comprehensive description of pulp embryology, anatomy, histology, physiology and pulp changes with age.

Features of Pulp which Distinguish it from Tissue found Elsewhere in the Body:

a. Pulp is surrounded by rigid walls and so is unable to expand in response to injury as a part of the inflammatory process. So, pulpal tissue is susceptible to a change in pressure affecting the pain threshold.
b. There is minimal collateral blood supply to pulp tissue which will reduce its capacity for repair following injury.
c. The pulp is composed almost entirely of simple connective tissue, yet at its periphery it is a layer of highly sophisticated cells, the odontoblasts. Secondary dentine is gradually deposited as a physiological process which reduces the blood supply and therefore, the resistance to infection or trauma.
d. The innervation of pulp tissue is both simple and complex. Simple in that there are only free nerve endings and consequently the pulp lacks proprioception. Complex because of innervation of the odontoblast processes which produces a high level of sensitivity to thermal and chemical change.

DEVELOPMENT OF DENTAL PULP

The pulp originated from ectomesenchymal cells of dental papilla. Dental pulp is identified when these cells mature and dentin is formed. Basically the development of tooth is divided into bud, cap and bell stage. The bud stage (Fig. 2.2) is initial stage where epithelial cells of dental lamina proliferate and produce a bud like projection into adjacent ectomesenchyme. The cap stage (Fig. 2.3) is formed when cells of dental lamina proliferate to form a concavity which produces cap like appearance. It shows outer and inner

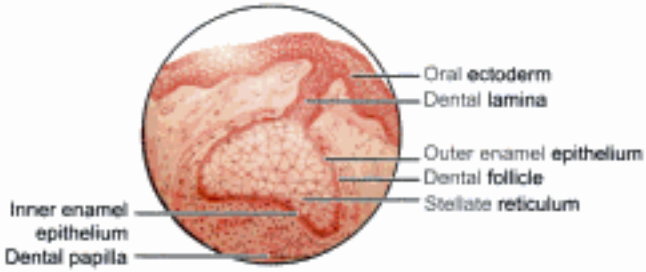


Fig. 2.3: Development of tooth showing cap stage

enamel epithelia and stellate reticulum. The rim of the enamel organ, i.e. where inner and outer enamel epithelia are joined is called cervical loop. As the cells of loop proliferate, enamel organ assumes bell shape (Fig. 2.4).

The differentiation of epithelial and mesenchymal cells into ameloblasts and odontoblasts occur during bell stage. The pulp is initially called as dental papilla; it is designated as pulp only when dentin forms around it. The differentiation of odontoblasts from undifferentiated ectomesenchymal cells is accomplished by interaction of cell and signaling molecules mediated through basal lamina and extracellular matrix. The dental papilla has high cell density and the rich vascular supply as a result of proliferation of cells with in it.

The cells of dental papilla appear as undifferentiated mesenchymal cells, gradually these cells differentiate into fibroblasts. The formation of dentin by odontoblasts heralds the conversion of dental papilla into pulp. The boundary between inner enamel epithelium and odontoblast form the future dentinoenamel junction. The junction of inner and outer enamel epithelium at the basal margin of enamel organ represent the future cementoenamel junction. As the crown formation with enamel and dentin deposition continues, growth and organization of pulp vasculature occurs.

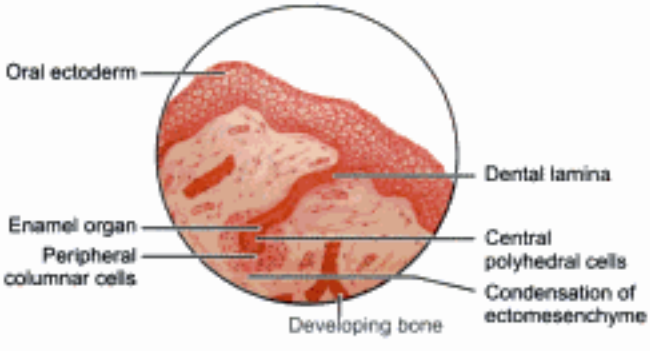


Fig. 2.2: Development of tooth showing bud stage

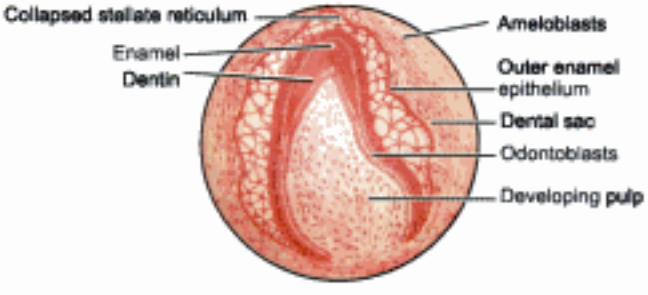


Fig. 2.4: Development of tooth showing "Bell stage"

According to Saunders (1966) and Cutright (1970), the blood supply of the developing tooth bud originates from an oval or circular reticulated plexus in the alveolar bone. Series of small vessels arise from the plexus, which then grows into dental papilla.

At the same time as tooth develops unmyelinated sensory nerves and autonomic nerves grow into pulpal tissue. Myelinated fibers develop and mature at a slower rate, plexus of Raschkow does not develop until after tooth has erupted.

HISTOLOGY OF DENTAL PULP

Basically the pulp is divided into the central and the peripheral region. The central region of both coronal and radicular pulp contains nerves and blood vessels.

The peripheral region contains the following zones (Fig. 2.5).

a. Odontoblastic layer
b. Cell poor zone
c. Cell rich zone

A. ***Odontoblastic layer***: As you know, the odontoblasts consists of cell bodies and their cytoplasmic processes. The odontoblastic cell bodies form the odontoblastic zone whereas the odontoblastic processes are located within predentin matrix. Capillaries, nerve fibers (unmyelinated) and dendritic cells may be found around the odontoblasts in this zone.

B. ***Cell free zone of Weil***: Central to odontoblasts is subodontoblastic layer, termed cell free zone of Weil. It contains plexuses of capillaries and small nerve fibers ramification.

C. ***Cell rich zone***: This zone lies next to subodontoblastic layer. It contains fibroblasts, undifferentiated cells which maintain number of odontoblasts by proliferation and differentiation.

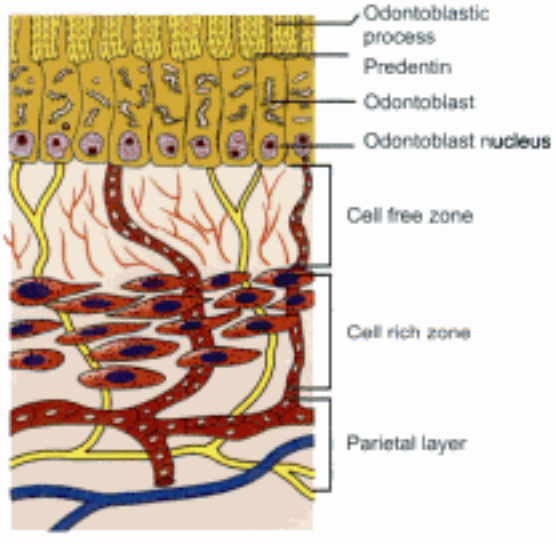


Fig. 2.5: Zones of pulp

CENTRAL PULP ZONE

Contents of the Pulp

I. Cells	1. Odontoblasts	
	2. Fibroblasts	
	3. Undifferentiated mesenchymal cells	
	4. Defense cells	- Macrophages Plasma cells Mast cells
II. Matrix	1. Collagen fibers	- Type I Type II
	2. Ground Substance	- Glycosaminoglycans - Glycoproteins - Water
III. Blood Vessels	- Arterioles, Venules, Capillaries	
IV. Lymphatics	- Draining to submandibular, submental and deep cervical nodes	
V. Nerves	- Subodontoblastic plexus of Raschkow	
	- Sensory afferent from Vth nerve and Superior cervical ganglion	

It is circumscribed by cell rich zone. It contains large vessels and nerves from which branches extend to peripheral layers. Principal cells are fibroblasts with collagen as ground substance.

Structural or Cellular Elements

a. ***Reserve cells/undifferentiated mesenchymal cells***: Undifferentiated mesenchymal cells are descendants of undifferentiated cells of dental papilla which can dedifferentiate and then redifferentiate into many cell types.

For example: depending on the stimulus, these cells may give rise to odontoblasts and fibroblasts. These cells are found throughout the cell-rich area and the pulp core and often are related to blood vessels. When these cells are examined under light microscope, they appear as large polyhedral cells possessing a large, lightly stained, centrally placed nucleus and displays abundant cytoplasm and peripheral cytoplasm extensions. In older

pulps, the number of undifferentiated mesenchymal cells diminishes, along with number of other cells in the pulp core. This reduction, along with other aging factors, reduces the regenerative potential of the pulp.

b. ***Fibroblasts***: The cells found in greatest numbers in the pulp are fibroblasts. 'Baume' refers them to mesenchymal cells/pulpoblasts or pulpocytes in their progressive levels of maturation. These are particularly numerous in the coronal portion of the pulp, where they form the cell-rich zone. These are spindle shaped cells which secrete extracellular components like collagen and ground substance. They also eliminate excess collagen by action of lysosomal enzymes. Fibroblasts of pulp are much like ***'Peter Pan'*** because they "never grow up" because they remain in relatively undifferentiated state (Fig. 2.6).

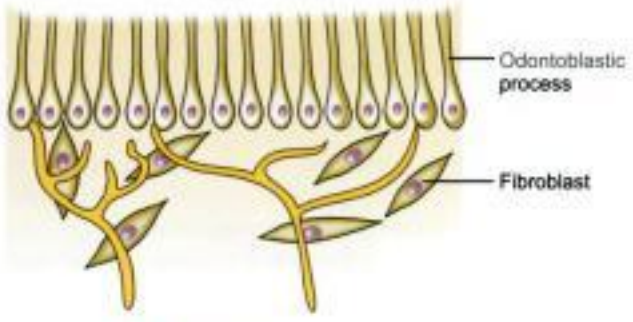


Fig. 2.6: Histology of pulp showing fibroblasts

c. ***Defence cells***: (Fig. 2.7)

I. ***Histiocytes and macrophages***: They originate from undifferentiated mesenchymal cells or monocytes. They appear as large oval or spindle shaped cells which are involved in the elimination of dead cells, debris, bacteria and foreign bodies, etc.

II. ***Polymorphonuclear leukocytes***: Most common form of leukocyte is neutrophil, though it is not present in healthy pulp. They are major cell type in micro abscesses formation and are effective at destroying and phagocytising bacteria and dead cells.

III. ***Lymphocytes***: In normal pulps, mainly T- lymphocytes are found but B-lymphocytes are scarce. They appear at the site of injury after invasion by neutrophils. They are associated with injury and resultant immune response. Thus their presence indicates presence of persistent irritation.

IV. ***Mast cells***: On stimulation, degranulation of mast cells release histamine which causes vasodilatation, increased vessel permeability and thus allowing fluids and leukocytes to escape.

V. ***Odontoblasts***: They are first type of cells encountered as pulp is approached from dentin. The number of odontoblasts has been found in the range of 59,000 to 76,000 per square millimeter in coronal dentin, with a lesser number in root dentin. In the crown of

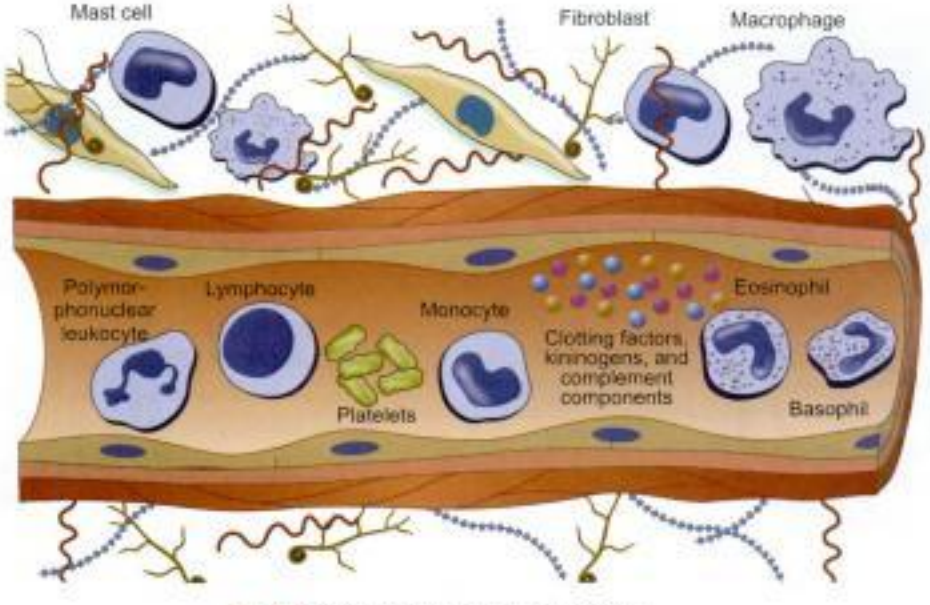


Fig. 2.7: Cells taking part in defence of pulp

the fully developed tooth, the cell bodies of odontoblasts are columnar and measure approximately 500 µm in height, whereas in the midportion of the pulp they are more cuboidal and in apical part, more flattened.

The morphology of odontoblasts reflects their functional activity and ranges from an active synthetic phase to a quiescent phase. Ultrastructure of the odontoblast shows large nucleus which may contain up to 4 nucleoli. Nucleus is situated at basal end (Fig. 2.8). Golgibodies is located centrally. Mitochondria, rough endoplasmic reticulum (RER), ribosome are also distributed throughout the cell body.

Odontoblasts synthesize mainly type I collagen, proteoglycans. They also secrete sialoproteins, alkaline phosphatase, phosphophoryn (phosphoprotein involved in extracellular mineralization).

Irritated odontoblast secretes collagen, amorphous material, and large crystals into tubule lumen which result in dentin permeability to irritating substance.

Similar Characteristic Features of Odontoblasts, Osteoblasts and Cementoblasts

1. They all produce matrix composed of collagen fibers and proteoglycans capable of undergoing mineralization.
2. All exhibit highly ordered RER, golgi complex, mitochondria, secretory granules, rich in RNA with prominent nucleoli.

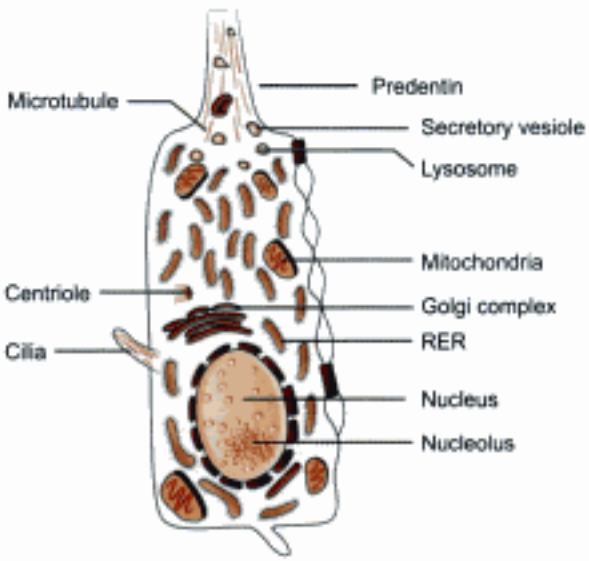


Fig. 2.8: Diagram showing odontoblasts

Difference between Odontoblasts, Osteoblasts and Cementoblasts

1. Odontoblasts are in columnar shape while osteoblasts and cementoblast are polygonal in shape.
2. Odontoblasts leave behind cellular processes to form dentinal tubules while osteoblasts and cementoblast are trapped in matrix as osteocytes and cementocytes.

Extracellular components: The extra cellular components include fibers and the ground substance of pulp:

Fibers: The fibers are principally type I and type III collagen. Collagen is synthesized and secreted by odontoblasts and fibroblasts whereas the overall collagen content of the pulp increases with age, while the ratio between types I and II remains stable. Fibers produced by these cells differ in the degree of cross-linkage and variation in hydroxyline content. Fibers secreted by fibroblasts don't calcify.

Fibers are more numerous in radicular pulp than coronal and greatest concentration of collagen generally occurs in the most apical portion of the pulp. This fact is of practical significance when a pulpotomy is performed during the course of endodontic treatment. Engaging the pulp with a barbed broach in the region of the apex affords a better opportunity to remove the tissue intact than does engaging the broach more coronally, where the pulp is more gelatinous and liable to tear.

Collagen with age becomes coarser and can lead to formation of pulp stones.

In peripheral pulp, collagen fibers have unique arrangement forming Von korff's fibers. These are corkscrew like originating between odontoblasts and pass into dentin matrix.

Ground Substance

The ground substance of the pulp is part of the system of ground substance in the body. It is a structureless mass with gel like consistency forming bulk of pulp. ***Chief Components of ground substance*** are

a. Glycosaminoglycans
b. Glycoproteins
c. Water

So, we can see that ground substance of the pulp is similar to that of connective tissue elsewhere in the body.

Functions of ground substance:

1. Forms the bulk of the pulp.
2. Supports the cells.

3. Acts as medium for transport of nutrients from the vasculature to the cells and of metabolites from the cells to the vasculature.
4. Alterations in the composition of ground substance caused by age or disease interfere with metabolism, reduced cellular function and irregularities in mineral deposition.

Depolymerization by enzymes produced by micro-organisms found in pulpal inflammation may change ground substance of the pulp. Alexander et al in 1980 found that these enzymes can degrade the ground substance of the pulp by disrupting the glycosaminoglycan – collagen linkage. Thus, the ground substance plays an important role in health and diseases of the pulp and dentin.

ANATOMY OF DENTAL PULP

Pulp lies in the centre of tooth and shapes itself to miniature form of tooth. This space is called pulp cavity which is divided into pulp chamber and root canal (Fig. 2.9).

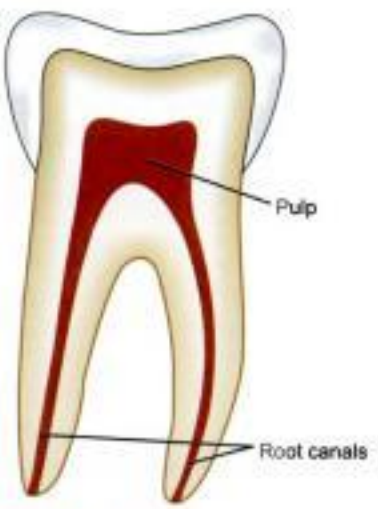


Fig. 2.9: Diagram showing pulp cavity

In the anterior teeth, the pulp chamber gradually merges into the root canal and this division becomes indistinct (Fig. 2.10). But in case of multirooted teeth, there is a single pulp chamber and usually two to four root canals (Fig. 2.11). As the external morphology of the tooth varies from person to person, so does the internal morphology of crown and the root. The change in pulp cavity anatomy results from age, disease, trauma or any other irritation.

Fig. 2.10: Diagram showing pulp anatomy of anterior tooth

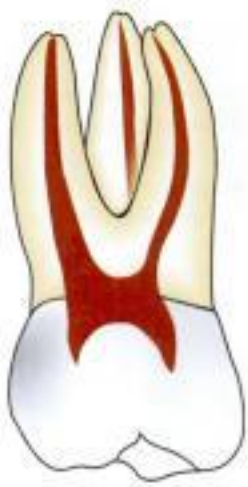


Fig. 2.11: Diagram showing pulp cavity of posterior tooth

PULP CHAMBER

Reflects the external form of enamel at the time of eruption, but anatomy is less sharply defined. The roof of pulp chamber consists of dentin covering the pulp chamber occlusally. Canal orifices are openings in the floor of pulp chamber leading into the root canals (Fig. 2.12).

A specific stimulus such as caries leads to the formation of irritation dentin while with time, pulp chamber shows reduction in size as secondary or tertiary dentin is formed (Fig. 2.13).

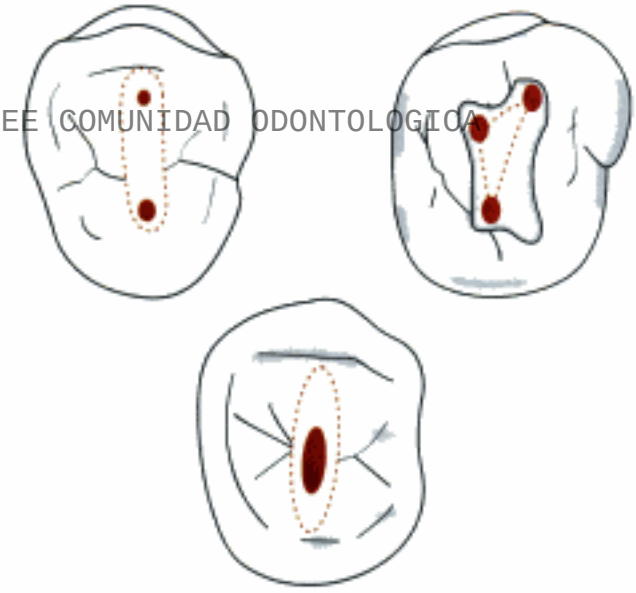


Fig. 2.12: Diagram showing opening of canal orifices in the pulp chamber

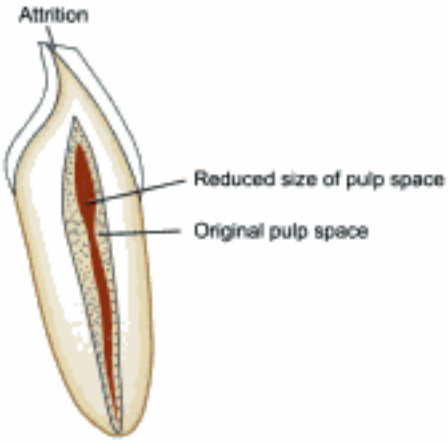


Fig. 2.13: Reduction in size of pulp cavity because of formation of secondary and tertiary dentin

ROOT CANAL

It is that portion of pulp cavity which extends from canal orifice to the apical foramen. The shape of root canal varies with size, shape, number of the roots in different teeth. A straight root canal throughout the entire length of root is uncommon. Commonly curvature is found along its length which can be gradual or sharp in nature (Fig. 2.14). In most cases, numbers of root canals correspond to number of roots but a root may have more than one canal.

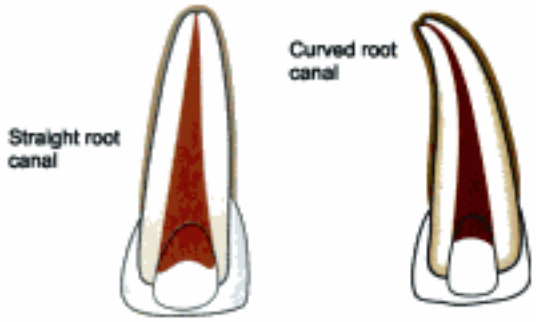


Fig. 2.14: Straight and curved root canal

According to Orban, the shape of the canal to large extent is largely determined by the shape of the root. Root canals can be round, tapering elliptical, broad, thin, etc.

'MEYER' stated that root which are round and cone shaped usually contain one canal but roots which are elliptical with flat or concave surface frequently have more than one canal (Fig. 2.15).

Fig. 2.15: Diagram showing relationship between shape of root and number of root canals

Change in shape and location of foramen is seen during post-eruptive phase due to functional forces (tongue pressure, mesial drift) acting on the tooth which leads to cementum resorption and deposition on the walls of foramen. This whole process resulted in new foramen away from the apex. The total volume of all permanent pulp organs is 0.38 cc with mean of 0.02 cc.

The apical foramen is an aperture at or near the apex of a root through which nerves and blood vessels of the pulp enter or leave the pulp cavity (Fig. 2.16). Normally, it is present near the apex but sometimes, opening may be present on the accessory and lateral canals of root surface forming the accessory foramina.

In young newly erupted teeth, it is wide open but as the root develops, apical foramen becomes narrower. The inner surface of the apex becomes lined with the cementum which may extend for a short distance into the root canal. Thus we can say that DCJ does not necessarily occur at the apical end of root, but may occur within the main root canal (Fig. 2.17).

Multiple foramina are frequent phenomenon in multirooted teeth. Majority of single rooted teeth have single

Fig. 2.16: Apical foramen through which nerves and blood vessels enter or leave the pulp cavity

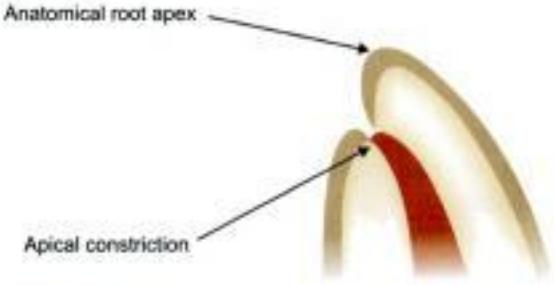


Fig. 2.17: Diagram showing cemento dentinal junction

canal which terminate in a single foramina. Continuous deposition of new layers of cementum causes change in foramen anatomy. Average size of maxillary teeth is 0.4 mm and of mandibular teeth is 0.3 mm.

Accessory canals: are lateral branches of the main canal that form a communication between the pulp and periodontium. Accessory canals contain connective tissue and vessels and can be seen anywhere from furcation to apex but tend to more common in apical third and in posterior teeth (Fig. 2.18).

In other words, more apical and farther posterior the tooth, the more likely the accessory canals will be present.

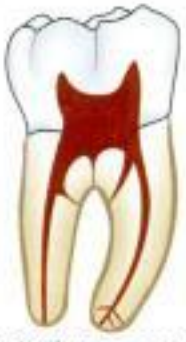


Fig. 2.18: Diagram showing accessory and lateral canals

Exact mechanism of their formation is not known but they occur in areas where there is premature loss of root sheath cells because these cells induce formation of odontoblasts. They also develop where developing root encounters a blood vessel. If vessel is located in this area, where dentin is forming; hard tissue may develop around it making a lateral canal from radicular pulp.

SUPPORTIVE ELEMENTS

Pulpal Blood Supply

Teeth are supplied by branches of maxillary artery. Mature pulp has an extensive and unique vascular pattern that reflects its unique environment. Blood vessels which are branches of dental arteries consisting of arterioles enter the dental pulp by way of apical and accessory foramina. One or sometimes two vessels of arterioler size (about 150 µm) enter the apical foramen with sensory and sympathetic nerve bundles. The arterioles course up through radicular pulp and give off branches which spread laterally towards the odontoblasts layer and form capillary plexus. As they pass into coronal pulp, they diverge towards dentin, diminish in size and give rise to capillary network in sub-odontoblastic region (Fig. 2.19). This network provides odontoblasts with rich source of metabolites.

Blood passes from capillary plexus into venules which constitute the efferent (exit) side of the pulpal circulation and are slightly larger than corresponding arterioles. Venules enlarge as they merge and advance toward the apical foramen. Efferent vessels are thin walled and show only scanty smooth muscle.

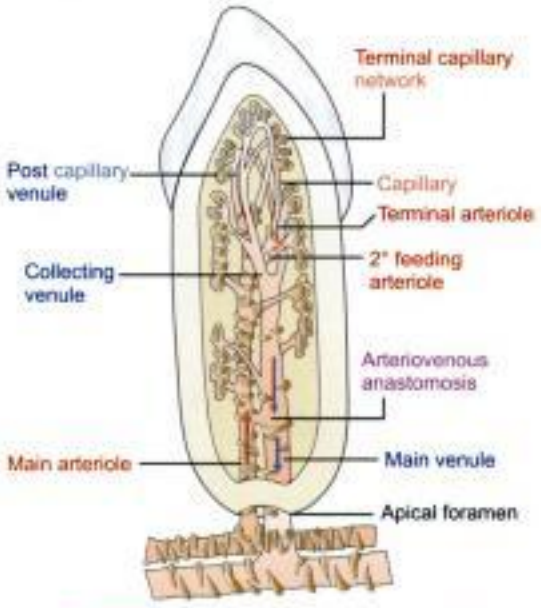
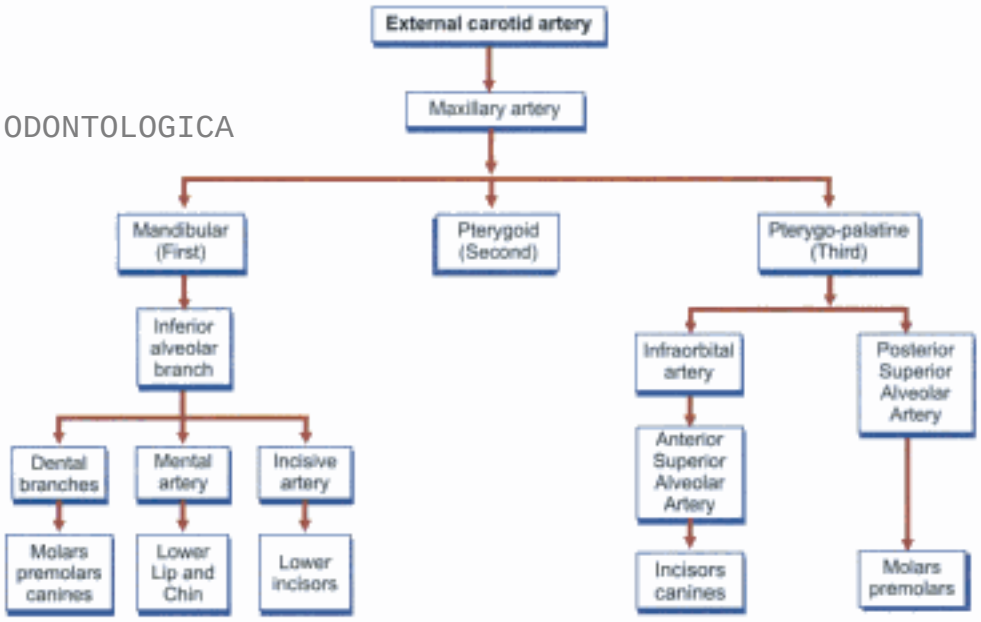


Fig. 2.19: Diagram showing circulation of pulp



(Arterial supply of teeth)

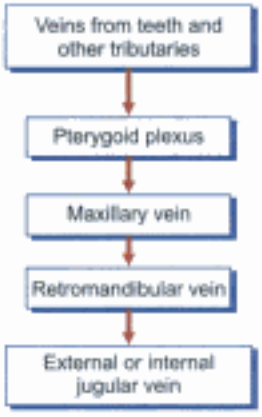
Lymphatic Vessels

Arise as small, blind, thin-walled vessels in the coronal region of the pulp and pass apically through middle and radicular regions of the pulp to exit via one or two large vessels through the apical foramen. Lymphatic can be differentiated from small venules in following ways:

1. Presence of discontinuities in vessel walls.
2. Absence of RBC in their lumina.

Regulation of Pulpal Blood Flow

Walls of arterioles and venules are associated with smooth muscles which are innervated by unmyelinated sympathetic fibers. When stimulated by electrical stimulus (epinephrine containing LA, etc.), muscle fiber contract, thus decrease the blood supply (Fig. 2.20).



Venous drainage from the teeth

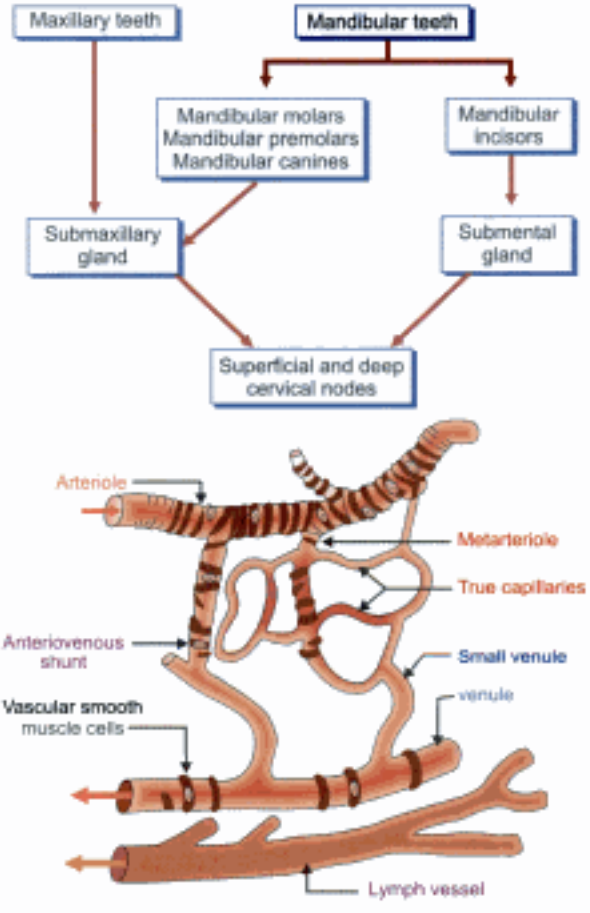


Fig. 2.20: Diagram showing regulation of pulpal blood flow

Pulpal Response to Inflammation

Whenever there is inflammatory reaction, there is release of lysosomal enzymes which cause hydrolysis of collagen and the release of kinins, etc. which in further leads to increased vascular permeability. The escaping fluid accumulates in the pulp interstitial space but space in the pulp is confined so, pressure within the pulp chamber rises. In severe inflammation, lymphatics are closed resulting in continued increasing fluid and pulp pressure which may result in pulp necrosis.

Effect of Posture on Pulpal Flow

In normal upright posture, there is less pressure effect in the structures of head. On lying down, the gravitational effect disappears; there is sudden increase in pulpal blood pressure and thus corresponding rise in tissue pressure which leads to pain in lying down position.

Another factor contributing to elevated pulp pressure on reclining position is effect of posture on the activity of sympathetic nervous system. When a person is upright, baroreceptors maintain high degree of sympathetic stimulation which leads to slight vasoconstriction. Lying down will reverse the effect leading to increase in blood flow to pulp. In other words, lying down increase blood flow to pulp by removal of both gravitational and baroreceptor effect.

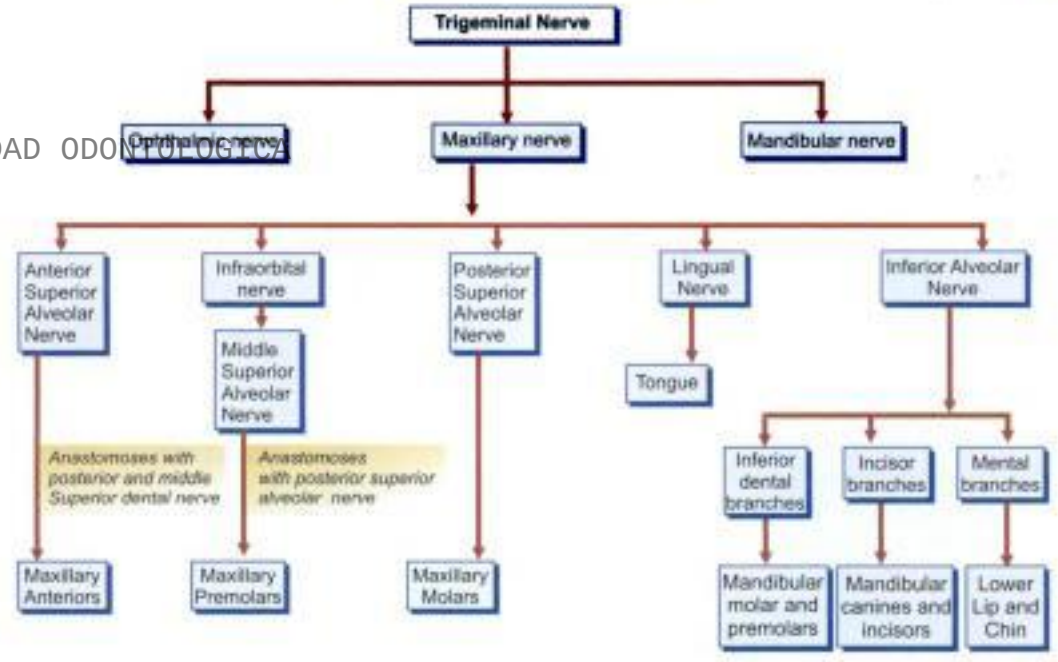
Clinical Correlation

1. ***Temperature changes***
   a. ***Increase of temperature***: A 10°-15°C increase in pulp temperature causes arteriolar dilation and increase in intrapulpal pressure of 2.5 mm Hg/°C but it is transient in nature. The irreversible changes occur when vasodilation is sustained by heating the pulp to 45°C for prolonged periods, resulting in persistent increase in pulp pressure.
   b. ***Decrease in Temperature***: It has been seen that at temperature lower than - 2°C, the pulp tissue exhibit immediate pulpal pathology such as vascular engorgement and necrosis.
2. ***Local anesthetics***: The effect of local anesthetics on pulp vasculature is mainly due to presence of vasoconstrictor in anesthetic solution. For example- presence of epinephrine in local anesthetic cause decrease in blood flow in the pulp which is due to stimulation of α – adrenergic receptors located in pulpal blood vessels.
3. ***General anesthetics***: General anesthetics have shown to produce effect on the velocity of blood flow in the pulp.
4. ***Endodontic therapy***: During endodontic therapy, if only some part of pulp is extirpated, the profuse bleeding occurs whereas there would be less hemorrhage if pulp were extirpated closer to the apex of tooth. This is because of increase diameter of the vessels in the central part of the pulp.
5. ***Aging***: With increasing age, pulp has shown decrease in vascularity, increase in fibrosis, narrowing of diameter of blood vessels, decrease circulation. Finally, the circulation becomes impaired because of atherosclerotic changes and calcifications in the blood vessel leading to cell atrophy and cell death.

INNERVATION OF PULP

Dental pulp is abundantly innervated by both sensory as well as autonomic nerve fibers, (Fig. 2.21). The nerve fibers enter the pulp through apical foramen along with blood vessels. After entering the pulp, the nerve bundles run coronally and divide into smaller branches until a single axons form a dense network near the pulp-dentin margin, termed as plexus of Raschkow. Also the individual axons may branch into numerous terminal filaments which enter the dentinal tubules (Fig. 2.22).

Pain is complex phenomenon which is in form of the evoked potential in the tooth that initiated signals to the brain. Regardless of the nature of sensory stimulus, i.e. mechanical, chemical or thermal, etc. almost all afferent impulses from the pulp result in pain. The dental pulp contains both sensory and motor nerves. The sensory nerves are encased in myelin sheath which is composed largely of fatty substances or lipids and proteins. Myelin appears to be internal proliferation of schwann cells. The unmyelinated fibers are surrounded by single layer of Schwann cells, but the myelin spirals are absent. The unmyelinated nerves are usually found in autonomic nervous system. The nerve fibers are classified according to their diameter, velocity of conduction and function. The fibers having largest diameter are classified as A fibers while those having smallest diameter are classified as C fibers (Fig. 2.23). The A delta fibers are faster conducting and are responsible for localized, sharp dentinal pain. The C fibers are slower conducting fibers and are considered responsible for dull and throbbing



Nerve supply of teeth

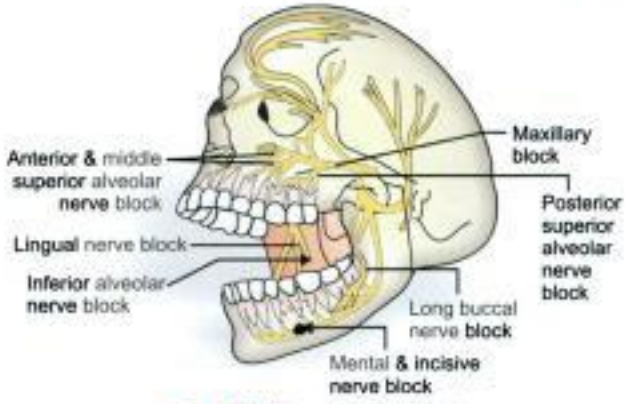


Fig. 2.21: Nerve supply of teeth

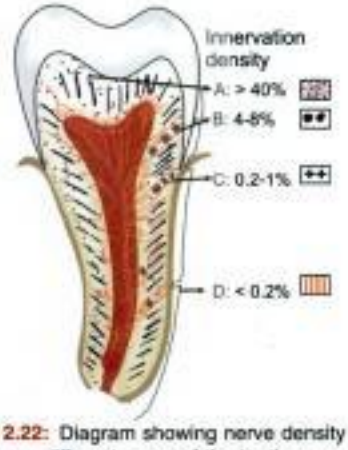


Fig. 2.22: Diagram showing nerve density at different areas of the tooth

Fig. 2.23: Diagram showing nerve fibers of pulp

pain. The pain receptors transmit their message to the central nervous system at different rates depending upon size, diameter and coating of the nerves.

Thermal, chemical or mechanical stimuli stimulate C fibers resulting in dull, poorly localized and throbbing pain.

Electrical pulp tester stimulates A delta fibers first because of their lower threshold. As the intensity of stimulus is increased along with A delta fibers, some of the C fibers also get stimulated resulting in strong unpleasant sensation.

Basic Structure of a Neuron

The basic unit of nervous system is the neuron. The cell membrane of the neuron is composed of bimolecular layer of lipid between two layers of protein (Fig. 2.24). The physiology of nerve conduction is related to changes in cell membrane. The resting potential of the neuron depends on the selective permeability of plasma membrane and sodium pump of the cell. Stimulation of a neuron causes it to depolarize in the region of stimulus and subsequently adjacent areas of cell membrane are also depolarized. This depolarization during passage of excitation along the neuron constitutes the action potential or a nerve impulse. The cell information, in form of action potential is transduced into chemical message. When the nerve is at rest, the Na^+ are more concentrated in extracellular fluid than cytoplasm of the nerve itself and the K^+ ions are more concentrated in cytoplasm rather in extracellular fluid. Because of this, unequal concentration of ion, nerve fiber membrane is polarized. During excitation, there is rapid increase of Na^+ in the cell and K^+ ion flow out to lesser extent. As the impulse moves away, the membrane is recharged by outward movement. Subsequently, Na^+ ion are expelled into extracellular fluid while K^+ pump return K^+ into the intracellular fluid (Fig. 2.25). When the impulse arrives at synaptic terminals, neurotransmitters are released form the synaptic vesicles. These neurotransmitters generate an electrical impulse in the receptors of the dendrite of other neurons.

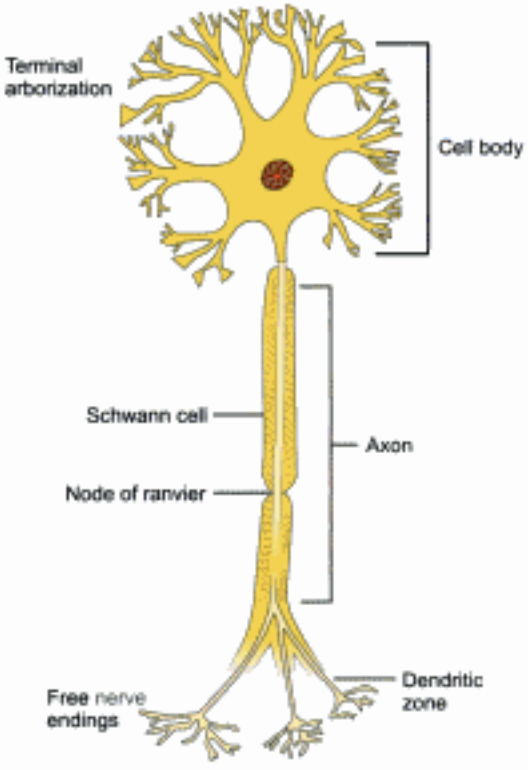


Fig. 2.24: Diagram showing structure of a neuron

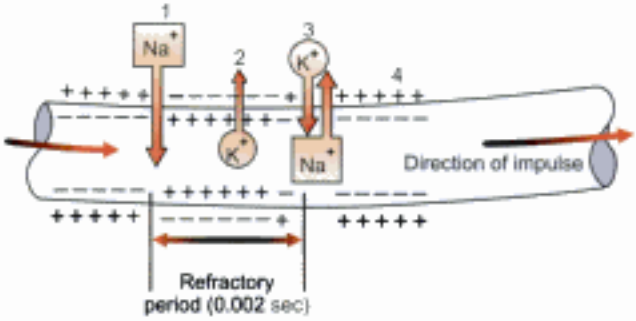


Fig. 2.25: Physiology of nerve conduction

FUNCTION OF PULP

The pulp lives for Dentin and the Dentin lives by the grace of the pulp.

Pulp Performs Four Basic Functions i.e.:
1. Formation of dentin
2. Nutrition of dentin
3. Innervation of tooth
4. Defense of tooth

1. Formation of Dentin

It is primary function of pulp both in sequence and importance. Odontoblasts are differentiated from the dental papilla adjacent to the basement membrane of enamel organ which later deposits dentin. Pulp primarily helps in:
- Synthesis and secretion of organic matrix.
- Initial transport of inorganic components to newly formed matrix.
- Creates an environment favorable for matrix mineralization.

2. Nutrition of Dentin

Nutrients exchange across capillaries into the pulp interstitial fluid, which in turn travels into the dentin through the network of tubules created by the odontoblasts to contain their processes.

3. Innervation of Tooth

Through the nervous system, pulp transmits sensations mediated through enamel or dentin to the higher nerve

centers. Pulp transmits pain, also senses temperature and touch.

Teeth are supplied by the maxillary and mandibular divisions of the trigeminal (V) nerve. The dental nerve divides into multiple branches as it traverses the bone. At the apical alveolar plate, the A delta and C axons enters the periodontal ligament. The nerves enters the apical foramina- unite to from common pulpal nerve- proceeds coronally with afferent blood vessels- divides into cuspal nerves at the coronal portion of the tooth- on a approaching the cell free zone of pulp, a mixture of myelinated and non-myelinated axons branch repeatedly- overlapping network of nerves plexus of Raschkow. The nerve twigs either end among the stroma of the pulp or terminate among the odontoblasts.

4. Defense of Tooth

Odontoblasts form dentin in response to injury particularly when original dentin thickness has been compromised as in caries, attrition, trauma or restorative procedure. Odontoblasts also have the ability to form dentin at sites when dentin continuity has been lost.

The formation of reparative dentin and sclerotic dentin are defense mechanisms of the tooth.

Pulp also has the ability to elicit an inflammatory and immunologic response in an attempt to neutralize or eliminate invasion of dentin by caries causing microorganisms and their by products.

AGE CHANGES IN THE PULP

Pulp like other connective tissues, undergoes changes with time. These changes can be natural or may be result of injury such as caries, trauma or restorative dental procedure. Regardless of the cause, the pulp shows changes in appearance (morphogenic) and in function (physiologic).

MORPHOLOGIC CHANGES

1. Continued deposition of intratubular dentin- reduction in tubule diameter.
2. Reduction in pulp volume due to increase in secondary dentin deposition (Fig. 2.26) - root canal appears very thin or seem to totally obliterated (Fig. 2.27).
3. Presence of dystrophic calcification and pulp stones (Fig. 2.27).
4. Decrease in the number of pulp cells-between 20-70 years. Cells density decreases by 50 percent.
5. Degeneration and loss of myelinated and unmyelinated axons -decrease in sensitivity.
6. Reduction in number of blood vessels- displaying arteriosclerotic changes.

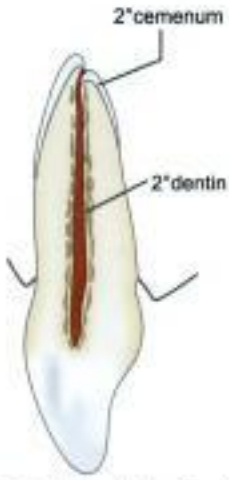


Fig. 2.26: Reduction in size of pulp volume

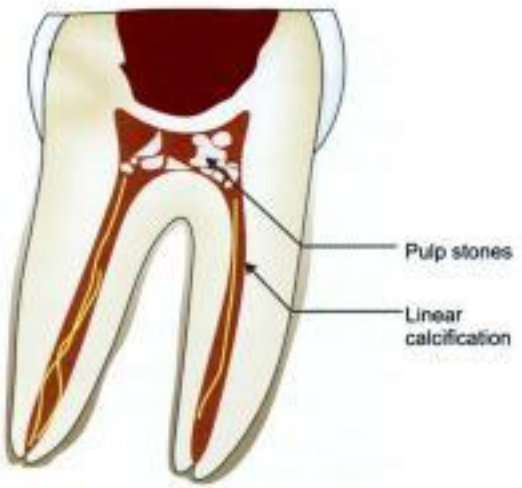


Fig. 2.27: Diagram showing pulp stones and reduced size of pulp cavity

7. Once believed that collagen content with age reduces, but recent studies prove that collagen stabilizes after completion of tooth formation. With age, collagen forms bundle making its presence more apparent.

PHYSIOLOGIC CHANGES

1. Decrease in dentin permeability provides protected environment for pulp- reduced effect of irritants.
2. Possibility of reduced ability of pulp to react to irritants and repair itself.

Pulpal Calcifications/Pulp Stones/Denticles

The larger calcifications are called denticles. It is seen that these stones are present in atleast 50 percent of teeth. These

stones may form either due to some injury or a natural phenomenon (Fig. 2.27).

Sometimes denticles became extremely large, almost obliterating the pulp chamber or the canal.

Pulp stones may be classified (1) according to structure (2) according to size (3) according to location.

Classification of pulp stone

1. According to structure
   a. True
   b. False
2. According to size
   a. Fine
   b. Diffuse
3. According to location
   a. Embedded
   b. Attached
   c. Free

According to Structure

They can be classified into true and false denticles. The difference between two is only morphologic and not chemical.

True Denticle

A true denticle is made up of dentin and is lined by odontoblasts. These are rare and are usually located close to apical foramen. Development of true denticle is caused by inclusions of remnants of epithelial root sheath within the pulp (Fig. 2.28) These epithelial remnants induce the cells of pulp to differentiate into odontoblast which form dentin masses called true pulp stones.

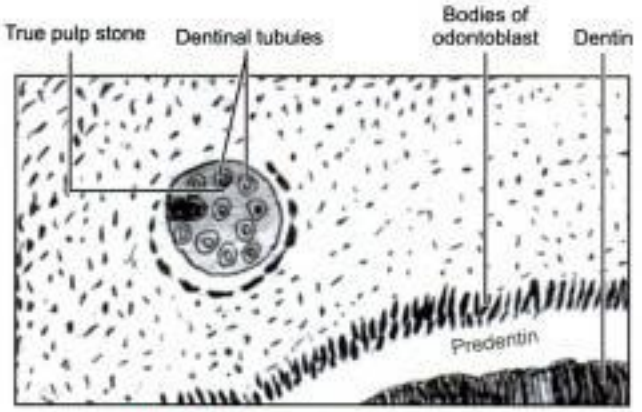


Fig. 2.28: Diagram showing true denticle

False Denticles

Appear as concentric layers of calcified tissue. These appear within bundles of collagen fibers. They may arise around vessels. Calcification of thrombi in blood vessels called, phleboliths, may also serve as nidi for false denticles. All denticles begin as small nodules but increase in size by incremental growth on their surface.

According to Size

According to size, there are fine, diffuse mineralizations, also known as fibrillar mineralizations, and denticles. The former are found more frequently in the root canals, but they may also be present in the coronal portion of the pulp.

According to Location they can be Classified as

- Free
- Attached
- Embedded

Free denticles are entirely surrounded by pulp tissue. ***Attached denticles*** are partially fused dentin (Fig. 2.29).

Embedded denticles are entirely surrounded by dentin calcifications, are seen more in older pulps. This may be due to increase in extent of cross linking between collagen molecules.

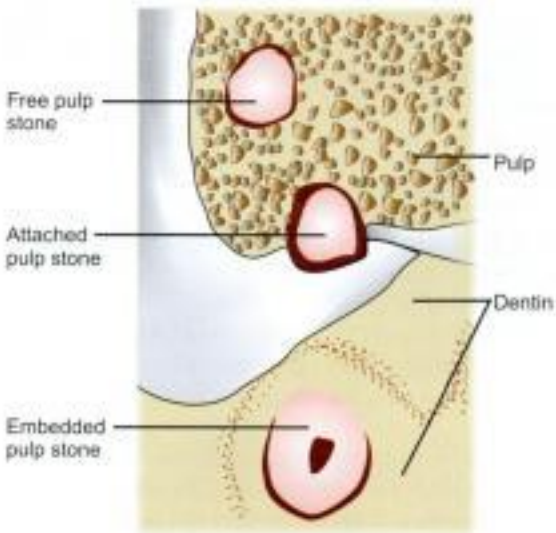


Fig. 2.29: Free, attached and embedded pulp stones

Clinical Significance of Pulp Stones

Presence of pulp stones may alter the internal anatomy of the pulp cavity. Thus, making the access opening of the tooth difficult. They may deflect or engage the tip of endodontic instrument. Since the pulp stone can originate in response to chronic irritation, the pulp chamber which appears to have diffuse and obscure outline may represent large number of irregular pulp stones which may indicate chronic irritation of the pulp.

Calcific Metamorphosis

Calcific metamorphosis is defined as a pulpal response to trauma that is characterized by deposition of hard tissue within the root canal space. It has also been referred to as pulp canal obliteration.

Calcific metamorphosis occurs commonly in young adults because of trauma. It is evident usually in the anterior region of the mouth and can partially or totally obliterate the canal space radiographically.

The ***clinical picture*** of calcific metamorphosis has been described by Patterson and Mitchell as a tooth that is darker in hue than the adjacent teeth and exhibits a dark yellow color because of a decrease in translucency from a greater thickness of dentin under the enamel.

The ***radiographic appearance*** of calcific metamor-phosis is partial or total obliteration of the pulp canal space with a normal periodontal membrane space and intact lamina dura. Complete radiographic obliteration of the root canal space, however, does not necessarily mean the absence of the pulp or canal space; in the majority of these cases there is a pulp canal space with pulpal tissue.

The pulps of 20 maxillary permanent incisors were evaluated microscopically by Lundberg and Cvek. The teeth were treated endodontically because of progressive hard tissue formation in the canal space. The tissue changes were characterized by a varied increase in collagen content and a marked decrease in the number of cells. Osteoid tissue with included cells was found adjacent to mineralized areas in the pulp, with only one pulp showing moderate lymphocytic inflammatory infiltrate because of further trauma. They concluded that tissue changes in the pulps of teeth with calcific metamorphosis do not indicate the necessity for root canal treatment.

The mechanism of hard tissue formation during calcific metamorphosis is characterized by an osteoid tissue that is produced by the odontoblasts at the periphery of the pulp space or can be produced by undifferentiated pulpal cells that undergo differentiation as a result of the traumatic injury. This results in a simultaneous deposition of a dentin-like tissue along the periphery of the pulp space and within the pulp space proper. These tissues can eventually fuse with one another, producing the radiographic appearance of a root canal space that has become rapidly and completely calcified.

The ***management of canals*** with calcific metamorphosis is similar to the management of pulpal spaces with any form of calcification.

To locate the calcified orifice, the one first mentally visualizes and projects the normal spatial relationship of the pulp space onto a radiograph of the calcified tooth. Access preparation is initiated, with the rotary instrument directed toward the presumed location of the pulpal space. This approach requires knowledge of the normal pulp chamber location, tooth canal anatomy, and the long axis of the roots. Accurate radiographs are essential for preoperative visualization and periodic assessment of bur penetration and orientation.

Helpful Considerations

- Irrigate copiously with NaOCl, which enhances dissolution of organic debris, lubricates the canal, and keeps dentin chips and pieces of calcified material in solution.
- Advance instruments slowly in calcified canals.
- Clean the instrument on withdrawal and inspect it before reinserting it into the canal.
- Use chelating pastes or solutions to assist in canal penetration.
- Use ultrasonic instruments in the pulp chamber to loosen debris in the canal orifices.
- Flare the canal orifice in a crown-down fashion and enlarge the negotiated canal space to improve tactile perception in continued canal penetration.

Symptomatic teeth that exhibit complete calcific metamorphosis radiographically or in which the canals cannot be negotiated must be treated with periradicular surgery.

PERIRADICULAR TISSUE

Periradicular tissue consists of cementum, periodontal ligament and alveolar bone.

Cementum

Cementum can be defined as hard, avascular connective tissue that covers the roots of the teeth (Fig. 2.30). It is light

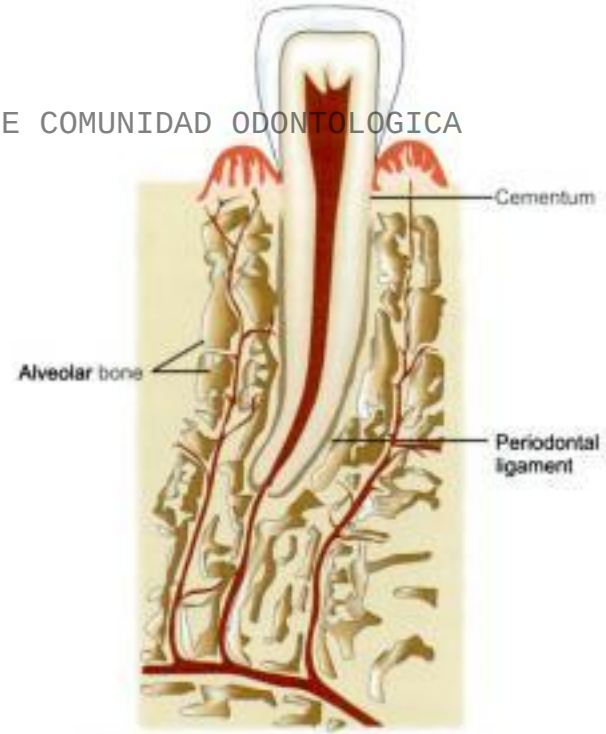


Fig. 2.30: Diagram showing periradicular tissue

yellow in color and can be differentiated from enamel by its lack of luster and darker hue. It is very permeable to dyes and chemical agents, from the pulp canal and the external root surface.

Types

There are two main types of root cementum
1. Acellular (Primary)
2. Cellular (Secondary)

Acellular Cementum

1. Covers the cervical third of the root
2. Formed before the tooth reaches the occlusal plane.
3. As the name indicated, it does not contain cells.
4. Thickness is in the range 30-230 µm.
5. Abundance of sharpey's fibers.
6. Main function is anchorage.

Cellular Cementum

1. Formed after the tooth reaches the occlusal plane.
2. It contains cells.
3. Less calcified than acellular cementum.
4. Sharpey's fibers are present in lesser number as compared to acellular cementum.
5. Mainly found in apical third and interradicular
6. Main function is adaptation.

Periodontal Ligament

Periodontal ligament is a unique structure as it forms a link between the alveolar bone and the cementum. It is continuous with the connective tissue of the gingiva and communicates with the marrow spaces through vascular channels in the bone. Periodontal ligament houses the fibers, cells and other structural elements like blood vessels and nerves.

The Periodontal ligament Comprises of the following Components

I. Periodontal fibers
II. Cells
III. Blood vessels
IV. Nerves

Periodontal Fibers

The most important component of periodontal ligament is principal fibers. These fibers are composed mainly of collagen type I while reticular fibers are collagen type III. The principal fibers are present in six arrangements (Fig. 2.31).

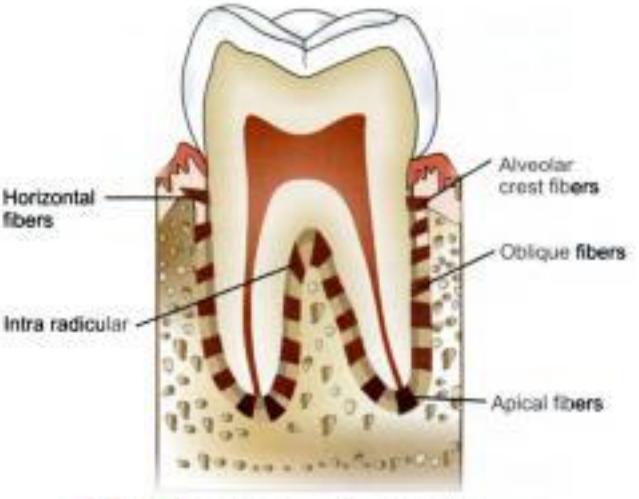


Fig. 2.31: Principal fibers of periodontal ligament

Horizontal Group

These fibers are arranged horizontally emerging form the alveolar bone and attached to the root cementum.

Alveolar Crest Group

These fibers arise from the alveolar crest in fan like manner and attach to the root cementum. These fibers prevent the extrusion of the tooth.

Oblique Fibers

These fibers make the largest group in the periodontal ligament. They extend from cementum to bone obliquely. They bear the occlusal forces and transmit them to alveolar bone.

Transeptal Fibers

These fibers run from the cementum of one tooth to the cementum of another tooth crossing over the alveolar crest.

Apical Fibers

These fibers are present around the root apex.

Interradicular Fibers

Present in furcation areas of multirooted teeth.

Apart from the principal fibers, oxytalan and elastic fibers are also present.

Cells

The cells present in periodontal ligament are

a. Fibroblast
b. Macrophages
c. Mast cells
d. Neutrophils
e. Lymphocytes
f. Plasma cells
g. Epithelial cells rests of Mallassez.

Nerve Fibers

The nerve fibers present in periodontal ligament, is either of myelinated or non-myelinated type.

Blood Vessels

The periodontal ligament receives blood supply form the gingival, alveolar and apical vessels.

Functions

Supportive

It supports the tooth and is suspended in alveolar socket.

Nutritive

This tissue has very rich blood supply. So, it supplies nutrients to adjoining structures such as cementum, bone and gingiva by way of blood vessels. It also provides lymphatic drainage.

Protective

These fibers perform the function of protection absorbing the occlusal forces and transmitting to the underlying alveolar bone.

Formative

The cells of PDL help in formation of surrounding structures such as alveolar bone and cementum.

Resorptive

The resorptive function is also accomplished with the cells like osteoclasts, cementoclasts and fibroblasts provided by periodontal ligament.

Alveolar Bone

Bone is specialized connective tissue which comprises of inorganic phases that is very well designed for its role as load bearing structure of the body.

Cells and Intercellular Matrix

Cells present in bone are:

a. Osteocytes
b. Osteoblasts
c. Osteoclasts

Intercellular Matrix

Bone consists of two third inorganic matter and one third organic matter. Inorganic matter is composed mainly of minerals calcium and phosphate along with hydroxyl apatite, carbonate, citrate etc. while organic matrix is composed mainly of collagen type I (90%).

Bone consists of two plates of compact bone separated by spongy bone in between. In some area there is no spongy bone. The spaces between trabeculae of spongy bone are filled with marrow which consists of hemopoitic tissue in early life and fatty tissue latter in life. Bone is a dynamic tissue continuously forming and resorbing in response to functional needs. Both local as well as hormonal factors play an important role in metabolism of bone. In healthy conditions the crest of alveolar bone lies approximately 2-3 mm apical to the cemento enamel junction but it comes to lie more apically in periodontal diseases. In periapical diseases, it gets resorbed easily.

3 Pathologies of Pulp and Periapex

- Introduction
- Etiology of Pulpal Diseases
- Progression of Pulpal Pathologies
- Diagnostic Aids for Pulpal Pathologies
- Classification of Pulpal Pathologies
- Periradicular Pathologies
- Etiology of Periradicular Diseases
- Diagnosis of Periradicular Pathologies
- Classification of Periradicular Diseases
- Diseases of Periradicular Tissues of Nonodontogenic Origin
- Histopathology of Periapical Response to Various Irritants

INTRODUCTION

Dental pulp consists of vascular connective tissue contained within the rigid dentin walls. It is the principle source of pain within the mouth and also the major site of attention in endodontics and restorative treatment.

Some important features of pulp are as follows (Fig. 3.1):

- Pulp is located deep within the tooth, so ***defies visualization***
- It gives radiographic appearance as ***radiolucent line***
- Pulp is a connective tissue with several factors making it unique and altering its ability to respond to irritation
- Normal pulp is a ***coherent soft tissue***, dependent on its normal hard dentin shell for protection and hence, once exposed, extremely sensitive to contact and to temperature but this pain does not last for more than 1-2 seconds after the stimulus removed
- Since pulp is totally surrounded by a hard tissue, ***dentin*** which ***limits the area for expansion*** and restricts the pulp's ability to tolerate edema (Fig. 3.2).

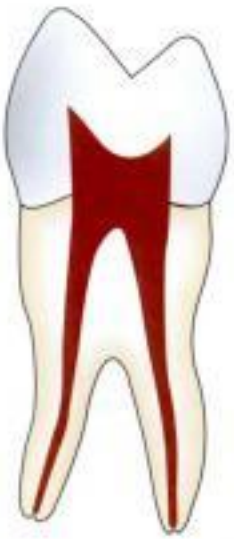


Fig. 3.1: Diagram showing presence of pulp within the tooth

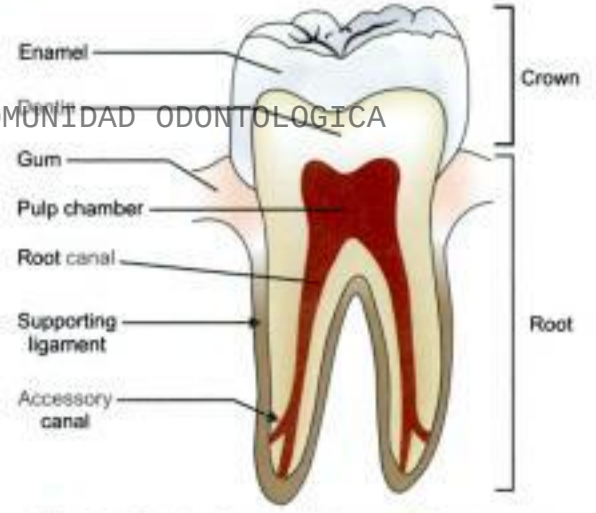


Fig. 3.2: Relation of pulp with its surrounding structures

- The pulp has almost a ***total lack of collateral circulation***, which severely limits its ability to cope with bacteria, necrotic tissue and inflammation
- The pulp ***possess*** unique cells the ***Odontoblasts***, as well as cells that can differentiate into hard-tissue secreting cells that form more dentin and/or irritation dentin in an attempt to protect itself from injury (Fig. 3.3)
- In spite of these circumstances, injured pulp has some capacity to recover, but the degree to which is uncertain
- Pulpal responses are unpredictable. "Some pulps die if you look at them cross eyes, while others won't die even if you hit them with an axe"
- Correlation of clinical signs and symptoms with corresponding specific histological picture is often difficult
- Thus the knowledge to pulp is essential not only for providing dental treatment, but also to know the rationale behind the treatment provided.

After all, "This little tissue has created big issue".

Fig. 3.3: Formation of irritation dentin

ETIOLOGY OF PULPAL DISEASES

I. Etiology of pulpal diseases can be broadly classified into:
   1. Physical
      - Mechanical
      - Thermal
      - Electrical
   2. Chemical
   3. Bacterial
   4. Radiation

II. *WEIN* classifies the causes of pulpal inflammation, necrosis or dystrophy in a logical sequence beginning with the most frequent irritant, microorganisms.
   1. ***Bacterial***

      Bacterial irritants: In 1891 WD Miller—bacteria were a possible cause of pulpal inflammation (Fig. 3.4). Most common cause for pulpal injury-bacteria or their products may enter pulp through a break in dentin either from:
      - Caries
      - Accidental exposure
      - Fracture
      - Percolation around a restoration
      - Extension of infection from gingival sulcus
      - Periodontal pocket and abscess (Fig. 3.5)
      - Anachoresis (Process by which microorganisms get carried by the bloodstream from another source localize on inflamed tissue).

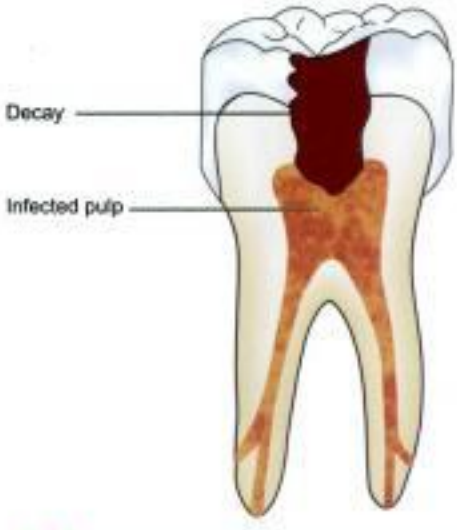


Fig. 3.4: Tooth decay causing pulpal inflammation

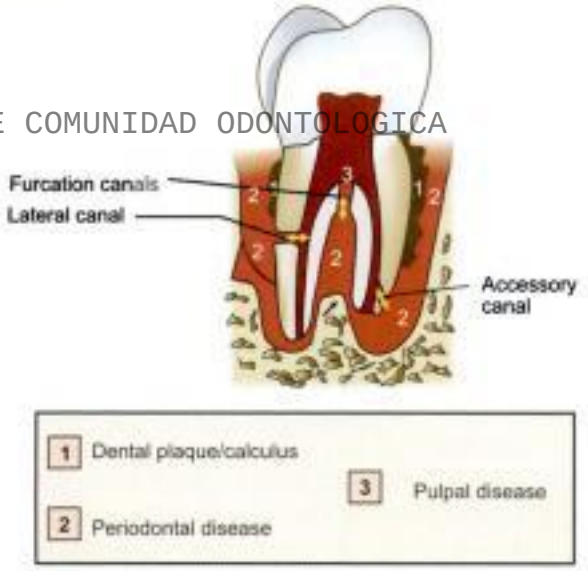


Fig. 3.5: Periodontal disease causing pulpal inflammation

Bacteria most often recovered from infected vital pulps are:

- Streptococci
- Staphylococci
- Diphtheroids, etc.

2. ***Traumatic***
   Acute trauma like fracture, luxation or avulsion of tooth (Fig. 3.6). Chronic trauma including para-functional habits like bruxism.
3. ***Iatrogenic*** (Pulp inflammation for which the dentists own procedures are responsible is designated as "Dentistogenic pulpitis.") *Various iatrogenic causes of pulpal damage can be*:
   a. *Thermal changes* generated by cutting procedures, during restorative procedures, bleaching of enamel, electrosurgical procedures, laser beam etc. can cause severe damage to the pulp if not controlled.
   b. Orthodontic movement
   c. Periodontal curettage
   d. Periapical curettage
   A use of chemicals like temporary and permanent fillings, liners and bases and use of cavity desiccants such as alcohol.
4. ***Idiopathic***
   a. Aging
   b. Resorption internal or external (Fig. 3.7)

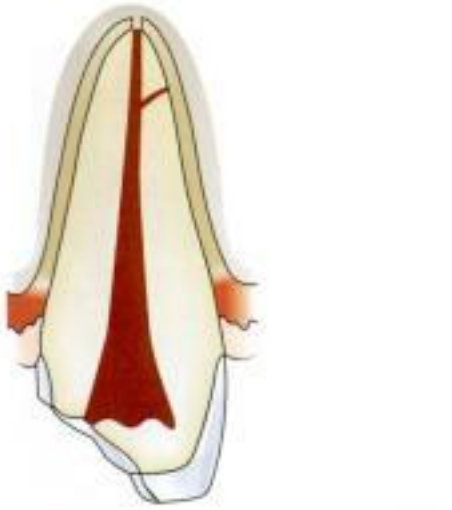


Fig. 3.6: Fracture of tooth can also cause pulpal inflammation

Fig. 3.7: Resorption of tooth

RADIATION INJURY TO PULP

Radiation damage to teeth depends on dose, source, type of radiation, exposure factor and stage of tooth development at the time of irradiation.

Radiation therapy affect pulps of fully formed teeth in patients exposed to radiation therapy. The pulp cells exposed to ionizing radiation may become necrotic, there may occur vascular damage and the interference on mitosis of cells. Also radiations affect the salivary glands causing decreased salivary flow, thereby increased disposition to dental caries and pulp involvement.

Laser beam causes cavitations by production of immense heat. Radium is generally taken from the public water supplies by teeth. Early lesions of radium intoxication include osteitis, osteomyelitis and loss of teeth. Delayed dental effects consist of unusual carious lesions, spontaneous tooth fracture and tooth resorption.

PROGRESSION OF PULPAL PATHOLOGIES

Pulp reacts to above mentioned irritants as do other connective tissues. Degree of inflammation is proportional to intensity and severity of tissue damage. For example slight irritation like incipient caries or shallow cavity preparation cause little or no pulpal inflammation, whereas extensive operative procedures may lead to severe pulpal inflammation.

Depending on condition of pulp, severity and duration of irritant, host response, pulp may respond from mild inflammation to pulp necrosis (Fig. 3.8).

These changes may not be accompanied by pain and thus may proceed unnoticed.

Microbial irritation is the main source of irritation of the pulp (Fig. 3.9).

Carious enamel and dentin contains numerous bacteria
↓
Bacteria decrease in deeper layers of carious dentin
↓
Pulp is affected before actual invasion by bacteria via their toxic byproducts
↓
Byproducts cause local chronic cell infiltration
↓
When actual pulp exposure occurs pulp tissue gets locally infiltrated by PMN's to form an area of liquefaction necrosis at the site of exposure
↓
Eventually necrosis spreads all across the pulp and periapical tissue resulting in severe inflammatory lesion.

Fig. 3.8: Response of pulp to various irritants

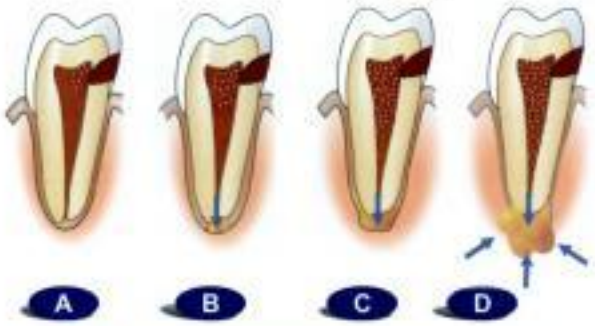


Fig. 3.9: Gradual response of pulp to microbial invasion

Degree and nature of inflammatory response caused by microbial irritants depends upon
1. Host resistance
2. Virulence of microorganisms
3. Duration of the agent
4. Lymph drainage
5. Amount of circulation in the affected area
6. Opportunity of release of inflammatory fluids

DIAGNOSTIC AIDS FOR PULPAL PATHOLOGY

- Subjective symptoms—most common being pain
- Objective symptoms
  1. Visual and tactile inspection – 3Cs –
     i. Color
     ii. Contour
     iii. Consistency
  2. Thermal tests
     i. *Heat tests* – isolation of tooth – use of:
        - Warm air
        - Hot water
        - Hot Burnisher
        - Hot Gutta Percha stick
     ii. *Cold tests*:
        - Ethyl chloride spray
        - Ice pencils
        - CO_2 snow – temperature 18 °C
  3. Electrical pulp testing
  4. Radiographs
  5. Anesthetic tests
  6. Test cavity

Recent advances in diagnostic aids for pulpal pathology include:
1. Laser Doppler flowmetry
2. Liquid crystal testing
3. Huges probeye camera
4. Infrared thermography
5. Thermo couples

6. Pulp-oximetry
7. Dual wavelength spectrophotometry
8. Plethysmography
9. Xenon – 133 radio isotopes

CLASSIFICATION OF PULPAL PATHOLOGIES

- ***Baume's classification***: Based on clinical symptoms
  1. Asymptomatic, vital pulp which has been injured or involved by deep caries for which pulp capping may be done.
  2. Pulps with history of pain which are amenable to pharmacotherapy.
  3. Pulps indicated for extirpation and immediate root filling.
  4. Necrosed pulps involving infection of radicular dentin accessible to antiseptic root canal therapy.
- ***Seltzer and Bender's classification***: Based on clinical tests and histological diagnosis.
  1. *Treatable*
     a. Intact uninflamed pulp
     b. Transition stage
     c. Atrophic pulp
     d. Acute pulpitis
     e. Chronic partial pulpitis without necrosis
  2. *Untreatable*
     a. Chronic partial pulpitis with necrosis
     b. Chronic total pulpitis
     c. Total pulp necrosis
- ***Grossman's clinical classification***
  1. *Pulpitis*
     a. Reversible
        – Symptomatic (Acute)
        – Asymptomatic (Chronic)
     b. Irreversible pulpitis
        i. Acute
           a. Abnormally responsive to cold
           b. Abnormally responsive to heat
        ii. Chronic
           a. Asymptomatic with pulp exposure
           b. Hyperplastic pulpitis
           c. Internal resorption
  2. *Pulp degeneration*
     a. Calcific (Radiographic diagnosis)
     b. Other (Histopathological diagnosis)
  3. *Necrosis*

A normal pulp gives moderate response to pulp test and this response subsides when the stimulus is removed. The tooth is free of spontaneous pain. Radiograph shows an intact lamina dura, absence of any pulpal abnormality, calcifications, and resorption (Fig. 3.10).

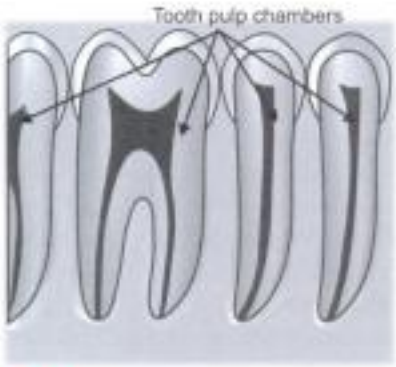


Fig. 3.10: Radiographic picture of normal teeth

Pulpitis is inflammation of the dental pulp resulting from untreated caries, trauma, or multiple restorations. Its principal symptom is pain. Diagnosis is based on clinical finding and in confirmed by X-ray. Treatment involves removing decay, restoring the damaged tooth, and, sometimes, performing root canal therapy or extracting the tooth.

Pulpitis can occur when caries progresses deeply into the dentin, when a tooth requires multiple invasive procedures, or when trauma disrupts the lymphatic and blood supply to the pulp. It starts as a reversible condition in which the tooth can be saved by a simple filling. If untreated, it progresses as swelling inside the rigid encasement of the dentin compromising the circulation, making the pulp necrotic, which predisposes to infection.

Infectious sequelae of pulpitis include apical periodontitis, periapical abscess cellulites, and osteomyelitis of the jaw (Fig. 3.11). Spread from maxillary teeth may cause purulent sinusitis, meningitis, brain abscess, orbital cellulitis,

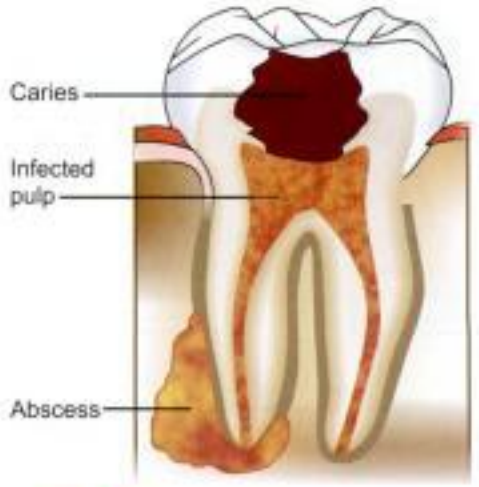


Fig. 3.11: Infectious sequelae of pulpitis

and cavernous sinus thrombosis. Spread from mandibular teeth may cause Ludwig's angina, parapharyngeal abscess, mediastinitis, pericarditis and empyema (Fig. 3.12).

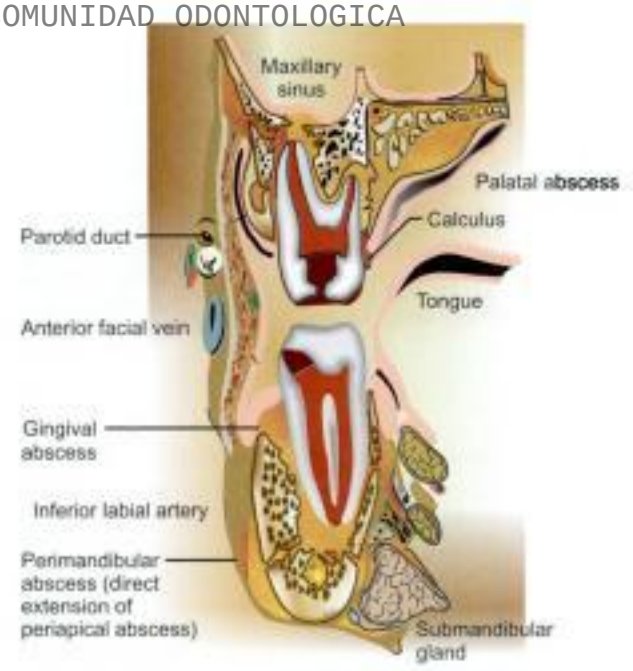


Fig. 3.12: Spread of pulpal inflammation to surrounding tissues

REVERSIBLE PULPITIS/HYPEREMIA/ HYPERACTIVE PULPALGIA

This is the first stage where the pulp is symptomatic. There is a sharp hypersensitive response to cold, but the pain subsides when stimulus is removed. The patient may describe symptoms of momentary pain and is unable to locate the source of pain. This stage can last for month or years.

Definition

"Reversible pulpitis is the general category which histologically may represent a range of responses varying from dentin hypersensitivity without concomitant inflammatory response to an early phase of inflammation."

It is an indication of peripheral A delta fiber stimulation. Determination of reversibility is the clinical judgment which is influenced by history of patient and clinical evaluation.

Etiology

Pulpal irritation to external stimuli is related to dentin permeability. Under normal circumstances, enamel and cementum act as impermeable barrier to block the patency of dentinal tubules at dentinoenamel junction or dentinocemental junction.

When caries and operative procedures interrupt this natural barrier, dentinal tubules become permeable. So inflammation can be caused by any agent which is capable of injuring pulp. It can be:

- Trauma
  - accident or occlusal Trauma
- Thermal injury
  - While preparing cavity
  - Overheating during polishing a filling
- Chemical stimulus—Like sweet or sour foodstuff
- Following insertion of a deep restoration (Fig. 3.13).

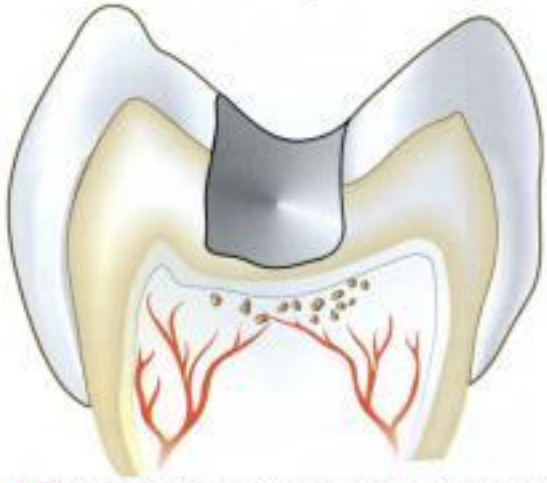


Fig. 3.13: Insertion of deep restoration causing pulp inflammation

Symptoms

Symptomatic reversible pulpitis is character by sharp pain lasting for a moment, commonly caused by cold stimuli. Pain doesn't occur spontaneously and doesn't continue when irritant is removed.

Asymptomatic reversible pulpitis may result from incipient caries and is resolved on removal of caries and proper restoration of tooth.

Histopathology

Reversible pulpitis may range from hyperemia to mild to moderate inflammatory changes limited to area of involved dentinal tubules. It shows—

1. Increased blood volume of pulp associated with increased intrapulpal pressure.
2. Edema of tissue.
3. White cell infiltration.
4. Reparative dentin formation.

Diagnosis

1. ***Pain***: It is sharp but of brief duration, ceasing when irritant is removed.
2. ***Visual examination and history***: may show caries, traumatic occlusion and undetected fracture.
3. ***Radiographs***: Show normal PDL and lamina dura.
   - Depth of caries or cavity penetration may be evident.
4. ***Percussion test***: Shows negative responses i.e. tooth is not tender to percussion.
5. ***Vitality test***: Pulp responds readily to cold stimuli. Electric pulp tester requires less current to cause pain.

Treatment

No endodontic treatment is needed for this condition. The best treatment of this condition is prevention.

Usually a sedative dressing will suffice, followed by permanent restoration when symptoms have completely subsided. Periodic care to prevent caries, desensitization of hypersensitive teeth and use of cavity varnish or base before insertion of restoration is recommended.

When reversible pulpitis is present, removal of noxious stimuli is done. If irritation of pulp continues, moderate to severe inflammation may develop into irreversible pulpitis and eventually pulpal necrosis.

Threshold to Pain Decreases in Reversible Pulpitis. It may be Attributed to –

- Release of mediators (endogenous alogenic agents) which initiate or lower the threshold of excitability.
- Neuropeptides released from unmyelinated C fibers mediate neurogenic inflammation which results in hyper excitability of nerve endings.

IRREVERSIBLE PULPITIS

Definition

"It is a persistent inflammatory condition of the pulp, symptomatic or asymptomatic, caused by a noxious stimulus". It has both acute and chronic stages in pulp.

Etiology

- Most common cause of pulpitis is bacterial involvement of pulp through caries
- Chemical, thermal, mechanical injuries of pulp may induce pulp inflammation
- Reversible pulpitis when left untreated deteriorates into irreversible pulpitis.

Symptoms

- A rapid onset of pain, which can be caused by sudden temperature change, sweet or acidic food. Pain remains even after removal of stimulus
- Pain can be spontaneous in nature which is sharp, piercing, intermittent or continuous in nature
- Pain exacerbated on bending down or lying down due to change in intrapulpal pressure
- Presence of referred pain
- In later stages, pain is severe, boring, throbbing in nature which increases with hot stimulus. Pain is so severe that it keeps the patient awake in night. The relief of pain can be simply done by use of cold water. The patient may report dental office with jar of ice water.

Diagnosis

1. ***Visual examination and history***: Examination of involved tooth may reveal previous symptoms. On inspection, one may see deep cavity involving pulp (Fig. 3.14) or secondary caries under restorations (Fig. 3.15).

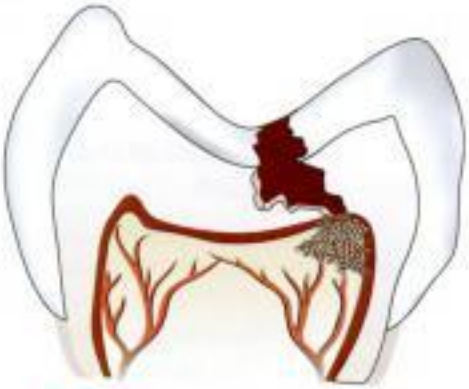


Fig. 3.14: Tooth decay causing pulpitis

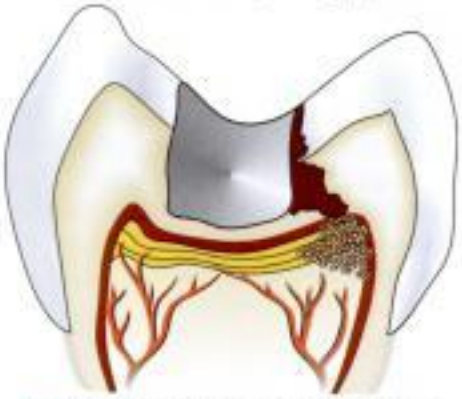


Fig. 3.15: Secondary caries under restoration

2. ***Radiographic findings***:
   - May show depth and extent of caries.
   - Periapical area shows normal appearance but a slight widening may be evident in advanced stages of pulpitis.

3. ***Percussion***: Tooth is tender on percussion (due to increased intrapulpal pressure as a result of exudative inflammatory tissue) (Fig. 3.16).

Differences between Transudate and Exudate

Feature	*Transudate*	*Exudate*
1. Definition	Filtrate of blood plasma but no changes in endothelial permeability.	Edema of inflamed tissue with increased vascular permeability
2. Character	Non-inflammatory - edema	Inflammatory edema
3. pH	Greater than 7.3	Less than 7.3
4. Specific Gravity	< 1.015	> 1.018
5. Protein Content	• Less than 3g/dl (low) • No tendency to coagulate	• More than 3g/dl (high) • Tendency to coagulate
6. Cells	Few cells	Many inflammatory cells

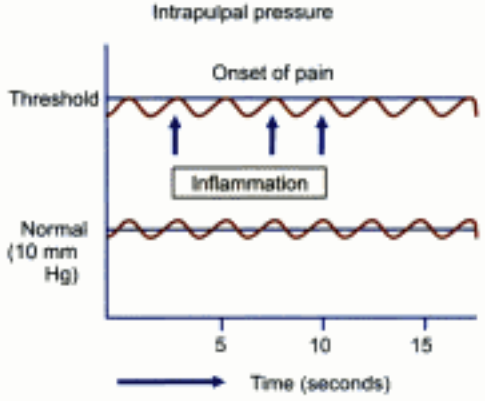


Fig. 3.16: Increased intrapulpal pressure causing pulpal pain

4. ***Vitality tests***:
   i. *Thermal test*: Hyperalgesic pulp responds more readily to cold stimulation than for normal tooth, pain may persist even after removal of irritant.
   As the pulpal inflammation progresses, heat intensifies the response because it has expansible effect on blood vessels. Cold tends to relieve pain because of its contractile effect on vessels, reducing the intrapulpal pressure.
   ii. *Electric test*: Less current is required in initial stages. As tissue becomes more necrotic, more current is required.

Differential diagnosis of reversible and irreversible pulpitis

Features	*Reversible pulpitis*	*Irreversible pulpitis*
1. Pain type	Sharp and fleeting pain, usually dissipates after stimulus is removed	Intense, continuous and prolonged pain due to pressure of secondary irritants
2. Stimulus	External stimulus for example—heat, cold, sugar	• No external stimulus • Dead or injured pulp tissue acts as secondary stimulant.
3. Pain at Night/ Postural	No	Yes
4. Pain Localization	Only with applied cold stimulus or PDL inflammation	Only with applied heat stimulus or PDL inflammation.
5. Referred Pain	Not usually found	Common finding
6. History	• Any History of recent dental procedure done • Sometimes cervical erosion/abrasion	History of: • Deep carries • Trauma • Extensive restoration
7. Percussion/ Occlusion	If due to occlusion, percussion test is positive, otherwise normal.	If inflamed, involved PDL-percussion test is positive otherwise normal
8. Pulp Tests		
(a) EPT	Normal response	Normal to elevated response
(b) Cold	Exaggerated response	Pain relieved by cold occasionally
(c) Heat	Normal-exaggerated response	Acute Pain
9. Color change	No	Yes
10. Radiograph	Caries, defective or unbased restoration	Caries, defective restorations, PDL space enlargement
11. Treatment	Removal of decay, repair of defect, restoration, ZOE dressing, occlusal adjustment	Pulpectomy (single root), Pulpotomy (multiple roots), occlusal adjustment

Treatment

Pulpectomy, i.e. root canal treatment.

A clinical guide as given by *Carrotte in 2003* to determine the status of dental pulp in irreversible pulpitis.

- A history of spontaneous bouts of pain which may last from a few seconds to several hours
- Hot and cold fluids exacerbating the pain. In the latter stages, heat will be more significant and cold will relieve the pain
- Pain radiating initially but once the periodontal ligament has become involved; the pain will be more localized by the patient
- The tooth may become tender to percussion once the inflammation has spread to the periodontal ligament
- A radiographically visible widening of the periodontal ligament may be seen.

CHRONIC PULPITIS

It is an inflammatory response of pulpal connective tissue to an irritant. Here pain is absent because of diminished exudative inflammatory activity and corresponding decrease in intrapulpal pressure to a point below threshold limits of pain receptors.

It can be of three types:

a. Ulcerative/open form
b. Hyperplastic form
c. Closed form of chronic pulpitis.

a. ***Ulcerative form*** is a chronic inflammation of cariously exposed pulp characterized by formation of an abscess at point of exposure (Fig. 3.17). Abscess is surrounded by granulomatous tissue. This condition is also known as pulpal granuloma.

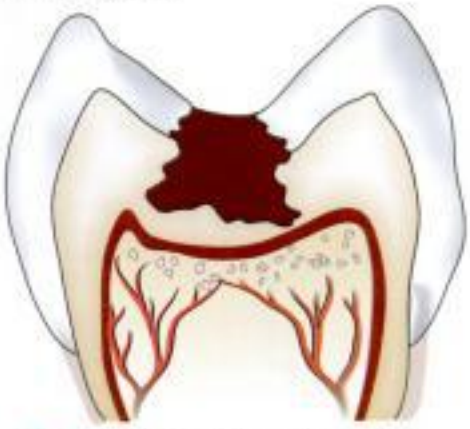


Fig. 3.17: Chronic inflammation of pulpitis resulting from tooth decay

b. ***Hyperplastic form*** is overgrowth of granulomatous tissue into carious cavity (Fig. 3.18).

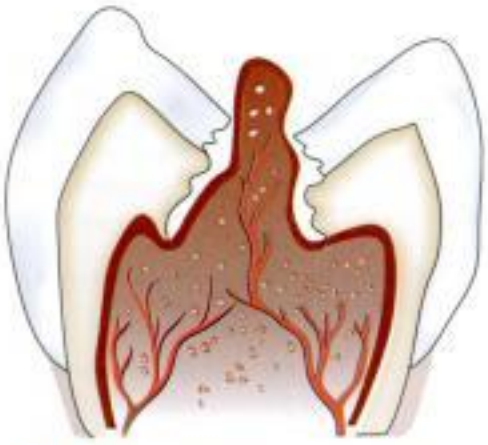


Fig. 3.18: Hyperplastic form of chronic pulpitis

c. ***Closed form of chronic pulpitis*** may occur from operative procedures, excessive orthodontic forces, trauma or periodontal lesions. Here carious lesion is absent.

Etiology

Etiology is same as that of irreversible pulpitis. It is normally caused by slow and progressive carious exposure of pulp. Nature of pulpal response depends on strength and duration of irritant, previous health of pulp and extent of tissue affected.

Signs and Symptoms

- Pain is absent because of low activity of exudative forces. Here proliferative granulomatous forces dominate
- Symptoms develop only when there is interference with drainage of exudate
- Hyperplastic form of chronic pulpitis is seen in teeth of children and adolescents in which pulp tissue has high resistance and large carious lesion permit free proliferation of hyperplastic tissue (Fig. 3.18). Since it contains few nerve fibers, it is non-painful but bleeds easily due to rich network of blood vessels.

Histopathology

- Formation of sclerotic and irritation dentin
- Minimal amount of vasodilation and infiltration of cell initially but when pulp is finally exposed, vasodilation and cellular infiltration increases
- Surface of pulp polyp is usually covered by stratified squamous epithelium which may be derived from gingiva, desquamated epithelial cells of mucosa and tongue.

Diagnosis

- ***Pain***: It is usually absent.
- Hyperplastic form shows a fleshy, reddish pulpal mass which fills most of pulp chamber or cavity. It is less sensitive than normal pulp but bleeds easily when probed.
- ***Radiographic changes*** show
  - Chronic apical periodontitis in long standing cases.
  - In young, patients low grade long standing irritation stimulates periapical bone deposition, i.e. condensing osteitis. Radiograph shows areas of dense bone around apices of involved teeth.

- ***Vitality Tests***
  - Tooth may respond feebly or not at all to thermal test, unless one uses extreme cold
  - More current than normal is required to elicit response by electric pulp tester.
- ***Differential Diagnosis***
  - Hyperplastic pulpitis should be differentiated from proliferating gingival tissue. It is done by raising and tracing the stalk of tissue back to its origin, i.e. pulp chamber.

Treatment

- Complete removal of pulp followed by its restoration should be the goal of treatment. In case of hyperplastic pulpitis, removal of polypoid tissue with periodontal curette or spoon excavator followed by extirpation of pulp should be done
- If tooth is in non-restorable stage, it should be extracted.

INTERNAL RESORPTION

Internal resorption is initiated within the pulp cavity and results in loss of substance from dentinal tissue (Fig. 3.19).

Fig. 3.19: Internal resorption of tooth

Etiology

Exact etiology is unknown.

Patient often presents with history of trauma or persistent chronic pulpitis, or history of pulpotomy.

Mechanism of Resorption

Pulp inflammation due to infection
↓
alteration or loss of predentine and odontoblastic layer
↓
undifferentiated mesenchymal cells come in contact with mineralized dentin
↓
differentiate into dentinoclasts
↓
resorption results

Symptoms

- Usually asymptomatic, recognized clinically through routine radiograph
- Pain occurs in cases of perforation of crown (Fig. 3.20)
- 'Pink Tooth' is the pathognomic feature of internal root resorption.

Fig. 3.20: Internal resorption of tooth causing perforation of root

Diagnosis

- ***Clinically*** - "Pink Tooth" appearance
- ***Radiographic changes***:
  - Radiolucent enlargement of pulp canal
  - Original root canal outline distorted
  - Bone changes are seen only when root perforation into periodontal ligament takes place.
- ***Pulp tests*** - positive, though coronal portion of pulp is necrotic, apical pulp could be vital.

Treatment

Pulp extirpation, stops internal root resorption.

- Surgically treatment is indicated if conventional treatment fails.

PULP NECROSIS

Pulp necrosis or death is a condition following untreated pulpitis. The pulpal tissue becomes dead and if the condition is not treated, noxious materials will leak from pulp space forming the lesion of endodontic origin (Fig. 3.21).

Necrosis may be partial or total, depending on extent of pulp tissue involvement.

The pulp necrosis is of ***two types***:

- ***Coagulation necrosis***: In coagulation necrosis protoplasm of all cells becomes fixed and opaque. Cell mass is recognizable histologically, intracellular details lost.

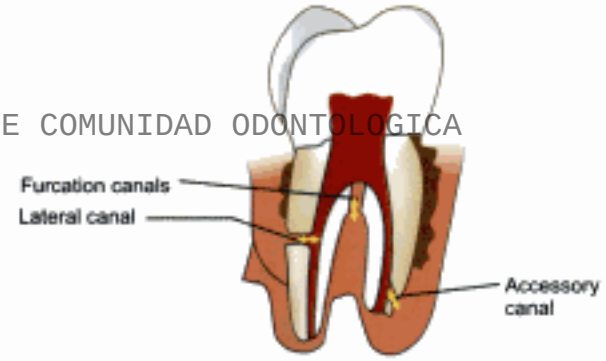


Fig. 3.21: Spread of pulpal inflammation into periodontium

- ***Liquefaction necrosis***: In liquefaction necrosis the entire cell outline is lost. The liquefied area is surrounded by dense zone of PMNL (dead or drying), chronic inflammatory cells.

Etiology

Necrosis is caused by noxious insult and injuries to pulp by bacteria, trauma, and chemical irritation.

Symptoms

- Discoloration of tooth—First indication of pulp death
- History from patient
- Tooth might be asymptomatic.

Diagnosis

1. ***Pain***: It is absent in complete necrosis.
2. ***History of patient*** reveals past trauma or past history of severe pain which may last for some time followed by complete and sudden cessation of pain.
3. ***Radiographic changes***: Radiograph shows a large cavity or filling or normal appearance unless there is concomitant apical periodontitis or condensing osteitis.
4. ***Vitality test***: Tooth is nonresponding to vitality tests. But multirooted teeth may show mixed response because only one canal may have necrotic tissue.

   Sometimes teeth with liquefaction necrosis may show positive response to electric test when electric current is conducted through moisture present in a root canal.
5. ***Visual examination***: Tooth shows color change like dull or opaque appearance due to lack of normal translucency.
6. ***Histopathology***: Necrotic pulp tissue, cellular debris and microorganisms are seen in pulp cavity. If there is concomitant periodontal involvement, there may be presence of slight evidence of inflammation.

Treatment

Complete removal of pulp followed by restoration or extraction of nonrestorable tooth.

PULP DEGENERATION

Pulp degeneration is generally present in older people. It may be the result of persistent mild irritation in the teeth of younger people. Usually pulp degeneration is induced by attrition, abrasion, erosion, bacteria, operative procedures, caries, pulp capping and reversible pulpitis.

It may occur in following forms:

1. ***Atrophic degeneration and fibrosis***
   - It is wasting away or decrease in size which occurs slowly as tooth grows old (Fig. 3.22). There is gradual shift in ratio and quality of tissue elements. In this condition nature collagen fibers / unit area increased leading to fibrosis. Number of pulp cells and size of cells decreased so cells appear as "shrunken solid particles in a sea of dense fibres"
   - Fibroblastic process are lost, cells have round and pyknotic nuclei
   - Dentinoblasts decrease in length, appear cuboidol or flattened.

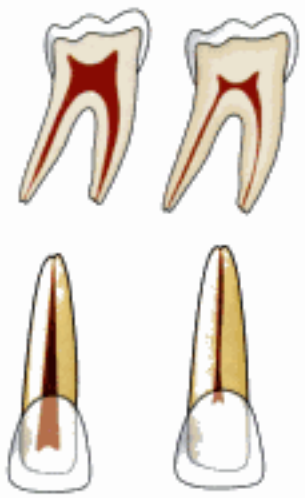


Fig. 3.22: Atrophic changes of pulp with age

2. ***Calcifications***

   In calcific degeneration, part of the pulp tissue is replaced by calcific material (Fig. 3.23). Mainly three types of calcifications are seen in pulp:
   - Dystrophic calcification

Fig. 3.23: Calcifications present in pulp

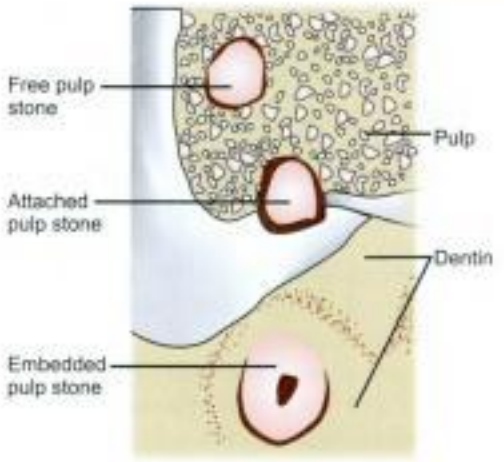


Fig. 3.24: Types of pulp stones

- Diffuse calcification
- Denticles/pulp stones.

Dystrophic Calcifications

They occur by deposition of calcium salts in dead or degenerated tissue. The local alkalinity of destroyed tissues attracts the salts. They occur in minute areas of young pulp affected by minor circulatory disturbances, in blood clot or around a single degenerated cell. It can also begin in the connective tissue walls of blood vessels and nerves and follow their course.

Diffuse Calcifications

They are generally observed in root canals. The deposits become long, thin and fibrillar on fusing.

Denticles / Pulp Stone

These are usually seen in pulp chamber.

Classification

According to location (Fig. 3.24)

- Free
- Embedded
- Attached

According to structure:

- True
- False

True denticles: It is composed of dentin formed from detached odontoblasts or fragments of Hertwig's enamel root sheath which stimulate and undifferentiated cells to assume dentinoblastic activity.

False denticles: Here degenerated tissue structures act as nidus for deposition of concentric layers of calcified tissues.

PERIRADICULAR PATHOLOGIES

Periradicular tissue contains apical root cementum, periodontal ligament and alveolar bone (Fig. 3.25).

Apical periodontium consists of cellular and extracellular components. Fibroblasts, cementoblasts, osteoblasts, undifferentiated mesenchymal cells, epithelial cells rests of malassez, blood vessels, lymphatics, sensory and motor nerve fibres form its components.

Alveolar bone proper lines the alveolus. It consists of:

- *Bundle bone*: Peripheral bone
- *Lamellated*: Centre of alveolar process

Lamina Dura is radiographic image of alveolar bone proper.

Cementum: Two types of collagen fibres are present in cementum.

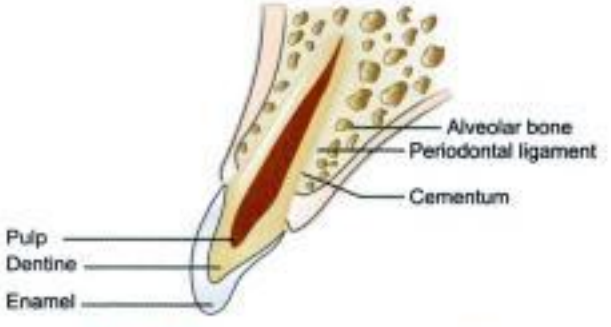


Fig. 3.25: Diagram showing periodontium

a. *Matrix fibres* – parallel to root surface; inter woven, mainly consists of cementoblasts.
b. *Sharpey's fibres* – fibroblast are the main component of Sharpey's fibres

ETIOLOGY OF PERIRADICULAR DISEASES

Bacterial

- Untreated pulpal infection leads to total pulp necrosis. If left untreated, irritants leak into periapical region forming periapex pathologies (Fig. 3.26). Severity of periapical inflammation is related to microorganisms in root canals and the length of exposure to infecting microorganisms. Anachoresis also accounts for microbial infection in teeth. Microorganisms may invade pulp from periodontal pocket and accessory canals leading to development of lesion of endodontic origin
- Root canal is unique, stringent ecological niche for bacterial growth because of lack of oxygen. For bacteria present here the primary nutrient sources is host tissues and tissue fluids.
- Microorganisms in chronically infected root canals are mainly anaerobic and gram-negative type. Most common microorganisms are:
  - *Streptococcus*
  - *Peptostreptococcus*
  - *Provotella*
  - *Porphyromonas*

    } *Black pigmented microorganisms*
  - *Enterococcus*
  - *Campylobacter*
  - *Fusobacterium*
  - *Eubacterium*
  - *Propionibacterium*

Trauma

- Physical trauma to tooth, or operative procedures which results in dental follicle desiccation or significant heat transfer cause sufficient damage to pulp and its blood supply. It results in inflammation with immediate response involving the production of endogenous inflammatory mediators which cause increase in vascular permeability, stasis and leukocyte infiltration
- In cases of severe trauma to tooth resulting in immediate interruption of blood supply, pulp becomes necrotic but is not infected
- Persistent periapical tissue compression from traumatic occlusion leads to apical inflammatory response.

Factors Related to Root Canal Procedures

Several complications can arise from improper endodontic technique which can cause periapical diseases:

1. It is impossible to extirpate pulp without initiating an inflammatory response because a wound is created.
2. Using strong or excessive amounts of intracanal medicaments between appointments may induce periapical inflammation.
3. Improper manipulation of instruments within root canal or over instrumentation can force dentinal debris, irrigating solution and toxic components of necrotic tissue in the periapex (Fig. 3.27).

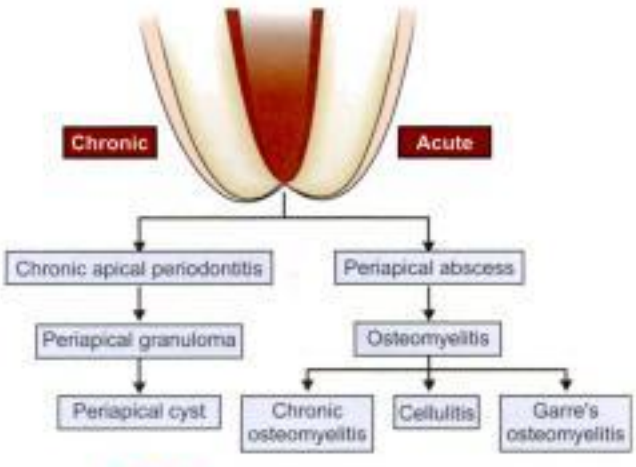


Fig. 3.26: Sequelae of pulpal inflammation

Fig. 3.27: Instrument should be confined to root canal only

4. Over extended endodontic filling material may induce periapical inflammation by directly inducing foreign body reaction which is characterized by presence of foreign body giant cells, histiocytes and other chronic inflammatory cells (Fig. 3.28).

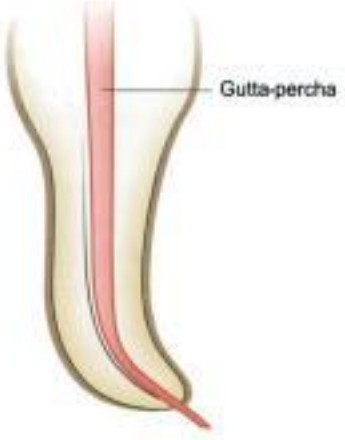


Fig. 3.28: Inflammation of periradicular tissue resulting from over extension of obturation material

DIAGNOSIS OF PERIRADICULAR PATHOLOGIES

1. *Chief complaint*: Patient usually complains of pain on biting, pain with swelling, pus discharge etc.
2. *Dental history*: Recurring episodes of pain, swelling with discharge, swelling which reduces on its own.

OBJECTIVE EXAMINATION

1. *Extraoral examination*: General appearance, skin tone, facial asymmetry, swelling, extraoral sinus, sinus-tract, tender or enlarged cervical lymph nodes.
2. *Intraoral examination*: It includes examination of soft tissues and teeth to look for dicoloration, abrasion, caries, restoration etc.

CLINICAL PERIAPICAL TESTS

1. ***Percussion***: Indicates inflammation of periodontium (Fig. 3.29).
2. ***Palpation***: Determines how far the inflammatory process has extended periapically.
3. ***Pulp vitality***:
   i. Thermal tests which can be heat or cold
   ii. Electrical pulp testing

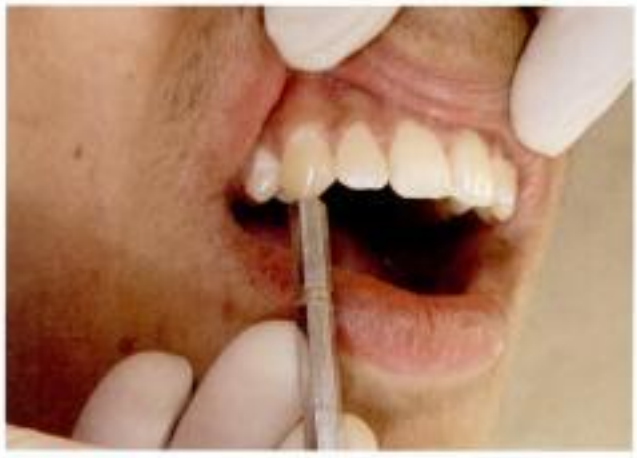


Fig. 3.29: Pain on percussion indicates inflammed periodontium

4. ***Periodontal examination***: It is important because periapical and periodontal lesion may mimic each other and require differentiation.
   a. *Probing*: Determines the level of connective tissue attachment. Probe can penetrate into an inflammatory periapical lesions that extends cervically (Fig. 3.30).
   b. *Mobility*: Determines the status of periodontal ligament.

Fig. 3.30: Probing of tooth determines the level of connective tissue attachment

5. ***Radiographic examination***: Periradicular lesions of pulpal origin have four characteristics:
   - Loss of lamina dura apically.
   - Radiolucency at apex regardless of cone angle.
   - Radiolucency resembles a hanging drop.
   - Cause of pulp necrosis is usually evident.

   Recent advances:
   - Digital subtraction radiography
   - Xeroradiography
   - Digital radiometric analysis
   - Computed tomography

- Radiovisiography
- Magnetic resonance imaging

CLASSIFICATION OF PERIRADICULAR PATHOLOGIES

I. *Grossman's classification*
   1. Acute periradicular disease
      a. Acute alveolar abscess
      b. Acute apical periodontitis
         i. Vital
         ii. Non vital
   2. Chronic Periradicular disease with areas of rarefaction:
      a. Chronic alveolar abscess
      b. Granuloma
      c. Cyst
   3. Condensing osteitis
   4. External root resorption
   5. Disease of the periradicular tissues of non-endodontic origin.

II. *WHO classification*

K 04.4 - Acute apical periodontitis
K 04.5 - Chronic apical periodontitis (apical granuloma)
K 04.6 - Periapical abscess with sinus
K 04.60 - Periapical abscess with sinus to maxillary antrum
K 04.61 - Periapical abscess with sinus to nasal cavity
K 04.62 - Periapical abscess with sinus to oral cavity
K 04.63 - Periapical abscess with sinus to skin
K 04.7 - Periapical abscess without sinus
K 04.8 - Radicular cyst (periapical cyst)
K 04.80 - Apical and lateral cyst
K 04.81 - Residual cyst
K04.82 - Inflammatory paradental cyst.

III. *Ingle's classification of pulpoperiapical pathosis*:
   A. *Painful pulpoperiapical pathosis*:
      1. Acute apical periodontitis
      2. Advanced apical periodontitis
         a. Acute apical abscess
         b. Phoenix abscess
         c. Suppurative apical periodontitis (chronic apical abscess)
   B. *Non painful pulpoperiapical pathosis*
      1. Condensing osteitis
      2. Chronic apical periodontitis both incipient and advanced stages.
      3. Chronic apical periodontitis
         a. Periapical granuloma
         b. Apical cyst
         c. Suppurative apical periodontitis.

ACUTE APICAL PERIODONTITIS (AAP)

Acute apical periodontitis is defined as painful inflammation of the periodontium as a result of trauma, irritation or inflection, through the root canal, regardless of whether the pulp is vital or non-vital. It is an inflammation around the apex of a tooth. The distinctive features of AAP are microscopic rather than roentgenographic, symptomatic rather than visible.

Etiology

a. In vital tooth it is associated with occlusal trauma, high points in restoration or wedging or forcing object between teeth.
b. In non-vital tooth AAP is associated with sequelae to pulpal diseases.
c. Iatrogenic causes can be over instrumentation of root canal pushing debris and microorganisms beyond apex, overextended obturation and root perforations.

Signs and Symptoms

- Dull, throbbing and constant pain
- Pain occurs over a short period of time
- Negative or delayed vitality test
- No swelling
- Pain on biting
- Cold may relieve pain or no reaction
- Heat may exacerbate pain or no reaction
- No radiographic sign; sometimes widening of periodontal ligament space.

Histopathology

Inflammatory reaction occur in apical periodontal ligament
↓
Dilatation of blood vessels
↓
Presence of polymorphonuclear leukocytes and round cells
↓
Serous exudate accumulation
↓
Distention of periodontal ligament and extrusion of tooth, slight tenderness
↓
If continued irritation occurs
↓
Loss of alveolar bone.

Treatment

Management of acute apical periodontitis

- Endodontic therapy should be initiated on the affected tooth at the earliest (Fig. 3.31)
- To control postoperative pain following initial endodontic therapy, analgesics are prescribed
- Use of antibiotics, either alone or in conjunction with root canal therapy is not recommended
- If tooth is in hyper-occlusion, relieve the occlusion
- For some patients and in certain situations, extraction is an alternative to endodontic therapy.

Fig. 3.31: Management of acute apical periodontitis

ACUTE APICAL ABSCESS

It is a localized collection of pus in the alveolar bone at the root apex of the tooth, following the death of pulp with extension of the infection through the apical foramen into periradicular tissue (Fig. 3.32).

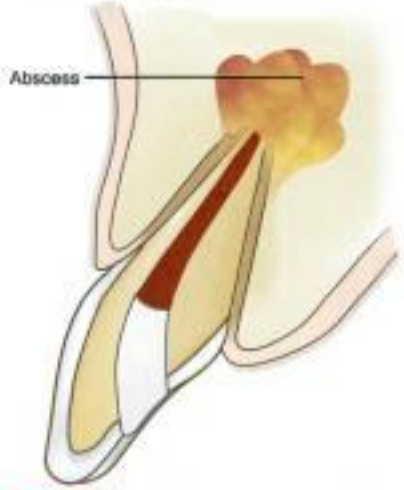


Fig. 3.32: Diagram showing periapical abscess

Etiology

Most common cause is bacterial invasion of dead pulp tissue but it can also occur by trauma, chemical or mechanical injury.

Tissue at surface of swelling appears taut and inflamed and pus starts to form underneath it. Surface tissue may become inflated from the pressure of underlying pus and finally rupture from this pressure. Initially the pus comes out in the form of a small opening but latter it may increase in size or number depending upon the amount of pressure of pus and softness of the tissue overlying it. This process is beginning of chronic abscess.

Features of Acute Apical Abscess

- Tooth is non-vital
- Pain
  - Rapid onset
  - Readily localized as tooth becomes increasingly tender to percussion
  - Slight tenderness to intense throbbing pain
  - Marked pain to biting
- Swelling
  - Palpable, fluctuant
  - Localized sense of fullness
- Mobility
  - May or may not be present
- Tooth may be in hyperocclusion
- Radiographic changes
  - No change to large periapical radiolucency

SYMPTOMS

In early stage there is tenderness of tooth which is relieved by continued slight pressure on extruded tooth to push it back into alveolus. Later on throbbing pain develops with diffuse swelling of overlying tissue. Tooth becomes more painful, elongated and mobile as infection increases in latter stages. Patient may have systemic symptoms like fever, increased WBC count. Spread of lesion towards a surface may take place causing erosion of cortical bone or it may become diffuse and spread widely leading to formation of cellulitis (Fig. 3.33). Location of swelling is determined by relation of apex of involved tooth to adjacent muscle attachment.

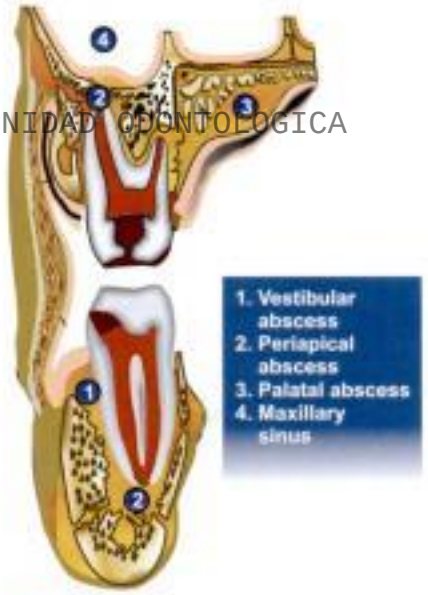


Fig. 3.33: Spread of apical abscess to surrounding tissues, if it is not treated

DIAGNOSIS

- Clinical examination
- Initially locating the offending tooth is difficult due to the diffuse pain. Location of the offending tooth is easier when there is extension of tooth following infection
- Pulp vitality tests give negative response
- Tenderness on percussion and palpation
- Radiography helpful in determining the affected tooth as it shows a cavity or evidence of bone destruction at root apex.

Differential diagnosis of acute alveolar abscess and periodontal abscess

Features	*Acute alveolar abscess*	*Periodontal abscess*
1. Pain Type	Pulsating, pounding, continuous	Dull
2. Pain Localization	Easily localized due to percussive tenderness	Upon probing
3. Pain at night/ Postural	Pain continuous	No
4. Mobility	Yes	Sometimes
5. Pulp tests		
I. EPT	No response	Normal
II. Cold	No response	Normal
III. Heat	No response	Normal
6. Swelling	Yes – often to large size	Occasionally
7. Radiograph	Caries, defective restorations	Possible foreign body or vertical bone loss
8. Treatment	• Establish drainage (Incision and drainage) • Antibiotics • NSAIDSs	• Removal of foreign body • Scaling • Curettage if necessary

HISTOPATHOLOGY

Polymorphonuclear leucocyte infiltrates
↓
Accumulation of inflammatory exudates in response to active infection
↓
Distention of periodontal ligament
↓
Elongation of tooth.
If the process continues, periodontal ligament separates
↓
Mobile tooth
↓
Bone resorption at apex
↓
Localized lesion of liquefaction necrosis containing polymorphonuclear leucocytes, debris, cell remnants and purulent exudates

MANAGEMENT OF AN ACUTE APICAL ABSCESS

- Drainage of the abscess should be initiated as early as possible. This may include:
  a. Non-surgical endodontic treatment (Root canal therapy) (Fig. 3.34)
  b. Incision and drainage
  c. Extraction
- Considerations regarding the treatment should be dependent on certain factors:
  a. Prognosis of the tooth
  b. Patient preference
  c. Strategic value of the tooth
  d. Economic status of the patient
- In case of localized infections, systemic antibiotics provide no additional benefit over drainage of the abscess
- In the case of systemic complications such as fever, lymphadenopathy, cellulitis or patient who is immuno compromised, antibiotics should be given in addition to drainage of the tooth

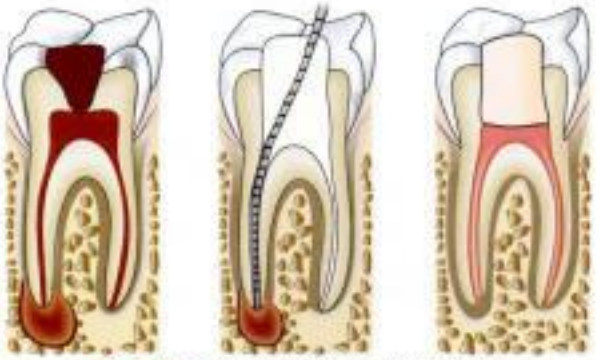


Fig. 3.34: Management of periapical abscess

- Relieve the tooth out of occlusion in hyper-occlusion cases
- To control postoperative pain following endodontic therapy, non-steroidal anti-inflammatory drugs should be given.

PHOENIX ABSCESS

Phoenix abscess is defined as an acute inflammatory reaction superimposed on an existing chronic lesion, such as a cyst or granuloma; acute exacerbation of a chronic lesion.

Etiology

Chronic periradicular lesions such as granulomas are in a state of equilibrium during which they can be completely asymptomatic. Because of influx of necrotic products from diseased pulp or because of bacteria and their toxin can cause the dormant lesion to react. This leads to initiation of acute inflammatory response. Lowered body defenses also trigger an acute inflammatory response.

Symptoms

- Clinically often indistinguishable from acute apical abscess
- At the onset—tenderness of tooth and elevation of the tooth from socket
- Tenderness on palpating the apical soft tissue.

Diagnosis

- Associated with initiation of root canal treatment most commonly
- History from patient
- Pulp tests show negative response
- Radiographs show large area of radiolucency in the apex created by inflammatory connective tissue which has replaced the alveolar bone at the root apex
- Histopathology of phoenix abscess shows areas of liquefaction necrosis with disintegrated polymorphonuclear leukocytes and cellular debris surrounded by macrophages, lymphocytes, plasma cells in periradicular tissues
- Phoenix abscess should be differentiated from acute alveolar abscess by patient's history, symptoms and clinical tests results.

Treatment

- Establishment of drainage
- Once symptoms subside—complete root canal treatment.

PERIAPICAL GRANULOMA

Periapical granuloma is one of the most common sequelae of pulpitis. It is usually described as a mass of chronically inflamed granulation tissue found at the apex of non-vital tooth (Fig. 3.35).

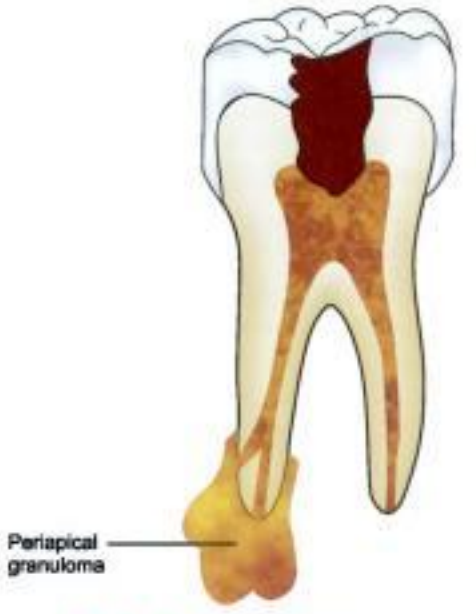


Fig. 3.35: Periapical granuloma

CLINICAL FEATURES

- Most of the cases are asymptomatic but sometimes pain and sensitivity is seen when acute exacerbation occurs
- Tooth is not sensitive to percussion
- No mobility
- Soft tissue overlying the area may / may not be tender
- No response to thermal or electric pulp test
- Mostly, lesions are discovered on routine radiographic examination.

RADIOGRAPHIC FEATURES

- Mostly discovered on routine radiographic examination
- The earliest change in the periodontal ligament is found to be thickening of ligament at the root apex
- Lesion may be well circumscribed or poorly defined
- Size may vary from small lesion to large radiolucency exceeding more than 2 cm in diameter
- Some amount of root resorption has been reported.

HISTOPATHOLOGIC FEATURES

- It consists of inflamed granulation tissue that is surrounded by a fibrous connective tissue wall

- The granulation consists of dense lymphocytic infiltrate which further contains neutrophils, plasma cells, histiocytes and eosinophils
- Sometimes, Rushel bodies may also be present.

TREATMENT AND PROGNOSIS

Main objective in treatment is to reduce and eliminate offending organisms and irritants from the periapical area.

- In restorable tooth, root canal therapy is preferred
- In non-restorable tooth, extraction followed by curettage of all apical soft tissue.

RADICULAR CYST

The radicular cyst is an inflammatory cyst which results because of extension of infection from pulp into the surrounding periapical tissues.

Etiology

- Caries
- Irritating effects of restorative materials
- Trauma
- Pulpal death due to development defects.

CLINICAL FEATURES

- The cyst is frequently asymptomatic and sometimes it is discovered when periapical radiographs are taken of teeth with non-vital pulps
- Incidence - Males are affected more than females.
- Age - Peak incidence in third or fourth decades.
- Site - Highest in anterior maxilla
  - In mandibular posterior teeth, separate small cysts arise from each apex of multi-rooted teeth.
- Slowly enlarging swelling sometimes attains a large size.
- As the cyst enlarges in size, the covering bone becomes thin in size and exhibits springiness due to fluctuation.
- In maxilla, palatal expansion is mainly seen in case of maxillary lateral incisor.
- The involved tooth / teeth usually found to be non-vital, discolored, fractured or failed root canal.

PATHOGENESIS

Periapical granulomas are initiated and maintained by the degradation products of necrotic pulp tissue. Stimulation of the resident epithelial rests of Malassez occurs in response to the products of inflammation. Cyst formation occurs as a result of epithelial proliferation, which helps to separate the inflammatory stimulus from the surrounding bone. When proliferation occurs within the body of the granuloma, it plugs the apical foramen which limits the egress of bacteria (Fig. 3.36). Sometimes, epithelial plugs protrude out from the apical foramen resulting in a pouch connected to the root and continuous with the root canal. This is termed as ***pocket or bay cyst*** (Fig. 3.37).

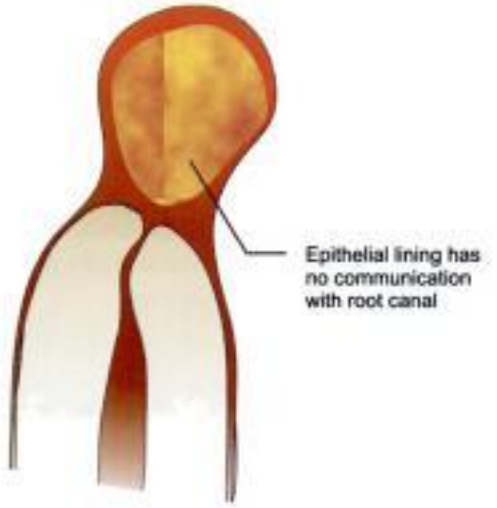


Fig. 3.36: Periapical cyst

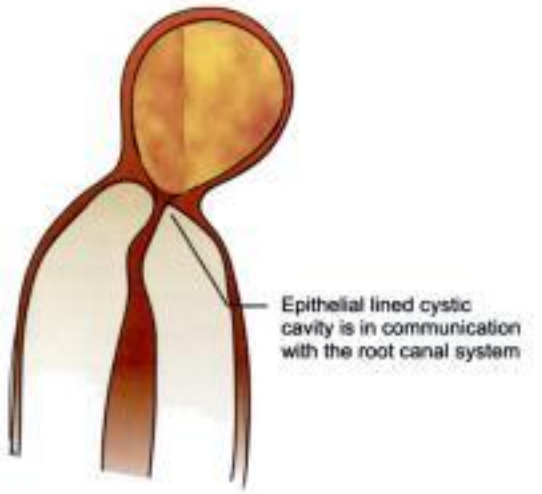


Fig. 3.37: Pocket or bay cyst

- Breakdown of cellular debris within the cyst lumen raises the protein concentration, producing an increase in osmotic pressure. The result is fluid transport across the epithelial lining into the lumen from the connective tissue side. Fluid ingress assists in outward growth of the cyst. With osteoclastic bone resorption, the cyst expands. Other bone-resorbing factors, such as prostaglandins, interleukins, and proteinases, from inflammatory cells and cells in the peripheral portion of the lesion permit additional cyst enlargement.

RADIOGRAPHIC FEATURES

Radiographically radicular cyst appears as round, pear or ovoid shaped radiolucency, outlined by a narrow radio-opaque margin (Fig. 3.38).

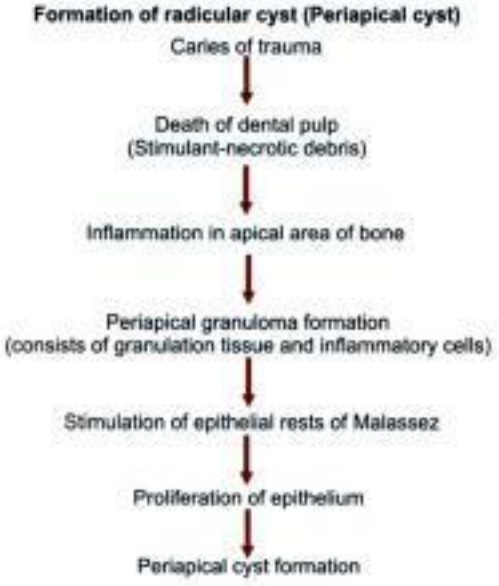
Fig. 3.38: Radiographic picture of a periapical cyst

Treatment

Different options for management of residual cyst are:
- Endodontic treatment
- Apicoectomy
- Extraction (severe bone loss)
- Enucleation with primary closure
- Marsupilization (in case of large cysts).

CHRONIC ALVEOLAR ABSCESS

Chronic alveolar abscess is also known as suppurative apical periodontitis which is associated with gradual egress of irritants from root canal system into periradicular area leading to formation of an exudate.



Etiology

It is similar to acute alveolar abscess. It also results from pulpal necrosis and is associated with chronic apical periodontitis that has formed an abscess. The abscess has burrowed through bone and soft tissue to form a sinus react stoma on the oral mucosa.

Symptoms

- Generally asymptomatic
- Detected either by the presence of a sinus tract or on routine radiograph
- In case of open carious cavity—drainage through root canal sinus tract prevents swelling or exacerbation of lesion—can be traced to apex of involved tooth.

Diagnosis

Chronic apical abscess may be associated with asymptomatic or slightly symptomatic tooth. Patient may give history of sudden sharp pain which subsided and has not reoccurred. Clinical examination may show a large carious exposure, a restoration of composite, acrylic, amalgam or metal, or discoloration of crown of tooth.

It is associated symptoms only if sinus drainage tract become blocked. Vitality tests show negative response because of presence of necrotic pulps.

Radiographic examination shows diffuse area of rarefaction. The rarefied area is so diffuse as to fade indistinctly into normal bone.

Differential Diagnosis

Chronic alveolar abscess must be differentially diagnosed from a granuloma or cyst, in which accurate diagnosis is made by studying the tissue microscopically. It should also be differentiated with cementoma which is associated with vital tooth.

Treatment

Removal of irritants from root canal and establishing drainage is main objective of the treatment. Sinus tract resolve following root canal treatment removing the irritants.

EXTERNAL ROOT RESORPTION

Resorption is a condition associated with either physiologic or a pathologic process that results in loss of substance from a tissue such as dentin, cementum or alveolar none (Fig. 3.39).

In external root resorption, root resorption affects the cementum or dentin of the root of tooth. It can be:

- Apical root resorption
- Lateral root resorption
- Cervical root resorption.

Fig. 3.39: External root resorption

Etiology

Periradicular inflammation due to

- Infected necrotic pulp
- Over instrumentation during root canal treatment
- Trauma
- Granuloma/cyst applying excessive pressure on tooth root
- Replantation of teeth
- Adjacent impacted tooth.

Symptoms

- Asymptomatic during development
- When root is completely resorbed, tooth becomes mobile
- When external root resorption extends to crown, it gives "Pink tooth" appearance
- When replacement resorption / ankylosis occur, tooth becomes immobile with characteristic high percussion sound (Fig. 3.40).

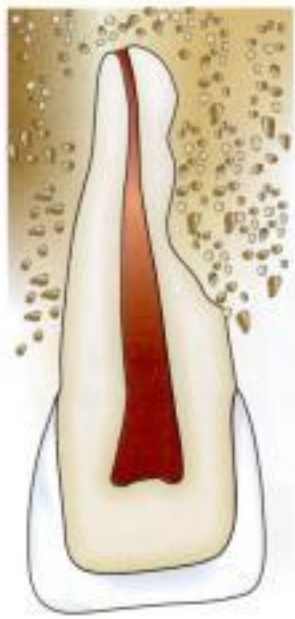


Fig. 3.40: Ankylosis of tooth without intermediate periodontal ligament

Radiographs Show

- Radiolucency at root and adjacent bone
- Irregular shortening or thinning of root lip,
- Loss of Lamina dura.

Treatment

- Remove stimulus of underlying inflammation.
- Non-surgical endodontic treatment should be attempted before surgical treatment is initiated.

DISEASES OF PERIRADICULAR TISSUE OF NON-ENDODONTIC ORIGIN

Periradicular lesions may arise from the remnants of odontogenic epithelium.

Benign Lesions

1. Early stages of periradicular cemental dysplasia
2. Early stages of monostatic fibrous dysplasia
3. Ossifying fibroma
4. Primordial cyst
5. Lateral periodontal cyst
6. Dentigerous cyst
7. Traumatic bone cyst
8. Central giant cell granuloma
9. Central hemangioma
10. Hyperparathyroidism
11. Myxoma
12. Ameloblastoma.

Radiographic Features of Lesions of Non-odontogenic Origin

- Radiolucent areas
- Intact lamina dura.

Diagnosis

Teeth associated with non-odontogenic lesions are usually vital. Final diagnosis is based on surgical biopsy and histopathological examination.

Malignant Lesions

They simulate endodontic periradicular lesions and are often meta static in nature

1. Squamous cell carcinoma.
2. Osteogenic sarcoma
3. Chondrosarcoma
4. Multiple myeloma.

Diagnosis

- ***Involved tooth is vital***. Occasionally disruption of pulp and sensory nerve may cause no response
- ***Radiographic features***: Lesions are associated with rapid and extensive loss of hard tissue, bone and tooth
- ***Biopsy***: Histological evaluation of diagnosis.

HISTOPATHOLOGY OF PERIAPICAL RESPONSE TO VARIOUS IRRITANTS

Root canal of teeth contains numerous irritants because of some pathologic changes in pulp. Penetration of these irritants from infected root canals into periapical area can lead to formation and perpetuation of periradicular lesions. In contrast to pulp, periradicular tissue have unlimited source of undifferentiated cells which can participate in inflammation and repair in inflammation and repair. Also these tissues have rich collateral blood supply and lymph drainage.

Depending upon severity of irritation, duration and host, response to periradicular pathosis may range from slight inflammation to extensive tissue destruction. Reactions involved are highly complex and are usually mediated by nonspecific and specific mediators of inflammation.

NONSPECIFIC MEDIATORS OF PERIRADICULAR LESIONS

Nonspecific mediators can be classified into cell derived and plasma derived mediators.

Cell Derived Mediators

Vasoactive amines

Vasoactive amines such as histamine, serotonin are present in mast cells, basophils and platelets. Their release cause increase in tissue permeability, vasodilation used vascular permeability (Fig. 3.41).

Leukotrienes

These are produced by activation of lipoxygenase pathway of arachidonic acid metabolism. Studies have shown the presence of LTB4, LTC4, LTD4 and LTE4 in periradicular lesions which cause different effects on the tissues as shown in (Fig. 3.42).

Platelet Activating Factor

It is released from IgE—sensitized basophils or mast cells. Its action include increase in vascular permeability, chemotaxis, adhesion of leucocytes to endothelium and bronchoconstriction.

Non Specific Mediators of Inflammation

I. Cell Derived Mediators
   1. Vasoactive amines
   2. Leukotriences (Metabolites via lipo-oxygenase pathway)
   3. Platelet activating factor
   4. Lysosomal enzymes
   5. Cytokines
   6. Prostaglandins (Metabolites via cyclo-oxygenase pathway).

II. Plasma Derived Mediators
   1. The fibrinolytic system
   2. The complement system
   3. The kinin system.

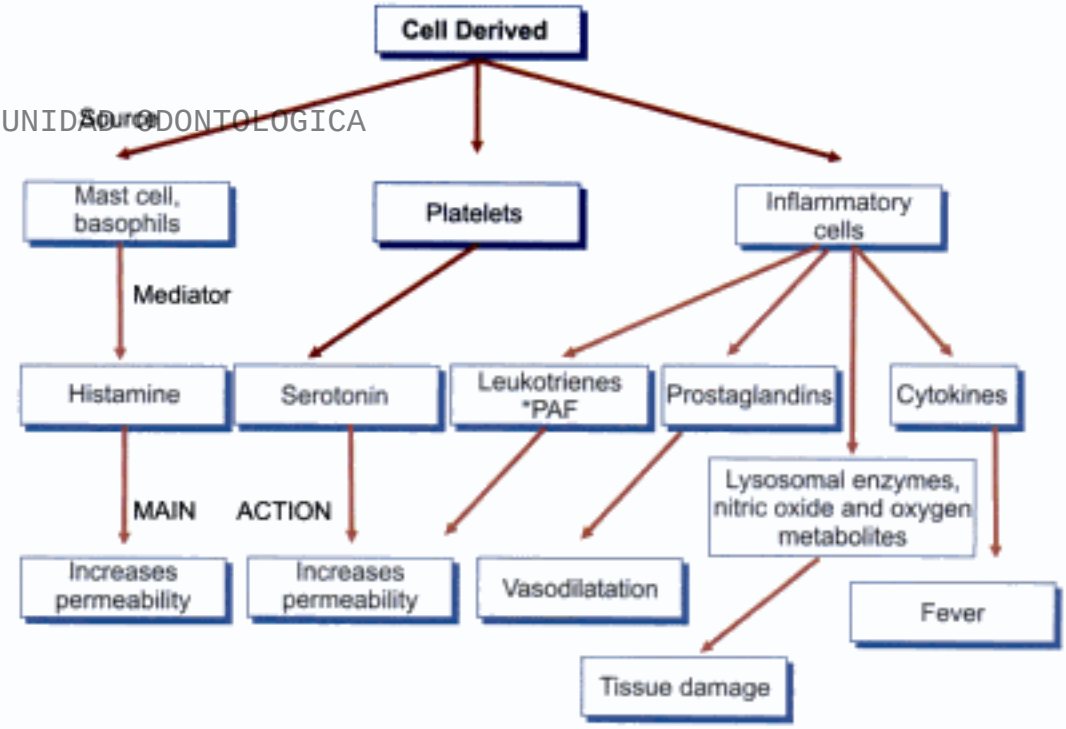


Fig. 3.41: PAF – Platelet activating factor

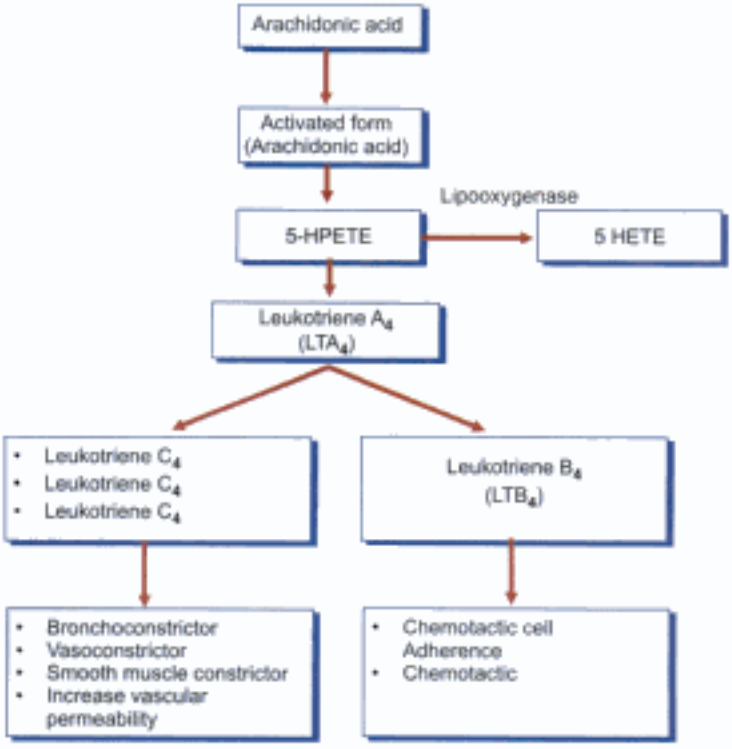


Fig. 3.42: Arachidonic acid metabolites via lipo-oxygenase pathway

Lysosomal Enzymes

Lysosomal enzymes such as alkaline phosphatase, lysozyme, peroxidases, collagenase cause increase in vascular permeability, leukocytic chemotaxis, bradykinin formation and activation of complement system.

Cytokines

Different cytokines such as interleukins and tumor necrosis factor (TNF) cause development and perpetuation of periradicular lesions. Various studies conducted on periradicular lesions shown the presence of IL-1 in areas of the bone resorption (Fig. 3.43)

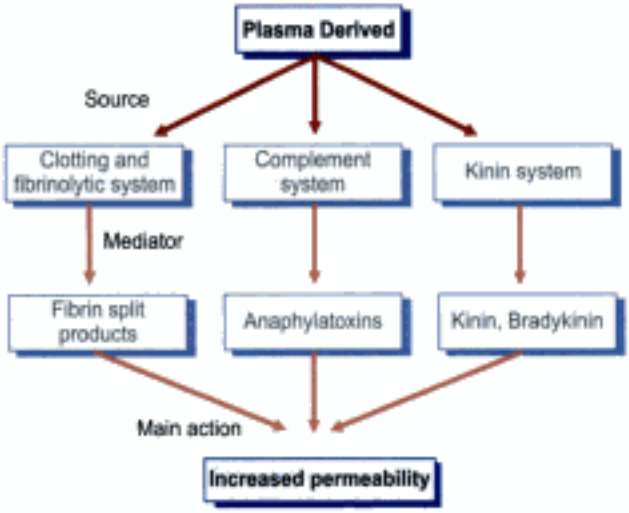


Fig. 3.43: Non specific mediators of inflammation

Prostaglandins

These are produced by activation of cyclo-oxygenase pathway of arachidonic acid metabolism (Fig. 3.44). Studies have shown high levels of PGE_2 in periradicular lesions. Torbinejad et al found that periradicular bone resorption can be inhibited by administration of indomethacin, an antagonist of PGs. This indicates that prostaglandins are also involved in the pathogenesis of periradicular lesions.

Plasma Derived Mediators

1. *The fibrinolytic system*
   The fibrinolytic system is activated by Hageman factor which causes activation of plasminogen. This results in release of fibrinopeptides and fibrin degradation products which cause increase in vascular permeability and leucocytic chemotaxis.
2. *The complement system*
   Trauma to periapex can result in activation of kinin

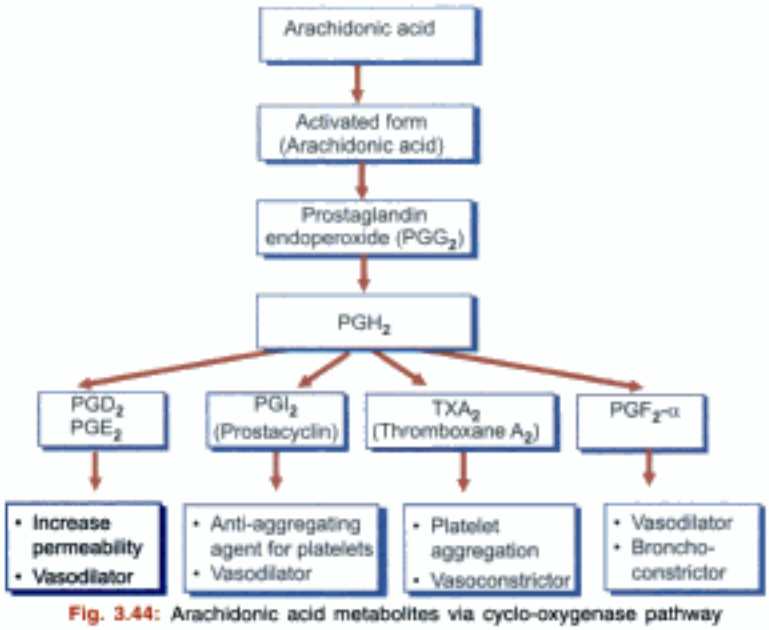


Fig. 3.44: Arachidonic acid metabolites via cyclo-oxygenase pathway

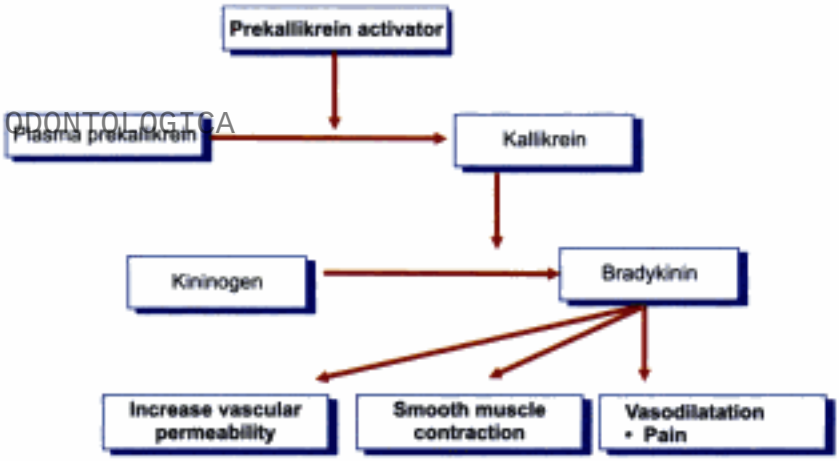


Fig. 3.45: Pathway of the kinin system

system which in turn activates complement system. Several studies have shown elevated levels of kinins and C_3 complement component in periradicular lesions. Products released from activated complement system cause swelling, pain and tissue destruction.

3. *The kinin system*
These are produced by proteolytic cleavage of kininogen. Release of kinins cause smooth muscle contraction, vasodilation and increase in vascular permeability (Fig. 3.45).

4 Endodontic Microbiology

- Introduction
- Portals of Entry for Microorganisms
- Microbial Virulence and Pathogenicity
- Microbial Ecosystem of Root Canal
- Primary Endodontic Infections
- Microbiology of Periradicular Endodontic Infections
- Microbiology of Root Canal Failures
- Endodontic Abscess and Cellulitis
- Identification of Bacteria
- How to Combat Microbes in the Endodontic Therapy

INTRODUCTION

Most of the pathologies of pulp and the periapical tissues are directly or indirectly related to the microorganisms. Therefore to effectively diagnose and treat endodontic infection, one should have the knowledge of bacteria associated with endodontic pathology. Since many years, the interrelationship of microorganisms and the root canal system have been proved. *Leeuwenhoek* observed infected root canal of a tooth and found "cavorting beasties". After this, it took 200 years for *W.D. Miller* to make the correlation between microorganisms and pulpal or periradicular pathologies.

Then in 1965, *Kakehashi et al* found that bacteria are the main etiological factors in the development of pulpal and periradicular diseases. *Kakehashi et al* proved that without bacterial involvement only minor inflammation occurred in exposed pulp.

So we have seen that a strong relationship occurs between microorganisms and pulpal or periradicular diseases. All the surfaces of human body are colonized by microorganisms. Colonization is the establishment of bacteria in a living host. It occurs if biochemical and physical conditions are available for growth. Permanent colonization in symbiotic relationship with host tissue results in establishment of normal flora.

Infection results if microorganisms damage the host and produce clinical signs and symptoms. The degree of pathogenicity produced by microorganisms is called as **virulence**.

PORTALS OF ENTRY FOR MICROORGANISMS

Microorganisms may gain entry into pulp through several routes. Most common portal of entrance for microorganisms to dental pulp is dental caries.

They can also gain entry into pulp cavity via mechanical or traumatic injury, through gingival sulcus and via bloodstream.

Entry of Microorganisms into Pulp Through

1. Open cavity
2. Open dentinal tubules
3. Periodontal ligament or gingival sulcus
4. Anachoresis
5. Faulty restorations

Entry Through Open Cavity

This is the most common way of entry of microorganisms into the dental pulp. When enamel and dentin are intact, they act as barrier to microorganisms (Fig. 4.1). But when these protective layers get destroyed by caries, bacteria gain entry into the pulp (Fig. 4.2).

Fig. 4.1: Showing normal tooth anatomy with protective layers of the pulp

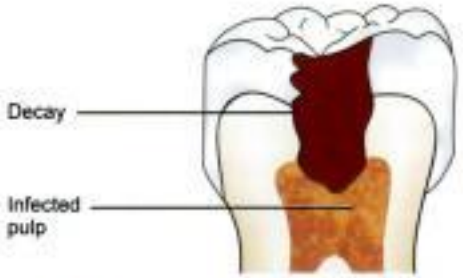


Fig. 4.2: Pulp infection from tooth decay

This protective barrier of enamel and dentine also gets destroyed by traumatic injuries, fractures, cracks or restorative procedures, thus allowing the access of microorganisms to the pulp.

Through Open Dentinal Tubules

It has been seen that microorganisms can pass into the dentinal tubules and subsequently to the pulp. Bacterial penetration into dentinal tubules has been shown to be greater in teeth with necrotic pulp (Fig. 4.3). Bacteria are preceded in the course of the tubules by their breakdown products which may act as pulp irritants.

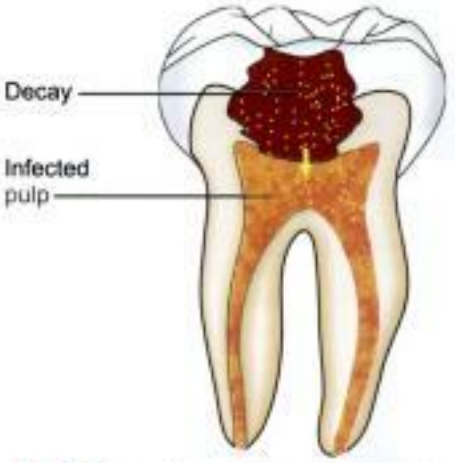


Fig. 4.3: Entry of bacteria through infected pulp

Through the Periodontal Ligament or the Gingival Sulcus

Microorganisms also gain entry into pulp via accessory and lateral canals which connect pulp and the periodontium. If periodontal disease or therapy destroys the protective covering, canal may get exposed to the microorganisms present in the gingival sulcus (Fig. 4.4). The removal of cementum during periodontal therapy also exposes dentinal fluids to oral flora.

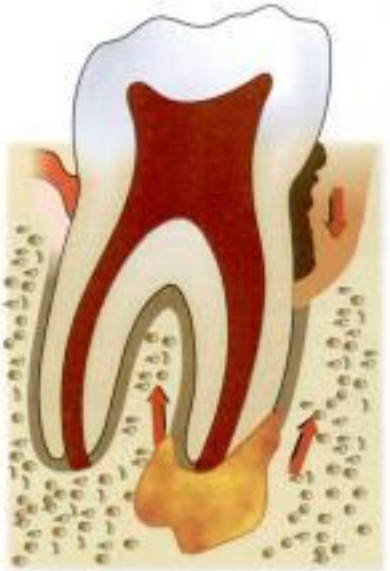


Fig. 4.4: Periodontal lesions causing inflammation of pulp

Anachoresis

A transient bacteremia is usually associated with many activities in a healthy individual. Anachoresis refers to the attraction of blood borne bacteria in the areas of inflammation. In other words anachoresis is a process by which microorganisms are transported in the blood to an area of inflammation where they establish an infection. But whether anachoresis contributes to pulpal or periradicular infection has not been determined.

Through Faulty Restorations

It has been seen that faulty restoration with marginal leakage can result in contamination of the pulp by bacteria. Bacterial contamination of pulp or periapical area can occur through broken temporary seal, inadequate final restoration and unused post space (Fig. 4.5).

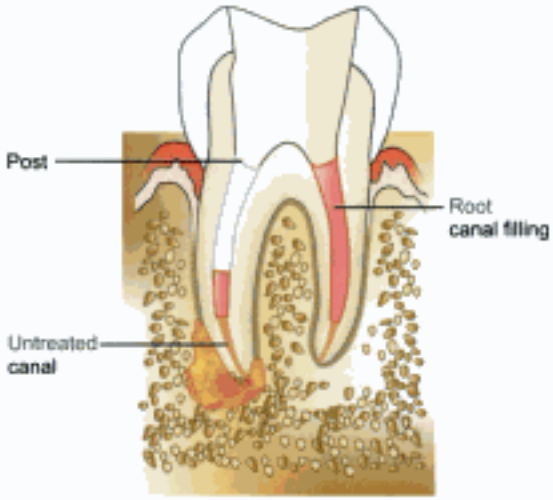


Fig. 4.5: Untreated canal and empty post space causing root canal failure

MICROBIAL VIRULENCE AND PATHOGENICITY

Under normal conditions pulp and periapical tissues are sterile in nature, when microorganisms invade and multiply in these tissues, endodontic infections result.

Pathogenicity is ability of microorganisms to produce a disease

Virulence is degree of pathogenicity.

Bacterial virulence factors include fimbriae, capsules, lipopolysaccharides, enzymes (collagenase, hyaluronidase, proteases), extracellular vesicles, short chain fatty acids, polyamines and low molecular weight products such as ammonia and hydrogen sulfide. Virulence factors may vary from strain to strain. Virulence is directly related to pathogenicity.

In *1965, Hobson* gave an equation showing the relation of number of microorganisms, their virulence, resistance of host and severity of the disease.

$$\frac{\text{Number of microorganisms} \times \text{Virulence of microorganisms}}{\text{Resistance of host}} = \text{Severity of the disease}$$

We can see that along with number of microorganisms, their virulence is also directly related to the severity of the disease.

Various Virulent Factors

1. Lipopolysaccharides (LPS)
2. Extracellular vesicles
3. Enzymes
4. Fatty acids
5. Polyamines
6. Capsule
7. Pilli

Lipopolysaccharides (LPS)

Lipopolysaccharides are present on the surface of gram-negative bacteria. LPS have nonspecific antigens which are not neutralized by antibodies. They exert numerous biologic functions when released from cells in the form of endotoxins. Endotoxins have capability to diffuse into the dentin. Various studies have shown the relationship between the endotoxins and the periradicular inflammation.

Extracellular Vesicles

Extracellular vesicles are produced by gram-negative bacteria in the form of endotoxins, outer membrane fragments or blebs. They have trilaminar structure similar to outer membrane of the parent bacteria. These vesicles contain various enzymes and toxic products which are responsible for hemagglutination, hemolysis and bacterial adhesion. Since they have antigenic properties similar to the parent bacteria, they may protect bacteria by neutralizing specific antibodies.

Enzymes

Enzymes produced by bacteria have numerous activities like they help in spread of the infection, neutralization of immunoglobulin and the complement components. PMN

leukocytes release hydrolytic enzymes which degenerate and lyse to form purulent exudates and have adverse effects on the surrounding tissues.

Fatty Acids

Various short chain fatty acids like propionic acid, butyric acid are produced by anaerobic bacteria. These cause neutrophil chemotaxis, degranulation, phagocytosis, and stimulate interleukin-1 production which further causes bone resorption and periradicular diseases.

Polyamines

These are biologically active chemicals found in the infected canals. Some of polyamines such as cadaverine, putrescine, spermidine help in regulation of the cell growth, regeneration of tissues and modulation of inflammation.

Other virulent factors like ***capsules*** present in gram-negative black pigmented bacteria, enable them to avoid phagocytosis. ***Pilli*** may play an important role in attachment of bacteria to surfaces and interaction with other bacteria.

New Nomenclature of Bacteroids Species

1. Porphyromonas – Dark-pigmented (asaccharolytic bacteroides species)
   - *Porphyromonas asaccharolyticus*
   - *Porphyromonas gingivalis**
   - *Porphyromonas endodontalis**
2. Prevotella—Dark-pigmented (saccharolytic bacteroides species)
   - *Prevotella melaninogenica*
   - *Prevotella denticola*
   - *Prevotella intermedia*
   - *Prevotella nigrescens**
   - *Prevotella corporis*
   - *Prevotella tannerae*
3. Prevotella – Nonpigmented (saccharolytic bacteroides species)
   - *Prevotella buccae**
   - *Prevotella bivia*
   - *Prevotella oralis*
   - *Prevotella oulorum*

*Most commonly isolated species of black-pigmented bacteria

MICROBIAL ECOSYSTEM OF THE ROOT CANAL

Since many years, various papers have been published regarding the microbial flora of the root canals, normal and infected both. But over past 5-10 years, difference in flora has reported because of improved technology in sampling, culture techniques, culture media as well as more advanced technology regarding isolation and identification of the microorganism.

Most commonly gram-positive organisms are found in the root canals, but gram-negative and obligate anaerobes have also been found in the root canals. Usually the microorganisms which can survive in environment of low oxygen tension and can survive the rigors of limited pabulums are found in root canals. Variety of micro-organisms enters the root canal system through various portals of entry but only those which fit for survival in such environment do survive. Most commonly seen bacteria in root canals is streptococci, others can be *staphylococcus*, gram-negative and anaerobic bacteria.

In necrotic pulp, a mix of bacterial species is found. In necrotic pulp, there is lack of circulation with compromised host defense mechanism; this makes pulp as a reservoir for invading microbes.

In necrotic pulps, tissue fluids and disintegrated cells from necrotic tissue, low oxygen tension and bacterial interactions are the main factors which determine which bacteria will predominate. The growth of one bacterial species may be dependent on the other bacterial species which supplies the essential nutrients. In the similar way, antagonistic relationship may occur in bacteria, i.e. byproducts of some bacterial species may kill or retard the growth of others species. In other words, some byproducts can act either as nutrient or as toxin depending on bacterial species (Fig.. 4.6).

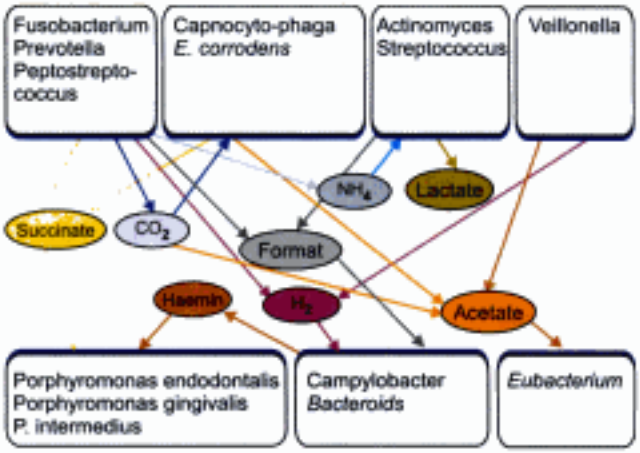


Fig. 4.6: Interrelationship of various root canal microorganisms

Naidorf summarized the few generalizations in relation to organisms isolated from the root canals:

1. Mixed infections are more common than single organisms.
2. Pulp contains flora almost similar to that of oral cavity.
3. Approximately 25 percent of isolated organisms are anaerobes.
4. Organisms isolated from flare-up as well as asymptomatic cases are almost similar.
5. Various researchers have identified wide variety of microorganisms in the root canals which is partially related to personal interest and culture techniques used by them.

PRIMARY ENDODONTIC INFECTIONS

In *1890, Miller* first investigated the association of presence of bacteria with pulpal diseases. Later in *1965, Kakehashi et al* proved the bacteria as main causative factor for endodontic diseases. It has been shown by various studies that endodontic infections are polymicrobial, though facultative bacteria predominate in early root canal infections, in latter stages they are replaced by strict anaerobic organisms.

A root canal containing a necrotic pulp becomes a selective habitat that allows some species of bacteria to grow in preference to others. The nutrients provided by the breakdown products of a necrotic pulp, tissue fluid and serum from surrounding tissues along with low oxygen tension, and bacterial by-products support the growth of selected microorganisms. The coronal portion of a root canal may harbor organisms different from those in the apical portion.

Some species of black-pigmented bacteria, peptostreptococci, Fusobacterium and Actinomyces species have been found related to clinical signs and symptoms. But because of polymicrobial nature of the endodontic infections, no absolute correlation has been found between bacterial species and severity of endodontic infections.

Coaggregation of different species of bacteria or self aggregation of the same species may present the organisms protection from the host's defenses and supply nutrients from the surrounding bacteria. The combination of *Fusobacterium nucleatum* with dark-pigmented bacteria *Prevotella* intermedia and Porphyromonas gingivalis has been shown to be more virulent than when the bacteria are in pure culture. This supports the concept that there is a synergistic relationship between bacteria in an endodontic infection.

Traditional methods of identifying microorganisms were based on gram staining, colonial morphology and biochemical tests, etc. Nowadays newer technologies like DNA method, molecular techniques like PCR (Polymerase chain reaction) SDS-PAGE have helped to identify various microorganisms. For example using DNA studies, black pigmented bacteria previously in génesis *Bacteroids* now have been placed in the genesis *Porphyromonas* and *Prevotella*. PCR technique has been used to show that geographical differences exist among the endodontic infections.

The virulence potential of dark-pigmented bacteria has been studied in animals. Strains of cultivable bacteria have been shown to possess the ability to resist phagocytosis, degrade immunoglobulins, and increase pathogenesis when in combination with other specific strains of bacteroids.

Microbiology of Infected Root Canal

Obligate anaerobes	*Facultative anaerobes*
Gram negative bacilli	*Gram negative bacilli*
Porphyromonas*	Capnocytophaga
Prevotella**	Eikenella
Fusobacterium	
Campylobacter	
Bacteroides	
Gram-negative cocci	*Gram-negative cocci*
Veillonella	Neisseria
Gram -positive bacilli	*Gram-positive bacilli*
Actinomyces	Actinomyces
Lactobacillus	Lactobacillus
Proprionibacterium	
Gram-positive cocci	*Gram-positive cocci*
Streptococcus	Streptococcus
Peptostreptococcus	Enterococcus
Spirochetes	*Fungi*
Treponema	Candida

*Dark pigmented bacteria
**Dark pigmented bacteria and nonpigmenting bacteria

The pathogenicity of Bacteroids is mainly related to the presence of lipopolysaccharides and peptidoglycans. These:

i. Induce hormones like cytokinins which play an important role in inflammations
ii. Stimulate B- lymphocytes.
iii. Activate complement cascade
iv. Release various enzymes like collagenase
v. Enhance production of various pain mediators like bradykinin, histamine and prostaglandins.
vi. LPS once released (as endotoxin) causes biological effects including inflammation and bone resorption.

Certain gram-positive bacteria have also been found associated with endodontic infections. Actinomyces may colonize periapical tissues. Aggregates of organisms may be recognized in periradicular biopsy sections as sulfur granules. A recent study using the PCR detected species of *Actinomyces (israelii, naeslundii, viscosus)* in 80 percent of the infected root canals and 46 percent of periradicular abscesses.

A. israelii is a bacterial species of endodontic infections which is resistant to conventional endodontic treatment. But at present combination of sodium hypochlorite and calcium hydroxide has proved to be an effective method in killing them. Surgery is another option to eliminate *A.israelii* from the periapical region.

Other gram-positive bacteria often cultured from endodontic infections include Peptostreptococci, Streptococcus, Enterococcus, and Eubacterium. Using PCR, Peptostreptococci was detected in 28 percent of the infected root canals tested. Recently DNA hybridization and PCR have been used for screening spirochetes.

Fungi have been cultivated and detected using molecular methods in infected root canal. When using PCR, ***Candida albicans*** was detected in 21 percent of infected root canals.

Viruses may also be associated with endodontic disease. Bacteriocins are viruses that infect bacteria and carry DNA into the genome of the bacteria. Usually the HIV, cytomegalovirus and Epstein Barr virus are seen to be associated with periapical pathologies.

Molecular methods such as DNA hybridization and PCR detect and identify many more microorganism than the so-called "gold standard" of culturing. Molecular methods are much more sensitive and offer precise identification at the DNA level. A disadvantage of molecular methods is not knowing whether the DNA was from an alive or dead organism. Currently antibiotic susceptibility tests cannot be accomplished without viable organisms. In the future, probes may be used to identify antimicrobial resistant genes in the DNA samples without need of growing the organisms for susceptibility tests.

MICROBIOLOGY OF PERIRADICULAR ENDODONTIC INFECTIONS

Once a necrotic pulp is infected, the root canal system becomes storehouse of microorganisms (Fig. 4.7). At the apical foramen and at the root ends of infected canal, plaque like biofilms have been found.

Extraradicular bacteria are usually associated with acute symptoms, the presence of a sinus tract, an infected cyst, or

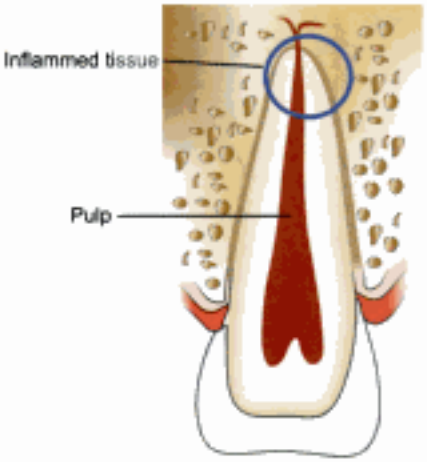


Fig. 4.7: Microorganisms in infected root canal

in cases not responding to endodontic treatment. Both acute and chronic periradicular abscesses are polymicrobial infections with large numbers of bacteria.

Periapical inflammatory lesions contain macrophages, lymphocytes (T-cells and B-cells), plasma cells and neutrophils. Their function is to prevent microorganisms from invading periradicular tissues. Both the pulp and periradicular inflammatory tissues have been shown to produce cellular and humoral responses to the microorganisms. Microbial invasion of periradicular tissues results in production of an abscess or cellulitis which presents the signs and symptoms because of both specific and nonspecific inflammation.

Several studies have shown that polymicrobial infections are found both in acute and chronic stages of periradicular infections. In many asymptomatic patients with chronic infection, sinus tracts have also been found. It has been seen that sinus tracts are associated with chronic periradicular abscess that always encompass a polymicrobial infection. They are relatively asymptomatic because the sinus tract provides a pathway of drainage.

Invasion of periradicular tissues is related to the virulence of the microorganisms and the host's resistance. Periapical inflammatory lesions are dynamic inflammatory events and may contain an abscess with bacteria, a cyst and surrounding inflammatory tissue simultaneously.

It is seen that most common cause of persistent periradicular infections is incomplete debridement of the root canal system (Fig. 4.8). Therefore three dimensional sealing of the root canal system is necessary for resolution of periapical pathologies.

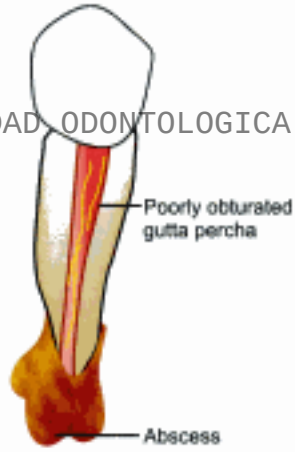


Fig. 4.8: Periradicular infection as a result of poorly obturated canal

MICROBIOLOGY OF ROOT CANAL FAILURES

The lack of periradicular healing following root canal treatment seems to be related to the persistence of microbes in the root canal system. This would appear to be related to an inability to effectively shape, clean, and seal the complete root canal system (Fig. 4.9). Interestingly, the microflora cultured from previously filled root canals with persistent apical lesions differs significantly for the microbes in untreated necrotic canals. Various researches have shown that microbiology of untreated canals is different from previously obturated canals.

Bacteria isolated from canals previously obturated but still associated with radiographic lesions tend to have more facultative bacteria rather than strict anaerobes. Instead of having approximately equal amounts of gram-negative and gram-positive bacteria, the bacteria isolated from previously obturated canals tend to have only one to two cultivable strains of mainly gram-positive bacteria. In several studies, previously filled canals had a relative increase in the presence of ***Enterococcus faecalis***.

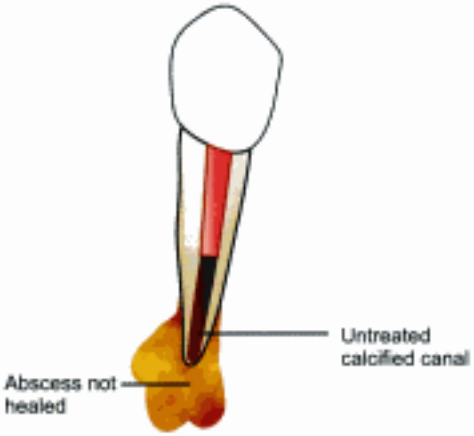


Fig. 4.9: Non-healing of periapical lesion because of untreated canal

Recently a study using PCR found the presence of ***E. faecalis***, ***Propionibacterium alactolyticus***, ***P. propionicum*** in previously filled root canal failure cases.

Filofactor alocis and Dialiste pneumosintes have not been isolated from previously filled root canals with periradicular lesions. They are anaerobic bacteria recently shown to be prevalent in untreated root canals. The presence of bacteria in all of the failed root canals supports the assertion that the vast majority of endodontic treatment failures are caused by intraradicular infections.

ENDODONTIC ABSCESSES AND CELLULITIS

The extent of endodontic infections beyond the root canal system is related to virulence of the bacteria, host response, and associated anatomy. Infections may localize or continue to spread. An abscess is a cavity of purulent exudates consisting primarily of bacteria, bacterial by-products, inflammatory cells, lysed cells and their contents. The content of inflammatory cells includes enzymes, which are damaging to the surrounding tissues. Endodontic abscesses are polymicrobial infections with organisms similar to those found in infected root canals. A diffuse cellulitis may have foci of abscesses. The spread of infection and ensuing edema associated with inflammation often produces an indurated swelling. Over the time, neutrophils accumulate and produce a fluctuant abscesses. This concept supports the rationale for early incision for drainage to provide a pathway for the drainage of bacteria, bacterial by-products, and inflammatory mediators.

IDENTIFICATION OF THE BACTERIA

Culture

Culture taking method though done less these days, but it still holds its importance because of wide range of bacteria found in the endodontic infections. The empirical administration of antibiotics may not produce satisfactory results, in such cases, culturing may provide a valuable information for better antibiotic selection. Thus we can say that with supportive information from culturing, an intelligent alteration in antibiotic therapy can be made.

Technique

1. The fluctuant space of abscess is palpated and the most dependent part of swelling is determined.

2. Mucosa in that area is disinfected with betadine or chlorhexidine.
3. An empty, sterile, syringe and attached 16 to 20 gauge needle is used to aspirate the exudate.
4. Sample is immediately injected into a container with prereduced transport media.
5. Sample should never be exposed to room air.
6. Gram staining is performed on the sample to determine type of microorganism.
7. Strict anaerobes not only require an atmosphere devoid of oxygen, but also require substances for growth which are not found in simply broths and agars. So the sample should be cultured accordingly.
8. Try to gain the results at the earliest possible in order to select antibiotics.
9. This holds great importance for medically compromised patients regarding the selection of antibiotics.

Disadvantages of Culturing Method

1. Unable to grow several microorganisms which can give false negative results
2. Strictly depend on mode of sample transport which must allow growth of anaerobic bacteria
3. Low sensitivity and specificity
4. Time consuming
5. Expensive and laborious.

To overcome these disadvantages, various molecular diagnostic methods have developed. These include DNA-DNA hybridization method, polymerase chain reaction method.

DNA-DNA Hybridization Method

This method uses DNA probes which target genomic DNA or individual genes. This method helps in simultaneous determination of the presence of a multitude of bacterial species in single or multiple clinical samples and is especially useful for large scale epidemiologic research.

Polymerase Chain Reaction Method (PCR Method)

PCR method involves in vitro replication of DNA, therefore it is also called as **"*genetic xeroxing*"** method. Multiple copies of specific region of DNA are made by repeated cycles or heating and cooling.

PCR has remarkable sensitivity and specificity because each distinct microbial species has unique DNA sequences.

PCR can be used to detect virtually all bacterial species in a sample. It is also used to investigate microbial diversity in a given environment. Clonal analysis of microorganisms can also be done by PCR method.

Advantages of Molecular Methods

Molecular methods have several advantages over the culturing methods. The molecular methods:

1. Can be helpful in detection of both cultivable and uncultivable microbial species
2. Are more sensitive tests
3. Have greater specificity
4. Are less time consuming
5. Do not need special control for anaerobic bacteria
6. Are useful when a large number of samples are needed to be analyzed for epidemiologic studies.

HOW TO COMBAT MICROBES IN THE ENDODONTIC THERAPY

The microbial ecosystem of an infected root canal system and inflammatory response caused by it will persist until source of irritation is completely removed. The main factor which is needed for successful treatment of pulp and periradicular inflammation is complete removal of the source of infection such as microorganisms and their by products etc.

Following measures should be taken to completely rid of these irritants:

1. ***Thorough cleaning and shaping of the root canal system***: Root canal system is thoroughly cleaned and shaped to remove the bacteria and substrates which can support microorganisms. Thorough cleaning and shaping followed by three dimensional obturation of the root canals have shown to produce complete healing of periradicular tissue (Fig. 4.10). Complete debridement of canal should be done with adjunctive use of irrigants like sodium hypochlorite which efficiently removes bacteria as well as their substrate from irregularities of canal system where instruments cannot reach such as fins, indentations, cul-de-sacs etc.

   NaOCl is considered an excellent antimicrobial agent with tissue dissolving properties. It can be used alone or in combination with other irrigants like chlorhexidine, EDTA, hydrogen peroxide etc.
2. ***Oxygenating a canal*** simply by opening it is detrimental to anaerobes. Use of oxygenating agents as Glyoxide can be of great help but care should be taken to avoid

Fig. 4.10: **A.** Infection of pulp which has progressed to alveolar abscess, **B.** Complete cleaning and shaping of root canal system, **C.** Successful root canal therapy and healed periapical bone

inoculation of these oxygenating agents into periapical tissues.

3. A tooth with serous or purulent or hemorrhagic exudate should be allowed to ***drain*** with rubber dam in place for a time under supervision. An abscess which is a potent irritant, has an elevated osmotic pressure. This attracts more tissue fluid and thus more edema and pain. Drainage by canal or by soft tissues decrease discomfort caused by inflammatory mediators.
4. ***Antibiotics*** should also be considered as adjunctive in severe infections. The choice of antibiotic agent should be done on the knowledge of microorganisms associated with the endodontic infections. The drug of choice for endodontic infections is the Penicillin VK because of its spectrum of microbial activity against most of the bacteria associated with endodontic infections and also because of its low toxicity.

   Other antibiotics which can be given to combat endodontic infections include amoxicillin because of its wider spectrum than penicillin, clarithromycin, azithromycin and metronidazole for their action against most of the anaerobes found in root canals. If patient is allergic to penicillin, clindamycin is recommended. It is effective against anaerobic bacteria, both obligate as well as facultative.
5. ***Intracanal medicaments*** play an important role in combating the microorganisms.
6. ***Use of calcium hydroxide in canals*** with necrotic pulps after instrumentation have shown to provide the beneficial results. Intracranial use of calcium hydroxide have shown to increase the efficiency of sodium hypochlorite and also the effectiveness of antimicrobial agent. Calcium hydroxide powder is mixed with water or glycerin to form a thick paste which is placed in pulp chamber with amalgam carrier or a syringe. This paste is covered with a sterile cotton pellet and access is sealed with temporary restoration.

Thus we can say that for successful endodontic outcome, one must have awareness of the close relationship between endodontic infections and microorganisms.

5 Diagnostic Procedures

- Introduction
- Case History
- Radiographs
- Pulp Vitality Tests
- Recent Advances in Pulp Vitality Testing
- Cracked Tooth Syndrome
- Diagnostic Perplexities
- Role of Radiographs in Endodontics
- Digital Dental Radiology
- RVG

INTRODUCTION

Diagnosis is defined as utilization of scientific knowledge for identifying a diseased process and to differentiate from other disease process. In other words, literal meaning of diagnosis is determination and judgment of variations from the normal.

It is the procedure of accepting a patient, recognizing that he/she has a problem, determining the cause of problem and developing a treatment plan which would solve the problem. There are various diagnostic tools of diagnosis, out of all these, art of listening is most important. It also establishes patient-doctor rapport, understanding and trust.

Although diagnostic testing of some common complaints may produce classic results but sometimes tests may produce wrong results, which need to be carefully interpreted by clinician.

The diagnostic process actually consists of ***four steps:***
First step: Assemble all the available facts gathered from chief complaints, medical and dental history, diagnostic tests and investigations.
Second step: Analyze and interpret the assembled clues to reach the tentative or provisional diagnosis.
Third step: Make differential diagnosis of all possible diseases which are consistent with signs, symptoms and test results gathered.
Fourth step: Select the closet possible choice.

The importance of making an accurate diagnosis cannot be overlooked. Many a times even after applying all the knowledge, experience and diagnostic tests, a satisfactory explanation for patient's symptoms is not determined. In many cases, nonodontogenic etiology is also seen as a source of chief complaint. To avoid irrelevant information and to prevent errors of omission in clinical tests, the clinician should establish a routine for examination, consisting of chief complaint, past medical and dental history and any other relevant information in the form of case history.

CASE HISTORY

The purpose of case history is to discover whether patient has any general or local condition that might alter the normal course of treatment. As with all courses of treatment, a comprehensive medical and previous dental history should be recorded. In addition, a description of the patient's symptoms in his or her own words should be noted.

Chief Complaint

It consists of information which promoted patient to visit a clinician. Symptoms are phenomenon or signs of deviation from normal and are indicative of illness. The form of notation should be in patient's own words.

History of Present Illness

Once the patient completes information about his/her chief complaint, a report is made which provides more descriptive analysis about this initial information. It should include signs and symptoms, duration, intensity of pain, relieving and exaggerating factors, etc. Examples of type of the questions which may be asked by the clinician in recording the patient's complaints are as below:

1. How long have you had the pain?
2. Do you know which tooth it is?
3. What initiates pain?
4. How would you describe the pain?
   a. Quality—Dull, Sharp, Throbbing, Constant
   b. Location—Localized, Diffuse, Referred, Radiating
   c. Duration—Seconds, Minutes, Hours, Constant
   d. Onset—Stimulation Required, Intermittent, Spontaneous
   e. Initiated—Cold, Heat, Palpation, Percussion
   f. Relieved—Cold, Heat, Any medications, Sleep

In other words, history of present illness should indicate severity and urgency of the problem.

If a chief complaint is toothache but symptoms are too vague to establish a diagnosis, then analgesics should be prescribed to help the patient in tolerating the pain until the toothache localizes. A history of pain which persists without exacerbation may indicate problem of nonodontogenic origins.

The most common toothache may arise from either pulp or periodontal ligament. Pulpal pain can be sharp piercing if A delta fibers are stimulated. Dull, boring or throbbing pain occurs if there is stimulation of C-fibers. Pulp vitality tests are usually done to reach the most probable diagnosis. If pain is from periodontal ligament, the tooth will be sensitive to percussion, chewing and palpation. Another hint that pain is of pulpal origin is its intensity. Patient is asked to mark the imaginary ruler with grading ranging from 0-10.

Mild to moderate pain can be of pulpal or periodontal origin but acute pain is commonly a reliable sign that pain is of pulpal origin. Localization of pain also tells origin of pain since pulp does not contain proprioceptive fibres; it is difficult for patient to localize the pain unless it reaches the periodontal ligament.

Medical History

There are no medical conditions which specifically contraindicate endodontic treatment, but there are several which require special care. **Scully** and **Cawson** have given a checklist of medical conditions which are needed to be taken a special care.

Checklist for Medical History (Scully and Cawson)

- Anemia
- Bleeding disorders
- Cardiorespiratory disorders
- Drug treatment and allergies
- Endocrine disease
- Fits and faints
- Gastrointestinal disorders
- Hospital admissions and attendance
- Infections
- Jaundice
- Kidney disease
- Likelihood of pregnancy or pregnant itself

If there is any doubt about the state of health of patient, consult medical practitioner before initiating endodontic treatment. Care should also be taken whether patient is on medication such as corticosteroids or anticoagulant therapy.

According to standards of American heart association, patient should be given antibiotic prophylaxis if there is high rise of developing bacterial endocarditis for example in cardiac conditions like prosthetic heart valves, rheumatic heart disease, previous bacterial endocarditis and complex cyanotic heart diseases.

Extra Oral Examination

Extra oral examination begins as soon as patient enters in the clinic and patient should be observed for unusual gait and habits, etc. which may suggest underlying systemic disease, drug or alcohol abuse if any.

Patient should be looked for any facial asymmetry or distention of tissues (Fig. 5.1). Dentist must examine any localized swelling, presence of bruises, abrasions, scars or any other signs of trauma if present. Patient should be looked for size of pupils which may signify systemic disease, premeditation or fear.

After extra oral examination of head and neck region, one should go for extra oral palpation. If any localized swelling is present, then look for:

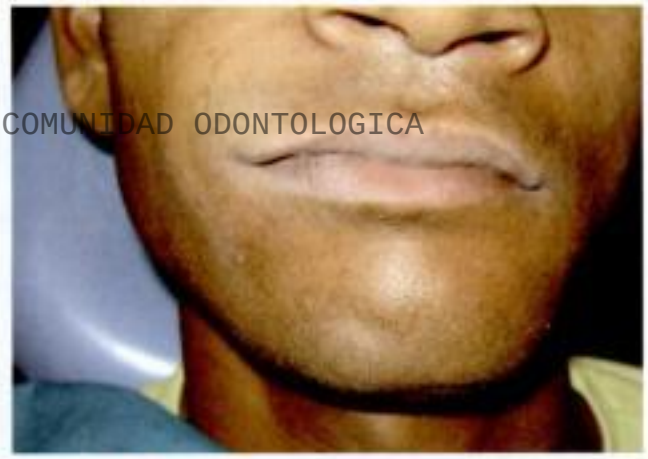


Fig. 5.1: Patient should be examined for facial asymmetry and swelling

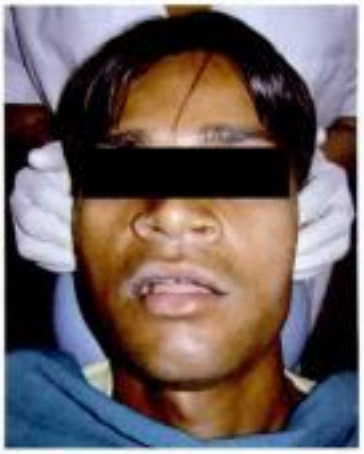


Fig. 5.3: Examination of TMJ

- Local rise in temperature
- Tenderness
- Extent of lesion
- Induration
- Fixation to underlying tissues etc.

Palpation of salivary glands should be done extra-orally. Submandibular gland should be differentiated from lymph nodes in the submandibular region by bimanual palpation (Fig. 5.2).

Palpation of TMJ can be done by standing in front of the patient and placing the index fingers in the preauricular region. The patient is asked to open the mouth and perform lateral excursion to notice (Fig. 5.3).

- Any restricted movement
- Deviation in movement
- Jerky movement
- Clicking
- Locking or crepitus

Palpation of lymph nodes should be done to note any lymph node enlargement, tenderness, mobility and consistency (Fig. 5.4). The lymph nodes frequently palpated are preauricular, submandibular, submental and cervical.

Lymph Nodes	*Probable Condition*
Tender, mobile, enlarged	Acute infection
Non-tender, mobile, enlarged	Chronic infection
Matted, Non-tender	Tuberculosis

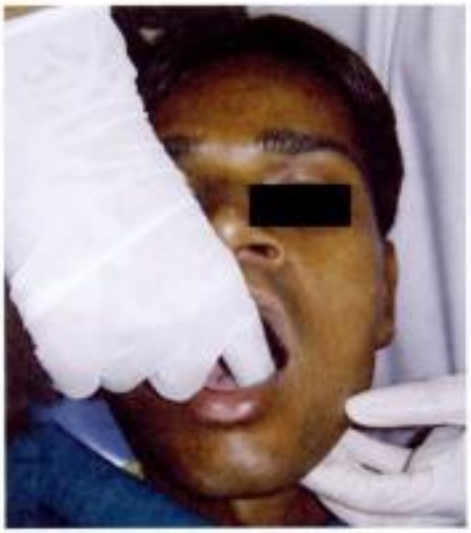


Fig. 5.2: Bimanual palpation of submandibular gland

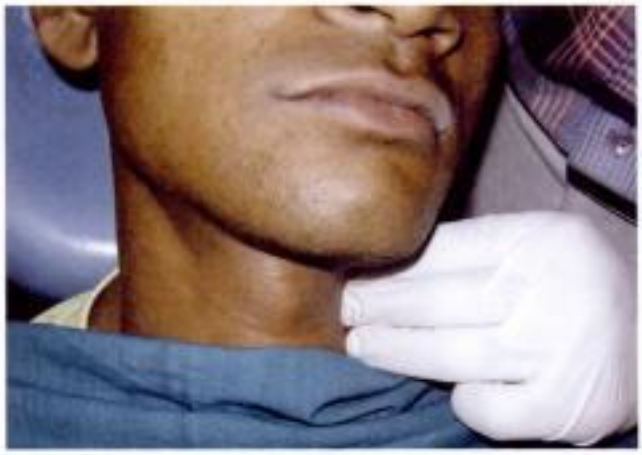


Fig. 5.4: Examination of lymph nodes

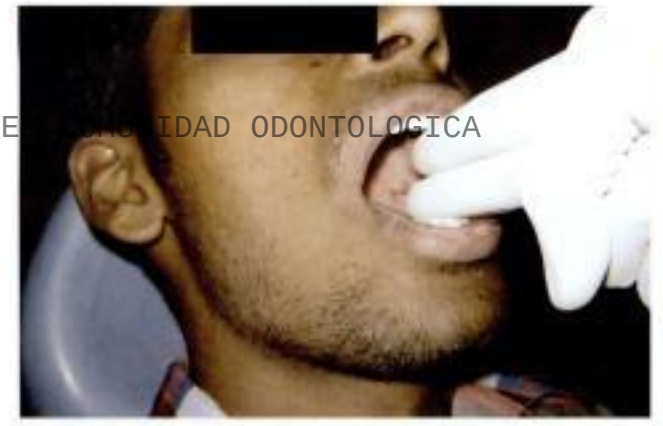


Fig. 5.5: Degree of mouth opening in a normal patient should be at least two fingers

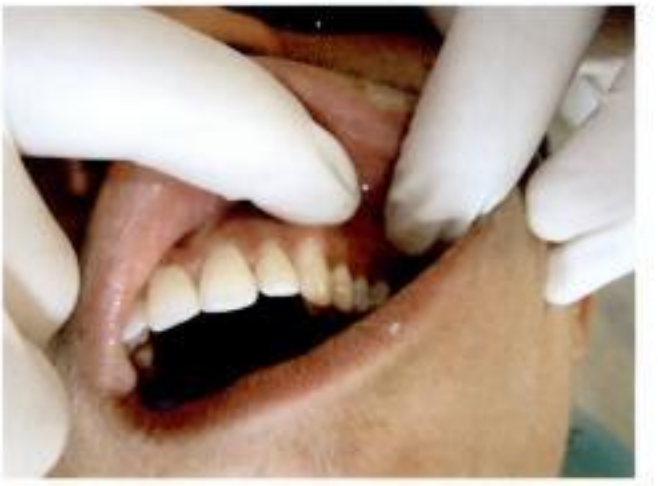


Fig. 5.6: Palpation of soft tissue using digital pressure

Intra Oral Examination

Before conducting intraoral examination check the degree of mouth opening. For a normal patient it should be at least two fingers (Fig. 5.5). During intra oral examination, look at the following structures systematically:

1. The buccal, labial and alveolar mucosa
2. The hard and soft palate
3. The floor of the mouth and tongue
4. The retromolar region
5. The posterior pharyngeal wall and facial pillars
6. The salivary gland and orifices

After examining this, ***general dental state*** should be recorded, which include:

a. Oral hygiene status
b. Amount and quality of restorative work
c. Prevalence of caries
d. Missing tooth
e. Presence of soft or hard swelling
f. Periodontal status
g. Presence of any sinus tracts
h. Discolored teeth
i. Tooth wear and facets

If patient's chief complaint includes symptoms which occur following specific events like chewing and drinking cold liquids, then specific intra oral examination should include tests which reproduce these symptoms. This will be helpful in establishing the diagnosis. For accurate diagnosis, evaluation of history, examination and clinical tests should be done properly. Clinical examination of tissue is done by palpation, percussion and other endodontic clinical tests.

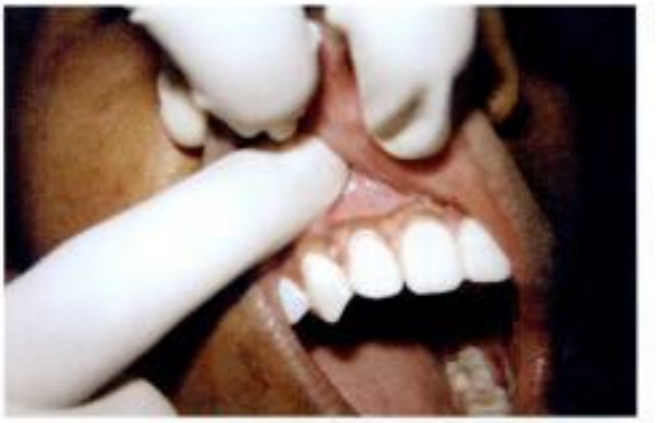


Fig. 5.7: Palpation of soft tissues

Palpation is done using digital pressure to check any tenderness in soft tissue overlying suspected tooth (Fig. 5.6). Sensitivity may indicate inflammation in periodontal ligament surrounding the affected tooth. Further palpation can tell any other information about fluctuation or fixation or induration of soft tissue, if any (Fig. 5.7).

Percussion of tooth indicates inflammation in periodontal ligament which could be due to trauma, sinusitis and/or PDL disease.

Percussion can be carried out by gentle tapping with gloved finger (Fig. 5.8) or blunt handle of mouth mirror (Fig. 5.9). Each tooth should be percussed on all the surfaces of tooth until the patient is able to localize the tooth with pain. Degree of response to percussion is directly proportional to degree of inflammation.

Periodontal evaluation can be assessed from palpation, percussion, mobility of tooth and probing (Figs 5.10 and

5.11). The mobility of a tooth is tested by placing a finger (Fig. 5.12) or blunt end of the instrument (Fig. 5.13) on either side of the crown and pushing it and assessing any movement with other finger.

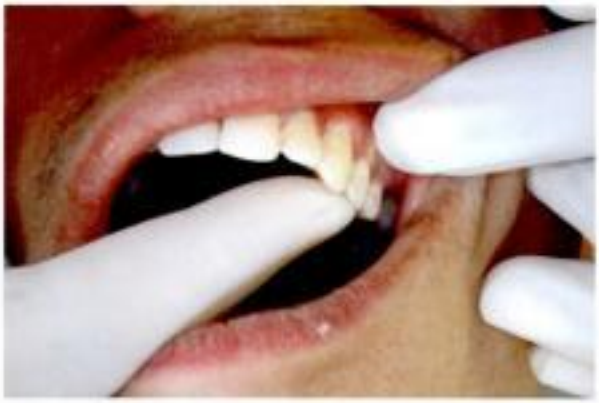


Fig. 5.8: Percussion of tooth using gloved finger

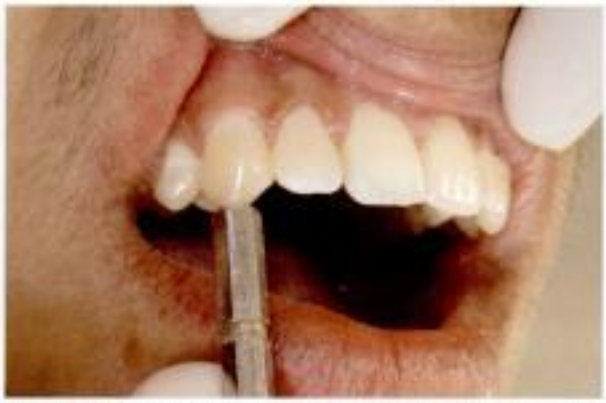


Fig. 5.9: Percussion of tooth using blunt handle of mouth mirror

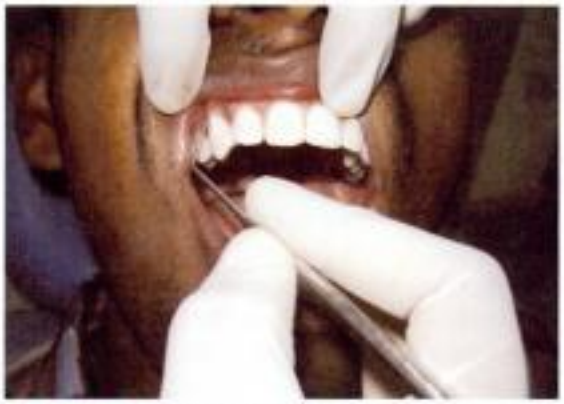


Fig. 5.10: Probing of a tooth determines the level of connective tissue attachment

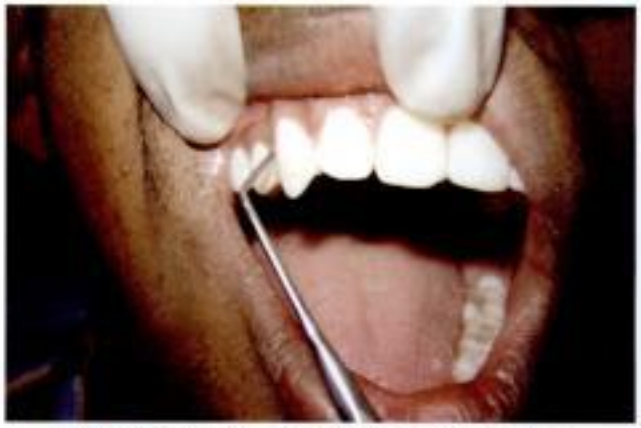


Fig. 5.11: Probing interdentally can identify any rough or overextended proximal restoration

Fig. 5.12: Checking mobility of a tooth by palpating with fingers

Fig. 5.13: Checking mobility of a tooth using blunt end of instrument

Mobility can be graded as:

1. Slight (normal)
2. Moderate mobility within a range of 1 mm.

3. Extensive movement (more than 1 mm) in mesiodistal or lateral direction combined with vertical displacement in alveolus.

RADIOGRAPH

The radiograph is one of most important tools in making a diagnosis. Without radiograph, case selection, diagnosis and treatment would be impossible as it helps examination of oral structure that would otherwise be unseen by naked eye.

In all endodontic cases, a good intra oral radiograph is mandatory as it gives excellent details and help in diagnosis and treatment planning (Fig. 5.14). They help to diagnose tooth related problems like caries, fractures, root canal treatment or any previous restorations, abnormal appearance of pulpal or periradicular tissues, periodontal diseases and the general bony pattern. Sometimes the normal anatomic landmarks like maxillary antrum, foraminas, tori, inferior alveolar canal etc may be confused with endodontic pathologies which may result in wrong diagnosis and thus improper treatment.

Generally the ***periapical lesions of endodontic origin have following characteristic features***:

- Loss of lamina dura in the apical region
- Etiology of pulpal necrosis is generally apparent
- Radiolucency remains at the apex even if radiograph is taken by changing the angle.

Radiographs help us in following ways:

a. Establishing diagnosis
b. Determining the prognosis of tooth
c. Disclosing the presence and extent of caries
d. Check the thickness of periodontal ligament
e. To see presence or absence of lamina dura
f. To look for any lesion associated with tooth

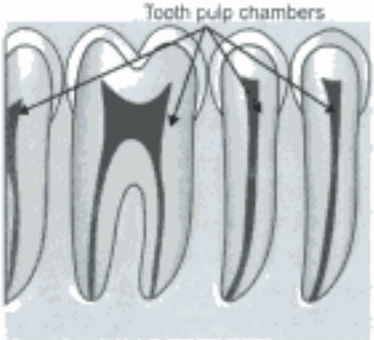


Fig. 5.14: A good radiographic image can detect normal and abnormal structures associated with teeth

g. To see the number, shape, length and pattern of the root canals
h. To check any obstructions present in the pulp space.
i. To check any previous root canal treatment if done
j. To look for presence of any intraradicular pins or posts
k. To see the quality of previous root canal filling
l. To see any resorption present in the tooth
m. To check the presence of calcification in pulp space
n. To see root end proximal structures
o. Help in determining the working length, length of master gutta percha cone and quality of obturation
p. During the course of treatment they help in knowing the level of instrumental errors like perforation, ledging and instrumental separation.

Lesions which commonly involve the periodontal ligament space include the periapical lesions which can be evident at apical part or at the lateral surface of the root, perforation lesions and fractured root lesions.

Following lesions should be differentiated from the lesions of endodontic origin while interpreting radiographs:

- Periodontal abscess
- Idiopathic osteosclerosis
- Cementomas
- Giant cell lesions
- Cysts
- Tumors
- Infections

Though the radiographs play an important role in dentistry but they have a few ***shortcomings:***

a. They are only two dimensional picture of a three dimensional object.
b. Pathological changes in pulp are not visible in radiographs
c. The initial stages of periradicular diseases produce no changes in the radiographs.
d. They don't help in exact interpretation for example radiographic picture of an abscess, inflammation and granuloma is almost same.
e. Misinterpretation of radiographs can lead to inaccurate diagnosis.
f. Radiographs can misinterpret the anatomical structures like incisive and mental foramen with periapical lesions
g. To know the exact status of multirooted teeth, multiple radiographs are needed at different angles which further increase the radiation exposure.

It must be emphasized that though radiographs play a critical role in diagnosis of an endodontic case but a poor

quality radiograph not only fails to yield diagnostic information but also causes unnecessary radiation to the patient.

PULP VITALITY TESTS

Pulp testing is often referred to as vitality testing. Pulp vitality tests play an important role in diagnosis because these tests not only determine the vitality of tooth but also the pathological status of pulp. Pulp testers should only be used to assess the vital or non-vital pulp as they do not quantify the disease, nor do they measure the health and thus, should not be used to assess the degree of pulpal disease.

Various types of pulp tests performed are:

1. Thermal test
   a. Cold test
   b. Heat test
2. Electrical pulp testing
3. Test cavity
4. Anesthesia testing
5. Bite test

1. Thermal Test

In thermal test, the response of pulp to heat and cold is noted. The basic principle for pulp to respond to thermal stimuli is that patient reports sensation but it disappears immediately. Any other type of response, i.e. painful sensation even after removal of stimulus or no response are considered abnormal.

a. Cold Test

It is the most commonly used test for assessing the vitality of pulp. It can be done in a number of ways. The basic step of the pulp testing, i.e. individually isolating the tooth with rubber dam is mandatory with all types. Use of rubber dam is specially recommended when performing the test using the ice-sticks because melting ice will run on to adjacent teeth and gingivae resulting in false-positive result.

1. The most commonly used method for performing pulp testing is ***spray with cold air*** directed against the isolated tooth.
2. The other frequently used method is application of cotton ***pellet saturated with ethyl chloride*** (Fig. 5.15).
3. The ***spray of ethyl chloride*** after isolating tooth with rubber dam is also employed. The ethyl chloride evaporates so rapidly that it absorbs heat and thus, cools the tooth.
4. The ***frozen carbon dioxide (dry ice)*** is reliable method of cold pulp testing. The frozen CO_2 is available in the form of solid sticks which is applied to the facial surface of the tooth. While testing with carbon dioxide, precaution should be taken to properly isolate the oral soft tissue and teeth with gauge or cotton roll so that CO_2 does not come into contact with these structures, because, of extremely low temperature of carbon dioxide, soft tissue burns may occur. The use of dry ice for pulp testing has been described by 'Ehrmann'. The advantage of using dry ice is that it can penetrate the full coverage restoration and can elicit a pulpal reaction to the cold because of its very low temperature (–69°F to –119°F/–56°C to –98°C)
5. One of the easy methods for cold test is to ***wrap an ice piece in the wet gauge*** and apply to the tooth. The ice sticks can also be prepared by filling the discarded anesthetic carpules with water and placing them in refrigerator.
6. ***Dichlorodifluoromethane (Freon)*** and the recently available material 1, 1, 1, 2-tetrafluoroethane are also used as cold testing material.

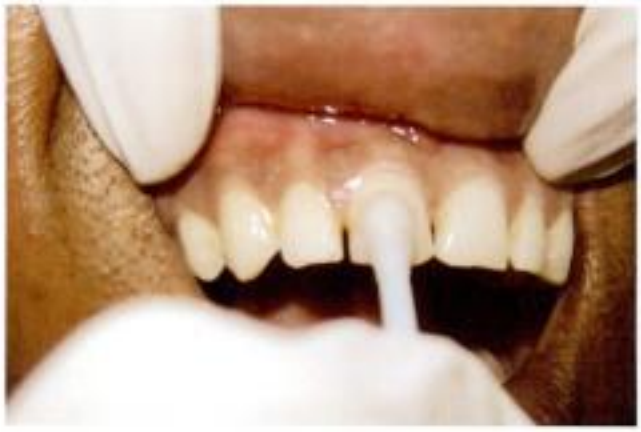


Fig. 5.15: Application of cotton pellet saturated with ethyl chloride

b. Heat Test

Heat test is most advantageous in the condition where patient's chief complaint is intense dental pain upon contact with any hot object or liquid. It can be performed using different techniques. Isolation of the area to be tested is the basic step for all the techniques.

1. The easiest method is to direct the ***warm air*** to the exposed surface of tooth and note the patient response.
2. If a higher temperature is needed to illicit a response, then other options like heated stopping stick, hot burnisher, hot water, etc. can be used.

Fig. 5.16: Application of heated gutta-percha stick on tooth for heat test

Among these, ***heated gutta-percha stick*** (Fig. 5.16) is most commonly used method for heat testing. In this method, tooth is coated with a lubricant such as petroleum jelly to prevent the gutta-percha from adhering to tooth surface. The heated gutta-percha is applied at the junction of cervical and middle third of facial surface of tooth and patient's response is noted. While using gutta-percha stick, precautions should be taken not to overheat it because in this state, it is at higher temperature than required for pulp testing and may result in pulpal injury.

3. The ***hot burnisher, hot compound*** or any other heated instrument may also be used for heat test (Fig. 5.17).
4. The other methods of heat testing is the use of ***frictional heat produced by rotating polishing rubber disc*** against the tooth surface.
5. One more method of heat test is to ***deliver warm water*** from a syringe on to the isolated tooth to determine the pulpal response. This method is especially useful for teeth with porcelain or full-coverage restoration. It also stimulates the existing conditions experienced by patient on taking hot foods or liquids.

[The preferred temperature for heat test is 150°F (65.5°C)]

The patient may respond to heat or cold test in following possible ways:

Mild, transitory response to stimulation show normal pulp. Absence of response in combination with other tests indicates pulp necrosis. An exaggerated and lingering response indicate irreversible pulpitis.

But there are certain ***conditions which can give false negative response,*** i.e. the tooth show no response but the pulp could be possibly vital. These conditions can be:

1. Recently erupted teeth with immature apex—due to incompletely developed plexus of Rashkow. Hence, incapable of transmitting pain.
2. Recent trauma—injury to nerve supply at the apical foramen or because of inflammatory exudates around the apex may interfere the nerve conduction
3. Excessive calcifications may also interfere with the nerve conduction.
4. Patients on premedication with analgesics or tranquillizers may not respond normally.

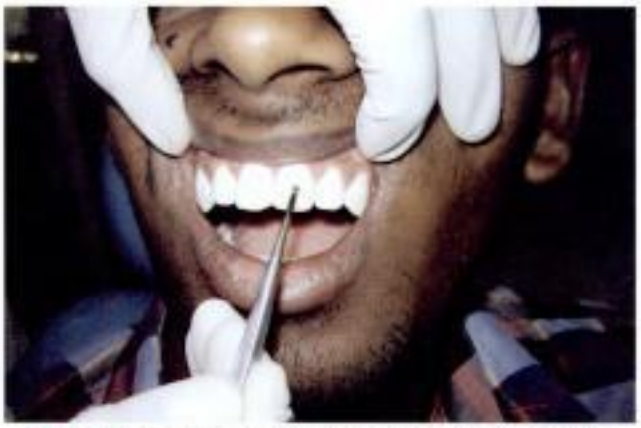


Fig. 5.17: Application of hot burnisher to check vitality of tooth

2. Electric Pulp Testing

Electric pulp tester is used for evaluation of condition of the pulp by electrical excitations of neural elements within the pulp. The pulp tester is an instrument which uses the gradations of electrical current to excite a response from the pulpal tissue. Pulp testers are available with cord which plug into electric outlets for power source (Fig. 5.18) they can also be available as battery operated instrument (Fig. 5.19). A positive response indicates the vitality of pulp. No response indicates non vital pulp or pulpal necrosis.

Fig. 5.18: Electric pulp tester

Fig. 5.19: Battery operated pulp tester

Procedure

1. Before starting the procedure, patient must be explained about the method. This will be helpful in reducing the anxiety of patient.
2. Isolation of the teeth to be tested is one of the essential steps to avoid any type of false positive response. This can be done by using 2" × 2" gauge piece.
3. Apply an electrolyte on the tooth electrode and place it on the facial surface of tooth (Fig. 5.20). Precaution should be taken to avoid it contacting adjacent gingival tissue or restorations; this will cause false positive response.
4. One should note that there should be a complete circuit from electrode through the tooth, to the body of the patient and then back to the electrode. If gloves are not used, the circuit gets completed when clinician's finger contact with electrode and patient's cheeks. But with gloved hands, it can be done by placing patient's finger on metal electrode handle or by clipping a ground attachment on to the patient's lip.
5. Once the circuit is complete, slowly increase the current and ask the patient to point out when the sensation occurs.
6. Each tooth should be tested 2-3 times and the average reading is noted. If the vitality of a tooth is in question, the pulp tester should be used on the adjacent teeth and the contralateral tooth, as control.

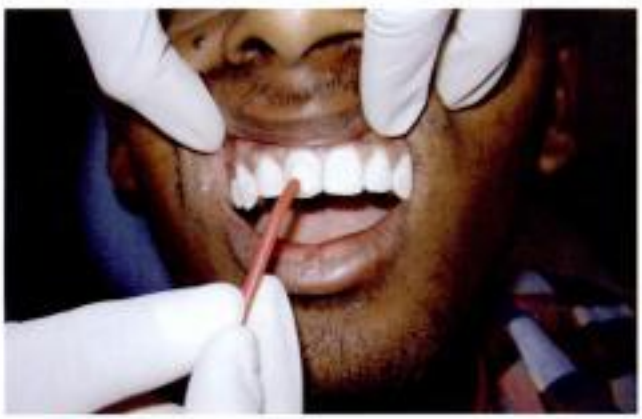


Fig. 5.20: Checking vitality of tooth using electric pulp tester

Disadvantages of Electric Pulp Testing

Electric pulp testing should not be considered solely for determination of pulp vitality; it should be used in combination with other tests. Various conditions can give rise to wrong results and thus misdiagnosis. These conditions can be as follows:

1. In teeth with acute alveolar abscess, false positive response is seen because gaseous or liquefied products within the pulp canal can transmit electric current.
2. Electrode may contact gingival tissue thus giving the false positive response.
3. In multirooted teeth, pulp may be vital in one or more root canals and necrosed in others, thus eliciting a false positive response.
4. In certain conditions, it can give false negative response for example:
   a. Recently traumatized tooth
   b. Recently erupted teeth with immature apex.
   c. Patients with high pain threshold
   d. Calcified canals
   e. Poor battery or electrical deficiency in plug in pulp testers.
   f. Teeth with extensive restorations or pulp protecting bases under restorations
   g. Patients premedicated with analgesics or tranquilizers etc.
   h. Partial necrosis of pulp some times is indicated as totally necrosis by electric pulp tester.

3. Test Cavity

This method should be used only when all other test methods are inconclusive in results. Here a test cavity is made with high speed number 1 or 2 round burs with appropriate air and water coolant. The patient is not anesthetized while performing this test. Patient is asked to respond if any painful sensation occurs during drilling. The sensitivity or the pain felt by the patient indicates pulp vitality. Here the procedure is terminated by restoring the prepared cavity. If no pain is felt, cavity preparation may be continued until the pulp chamber is reached and later on endodontic therapy may be carried out.

4. Anesthesia Testing

When patient is not able to specify the site of pain and when other pulp testing techniques are inconclusive, the selective anesthesia may be used. The main objective of this test is to anesthetize a single tooth at a time until the

pain is eliminated. It should be accomplished by using intraligamentary injection. Injection is administered to the most posterior tooth in the suspected quadrant. If the pain persists until that tooth has been fully anaesthetized, then repeat the procedure to the next tooth mesial to it. It is continued until the pain disappears. If source of pain can not be determined, repeat the same technique on the opposite arch.

5. Bite Test

This test helps if patient complains of pain on mastication. Tooth is sensitive to biting if pulpal necrosis has extended to the periodontal ligament space or if a crack is present in a tooth. In this patient is asked to bite on a hard object such as cotton swab, tooth pick or orange wood stick with suspected tooth and the contralateral tooth (Fig. 5.21). Tooth slooth is another commercially available device for bite test. It has a small concave area on its top which is placed in contact with the cusp to be tested (Fig. 5.22). Patient is asked to bite on it. Pain on biting may indicate a fractured tooth.

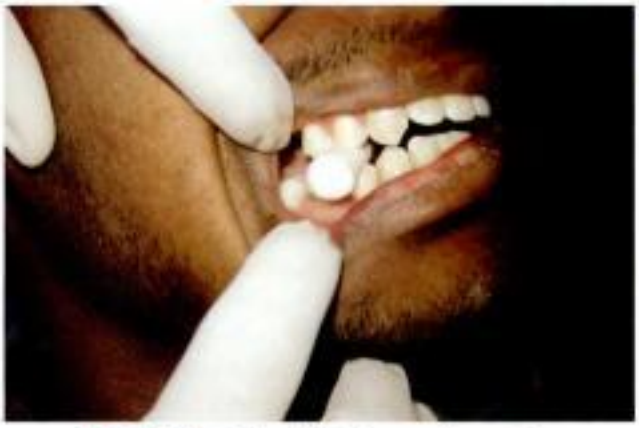


Fig. 5.21: Patient is asked to bite on cotton swab or hard object for bite test

Fig. 5.22: Tooth slooth

RECENT ADVANCES IN PULP VITALITY TESTING

The assessment of pulp vitality is a crucial diagnostic procedure in the practice of dentistry. Current routine methods rely on stimulation of A delta nerve fibers and give no direct indication of blood flow within the pulp. These include thermal stimulation, electrical or direct dentine stimulation. These testing methods have the potential to produce an unpleasant and occasionally painful sensation and inaccurate results. In addition, each is a subjective test that depends on the patient's perceived response to a stimulus as well as the dentist's interpretation of that response.

Recent studies have shown that blood circulation and not innervation is the most accurate determinant in assessing pulp vitality as it provides an objective differentiation between necrotic and vital pulp tissue.

Recently Available Pulp Vitality Tests Are:

- Laser Doppler flowmetry (LDF)
- Pulp oximetry
- Dual wavelength spectrophotometry
- Measurement of temperature of tooth surface
- Transillumination with fiber-optic light
- Plethysmography
- Detection of interleukin—1 beta
- Xenon—133
- Hughes probeye camera
- Gas desaturation
- Radiolabeled microspheres
- Electromagnetic flowmetry

Laser Doppler Flowmetry (LDF)

Laser Doppler flow meter was developed by Tenland in 1982 and later by Holloway in 1983. The technique depends on Doppler principle in which a low power light from a monochromatic laser beam of known wavelength along a fiber optic cable is directed to the tooth surface, where the light passes along the direction of enamel prisms and dentinal tubules to the pulp.

The light that contacts a moving object is Doppler shifted, and a portion of that light will be back scattered out of tooth into a photodetector. Some light is reflected off moving red blood cells in pulpal capillaries and as a consequence frequency broadened. The reflected light is passed back to the flow meter where the frequency broadened light, together with laser light scattered from static tissue, is photo-detected for strength of signal and pulsatility (Fig. 5.23).

Since, red blood cells represents the majority of moving objects within the tooth, measurements of Doppler shifted back scattered light may be interpreted as an index of pulpal blood flow.

The resulting photocurrent is processed to provide a blood flow measurement. The blood flow measured by laser Doppler technique is termed as 'flux', which is proportional to the product of average speed of blood cells and their concentration.

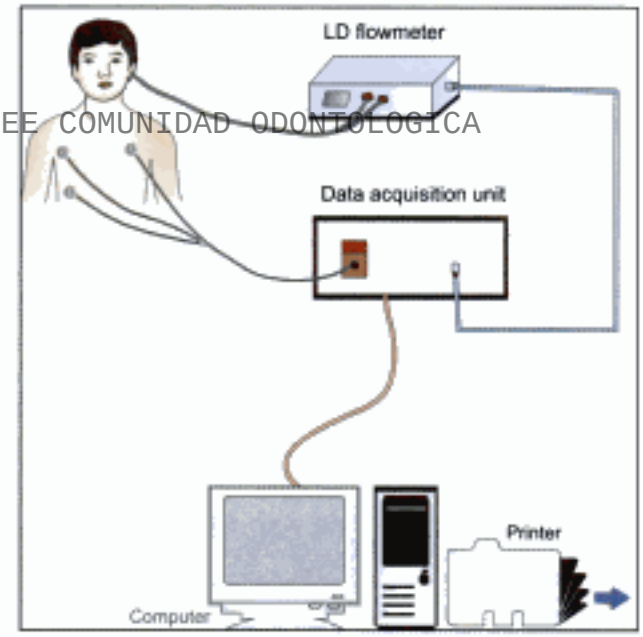


Fig. 5.23: Working of LDF

Pulp is a highly vascular tissue, and cardiac cycle blood flow in the supplying artery is transmitted as pulsations. These pulsations are apparent on laser doppler monitor of vital teeth and are absent in non-vital teeth. The blood flux level in vital teeth is much higher than for non-vital teeth. Currently available flow meters display the signal on a screen, from which the clinician can interpret whether pulp is vital or nonvital.

Advantages of Laser Doppler Flowmetry
1. An objective test
2. Accurate to check vitality

Disadvantages of Laser Doppler flowmetry
1. Cannot be used in patients who cannot refrain from moving or if tooth to be tested cannot be stabilized
2. Medications used in cardiovascular diseases can affect the blood flow to pulp
3. Requires higher technical skills to achieve
4. Use of nicotine also affect the blood flow to pulp
5. Expensive

Pulp Oximetry

Pulp oximetry is a non-invasive device for determining pulp vitality. The principle of this technology is based on modification of Beer's law and the absorbency characteristics of hemoglobin in red and infrared range.

The pulp oximeter is a non-invasive oxygen saturation monitor in which liquid crystal display oxygen saturation, pulse rate and plethysmographic wave form readings. The probe consists of red and infrared light-emitting diodes opposite a photoelectric detector. Clinically the detection of a pulse should be enough to establish pulp vitality or necrosis.

Pulp oximetry is especially helpful in cases of traumatic injury to the teeth during which nerve supply of the pulp may be injured, but the blood supply stays intact.

A distinctive advantage of this technique is its objectivity and lack of dependence on sensory response which eliminates the need for application of an unpleasant stimulus to the patient.

Advantages of Pulp Oximetry
1. Effective and objective method to evaluate pulp vitality
2. Useful in cases of traumatic injuries where the blood supply remains intact but nerve supply is damaged
3. Pulpal circulation can be detected independent of gingival circulation
4. Easy to reproduce pulp pulse readings
5. Smaller and cheaper pulp oximeters are now available

Disadvantages of Pulp Oximetry
1. Background absorption associated with venous blood.

Dual Wavelength Spectrophotometry

Dual wavelength spectrophotometry (DWLS) is a method independent of a pulsatile circulation. The presence of arterioles rather than arteries in the pulp and its rigid encapsulation by surrounding dentine and enamel make it difficult to detect a pulse in the pulp space. This method measures oxygenation changes in the capillary bed rather than the supply vessels and hence does not depend on a pulsatile blood flow.

Advantages of DWLS
1. In case of avulsed and replanted teeth with open apices where the blood supply is regained within first 20 days but the nerve supply lags behind. Repeated readings for 40 days in such teeth reveal the healing process.
2. It uses visible light which is filtered and guided to the tooth by fiber optics, unlike laser light where eye protection is necessary for patient and the operator.
3. Non-invasive test
4. An objective test
5. Instrument is small, portable and inexpensive

DWLS detects the presence or absence of oxygenated blood at 760 nm and 850 nm. The blood volume or concentration channel (760 nm plus 850 nm) is arranged to respond linearly to the increase in light absorption. The oxygenation channel (760 nm minus 850 nm) senses the oxygenated blood because of the greater absorption at 850 nm as compared to 760 nm.

Measurement of Surface Temperature of Tooth

This method is based on the assumption that if pulp becomes non-vital, the tooth no longer has internal blood supply, thus should exhibit a lower surface temperature than that of its vital counterparts.

Fanibund is 1985 showed that it is possible to differentiate by means of crown surface temperature, distinct difference between vital and non-vital teeth. He used a thermistor unit consisting of two matched thermistors connected back to back, one measuring the surface temperature of the crown (measuring thermistor) while the other acting as a reference thermistor. The tooth to be tested was dried with gauze and the thermistor unit was positioned so that the measuring thermistor contacted the center of the buccal surface of the crown. The reference thermistor was suspended in air, close to it, but not touching either the measuring thermistor or the enamel surface.

Equilibrium was next achieved between the temperatures of the thermistors, the crown surface and the immediate environment by holding the measuring unit in the described position until a steady state was established for at least 20 seconds. Stimulation of the crown surface was carried out by means of a rubber-polishing cup fitted to a dental contra-angle hand piece. The recordings were continued for a period of time following the stimulation period. It was found that a difference was obtained between the critical period for vital and non-vital teeth and the difference corresponded with a specific temperature change.

Transillumination with Fiber Optic Light

It is a system of illumination whereby light is passed through a finely drawn glass or plastic fibers by a process known as ***total internal reflection.***

By this method, a pulpless tooth that is not noticeably discolored may show a gross difference in translucency when a shadow produced on a mirror is compared to that of adjacent vital teeth.

Detection of Interleukin-I Beta in Human Periapical Lesion

The inflammatory periapical lesions are common sequelae of infected pulp tissue. Numerous cell types including PMN leucocytes, T and B lymphocytes, macrophages and plasma cells are found in these tissues.

These inflammatory cells produce interleukin-I (1L-1), which acts as a mediator of various immunologic and inflammatory responses.

This lymphocyte activating factor 1L-1 is responsible for osteoclast activation which results in bone resorption which is frequently a feature of inflammatory response.

Plethysmography

It is a method for assessing the changes in volume and has been applied to the investigation of arterial disease because the volume of the limb or organ exhibits transient changes over the cardiac cycle. Plethysmography in limb or digit can be performed using air filled cuffs or mercury in rubber strain gauges. As the pressure pulse passes through the limb segment, a wave form is recorded which relates closely to that obtained by intraarterial cannulation. The same principle can be used to assess tooth vitality. Presence or absence of a wave form can indicate the status of the tooth.

CRACKED TOOTH SYNDROME

The crack tooth syndrome means incomplete fracture of a tooth with vital pulp. The fracture commonly involves enamel and dentin but sometimes pulp and periodontal structure may also get involved (Fig. 5.24).

Etiology

The etiology of cracked tooth syndrome is not specific but is commonly seem to be associated in teeth with large and complex restorations, leaving the teeth more susceptible to cracks. Moreover stressful lifestyle, parafunctional habits and high masticatory forces are important contributing factors.

Fig. 5.24: Cracked teeth

Signs and Symptoms

- Erratic pain on mastication especially with release of biting pressure
- Sensitivity to thermal changes
- If pulp is involved, sign and symptoms of irreversible pulpits or pulpal necrosis with periapical pathology
- Generally, tooth is not tender on percussion

Diagnosis of Cracked Tooth Syndrome

The patient with cracked tooth syndrome gives history of variable signs and symptoms which are difficult to diagnose. Even the radiographs are inconclusive. The careful history of the patient, examination, diagnosis tests, radiographs and sometimes surgical exposure are needed for accurate diagnosis of cracked tooth syndrome.

i. History of the Patient

History of the patient includes:

a. A detailed history regarding dietary and parafunctional habits.
b. History of any previous trauma.

ii. Visual Examination

a. Look for any wear facets and steep cusps.
b. Check the cracked restorations or unusual gaps between restorations and tooth structure.
c. The removal of restorations is sometimes required for examination of cavity.

iii. Tactile Examination

During tactile examination pass the tip of sharp explore gently along the tooth surface, it may catch the crack.

iv. Periodontal Probing

Thorough periodontal probing along the involved, tooth may reveal a narrow periodontal pocket.

v. Bite Test

Orange wood stick, rubber wheel or the tooth slooth are commonly used for detection of cracked tooth. Tooth slooth is small pyramid shaped, plastic bite block with small concavity at the apex which is placed over the cusp and patient is asked to bite upon it with moderate pressure and release. The pain during biting or chewing especially upon the release of pressure is classic sign of cracked tooth syndrome.

vi. Transillumination

The use of fiber-optic light to transilluminate a fracture line is also method to diagnosis.

vii. Use of Dyes

Staining of fractured teeth with a dye such as methylene blue dye can aid in diagnosis. Dye can be directly applied to the tooth to identify fracture (Fig. 5.25), or it can be incorporated into a temporary restoration like ZOE and placed in the prepared cavity (Fig. 5.26) or patient can be asked to chew a disclosing tablet (Fig. 5.27). The dark stain present on the fracture line helps in detecting the fracture.

Fig. 5.25: Application of dye directly on tooth to identify fracture

Fig. 5.26: Incorporation of dye in temporary restoration to identify crack

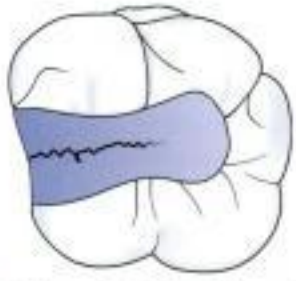


Fig. 5.27: Chewing a disclosing tablet can also identify cracked tooth

viii. Radiographs

1. Radiographs are not of much help especially, if, crack is mesiodistal in direction. Even the buccolingual cracks will only appear if there is actual separation of the segments or the crack happens to coincide with the X-ray beam.
2. Taking radiographs from more than one angle can help in locating the crack (Fig. 5.28).
3. A thickened periodontal ligament space, a diffused radiolucency especially with elliptical shape in apical area may indicate crack.

xi. Surgical Exposure

If a fracture is suspected, a full thickness mucoperiosteal flap should be reflected for visual examination of root surface.

Classification of Cracked Teeth

Cracked teeth can be classified on the basis of pulpal or periodontal involvement and the extent of crack.

Class A: Crack involving enamel and dentin but not pulp.
Class B: Crack involving pulp but not periodontal apparatus.
Class C: Crack extending to pulp and involving periodontal apparatus.
Class D: Complete division of tooth with pulpal and periodontal apparatus involvement.
Class E: Apically induced fracture.

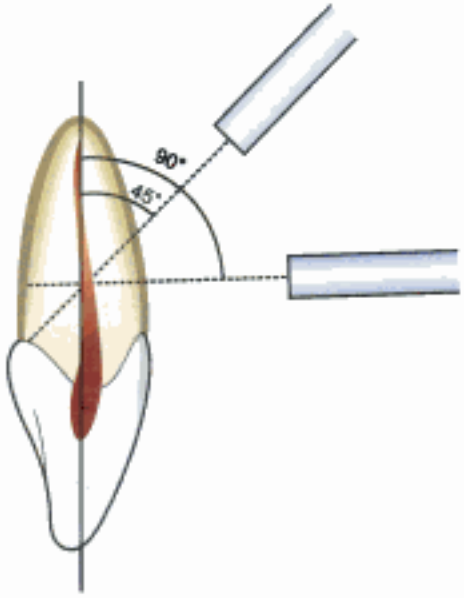


Fig. 5.28: Taking radiograph from more than one angle helps in locating the crack

Classification	*Involvement of enamel/dentin*	*Involvement of pulp*	*Involvement of periodontium*	*Prognosis*
Class A	Yes	No	May be	Excellent
Class B	Yes	Yes	No	Good
Class C	Yes	Yes	Yes	Average
Class D	Yes	Yes	Yes	Poor
Class E	Root Involvement	Yes	Yes	Poor

Differential Diagnosis of Cracked Tooth Syndrome

The crack is commonly invisible to naked eye and symptoms may vary; these may include pain on chewing, varied patterns of referred pain and sensitivity to thermal changes. Furthermore inconclusive radiographs make the diagnosis of cracked tooth difficult. There must be differentiation of a cracked tooth from a fractured cusp. The tooth crack occurs more towards the centre of the occlusal surface as compared to the cusp fracture which is more peripheral in position.

- If the crack has progressed to involve the pulp or periodontium, patient may have thermal sensitivity which lingers after removal of the stimulus or slight to very severe spontaneous pain consistent with irreversible pulpitis, pulp necrosis or apical periodontitis.
- When crack is mesiodistal across both the marginal ridges, splitting the tooth in two segments, patient may complain of pain on chewing and soreness of gums of the affected area. It should be differentiated from periodontal abscess.

Treatment of Cracked Teeth

The treatment plan for cracked teeth varies with location and extent of the crack. Even when crack can be located, the extent is still difficult to determine.

- Urgent care of the cracked tooth involves the immediate reduction of its occlusal contacts by selective grinding at the site of the crack or its antagonist.
- Definitive treatment of the cracked tooth aims to preserve the pulpal vitality by providing full occlusal coverage for cusp protection.
- When crack involves the pulpal floor, endodontic access is needed but one should not make attempts to chase down the extent of crack with a bur, because the crack may become invisible long before it terminates. Endodontic treatment can alleviate irreversible pulpal symptoms.

- If the crack is partially visible across the floor of the chamber, the tooth may be bonded with a temporary crown or orthodontic band. This will aid in determining the prognosis of the tooth and protect it from further deterioration till the endodontic therapy is completed (Fig. 5.29).

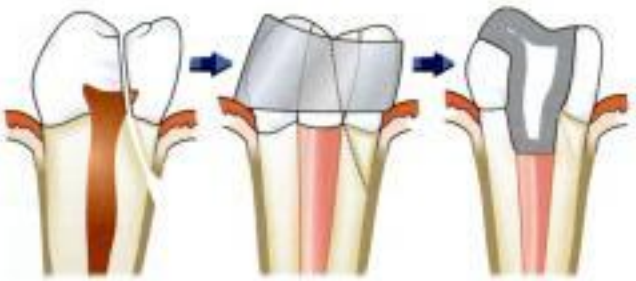


Fig. 5.29: Management of cracked tooth (a) Crack extending on to the root (b) Placing a matrix band and completing root canal treatment (c) Restoration of tooth using crown

- Apically extension and future migration of the crack apically onto the root determines the prognosis. If the fracture is not detected, pulpal degeneration and periradicular pathosis may be the initial indication, that complete vertical fracture is present. Depending upon the treatment may involve extractions, root resection, or hemisection.

To conclude, it can be stated that the diagnosis of a cracked tooth can be difficult, but it should be done without the sense of urgency, so as to alleviate the painful symptoms, initiate an appropriate treatment plan and to improve the prognosis of the cracked tooth.

Diagnostic Perplexities

There are certain conditions in which it is difficult to reach proper diagnosis even after detailed history and examination. These conditions can be:

- Idiopathic tooth resorption
- Treatment failures
- Cracked tooth syndrome
- Persistent discomfort
- Unusual radiographic appearances
- Paresthesia

DIAGNOSTIC FINDINGS

Once the patient has been evaluated and the clinical examination along with tests completed diagnosis is made. The findings of examination are arranged in a rational manner so as to reach at the pulpal or periapical diseases. Once the correct diagnosis is made, the treatment plan should be made. Basically the pulpal diseases can be reversible pulpitis, irreversible pulpitis or the necrotic pulp. The periapical diseases can be acute apical periodontitis, chronic apical periodontitis, acute or chronic apical abscess or condensing osteitis. These have been summarized in the table below.

Diagnostic findings of common dental problems

	Symptoms	*X-ray findings*	*Pulp vitality tests*
Reversible pulpitis	Asymptomatic or slight symptoms to thermal stimulus	No changes	Gives response to vitality tests
Irreversible pulpitis	Asymptomatic or may have spontaneous or severe pain to thermal stimuli	No changes, except in long standing cases condensing osteitis	Gives response
Pulp necrosis	None	Depends on periapex status	No response
Acute apical periodontitis	Pain on biting or pressure	Not significant	Depending on status of pulp, response or no response
Chronic apical periodontitis	Mild or none	Not significant	Depending on pulp status, response or no response
Acute apical abscess	Pain and/or swelling	Radiolucency at apical end	No response
Chronic apical abscess	Draining sinus	Radiolucency	No response
Condensing osteitis	Varies according to status of pulp or periapex	Increased trabecular bone	Depending on pulp status response or no response

ROLE OF RADIOGRAPHS IN ENDODONTICS

Radiographs play an important role in diagnosis of the dental diseases. The interpretation of radiographs should be done in a systematic manner. The clinician should be familiar with normal radiographic landmarks.

Normal radiographic landmarks are (Fig. 5.30):
Enamel: It is the most radiopaque structure.
Dentin: Slightly darker than enamel
Cementum: Similar to dentin in appearance
Periodontal ligament: Appears as a narrow radiolucent line around the root surface.

Fig. 5.30: Normal radiographic features of teeth

Lamina dura: It is a radiopaque line representing the tooth socket.

Pulp cavity: Pulp chamber and canals are seen as radiolucent lines within the tooth.

Though the radiographs play an important role in dentistry but they have a few shortcomings. For example pathological changes in pulp are not visible in radiographs, also the initial stages of periradicular diseases produce no changes in the radiographs. They are only two dimensional picture of a three dimensional object. It must be emphasized that a poor quality radiograph not only fails to yield diagnostic information but also causes unnecessary radiation to the patient.

To reduce the amount of radiation exposure and to improve the quality of radiograph, continuous efforts have been made since the discovery of X-ray in 1895.

In order to decrease the radiation exposure, certain newer methods have been introduced which include:

a. Use the paralleling technique instead of bisecting angle technique
b. Faster radiographic films.
c. Digital radiographic techniques.
d. Use of Electronic apex locator to assist in endodontic treatment.

History of Dental Radiology

1895	WC Roentgen	Discovery of X-rays
1896	O Walkhoff	First dental radiograph
1901	WH Rollins	Presented first paper on dangers of X-rays
1904	WA Price	Introduction of bisecting technique
1913	Eastman Kodak company	Introduction of pre-wrapped dental films
1920	Eastman Kodak company	Introduction of machine made film packets
1925	HR Raper	Introduction of bitewing technique
1947	FG Fitzgerald	Introduction of paralleling cone technique

Principles of Radiography

For diagnosis purposes in endodontics, the number of radiographs required, depends on situations. A properly placed film permits the visualization of approximately three teeth and at least 3-4 mm beyond the apex. In most of the cases, a single exposure is needed to get the information on root and pulp anatomy. Basically there are two types of techniques for exposing teeth viz; ***bisecting angle technique*** and ***paralleling technique.***

In ***bisecting angle technique*** (Fig. 5.31) the X-ray beam is directed perpendicular to an imaginary plane which bisects the angle formed by recording plane of X-ray film and the long axis of the tooth. This technique can be performed without the use of film holders, it is quick and comfortable for the patient when rubber dam is in place. But it also has certain disadvantages like incidences of cone cutting, image distortion, superimposition of anatomical structures and difficulty to reproduce the periapical films.

In ***paralleling technique*** (Fig. 5.32), the X-ray film is placed parallel to the long axis of the tooth to be exposed and the X-ray beam is directed perpendicular to the film. Various advantages of paralleling technique are:

- Better accuracy of image
- Reduced dose of radiation
- Reproducibility
- Better images of bone margins, interproximal regions and maxillary molar region

Disadvantages associated with this technique are difficult to use in patients with shallow vault, gag reflex and when rubber dam is in place.

Cone angulation is one of the most important aspects of radiography because it affects the quality of image. As we

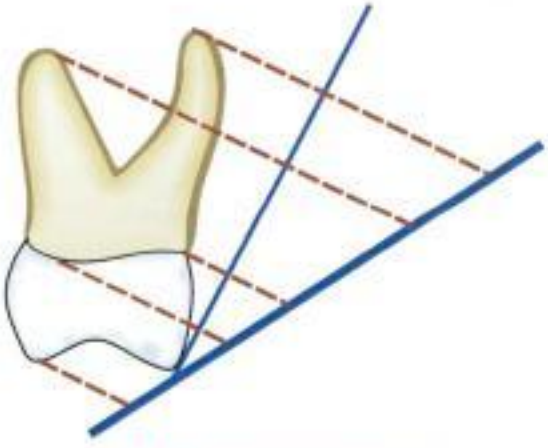


Fig. 5.31: Bisecting angle technique

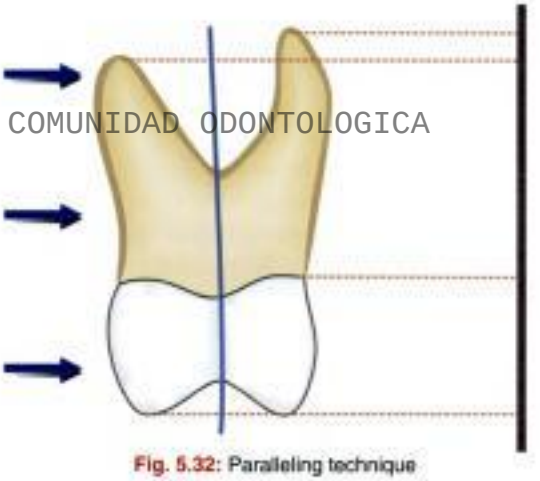


Fig. 5.32: Paralleling technique

have seen that paralleling technique has been shown to superior to bisecting angle technique especially in reproduction of apical anatomy of the tooth. As the angle increases away from parallel, the quality of image decreases. This happens because as the angle is increased, the tissue that the X-rays must pass through includes greater percentage of bone mass thus anatomy becomes less predictable. To limit this problem, **Walton** gave a ***modified paralleling technique*** in which central beam is oriented perpendicular to radiographic film but not to teeth. Modified paralleling technique is also beneficial for some special situations in which paralleling technique is not feasible, for example shallow palatal vault, maxillary tori, extremely long roots, uncooperative and gagging patient.

There are some special situations during endodontic therapy which require special considerations to obtain best quality of radiographs. These radiographs are neither parallel nor bisecting angle, but in such radiograph some modifications are made by varying the horizontal cone angle, cone placement and modified film placement. Commonly, modified paralleling technique is used in such radiographs.

Cone Image Shift Technique

An understanding of cone shift technique is essential to endodontic treatment. The proper application of this technique helps in distinguishing the objects which have been superimposed, differentiating the different types of resorption, and to locate additional number of roots or canals (Fig. 5.33). ***The main concept of technique*** is that as the vertical or horizontal angulations of X-ray tube head changes, the object buccal or closest to the tube head moves to opposite side of radiograph when compared to lingual object (Fig. 5.34). In other words, we can say that the cone image

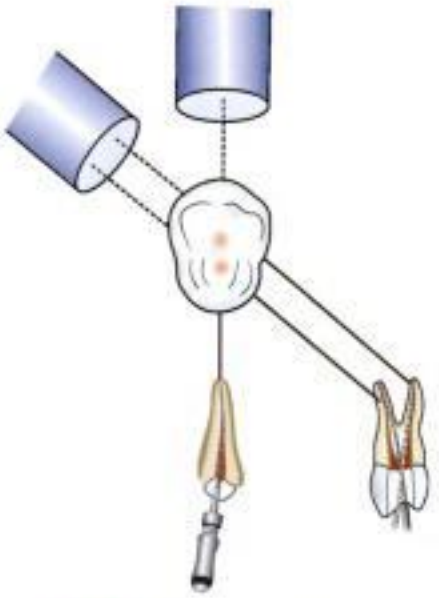


Fig. 5.33: Cone-shift technique

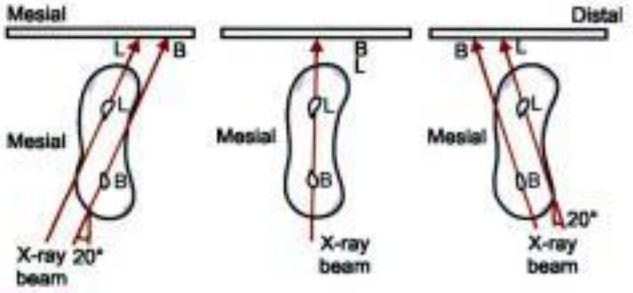


Fig. 5.34: As X-ray tube head changes, the object buccal or closet to the tube head moves to opposite side of radiograph when compared to lingual object

shift technique separates and identifies the facial and lingual structures.

As the cone position moves from parallel either towards horizontal or vertical, the object on the film shifts away from the direction of cone, i.e. in the direction of central beam.

In other words, when two objects and the film are in fixed position and the tube head is moved, images of both objects moving in opposite direction, the resultant radiograph shows lingual object that moved in the same direction as the cone and the buccal object moved in opposite direction. This is also known as "SLOB" rule (same lingual opposite buccal).

Synonyms of Cone Image Shift Technique
1. BOR (Buccal object rule)
2. SLOB (Same lingual opposite buccal)
3. BOMM (Buccal object moves most)
4. Clark's Rule
5. Walton's Projection

To simplify understanding of this rule, Walton gave an easy method. Place two fingers directly in front of open eyes so that one finger is superimposed on the other. By moving the head, from one way and the other, the position of finger, relative to each other shifts. The same effect is produced with two superimposed roots when centre beam is shifted.

Advantages of 'SLOB' Rule

1. It helps in separation of overlapping canals for example in case maxillary premolars and mesial canals of mandibular molars.
2. The working length radiographs are better traced from orifice to the apex by this technique.
3. It helps to locate the root resorptive processes in relation to tooth.
4. This technique is helpful in locating a canal in relation to radiopaque margin such as bur in the access opening.
5. It is useful in identification of anatomic landmarks and pathosis.
6. This rule is also used to increase the visualization of apical anatomy by moving anatomic landmarks such as Zygomatic process or the impacted tooth.
7. It also helps to identify the angle at which particular radiograph was made, even if information was not recorded
8. This technique is advantageous during access preparation. It helps to identify the missed canals or calcified canals and sometimes the canal curvature.

Disadvantages of 'SLOB' Rule

1. It results in blurring of the object which is directly proportional to cone angle. The clearest radiograph is achieved by parallel technique so when the central beam changes direction relative to object and the film, object become blurred.
2. It causes superimposition of the structures. Objects which have natural separation on parallel technique, with cone shift; they may move relative to each other and become superimposed. For example in case of maxillary molars, all three separate roots are visible on parallel radiographs but an angled radiograph may move palatal root over the distobuccal or mesiobuccal root and thus decreasing the ability to distinguish apices clearly.

FILM EXPOSURE AND QUALITY

Several choices are available regarding the film and processing. Studies have shown that there is no significant difference in the diagnostic quality of 'E' (EKTA speed) films when compared with 'D' films (Ultra speed). These, E plus films should be used to reduce the radiation exposure to the patient. The optimal setting for maximal contrast between their radiopaque and radiolucent structure is 70 kV. To maximize the clarity of the film, exposure time and milliamperage should be set individually on each machine. The rapid processing chemicals are also used in endodontic to expedite the development of the film for treatment radiograph.

FILM HOLDERS

Film holders are basically required for paralleling technique because their use decreases the distortion of X-rays caused by misorientation of film, tooth or X-ray beam (Fig. 5.35). The use of film holder has shown to provide more predictable results, this alignment device has following advantages:

1. True image of tooth, its length and anatomical features can be obtained

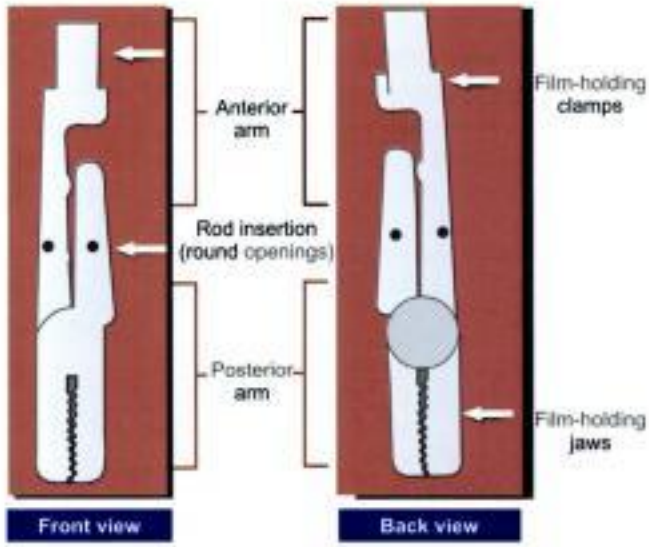


Fig. 5.35: Film holder

2. Subsequent films taken with same holder can be more accurately compared especially while assessing the degree of healing of periapical lesion.
3. Films can be taken more rapidly, and improve the quality of radiograph.
4. They eliminate the patient's finger from the field of exposure and thus reduce the risk of displacing the film.

Commonly used device for holding X-ray film is hemostat. Though various commercial devices are available as film holder. Various commercially available film holding devices include Endo Ray II film holder, Snap-H-Ray film holder, Uni Bite film holder and the Stable disposable film holder.

ADVANTAGES OF RADIOGRAPHS IN ENDODONTICS

In all the endodontic cases, a good intra oral periapical radiograph of the root and its related structures is mandatory. Radiography is the most reliable of all the diagnostic tests and provides the most valuable information.

In endodontics, the radiographs perform essential functions in three main areas that are diagnosis, treatment and recall.

1. Diagnosis

a. Radiographs help to know the presence of caries which may involve or on the verge of involving the pulp. Depth of caries, restoration, evidence of pulp capping or pulpotomy etc. could be evaluated on seeing the radiograph.
b. The radiographs help to know the root and pulpal anatomy, i.e. normal and abnormal root formation, curvature of the canal, number of roots and the canals, any calcifications if present in the canal and variation in the root canal system, i.e. presence of fused or extra roots and canals, any bifurcation or trifurcation in the canal system if present.
c. Radiographs help to know the pulp conditions present inside the tooth like pulp stones, calcification, internal resorption etc.
d. A good quality preoperative radiograph provide information on orientation and depth of the angulation of handpiece (Fig. 5.36).
e. Other conditions like resorption from the root surface i.e. external resorption, thickening of periodontal ligament and extent of periapical and alveolar bone destruction can be interpreted by viewing the radiographs.

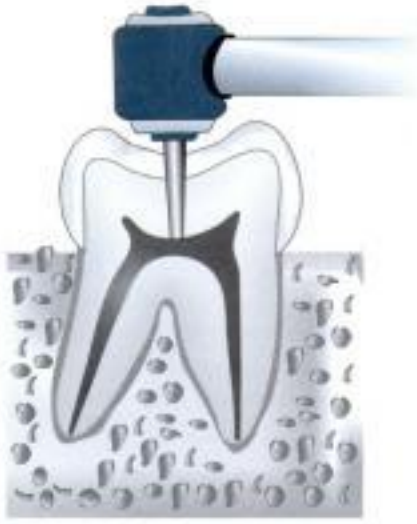


Fig. 5.36: Pre-operative radiograph can provide information on orientation and depth of the angulation of handpiece

f. They also help to identify the numerous radiolucent and radiopaque structures which often lie in the close proximity. These must be distinguished and differentiated from the pathological lesions.

2. Treatment

The radiographs exposed during the treatment phase are known as ***working radiographs.*** Working radiographs are made while rubber dam is in place, i.e. these radiographs are exposed during treatment phase.

a. Working Length Determination

In this, radiograph establishes the distance from the reference point to apex at which canal is to be prepared and obturated. By using special cone angulations, some superimposed structures can be moved to give clear image of the apex.

b. Master Cone Radiographs

It is taken in the same way as with working length radiograph. Master cone radiograph is used to evaluate the length and fit of master gutta percha cone.

c. Obturation

Radiographs help to know the length, density, configuration and the quality of obturation.

3. Recall

1. Radiographs are essential to evaluate the post treatment periapical status.

2. The presence and nature of lesion that have occurred after the treatment are best detected on radiograph. These lesions may be periapical, periodontal or nonendodontic.
3. Recall radiographs help to know the success of treatment by evaluating the healing process.

Thus, we can say that radiographs provide valuable information concerning the case selection, diagnosis, treatment planning and prognosis but one must not be totally dependent on the radiograph. In fact, radiographs can be misleading, thus must be viewed with caution and should be correlated with other clinical findings for example radiographs cannot differentiate granuloma, cyst or chronic abscess. For accurate diagnosis, histopathological examination is must. Similarly, presence of periapical radiolucencies on tooth does not indicate a diseased tooth. It could be due to normal anatomic structures like incisive or mental foramen or maxillary sinus or due to other odontogenic lesions such as ameloblastoma, early stages of cementoma or ossifying fibroma etc. In ossifying fibroma, the pulp vitality confirms the diagnosis. It is associated with the vital tooth. Perplexity of radiograph is that it does not always the correct interpretation yet it has to be used as an important diagnostic tool. For reaching an accurate diagnosis, one should not rely only on radiographs but it has to be used in conjunction with other tests.

DIGITAL RADIOGRAPHY

Most digital imaging in dentistry used standard radiology techniques with film to record the image, and then subjects the finished image to digital processing to produce the final result.

The backlot film image is converted to a digital signal by a scanning device, such as videocamera. First the image is divided into a grid or matrix of uniformly sized pixels, each of which is assigned a grey scale value based on its optical diversity. This value is stored in computer.

Once the image is in computer, a number of operations can be performed on computer program used.

One of the most useful operations is a comparison of images called digital subtraction. The computers can compare two images. Some use for this type includes determination of progression of disease over time and evaluation of treatment outcomes for periodontal and endodontic therapy.

For digital subtraction this is standardization of the process used to make the images as well as adjustments of the film density and contrast.

Another use is in detection of lesion on radiographs. This is where an endodontist can use this application for diagnosis. The computers can detect lesion with pattern recognition and boundary determination. Sometimes density changes on radiographs are so subtle that human eye has trouble identifying them, but machines are able to discriminate at density level beyond what human eye can see.

Disadvantages

"The radiation dose to the patient is the same as that used to radiographs. However, depending on the task required, less dense radiographs requiring low doses, could be used as computer can differentiate between areas of low contrast better than the human eye can" Disadvantage being equipment to print photographs or even earlier scanning of radiographs.

Advantages

1. The amount of information available from these radiographs is greater than from radiographs that have not been digitized.
2. The storage of radiographs and quality of image is better.
3. Photographs of radiographs can be produced.

DIGITAL DENTAL RADIOLOGY

Images in digital form can be readily manipulated, stored and retrieved on computer. Furthermore, technology makes the transmission of images practicable. The general principles of digital imaging are:

1. The chemically produced radiograph is represented by data that is acquired in a parallel and continuous fashion known as analogue.
2. Computers use binary (0 or 1) language, where information is usually handled in 8 character words called bytes.
3. If each character can be either 0 or 1. This result in 28 possible combination (words) that is 256 words. Thus digital dental images are limited to 256 shades of grey.
4. Digital images are made up of pixels (picture elements), each allocated a shade of grey.
5. The spatial resolution of a digital system is heavily dependent upon the number of pixels available per millimeter of image.

Digital Dental Radiology is Possible with Two Methods

1. One uses charged couple devices.
2. Other uses photo stimulable phosphor imaging plates.

Both methods can be used in dental surgery with conventional personal computers.

Digital imaging systems require an electronic sensor or detector, an analog to digital converter, a computer, and a monitor or printer for image of the components of imaging system. It instructs the X-ray generator when to begin and end the exposure, controls the digitizer, constructs the image by mathematical algorithm, determines, determines the method of image display, and provides for storage and transmission of acquired data.

The most common sensor is the CCD, the other being phosphor images.

When a conventional X-ray unit is used to project the X-ray beam on to the sensor, an electronic charge is created, an analog output signal is generated and the digital converter converts the analog output signal from CCD to a numeric representation that is recognizable by the computer.

The radiographic image then appears on the monitor and can be manipulated electronically to alter contrast, resolution, orientation and even size.

THE CCD SYSTEM

The CCD is a solid state detector composed of array of X-ray or light sensitive phosphores on a pure silicon chip. These phosphors convert incoming X-rays to a wavelength that matches the peak response of silicon.

RVG

RVG is composed of three major parts. The radio part consists of a conventional X-ray unit, a precise timer for short exposure times and a tiny sensor to record the image. The sensor has a small (17 × 24 mm) receptor screen which transmits information via fiber optic bundle to a miniature CCD. The sensor is protected from X-ray degradation by a fiber optic shield and can be cold sterilized for infection control, disposable latex sheaths are used to cover the sensor when it is in use (Fig. 5.37).

Because the sensor does not need to be removed from mouth after each exposure, the time to take multiple images is greatly reduced.

The 'visio' portion of the system receives and stores incoming signals during exposure and converts them point by point into one of 256 discrete gray levels. It consists of a video monitor and display processing unit (Fig. 5.38). As the image is transmitted to the processing unit, it is ***digitized*** and memorized by the computer. The unit magnifies the image four times for immediate display on video monitor and has additional capability of producing colored images

Fig. 5.37: Sensor used for RVG system

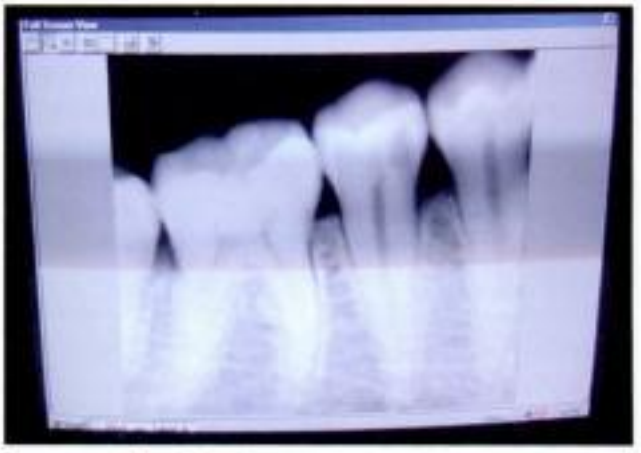


Fig. 5.38: Video monitor displaying image

(Figs 5.39 and 5.40). It can also display multiple images simultaneously, including a full mouth series on one screen. A zoom feature is also available to enlarge a portion of image upto face-screen size.

The 'graphy' part of RVG unit consists of digital storage apparatus that can be connected to various print out or mass storage devices for immediate or later viewing.

Advantages

1. Low radiation dose. Almost 90 percent less exposures and even higher reductions are possible in future.
2. Diagnostic capability is increased through digital enhancement and enlargement of specific areas for closer examination.
3. Image distortion from bent radiographic film is eliminated.
4. Contrast and resolution can be altered, and images can be viewed in black and white colour. The high contrast images are ideal for endodontic diagnosis and treatment.

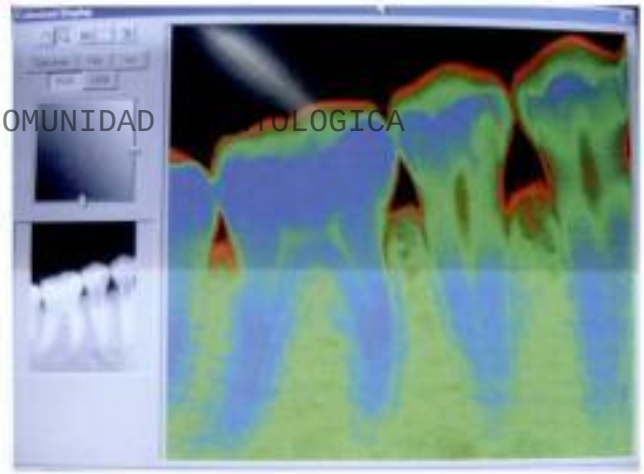


Fig. 5.39: Colored image produced in RVG

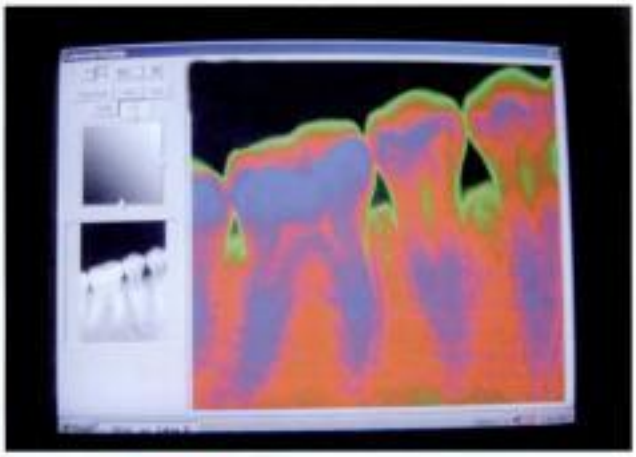


Fig. 5.40: Colored image produced in RVG

5. Images are displayed instantly.
6. Film less X-rays means, no dark room, no messy processing and no any problems/faults associated with developing of film.
7. Full mouth radiographs can be made within seconds.
8. Storages and archiving of patient information.
9. Transfer of images between institutions (teleradiology).
10. Infection control and toxic waste disposal problems associated with radiology are eliminated.

Disadvantages

1. Expensive
2. Large disc space required to store images.
3. Bulky sensor with cable attachment, which can make placement in mouth difficult.
4. Soft tissue imaging is not very nice.

PHOSPHOR IMAGING SYSTEM

Imaging using a photostimulable phosphor can also be called as an indirect digital imaging technique. The image is captured on a phosphor plate as analogue information and is converted into a digital format when the plate is processed.

Two sizes of phosphor plates, similar in size to conventional intraoral film packets are provided. They have to be placed in plastic light-tight bags, before being used in the mouth. They are then positioned in the same manner as film packets, using holders, incorporating beam - aiming devices, and are exposed using conventional dental X-ray equipment. The dose is highly reduced. The image is displayed and manipulated. A hard copy can be obtained if necessary.

Advantages

- Low radiation dose (90% reduction)
- Almost instant image (20 - 30 seconds)
- Wide exposure latitude (almost impossible to burn out information)
- Same size receptor as films
- X-ray source can be remote from PC
- Image manipulation facilities.

Disadvantages

- Cost
- Storage of images (same as with CCD systems)
- Slight in convenience of plastic bags.

6 Differential Diagnosis of Orofacial Pain

- Introduction
- Pain
- Diagnosis of Pain
- Transmission of Pain
- Pain Modulation and Perception
- Sources of Odontogenic Pain
- Pulpal Pain
- Periodontal Pain
- Sources of Non-odontogenic Pain
  - Myofascial Toothache
  - Neurovascular Toothache
  - Cardiac Toothache
  - Neuropathic Pain
  - Sinus or Nasal Mucosal Pain
  - Psychogenic Pain

INTRODUCTION

Orofacial pain is the field of dentistry related to diagnosis and management of chronic, complex facial pain and orofacial disorders. The dentist assumes a great responsibility for the proper management of pains in and around the mouth, face and neck. To do this, he or she must have accurate knowledge of pain problems arising from other than oral and maxillofacial area. This specialty in dentistry has been developed over a number of years for diagnosis of orofacial pain which did not seem to have a clearly defined medical problem.

Orofacial pain, like pain elsewhere in the body, is usually the result of tissue damage and the activation of nociceptors, which transmit a noxious stimulus to the brain. Orofacial disorders are complex and difficult to diagnose due to rich innervations in head, face and oral structures. Ninety percent of orofacial pain arises from teeth and adjoining structures. As a dentist, one must be trained to diagnose and treat acute dental pain problems.

After ruling out dental problems, myofacial and neuropathic pain conditions are the most common causes of dental pain. For accurate diagnosis and treatment planning, one must have thorough understanding of basic concepts of pain and its transmission.

PAIN

Dorland's Medical Dictionary defines pain as "A more or less localized sensation of discomfort, distress or agony resulting from the stimulation of nerve endings". It indicates that pain is a protective mechanism against injury. ***International Association for the Study of Pain (IASP) has defined pain as*** "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."

It is usually found that the more severe the pain and the more distressed the patient more emotional are his responses leading to greater impact on his ability of function.

DIAGNOSIS

The most important component of managing pain is in understanding the problem and establishing the proper diagnosis. For establishing the correct diagnosis, the dentist must record all relevant information regarding signs, symptoms, history of present complaint, past medical and dental history. The history is an important part of diagnosis, it should assess the present location of the pain, its causative and aggravating factors and a detailed description of the pain since its origin. The clinician should record all the factors related to the pain such as emotional, physical and influence of any prior trauma, infection or systemic illness. For establishing differential diagnosis, an accurate history taking is the most important aspect.

HISTORY OF PAIN

History of pain includes the following:

Chief Complaint

- Location
- Onset
- Chronology
- Quality
- Intensity
- Aggravating factors
- Precipitating factors
- Past medical and dental history
- Psychologic analysis
- Review of systems

Location

The patient's ability to locate the pain usually helps in diagnosis but sometimes wrong localization of pain problems for clinician. The patient's description of the location of his or her complaint identifies only site of pain. So, it is the dentist's responsibility to determine whether it is the true source of pain or the referred pain.

Onset

It is important to record the conditions associated with initial onset of pain. Sometimes it may facilitate in recognizing the etiology of pain.

Chronology

Chronology of pain should be recorded in a following pattern:

- Initiation
- Clinical course and temporal pattern
  - A. Mode
  - B. Periodicity
  - C. Frequency
  - D. Duration

Quality

It should be classified according to how pain makes the patient feel.

- Dull, grawing or aching
- Throbbing, pounding or pulsating
- Sharp, recurrent or stabbing pain
- Squeezing or crushing pain.

Intensity

Intensity of pain is usually established by distinguishing between mild, moderate and severe pain. A visual analog scale is used to assess the intensity of pain. The patient is given a line on which no pain is written on one end and at other end there is most severe pain which patient has experienced. A scale of 0-10 is used to assess the pain, 0-being no pain while 10 being the maximum pain possible.

- Pain index : 0-10 (Fig. 6.1)
- Pain classification : Mild
  Moderate
  Severe

Fig. 6.1: Rating scale to check intensity of pain ranging from (0-10)

Aggravating Factors

Aggravating factors always help the clinician in diagnosis. These can be local or conditional. Local factors can be in form of irritants like heat, cold, sweets and pain on biting etc. Conditional factors include change of posture, activities and hormonal changes, etc.

Local factors	*Conditional factors*
• Sweets	• Change of posture
• Chewing	• Time of day
• Palpation	• Activities
• Heat	• Hormonal
• Cold	
• Percussion	

TRANSMISSION OF PAIN

Pain sensation from the intraoral and extraoral structures of the head and face are carried to central nervous system by trigeminal system. The term "***trigeminal system***" refers to complex arrangement of nerve fibers, synaptic connections and interneurons which process incoming information from three divisions of the trigeminal nerve. Sensory information from the face and oral cavity (except proprioception) is carried out by primary afferent neurons. These neurons further transmit information to higher centers by forming synapses on neurons located within the trigeminal brainstem complex, which span the midbrain and cervical spinal cord. This complex receives afferent input primarily from trigeminal nerve but also receives afferent axons from the facial, glossopharyngeal, vagus and upper cervical nerves (C 2, 3). The connection between the upper cervical nerves and trigeminal spinal tract nucleus may perhaps be a mechanism involved in facial pain and headaches. The trigeminal brainstem sensory nuclear complex can be separated into trigeminal main sensory nucleus and trigeminal spinal tract nucleus. The spinal tract nucleus is composed of three separate nuclei proceeding from superior to inferior (caudal) direction (Fig. 6.2).

- Subnucleus oralis
- Subnucleus interpolaris
- Subnucleus caudalis

Subnucleus caudalis, the most inferior one is located in the medulla, at times extending to level of C_2 and C_3 and is the principal brain relay site of nociceptive information arising from the orofacial region (Fig. 6.3). Because the subnucleus caudalis is continuous and structurally similar to spinal cord dorsal horn and it also extends into the medulla. So, it is often referred to as the medullary dorsal horn. Both incoming nociceptive signals to subnucleus caudalis and projected nociceptive signals on their way to the thalamus can be modified by descending nerve fibers from higher levels of the central nervous system or by drugs.

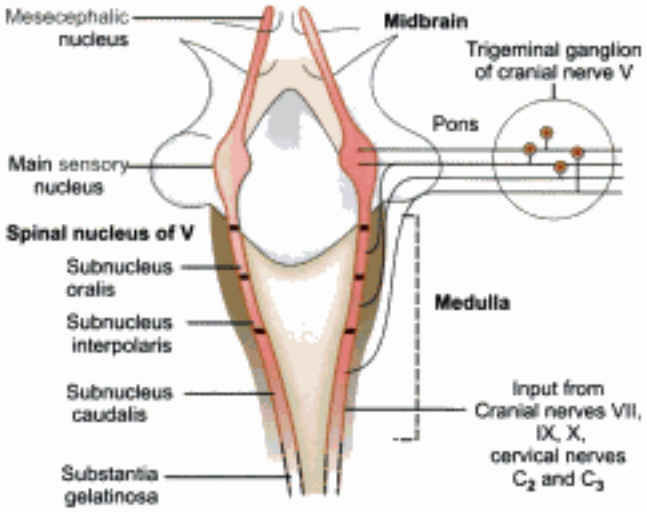


Fig. 6.2: Trigeminal brain stem sensory nuclear complex

Convergence of neurons with in the trigeminal spinal tract

Primary nociceptors from both visceral and cutaneous neurons often converge onto the same second-order pain transmission neurons in the spiral cord. In many cases, the brain is unable to interpret the exact location of the original oral stimulus for example early stages of pulpal pain. In an inflammatory or pain process, a signal is sent only when a certain critical level of insult is reached, for example in cases of deep caries.

The brain may appreciate that there is toothache somewhere but can not localize it and due to convergence factors, the brain may experience more difficulty in localizing the pain.

Second Order Neurons

These neurons can be classified into three types of transmission cells (Fig. 6.4).

1. Wide dynamic range neurons (WDR)
2. Nociceptive-specific neurons (NS)
3. Low threshold mechanoreceptive neurons (LTM)

1. Wide dynamic range neurons (WDR)

These neurons are excited by both noxious stimuli and non-noxious tactile stimuli over a wide range of intensities.

2. Nociceptive-specific neurons (NS)

These neurons are excited by intense noxious mechanical and thermal stimuli by thin nociceptive fibers.

The WDR and NS neurons predominate in laminar I, II, V, and comprise the trigeminal nociceptive pathways. They all receive input from cutaneous structures and at least half of them receive input from deep structures of the mouth and face also (Fig. 6.5) .

3. Low threshold mechanoreceptive (LTM)

These neurons are normally non-nociceptive and respond to light tactile stimuli. They mainly lie in laminar III and IV. These neurons appear to be excited by strong electrical

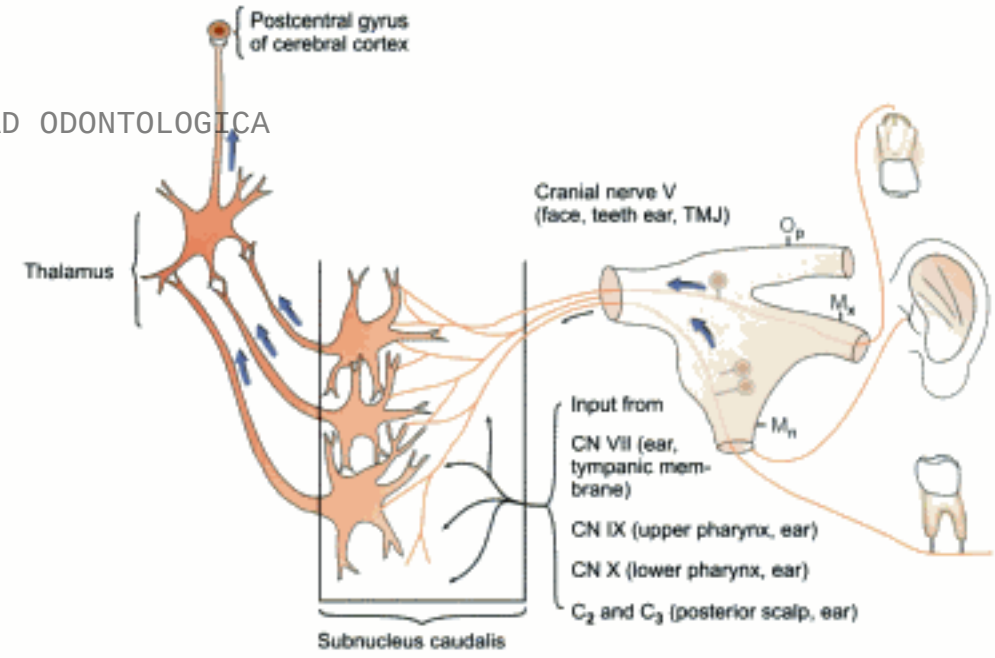


Fig. 6.3: Diagram showing subnucleus caudalis

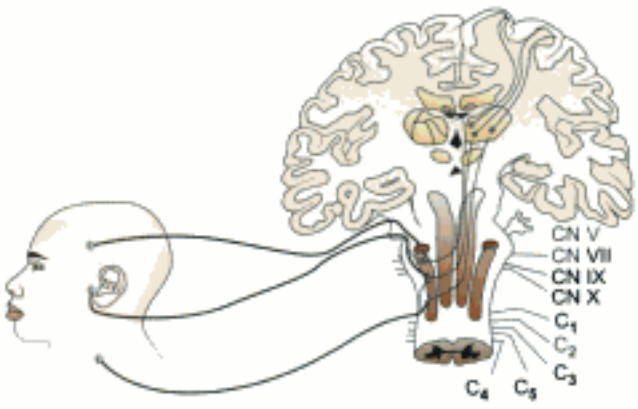


Fig. 6.4: Second order neurons showing pain transmission

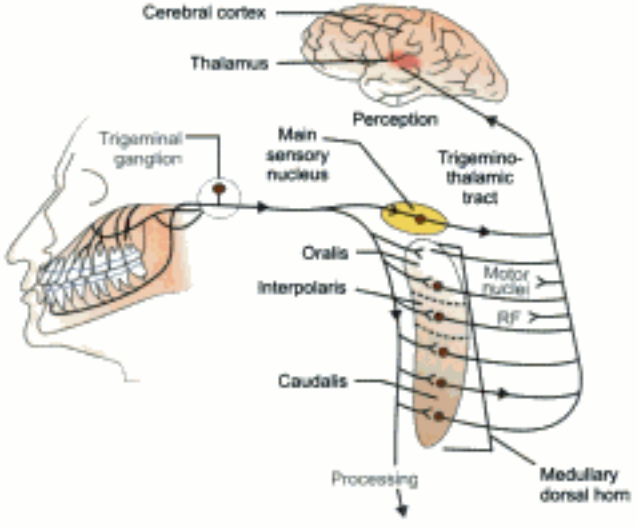


Fig. 6.5: Pathway of transmission of nociceptive signals from the orofacial region

stimulation of tooth pulp and may involve in pathological conditions of pulp.

Third Order Neurons

The next major synaptic transmission in pain transmission is in the thalamus where axons travelling in trigeminothalamic tract synapse with third order neurons. All sensory information from spinal cord and brainstem passes through the thalamus making it one of the primary relay stations between the brainstem and different parts of the somatosensory cerebral cortex (Fig. 6.6). For all nerve pathways from the thalamus to the cerebral cortex, there are reciprocal

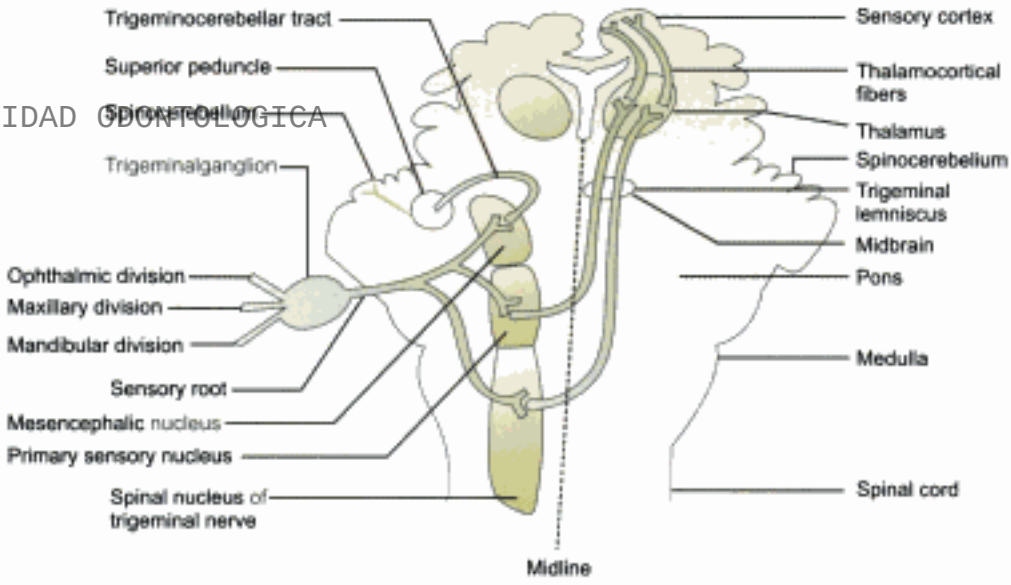


Fig. 6.6: Trigeminal pathway

connections from the cortex to the thalamus. Sensory information reaching the thalamus may also be relayed to several distinct nuclei in the thalamus.

At the thalamic level, the action potential will be subjected to extensive processing through interactions among its various nuclei and by interconnections with limbic, hypothalamic and cortical regions of the brain. It should be noted that until the nociceptive signal reaches the level of the thalamus, most of the reactions in the CNS are reflex in nature. Only when thalamus is involved, elements of consciousness and alertness introduced.

PAIN MODULATION AND PERCEPTION

Modulation is defined as the ability to alter the intensity of nociceptive signals and reduce the pain experience. In human nervous system, the key pathways start in periqueductal gray area of midbrain. The signals from this area descend to dorsal horn of spinal cord where neuron interactions further reduce the transmission of primary pain signal from the peripheral nociceptive receptors to the second order neuron.

Finally, the perception of pain takes place in the posterior parietal cortex of the brain. The differences in the size of sensory map on the brain reflect the differences in functional importance.

Orofacial pain can be basically divided into odontogenic(dental pain) and nonodontogenic pain (non dental). Dental pain may have origin in the pulpal tissue or the periradicular tissue. *Nondental pain can be in form of myofacial toothache, vascular headache, cluster headache, sinusitis, trigeminal neuralgia,*

SOURCES OF ODONTOGENIC PAIN

a. Dental Pain of Pulpal Origin

Dental pulp is richly innervated by A and C nerve fibers. The nerves of the pulp include primary afferent fibers that are involved in pain transmission and sympathetic efferent fibers which modulate the microcirculation of the pulp. The sympathetic efferent fibers reduce the flow of the blood through pulp by stimulating smooth muscle cells encircling the arterioles. Also, four types of nerve endings are present in pulp;

a. Marginal fibers
b. Simple predentinal fibers
c. Complex predentinal fibers
d. Dentinal fibers

Both pain and touch sensations have been related to free nerve endings but stimulation of the pulp give rise only to pain. Fast pain is associated with A fibers and slow pain is with C fibers. ***Stimulation of A fibers produce a sharp, piercing or stabbing sensation while C fibers produce dull, burning, and aching sensation that is usually harder to endure*** (Fig. 6.7).

The pulpal pain is of threshold type, that is, no response occurs until threshold level is increased. Pulp may respond

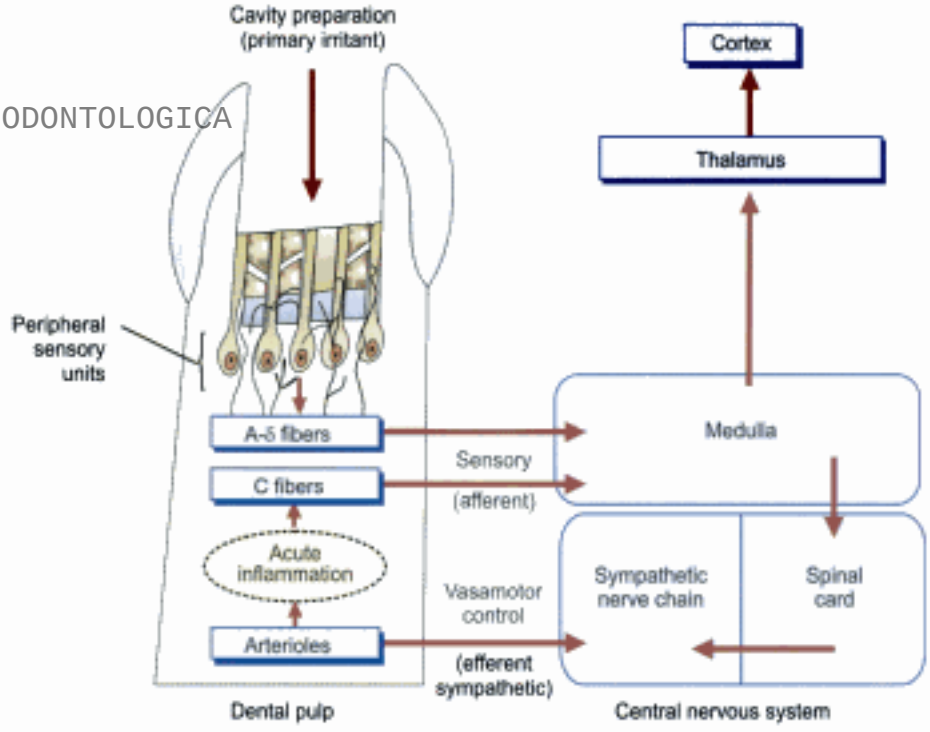


Fig. 6.7: Neurophysiology of pulpal pain

to chemical, mechanical, electrical or thermal stimulation but not to ordinary masticatory functions. Pulpal pain can not be localized by the patient. A basic feature of pain of pulpal origin is that it does not remain the same for long periods. Generally it resolves, becomes chronic or involves the periodontal structure.

b. Dental Pain of Periodontal Origin

Periodontal pain is deep somatic pain of the musculoskeletal type because of presence of proprioceptor fibers. Patient can localize the pain of periodontal origin. Therefore, periodontal pain presents no diagnostic problems because the offending tooth can be readily identified. This localization can be identified by applying pressure to the tooth axially and laterally.

When the periodontal pain involves many teeth, one may consider occlusal overstressing which could be due to occlusal interferences or parafunctional habits such as bruxism.

Sources of Odontogenic Pain

Pulpal pain

1. Dentinal sensitivity
2. Reversible pulpitis
3. Irreversible pulpitis
4. Necrotic pulp

Periodontal pain

1. Acute apical periodontitis
2. Acute apical abscess
3. Chronic apical periodontitis

PULPAL PAIN

1. DENTINAL SENSITIVITY

In the absence of inflammation, dentinal sensitivity is the mildest form of pulp discomfort. The pain is often characterized as a short, sharp, shock and it is brought on by some stimulating factor such as hot or cold, sweet, sour, acid or touch. It is not pathologic, but is rather, fluid flow in the dentinal tubules which stretches or compresses the nerve endings that pass alongside the tubular extensions of the pulp odontoblasts.

Dentinal sensitivity may also develop when dentin is exposed from gingival recession or following periodontal surgery. Nerves in these exposed tubules not only respond to hot, cold, sweet and sour but also to scratching with a finger nail or during tooth brushing.

Diagnosis

1. Apply the irritant which starts the painful reaction-hot or cold, sweet or sour or scratching with an instrument.
2. All diagnostic tests such as electric pulp test, percussion and radiographs are generally negative.

Treatment

After diagnosis, dentinal hypersensitivity can be treated by home use of desensitizing dentifrices containing strontium

chloride, fluorides and potassium nitrate. Since rapid movement of fluid in the dentinal tubules is responsible for pain by activating intradental sensory nerves, the treatment of hypersensitive teeth should be aimed at reducing the anatomical diameter of the tubules so as to limit the fluid movement. Various agents can be use to occlude the dentinal tubules like cavity varnishes, calcium compounds, fluoride compounds like sodium fluoride, stannous fluoride, iontophoresis, restorative resins and dentin bonding agents.

2. REVERSIBLE PULPITIS

In reversible pulpitis, pain occurs when a stimulus (usually cold or sweets) is applied to the tooth. When the stimulus is removed, the pain ceases with in 1 to 2 second, i.e. it should return to normal with removal of cause. The common causes of reversible pulpitis are caries, faulty restorations, trauma or any recent restorative procedures. Pulpal recovery is usually seen if reparative cells in the pulp are adequate.

Diagnosis

Diagnosis is made by careful history and clinical examination. If there is discrepancy between the patient's chief complaint, symptoms and clinical examination, obtain more information from the patient. It is important to note that both pulpal and periapical diagnosis should be made before treatment is initiated. If tooth is sensitive to percussion, then look for bruxism and hyperocclusion.

Treatment

1. Removal of the cause if present (caries; fractured restoration; exposed dentinal tubules).
2. If recent operative procedure or trauma has taken place, then postpone the additional treatment and observe the tooth.
3. If pulp exposure is detected, go for root canal treatment.

3. IRREVERSIBLE PULPITIS

Irreversible pulpitis develops, if inflammatory process progresses to involve pulp. Patient may have history of spontaneous pain or exaggerated response to hot or cold that lingers even after the stimulus is removed. The involved tooth usually presents an extensive restoration and/or caries.

Diagnosis

Diagnosis is usually made after taking thorough history and clinical examination of the patient.

1. Patient usually gives a history of spontaneous pain.
2. Tooth is hypersensitive to hot or cold that is prolonged in duration.
3. Pulp may be vital or partially vital.
4. In certain cases of irreversible pulpitis, the patient may arrive at the dental chair with a glass of ice/cold water. In these cases, cold actually alleviates the patient's pain and thus, can be used as a diagnostic test. Cooling of the dentin and the resultant contraction of the fluid in the tubules relieves the pressure on pulpal nerve fibers caused by edema and inflammation of the pulp.

Treatment

Complete removal of pulpal tissue should be done, i.e. endodontic therapy.

4. NECROTIC PULP

It results from continued degeneration of an acutely inflamed pulp. Literal meaning of necrosis is death, that is pulpal tissue becomes dead because of untreated pulpal inflammation. In pulpal necrosis, there is progressive breakdown of cellular organization with no reparative function. It is frequently associated with apical radiolucent lesion. In case of multirooted teeth, one root may contain partially vital pulp, whereas other roots may be nonvital.

Diagnosis

1. Tooth is usually asymptomatic; may give moderate to severe pain on biting pressure (It is not symptom of necrotic pulp but it indicates inflammation).
2. Pulp tests show negative response but in case of multirooted teeth , it can give false positive results .

Treatment

Complete removal of pulpal tissue that is root canal treatment.

PERIODONTAL PAIN

1. ACUTE APICAL PERIODONTITIS

It is the inflammation of periodontal ligament which is caused by tissue damage, extension of pulpal pathology or occlusal trauma. Tooth may be elevated out of the socket because of the built up fluid pressure in the periodontal ligament. Pain remains until the bone is resorbed, fluid is drained or irritants are removed.

Diagnosis

1. Check for decay, fracture lines, swelling, hyperocclusion or sinus tracts.
2. Patient has moderate to severe pain on percussion.
3. Mobility may or may not be present.
4. Pulp tests are essential and their results must be correlated with other diagnostic information in order to determine if inflammation is of pulpal origin or from occlusal trauma.
5. Radiographs may show no change or widening of periodontal ligament space in some cases.

Treatment

1. Complete removal of pulp.
2. Occlusal adjustment.

2. ACUTE PERIAPICAL ABSCESS

Acute periapical abscess is an acute inflammation of periapical tissue characterized by localized accumulation of pus at the apex of a tooth. It is a painful condition that results from an advanced necrotic pulp. Patients usually relate previous painful episode from irreversible pulpitis or necrotic pulp. Swelling, tooth mobility and fever are seen in advanced cases.

Diagnosis

1. Spontaneous dull, throbbing or persistent pain is present.
2. Tooth is extremely sensitive to percussion.
3. Mobility may be present.
4. On palpation, tooth may be sensitive.
5. Vestibular or facial swelling in seen in these patients.
6. Pulp tests show negative results.

Treatment

1. Drainage
2. Complete extirpation of pulp.
3. Appropriate analgesics and antibiotics if necessary.

3. CHRONIC APICAL PERIODONTITIS

It is caused by necrotic pulp which results from prolonged inflammation that erodes the cortical plate making a periapical lesion visible on the radiograph. The lesion contains granulation tissue consisting of fibroblasts and collagen.

Diagnosis

1. It is usually asymptomatic but in acute phase may cause a dull, throbbing pain.
2. Pulp tests show non-vital pulp.
3. There is no pain on percussion.
4. Radiographically, it is usually associated with periradicular radiolucent changes.

SOURCES OF NONODONTOGENIC PAIN

As dental pain is considered one of the commonest cause of orofacial pain, so dentist can be easily drawn to this diagnosis. Tooth aches of nondental origin are usually suspected when patient convincingly reports that pain is usually felt in this particular tooth. But dentist should be aware of this fact that some tooth aches felt in the tooth/teeth do not originate from these structures. There are many structures in the head and neck region which can stimulate pain. Such types of pain are classified under heterotopic pain. Heterotopic pain can be defined as any pain felt in an area other than its true source.

There are three general types of pain:

i. Central pain
ii. Projected pain
iii. Referred pain.

Referred pain is a heterotopic pain that is felt in an area innervated by a different nerve, from the one that mediates the primary pain. Referred pain is wholly dependent upon the original source of pain. It can not be provoked by stimulation where the pain is felt while it can be accentuated only by stimulation the area where primary source of pain is present.

Myofascial Toothache

Any deep somatic tissue in the head and neck region has tendency to induce referral pain in the teeth. In these structures, pains of muscular origin appear to be the most common. Muscles which are commonly affected are masseter, temporalis but in some cases medial, and lateral pterygoid and digastric muscles are also affected.

Characteristic Findings of Muscular Toothache are:

1. Non-pulsatile, diffuse, dull and constant pain.
2. Pain increases with function of masticatory muscles. For example pain is increased when chewing is done because of effect on masseter muscle.

3. Palpation of the involved muscles at specific points (trigger points) may induce pain.
   *Trigger points are hyperexcitable muscle tissues which may feel like taut bands or knots.

4. Usually arise with or without pulpal or periradicular pathology.
5. Tooth pain is not relieved by anesthetizing the tooth; rather local anesthesia given at affected muscle may reduce the toothache.

Diagnosis of these muscular pains as nonodontogenic tooth is purely based on lack of symptoms after diagnostic tests such as pulp testing, percussion and local anesthesia block.

Several therapeutic options used in the ***management*** of these muscular pain are:

i. Restriction of functional activities within painless limit
ii. Occlusal rearrangement
iii. Deep massage
iv. Spray and stretch technique
v. Ultrasound therapy
vi. Local anesthesia at the site of trigger points
vii. Analgesics
viii. Anxiolytics.

Neurovascular Toothache

The most common neurovascular pain in the mouth and face is migraine. This category of pain includes three subdivisions of primary headache. These are:

i. ***Migraine***
ii. ***Tension type headache***
iii. ***Cluster headache.***

These neurovascular entities can produce relatively localized pains that match with sign and symptoms with the toothache. These accompanying toothaches are usually mistaken for true odontogenic pains and can be treated as separate entities. Unfortunately there are several clinical characteristics that could misguide clinician in diagnosis and treatment.

The ***following characteristics*** are usually found common in ***neurovascular toothache*** are:

1. The pain is ***deep, throbbing, spontaneous*** in onset, variable in nature and pulsatile. These are characteristics which simulate pulpal pain.
2. The ***pain*** is predominantly ***unilateral***.
3. Accompanying ***toothache*** shows periods of ***remission*** that imitates the pain-free episodes or temporal behavior found in neurovascular pain.
4. ***Headache*** is considered as the main symptom. It is most often accompanied by toothache.
5. ***Recurrence*** is characteristic finding in neurovascular pain. Sometimes, the pain may undergo remission after dental treatment has been performed in these teeth. It usually appears for certain period of time and may even spread to adjacent teeth, opposing teeth or the entire face.
6. ***Autonomic effects*** such as nasal congestion, lacrimation, rhinorrhea and edema of the eyelids and face is seen. Sometimes edema of the eyelids and face might lead to confusion in diagnosis as these features bear a resemblance to abscess.

Migraine

Migraine has been divided into two main types:

1. ***Migraine with aura.***
2. ***Migraine without aura.***

Features of Migraine

- Commonly found between the age group ***20-40 years***.
- ***Visual auras*** are most common. These usually occur 10-30 minutes prior to the onset of headache pain. (Migraine with aura)
- Pain is usually unilateral, pulsatile or throbbing in nature
- More common in ***females***
- Patient usually experiences nausea, vomiting, photophobia
- Various drugs used in the ***management*** of migraine are sumatriptan, β-Blockers, tricyclic antidepressants and calcium channel blockers.

CLUSTER HEADACHE

- Commonly found in the age group 20-50 years
- Cluster headaches derive their name from the temporal behavior and usually occur in series, i.e. one to eight attacks per day
- More common in males than females
- Pain is unilateral, excruciating and continuous in nature and usually found in orbital, supraorbital or temporal region
- Autonomic symptoms such as nasal stuffiness, lacrimation, rhinorrhea or edema of eyelids and face are usually found
- Standard treatment is inhalation of 100 percent oxygen.

The behavior of neurovascular variants should be well appreciated to avoid any unnecessary treatment and

frustration felt by patient and clinician. Although the term neurovascular toothache is nondescriptive, but it has given the dentist an important clinical entity that has been misdiagnosed and mistreated in the past. Some sign and symptoms of neurovascular headache that mimic the toothache are:

- Periodic and recurrent nature
- Precise recognition of painful tooth
- Absence of local dental etiology.

CARDIAC TOOTHACHE

Severe referred pain felt in mandible and maxilla from area outside the head and neck region is most commonly from the heart. ***Cardiac pain is clinically characterized by heaviness, tightness or throbbing pain in the substernal region which commonly radiates to left shoulder, arm, neck and mandible***. Cardiac pain is most commonly experienced on the left side rather than right. In advanced stages, the patient may complain of severe pain and rubs the jaw and chest. In present time, dentist should be aware of incidence of jaw pain that is occurring in number of patients secondary to cardiac pain. Sometimes, patient presents dental complaints as the chief complaint rather than having pain in substernal region, it creates confusion in diagnosis for dental pain. A lack of dental cause for dental pain should always be an alerting sign. Anesthetizing the lower jaw or providing dental treatment does not decrease the tooth pain, it indicates that primary source of pain is not the tooth. ***Usually the cardiac toothache is decreased by taking rest or a dose of sublingual nitroglycerin.*** A complete medical history should be taken when cardiac toothache is suspected and should be immediately referred to cardiac unit in hospital.

In brief, the following characteristics of cardiac toothache are:

1. Pain is of sudden in onset, gradually increasing in intensity, diffuse with cyclic pattern that vary in intensity from mild to severe.
2. Tooth pain is increased with physical activities.
3. Chest pain is usually associated.
4. Pain is not relieved by anesthesia of lower jaw or by giving analgesics.

NEUROPATHIC PAIN

Neuropathic pain is usually caused by abnormalities in the neural structures themselves. Neuropathic pain is sometimes misdiagnosed as psychogenic pain because local factors can not be visualized.

Neuropathic pain can be classified into different categories:

i. ***Neuralgia***
ii. ***Neuritis***
iii. ***Neuropathy***

Neuralgia

Paroxysmal, unilateral, severe, stabbing or lancinating pain, usually are the characteristics of all ***paroxysmal neuralgias.*** The pain is usually of short duration and lasts for few seconds.

Trigeminal Neuralgia

- It is also known as **'Tic Doulourex'** which has literal meaning of ***painful jerking***
- Usually characterized by paroxysmal, unilateral, sharp, lancinating pain typically confined to one or more branches of 5th cranial nerve
- Even slight stimulation of 'Trigger points' may elicit sharp, shooting pain
- Sometimes trigger points are present intraorally. These are stimulated upon chewing which may led to diagnosis of odontogenic pain. Intraoral trigger points always create confusion in diagnosis if not properly evaluated
- Local anesthesia given at the trigger point reduces the attacks
- It rarely crosses midline
- Frequently occur in persons over the age of 50 years
- Attacks generally do not occur at night
- Absence of dental etiology along with symptoms of paroxysmal, sharp, shooting pain always alert the dentist to include neuralgia in the differential diagnosis
- ***Treatment*** includes surgical and medicinal. Usually medicinal approach is preferred. It includes administration of carbamazepine, baclofen, phenytoin sodium and gabapentin, etc.

Neuritis

Neuritis literally means inflammation of nerve. It is usually observed as heterotopic pain in the peripheral distribution of the affected nerve. It may be caused by traumatic, bacterial and viral infection. In neuritis, the inflammatory process elevates the threshold for pricking pain but lowers it for burning pain. The characteristics of pain in neuritis are:

1. Pain has a characteristic burning quality along with easily localization of the site.

2. It may be associated with other sensory effects such as hyperesthesia, hypoesthesia, paresthesia, dysesthesia and anesthesia.
3. Pain is non-pulsatile in nature.
4. Pain may vary in intensity.

Peripheral neuritis is an inflammatory process occurring along the course of never trunk secondary to traumatic, bacterial, thermal or toxic causes. Neuritis of superior dental plexus has been reported when inflammation of sinus is present. The dental nerves frequently lie just below the lining mucosa or are separated by very thin osseous structure. These nerves are easily involved due to direct extension. Symptoms usually seen along with antral disease are pain, paresthesia and anesthesia of a tooth, gingiva or area supplied by infraorbital nerve. ***Mechanical nerve trauma*** is more common in oral surgery cases. It usually arises from inflammation of the inferior dental nerve either due to trauma or infection.

Acute neuritis cases are always misdiagnosed and remain untreated. Most of the times, dental procedures are done to decrease the symptoms of neuritis as these are difficult to diagnose. These unnecessary dental procedures further act as aggravating factors for neuritis, making it chronic.

TREATMENT OF NEURITIS

- Treatment of acute neuritis is based on its etiology
- If bacterial source is present, antibiotics are indicated
- If viral infection is suspected, antiviral therapy should be started
- If there is no infections, steroids should be considered.

Neuropathy

This is the term used for localized and sustained pain secondary to an injury or change in neural structure. Atypical odontalgia has been included in neuropathy. Atypical odontalgia means toothache of unknown cause. It is also known as ***"Phantom tooth pain"*** or ***"dental migraine"***. Most patients who report with atypical odontalgia usually have multiple dental procedures completed before reaching a final diagnosis.

Clinical Characteristics of Neuropathy (Atypical Odontalgia)

1. More common in women.
2. Frequently found in 4th or 5th decades of life.
3. Tooth pain remains constant or unchanged for weeks or months.
4. Constant source of pain in tooth with no local etiology.
5. Pain usually felt in these patients is – dull, aching and persistent.
6. Most commonly affected teeth are maxillary premolar and molar region.
7. Response to local anesthesia is equal in both pulpal toothache and atypical odontalgia.

SINUS OR NASAL MUCOSAL TOOTHACHE

Sinus and Nasal Mucosal pain is also another source which can mimic toothache. It is usually expressed as pain throughout the maxilla and maxillary teeth.

Clinical characteristics of sinus or nasal mucosal toothache are:

1. Fullness or pressure below the eyes.
2. Increased pain when palpation is done over the sinus.
3. Increased pain sensation when head is placed lower than the heart.
4. Local anesthesia of referred tooth/teeth does not eliminated pain while topical anesthesia of nasal mucosa will eliminate the pain if etiology lies in nasal mucosa.
5. Different diagnostic aids used to diagnose sinus disease include paranasal sinus view, computed tomography imaging and nasal ultrasound.

PSYCHOGENIC TOOTHACHE

This is a category of mental disorders in which a patient may complain of physical condition without the presence of any physical signs.

In these cases, always think of psychogenic toothache. No damage to local tissue is typical in heterotrophic pain entities.

It must be noted that psychogenic pain is rare. So, all other possible diagnoses must be ruled out before making the diagnosis of psychogenic pain.

The following features are usually found in these diseases are:

1. Pain is observed in multiple teeth.
2. Precipitated by severe psychological stress.
3. Frequent changes in character, location and intensity of pain.
4. Response to therapy varies which can include lack of response or unusual response.
5. Usually referred to ***psychiatrist*** for further management.

7 Case Selection and Treatment Planning

- Introduction
- When to Do Endodontic Therapy
- Contraindications of Endodontic Therapy
- Treatment Planning
- Medical Conditions Influencing Endodontic Treatment Planning
- Asepsis in Endodontics
- Pain Control
- Isolation of the Tooth
- Technique for anesthetizing Maxillary Teeth
- Technique for Anesthetizing Mandibular Teeth

INTRODUCTION

In the past, injured or diseased teeth frequently were indicated for extraction. But now the trend has been changed. The teeth now can be saved through endodontic treatment, because in modern scenario, the clinician as well as the general population is becoming more aware of the importance of natural teeth. Now it has become common practice to make additional efforts to retain the teeth which are pulpally or periradicularly involved. The endodontic therapy has become the recent trend among general population. Infact during conversation the people proudly claim that they have got the root canal done. Especially for last 30-40 years, role of endodontics have broadened in scope. Many factors such as increased awareness among people, development of latest instruments and techniques which further added comfort to both patient as well as clinician, are responsible for this. Infact high predictability of endodontic success plays an important role for its growth. Various studies have shown that if properly done, the success rate of 95 percent can be achieved in endodontic therapy. To further success of endodontic therapy, continuous education programme and professional study along with clinical experience are required.

WHEN TO DO ENDODONTIC THERAPY?

This is sometimes difficult to decide simply when there is doubt about the case selection for endodontic therapy. Sometimes patients come to clinic having no symptoms with large restorations on teeth which require crowns. But the clinician suggests that endodontic treatment is needed for that particular tooth. So one can say that sometimes decision to do endodontic therapy becomes complicated because multiple factors are involved in deciding the need, treatment planning and prognosis of tooth.

Following factors are important in deciding the need and success of the overall endodontic therapy:

i. Actual Reason for Endodontic Therapy

If there is pulp involvement due to caries, trauma, etc. (Fig. 7.1) the tooth must be treated endodontically and restored with proper restoration.

ii. Elective Endodontics

Sometimes elective endodontic is done with crack or heavily restored tooth, to prevent premature loss of cusp during their restoration (usually crown preparation) and eliminate fear

Fig. 7.1: An infected pulp is to be removed by endodontic therapy

of pulp exposure. Elective endodontics allows to do more predictable and successful restorative dentistry.

iii. Inadequate Restorations

Patients with cracked or carious teeth having crowns, when want patch up of the crown margins or use preexisting crown even after another restorative procedures show high degree of restorative failure. In such cases endodontic treatment followed by optimal restoration of the tooth provide high success rate.

iv. Devitalization of Tooth

In patients with rampant caries or recurrent decay and smooth surface defects, it is wise to do desensitization of the teeth so that patients do not feel discomfort to cold or sweets.

v. Endodontic Emergency

Sometimes patient comes with acute dental pain, in such cases endodontic therapy is often indicated before a complete examination and treatment plan has performed. It is important to place endodontic case in perspective with the needs of patient's entire mouth.

CONTRAINDICATIONS OF ENDODONTIC THERAPY

There are only few true contraindications of the endodontic therapy. Otherwise any tooth can be treated by root canal treatment. *Mainly there are following four factors which influence the decision of endodontic treatment:*

a. Accessibility of apical foramen.
b. Restorability of the involved tooth.
c. Strategic importance of the involved tooth.
d. General resistance of the patient.

Therefore, before deciding the endodontic treatment, multiple factors should be considered. In general, following cases are considered ***poor candidates for endodontic treatment***:

1. ***Non restorable teeth*** such as teeth with extensive root caries, furcation caries, poor crown/root ratio and with fractured root are contraindicated for endodontic treatment. Because in such cases even the best canal filling is futile if it is impossible to place the restoration.
2. ***Teeth in which instrumentation is not possible*** such as teeth with sharp curves, dilacerations, calcifications, dentinal sclerosis are ***treatment difficulties.*** Though use of recent instruments and techniques may help sometimes like NiTi files, anticurvature filing may help sometimes.

   Several teeth with previous treatment show canal blockage by broken instruments, fillings, posts, ledges, and untreatable perforations and canal transportations. So, careful evaluation is needed before starting treatment in such teeth.
3. ***Poor accessibility:*** Occasionally trismus or scarring from surgical procedures or trauma, systemic problems etc. may limit the accessibility due to limited mouth opening. These result in poor prognosis of the endodontic therapy.
4. ***Untreatable tooth resorption:*** Resorptions which are extremely large in size make the endodontic treatment almost impossible for such teeth.
5. ***Vertical tooth fracture:*** Teeth with vertical root fractures pose the hopeless prognosis.
6. ***Non strategic teeth:*** There are two major factors which relegate a strategic tooth to the hopeless status; restorability and periodontal support. The tooth that cannot be restored or that has inadequate, unmaneable periodontal support is hopeless. Evaluation of the oral cavity can decide whether tooth is strategic or not, for example if a person has multiple missing teeth, root canal of third molar may be needed. But in case of well maintained oral hygiene with full dentition, an exposed third molar can be considered for extraction.
7. ***Evaluation of the clinician:*** Clinician should be honest while dealing with the case. Self evaluation should be done for his experience, capability to do the case, equipment he has or not for the completion of the case.

8. ***Systemic conditions:*** Most of the medical conditions don't contraindicate the endodontic treatment but patient should be thoroughly evaluated in order to manage the case optimally.

For predictable and successful endodontic therapy certain steps are needed and skipping a step may lead to the endodontic failure or less than desirable result.

These steps are as follows:

a. Take proper history and medical history of the patient
b. Make accurate diagnosis and treatment planning
c. Obtain adequate anesthesia
d. Isolate the tooth using rubber dam
e. Utilize adequate visualization and lighting
f. Obtain straight line access to the canals
g. Complete biomechanical preparation of the tooth
h. Efficient and safe use of nickel titanium files
i. Copiously irrigate at all stages
j. Obturate the canal three dimensionally
k. Give the coronal restoration to tooth.

TREATMENT PLANNING

The treatment planning signifies the planning of the management of the patient's dental problems in systematic and ordered way that assumes a complete knowledge of patient needs, nature of problem and prognosis of the treatment to be done very rarely, both dentist as well as the patient have complete picture of considerations mentioned above.

Thus the stage of assessment of a complete picture overlaps with the stages of decision making, treatment planning and treatment phase.

The Treatment Planning Consists of Following Phases

- Establishing the nature of the problem
- Decision making
- Planning required to deliver the selected treatment

A treatment plan for gaining the patient compliance and to have success in the pain management should progress as follows:

1. Treatment of acute problem which includes first step of endodontic treatment comprising of access opening, extirpation of pulp and allowing drainage through pulp space.
2. Oral hygiene instructions, diet instructions.
3. Temporary restoration of carious teeth, scaling and polishing.
4. Definitive restorations of carious teeth.
5. Complete root canal treatments of required teeth.
6. Do endodontic surgery if needed.
7. Evaluate the prognosis of treated teeth.
8. Provide post endodontic restorations.

Factors Influencing the Treatment Planning

There are several factors which influence the treatment planning. The clinician must be aware of problems encountered during treatment, short and long term goals of the treatment and patient's expectations about the treatment.

Factors Affecting Treatment Planning

1. Chief complaint regarding pain and swelling requires urgent treatment and planning for definitive solution.
2. Previous history of dental treatment (solve the residual problems of previous dental treatment).
3. Medical history (identify factors which can compromise dental treatment).
4. Intraoral examination (to know the general oral condition first before focusing on site of complaint so as not to miss the cause).
5. Extraoral Examination (to differentially diagnose the chief complaint).
6. Oral Hygiene.
7. Periodontal status (to see the periodontal foundation for long term prognosis of involved tooth).
8. Teeth and restorative status (to identify replacement of missing teeth, status of the remaining dentition).
9. Occlusion (to check functional relationship between opposing teeth, parafunctional habits, etc.).
10. Special Tests (to explore the unseen tissues).
11. Diagnosis (repeat the series of conclusions).
12. Treatment options (evaluate various options to decide the best choice for long term benefit of the patient).

MEDICAL CONDITIONS INFLUENCING ENDODONTIC TREATMENT PLANNING

Medical condition	*Modifications in treatment planning*
1. **Patients with valvular disease and murmers** Patients are susceptible to bacterial endocarditis secondary to dental treatment	Prophylactic antibiotics are advocated before initiation of the endodontic treatment
2. **Patients with Hypertension** i. In these patients, stress and anxiety may further increase chances of myocardial infarction or cerebrovascular accidents	• Give premedication • Plan short appointments

ii. Sometimes antihypertensive drugs may cause postural hypotension	• Use local anesthetic with minimum amount of vasoconstrictors
3. Myocardial Infarction	
i. Stress and anxiety can precipitate myocardial infarction or angina	• Elective endodontic treatment is postponed if recent myocardial infarction is present i.e. < 6 months
ii. Some degree of congestive heart failure may be present	• Reduce the level of stress and anxiety while treating patient
iii. Chances of excessive bleeding when patient is on aspirin	• Keep the appointments short and comfortable
iv. If pacemaker is present, apex locators can cause electrical interferences	• Use local anesthetics without epinephrine
	• Antibiotic prophylaxis is given before initiation of the treatment
4. Prosthetic Valve or Implants	
i. Patients are at high risk for bacterial endocarditis	• Prophylactic antibiotic coverage before initiation of the treatment
ii. Tendency for increased bleeding because of prolonged use of antibiotic therapy	• Consult physician for any suggestion regarding patient treatment
5. Leukemia	
Patient has increased tendency for:	• Consult the physician.
i. Opportunistic infections	• Avoid treatment during acute stages
ii. Prolonged bleeding	• Avoid long duration appointment
iii. Poor and delayed wound healing	• Strict oral hygiene instructions.
	• Evaluate the bleeding time and platelet status
	• Use of antibiotic prophylaxis
6. Cancer	
Usually because of radiotherapy and chemotherapy,	• Consult the Physician prior to treatment.
i. These patients suffer from xerostomia, mucositis, trismus and excessive bleeding	• Perform only emergency treatment if possible
	• Symptomatic treatment of mucositis, trismus and xerostomia
ii. Prone to infections because of bone marrow suppression	• Optimal antibiotic coverage prior to treatment.
	• Strict oral hygiene regimen
7. Bleeding Disorders	
In cases of hemophilia, thrombocytopenia, prolonged bleeding due to liver disease, broad spectrum antibiotics, patients on anticoagulant therapy patient experiences	• Take careful history of the patient
i. Spontaneous bleeding	• Consult the physician for suggestions regarding the patient
ii. Prolonged bleeding	• Avoid aspirin containing compounds and NSAIDs
iii. Petechiae, ecchymosis and hematoma	• In thrombocytopenia cases, replacement of platelets is done before procedure
	• Prophylactic antibiotic coverage to be given
	• Incase of liver disease, avoid drugs metabolized by liver
8. Renal Disease	
i. In this patient usually has hypertension and anemia	• Prior consultation with Physician
ii. Intolerance to nephrotoxic drugs	• Check the blood pressure before initiation of treatment.
iii. Increased susceptibility to opportunistic infections	• Antibiotic Prophylaxis Screen the bleeding time
iv. Increased tendency for bleeding	• Avoid drugs metabolized and excreted by kidney
9. Diabetes Mellitus	
i. Patient has increased tendency for infections and poor wound healing	• Take careful history of the patient
ii. Patient may be suffering from diseases related to cardiovascular system, kidneys and nervous system like myocardial infarction, hypertension, congestive heart failure, renal failure and peripheral neuropathy.	• Consult with physician prior to treatment
	• Note the blood glucose levels.
	• Patient should have normal meals before appointment
	• If patient is on insulin therapy, he/she should have his regular dose of insulin before appointment
	• Schedule the appointment early in the mornings
	• Antibiotics may be needed
	• Have instant source of sugar available in clinic
	• Patient should be evaluated for the presence

	of hypertension, myocardial infarction or renal failure
10. Pregnancy i. In such patients the harm to patient can occur via radiation exposures, medication and increased level of stress and anxiety ii. In the third trimester, chances of development of supine hypotension are increased	• Do the elective procedure in second trimester • Use the principles of *ALARA while exposing patients to the radiation • Avoid any drugs which can cause harm to the foetus • Consult the physician to verify the physical status of the patient and any precautions if required for the patient • Reduce the number of oral microorganism (by chlorhexidine mouthwash) • In third semesters, don't place patient in supine position for prolonged periods
11. Anaphylaxis Patient gives history of severe allergic reaction on administration of: i. Local anesthetics ii. Certain drugs iii. Latex gloves and rubber dam sheets	• Take careful history of the patient • Avoid use of agents to which patient is allergic • Always keep the emergency kit available • In case the reaction develops: – Identify the reaction – Call the physician – Place patient in supine position – Check vital signs – If vital signs are reduced, inject epinephrine into tongue – Provide CPR if needed – Admit the patient

*ALARA – As low as Reasonably Achievable

Sequence of Treatment Delivery Consists of Three Stages

- Initial treatment
- Definitive treatment
- Patient recall check up

Sequence of Treatment Delivery

a. ***Initial treatment:*** The initial treatment mainly aims at providing the relief from symptoms for example incision and drainage of an infection with severe pain and swelling, endodontic treatment of a case of acute irreversible pulpitis, etc.

Halting the progress of primary disease, i.e. caries or periodontal problem comes thereafter. Finally the patient is made to understand the disease and its treatment which further increases his/her compliance to the treatment. This approach is beneficial for the long term prevention of the dental caries and periodontal disease.

b. ***Definite treatment:*** Definitive treatment involves root canal treatment, surgical treatment, endodontic retreatment or the extraction of teeth with hopeless prognosis. In this phase tooth is given endodontic treatment with final restoration to maintain its form, function and aesthetics.

c. ***Patient recall check up:*** Regular patient recall is integral part of the planning process. It involves taking patient history, examination, diagnosing again for assessment of the endodontic treatment.

Before the clinician starts the endodontic therapy, a number of issues arise related to the treatment planning. These include maintaining asepsis of the operatory and infection control measures, premedication and administration of local anesthesia followed by review of radiographs and complete isolation of the operating site.

ASEPSIS IN ENDODONTICS

Endodontics has long emphasized the importance of aseptic techniques using sterilized instruments, disinfecting solutions and procedural barriers like rubber dam. Dental professionals are exposed to wide variety of microorganisms in the blood and saliva of patients, making infection control procedures of utmost importance. The common goal of infection control is to eliminate or reduce number of microbes shared between the people. The procedures of infection control are designed to kill or remove microbes or to protect against contaminations.

Infection Control

The basic principles of asepsis and infection control used in general dentistry apply to endodontics with little variance.

Universal use of gloves, gowns, eye shields, goggles, plastic aprons; use of aseptic techniques; proper use of disinfectants and instruments sterilization; appropriate handling and disposal of sharps and infectious waste work

together to provide a professional health care atmosphere that provides adequate protection and treatment according to principles of infection control.

Guidelines for Universal Precautions for Maintaining Asepsis in the Operatory Field

- Use of barrier technique, i.e. use of gloves, gowns, protective eye wear, head cap and aprons.

Steps to Infection Control include:

- Patient screening
- Personal protection
- Instrument sterilization
- Use of aseptic technique
- Surface disinfection
- Procedural barriers
- Waste disposal
  - Disinfection and sterilization procedures
  - Appropriate handling and disposal of waste
  - Use of proper hand washing techniques

Principles of Infection Control

A successful infection control strategy is based on the following principles:

1. Appreciation of basic microbiology and modes of disease transmission.
2. Conscientious hygiene, including appropriate hand washing and cleaning of work area, equipments and materials in dental office.
3. Use of recommended sterilization and disinfection procedure.
4. Use of disposable or sterilisable equipments.
5. Surveillance of nosocomial/iatrogenic and occasionally acquired infection.

Routes of Disease Transmission

In endodontics, disease can be transmitted from patient to dentist, dentist to patient and patient to patient through cross contamination.

For an Infection to be Transmitted, the following Conditions are Required

- A pathogenic organism.
- A source which allows pathogenic organism to survive and multiply.
- Mode of transmission.
- Route of entry.
- A susceptible host.

Mode of Transmission

- Direct contact from one person to another.
- Indirect contact via instruments.
- Droplets or aerosol/spray.

Routes of Entry of Microorganisms can be:

- Inhaled
- Implanted
- Injected
- Splashed on to the skin/mucosa

Sterilization

Sterilization is the process of destroying all microbial life from an article or surface, including spores. The term sterilization can only be applied to instruments, and not to skin, where only antisepsis can be achieved.

Classification of Instrument Sterilization

The center for disease control and prevention categorize patient care items into three categories. These categories are also known as ***Spaulding classification (given in 1968)***.

Classification of Instrument Sterilization

Category	*Definitions*	*Examples*
Critical	Where instruments enter or penetrate into sterile tissue, cavity or blood stream	• Surgical blades and instruments • Surgical dental bur
Semicritical	Which contact intact Mucosa or nonintact skin	• Amalgam condenser • Dental handpieces • Mouth mirror (dental) • Saliva ejectors
Noncritical	Which contact intact skin	• Pulse oximeter • Stethoscope • Light switches • Dental chair

Various Methods of Sterilization are:

- Moist heat/steam heat (autoclave)
- Dry heat (hot air oven, glass bead sterilizer)
- Chemical vapor pressure sterilization
- Ethylene oxide sterilization

The Entire Process of Sterilization can be Divided into Three Steps:

- Cleaning of instruments (presterilization cleaning)
- Sterilization process
- Storage

Cleaning of Instruments

All surgical instruments must be cleansed of debris including blood, saliva and necrotic material, which can interfere with

sterilization process. The instruments should be cleaned thoroughly by scrubbing with soap and water or a detergent solution or with ultrasonic cleaning devices. Ultrasonic cleaner is considered more effective than normal cleaning with hand.

Sterilization Process

Moist heat/steam heat (autoclave): It is considered as a better method of sterilization due to higher efficiency of penetration of moist heat than dry heat. The commonly accepted specifications for autoclave are 121°C (250°F) at 15 psi with minimum holding time 15 minutes (Fig. 7.2).

Fig. 7.2: Autoclave for moist heat sterilization

Advantages

i. Better penetration of moist heat
ii. Instruments can be wrapped prior to sterilization, this increases the shelf life of instruments
iii. Effective and rapid method of sterilization.
iv. Steam under pressure does not destroy cotton or cloth products
v. Most of the dental instruments can be autoclaved.
vi. Sterilization is confirmable.

Disadvantages

i. Dulling and corrosion of sharp instruments.
ii. Items sensitive to elevated temperatures and moisture cannot be effectively autoclaved.
iii. Damage to plastic and rubber products.
iv. Instruments need to be air dried at the end of cycle.

Flash Autoclave Sterilizer

These are rapid speed autoclaves with shorter sterilization cycle, i.e. 3-5 minutes. These have smaller chamber size and are primarily indicated for use in dentistry.

Advantages:

- Takes less time for sterilization
- Better penetration of moist heat
- Effective and rapid method of sterilization.

Dry Heat Sterilization

It is an alternative method for sterilization of instruments, particularly, the sharp instruments. Dry heat kills the microorganisms by denaturation and oxidation process.

Hot Air Oven

The instruments are packed loosely inside the oven and a temperature of 160°C is achieved with a holding time of one hour.

Advantages

1. No corrosion is seen in carbon steel instruments and burs.
2. Instruments are dry after cycle.
3. Low cost of equipment.
4. Maintains the sharpness of cutting instruments.
5. Sterilization is confirmable.

Disadvantages

1. Poor penetrating capacity of dry heat.
2. Long cycle is required for sterilization (period).
3. Damage to plastic and rubbers.
4 Instruments must be thoroughly dried before placing them in sterilization.
5 Higher temperature of sterilizer may damage the instruments.
6 Dry heat sterilizer must be calibrated and monitored.

Glass Bead Sterilizer

It is rapid method of sterilization which is used for sterilization of endodontic instruments such as reamers, files and broaches, etc. (Fig. 7.3A and B). It usually uses table salt which consists approximately of 1 percent sodium silicoaluminate, sodium carbonate or magnesium carbonate so it pours more readily and does not fuse under heat. Salt can be replaced by glass beads provided the beads are

A

B

Figs 7.3A and B: Glass bead sterilizer for endodontic instruments

smaller than 1 mm in diameter because larger beads are not efficient in transferring the heat to endodontic instruments due to presence of large air spaces between the beads.

The instruments can be sterilized in **5 to 15** seconds at a temperature of **437-465° F(260°C)** even when inoculated with spores.

The specific **disadvantage** of these sterilizers is that the handle portion is not sterilized and therefore these articles are not entirely 'sterile'. These are not recommended unless absolutely required.

Advantages

- Commonly used salt is table salt which is easily available and cheap.
- Salt does not clog the root canal. If it is carried in to the canal, it can be readily removed by irrigation.

Chemical Vapor Pressure Sterilization

The chemical vapor pressure sterilization usually operate at **127 °C to 131°C at 20 psi for 20 minutes**. This method of sterilization is based on the factor of heat, water and chemical. The chemicals include formaldehydes, acetones and alcohols.

Advantages

1. Lack of corrosion of instruments.
2. Shorter duration of sterilization cycle.
3. Low temperature does not damage delicate instruments.
4. Instruments can be wrapped during the process, this increase their shelf life.
5. Sterilization is confirmable.

Disadvantages

1. Adequate ventilation is required to expel chemical vapors.
2. Instruments must be dried before sterilization because water present on instruments can interfere with sterilization procedure.
3. Chemical vapors can damage sensitive instruments.
4. May emit offensive vapor smell.

Ethylene Oxide Sterilization

This method of sterilization is used for sterilizing heat sensitive instruments. It is highly penetrating, non corrosive agent with cidal action against bacteria, spores and viruses.

Advantages

1. It penetrates extremely well.
2. It is non toxic in nature.
3. Many heat sensitive articles, i.e. plastic, rubber can be sterilized.

Disadvantages

1. High cost of the equipment.
2. Explosive and inflammable in nature.
3. Toxicity of the gas.

Disinfection

Disinfection can be defined as the destruction of all pathogenic organisms but not spores from inanimate surfaces. There are three categories of disinfectants.

- ***High level disinfectant:*** It destroys all microorganisms except for some bacterial spores. For example gluteraldehyde and hydrogen peroxide.
- ***Intermediate level disinfectant:*** It destroys all bacteria, fungi and viruses except spores. It is tuberculocidal in action. For example iodophores, quaternary ammonium compounds with alcohol, etc.
- ***Low level disinfectant:*** It destroys some bacteria, fungi and viruses. It does not kill mycobacterium tuberculosis and spores for example quaternary ammonium compounds.

Personal Barrier Protection

Barrier protection in infection control is an important factor in disease prevention (Fig. 7.4).

In dentistry, this involves the use of protective barriers such as gloves, masks, protective eyewear and protective clothing.

Fig. 7.4: Barrier protection using gloves, mask, protective eyewear

Hand Washing and Gloving

Hand washing continues to be important mean for personal protection and of diseases prevention. All jewellery, rings and stones should be removed before washing. The nails should be checked for cleanliness. Hand washing serves two purposes; first, it helps in removing the superficial contaminants. Second, it reduces bacterial count on the skin. Before surgery, one should use a prescribed surgical scrub washing and rinsing from hands towards elbow. Various hand cleansers are available in the market such as Hibiscrub (4% chlorhexidine gluconate) and betadine (7.5% povidone-iodine).

Sterilization of the dental equipment

S.No.	*Instrument*	*Method*
1.	Mouth Mirror Probes Explorer Tweezers	Autoclave
2.	Endodontic Instruments—Files, reamers, broaches	Autoclave
3.	Steel, burns	Disposable
4.	Carbide and Diamond burs	Autoclave
5.	Local anaesthetic cartridges	Presterilized disposable
6.	Needles	Disposable
7.	***Rubber dam equipment***	
	a. Carbon steel clamps and metal frames	Dry heat ethylene oxide autoclave
	b. Punch	Dry heat ethylene oxide
8.	Gutta percha points	Dip in 5.2% sodium hypochlorite for 1 min and then rinse with hydrogenperoxide and dry it

Gloves should be worn all the time during treatment procedure. Gloves help in protecting the dental team members from direct contact with patient microbes and also patients from contact with microbes on the hands of the dental team members. Cuts and abrasions on the hands are suggested as main reason for high recurrence of hepatitis B in those who do not use gloves routinely.

Sterile latex or vinyl gloves should be used for surgical procedure. Gloves should not be reused. Another important aspect about glove is that one must remove the torn glove as soon as they are noticed, followed immediately by hand washing and use of fresh gloves.

Face Masks

This is also another important protective barrier which provide protection from inhalation of aerosols generated by handpieces and air water syringes. Masks may be composed of synthetic fiber, paper or gauze. Disposable masks made of synthetic fibers are better.

Protective Eyewear

It is advisable to use protective eyewear whenever there are chances of contamination of the eyes with aerosols or splashes of body fluids is possible. The dentist should also make arrangements for patients eye protection during treatment.

Protective Clothing

As with eyewear or masks, protective clothing is also worn under conditions generating sprays, spatter, splashes of body fluids or contaminated solutions.

PAIN CONTROL

Pain control is one of the most important step while performing root canal therapy. The clinician must have accurate knowledge of local anesthesia and various techniques of administration of painless anesthesia.

Local Anesthesia

Definition

It is defined as a loss of sensation in a circumscribed area of the body caused by depression of excitation in nerve endings or an inhibition of the conduction process in peripheral nerves.

Classification of Local Anesthetic Agents

All local anesthetics except cocaine are synthetic. They are broadly divided into two groups, i.e. ester and amide (non ester) group.

A. Based on chemical structure

I. ***Ester group***
   1. Cocaine
   2. Benzocaine
   3. Procaine
   4. Tetracaine

II. ***Amide (Nonester group)***
1. Lidocaine
2. Mepivacaine
3. Prilocaine
4. Etidocaine
5. Bupivacaine

B. Based on duration of action

1. Short acting
   - Procaine
2. Intermediate acting
   - Lidocaine
3. Long acting
   - Bupivacaine

The primary action of the local anesthetics agent in producing a nerve conduction block is to decrease the nerve permeability to sodium (Na^+) ions, thus preventing the inflow of Na^+ ions into the nerve. Therefore local anesthetics interfere with sodium conductance and inhibit the propagation of impulse along the nerve fibres (Fig. 7.5A).

In tissues with lower pH, local anesthetics show slower onset of anesthesia while in tissues with higher pH, local anesthetic solution speeds the onset of anesthesia. This happens because at alkaline pH, local anesthetic is present in undissociated base form and it is this form penetrates the axon.

Composition of a Local Anesthetic Agent

- Local anesthetic - salt form of lidocaine hydrochloride.
- Vasoconstrictor - epinephrine
- Preservative for vasoconstrictor—sodium bisulfite
- Isotonic solution—sodium chloride
- Preservative—methyl paraben
- Sterile water to make the rest of the volume

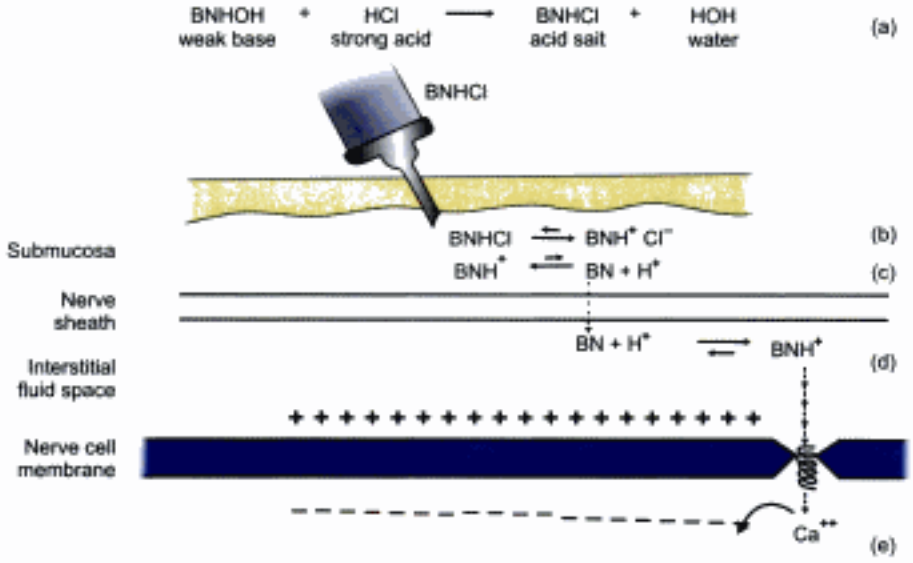


Fig. 7.5A: Mechanism of action of local anaesthetic at normal pHA

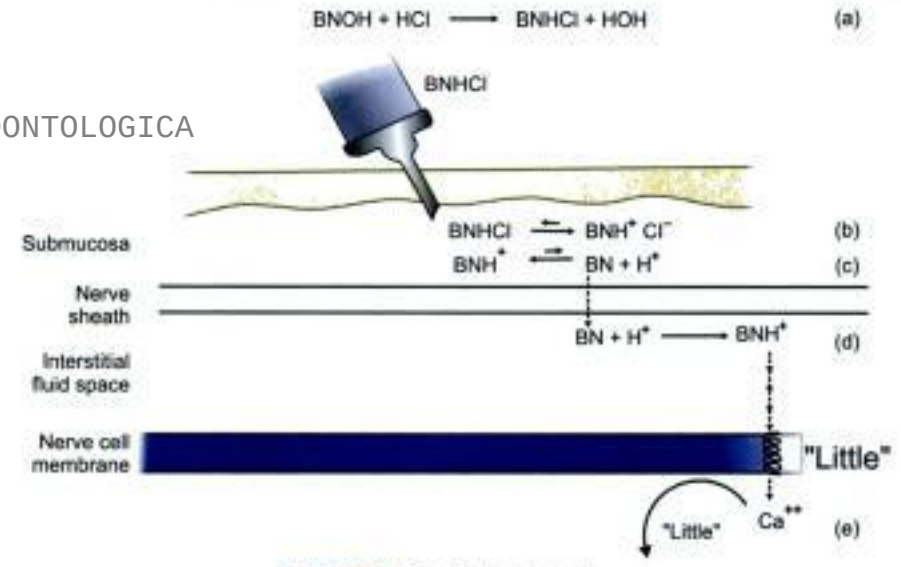


Fig. 7.5B: Action of LA at low pH

Various Techniques of Local Anesthesia

- Local infiltration technique
- Supraperiosteal technique
- Field block technique
- Nerve block technique

Techniques Used for Maxillary Tissues

1. Supraperiosteal technique
*2. Anterior and middle superior alveolar nerve block
3. Posterior superior alveolar nerve block
4. Greater palatine nerve block (anterior palatine nerve block)
5. Nasopalatine nerve block
*6. Maxillary nerve block
7. Periodontal ligament injection

* Both can be given intraorally and extraorally while all other are given intraorally only.

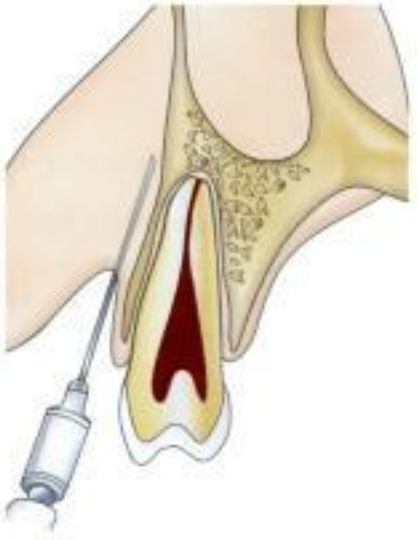


Fig. 7.6: Supraperiosteal technique of local anesthesia

Techniques for Anesthetizing Maxillary Teeth

1. Supraperiosteal Technique

It is also known as local infiltration and is most frequently used technique for obtaining anesthesia in maxillary teeth.

Technique

The needle is inserted through the mucosa and the solution is slowly deposited in close proximity to the periosteum, in the vicinity of the apex of the tooth to be treated (Fig. 7.6).

Advantages

- It is simple to learn.

Disadvantages

- Multiple injections are required for large area.

2. (a) Anterior Superior Alveolar Nerve Block

Nerve anaesthesized with this block are anterior, and middle superior alveolar nerve and infraorbital nerve; inferior palpebral, lateral nasal, superior labial nerves. It is given

for anaesthesizing the maxillary incisors, canines, premolars and mesiobuccal root of first molar (in 70% of cases). In this the target area is infraorbital foramen.

Technique

Needle is inserted in the mucobuccal fold over the maxillary first premolar and directed towards the infraorbital foramen, once you have palpated. After aspirating, slowly deposit the solution 0.9 to 1.2 ml in the vicinity of the nerve (Fig. 7.7).

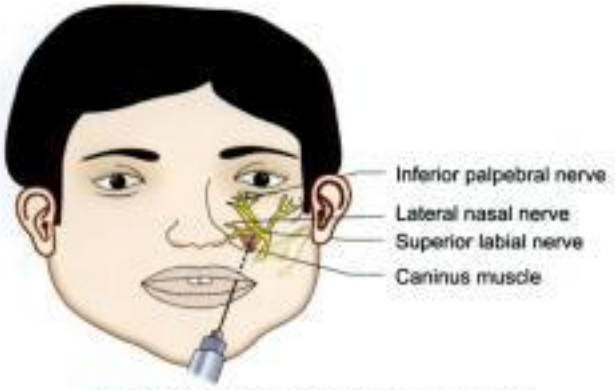


Fig. 7.7: Anterior superior alveolar nerve block

2. (b) Middle Superior Alveolar Nerve Block

It is used for anaesthesizing the middle superior alveolar nerve and its terminal branches. It is given for anaesthesizing the maxillary first and second premolars and mesiobuccal root of the first molar.

Technique

Needle is inserted into the mucobuccal fold above the second premolar. After aspirating, slowly deposit local anaesthetic solution (i.e. 0.9 to 1.2 ml).

3. Posterior Superior Alveolar Nerve Block

It is used for posterior superior alveolar nerves. It is given for anaesthesizing the maxillary third, second and first molar (sometimes mesiobuccal root is not anaesthesized) (Fig. 7.8) and overlying structures (buccal mucosa and bone).

Technique

Needle is inserted distal to the zygomatic process in the mucobuccal fold over the maxillary molar teeth. After aspirating slowly deposit local anaesthetic solution.

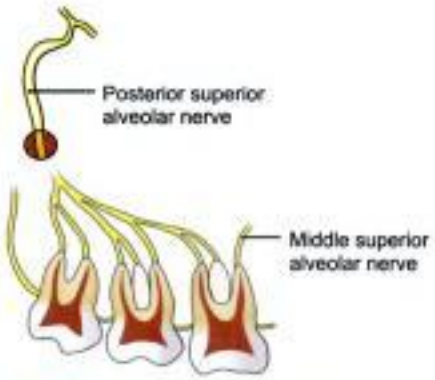


Fig. 7.8: Posterior superior alveolar nerve

4. Greater Palatine Nerve Block

It is used for anaesthesizing greater palatine nerve. It is given for anaesthesizing posterior portion of hard palate and its overlying soft tissue, up to the first bicuspid.

Technique

In this target area is greater palatine foramen. The needle is inserted from the opposite side of mouth at a right angle to the foramen which lies 1 cm from palatal gingival margin towards midline (Fig. 7.9). After aspirating, deposit the solution slowly.

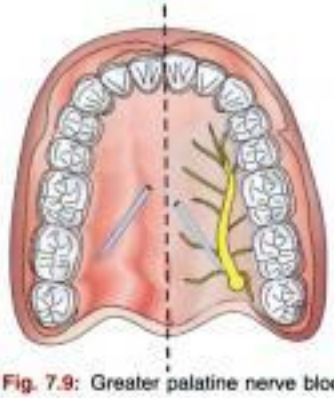


Fig. 7.9: Greater palatine nerve block

5. Nasopalatine Nerve Block

It is used for anaesthesizing anterior portion of the hard palate (soft and hard tissues), extending from one side premolar to other side of first premolar.

Technique

Needle is inserted in intraseptal tissue between the maxillary central incisors. Deposit slowly the local anaesthetic solution in the tissue (Fig. 7.10).

Fig. 7.10: Nasopalatine nerve block

6. Maxillary Nerve Block

It is used for anesthesizing the maxillary nerve of trigeminal nerve. In this, different techniques which can be used are:

i. High tuberosity approach
ii. Greater palatine canal approach
iii. Extraoral technique

7. Periodontal Ligament Injection

It is used for anaesthesizing terminal nerve endings in vicinity of the injection. The local anaesthetic solution is deposited into the periodontal ligament or membrane.

Techniques

Needle is inserted along the long axis of the tooth either on mesial or distal of the root (Fig. 7.11). Deposit local anesthetic solution (0.1-0.2 ml) slowly.

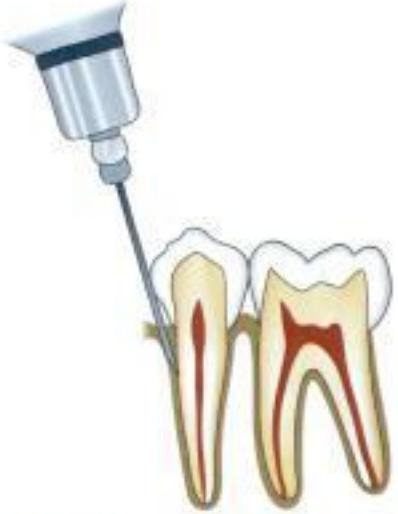


Fig. 7.11: Periodontal ligament injection

Advantages

- Rapid onset of action
- It is a useful adjunct to normal local anaesthesia
- Provides specific analgesia to isolated tooth.

Disadvantages

- Post injection discomfort due to temporary extrusion.

Various Mandibular Anesthesia Techniques

1. Inferior alveolar nerve block
2. Long buccal nerve block
3. Mandibular nerve block
   - Gow-gates technique
   - Extra oral approach
4. Vazirani-Akinosi closed mouth technique
5. Mental nerve block

Techniques of Anesthetizing Mandibular Teeth

1. Inferior Alveolar Nerve Block

It is used for anaesthesizing inferior alveolar nerve, lingual nerve and its terminal branches, i.e. mental and incisive.

The areas anesthesized are:

i. Mandibular teeth
ii. Body of the mandible and inferior portion of the ramus
iii. Buccal mucous membrane and its underlying tissues only up to first molar
iv. Anterior two third of tongue, lingual soft tissues, floor of the oral cavity.

Technique

The target in this technique is inferior alveolar nerve. The operator should first palpate the anterior border of the ramus. Its deepest concavity is known as coronoid notch which determines the height of injection. The thumb is placed over the coronoid notch and also in contact with internal oblique ridge. The thumb is moved towards the buccal side, along with buccal sucking pad. This gives better exposure to pterygomandibular raphe (Fig. 7.12). Insert the needle parallel to occlusion of mandibular teeth from opposite side of mouth. Needle is finally inserted lateral to pterygomandibular raphe in pterygomandibular space.

Bone must be contacted as it determines the penetration depth. Solution required in this block vary from 1.5-1.8 ml.

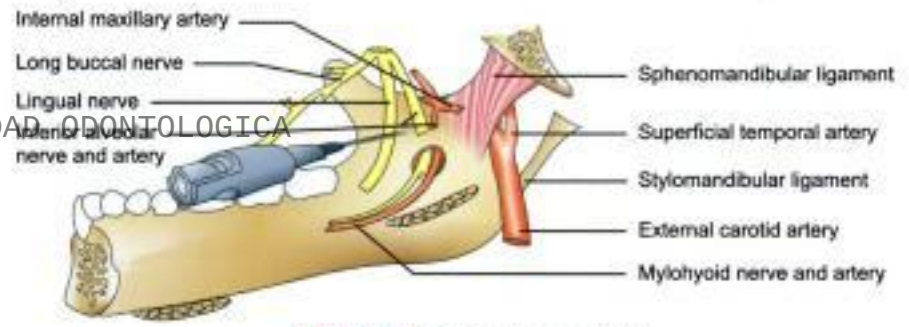


Fig. 7.12: Inferior alveolar nerve block

2. Long Buccal Nerve Block

It is used for anesthesizing buccal mucosa of mandibular molar teeth.

Technique

In this, target is buccal nerve. Insert needle in the mucosa distal and buccal to last lower molar tooth in the oral cavity (Fig. 7.13).

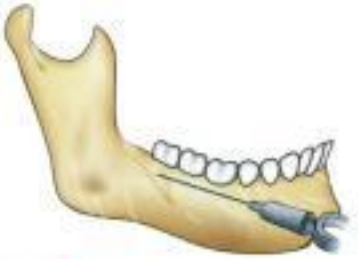


Fig. 7.13: Long buccal nerve block

3. Mandibular Nerve Block

For complete anesthesizing the mandibular nerve, following techniques may be used

i. Gow-gates technique
ii. Extra oral approach

4. Vazirani-Akinosi Closed Mouth Technique

It is usually preferred in patients who have limited/restricted mouth opening. The areas anaesthesized by this technique is very much similar to the area anaesthesized by inferior alveolar nerve block. Target area is pterygomandibular space.

Technique

In this technique first patient is asked to bring teeth in the occlusion. Needle is positioned at the level of mucogingival junction of maxillary molars. Needle is penetrated through the mucosa in the embrasure just medial to the ramus (Fig. 7.14). When tip of the needle reaches the target area, approximate 2 ml of solution is deposited slowly.

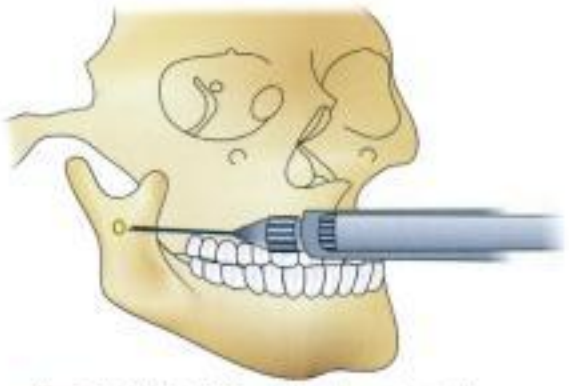


Fig. 7.14: Vazirani-Akinosi closed mouth technique

5. Mental Nerve Block

It is used for anesthesizing the buccal soft tissues anterior to the mental foramen and up to the midline.

Technique

Insert the needle in the mucobuccal fold just anterior to mental foramen (Fig. 7.15). Slowly deposit the solution into the tissue.

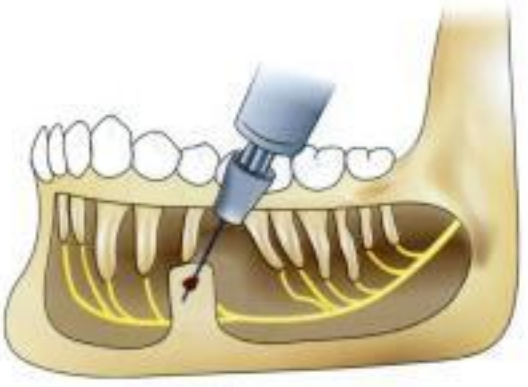


Fig. 7.15: Mental nerve block

Intrapulpal Injection

Adequate pulpal anesthesia is required for treatment of pulpally involved tooth. Mandibular teeth usually offers some problems in obtaining profound anesthesia. This injection control spain, both by applying pressure and utilizing the pharmacologic action of local anaesthetic agent.

Indications

- Lack of obtaining profound anesthesia in pulpally involved teeth by other techniques (mentioned above).

Nerves Anesthesized

- Terminal nerve endings at the site of injection.

Technique

1. Insert 25 or 27 gauge needle firmly into the pulp chamber (Fig. 7.16).

Fig. 7.16: Intrapulpal injection

2. Before inserting the needle, patient must be informed that he/she may experience a brief period of sensitivity (mild to very painful) after giving the injection.
3. Always deposit local anesthetic solution under pressure as back pressure is shown to be the major factor is producing anesthesia (Fig. 7.17).
   - For creating back-pressure, block the access with stoppers (cotton pellet). To prevent back-flow, other stoppers which can be used are gutta-percha, waxes or pieces of rubber.
   - Deposit a very small amount of solution (0.2-0.3 ml) under pressure (5-10 seconds).

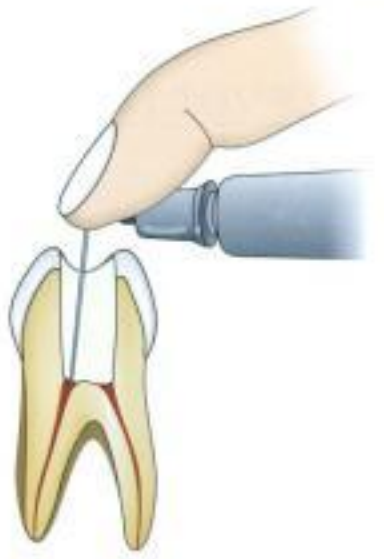


Fig. 7.17: Inject local anesthetic solution under pressure to prevent backflow while giving intrapulpal injection

   - Sometimes, bending of needle is done for gaining access to the canal.

Advantages

1. Requires less volume
2. Early onset
3. Easy to learn

Disadvantages

1. Results are not predictable as it may vary (it should always be given under pressure).
2. Taste of local anaesthetic drug is not accepted by patients as it may spill during administration of intrapulpal injection.
3. Brief pain during or after insertion of solution (not tolerated by some patients).

ISOLATION OF THE TOOTH

Isolation of the tooth requires proper placement of the rubber dam/ dental dam. It helps to isolate the pulp space from saliva and protects oral tissues from irrigating solutions, chemicals and other instruments. Rubber dam was introduced by ***Barnum, a New York dentist in 1863*** (Fig. 7.18).

Rubber dam can be defined as a flat thin sheet of latex/ non-latex that is held by a clamp and frame which is

Fig. 7.18: Rubber dam

perforated to allow the tooth/teeth to protrude through the perforations while all other teeth are covered and protected by sheet.

Advantages of Using a Rubber Dam

1. It is raincoat for the teeth.
2. It helps in improving accessibility and visibility of the working area.
3. It gives a clean and dry field while working.
4. It protects the lips, cheeks and tongue by keeping them out of the way.
5. It helps to avoid unnecessary contamination through infection control.
6. It protects the patient from inhalation or ingestion of instruments and medicaments.
7. It helps in keeping teeth saliva free while performing a root canal so that tooth does not get decontaminated by bacteria present in saliva.
8. It improves the efficiency of the treatment.
9. It limits bacterial laden splash and splatter of saliva and blood.
10. It potentially improves the properties of dental materials.
11. It provides protection of patient and dentist.

Rubber Dam Sheet (Fig. 7.19)

The rubber dam is available in size 6 × 6 squares and colours are usually green or black. It is available in three thicknesses, i.e. light, medium and heavy. The middle grade is usually preferred as finest is more prone to tearing and thickest more difficult to apply. Latex free dam is necessary as number of patients are increasing with latex allergy. ***Flexi Dam*** is latex free dam of standard thickness with no rubber smell.

Thickness of Rubber Dam Sheet

Thin	-	0.15 mm
Medium	-	0.2 mm
Heavy	-	0.25 mm
Extra Heavy	-	0.30 mm
Special Heavy	-	0.35 mm

Fig. 7.19: Rubber dam sheets

Rubber dam clamps, to hold the rubber dam on to the tooth are available in different shapes and sizes (Fig. 7.20). Clamps mainly serve two functions.

i. They anchor the rubber dam to the tooth (Fig. 7.21)
ii. Help in retracting the gingivae.

Fig. 7.20: Rubber dam clamps

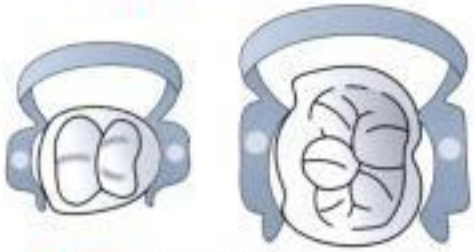


Fig. 7.21: Use of rubber dam clamp on tooth

Rubber dam clamps can be divided into two main groups on the basis of jaw design.

i. Bland
ii. Retentive

i. ***Bland clamps*** are usually identified by the jaws, which are flat and point directly towards each other. In these clamps, flat jaws usually grasp the tooth at or above the gingival margin. They can be used in fully erupted tooth where cervical constriction prevents clamp from slipping off the tooth.
ii. ***Retentive clasps:*** As the name indicates, these clasps provide retention by providing four-point contact with the tooth. In these, jaws are usually narrow, curved and slightly inverted which displace the gingivae and contact the tooth below the maximum diameter of crown.

Both flanges are further subdivided into:

- Winged
- Wingless

Rubber dam clamp can also be divided on the basis of material used.

i. Metallic
ii. Non-metallic

Metallic

Traditionally, clamps have been made from tempered carbon steel and more recently from stainless steel.

Non-metallic

Non-metallic are made from polycarbonate plastic. An advantage of these clamps over metallic is radiolucency.

A good length of dental floss should always be passed through the holes in the clamp as a security in case it is dropped in the month or the bow fractures.

Rubber Dam Clamps

22 Similar to #207, but wingless
27 Similar to #206, but wingless, festooned
29 For upper and lower bicuspids, with broad beaks
206 For upper and lower bicuspids, with festooned beaks
207 For upper and lower bicuspids, with flat beaks
208 For bicuspids (large), with similar pattern to #207
209 For lower bicuspids, with flat beaks
0 For small bicuspids and primary central incisors
00 For very small bicuspids and primary central incisors
1 For roots, with deep festooned beaks
2 For lower bicuspids, with flat beaks
2A Similar to #2, but with large beaks
W2A Similar to #2A, but wingless
P-1,#P-2 For children's first molars

Rubber Dam Forceps

Rubber dam forceps are used to carry the clamp to the tooth and it is essential that these do not have deep grooves at their tips or they become very difficult to remove once the clamp is in place (Fig. 7.22).

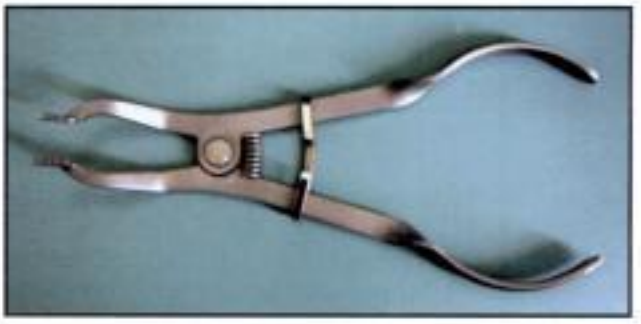


Fig. 7.22: Rubber dam forcep

Rubber Dam Frame

Rubber dam frames support the edges of rubber dam. They have been improved dramatically since the old style with the huge "butterflies". Modern frames have sharp pins which easily grip the dam. These are mainly designed with the pins that slope backwards.

Rubber dam frames serve following purposes:

i. Supporting the edges of rubber dam
ii. Retracting the soft tissues
iii. Improving accessibility to the isolated teeth.

Rubber dam frames are available in either metal or plastic. Plastic frames have advantage of being radiolucent.

As we see when taut, rubber dam sheet exerts too much pull on the rubber dam clamps, causing them to come loose, especially clamps attached to molars. To overcome this problem, a new easy-to-use rubber dam frame ***(Safe-T-Frame)*** has been developed that offers a secure fit without stretching the rubber dam sheet. Instead, its "snap-shut" design takes advantage of the clamping effect on the sheet caused when its two mated frame members are firmly pressed together. In this way the sheet is securely attached, but without being stretched. Held in this manner, the dam sheet is under less tension, and hence, exerts less tugging on clamps—especially on those attached to molars.

Rubber Dam Punch

Rubber dam punch is used to make the holes in the rubber sheet through which the teeth can be isolated (Fig. 7.23). The punch must produce a clean cut hole every time. Two

Fig. 7.23: Rubber dam punch

types of holes are made, single and multi-hole. Single holes are used in endodontics mainly. If rubber dam punch is not cutting cleanly and leaving behind a tag of rubber, the dam will often split as it is stretched out.

Rubber Dam Accessories

A ***lubricant or petroleum jelly*** is usually applied on the undersurface of the dam. It is usually helpful when the rubber sheet is being applied to the teeth.

Dental floss: It is used as flossing agent for rubber dam in tight contact areas. It is usually required for testing interdental contacts.

Rubber dam napkin: which is a sheet of absorbent materials usually placed between the rubber sheet and soft tissues. It is generally not recommended for isolation of single tooth.

Recent Modifications in the Designs of Rubber Dam

Insti-Dam is recently introduced rubber dams for quick, convenient rubber dam isolation.

Salient Features of Insti–Dam:

- It is natural latex dam with prepunched hole and built-in white frame.
- Its compact design is just the right size to fit outside the patient's lips.
- It is made up of stretchable and tear-resistant, medium gauge latex material.
- Radiographs may be taken without having to remove the dam.
- Built-in flexible nylon frame eliminates bulky frames and sterilization.
- Off-center, pre-punched hole customizes fit to any quadrant — add more holes if desired.

Another recently introduced dam is **Handi dam.** This is preframed rubber dam eliminates the need for traditional frame (Fig. 7.24).

Fig. 7.24: Handi-dam

Handi dam is easy to place and saves time of both patient as well as doctor. It allows easy access to oral cavity during the procedure. Another newer type of rubber dam is also available which does not require a frame **'dry dam'**.

Before placement of rubber dam, following procedures should be done:

1. Thorough prophylaxis of the oral cavity.
2. Check contacts with dental floos.
3. Check for any rough contact areas.
4. Anesthesize the gingiva if required.
5. Rinse and dry the operated field.

Methods of Rubber Dam Placement

Method I

Clamp placed before rubber dam.

- Select an appropriate clamp according to the tooth size.
- Tie a floss to clamp bow.
- Larger holes are required in this technique as rubber dam has to be stretched over the clamp. Usually two or three overlapping holes are made.
- Stretching of the rubber dam over the clamps can be done in following sequence.
  - Stretch the rubber dam sheet over the clamp
  - Then stretch the sheet over the buccal jaw and allow to settle into place beneath that jaw
  - Finally, the sheet is carried to palatal/lingual side and released.

This method is mainly used in posterior teeth in both adults and children except third molar.

Method II

Placement of Rubber Dam and Clamp Together

1. Select an appropriate clamp according to tooth anatomy.
2. Tie a floss around the clamp and check the stability.
3. Punch the hole in rubber dam sheet.
4. Clamp is held with clamp forceps and its wings are inserted into punched hole.

5. Both clamp and rubber dam are carried to the oral cavity and clamp is tensed to stretch the hole.
6. Both clamp and rubber dam is advanced over the crown. First, jaw of clamp is tilted to the lingual side to lie on the gingival margin of lingual side.
7. After this, jaw of the clamp is positioned on buccal side.
8. After seating the clamp, again check stability of clamp.
9. Remove the forcep from the clamp.
10. Now, release the rubber sheet from wings to lie around the cervical margin of the tooth.

Method III

This method is "Split dam technique" in which rubber dam is placed to isolate the tooth without the use of rubber dam clamp. In this technique, two overlapping holes are punched in the dam. The dam is stretched over the tooth to be treated and over the adjacent tooth on each side. Edge of rubber dam is carefully teased through the contacts of distal side of adjacent teeth.

This technique is ***indicated***:

- To isolate anterior teeth.
- When there is insufficient crown structure.
- When isolation of teeth with porcelain crown is required. In such cases placement of rubber dam clamp over the crown margins can damage the cervical porcelain.

Management of Difficult Cases

1. Malpositioned teeth:

To manage these cases, following ***modifications*** are done.

i. Adjust the spacing of the holes.
ii. In tilted teeth, estimate the position of root center at gingival margin rather than the tip of the crown.
iii. Another approach is to make a customized cardboard template.
iv. Tight broad contact areas can be managed by
   a. Wedging the contact open temporarily for passing the rubber sheet
   b. Use of lubricant

Extensive Loss of Coronal Tissue

When sound tooth margin is at or below the gingival margin because of decay or fracture, the rubber dam application becomes difficult. In such cases, to isolate the tooth:

i. Use retentive clasps
ii. Punch a bigger hole in the rubber dam sheet so that it can be stretched to involve more teeth, including the tooth to be treated.
iii. In some cases, the modification of gingival margin can be tried so as to provide supragingival cavity margin. This can be accomplished by gingivectomy or the flap surgery.

Crowns with Poor Retentive Shapes

Sometimes anatomy of teeth limits the placement of rubber dam. (lack of undercuts and retentive areas) In such cases, following can be done:

i. Placing clamp on another tooth.
ii. By using clamp which engages interdental spaces below the contact point
iii. By building retentive shape on the crown with composite resin bonded to acid etched tooth surface.

Teeth with Pocelain Crowns

In such cases, placing a rubber dam may cause damage to porcelain crown. To avoid this:

i. Clamp should be placed on another tooth.
ii. Clamp should engage below the crown margin
iii. Do not place clamp on the porcelain edges
iv. Place a layer of rubber dam sheet between the clamp and the porcelain crown which acts as a cushion and thus minimizes and localized pressure on the porcelain.

Leakage

Sometimes leakage is seen through the rubber dam because of the accidental tears or holes. Such leaking gaps can be sealed by using cavit, periodontal packs, liquid rubber dam or oraseal. Now-a-days, the rubber dam adhesive can be used which can adhere well to both tooth as well as rubber dam. For sealing the larger gaps, the rubber dam adhesives in combination with orabase can be tried. If leakage persists in spite of these efforts, the rubber dam sheet should be replaced with new one.

Contraindications of use of rubber dam

- Asthmatic patients
- Allergy to latex
- Mouth breathers

Summary

Efficient and successful endodontics begins with proper case selection. The clinician must know his/her limitations and select cases accordingly. Since success of endodontic treatment depends upon many factors which can be modified to get better before initiating the treatment. Therefore accurate and thorough preparation of both patient as well as tooth to be treated should be carried out to achieve the successful treatment results.

8 Endodontic Instruments

INTRODUCTION

Although variety of instruments used in general dentistry, are applicable in endodontics, yet some special instruments are unique to endodontic purpose.

In early 1900's, there was availability of variety of tools like path finders, barbed broaches, reamers, files, etc. In that time, every clinical picture presented was tackled with unique formulae utilizing permutations and combinations of tools, medicaments and sealants available. In other words there was little uniformity in quality control, taper of canal or instrument and filling materials in terms of size and shape.

The year 1958 was hallmark year in the history of endodontic instrumentation. The manufacturers came together and a consensus was reached on instruments and obturation materials for root canal therapy. Then in 1959, standardized instruments and filing materials were introduced. In this standardization:

a. For each instruments and filling materials a formula for diameter and taper was made.
b. Formulae for graduated increment in size from one instrument to another were given.
c. Based on instrument diameter, numbering system for instruments was developed.

In 1968, Jack Jacklich of Loyola University formed a group with other dentists and performed endodontic therapy. The tedium of hand instrumentation and its ineffectiveness soon resulted in what he called ***"the scourge of digital hyperkeratosis"***. Also the time and patience required for handling of gutta percha points for lateral condensation technique led him on the path of discovery, which led to many innovations in techniques and tools. The result was a huge paradigm shift in the logic and technique.

Then in 1989, American National Standards Institute (ANSI)" granted the approval of "ADA Specification No.28 for endodontic files and reamers. It established the requirements for diameter length, resistance to fracture, stiffness, etc.

Latter on, in 1996, the specification No. 28 was again modified. Initially manufactures of endodontic instruments adhered closely to these specification but nowadays several variations regarding diameter, taper, tip feature, stiffness and metal type used have been noted.

CLASSIFICATION OF ENDODONTIC INSTRUMENTS

ISO - FDI (Federation Dentaire International) grouped root canal instruments according to their method of use:

Group I : *Hand use only* for example K and H-files, reamers, broaches, etc.

Group II : *Latch type Engine driven*: Same design as group I but can be attached to hand piece.

Group III : *Drills or reamers Latch type Engine driven* for example Gates-Glidden, Peeso reamers.

Group IV : *Root Canal points* like gutta-percha, silver point, paper point.

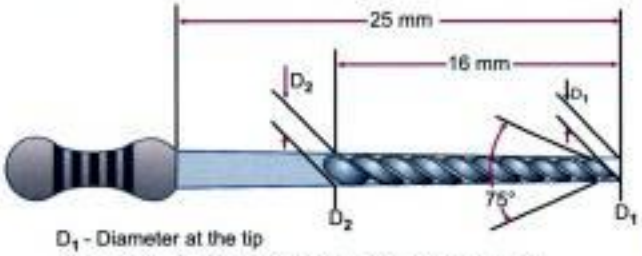
HAND OPERATED INSTRUMENTS

A hand operated instrument reamer or file begins as a round wire which is modified to form a tapered instrument with cutting edges. Several shapes and forms of such instruments are available. These are manufactured by two techniques

a. By machining the instrument directly on the lathe for example H-file and NiTi instruments are machined.
b. By first grinding and then twisting. Here the raw wire is ground into tapered geometric blanks, i.e. square, triangular or rhomboid. These blanks are then twisted counterclockwise to produce cutting edges.

Ingle and LeVine using and electronic microcomparator found variation in the diameter and taper for same size of instrument. They suggested few guidelines for instruments for having uniformity in instrument diameter and taper. The guidelines were:

1. Instruments are numbered from 10-100. Each number represent diameter of instrument in 100th of millimeter at the tip.
2. Working blade begins at tip (D_1) and extends 16 mm up the shaft (D_2). D_2 is 0.32 mm greater than D_1, ensuring that there is constant increase in taper, i.e 0.02 mm per mm of instrument (Fig. 8.1).



D_1 - Diameter at the tip
D_2 - Diameter at end of cutting blade (16 mm from D_1)
Tip angle is 75° ± 15°

Fig. 8.1: Diagrammatic representation of an endodontic instrument in accordance with ANSI specification No. 57

Fig. 8.2: Color coding of endodontic instruments

3. Tip angle of instrument varies as 75 ± 15°.
4. Instruments handles are color coded for their easier recognition (Pink, grey) (Fig. 8.2).

Color code	*Instrument number*
Pink	06
Grey	08
Purple	10
White	15
Yellow	20
Red	25
Blue	30
Green	35
Black	40
White	45
Yellow	50
Red	55
Blue	60
Green	70
Black	80

5. Instruments available in length 21, 25, 28 and 30 mm are used for root canal therapy, and those of 40 mm size are used in preparing root canals for the endodontic implants.

Broaches and Rasps

1. They are one of the oldest intracanal instruments with specifications by ANSI No. 63 and ISO No. 3630-1.

2. Broaches and rasps are manufactured from round wires, smooth surface of which has been notched to form barbs (Fig. 8.3).

Fig. 8.3: Broaches with color coding

3. They are specifically designed to remove the pulp (Fig. 8.4).

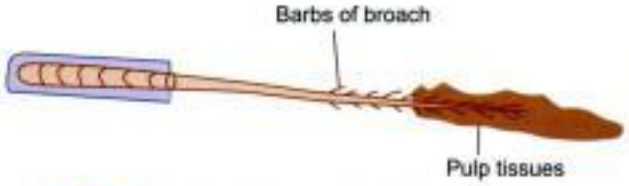


Fig. 8.4: Removal of pulp mass in whole using a broach

4. Both have similar design except in taper and barb size. Barb size is larger in broach than rasp (Fig. 8.5).
5. Broach does not cut the dentin but can effectively be used to remove cotton or paper points which might have lodged in the canal.
6. Broach should not be forced apically into the canal, as its barbs get compressed by the canal wall. While removing this embedded instrument, barbs get embedded into dentin and broach may break on applying pressure.
7. Smooth broach is free of barbs. It is used as pathfinder.

Technique of Pulp Extirpation (Healey, 1984)

Penetrate the barbed broach along the canal wall towards the apex
↓
As it reaches to the apical constriction, move it into the centre of mass of pulp tissue
↓
Rotate the broach several times in a watch winding manner to entrap the pulp which is then withdrawn from the canal.

Reamers

1. Reamers are K-type instruments (manufactured by Kerr company), which are used to ream the canals. They cut by inserting into the canal, twisting clockwise one quarter to half turn and then withdrawing, i.e. penetration, rotation and retraction.

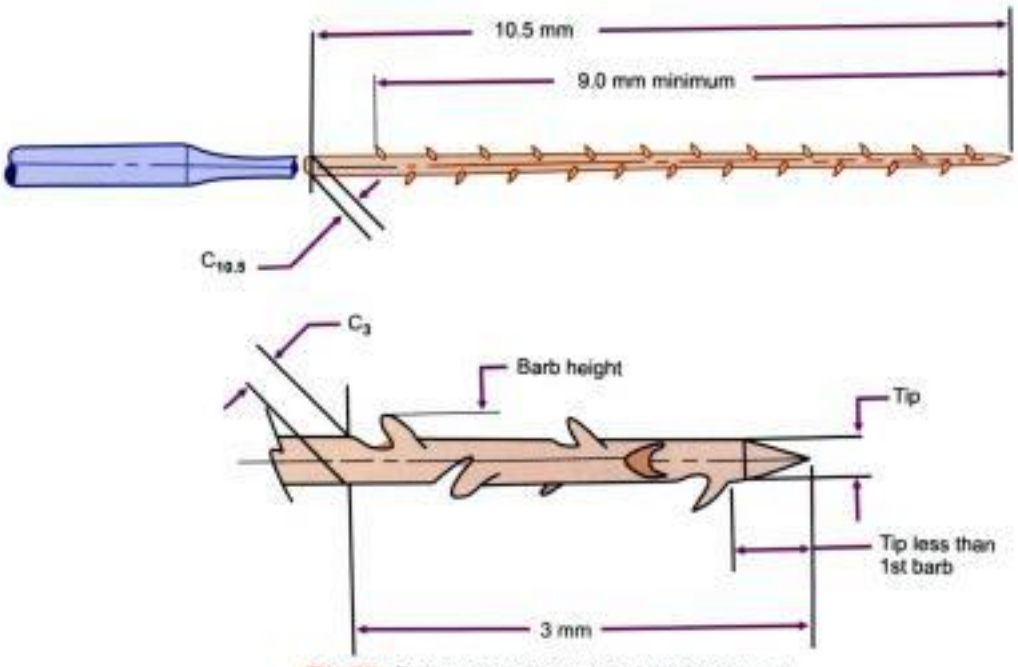


Fig. 8.5: Diagrammatic picture of a broach and rasp

2. Reamers have triangular blank and lesser number of flutes than files. Numbers of flutes in reamer are $^{1}/_{2}$-1 / mm, while in files the flutes are $1^{1}/_{2}$-2 / mm (Fig. 8.6).

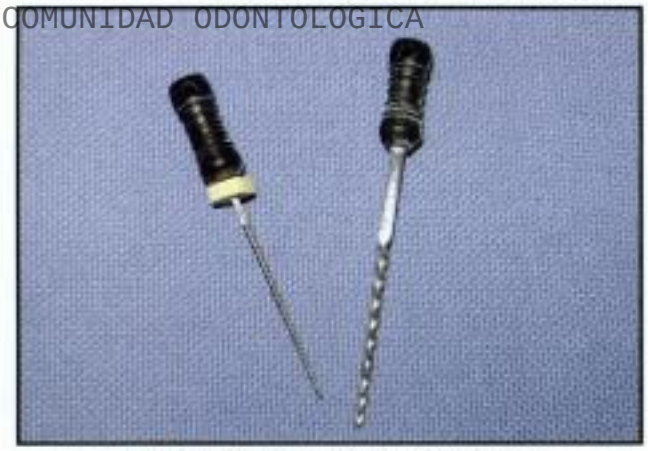


Fig. 8.6: Reamer has lesser number of flutes when compared with files

3. Though reamer has fewer numbers of flutes than file, cutting efficiency is same as that of files because more space between flutes causes better removal of debris (Fig. 8.7).

Fig. 8.7: Diagrammatic representation of reamer

4. Reamer tends to remain self centered in the canal resulting in less chances of canal transportation.

Files

Files are the instruments used during cleaning and shaping of the root canals for machining of the dentin. Since Kerr manufacturing company was first to produce them, the files were also called K-files (Fig. 8.8).

Fig. 8.8: K-files

Files are predominantely used with filing or rasping action in which there is little or no rotation in the root canals. It is placed in root canal and pressure is exerted against the canal wall and instrument is withdrawn while maintaining the pressure (Fig. 8.9).

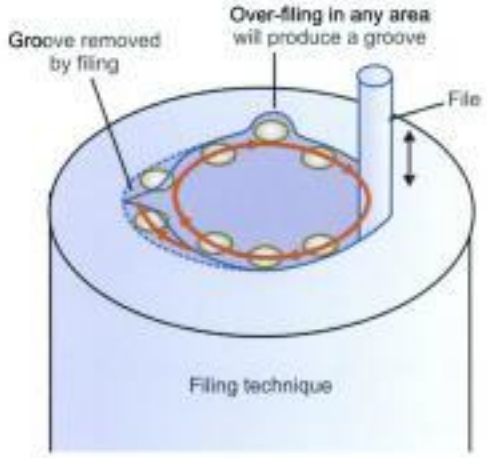


Fig. 8.9: Filing technique

Commonly Used Files:

1. K-file
2. K-Flex file
3. Flexofile
4. Flex-R file
5. Hedstrom file
6. Safety H-file
7. S-file

K-File

- It is triangular or square in cross section, manufactured from stainless steel wire, which is grounded into desired shape.
- Tighter twisting of the file spirals increases the number of flutes in file (more than reamer).
- Triangular cross sectioned files show superior cutting and increased flexibility than the file or reamers with square blank (Fig. 8.10).

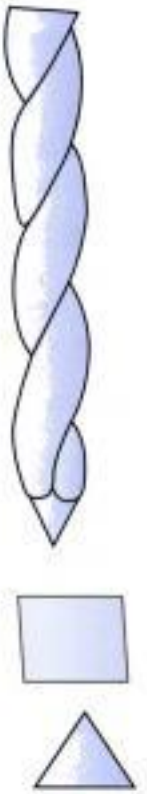


Fig. 8.10: Diagrammatic representation K-file

K-Flex Files

- They were introduced by Kerr manufacturing company in 1982. It was realized that square blank of file results in total decrease in the instrument flexibility. To maintain shape and flexibility of these files, K-flex files were introduced (Fig. 8.11).
- K-flex files are rhombus in cross section having two acute angles causing increased sharpness and two obtuse angles which make more space for debris removal (Fig. 8.12). Also the decrease in contact of instrument with canal walls provides more space for irrigation.

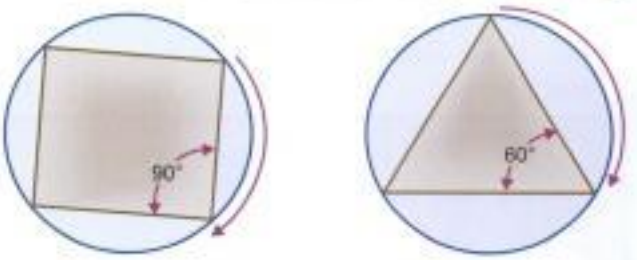


Fig. 8.11: Cross section of instrument

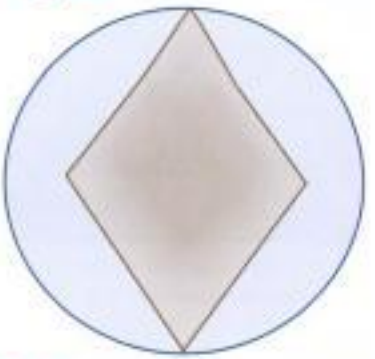


Fig. 8.12: Rhombus cross section of K-flex File

Flexo File

- These are similar to the K-Flex files *except* that they have triangular cross section. This feature provides them more flexibility and thus ability to resist fracture.

Flex-R File

- Flex-R files are made by removing the sharp cutting edges from the tip of instrument (Fig. 8.13A). This design reduces the ledge formation, canal trans-portation and other procedural accidents when used with balanced force technique.
- The noncutting tip enables the instrument to traverse along the canal rather than gouge into it (Fig. 8.13 B).

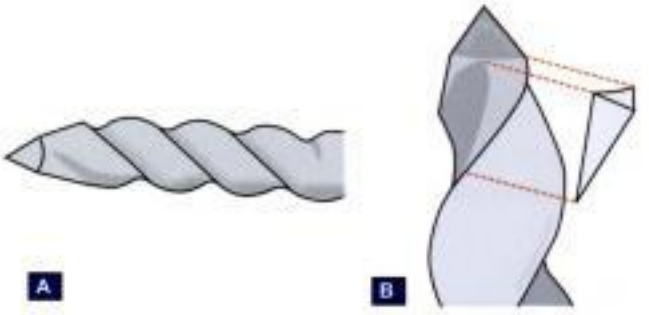


Fig. 8.13: Flex R-file

- Another feature of flex-R file is presence of triangular cross section which provides it flexibility to be used in curved canals.

Hedstrom Files (H-files)

- Hedstrom files have flutes which resemble successively triangles set one on another (Fig. 8.14).
- They are made by cutting the spiral grooves into round, tapered steel wire in the same manner as wood screws are made. This results information of a sharp edge which cuts on removing strokes only (Fig. 8.15).
- Hedstrom files cut only when instrument is withdrawn because its edges face the handle of the instrument.
- When used in torquing motion, their edges can engage in the dentin of root canal wall and causing H - files to fracture.
- Rake angle and distance between the flutes are two main features which determine working of the file.
- H-Files have positive rake angle, i.e. its cutting edge is turned in the same direction in which the force is applied which makes it dig into the dentin making it more aggressive in cutting.
- Hedstrom files should be used to machine straight canals because they are strong and aggressive cutters. Since they lack the flexibility and are fragile in nature, the H-files tend to fracture when used in torquing action.

Fig. 8.14: Diagrammatic view of Hedstrom file

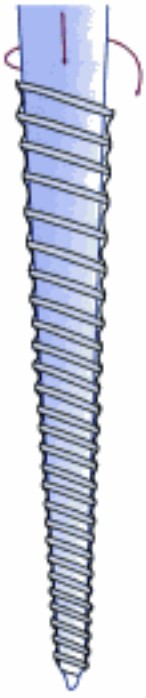


Fig. 8.15: Screw entering a piece of wood

Safety Hedstrom File

- This file has noncutting safety side along the length of the blade which reduces the chances of perforations. The non cutting side is directed to the side of canal where cutting is not required. The non cutting side of safety file prevents lodging of the canals (Fig. 8.16).

Fig. 8.16: Safety Hedstrom file

S - File

- S-File is been produced by grinding, which makes it stiffer than Hedstrom file. These files are designed with two spirals for cutting blades, forming double helix design (Fig. 8.17).

Fig. 8.17: Cross section and longitudinal shape of S-file

- This file shows 'S' shape in the cross section. S-File has good cutting efficiency in either filling or reaming action, thus this file can also be classified as a hybrid design.

ENGINE DRIVEN INSTRUMENS

Gates Glidden Burs

1. Traditional engine driven instruments include Gates-Glidden drills which have flame shaped cutting point mounted on long thin shaft attached to a latch type shank (Fig. 8.18).

Fig. 8.18: Gates glidden drills

2. Gates-Gliddens are available in a set from 1 to 6 with the diameters from 0.5 to 1.5 mm.
3. Due to their design Gates-Glidden drills are side cutting instruments with safety tips. They should be used at the speed of 750-1500 rpm.
4. If its cutting tip jams against the canal wall, fracture should occur at the junction of shank and the shaft but not at the tip of the instrument. This makes the easy removal of fractured drill from the canal.
5. They can be used both in crown down as well as step back fashion (Fig. 8.19).

Fig. 8.19: Use of gates gliddens for canal preparation

Uses of Gates-Glidden Drills

1. For coronal flaring during root canal preparation.
2. During retreatment cases or post space preparation for removal of gutta percha.
3. During instrument removal, if used incorrectly for example using at high rpm, incorrect angle of insertion, forceful drilling, the use of Gates-Glidden can result in procedural accidents like perforations, instrument separation, etc.

But nowadays in many ways nickel titanium instruments have replaced the Gates-Glidden. The rotary nickel titanium instruments have emerged as a great breakthrough in endodontics, these files can be rotated continuously in a handpiece through 360°. These instruments have many advantages over the traditional instruments in that they allow increased debris removal because of continuous rotation, smoother and faster canal preparation with less clinician fatigue.

PEESO REAMERS

They are rotary instruments used mainly for post space preparations. Disadvantages of using peeso reamers are:

1. They don't follow the canal curvature and may cause perforation by cutting laterally.
2. They are stiff instruments
3. Unable to be used repeatedly to avoid iatrogenic errors.

NICKEL TITANIUM (NITI) ENDODONTIC INSTRUMENTS

When using the stainless steel files, occurrence of procedural errors cannot be avoided specially in case of curved canals. Deviation from the original shape, ledge formation, zipping, stripping and perforations are the common problems which are seen in such cases. But the superelasticity of NiTi alloy allows these instruments to flex more than the stainless steel instruments before exceeding their elastic limit, thereby allowing canal preparation with minimal procedural errors.

NiTi was developed by Buchler 40 years ago. NiTi is also known as the NiTinol (NiTi Navol Ordinance Laboratory in US). In endodontics commonly used NiTi alloys are called 55 NiTi nol (55% weight Ni and 45% Ti) and 60 NiTi nol (60% weight of Ni, 40% Ti).

First use of NiTi in endodontics was reported in 1988, by Walia et al when a 15 No. NiTi file was made from orthodontic wire and it showed superior flexibility and resistance to tortional fracture. This suggested the use of NiTi files in curved canals.

Properties of NiTi Alloys:
1. Shape memory
2. Superelasticity
3. Low modulus of elasticity
4. Good resiliency
5. Corrosion resistance
6. Softer than stainless steel

Superelasticity and shape memory of NiTi alloys is because of phase transformation in their crystal structures when cooled from the stronger, high temperature form (Austenite) to the weaker low temperature form (Martensite). This phase transformation is mainly responsible for the above mentioned qualities of NiTi alloys.

Manufacturing of NiTi Files

Because of the presence of superelasticity and shape memory, the NiTi files cannot be manufactured by twisting as is done with K-Files. Infact the NiTi files have to be grounded for their manufacturing.

Earlier there used to be NiTi hand files but latter automated use of NiTi files was developed to increase the efficiency of clinical treatment.

Instrument Deformation and Breakage

An unfortunate thing about NiTi instruments is that their breakage can occur without any visible sign of unwinding or permanent deformation. In other words visual examination is not a reliable method for evaluation of any NiTi instrument.

Basically there are two modes of rotary instrument separation viz. Torsional fracture and flexural fracture.

Torsional Fracture

Occurs when torque limit is exceeded. The term torque is used for the forces which act in the rotational manner. The amount of torque is related to mass of the instrument, canal radius and apical force when worked in the canal. As the instrument moves apically, the torque increases because of increased contact area between the file and the canal wall.

Theoretically an instrument used with high torque is very active but chances of deformation and separation increase with high torque. Thus as the file advances further into the canal, the pressure should be loosened to prevent the torque.

A variety of speeds for different rotary instrumentation have been recommended by various companies. Depending on the manufacturer and condition of the handpiece, each handpiece has different degree of effectiveness depending upon the torque values. Thus one must take care while choosing appropriate handpiece, according to required speed and torque.

Role of Handpiece

Handpiece is a device for holding instruments, transmitting power to them and position them intraorally. Both speed and torque in a handpiece can be modified by incorporation of the gear system. Various types of gearing systems can be incorporated in the handpieces but gearing is limited by the need to maintain the drive concentrically through handpiece and the head (Fig. 8.20).

Torque control motors allow the setting of torque produced by the motor. In low torque control motors, torque values set on the motor is less than the value of torque at deformation and separation of the instruments. Where as in high torque motors, the torque value is higher as compared to torque at deformation and separation of the rotary instruments. During root canal preparation, all the

Fig. 8.20: Torque control handpiece

instruments are subjected to different levels of torque. If torque level is equal or greater than torque at deformation the instrument will deform or separate. Thus, with low torque control motors, motor will stop rotating and may even reverse the direction of rotation when instrument is subjected to torque level equal to torque value set at the motor. Thus instrument failure can be avoided. Where as in high torque motors, instruments may deform or separate before the torque value of motor is achieved. Hence we can say that torque control is an important factor to reduce NiTi fracture.

Flexural Fracture

When an instrument rotates in a curve, it gets compressed on the inner side of a curve, where as it gets stretched on the outer side of the curve. With every 180° of rotation, instrument flexes and stretches again and again resulting in the cyclic fatigue and subsequent fracture of the instrument.

In large size files because of more metal mass, more of tensile and compressive forces occurs, which may result in early fatigue of the instrument.

The elastic and fracture limit of NiTi rotary instruments are dependent on design, size and taper of the instrument. Thus to prevent instrument deformation and fracture, right torque value for each instrument should be calculated. Also the motors should have fine control of torque values.

Conventional endodontic motors do not allow precise setting of torque values. The latest development with regard to torque control is incorporation of the gear system with in the handpieces which regulate torque depending on size of the rotary system.

Rotary Nickel Titanium Files (RNT) are the least resistant files to breakage. It has been reported in the literature that NiTi begins to microfracture as soon as it is used in the root even with light touch. In the rotary handpiece, the combination of compressive and tensile stress cause files to break even sooner. Unfortunately the NiTi instruments tend to fracture without visible warning.

Two things can be done to reduce the risk of NiTi fracture:

1. Examine the file every time before placing it into the canal.
2. Bend the file to at least 80 degree angle, every time before placing into the canal, to see if it will fracture (Fig. 8.21).

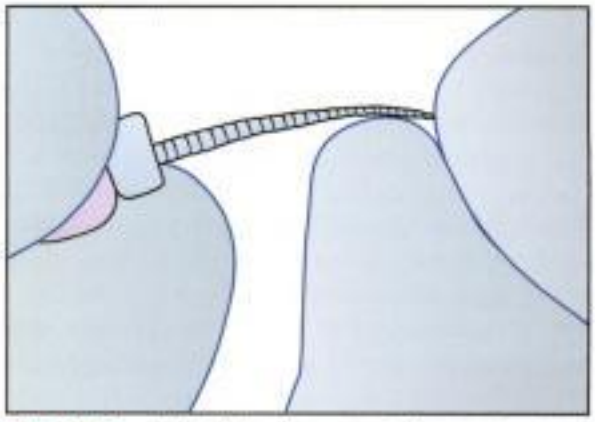


Fig. 8.21: To check whether instrument will fracture, bend the file to 80° angle before placing into the canal

Prevention of Breakage of Instruments when using Nickel - Titanium Rotary Instruments:

1. Use only torque controlled electric handpiece for these instruments.
2. Proper glide path must be established before using rotary files, i.e. getting the canal to at least size 15 before using them (Fig. 8.22).

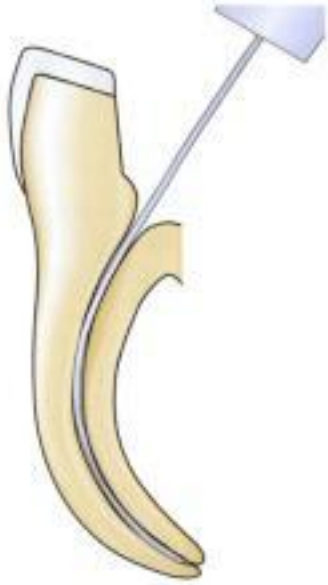


Fig. 8.22: Establish glide path before using rotary instrument

3. Use crown down method for canal preparation. By this apical curves can be negotiated safely (Fig. 8.23).

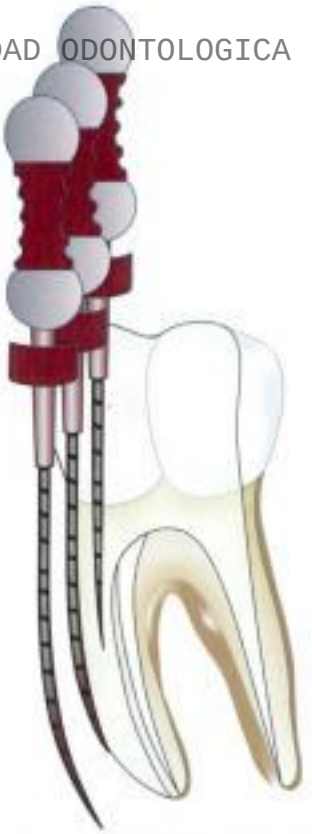


Fig. 8.23: Use of crown down technique for canal preparation

4. Frequent cleaning of flutes should be done as it can lessen the chances that debris will enter the micro-fractures and resulting in propagation of original fracture and finally the separation (Fig. 8.24).

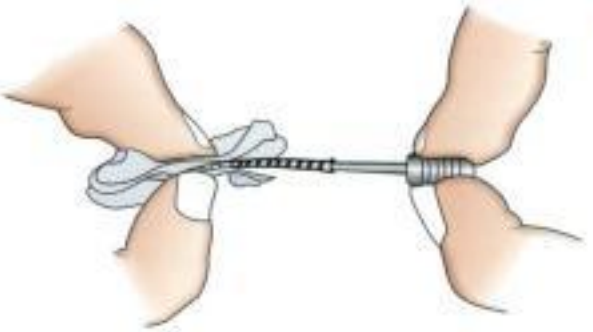


Fig. 8.24: Frequent cleaning of flutes should be done to remove debris

5. Do not force the file apically against resistance. Motion of file going into canal should be smooth, deliberate with 1-2 mm deep increments relative to the previous instrument.
6. Remove the maximum possible pulp tissue with broach before using rotary files.
7. Canals should be well lubricated and irrigated. This reduces the friction between instrument and the dentinal walls.
8. Dentin mud collected in the canal increases the risk of fracture, it should be cleared off by frequent irrigation.
9. Discard a file if it is bent, stretched or has a shiny spot.
10. Do not use rotary nickel titanium files to true working length specially in teeth with S - shaped canals, canals with multiple and sharp curves and if there is difficult access of orifice because it can place stresses on the instrument which will cross the breaking torque value. In such cases apical portion of canal should be prepared by hand files.
11. A file should be considered disposable when:
    a. It has been used in curved canals.
    b. Despite of existence of excellent glide path, if it doesn't cut dentin properly.

VARIOUS ROTARY NICKEL TITANIUM SYSTEM (RNT)

Since last many years innumerable amount of RNT file system have been made available. Though no system is perfect, but if used in proper way, they can result in desired canal shape. Various rotary nickel titanium systems available in the market are Profile, Greater Taper files, ProTaper, Quantec, Light Speed System, K_3 system and HERo 642, RaCe and Real World Endo Sequence file system.

PROFILE SYSTEM

- Profile instruments made by Tulsa Dental were one of the first NiTi instruments available commercially. This system was introduced by Dr Johnson in 1944 (Fig. 8.25).
- Earlier Profile system was sold as series 29 instruments. In series 29, at the constant rate of 29 percent, there has advantage of smooth transition among the smaller files but in larger files, the greater gap may create difficulties during cleaning and shaping of the root canals. After this profile series were introduced with greater tapers of 19 mm lengths and ISO sized tips.

Fig. 8.25: Profiles

- Recommended rotational speed for profiles is 150-300 RPM.
- Cross section of profiles show three equally shaped U-shaped grooves with radial lands (Fig. 8.26).
- Central parallel core present in profiles increase their flexibility.
- They have negative rake angle (–20°) which makes them to cut dentin in planning motion. Profile instruments tend to pull debris out of the canal because of presence of 20° helical angle.

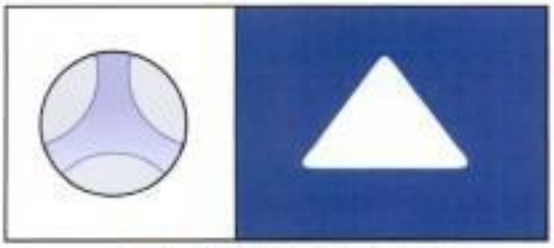


Fig. 8.26: Cross section of profiles

GREATER TAPER FILES (GT FILES)

1. The GT rotary instruments possess a U-shaped file design with ISO tip sizes of 20, 30 and 40 and tapers of 0.04, 0.06, 0.08, .010 and 0.12.
2. Accessory GT files for use as orifice openers of 0.12 taper in ISO sizes of 35, 50, 70 and 90 are also available.
3. The maximum diameter of these instruments is 1.50 mm.
4. Recommended rotational speed for GT files is 350 rpm.
5. Negative rake angle of these files makes them to scrape the dentin rather than cutting it.

PROTAPER FILES

1. The ProTaper file has a triangular cross section and is variably tapered across its cutting length (Fig. 8.27). A unique feature of the ProTaper files is each instrument has changing percentage tapers over the length of cutting blades. This progressively tapered design improves flexibility, cutting efficiency and the safety of these files.

Fig. 8.27: ProTaper file

2. Convex triangular cross section of these instrument decrease the friction between the blade of file and the canal wall while increasing its cutting efficiency (Fig. 8.28).

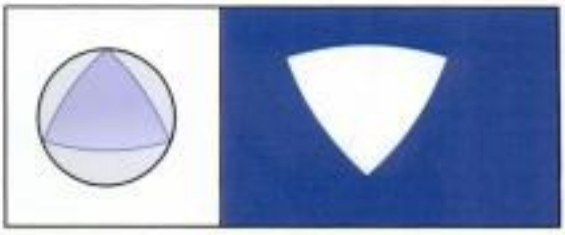


Fig. 8.28: Cross section of ProTaper files

3. ProTaper file has modified guiding tip which allows one to follow canal better and variable tip diameters allow file to have specific cutting action in defines area of canal without stressing instrument in other sections.
4. ProTaper file has a changing helical angle and pitch over their cutting blades which reduces the instrument from screwing into the canal and allows better removal of debris.
5. ProTaper file acts in active motion, this further increases its efficiency and reduces torsional strain.
6. Length of file handle is reduced from 15 to 12.5 mm. which allows better access in posterior areas.
7. The ProTaper system consists of just three shaping and three finishing files.

Shaping Files

These are termed as S_x, S_1 and S_2. (Fig. 8.29)

Fig. 8.29: Shaping files; S_x, S_1, S_2

S_x

- No identification ring on its gold colored handle
- Shorter length of 19 mm
- Do diameter is 0.19 mm
- D_{14} diameter is 1.20 mm
- There is increase in taper up to D_9 and then taper drops off up to D_{14} which increases its flexibility
- Use is similar to Gates-Glidden drills or orifice shapers.

S_1

- Has purple identification ring on its handle
- Do diameter is 0.17 mm and D_{14} is 1.20 mm
- Used to prepare coronal part of the root.

S_2

- Has white identification ring on its handle
- Do diameter is 0.20 mm and D_{14} is 1.20 mm
- Used to prepare middle third of the canal.

Finishing Files

Three finishing files F_1, F_2, F_3 are used to prepare and finish apical part of the root canal (Fig. 8.30).

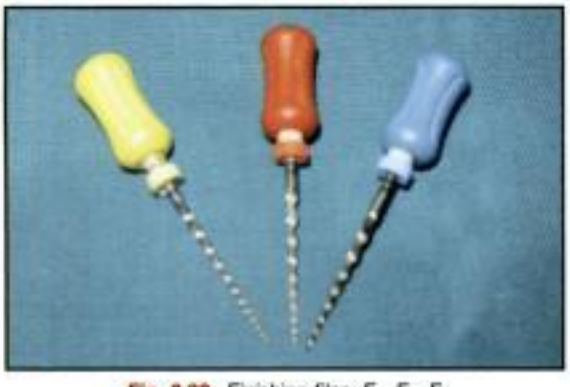


Fig. 8.30: Finishing files; F_1, F_2, F_3

F_1

- Yellow identification ring
- Do diameter and apical taper is 20 and 0.07.

F_2

- Red identification ring on handle
- Do diameter and taper is 25 and 0.08.

F_3

- Blue colored ring on handle
- Do diameter and taper is 30 and 0.09.

Each instrument has decreasing percentage of taper from D_4 to D_{14}. This improves flexibility and decrease the potential for taper lock.

QUANTEC FILE SYSTEM

1. Quantec file series are available in both cutting and noncutting tips with standard size of 25 No. in 0.12, 0.10, 0.08, 0.06, 0.05, 0.04, 0.03 and 0.02 tapers. 0.02 tapered Quantec file are also available in size 15-60 No.
2. Quantec system has a positive blade angle with two wide radial lands and relief behind the lands (Fig. 8.31).

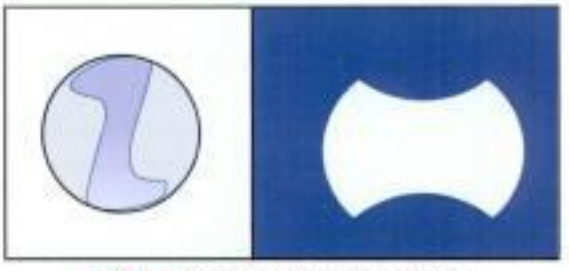


Fig. 8.31: Cross section of quantec file

3. This unique design minimizes its contact with the canal, thereby reducing the torque, also this design increases the strength of the instrument.
4. Quantec system utilizes the "graduated taper technique" to prepare a canal. It is thought that using a series of files of single taper result in the decreases efficiency as the larger instruments are used. This happens because more of file comes in contact with the dentinal wall which makes it more difficult to remove dentin. Thereby retarding the proper cleaning and shaping of the canal. But in graduated taper technique, restricted contact of area increases the efficiency of the instrument because now forces are concentrated on smaller area.

LIGHT SPEED SYSTEM

1. Light speed system is engine driven endodontic instrument manufactured from nickel-titanium. These are so named because a "light" touch is needed as "speed" of instrumentation is increased.
2. Light speed instrument are slender with thin parallel shaft and have non cutting tip with gates glidden in configuration.
3. They are available in 21, 25, 31 and 50 mm length and ISO No. 20-140.

   Half sizes of light speed instrument are also available viz. 22.5, 27.5, 32.5. The half sizes are also color coded as full ones with only difference in that half size instruments have white or black rings on their handles.
4. Cutting heads of light speed system has three different geometric shapes -
   i. Size 20-30-short, non cutting tips at 75° cutting angle.
   ii. Size 32.5 - longer non cutting tip at 33° cutting angle.
   iii. Size 35-140-longer non cutting tip with 21° cutting angle.
5. Cutting heads basically have three radial lands with spiral shaped grooves in between.

K_3 ROTARY FILE SYSTEM

Dr John Mc Spadden in 2002 in North America introduced K_3 system.

1. K_3 files are available in taper of 0.02, 0.04 or 0.06 with ISO tip sizes. An Axxess handle design shortens the file by 5 mm without affecting its working length.
2. They are flexible because of presence of variable core diameter.
3. Cutting head of K_3 system show three radial lands with relief behind two radial lands. Asymmetrically placed flutes make the K_3 system with superior canal tracking ability, add peripheral strength to K_3 system, and prevent screwing into the canal (Fig. 8.32).

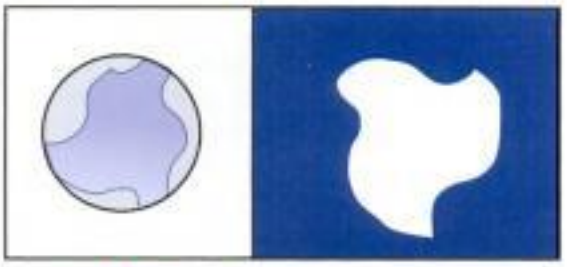


Fig. 8.32: Cross section of K_3 file system

4. K_3 files have positive rake angle providing them an effective cutting surface.
5. They are color coded to differentiate various tip sizes and tapers.
6. Body shapers available in taper 0.08, 0.10, and 0.12 all with tip size 25, are used to prepare the coronal third of the canal.

HERO 642

Hero	-	High elasticity in rotation
642	-	0.06., 0.04 and 0.02 tapers

HERO 642 (High Elasticity in Rotation, 0.06, 0.04 and 0.02 tapers) is used in "Crown down" technique, between 300 and 600 rotations per minute (rpm) in a standard slow speed contra angle air driven or electric motors.

Features

1. It has trihelical Hedstorm design with sharp flutes (Fig. 8.33).
2. Due to progressively increasing distance between the flutes-reduced risk for binding of the instrument in root canal.
3. Larger central core - provides extra strength.
4. Used at speed - 300 - 600 rpm.
5. Available in size - 0.20 - 0.45.

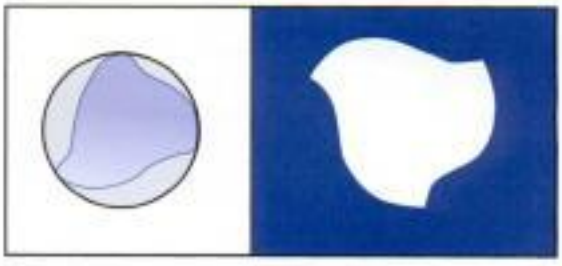


Fig. 8.33: Cross section of HERO 642

RACE FILES (Reamers with Alternating Cutting Edges)

- Race has safety tip and triangular cross section. This file has two cutting edges, first alternates with a second which has been placed at different angle (Fig. 8.34)
- This file has an alternating spiral and has a cutting shank giving variable pitch and helical angles
- Variable helical angle and pitch prevents the file from screwing into the canal during its working
- Electrochemical treatment of these files provides better resistance to corrosion and metal fatigue.

Fig. 8.34: Cross-section of RACE files

Advantages of RACE Files

1. Non cutting safety tip helps in:
   a. Perfect control of the instrument.
   b. Steers clear of lateral canals.
2. Alternating cutting edges
   a. Reduced working time.
   b. Lowest operation torque.
   c. Non - threading or blocking effect
3. Sharp cutting edges
   a. Improved efficiency.
   b. Better debris evacuation.
4. Electrochemical treatment
   a. Better resistance to torsion and metal fatigue.

REAL WORLD ENDO SEQUENCE FILE

1. Real World Endo sequence file system is recently introduced in NiTi rotary system. During manufacturing, these files are subjected to electropolishing for metal treatment. Electropolishing reduces the tendency of NiTi files for crack propagation by removing the surface imperfections.
2. Real World Endo sequence file system has a blank design in such a way that alternating contact points (ACPs) exist along the shank of the instrument. Because of presence of ACPs, there is no need of radial lands, which further make the instrument shaper and thus more effective.
3. These files are available in 0.04 and 0.06 taper having the precision tip. Precision tip is defined as a noncutting tip which becomes active at D_1. This results in both safety as well as efficiency.
4. Sequence files have variable pitch and helical angle which further increase its efficiency by moving the debris out of canal and thus decreasing the torque caused by debris accumulation.
5. They are worked at the speed of 450-600 RPM. Sequence files come in package of four files each, i.e. Expeditor file, 0.06 taper files in extra small, small, medium and large sizes.

SONICS AND ULTRASONICS IN ENDODONTICS

The concept of using ultrasonics in endodontics was suggested by Richman in 1957. The pioneer research on endosonics was done by Cunningham and Martin in early 1980. But these machines showed lots of problems like tip design of instrument was not with sodium hypochlorite as an irrigant, machines used to get blocked due to crystallization of NaOCl in irrigation lines. Since then a lot of changes have been made in endosonics for example tip design has been specifically turned for endodontic use, etc.

Ultrasonic Endodontics

Ultrasonic Endodontics is based on a system on which sound as an energy source (at 20 to 42 kHz), activates an endodontic file resulting in three dimensional activation of the file in the surrounding medium. The ultrasonic systems involve a power source to which an endodontic file is attached with a holder and an adapter. Ultrasonic handpiece uses K - file as a canal instrument (Fig. 8.35). Before a size 15 can fully function, the canal must be enlarged with hand instruments to a size 20. The irrigants are emitted from cords on the power source and travel down the file into the canal to be energized by the vibrations (Fig. 8.36).

The only similarity between the ultrasonic and sonic instruments is in imparting vibrational type of movement for the root canal instrument, which they activate. The rest of the features such as the source of power, frequency of vibration, type of handpiece and root canal instruments used are all different. Sonic instruments use compressed air line at a pressure of 0.4 MPa, which is already available in the

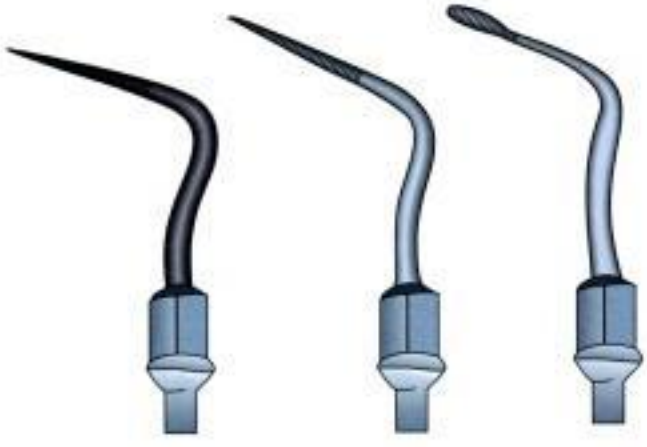


Fig. 8.35: Endosonic files

Fig. 8.36: Diagram showing energized irrigating solution using ultrasonics

dental unit setup, as its source of power. There are two options for irrigating the root canal while using sonic handpieces. Either the water line of the dental units can be attached to the sonic handpiece, or the water can be cut off and the dental assistant can introduce sodium hypochlorite from a syringe. Sonic handpieces impart vibrations usually in the frequency of 1.5-3 kHz with provision for adjusting the frequency, in the form of a ring on the handpiece.

Files that are used with sonic handpieces are the Rispi sonic, Shaper sonic and the Helio sonic. All these instruments have safe ended non cutting tip 1.5-2.0 mm in length. The ISO sizes of these instruments range from 15 to 40.

Cavitation and Acoustic Streaming

Cavitation is defined as the growth and subsequent violent collapse of a small gas filled pre existing inhomogeneity in the bulk fluid .This motion results in development of shock wave, increased temperature and pressure and free radical formation in the fluid (Fig. 8.37).Cavitation has been shown useful in removal of tooth deposits in scaling procedure but during its use in root canal regarding cavitation phenomenon, following points are to be considered:-

- Threshold power setting at which this phenomenon occurs is beyond the range that is normally used for endodontic purpose.
- Cavitation depends on free displacement amplitude of the file. During root canal therapy, when file movement is restricted, this phenomenon is impossible to achieve.

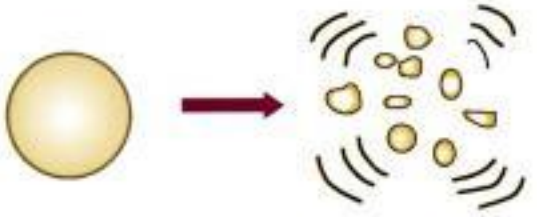


Fig. 8.37: Cavitation produced by ultrasonics

Acoustic Streaming

Acoustic streaming is defined as the generation of time independent, steady unidirectional circulation of fluid in the vicinity of a small vibrating object (Fig. 8.38). This flow of liquid has a small velocity, of the order of a few centimeters per second, but because of the small dimensions involved the rate of change of velocity with from the is high. This results in the production of large hydrodynamic shear stresses around the file ,which are more than capable of disrupting most biological material. In endodontic file the greatest shear stresses around the points of maximum displacement, such as the tip of file and the antinodes along its length in the canal., therefore it may be of benefit to preheat the irrigant in the reservoir within the endosonic unit.

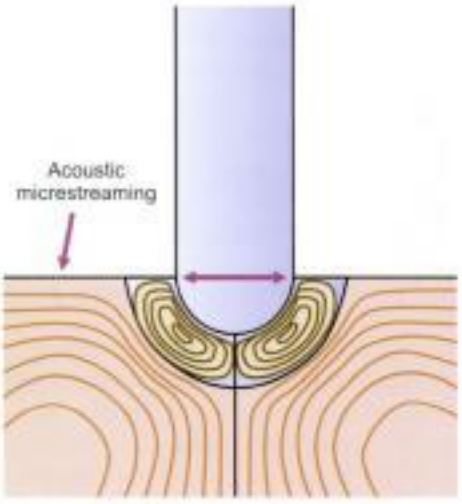


Fig. 8.38: Acoustic streaming

Uses of Endosonics

Access Enhancement

Performing access preparation with burs can lead to gauging of the pulpal floor and sides of the chamber. But use of round or tapered ultrasonically activated diamond coated tips has shown to produce smooth shapes of access cavity.

Orifice Location

Ultrasonic instruments are very useful in removal of the chamber calcifications as well as troughing for canals in isthmus and locating the canal orifices

The troughing concept involves using an endodontic tip in back and forth brushing motion along a groove in the pulpal floor. By troughing there is better removal of tissue

in the isthmus between the canals, and conservation of the tooth structure.

Irrigation

Studies have shown the use of endosonics have resulted in cleaner canals. There is a synergetic action of the physical action of the tip along with the chemical action of the sodium irrigant.

Acoustic streaming forces with in the irrigant together with the oscillation of the instrument are useful for dislodging out the debris out of the canal.

Sealer Placement

One of the method of sealer placement is by using an ultrasonic file which runs without fluid coolant. A recent study found ultrasonic endodontic sealer placement significantly superior to hand reamer placement. A common problem encountered with this technique is the "whipping up" of the cement in the canal and causing it to set prematurely. This problem can be solved to an extent by replacing the ZnO-Eugenol sealer with a resin sealer like AH-26.

Gutta Percha Obturation

Moreno first suggested the technique of plasticizing gutta-percha in the canal with an ultrasonic instrument. His technique advocated the placement of gutta-percha points to virtually fill the canal. The attached endodontic instrument is then inserted into the mass and the ultrasonic instrument is then activated. The gutta-percha gets plasticized by the friction being generated. Final vertical compaction is done with hand or finger pluggers.

MTA Placement

MTA is a wonderful material with many uses. But its placement is difficult because of its sandy consistency. Low powered ultrasonics can be used to vibrate the material into position with no voids.

Endodontic Retreatment

i. ***Intraradicular post removal***
Teeth that present with intraradicular posts and periapical infections can be difficult to retreat from a coronal approach. Many techniques have been devised to aid in removal of posts from root canal spaces, of which ultrasonics are highly efficient as post removal can be achieved with minimal loss of tooth structure and decreased risk of other root damage. Ultrasonic instrumentation for post removal typically involves removing coronal cement and buildup material from around the post, then activating the tip of the ultrasonic instrument against the metal post. The ultrasound energy transfers to the post and breaks down the surrounding cement until the post loosens and is easily removed.

ii. ***Gutta-percha removal***
Studies have shown that Ultrasonic instrumentation alone or with a solvent is as effective as hand instrumentation in removing gutta percha from root canals. Although ultrasonic instrumentation requires less time than hand instrumentation to attain the working length, it uses significantly more solvent (chloroform or halothane) because the solvent not only acts as an irrigant but also gets volatilized.

iii. ***Silver point removal***
A conservative approach for removing defective silver points has been suggested by Krell. In this technique, a fine Hedstrom file is placed down into the canal alongside the silver point. The file is then enervated by the ultrasonic tip and slowly withdrawn. Ultrasonics with copious water irrigation along with gentle up and down strokes is quite effective in not only removing silver points and broken files, but spreader and burs tips as well.

It is apparent that no automated device will answer all needs in cleaning and shaping. Hand instrumentation is essential to prepare and cleanse the apical canal, no matter which device, sonic and ultrasonic is used later. Both ultrasonics and sonics will be a useful adjunct in endodontics if their limitations and characteristics are fully understood.

INSTRUMENTS USED FOR FILLING ROOT CANALS

Spreaders and pluggers are the instruments used to compact the gutta-percha into root canal during obturation (Figs 8.39 and 8.40). The use of instrument depends on the technique employed for obturation.

Earlier there used to occur the discrepancy in spreader size and shape with the gutta-percha points but in 1990, ISO/ADA endodontic standardization committee recommended the size of 15-45 for spreaders and 15-140 for pluggers. Hand spreaders are made from stainless steel

Fig. 8.39: Spreaders

and are designed to facilitate the placement of accessory gutta-percha points around the master cone during lateral compaction technique (Fig. 8.41). These spreaders do not have standardized size and shape.

Finger spreaders are shorter in length which allows them to afford a great degree of tactile sense and allow them to rotate freely around their axis. They are standardized and color coded to match the size of gutta-percha points (Fig. 8.42).

They can be manufactured from stainless steel or now a day from nickel titanium. Stainless steel spreaders may pose difficulty in penetration in curved canals, may cause wedging and root fracture if forced during compaction. Stainless steel spreaders also produce great stresses while compaction.

Fig. 8.40: Plugger

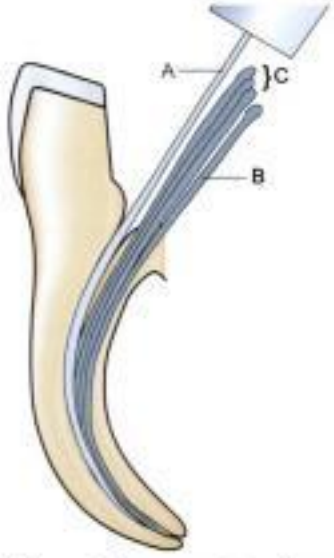


Fig. 8.41: Use of plugger during compaction of warm Gutta-percha

Fig. 8.42: Spreader with pointed end

NiTi spreaders are recently introduced spreaders which can penetrate the curved canals and produce less stresses during compaction. But they may bend under pressure during compaction. So, we can say that combination of both types of spreaders, i.e. stainless steel and NiTi is recommended for compaction of gutta-percha, NiTi spreaders in apical area and stainless steel in coronal part of the root canal.

Pluggers consist of diameter larger than spreader and have blunt end (Fig. 8.43). They are used to compact the warm gutta-percha vertically and laterally into the root canal. They may also be used to carry small segments of gutta-percha into the canal during sectional filling technique (Fig. 8.44).

Fig. 8.43: Plugger with flat end

Fig. 8.44: Use of plugger during compaction of warm gutta-percha

Calcium hydroxide or MTA like materials may also be packed into the canals using pluggers.

Care should be taken with spreaders and pluggers while compacting the gutta percha in canals. They should be cleaned prior to their insertion in to the canal; otherwise the set sealer from previous insertion may roughen their surface and may pull the cone outside the canal rather than packing it. Also one should discard the instrument when it has become bent or screwed to avoid instrument separation while compaction.

SURGICAL OPERATING MICROSCOPE (SOM)

Use of microscope in endodontics was first introduced in 1990's and since its introduction in endodontics there has been made great change in the way endodontics is done and has also affected the success rate of endodontic therapy.

Most of surgical microscope come with three to five steps of magnification ranging from 3x to 27x. The light source is usually 100 to 150 watt halogen bulb connected to the microscope via a high efficiency fiber optic cable.

To use SOM in endodontics, one should have specially designed micro instruments for example files specially designed for this are called microopeners, similarly other instruments like micromirrors are used with SOM.

Before using SOM, rubber dam placement is necessary because direct viewing through the canal with microscope is difficult, so a mirror is needed to reflect the canal. But without the use of rubber dam, mirror will fog soon.

To maximize the access and quality of view there should be 45° angle between the microscope and the mirror.

USES OF ENDOMICROSCOPE

SOM is useful in all aspects of endodontic therapy from diagnosis to evaluation of final obturation.

1. *Diagnosis*
   a. SOM allows calcified, irregularity positioned or accessory canals to be found with ease and thereby increasing the success rate and decreasing stress.
   b. SOM helps to detect microfractures which are not visible with naked eye.
   c. Missing canals (most common MB_2 of maxillary molar) can be successfully located by use of endo-microscope.
2. Removal of foreign materials like cast post and cement filing material, can be easily accomplished by its use.
3. The endodontic retreatment involving the removal of screw posts, separated instruments, silver points can be guided by use of endomicroscope.
4. Perforation repair can be precisely done by use of SOM by accurate placement of the repair material and by précised manipulation of the tissue.
5. Evaluation of the canal preparation can be accurately done by use of endomicroscope.
6. SOM is also useful in evaluation of the final obturation of root canals. With the help of SOM one can assess the irregularly shaped and poorly obturated canals, and quality of apical seal.
7. Intracanal isthmus communication can be well assessed by use of endomicroscope.

MINERAL TRIOXIDE AGGREGATE (MTA)

Mineral trioxide aggregate (MTA) was developed by Dr Torabinejad at Loma Linda University in 1993.

Composition

It is available in two colors - White and Grey color

Grey color - It contains tricalcium silicate,
Dicalcium silicate,
Tricalcium aluminate
Bismuth oxide
Calcium sulfate
Tetracalcium aluminoferrite

White colour-It has same composition as that of grey color MTA except the lack of tetracalcium alumino-ferrite. Consequently, it is white in color.

Properties

1. P_H of MTA is 12.5 (When set) so, it has biological and histological properties similar to calcium hydroxide.

2. Setting time is 2 hours and 45 minutes.
3. Compressive strength is 40 MPa immediately after setting and 70 MPa after 21 days.
4. Contrast to $Ca(OH)_2$ it produces hard setting nonresorbable surface.
5. It sets in a moist environment (hydrophilic in nature).
6. It has low solubility.
7. It shows resistance to marginal leakage.
8. It also reduces bacterial migration.
9. It exhibits excellent biocompatibility in relation with vital tissues.
10. The compressive strength of MTA is equal to IRM and Super EBA but less than that of amalgam.
11. MTA is also known as ***Portland's cement*** except for addition of bismuth oxide which is added for modifying its setting properties. Its consistency is similar to very hard cement, which can be compared to concrete.

Commercially it is available under the name Pro Root MTA (Dentsply).

Manipulation of MTA

To prepare MTA, a small amount of *liquid and powder are mixed to putty consistency*. Since, MTA mixture is a *loose granular aggregate* (like concrete cement) it *doesn't stick* very well *to any instrument*. It can't be carried out in cavity with normal cement carrier and thus has to be *tried with Messing gun, amalgam carrier* or *specially designed carrier*. Once MTA is placed, it is *compacted with burnishers and micropluggers*. Unless compacted very lightly, the loosely bound aggregate will be pushed out of the cavity. Next, a *small damp cotton pellet is used to gently clean the resected surface* and *to remove any excess MTA from cavity*.

Advantages of MTA

1. Water based chemistry, so requires moisture for setting
2. Excellent biocompatibility
3. Normal healing response without inflammation
4. Least toxic of all the filling materials
5. Reasonably radiopaque
6. Bacteriostatic in nature
7. Resistance to marginal leakage.

Disadvantages of MTA

1. Difficult to manipulate
2. Long setting time (3 - 4 hours)
3. Costly.

Precautions to be taken for MTA

1. MTA material should be kept in closed container to avoid moisture
2. MTA must be stored in dry area
3. MTA material should be immediately placed after mixing with liquid, to prevent dehydration during setting
4. Do not irrigate after placing MTA, remove excess water with moist cotton pallet
5. Adding too much or too little liquid will reduce the ultimate strength of the material
6. MTA material usually takes 3 - 4 hours but the working time is about five minutes. If more working time is needed, the mixed material should be coverted with a moist gauge pad to prevent evaporation.

Indications of use of MTA

1. As a pulp capping material.
2. For the repair of root canals as an apical plug during apexification.
3. For the repair of root perforations during root canal therapy.
4. For the repair of root resorptions.
5. As a root end filling material.

Clinical Applications of MTA (Fig. 8.45)

1. ***Pulp capping***: Vital pulp therapy is indicated in some cases. By placing MTA over the exposed area often

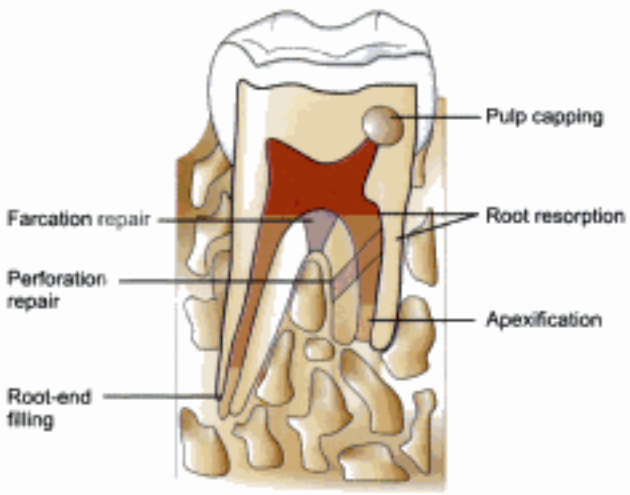


Fig. 8.45: Clinical applications of MTA

allows healing and preservation of vital pulp without further treatment. Rinse the cavity with sodium hypochlorite to disinfect the area. Mix the MTA with enough sterile water to give it a putty consistency. Apply it over the exposed pulp and remove the excess. Blot the area dry with a cotton pellet and restore the cavity with an amalgam or composite filling material.

2. ***Apexification***: MTA is excellent material for apexification because it creates a permanent apical plug at the outset of treatment.

   Vital pulp: Isolate the tooth with a rubber dam and perform a pulpotomy procedure. Place the MTA over the pulp stump and close the tooth with temporary cement until the apex of the tooth closes up.

   Non vital pulp: Isolate the tooth with a rubber dam and perform root canal treatment. Mix the MTA and compact it to the apex of the tooth, creating a 2 mm thickness of plug. Wait for it to set; then fill in the canal with cement and gutta percha.

3. ***Internal and external root resorption***: The root resorption is an idiopathic condition resulting in the breakdown or destruction of the root structure. In the case of *internal root resorption*, isolate the tooth and perform RCT in the usual manner. Once the canal has been cleaned and shaped, prepare a putty mixture of MTA and fill the canal with it, using a plugger or gutta percha cone and obturate the canal. In the case of *external resorption*, complete the root canal therapy for that tooth. Raise a flap and remove the defect on the root surface with a round bur. Mix the MTA in the same manner as above and apply it to the root surface. Remove the excess cement and condition the surface with tetracycline. Graft the defect with decalcified freeze dried bone allograft and a calcium sulfate barrier.

4. ***Perforation***: Perforations are the result of procedural error in which communication between the pulp canal and periodontal tissues occur. First finish cleaning and shaping of the perforated canal. Irrigate the canal really well with sodium hypochlorite and dry it with a paper point. If the perforation is down at the mid to apical third, then follow the directions for treating an internal resorption. If the perforation is closer to the coronal third, then obturate the canal with gutta percha as usual. Next, remove the gutta percha below the perforation using the Pesso reamer. Mix the MTA and fill the rest of the canal up with a plugger.

5. ***Root - end filling***: Root end filling is required when an endodontic case can best be treated or retreated with a surgical (extra radicular) rather than intra-radicular approach. MTA has shown excellent sealing ability and allows periradicular healing when used as a root end filling material during periradicualr surgery.

   First, gain access to the root-end and resect the root end with a surgical bur. Prepare a class I cavity preparation. Isolate the area and achieve hemostasis. Mix the MTA according to manufacture's instructions. Condense the MTA material into the cavity using a small plugger. Excess cement is removed with the help of moist gauge. Confirm with the help of radiograph.

9 Internal Anatomy and Access Cavity Preparation

INTRODUCTION

For the success of endodontic therapy, the knowledge of pulp anatomy cannot be ruled out. It is essential to have the knowledge of normal and usual configuration of the pulp cavity and its variations from the normal.

Before starting the endodontic therapy after correct diagnosis and treatment planning, one must have thorough knowledge of pulp anatomy. The pulp cavity must be mentally visualized three dimensionally. In addition to general morphology, variations in canal system must be kept in mind while performing the root canal therapy.

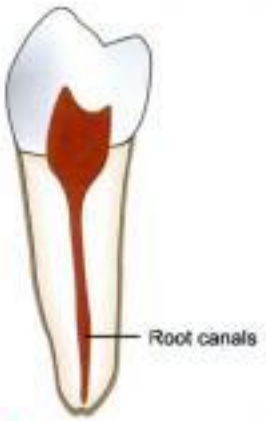


Fig. 9.1: Diagram showing pulp cavity

PULP CAVITY

Various studies have been conducted regarding the anatomy of pulp cavity of teeth. The pulp cavity lies within the tooth and is enclosed by dentin all around except at the apical foramen. It is divided into two—a coronal and a radicular portion. The coronal portion, i.e. pulp chamber (Fig. 9.1) reflects the external from of crown. The roof of pulp chamber consists of dentin covering the pulp chamber occlusally or incisally. The floor of pulp chamber merges into the root canal at the orifices. Thus canal orifices are the openings in the floor of pulp chamber leading into the root canals (Fig. 9.2).

The root canal extends from canal orifice to the apical foramen. In anterior teeth, the pulp chamber merges into

the root canal (Fig. 9.3) but in multirooted posterior teeth this division becomes quite obvious (Fig. 9.4).

Usually a root canal has curvature or constriction before

apex. Curvature can be smooth or sharp, single or double in form of letter "S".

The ***cementodentinal junction*** is the point in the canal, where cementum meets dentin. The position of CDJ varies but usually it lies 0.1 mm from the apical foramen (Fig. 9.5).

The root canal treatment of apical part of root is difficult sometimes because of presence of accessory and lateral canals, pulp stones, varying amounts of irregular secondary dentin and areas of resorption.

The main problems associated with apical part of root are its variability and unpredictability. Because of great variation in size and shape, problems may occur during the endodontic treatment. In teeth with multiple canals, isthmus is often found. An isthmus is a narrow, ribbon shaped communication between two root canals which can be complete (Fig. 9.6) or incomplete, i.e. a faint communication (Fig. 9.7). It contains pulp or pulpally derived tissue and

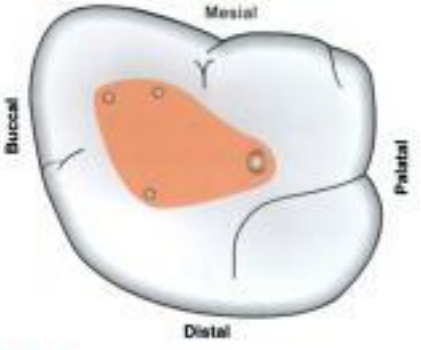


Fig. 9.2: Pulp orifices indicating openings of root canals in the floor of pulp chamber

Fig. 9.3: Pulp cavity of anterior teeth

Fig. 9.4: Pulp cavity of posterior teeth

Fig. 9.5: Variations in position of CDJ

Fig. 9.6: Isthmus-complete

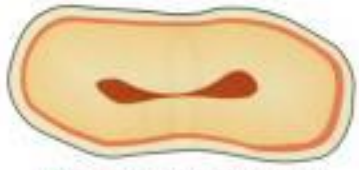


Fig. 9.7: Incomplete isthmus

acts as store house for bacteria so the isthmus should be well prepared and filled if seen on resected root surface.

COMMON CANAL CONFIGURATION

Various researches have been conducted to study normal and variations in normal anatomy of pulp cavity, but

exhaustive work on canal anatomy has done by ***Hess***. He studied branching, anastomoses, intricate curvatures, shape, size and number of root canals in different teeth. Others who have contributed to the study of anatomy of pulp are Wheeler, Rankine-Wilson, Weine, Perth, etc.

In most cases, number of root canals corresponds with number of roots but a root may have more than one canal. Despite of many combinations of canals which are present in the roots of teeth, the four categories of root canal system can be described. These are as follows:

1. ***Type I:*** Single canal from pulp chamber to apex (Fig. 9.8).

Fig. 9.8: Type I root canal system

2. ***Type II:*** Two separate canals leaving the chamber but exiting as one canal (Fig. 9.9).

Fig. 9.9: Type II root canal system

3. ***Type III:*** Two separate canals leaving the chamber and exiting as two separate foramina (Fig. 9.10).

Fig. 9.10: Type III root canal system

4. ***Type IV:*** One canal leaving the chamber but dividing into two separate canals and exiting in two separate foramina (Fig. 9.11).

Vertucci have also studied the canal anatomy extensively. He established eight different classification of pulp anatomy

Fig. 9.11: Type IV root canal system

rather than four. Classification for root canal system as given by Vertucci (Fig. 9.12)

- ***Type I:*** Single canal from orifice to apex.
- ***Type II:*** Two canals leaving the pulp chamber but joining shortly before apex
- ***Type III:*** One canal dividing into two within the body of the root and then again forming one canal.
- ***Type IV :*** Two canals exiting into two apices.
- ***Type V:*** One canal leaving the chamber dividing into two with two apices.
- ***Type VI:*** Two canals leaving the chamber merging in body and then redividing into two apices.
- ***Type VII:*** One canal leaving the chamber, dividing and then rejoining in body of the root and finally redividing into two apices.
- ***Type VIII:*** Three canals from chamber to apex.

This classification does not consider possible positions of auxilliary canals or portion at which apical foramen exit the root.

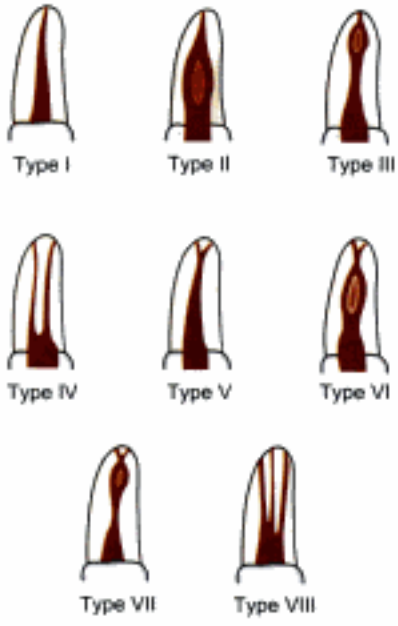


Fig. 9.12: Vertucci's classification of root canal system

Methods of Determining Pulp Anatomy

1. *Anatomic studies:* The knowledge of anatomy gained from various studies and textbooks is commonly used method.
2. *Radiographic method:* Radiographs are also useful in assessing the root canal anatomy. But very good quality of radiographs is needed for this purpose. Since radiographs are two dimensional picture of a three dimensional object, some errors may occur by them.

VARIATION IN THE INTERNAL ANATOMY OF TEETH

The canal configuration can vary in some cases because of numerous reasons like development anomalies, hereditary factors, trauma, etc. Usually the variations in root morphologies tend to be bilateral.

Commonly seen Anomalies of Pulp Cavities are as follows:

Lingual Groove

It is a surface in-folding of dentin directed from the cervical portion towards apical direction. It is frequently seen in maxillary lateral incisors.

It is usually associated with deep narrow periodontal pocket which often communicates with pulp causing endodontic-periodontal relationship. Prognosis of such teeth is poor and treatment is difficult.

High Pulp Horns

Commonly high pulp horns are found in recently erupted teeth (Fig. 9.13). So there are chances of pulp exposure in these teeth by caries or accidental cavity preparation.

C-shaped Canals

This type of canal is usually found in mandibular molars. They are named so because of its morphology. Pulp chamber in C-shaped molar is single ribbon shaped with 180 degree arc or more (Fig. 9.14). Root structure of C-shaped molar has various anatomic variations below the orifice level. It has been divided into two basic groups:

1. Single ribbon shaped canal from orifice to apex
2. Three or more distinct canals below C-shaped orifice.

The prognosis of root canal treatment in such teeth is questionable because of complex internal anatomy. Therefore, to increase the success rate in such cases, additional treatment measures such as use of surgical operating microscope or microtomography of root canals should be taken.

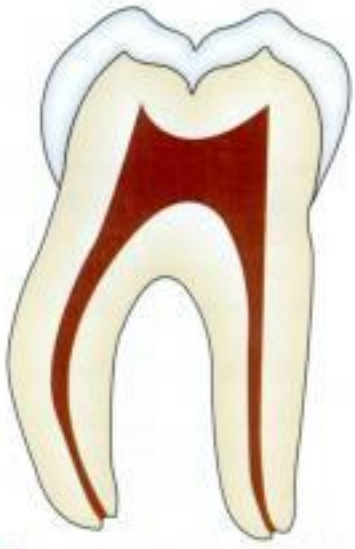


Fig. 9.13: Young permanent tooth showing high pulp horns

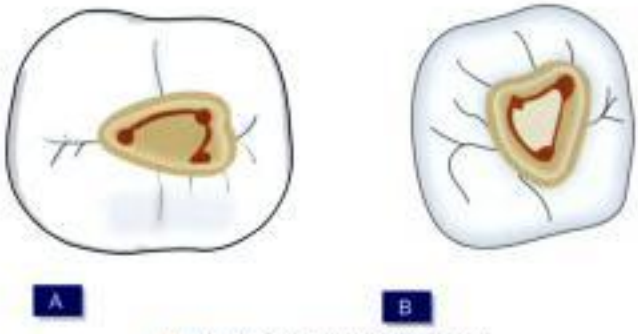


Fig. 9.14: C-shaped canal system

Presence of Extracanals

More than 70 percent of maxillary first molar have shown the occurrence of second mesiobuccal canal, and this is found to be most common reason for retreatment of maxillary molars. Location of orifice can be made by visualizing a point at the intersection between a line running from mesiobuccal to palatal canal and a perpendicular from the distobuccal canal.

In mandibular molars extracanals are found in 38 percent of the cases. A second distal canal is suspected when distal canal does not lie in midline of the tooth.

Two canals in mandibular incisors are reported in 41 percent of the cases. And among mandibular premolars more than 11 percent of teeth have shown the presence of two canals. In most of the cases no separate orifices are located

for two different canals. Usually the lingual canal projects from the wall of main buccal canal at an acute angle.

Dilacerations

Dilacerations is an extraordinary curving of the roots of the teeth. Etiology of dilacerations is usually related to trauma during the root development in which movement of the crown and a part of root may result in sharp angulation after tooth completes development (Fig. 9.15).

Fig. 9.15: A dilacerated root with sharp angulation

Dens in dente or Dens Invaginatus

This condition represents an exaggeration of the lingual pit (Fig. 9.16). Most commonly involved teeth are permanent maxillary lateral incisors. This condition may range from being superficial that is involving only crown part to pit in which both crown and root are involved. Tooth with dens invaginatus has tendency for plaque accumulation which predisposes it to early decay and thus pulpitis.

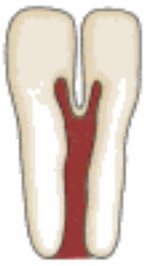


Fig. 9.16: Dens in dente

Dens Evaginatus

In this condition an anomalous tubercle or cusp is located on the occlusal surface (Fig. 9.17). Because of occlusal abrasion, this tubercle wears off fast causing early exposure of accessory pulp horn that extends into the tubercle. This may further result in periradicular pathology in otherwise caries free teeth even before completion of the apical root development. This condition is commonly seen in premolar teeth.

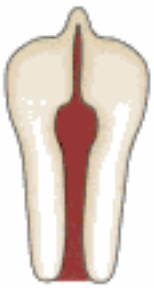


Fig. 9.17: Dens evaginatus

Taurodontism

This condition is named so because it resembles teeth of bull. In taurodontism, teeth show elongated crowns or apically displaced furcations resulting in pulp chambers which have increased apico-occlusal height. This condition is commonly seen associated with syndromes like ***Klinefelter syndrome*** and ***Down syndrome***.

Variations in the Internal Anatomy:
- Lingual groove
- High pulp horns
- C-shaped canals
- Presence of extra canals
- Dilacerations
- Dens in dente
- Dens evaginatus
- Taurodontism

FACTORS AFFECTING INTERNAL ANATOMY

Internal anatomy of teeth, i.e. pulp cavity though reflects the tooth form, yet various environmental factors whether physiological or pathological affect its shape and size because of pulpal and dentinal reaction to them. These factors can be enlisted as:

1. ***Age:*** With advancing age, there is continued dentin formation causing regression in shape and size of pulp cavity (Fig. 9.18). Clinically it may pose problems in locating the pulp chamber and canals.
2. ***Irritants:*** Various irritants whether in form of caries, periodontal disease, attrition, abrasion, erosion, cavity preparation and other operative procedures may stimulate dentin formation at the base of tubules in the underlying pulp leading to alteration in internal anatomy.

3. ***Calcific metamorphosis:*** Result commonly as a result of trauma to a recently erupted tooth.
4. ***Calcifications:*** In form of pulp stones or diffuse calcifications are usually present in chamber and the radicular pulp. These are either normal or may form as a result of irritation. These alter the internal anatomy of teeth and may make the process of canal location difficult (Fig. 9.19).
5. ***Resorption:*** Chronic inflammation or for unknown cause internal resorption may result in change of shape of pulp cavity making the treatment of such teeth challenging (Fig. 9.20).

Factors Affecting Internal Anatomy:
- Age
- Irritants
- Calcific metamorphosis
- Canal calcifications
- Resorption

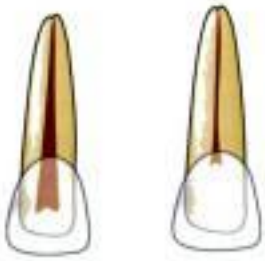


Fig. 9.18: Atrophy in size of pulp cavity with advancing age

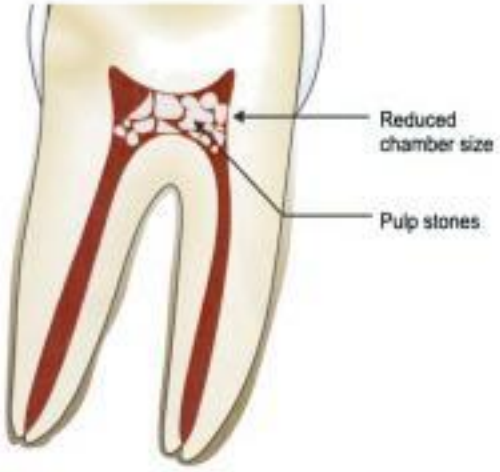


Fig. 9.19: Pulp stones makes the process of canal location difficult

Fig. 9.20: Internal resorption

INDIVIDUAL TOOTH ANATOMY

Maxillary Central Incisor (Fig. 9.21)

Average Tooth Length

The average length of the maxillary central incisor is 22.5 mm. The average pulp volume of this tooth is 12.4 mm^3.

Pulp Chamber

- It is located in the center of the crown, equal distance from the dentinal walls.
- Mesiodistally, pulp chamber follows the outline of the crown and it is ovoid in shape.
- Buccopalatally the pulp chamber is narrow as it transforms into the root canal with a constriction just apical to the cervix.

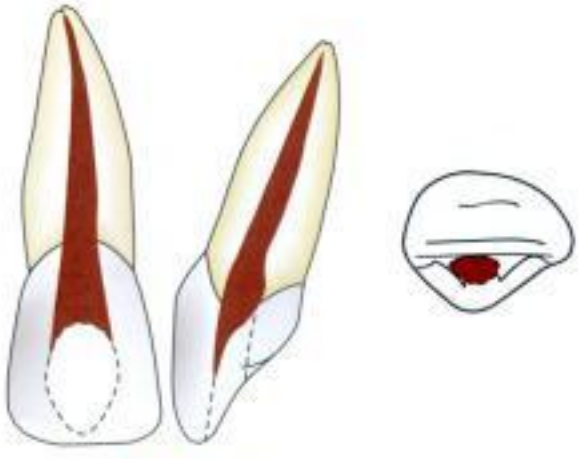


Fig. 9.21: Maxillary central incisor

- In young patients, central incisors usually have three pulp horns that correspond to enamel mammelons on the incisal edge.

Root Canal

- Usually central incisor has one root with one root canal.
- Coronally, the root canal is wider buccopalatally.
- Coronally or cervically, the canal shape is ovoid in cross-section but in apical region, the canal is round.
- The root canal differs greatly in outline in mesiodistal and labiopalatal view.
  a. Mesidistal view shows a fine straight canal.
  b. In labiopalatal view the canal is very much wider and often shows a constriction just apical to the cervix.
- Usually lateral canals are found in apical third.
- Most of the time, the root of central incisor is found to be straight.

Clinical Considerations

- A pulp horn can be exposed following a relatively small fracture of an incisal corner in the young patient.
- Placing the access cavity too far palatally makes straight line access difficult.
- In order to clean a ribbon shaped canal effectively, the operator relies on the effectiveness of irrigant solutions.

Maxillary Lateral Incisor (Fig. 9.22)

Average Length

The average length of maxillary lateral incisor is 21 mm with average pulp volume of 11.4 mm^3.

Pulp Chamber

The shape of pulp chamber of maxillary lateral incisor is similar to that of maxillary central incisor but there are few differences.

I. The incisal outline of the pulp chamber tends to be more rounded.
II. Lateral incisors usually have two pulp horns, corresponding to the development mammelons.

Root Canal

- Root canal has finer diameter than that of central incisor though shape is similar to that.
- Labiopalatally, the canal is wider and usually shows constriction just apical to the cervix.
- Canal is ovoid labiopalatally in cervical third, ovoid in middle third and round in apical third.
- Apical region of the canal is usually curved in a palatal direction.

Clinical Considerations

- Cervical constriction may need to be removed during coronal preparation to produce a smooth progression from pulp chamber to root canal.
- Since palatal curvature of apical region is rarely seen radiographically, during cleaning and shaping ledge formation may occur at this curve. This may result in root canal filling short of apex and other problems.
- Apical curvature can also complicate surgical procedures like root end cavity preparation and root resection.

Maxillary Canine (Fig. 9.23)

Average Tooth Length

It is the longest tooth with an average length of 26.5 mm with average pulp space volume of 14.7 mm^3.

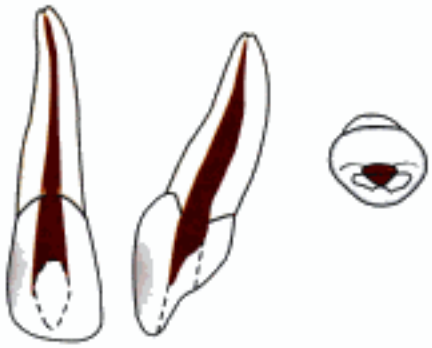


Fig. 9.22: Maxillary lateral incisor

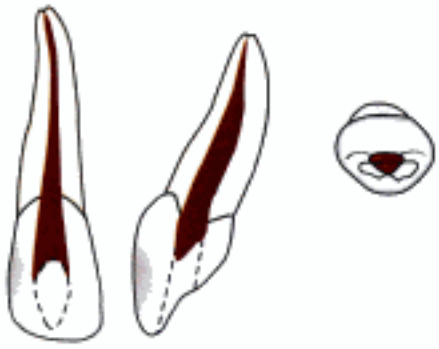


Fig. 9.23: Maxillary canine

Pulp Chamber

- Labiopalatally, the pulp chamber is almost triangular shape with apex pointed incisally
- Mesiodistally it is narrow, sometimes resembling a flame. At cervix, there can be constrictions sometimes
- In cross-section it is ovoid in shape with larger diameter labiopalatally
- Usually one pulp horn is present corresponding to one cusp.

Root Canal

- Normally there is single root canal which is wider labiopalatally than in mesiodistal aspect
- Cross-section at cervical and middle third show its oval shape, at apex it becomes circular
- Canal is usually straight but may show a distal apical curvature.

Clinical Considerations

- Cervical constriction needs to be shaped during coronal flaring to produce uniformly tapered preparation
- When long, sclerosed canal is being prepared, care must be taken to avoid blockage of the root canal
- Surgical access sometimes becomes difficult because of their long length.

Maxillary First Premolar (Fig. 9.24)

Average Tooth Length

This tooth has generally two roots with two canals and average length of 21 mm. The pulp space volume of maxillary first premolar is 18.2 mm^3.

Pulp Chamber

- Pulp chamber is wider buccopalatally with two pulp horns, corresponding to buccal and palatal cusps
- Roof of pulp chamber is coronal to the cervical line
- Floor is convex generally with two canal orifices.

Root Canal

- Maxillary first premolar has two roots in most (>60%) of the cases but cases with single root or three roots have also been reported
- Buccal canal is directly under the buccal cusp and palatal canal is directly under the palatal cusp
- Cross-section of root canals shows ovoid shape in cervical third with larger dimensions buccopalatally At middle and apical third, they show circular shape in cross-section
- The root canals are usually straight and divergent.

Clinical Considerations

- To locate both the canals properly, a good quality of radiograph should be taken from an angle so as to avoid superimposition of canals.
- Avoid over flaring of the coronal part of the buccal root to avoid perforation of palatal groove present on it.
- Surgical procedures on first premolar should be given more consideration since palatal root may be difficult to reach.
- In maxillary first premolar, failure to observe the distal—axial inclination of the tooth may lead to perforation.

Maxillary Second Premolar (Fig. 9.25)

Average length of maxillary second premolar is 21.5 mm. Average pulp volume is 16.5 mm^3.

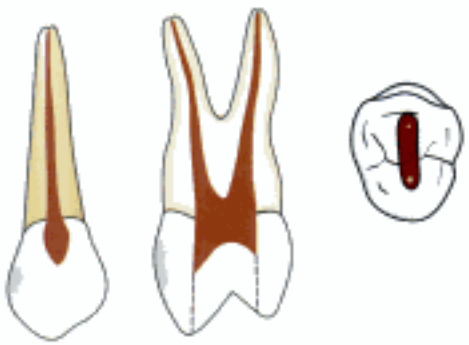


Fig. 9.24: Maxillary first premolar

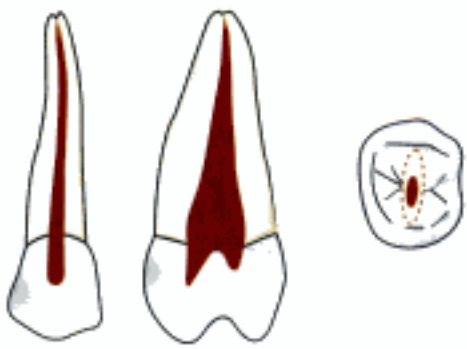


Fig. 9.25: Maxillary second premolar

Pulp Chamber

- Maxillary second premolar usually has one root with a single canal, but shape of canal system is variable
- Pulp chamber is wider buccopalatally and narrower in mesiodistal direction
- In cross-section, pulp chamber has narrow and ovoid shape.

Root Canals

- In more than 60 percent of cases single root with single canal is found. There may be a single canal along the entire length of the root
- If there are two canals, they may be separated or distinct along the entire length of the root or they may merge to form a single canal as they approach apically
- Canal is wider buccopalatally forming a ribbon like shape
- At cervix, cross-section shows ovoid and narrow shape, at middle third it is ovoid which becomes circular in apical third.

Clinical Considerations

- Narrow ribbon like canal is often difficult to clean and obturate effectively
- Care should be taken to explore, clean and obturate the second canal of maxillary second premolar (40 % of the cases).

Maxillary First Molar (Fig. 9.26)

Average Tooth Length

The average tooth length of this tooth is 21 mm and average pulpal volume is 68.2 mm^3.

Pulp Chamber

- Maxillary first molar has the largest pulp chamber with four pulp horns, viz. mesiobuccal, mesiopalatal, distobuccal and distopalatal.

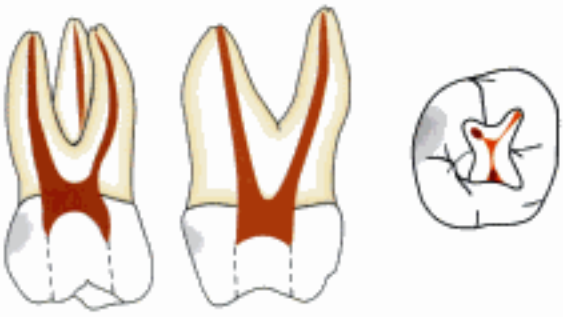


Fig. 9.26: Maxillary first molar

- Bulk of pulp chamber lies mesial to the oblique ridge across the surface of the tooth
- The four pulp horns are arranged in such a fashion which gives it rhomboidal shape in the cross-section. The four walls forming roof converge towards the floor, where palatal wall almost disappears making a triangular form in cross-section
- Orifices of root canals are located in the three angles of the floor; Palatal orifice is largest and easiest to locate and appears funnel like in the floor of pulp chamber.
- Distobuccal canal orifice is located more palatally than mesiobuccal canal orifice
- More than 60 percent of maxillary first molars have shown the presence of two canals in mesiobuccal root. The minor mesiobuccal canal (MB_2) is located on a line between palatal canal orifice and the main mesiobuccal canal orifice.

Root Canals

- Maxillary is generally three rooted with three or four canals
- Two canals in mesiobuccal root are usually closely interconnected and sometimes merge into one canal
- Mesiobuccal canal is the narrowest of the three canals, flattened in mesiodistal direction at cervix but becomes round as it reaches apically
- Distobuccal canal is narrow, tapering canal, sometimes flattened in mesiodistal direction but generally it is round in cross-section
- The palatal root canal has largest diameter which has rounded triangular cross-section coronally and becomes round apically
- Palatal canal can curve buccally in the apically one third
- Lateral canals are found in 40 percent of three canals at apical third and at trifurcation area.

Clinical Considerations

- Buccal curvature of palatal canal may not be visible on radiographs, leading to procedural errors
- MB_2 should be approached from distopalatal angle since the initial canal curvature is mesial
- Sometimes isthmus is present between mesiobuccal canals, it should be cleaned properly for success of the treatment
- Mesiobuccal canals show curvature sometimes which is not visible radiographically. So, care should be taken while doing endodontic therapy

- Since pulp chamber lies mesial to oblique ridge, pulp cavity is cut usually mesial to oblique ridge
- Caries, previous restorative procedures, attrition etc can lead to formation of secondary dentin causing alteration in pulp cavity. So careful study of preoperative radiographs is mandatory to avoid any procedural errors
- Perforation of a palatal root is commonly caused by assuming canal to be straight.

Maxillary Second Molar (Fig. 9.27)

Average Tooth Length

The average tooth length of this tooth is 20 mm and average pulp volume is 44.3 mm^3.

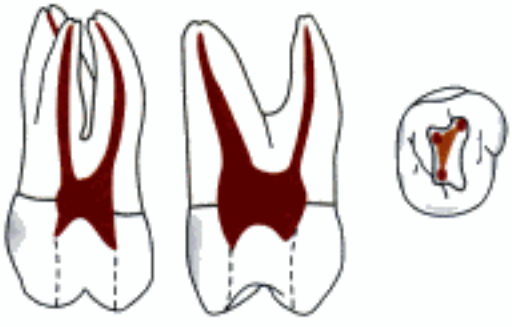


Fig. 9.27: Maxillary second molar

Pulp Chamber

- It is similar to maxillary first molar except that it is narrower mesiodistally
- Roof of pulp chamber is more rhomboidal in cross-section and floor is an obtuse triangle
- Mesiobuccal and distobuccal canal orifices lie very close to each other, sometimes all the three canal orifices lie in a straight line.

Root Canal

- Similar to first molar except that in maxillary second molar roots tend to be less divergent and may be fused
- Fewer lateral canals are present in roots and furcation area than in first molar.

Clinical Considerations

- Similar to maxillary first molar.

Maxillary Third Molar

Average Tooth Length

Average length of tooth is 16.5 mm.

Pulp Chamber and Root Canal

It is similar to second molar but displays great variations in shape, size, and form of both pulp chamber as well as root canal.

There may be presence of one, two, three or more canals sometimes.

Mandibular Teeth

Central Incisor (Fig. 9.28)

Average Tooth Length

Average length of this tooth is 21 mm.
Average pulp volume is 6.1 mm^3.

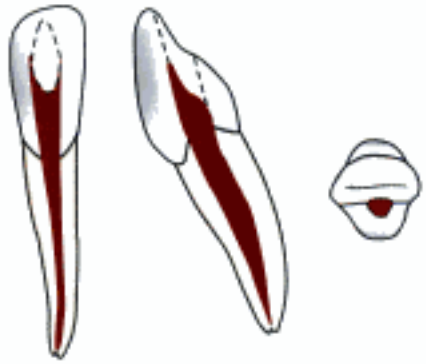


Fig. 9.28: Mandibular central incisor

Pulp Chamber

Mandibular central incisor is the smallest tooth in the arch.

- Pulp chamber is similar to maxillary central incisor being wider labiolingually pointed incisally with three pulp horns
- Cross-section of pulp chamber shows its ovoid shape.

Root Canals

- Various root canal formations have been seen in mandibular incisors. There can be a single canal from orifice to apex or a single canal by bifurcate into two canals or sometimes two separate canals are also found. Incidence of two canals can be as high as 41 percent
- Cross-section of root canals show wider dimension in labiolingual direction making it ovoid shape whereas round in the apical third
- Since canal is flat and narrow mesiodistally and wide buccopalatally, ribbon shaped configuration is formed.

Clinical Considerations

- If root canals are overprepared, because of presence of groove along the length of root and narrow canals, weakening of the tooth structure or chances of strip perforations are increased
- It is common to miss presence of two canals on preoperative radiograph if they are superimposed
- Since apex of mandibular central incisor is inclined lingually, the surgical access may become difficult to achieve.

Mandibular Lateral Incisor (Fig. 9.29)

Average Tooth Length

- Average length of mandibular lateral incisor is 21 mm.
- Average pulp volume is 7.1 mm^3.

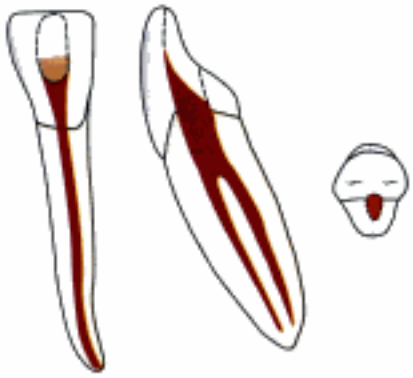


Fig. 9.29: Mandibular lateral incisor

Pulp Chamber

The configuration of pulp chamber is similar to that of mandibular central incisor except that it has larger dimensions.

Root Canals

- It has features similar to those of mandibular central incisor
- Usually the roots are straight or curved distally or labially, but distal curve is sharper than those of mandibular central incisors.

Clinical Considerations

They are similar to central incisor.

Mandibular Canine (Fig. 9.30)

- ***Average tooth length:*** Average length of the tooth is 22.5 mm
- Average pulp volume is 14.2 mm^3.

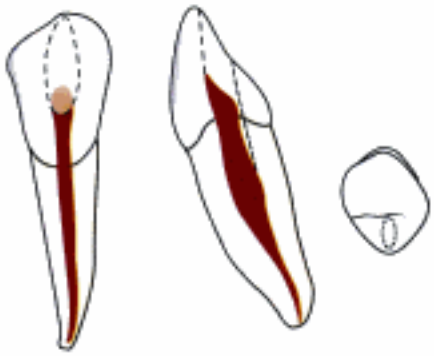


Fig. 9.30: Mandibular canine

Pulp Chamber

- On viewing labiolingually, the pulp chamber tapers to a point in the incisal third of the crown
- In cervical third of tooth, it is wider in dimensions and ovoid in cross-section at this level
- Pulp chamber appears to narrower mesiodistally
- Cervical constriction is also present.

Root Canals

- Mandibular canine usually has one root and one canal but can occasionally have two (14 % cases)
- Coronally, the root canal is oval in cross-section, becomes round in the apical region.
- Lateral canals are present in 30 percent of cases.

Clinical Consideration

- In older patients, where there is deposition of secondary dentine, it is necessary to incorporate the incisal edge into the access cavity for straight line access.

Mandibular First Premolar (Fig. 9.31)

Average Tooth Length

Average length of the tooth is 21.5 mm and average mature pulp volume is 14.9 mm^3.

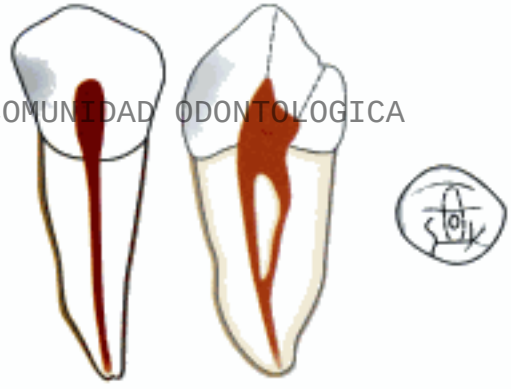


Fig. 9.31: Mandibular first premolar

Pulp Chamber

- Mesiodistally, the pulp chamber is narrow in dimension
- Pulp chamber has two pulp horns, the buccal horn being most prominent
- Buccolingually, the pulp chamber is wide and ovoid in cross-section.

Root Canal

- Mandibular first premolars usually have one root and one canal. Sometimes teeth have shown presence of second canal
- Mesiodistally, the canal is narrower in dimension
- Buccolingually, root canal cross-sections tend to be oval until the most apical extents, where they become round
- Lateral canals are present in 44 percent of the cases.

Clinical Considerations

- The access cavity in these teeth should have extended on to the cusp tip, in order to gain straight line access
- Surgical access to the apex of the mandibular first premolar is often complicated by the proximity of the mental nerve
- The lingual canal when present, is difficult to instrument. Access can usually be gained by running a fine instrument down the lingual wall of the main buccal canal until the orifice is located
- Perforation at the distogingival is caused by failure to recognize the distal tilt of premolar
- Apical perforation should be avoided by taking care of buccal curvature of the canal at the apex.

Mandibular Second Premolar (Fig. 9.32)

Average Tooth Length

The average length of this tooth is 22.5 mm and average mature pulp volume is 14.9 mm^3.

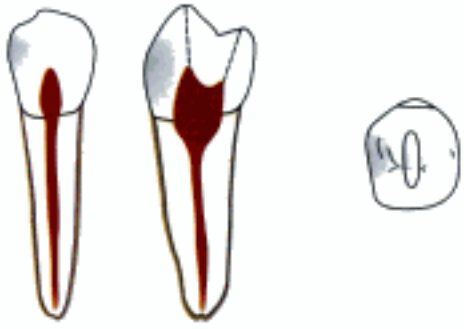


Fig. 9.32: Mandibular second premolar

Pulp Chamber

- It is similar to that of mandibular first premolar except that lingual pulp horn is more prominent
- Cross-section of pulp chamber shows oval shape with greatest dimensions buccolingually.

Root Canal

- Usually has one root and one canal and in 11 percent of the teeth, has a second canal
- Buccolingually, it is wider than that of mandibular first premolar
- Root canal cross-sections tend to be oval coronally and round apically.

Clinical Considerations

They are similar to mandibular first premolar.

Mandibular First Molar (Fig. 9.33)

Average Tooth Length: The average length of this tooth is 21 mm and an average pulp volume is 52.4 mm^3.

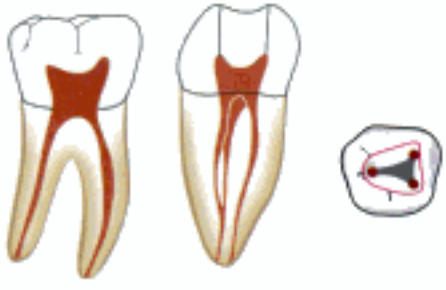


Fig. 9.33: Mandibular first molar

Pulp Chamber

- It is quadrilateral in cross-section at the level of the pulp floor and is wider mesially than distally
- The roof of the pulp chamber is rectangular in shape with straight mesial wall and rounded distal wall
- There may be presence of four or five pulp horns
- Mesiobuccal orifice is present under the mesiobuccal cusp
- The mesiolingual orifice is located in a depression formed by mesial and the lingual walls. Usually a connecting groove is present between mesiobuccal and mesiolingual orifices
- Distal orifice is the widest of all three canals. It is oval in shape with greater diameter in buccolingual direction.

Root Canals

Mandibular first molar usually has two roots with three canals. But teeth with three roots and four or five canals have also been reported.

- Mesial root has two canals, viz. mesiobuccal and mesiolingual which may exit in two foramina (>41% cases), exit in single foramen (30%) and may also exit in different pattern
- Mesiobuccal canal is usually curved and often exit in pulp chamber in a mesial direction
- Distal root generally has one canal (> 70% cases). But two canals are also seen in some cases (Fig. 9.34)
  A single distal canal is ribbon shaped and has largest diameter buccolingually. But when two canals are present in distal root, they tend to be round in the cross-section.

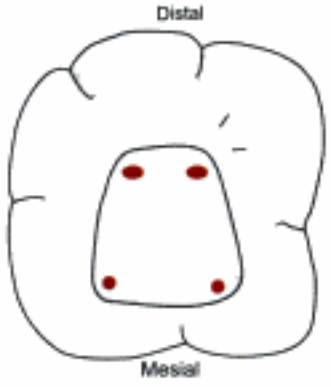


Fig. 9.34: Mandibular first molar showing two distal canals

Clinical Considerations

- Over-enlargement of mesial canals should be avoided to prevent procedural errors
- To avoid superimposition of the mesial canals, radiograph should be taken at an angle.

Mandibular Second Molar (Fig. 9.35)

Average Tooth Length

The average tooth length of this tooth is 20 mm and average pulp volume is 32.9 mm^3.

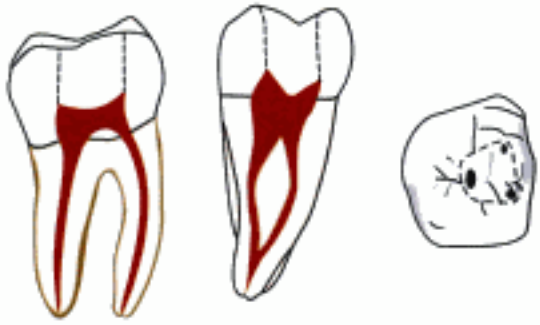


Fig. 9.35: Mandibular second molar

Pulp Chamber

- It is similar to that of mandibular first molar except that it is smaller in size
- Root canal orifices are smaller and closer together.

Root Canals

- Usually it contains two roots with three canals but variation in their presence (one or three roots, 2 canals) is also seen
- C-shaped canals are also seen, i.e. mesial and distal canals become fused into a fin.

Clinical Considerations

- C-shaped canals make the endodontic procedures difficult so care should be taken while treating them
- There may be only one mesial canal. The mesial and distal canals may lie in midline of the tooth
- Perforation can occur at mesial cervical region if one fails to recognize the mesially tipped molar.

Mandibular Third Molar

- Average Tooth Length
- Average length of this tooth is 17.5 mm.

Pulp Chamber and Root Canals

It resembles to that of mandibular first and second molar but with enormous variations, i.e. there may be presence of one, two or three canals. Anomalous configurations such as "C-shaped" root canal orifices are also seen commonly.

ACCESS CAVITY PREPARATION

It is a well established fact that success of endodontic therapy depends on the main three factors:

a. Cleaning and shaping
b. Disinfection
c. Three dimensional obturation of the root canal system.

Proper cleaning and shaping establishes the necessary conditions for next two factors. However, there is one step which precedes these factors, the error in this preliminary step would compromise the whole subsequent work. This preliminary step is the preparation of the access cavity, i.e. opening through the coronal portion of tooth which allows localization, cleaning, shaping, disinfection and a three dimensional obturation of the root canal system. Thus we can say that coronal access forms the foundation of pyramid of endodontic treatment (Fig. 9.36).

As we have seen success of endodontic therapy depends on proper evaluation and thereafter placement of this step. Any improperly prepared access cavity can impair the instrumentation, disinfection and therefore obturation resulting in decreased prognosis of the treatment.

Before going for access cavity preparation, after evaluating other factors, a study of preoperative periapical radiograph is necessary with a paralleling technique. It helps in knowing:

i. Morphology of the tooth.
ii. Anatomy of root canal system.
iii. Number of canals.
iv. Curvature of branching of the canal system.
v. Length of the canal
vi. Position and size of the pulp chamber and its distance from occlusal surface.
vii. Position of apical foramen.
viii. Calcification, resorption present if any.

The main objective of the access cavity preparation is to create a smooth, straight line access to the canal system and the apex. The optimal access cavity results in the straight entry into the canal orifices with line angles forming a funnel which drops smoothly into the canals (Fig. 9.37). Sometimes depending upon the location and number of canals, modification of the outline form may be needed.

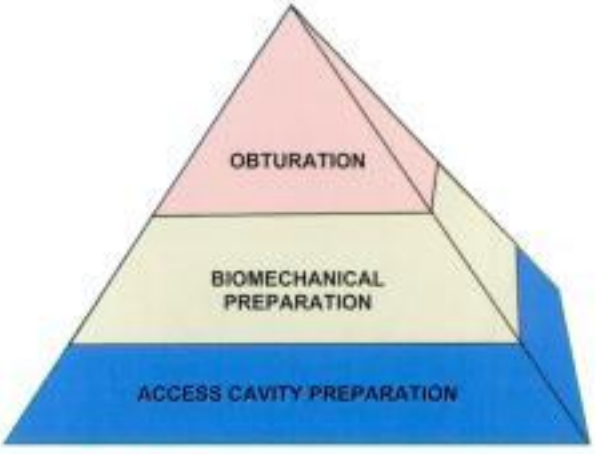


Fig. 9.36: Pyramid of endodontic treatment

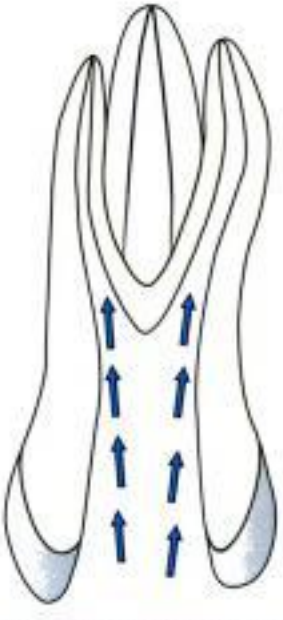


Fig. 9.37: Smooth, straight line access to root canal system

An ideal access preparation should have following qualities:

1. An unobstructed view into the canal.
2. A file should pass into the canal without touching any part of the access cavity.
3. No remaining caries should be present in access cavity.
4. Obturating instruments should pass into the canal without touching any portion of the access cavity.

Removal of coronal contacts on instruments reduce the adverse unidirectional forces directed on the instruments which may result in instrumental errors like ledging and perforation (Fig. 9.38).

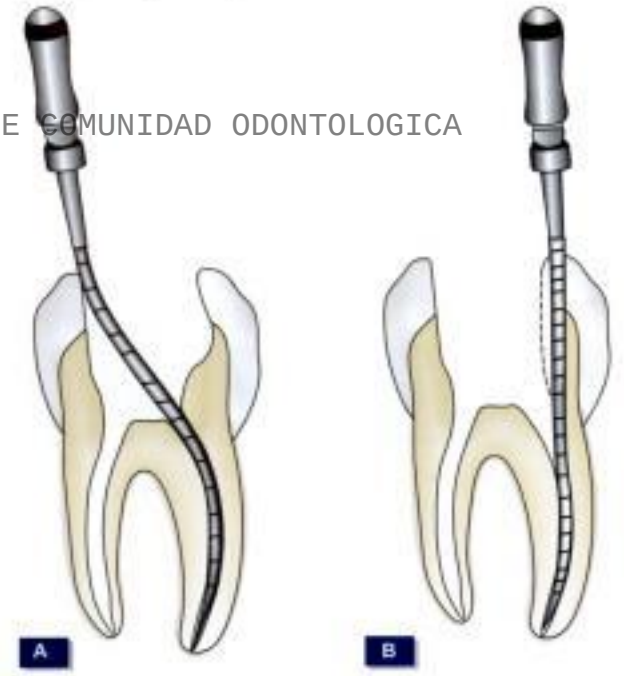


Fig. 9.38A: Not removing dentin from mesial wall causes bending of instrument while inserting in canal leading to instrumental errors

Fig. 9.38B: Removal of extra-dentin from access opening gives straight line access to the canal without any undue bending

Objectives of Access Cavity Preparation

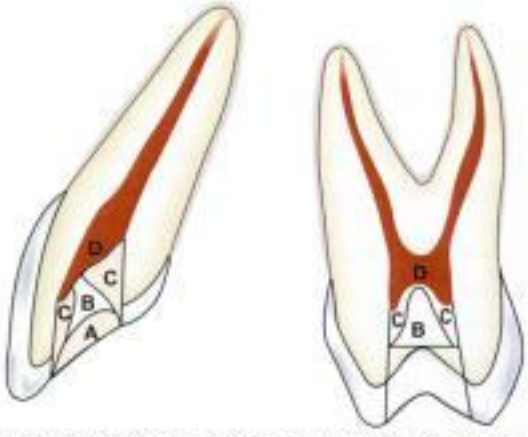
1. The main objective of access cavity preparation is to gain the direct access to the apical foramen.
2. Remove the entire roof of pulp chamber so that pulp chamber can be debrided.
3. Conserve as much sound tooth structure as possible and as consistent with treatment objectives.
4. Before starting the treatment, must make a three dimensional picture of internal anatomy of tooth.

Guidelines for Access Cavity Preparation (Fig. 9.39)

1. Before starting the access cavity preparation one should check the depth of preparation by aligning the bur and handpiece against the radiograph. This is done so as to note the position and depth of the pulp chamber (Fig. 9.40).
2. Place a safe ended bur in handpiece complete the outline form. The bur is penetrated into the crown until the roof of pulp chamber is penetrated.

Commonly recommended access opening bur is round bur, that is to penetrate the pulp chamber (Fig. 9.41). It prevents the overpreparation. Once the "drop in" into the pulp chamber is obtained, round bur is replaced by tapered fissured bur (Fig. 9.42).

One should avoid using flat ended burs which make highly irregular access walls, causing multiple ledges. Round ended carbide burs are used for access opening into cast restorations because they have distinct tactile sense when dropping into the pulp chamber.



A Penetration into enamel with No. 2 or No. 4 high speed round bur
B Exposure of pulp chamber with tapered fissure bur
C Refinement of the pulp chamber and removal of pulp chamber roof using round bur from inside to outside
D Complete debridement of pulp chamber space

Fig. 9.39: Guidelines for access cavity preparation

Fig. 9.40: Preoperative radiograph can help to note the position and depth of pulp chamber

Fig. 9.41: Entry into pulp chamber with round bur

Fig. 9.42: Round bur is replaced by tapered fissure bur once the "drop in" into the pulp chamber is obtained

Access finishing is best carried out by using burs with safe non-cutting ends. ***Advantage*** of using these burs is that they are less likely to damage or perforate the pulp chamber floor. ***Disadvantages*** of using these burs are that they can cut laterally and they cannot drop into small orifices to funnel the point of transition between the access cavity and walls.

3. When locating the canal orifices is difficult, one should not apply rubber dam until correct location has been confirmed.
4. Remove all the unsupported tooth structure to prevent tooth fracture during treatment.
5. Remove the chamber roof completely as this will allow the removal of all the pulp tissue, calcifications, caries or any residuals of previous fillings (Fig. 9.43). If pulp chamber is not completely deroofed, it can result in–
   a. Contamination of the pulp space.
   b. Discoloration of endodontically treated tooth.
6. The walls of pulp chamber are flared and tapered to form a gentle funnel shape with larger diameter towards occlusal surface (Fig. 9.44).
7. Endodontic access cavity is prepared through the occlusal or lingual surface never through proximal or gingival surface. If access cavity as made through wrong entry, it will cause inadequate canal instrumentation resulting iatrogenic errors (Fig. 9.45).
8. Inspect the pulp chamber for determining the location of canals, curvatures, calcifications using well magnification and illumination.

Fig. 9.43: Completely deroof the pulp chamber

Fig. 9.44: Complete access cavity preparation

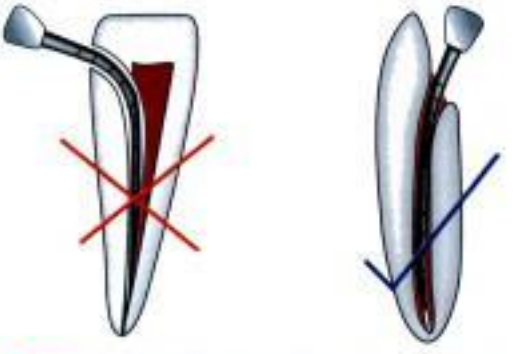


Fig. 9.45: Correct position for entering into the pulp cavity

ACCESS CAVITY OF ANTERIOR TEETH

- The access cavity shape varies with size and shape accordingly the internal anatomy of the pulp chamber, so internal anatomy of tooth must be determined before starting the treatment.
- Remove all the caries and any defective restorations so as to prevent contamination of pulp space as well as to have a straight line access into the canals.
- Access opening is started at centre of the lingual surface (Fig. 9.46). If it is made too small and too close to the cingulum the instrument tends to bind the canal walls and thus may not work optimally.
- Direct a round bur perpendicular to the lingual surface at its centre to penetrate the enamel. Once enamel is penetrated, bur is directed parallel to the long axis of the tooth, until 'a drop' in effect is felt (Fig. 9.47).
- Now when pulp chamber has been penetrated, the remainder of chamber roof is removed by working a round bur from inside to outside. This is done to remove all the obstructions of enamel and dentin overhangs that would entrap debris, tissues and other materials.
- Now locate the canal orifices using endodontic explorer. Sharp explorer tip is used to locate the canal orifices, to penetrate the calcific deposits if present, and also to evaluate the straight line access.
- Once the canal orifices have been located, the lingual shoulder is removed using Gates-Glidden drills or safe tipped diamond or carbide burs. Lingual shoulder is basically a prominence of dentin formed by removal of lingual roof which extends from the cingulum to approximately 2 mm apical to the orifice (Fig. 9.48).

During the removal of lingual shoulder, the orifice should also be flared so that it becomes confluent with all the walls of access cavity preparation. By this a straight line access to the apical foramen is attained, i.e. an endodontic file can reach up to apical foramen without bending or binding to the root canal wall. Any deflection of file occurs should be corrected because it can lead to instrumental errors (Fig. 9.49). The deflected instruments work under more stress, more chance of instrument separation is there.

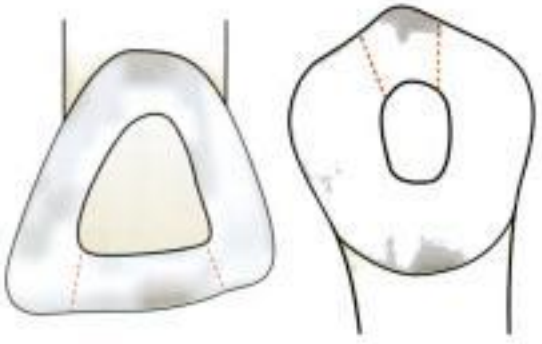


Fig. 9.46: Access opening is initiated at centre of lingual surface

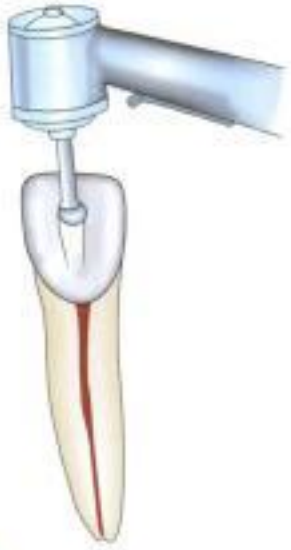


Fig. 9.47: Once enamel is penetrated, bur is directed parallel to long axis of tooth

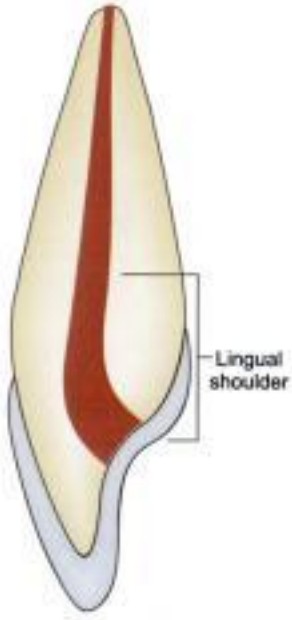


Fig. 9.48: Lingual shoulder is prominence of dentin formed by lingual roof. It extends from cingulum to 2 mm apical to the canal orifice

Fig. 9.49: Improper access cavity preparation causing deflection of instrument

Deflected instruments also result in procedural accidents like canal transportations, perforations, ledging and zipping.

- After the straight line access of the canal is confirmed by passing a file passively into the canal, one should evaluate the access cavity using magnification and illumination.
- Finally smoothening of the cavosurface margins of access cavity is done because rough or irregular cavity margins can cause coronal leakage through restorations. Also smooth cavity margins allow better and précised placement of final composite restoration with minimal coronal leakage.

Since the outline form of access cavity reflects the internal anatomy of the pulp space, though technique of the access opening of anterior teeth is the same, the shape may vary according to internal anatomy of each tooth.

Maxillary Central Incisor

The outline form of access cavity of maxillary central incisor is a rounded triangular shape with base facing the incisal aspect (Fig. 9.50). The width of base depends upon the distance between mesial and distal pulp horns. Shape may change from triangular to slightly oval in mature tooth because of less prominence of mesial and distal pulp horns.

Fig. 9.50: Outline of access cavity of maxillary incisor

Maxillary Lateral Incisor

The shape of access cavity is almost similar to that of maxillary central incisor except that:

i. It is smaller in size.
ii. When pulp horns are present, shape of access cavity is rounded triangle.
iii. Generally the pulp horns are missing so shape of access cavity which results is oval.

Maxillary Canine

Shape of access cavity of canine though is quite similar to incisors with following differences:

i. Canine doesn't have pulp horns
ii. Access cavity is oval in shape with greater diameter labiopalatally (Fig. 9.51).

Fig. 9.51: Outline of access cavity of maxillary canine

Mandibular Incisors

Mandibular central and lateral incisors are similar in shape of access cavity and the root canal system. Shape of access cavity of mandibular incisors is different from maxillary incisors in following aspects (Fig. 9.52):

i. It is smaller in shape.
ii. Shape is long oval with greatest dimensions directed incisogingivally.

Mandibular Canine

The shape of access opening of mandibular canine is similar to that maxillary of canine except that:

i. It is smaller in size.

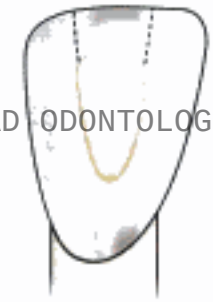


Fig. 9.52: Outline of access cavity of mandibular incisor

ii. Root canal outline is narrower in mesiodistal dimension
iii. Generally two canals are present in mandibular canine.

ACCESS CAVITY PREPARATION FOR PREMOLARS

- The basic step of access cavity preparation is removal of the caries and any other permanent restoration material if present.
- Determine the site of access opening on the tooth. In premolars, it is in the centre of occlusal surface between buccal and the lingual cusp tips (Fig. 9.53).

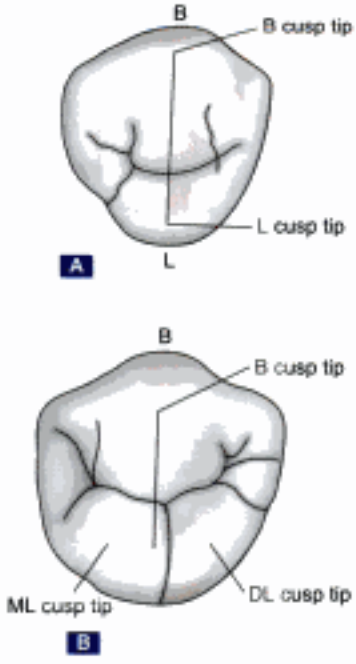


Fig. 9.53: Outline of access cavity of premolars

- Slight variations exist between mandibular and maxillary premolars because of the lingual tilt of mandibular premolars.
- Penetrate the enamel with No. 4 round bur in high speed contra angle handpiece. The bur should be directed parallel to the long axis of tooth and perpendicular to the occlusal table. Generally the external outline form for premolars is oval in shape with greater dimensions of buccolingual side (Fig. 9.54).

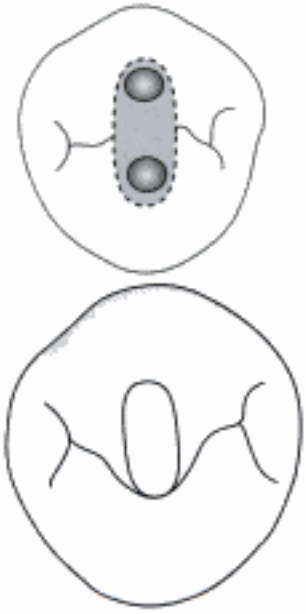


Fig. 9.54: Oval shaped access cavity of premolars

- Once the clinician feels "drop" into the pulp chamber, penetrate deep enough to remove the roof of pulp chamber without cutting the floor of pulp chamber. To remove the roof of pulp chamber and pulp horns, place the bur alongside the walls of pulp chamber and work from inside to outside.
  For removal of pulp chamber roof, round bur, a tapered fissure or a safety tip bur can be used.
- After removal of roof of pulp chamber, locate the canal orifices with the help of sharp endodontic explorer Ideally the canal orifices should be located at the corners of final preparation. Extension of orifices to the axial walls results in "Mouse Hole Effect".

  Mouse hole effect is caused because of under extension of the access cavity. This may result in hindrance to the straight line access which may further cause procedural errors (Fig. 9.55).

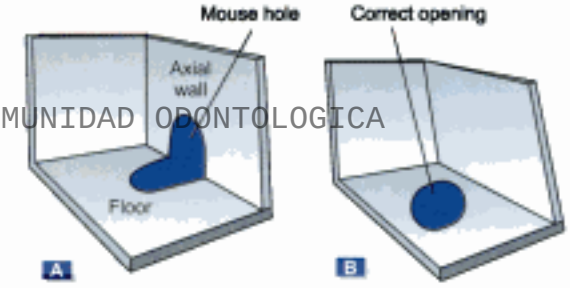


Fig. 9.55: Mouse hole effect: **A.** Mouse hole effect—Due to under extension of axial wall, orifice opening appears partly in axial wall and partly in floor, **B.** Correct opening

- Remove any remaining cervical bulges or obstructions using safety tip burs or Gates-Glidden drills and obtain a straight line access to the canals.

  It can be confirmed by passing a file passively into the canal which should reach the apex or the first point of curvature without any deflection.
- The walls of access cavity are then smoothened and sloped slightly towards the occlusal surface. The divergence of access cavity walls creates a positive seat for temporary restorations.

  Access cavity preparation for all premolars is same except for some differences.

Maxillary First Premolar

Shape of access cavity is ovoid in first premolar in which boundaries should not exceed beyond half the lingual incline of buccal cusp and half the buccal incline of lingual cusp.

Maxillary Second Premolar

It is similar to that of maxillary first premolar and varies only by anatomic structure of the pulp chamber.

Mandibular First Premolar

Following differences are seen in case of mandibular first premolar from the maxillary premolars:

i. There is presence of 30° lingual inclination of the crown to the root, hence the starting point of bur penetration should be halfway up the lingual incline of the buccal cusp on a line connecting the cusp tips (Fig. 9.53).
ii. Shape of access cavity is oval which is wider mesiodistally when compared with its maxillary counterpart.

Mandibular Second Premolar

The access cavity preparation is similar to mandibular first premolar except that in mandibular second premolar:

i. Enamel penetration is initiated in the central groove because its crown has smaller lingual tilt.
ii. Because of better developed lingual half, the lingual boundary of access opening extends halfway upto the lingual cusp incline, i.e. pulp chamber is wider buccolingually.
iii. Root canals are more often oval than round.
iv. Ovoid access opening is wider mesiodistally.

ACCESS CAVITY PREPARATION FOR MAXILLARY MOLARS

Though the technique of access cavity preparation of molar is similar to that of anterior teeth and premolar but because of anatomic differences, they are discussed separately.

- Remove all the carious portion or any restoration if present.
- Determine the shape and size of access opening by measuring the boundaries of pulp chamber mesially and distally and coronally on the radiograph.
- Determine the starting point of bur into the enamel. It is determined by mesial and distal boundary. Mesial boundary is a line joining the mesial cusps and the distal boundary is the oblique ridge. The starting point starting point of bur penetration is on the central groove midway between mesial and distal boundaries (Fig. 9.56).

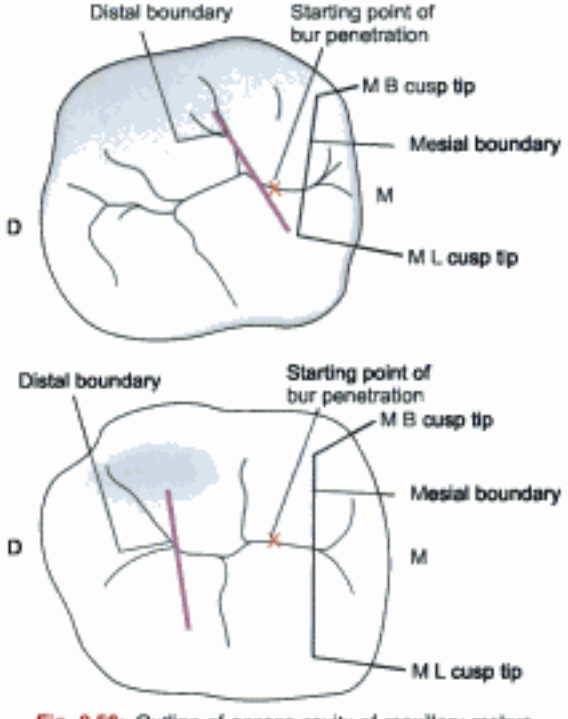


Fig. 9.56: Outline of access cavity of maxillary molars

- Now penetrate the enamel with No. 4 round bur in the central groove directed palatally and prepare an external outline form.
- Remove the bur deeper into the dentin until the clinician feels "drop" into the pulp chamber. Now remove the complete roof of pulp chamber using tapered fissure, round bur or safety tip diamond or the carbide bur working from inside to outside. The shape and size of the internal anatomy of pulp chamber guides the cutting.
- Explore the canal orifices with sharp endodontic explorer. All the canal orifices should be positioned entirely on the pulp floor and should not extend to the axial walls.
- After the canal orifices has been located, remove any cervical bulges, ledges or obstruction if present.
- Smoothen and finish the access cavity walls so as to make them confluent within the wall of pulp chamber and slightly divergent towards the occlusal surface.

Maxillary First Molar

- The shape of pulp chamber is rhomboid with acute mesiobuccal angle, obtuse distobuccal angle and palatal right angles (Fig. 9.57).

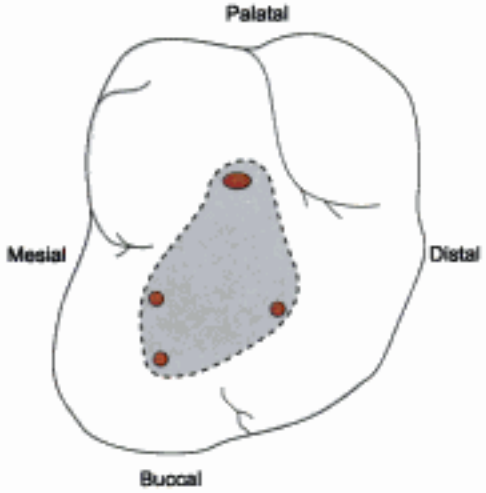


Fig. 9.57: Position of root canal orifices of maxillary first molar

- Palatal canal orifice is located palatally. Mesiobuccal canal orifice is located under the mesiobuccal cusp. Distobuccal canal orifice is located slightly distal and palatal to the mesiobuccal orifice.
  A line drawn to connect all three orifices (i.e. MB, DB and palatal) forms a triangle, termed as ***molar triangle***.
- Almost always a second mesiobuccal canal, i.e. MB_2 is present in first maxillary molars, which is located palatal and mesial to the MB_1. Though its position can vary sometimes it can lie a line between MB_1 and palatal orifices.
- Because of presence of MB_2, the access cavity acquires a rhomboid shape with corners corresponding to all the canal orifices, i.e. MB_1, MB_2, DB and palatal.

Luebke has shown that an entire wall is not extended to search and facilitate cleaning, shaping and obturation of extracanal. He recommended extension of only that portion of the wall where extracanal is present, and this may result in "***cloverleaf appearance***" in the outline form. Luebke referred this to as a ***shamrock preparation***.

Maxillary Second Molar

Basic technique is similar to that of first molar but with following differences:

i. Three roots are found closer which may even fuse to form a single root.
ii. MB_2 is less likely to be present in second molar.
iii. The three canals form a rounded triangle with base to buccal.
iv. Mesiobuccal orifice is located more towards mesial and buccal than in first molar.

ACCESS CAVITY PREPARATION FOR MANDIBULAR MOLARS

- It is similar to that of any other access cavity preparation in removal of caries and any restorative material if present.
- The enamel is penetrated with No. 4 round bur on the central fossa midway between the mesial and distal boundaries. The mesial boundary is a line joining the mesial cusp tips and the distal boundary is the line joining buccal and the lingual grooves (Fig. 9.58).

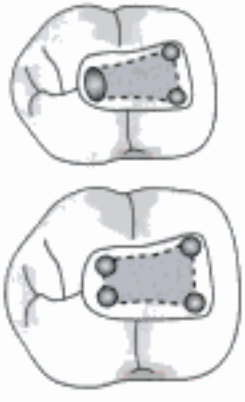


Fig. 9.58: Outline of access cavity of mandibular molars

- Bur is penetrated in the central fossa directed towards the distal root. Once the "drop" into pulp chamber is felt, remove whole of the roof of pulp chamber working from inside to outside with the help of round bur, tapered fissure bur or the safety tip diamond or the carbide bur as it was done in maxillary molars.
- Explore the canal orifices with sharp endodontic explorer and finally finish and smoothen the cavity with slight divergence towards the occlusal surface.

v. Distobuccal orifice is located midway between mesiobuccal and the palatal orifice.
vi. Second molars with fused roots usually have two canals, buccal and palatal though the number, type, shape and form of canals may vary.
vii. When four canals are present, the shape of access cavity is rhomboid but when two canals are present, access cavity is oval in shape with wider dimensions buccolingually.
viii. Shape and size of the access cavity may vary according to the size, shape and location of the canal orifices.

Mandibular First Molar

Mesiobuccal orifice is under the mesiobuccal cusp. Mesiolingual orifice is located in a depression formed by mesial and the lingual walls. The distal orifice is oval in shape with largest diameter buccolingually, located distal to the buccal groove.

Orifices of all the canals are usually located in the mesial two third of the crown (Fig. 9.58).

Cases have also been reported with an extramesial canal, i.e. middle mesial canal (1-15%) lying in the developmental groove between mesiobuccal and mesiolingual canals. Distal root has also shown to have more than one orifices, i.e. distobuccal, distolingual and middle distal. These orifices are usually joined by the developmental grooves.

- The shape of access cavity is usually trapezoidal or rhomboid irrespective of number of canals present.

  The mesial wall is straight, the distal wall is round. The buccal and lingual walls converge to meet the mesial and distal walls.

Mandibular Second Molar

Access opening of mandibular second molar is similar to that of first molar except for few differences. In mandibular second molar:

i. Pulp chamber is smaller in size.
ii. One, two or more canals may be present.
iii. Mesiobuccal and mesiolingual canal orifices are usually located closer together.
iv. When three canals are present, shape of access cavity is almost similar to mandibular first molar, but it is more triangular and less of rhomboid shape.
v. When two canal orifices are present, access cavity is rectangular, wide mesiodistally and narrow buccolingually.
vi. Because of buccoaxial inclination, sometimes it is necessary to reduce a large portion of the mesiobuccal cusp to gain convenience form for mesiobuccal canal.

MANAGING DIFFICULT CASES FOR ACCESS OPENING

Extensive Restorations

If extensive restorations or full veneer crowns are marginally intact with no caries, then they can be retained with access cavity being cut through them (Fig. 9.59). Restorative materials often alter the anatomic landmarks making the access cavity preparation difficult (Fig. 9.60).

If possible, complete removal of extensive restoration allows the most favorable access to the root canals. When restoration is not removed, and access cavity is made through it, following can occur:

i. Coronal leakage because of loosening of fillings due to vibrations while access preparation.

Fig. 9.59: When full veneer crown is marginally intact with no caries, access can be made through the crown

Fig. 9.60: Perforation caused during access cavity preparation while gaining entry through already placed crown

ii. Poor visibility and accessibility.
iii. Blockage of canal, because broken filling pieces may struck into the canal system.
iv. Misdirection of bur penetration (because in some cases restorations are placed to change the crown to root angulations so as to correct occlusal discrepancies).

If the restorations show no defect, leaky margins, fractures or caries, access can be made through them. For cutting porcelain restorations diamond burs are effective and for cutting through metal crowns, a fine cross-cut tungsten carbide bur is very effective.

Tilted and Angulated Crowns

If tooth is severely tilted, access cavity should be prepared with great care to avoid perforations. Preoperative radiographs are of great help in evaluating the relationship of crown to the root (Fig. 9.61). Sometimes it becomes necessary to open up the pulp chamber without applying the rubber dam so that bur can be placed at the correct angulation.

If not taken care, the access cavity preparation in tilted crowns can result in:

i. Failure to locate canals
ii. Gouging of the tooth structure
iii. Procedural accidents such as
   a. Instrument separation
   b. Perforation
   c. Improper debridement of pulp space

Calcified Canals

Calcifications in the pulp space are of common occurrence. Pulp space can be partially or completely obliterated by the pulp stones. Teeth with calcifications result in difficulty in locating and further treatment of the calcified canals.

Special tips for ultrasonic handpieces are best suited for treating such cases. They allow the précised removal of dentin from the pulp floor while locating calcified canals (Fig. 9.61). But magnification and illumination are the main requirements before negotiating a calcified canal.

If special tips are not available then a pointed ultrasonic scaler tip can be used for removal of calcifications from the pulp space. One should avoid over cutting of the dentin in order to locate the canals, this will further result in loss of landmarks and the tooth weakening. At the first indication that canal is found, introduce the smallest instrument with gentle passive motion both rotational and apical in the canal to negotiate it (Fig. 9.62). Use of chelating agents is also of great help while negotiating the calcified canals.

Fig. 9.61: Use of ultrasonic tip to remove dentin while locating calcified canals

Fig. 9.62: Introduce the smallest instrument into the canal at first indication of canal orifice

Sclerosed Canals

Sometimes sclerosed canals are found in teeth which make the endodontic treatment a challenge. For visualization, magnification and illumination are the main requirements. Dyes can be used to locate the sclerotic canals. While negotiating, the précised amount of dentin should be removed with the help of ultrasonic tips to avoid over cutting. Long shanked low speed number 2 round burs can also be used (Fig. 9.63). Use of chelating agents in these cases is not of much help because it softens the dentin indiscriminately, resulting in procedural errors such as perforations.

Fig. 9.63: Use of long shanked round bur to negotiate the sclerosed canal

Teeth with No or Minimal Crown

Though it seems to be quite simple to prepare access cavity in such teeth but some precautions are needed while dealing such cases:

a. Evaluate the preoperative radiograph to assess the root angulation.
b. Start the cavity preparation without applying rubber dam.
c. Evaluate the depth of penetration from preoperative radiograph.
d. Apply rubber dam as soon as the canals have been located.

If precautions are not taken in case of missing crown, there are chances of occurrence of iatrogenic errors like perforations due to misdirection of the bur. In such cases, sometimes it becomes imperative to rebuild the tooth previous to endodontic treatment. In teeth with weakened walls, it is necessary to reinforce the walls before initiating endodontic treatment. In other words, it is necessary to restore the natural form of a crown of the tooth to achieve following goals:

a. Return the tooth to its normal form and function.
b. Prevent coronal leakage during treatment.
c. Allow use of rubber dam clamps.
d. Prevent fracture of walls which can complicate the endodontic procedure.

10

Working Length Determination

- Introduction and History
- Definitions
- Significance of Working Length
- Different Methods of Working Length Determination
- Radiographic Method of Working Length Determination
- Mathematic Method of Working Length Determination
- Electronic Apex Locators

INTRODUCTION AND HISTORY

Determination of an accurate working length is one of the most critical steps of endodontic therapy. The cleaning shaping and obturation of root canal system cannot be accomplished accurately unless working length is determined precisely. Prognosis and microbiologic studies as well as histologic experiments involving healing after obturation show that it is preferable to confine instruments, irrigating solutions and obturating materials to the canal space. Thus we can say that predictable endodontic success demands accurate determination of working length of root canal and strict adherence to it, in order to create a small wound site and good healing conditions.

Historical Perspectives

- At the end of nineteenth century - Working length is usually calculated when file is placed in the canal and patient experiences pain.
- 1899 Kells - Introduced X-rays in dentistry
- 1918 Hatton - Microscopically studied the diseased periodontal tissues.
- 1929 Collidge - Studied the anatomy of root apex in relation to treatment problem.
- 1955 Kuttler - Microscopically investigated the root apices.
- 1962 Sunada - Found electrical resistance between periodontium and oral mucous membrane.
- 1969 Inove - Significant contribution in evolution of Electronic apex locator

Before we discuss various methods of determination of working length, we need to understand the anatomic consideration regarding it.

DEFINITIONS

According to endodontic glossary *working length* is defined as "the distance from a coronal reference point to a point at which canal preparation and obturation should terminate" (Fig. 10.1).

Reference point: Reference point is that site on occlusal or the incisal surface from which measurements are made. A

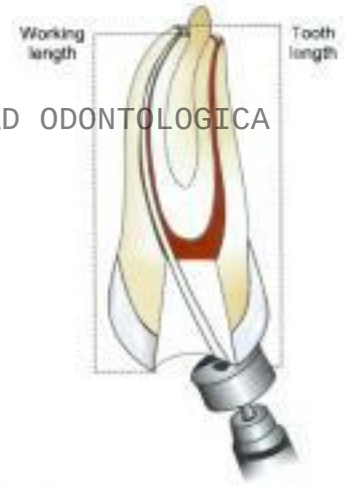


Fig. 10.1: Diagram showing the working length. (1 mm from the radiographic apex)

reference point is chosen which is stable and easily visualized during preparation. Usually this is the highest point on incisal edge of anterior teeth and buccal cusp of posterior teeth (Fig. 10.2). Reference point should not change between the appointments. For example in case of teeth with undermined cusps and fillings, they should be reduced considerably before access preparation.

Fig. 10.2: Usually the reference point is highest point on incisal edge of anterior teeth and cusp tip of posterior teeth

Anatomic apex is "tip or end of root determined morphologically."

Radiographic apex is "tip or end of root determined radiographically."

Apical foramen is main apical opening of the root canal which may be located away from anatomic or radiographic apex.

Apical constriction (minor apical diameter) is apical portion of root canal having narrowest diameter. It is usually 0.5 -1 mm short of apical foramen (Fig. 10.3). The minor diameter widens apically to foramen i.e. major diameter (Fig. 10.4).

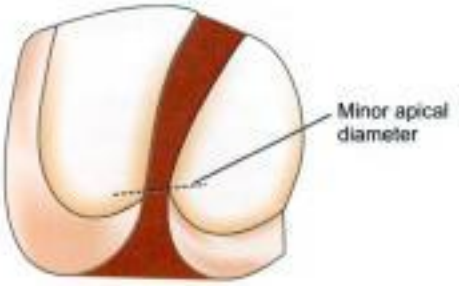
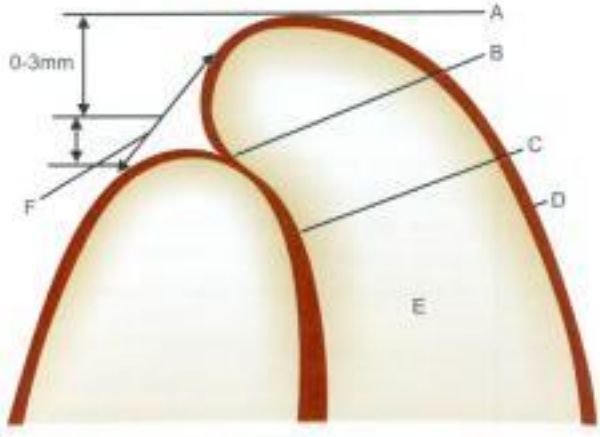


Fig. 10.3: Minor apical diameter



A	=	Root apex	B	=	Apical constriction
C	=	Root canal	D	=	Cementum
E	=	Dentine	F	=	Apical foramen

Fig. 10.4: Major apical diameter

The *cementodentinal junction* is the region where cementum and dentin are united, the point at which cemental surface terminates at or near the apex of tooth. It is not always necessary that CDJ always coincide with apical constriction. Location of CDJ ranges from 0.5 - 3 mm short of anatomic apex (Fig. 10.5).

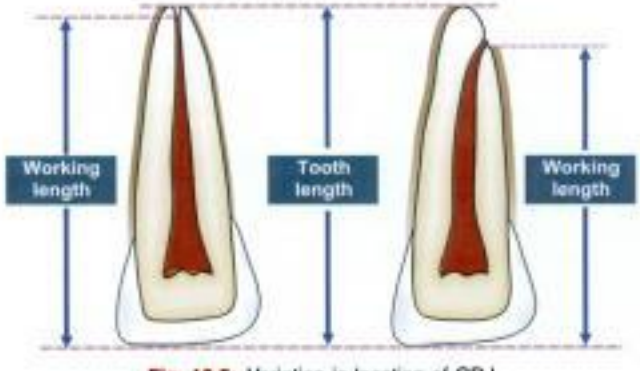


Fig. 10.5: Variation in location of CDJ

SIGNIFICANCE OF WORKING LENGTH

- Working length determines how far into canal, instruments can be placed and worked.
- It affects degree of pain and discomfort which patient will experience following appointment by virtue of over and under instrumentation.
- If placed in correct limits, it plays an important role in determining the success of treatment.
- Before determining a definite working length, there should be straight line access for the canal orifice for unobstructed penetration of instrument into apical constriction.
- Once apical stop is calculated, monitor the working length periodically because working length may change as curved canal is straightened.
- Failure to accurately determine and maintaining working length may result in length being over than normal which will lead to post operative pain, prolonged healing time and lower success rate because of incomplete regeneration of cementum, periodontal ligament and alveolar bone.
- When working length is made short of apical constriction it may cause persistent discomfort because of incomplete cleaning and underfilling. Apical leakage may occur into uncleaned and unfilled space short of apical constriction. It may support continued existence of viable bacteria and contributes to the periradicular lesion and thus poor success rate.

DIFFERENT METHODS OF WORKING LENGTH DETERMINATION

Various methods for determining working length include using average root lengths from anatomic studies, preoperative radiographs, tactile sensation, etc. Other common methods include use of paper point, working length radiograph, electronic apex locators or any combination of the above.

In this era of improved illumination and magnification, working length determination should be to the nearest 1.5 mm. So to achieve the highest degree of accuracy a combination of several methods should be used.

Different Methods of Determining Working Length

- Average root length from anatomic studies
- Radiographs
- Mathematics method
- Tactile sensation
- Bleeding on paper point
- Apical periodontal sensitivity
- Electronic apex locator

RADIOGRAPHIC METHOD OF WORKING LENGTH DETERMINATION

Radiographic apex has been used as termination point in working length determination since many years and it has showed promising results. But there are two school of thoughts regarding this:

Those who follow this concept say cementodentinal junction is impossible to locate clinically and the radiographic apex is the only reproducible site available for length determination. According to it, a patent root tip and larger files kept with in the tooth may result in excellent prognosis.

Those who don't follow this concept say that position of radiographic apex is not reproducible. Its position depends on number of factors like angulation of tooth, position of film, film holder, length of X-ray cone and presence of adjacent anatomic structures etc.

When radiographs are used in determining working length the quality of the image is important for accurate interpretations.

Among the two commonly used techniques, paralleling techniques have been demonstrated as superior to bisecting angle technique in determination and reproduction of apical anatomy.

*As the angle increases away from parallel, the quality of image decreases. This occurs because as the angle as increased, the tissue that X-rays must pass through include a greater percentage of bone mass and root anatomy becomes less apparent.

Parallel working length radiographs can be difficult to attain because of disorientation, shallow palatal vault and tori etc. But good film holders like Endo Ray II film holder may help in improving the results.

Before studying the X-rays for endodontics, understanding of buccal object rule is essential. The basic concept of the rule is that as the vertical or horizontal angulation of X-ray tube changes, the object buccal or closest to tube head moves to opposite side of radiograph compared to the lingual object.

To separate buccal and lingual roots (for example in maxillary first premolar) to visualize the working length, tube head should be moved from a 20° mesial angulation. This captures the buccal root to the opposite or distal side of radiograph and lingual root on mesial side of the radiograph. It is also known as SLOB rule that is same lingual opposite buccal. Although the individual canals can usually be distinguished by applying the SLOB rule and knowing the angle at which the radiographs was made,

misinterpretation is still possible. This can be reduced by using different types of files like K and H-files or different file sizes in different canals.

Radiographic method of length determination involves measurement of radiographic apex and then subtracting a specific value from that length.

Before access opening, fractured cusps, cusps weakened by caries or restorations are reduced to avoid fracture of weakened enamel during the treatment. This will avoid the loss of initial reference point and thus the working length (Fig. 10.6).

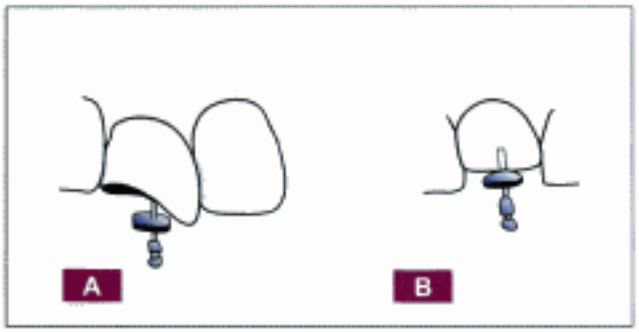


Fig. 10.6: (a) Reference point made at weakened or fractured tooth surface can cause error in working length (b) Weakened tooth surface is flattened to avoid errors

In this method, preoperative periapical radiograph is used to calculate the working length for endodontic treatment. OrthoPantograph (OPG) radiographs are not advocated for calculating tentative working length because of gross magnification of 13-28 percent employed in OPG which will lead to errors in calculation of accurate readings.

This measurement is used as estimated working length which can then be confirmed by placing an endodontic instrument into the canal and taking a second radiograph (Fig. 10.7).

The instrument inserted in the canal should be large enough not to be loose in the canal as it will move while taking the radiograph and thus may cause errors in determining the working length.

Fine instruments are often difficult to see in their entirely in a radiograph. The new working length is calculated by adding or subtracting the distance between the instrument tip and desired apical termination of the root.

The correct working length is finally calculated by subtracting 1 mm as safety factor from this new length. This technique was first introduced by John Ingle. Weine modified this subtraction rule (Fig. 10.8) as follows:

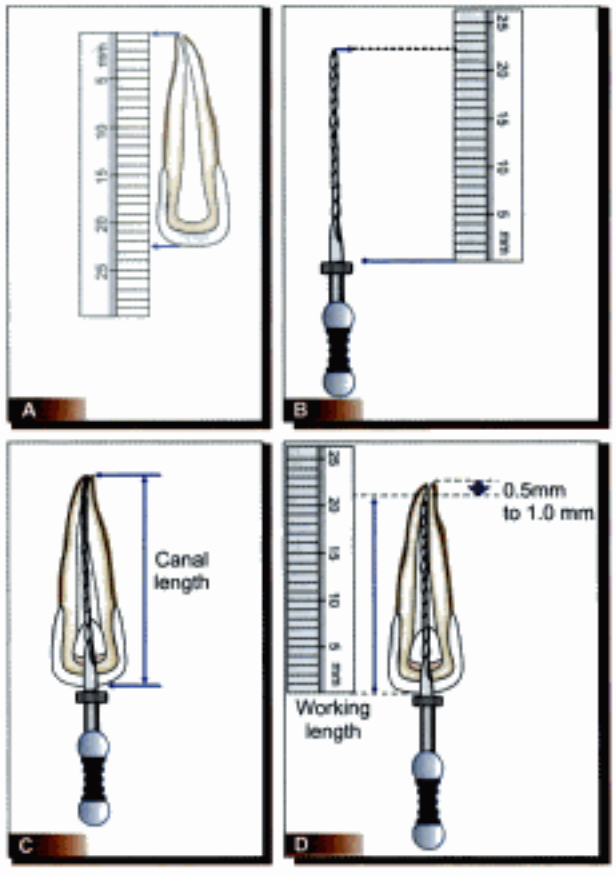


Fig. 10.7: Radiographic method of length determination

a. If radiograph shows absence of any resorption i.e. bone or root apex, shorten the length by 1 mm (Fig. 10.8A).
b. If periapical bone resorption is present, shorten it by 1.5 mm (Fig. 10.8B).
c. If both bone and root resorption is seen, shorten it by 2 mm. This is done because if there is root resorption, loss of apical constriction may occur in such cases (Fig. 10.8C).

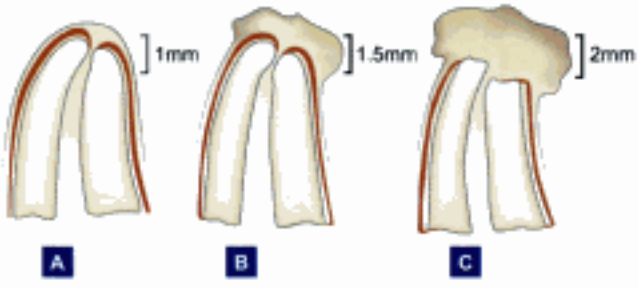


Fig. 10.8: Modification of length substraction in case of resorption

In curved canals, canal length is reconfirmed because final working length may shorten up to 1 mm as canal is straightened out by instrumentation. If root contains two canals, the tube should be bent to a 20 to 30 degree horizontal deviation from the standard facial projection.

Radiographic Method of Length Determination

1. Measure the estimated working length from preoperative periapical radiograph.
2. Adjust stopper of instrument to this estimated working length and place it in the canal up to the adjusted stopper.
3. Take the radiograph.
4. On the radiograph measure the difference between the tip of the instrument and root apex. Add or subtract this length to the estimated working length to get the new working length.
5. Correct working length is finally calculated by subtracting 1 mm from this new length.

Modification in the Length Subtraction (Fig. 10.8)

1. No resorption - subtract 1 mm
2. Periapical bone resorption - subtract 1.5 mm
3. Periapical bone + root apex resorption - subtract 2 mm

MATHEMATIC METHOD OF WORKING LENGTH DETERMINATION

It is based on simple mathematical formulations to calculate the working length. In this, an instrument is inserted into the canal, stopper is fixed to the reference point and radiograph is taken. The formula to calculate actual length of the tooth is as follows:

$$\frac{\text{Actual length of the tooth}}{\text{Actual length of the instrument}} = \frac{\text{Apperent length of tooth in radiograph}}{\text{Apparent length of instrument in radiograph}}$$

By above, as we see those three variables are known and by applying the formula, 4th variable i.e. actual length of tooth can be calculated.

$$\text{Actual length of tooth} = \frac{\text{Actual length of the instrument} \times \text{Apparent length of tooth in radiograph}}{\text{Apparent length of instrument in radiograph}}$$

Disadvantages

1. Wrong readings can occur because of:
   a. Variations in angles of radiograph
   b. Curved roots
   c. S-shaped, double curvature roots

Other methods of working length determination include employing ***tactile sense by instrument***, ***apical periodontal sensitivity*** and ***using paper point measurement*** etc.

But these methods don't always provide the accurate readings, for example in case of narrow canals, instrument may feel increased resistance as file approaches apical 2-3 mm and in case of teeth with immature apex instrument can go beyond apex. Similarly while performing apical periodontal sensitivity test in cases of canal with necrotic pulp, instrument can pass beyond apical constriction and in case of vital or inflamed pulp, pain may occur several mm before periapex is crossed by the instrument. These methods should not be used alone for determining working length, they should be used as supplement to radiographs apex locators etc.

ELECTRONIC APEX LOCATORS

Radiographs are often misinterpreted because of difficulty in distinguishing the radicular anatomy and pathosis from normal structures. Electronic apex locators (EAL) are used for determining working length as an adjunct to radiography. They are basically used to locate the apical constriction or cementodentinal junction or the apical foramen, and not the radiographic apex. Hence the term apex locator is a misnomer one.

The ability to distinguish between minor diameter and major diameter of apical terminus is most important for the creation of apical control zone (Fig. 10.9). ***The apical control zone*** is the mechanical alteration of the apical terminus of root canal space which provides resistance and retention form to the obturating material against the condensation pressure of obturation. Various studies have shown that electronic apex locators have provided more accurate results when compared to conventional radiographs.

History of EALs goes back to 1918, when Custer first reported the use of electronic current to determine working length. In 1960's, Gordon reported the use of a clinical device for electrical measurement of root canals.

In 1942, Suzuki's discovered that electrical resistance between an instrument inserted into root canal and an electrode attached to oral mucosa registered a constant value.

In 1962, Sunada using a direct current device with simple circuit demonstrated that consistent electrical resistance between periodontium and mucous membrane was 6.5 K ohms (Fig. 10.10).

In 1970's, frequency measurements were taken through the feedback of an oscillator loop by calibration at periodontal pocket depth of each tooth.

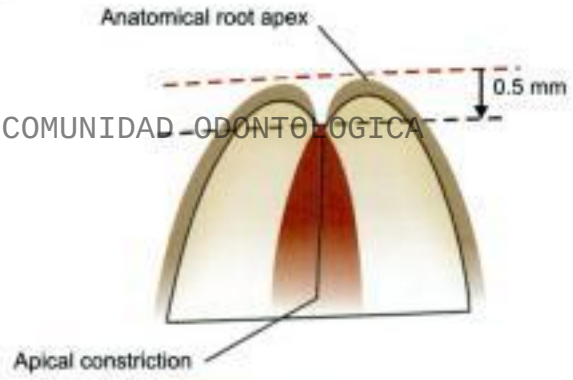


Fig. 10.9: Location of CDJ

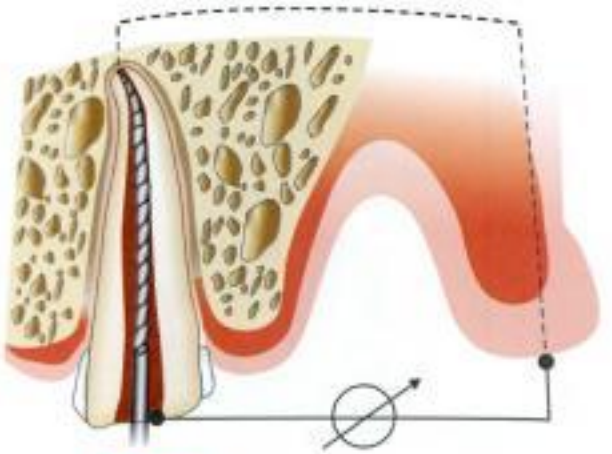


Fig. 10.10: Diagramatic representation of working of resistance type of apex locator

In mid 80's, there occurred the development of a relative value of frequency response method where apical constriction was picked by filtering the difference between two direct potentials after 1 KHz wave was applied to canal space.

A third generation electronic apex locator was developed in late 80's by Kobayashi. He used multiple channel impedance ratios based technology to simultaneously measure the impedance of two different frequencies; calculate the quotient of impedance and express it in terms of the position of electrode i.e. file in the canal.

Historical Review of EALs

1918 -	Custer -	Use of electric current for working length
1942 -	Suzuki -	Conducted scientific study of apex locator
1960's -	Gordon -	Use of clinical device for measure-ment of length.
1962 -	Sunada -	Found electrical resistance between periodontium and oral mucous membranes
1969 -	Inove -	Significant contribution in evolution of EAL
1996 -	Pratten and McDonald	Compared the efficacy of three parallel radiographs and Endex apex locators in cadaver.

Thus we can say that all of these apex locators function by using human body to complete an electrical circuit. One side of apex locator circuit is connected to endodontic instrument and other side is connected to patient's body. Circuit is completed when endodontic instrument is advanced into root canal until it touches the periodontal tissue.

Classification of EALs

This classification is based on type of the current flow and opposition to current flow as well as number of frequencies involved. Following classification is modification of classification given by Mc Donald [DCNA 1992; 36:293].

First Generation Apex Locators: (Resistance Apex Locators)

They are also known as resistance apex locators which measure opposition to flow of direct current i.e. resistance. It is based on the ***principle*** that resistance offered by periodontal ligament and oral mucous membrane is the same, i.e. 6.5 k ohms. Initially ***Sono - Explorer*** was imported from ***Japan*** by ***Amadent***, but now a days first generation apex locators are off the practice. Blood, pus, chelating agents, irrigants and other materials used within the canal can give false readings.

Technique for Using Resistance Based EAL

1. Turn on the device and attach the lip clip near the arch being treated. Hold a 15 number. file and insert it approx 0.5 mm into sulcus of tooth (like PD probe). Adjust the control knob until the reference needle is centered on the meter scale and produces audible beeps. Note this reading.
2. Prepare the access cavity and apply rubber dam and remove pulp, debris etc.
3. Using preoperative radiograph estimate the working canal width. Clean the canal if bleeding form vital pulp is excessive, dry it with paper points.
4. Insert the file into canal unless the reference needle moves from extreme left to centre of scale and alarm

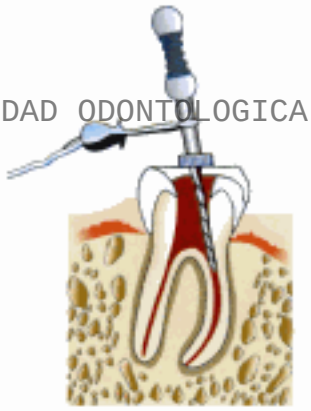


Fig. 10.11A: File is being introduced into the canal

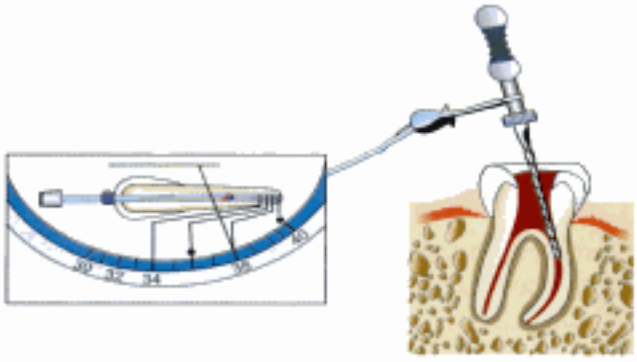


Fig. 10.11B: Steady increase in reading of electrical resistance

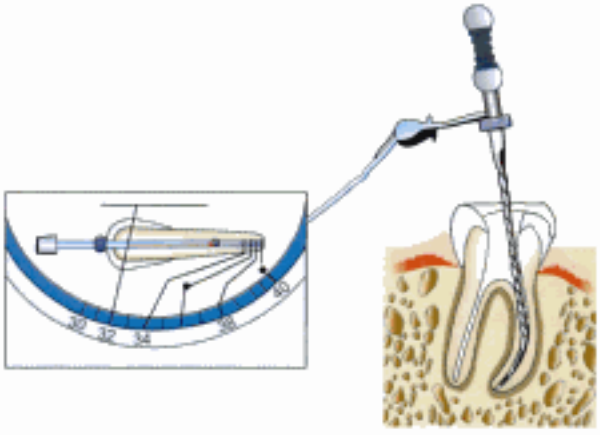


Fig. 10.11C: Reading showing that file has reached the apical foramina

beeps sound. Reset the stop at reference point and record the lengths (Figs 10.11A to C).

5. Take the radiograph with file in place at the length indicated by apex locator. If length is longer / shorter, it is possible that preoperative film can be elongated or apex locator is inaccurate.

Advantage

1. It is possible to get objective information with high degree of accuracy.
2. Used where radiographs can't be used accurately like
   a. Maxillary molars (due to zygomatic process).
   b. Mandibular molars (obstruction due to mandibular tori).
3. Patient with gag reflex also benefit from apex locator.

Disadvantages

Many factors can lead to wrong readings like low battery, tissue present in canal, wet canal, too narrow canal or canal with blockage and problems of lip clip.

Second Generation Apex Locators (Impedence Apex Locators)

They are also called as ***impedance apex locator*** which measure opposition to flow of alternating current or impedance. A major ***disadvantage*** of second generation apex locators is that root canal has to be free of electro-conductive materials to obtain accurate readings. Presence of electro-conductive irrigants changes the electric characteristics and lead to inaccurate readings. Various second generation apex locators are given below.

Various Second Generation Apex Locators

- Sono explorer
- The apex finder (has digital LED indicator and is self calibrating)
- Endo analyzer (combination of apex locator and pulp tester)
- Digipex (has digital LED indicator but requires calibration)
- Digipex II (combination of apex locators and pulp tester)
- Formation IV (digital LED light and display, self calibrating).

Third Generation Apex Locator

They are also called ***frequency dependent apex locator***. They are based on the fact that different sites in canal give difference in impedance between high (8KHz) and low (400Hz) frequencies. The difference in impedance is least in the coronal part of canal. As the probe goes deeper into canal, difference increases. It is the greatest at cemento-dentinal junction.

Since impedance of a given circuit may be substantially influenced by the frequency of current flow, these are also known as frequency dependent. More appropriately, they should be termed as "Comparative Impedance" because they measure relative magnitudes of impedance which are converted into length information.

Endex is original third generation apex locator which was described by Yamaoka et al. It uses two frequencies 5 and 1 KHz. As the instrument is moved apically, difference between the two signals is measured. Some of the available third generation apex locators are mentioned in below:

Various Third Generation Apex Locators	
Endex	Original 3rd generation apex locator
Neosomo Ultimo EZ apex locator	Apex locator with pulp tester
Mark V plus	Apex locator with pulp tester
Root Zx	Shaping and cleaning of root canals with simultaneous monitoring of working length
Combination of Apex Locator and Endodontic Handpiece	
Tri Auto Zx	Cordless electrical handpiece with three safety mechanism
Endy 7000	Reverses the rotation when tip reaches apical constriction
Sofy Zx	Monitor the location of file during instrumentation

Fourth Generation Apex Locators

Recently fourth generation electronic apex locators have been developed which measure resistance and capacitance separately rather than the resultant impedance value. There can be different combination of values of capacitance and resistance that provides the same impedance, thus the same foraminal reading. But by using fourth generation apex locator, this can be broken down into primary components and measures separately for better accuracy and thus less chances of occurrence of errors.

Though it is claimed to provide high accuracy but more studies are required for its confirmation.

Combination Apex Locators and EndodonticHandpiece

Tri Auto ZX (J. Morita Calif) is cordless electric endodontic handpiece with built in root ZX apex locator. It has three safety mechanisms (Fig. 10.12).

Autostart stop mechanism: Handpiece starts rotation when instrument enters the canal and stops when it is removed.

Auto torque reverse mechanism: Handpiece automatically stops and reverses rotation when torque threshold (30 gm/cm) is exceed. It prevents instrument breakage.

Auto apical-reverse mechanism: It stops and reverses rotation when instrument tip reaches a distance from apical

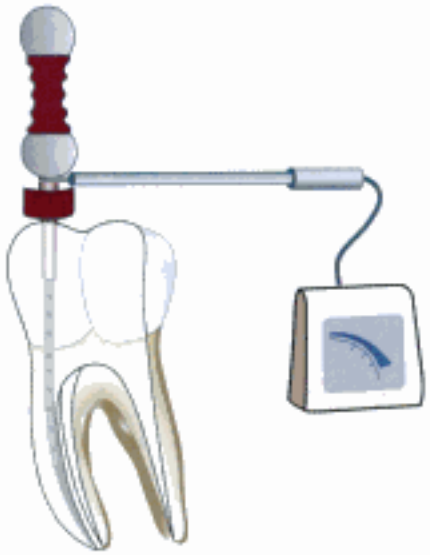


Fig. 10.12: Diagrammatic representation of use of Root Zx apex locator

constriction taken for working length. It prevents apical perforation. Endy 7000 reverse the rotation when tip reaches the apical constriction. Sofy Zx (J. Morita Calif) uses Root Zx to electronically monitor the location of file tip during whole of the instrumentation procedure.

Basic Conditions for Accuracy of EALs

Whatever is the generation of apex locator; there are some basic conditions, which ensure accuracy of their usage.

1. Canal should be free from most of the tissue and debris.
2. The apex locator works best in a relatively dry environment. But extremely dry canals may result in low readings i.e. long working length.
3. Cervical leakage must be eliminated and excess fluid must be removed from the chamber as this may cause inaccurate readings.
4. If residual fluid is present in the canal, it should be of low conductivity value, so that it does not interfere the functioning of apex locator.

The descending order of conductivity of various irrigating solutions is:
5.25 percent NaOCl > 17 percent EDTA > Saline.

5. Since EALs work on the basis of contact with canal walls and periapex. The better the adaptation of file to the canal walls, the more accurate as the reading.

6. Canals should be free from any type of blockage, calcifications etc.
7. Battery of apex locator and other connections should be proper.

Basic Conditions for Accuracy of EAL

- Canal should be free from debris
- Canal should be relatively dry.
- No cervical leakage
- Proper contact of file with canal walls and periapex
- No blockages or calcifications in canal

Uses of Apex Locators

1. They provide objective information with high degree of accuracy.
2. They are useful in conditions where apical portion of canal system is obstructed by:
   a. Impacted teeth
   b. Zygomatic arch
   c. Tori
   d. Excessive bone density
   e. Overlapping roots
   f. Shallow palatal vault.

   In such cases, they can provide information which radiographs cannot.
3. They are useful in patient who cannot tolerate X-ray film placement because of gag reflex.
4. In case of pregnant patients, to reduce the radiation exposure, they can be valuable tool.
5. They can also be used in children who may not tolerate taking radiographs, disabled patients and patients who are heavily sedated.
6. They are valuable tool for:
   a. Detecting site of root perforations (Fig. 10.13)
   b. Diagnosis of external and internal resorption which have penetrated root surface
   c. Detection of horizontal and vertical root fracture
   d. Determination of perforations caused during post preparation
   e. Testing pulp vitality
7. They are helpful in root canal treatment of teeth with incomplete root formation, requiring apexification and to determine working length in primary teeth.

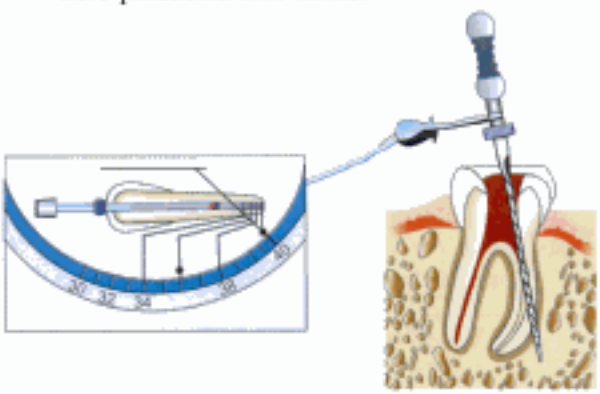


Fig. 10.13: Sudden rise in reading indicates perforation

Uses of Apex Locators

1. Provide objective information with high degree of accuracy
2. Useful in conditions where apical portion is obstructed by anatomic structures such as zygomatic arch, tori, exostoses etc.
3. Useful in patients with gag reflex
4. Pregnant patients
5. Root perforations, resorptions, root fracture
6. Pulp vitality
7. RCT of teeth with incomplete root formation

Advantages of Apex Locators (EAL)

- Accurate
- Objective measurements
- Easy and fast
- Reduction of exposure to radiation
- Perforations can be detected
- Can measure pulp space exactly to the constriction
- Can detect resorption and root fracture

Contraindications to the Use of Apex

Apex locators are contraindicated in the patients who have cardiac pacemaker functions. Electrical stimulation to such patients can interfere with pace maker function.

It has been shown that in teeth with periapical radiolucencies, and necrotic pulps associated with root resorption etc. the use of apex locators is not much beneficial. In such cases there is alteration of apical constriction and lack of viable periodontal ligament tissue to respond to EAL which may cause abnormally long readings.

SUMMARY

The most important thing to understand when determining working length is morphology of apical one third of the canal. The consideration should be given to adopt the

parameters of 0.5 - 0.0 mm (from the apical constriction) as most ideal terminating point in canal. One should use as many of techniques as possible during the course of treatment. First of all there should be a stable coronal reference point. Next step is to estimate working length from average anatomical length and preoperative radiograph. Finally calculate the correct working length using combination of various mentioned techniques i.e. tactile sense, radiography, electronic apex locators etc. Multiple measurements should be taken to determine accurate readings of working length. With so many advances coming up in the branch of endodontics we expect the future of apex locators to provide accurate readings in all the conditions of root canal without the need of calibrations.

11 Irrigation and Intracanal Medicaments

- Introduction
- Properties of an Irrigating Solution
- Functions of Irrigants
- Commonly used Irrigating Solutions
- Factors Modifying Action of Irrigating Solution
- Sodium Hypochlorite
- Urea
- Hydrogen Peroxide
- Urea Peroxide
- Chlorhexidine
- Chelating Agents
- Ultrasonic Irrigation
- Newer Irrigating Solutions
- Method of Irrigation
- Intracanal Medicaments
- Commonly used Intracanal Medicaments
- Essential Oils
- Phenolic Compounds
- Calcium Hydroxide
- Halogens
- Chlorhexidine Gluconate
- Antibiotics
- Corticosteroid-antibiotic Combination

INTRODUCTION

During the past 20 years, endodontics has begun to appreciate critically the important role of irrigation in successful endodontic treatment. The objective of endodontic treatment is to prevent or eliminate infection within the root canal. Over the years, research and clinical practices have concentrated on instrumentation, irrigation and medication of root canal system followed by obturation and the placement of coronal seal. It's truly said, "Instruments shape, irrigants clean". Every root canal system has spaces that cannot be cleaned mechanically. The only way we can clean webs, fins and anastomoses is through the effective use of an irrigation solution (Fig. 11.1). In order to get maximum efficiency from the irrigant, irrigant must reach the apical portion of the canal. Irrigation is an important part of root canal treatment because it assists us in (a) removing bacteria and debris (b) configuring the system so that it can be obturated to eliminate dead space. It has been found that use of saline as an irrigant before and after instrumentation, markedly resulted in a 100 to 1,000 fold reduction in bacterial counts (Bystrom et al; 1981).

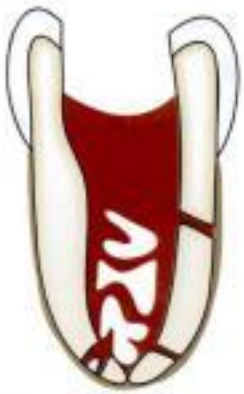


Fig. 11.1: Complicated root canal system

An ideal irrigant solution must fulfill the following criteria:

1. It must have broadspectrum antimicrobial properties.
2. It must aid in the debridement of the canal system.

3. It should have the ability to dissolve necrotic tissue or debris.
4. It should have low toxicity level.
5. It should be a good lubricant.
6. It should have low surface tension so that it can easily flow into inaccessible areas.
7. It should be able to effectively sterilize the root canal (or at least disinfect them).
8. It should be able to prevent formation of smear layer during instrumentation or dissolve the latter once it is formed.
9. It should inactivate endotoxin.

Other desirable properties of ideal irrigants include availability, cost, ease of use, convenience, adequate shelf life and ease of storage. In addition to these properties, if endodontic irrigants come in contact with vital tissue, these should be systemically nontoxic, noncaustic to the periodontal tissue and have little potential to cause an anaphylactic reaction.

Properties of ideal irrigant solution

1. Broad spectrum antimicrobial properties.
2. Aid in debridement of the root canal system.
3. Ability to dissolve necrotic tissue or debris.
4. Low toxicity level.
5. Good lubricant.
6. Low surface tension to flow into inaccessible area.
7. Ability to sterilize the canal.
8. Prevent/dissolve smear layer.
9. Inactivate endotoxin.

FUNCTIONS OF IRRIGANTS

1. Irrigants perform physical and biologic functions. Dentin shavings get removed from canals by irrigation (Fig. 11.2). Thus, they do not get packed at the apex of root canal (Fig. 11.3).
2. Instruments do not work properly in dry canals. Their efficiency increases by use in wet canals. Instruments are less likely to break when canal walls are lubricated with irrigation.
3. Irrigants act as solvent of necrotic tissue, so they loosen debris, pulp tissue and microorganisms from irregular dentinal walls (Fig. 11.4).
4. Irrigants help in removing the debris from accessory and lateral canals where instruments cannot reach.
5. Most irrigants are germicidal but they also have antibacterial action.
6. Irrigants also have bleaching action to lighten teeth discolored by trauma or extensive silver restorations.

Fig. 11.2: Dentin shavings packed at apical third

Fig. 11.3: Use of irrigating syringe to remove debris

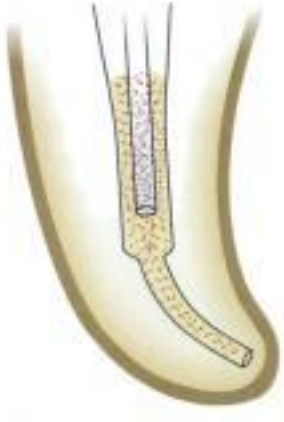


Fig. 11.4: Irrigation helps in loosening of debris

7. Though presence of irrigants in canal facilitate instrumentation but simultaneous use of some lubricating agents (RC prep, REDTAC, Glyde, etc.) make the instrumentation easier and smoother.

Functions of irrigants

1. Perform physical and biologic functions-remove dentinal shavings by irrigation.
2. Contain the debris in solution.
3. Dissolve necrotic tissue.
4. Remove debris from lateral and accessory canals.
5. Germicidal as well as antibacterial properties.
6. Bleaching action.
7. Irrigants with lubricating agent further increase the efficiency.
8. Opening of dentinal tubules by removal of smear layer.

FACTORS THAT MODIFY ACTIVITY OF INTRACANAL IRRIGATING SOLUTIONS

It is clear that there are several factors associated with the efficacy of the agents used. Some modifying factor such as host resistance, bacterial virulence, microbial resistance or susceptibility, etc. are beyond our control.

Other factors which can be controlled or at least predicted, such factors are:

1. ***Concentration:*** Several studies have revealed that the tissue dissolving ability of sodium hypochlorite is greater at a concentration of 5.2 percent than at 2.5 percent and 0.5 percent. But it has also been clearly demonstrated that higher concentrations are more cytotoxic than lower concentrations. This direct relationship of effectiveness and toxicity to concentration is generally true for all intracanal agents.
2. ***Contact:*** To be effective, the intracanal agent must contact the substrate (i.e. organic tissue or microbes). In case of an irrigating solution, it neither dissolves nor physically flushes out the debris if it is not in contact with the substrate. So, it is critical that the canals should be mechanically enlarged to carry the solution to the apical extent of canal preparation. When the canals are sufficiently enlarged, the solution can be deposited directly in the apical area of the preparation with a fine irrigating needle (Fig. 11.3).
3. ***Presence of organic tissue:*** Presence of the organic tissues decreases the effectiveness of intracanal medicaments, so the organic tissue must be removed mechanically or chemomechanically by simultaneous use of instruments and irrigating solutions so that medications can therefore be used.

   If organic debris are present in root canal space, its protein content will coagulate as a result of its reaction with the medicament. This coagulation serves as a barrier to further penetration of medicament, thus limiting its effectiveness.
4. ***Quantity:*** Baber et al proved that ability of solution to debride was directly related to the quantity of irrigating solution. In case of medicament, increase in quantity results in increase in cytotoxicity.
5. ***Temperature:*** It is shown in studies that if sodium hypochlorite is warmed before irrigation, it is much (60-70°C) more effective as a tissue solvent .

Factors modifying the activity of irrigating solutions

1. *Concentration:* Tissue dissolving capability of NaOCl is higher at 5.2%.
2. *Contact:* To effective, irrigant must come in contact with the substrate
3. *Presence of organic tissue:* Organic tissues must be removed for effective irrigation
4. *Quantity:* Increase in quantity increases the effectiveness
5. *Temperature:* Warming the NaOCl increases its efficacy.

CHOICE OF MAIN IRRIGANT SOLUTION

Currently, there is no single irrigant that can fulfill all of these criteria and so we have to rely on different irrigating solutions and sometimes their combination. The main irrigants include sodium hypochlorite, chlorhexidine and ethylene diamine tetraacetic acid. Unfortunately, this does not seem to be one clear regimen that should be followed to maximize the benefits of each of these materials.

COMMONLY USED IRRIGATING SOLUTIONS

Chemically non-active solution

- Water
- Saline
- Local anesthetic

Chemically active materials:

- Alkalis: Sodium hypochlorite 0.5-5.25%
- Chelating agents: ethylene diamine tetra acetic acid (EDTA)
- Oxidizing agents: hydrogen peroxide, carbamide peroxide
- Antibacterial agents: chlorhexidine, Bisdequalinium acetate
- Acids: 30% hydrochloric acid
- Enzymes: streptokinase, papain, trypsin
- Detergents: sodium laruyl sulphate

Sodium Hypochlorite

Sodium hypochlorite is a clear, pale, green-yellow liquid with strong odor of chlorine (Fig. 11.5). It is easily miscible with water and gets decomposed by light.

Fig. 11.5: Sodium hypochlorite

Historical Review

1. Sodium hypochlorite was first introduced during the World War I by chemist **Henry Drysdale Dakin** for treating infected wounds. It is also known as **"Dakin's solution"**. The original concentration suggested by Dakin was 0.5 percent but concentration commonly used in practice is 5.25 percent.
2. Walker in 1936 first suggested its use in root canal therapy.
3. Grossman in 1941 demonstrated the tissue dissolving ability of Chlorinated soda when used double strength and recommend its use as intracanal medicament (Double strength chlorinated soda is same as 5 percent NaOCl).
4. Spangberg in 1973 said that 0.5 percent NaOCl was sufficient to provide germicidal activity but was les toxic to tissue culture than 5 percent NaOCl.
5. Madden in 1977 compared 5, 2.5 and 0.5 percent NaOCl and found that 5 percent and 2.5 percent solution was better than 0.5 for tissue dissolving.
6. Foley et al in 1983 tested effectiveness of 0.5 percent NaOCl and Glyoxide and concluded that both can effectively kill Bacteriod melaninogenicus and Peptostreptococci.
7. Kozol et al concluded that Sodium hypochlorite was detrimental to neutrophilic chemotaxis and toxic to fibroblasts and endothelial cells.

1. Introduced in World War Ist by Dakin.
2. Walker-1936—first suggested its use in root canal therapy.
3. Grossman-1941—used it as an intracanal medicament.
4. Spangberg-1973-0.5%—NaOCl has good germicidal activity.
5. Madden-1977—compared the different concentrations of Sodium hypochlorite.
6. Foley et al-1983—compared effectiveness of 0.5% NaOCl and Glyoxide.

Mechanism of Action of Sodium Hypochlorite

Sodium hypochlorite contains 5 percent of free chlorine which is important for breakdown of proteins into amino groups. At body temperature, reactive chlorine in aqueous solution exists in two forms-hypochlorite (OCl^-) and hypochlorous acid (HOCl). State of available chlorine depends on pH of solution, i.e. above pH of 7.6, mainly hypochlorite form and below this hypochlorous acid.

The pH of commonly used sodium hypochlorite is 12, at which the OCl form exits. But as we have seen that hypochlorous acid is more bactericidal, so to increase the efficacy of NaOCl solution, 1 percent sodium bicarbonate is added as buffering agent. Buffering makes the solution unstable, thus decreases its shelf life to even less than one week. Buffered and diluted sodium hypochlorite should be stored in dark and cool place.

Methods by which we can increase the efficacy of sodium hypochloride are:

1. *Time:* Since antimicrobial effectiveness of sodium hypochlorite is directly related to its contact time with the canal, greater the contact time, more effective it is. This is especially important in necrotic cases.
2. *Heat:* It has been shown that warming sodium hypochlorite to 60-70°, increases its solvent properties and tissue dissolving properties (Fig. 11.6). But one should be careful not to overheat the solution because this can cause breakdown of sodium hypochlorite constituents and thus may damage the solution.
3. *Specialized irrigating syringes:* Most researches have shown that unaided irrigation requires at least a size #25 apex for it to reach the more apical portions of canals. Newer specialized side venting endodontic syringes with narrower diameter (32 gauge) are available which aid in getting irrigant closer to apex and help the irrigant to move sideways (Fig. 11.7).

Fig. 11.6: To warm NaOCl, syringes filled with NaOCl are placed in 60-70°C 140°F water bath

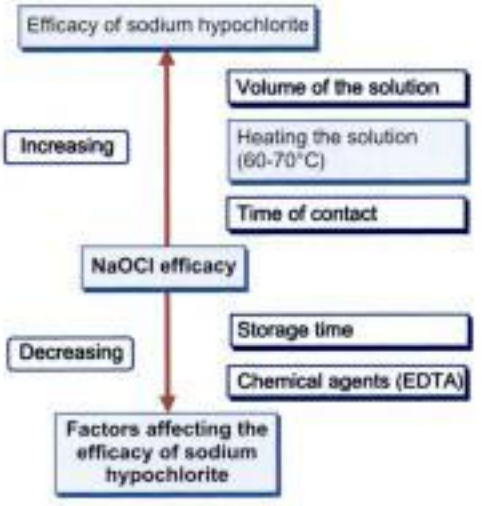


Fig. 11.7: Needle with side venting helps to move the irrigant sidways in whole canal

Fig. 11.8: Ultrasonic activation of irrigating solution

4. *Ultrasonic activation of* sodium hypochlorite has also shown to accelerate chemical reaction, create cavitational effect and thus achieve a superior cleansing action (Fig. 11.8). But it is seen that if ultrasonic activation of sodium hypochlorite is used, it is better to apply ultrasonic instrument after canal preparation. A freely oscillating instrument causes better effect than the instrument which binds to canal walls.

Also the ultrasonic files cause more uncontrolled cutting of canal walls especially if used during preparation. Thus use of a noncutting instrument after canal preparation is best option for optimal effects.

Precautions to be Taken While Using Sodium Hypochlorite Solution

It is important to remember that though sodium hypochlorite is nontoxic during intracanal use but 5.25 percent NaOCl can cause serious damage to tissue if injected periapically (Fig. 11.9).

If sodium hypochlorite gets extruded into periapical tissues, it causes excruciating pain, periapical bleeding and swelling. As potential for spread of infection is related to tissue destruction, medication like antibiotics, analgesics, antihistamine should be prescribed accordingly. In addition to these, reassurance to the patient is the prime consideration.

Fig. 11.9: Forceful irrigation can cause periapical extrusion of sodium hypochlorite solution

Thus irrigation with sodium hypochlorite solution should always be performed passively especially in cases with larger apical diameters and needles with very small diameter, also the syringe should never be locked in the canal.

Use of Sodium Hypochlorite in Combination with other Medicaments

Many studies have shown that efficacy of sodium hypochlorite as antimicrobial agent is increased when it is used in combination with other solutions such as calcium hydroxide, EDTA or chlorhexidine.

The tissue dissolving capacity of sodium hypochlorite or chlorhexidine is found to be increased when tissue is pretreated with calcium hydroxide (Hasselgren et al).

Wadachi et al in their study have shown that combination of calcium hydroxide and sodium hypochlorite was better than either of medicament alone. Various studies have shown that combination of sodium hypochlorite and EDTA has more bactericidal effect which is probably due to removal of contaminated smear layer by EDTA.

The alternate use of sodium hypochlorite and chlorhexidine results in greater reduction of microflora than the use of either alone as shown by ***Kuruvilla and Kamath***.

UREA

It is a white, odorless, crystalline powder. It was used in World War first as a therapeutic agent for infected wounds. Urea solution (40% by weight) is mild solvent of necrotic tissue and pus and is mild antiseptic too. In 1951, Blechman and Cohen suggested that 30 percent urea solution can be used as root canal irrigant in patients with vital pulp as well as those with necrotic pulp.

Mechanism of Action

1. *Denaturation of protein:* Urea denatures the protein by destroying bonds of the secondary structure resulting in loss of functional activity of protein. This mode of action is responsible for its antiseptic property.
2. It has the property of chemically debriding the wound by softening the underlying substrate of fibrin.

Uses

1. It is excellent vehicle for antimicrobials such as sulfonamides.
2. It has low toxicity and so, it can be used in patients where vital uninfected pulp has been removed.
3. It can be used in open apex or in areas of resorptive defects.

HYDROGEN PEROXIDE

It is clear, odorless liquid. It is mainly the 3 percent solution which is used as an irrigating agent (Fig. 11.10).

Mechanism of Action

1. It is highly unstable and easily decomposed by heat and light. It rapidly dissociates into H_2O + [O] (water and nascent oxygent). While on contact with tissue enzymes catalase and peroxidase, the liberated [O] has bactericidal effect but this effect is transient and diminishes in presence of organic debris.
2. It causes oxidation of bacterial sulfhydryl group of enzymes and thus interferes with bacterial metabolism.

Fig. 11.10: Hydrogen peroxide

3. The rapid release of [O] nascent oxygen on contact with organic tissue results in effervescence or bubbling action which is thought to aid in mechanical debridement by dislodging particles of necrotic tissue and dentinal debris and floating them to the surface.

Use

It is used as an irrigating solution either alone or alternatively with sodium hypochlorite. The advantage of using alternating solutions of 3 percent H_2O_2 and 5.2 percent NaOCl are:

1. Effervescent reaction by Hydrogen peroxide bubbles pushes debris mechanically out of root canal.
2. Solvent action of sodium hypochlorite on organic debris.
3. Disinfecting and bleaching action by both solutions.

Always use sodium hypochlorite last because Hydrogen peroxide can react with pulp debris and blood to form gas (nascent oxygen) which builds up pressure on closing the tooth and causes pain.

UREA PEROXIDE

It is white crystalline powder with slight odor. It is soluble in water, alcohol and glycerine.

Mechanism of Action

It decomposes rapidly when exposed to heat, light or moisture. It dissociates into urea and hydrogen peroxide.

$$\text{Urea peroxide} \rightarrow \text{Urea} + H_2O_2$$

Its mechanism of action combines the effects of urea and hydrogen peroxide.

2. The anhydrous glycerol increases the stability of urea peroxide.

Use

1. 10 percent solution of urea peroxide in anhydrous glycerol base is commonly available as (Glyoxide).The advantage of adding glycerol are:
   i. It increases the stability of solution, thus increases shelf life.
   ii. It acts as a good lubricant, so facilitates negotiation and instrumentation of thin, tortuous root canals.
   iii. Glyoxide can be used along with EDTA to clean the walls of the canal.

Disadvantages

1. It dissociates more slowly than hydrogen peroxide (H_2O_2). So, its effervescence is prolonged but not as pronounced. This can be overcome by alternating irrigation with sodium hypochlorite.

CHLORHEXIDINE

Chlorhexidine was developed in the late 1940's in the research laborites. Chlorhexidine was the most potent of the tested bisbiguanides. It has strong base and is most stable in the form of its salts, i.e. chlorhexidine gluconate. It is a potent antiseptic which is widely used for chemical plaque control in the oral cavity in concentrations of 0.2 percent while 2 percent is the concentration, used for irrigating root canal.

Mechanisms of Action

Chlorhexidine is broad spectrum antimicrobial agent. The antibacterial mechanism of chlorhexidine is related to its cationic bisbiguanide molecular structure. The cationic molecule is absorbed to the negatively charged inner cell membrane and cause leakage of intracellular components. At low concentration, it acts as a bacteriostatic, while at higher concentrations; it will cause the coagulation and precipitation of cytoplasm and therefore, is bactericidal.

In addition, chlorhexidine has the property of substantivity (residual effect). Both 2 and 0.2 perent chlorhexidine can cause residual antimicrobial activity for 72 hours, if used as an endodontic irrigant.

Advantages and Uses

1. A 2 percent solution is used as root irrigant in canals.
2. A 0.2 percent solution can be used in controlling plaque activity.
3. It is more effective on gram-positive bacteria than gram-negative bacteria.

Disadvantages

1. It is not considered as the main irrigant in standard endodontic therapy.
2. It is unable to dissolve necrotic tissue remnants.
3. It is less effective on gram-negative than on gram-positive bacteria.

CHELATING AGENTS

After canals are instrumented, an organic layer remains which covers the dentinal tubules. Controversies still exist whether to keep or to remove smear layer as it relates to permeability of dentin. However most of studies have

Fig. 11.11: Chelating agent

recommended removal of smear layer because it is the source of microorganisms and also the closest possible adaptation of endodontic filling is possible only after its removal.

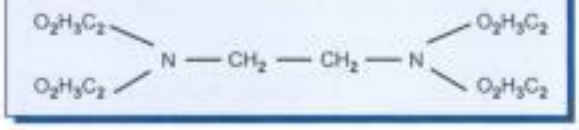
Though sodium hypochlorite is thought to be almost ideal irrigating solution but it does not possess chelating properties. EDTA and other chelating agents like citric acid, polyacrylic acids are used for this purpose (Fig. 11.11).

Chelating agent is defined as a chemical which combines with a metal to form chelate.

EDTA is most commonly used chelating agent. It was introduced in dentistry by Nygaard Ostby for cleaning and shaping of the canals.

The basic purpose of a chelator is lubrication, emulsification and holding debris in suspension. EDTA functions by forming calcium chelate solution with calcium ions of dentin which makes it more friable and thus dentin becomes easier to manipulate by instrumentations.

The structural formula of EDTA is :



It contains four acetic acid groups attached to ethylenediamine. EDTA is relatively nontoxic and slightly irritating in weak solutions.

The effect of EDTA on dentin depends on the concentration of EDTA solution and length of time it is in contact with dentin.

Serper and Calt in their study observed that EDTA was more effective at a neutral pH than at a pH 9.0. They showed that for optimal cleaning and shaping of canals EDTA should be used at neutral pH and with lower concentrations.

It is commercially available as 15 percent solution and pH of 7.3 under the name EDTAC because it contains cetavelon, a quaternary ammonium compound which has been added to it for its disinfecting properties. Also the introduction of surfactant reduces the contact angle of EDTA when placed on dentin surface and thus enhances its cleaning efficacy.

A chelating agent can be applied in liquid or paste form. The use of paste type preparation was first advocated by Stewart who devised a combination of urea peroxide with glycerol. Later this product was modified by combining EDTA, urea peroxide and water soluble carbowax, i.e. polyethylene glycol as vehicle. This product is commercially available as RC Prep. It is an effective lubricating and cleaning agent. Presence of glycol makes it a lubricant and coats the instrument which facilitates its movement in the canal.

A viscous suspension of chelator promotes the emulsification of organic debris and facilitates negotiation of the canal. Collagen is the major constituent of vital pulp which can be packed into glue like mass which contributes to iatrogenic blocks. Without the use of a chelator, vital tissue tends to collapse and readheres to itself but use of chelator does not allow this phenomenon to occur and accelerate emulsification of tissue.

Various studies have shown that combined use of sodium hypochlorite and RC Prep causes an efficient cleaning of canals. Their combination causes release of nascent oxygen which kills anaerobic bacteria and effervescence action which mechanically pushes the debris out of canal.

Citric Acid

Other commonly used chelating agent for removal of smear layer as irrigating solution is citric acid.

It can be used alone or in combination with other irrigants but EDTA or citric acid should never be mixed with sodium hypochlorite because EDTA and citric acid strongly interact with sodium hypochlorite. This immediately reduces the available chlorine in solution and thus making it ineffective against bacteria.

Another chelating agent suggested as irrigant is polyacrylic acid, commercially available as Durelon and Fuji II liquid.

Hydroxyethylidene bisphosphonate (HEBP) also known as Etidronate having chelating properties, is also suggested as an irrigating solution. The advantageous property of HEBP as chelating agent is that it shows only short term interference with sodium hypochlorite.

ULTRASONIC IRRIGATION

Ultrasonic irrigation have shown to clean the root canals or eliminates bacteria from the walls better than conventional methods (hand instrumentation alone).

Use of ultrasonics causes continuous flow of an irrigant in the canal, thus prevents accumulation of debris in the canal. (Fig. 11.8)

Mechanism of Action

When a small file is placed in canal and ultrasonic activation is given. The ultrasonic energy passes through irrigating solution and exerts its 'acoustic streaming or scrubbing' effect on the canal wall (Fig. 11.12). This mechanical energy warms the irrigant solution (Sodium hypochlorite) and dislodges debris from canal. The combination of activating and heating the irrigating solution is adjunct in cleaning the root canal.

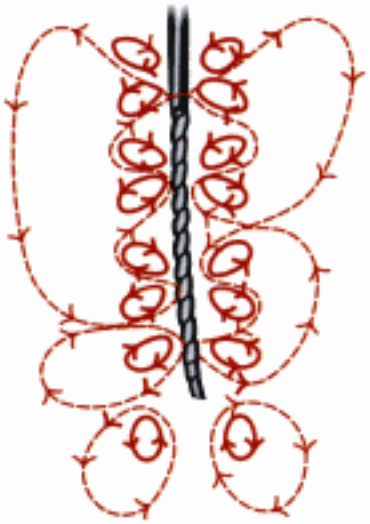
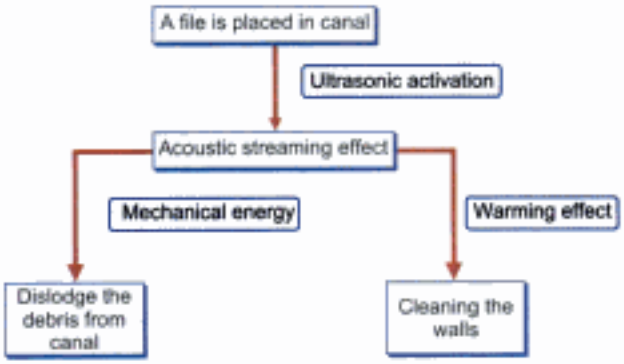


Fig. 11.12: Ultrasonic irrigation



Advantages

1. It cleans the root canal walls better than conventional ones.
2. It removes the smear layer efficiently.
3. It dislodges the debris from the canal better due to acoustic effect.

Disadvantages

1. Ultrasonic preparation of the canal is found to be unpredictable.
2. It can lead to excessive cutting of canal walls and may damage the finished preparation.

NEWER IRRIGATING SOLUTIONS

Electrochemically Activated Solution

It is one of newer irrigant solution which is produced from the tap water and low concentrated salt solutions. Further, electrochemical treatment results in synthesis of two type of solutions, i.e. anolyte (produced in anode chamber) and catholyte (produced in cathode chamber).

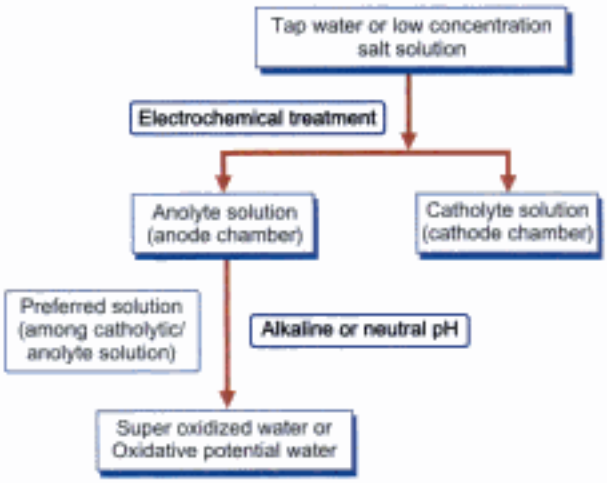
Anolyte solution has also been termed as super oxidized water or oxidative potential water but now a days neutral and alkaline solutions has been recommended for clinical application.

Advantages of electrochemically activated solution:

1. Non-toxic in contact with biological tissues
2. Effective over wide range of microbial spectra.

Ozonated Water Irrigation

Ozonated water is newer irrigant solution which is shown to be powerful antimicrobial agent against bacteria, fungi,



Formation of electrochemically activated solution

protozoa and viruses. It is suggested that ozonated water may proved to be useful to control oral infectious micro organism.

Its advantages include:

- Its potency
- Ease of handling
- Lack of mutagenicity
- Rapid microbial effects.

Ruddle's Solution

It is a new experimental irrigating solution, introduce in the endodontics in an attempt to visualize the microanatomy of the canal system.

Composition

Ruddle's solution consists of:

a. 17 percent EDTA
b. 5 percent NaOCl
c. Hypaque which is an aqueous solution of Iodide salts viz; Ditrizoate and sodium iodine.

Mechanism of Action

1. The solvent action of sodium hypochlorite, improved penetration due to EDTA and radiopacity because of hypaque helps to visualize the shape and microanatomy of canals and dentin thickness during endodontic therapy.
2. The solvent action of sodium hypochlorite clears the contents of root canal system and thus enables hypaque component to flow into every nook and corner of the canal system such as fracture, missed canals and defective restoration.

So Ruddle's solution can be helpful for improving diagnostic accuracy, treatment planning, management of procedural accidents, but further studies are needed to prove it as effective irrigating solution.

Photo Activated Disinfection (PAD)

PAD is a breakthrough in the fight against pathogenic bacteria. It is a fast, effective and minimally invasive disinfection system which is considered to kill more than 99.99 percent of bacteria in the endodontic biofilm.

Mechanism of PAD

Here, low powered laser light is transmitted through the disposable fibro-optic tip to activate the PAD antibacterial solution. Within 1-3 minutes, the PAD system eliminates more than 99.99 percent bacteria found in root canals.

Advantages of PAD

1. It has shown to be the most effective antimicrobial agent. It can effectively kill gram-negative, gram-positive, aerobic and anaerobic bacteris, in other words it eliminates all types of bacteria.
2. It overcomes the problems of antibiotic resistance.
3. It can kill bacteria present in complex bio-film such as subgingival plaque which is typically resistant to action of anti microbial agents.
4. PAD does not pose any thermal risk due to low power of PAD laser
5. It does not cause any sensitization
6. Neither the PAD solution nor its products are toxic to patients.

MTAD (A Mixture of a Tetracycline Isomer, an Acid and a Detergent)

Recently MTAD has been introduced as a final rinse for disinfection of root canal system. Torabinejad et al have shown that MTAD is able to safely remove the smear layer and is effective against Enterococcus faecalis, a microorganism resistant to the action of antimicrobial medication.

Purpose of MTAD

The purpose of using MTAD is to:

a. Disinfect the dentin
b. Remove the smear layer
c. Open the dentinal tubules and allow the antimicrobial agents to penetrate the entire root canal system.

Composition

It consists of:

a. Tetracycline isomer (doxycycline)
b. An acid (citric acid)
c. Detergent (Tween-80)

Advantages

1. It is an effective solution for removal of most of the smear layer.
2. It kills most significant bacterial stains, i.e. E faecalis which has been shown to resistant to many intracanal medicaments and irrigants
3. It is biocompatible

4. It has minimal effect on properties of teeth
5. MTAD has similar solubilizing effects on pulp and dentin to those of EDTA
6. The MTAD affinity for collagen present in MTAD for dentin allows prolonged antibacterial effect. (it's the main difference between MTAD and EDTA).

METHOD OF IRRIGATION

Although the technique of irrigation is simple and easy, still, care should be taken while irrigating with different syringes or system. Following points should be in mind while irrigating the canal:

1. The solution must be introduced slowly and passively into the canal.
2. Needle should never be wedged into the canal and should allow an adequate back-flow (Fig. 11.13).
3. Blunted needle of 25 gauge or 27 gauge are preferred.

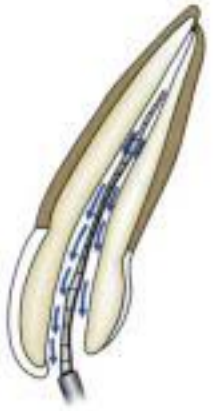


Fig. 11.13: Loose fitting needle providing space for optimal flow of irrigant

4. In case of small canals, deposit the solution in pulp chamber. Then file will carry the solution into the canal. Capillary action of narrow canal will stain the solution. To remove the excess fluid, either the aspirating syringe or 2 × 2 inches folded gauge pad is placed near the chamber (Fig. 11.14). To further dry the canal, remove the residual solution with paper point.
5. Canal size and shape are crucial for irrigation of the canal. For effective cleaning of apical area, the canals must be enlarged to size 30 and to larger size (Fig. 11.15).
6. Regardless of delivery system, irrigants must never be forcibly inserted into apical tissue rather gently placed into the canal.

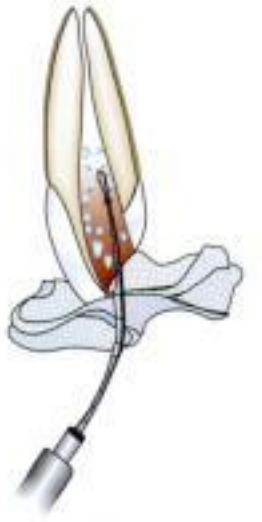


Fig. 11.14: A sterile gauge piece is placed near access opening to absorb excess irrigating solution and to check the debris from root canal

7. For effective cleaning, the needle delivering the solution should be in close proximity to the material to be removed.

Fig. 11.15: A well prepared canal allows better use of irrigant

Fig. 11.16: 30° angle bend given in irrigation needle for efficient irrigation

8. In case of large canals, the tip of needle should be introduced until resistance is felt, then withdraw the needle 2-3 mm away from that point and irrigate the canal passively. For removal of the solution sterile gauge pack or paper points should be used.

9. In order to clean effectively in both anterior and posterior teeth canals, a blunt bend of 30° in the center of needle can be given to reach the optimum length to the canal (Fig. 11.16).
10. Volume of Irrigation solution is more important than concentration or type of irrigant.

Various delivery systems for irrigation:

1. 25 or 27 gauge disposable plastic needle.
2. 27gauge needle with notched tip
3. Monojet endodontic needle
   a. 23 gauge
   b. 27 gauge
4. ProRinse—25, 28, 30 gauge probes
5. Ultrasonic handpiece.

Ideal properties of irrigating needle

1. Needle should be blunt.
2. It should allow back-flow.
3. It should be flexible.
4. Longer in length.
5. Easily available.
6. Cost-effective.

Facts

1. ***27 gauge needle with Notched tip:*** This needle is preferred as its notched tip allows backflow of the solution and does not create pressure in the periapical area. So, it ensures optimum cleaning without damage to periapical area (Fig. 11.17).

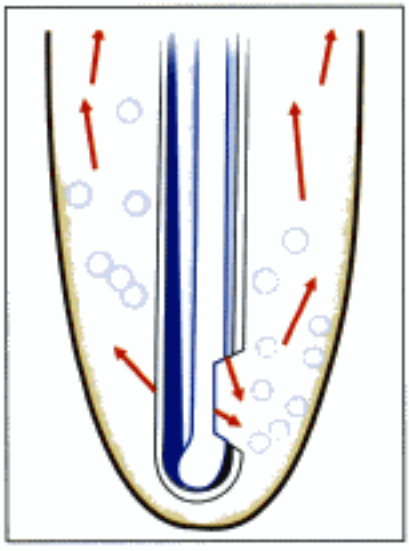


Fig. 11.17: Needle with notched tip

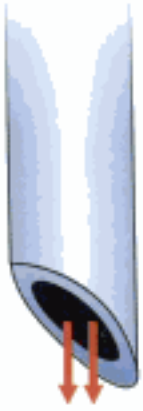


Fig. 11.18: Needle with bevel

Fig. 11.19: Monojet endodontic needle

2. ***Needle with bevel:*** Needle with bevel if gets lodged into the canal, there is risk of forcing irrigant past the apex (Fig. 11.18).
3. ***Monojet endodontic needle:*** This needle is also considered to be efficient one as the long blunt needles can be inserted to the full length of the canal to ensure optimum cleaning (Fig. 11.19). The only drawback observed is that if needles are placed near to the periapical area, it can cause damage.
4. ***ProRinse probes:*** This probe is proved to be highly effective in all gauges but 27gauge notch tip needle is proved to be highly effective as it can clean the periapical area without placing near the apical foramen. Its efficiency lie in its design as it has a blunt tip, with lumen 2 mm from the tip. Fluid from the lumen creates turbulence in all directions.

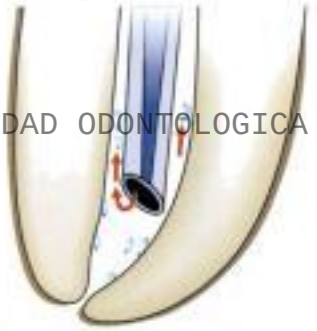


Fig. 11.20: Needle should not be wedged into the canal

PRECAUTIONS TO BE TAKEN WHILE IRRIGATION

1. Avoid wedging the needle into the canal (Fig. 11.20).
2. Avoid forcing the solution into the canal.
3. Avoid placing the needle beyond the apical area or very near to apical area
4. Avoid using larger gauge needle.
5. Avoid using metallic, autoclavable syringe as they more prone to breakage.

INTRACANAL MEDICAMENTS

Originally endodontics was mainly a therapeutic procedure in which drugs were used to destroy microorganisms, fix or mummify vital tissue and affect a sealing of the root canal space.

The drugs commonly used were caustics such as phenol and its derivatives which were shown to produce adverse effects on the periapical tissues. Gradually the reliance on drugs has been replaced by emphasis on thorough canal debridement. But drugs are still being used as intra-treatment dressings, although an ever increasing number of endodontists use them only for symptomatic cases.

Functions

- Destroy the remaining bacteria and also limits the growth of new arrivals.
- Useful in treatment of apical periodontitis for example in cases of inflammation caused due to over instrumentation.

A root canal disinfectant should have following properties:

1. It should be effective germicide and fungicide.
2. It should be non-irritating to pulpal tissue.
3. It should remain stable in the solution.
4. It should have prolonged antimicrobial action.
5. It should remain active in presence of blood and pus, etc.
6. It should have low surface tension.
7. It should not interfere with repair of periapical tissue.
8. It should not stain tooth.
9. It should be capable of inactivation in the culture media.
10. It should not induce immune response.

Various intracanal medicaments used are

1. Essential oils — eugenol
2. Phenolic compounds —
   - i. Phenol
   - ii. Paramonochlor
   - iii. Camphorated phenol
   - iv. Cresatin
   - v. Aldehydes
     - a. Formocresol
     - b. Paraformaldehyde
     - c. Glutaraldehyde
3. Calcium hydroxide
4. Halogens
   - i. Chlorine-sodium Hypochlorite
   - ii. Iodine
     - 2% I_2 in 5% KI solution, i.e. iodophores.
     - 5 percent I_2 in tincture of alcohol
5. Chlorhexidine gluconate
6. Antibiotics
7. Corticosteroid-antibiotic combination

CHARACTERISTICS OF INTRACANAL MEDICAMENTS

ESSENTIAL OILS

Eugenol

It has been used in endodontics for many years. It is a constituent of most root canal sealers and is used as a part of many temporary sealing agents (Fig. 11.21). This substance is the chemical essence of oil of clove and is related to phenol. Effects of eugenol are dependent on tissue concentrations of the eugenol (Fig. 11.22). These are divided into low dose (beneficial effects) and high dose (toxic effects).

Low doses show anti-inflammatory activity while high doses exert cytotoxic effects.

Eugenol

Low dose (beneficial effects)	*High dose (toxic effects)*
1. Inhibits prostaglandins synthesis	1. Induces cell death
2. Inhibits nerve activity	2. Inhibits cell respiration
3. Inhibits white cell chemotaxis	

Uses of Eugenol

1. Used as an intracanal medicament.
2. Used as a root canal sealers.
3. Part of temporary sealing agents.

Phenolic Compounds

Phenol

It was used for many years for its disinfectant and caustic action. However, it has strong inflammatory potential, so, at present, it is rarely used as an intracanal medicament.

Liquefied phenol (Carbolic acid) consists of 9 parts of phenol and 1 part of water.

Uses

a. It is used for disinfection before periapical surgery.
b. It is also used for cauterizing tissue tags that resist removal with broaches or files.

Parachlorophenol

Parachlorophenol has been a very popular component of dressing as phenol is no longer used in endodontics because of its high toxicity to efficacy ratio.

Composition

This is substitution product of phenol in which chlorine replaces one of the hydrogen atoms (C_6H_4OHCl). On trituration with gum camphor, these products combine to form an oily liquid.

Concentration

1 percent aqueous solution is preferred:

Fig. 11.21: Zinc oxide eugenol used as temporary restorative material

Fig. 11.22: Eugenol

Fig. 11.23: Camphorphenol

Uses

Used as a dressing of choice for infected tooth.

Camphorated Monoparachlorophenol (CMCP)

It is probably the most commonly used medicament in endodontics presently, even though its use has decreased considerably in the past few years (Fig. 11.23).

Composition

2 parts of para-chlorophenol
+
3 parts gum camphor
↓
Camphorated monochlorophenol (CMCP)

Camphor is added to Parachlorophenol (PCP) because it:

1. Has diluent action
2. Prolongs the antimicrobial effect
3. Reduces the irritating effect of PCP
4. Serves as a vehicle for the solution

Uses

Used as a dressing of choice for infected teeth.

Cresatin

As reported by Schilder and Amsterdam, Cresatin possesses the same desirable qualities and actions as that of CMCP, yet even less irritating to periapical tissues.

Composition

This substance is clear, stable, oily liquid of low volatile nature known as Metacresyl acetate.

Aldehydes

Formaldehyde, paraformaldehyde and glutaraldehyde are commonly used intracanal medicaments is root canal therapy. These are water soluble protein denaturing agents and are considered among the most potent disinfectants. They are mainly applied as disinfectants for surfaces and medical equipment which can not be sterilized, but they are quite toxic and allergic and some even may be carcinogenic.

Formocresol

Formocresol contains formaldehyde as its main ingredient and is still widely used medicament for pulpotomy procedures in primary teeth but its toxic and mutagenic properties are of concern (Fig. 11.24).

Composition of Formocresol

Formaldehyde	—	19 percent
Cresol	—	35 percent
Water and glycerine	—	46 percent

Uses

Used as dressing for pulpotomy to fix the retained pulpal tissue.

Paraformaldehyde

It is polymeric form of formaldehyde and is commonly found as component of some root canal obturating materials like endomethasone. It slowly decomposes to give out formocresol, its monomer. Its properties are similar to formaldehyde that is toxic, allergenic and genotoxic in nature.

Fig. 11.24: Formocresol

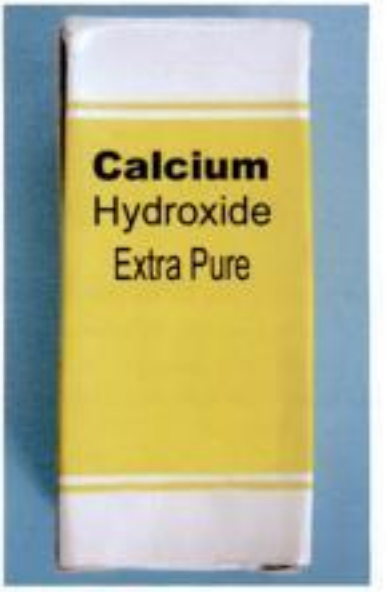


Fig. 11.25: Calcium hydroxide

All phenolic and similar compounds are highly volatile with low surface tension. Therefore, if they are placed on a cotton pellet in the chamber of a tooth during treatment, the vapors will permeate the entire canal preparation, so, placement on a paper point is unnecessary. Only tiny quantity of medication is needed for effectiveness, otherwise, chances of periapical irritation are increased.

Calcium Hydroxide

The use of calcium hydroxide in endodontics was introduced by Hermann in 1920. It has acquired a unique position in endodontics (Fig. 11.25). After its successful clinical applications for variety of indications, multiple biological functions have been attributed to calcium hydroxide. The various functions of calcium hydroxide which have been reported are as follows:

1. It shows antiseptic action probably because of its high pH and its leaching action on necrotic pulp tissues. It also increases the pH of circumpulpal dentin when placed into the root canal.
2. It hydrolyses the lipid part of bacterial lipopolysaccharide (LPS) and thus inactivates the activity of LPS. This is a desirable effect because dead cell wall material remains after the killing of bacteria which may cause infection.
3. It is also used as an intracanal medicament when one anticipates an excessive delay between appointments because it is effective as long as it remains within root canal.

Use of Calcium Hydroxide in Weeping Canal Cases

Sometime a tooth undergoing root canal treatment shows constant clear or reddish exudation associated with periapical radiolucency. Tooth can be asymptomatic or tender on percussion. When opened in next appointment, exudates stops but it again reappears in next appointment. This is known as "Weeping Canal"

In these cases, tooth with exudates is not ready for filling, since culture reports normally show negative bacterial growth so, antibiotics are of no help in such cases. For such teeth, dry the canals with sterile absorbent paper points and place calcium hydroxide in the canal. By next appointment, one finds a dry canal, ready for obturation . It happens because pH of periapical tissues is acidic in weeping stage which gets converted into basic pH by Calcium hydroxide. Some say that caustic effect of Calcium hydroxide burns the residual chronic inflamed tissue and also Calcium hydroxide builds up the bone in the lesion due to its calcifying action.

Root Canal Disinfectants

Halogens

Chlorine

Irrigating solution: Sodium hypochlorite 0.5 to 5.25% in aqueous solution.

Iodine

Irrigating solution: 2% I_2 in 5% KI aqueous solution; iodophors.
Surface disinfection: 5% I_2 in tincture of alcohol.

Chlorhexidine

Chlorhexidine gluconate Irrigating solution: 0.12-2.0% aqueous solution.

Calcium hydroxide

Dressing: aqueous or viscous formulation with varying amounts of salts added. Antibacterials like iodine, chlorphenols, chlorhexidine may also be added.

Aldehydes

Formocresol

Dressing: 19% formaldehyde, 35% cresol, 46% water and glycerine.

Phenols

Camphorated phenol
Paramonochlorphenol(PMCP)

Irrigating solution; 2% aqueous solution .
Dressing: CMCP ; 65% camphor, 35% PMCP.

Eugenol

Halogens

Halogens include chlorine and iodine which are used in various formulations in endodontics. They are potent oxidizing agents with rapid bactericidal effects.

Chlorine

Sodium hypochlorite: This compound is sometimes used as an intracanal medicament. In general, the disinfectant action of halogens is inversely proportional to their atomic weights. Chlorine (lowest atomic weight), has the greatest disinfectant action among the members of this group. Chlorine disinfectants are not stable compounds because they interact rapidly with organic matter. Mentz found sodium hypochlorite as effective intracanal medicament as well as irrigant. As the activity of sodium hypochlorite is intense but of short duration, the compound should preferably be applied to the root canal every other day.

Iodides: These compounds have been used as antiseptics for more than a century. Iodine is highly reactive, combining with proteins in a loosely bound manner so that its penetration is not impeded. It probably destroys micro-organisms by forming salts that are *inimical* to the life of the organism. Iodine is used as iodine potassium iodide and in Iodophors, which are organic iodine containing compounds that release iodine over time. It is also a very potent antibacterial agent of low toxicity, but may stain clothing if spilled. As Iodophors, it was used in a paste formulation to serve as a permanent root canal filling. Current applications of iodine compounds are as an irrigating solution and short-term dressing in a 2 percent solution of iodine in 4 percent aqueous potassium iodide and more recently, as a constituent in gutta-percha points for filling.

Chlorhexidine Gluconate

Chlorhexidine gluconate has been widely used in periodontics because of its antibacterial activity. Its use as an irrigant solution has been compared with sodium hypochlorite.

Chlorhexidine exhibit substantivity (persistence in the area of interest), broadspectrum activity and low toxicity, these properties make it well suited for irrigation and dressing applications in endodontics. Effective concentrations commonly used are in range of 0.2-2 percent range. Innovative attempts are being made to utilize the disinfecting properties of chlorhexidine in gutta-percha points.

PBSC Paste

As mentioned by Grossman, PBSC has enjoyed wide use among dentists. The constituents of the paste are as follows:

Penicillin-effective against gram-positive microorganisms
Bacitracin-effective against penicillin-resistant microorganisms
Streptomycin-effective against the gram-negative microorganisms
Caprylate (*sodium salt*)-effective against fungi.

Nystatin replaces sodium caprylate as the an antifungal agent and is available in form of PBSN. Both are available in a paste form that may be injected into root canals or impregnated on paper points. Because there is no volatility, the drug must be placed in the canal to have effect in that area.

PBSC may interfere with subsequent culturing procedures, therefore penicillinase may be added to culture media to inactivate penicillin. Reports of allergic reaction to the drug have been presented, if the patient reports history of allergy to any of the constituents, the drug should not be used. With the decline in popularity of intracanal drugs in general, and because of the potential for sensitivity due to topical use of antibiotics, PBSN largely has fallen into disuse.

Sulfonamides

Sulfanilamide and Sulfathiazole are used as medicaments by mixing with sterile distilled water or by placing a moistened paper point into a fluffed jar containing the powder. Yellowish tooth discoloration has been reported after use. Sulfonamides are usually recommended while giving closed dressing in a tooth which had been left open after an acute periapical abscess.

Corticosteroid-antibiotic Combinations

Medications that combine antibiotic and corticosteroid elements are highly effective in the treatment of over instrumentation; they must be placed into the inflamed periapical tissue by a paper point or reamer to be effective. Tetra-Cortril, Corti-sporin, Mycolog, and other combinations are available for their use in endodontics. The corticosteroid constituent reduces the periapical inflammation and gives almost instant relief of pain to the patient who complains of extreme tenderness to percussion after canal instrumentation. While the antibiotic constituents present in the corticosteroid antibiotic combination prevent the overgrowth of microorganisms when the inflammation subsides.

PLACEMENT OF INTRACANAL MEDICAMENT

1. Copiously irrigate the canal to remove debris present if any (Fig. 11.26).
2. Place the master apical file in the canal (Fig. 11.27).

Fig. 11.26: Copiously irrigate the canal

Fig. 11.27: Place the master apical file in the canal

Fig. 11.28: Dry the canal using absorbent paper points

Fig. 11.29: Intracanal medicament

Fig. 11.30: Intracanal medicament on a cotton pellet is applied and placed in pulp chamber. Over it, a sterile dry cotton pellet is placed which is finally sealed with a temporary filling material

3. Dry the canal using absorbent paper points (Fig. 11.28)
4. Place the intracanal medicament on a sterile cotton pellet and place it in the pulp chamber (Fig. 11.29)
5. Over this another sterile cotton pellet is placed, which is finally sealed with a temporary restorative material (Fig. 11.30).

12 Cleaning and Shaping of Root Canal System

- Introduction
- Schilder's Objectives of Canal Preparation
- Objectives of Biomechanical Preparation
- Various Movements of Instruments
- Basic Principles of Canal Preparation
- Techniques of Canal Preparation
- Engine Driven Preparation with NiTi Instruments
- Canal Preparation Using Ultrasonic Instruments
- Canal Preparation Using Sonic Instruments
- Laser Assisted Canal Therapy
- Evaluation of Canal Preparation
- Special Anatomic Problems in Cleaning and Shaping

INTRODUCTION

Endodontic treatment mainly consists of **three steps:**

1. Cleaning and shaping of the root canal system
2. Disinfection
3. Obturation

Cleaning and shaping is one of the most important step in the root canal therapy for obtaining success in the root canal treatment.

Cleaning

The removal of all potentially pathogenic contents of the root canal system.

Shaping

The establishment of a specifically shaped cavity which performs the dual role of three dimensional progressive access into the canal and creating an apical preparation which will permit the final obturation instruments and materials to fit easily (Fig. 12.1).

Fig. 12.1: Complete cleaning and shaping of root canal system

For the success of endodontic treatment one must remove all the contents of the root canal completely because any communication from root canal system to periodontal space acts as portal of exit which can lead to formation of lesions of endodontic origin (Fig. 12.2).

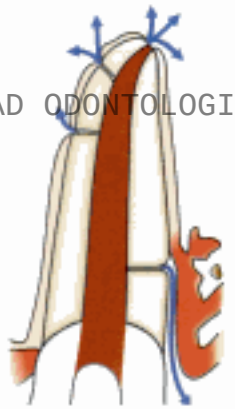
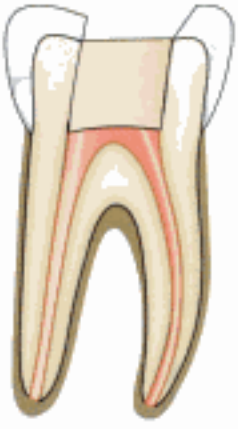


Fig. 12.2: Portals of communication of root canal system and periodontium

Cleaning shaping, i.e. biomechanical preparation of the root canal system was a hit and trial method, before **Dr. Schilder** introduce the concept of cleaning and shaping.

Initially the root canals were manipulated primarily to allow the placement of intracanal medicaments. But with the advent of role of radiographs in endodontics, one could see the lesion of bone and teeth and concept was changed to remove the pathogenic cause from the tooth. As the root canal therapy has developed, the preparation has been described as instrumentation, chemomechanical instrumentation, biomechanical preparation, etc. which described the mode of root canal therapy, but the ultimate goal is of cleaning and shaping of the root canal system.

The two concepts, cleaning and shaping and the three dimensional obturation are interdependent. Obturation of root canal can not be better achieved if canals are not thoroughly cleaned and shaped (Fig. 12.3).



Figs 12.3: Three dimensional obturation of root canal system

The mechanics of cleaning and shaping may be viewed as an extension of the principles of coronal cavity preparation to the full length of the root canal system. **Schilder** gave five mechanical objectives for successful cleaning and shaping 30 years ago. The objectives taught the clinicians to think and operate in three dimensions.

The objectives given by **Schilder** are:

1. ***The root canal preparation should develop a continuously tapering cone:*** This shape mimics the natural canal shape (Fig. 12.4). Funnel shaped preparation of canal should merge with the access cavity so that instruments will slide into the canal. Thus access cavity and root canal preparation should form a continuous channel.

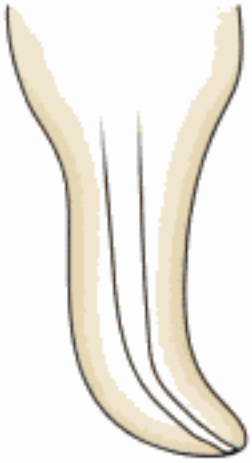


Fig. 12.4: Prepared root canal shape should be continuous tapered

2. ***Making the preparation in multiple planes which introduces the concept of "flow":*** This objective preserves the natural curve of the canal.
3. ***Making the canal narrower apically and widest coronally:*** To create a continuous tapers up to apical third which creates the resistance form to hold gutta percha in the canal (Fig. 12.5).
4. ***Avoid transportation of the foramen:*** There should be gentle and minute enlargement of the foramen while maintaining its position (Fig. 12.5).
5. ***Keep the apical opening as small as possible:*** The foramen size should be kept as small as possible as overlapping of foramen contributes to number of iatrogenic problems. Doubling the file size apically increases the surface area of foramen four folds (πr^2) (Fig. 12.6). This overlapping of apical foramen should be avoided.

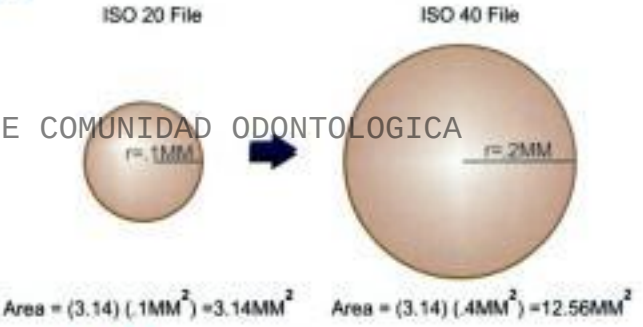


Fig. 12.5: Doubling the file size apically, increases the surface area of foramen four times

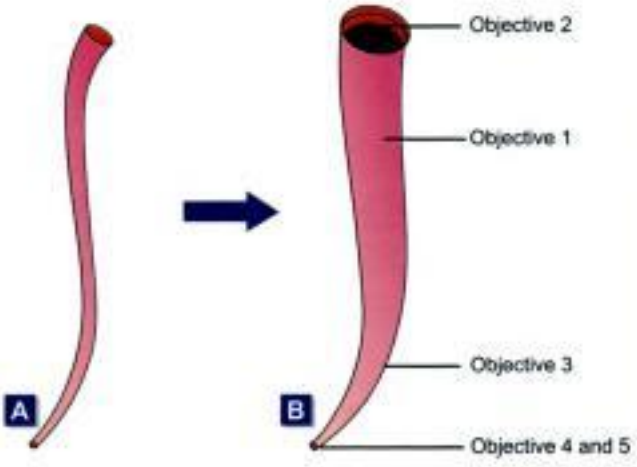


Fig. 12.6: Diagrammatic representation of objectives of canal preparation

Mechanical Objectives of Root Canal Preparation (given by Schilder)

- The root canal preparation should develop a continuously tapering cone.
- Making the preparation in multiple plane which introduces the concept of "Flow".
- Making the canal narrower apically and widest coronally.
- Avoid transportation of foramen.
- Keep the apical opening as small as possible.

Objectives of Biomechanical Preparation

Biologic objectives of biomechanical preparation are to remove the pulp tissue, bacteria and their by-products from the root canal space.

Clinical Objectives: Before starting the endodontic therapy, the clinicians should plan the whole treatment so as to obtain the successful treatment results.

The clinician should evaluate the tooth to be treated to ensure that the particular tooth has favorable prognosis (Fig. 12.7). Before performing cleaning and shaping, the straight line access to canal orifice should be obtained. All the overlying dentin should be removed and there should be flared and smooth internal walls to provide straight line access to root canals (Fig. 12.8). Since shaping facilitates cleaning in properly shaped canals, instruments and irrigants can go deeper into the canals to remove all the debris and contents of root canal and thus creating a smooth tapered opening to the apical terminus for obtaining three dimension obturation of the root canal system.

After obturation, there should be complete sealing of the pulp chamber and the access cavity to prevent microleakage into the canal system (Fig. 12.9). Tooth should be restored with permanent restoration to maintain its form, function and aesthetics and patient should be recalled on

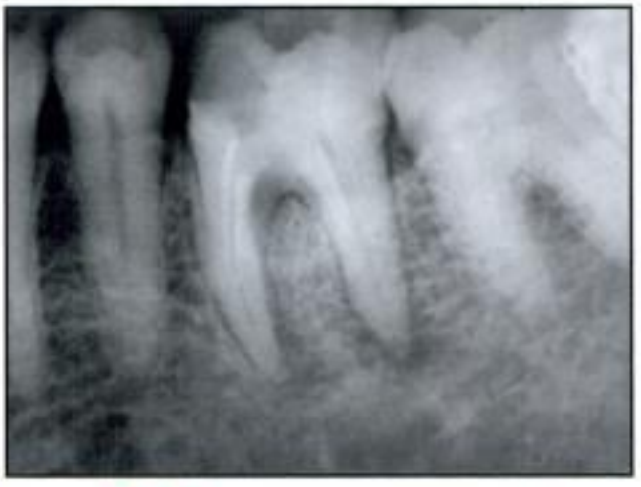


Fig. 12.7A: Radiograph showing carious molar with pulp exposure

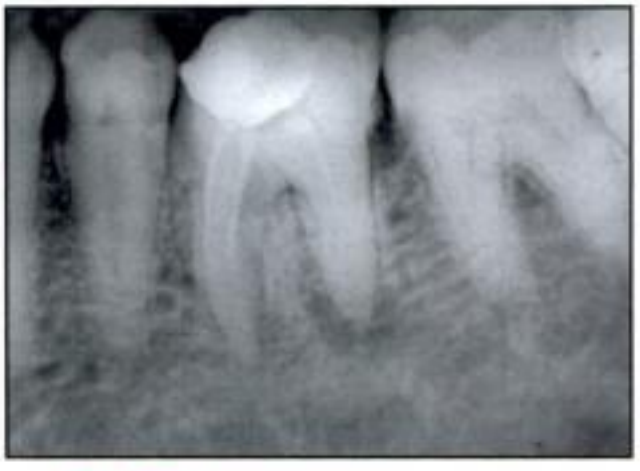


Fig. 12.7B: Completed endodontic therapy of molar

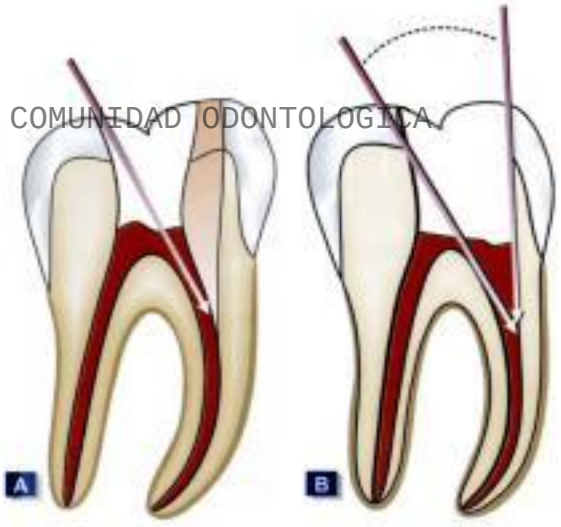


Fig. 12.8: Removal of overlying dentin causes smooth internal walls and provide straight line access to root canals

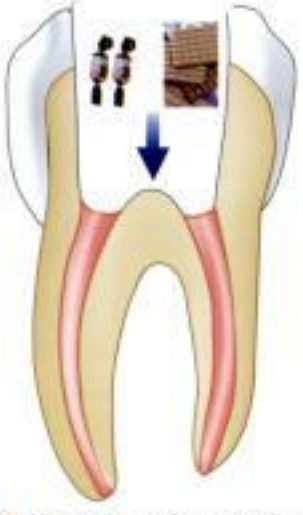


Fig. 12.9: Microleakage of root canal system in nonrestored tooth

the regular basis to evaluate the success of the treatment. For past many years, there has been a gradual change in the ideal configuration of the prepared root canal.

Earlier a round tapered and almost parallel shape was considered an ideal preparation but later when **Schilder** gave the concept of finished canal with gradually increasing the taper having the smallest diameter apically and widest diameter at the coronal orifice.

Since long, many techniques have been suggested for complete debridement and shaping of the root canal system with different success rates.

VARIOUS MOVEMENTS OF INSTRUMENTS

Reaming

In Layman's terms, ream indicates use of sharp edged tool for enlarging holes.In endodontic practice, reaming is commonly done by use of reamers , though files can also be used. It involves clockwise rotation of an instrument. The instrument may be controlled from insertion to generate a cutting effect (Fig. 12.10).

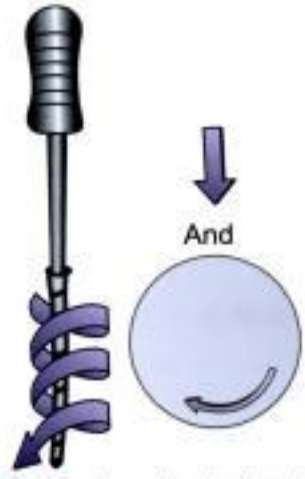


Fig. 12.10: Reaming motion involving clockwise rotation of instrument

Fig. 12.11: Filing motion showing push and pull action of instrument

Filing

The term filing indicates push-pull motion with the instrument (Fig. 12.11). This method is commonly used for canal preparation.

But this active insertion of instrument with cutting force is a combination of both resistance to bending and apically directed hand pressure. This may lead to canal ledging, perforation and other procedural errors.

Fig. 12.12: Combination of reaming and filing

But nowadays, to avoid such errors current techniques employ the technique of passive insertion of instrument, precurving of instruments and a quarter turn insertion.

Combination of Reaming and Filing (Fig. 12.12)

In this technique file is inserted with a quarter turn clockwise and apically directed pressure (i.e. reaming) and then is subsequently withdrawn (i.e. filing).

File edges get engaged into dentin while insertion and breaks the loose dentin during its withdrawal. By performing this combination of reaming and filing repeatedly, canal enlargement takes place.

Frequent ledge formation, perforation and other procedural errors.

To overcome these shortcomings, this technique was modified by Schilder. He suggested to give a clockwise rotation of half revolution followed by instrument apically. In this method every time when a file is withdrawn, it is followed by next in the series. Though this method is effective in producing clean canals but it is very laborious and time consuming.

Balanced Force Technique

This technique involves oscillation of instrument right and left with different arcs in either direction. Instrument is first inserted into the canal by moving it clockwise with one quarter turn. Then to cut dentin, file is rotated counter clockwise and simultaneously pushing apically to prevent it from backing out of the canal. Finally, the file is removed by rotating file clockwise simultaneously pulling the instrument out of the canal (Fig. 12.13).

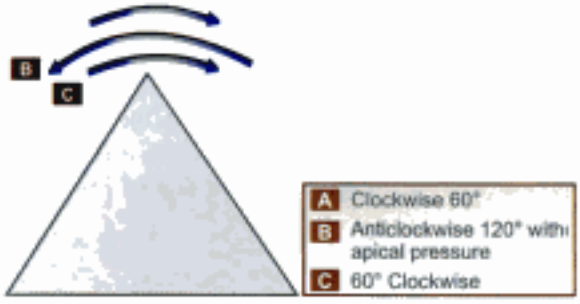


Fig. 12.13: Balanced force technique

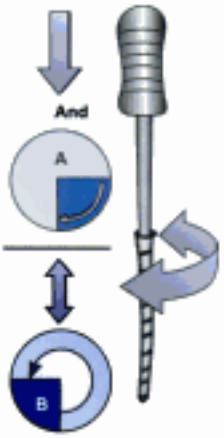


Fig. 12.14: If excessive force is applied, instrument may lock into the root canal system during rotation

This technique offers most efficient dentin cutting but care should be taken not to apply excessive force with this technique because it may lock the instrument out of the canal (Fig. 12.14).

Since H-files and broaches do not possess left hand cutting efficiency, they are not used with this technique. Simultaneous apical pressure and anticlock-wise rotation of the file maintains the balance between tooth structure and the elastic memory of the instrument, this balance locates the instrument near the canal axis and thus avoids transportation of the canal.

Watch Winding

It is back and forth oscillation of the endodontic instrument (file or reamer) right and left as it is advanced into the canal. The angle of rotation is usually 30 to 60 degrees (Figs 12.15 and 12.16).

Fig. 12.15: Watch winding motion

This technique is efficient with K-type instruments. This motion is quite useful during biomechanical preparation of the canal. Watch winding motion is less aggressive than clockwise and anticlockwise motion. As this motion, the instrument tip is not forced in to the apical area with each motion, thereby reducing the frequency of instrumental errors.

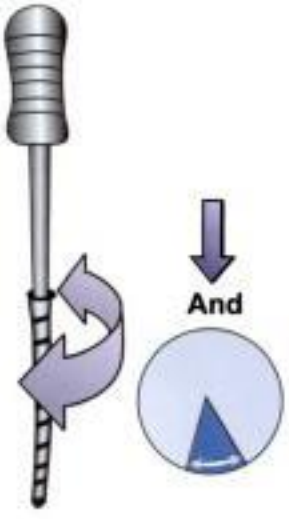


Fig. 12.16: Rotation of file in watch winding motion

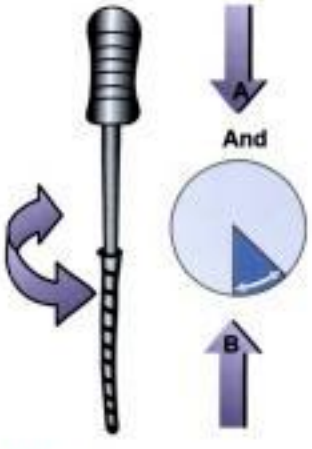


Fig. 12.17: Watch winding and pull motion

Watch Winding and Pull Motion

In this, first instrument is moved apically by rotating it right and left through an arc. When the instrument feels any resistance, it is taken out of the canal by pull motion (Fig. 12.17). This technique is primarily used with Hedstroem files. When used with H-files, watch winding motion can not cut dentin with backstroke because H- files can cut only during pull motion.

BASIC PRINCIPLES OF CANAL INSTRUMENTATION

1. There should be a straight line access to the canal orifices. Creation of a straight line access by removing overhang dentine influence the forces exerted by a file in apical third of the canal. (Fig. 12.18)
2. Files are always worked with in a canal filled with irrigant, so copious irrigation is done between the instrumentation, i.e. canal must always be prepared in wet environment
3. Preparation of canal should be completed while retaining its original form and the shape (Fig. 12.19).
4. Exploration of the orifice is always done with smaller file to gauge the canal size and the configuration.

Fig. 12.18: Straight line access to root canal system

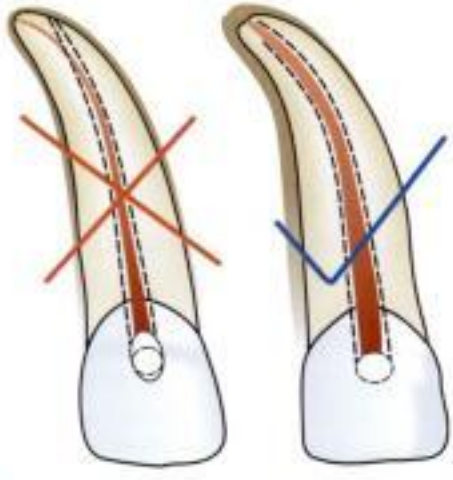


Fig. 12.19: Prepared canal should retain its original form and shape

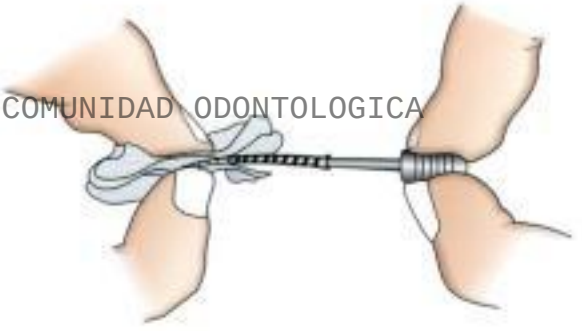


Fig. 12.20: Cleaning of flutes should be done after each instrumentation

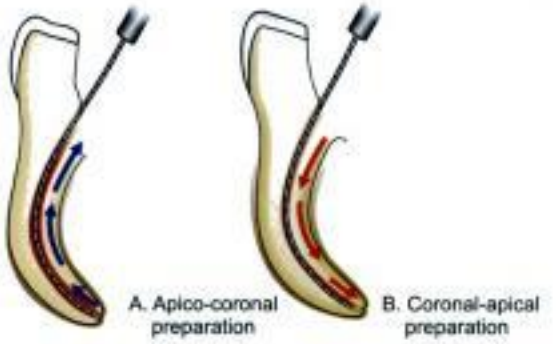


Fig. 12.21: Techniques of biomechanical preparation

5. Canal enlargement should be done by using instruments in the sequential order without skipping sizes.
6. All the working instruments should be kept in confines of the root canal to avoid any procedural accidents.
7. Instrument binding or dentin removal on insertion should be avoided.
8. After each insertion and removal of the file, its flutes should be cleaned and inspected (Fig. 12.20).
9. Smaller number instruments should be used extravagantly.
10. Recapitulation is regularly done to loosen debris by returning to working length. The canal walls should not be enlarged during recapitulation.
11. Over preparation and too aggressive over enlargement of the curved canals should be avoided.
12. Creation of an apical stop may be impossible if apical foramen is already very large. Overusing of larger files should be avoided in such cases as it may create even a larger apical opening.
13. Never force the instrument in the canal. Forcing or continuing to rotate an instrument may break the instrument.
14. Establish the apical patency before starting the biomechanical preparation of tooth. Apical patency of the canal established and checked, by passing a smaller number file (No. 10) across the apex. The aim is to allow for creation of a preparation and filling extending fully to the periodontal ligament. Establishing the patency is considered a non-harmful even considering the blood supply and immune response present in the periapical area.

TECHNIQUES OF ROOT CANAL PREPARATION

Basically, there are ***two approaches*** used for ***biomechanical preparation***, either starting at the apex with fine instruments and working up to the orifice with progressively larger instruments, this is ***Step back technique*** or starting at the orifice with larger instrument and working up to apex with larger instruments, this is ***Crown down technique (Fig. 12.21).***

Various other techniques have been modified out of these two basic techniques. Whichever the techniques is used for canal preparation one should ensure of staying within the confines of root canal and resulting in continuous tapered preparation of the canal.

Fig. 12.22: Tapered canal preparation

STEP BACK TECHNIQUE

Step back technique is also known as ***Telescopic canal preparation*** or ***serial root canal preparation.*** Step back technique emphasizes keeping the apical preparation small, in its original position and producing a gradual taper coronally (Fig. 12.22). This technique was first described in 1960 by Mullaney.

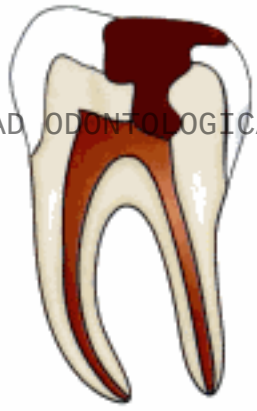


Fig. 12.23: Tooth decay causing pulp exposure

Fig. 12.25: Watch winding motion with gentle clockwise and anticlockwise motion of the file

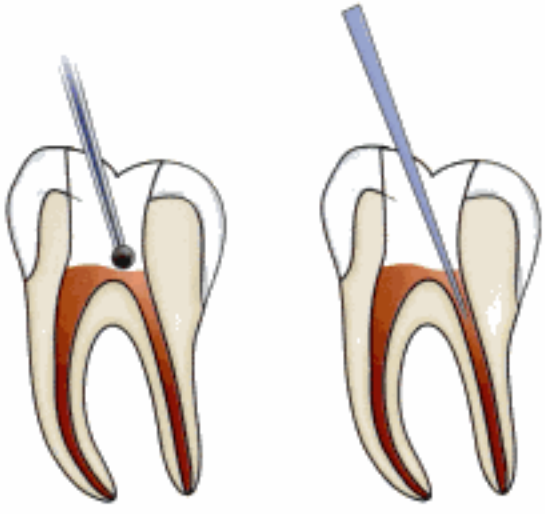


Fig. 12.24: Prepare the access cavity and locate the canal orifices

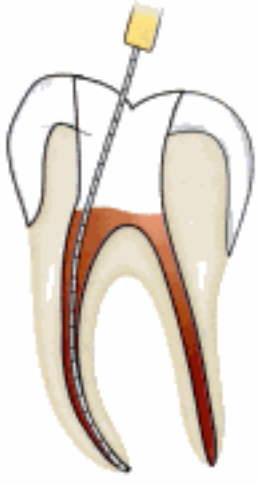


Fig. 12.26: Place file to working length

Basically this technique involves the canal preparation into ***two phases; phase I*** involves the preparation of apical constriction and ***phase II*** involves the preparation of the remaining canal.

Phase I

1. Initially prepare the access cavity and locate the canal orifices (Figs 12.23 and 12.24).
2. Establish the working length of the tooth using pathfinder.
3. Now insert the first instrument into the canal with watch winding motion. In watch winding motion, a gentle clockwise and anticlockwise rotation of file with minimal apical pressure is given (Fig. 12.25).
4. Remove the instrument and irrigate the canal.
5. Don't forget to lubricate the instrument for use in apical area because it is shown that lubricant emulsify the

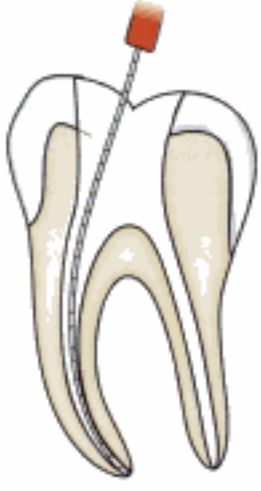


Fig. 12.27: 25 No. file at working length

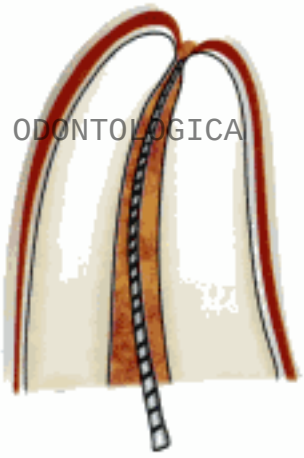


Fig. 12.28: Recapitulation using smaller file

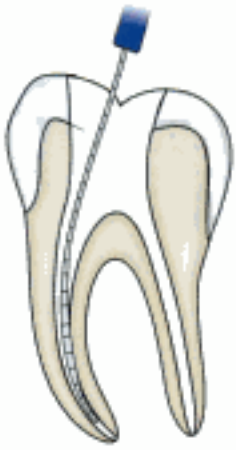


Fig. 12.29: 30 No. file 1 mm short of working length

fibrous pulp tissue allowing the instrument to remove it whereas irrigants may not reach the apical area to dissolve the tissues.

6. Place the next larger size file to the working length in similar manner and again irrigate the canal (Fig. 12.26).
7. Don't forget to recapitulate the canal with previous smaller number instrument. This breaks up apical debris which are washed away with the irrigant.
8. Repeat the process until a size 25 K-file reaches the working length (Fig. 12.27). Recapitulate between the files by placing a small file to the working length (Fig. 12.28).

Phase II

1. Place next file in the series to a length 1 mm short of working length. Insert the instrument into the canal with watch winding motion, remove it after circumferential filing, irrigate and recapitulate (Fig. 12.29).
2. Repeat the same procedure with successively larger files at 1 mm increments from the previously used file (Fig. 12.30).
3. Similarly mid canal area and coronal part of the canal is prepared and shaped with larger number files (Figs 12.31 to 33).
4. Finally refining of the root canal is done by master apical file with push-pull strokes to achieve a smooth taper from of the root canal.

Variations in the Original Step Back Technique involves

a. Initial enlarging of the coronal part of root canal with Gates-Glidden drills (Fig. 12.34).

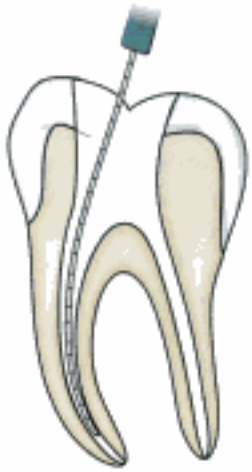


Fig. 12.30: 35 No. file 2 mm short of working length

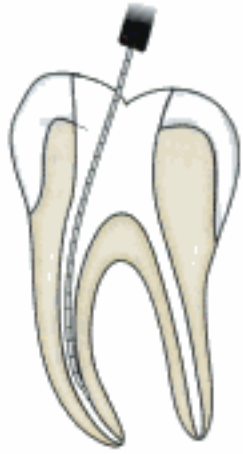


Fig. 12.31: 40 No. file 3 mm short of working length

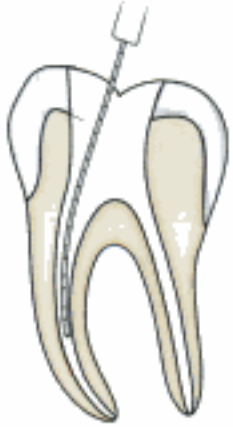


Fig. 12.32: 45 No. file 4 mm short of working length

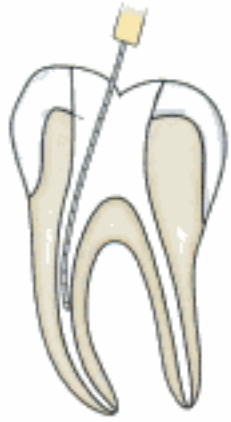


Fig. 12.33: 50 No. file for canal preparation

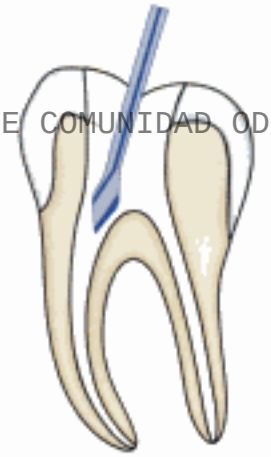


Fig. 12.34: Enlargement of canal using Gates-Glidden drill

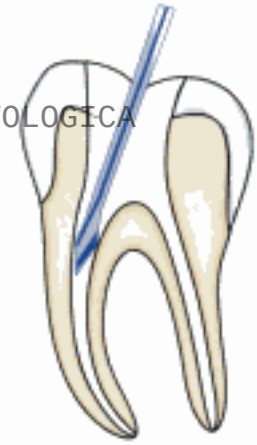


Fig. 12.35: Use of smaller Gates-Glidden to prepare mid root area

b. Use of smaller Gates-Glidden drills to prepare the mid root level (Fig. 12.35).
c. Use of Hedstorm files to flare the preparation.

Before using Gate-Glidden, one must gain access to the canal orifices by removing the overlying dentinal structure. Thus we see that step back technique creates small apical preparation with larger instruments used at successively decreasing lengths to create a taper. The taper of canal preparation could be altered by changing the interval between the consecutive instruments for example taper of prepared canal could be increased by reducing the intervals between each successive file from 1 to 0.5 mm.

Though this technique has been used since long time, but various studies have shown that this apical to coronal preparation of root canals can cause deformation of the canal shape, instrument separation, zipping of the apical area, significant apical extrusion of debris, apical blockage, etc. (Figs 12.36A to C).

MODIFIED STEP BACK TECHNIQUE

In this technique, the preparation is completed in apical third of the canal after this step back procedure is started 2-3 mm up the canal to give an almost parallel retention form at the apical area. This receives the primary gutta-percha point which should show a slight tug back, when the point is removed. This explains that cone fits snuggly into the last 2-3 mm of the prepared canal.

PASSIVE STEP BACK TECHNIQUE

This technique was developed by ***Torabinajed*** which involves combination of hand instruments (files) and the rotary instruments (Gates-Glidden drills and Pesso reamers) to attain an adequate coronal flare before apical root canal preparation.

This technique provides gradual enlargement of the root in an apical to coronal direction without applying force, thereby reducing the occurrence of procedural errors like transportation of the canal, ledge or zip formation and this is convenient to both patient as well as doctor.

Technique

1. First of all, after preparation of the access cavity, locate the canal orifices and flare the walls of access cavity using tapered diamond burs.
2. Now establish the correct working length using a number 15 file. A number 15 file is inserted to the corrected working length with very light pressure to one eighth to one quarter turn with push-pull stroke to establish the apical patency.
3. After this additional files of number 20, 25, 30, 35 and 40 are inserted into the canal passively. This step removes the debris and creates a mildly flared preparation for insertion of Gates-Glidden drills.
4. Copious irrigation of the canal system is frequently done with sodium hypochlorite.
5. After this, number 2 Gate-Glidden drill is inserted into mildly flared canal to a point, where it binds slightly. It is pulled back 1 to 1.5 mm and then activated.

   With up and down motion and slight pressure, the canal walls are flared. In the similar fat fashion numbers 3 and 4 Gates-Gliddens are then used coronally.

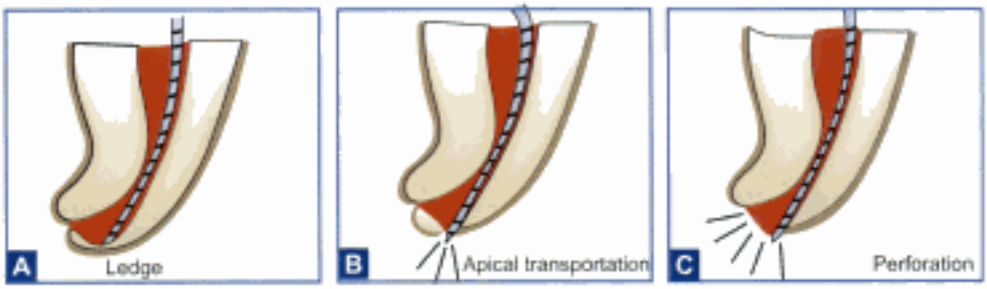


Fig. 12.36: Procedural errors

6. Because flaring and removed of curvatures reduce the working length so reconfirmation of the working length is done before apical pressure.
7. After this, a number 20 file is inserted into the canal [illegible] working length. The canal is then prepared with sequential use of progressively larger instruments placed successively short of the working length. Narrow canal should not be enlarged beyond the size of number 25 or 30 files.

Advantages of Passive Step Back Technique

1. Removal of debris and minor canal obstructions.
2. Knowledge of the canal morphology.
3. Gradual passive enlargement of the canal in an apical to coronal direction.
4. This technique can also be used with ultrasonic instruments.

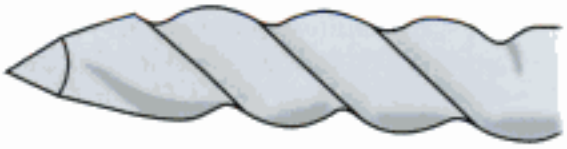
BALANCED FORCE TECHNIQUE

This technique was developed by ***Roane***. It involves the use of instrument with noncutting tip. Since the K-type files have pyramidal tips with cutting angles which can be quite aggressive with clockwise rotation. For this technique, use of triangular cross sectioned instruments should be done because the decreased mass of the instrument and deeper cutting flutes improve flexibility and decrease the restoring force of the instrument when placed in curved canals.

Use of Flex-R files is recommended for this technique which has "safe tip design" with a guiding land area behind the tip which allows the file to follow the canal curvature without binding in the outside wall of the curved canal (Fig. 12.37).

Technique

1. In balanced force technique, first file to bind short of working length is inserted into the canal and rotated clockwise a quarter of a turn. This causes flutes to engage a small amount of dentin (Fig. 12.38).



Flex-R file

Fig. 12.37: Use of flex-R file for balanced force technique

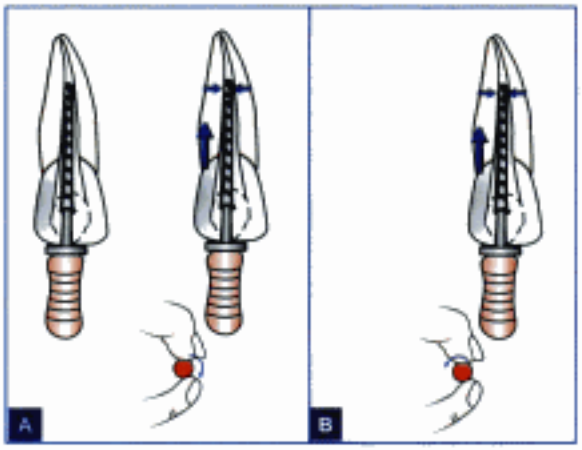


Fig. 12.38: (A) Engaging dentin with quarter clockwise turn (B) Cutting action by anticlockwise motion with apical pressure

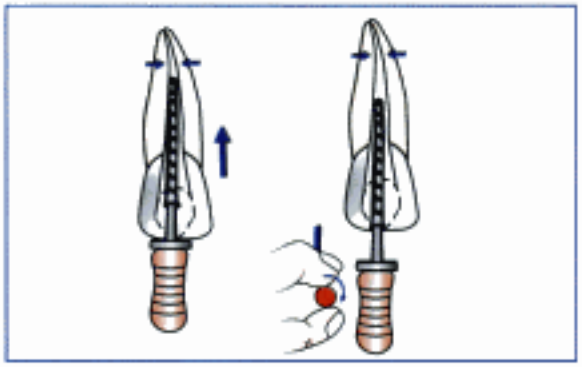


Fig. 12.39: Now file is turned quarter clockwise. It picks the debris and withdraws the instrument

2. Now file is rotated counterclockwise with apical pressure at least one third of a revolution (Fig. 12.39). It is the counterclockwise rotation with apical pressure which actually provides the cutting action by shearing off small amount of dentin engaged during clockwise rotation.
3. If there is little curvature or if instrument does not bind, only one or two counterclockwise motions are given. It should not be forced to give the counterclockwise rotation because it may lead to fracture of the instrument.
4. Then a final clockwise rotation is given to the instrument which loads the flutes of file with loosened debris and the file is withdrawn.

Advantages of Balanced Force Technique

1. With the help of this technique, there are lesser chances of canal transportation.
2. One can manipulate the files at any point in the canal without creating a ledge or blockage.

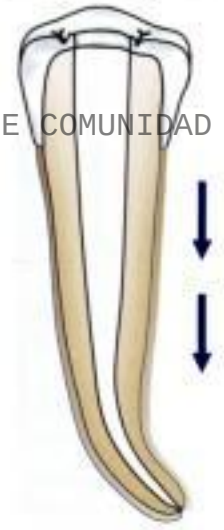


Fig. 12.40: Crown-down technique

Fig. 12.41: Straight line access to root canal system

Fig. 12.42: Filling the chamber with irrigant solution

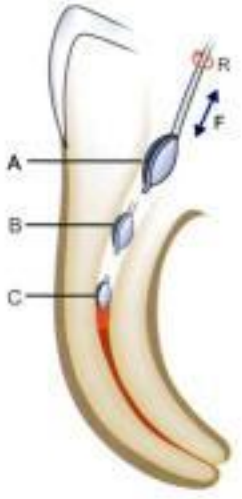


Fig. 12.43: Use of Gates-Glidden for preflaring

CROWN DOWN TECHNIQUE

In the crown down technique, the dentist prepares the canal from crown of the tooth, shaping the canal as he / she move towards the apical portion of the canal (Fig. 12.40). Morgan and Montgomery found that this "crown down pressureless" techniques resulted in a rounder canal shape when compared to usual step back technique. Moreover many studies have shown that greater apical enlargement without causing apical transportation can be achieved if coronal obstructions are eliminated.

Technique of Crown Down Preparation

1. First step in the crown down technique is the access cavity preparation with no pulp chamber obstructions (Fig. 12.41). Locate the canal orifices with sharp explorer which shows binding in the pulp chamber.
2. Now fill the access cavity with an irrigant and start preflaring of the canal orifices (Fig. 12.42). Preflaring of the coronal third of the canal can be done by using hand instruments, Gates-Glidden drills or the Nickle - titanium rotary instruments.
3. Gates-Glidden drills can be used after scouting the canal orifices with number 10 or 15 files. The crown down approach begins with larger Gates-Glidden first (Fig. 12.43). After using this subsequent, smaller diameter Gates-Glidden are worked into the canal with additional mm to complete coronal flaring. One should take care to avoid carrying all the Gates-Glidden drills to same level which may lead to excessive cutting of the dentin, weakening of the roots and thereby "Coke Bottle Appearance" in the radiographs (Fig. 12.44).
4. Frequent irrigation with sodium hypochlorite and recapitulation with a smaller file (usually No. 10 file) to prevent canal blockage.
5. After establishing coronal and mid root enlargement explore the canal and establish the working length with small instruments (Fig. 12.45).
6. Introduce larger files to coronal part of the canal and prepare it (Fig. 12.46). Subsequently introduce progressively smaller number files deeper into the canal in sequential order and prepare the apical part of the canal (Fig. 12.47 and 48).
7. Final apical preparation is prepared and finished along with frequent irrigation of the canal system.

The classical apical third preparation should have a tapered shape which has been enlarged to at least size 20 at apex and each successive instrument should move away from the foramen by $^1/_2$ mm increments.

Apical Gauging

1. The function of apical gauging is to measure the apical diameter of the canal prior to cutting the final shape. This is necessary to insure that the final tapered preparation extends all the way to the terminus of the canal (Fig. 12.49).
2. Use NiTi K-files for gauging. The flexibility allows for much more accurate apical gauging in curved canals than with stainless steel, insuring the apical accuracy of obturation.

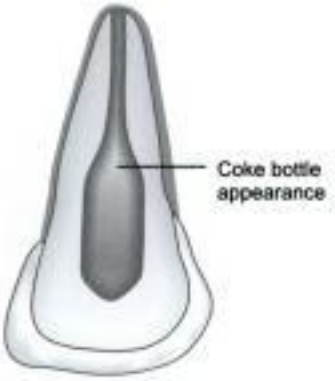


Fig. 12.44: "Coke-bottle appearance" caused by excessive use of Gates-Glidden drills

Fig. 12.45: Establishing working length using a small instrument

Fig. 12.46: Use of larger files to prepare coronal third

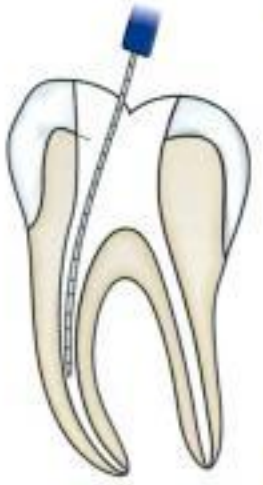


Fig. 12.47: Preparation of canal to deeper levels

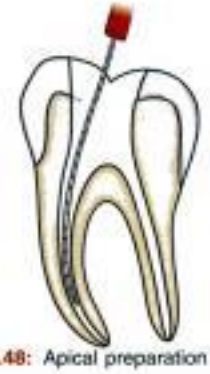


Fig. 12.48: Apical preparation of canal

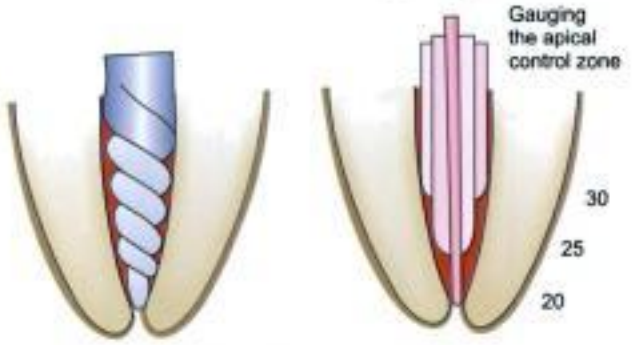


Fig. 12.49: Apical gauging of root canal

3. No effort is made to cut dentin during apical gauging. The gauging instruments are inserted straight in and are pulled straight out with no rotation.
4. Always use ***17 percent aqueous EDTA*** as an irrigant during gauging to remove the smear layer.

Biological Benefits of Crown Down Technique

1. Removal of tissue debris coronally, thus minimizing the extrusion of debris periapically.
2. Reduction of post operative sensitivity which could result from periapical extrusion of debris.
3. Greater volumes of irrigants can reach in canal irregularities in early stages of canal preparation because of coronal flaring.
4. Better dissolution of tissue with increased penetration of the irrigants.
5. Rapid removal of contaminated and infected tissues from the root canal system.

Clinical Advantages of the Crown Down Technique

1. Enhanced tactile sensation with instruments because of removal of coronal interferences (Fig. 12.50).
2. Flexible (smaller) files are used in apical portion of the canal; where as larger (stiffer) files need not be forced but kept short of the apex.
3. In curved canals, after doing coronal flaring, files can go upto apex more effectively due to decrease deviation of instruments in the canal curvature.
4. Provides more space of irrigants.
5. Straight line access to root curves and canal junctions.
6. Enhanced movement of debris coronally.
7. Desired shape of canal can be obtained that is narrow at apex, wider at coronal (Figs 12.51 and 12.52).

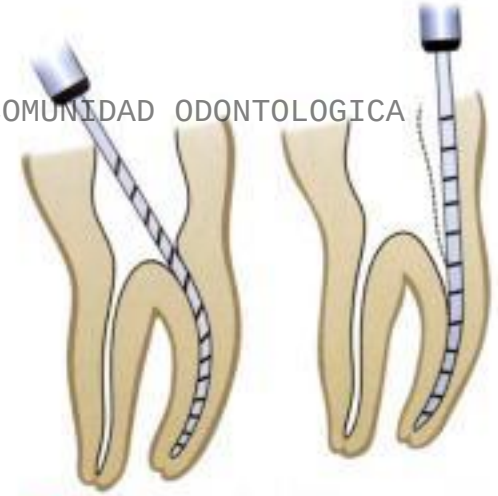


Fig. 12.50: Preflaring of canal causes removal of coronal interferences

Fig. 12.53: Ledge formation caused by use of stiff instrument in curved canals

Fig. 12.51: A well prepared canal mandibular premolar

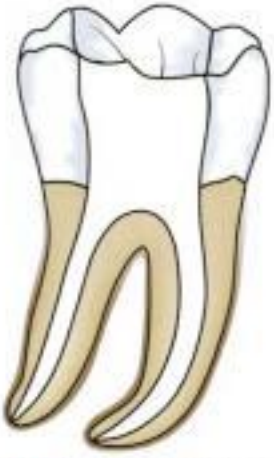


Fig. 12.52: A well prepared tapered preparation of mandibular molar

8. Predictable quality of canal cleaning and shaping.
9. Decreased frequency of canal blockages.

The crown down is often suggested as a basic approach using nickel-titanium rotary instruments. Very few limitations exist in the application of the crown-down technique. Its application may be limited however to the use of hand instruments in specific situations. As with any technique, there will be a learning curve in its implementation, and the achievements with this technique of root canal cleaning and shaping may be affected by:

1. Operator desire to learn and skill level developed in application.
2. The use of end cutting rotary instruments in small or partially calcified canals may predispose to perforation as the instrument moves apically.
3. In canals that curve severely the rotary instruments cannot be precurved for placement and ease of penetration to enlarge the coronal third of the canal. In these cases, the crown down technique can still be implemented by using hand instruments prior to rotary instruments.
4. If large, less flexible rotary instruments are used too rapidly and deeply in the root canal, a ledge may form (Fig. 12.53).

ENGINE DRIVEN PREPARATION WITH NITI INSTRUMENTS

These instruments were introduced in early 1990s, and since then they have become indispensable tools for canal enlargement. Before using these instruments one should take care to have a straight line access to the canal system. Canals should be thoroughly explored and passively enlarged before using rotary instrument. Instruments should be constantly moving and speed of rotation of each instrument should be known.

All of these NiTi rotary systems incorporate:

- Crown down preparation.
- Apical preparation as finale.
- Increasing taper instruments.

PROFILE SYSTEM

Profile instruments system was introduced by Dr Johnson in 1944. Earlier profile system was sold as series 29 instru-

ments. After this profile series were introduced with greater tapers of 19 mm lengths and ISO sized tips. Suggested rotational speed for profiles is 150-300 RPM. Cross section of profiles have a central pulpal core with three equally shaped U-shaped grooves along with radial lands. The negative rake angle of profiles makes them to cut dentin in planning motion.

CLINICAL TECHNIQUE FOR USE OF PROFILES

1. Make a straight line access to the canal orifice.
2. Estimate the working length of the canal from preoperative radiograph.
3. Create a glide path before using orifice shapers. Establish this path with a small, flexible, stainless steel number 15 or 20 file.
4. Use orifices shapers sizes 4, 3, 2, and 1 in the coronal third of the canal.
5. Perform crown down technique using the profile instruments of taper/size 0.06 /30,0.06/25,0.04/30 and0.04/25 to the resistance. For larger canals use 0.06/35,0.06/30,0.04/35 and 0.04/30.
6. Now determine the exact working length by inserting conventional number 15 K-file (2% taper).
7. After establishing the exact working length complete the crown down procedure up until this length. Use profile 0.04/25, 0.04/30 for apical preparatio.
8. Now final flaring is done using profile 0.06/25 short of working length to merge coronal and apical preparation.

In summary the profile instruments are used in both descending order of the diameter (i.e. for crown down from largest to the smallest) and in ascending order of diameter (for preparation to the exact working length and for final flaring from smallest to the largest).

Advantages

1. Presence of radial land and noncutting tip keep the Profiles self centered in root canal thus preserving the natural canal path. It also avoids risk of zip or transportation of canal.
2. Presence of 20° helical angle allows effective removal of dentin debris, thus eliminating the risk of debris blocking the canal or being pushed into the periapical area.
3. Presence of radial land prevents its screwing into the canal and thus reduces the fracture risk.
4. Because of presence of modified tip without transition angle and negative rake angle, profiles work the dentin in planning motion.

GREATER TAPER FILES (GT FILES)

The G T rotary instruments possess a U - shaped file design with ISO tip sizes of 20, 30 and 40 and tapers of 0.04, 0.06, 0.08,.010 and 0.12. Accessory GT files for use as orifice openers are available in sizes of 0.12 taper in ISO sizes of 35, 50, 70 and 90.Negative rake angle of these files makes them to cut the dentin in planning motion.

CLINICAL TECHNIQUE

1. Obtain a straight line access to the canal orifice and establish the glide path using No. 15 stainless steel file (Fig. 12.54).

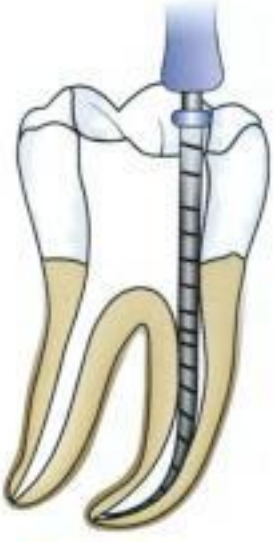


Fig. 12.54: Straight line access to canal orifices

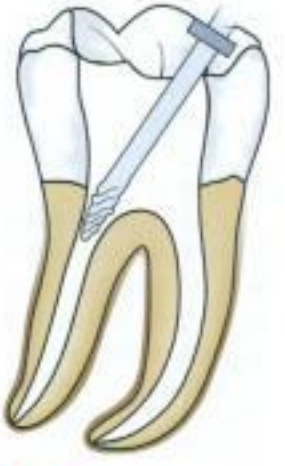


Fig. 12.55: Use of 0.12 GT files for coronal preparation

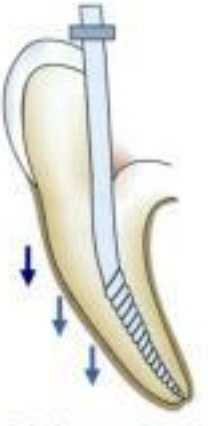


Fig. 12.56: Apical preparation using GT files

2. Lubricate the canal and use GT files (0.12, 0.10, 0.08 and 0.06 taper) in crown down fashion at 150 - 300 rpm (Fig. 12.55).
3. With GT files reach the 1/2 to 2/3rd of estimated working length, establish the correct working length using 15 number stainless steel file.
4. Select the final shaping instrument and penetrate in canal steadily. Remove the instrument, irrigate the canal and reinsert it. Continue to progress apically until working length is achieved (Fig. 12.56).
5. Apical gauging is done thereafter to assure smooth apical taper.

PROTAPER FILES

As we have seen that ProTaper files have a triangular cross section and is variably tapered across its cutting length. The progressively tapered design improves flexibility, cutting efficiency and the safety of these files.

The ProTaper system consists of three shaping and three finishing files.

1. Shaping files are termed as S_x, S_1 and S_2.
   - S_x these are files of shorter length of 19 mm with Do diameter of 0.19 mm and D_{14} diameter of 1.20 mm. The increase of taper up to D_9 and then taper decrease up to D_{14} increases its flexibility.
   - S_1 has Do diameter of 0.17 mm and D_{14} of 1.20 mm. it is used to prepare coronal part of the root.
   - S_2 has Do diameter of 0.20 mm and D_{14} of 1.20 mm. it is used to prepare middle third of the canal.
2. Finishing files F_1, F_2, F_3 are used to prepare and finish apical part of the root canal.
   - F_1 Do diameter and apical taper is 20 and 0.07.
   - F_2 Do diameter and taper is 25 and 0.08.
   - F_3 Do diameter and taper is 30 and 0.09.

CLINICAL TECHNIQUE

1. The foremost step is gaining straight line access to the canal orifices.
2. Establish a smooth glide path before doing any instrumentation with ProTaper system.
3. Now prepare the coronal third of the canal by inserting S_1 into the canal using passive pressure. Don't go more then third fourth of the estimated canal length (Fig. 12.57).
4. Irrigate and recapitulate the canal using number 10 file.
5. In shorter teeth, use of S_x is recommended.
6. After this S_2 is worked up to the estimated canal length.

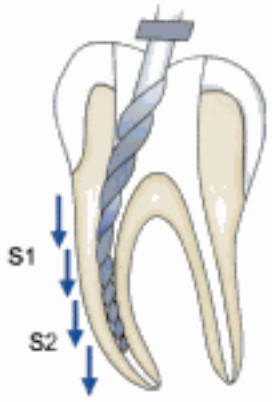


Fig. 12.57: Use of S_1 and S_2 file for canal preparation

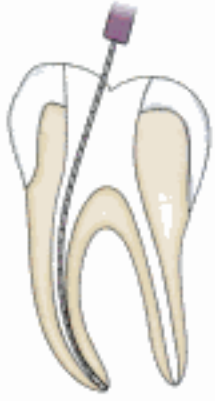


Fig. 12.58: Confirmation of working length using hand files

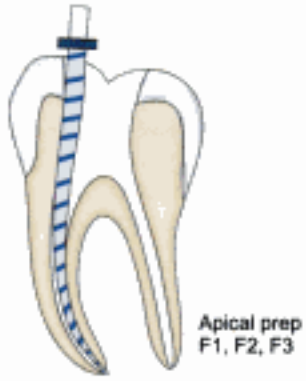


Fig. 12.59: Apical preparation using F1, F2, F3.

7. Now confirm the working length using small stainless steel K- files up to size 15 by electronic apex locators and/or with radiographic confirmation (Fig. 12.58).
8. Use F_1, F_2 and F_3 (if necessary) finishing files up to established working length and complete the apical preparation. Then refine the apical preparation using corresponding stainless steel file to gauge the apical foramen and to smoothen the canal walls (Fig. 12.59).

Advantages of ProTaper Files

1. ProTaper file has modified guiding tip which allows one to follow canal better and variable tip diameters allow file to have specific cutting action in defines area of canal without stressing instrument in other sections.
2. ProTaper file has a changing helical angle and pitch over their cutting blades which reduces the instrument from screwing into the canal and allows better removal of debris.

3. ProTaper file acts in active motion, this further increases its efficiency and reduces torsional strain.
4. Length of file handle is reduced from 15 to 12.5 mm. which allows better access in posterior areas.

QUANTEC FILE SYSTEM

Quantec file series are available in both cutting and non-cutting tips with standard size of 25 No. in 0.12, 0.10, 0.08, 0.06, 0.05, 0.04, 0.03 and 0.02 tapers. 0.02 tapered
This unique design of Quantec system that is a positive blade angle with two wide radial lands and relief behind the lands reduces its contact with the canal, thereby minimizing the torque. Quantec system utilizes the "graduated taper technique" to prepare a canal. It is thought that using a series of files of single taper result in the decreases efficiency as the larger instruments are used. This happens because more of file comes in contact with the dentinal wall which makes it more difficult to remove dentin. Thereby retarding the proper cleaning and shaping of the canal. But in graduated taper technique, restricted contact of area increases the efficiency of the instrument because now forces are concentrated on smaller area.

CLINICAL TECHNIQUE

1. Obtain the straight line access to the canal orifices.
2. Establish the patency of canal using number 10 or 15 stainless steel files.
3. Insert the Quantec number 25, taper 0.06 file passively into the canal.
4. After negotiation of the canal using Quantec file, prepare the canal from 0.12 to 0.03 taper.
5. Finally complete the apical preparation of canal using 40 or 45 No., 0.02 taper hand or rotary files.

LIGHT SPEED SYSTEM

These are so named because a "light" touch is needed as "speed" of instrumentation is increased.
Light speed instrument have non-cutting tip with Gates-Glidden in configuration and are available in 21, 25, 31 and 50 mm length and ISO numbers 20 -140.

Half sizes of light speed instrument are available in numbers 22.5, 27.5, 32.5.

CLINICAL TECHNIQUE

While doing cleaning and shaping using the light speed system, three special instruments are used -

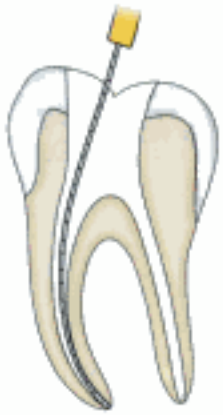


Fig. 12.60: Obtain straight line access

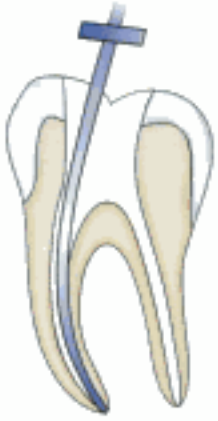


Fig. 12.61: Use of initial apical rotary in canal

- Initial Apical Rotary (IAR) (begins to cut canal walls at working length).
- Master Apical Rotary (MAR) (Last instrument to perform the apical preparation).
- Final Rotary (FR) (Last step back instrument which completes the step back procedure).

1. Obtain a straight line access to the canal orifice and establish a glide path using number 15 stainless steel file (Fig. 12.60).
2. Slightly (1-2mm) enlarge the canal orifice with the help of Gates-Glidden driils.
3. Determine the working length using number 15 stainless steel file.
4. Use initial apical rotary up to working length (Fig. 12.61).
5. Now prepare the canal using light speed instruments in forward and backward movement from smaller to larger number (Fig. 12.62).

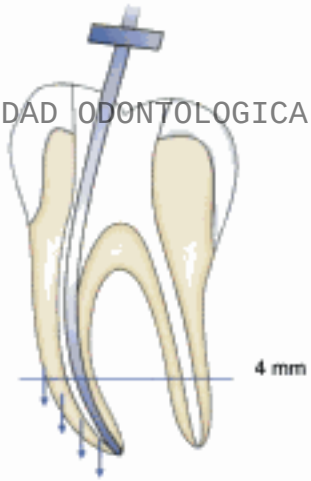


Fig. 12.62: Use of light speed instrument in forward and backward movement

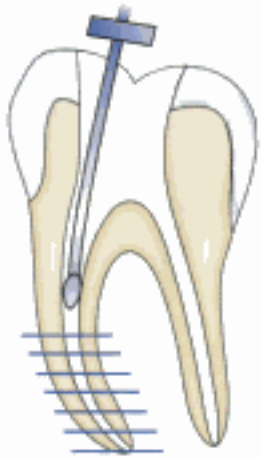


Fig. 12.63: Use of light speed instrument in step back procedure

6. Last instrument used in canal for canal preparation is master apical rotary which could be 5-12 size larger than the initial apical rotary instrument.
7. After using MAR, light speed instruments are used in step back procedure to complete the canal preparation (Fig. 12.63). Use the final rotary (FR) as the last back instrument.
8. Finally recapitulated with Master Apical Rotary (MAR) up to the working length.

ADVANTAGES OF LIGHT SPEED SYSTEM

1. Short cutting blades provide more accurate tactile feedback of canal preparation.
2. Flexibility of light speed system keeps it centered, virtually eliminating ledging, perforation or zipping of canal.
3. Light speed instrumentation is conservative which prevents weakening of the root.
4. When used correctly, risk of instrument separation is low but if it does separate, it is designed to separate 18 mm from the tip which makes its removal fast.
5. Short cutting blades with non-cutting shaft minimize the torque and stress on the instrument.

K_3 ROTARY FILE SYSTEM

K_3 files are available in taper of 0.02, 0.04 or 0.06 with ISO tip sizes.The presence of variable core diameter makes them flexible.K_3 files have positive rake angle providing them an effective cutting surface. Body shapers available in taper 0.08, 0.10, and 0.12 all with tip size 25, are used to prepare the coronal third of the canal.

CLINICAL TECHNIQUE

1. Obtain a straight line access to the canal orifices and enlarge them with K_3 shaper files. The shaper files are used to light resistance which is usually 3 to 4 mm apically.
2. After preparing coronal third of canal with shaper file, prepare the middle third of the canal.
   Obtain the glide path using number 15 stainless steel hand file before using K_3 system.
3. Then 0.06/40 K_3 can be inserted up to middle third of the canal. If it is difficult to use, switch over smaller files (0.06/35). On the whole 0.06/40 is used first followed by 0.06/35,0.06/30 etc. until the middle third and apical third is reached.
4. Don't forget to irrigate and recapitulate in between the files.
5. In narrow canals use 0.04 tapered files instead of 0.06 taper.
6. Now prepare the apical third of the canal using smaller K_3 files upto estimated working length.

REAL WORLD ENDO SEQUENCE FILE

A recently introduced in NiTi world is Real World Endo Sequence File system. Electropolishing treatment of these files during manufacturing lessens the propensity of NiTi files for crack propagation. The blank design with alternating contact points (ACPs) and absence of radial lands, makes the instrument shaper and more efficient in cutting.

CLINICAL TECHNIQUE

1. Gain the straight line access to canal orifice and confirm the coronal patency with number 10 or 15 stainless steel hand file.
2. Use Expeditor file first into canal to determine the appropriate size of the canal.
3. Remove the Expeditor file from the canal, irrigate the canal and choose appropriate sequence file according to the canal size and perform crown down technique.
4. Now establish the working length of the canal, after using the second rotary file.
5. Complete the crown down technique up to the established working length.

Advantages of Real World Endo Sequence File System

1. These files are available in 0.04 and 0.06 taper having the precision tip. Presence of precision tip results in both safety as well as efficiency.
2. These files keep themselves centered in the canal and produce minimal lateral resistance because of
   i. Presence of ACPs
   ii. Electropolishing
   iii. Absence of radial lands.

Sequence files have variable pitch and helical angle which further increase its efficiency by moving the debris out of canal and thus decreasing the torque caused by debris accumulation.

HERO 642

HERO 642 (High Elasticity in Rotation, 0.06. 0.04 and 0.02 tapers-) has trihelical hedstorm design with sharp flutes. It is used in "Crown down" technique, between 300 and 600 rotations per minute (rpm) in a standard slow speed contraangle air driven or electric motors.

Due to progressively increasing distance between the flutes-reduced risk for binding of the instrument in root canal.

Technique

In this crown down technique is achieved using variable size and taper. First and foremost step of canal preparation is to obtain straight line access to the canal orifices.

1. Start with size 30 of 0.06 taper, penetrate it in the canal with light up and down motion at the speed of 300-600 RPM and prepare the coronal part of the canal (Fig. 12.64).

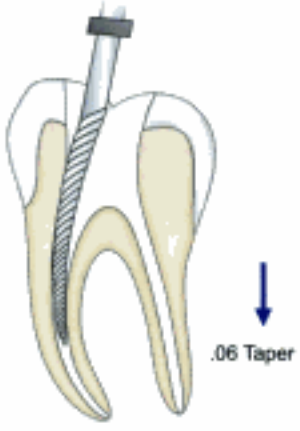


Fig. 12.64: 0.06 taper file to prepare coronal two third of the canal

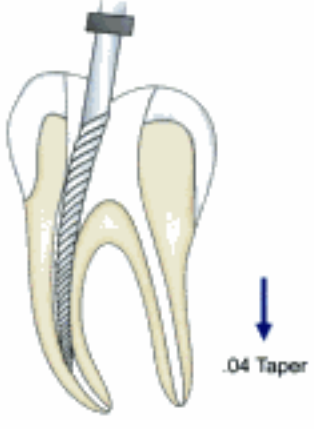


Fig. 12.65: Use of 0.04 taper file for mid root preparation

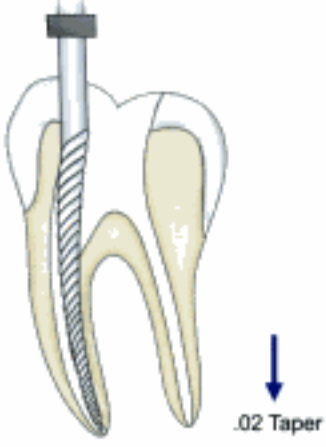


Fig. 12.66: Use of 0.02 tapered instrument for final apical preparation

2. Remove the file, irrigate the canal
3. Now insert file size 30, 0.04 taper and continue the canal preparation up to the level of 2mm of working length (Fig. 12.65).
4. Finally complete the apical preparation using NiTi hand instruments or 0.02 taper rotary (Fig. 12.66).

CANAL PREPARATION USING ULTRASONIC INSTRUMENTS

The concept of using ultrasound in endodontic therapy was suggested in 1957 by **Richman**. But it was the late 1970's, when ultrasonic scaling units began to be adapted for use in endodontics resulting in endosonics, since that time lot of research is being done on endosonics to make it an integral part of endodontics. The machines used for this purpose are designed to transmit low frequency ultrasonic vibration by conversion of electromagnetic energy to the mechanical energy to produce oscillation of file. File oscillates at the frequency of 20,000–25, 000 vibrations/seconds (Fig. 12.67).

During the oscillation of file, there is continuous flow of irrigants solutions from the handpiece along the file. This causes formation of cavitation, i.e. by the movement of the file within water supply. Cavitation is growth and subsequent violent collapse of the bubbles in the fluid which results in formation of a shock wave, increase temperature, pressure and the free radical formation in the fluid. Cavitation is considered as one of the primary beneficial effect in endosonics.

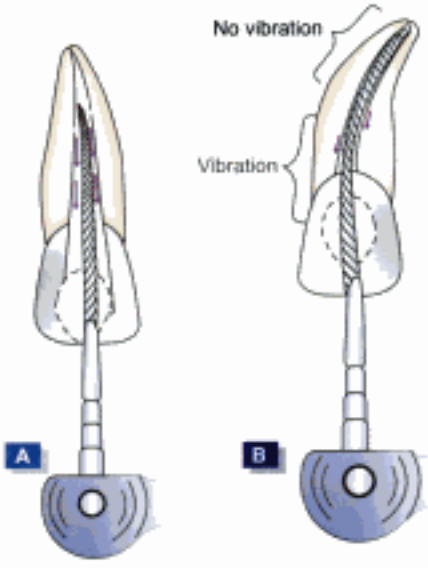


Fig. 12.67: **(A)** Ultrasonic instrument and irrigation work actively in straight canal. **(B)** Curvature in canal may impede vibration

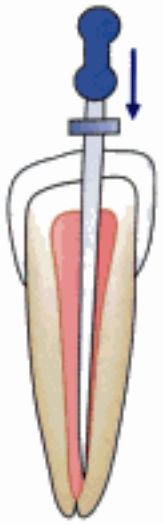


Fig. 12.68: Spreader should reach 1 mm short of apex in a well prepared canal

Another effect is acoustic steaming which is produced around an object oscillating in a liquid. In this, there is production of shear forces which are capable of dislodging the lumps of material. Thus, acoustic steaming may be useful in reducing the number of smear layer and loosening the aggregates of the bacteria (Fig. 12.68).

Technique

1. Before starting with ultrasonic instrumentation apical third of the canal should be prepared to at least size 15 file.
2. After activation, ultrasonic file is moved in the ***circumferential*** manner with push - pull stroke along the walls of canal.
3. File is activated for one minute. This procedure is repeated till the apex is prepared to at least size 25.

The root canal debridement depends on:

1. Choice of irrigant solution (sodium hypochlorite is irrigant of choice).
2. Oscillation of file.
3. The form of irrigation with ultrasonic irrigation being supplied.

Advantages of Ultrasonic Canal Preparation

1. Less time consuming.
2. Produce cleaner canals because of synergetic relationship between the ultrasound and the sodium hypochlorite.

3. Heat produced by ultrasonic vibration increase the chemical effectiveness of the sodium hypochlorite.

Disadvantages

1. Increased frequency of canal transportations.
2. Increased chances of overinstrumentation.

CANAL PREPARATION USING SONIC INSTRUMENTS

Design of sonic instruments is similar to that of ultrasonics. They consist of a driver on to which an endosonic file is attached. The oscillatory pattern of driver determines the nature of movement of the attached file. In sonic instruments, there is longitudinal pattern of the vibration when activated in the root canal. This longitudinal file motion produces superior cleaning of the root canal walls.

Sonic system uses three types of file system for root canal preparation viz. Heliosonic, Rispisonic and the canal shaper instruments. These files have spiral blades protruding along their length and non cutting tips.

Technique

1. After gaining the straight line access to the canal orifices, penetrate small number file in the canal. Enlarge the canal upto 20 or 25 number file upto 3 mm of the apex to make some space of sonic file.
2. Now insert the sonic file 0.5 - 1 mm short of number 20 file, and do circumferential filling with up and down motion for 30 - 40 seconds.
3. Next use the larger number sonic file and do the coronal flaring.
4. After completion with this, determine the working length and prepare the apical third of the canal with hand files.
5. Finally blend the apical preparation with coronal preflaring with smaller number sonic file.

Though sonic files have shown to enlarge and debride the canals effectively in lesser time but care should be taken not to force the file apically to prevent instrument separation, ledge formation or canal transportation.

LASER ASSISTED ROOT CANAL THERAPY

Weichman and Johnson in 1971 were the first to suggest the use of lasers in endodontics. The most important benefits of this revolutionary technology for endodontic treatment is the ease of using it and great degree of patient comfort during and after the procedure.

Nd:YAG, Ar, Excimer, Holium, Ebrium laser beam are delivered through the optical fiber with the diameter of 200-400 µm equivalent to size 20 - 40 number file. Studies have shown different results with lasers.

Bahcall et al in 1992 found that though the use of Nd:YAG laser can be produce cleaner canals, but heat produced by it may damage the surrounding supporting tissues, i.e. bone and PDL. Hibst et al showed that use of Er:YAG laser may pose less thermal damage to the tissues because it causes localized heating thereby minimizing the absorption depth.

Recently a new root canal treatment using the Er, Cr:YSGG (erbium, chromium : Yettruim scandium gallium garnet) has been introduced to help reduce the patient fear and provide better comfort to the patient. The device which provides such a treatment is the waterlase—Hydrokinetic Hard and Soft tissue laser, the only laser system to receive FDA clearance for complete endodontic therapy and other root canal procedures. This laser uses specialized fibers of various diameters and lengths to effectively clean the root canal walls and prepare the canal for obturation. By using hydrokinetic process in which water is energized by the YSGG laser photons to cause molecular excitation and localized microexpansion, hard tissues are removed precisely with no thermal side effects.

With this technique there is minimal patient discomfort, and postoperative complications such as inflammation, swelling and pain. Moreover, antibacterial action of YSGG laser has reduced the use of postoperative antibiotics therapy. Thus we see that laser is one of the important revolution in endodontics. The intracanal irradiation with laser has shown to reduce the microbial reduction, inflammation, and other post operative complications, simultaneously providing the comfort to patient. However performance of the equipment safety measures, temperature rise and level of microbial reduction should be well documented before it becomes a current method of choice for treatment.

EVALUATION CRITERIA OF CANAL PREPARATION

1. Spreader should be able to reach within 1 mm of the working length if spreader does not reach the estimated length, it indicates canal is not well prepared (Fig. 12.68).
2. After canal preparation, when master apical file is pressed firmly against each walls should feel smooth (Fig. 12.69).

Recently a three dimensional, nondestructive technique has developed for detailed study of root canal geometry. A micro - computed tomography scanner is used to record

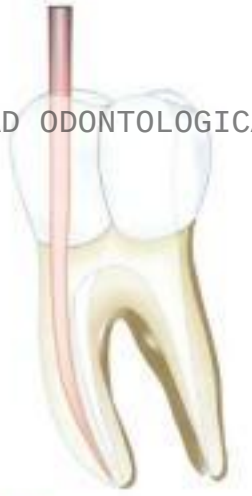


Fig. 12.69: Master apical file should feel smooth in a well prepared canal

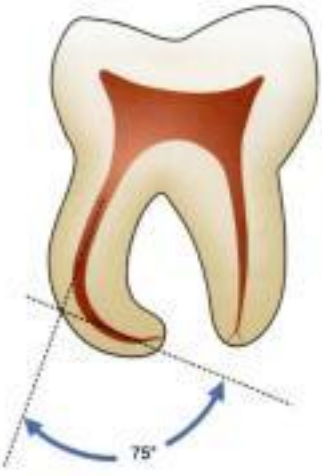


Fig. 12.70: Internal angle formed by interaction of lines forms the angle of curvature

the precise canal anatomy before and after the instrumentation. A three dimensional analysis of root canal geometry by high resolution CT is then performed.

SPECIAL ANATOMIC PROBLEMS IN CANAL CLEANING AND SHAPING

1. Management of curved canals.
2. Management of calcified canals.
3. Management of C-shaped canals.
4. Management of S-shaped canals

1. Management of Curved Canals

The controlled, uniformly tapered radicular preparation is a great challenge in endodontics. But now a days, introduction of flexible non-ISO taper NiTi instruments have brought a major change to overcome the problems of cleaning and shaping in curved canals earlier caused by use of stiff stainless steel files in push-pull motion.

In management of curved canals first of all estimate the angle of curvature. To calculate angle of curvature, imagine a straight line from orifice towards canal curvature and another line from apex towards apical portion of the curve. The internal angle formed by interaction of these lines is the angle of curvature (Fig. 12.70).

In curved canals, frequently seen problem is occurrence of uneven cutting. File can cut dentine evenly only if it engages dentine around its entire circumference. Once it becomes loose in a curved canal, it will tend to straighten up and will contact only at certain points along its length. These areas are usually outer portion of curve apical to the

Fig. 12.71: Formation of ledge in a curved canal

curve, on inner part of curve at the height of curve and outer or inner curve coronal to the curve. All this can lead to occurrence of procedural errors like formation of ledge, transportation of foramen, perforation or formation of elbow and zip in a curved canal (Fig. 12.71). To avoid occurrence of such errors there should be even contact of file to the canal dentine. But because file has tendency to straighten up to its original shape and it is difficult to control removal of dentine along the entire length of file in push- pull motion, the above errors can be reduced by:

- Decreasing the restoring force by means of which straight files apt to bend against the curved dentine surface.
- Decreasing the length of file which is aggressively cutting at the given span.

Decrease in the force can be done by :

i. ***Precurving the file:*** A precurved file has shown to traverse the curve better than a straight file. Two types of precurving are done : (Fig. 12.72)

- Placing a gradual curve for the entire length of the file.
- Placing a sharp curve of nearly 45 degrees near the apical end of the instrument. This type of curved file is used in cases when a sharp curve or an obstruction is present in the canal. Curve can be placed by grasping the flutes with gauze sponge and carefully bending the file until the preferred curvature is attained.

  Once the precurved file is placed in the canal, there are chances of loosing the direction of curve. To avoid this problem teardrop shape rubber stopper is usually recommended with point showing the direction of the curve.

ii. ***Extravagant use of smaller number files:*** Since smaller sized instruments can follow the canal curvature because of their flexibility, they should be used until the larger files are able to negotiate the canal without force.

iii. ***Use of intermediate sizes of files:*** It has been seen that increment of 0.05 mm between the instruments is too large to reach the correct working length in curved canals. To solve this problem, by cutting off a portion of the file tip a new instrument size is created which has the size intermediate to two consecutive instruments. There is increase of 0.02 mm of diameter per millimeter of the length, cutting 1 mm of the tip of the instrument creates a new instrument size, for example cutting 1 mm of a number15 file makes it number 17 file. In severely curved canals the clinician can cut 0.0 5mm of the file to increase the instrument diameter by 0.01mm. This allows the smoother transition of the instrument sizes to cause smoother cutting in curved canals.

iv. ***Use of flexible files:*** It has been seen that use of flexible files cause less alteration of the canal shape than the stiffer files. Flexible files help in maintaining the shape of the curve and avoid occurrence of procedural errors like formation of ledge, elbow or zipping of the canal.

Decrease in length of actively cutting file can be achieved by:

a. ***Anticurvature filing:*** In some roots like mesial root of mandibular molar and mesiobuccal root of maxillary molars, if care is not taken while preparing them, incidence of strip formation are

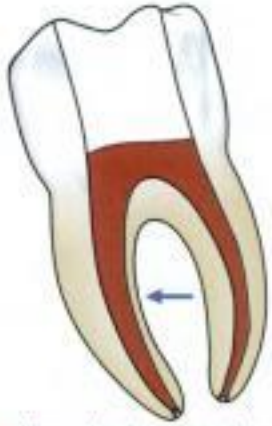


Fig. 12.73A: Arrow showing area where chances of strip perforation are more

Fig. 12.72: Precurving of file

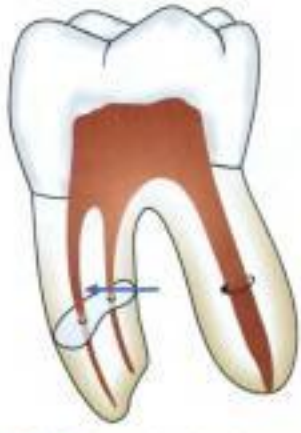


Fig. 12.73B: Arrow showing danger zone

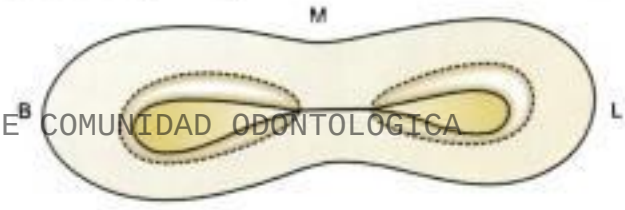


Fig. 12.74: Removal of dentin should be done more in shaded area to avoid perforation

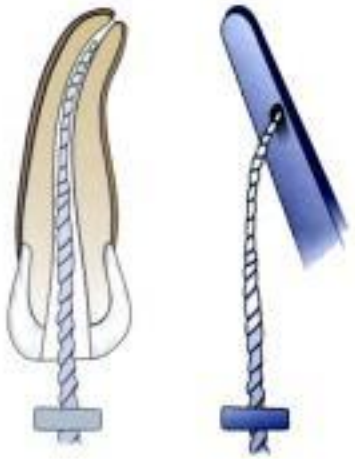


Fig. 12.75: Duling of flutes is done with the help of diamond file

Fig. 12.76: Crown down technique for curved canals

seen. The canal wall facing the curve or furcation makes the danger zone because in this portion there is lees of the tooth structure as compared to the outer portion (safety zone) (Figs 12.73A and B). In these teeth, to reduce the incidence of strip perforation, the concept of anticurvature filing is employed as given by Lim and Stock. It involves lesser filing of the canal wall which is facing the curvature (Fig. 12.74). For example in case of filing of mesiobuccal canal of mandibular molars, more filing is done on mesial and buccal wall as compared to distal and lingual walls.

b. ***Modifying cutting edges of the instrument:*** The cutting edges of the curved instrument can be modified by dulling the flute on outer portion of the apical third and inner portion of the middle third. Dulling of the flutes can be done with the help of diamond file (Fig. 12.75).

c. ***Changing the canal preparation techniques:*** crown down technique, i.e. preparation of coronal part of the canal before apical part removes the coronal interferences and allow the files to reach up to the apex more effectively (Fig. 12.76).

2. Management of Calcified Canals

Calcifications in the root canal system are commonly met problem in root canal treatment. The dentist must recognize that pulpal calcifications are signs of the pathosis, not the cause (Fig. 12.77). Various etiological factors seen to be associated with calcifications are caries, trauma, drugs and aging.

Success in negotiating small or calcified canals is predicted on a proper access opening and identification of the canal orifice or orifices.

To locate the calcified orifice, first mentally visualize and plan the normal spatial relationship of the pulp space

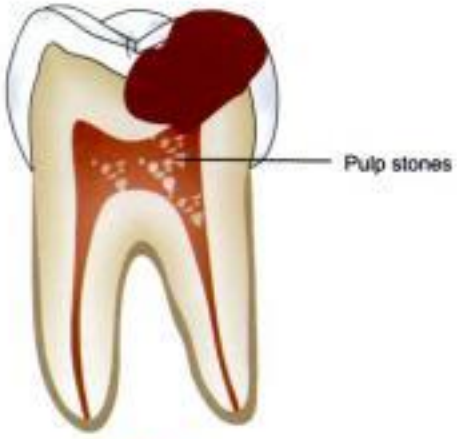


Fig. 12.77: Calcified canals

Fig. 12.78: Rotary instrument is directed towards assumed pulpal space

Fig. 12.79: Radiographic evaluation of a calcified tooth helps in knowing location of pulp chamber.

onto a radiograph of calcified tooth. Then the two dimensional radiographic image is correlated with the three-dimensional radiographic image. After this access preparation is initiated, with the rotary instrument directed toward the assumed location of pulpal space (Fig. 12.78). Accurate radiographic visualization and intermittent evaluation of bur penetratio 006E and orientation helps to recognize the calcified orifice.

In a tooth with a calcified pulp chamber, the distance from the occlusal surface to the pulp chamber is measured from the preoperative radiograph (Fig. 12.79). The geometric patterns of canal orifices and their variations have to be mentally projected on the calcified pulp chamber floor.

Fig. 12.80: Use of sharp endodontic explorer in tentative location of orifice

Location of the Orifice and Penetration

The most significant instrument for orifice location is the DG – 16 explorer. If an orifice is present, firm pressure will force the instrument slightly into the orifice, and it will "stick" (Fig. 12.80). At this suspected point a fine instrument number 8 or 10 K-file, is placed into the orifice, and an effort is made to negotiate the canal. An alternative choice is to use instruments with reduced flutes, such as a canal pathfinder which can penetrate even highly calcified canals. Although most of the attempts to locate canal orifices with calcifications are successful still there is a probability for perforation. Probing with the explorer yields a characteristic "stick" but if explorer lies too close to the root surface, it actually penetrates a thin area of remaining dentin. The most common sign of accidental perforation is bleeding, but bleeding may also indicate that the pulp in the calcified canal is vital. If there is any doubt as to whether the orifice has actually been found, place a small instrument in the opening and take a radiograph.

Penetration and Negotiation of the Calcified Canal

Once the orifice has been located, No. 8 K-file is penetrated into the canal to negotiate the calcified canal (Fig. 12.81). A No. 10 K-file is too large, and a No. 6 K-file is too weak to apply any firm apical pressure. Also the use of Nickel-Titanium files is contraindicated for this purpose because of lack of strength in the long axis of the file. Before the

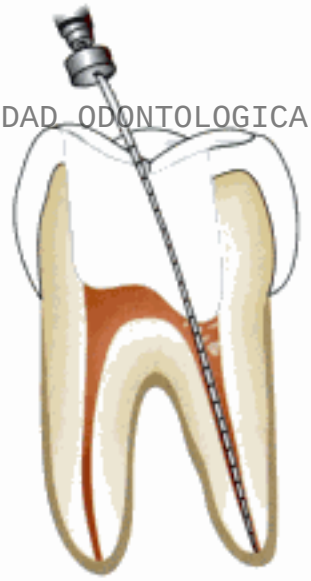


Fig. 12.81: Use of sharp, small instrument to negotiate calcified canal

file is inserted into the canal, a small curve is placed in its apical 1 mm. In negotiating the fine-curved canal, the precurved instrument must be positioned along the pathway the canal is most likely to follow; as a result it is important to know in which direction the curve in the instrument is pointed. This is easily accomplished by observing the rubber stop on the instrument shaft.

The recurrent effect of forceful probing with fine instruments and chelating agents is the creation of a false canal and continued instrumentation in a false canal results in perforation. In a calcified canal, it is necessary to confirm the position of the instrument with a radiograph. In cases of teeth with calcified canals, the prognosis of the root canal treatment depends on the continued health of the pulp or the periradicular tissues on the apical side of the blockage. In the absence of symptoms or evidence of apical pathosis, it is clinically practical and satisfactory to instrument and fill the canal to the level negotiated, followed by regular recall of the patient.

Guidelines for Negotiating Calcified Canals

1. Copious irrigation at all times with 2.5 to 5.25 percent NaOCl enhances dissolution of organic debris, lubricates the canal, and keeps dentin chips and pieces of calcified material in solution.
2. Always advance instruments slowly in calcified canals.
3. Always clean the instrument on withdrawl and inspect before reinserting it into the canal.
4. When a fine instrument has reached the approximate canal length, do not remove it; rather obtain a radiograph to ascertain the position of the file.
5. Use chelating agents to assist in canal penetration.
6. Flaring of the canal orifice and enlargement of coronal third of canal space improves tactile perception.
7. The use of recent nickel - titanium rotary orifice penetrating instruments is also beneficial.
8. Well angulated periapical and bite using radiographs should be taken. They not only indicate the position of canals but also give important information about the relative position of canal orifice in calcified cases. Failure to recognize changes in the axis of the tooth that occurs during crown restoration, can lead to perforations. Proximal restorations can be used as guide to locate canals.
9. Not anesthetizing the patient while performing access opening can be useful in some cases. Patient should be told to indicate when he/she feels a sharp sensation during access with a bur. At that point a sharp DG 16 Endo explorer is used to locate the canal. It is easy to tell the difference between PDL and pulp with a small file. If file is inserted only a mm or two into the pulp, the reaction will be sharp. If it is in PDL, reaction is often less sharp.
10. Avoid removing large amount of dentin in the hope of finding a canal orifice. By doing this all the pulp floor landmarks are lost also the strength and dentinal thickness of tooth gets compromised.
11. Small round burs should be used to create a glide path to the orifice. This will further ease the instruments into the proper lane to allow effortless introduction of files into the canals.

3. Management of C-shaped Canals

Though the prevalence of C-shaped canals is low, but those requiring endodontic treatment present a diagnostic and treatment difficulties to the clinician. Some C-shaped canals are difficult to interpret on radiographs and often are not identified until an endodontic access is made. These are commonly seen in mandibular second molars and maxillary first molars especially when roots of these teeth appear very close or fused (Fig. 12.82).

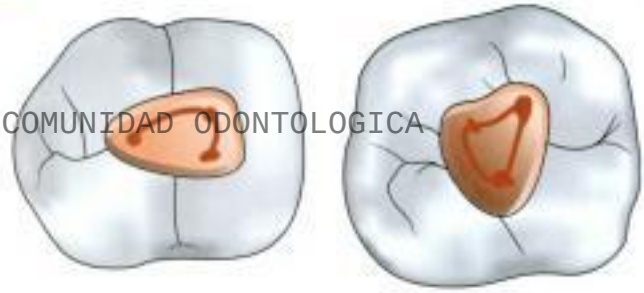


Fig. 12.82: Maxillary and Mandibular molar showing C-shaped canal anatomy

In maxillary molars, the C-shaped canal includes mesiobuccal and palatal canals or the distobuccal and palatal canals. In the mandibular second molar, the C-shaped canal includes mesiobuccal and distal canals.

In any of these cases, canal orifices may be found within the C-shaped trough or the C-shape may be continuous throughout the length of the canal.

Major problems come across during bio-mechanical preparation of C-shaped canals are difficulty in removing pulp tissue and necrotic debris, excessive hemorrhage, and persistent discomfort during instrumentation. Because of large volumetric capacity of the C-shaped canal system, along with transverse anastomoses and irregularities, continuous circumferential filing along the periphery of the C with copious amounts of 5.25 percent NaOCl is necessary for maximum tissue removal and for control of bleeding. If hemorrhage continues, ultrasonic removal of tissue or placement of calcium hydroxide between appointments may be used to enhance tissue removal and control hemorrhage. Over preparation of C-shaped canals should be avoided, because of presence of only little dentin between the external root surface and the canal system in these teeth.

4. Management of S-shaped Canals

S-shaped or bayonet shaped canals pose great problems while endodontic therapy, since they involve at least two curves, with the apical curve having maximum deviations in anatomy. These double curved canals are usually identified radiographically if they cross in mesiodistal direction. If they traverse in a buccolingual direction, they may be recognized with multi-angled radiographs, or when the initial apical file is removed from the canal and it simulates multiple curves. S-shaped canals are commonly found in maxillary lateral incisors, maxillary canines, maxillary premolars, and mandibular molars.

For optimal cleaning and shaping of S-shaped canals, the three-dimensional nature of these canals must be visualized with special consideration and evaluation to the multiple concavities along the external surfaces of the root. Failure to know these may result in stripping of the canal along the inner surface of each curve. During initial canal penetration, it is essential that there be an unrestricted approach to the first curve. For this, the access preparation is flared to allow for a more direct entry. Once the entire canal is negotiated, passive shaping of the coronal curve is done first, to facilitate the cleaning and shaping of the apical curve. Constant recapitulation with small files and copious irrigation is necessary to prevent blockage and ledging in the apical curve. Over curving the apical 3mm of the file aids in maintaining the curvature in the apical portion of the canal as the coronal curve becomes almost straight during the later stages of cleaning and shaping. Gradual use of small files with short amplitude strokes is essential to manage these canals effectively. To prevent stripping in the coronal curve, anticurvature or reverse filing is recommended, with primary pressure being placed away from curve of coronal curvature.

13 Obturation of Root Canal System

- Introduction
- History
- Timing of Obturation
- Materials Used for Obturation
- Root Canal Sealers
- Classification of Root Canal Sealers
- Obturation Techniques
- Lateral Compaction Technique
- Chemical Alteration of Gutta-Percha
- Vertical Compaction Technique
- System-B Continuous Wave of Condensation Technique
- Lateral/Vertical Compaction Warm Gutta-Percha
- Sectional Method of Obturation
- MC Spadden Compaction
- Thermomechanical Injectable Gutta-percha
- Solid Core Carrier Technique
- Silver Cone Obturation
- Apical Third Filling
- Post Obturation Instructions

INTRODUCTION

Root canal therapy may be defined as the complete removal of the irreversibly damaged dental pulp followed by thorough cleaning, shaping and filling of the root canal system that the tooth may remain as a functional unit with in the dental arch (Fig. 13.1). The rationale of root canal treatment relies on the fact that the nonvital pulp, being avascular, it has no defense mechanisms. The damaged tissue within the root canal undergoes autolysis and the resulting break down products will diffuse into the surrounding tissues and cause periapical irritation associated with the portals of exit even in the absence of bacterial contamination. The complex anatomy of the apical delta of many root canals makes complete debridement virtually impossible. It is essential therefore, that endodontic therapy must include sealing of the root canal system to prevent tissue fluids from percolating in the root canal and prevent toxic by-products from both necrotic tissue and microorganisms regressing into the periradicular tissues (Figs 13.2 and 13.3).

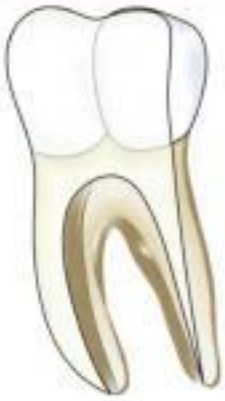


Fig. 13.1: Complete cleaning and shaping of root canal system

The current accepted method of obturation of prepared canals employs a solid or a semisolid core such as gutta-percha and a root canal sealer. Gutta-percha has no adhesive qualities to dentin regardless of the obturation technique used. Therefore root canal sealers along with solid core

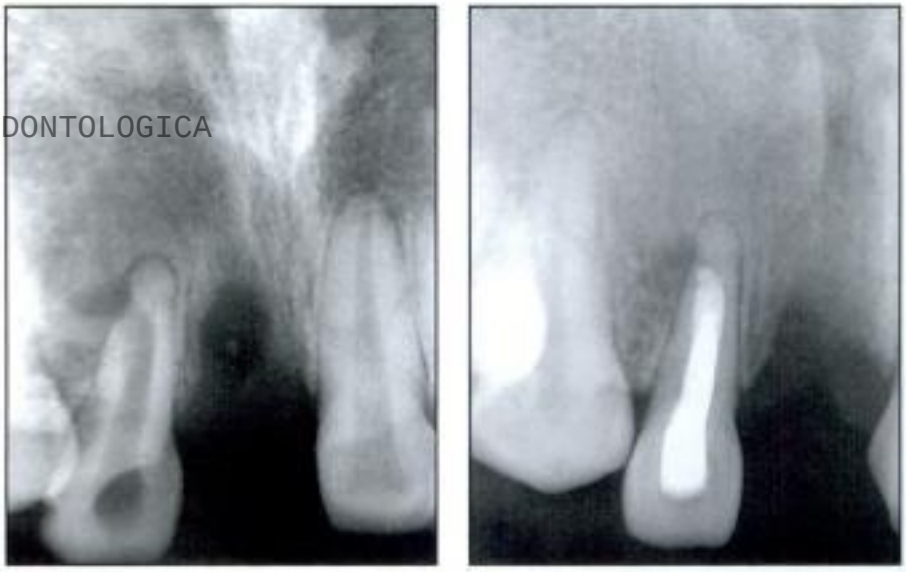


Fig. 13.2: Radiograph showing three dimensional obturation of canal in pulpally involved maxillary lateral incisor (maxillary)

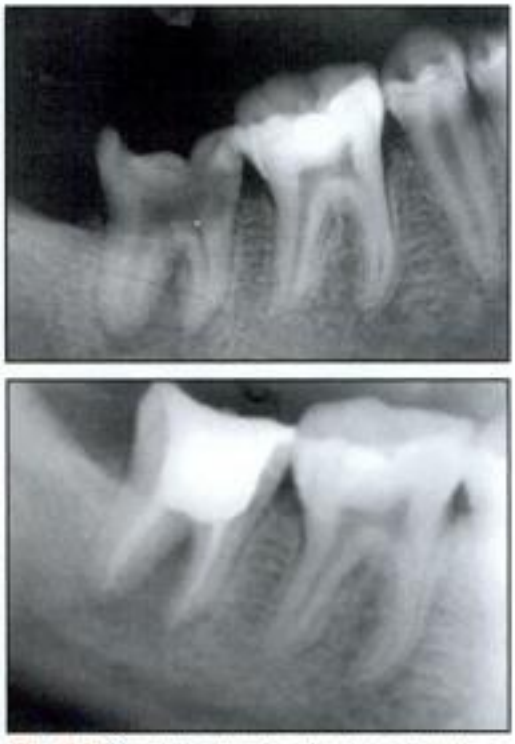


Fig. 13.3: Complete obturation of root canal system in grossly carious mandibular second molar

play a major role in achieving the hermetic seal by filling the accessory root canals, voids, spaces and irregularities and hence reducing the chances of failure of root canal treatment.

Not all teeth with positive bacterial cultures fail, nor do all teeth with negative cultures succeed. Thus entombing residual microorganisms and irritants by sealing them within the root canal system may have a major influence on clinical outcome. Leakage of fluids through an obturated root canal can occur between the sealer and dentin, the sealer and gutta-percha, or through the voids with in the sealer (Fig. 13.4). Although sealers enhance sealing ability by filling in any residual spaces and bonding to dentin, the optimal outcome of obturation is to maximize the volume of the core material and minimize the amount of sealer between the inert core and the canal wall.

Fig. 13.4: Leakage in an obturated canal leading to root canal failure

Thus we can say that a three dimensional well fitted root canal with fluid tight seal is the main objective of the root canal obturation. It serves the following:

a. Prevents percolation and microleakage of periapical exudate into the root canal space.
b. Prevents infection by completely obliterating the apical foramen and other portals of communication.
c. Creates a favorable environment for process of healing to take place.

History

1757 - Carious teeth were extracted, filled with Gold/lead and replanted again.
1847 - Hill's stopping was developed.
1867 - CA Bowman claimed to be the first to use gutta-percha for root canal filling.
1914 - Lateral condensation technique was developed by Callahan.
1953 - Acerbach advised filling of root canals with silver wires.
1961 - Use of stainless steel files in conjunction with root canal sealer as given by Sampeck.
1979 - Mc Spadden technique.

TIMING OF OBTURATION

Various factors like patient symptoms, pulp and periradicular status and procedural difficulties affect the timings of obturation and number of appointment.

Patient Symptoms

- If patients presents with sensitivity on percussion, it indicates inflammation in periodontal ligament space, canal should not be obturated before the inflammation has subsided.
- In case of irreversible pulpitis, obturation can be completed in single visit if the main source of pain, i.e. pulp has been removed.

Pulp and Periradicular Status

- Teeth with vital pulp can be obturated in same visit.
- Teeth with necrotic pulp may be completed in single visit if tooth is asymptomatic.
- Presence of even a slight purulent exudate may indicate possibly of exacerbation. If canal is sealed, pressure and subsequent tissue destruction may proceed rapidly.

Negative Culture

The reliance on negative cultures has decreased now since the researches have shown that false negative results can give inaccurate assessment on microbial flora, also the positive results do not indicate the potential pathogenicity of bacteria.

MATERIALS USED FOR OBTURATION

After the pulp space has been prepared appropriately, it must be obturated with a material which is capable of completely preventing communication between oral cavity and periapical tissue. The prepared apical connective tissue wound area can not heal with epithelium, thus root canal filling material placed against this wound serves as an alloplastic implant. There are expectations which make the selection of a good obturation material. These materials may be introduced into the canals in different forms and may be manipulated by different ways. Grossman grouped acceptable filling materials into plastics solids, cements and pastes. He also delineated ***ten requirements for an ideal root canal filling materials.***

Characteristics of An Ideal Root Canal Filling Material

- Easily introduced in the canal.
- Seal canal laterally and apically.
- Dimensionally stable after being inserted.
- Impervious to moisture.
- Bacteriostatic or at least should not encourage bacterial growth.
- Radiopaque.
- Non staining to tooth structure.
- Non irritating.
- Sterile/easily sterilized.
- Removed easily from canal if required.

Most commonly used material for obturation of root canal space is the Gutta-Percha, though silver points are also used for root canal obturation.

Gutta-Percha

Gutta-percha was initially used as a restorative material and later developed into an indispensable endodontic filling material.

Gutta-percha is derived from two words.
"GETAH" - meaning gum
"PERTJA" - name of the tree

History

- In 1843, it was first introduced by Sir Jose d Almeida to Royal Asiatic society of England.
- Edwin Truman was the first man who introduced gutta-percha to dentistry as a temporary filling material.

- Hill (1847) introduced a new restorative material under the name of "Hill's Stopping" (a mixture of bleached gutta-percha and carbonate of lime and quartz).
- Bowman (1867) was first to use gutta-percha as root canal filling material.
- Perry (1883) used Gold wire wrapped with gutta-percha to pack it in to the root canal.
- SS White (1887) was first company to start the commercial manufacture of gutta-percha points.
- Rollins (1893) used gutta-percha along with pure oxide of mercury into root canal filling.
- Callahan (1914) has done softening and dissolution of gutta-percha with use of rosins and then used in obturating the canals.
- Ingle and Levine (1959) were first persons to propose standardization of root canal instruments and filling materials.

Historical Background

- 1843 : ***Sir Jose d Almeida*** – First introduced
- In Dentistry : ***Edwin Truman*** – First introduced
- 1847 : ***Hill*** – Introduced Hill's stopping
- 1867 : ***Bowman*** – As root canal filling material
- 1883 : ***Perry*** – Packed gold wire wrapped with gutta-percha in root canals
- 1887 : ***SS White company*** – Commercial manufacturing
- 1893 : ***Rollins*** – Gutta-percha with pure oxide of mercury in root canals
- 1914 : ***Callahan*** – Softening and dissolution with rosins to use in root canals
- 1959 : ***Ingle and Levine*** – Standardization of root canal instruments and filling materials.

Despite its widely usage in endodontics, gutta-percha was earlier used as splints for holding fractured joints, to control hemorrhage in extracted sockets, in various skin diseases such as psoriasis, eczema and in manufacturing of golf balls (known as ***"Gutties"*** in past).

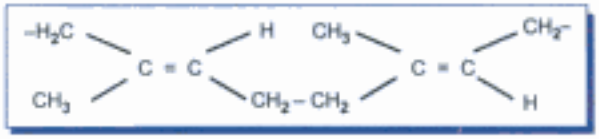
Sources

Gutta-percha is a dried coagulated extract which is derived from plants of ***Brazillian tree (Palaquium)***, belongs to family of Sapotaceae family. In India, these trees are found in ***Assam and Western Ghats***.

Chemistry

Its molecular structure is close to natural rubber, which is also a cis-isomer of polyisoprene.

Chemical Structure



In Crude Form, the Composition of Gutta-percha is

Gutta	-	75 - 82%
Alban	-	14 - 16 %
Fluavil	-	04 - 06%

* Also contains tannins, salts and saccharine.

Composition of Commercially Available Gutta-Percha (Given by Friedman et al)

• Matrix (Organic)	Gutta-percha	20%
• Filler (Inorganic)	Zinc Oxide	66%
• Radioopacifiers (Inorganic)	Heavy metal sulfates	11%
• Plasticizers (Organic)	Waxes or resins	3%

In other words,

- Organic Content – Gutta-percha + Waxes = 33%
- Inorganic content – Zno + Metal Sulfates = 77%

Chemically pure gutta-percha exists in two distinctly different crystalline forms, i.e. α and β forms which differ in molecular repeat distance and single bond form. Natural Gutta-percha coming directly from the tree is in α – form while the most commercial available product is in β – form (Fig. 13.5).

During Process of Manufacturing, if Gutta-percha is:

- Heated at 115°F (46°C) - β changes to α form
- Cooled slowly (less than 0.5° C/hr) - α forms
- Cooled rapidly - β forms

* Heated at temperature 140°F (54°C)-unstable (γ) form

Phases of Gutta-percha

These phases are interconvertible.

- α - runny, tacky and sticky (lower viscosity)
- β - solid, compactable and elongatable (higher viscosity)
- γ - unstable form
- On heating, gutta-percha expands which accounts for increased volume of material which can be compacted into the root canal. Gutta-percha shrinks as it returns to normal temperature. So, vertical pressure should be

Fig. 13.5: Gutta-percha cones

applied in all warm gutta-percha technique to compensate for volume change when cooling occurs (Schilder et al)

- Aging of gutta-percha causes brittleness because of the oxidation process (Fig. 13.6). Storage under artificial light also speeds up their deterioration. This brittle gutta-percha can be rejuvenated by a technique described by ***Sorien and Oliet***. In this, gutta-percha is immersed in hot water (55°C) for one or two seconds and then immediately immersed in cold water for few seconds.

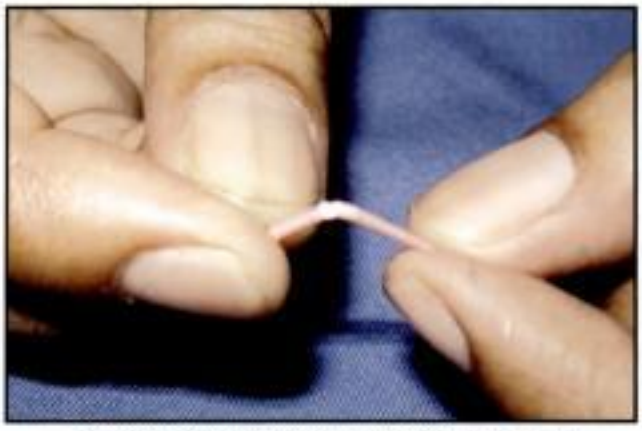


Fig. 13.6: Brittle gutta-percha point breaks on bending

- Gutta-percha can not be heat sterilized. For disinfection of gutta-percha points, they should be immersed in 5.25 percent NaOCl for one minute (Fig. 13.7). Then, gutta-percha should be rinsed in Hydrogen Peroxide or ethyl alcohol. The aim of rinsing is to remove crystallized NaOCl before obturation, as these crystallized particles impair the obturation.
- Gutta-percha should always be used with sealer and cement to seal root canal space as gutta-percha lacks adhering qualities.

Fig. 13.7: Sterilization of gutta-percha by immersing them in 5.25% sodium hypochlorite for one minute

- Gutta-percha is soluble in certain solvents like chloroform, eucalyptus oil, etc. This property can be used to plasticize Gutta-percha by treating it with the solvent for better filling in the canal. But it has shown that gutta-percha shrinks (1-2%) when solidifies.
- Gutta-percha also shows some tissue irritation which is due to high content of zinc oxide.

Current Available Forms of Gutta-percha

Various forms of gutta-percha currently available in the market are:

1. Solid core gutta-percha points
   - Standardized
   - Non standardized
   - i. Standardized cones are of same size and shape as the ISO endodontic instruments.
   - ii. Non standardized form is cone shaped to conform to perceived form of root canal.
2. Thermo-mechanical compactable gutta-percha.
3. Thermo-plasticized gutta-percha
   - Solid core system
   - Injectable form
4. Medicated gutta-percha

Advantages of Gutta-percha

- ***Compactibility:*** adaptation to canal walls
- ***Inertness:*** makes it non-reactive material
- ***Dimensionally stable***
- ***Tissue tolerance***
- ***Radiopacity:*** easily recognizable on radiograph
- ***Plasticity:*** becomes plastic when heated
- ***Dissolve in some solvents*** like chloroform, eucalyptus oil, etc. This property makes it ***more versatile as canal filling material.***

Disadvantages

- Lack of rigidity: Bending of gutta-percha is seen when lateral pressure is applied. So, difficult to use in smaller canals.
- Easily displaced by pressure
- Lacks adhesive quality.

Silver Points

They have been used in dentistry since 1930's but now a days their use has been declined, because of corrosion caused by them.

Silver cones contain traces of metal like copper, nickel which add up the corrosion of the silver points. It has been seen that silver corrosion products are toxic in nature and thus may cause tissue injury.

Due to stiffness of silver cones, they are mainly indicated in round, tapered and narrow canals. They cannot conform with the shape of root canal because they lack plasticity; the use of silver points is not indicated in filling of large, triangular canals as in maxillary anterior teeth.

Silver cones do not possess adhering qualities, so a sealer is required to adequately seal the canal.

ROOT CANAL SEALERS

The purpose of sealing root canals is to prevent periapical exudates from diffusing into the unfilled part of the canal, to avoid reentry and colonization of bacteria and to check residual bacteria from reaching the periapical tissues. Therefore to accomplish a fluid tight seal, a root canal sealer is needed.

The sealer performs several functions during the obturation of a root canal system with gutta-percha; it lubricates and aids the seating of the master gutta-percha cone, acts as a binding agent between the gutta-percha and the canal wall and fills anatomical spaces where the primary filling material fails to reach. Root canal sealers, although used only as adjunctive materials in the obturation of root canal systems, have been shown to influence the outcome of root canal treatment.

The adequate combination of sealing ability and biocompatibility of root canal sealer is important for a favorable prognosis of the root canal treatment. Many studies have shown that most commercially available sealers can irritate the periapical tissues. Initially some type of cytotoxic reaction may even be partially beneficial with respect to eventual periapical healing. So, for a root canal filling material this toxicity should be minimal and clinically acceptable at the time of obturation. At a later time period, the material should become as inert as possible.

There are a variety of sealers that have been used with different physical and biological properties. The clinician must be careful to evaluate all characteristics of a sealer before selecting.

Requirements of an Ideal Root Canal Sealer

Grossman listed eleven requirements and characteristics of a good root canal sealer:

1. It should be tacky when mixed to provide good adhesion between it and the canal wall when set. Only polycarboxylates, glass ionomers and resin sealers satisfy the requirement of good adhesion to dentin.
2. It should create hermetic seal.
3. It should be radiopaque so that it can be visualized in the radiograph. Radiopacity, is provided by salts of heavy metals such as silver, barium, bismuth.
4. The particles of powder should be very fine so that they can mix easily with the liquid.
5. It should not shrink upon setting. All of the sealers shrink slightly on setting, and gutta-percha also shrinks when returning from a warmed or plasticized state.
6. It should not stain tooth structure. Grossman's cement, zinc oxide-eugenol, Endomethasone, and N2 induce a moderate orange-red stain, Diaket and Tubli-Seal cause a mild pink discoloration, AH-26 gives a distinct color shift towards grey, Riebler's paste cause a severe dark red stain. Diaket causes the least discoloration. Leaving any sealers or staining cements in the tooth crown should be avoided.
7. It should be bacteriostatic or at least not encourage bacterial growth. All root canal sealers exert antimicrobial activity to a varying degree and those containing paraformaldehyde to a greater degree initially.
8. It should set slowly. The working and setting times of sealers are dependent on the constituent components, their particle size, temperature and relative humidity. There is no standard working time for sealers, but it must be long enough to allow placement and adjustment of root filling if necessary.
9. It should be insoluble in tissue fluids.
10. It should be tolerant, nonirritating to periradicular tissue.
11. It should be soluble in a common solvent if it is necessary to remove the root canal fitting.

The following were added to Grossman's 11 basic requirements

12. It should not provoke an immune response in periradicular tissue.
13. It should be neither mutagenic nor carcinogenic.

Requirements of an Ideal Root Canal Sealer

- Should be tacky when mixed to provide good adhesion between it and the canal wall when set.
- Should create hermetic seal.
- Should be radiopaque
- Particles of powder should be very fine, for easy mixing with liquid.
- Should not shrink upon setting.
- Should not stain tooth structure.
- Should be bacteriostatic.
- Should set slowly.
- Should be insoluble in tissue fluids
- Should be non-irritating to periradicular tissue.
- Should be soluble in a common solvent.
- Should not provide immune response in periradicular tissue.
- Should not be mutagenic or carcinogenic.

Functions of Root Canal Sealers

Root canal sealers are used in conjunction with filling materials for the following purposes:

1. ***Antimicrobial agent:*** All the popularly used sealers contain some antibacterial agent, and so a germicidal quality is excreted in the period of time immediately after its placement.
2. Sealers are needed to ***fill in the discrepancies*** between the filling material and the dentin walls (Fig. 13.8).
3. ***Binding agent:*** Sealers act as binding agent between the filling material and the dentin walls.

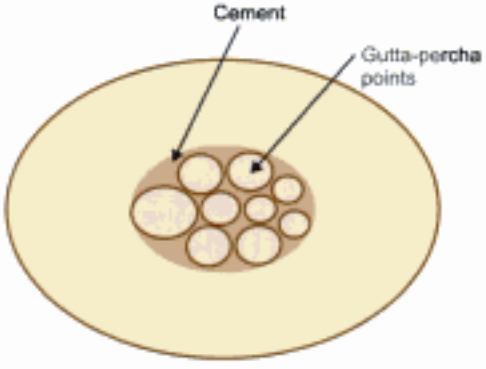


Fig. 13.8: Sealer fills the space between gutta-percha points

4. ***As lubricant:*** With the use of semisolid materials, the most important function for the sealer to perform is its action of lubrication.
5. ***Radiopacity:*** All sealers display some degree of radiopacity; thus they can be detected on a radiograph. This property can disclose the presence of auxiliary canals, resorptive areas, root fractures, and the shape of apical foramen.
6. ***Certain techniques dictate the use of particular sealer.*** The choropercha technique, for instance, uses the material as sealer as well as a solvent for the master cone, allowing the shape of a previously machine made cone to be altered to conform to the prepared canal.

Functions of Root Canal Sealers

- As antimicrobial agent
- Fill the discrepancies between the materials and dentin walls
- As binding agent
- As lubricant
- Give radiopacity
- As canal obturating material

Classification

There are numerous classifications of root canal sealers. Classifications according to various authors are discussed below.

Sealers may be broadly classified according to their composition

1. Eugenol
2. Non-eugenol
3. Medicated

Among these, eugenol containing sealers are widely accepted.

1. **Basically the eugenol group may be divided into sub groups namely.**
   a. Silver containing
   b. Silver free

a. *Silver containing cements:*
   - Kerr sealer (Rickert, 1931)
   - Procosol radaiopaque silver cement (Grossman, 1936)

b. *Silver free cements:*
   - Procosol nonstaining cement (Grossman, 1958)
   - Grossman's sealer (Grossman, 1974)
   - Tubliseal (Kerr, 1961)
   - Wach's paste (Wach)

2. **Non-eugenol**
   These sealers do not contain eugenol and consist of wide variety of chemicals.

Contd...

Contd...

Examples:
- Diaket
- AH26
- Chloropercha and Eucapercha
- Nogenol
- Hydron
- Endofil
- Glass ionomer
- Polycarboxylate
- Calcium Phosphate cement

3. **Medicated:**
These include the group of root canal sealers which have therapeutic properties. These materials are usually used without core materials. The claim is made, particularly for the formulas containing iodoform, that failures of the materials to provide a compact root canal filling are compensated by their prolonged or permanent therapeutic properties.

Examples:
- Diaket-A
- N2
- Endomethasone
- SPAD
- Iodoform paste
- Riebler's paste
- Mynol cement
- $Ca(OH)_2$ paste

According to Grossman
- Zinc oxide resin cements
- Calcium hydroxide cements
- Paraformaldehyde cements
- Pastes

According to Cohen (ADA and ANSI)

According to the intended use:

Type I Material's intended to be used with core material.

Type II Intended for use with or without core material or sealer.

Type I: Divided into three classes

Class I: Includes materials in the form of powder and liquid that set through a non polymerizing process.

Class II: Includes materials in the form of two pastes that set though a non polymerizing process.

Class III: Includes polymers and resin systems that set through polymerization.

Type II

Class I: Powder and liquid-non polymerizing

Class II: Paste and paste-non polymerizing

Class III: Metal amalgams

Class IV: Polymer and resin systems-polymerization

According to Clark
- Absorbable
- Non-absorbable

According to Ingle
- Cements
- Pastes
- Plastic
- Experimental sealers

Zinc Oxide Eugenol Sealers

Kerr Root Canal Sealer or Rickert's Formula

The original zinc oxide-eugenol sealer was developed by Rickert. This is based on the cement described by Dixon and Rickert in 1931. This was developed as an alternative to the gutta-percha based sealers (chloropercha and eucapercha sealers) as they lack dimensional stability after setting.

Composition:

Powder:	
Zinc oxide	34-41.2%
Precipitated silver	25-30.0%
Oleo resins	30-16%
Thymol iodide	11-12%
Liquid:	
Oil of cloves	78-80%
Canada balsam	20-22%

Advantages

1. Excellent lubricating properties.
2. It allows a working time of more than 30 min, when mixed in 1:1 ratio.
3. Germicidal action and biocompatibility.
4. Greater bulk than any sealer and thus makes it ideal for condensation techniques to fill voids, auxilliary canals and irregularities present lateral to gutta-percha cones.

Disadvantages

The major disadvantage is that the presence of silver makes the sealer extremely staining if any of the material enters the dentinal tubuli. So sealers must be removed carefully from the pulp chamber with xylol.

Manipulation

Powder is contained in a pellet and the liquid in a bottle. One drop of liquid is added to one pellet of powder and

mixed with a heavy spatula until relative homogenicity is obtained.

Kerr pulp canal sealer completely sets and is inert within 15-30 min, thus producing the inflammatory responses, compared to other that take 24-36 hours to set.

Procosol Radiopaque-silver Cement (Grossman, 1936)

Composition	
Powder	
Zinc oxide	45%
Precipitated Silver	17%
Hydrogenated resins	36%
Magnesium oxide	2%
Liquid	
Eugenol	90%
Canada balsam	10%

Procosol Non Staining Cement (Grossman's 1958)

Composition	
Powder	
Zinc oxide (reagent)	40%
Staybelite resin	27%
Bismuth Subcaronate	15%
Barium Sulfate	15%
Liquid	
Eugenol	80%
Sweet oil of almond	20%

Grossman's Sealer

Composition	
Powder	
Zinc oxide (reagent)	40 parts
Staybelite resin	30 parts
Bismuth Subcaronate	15 parts
Barium Sulfate	15 parts
Sodiumborate	1 part
Liquid	
Eugenol	

Properties

1. It has plasticity and slow setting time due to the presence of sodiumborate anhydrate.
2. It has good sealing potential.
3. Zinc eugenolate is decomposed by water through continuous loss of eugenol, which makes zinc oxide eugenol a weak unstable compound.

Disadvantage

Resin is of coarse particle size, unless the material is spatulated vigorously during mixing, a piece of resin may lodge on the walls of the canal.

Setting Time

Cement hardens in approximately 2 hours at 37°C.

The setting time is influenced by:

1. Quality of the ZnO and pH of the resin used.
2. Technique used in mixing the cement.
3. Amount of humidity in the temperature
4. Temperature and dryness of the mixing slab and spatula.

Wach's Sealer

Composition	
Powder	
Zinc oxide	10 g
Tricalcium phophate	2 g
Bismuth subnitrate	3.5 g
Bismuth subiodide	0.3 g
Heavy magnesium oxide	0.5 g
Liquid	
Canada balsam	20 ml
Oil of clove	6 ml

Properties

1. Medium working time.
2. Medium lubricating quality.
3. Minimal periapical irritation.
4. It is sticky due to the presence of Canada balsam
5. Increasing the thickness of the sealer lessens its lubricating effect. So this sealer is indicated when there is a possibility of over extension beyond the confines of the root canal.

Advantages

1. It is germicidal
2. Less periapical irritation.

Disadvantages

1. Odor of liquid

Tubliseal (1961)

Slight modifications have been made in Ricket's formula to eliminate the staining property. It has marketed as 2 paste system containing base and catalyst.

Composition

Base

Zinc oxide	57 - 59%
Oil of cajeput	18.5 - 21.25%
Bismuth trioxide	7.5 %
Thymol iodide	3.75 - 5%
Oil and waxes	10%

Catalysts

Eugenol
Polymerized resin
Annidalin

Setting Time

- 20 minute on the glass slab.
- 5 minute in the root canal.

Advantages

1. Easy to mix
2. Extremely lubricated.
3. Does not stain the tooth structure.
4. Expands after setting.

Disadvantages

1. Irritant to periapical tissue.
2. Very low viscosity makes extrusion through apical foramen.
3. Short working time.

Indications

1. When apical surgery is to be performed immediately after filling.
2. Because of good lubricating property, it is used when it becomes difficult for a master cone to reach last millimeter of preparation.

Setting Reaction of Zinc Oxide Eugenol Cement

Zinc oxide and eugenol sets because of a combination of physical and chemical reaction, yielding a hardened mass of zinc oxide embedded in matrix of long sheath like crystals of zinc eugenolate (Fig. 13.9). Hardening of the mixture is due to formation of zinc eugenolate. The presence of free eugenol tends to weaken the set. The significance of free eugenol is most apparent on increased cytotoxicity rather than thorough alteration of the physical properties of dentin. Practically all ZOE sealer cements are cytotoxic and invoke an inflammatory response in connective tissue.

Fig. 13.9: Eugenol and zinc oxide powder

Advantages of Zinc Oxide Eugenol Cement:

- Ease of manipulation.
- Adhesion to the dentinal walls and has only slight dimensional change.
- Radiopaque with some germicidal properties.
- Minimal staining.
- Ample working time

Disadvantages

- Allows some fluid absorption and is irritant to periapex.
- Not easily absorbed from the apical tissues.

Root Canal Sealers Without Eugenol

Kloroperka N - Ø Sealers

This formula was given by Nyborg and Tullin in 1965.

Composition

Power

Canada balsam	19.6%
Rosin	11.8%
Gutta-percha	19.6%
Zinc oxide	49%

Liquid

Chloroform

Kloroperka N-Ø was first introduced in 1939. The powder is mixed with liquid chloroform. After insertion the chloroform evaporates, leaving voids. It has been shown to be associated with a greater degree of leakage than other materials.

Chloropercha

This is a mixture of gutta-percha and chloroform.

Modified Chloropercha Methods

There are two modifications:

1. Johnston-Callahan
2. Nygaard-Ostby

1. ***Johnston-Callahan Method:*** In this method the canal is repeatedly flooded with 95 percent alcohol and then dried. After this, it is flooded with Callahan resin cement and a fitted gutta-percha cone is inserted and compressed laterally and apically with a plugger until the gutta-percha is dissolved completely in the chloroform solution in the root canal. Additional points are added and dissolved in the same way.
2. ***Nygaard Ostby:*** It consists of Canada balsam; colophonium and zinc oxide powder mixed with chloroform. In this technique, the canal walls are coated with Kloroperka, the primary cone dipped in sealer is inserted apically pushing partially dissolved tip of the cone to its apical seal. Additional cones dipped in sealer are packed into the canal to obtain a good apical seal.

Hydron

Hydron is a rapid setting hydrophilic, plastic material used as a root canal sealer without the use of a core. This was introduced by Wichterle and Lim in 1960. It is available as an injectable root canal filling material.

Advantages

1. A biocompatible material.
2. Conforms to the shape of the root canal because of its plasticity.

Disadvantages

1. Short working time.
2. Its radiopacity is very low.
3. Irritant to periapical tissues.
4. Difficult to remove from the canals.

Nogenol

Nogenol was developed to overcome the irritating quality of eugenol. Base is ZnO with Barium sulfate as radiopacifier along with vegetable oil. Set is accelerated by hydrogenated rosin, chlorothymol and salicylic acid.

Appetite Root Canal Sealer

Several root canal sealers composed of hydroxyapatite and tricalcium have been promoted.

There are three types.

Type I:

Powder	
Tricalcium phosphate	80%
Hydroxy apatite	20%
Liquid	
Polyacrylic acid	25%
Water	75%

This is used for vital pulpectomy.

Type II:

Powder	
Tricalcium phosphate	52%
Hydroxyapatite	14%
Iodoform	30%
Liquid	
Polyacrylic acid	25%
Water	75%

Type III:

Powder	
Tricalcium phosphate	80%
Hydroxyapatite	14%
Iodoform	5%
Bismuth subcarbonate	1%
Liquid	
Polyacrylic acid	25%
Water	75%

Resin Based Sealers

Diaket

Diaket is a polyvinyl resin (Polyketone), a reinforced chelate formed between zinc oxide and diketone.

AH-26

This is an epoxy resin recommended by Shroeder in 1957. Epoxy resin based sealers are characterized by the reactive epoxide ring and are polymerized by the breaking this ring. Feldman and Nyborg gave the following composition.

Composition	
Powder	
Bismuth oxide	60%
Hexamethylene tetramine	25%
Silver powder	10%
Titanium Oxide	5%
Liquid	
Bisphenol diglycidyl ether	

The formulation has been altered recently with the removal of silver as one of the constituent to prevent tooth discoloration.

Properties

1. It has good adhesive property.
2. It has good flow
3. Antibacterial
4. It contracts slightly while hardening
5. Low toxicity and well tolerated by periapical tissue.
6. The addition of a hardener, hexamethylene tetramine, makes the cured resin chemically and biologically inert.

AH 26 consists of a yellow powder and viscous resin liquid and mixed to a thick creamy consistency. The setting time is 36 to 48 hours at body temperature and 5-7 days at room temperature.

AH 26 produces greater adhesion to dentin especially when smear layer is removed. Smear layer removal exposes the dentinal tubules creating an irritating surface thus enhancing adhesion.

Thermaseal

Thermaseal has a formulation very similar to that of AH-26. It has been tested in several studies in the united states and is highly rated for both sealing ability and periapical tolerance. Thermaseal may be used with condensation techniques other than Thermafil.

AH Plus

AH Plus is an Epoxide-Amine resin pulp canal sealer, developed from it predecessor AH26. Its shade and color stability make it material of choice where aesthetic demands are high (Fig. 13.10). This easy-to-mix sealer adapts closely to the walls of the prepared root canal and provides minimal shrinkage upon setting as well as outstanding long-term dimensional stability and sealing properties.

Composition

AH Plus Paste A
- Epoxy Resins
- Calcium tungstate
- Zirconium Oxide
- Silica
- Iron Oxide

AH Plus Paste B
- Adamantianeamine
- N,N-Dibenzyl-5-Oxanonane-diamine-1,9,TCD-diamine
- Calcium tungstate
- Zirconium Oxide
- Silica
- Silicone Oil

Fig. 13.10: AH plus root canal sealer

Although pure AH plus contains calcium tungstate, but calcium release is absent from this material. Durarte et al in 2003 suggested addition of 5 percent calcium hydroxide so that it leads to low viscosity material, as well as it provides a more alkaline pH and calcium release. This higher alkalinity and enhanced calcium release leads to improved biological and microbiological behavior, as a more alkaline pH favors the deposition of mineralized tissue and exerts an antimicrobial action.

Dosage and Mixing

Mix equal volume units (1:1) of Paste A and Paste B on a glass slab or mixing pad using a metal spatula. Mix to a homogeneous consistency.

Fiberfill

Fiberfill is a new methacrylate resin-based endodontic sealer. Fiberfill root canal sealant is used in combination with a self-curing primer (Fiberfill primers A and B). Its composition resembles that of dentin bonding agents.

Composition

Fiberfill Root Canal Sealant
- Mixture of UDMA, PEGDMA, HDDMA, Bis-GMA resins
- Treated barium borosilicate glasses
- Barium sulfate
- Silica
- Calcium hydroxide
- Calcium phosphates
- Initiators
- Stabilizers
- Pigments
- Benzoyl peroxide

Fiberfill Primer A
- Mixture of acetone and dental surface active monomer
- NTG-GMA magnesium

Fiberfill Primer B
- Mixture of acetone and dental methacrylate resins of PMGDMA, HEMA Initiator

The bond between the adhesive systems and dentin depends on the penetration of monomers into the dentin surface, to create micromechanical interlocking between the dentin collagen and resin, thus to form a hybrid layer.

Manipulation

Mix equal number of drops of Fiberfill primer A and B. Apply this mix into the root canal. The main sealer which is a mix of base and the catalyst is then applied as an ordinary root canal sealer.

Calcium Hydroxide Sealers

Calcium hydroxide has been used in endodontics as a root canal filling material, intra canal medicaments or as a sealer in combination with solid core materials. The pure calcium hydroxide powder can be used alone or it can be mixed with normal saline solution. The use of calcium hydroxide paste as a root canal filling material is based on the assumption that there is formation of hard structures or tissues at the apical foramen (Fig. 13.11). The alkalinity of calcium hydroxide stimulates the induction of mineralized tissue.

Fig. 13.11: Metapex

Calcium hydroxide and/or calcium hydroxide sealers can:

- Induce mineralization
- Induce apical closure via cementogenesis
- Inhibit root resorption subsequent to trauma
- Inhibit osteoclast activity via an alkaline pH
- Seal or prevent leakage as good as or better than ZOE sealers
- Less toxic than ZOE sealers

Disadvantages

- Calcium hydroxide content may dissolve, leaving obturation voids.
- There is no objective proof that a calcium hydroxide sealer provides any added advantage of root canal obturation or has any of the its desirable biological effects.
- Although calcium hydroxide has dentin regenerating properties, the formation of secondary dentin along the canal wall is prevented by the absence of vital pulp tissue.

Seal apex

It is a non eugenol calcium hydroxide polymeric resin root canal sealer.

Composition

Base

Calcium hydroxide	25%
Zinc oxide	6.5%
Calcium oxide	
Butyl benzene	
Fumed silica (silicon dioxide)	

Catalyst

Barium Sulfate	18.6%
Titanium dioxide	5.1%
Znic stearate	1.0%
Isobutyl salicylate	
Disalicylate	
Trisalicylate	
Bismuth trioxide	

In 100 percent humidity it takes three weeks to reach a final set. It never sets in a dry atmosphere.

Calcibiotic root canal sealer (CRCS)

Composition

Powder

Zinc Oxide	Hydrogenated resin
Barium sulfate	Calcium hydroxide
Bismuth subcarbonate	

Liquid

Eugenol
Eucalyptol

CRCS is a zinc oxide eugenol eucalyptol sealer to which calcium hydroxide has been added for its osteogenic effect. CRCS takes three days to set fully in either dry or humid environment. Because of little water resorption property, it is quite stable. Though sealing is improved, but since calcium hydroxide is not released from the cement, its main role (osteogenic effect) becomes questionable.

Apexit

Apexit is a calcium hydroxide-based root canal sealer available in syringes.

Composition		
Base		
Calcium hydroxide	31.9%	
Hydrogenated colophony	31.5%	
Highly dispersed silicon dioxide	8.1 %	
Calcium oxide	5.6%	
Zinc oxide	5.5%	
Tricalcium phophate	4.1%	
Polydimethylsiloxane	2.5%	
Zinc Stearate	2.3%	
Alkyl ester of phosphoric acid		
Paraffin Oil		
Pigments		
Activator		
Trimethyl hexanedioldisalicylate	25.0%	
Bismuth carbonate	18.2%	
Bismuth oxide	18.2%	
Highly dispersed silicon dioxide	15.0%	
1.3 Butanedioldisalicylate	11.4%	
Hydorgenized Colophony	5.4%	
Tricalcium phosphate	5.0%	
Zinc Stearate		1.4%
Alkyl ester of phosphoric acid		

Advantages

- Biocompatible calcium hydroxide base.
- Easy to mix due to paste delivery form
- Radiopaque.
- Hard setting.

Medicated Sealers

N_2

N_2 was introduced by Sargenti and Ritcher (1961). N_2 refers to the so called second nerve. ***(Pulp is referred to as first nerve)***

Composition of N_2	
Powder	
Zinc oxide	68.51 g
Lead tetraoxide Para	12.00 g
Paraformaldehyde	4.70 g
Bismuth subcarbonate	2.60 g
Bismuth subnitrate	3.70 g
Titanium dioxide	8.40 g
Phenyl mercuric borate	0.09 g
Liquid	
Eugenol	
Oleum Rosae	
Oleum Lavandulae	

The corticosteroids are added to the cement separately as hydrocortisone powder or Terra-Cortril.

The object of introducing formaldehyde within the root-filling is to obtain a continued release of formaldehyde gas, resulting in a prolonged fixation and antiseptic action.

Toxicity

Degree of irritation is severe with the over filling when N_2 is forced into the maxillary sinus or mandibular canal, persisting paresthesia was observed.

Endomethasone

The formation of this sealer is very similar to N_2 composition.

Composition	
Powder	
Zinc oxide	100.00 g
Bismuth subnitrate	100.00 g
Dexamethasone	0.019 g
Hydrocortisone	1.60 g
Thymol iodide	25.0 g
Paraformaldehyde	2.20 g
Liquid	
Eugenol	

Silicone-based Root Canal Sealers

Endo Fill

Endo-fill is an injectable silicone resin endodontic sealant known as Lee Endo fill. Endofill, a silicone elastomer, consists essentially of a silicone monomer and a silicone-based catalyst plus bismuth subnitrate filler.

Active ingredients are hydroxyl terminated dimethyl polysiloxane, benzyl alcohol and hydrophobic amorphous silica. Catalysts are tetra ethylorthosilicate and polydimethyl siloxane.

Setting time can be controlled form 8 to 90 minutes by varying the amount of catalyst used. The more drops of catalyst used, the quicker the material sets and greater the shrinkage.

Advantages

1. Ease of penetration
2. Adjustable working time
3. Low working viscosity
4. Rubbery consistency
5. It is non-resorbable material.

Disadvantages

1. Cannot be used in presence of hydrogen peroxide.
2. Canal must be absolutely dry.
3. Shrinks upon setting but has affinity for flowing into open tubuli.
4. Difficult to remove from the canals.

Glass Ionomer Sealer (Ketac-Endo)

Recently glass ionomer cements have been introduced as endodontic sealers (Ketac-Endo). Glass ionomer cements are reaction product of an ion-leachable glass powder and a polyanion in aqueous solution. On setting they form a hard polysalt gel, which adhere tightly to enamel and dentin. Because of their adhesive qualities, they can be used as root canal sealers (Fig. 13.12).

Fig. 13.12: Ketac endo sealer

Composition

Powder

Calcium aluminium lanthanum flurosilicate glass
Calcium volframate
Cilicic acid
Pigments

Liquid

Polyethylene polycarbonic acid/ Maleic acid
Copolymer
Tartaric acid
Water

Advantages

1. It has optimal physical qualities.
2. It shows bonding to dentin
3. Shows minimum number of voids.
4. Lowest surface tension.
5. Optimal flow property.

Disadvantages

It cannot be removed from the root canal in the event of retreatment as there is no known solvent for glass ionomer. However, Toronto/Osract group has reported that Ketac-endo sealer can be effectively removed by hand instruments or chloroform solvent followed by 1 minute with an ultrasonic No. 25 file.

Newer Sealers

A new material, Resilon (Epiphany, Pentron Clinical Technologies; Wallingford, CT; RealSeal, SybronEndo; Orange, CA) has been developed to replace gutta-percha and traditional sealers for root canal obturation.

It offers solutions to the problems associated with gutta-percha:

1. Shrinkage of gutta-percha after application of heat.
2. The other problem that occurs because of the shrinkage during cooling is that gutta-percha does not physically bind to sealer. It is seen that a gap is present between the sealer and the gutta-percha because of this shrinkage during cooling.

This resilon core material only shrinks 0.5 percent and is physically bonded to the sealer by polymerization. When it sets, no gaps are present due to shrinkage.

Resilon system is comprised of:

1. ***Primer*** a self etch primer, which contains a sulfonic acid terminated functional monomer, HEMA, water and a polymerization initiator.
2. ***Resilon sealer*** a dual-curable, resin-based composite sealer. The resin matrix is comprised of Bis-GMA, ethoxylated BisGMA, UDMA, and hydrophilic difunctional methacrylates. It contains fillers of calcium hydroxide, barium sulphate, barium glasss, bismuth oxychloride and silica. The total filler content is approximately 70 percent by weight. The preparation of the dentin through these chemical agents may prevent shrinkage of the resin filling away form the dentin wall and aid in sealing the roots filled with resilon material.
3. ***Resilon core material*** is a thermoplastic synthetic polymer based (polyester) root canal core material that contains bioactive glass, bismuth oxychloride and barium sulphate. The fillers content is approximately 65 percent by weight.

This new material has shown to be biocompatible, non-cytotoxic and non-mutagenic. The excellent sealing ability of the resilon system may be attributed to the "mono block" that is created by the adhesion of the resilon cone to the Epiphany sealer, which adheres and penetrates into the dentin walls of the root canal system.

The Monoblock Concept

An ideal endodontic filling material would create a "monoblock". This term refers to a continuous solid layer that consists of: - an etched layer of canal dentin impregnated with resin tags that are attached to a thin layer of resin cement that is bonded to a core layer of resilon which makes up the bulk of the filling material. ***The monoblock concept means the creation of a solid, bonded, continuous material from one dentin wall of the canal to the other. One added benefit of the monoblock is that research has shown that it strengthens the root by approximately 20 percent.***

Method of Use

1. Canal is prepared with normal preparation method.
2. ***Smear layer removal:*** Sodium hypochlorite should not be the last irrigant used within the root canal system due to compatibility issues with resins. Use 17 percent EDTA or 2 percent Chlorhexidine as a final rinse.
3. ***Placement of the primer:*** After the canal is dried with paper points, the primer is applied up to the apex. Dry paper points are then used to wick out the excess primer from the canal. The primer is very important because it creates a collagen matrix that increases the surface area in which to bond. The low viscosity primer also draws the sealer into the dentinal tubules.
4. ***Placement of the sealer:*** The sealer can be placed into the root canal system using a lentulospiral at low rpm or by generously coating the master cone.
5. ***Obturation:*** The root canal system is then obturated by preferred method (lateral or warm vertical etc.)
6. ***Immediate cure:*** The resilon root filling material can be immediately cured with a halogen curing light for 40 seconds.
7. ***Coronal restoration:*** A coronal temporary or permanent restoration should then be placed to properly seal the access cavity.

Sealer Placement

Various methods are employed for placing sealer prior to inserting master cone. The common methods are:

1. Coating the master cone and placing the sealer in the canal with a pumping action.
2. Placing the sealer in the canal with a lentulo spiral (Fig. 13.13).
3. Placing the sealer on the final file used at the corrected working length and turning the file counterclockwise.
4. Injecting the sealer with special syringes (Fig. 13.14).

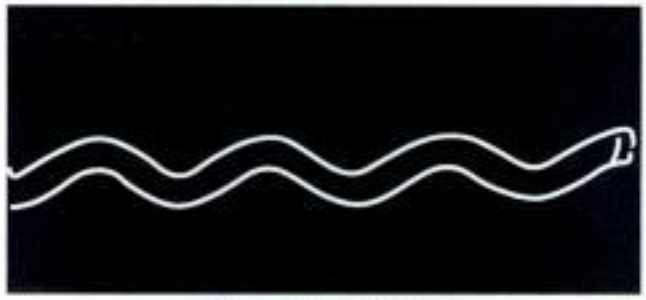


Fig. 13.13: Lentulospiral

Fig. 13.14: Injectable syringe for placement of sealer

Sealer placement techniques vary with the status of apical foramen. If apex is open, only apical one third of master cone is coated with sealer to prevent its extrusion into periapical tissues. In closed apex root, any of above techniques can be used.

OBTURATION TECHNIQUES

The main objective of root canal obturation is the three dimensional sealing of the complete root canal system. As we have seen that gutta-percha is the most common material used for root canal obturation, however, it must be stressed that a sealer is always required to lute the material to the root canal wall and to fill the canal wall irregularities.

Various root canal obturation techniques have been portrayed in the literature, each technique having its indications, contraindications, advantages and disadvantages (Fig. 13.15). Generally speaking the root canal obturation with gutta-percha as filling material, can be mainly divided in to following groups:

1. Use of cold gutta-percha
   - Lateral compaction technique
2. Use of chemically softened gutta-percha
   - Chloroform
   - Halothane
   - Eucalyptol

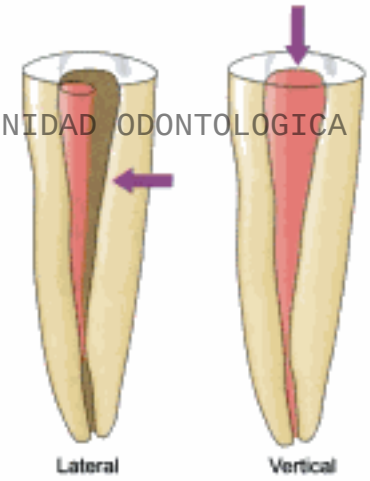


Fig. 13.15: Lateral and vertical compaction of gutta-percha

3. Use of heat softened gutta-percha
   - Vertical compaction technique
   - System B continuous wave condensation technique
   - Lateral/vertical compaction
   - Sectional compaction technique
   - McSpadden compaction of gutta-percha
   - Thermoplasticized gutta-percha technique including
     - Obtura II
     - Ultrasonic plasticizing
     - Ultrafil system
   - Solid core obturation technique including
     - Thermafil system
     - Silver point obturation

LATERAL COMPACTION TECHNIQUE

It is one of the most common methods used for root canal obturation. It involves placing tapered gutta-percha cones in the canal and then compacting them under pressure against the canal walls using a spreader. A canal should have continuous tapered shape with a definite apical stop, before it is ready to be filled by this method (Fig. 13.16).

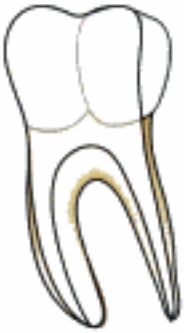


Fig. 13.16: Tapered preparation of root canal system

Technique

1. Following the canal preparation, select the master gutta-percha cone whose diameter is consistent with largest file used in the canal up to the working length. One should feel the tugback with master gutta-percha point (Fig. 13.17). master gutta-percha point is notched at the working distance analogous to the level of incisal or occlusal edge reference point (Fig. 13.18).

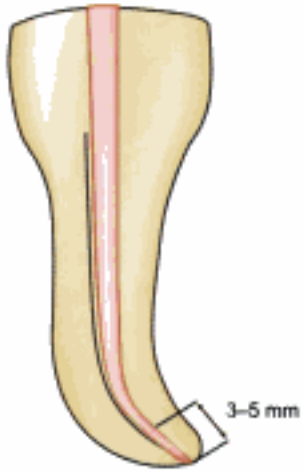


Fig. 13.17: Tugback with master gutta-percha cone

Fig. 13.18: Notching of gutta-percha at the level of reference point

2. Check the fit of cone radiographically. If found satisfactory, remove the cone from the canal and place it in sodium hypochlorite. If cone fits short of the working length, check for dentin chip debris, any ledge or curve in the canal and treat them accordingly (Fig. 13.19) and (Fig. 13.20).

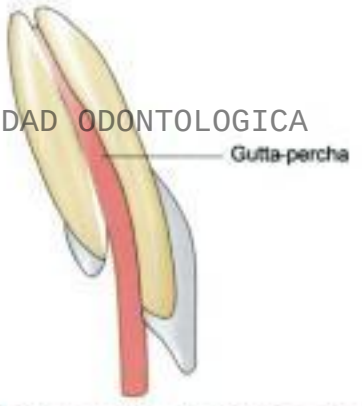


Fig. 13.19: Gutta-percha showing tight fit in middle and space in apical third

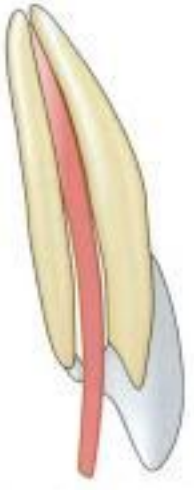


Fig. 13.20: Gutta-percha cone showing tight fit only on apical part of sort canal

If cone selected is going beyond the foramen, either select the larger number cone or cut that cone to the working length (Fig. 13.21).

If cone shows "s" shaped appearance in the radiograph that means cone is too small for the canal. Here a larger cone must be selected to fit in the canal.

3. Select the size of spreader to be used for lateral compaction of that tooth. It should reach 1-2 mm of true working length (Fig. 13.22).
4. Dry the canal with paper points and apply sealer in the prepared root canal (Fig. 13.23).
5. Now premeasured cone is coated with sealer and placed into the canal. After master cone placement, spreader is placed into the canal alongside the cone (Fig. 13.24). Spreader helps in compaction of gutta-percha. It act as a wedge to squeeze the gutta-percha laterally under

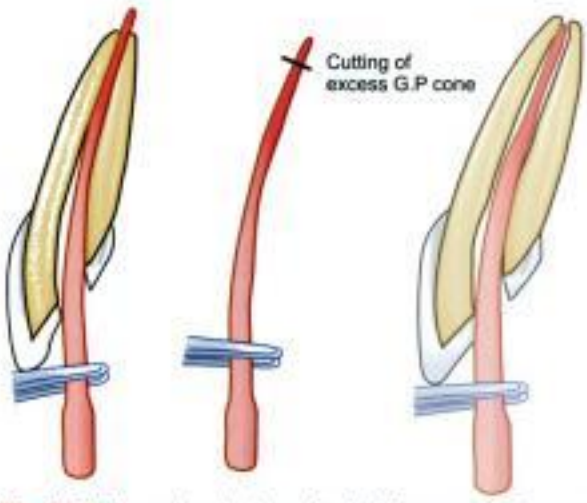


Fig. 13.21: If cone is going beyond apical foramen, cut the cone to working length or use larger no. cone

Fig. 13.22: Spreader should match the taper of canal

Fig. 13.23: Apply sealer in the prepared canal

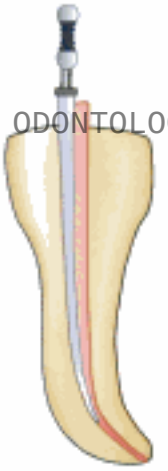


Fig. 13.24: Placing spreader along gutta-percha cone

vertical pressure not by pushing it sideways (Fig. 13.25). It should reach 1-2 mm of the prepared root length.

6. After placement, spreader is removed from the canal by rotating it back and forth. This compacts the gutta-percha and a space gets created lateral to the master cone (Fig. 13.26).
7. An accessory cone is placed in this space and the above procedure is repeated until the spreader can no longer penetrate beyond the cervical line (Fig. 13.27).
8. Now sever the protruding gutta-percha points at canal orifice with hot instrument (Fig. 13.28).

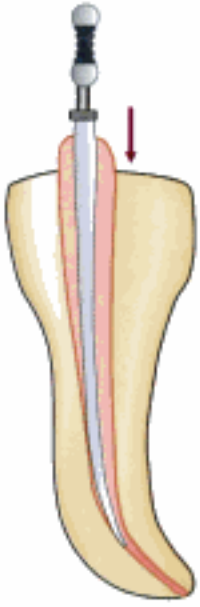


Fig. 13.25: Compaction of gutta-percha using spreader

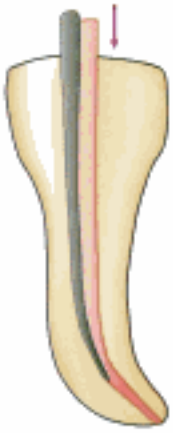


Fig. 13.26: Placing accessory cone along master cone

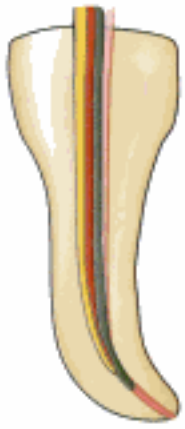


Fig. 13.27: Use of more accessory cones to complete obturation of the canal

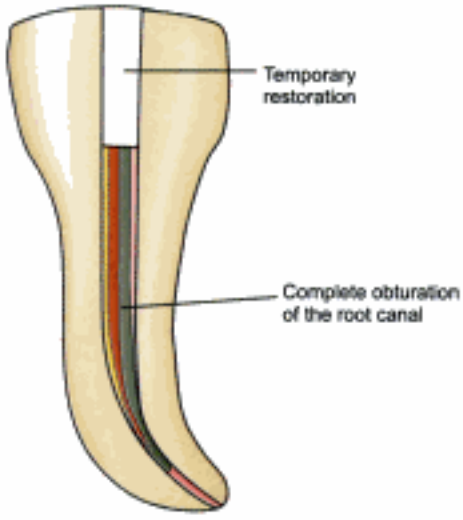


Fig. 13.28: Cut the protruding gutta-percha points at orifice with hot instrument

Advantages of Lateral Compaction Technique

1. Can be used in most clinical situations.
2. During compaction of gutta-percha, it provides length control, thereby prevent overfilling.

Disadvantages

1. May not fill the canal irregularities efficiently.
2. Does not produce homogenous mass.
3. Space may exist between accessory and master cones (Fig. 13.29).

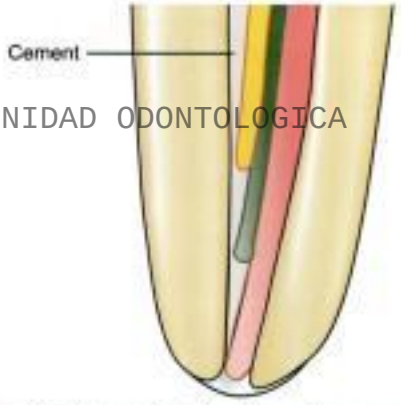


Fig. 13.29: In lateral compaction of gutta-percha, cones never fit as homogenous mass, sealer occupies the space in between the cones

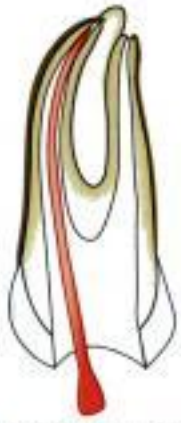


Fig. 13.31: Checking the fit of gutta-percha cone

CHEMICAL ALTERATION OF GUTTA-PERCHA

Gutta-percha is soluble in number of solvents viz: chloroform, eucalyptol, xylol. This property of gutta-percha is used to adapt it in various canal shapes which are amenable to be filled by lateral compaction of gutta-percha technique. For example:

- In teeth with blunderbuss canals
- Root ends with resorptive defects, delta formation.
- In teeth with internal resorption.

In these cases an imprint of apical portion of the canal is obtained by following method:

- Root canal is cleaned and shaped properly (Fig. 13.30).
- The cone is held with a plier which has been adjusted to the working length (Fig. 13.31).
- The apical 2-3 mm of cone is dipped for a period of 3-5 seconds into a dappen dish containing solvent (Fig. 13.32).

Fig. 13.32: Softening of gutta-percha cone by placing in chloroform

- Softened cone is inserted in the canal with slight apical pressure until the beaks of plier touch the reference point (Fig. 13.33).
- Here take care to keep the canal moistened with irrigation, otherwise some of softened gutta-percha may stick to the desired canal walls. Though this detached segment can be easily removed by use of H-file.

Fig. 13.30: Cleaned and shaped canal

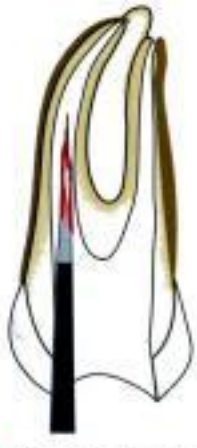


Fig. 13.33: Application of sealer in the canal

- Radiograph is taken to verify the fit and correct working length of the cone. When found satisfactory, cone is removed from the canal and canal is irrigated with sterile water or 99 percent isopropyl alcohol to remove the residual solvent.
- After this canal is coated with sealer (Fig. 13.33). Cone is dipped again for 2-3 seconds in the solvent and thereafter inserted into the canal with continuous apical pressure until the plier touches the reference point (Fig. 13.34).
- A finger spreader is then placed in the canal to compact the gutta-percha laterally (Fig. 13.35).
- Accessory gutta-percha cones are then placed in the space created by spreader (Fig. 13.36).
- Protruding gutta-percha points are cut at canal orifice with hot instrument (Fig. 13.37).

Though this method is considered good for adapting gutta-percha to the canal walls but chloroform dip fillings have shown to produce volume shrinkage which may lead to poor apical seal.

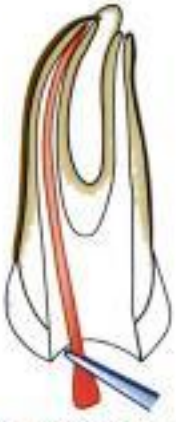


Fig. 13.34: Softened gutta-percha placed in the canal

Fig. 13.35: Compaction of gutta-percha using spreader

Fig. 13.36: Complete obturation of the canal using accessory cones

Fig. 13.37: Severe the protruding gutta-percha cones using hot burnisher

VERTICAL COMPACTION TECHNIQUE

It has been shown that root canal is not just a tubular structure present in the root. Infact presence of many lateral and accessory canals and anastomoses give it a complex shape (Fig. 13.38). Vertical compaction of warm gutta-percha method of filling the root canal was introduced by Schilder with an objective of filling all the portals of exit with maximum amount of gutta-percha and minimum amount of sealer. This is also known as Schilder's technique of obturation. In this technique using heated pluggers, pressure is applied in vertical direction to heat softened gutta-percha which causes it to flow and fill the canal space (Fig. 13.39).

Fig. 13.38: (A) Theoretical canal formation (B) Actual root canal configuration with branches and ramifications

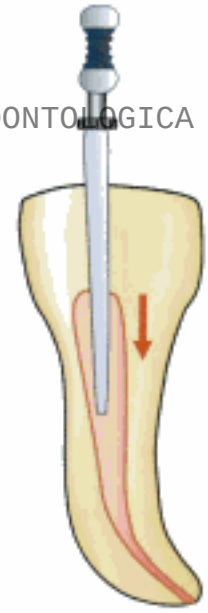


Fig. 13.39: Vertical compaction of gutta-percha using plugger

Basic requirements of a prepared canal to be filled by this technique are:

a. Continuous tapering funnel shape from orifice to apex (Fig. 13.40).
b. Apical opening kept as small as possible.
c. Decreasing the cross sectional diameter at every point apically and increasing at each point as canal is approached coronally.

Technique

- Select a master cone according to shape and size of the prepared canal. Cone should fit in 1-2 mm of apical stop because when softened material moves apically into prepared canal, it adapts more intimately to the canal walls (Fig. 13.41).
- Confirm the fit of cone radiographically, if found satisfactory, remove it from the canal and place in sodium hypochlorite.
- Irrigate the canal and then dry by rinsing it with alcohol and latter using the paper points.
- Select the heat transferring instrument and pluggers according to canal shape and size. Pluggers are prefitted at 5 mm intervals so as to capture maximum cross section area of the softened gutta-percha (Fig. 13.42 to Fig. 13.44).
- Lightly coat the canal with sealer.

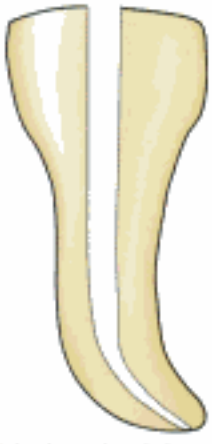


Fig. 13.40: Completely cleaned and shaped tapered preparation

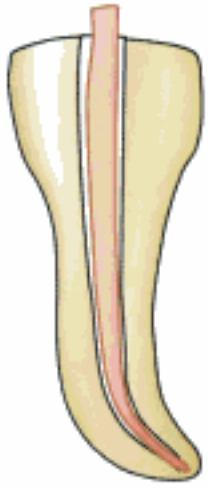


Fig. 13.41: Select the master gutta-percha cone

Fig. 13.42: Select the plugger according to canal shape and size

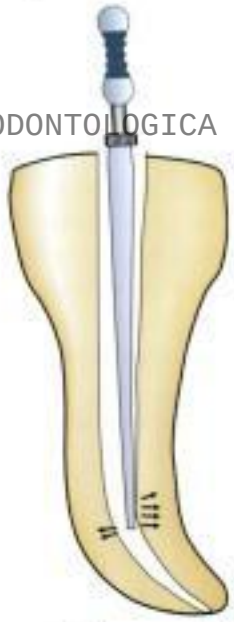


Fig. 13.43: Larger sized plugger may bind the canal and may split the root

Fig. 13.44: Small plugger is ineffective for compaction

- Cut the coronal end of selected gutta-percha at incisal or occlusal reference point.
- Now use the heated plugger to force the gutta-percha into the canal. The blunted end of plugger creates a deep depression in the centre of master cone (Fig. 13.45). The outer walls of softened gutta-percha are then folded inward to fill the central void, at the same time mass of softened gutta-percha is moved apically and laterally. This procedure also removes 2-3 mm of coronal part of gutta-percha.
- Once apical filling is done, complete obturation by doing backfilling. Obturate the remaining canal by heating small segments of gutta-percha, carrying them into the canal and then compacting them using heated pluggers as described above (Fig. 13.46).
- Take care not to overheat the gutta-percha because it will become too soft to handle.
- Do not apply sealer on the softened segments of gutta-percha because sealer will prevent their adherence to the body of gutta-percha present in the canal.

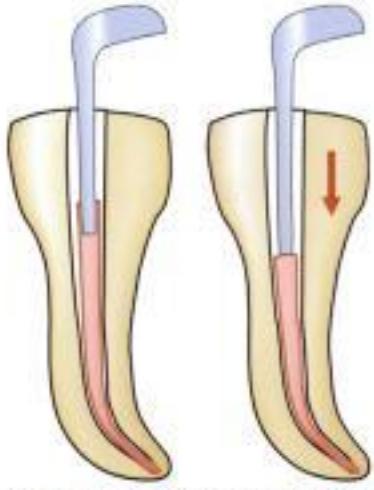


Fig. 13.45: Heated plugger used to compact gutta-percha

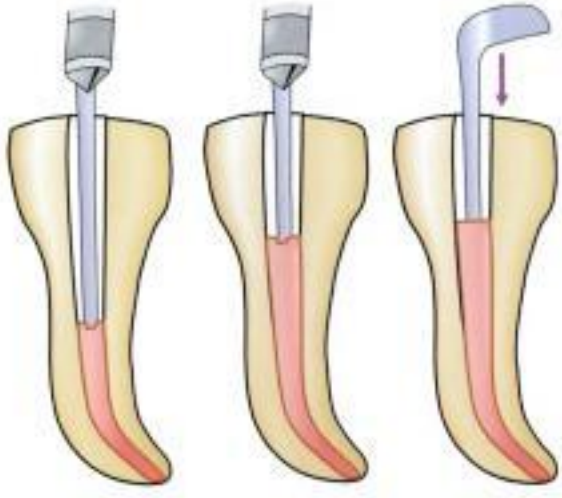


Fig. 13.46: Back filling of the canal

- After completion of obturation, clean the pulp chamber with alcohol to remove remnants of sealer or gutta-percha.

Advantages of Vertical Compaction Technique

- Excellent sealing of canal apically, laterally and obturation of lateral as well as accessory canals.

Disadvantages of this Technique

- Increased risk of vertical root fracture.
- Overfilling of canals with gutta-percha or sealer from apex.
- Time consuming.

SYSTEM B: CONTINUOUS WAVE OF CONDENSATION TECHNIQUE

System B is newly developed device by ***Buchanan*** for warming gutta-percha in the canal. It monitors temperature at the tip of heat carrier pluggers, thereby delivering a précised amount of heat.

To have satisfactory, three dimensional obturation by using system B technique, following precautions should be taken

- Canal shape should be continuous perfectly tapered.
- Do not set the system B at high temperature because this may burn gutta-percha.
- While down packing, apply a constant firm pressure.

Technique

1. Select the Buchanan plugger which matches the selected gutta-percha cone (Fig. 13.47). Place rubber stop on the plugger and adjust it to its binding point in the canal 5-7 mm short of working length.
2. Confirm the fit of the gutta-percha cone (Fig. 13.48).
3. Dry the canal, cut the gutta-percha ½ mm short and apply sealer in the canal.
4. With the system B turned on to "use", place it in touch mode, set the temperature to 200°C and dial the power setting to 10. Sever the cone at the orifice with preheated plugger. Afterwards plugger is used to compact the softened gutta-percha at the orifice. Push the plugger smoothly though the gutta-percha to with 3-4 mm of the binding point (Fig. 13.49).
5. Release the switch. Held the plugger here for 10 seconds with a sustained pressure to take up any shrinkage which might occur upon cooling of gutta-percha (Fig. 13.50).

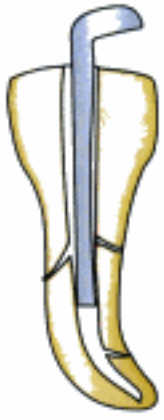


Fig. 13.47: Selection of plugger according to shape and size of the canal

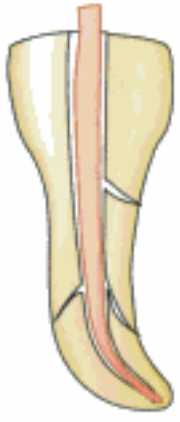


Fig. 13.48: Confirm fit of the cone

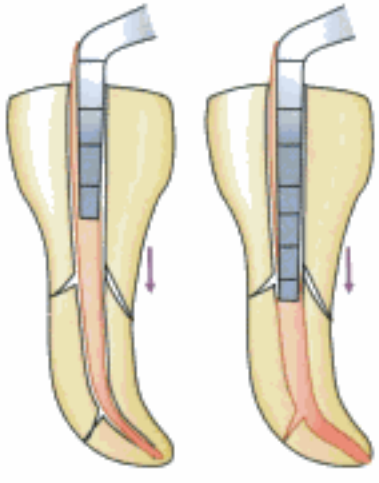


Fig. 13.49: Filling the canal by turning on system-B

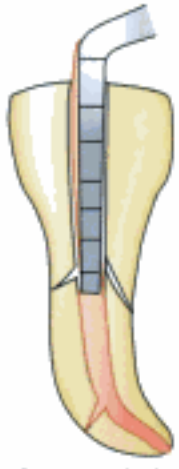


Fig. 13.50: Compaction of gutta-percha by keeping the plugger for 10 sec with sustained pressure

6. Maintaining the apical pressure, activate the heat switch for 1 second followed by 1 second pause, and then remove the plugger (Fig. 13.51).
7. Apply lateral pressure with the small flexible end of another plugger with pressure to confirm that apical mass of gutta-percha has not dislodged, has cooled and set (Fig. 13.52).

Following radiographic confirmation canal is ready for the backfill by any means.

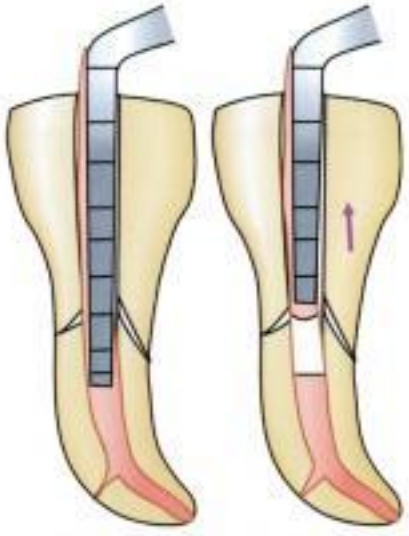


Fig. 13.51: Removal of plugger

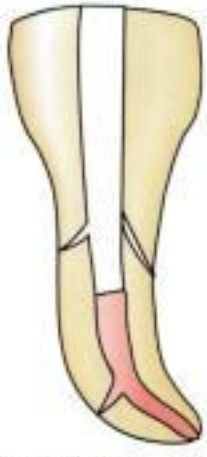


Fig. 13.52: Apical filling of root canal completed

Advantages of System B

- It creates single wave of heating and compacting thereby compaction of filling material can be done at same time when it has been heat softened.

Contd...

Contd...

- Excellent apical control.
- Less technique sensitive
- Fast, easy, predictable.
- Thorough condensation of the main canal and lateral canals.
- Compaction of obturating materials occurs at all levels simultaneously throughout the momentum of heating and compacting instrument apically.

LATERAL/VERTICAL COMPACTION OF WARM GUTTA-PERCHA

Vertical compaction causes dense obturation of the root canal, while lateral compaction provides length control and satisfactory ease and speed.

Advantages of both of these techniques are provided by a newer device viz ***Endotec II*** which helps the clinician to employ length control with warm gutta-percha technique. It comes with battery which provides energy to heat the attached plugger and spreader.

Technique

1. Adapt master gutta-percha cone in canal.
2. Select Endotec plugger and activate the device.
3. Insert the heated plugger in canal beside master cone to within 3-4 mm of the apex using light apical pressure.
4. Afterwards unheated spreader can be placed in the canal to create more space for accessory cones. This process is continued until canal is filled.

Advantages

- Three dimensional obturation of canal.
- Better sealing of accessory and lateral canals.
- ***Endotec*** can also be used to soften and remove the gutta-percha.

SECTIONAL METHOD OF OBTURATION

In this technique, small pieces of gutta-percha cones are used to fill the sections of the canal. It is also known as ***Chicago technique*** because it was widely promoted by ***Coolidge, Lundquist, Blayney,*** all form ***Chicago***.

Technique

1. A gutta-percha cone of same size of the prepared root canal is selected and cut into sections of 3 to 4 mm long.
2. Select a plugger which loosely fits within 3 mm of working length.

3. Apply sealer in the canal.
4. One end of gutta-percha is mounted to heated plugger and is then carried into the canal and apical pressure is given. After that plugger is disengaged from gutta-percha by rotating it.
5. Radiograph is taken to confirm its fit.

If found satisfactory, remainder of the canal is filled in same manner.

Advantages
- It seals the canals apically and laterally.
- In case of post and core cases, only apical section of canal is filled.

Disadvantages
- Time consuming.
- If canal gets overfilled, difficult to remove sections of gutta-percha.

McSPADDEN COMPACTION/ THERMOMECHANICAL COMPACTION OF THE GUTTA-PERCHA

McSpadden introduced a technique in which heat was used to low the viscosity of gutta-percha and thereby increasing its plasticity. This technique involves the use of a compacting instrument (McSpadden compacter) which resembles reverse Hedstorm file (Fig. 13.53). This is fitted into latch type handpiece and rotated at 8000 - 15000 rpm alongside gutta-percha cones inside the canal walls. At this speed, heat produced by friction softens the gutta-percha and designs of blade forces the material apically.

Because of its design, the blades of compaction break easily if it binds, so it should be used only in straight canals. But now days, its newer modification in form of Microseal condenser has come which is made up of nickel - titanium. Because of flexibility they may be used in curved canals.

Fig. 13.53: Thermomechanical compaction of gutta-percha

Advantages
- Requires less chair side time.
- Ease of selection and insertion of gutta-percha.
- Dense, three dimensional obturation.

Disadvantages
- Liability to use in narrow and curved canals.
- Frequent breakage of compactor blades.
- Overfilling of canals.
- Shrinkage of gutta-percha on cooling.

THERMO PLASTICIZED INJECTABLE GUTTA-PERCHA OBTURATION

Obtura II Heated Gutta-Percha System/High Heat System

This technique was ***introduced in 1977 at Harvard institute***. It consists of an electric control unit with pistol grip syringe and specially designed gutta-percha pellets which are heated to approximately 365 - 390°F (185 - 200°C) for obturation. In this, regular Beta phase of gutta-percha is used. For canals to filled by Obtura II they need to have:

a. Continuous tapering funnel shape for unrestricted flow of softened gutta-percha (Fig. 13.54).
b. A definite apical stop to prevent overfilling.

Fig. 13.54: Tapering funnel shaped of prepared canal is well suited for obturation using obtura II

Indication

- Roots with straight or curved canals.
- For backfilling of canals.
- For obturation of roots with internal resorption, perforations, etc.

Technique

a. Before starting the obturation, applicator needle and pluggers are selected. The needle tip should reach ideally 3-5 mm of the apical terminus passively (Fig. 13.55).
b. Apply sealer along the dentinal walls to fill the interface between gutta-percha and dentinal walls.
c. Place obtura needle loosely 3 -5 mm short of apex, as warm gutta-percha flows and fills the canal, back pressure pushes the needle out of the canal (Fig. 13.56).

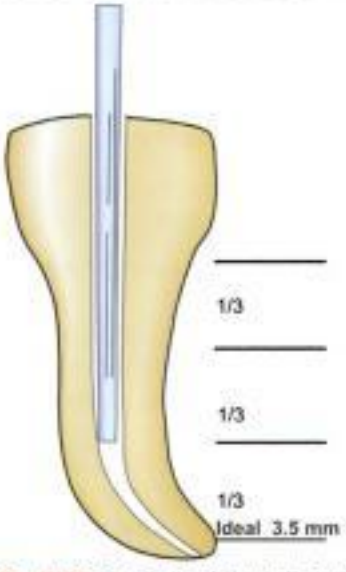


Fig. 13.55: Needle tip of obtura-II should reach 3-5 mm of apical end

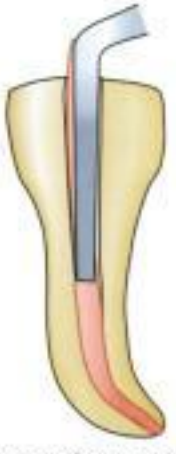


Fig. 13.56: Compaction of gutta-percha using plugger

d. Now use pluggers to compact the gutta-percha, pluggers are dipped in isopropyl alcohol or sealer to prevent sticking of the gutta-percha.

Continuous compaction force should be applied throughout the obturation of whole canal to compensate shrinkage and to close any voids if formed.

Variations in Thermoplasticizing Technique of Gutta-Percha

1. Ultrasonic Plasticizing of Gutta-Percha

It has been seen that ultrasonics can be used to fill the canals by plasticizing the gutta-percha.

Earlier cavitron US scaler was used for this purpose but its design limited its use only in anterior teeth. Recently Enac Ultrasonic unit comes with an attached spreader which has shown to produce homogenous compaction of gutta-percha.

2. Ultrafil System

This system uses low temperature (i.e. 70°C) plasticized alpha phase gutta-percha. Here gutta-percha is available in three different viscosities for use in different situations.

Regular set and the firm set with highest flow properties primarily used for injection and need not be compacted manually. Endoset is more of viscous and can be condensed immediately after injection.

Technique

1. Cannula needle is checked in canal for fitting. It should be 6-7 mm from apex (Fig. 13.57). After confirmation

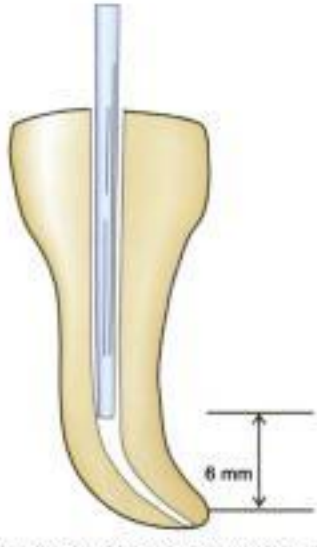


Fig. 13.57: Needle should reach 6-7 mm from the apical end

it is placed in heater (at 90°) for minimum of 15 minutes before use.
2. Apply sealer in the canal and passively insert the needle into the canal. As the warm gutta-percha fills the canal, its backpressure pushes the needle out of the canal.
3. Once needle is removed, prefitted plugger dipped in alcohol is used for manual compaction of gutta-percha.

SOLID CORE CARRIER TECHNIQUE

Thermafil Endodontic Obturators

Thermafil endodontic obturators are specially designed flexible steel, titanium or plastic carriers coated with alpha phase gutta-percha. Thermafil obturation was devised by Johnson in 1978.This technique became popular because of its simplicity and accuracy.

Thermafil obturators are available in sizes ranging from 20 to 140 and are color coded to correspond to endodontic instruments. The carrier is not the primary cone for obturation. It acts as carrier and condenser for thermally plasticized gutta-percha (Fig. 13.58 and 58A).

Fig. 13.58: Thermafil obturator

Fig. 13.58A: Thermafil obturator

Technique

1. Select a thermafil obturator of the size and shape which fits passively at the working length (Fig. 13.59). Verify it by taking a radiograph.

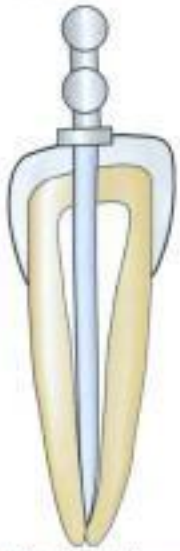


Fig. 13.59: Selection of thermafil obturator

2. Now disinfect the obturator in 5.25 percent sodium hypochlorite for one minute and then rinse it in 70 percent alcohol.
3. Obturator is preheated in "Therma Prep" oven for sometime. This oven is recommended for heating obturator because it offers a stable heat source with more control and uniformity for plasticizing the gutta-percha.
4. Canal is dried and lightly coated with sealer. Heated obturator is placed into the canal with a firm apical pressure (Fig. 13.60) to the marked working length (Fig. 13.61).
5. Working time is 8-10 seconds after removal of obturator from oven. If more obturators are required, they should be inserted immediately.
6. Verify the fit of obturation in radiograph. When found accurate, while stabilizing the carrier with index finger, sever the shaft level with the orifice using a prepi bur or an inverted cone bur in high speed handpiece (Fig. 13.62).
7. Do not use flame heated instrument to sever the plastic shaft because instrument cools too rapidly and thus may cause inadvertent obturator displacement from the canal.
8. Now a small condenser coated with vaseline or dipped in alcohol, is used to condense gutta-percha vertically around the shaft.

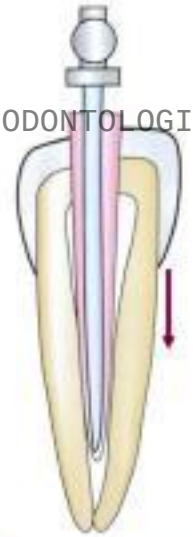


Fig. 13.60: Placing heated obturator in the canal with firm pressure

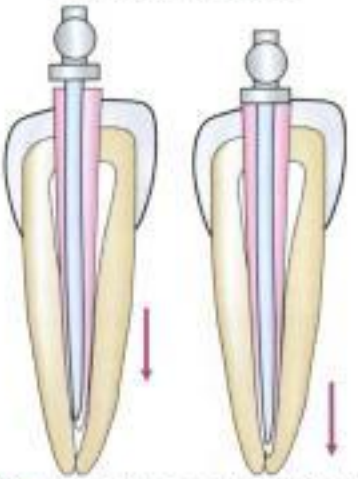


Fig. 13.61: It should reach up to the working length

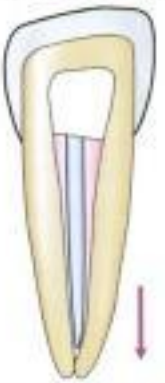


Fig. 13.62: Severe the thermafil obturator using prepi bur or inverted cone bur

9. When the use of post is indicated, the obturator is severed with the fissure bur at the selected length and counter clockwise rotation of shaft following insertion can disengage the instrument.

Advantages

- Requires less chairide time.
- Dense three dimensional obturation as gutta-percha flows into canal irregularities such as fins, anastomoses, and lateral canals etc.
- No need to precurve obturators because of flexible carriers.
- Since this technique requires minimum compaction so less strain while obturation with this technique.

OBTURATION WITH SILVER CONE

Silver cones are most usually preferred method of canal obturation mainly because of their corrosion. Their use is restricted to teeth with fine, tortuous, curved canals which make the use of gutta-percha difficult (Fig. 13.63).

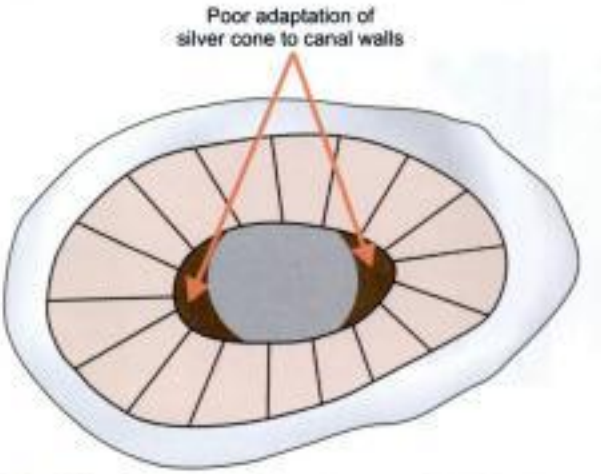


Fig. 13.63: Cross-section of canal obturated with silver cone showing poor adaptation of the cone in irregularly shaped canal

Steps

1. Select a silver cone conforming the final shape and size of the prepared canal. Check its fit radiographically. If found satisfactory, remove it from the canal and sterilize it over an alcohol flame.
2. Dry the canal and coat the canal walls with sealer.
3. Insert the cone into the canal with sterile cotton plier or Stieglitz forceps.

4. Take a radiograph to see the fit of cone. It satisfactory, fill the remaining canal with accessory gutta-percha cones.
5. Remove excess of sealer with cotton pellet and place restoration in the pulp chamber.

Apical Third Filling

Sometimes apical barriers are needed to provide apical stop in cases of teeth with incomplete root development, over instrumentation and apical root resorption. Various materials can be used for this purpose. They are designed to allow the obturation without apical extrusion of the material in such cases.

Commonly advocated materials are:

- Dentin chips
- Calcium hydroxide
- Hydroxyapatite
- Tricalcium phosphate
- Collagen
- MTA

Dentin Chip Filling

Dentin chip filling forms a ***Biologic seal***. In this technique after through cleaning and shaping of canal, H - file is used to produce dentin powder in central portion of the canal, which is then packed apically with butt end of paper point (Figs 13.64 to 13.66).

Though it is biocompatible but care must be taken in this technique because infected pulp tissue can be present in the dentinal mass (Fig. 13.67).

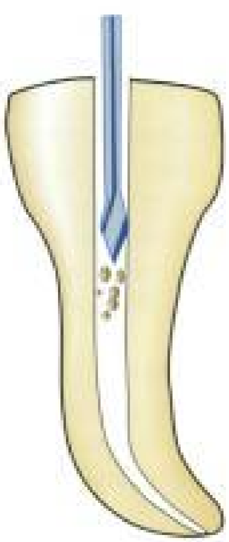


Fig. 13.64: Dentin chips produced by use of gates glidden drills

Fig. 13.65: Chips being compacted with blunt end of instrument/paper point

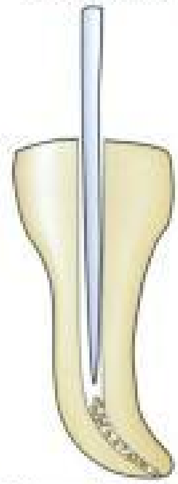


Fig. 13.66: Compaction of dentin chips apically

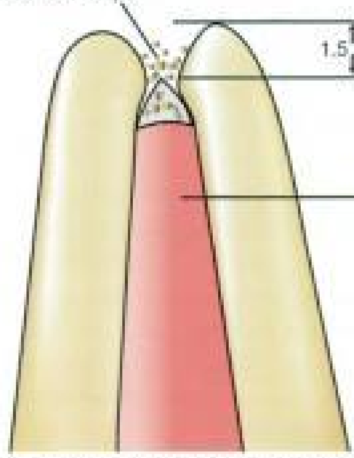


Fig. 13.67: Compaction of dentin chips in apical 2 mm from working length to stimulate hard tissue formation

Calcium hydroxide

It has also been used frequently as apical barrier. Calcium hydroxide has shown to stimulate cementogenesis. It can be used both in dry or moist state.

Moist calcium hydroxide is placed with the help of plugger and amalgam carrier, injectable syringes or by lentulospirals.

Dry form of $Ca(OH)_2$ is carried into canal by amalgam carrier which is then packed with pluggers (Fig. 13.68). Calcium hydroxide has shown to be a biocompatible material with potential to induce an apical barrier in apexification procedures.

Fig. 13.68: Placement of $Ca(OH)_2$ in the canal

Fig. 13.69: Mineral trioxide aggregate

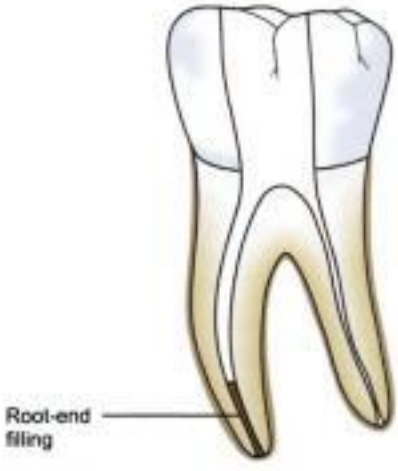


Fig. 13.70: MTA placed at the apical end

Mineral trioxide aggregate (MTA)

MTA was developed by Dr Torabinejad in 1993 (Fig. 13.69). It contains tricalcium silicate, dicalcium silicate, tricalcium aluminate, bismuth oxide, calcium sulfate and Tetracalcium aluminoferrite.

pH of MTA is 12.5, thus having its biological and histological properties similar to calcium hydroxide. Setting time is 2 hours and 45 minutes. In contrast to $Ca(OH)_2$, it produces hard setting nonresorbable surface.

Because of being hydrophilic in nature, it sets in a moist environment. It has low solubility and shows resistance to marginal leakage. It also exhibits excellent biocompatibility in relation with vital tissues.

To use MTA, mix a small amount of liquid and powder to putty consistency. Since, MTA mix is a loose granular aggregate, it can't be carried out in cavity with normal cement carrier and thus has to be carried in the canal with Messing gun, amalgam carrier or specially designed carrier. After its placement, it is compacted with micropluggers (Fig. 13.70).

Advantages of MTA include its excellent biocompatibility, least toxicity of all the filling materials, radiopaque nature, bacteriostatic nature and resistance to marginal leakage. However it is difficult to manipulate with long setting time (3-4 hours).

POST OBTURATION INSTRUCTIONS

Sometimes patient should be advised that tooth may be slightly tender for a few days. It may be due to sensitivity to excess of filling material pushed into periapical tissues.

For relief of pain, NSAID and warm saline rinses are advised. Anti inflammatory drugs such as corticosteroids and antibiotics should be prescribed in severe cases. Patient is advised not to chew unduly on the treated tooth until it is protected by permanent restoration.

Patient Recall

Patient should be recalled regularity to evaluate tissue repair and healing progress.

In case of periapical radiolucency, radiographs should be taken at 3, 6 and 9 months internal period to see continued new bone formation.

The radiograph of a successful filling should show uniformly thickened periodontal ligament and continuous lamina dura along the lateral surfaces of root and around the apex. The tooth should be completely comfortable to patient.

14 Mid Treatment Flare-Ups in Endodontics

- Introduction
- Etiology of Flare-Up
- Microbiology and Immunology of Flare-Ups
- Diagnosis and Management of Flare-Ups
- Prevention of Flare-Ups

INTRODUCTION

Flare-up is described as the occurrence of pain, swelling or the combination of these during the course of root canal therapy, which results in unscheduled visits by patient. Pain may occur soon after initiating endodontic treatment for an asymptomatic tooth or shortly after the initial emergency treatment or during the course of treatment.

American Association of Endodontics (AAE) defines a flare-up "as an acute exacerbation of periradicular pathosis after initiation or in continuation of root canal treatment." Mor C et al in 1992 has suggested that incidence of inter-appointment emergency with endodontic treatment is 4.2 percent and it is not related to patient's sex, age or tooth location.

The mid treatment flare-up is a true emergency and is so severe that an unscheduled patient visit and treatment are required. Despite judicious and careful treatment procedures, complications such as pain, swelling or both may occur. These inter-appointment emergencies are undesirable and disruptive events, so it should be resorted quickly. Occasionally flare-ups are unexpected; sometimes these are predicated due to patient presenting factors.

Flare-ups may even occur with the best of the therapy but these flare-ups usually happen due to improper treatment or when insufficient time is allowed for specific modalities in therapy (***Weine***).

Acute periapical inflammation is the most common cause of mid treatment pain and swelling. Mid treatment emergencies are usually due to irritants left within root canal system or iatrogenic factors such as operator's fault and host factors. The occurrence of mild pain is relatively common following root canal therapy; it should be expected and anticipated by patients, whereas severe pain and swelling associated with flare-up is a rare occurrence (1.4-16%) (Fig. 14.1).

excessive exudate creates pain by causing pressure on nerve endings. Root canals of such teeth when kept open, exudate comes out but in teeth with less periapical [illegible] and the irritants gets aspirated into periapical area leading to pain.

3. ***Microbial factors***: It has been shown by many studies that bacteria are main causative factor in the pulpal and periapical disease. After 1970, with the advent of new technique of culture, the role of flora in root canal is clearly shown. Studies have shown that enzymes, endotoxin and other chemicals produced by anaerobes may cause persistence of periapical lesion. Most commonly found anaerobes are Bacteroides melaninogenicus. It produces enzymes which is collagenolytic, fibrinolytic and endotoxin which activates the Hageman factor which further leads to production of bradykinins and pain mediator. Many studies have shown that teichoic acid present in coating of gram-positive bacteria is a potent immunogen producing humoral antibodies like IgM, IgG and IgA which may cause pain.
4. ***Effect of chemical mediators***: Chemical mediators can be in form of cell mediators, plasma mediators and in form of neutrophils products (Fig. 14.11). Cell mediators include histamine, serotonin, prostaglandins, platelet activating factor and lysosomal components which may lead to pain. The plasma mediators are present in circulation in inactive precursor form and get activated on coming in contact with irritants. For example Hageman factor when gets activated after in contact with irritants, produce multiple effects like production of bradykinin and activation of clotting cascade which may cause vascular leakage . On instrumentation of root canal, acute inflammatory response is initiated resulting in polymorphonuclear leucocytes infiltration, which on further, release collagenase, peroxidase, amylase, lipase and other lytic enzymes resulting in severe pain and swelling.
5. ***Changes in cyclic nucleotides***: Bourne et al have shown that character and intensity of inflammatory and immune response is regulated by hormones and the mediators. For example increased levels of cAMP inhibits mast cell degranulation which helps in reducing pain where as increase in cGMP levels stimulate mast cell degranulation which results in increase in pain (Fig. 14.12). Studies have shown that during flare-up, there is increased level of cGMP over cAMP concentrations.
6. ***Immunological response***: In chronic pulpitis and periapical disease, presence of macrophages and lymphocytes indicates both cell mediated and humoral response. Despite of their protective effect, the immunologic response also contributes to destructive phase of

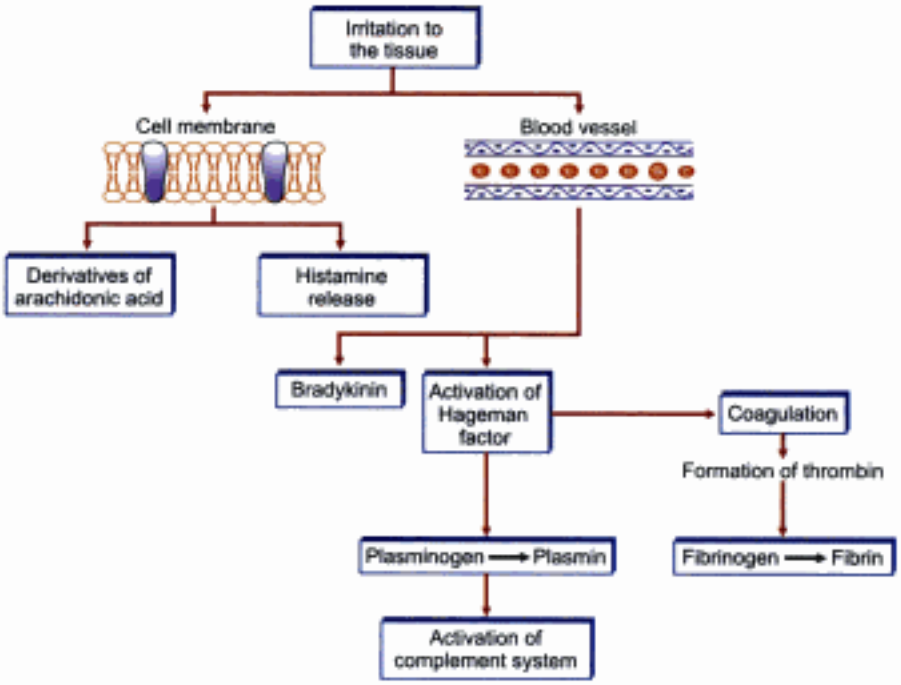


Fig. 14.11: Tissue response to irritation

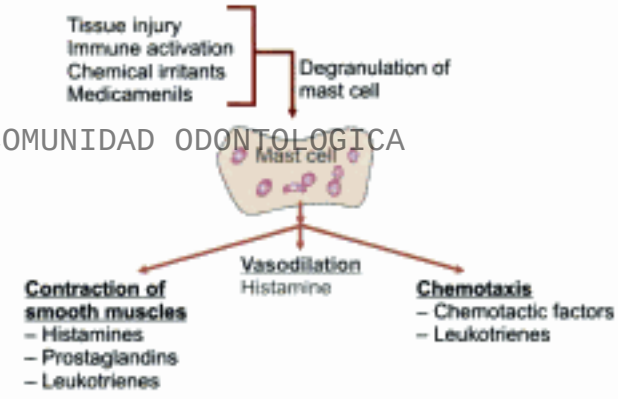


Fig. 14.12: Release of inflammatory mediators from mast cell degranulation

reaction which can occur, causing perpetuation and aggravation of inflammatory process.

7. ***Psychological factor***: Anxiety, apprehension , fear and previous history of dental experience appears to play an important role in mid treatment flare-ups.

DIAGNOSIS AND MANAGEMENT OF FLARE-UPS

Establishing the cause of flare-up is an important step towards management of mid-treatment pain. It is necessary to warn the patient that he or she may experience slight pain after the appointment. So analgesic should be prescribed when patient experience moderate to severe pain after the first appointment. The clinician must review the diagnosis to ensure the tooth under treatment has been identified correctly as the source of pain.

Inter-appointment emergencies are divided into patients with an initial diagnosis of a vital or a necrotic pulp and with or without swelling.

Previously Vital Pulps with Complete Debridement

In these conditions, chances of flare-ups are less, only patient reassurance and the prescription of mild to moderate analgesic often will suffice.

There is no need of reopening and placing corticosteroids in the canal to reduce the pain and swelling as nothing much is gained by these methods. Studies have shown that flare-ups are not prevented by steroids, whether they are placed intracanally or systemically.

Previously Vital Pulps with Incomplete Debridement

It is most commonly seen that pulp remnants left during debridement, considered to be a major irritant, causing severe pain.

To manage this condition, the working length should be rechecked and the canals should be carefully cleaned with copious irrigation of sodium hypochlorite. A dry cotton pellet is then placed followed by a temporary filling (Fig. 14.13) and a mild analgesic is prescribed, along the tooth relieving from occlusion.

In some cases, if tooth become tender, it indicates that inflammation process has reached periapical tissues. Again the management is same as described above.

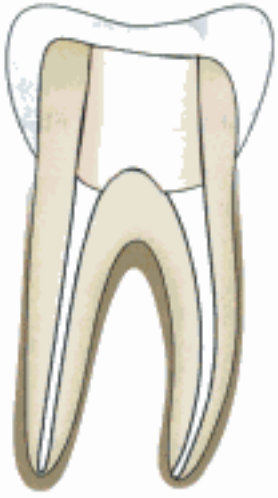


Fig. 14.13: Complete debridement of pulp followed by temporary restoration

Previously Necrotic Pulps without Swelling

Studies have suggested that incidence of flare-ups is higher with necrotic tooth than in vital tooth. It is found to be 7.17 percent (Tayfun Alacam et al) teeth with necrotic pulp often develop as acute apical abscess after the initial appointment. As the lesion is confined to bone, there occurs the severe pain.

The best method of managing the necrotic pulp is to establish accurate working length, complete instrumentation of root canal. In these cases, the tooth is opened and the canal is gently recleaned and irrigated with copious amount of sodium hypochlorite. Drainage must be established if possible. If there is drainage from tooth after opening, then tooth is again cleaned and debrided completely and irrigated

with sodium hypochlorite. After drying the canal, calcium hydroxide dressing is placed and access is sealed.

If there is no drainage from the canal, still gently open the tooth, lightly instrument and completely debride the canal. After debridement, copious irrigation with sodium hypochlorite is done. Then, again use the calcium hydroxide paste and close the access.

It has also been advocated that an analgesic and antibiotic should be prescribed. Leaving the root canal open for drainage is controversial as exposure to oral flora serves no purpose and may lead to flare-ups.

Previously Necrotic Pulp with Swelling

These cases are best managed by incision and drainage. Also, canals should be opened, debrided and gently irrigated with sodium hypochlorite solution. Then calcium hydroxide paste should be placed and closed.

GENERAL MANAGEMENT OF FLARE-UPS

Studies have shown that postoperative pain diminishes in 72 hrs. During this patient experiences pain which must be relieved by the clinician.

General Management of Flare-Ups

1. Reassurance to the patient
2. Complete debridement of root canal system
3. Establishment of drainage
4. Relief of occlusion
5. Calcium hydroxide therapy
6. Intracanal medicaments
7. Medications
   a. Analgesic
   b. Antibiotic
   c. Corticosteroid
8. Placebo

1. ***Reassurance to the patient***: Reassurance is the most important aspect of treatment, so, explain the patient about procedure completely to reduce the anxiety and gaining confidence. The explanation should include that the flare-up is not unusual and can be managed effectively. Also, general anesthesia and conscious sedation are also good adjuncts in treating some cases especially uncooperative and fearful patients.
2. ***Complete debridement of root canal system***: Single most effective method to reduce flare-ups is complete debridement by cleaning and shaping of root canal system (Fig. 14.14). Patency of apex and shaping and cleaning by crown-down technique are two important factors in the management of flare-ups.

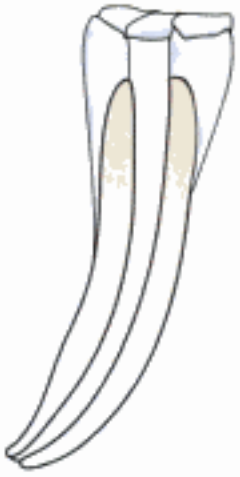


Fig. 14.14: Complete cleaning and shaping of root canal system

3. ***Establishment of drainage***: In the presence of suppuration, drainage of exudate is the most effective method for reducing pain and swelling (Fig. 14.15). In most cases, accumulated exudate will surge from root canal affording immediate relief. But in some cases, root canal instruments are needed to pass through packed dentinal shavings in the apical third of canal. After exudation, access cavity is closed again to avoid exposure of canal to oral flora and salivary products. These salivary products and oral flora may introduce new microorganisms into the canal which may activate complement system thus leading to exacerbation of pain.
4. ***Relief of occlusion***: It has been advocated by Cohen for prevention of postoperative pain and/or to reduce intra-appointment pain.

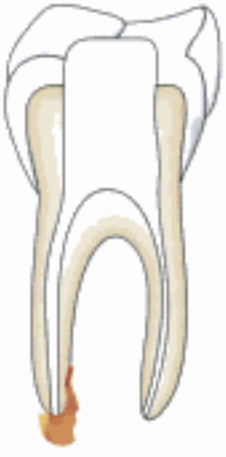


Fig. 14.15: Opening of pulp chamber to allow drainage

5. ***Calcium hydroxide therapy***: It is intracanal dressing used as therapeutic in prevention or treatment of flare-ups which is described as below:

   - Calcium hydroxide is intended to reduce bacterial colonies and their toxic byproducts. The effect of antimicrobial remains in canal for one week.
   - Calcium hydroxide also hydroxylates the lipid moiety of bacterial lipopolysacchride, rendering it incapable of producing biologic effects and complement activation
   - Calcium hydroxide absorbs carbon dioxide (CO_2), by this it deprives the capnophilic organism for carbon dioxide in root canal system.
   - It also obliterates root canal space which minimizes the ingress of tissue exudate, a potential source of nourishment for remaining bacteria.
   - Extrusion of calcium hydroxide periapically reduces inflammatory reaction by reducing substrate adherence capacity of macrophages.
   - Calcium hydroxide has soft tissue dissolving property because of its high pH. As it can dissolve necrotic tissue, denaturing effect of $Ca(OH)_2$ on protein allows sodium hypochlorite to dissolve remaining tissue more easily.

   Placement of calcium hydroxide in an inflamed canal is shown in Fig. 14.16.

6. ***Intracanal medicaments***: For relief of pain during root canal treatment, commonly used intracanal medicaments are antimicrobial agents, irrigating solutions and corticosteroids. The antimicrobial agents such as formocresol, cresatin, CMCP have been tried to provide relief of pain (Fig. 14.17). It has been seen that micro organisms are responsible for exaggeration of inflammation. Among the irrigating solutions, sodium hypochlorite have been proved to provide better effects against the gram-negative anaerobes which are thought to be the main causative factor in flare-up.

   The corticosteroids have been tried because of their anti-inflammatory activity. These inhibit the formation

Fig. 14.16: Placement of $Ca(OH)_2$ in canal

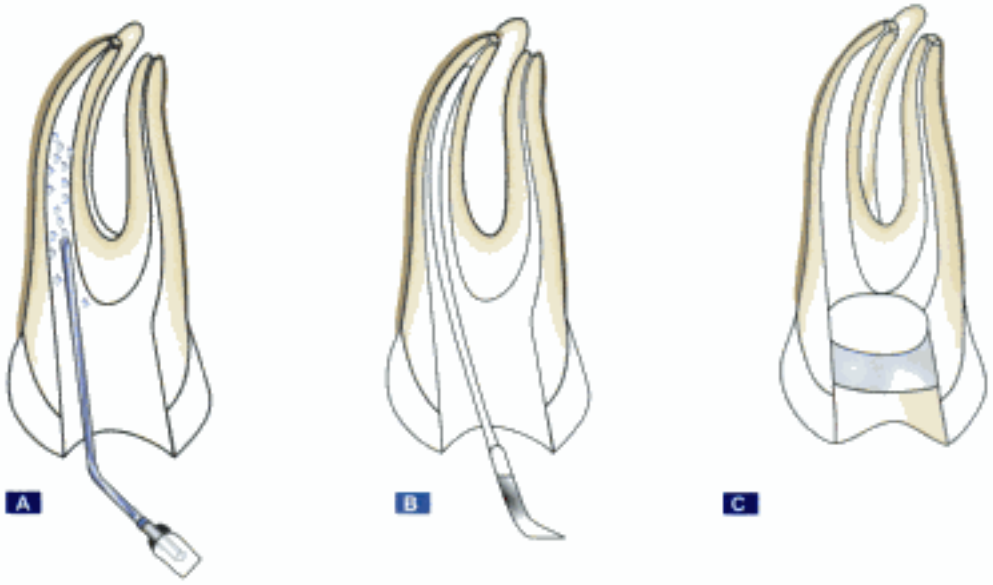


Fig. 14.17: **A.** Irrigation of canal for final cleaning of the canal, **B.** Drying of the canal using absorbent paper point, **C.** Placement of intracanal medicament

of arachidonic acid from the membrane phospholipids and increases the production of cyclic AMP which further inhibits mast cell degranulation thus reducing the inflammatory response. Corticosteroids also hyperpolarizes the nerve present in inflamed area and thus reduces the nerve impulse transmission. The disadvantage of using steroids is that they interfere with phagocytosis and nerve synthesis.

7. ***Medications***: Commonly used systemic drugs are
   a. Analgesic
   b. Antibiotic
   c. Corticosteroid

   Among analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly used for relieving the pain. Commonly used drugs are ibuprofen, diclofenac sodium,aspirin and etoricoxib.

   Narcotic analgesics provide relief from pain by acting on neural receptors of brain cells. They raise pain threshold by causing relaxation, apathy and freedom from anxiety. Commonly used drugs are morphine, codeine, meperidine, tramodol and propoxyphene.

   Among antibiotics, pencillins and its derivatives are commonly prescribed. Metronidazole, tinidazole, ornidazole and clindamycin are also used because of their effectivity against anaerobic infections.
8. ***Placebo***: Placebos are pharmacologically inert substances which do not have therapeutic effect, they act as analgesics and alleviates anxiety. They mimic the action of active drugs. Studies have shown that patients experience the relief of pain after receiving placebo.

Placebo effects are based on patients comprehensation and emotional response to drug administration. The observed effects of drug administration are combination of pharmacological actions and placebo. Clinician himself exerts a potent placebo effect, his conviction that an analgesic is effective, if communicated to anxious patient, increase the expectations of patients relief. Also instruction or suggestions given to patients are powerful aids in controlling of pain. Placebos enhance the levels of endorphin productions and thus resulting in pain relief.

Precautions to be Taken to Prevent Flare-Ups

- Proper diagnosis of the case
- Determination of correct working length
- Complete extirpation of the vital pulp
- Avoid filing too close to the radiographic apex
- Reduce tooth from occlusion especially if apex is severely violated by over instrumentation
- Placement of intracanal medicaments
- Prescription of analgesics and antibiotics whenever condition warrants it.

CONCLUSION

The occurrence of mild pain and discomfort is common even if the treatment rendered is of highest standard. But still psychological preparation of the patient, complete cleaning and shaping of root canal system, use of long acting anaesthetic and analgesics decrease the incidence of inter appointment flare-ups in mild to moderate levels.

Prompt and effective treatment of flare-ups is an essential part of overall endodontic treatment.

15

Endodontic Emergencies

- Introduction
- Diagnosis and Treatment Planning
- Pretreatment Endodontic Emergencies
- Intratreatment Emergencies
- Postobturation Emergencies

INTRODUCTION

It has been seen that more than 80 percent of the patient who report in the dental clinic with emergency symptoms have endodontically related pain. Therefore, the knowledge and skill in various aspects of endodontics are required to achieve successful outcome of endodontic treatment. Incorrect diagnosis and treatment may cause aggravation of the situation. One must have knowledge of pain mechanism, patient management, diagnosis and appropriate treatment measures for both hard and soft tissues. The emergencies are matter of great concern to the both patient and dentists. Emergencies can occur with varying frequencies of pain and swelling in patients before, during and after the root canal treatment. These emergencies are result of combination of various factors which induce severe inflammation in pulp and periapical tissues. The irritation of periradicular tissues result in inflammation and the release of various chemical mediators which initiate inflammation. The pain in endodontic emergencies is mainly related to two factors, chemical mediators and pressure. ***Chemical mediators*** cause pain directly by lowering the pain threshold of sensory nerve fibers or by increasing vascular permeability and producing edema.

Increased fluid ***pressure*** resulting from edema directly stimulates the pain receptors.

In this chapter, we will discuss the diagnosis and treatment of various conditions requiring emergency endodontic treatment.

Endodontic emergency is defined as the condition associated with pain and/or swelling which requires immediate diagnosis and treatment. The main ***causative factors*** responsible for occurrence of endodontic emergencies are:

1. Pathosis in pulp and periradicular tissues
2. Traumatic injuries

Endodontic Emergencies are Categorized into Three Main Types

1. Pretreatment
2. Intraappointment
3. Postobturation

In pretreatment emergencies, patient initially comes with pain and swelling, while intraappointment and post-obturation emergencies occur during or after the initiation of endodontic therapy. Before managing endodontic emergency, one should differentiate a true emergency and a less critical urgency. A ***true emergency*** is the condition which requires unscheduled visit with diagnosis and treatment at that time. But ***urgency*** indicates a less severe problem in which next visit may be scheduled for mutual convenience of both patient as well as the dentist. These conditions can be differentiated by a system of diagnosis.

DIAGNOSIS AND TREATMENT PLANNING

Complete history of the patient along with clinical examination are the two basic steps for successful management of endodontic emergencies. The patient should be asked about the pain, swelling or any other symptom associated with emergency.

The clinician should follow a systematic approach to reach at accurate diagnosis. Certain factors like emotional status of the patient, stress of the clinician and shortage of time, should not affect an orderly approach.

History of the Patient

Pain is frequently the main component of chief compliant. The initial question should help establish ***two basic components*** of pain; ***time (chronicity)*** and ***severity (intensity)***. The patient should be asked questions such as "How painful the tooth is?", "When does it hurt?" "What makes it worse?", etc. A complete history regarding the pain chronology, i.e. mode, periodicity, frequency and duration, pain quality, i.e. sharp, dull, recurrent stabbing, throbbing should be taken.

Pain is a complex, physiological and psychological phenomenon. The psychological components of pain perception and pain reaction comprise emotional and symbolic factors. The emotional status and anxiety level alter the pain reaction threshold. Thus, for accurate history and to receive co-operation during treatment, the anxiety of patient should be reduced (Fig. 15.1). It can be done by gaining the confidence of the patient, providing attention and sympathy and treating the patient as an important

Fig. 15.1: Patient in endodontic emergency presents lot of anxiety and apprehension

individual. Thus we can say that psychological management of the patient is most important factor in emergency treatment. After the patient has provided complete history regarding his or her problem, both subjective questioning and objective examination are performed carefully.

Subjective Examination

A patient should be asked questions about history, location, duration, severity and aggravating factors of pain. For example if pain occurs on mastication or when teeth are in occlusion and is localized in nature, it is ***periodontal in origin*** but if thermal stimuli lead to severe explosive pain and the patient is unable to localize, it is ***pulpal in origin***. Basically quality, quantity, intensity, spontaneity and duration of pain should be asked. A tentative diagnosis achieved by means of thorough subjective examination, is confirmed by objective tests and radiographic findings.

Objective Examination

In objective examination, tests are done to reproduce the response which mimics what the patient reports subjectively. For example if patient complains of pain to thermal changes and on mastication, same pain can be reproduced by applying cold and pressure, thus identifying the offending tooth. The objective examination includes extraoral examination, intraoral examination and diagnostic tests for periradicular as well as pulp tissues. Various pulp evaluating tests are:

- Thermal tests which include heat and cold test.
- Electric pulp test.
- Direct dentin stimulation.

The tests done for evaluation of periradicular status include:

- Periodontal Probing
- Palpation over the apex
- To check the mobility of tooth
- Selective biting on an object

Radiographic Examination

Radiographs are helpful but have many limitations. Though it is impossible to obtain good information without the use of radiograph but one needs to have excellent quality films for correct interpretation. Also one should not become totally dependent on radiographs. It is mandatory that other tests should be used in conjunction with radiographs. Intraoral periapical and bitewing radiographs may detect caries, restorations, pulp exposures, root resorption-external or internal and periradicular pathologies.

So, we can see that by following the system of diagnosis as mentioned above, the offending tooth and the tissue, i.e. pulpal or periradicular can be identified. If diagnosis is not clear by a general practitioner, the patient should be referred. No treatment is initiated without clear diagnosis.

Common Features of Oral Pain

Origin	*Associated sign*	*Useful test*	*Radiograph*
Pulp	Deep caries, previous treatment extensive restoration	heat or cold test	caries, extensive restoration
Periradicular tissue	swelling, redness tooth mobility	percussion palpation and Probing	caries, sometimes periradicular signs
Dentin	caries, defective restoration	hot, cold test, scratching	caries, poor restorations
Gingiva	gingival inflammation	percussion visual examination	none

TREATMENT PLANNING

The main objective of treatment should be immediate relief of pain. Since pain is basically caused by inflammation and increase tissue pressure, the reduction of irritants or pressure or removal of inflamed pulp should be the main goal of treatment.

Definitive Treatment

- Reduction in tissue pressure in the pulp and periradicular tissue and/or removal of inflamed pulp tissue usually results in pain relief / reduction
- Eliminate peripheral components of allodynia and hyperalgesia.

Before starting the treatment for an emergency condition, two fundamental steps, i.e. patient management and obtaining profound anesthesia of inflamed tissues should be always kept in mind.

The ***patient management*** is the most critical factor which affects the prognosis of treatment. An anxious and frightened patient may lose confidence in clinician and may even assume that extraction is necessary. So, reassurance is the most important aspect of the treatment.

To ***obtain an adequate anesthesia*** of inflamed tissues is the challenge. To provide adequate pulpal anesthesia in the mandible, inferior alveolar, lingual and long buccal injection should be preferred. If anesthesia is required in the lower premolars, canine and incisor, then other alternative such as mental nerve block, periodontal ligament injection (Fig. 15.2), intraosseous anesthesia and intrapulpal injection (Fig. 15.3) are given in painful irreversible pulpitis along with classical nerve block. In contrast to mandible, maxillary anesthesia is easier to obtain by giving infiltration or block injections in the buccal or palatal region. These include posterior superior alveolar (PSA), middle superior alveolar (MSA) and infraorbital nerve block.

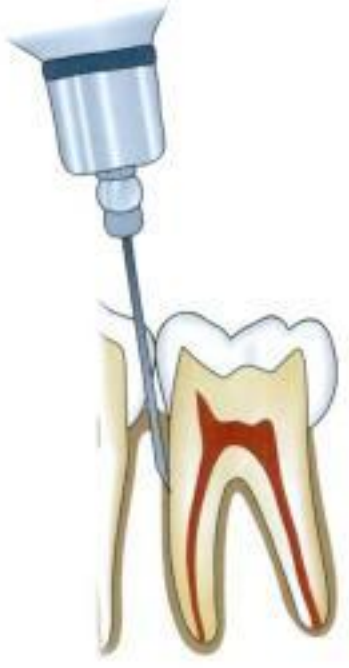


Fig. 15.2: Intraligamentary injection

Fig. 15.4: Extirpation of pulp chamber

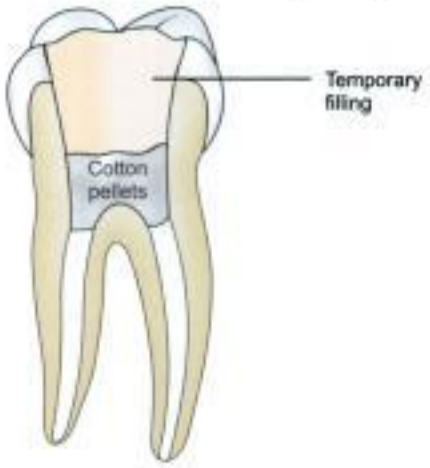


Fig. 15.6: Placement of sedative dressing

5. Thorough irrigation and debridement of the pulp chamber.
6. Determination of the working length.
7. Total extirpation of the pulp followed by cleaning and shaping of the root canal (Fig. 15.5).
8. Thorough irrigation of the root canal system.
9. Drying of the root canal with sterile absorbent points.
10. Placement of a dry cotton pellet or pellet moistened with CMCP, formocresol or eugenol in the pulp chamber and sealing it with the temporary restoration (Fig. 15.6).
11. Relief of the occlusion.
12. Appropriate analgesics therapy and antibiotics, if needed.

Acute periapical abscess: Acute periapical abscess is characterized by following features:

1. Large numbers of bacteria get past the apex into the periradicular tissues (Fig. 15.7).
2. Formation of a periapical abscess implies the breakdown of body's immune system because it should have been able to contain the microbes inside the root canal system.
3. Results in local collection of purulent exudates.
4. May not have radiographic evidence of bone destruction because fluids are rapidly spread away from the tooth.
5. Clinically, swelling to various degrees is present along with pain and a feeling that tooth is elevated in the socket.
6. Systemic features such as fever and malaise may also be present.
7. Mobility may or may not be present.

Fig. 15.5: Cleaning and shaping of the root canal

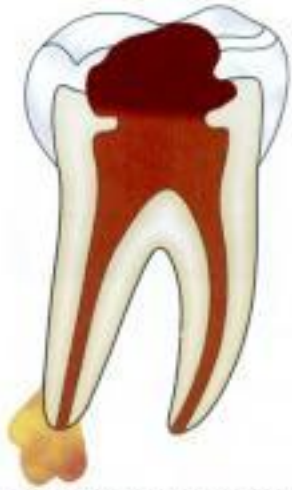


Fig. 15.7: Periapical abscess resulting from tooth decay

3. Total extirpation of pulp in pulp chamber
4. Determination of working length
5. Total extirpation of the pulp
6. Cleaning and shaping of the root canal
7. Thorough irrigation
8. Placement of sedative dressing followed by closed dressing
9. Relieve occlusion if indicated
10. Prescribe analgesics to reduce the pain.

INTERATREATMENT EMERGENCIES

The inter-appointment flares up form a true emergency which requires unscheduled patient visit and immediate active treatment for pain relief. Despite of providing careful treatment procedures complications in the form of pain and/or swelling may occur. Incidence of inter-appointment flare ups vary from approximately (2-25%), variations are because of different factors, conditions and criteria for studies. We have discussed inter-appointment emergencies in detail in next chapter (Chapter 16). To summarize, it.

Etiology of Mid-treatment Flare-ups

1. Over-instrumentation (Fig. 15.11)
2. Inadequate debridement (Fig. 15.12)
3. Missed canal
4. Hyper occlusion
5. Debris extrusion
6. Procedural complications (Figs 15.13 to 15.15)

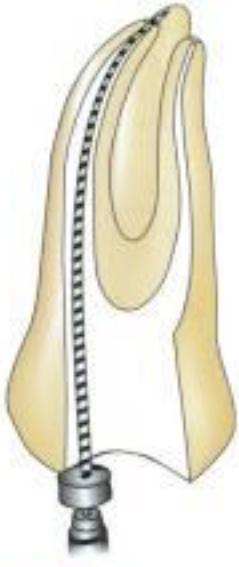


Fig. 15.11: Overinstrumentation

Fig. 15.12: Inadequate debridement

Fig. 15.13: Missed canal

Fig. 15.14: Perforation of root canal

16 Procedural Accidents

- Introduction
- Various Procedural Accidents
- Inadequate Cleaning and Shaping of Root Canal System
- Missed Canal
- Instrument Separation
- Zipping
- Strip Wall Perforation
- Canal Transportation
- Inadequate Canal Preparation
- Perforations
- Underfilling of Root Canals
- Overfilling of Root Canals
- Vertical Root Fracture
- Instruments Aspiration

INTRODUCTION

Like any other field of dentistry, a clinician may face unwanted situations during the root canal treatment which can affect the prognosis of endodontic therapy. These procedural accidents are collectively termed as ***endodontic mishaps.***

Accurate diagnosis, proper case selection, and adherence to basic principles of endodontic therapy may prevent occurrence of procedural accidents. Whenever any endodontic mishap occurs; inform the patient about -

a. The incident and nature of mishap
b. Procedures to correct it
c. Alternative treatment options
d. Prognosis of the affected tooth.

Endodontic mishaps may have dentolegal consequences. Thus their prevention is the best option both for patient as well as dentist. Knowledge of etiological factors involved in endodontic mishaps is mandatory for their prevention. Recognition of a procedural accident is first step in its management.

Procedural accidents can occur at any stage of the root canal treatments which may lead to endodontic failures. Grossly we can categorize the procedural errors as following:

VARIOUS PROCEDURAL ACCIDENTS

1. Inadequately cleaned and shaped root canal system.
   a. Loss of working length
   b. Canal blockage
   c. Ledging of canal
   d. Missed canals
2. Instrument separation
3. Deviation from normal canal anatomy
   a. Zipping
   b. Stripping or lateral wall perforation
   c. Canal transportation
4. Inadequate canal preparation
   a. Over instrumentation
   b. Over preparation
   c. Under preparation

contd...

contd...

5. Perforations
   a. Coronal perforations
   b. Root perforations
      i. Cervical canal perforations
      ii. Mid root perforations
      iii. Apical perforations
   c. Post space perforations
6. Obturation related
   a. Over obturation
   b. Under obturation
7. Vertical root fracture
8. Instrument aspiration

INADEQUATELY CLEANED AND SHAPED ROOT CANAL SYSTEM

Regardless of the instrumentation technique used for cleaning and shaping of the root canal system, the main objectives of biomechanical preparation are to remove pulp tissue, debris and bacteria, as well as to shape the canal for obturation (Fig. 16.1).

Fig. 16.1: Complete cleaning and shaping of root canal system

As with access opening preparations, failure to pay close attention to detail during canal cleaning and shaping will result in violation of the principles of biomechanical canal preparation. These procedural errors and their sequelae can adversely affect the prognosis of treatment. The errors that most often occur during canal preparation include:

1. Loss of working length
2. Deviations from normal canal anatomy
3. Inadequate canal preparation
4. Perforations

LOSS OF WORKING LENGTH

Loss of working length during cleaning and shaping is a common procedural error. The problem may be noted only on the master-cone radiograph or when the master apical file is short of established working length (Fig. 16.2).

The loss of working length is actually secondary to other endodontic procedural errors, since blockages, ledges and fractured instruments can all result in loss of working length. However, these problems are usually recognized during cleaning shaping procedures.

In most instances, loss of working length can be attributed to rapid increase in the file size and the accumulation of dentinal debris in the apical third of the canal (Fig. 16.3). Preventive measures include frequent irrigation with NaOCl, recapitulation and periodic radiographic verification of working length (Fig. 16.4). In other instances, lack of attention to detail, such as malpositioned instrument stops, variations in reference points, poor radiographic technique and improper use of instruments, can contribute to this problem.

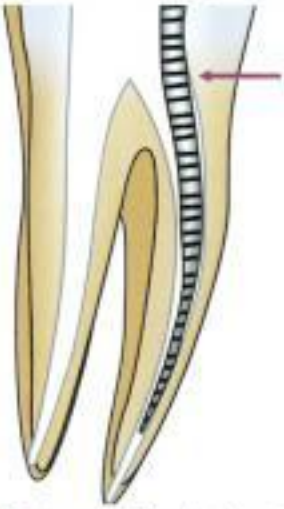


Fig. 16.2: Master apical file short of working length

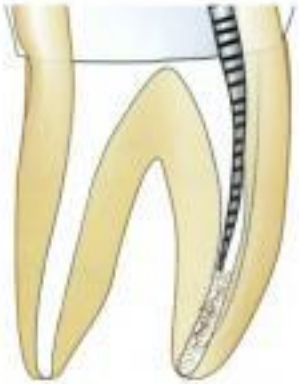


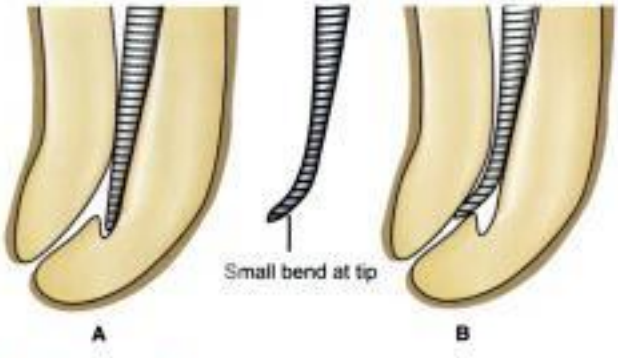
Fig. 16.3: Accumulation of dentinal debris in apical third because of loss of working length

dentinal wall. Rapid advancement in file sizes or skipping file size can also result in ledge formation. Ledges occur on the outer wall of the canal curvature. One may get suspicious that ledge has been formed when there is loss of tactile sensation at the tip of the instrument, loose feeling instead of binding at the apex. Above all the instrument can no longer reach its estimated working length. When in doubt a radiograph of the tooth with the instrument in place is taken to provide additional information.

Treatment

To negotiate a ledge, choose a smaller number file, usually No. 10 or 15. Give a small bend at the tip of the instrument (Fig. 16.11) and penetrate the file carefully into the canal. Once the tip of the file is apical to the ledge, it is moved in and out of the canal utilizing ultra short push-pull movements with emphasis on staying apical to the defect. When the file moves freely, it may be turned clockwise upon withdrawal to rasp, reduce, smooth or eliminate the ledge. When the ledge can be predictably bypassed, then efforts are directed towards establishing the apical patency with a No. 10 file. Gently passing 0.02 tapered 10 file 1 mm through the foramen ensures its diameter is at least 0.12 mm and makes the way for the 15 file. Nowadays, a significant improvement in ledge management is the utilization of nickel-titanium (NiTi) hand files that exhibit tapers greater than ISO files. Progressively tapered NiTi files can be introduced into the canal when the ledge has been bypassed, the canal negotiated and patency established.

Not all ledges can or should be removed. Clinicians must weigh risk versus benefit and make every effort to maximize remaining dentin.



Figs 16.11A and B: **A.** Formation of ledge by use of stiff instrument in curved canal. **B.** Correction of ledge; Ledge is bypassed by making a small bend at tip of instrument. Bent instrument is passed along canal wall to locate original canal

MISSED CANAL

Sometimes endodontic failure can occur because of untreated missed canals which are store house of tissue, bacteria and other irritants (Fig. 16.12). To avoid such problem to occur, one should have through knowledge of the root canal anatomy. There are several teeth which have predisposition for extra canal which might be missed if not explored accurately while treatment. For example maxillary premolars may have three canals (mesiobuccal, distobuccal and palatal), upper first molars usually have four canals, mandibular incisors usually have extra canal, mandibular premolars often have complex root anatomy and mandibular molars may have extra mesial and /or distal canal in some cases.

Missed canals can be located by:

1. Taking radiographs.
2. Use of magnifying glasses, endomicroscope.
3. Accurate access cavity preparation.
4. Use of ultrasonics.
5. Use of dyes such as methylene blue.
6. Use of sodium hypochlorite: After thorough cleaning and shaping, pulp chamber is filled with sodium hypochlorite. If bubbles appear in, it indicates either there is residual tissue present in a missed canal or

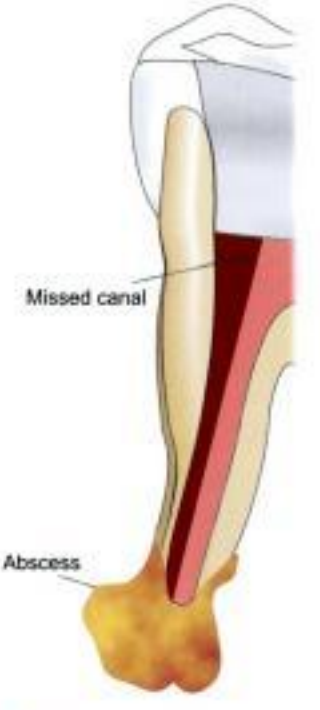


Fig. 16.12: Missed canal leading to root canal failure

residual chelator in the prepared canal. This is called as "***Champagne test***".

Missed canals contribute to endodontic failure because bacterial and other irritants. The tooth should be retreated first conservatively if endodontic failure exists, before going for endodontic surgery procedure (Figs 16.13A and B).

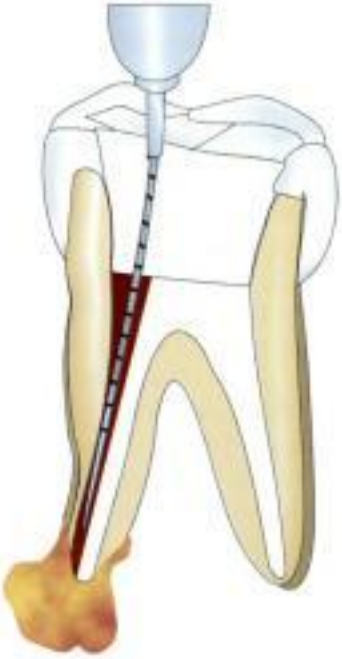


Fig. 16.13A: Retreatment of endodontically treated tooth with failure due to poor obturation

Fig. 16.13B: Completion of endodontic retreatment. Three dimensional obturation of root canal system is main aim of endodontic therapy

INSTRUMENT SEPARATION

Instrument breakage is a common and frustrating problem in endodontic treatment which occurs by improper or overuse of instruments. Any time during the cleaning and shaping of root canal file, reamer, broach or Gates Glidden may break especially while working in curved, narrow or tortuous canals (Fig. 16.14).

Certain factors affect the instrument separation and their removal for example cross sectional diameter, curvature and length of the canal (Fig. 16.15) , location of the separated instrument and type of the broken material, i.e. whether stainless steel or NiTi.

Studies have shown that instrument separation in root filled teeth with necrotic pulps results in a poorer prognosis. But if an instrument can be bypassed and incorporated in the root canal filling, prognosis becomes favorable. Also if

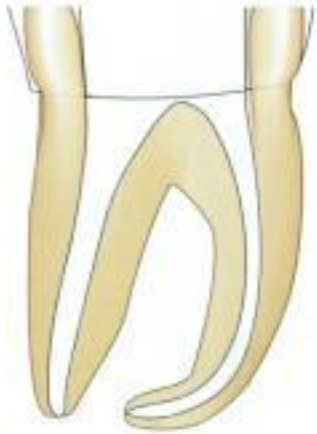


Fig. 16.14: Curved, narrow and tortuous canals are more prone for instrument fracture

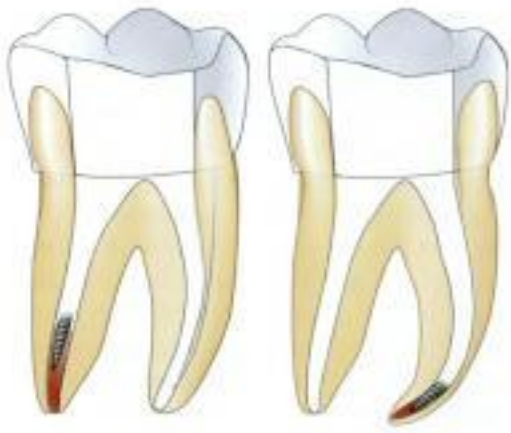


Fig. 16.15: Diameter, curvature of canal and location of instrument affects its removal

instrument separates at later stages of instrumentation and close to apex, prognosis is better than if it separates in undebrided canals, short of the apex or beyond apical foramen. Separated instrument is not the prime cause of endodontic failure but separated instruments impede mechanical instrumentation of the canal, which may cause endodontic failure.

Before going for removal of broken instrument, evaluate the tooth radiographically. Before starting with the instrument retrieval efforts, one should ensure straight line access to the canal orifice.

File Bypass Technique

The key to bypass a file is establishing straight line access and patency with small instruments (Fig. 16.16). The initial attempts should be made with number 6 or 8 file. In order

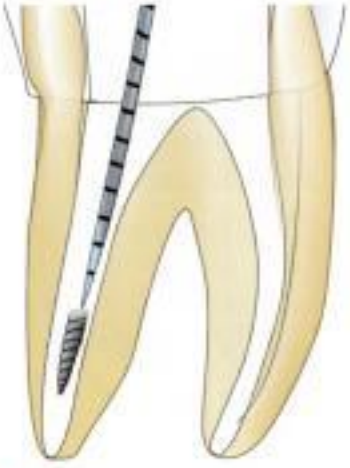


Fig. 16.16: Straight line access to instrument is primary requirement

to get past the broken instrument fragment, a small sharp bend should be given at the end of the instrument. Insert the file slowly and carefully into the canal. When the negotiation occurs past the fragment, one will find a catch. Don't remove file at this point. Use a small in and out movements along with copious irrigation of the root canal.

While doing these movements, sometimes file may kink, and one may not be able to place the file in the canal to the same length. In such cases, use new file with similar bend and repeat the above procedure. Once the patency is achieved with No.10 file, stick to it. It may get kinked or bent, discard it and start with new file. While doing this procedure, if patency is lost again repeat the procedure, starting with smaller number files.

Once patency with a No 15 instrument is achieved, go to K reamers. Use a "place-pull/rotate/withdrawal" movement rather than a filling motion. Two things will occur in response to this:

1. The reamer will be deflected by the fragment and then there is need to find a consistent path of instrument insertion that is probably different than the initial path.
2. Every time one rotates the reamer, there will be a "clicking" sound as the flutes brush up against the file fragment. This is normal. One must avoid placing an instrument directly on top of the broken file. This can push it deeper resulting in loss of patency. If the file is visible at this point, it is possible to use a small tipped ultrasonic instrument or $^{1}/_{4}$ turn withdrawal-type handpiece to dislodge and remove it.

In order to attempt file removal, exposure of fragment is mandatory. Instrument can be visualized using microscope. Modified Gates Gliddens can also be used to expose the instrument.

Gates-Glidden is modified by removing their bottom half and thus creating a flat surface. The crown down technique using Gates-Glidden burs is carried out. Once it is accomplished, use modified Gates-Glidden to enlarge the canal to a point where instrument is located; this way a platform is created which enable to visualize the broken fragment (Fig. 16.17). It creates a flat area of dentin surrounding the file fragment. Thereafter, small tipped

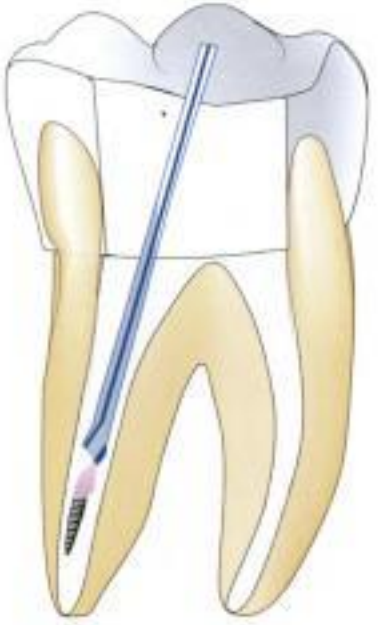


Fig. 16.17: Gates-Glidden modified to form a platform which enables to visualize broken fragment

ultrasonic instruments are used to through around the instrument and eventually vibrate the file out of the canal (Fig. 16.18). The tip is used in a counter clockwise motion to loosen the file. Irrigation with sodium hypochlorite and ultrasonics can frequently flush it out at this point. If sufficient file is exposed, an instrument removal system can be used.

If it is very difficult to remove the fractured instrument, incorporate the instrument fragment in the final obturation. (Fig. 16.19).

Surgical treatment for removal of broken fragment is indicated when:

1. Broken file is behind the curve.
2. File fragment is not visible because of the curved root.
3. Instrument is in the apical part of the canal and is difficult to retrieve (Fig. 16.20).
4. Much of dentin has to be removed to allow file removal.

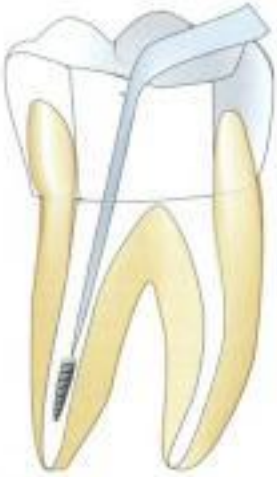


Fig. 16.18: Use of ultrasonic instrument to remove fractured instrument

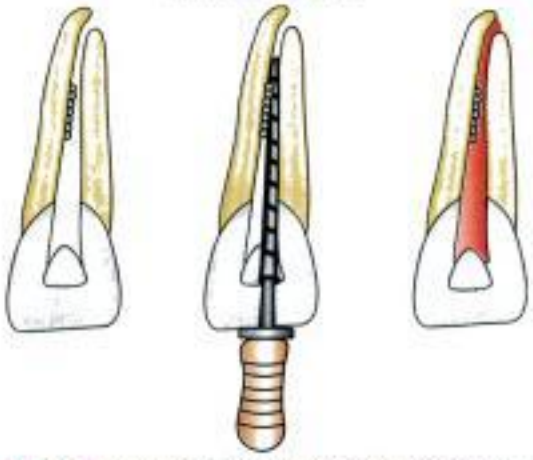


Fig. 16.19: If unable to remove the fractured instrument incorporate it in final obturation

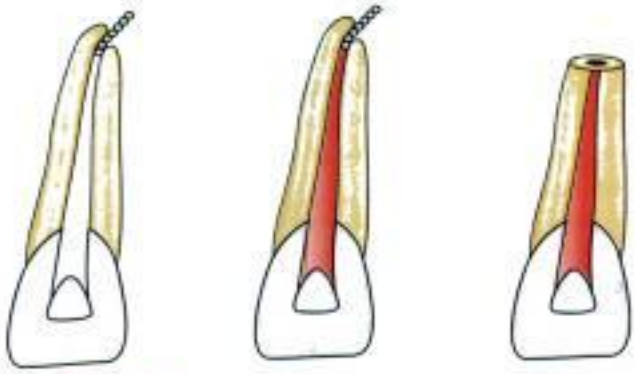


Fig. 16.20: Surgical removal of fractured instrument

PREVENTION OF INSTRUMENT SEPARATION

1. Instead of using carbon steel, use stainless steel files.
2. Use smaller number of instruments only once.
3. Examine each instrument before placing it into the canal.
4. Always use the instruments in sequential order.
5. Never force the instrument into the canal.
6. Canals should be copiously irrigated during cleaning and shaping procedure.
7. Never use instruments in dry canals.
8. Always clean the instrument before placing it into the canal. Debris collected between the flutes retard the cutting efficiency and increase the frictional torque between the instrument and canal wall.
9. Don't give excessive rotation to instrument while working with it.

ZIPPING

Zipping is defined as transposition of the apical portion of the canal (Fig. 16.21). This is commonly seen in curved canals because of following reasons:

Fig. 16.21: Zipping

1. Failure to precurve the files.
2. Forcing instruments in curved canal
3. Use of large, stiff instruments to bore out a curve canal. In this situation the apical foramen will tend to become a tear drop shape or elliptical and be transported from the curve of the canal.

Files placed in curved canal will cut more on the outer portion of the canal wall at its apical extent, thus causing movement of the canal away from the curve and its natural path. In contrast, the coronal third of the flutes will remove more on the inner most aspect of the canal wall causing an uneven reduction of the dentin in the coronal third.

When a file precurved or not, is rotated in a curved canal a biomechanical defect known as an elbow will form coronally to the elliptically shaped apical seat. This is the narrowest portion of the canal (Fig. 16.22). In many cases the obturating material terminates at the elbow leaving an unfilled zipped canal apical to elbow. This is the common occurrence with laterally compacted gutta-percha technique. Use of vertical compaction of warm gutta-percha or thermoplastisized gutta-percha would be ideal in these cases to compact a solid core material into the apical preparation without using excessive amount of sealer.

If a created elbow prevents optimal compaction in the apical portion of the canal, elbow becomes the apical seat. So, the obturating material is compacted against the elbow and patient is recalled or regular basis.

Zipping can be prevented by:

1. Using pre-curved files for curved canals.
2. Using incremental filling technique.
3. Using flexible files.

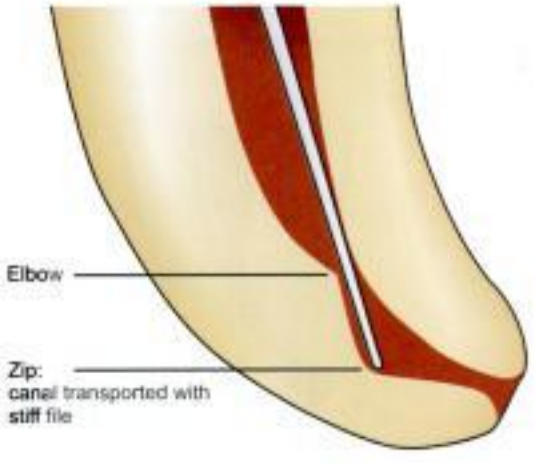


Fig. 16.22: Elbow formed in a curved canal

4. Removing flutes of file at certain areas, for example file portion which makes contact with outer dentinal wall at the apex and portion which makes contact with inner dentinal wall especially in the mid root area (Fig. 16.23).
5. Over curving in apical part of the file specially when working for severely curved canals.

Fig. 16.23: Modification of flutes

STRIPPING OR LATERAL WALL PERFORATION

"Stripping" is a lateral perforation caused by over instrumentation through a thin wall in the root and is most likely to happen on the inside or concave wall of a curved canal such as distal wall of mesial roots in mandibular first molars (Fig. 16.24). Stripping is easily detected by sudden appearance of hemorrhage in a previously dry canal or by a sudden complaint by patient.

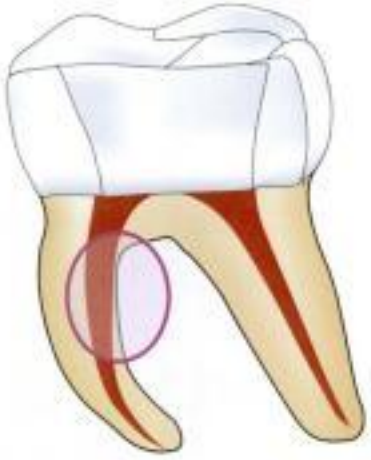


Fig. 16.24: Strip perforation occurs more commonly on inner side of curve

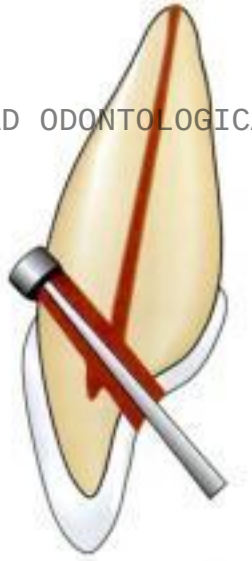


Fig. 16.32: Perforation caused during access cavity preparation

Fig. 16.33: Perforation caused by misdirection of bur during access cavity preparation of a molar with previously placed crown

third of the root canals. Sudden appearance of blood from canal is the first sign of perforation.

b. ***Mid root perforations:*** They commonly occur in the curved canals when a ledge has formed during instrumentation or along inside the curvature of root canal, as it is straightened out, i.e. strip perforation has resulted (Fig.16.35). Usually it is caused by over-instrumentation and over-preparation of the thin wall of root or concave side of the curved canals. Sudden appearance of bleeding is the pathognomic feature.

c. ***Apical root perforations:*** Apical root perforations occur-

I. When instrument goes into periradicular tissue, i.e. beyond the confines of the root canal (Fig.16.27).

II. Overuse of chelating agents along with straight and stiffer large sized instruments to negotiate ledging, canal blockage or zipping, etc.

Occurrence of a perforation can be recognized by:

1. Placing an instrument into the opening and taking a radiograph.
2. Using paper point (Fig.16.28).
3. Sudden appearance of bleeding.
4. Complain of pain by patient when instrument touches periodontal tissue

Repair of the Perforation

Treatment of the endodontics perforation depends on recognition of the condition, location, size, level of the perforation, timing of therapeutic intervention and clinician's skill and experience. Prognosis of endodontically treated teeth with perforation depends upon prevention of bacterial infection of the perforation site.

Location greatly influences the prognosis. When perforation is located at alveolar crest or coronal to it, prognosis is poor because of epithelial migration and periodontal pocket formation. Perforation in the furcation

Fig. 16.34: Misorientation of bur causing perforation during access cavity preparation

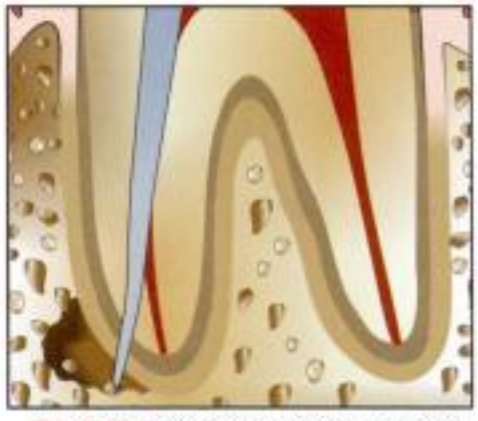


Fig. 16.35: Perforation caused by use of stiff instruments in a curved canal

area has the poor prognosis. Perforation in coronal third of root and surrounded by a healthy periodontium, i.e. which doesn't communicate with the gingival sulcus has good prognosis. Perforation occurring in midroot and apical part of root doesn't have communication with oral cavity and thus has good prognosis.

Size of perforation too affects the prognosis. A small perforation has less tissue destruction and inflammation, thus having better prognosis than larger sized perforation.

Visibility, accessibility also affects the perforation repair.

Time between perforation repair affects the prognosis greatly. The perforation should be repaired as soon as possible to discourage further loss of attachment and prevent sulcular breakdown. Early treatment enhances the success.

Associated periodontal condition and strategic importance of tooth also influence the treatment plan of the perforation. If attachment apparatus is intact without pocketing, nonsurgical repair is recommended where as in case of loss of attachment, surgical treatment should be planned.

In addition **esthetics** influences the perforation repair and material to be used for repair of the perforation.

MATERIAL USED FOR PERFORATION REPAIR

Various materials have been tried for perforation repair since long with variable degree of the success.

An Ideal Material for Perforation Repair should

- Adhere to preparation walls of the cavity and seal the root canal system.
- Be non toxic
- Be easy to handle
- Be radiopaque
- Be dimensionally stable
- Be well tolerated by periradicular tissue
- Be non absorbable
- Not corrode
- Not be affected by moisture
- Not stain periradicular tissues.

Some of the most investigated materials for perforation repair include amalgam, calcium hydroxide, IRM, Super EBA, gutta-percha, MTA, other materials tried for repair include dentin chips, hydroxyapatite, glass ionomer cements and plaster of paris.

For perforation repair, hemostatics are needed to control the hemorrhage and make the area dry so that optimal placement of restorative material can be accomplished. Materials which can be used as hemostatics include calcium hydroxide, calcium sulphate, freezed dried bone and/or MTA.

Which ever is the material used, the ultimate goal is to seal the defect with a biocompatible material and maintain an intact periodontal attachment apparatus.

Management of the Coronal Third Perforations

In these cases esthetics is the main concern. Here the materials used for perforation repair could be calcium sulphate barrier along with composites, glass ionomer cements and white MTA. But in posterior teeth where aesthetics is not the main criteria, super EBA, amalgam, MTA can be tried.

Management of Perforations in Mid Root Level

In these cases, the success of perforation repair depends on the hemostasis, accessibility and visibility, use of microinstrumentation techniques and selection the material for repair.

If the defect is small and hemostasis can be achieved, perforation can be sealed and repaired during three dimensional obturation of the root canal. But in case the perforation defect is large and moisture control is difficult, then one should prepare the canal before going for perforation repair.

Lemon in 1992 gave the internal matrix concept for the repair of inaccessible strip perforations using microsurgical technique. The rationale behind this concept was that a matrix was needed to control the material and thus preventing overfilling of the repair material into the periradicular tissues.

Several materials meet these criteria like cavit, amalgam, zinc oxide eugenol, gutta-percha and sealer. But none of these can overcome contamination, underfilling and overfilling. According to Lemon, hydroxyapatite could be used for accessible perforations.

A Material to be Used as Internal Matrix should

a. Be biocompatible
b. Be sterile
c. Be easy to manipulate
d. Stimulate osteogenesis

Technique of Placement of Matrix

1. Attain the hemostasis and place files, silver cones or gutta-percha points in the canals to maintain their patency.
2. The hydroxyapatite (HA) particles are wetted with saline and clumped together for their easy transportation.

MTA is composed of Tricalcium silicate, Dicalcium silicate, Tricalcium aluminate, Tetracalcium aluminaferrite, calcium sulfate and bismuth oxide. The main advantage of this material is that being hydrophilic it requires moisture to set. It sets in brick hard consistency and shown to induce cementogenesis, bone deposition with almost absence of inflammatory response. All these make MTA a material of choice in treatment of perforations (Fig.16.39).

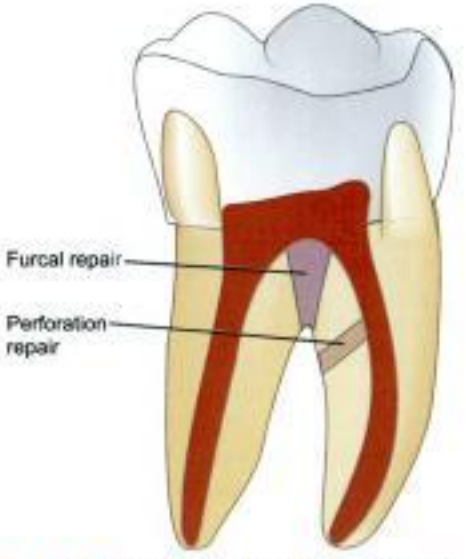


Fig. 16.39: Use of MTA for repair of perforation

Perforations can be avoided by:

1. Evaluation of the anatomy of the tooth before starting the endodontic therapy.
2. Using the smaller, flexible files for curved canals.
3. Not skipping the filling sizes.
4. Recapitulation with smaller files between sizes.
5. Confirming the working length and maintaining the instruments with in the confines of working length.
6. Using anticurvature filling techniques in curved canals to selectively remove the dentin.
7. Minimizing the overuse of Gates Gliddens too deep or too large especially in curved canals.
8. Avoiding overuse of chelating agents, larger stiff files in order to negotiate procedural errors like ledges, canal blockages, etc.
9. Copious irrigation of the canal to prevent the canal blockage by dentin chips or tissue debris.

POST SPACE PERFORATIONS

Post preparation is an integral part of restoration of endodontically treated tooth. Iatrogenic perforations during post space preparations can severely impair the prognosis

Fig. 16.40: Post space perforation caused by misdirection of post preparing drills

of the tooth. They are usually caused by poor clinical judgment and improper orientations of the post preparing drills (Fig.16.40). Perforation can be recognized by sudden appearance of blood in the canal or radiographically. The error of this iatrogenic perforation is compounded when clinician is unaware of the perforation and proceeds with post placement in the perforated site.

Treatment of this perforation involves the same principles as of other perforation repair, i.e. sealing of the perforation site is the primary goal. Perforation can be repaired in several ways. The defect can be accessed both surgically as well as non-surgically. Various materials like dental amalgam, calcium hydroxide, glass ionomer, composite resins, cavity, freezed dried bone and tri-calcium phosphate can be used to repair the perforation.

Prevention of Post-related Perforation

1. One must know anatomic features of root including radicular considerations of root anatomy as well its variations.
2. One should opt preparing post space at the time when obturation of root is done.
3. Avoid excessive use of Gates Gliddens or peeso reamers to cut the dentin.

UNDER FILLING/INCOMPLETELY FILLED ROOT CANALS

Under filling, i.e. more than 2 mm short of radiographic apex occurs commonly because of procedural errors like ledge formation, blockage or incomplete instrumentation of the root canal.

Inaccurate working length determination, inadequate irrigation and recapitulation during biomechanical preparation can lead to accumulation of dentin chips and tissue debris which will result in canal blockage and incomplete instrumentation of the canal (Fig.16.41).

Ledge can be caused by using (Fig.16.42)

- Large stiff files in curved canals.
- Inadequate straight line access to canals apices
- Inadequate irrigation.
- Skipping the file sizes during biomechanical preparation.
- Packing dentin chips, tissue debris in apical portion of the canal.

All these lead to inadequate removal of infected necrotic tissue remaining in the apical portion of the root canal. In teeth with periapical pathosis bacteria get colonized at and around the apical foramen. Thus persistent bacterial infection in the root canal may initiate or perpetuation of existing periapical pathosis will result (Fig.16.43). Fillings short of apex have shown poorer prognosis, especially those with necrotic pulp and periradicular pathosis.

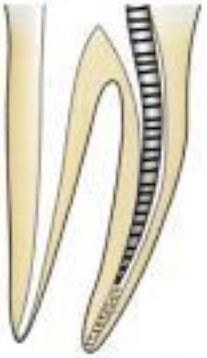


Fig. 16.41: Accumulation of dentin chips and tissue debris resulting in incomplete instrumentation

Fig. 16.42: Inadequate instrumentation of curved canal causing ledge formation

Fig. 16.43: Persistent bacterial infection in root canal with filling short of apex causes treatment failure

Various research studies have shown that underfillings per se don't have direct effect on the success of endodontic treatment but it is the remaining infected necrotic tissue in inadequately, instrumented and incompletely filled canal which lead to continued irritation to the periradicular tissues. Consequently for the ultimate success of endodontic therapy the practice of underfilling should be avoided. This can be prevented by:

1. Obtaining straight line access to canal orifices to apex.
2. Precurving the files before using in curved canals.
3. Copious irrigation and recapitulation of the canal.
4. Attaining apical patency.
5. Using EDTA in vital cases especially to emulsify the pulp and remove it completely.
6. Using the files sequentially.
7. Clinician should feel the tensional binding of the file which exists at minor constriction of the apical foramen.

OVERFILLING OF THE ROOT CANALS

Overfilling of the root canals, i.e. filling more than 2 mm beyond the radiographic apex (Fig.16.44) often occurs because of:

1. Over instrumentation of the root canal.
2. Inadequate determination of the working length.
3. Incompletely formed root apex.
4. Inflammatory apical root resorption.
5. Improper use of reference points for measuring working length.

Not all teeth can lead to endodontic failures. The response of periradicular tissues to root canal filling materials depend

2. Use less tapered and more flexible compacting instruments to control condensation forces while obturation.
3. Root should be dentinal thickness are necessary to retain a tooth.

INSTRUMENTS ASPIRATION

Aspiration of instruments can occur during endodontic therapy if accidentally dropped in the mouth. It occurs specially in absence of rubber dam. It is a type of emergency which has to be tackled as soon as possible. Patient must be provided medical care for examination which includes radiograph of chest and abdomen.

High volume suction tips, hemostats or cotton pliers can be helpful only in some cases, when the objects are readily accessible in throat otherwise medical care is needed. This accident can be prevented by:

1. Use of rubber dam.
2. Tying up the rubber dam clamp or endodontic instrument with floss.

17 Endodontic Failures and Retreatment

- Introduction
- Definitions Related to Endodontic Treatment Outcome
- Evaluation of Success of Endodontic Treatment
- Causes of Endodontic Failures
- Case Selection and Endodontic Retreatment
- Steps of Retreatment

INTRODUCTION

Since long, many studies are being conducted to determine success and failures of endodontic treatments. In different studies success rate ranges from 54 percent to 95 percent.

The definition of success is ambiguous; it has different meaning when referring to different criteria. For example success in endodontic therapy is ambiguous with requirements ranging from stringent (radiographic and clinical normalcy) to the lenient (only clinical normalcy). Obviously the more lenient definition increases the success rate when compared with the stringent one.

Success is defined by goals established to be achieved. The usual goal of endodontic therapy is to prevent or heal the disease. Accordingly, endodontic treatment outcomes should be defined in reference to healing and disease.

Definitions Related to Endodontic Treatment Outcome

Healed: Both clinical and radiographic presentations are normal

Healing: It is a dynamic process, reduced *radiolucency* combined with normal clinical presentation

Disease: No change or increase in *radiolucency*, clinical signs may or may not be present or vice versa.

The properly executed root canal treatment has shown the success rate in 95 percent of the cases. Yet failures occur and the root canal failures are commonly caused by ramifications of the infected tooth, periapical or the surrounding periodontium (Fig. 17.1).

A clear definition of what constitute a failure following endodontic therapy is not yet clear. Failures can not be subscribed to any particular criteria of evaluation; instead success or failures following endodontic therapy could be evaluated from combination of various criteria like clinical, histopathological and radiographical criteria.

Fig. 17.1: Non-healing periapical abscess due to poor obturation

EVALUATION OF SUCCESS OF ENDODONTIC TREATMENT

Clinical Evaluation

Presence of symptoms though indicates the presence of pathology, but absence of a pain or any other symptoms does not confirm the absence of a disease. A little correlation exists between the presence of symptoms and the periapical disease.

Clinical Criteria for Success

- No tenderness to percussion or palpation
- Normal tooth mobility
- No evidence of subjective discomfort
- Tooth having normal form, function and aesthetics
- No sign of infection or swelling
- No sinus tract or integrated periodontal disease
- Minimal to no scarring or discoloration.

Radiographic Evaluation

The radiographic criteria for failures are development of radiographic periapical areas of rarefaction after the endodontic treatment, in cases where they were not present before the treatment or persistence or increase in sizes of the radioleucency after the treatment. To predict the success or failure, one should be able to accurately compare the radiographs that are taken at different times (Fig. 17.2).

Prognosis is prediction of whether an endodontic treatment will be successful or a failure and if successful to what degree it will be. Normally, the development of apical periodontitis is indication of endodontic failures, is frequently asymptomatic clinically, and the radiograph is the way to determine the success here.

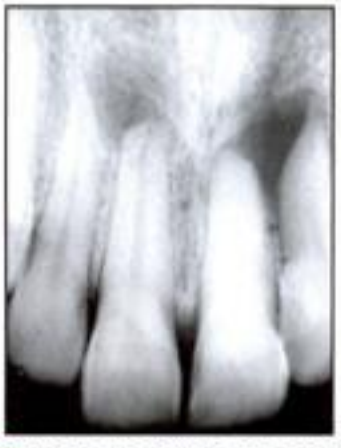


Fig. 17.2: Radiograph showing periapical abscess in relation to central incisor

Radiographic Criteria for Success of Endodontic Treatment

- Normal or slightly thickened periodontal ligament space
- Reduction or elimination of previous rarefaction
- No evidence of resorption
- Normal lamina dura
- A dense three dimensional obturation of canal space.

Histological Evaluation

Histological criteria for success or failure of endodontic therapy may include absence of inflammation and regeneration of periodontal ligament, bone and cementum following endodontic therapy. Histologically, the success of endodontically treated tooth is reduced because chronic inflammation may persist for long even without any symptoms.

Histological Criteria for Success

- Absence of inflammation
- Regeneration of periodontal ligament fibers
- Presence of osseous repair
- Repair of cementum
- Absence of resorption
- Repair of previously resorbed areas.

CAUSES OF THE ENDODONTIC FAILURES

Most commonly the causes of root canal failures are directly or indirectly related to bacteria somewhere in the root canal system. The treatment failures can occur despite of the strict adherence to the basic treatment principles. Multitude of factors affect the success or failure of the endodontic treatment but there are certain factors which are common in all the cases for their success or failure and in some cases, success or failure is particularity related to that individual case.

Factors Affecting Success or Failure of Endodontic Therapy in Every Case:

- Diagnosis and the treatment planning
- Radiographic interpretation
- Anatomy of the tooth and root canal system
- Debridement of the root canal space
- Asepsis of treatment regimen
- Quality and extent of apical seal
- Quality of post endodontic restoration
- Systemic health of the patient
- Skill of the operator.

Overfilling of root canals: Overfilling of root canals i.e. obturation of the canal extending more than 2 mm beyond radiographic apex. It occurs because of apical root resorption, improper working length of roots and over instrumentation of the root canal system. Overfilling of the root canals may cause endodontic failure because of continuous irritation of the periapical tissues (Fig. 17.14). The filling material acts as a foreign body which may generate immunological response. Biofilms (accumulation of microorganisms embedded in self produced extracellular polysaccharide matrix, adherent to solid surface) are seen on the extruded material which contains the treatment resistant bacteria.

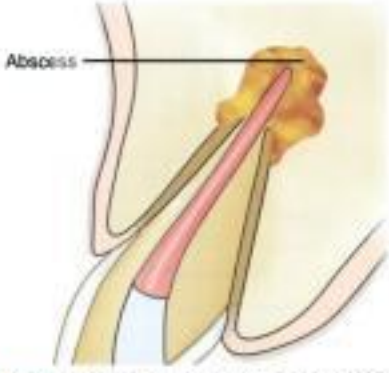


Fig. 17.14: Overfilling of root canal resulting in non healing of periapical abscess

Corrosion of root canal fillings: Corrosion is the tendency of most of the metals to revert to their lower form by oxidation. Silver cones have shown to produce corrosion. The main area of corrosion of silver cones is coronal and the apical portions, the areas which contact tissue fluids either periapical exudation or saliva. The corrosion products are cytotoxic and may act as tissue irritants causing persistent periapical inflammation.

Anatomic factors: Presence of overly curved canals, calcifications, numerous lateral and accessory canals, bifurcations, aberrant canal anatomy like C or S shaped canals may pose problems in adequate cleaning and shaping and thereby incomplete filling of the root canals (Figs 17.15 to 17.17). These can lead to endodontic failure.

Root Fractures (Fig. 17.18): Endodontic failures can occur by partial or complete fractures of the roots. Prognosis of teeth with vertical root fracture is poorer than horizontal fractures.

Fig. 17.15: Curved root canal

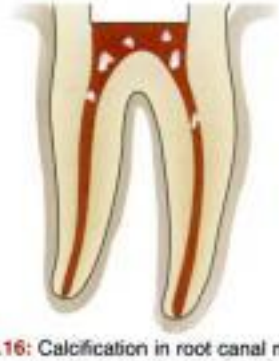


Fig. 17.16: Calcification in root canal making the endodontic treatment difficult

Fig. 17.17: Numerous lateral and accessory canals making it difficult to completely debride the root canal system

Fig. 17.18: Root fracture

Traumatic occlusion: Traumatic occlusion has also been reported to cause endodontic failures because of its effect on periodontium.

Periodontal considerations: An endodontic failure may occur because of communication between the periodontal ligament and the root canal system. Also the recession of attachment apparatus may cause exposure of lateral canals to the oral fluids which can lead to reinfection of the root canal system because of percolation of fluids (Fig. 17.19).

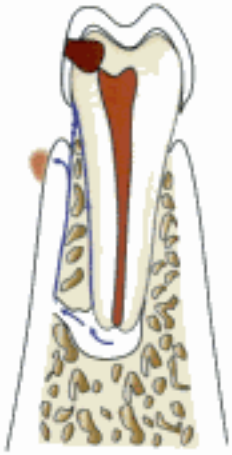


Fig. 17.19: Communication between root canal system and periodontal area due to recession of attachment apparatus

Systemic Factors

Systemic factors influence the success or failure of the endodontic therapy. The systemic diseases may influence the local tissue resistance and thus interfering with the normal healing process.

When systemic disease is present, the response of the periapical tissues may get intensified if there is increase in concentration of irritants during endodontic therapy. Thus a severe reaction may occur following cleaning and shaping, i.e. mechanical irritation and chemical irritation from medicaments and irrigants, causing dispersion of the microorganisms. Healing is also impaired in patients with systemic disease. Various systemic factors which can interfere in the success of endodontic therapy are nutritional deficiencies, diabetes mellitus, renal failure, blood dyscrasias, hormonal imbalance, autoimmune disorders, opportunistic infections, aging, and patients on long term steroid therapy Thus before starting endodontic therapy, a complete medical history is essential to predict the prognosis of the tooth.

Factors Responsible for Endodontic Failures

Local	*Systemic*
• Infection	• Nutritional deficiencies
• Incomplete debridement of the root canal system	• Diabetes mellitus
• Excessive hemorrhage	• Renal failure
• Over instrumentation	• Blood dyscrasias
• Chemical irritants	• Hormonal imbalance
• Iatrogenic errors	• Autoimmune disorders
– Separated instruments	• Opportunistic infections
– Canal blockage and ledge formation	• Aging
– Perforations	• Patients on long term steroid therapy
– Incompletely filled teeth	
– Overfilling of root canals	
• Corrosion of root canal fillings	
• Anatomic factors	
• Root fractures	
• Traumatic occlusion	
• Periodontal considerations	

The tooth which has experienced the endodontic failure could be treated either by nonsurgical retreatment, surgical or combination of both procedures. In endodontic retreatment, one attempt to eliminate the root canal microorganisms but in case of surgical correction, there is an attempt to confine the microorganisms within the root canal.

Before going for endodontic retreatment, following factors should be considered:

a. When should treatment be considered, i.e. if patient is asymptomatic even if treatment is not proper, the retreatment should be postponed.
b. Patient's needs and expectations.
c. Strategic importance of the tooth.
d. Periodontal evaluation of the tooth.
e. Other interdisciplinary evaluation.
f. Chair time and cost.

Retreatment can be differentiated from the normal endodontic therapy in its unique considerations and techniques.

Before performing endodontic retreatment following points should be considered:

a. Retreatment may be performed to prevent the potential disease.
b. To gain access into root canal extensive coronal restoration has to be removed and remade.
c. Technical problems may result from previous treatment or aberrant canal anatomy.

d. Even after retreatment sometimes better results may not be achieved.
e. Root canal filling materials have to be removed during retreatment.
f. Prognosis of retreatment could be poorer than the initial endodontic therapy.
g. Patient might be more apprehensive than with initial treatment.

CASE SELECTION FOR ENDODONTIC RETREATMENT

Retreatment is usually indicated in symptomatic endodontically treated teeth or in asymptomatic teeth with improperly done, initial endodontic therapy to prevent future emergence of the disease.

1. Careful history of patient should be taken to know the nature of case, pathogenesis and urgency of the treatment etc.
2. Evaluate the anatomy of root canal in relation to canal curvature, calcifications, unusual configurations etc.
3. Evaluate the quality of obturation of primary endodontic treatment.
4. Check for iatrogenic complications like separated instruments, ledges, perforations, zipping, canal blockages etc.
5. Consider the cooperation of the patient which is mandatory for retreatment procedure.

Factors Affecting Prognosis of Endodontic Treatment

- Presence of any periapical radioleucency
- Quality of the obturation
- Apical extension of the obturation material
- Bacterial status of the canal
- Observation period
- Post-endodontic coronal restoration
- Iatrogenic complication

Contraindications of Endodontic Retreatment

- Unfavourable root anatomy (shape, taper, remaining dentin thickness)
- Presence of untreatable root resorptions or perforations.
- Presence of root or bifurcation caries.
- Insufficient crown/root ratio.

Problems Commonly Encountered during Retreatment

- Unpredictable result.
- Frustration
- Cost factor
- Time consuming

STEPS OF RETREATMENT

- Coronal disassembly
- Establish access to root canal system
- Remove canal obstructions
- Establish patency
- Thorough cleaning, shaping and obturation of the canal.

Coronal Disassembly

Endodontic retreatment procedures commonly require removal of the existing coronal restoration (Fig. 17.20). But in some cases access can be made through the existing restoration (Fig. 17.21).

Fig. 17.20: Retreatment of a premolar with endodontic failure and Access is made by removing coronal restoration

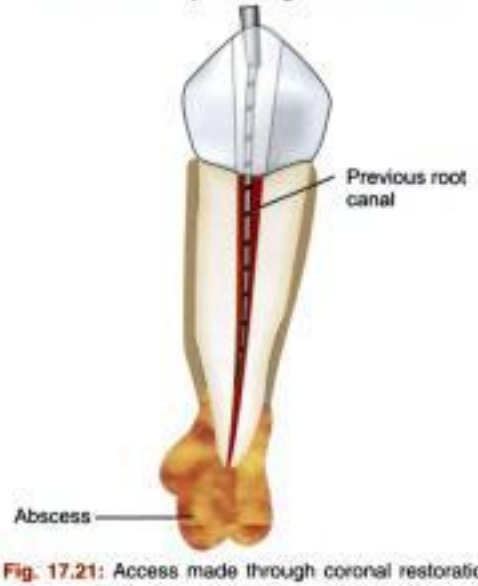


Fig. 17.21: Access made through coronal restoration

Gaining access through original restoration helps in:

a. facilitating rubber dam placement
b. maintaining form, function and aesthetics
c. reducing the cost of replacement.

But disadvantages of retaining a restoration include:

a. Reduced visibility and accessibility
b. Increased risks of irreparable errors.
c. Increased risks of microbial infection if crown margins are poorly adapted.

It is advisable to remove the existing restoration especially if it has poor marginal adaptation, secondary caries to avoid procedural errors (Fig. 17.22). To maintain form, function and aesthetics, temporary crown can be placed.

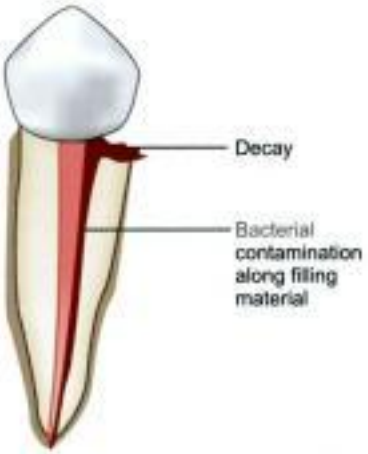


Fig. 17.22: Previous coronal restoration with secondary caries is indicated for removal

Establish Access to Root Canal System

Some teeth are restored with post and core which need to be removed for gaining access to root canal system (Figs 17.23 and 17.24) or sometimes they can be perforated to gain access (Fig. 17.25).

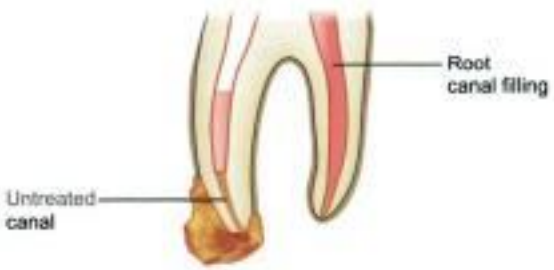


Fig. 17.23: Previously treated tooth with endodontic failure

Fig. 17.24: Access made by removal of coronal restoration

Fig. 17.25: Access made through coronal restoration

Posts can be removed by various methods. These are:

1. Weakening retention of posts by use of ultrasonic vibration (Fig. 17.26).

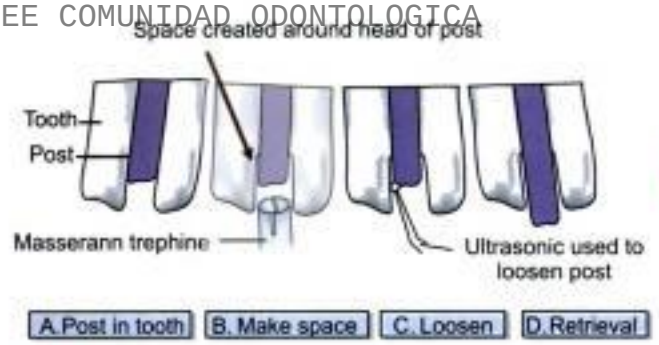


Fig. 17.26: Weakening retention of post using ultrasonic vibration

2. Forceful pulling of posts but it increases the risk of root fracture.
3. Removing posts with the help of special pliers using post removal systems.
4. Occasionally access can be made through the core for retreatment procedure without disturbing the post.

The recently developed Post Removal System (PRS) simplified the removal of post from the canal. For use of PRS kit, one should have straight line access to the canal and also the post should be easily visualized from the chamber.

PRS kit consists of five variously designed trephines and corresponding taps, a torque bar, a transmeatal bur, rubber bumpers and extracting pliers.

1. Initially a transmeatal bur is used for efficiently dooming of the post head (Fig. 17.27).

Fig. 17.27: Use of transmeatal bur over post head

2. Then a drop of lubricant such as RC Prep is placed on the post head to further facilitate the machining process.
3. After that select the largest trephine to engage the post and to machine down the coronal 2-3 mm of the post (Fig. 17.28).

Fig. 17.28: Place trephine over 2-3 mm coronal portion of the post

4. Followed by a PRS microtubular tap is inserted against the post head and screwed it into post with counter clockwise direction. Before doing this rubber bumper is inserted on the tap to act as cushion against forces (Fig. 17.29).

Fig. 17.29: Insertion of microtubular tap against post head

5. When tubular tap tightly engages the post, rubber bumper is pushed down to the occlusal surface.
6. Mount the post removal plier on tubular tap by holding it firmly with one hand and engaging it with other hand by turning screw knob clocking if post is strongly bonded in the canal, then ultrasonic instrument is vibrated on the tap or a torque bar is inserted onto the handle to increase the leverage, thereby facilitating its removal (Fig. 17.30).

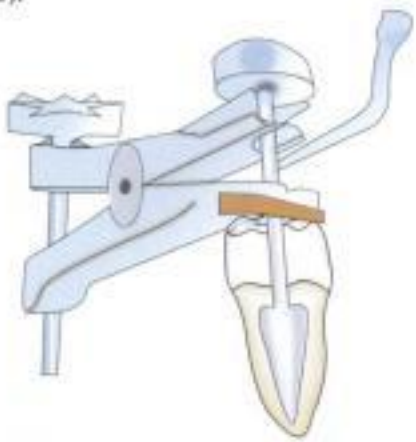


Fig. 17.30: Activation of extracting plier with ultrasonic tip

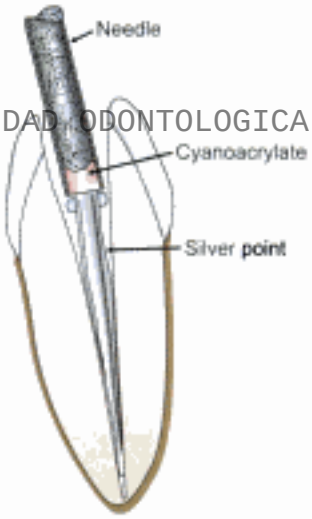


Fig. 17.34: Use of hypodermic needle for removal of silver point

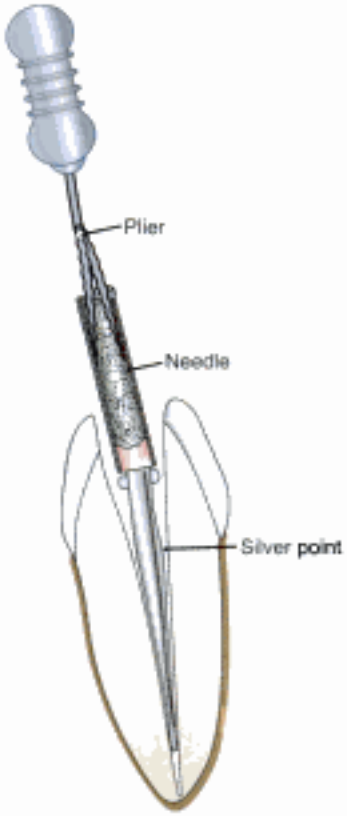


Fig. 17.35: Use of plier for removal of silver point

e. By tap and thread option using microtubular taps from post removal the post removal system kit.
f. Using instrument removal system.

Gutta-Percha Removal

The relative difficulty in removing gutta-percha is influenced by length, diameter, curvature and internal configuration of the canal system. Irrespective of the technique gutta-percha is best removed from root canal in progressive manner to prevent its extrusion periapically. Coronal portion of gutta-percha should always be explored by Gates Gliddens to:

a. Remove gutta-percha quickly.
b. Provide space for solvents.
c. Improve convenience form.

Gutta-percha can be removed by:

- Using solvents
- Using hand instruments
- Using rotary instruments
- Using microdebrider.

Use of solvents to remove gutta-percha: Since it has been seen that gutta-percha is soluble in chloroform, methyl chloroform, benzene, xylene, eucalyptol oil, halothane and rectified white turpentine, it can removed from the canal by dissolving it among these solvents (Fig. 17.36).

Being highly volatile, chloroform is most effective so commonly used. Since at high concentrations, it has shown to be carcinogenic, its excessive filling in pulp chamber is avoided.

Gutta-percha dissolution has to be supplemented by further negotiation of the canal and removing the dissolved material from it (Fig. 17.37).

Fig. 17.36: Use of solvent for removal of gutta-percha

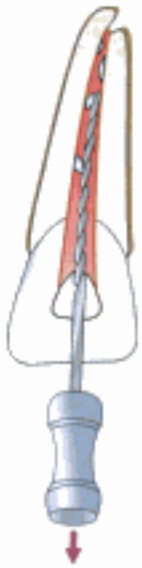


Fig. 17.37: Further removal of dissolved gutta-percha using hand instrument

Use of hand instruments: Hand instruments are mainly used in the apical portion of the canal. Poorly condensed gutta-percha can be easily pulled out by use of files. Hedstroem files are used to engage the cones so that they can be pulled out in single piece (Fig. 17.38). Removal of gutta-percha can also be done by using hot endodontic instrument like file or reamer (Fig. 17.39). Reamers or files can be used to bypass the gutta-percha sometimes.

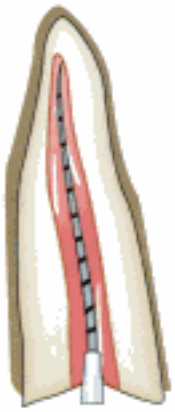


Fig. 17.38: Use of H-file for removal of gutta-percha

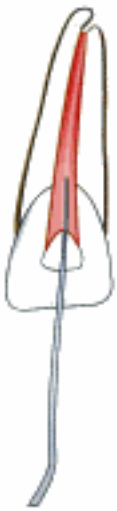


Fig. 17.39: Use of hot endodontic instrument for removal of gutta-percha

With overextended cones, files sometimes have to be extended periapically to avoid separation of the cone at the apical foramen. Sometimes cones which get separated at apex may not be retrieved.

Use of rotary instrumentation: Rotary instruments are safe to be used in straight canals. Recently in May 2006, new ProTaper universal system was introduced consisting of D_1, D_2, and D_3 to be used at 500-700 rpm.

D_1 file:
- Removes filling from coronal third
- 11 mm handle
- 16 mm cutting surface
- White ring for identification
- ISO 30 active tip for easier penetration of obturation material
- 9 percent taper file.

D_2 file:
- Removes filling from middle third.
- 11 mm handle
- 18 mm cutting blades
- Two white rings for identification
- ISO 25, non-active rounded tip to follow canal path
- 8 percent taper file.

D_3 file:
- Removes filling from apical third.
- 11 mm handle
- 22 mm cutting blades
- Three white rings for identification
- ISO 20 non-active rounded tip to follow canal path
- 7 percent taper file.

Using microdebriders: These are small files constructed with 90 degrees bends and are used to remove any remaining gutta-percha on the sides of canal walls or isthmuses after the repreparation.

Pastes and Cements

Pastes or cements can be of varying consistency and hardness.

Soft setting pastes can be removed using the normal endodontic instruments preferably using crown down technique (Fig. 17.40).

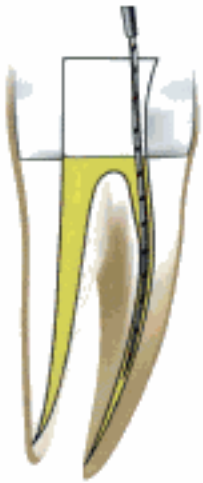


Fig. 17.40: Removal of soft setting paste using endodontic instrument

Hard setting cements like resin cements can be first softened using solvents like xylene, eucalyptol etc. and then removed using endodontic files. Ultrasonic endodontic devices can also be used to dislodge the pastes by vibrations and thus facilitate their removal (Fig. 17.41).

Hard setting pastes can also be drilled out using lone shank, small round burs.

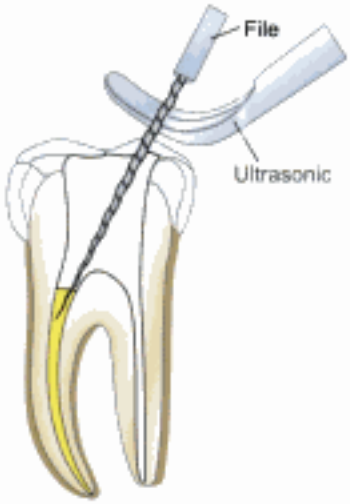


Fig. 17.41: Use of ultrasonics for removal of hard setting cement

Separated Instruments and Foreign Objects

Broken instruments or foreign objects can be retrieved from the canals but primary requirement for their removal is their accessibility and visibility. So, we one can say that if root canal is obstructed by foreign object in coronal third then attempt retrieval, in middle third, attempt retrieval or bypass and if it is in apical third leave or surgically treat.

The secret of removing the instruments broken in coronal and middle third is to recognize how much coronal tooth structure one has to remove to gain access to the instrument. If over preparation of canal compromises the dentin thickness, one should leave the instrument in place rather than compromising the coronal dentin.

If instrument is readily accessible, remove it by holding with instruments like Stieglitz pliers and Massermann extractor. Massermann extractor comprises a tube with a constriction into which a stylet is introduced to grasp the fractured instrument (Fig. 17.42).

Ultrasonics can also be used to remove the instruments by their vibration effect (Fig. 17.41).

Broken instruments can also be attempted for their removal using modified Gates Gladden bur and then creating a staging platform before using an ultrasonic tip to rotate around the file in a counterclockwise direction to remove it (Figs 17.43 and 17.44).

Fig. 17.42: Use of massermann extractor to remove fractured instrument

Fig. 17.43: Use of Gates Glidden drills to create staging plateform

Fig. 17.44: Removal of fractured instrument

When it is not possible to remove the foreign objects, attempts should be made to bypass the object and complete biomechanical preparation of the canal system.

Bypassing of instrument can be attempted using hand instruments like reamers and files. These instruments are inserted alongside the broken instrument to soften its cementation and thus facilitating its removal. While making efforts to the bypass the instrument, copious irrigation is needed. Irrigation with sodium hypochlorite, hydrogen peroxide and RC Prep may float the object coronally through the effervescence they create.

Use of ultrasonic K-file No. 15 or 20 with vibration and copious irrigation may also pull the instrument coronally.

Completion of the Retreatment

After gaining access to the root canal system, with its thorough cleaning and shaping and managing other complications, the treatment is completed using the routine procedures. But some times the retreatment may become

difficult due to presence of therapy resistant microorganisms like *Enterococcus faecalis*. During retreatment procedures a wide coronal access is needed and canal is slightly over enlarged than the previous one to completely remove the residues of previous treatment. Clinician may face difficulty in retreating a case especially if access is to be made through the previously placed restoration, post removal is impossible and if there is overextended gutta-percha, presence of gutta-percha or foreign objects, or hard setting pastes which are amenable to remove shows complete retreatment of an endodontic treatment failure in a premolar with periapical abscess.

The outcome of retreatment can be divided into short term and long-term. The short-term outcome may be associated with postoperative discomfort including pain and swelling. Long-term outcomes of retreatment depend mainly on regaining the canal patency and the obturation of the root canal system. It has been seen that retreatment is most frequently associated with the procedural compli-cations than the primary treatment. Thus an effective communi-cation is required between clinician and the patient about the potential problems before the treatment is initiated to avoid frustration.

18 Surgical Endodontics

- Introduction
- Indications and Contraindications
- Classification
- Presurgical Consideration
- Incision and Drainage
- Periradicular Surgery
- Anesthesia and Hemostasis
- Flap Designs and Incisions
- Hard Tissue Management
- Periradicular Currettage
- Root-end Resection
- Root-end Preparation
- Retrograde filling
- Root-end filling material
- Postsurgical Care
- Suturing materials and Techniques
- Postsurgical Complications

INTRODUCTION

Endodontic surgery is defined "as removal of tissues other than the contents of root canal to retain a tooth with pulpal or periapical involvement". Surgical intervention is required for cases where retreatment has failed or is not an option, and the tooth is to be retained rather than extracted. Endodontic therepy eliminates rooth canal flora by chemomechanical debridement followed by obturation of the canal to achieve a seal. Clinical radiographic and histological observation are the criteria for evaluating success. The percentage of success reported has been consistently high. Failures may arise due to inadequate control of infection, poor design of the access cavity, inadequate instrumentation and obturation, missed canals and coronal leakage. Factors for persistence of periapical radiolucencies after conventional canal treatment in well treated cases may be intraradicular infection, extraradicular infection, foreign body reaction and true cysts. Endodontic failures may be retreated, receive endodontic surgery or the problem tooth extracted.

The First cases of endodontic surgery were those performed by ***Abulcasis*** in the ***11th century***. A root end resection procedure to manage a tooth with a necrotic pulp and an alveolar abscess was documented in 1871 and root end resection with retrograde cavity preparation and filling with amalgam in the 1890's. Endodontic surgery was often considered as an alternative to root canal treatment and indications for surgery were proposed first in the 1930's. The rational for performing surgical treatment has changed over the past 120 years. The current view is that retreatment by conventional methods is the preferred alternative to apicoectomy.

In the 1960's surgery was indicated in a non-vital tooth if instrument had not reached within 2-3 mm of the root apex. The level of unfilled or poorly filled canal determined the level of resection, and at that time the cut was beveled towards the labial surface to improve visualization and ease

of placement of a root and filling. There was little evidence to show any increase in the rate of success by preparing and delaying the root canal filling until two consecutive negative cultures were obtained following antibacterial treatment. At that time appropriate anaerobic culturing techniques were not available, and calcium hydroxide was not in major use as a medicament. Also, in the 1960's it was believed that the main cause of endodontic failure was a poor apical seal.

Radiographic evaluation is important criteria to see the success of endodontic therapy, with increase in size or development of a lesion considered to be a failure. Usually a radiographic follow-up to 4 years is satisfactory for determining success. Biological and technical failures continue to occur as there are parts of root canal where debridement and obturation are not adequate. The reason for persistence of a radiolucency in what seem well treated cases are not yet fully understood. New devlopments in root canal treatment in the last 10 years include an increased use of microscopes, preparations using ultrasound, canal preparations.

INDICATIONS

Indications of Endodontic Surgery—As given by ***Luebke, Click and Ingle***

1. Need for surgical drainage
2. Failed non-surgical treatment:
   - Irretrievable root canal filling material.
   - Irretrievable intraradicular post.
   - Continuous postoperative discomfort
   - Recurring exacerbations of non surgical endodontic treatment.
3. Calcific metamorphosis of the pulp space.
4. Horizontal fracture at the root tip with associated periapical disease.
5. Procedural errors:
   - Instrument separation
   - Non-negotiable ledging
   - Root perforation
   - Severe apical transportations
   - Symptomatic over filling
6. Anatomic Variations
   - Root dilacerations
   - Apical root fenestrations
   - Non negotiable root curvatures.

6. Biopsy
7. Corrective surgery
   a. Root resorptive defects
   b. Root caries
   c. Root resection
   d. Hemi-section
   e. Bi-cuspidization.
8. Replacement surgery
   a. Intentional replantation
   b. Post traumatic replantation.
9. Implant surgery
   a. Endodontic implants
   b. Osseo-integrated implants.
10. Exploratory surgery.

CONTRAINDICATIONS

1. Periodontal health of the tooth: Tooth mobility and periodontal pockets are two main factors affecting the treatment plan.
2. Patients health considerations
   - Leukemia or neutropenia in active state leading to more chances of infection after surgery, and impaired healing
   - Uncontrolled Diabetes Mellitus—Defective leukocyte function, defective wound healing commonly occurs in severe diabetic patients
   - Recent serious cardiac or cancer surgery
   - Very old patients—Old age is usually associated with complications like cardiovascular pulmonary disorders decreased kidney functions, liver functions.
   - Uncontrolled hypertension
   - Uncontrolled bleeding disorders
   - Immuno-compromised patients
   - Recent myocardial infarction or patient taking anticoagulants
   - Patients who have undergone radiation treatment of face because in such cases incidence of osteoradio-necrosis and impaired healing is high
   - Patient in first trimester of pregnancy – It is during this period the fetus is susceptible to insult, injury and environmental influences that may result in postpartum disorders.
3. Patient's mental or psychological status:
   - Patient does not desire surgery
   - Very apprehensive patient
   - Patient unable to handle stress for long complicated procedures.
4. Surgeon's skill and ability—Clinician must be completely honest about their surgical skill and knowledge. Beyond their abilities, case must be referred to endodontist or oral surgeon.

2. If swelling is diffuse or has spread into extraoral musculo-facial tissues or spaces, then go for surgical drainage and prescribe systemic antibiotics to the patient.
3. If there is a localized abscess or diffuse swelling—Allow it to localize and became soft and fluctuant before incision and drainage.
4. To anesthetize the tooth local anesthesia is given, here nerve block is preferred which is supplemented with infiltration.
5. Use of nitrous oxide analgesia is also advocated sometimes to reduce anxiety and lowering pain.
6. Incision to the most dependent part of swelling is given with scalpel blade, No. 11 or 12. Horizontal incision is placed at dependent base of the fluctuant area for effective drainage to occur (Fig. 18.3).

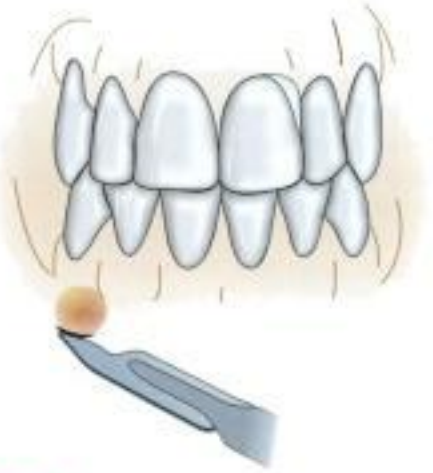


Fig. 18.3: Incision is made at most dependent part of swelling

PERIRADICULAR SURGERY

Before proceeding for periradicular surgery, the clinician must take care of the factors which affect the prognosis of the tooth like taking the complete dental and medical history of the patient, evaluating accessibility to the surgical site, conducting suitable vitality tests and radiographs and assessing the compliance of patient.

ARMAMENTARIUM FOR PERIRADICULAR SURGERY

It consists of all the sterile instruments needed from initiating till the completion of the procedure. It includes:

- Anesthesia like lidocaine with adrenaline and disposable syringes
- Sterilized gauze pieces and cotton pellets
- Bard parker handle, No. 15 and 12 blade
- Mirrors
- A periodontal probe
- Endodontic explorer
- Periosteal elevator
- Periodontal and surgical curettes
- Hemostats
- Scissors
- Cotton forceps
- Flap retractor
- Suturing material
- Surgical and regular length burs.

LOCAL ANESTHESIA AND HEMOSTASIS

1. Local anesthetics with vasopressors are the main choice in surgical endodontic procedures to obtain profound anesthesia and optimal hemostasis (Fig. 18.4).
2. Lidocaine with vasopressor adrenalin is the local anesthesia of choice for surgical procedures.
3. If amide is contraindicated, then ester agent, i.e. procaine, propoxicaine with levonordeffrin is indicated.
4. For nerve blocks 2 percent lidocaine with 1: 100000 or 1: 200000 adrenalin is used. But for obtaining hemostasis adrenalin concentration should be 1: 50000 (Fig. 18.5) to (Fig. 18.8).

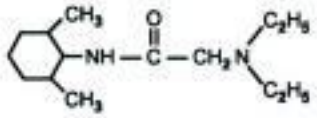


Fig. 18.4: Structure of commonly used local anesthesia

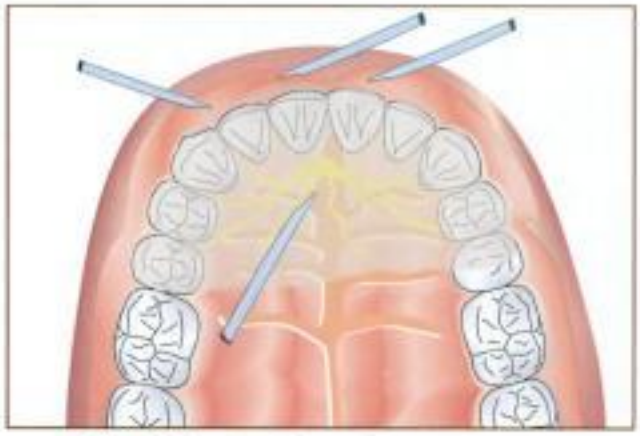


Fig. 18.5: Anesthesia for maxillary anterior teeth

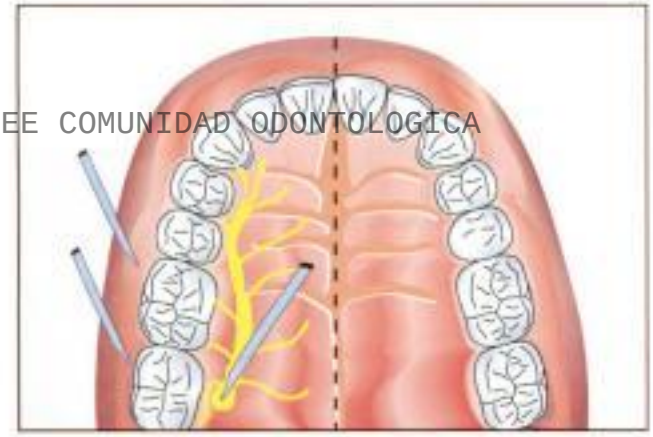


Fig. 18.6: Anesthesia of maxillary posterior area

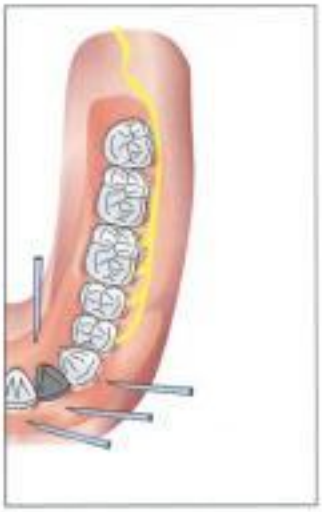


Fig. 18.7: Anesthesizing mandibular anterior region

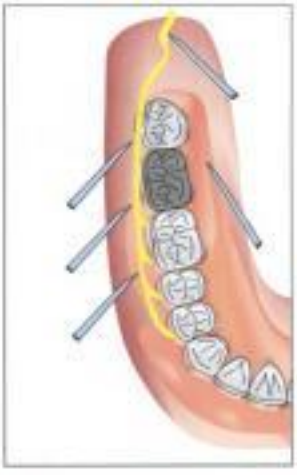


Fig. 18.8: Anesthesizing mandibular posterior area

5. Rate of injection should be 1 ml/minute with maximum safe rate of 2 ml/minute.
6. Submucosal infiltration for hemostasis should be given with 30 gauge needle with the bevel towards bone and penetration just superficial to periosteum at the level of root apices. 0.25-0.5 ml should be injected slowly at a place.
7. Rapid injection produces localized pooling of solution in the injected site resulting in delayed and limited diffusion into the adjacent tissue, thus surface contact with microvasculature is reduces, resulting in reduced hemostasis.

Amount of Local Anesthesia

It depends on the size of surgical site.

- Small site involving only few teeth require 1.8 ml with 1: 50000 adrenalin.
- For extensive surgery involving multiple teeth, one needs 3.6 ml with 1: 50000 adrenalin.
- Delay the incision for 4-5 minutes following injection is advised until good hemostasis is achieved. It is clinically indicated by a blanching of the soft tissues through out the surgical site is achieved.

RECEPTORS AND MECHANISM OF HEMOSTASIS

- There are two types of adrenergic receptors viz. alpha and beta receptors
- ***Gage*** demonstrated that the action of a vasopressor drug on microvasculature depends on
  a. Predominant receptor type
  b. Receptor selectivity of vasopressor drug.
- Alpha receptor predominate in oral mucosa and gingiva
- Beta receptors predominate in skeletal muscles
- Adrenalin receptor selectivity approximately equal for both alpha and beta
- Stimulation of alpha receptors results in vasoconstriction and thus decrease in the blood flow
- Stimulation of beta receptors causes vasodilatation which results in increased blood flow.

Reactive Hyperemia: The Rebound Phenomenon

This phenomenon occurs due to rebound from an alpha to a beta response. In this condition concentration of vaso-constrictor decreases so that it does not cause alpha adrenergic response but after some time blood flow increases more than normal leading to reactive hyperemia. Rebound

join a horizontal intrasulcular incision at obtuse angles (Fig. 18.13).

Fig. 18.13: Trapezoidal flap

Disadvantages

- Wound healing by secondary intention
- Pocketing or clefting of soft tissue
- Compromise in blood supply
- Contraindicated in periradicular surgery.

Envelope Flap

It is formed by a single horizontal intrasulcular incision and is usually recommended for corrective endodontic surgery.

Indications

1. for repair of perforation defects
2. for root resections
3. in cases of hemisections

Advantages

1. Improved wound healing
2. Easiness of wound closure and postsurgical stabilization.

Disadvantages

1. Extremely limited surgical access.
2. Essentially impractical for periradicular surgery. But some use it for palatal surgery.

Potential Disadvantages of Full Mucoperiosteal Flaps

1. Loss of soft tissue attachment level: A key element in preventing loss is ensuring that root attached tissues are not damaged or removed during surgery.
2. Loss of crestal bone height.
3. Postsurgical flap dislodgement.

LIMITED MUCOPERIOSTEAL FLAPS

Semilunar Flap

It was first given by ***Partsch,*** also known as ***Partsch incision.*** It is formed by a single curved incision. This flap is called as semilunar flap because horizontal incision is modified to have a dip towards incisal aspect in centre of the flap, giving resemblance to the half moon (Fig. 18.14). This flap has no primary advantage and it is ***not preferred in modern endodontic*** practice because of its numerous disadvantages.

Fig. 18.14: Semilunar flap

Disadvantages

1. Limited surgical access
2. Difficult wound closure
3. Poor apical orientation
4. Potential for postsurgical soft tissue defects by incising through tissues unsupported by bone.
5. Maximum disruption of blood supply to un-flapped tissues.

Ochsenbein-Luebke Flap

This is also known as ***submarginal retaan flap.*** In 1926, Neumann described a surgical technique for management of periodontal diseases which is very much similar to this flap. This flap is modification of the rectangular flap (Fig. 18.15).

Fig. 18.15: Ochsenbein-Luebke flap

Flap design—in this scalloped horizontal incision is given in the attached gingival which forms two vertical incisions made on each side of surgical site (Fig. 18.16).

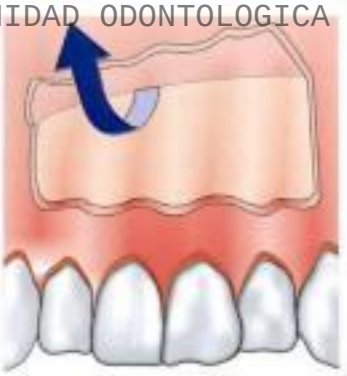


Fig. 18.16: Reflection of flap

The flap is developed by an endodontic and periodontist.

This flap gives advantage of vertical flap along with semilunar flap.

Advantages

1. Marginal and inter-dental gingiva are not involved
2. Unaltered soft tissue attachment level
3. Crestal bone is not exposed
4. Adequate surgical access
5. Good wound healing potential—as compared to semi-lunar flap

Indication

1. In presence of gingivitis or periodontitis associated with fixed prosthesis.
2. Where bony dehiscence is suspected

Disadvantages

1. Disruption of blood supply to un-flapped tissues
2. Flap shrinkage
3. Difficult flap re-approximation and wound closure
4. Untoward postsurgical sequelae
5. Healing with scar formation
6. Limited apical orientation
7. Limited or no use in mandibular surgery.

FLAP DESIGN CONSIDERATION IN PALATAL SURGERY

Two flap designs mainly indicated for palatal surgery are:

1. Triangular
2. Horizontal

In triangular flap, the vertical releasing incision extends from the marginal gingiva mesial to first premolar to a point near the palatal midline and is joined by a horizontal instrasulcular incision which extends distally far as to provide access (Fig. 18.17).

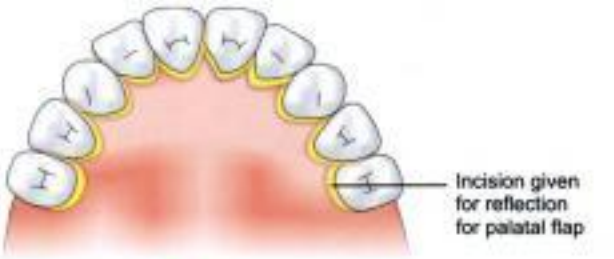


Fig. 18.17: Flap for palatal surgery

Relaxing incisions are given between first premolar and canine to decrease the chances for severance of blood vessels.

The bony surface of posteriors part of palate is pebblier which makes it difficult area during positions elevation. In these area, scalpal can be used to partially dissect the tissues for modified thickness flap. In palatal flap, retraction of flap is difficult, so sling suture be given for flap retraction (i.e., suture the edge of flap and tie it tightly to tooth on opposite side of arch when surgery is completed, suture is cut.

Indications of Palatal Flaps

1. Surgical procedures for palatal roots of maxillary molars and premolars for retrofilling, perforation repair or root amputation.
2. Perforation or resorption repair of palatal surfaces of anterior teeth or in case of palato-gingival groove.

FLAP REFLECTION AND RETRACTION

Without proper attention to these simple procedures there can be severe damage to delicate tissues that play an important role in wound closure.

For Full Mucoperiosteal Flap

Marginal gingiva is very delicate and easily injured. Therefore, it is not appropriate to begin the reflection process in the horizontal incision. For supracrestal root, attached tissues are of greater clinical significance. These are easily damaged by direct reflective forces. If damaged, they lose their viability causing apical epithelial down growth resulting in increased sulcular depth and loss of soft tissue attachment level.

and less resistant to injury. So, any small changes during bone removal can affect bone physiology and viability.

Speed of Cutting

1. ***At 8000 rpm:*** Almost similar tissue response are seen when irrigation is done with or without a coolant or with a mixture of blood and saliva or water.
2. ***At high speed (up to 300000 rpm)*** favorable tissue response are noted when other parameters (coolant, pressure, type of bur) are controlled.

Use of Coolant

- Various studies have supported the use of a liquid coolant (water, saline) to dissipate the heat generated during the cutting osseous tissue, and by keeping the cutting flutes of instrument free of debris there by reducing friction and using cutting efficacy of bur.
- For coolant to be effective, it must be directed on the head of the bur enough to prevent tissue debris from clogging the flutes (Fig. 18.20).

Fig. 18.20: Coolant must be directed on head of bur for efficient action

- Use of coolant with high speed rotary instruments can contaminate a sterile field due to back splash effect during cutting.
- There are certain guidelines which helps in controlling the bacterial population:
  1. Thorough rinsing with mouthwash for one minute before surgery.
  2. Waterlines connected to dental unit should be thoroughly clean with sufficient amount of water or hypochlorite solution.
  3. Handpiece should also be flushed with sufficient amount of normal saline.
  4. Handpiece should always be sterilized before every use.

Studies have shown that using high speed handpiece with 45° angled heat increase visibility and efficiency of cutting instead of using slow speed handpiece. ***Impact air 45° high speed handpiece*** has added advantage that air is exhausted to rear of turbine rather towards bur and surgical site. So, it decreases the splatter and chances of tissue emphysema, pyema and pneumomediastinum, etc.

Bur Types

Shape of bur and flute design plays a very important role.

- Cutting of osseous tissue with a No. 6 or No. 8 round bur produces less inflammation and results in a smoother cut surface and a shorter healing time than when a fissure or diamond bur is used.
- Burs with the ability to cut sharply and cleanly with the largest space between cutting flutes, regardless of the speed of rotation, leave defects that heal in the shortest postsurgical time.
- Cutting bone with a diamond stone is the most inefficient as defects produced by these burs heal at very slow rate.

Pressure and Time during Cutting Procedure

Pressure should be minimum possible and time the bur stays in contact with bone should be as short as possible. This reduced time factor along with light pressure can be achieved by employing the tech of "Brush stroke' cut method.

PRINCIPLES OF SURGICAL ACCESS TO ROOT STRUCTURE

Normally when radiolucent area is present around apex of tooth, tooth root is visible through cortical plate. It is difficult when bone is to be removed to gain access to tooth especially when no periapical radiolucent area is present. Guidelines which should be strictly followed to accurately determine and locate the root apices are:

1. Angulation of crown of tooth to root should be assessed
2. Measurement of entire tooth to root should be assessed.
3. Locate root from coronal to apex where bone covering root is thinner. Once it is located, then covering bone is removed slowly with light brush strokes working in apical direction.
4. Exposing radiographs from both a mesial and distal angulation in addition to straight view.
5. Probing can be done forcibly with instruments endodontic explorer or straight curette in the apical region to know whether a small defect is present or not.

When a small defect is present in the bone, then a small piece of lead sheet, gutta-percha point or a plug of alloy can be placed to know the position of apex.

There are some characteristics (*given by Barnes*) which are helpful in differentiating root surface from surrounding osseous tissue:

1. Root structure is yellowish in color
2. Texture of root is smooth and hard while that of bone is porous and granular
3. Root doesn't bleed when probed
4. Root is surrounded by periodontal ligament. The methylene blue dye can be used to identify the periodontal ligament.

PERIRADICULAR CURETTAGE

It is a ***surgical procedure to remove diseased tissue from the alveolar bone in the apical or lateral region surrounding a pulpless tooth.***

Indications

a. Access to the root structure for additional surgical procedures.
b. For removing the infected tissue from the bone surrounding the root.
c. For removing overextended fillings.
d. For removing necrotic cementum.
e. For removing a long standing persistent lesion especially when a cyst is suspected.
f. To assist in rapid healing and repair of the periradicular tissues.

Surgical Techniques

1. Inject local anesthetic with vasoconstrictor into soft tissue. This will help in controlling hemorrhage during surgery.
2. Design flap depending upon condition of the patient and preference of the clinician.
3. Expose the surgical site.
4. Use the bone curette to remove the pathologic tissue surrounding the root.
5. Insert the curette between the soft tissue and bone, apply the pressure against the bone.
6. After removing the tissue from the bony area, grasp the soft tissue with the help of tissue forceps.
7. Send the pathological tissue for histopathological examination.

ROOT-END RESECTION (APICOECTOMY, APICECTOMY)

It is the ablation of apical portion of the root-end attached soft tissues.

The current indications of root-end resection are:

a. Inability to perform nonsurgical endodontic therapy due to anatomical, pathological and iatrogenic defects in root canal.
b. Persistent infections after conventional endodontic treatment.
c. Need for biopsy.
d. Need to evaluate the resected root surface for any additional canals or fracture.
e. Medical reasons.
f. Lack of time.
g. For removal of iatrogenic errors like ledges, fractured instruments, and perforation which are causing treatment failure.
h. For evaluation of apical seal.
i. Blockage of the root canal due to calcific metamorphosis or radicular restoration.

Guidlines for Bone Removal

1. Adequate anesthesia and hemostasis is necessary.
2. Always sterilize the handpiece before use.
3. Flush the water lines connected to dental unit thoroughly before use.
4. Use sharp and sterile round burs.
5. Amount of pressure should be light while cutting the bone.
6. Handpiece either high speed or low speed should be used with coolant.
7. Cut bone in a shaving or brush stroke method.
8. Visibility of the operative site should be good in order to increase the success of procedure. Position handpieces, bur, suction tip and operating light in right direction to increase the visibility.
9. Avoid deep penetration (3-5 mm) during cutting.

Factors to be considered before root end resection are:

1. Instrumentation
2. Extent of resection
3. Angle of resection.

1. ***Instrumentation:*** High speed handpiece with surgical length fissure bur usually results in satisfactory resection. Use of round bur may result in gouging of root surface where as crosscut fissure burs can lead to uneven and rough surface.

In a study by ***Nedderman et al*** it was found that use of round burs produce ditching of the root surface where as crosscut fissure burs produce the roughest root surface. Use of low speed tissue shaved to produce the smoothest root surface.

Recently studies have shown the use of Er:YAG laser and Ho:YAG laser for root end resection but among these Er:YAG laser is better as it produces clean and smooth root surface. ***Advantages of use of laser in periradicular surgery over the traditional methods include:***

1. Reduction of postoperative pain.
2. Improved hemostasis.
3. Reduction of permeability of root surface.
4. Potential sterilization of the root surface.
5. Reduction of discomfort.

2. ***Extent of resection:***

Historically it was thought that since root-end is surrounded by granulation tissue, failure to remove all foci of infection should result in persistent disease process so it was advised to resect the root surface to the level of healthy bone.

But study by Andreasen and Rud showed no correlation between presence of microorganisms in dentinal tubules and degree of the periapical inflammation.

Factors to be considered while performing root-end resection are:

1. Access and visibility of surgical site.
2. Anatomy of the root, i.e. its shape, length, etc.
3. Anatomy of the resected root surface to see number of canals.
4. Presence and location of iatrogenic errors.
5. Presence of any periodontal defect.
6. Presence of any root fracture.
7. Need to place root-end filling into sound tooth structure.

According to Cohen et al the length of root tip for resection depends upon the frequency of lateral canals and apical ramifications at the root-end. They found that when 3 mm of apex is resected, the lateral canals are reduced by 93 percent and apical ramifications decreased by 98 percent (Fig. 18.21). Whereas a root resection of 3 mm at a 0 degree bevel angle eliminates most of the anatomic features that are possible cause of failure (Fig. 18.22).

Angle of Root-End Resection

Earlier it was thought that root-end resection at 30-45° from long axis of root facing buccally or facially provides:

- Improved visibility of the resected root-end.
- Improved accessibility

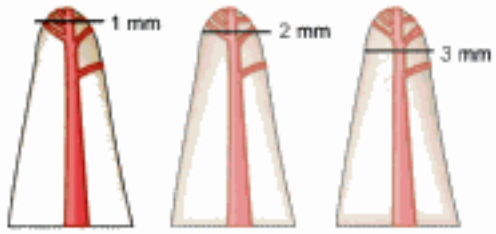


Fig. 18.21: Frequency of ramifications at different levels of root canal

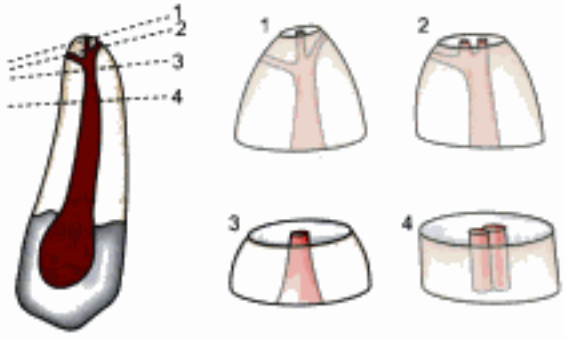


Fig. 18.22: Frequency of canals found at different levels of root canals

Recently, several authors presented evidence that beveling of root-end results in opening of dentinal tubules on the resected root surface that may communicate with the root canal space and result in apical leakage, even when a retrofilling has been placed (Fig. 18.23). Nowadays a ***bevel of 0°-10°*** is recommended with ressection at the level of 3 mm.

Fig. 18.23: Bevelling of root end results in more exposure of dentinal tubules and thus leakage

Steps of Root-End Preparation by Ultrasonic Instruments

1. First of all examination is done using magnification and staining.
2. Thereafter cavity design is planned and outlined by sharp point of a CT-S ultrasonic tip, without irrigation.
3. Prepared cavity design is deepened with appropriately sized and angled ultrasonic tip with irrigation (Fig. 18.26).
4. At completion, cavity is thoroughly irrigated with sterile saline, dried and finally examined under magnification.

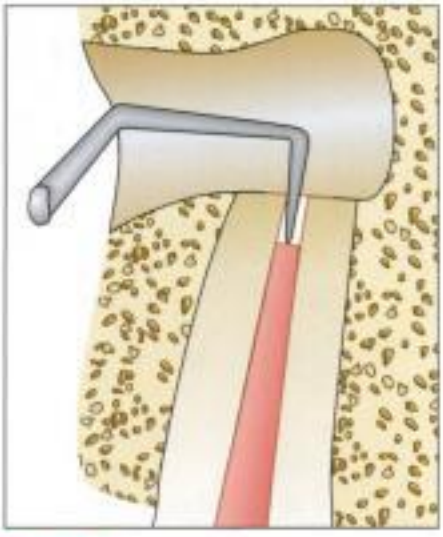


Fig. 18.26: Retrograde cavity preparation using ultrasonic handpiece

RETROGRADE FILLING

The main aim of the endodontic therapy whether nonsurgical or surgical is three dimensional obturation of the root canal system. Therefore after the apical surgery, placement of a root-end filling material is an equally important step. Root canal filling material is placed in the prepared root-end in a dry field. To place a material in the retropreparation, it is mixed in the desired consistency, carried on the carver (hollenback) and placed carefully into the retropreparation (Fig. 18.27) and compacted with the help of burnisher. After the material is set, excess of it is removed with carver or periodontal curette (Fig. 18.28). Finally the root-end filling is finished with carbide finishing bur and a radiograph is exposed to confirm the correct placement of the filling.

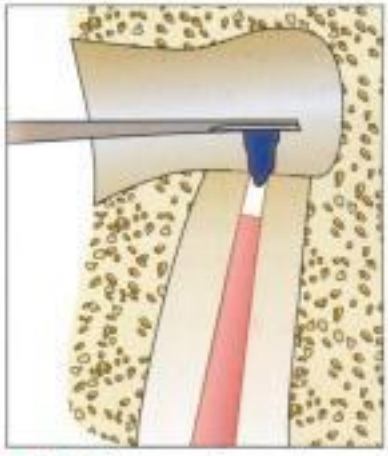


Fig. 18.27: Placement of restorative material

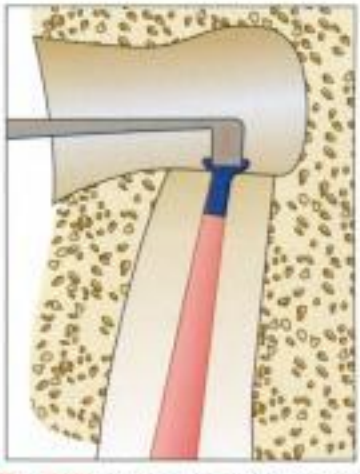


Fig. 18.28: Removal of excess material

ROOT-END FILLING MATERIALS

Ideal properties of a root-end filling material are that it:

1. Should be well tolerated by periapical tissues
2. Should adhere to tooth surface.
3. Should be dimensionally stable
4. Should be resistant to dissolution
5. Should promote cementogenesis
6. Should be bactericidal or bacteriostatic
7. Should be non corrosive
8. Should be electrochemically inactive
9. Should not stain tooth or periradicular tissue
10. Should be readily available and easy to handle
11. Should allow adequate working time, then set quickly
12. Should be radioopaque.

Commonly used root-end filling materials are:

1. Amalgam

2. Gutta-percha
3. Gold foil
4. Titanium screws
5. Glass ionomers
6. Zinc oxide eugenol
7. Cavit
8. Composite resins
9. Polycarboxylate cement
10. Poly HEMA
11. Bony cements
12. Super EBA
13. Mineral Trioxide Aggregate

Amalgam

It is one of the most popular and widely used retrograde filling material since last century.

Advantages

1. Easy to manipulate
2. Readily available
3. Well tolerated by soft tissues
4. Radiopaque
5. Initially provides tight apical seal

Disadvantages

1. Slow setting
2. Dimensionally unstable
3. It shows leakage
4. Stains overlying soft tissues, resulting in formation of tattoo.
5. More cytotoxic than IRM, super EBA or MTA.

Zinc Oxide Eugenol Cements

1. Unmodified ZOE cements are weak and have a long setting time.
2. They tend to be absorbed overtime because of high water solubility.
3. On contact with moisture this releases free eugenol which is responsible for most of the effects caused by zinc oxide eugenol cements.

Effects of free Eugenol

1. Competitively inhibit prostaglandin synthetase by preventing biosynthesis of cyclo-oxygenase.
2. Inhibits sensory nerve activity.
3. Inhibits mitochondrial respiration.
4. Kills a range of natural oral microorganisms.
5. Can act as allergen.

Intermediate Restorative Material (IRM)

1. IRM is a ZOE cement reinforced by addition of 20 percent polymethacrylate by weight to zinc oxide powder.
2. This reinforcement eliminated the problem of absorbability.
3. Milder reaction than unmodified ZOE cements.
4. Mild to zero inflammatory effect after 30 days.
5. Have statistically significant higher success rate compared to amalgam.

Super EBA

It is a ZOE cement modified with Ethoxy Benzoic Acid (EBA) to alter the setting time and increase the strength of mixture.

Powder contains:

- 60 percent zinc oxide
- 34 percent silicone dioxide
- 6l percent natural resin.

Liquid contains:

- EBA—62.5 percent
- Eugenol—37.5 percent

Advantages

1. Neutral pH
2. Low solubility
3. Radiopaque
4. Strongest and least soluble of all ZOE formulations
5. Yield high compressive and tensional strength
6. Significantly less leakage than amalgam
7. Non resorbable
8. Good adaptation to canal walls compared with amalgam

Disadvantages

1. Difficult to manipulate because setting time is short and greatly affected by humidity.
2. Tends to adhere to all surfaces—Difficult to place and comfort.

Mineral Trioxide Aggregate (MTA)

1. MTA is composed of Tri-calcium silicate, Tri-calcium aluminate; Tri-calcium oxide and Silicate oxide.
2. Bismuth oxide is added to the mixture for radiopacity.
3. pH-12. 5 (when set).

The Swage

The suture attachment and creates a single, continuous unit of suture and needle. These type of needles are called atraumatic needles while traumatic needles are needles with holes or eyes which are supplied to the hospital from their suture head. The suture must be threaded on site as is done when sewing at home.

In atraumatic needle with sutures, an eyeless needle attached to a specific length of suture thread. The suture manufacturer swages the suture thread to the eyeless atraumatic needle at the factory. It has certain advantages over traumatic needles such as (i) The doctor assistant does not have to spend time threading the suture on needle. (ii) It is less traumatic.

Several shapes of surgical needles

- Straight
- Half curved
- ¼ circle
- 3/8 circle
- ½ circle
- 5/8 circle

Needles can also be classified according to point geometry (Fig. 18.30)

- Taper (round)—needle body is sound and tapers smoothly to a point, e.g. used in subcutaneous layers periosteum
- Cutting—needle body is triangular and has sharpened cutting edge on the inside
- Side cutting—flap on top and bottom with a cutting edge along the front to one side e.g. used in ophthalmic procedures
- Reverse cutting—cutting edge on the outside, e.g. skin, oral mucosa and tendon sheaths.

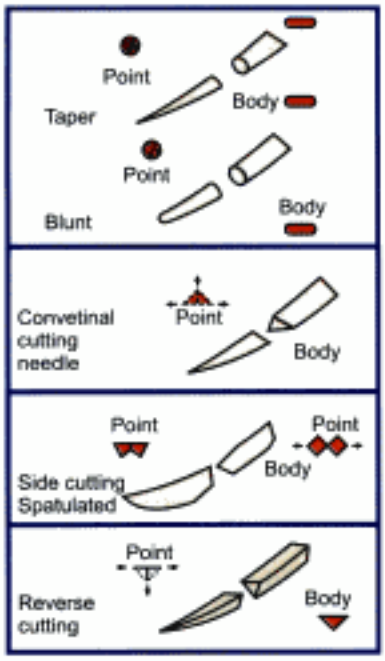


Fig. 18.30: Different types of needle

Principles of Suturing

1. The needle should enter the mucosal skin perpendicular to the surface of tissue.
2. The needle should always pass from free tissue to fixed tissue.
3. The needle should always be inserted at an equal depth and distance from incision line on both sides.
4. The suture knot should never lie on the incision line.
5. The suture should not be too tight. Sutures are given to approximate the tissues, not to blanch the tissues. If sutures are too tight, there will be local ischemia underneath the suture tracks.
6. The needle should always from thinner to thicker tissues.
7. Tissues should not be closed under tension.
8. The needle should be held in needle holders two thirds of way from needle tip to swage.
9. The needle should take smooth semicircular course to exit at 90 degrees to the wound edge.
10. Sutures should be spaced evenly.
11. After tying, knot should be left to one side.

SUTURING TECHNIQUES

Interrupted Suture (Fig. 18.31)

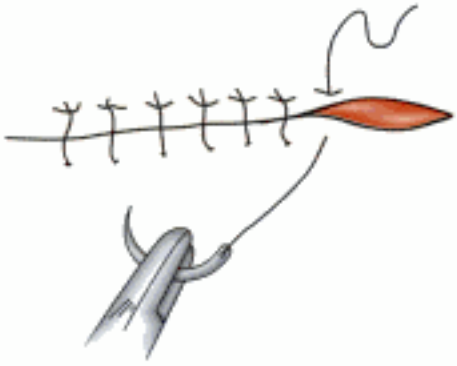


Fig. 18.31: Interrupted suture

Contd...

Suture	*Classification*	*Origin*	*Absorption*	*Disadvantages properties*	*Advantages and*	*Uses*
					• Stronger than silk suture	
5. Polypropylene (prolene)	Synthetic monofilament	• Stereoisomer of linear propylene isomer	Not available	• Difficult in handling	• Biologically inert • Minimal tissue reaction • Resistance to bacterial contamination • Retention of strength (upto 2 years)	• Useful in contaminated and infected wound • Plastic and oral surgery

3. Application of hot moist towel is recommended after 24 hours (↑ ing the temperature causes ↑se in blood flow in that area which enhances inflammatory and healing process).
2. Postoperative bleeding: Slight oozing of blood is usually seen after surgery for several hours. This slight oozing of the blood is normal, but significant bleeding is uncommon and may require attention.

   Postoperative bleeding can be reduced by compression of the surgical flap both before and after suturing.

Management

1. First and foremost step in managing bleeding is apply firm pressure over the area for 10-20 minutes. This can be applied with either moistened cotton gauge or a tea bag or ice pieces placed in cotton gauge.
2. Some prefer pressure to the area along with local anesthetic containing epinephrine (1:50,000 or 1:1,00000).
3. If bleeding still continues, then sutures should be removed and then search for blood vessels causing bleeding. Cauterization should be done either by using thermal (heating an instrument) or electrical method (electrocautery if available). In these cases, local hemostatic agents can also be tried.
   - If bleeding is still unmanageable, then hospitalization of patient is necessary. Review the medical status of the patient.

Extraoral Ecchymosis (Extraoral discoloration)

Discoloration/ecchymosis usually results when blood has leaked into the surrounding tissues. This condition is self limiting in nature and lasts up to 2 weeks and does not affect the prognosis.

Management

1. Application of moist hot for 2 weeks is helpful as heat promotes fluid exchange and also speeds up resorption of discoloring agents from tissues.

Pain

Postoperative pain usually maximum on the day of surgery and it decreases thereafter.

Management

1. Pain can be managed by prescribing NSAIDs
2. If severe pain is present, opoid analgesics may be combined with NSAIDs
3. Long acting anesthetics like bupivacaine has also been advocated.

Infection

Postoperative infection usually occurs due to inadequate aseptic technique and improper soft tissue handling, approximation and stabilization. The symptoms usually appear 36-48 hours after surgery. Suppuration, elevated temperature and lymphadenopathy is seen in some cases.

Management

1. Systemic antibiotics should be prescribed antibiotic of choice in these cases is pencillin. If person is allergic to pencillin, then clindamycin should be given (initial dose-600 mg, maintenance 150-300 mg).

Miscellaneous

i. Maxillary sinusitis
ii. Paresthesia

19 Endodontic Periodontal Relationship

- Introduction
- Pathways of Communication Between Pulp and Periodontium
- Effect of Pulpal Disease on Periodontium
- Effect of Periodontal Disease on Pulp
- Classification of Endo-perio Lesions
- Diagnosis of Endo-perio Lesions
- Primary Endodontic Lesions
- Primary Endodontic Lesions with Secondary Periodontal Involvement
- Primary Periodontal Lesions
- Primary Periodontal Lesions with Secondary Endodontic Involvement
- True Combined Endo-perio Lesions

INTRODUCTION

The health of periodontium is important for proper function of the tooth. The periodontium consists of gingiva, cementum, periodontal ligament and alveolar bone. This is the fact that the periodontium is anatomically interrelated with dental pulp by virtue of apical foramina and lateral canals which create pathways for exchange of noxious agents between these two tissues (Fig. 19.1). When the pulp becomes infected, the disease can progress beyond the apical foramen and affects the PDL. The inflammatory process results in formation of inflammatory tissue, which if not treated can result in resorption of alveolar bone, cementum and dentin.

Besides going through apical foramen, pulpal disease can progress through lateral canals, commonly present in the apical third and the furcation areas.

Not only the interaction between periodontium and pulp, produce or aggravate the existing lesion, they also present challenges in deciding the direct cause of an inflammatory condition. So a correct diagnosis should be made after careful history taking and clinical examination.

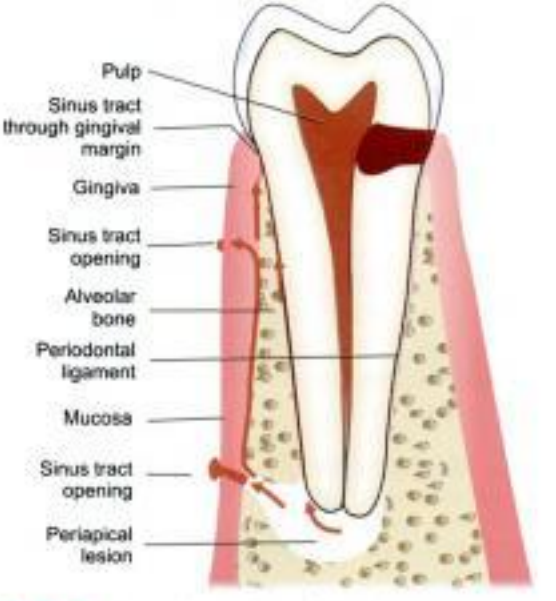


Fig. 19.1: Pathway for exchange of noxious agents between endodontic and periodontal tissue

Pathways of Communication Between Pulp and Periodontium

Physiologic Pathways

- Dentinal tubules
- Lateral and accessory canals
- Apical foramen
- Palatogingival groove

Non-physiologic Pathways

- Perforations
- Vertical root fracture

PHYSIOLOGIC PATHWAYS

Dentinal Tubules

In roots, the dentinal tubules extend from dentin from cementum-dentin junction to the pulp predentin junction. Dentin-tubules follow a straight course in root dentin instead of S-shaped contours present in the crown portion (Fig. 19.2). Usually they are patent but their potency may decrease with age, sclerosis or calcifications. Cementum acts as protective barrier to the dentin but because of periodontal disease, periodontal therapy (root planning) or other irritants, if cementum is destroyed a direct communication between dentinal tubules and the oral cavity may occur.

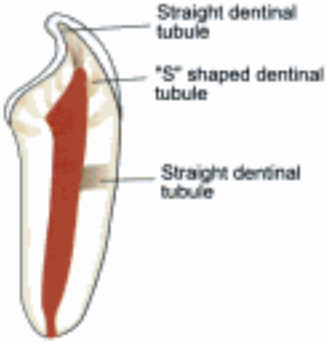


Fig. 19.2: Pattern of dentinal tubules

Dentinal Tubules

- Traverse from pulpodentinal junction to cementodentinal or dentinoenamel junction.
- In radicular area-straight course.
- In coronal portion-S-shaped course.
- Greater density at pulpodentinal junction than CDJ.
- Congenital absence of cementum, cemental exposure by periodontal disease, caries, root surface instrumentation–exposes dentinal tubules.
- Communication between pulp and periodontium.

Lateral or Accessory Canals

Lateral or accessory canals may exist anywhere on the root surface, though majority of them are found in apical third and furcation area of the root. It has been seen that up to 40 percent of teeth have lateral or the accessory canals (Fig. 19.3). As the periodontal disease progresses down the root surface, more of the accessory and lateral canals get exposed to oral cavity. Clinically, positive identification of the presence of lateral canal can be made when an isolated lateral lesion associated with a nonvital tooth is seen radiographically or during obturation, when some of the filling material got extruded into the lateral canal.

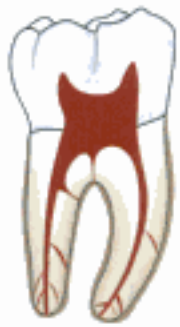


Fig. 19.3: Lateral and accessory canals can exist anywhere on the root surface

Lateral and Accessory Canals

- Most common in apical third of posterior teeth.
- Difficult to identify on radiographs.
- Identified by isolated defects on the lateral surface of roots or by post-obturation radiographs showing sealer puffs.

Condition when pulp is necrotic and lateral canal is exposed, the periodontal reattachment to root surface can be inhibited if periodontal therapy is done before endodontic treatment. Thus in cases where pulp is nonvital and periodontal prognosis is good, then endodontic treatment should precede the periodontal therapy.

Apical Foramen

One of the major pathways of communication between the dental pulp and the periodontium is through apical foramen (Fig. 19.4).

Apical Foramen

- Major pathway of communication
- Inflammatory factors exit through apical foramen and irritate periodontium.

Palato Gingival Grooves

These are developmental anomalies of maxillary incisor teeth. These grooves begin in the central fossa, cross the cingulum and extent apically at varying distances.

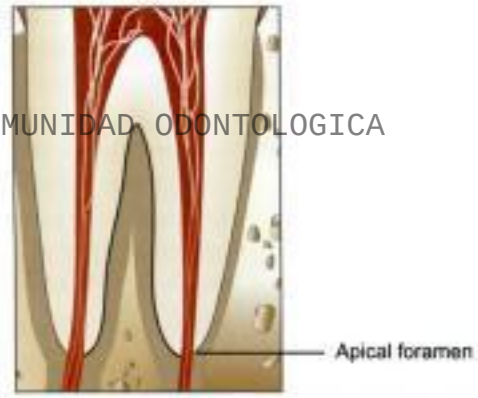


Fig. 19.4: Apical foramen communicating endodontic and periodontal system

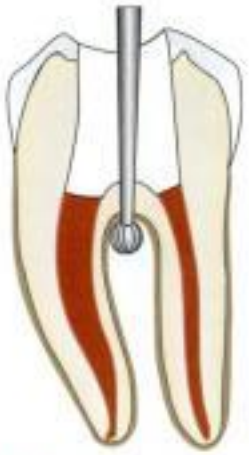


Fig. 19.5: Furcation of root creates communication between root canal system and periodontium

NON-PHYSIOLOGICAL PATHWAYS

Perforation of the Root

Perforation creates an artificial communication between the root canal system and the periodontium (Fig. 19.5). Closer the perforation to the gingival sulcus, greater is the chances of apical migration of the gingival epithelium in initiating a periodontal lesion.

Vertical Root Fracture

Vertical root fracture can form a communication between root canal system and the periodontium. The fracture site provides entry for bacteria and their toxic products from root canal system to the surrounding periodontium.

Diagnosis of the combined endodontic and periodontal lesions is often multifaceted and exasperating. A growing periapical lesion with secondary involvement of the periodontal tissue may have the similar radiographic appearance as a chronic periodontal lesion which has reached to the apex. Images of bone resorption, including apex furcation and marginal areas may confuse the diagnosis.

Pulp tests may be done sometimes to rule out an endodontic etiology, but they are not always reliable. Partial pulpal necrosis in multirooted teeth can respond positively to the vitality tests indicating pulp vitality despite of a combined lesion.

An endodontically treated tooth or a nonvital tooth associated with periodontal lesion can pose greater diagnostic problem as in such cases pulpal inflammation is frequently associated with inflammation of periodontal tissue.

Thus, a careful history taking, visual examination, diagnostic tests involving both pulpal and periodontal testing and radiographic examination are needed to diagnose such lesions.

Tooth with combined endodontic-periodontal lesions must fulfill the following criteria:

- Tooth involved must be pulpless.
- There must be destruction of the periodontal attachment which can be diagnosed by probing from gingival sulcus to either apex of the tooth or to the level of involved lateral canal.
- Both endodontic therapy and periodontal therapy are required to resolve the lesion completely.

IMPACT OF PULPAL DISEASES ON THE PERIODONTIUM

Pulpal infection may cause a tissue destructive process which may progress from apical region to the gingival margin, termed as "retrograde periodontitis" (Fig. 19.6).

Caries restorative procedures and/or traumatic injuries may cause inflammatory changes in the pulp, though it is still vital. It has been seen that even in presence of significant inflammation, a vital pulp does not affect the periodontium. But necrosis of pulp is frequently seen to be associated with the involvement of the periodontal tissue. Commonly, the areas of bone resorption are seen at apex, furcation areas and on the lateral surface of the root. These lesions can be in form of cyst, granuloma or abscess.

The inflammatory process in periodontium associated with necrotic pulps is similar to periodontal disease, that is an infectious etiology. The only difference lies is the source of infection.

Inflammatory lesions may also form from a root canal infection through lateral and accessory canals present on the lateral surface of root and furcation areas. These lesions

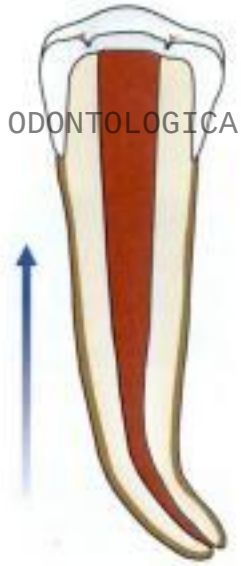
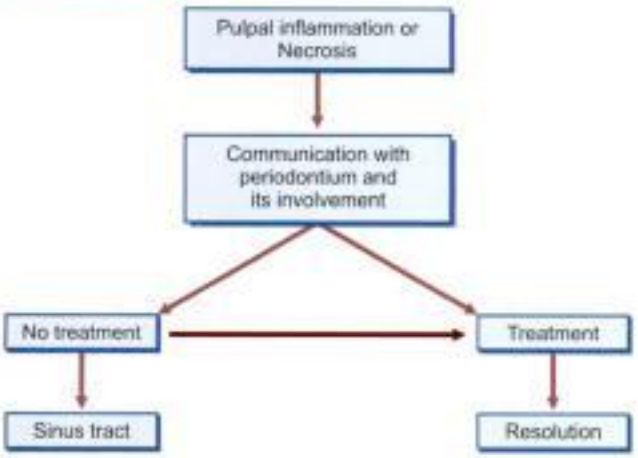


Fig. 19.6: Retrograde periodontitis

are induced and maintained by the bacterial products which reach the periodontium through lateral canals.

EFFECT OF PULPAL DISEASES ON PERIODONTIUM



IMPACT OF PERIODONTAL DISEASE ON PULPAL TISSUE

The pathogenic bacteria and inflammatory products of periodontal diseases may enter into the root canal system via accessory canals, lateral canals, apical foramen, dentinal tubules or iatrogenic errors. Though irreversible pulpitis or pulpal necrosis is not the common occurrence, but inflammatory and pathologic changes may occur once the periodontal disease reaches the terminal stage, i.e. when plaque involves the apical foramina. As periodontal disease extends from gingival sulcus towards apex, the auxillary canals get affected which results in pulpal inflammation. It becomes more serious if these canals get exposed to oral cavity because of loss of periodontal tissues by extensive pocket depth.

Periodontal therapy also affects the pulp. Periodontal instruments like ultrasonic scalers, vibrators, curettes may cause harm to the pulp specially if used when the remaining dentin thickness is <2 mm. Also the chemicals and medicaments used during periodontal therapy may cause pulpal damage.

Effect of Periodontal Disease on Pulp

- Periodontal disease may involve pulp through apical foramen, lateral and accessory canals, dentinal tubules or iatrogenic errors.
- Periodontal procedures; scaling, root planning, surgically expose dentinal tubules due to removal of cementum and dentin.
- Chemical irritants and medicaments irritate pulpal tissue.
- Effects depend on remaining dentin thickness.

ETIOLOGY OF ENDODONTIC-PERIODONTAL PROBLEMS

It has been proved since ages that primary etiologic agent in periodontitis is bacterial plaque. Besides this primary factor, there are secondary factors which contribute to the disease process either by increasing the chances of plaque accumulation or by altering the response of host to the plaque.

It is also seen that irreversible pulpal disease occurs when trauma inflicted on pulpal tissue exceeds its reparative capacity. Such insult can occur through bacteria, chemical, mechanical, thermal or electrical trauma to the pulp.

Pulpal diseases can result in the periodontal problems and vice versa. It is the length of time that the etiological factor persists in the susceptible environment which is directly related to the probability of occurrence of combined lesions.

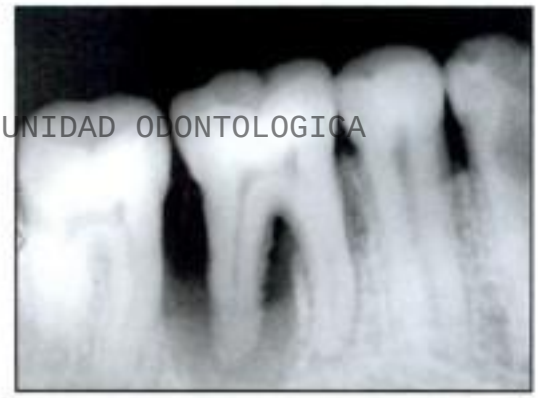


Fig. 19.10: Radiograph showing endo-perio lesion with bone resorption in molar in right mandibular molar

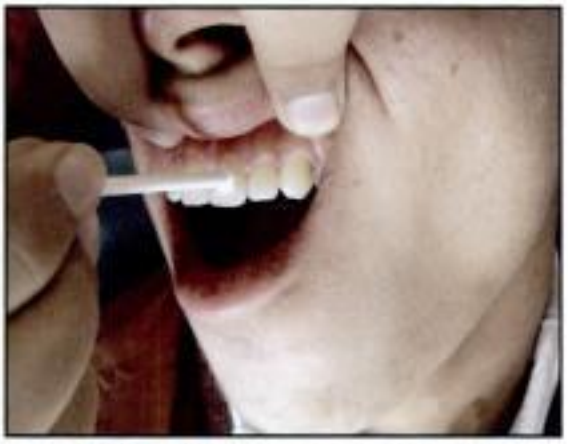


Fig. 19.11: Checking vitality of pulp using hot gutta-percha stick

used pulp vitality tests are cold test, electric test, blood flow test and cavity test (Fig. 19.11). Recent advances in the diagnosis include the use of Laser Doppler Flowmetery, pulp oximetery and magnetic resonance imaging.

iv. ***Tracking sinus or fistula:*** Tracking the fistula may aid the clinician to differentiate the source.

v. ***Pocket probing:*** Pocket probing helps in knowing location and extent of the pockets, depth of pocket and any furcation involvement if any (Fig. 19.12).

vi. ***Microbiological examination:*** Occasionally the microbiological analysis can provide an important information regarding the main source of the problem.

Treatment and Prognosis

Treatment planning and prognosis depend mainly on diagnosis of the specific endodontic and/or periodontal disease. In teeth with combined endodontic-periodontal

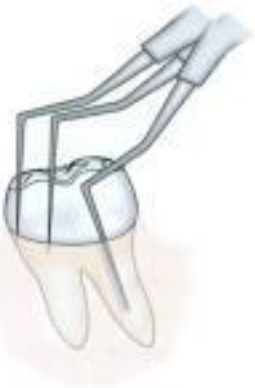


Fig. 19.12: Probing of tooth helps in knowing extent of pockets

lesions, the prognosis depends on extent of destruction caused by the periodontal disease. If lesion is of endodontic origin, an adequate endodontic treatment has good prognosis. Thus in combined disease, prognosis depends on efficacy of periodontal therapy.

Clinical Considerations

Primary Endodontic Lesions (Fig. 19.13)

- Sometimes an acute exacerbation of chronic apical lesion in a nonvital tooth may drain coronally through periodontal ligament into the gingival sulcus, thus mimic clinically the presence of periodontal abscess.
- The lesion presents as an isolated pocket or the swelling on the side of the tooth.

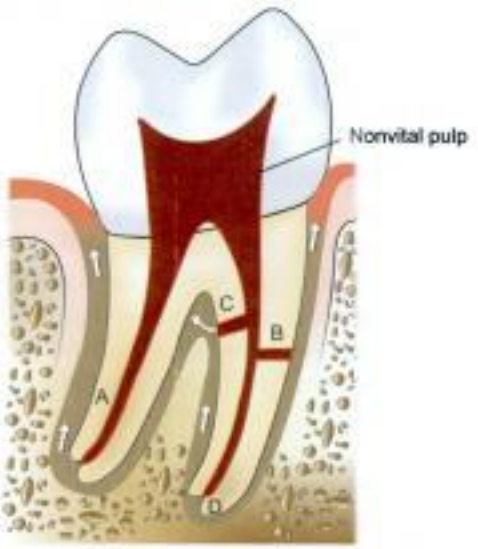


Fig. 19.13: Spread of infection can occur (A) from apical foramen to gingival sulcus via periodontium (B) from lateral canal to pocket (C) from lateral canal to furcation (D) from apex to furcation

- Patient is usually asymptomatic, but history of acute exacerbation may be present.
- Since tooth is associated with necrotic pulp, pulp does not show responses to vitality tests.
- Sinus tract may be seen from apical foramen, lateral canals or the furcation area.
- Probing shows true pockets. Pocket is associated with minimal plaque or calculus. The significant sign of this lesion is that patient does not have periodontal disease in other areas of oral cavity.
- The prognosis after endodontic therapy is excellent. In fact, periodontal therapy is performed without considering pulpal problem, prognosis becomes poor.

Diagnosis

- Necrotic pulp draining through periodontal ligament into gingival sulcus.
- Isolated pocket on side of tooth.
- Pocket associated with minimal amount of plaque or calculus.
- Patient asymptomatic with history of acute exacerbations.

Treatment

- Root canal therapy
- Good prognosis

Primary Endodontic Lesion with Secondary Periodontal Involvement (Fig. 19.14)

- These lesions appear if primary endodontic lesion is not treated. The endodontic disease will continue, resulting in destruction of periapical alveolar bone, progression into the interradicular area, and finally causing break down of surrounding hard and soft tissues. As the drainage persists through periodontal ligament space, accumulation of irritants result in periodontal disease and further migration of attachment.
- Isolated deep pockets are seen though there may be the presence of generalized periodontal disease.
- In such cases, endodontic treatment will heal part of the lesion but complete repair will require periodontal therapy.
- Treatment involves endodontic treatment or retreatment in cases in which poor quality obturation or root fracture etc. may give rise to periodontal problems.
- In case, vertical root fracture is causing the endo-perio lesions, tooth is extracted, otherwise the prognosis is good.

Fig. 19.14: Primary endodontic lesion with secondary periodontal involvement

Primary Endodontic Lesion with Secondary Periodontal Involvement

Diagnosis

- Continuous irritation of periodontium from necrotic pulp or from failed root canal treatment.
- Isolated deep pockets.
- Periodontal breakdown in the pocket.

Treatment

- Root canal treatment to remove irritants from pulp space.
- Re-treatment of failed root canal therapy.
- Concomitant periodontal therapy
- Extraction of teeth with vertical root fracture if prognosis is poor.
- Good prognosis.

Primary Periodontal Lesions (Fig. 19.15)

- Primarily these lesions are produced by the periodontal disease. In these lesions periodontal breakdown slowly advances down to the root surface until the apex is reached. Pulp may be normal in most of the cases but as the disease progress, pulp may become affected.
- Periodontal probing may show presence of plaque and calculus within the periodontal pocket.
- Due to attachment loss, tooth may become mobile.
- Usually generalized periodontal involvement is present.
- Treatment involves scaling, root planing, oral prophylaxis and oral hygiene instructions.
- Periodontal surgeries, root amputations may be required in advanced cases.
- Prognosis becomes poor as the disease advances.

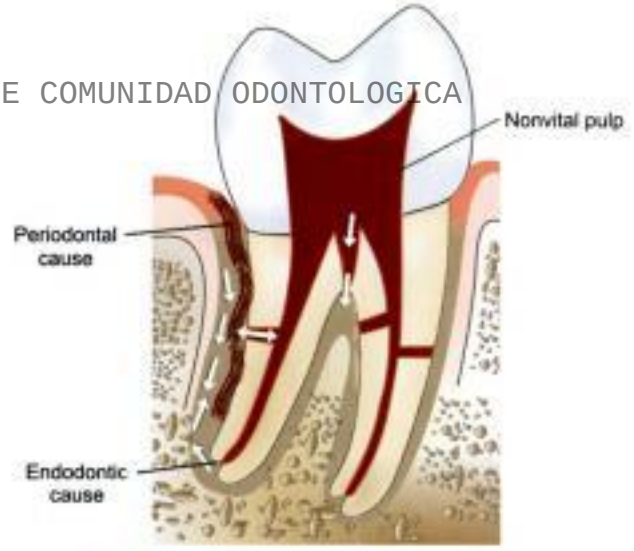


Fig. 19.17: True combined endo-perio lesion

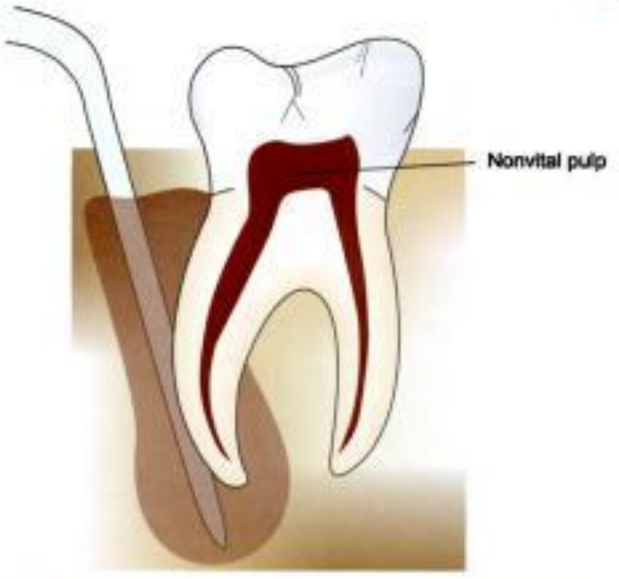


Fig. 19.18: In true combined endodontic periodontal lesion at the base of periodontal lesion the probe abruptly drops down the root and extend to tooth apex

- Though both periodontal and endodontic lesions are present concomitantly but they can not be designated as true combined endo-perio lesions because there is no demonstrable communication between these two lesions.
- Root canal treatment is needed for treating pulp space infection.
- Periodontal therapy is required for periodontal problem. Prognosis of the tooth depends on the periodontal prognosis.

True Combined Endo-Perio Lesions (Fig. 19.17)

- The true combined lesions are produced when one of these lesion (pulpal or periodontal) which are present in and around the same tooth coalesce and become clinically indistinguishable. These are difficult to diagnose and treat.
- Periodontal probing reveals conical periodontal type of probing, and at base of the periodontal lesion the probe abruptly drops farther down the root surface and may extend the tooth apex (Fig. 19.18).
- Radiograph may show bone loss form crestal bone extending down the lateral surface of root.
- Prognosis of the tooth depends upon prognosis of the periodontal disease. First see whether periodontal condition is treatable, if promising, and then go for endodontic therapy. The endodontic therapy is completed before initiation of the definitive periodontal therapy.
- After completion of endodontic therapy, periodontal therapy is started which may include scaling, root planning, surgery along with oral hygiene instructions.

Differential diagnosis between pulpal and periodontal disease

Features	*Periodontal*	*Pulpal*
Etiology	Periodontal infection	Pulpal infection
Plaque and calculus	Commonly seen	No relation
Tooth vitality	Tooth is vital	Non vital
Restorations	No Relation	Usually show deep and extensive restoration
Periodontal destruction	Usually present, and generalized	If present single, isolated
Gingiva and epithelial attachment	Recession of gingival with apical migration of attachment	Normal
Pattern of disease	Generalized	Localized
Radiolucency	Usually not related	Perapical radiolucency
Inflammatory and granulation tissue	Usually present on coronal part of tooth	Commonly seen on apical part of tooth
Treatment	Periodontal therapy	Root canal therapy

20 Restoration of Endodontically Treated Teeth

- Introduction
- Effect of Endodontic Treatment on Teeth
- Planning Postendodontic Restoration Procedure
- Posts
  - Custom Made Posts
  - Prefabricated Posts
- Classification and Description of Posts
- Factors to be Considered while Planning Posts and Core

INTRODUCTION

Endodontic treatment is an attempt at preserving a tooth with damaged pulp in function. Success of endodontic therapy depends upon combination of a three dimension fluid tight obturation along with adequate post endodontic restoration to make the pulpless teeth to function as an integral part of the dental arch (Fig. 20.1). The post endodontic restorations are equally important as the instrumentation and obturation of the root canal.

Before starting the endodontic therapy, the clinician should evaluate the tooth completely. Various studies have shown the more endodontic treatments have shown failures because of restorative difficulties after endodontic therapy than because of endodontic treatment itself.

Fig. 20.1: Restoration of endodontically treated tooth

EFFECTS OF ENDODONTIC TREATMENT ON THE TOOTH

Structural Changes

In general the crowns of endodontically treated teeth could be weakened by caries, trauma and/or during access cavity preparation (Fig. 20.2). This weakened crown portion becomes unable to perform its normal function even after successful endodontic therapy. This weakened tooth structure is further prone to fracture. The compromised structural integrity makes the tooth insufficient to perform its function because of loss of occlusion with its antagonist and adjacent teeth. Also the excessive removal of radicular dentin during canal preparation compromises the root (Fig. 20.3).

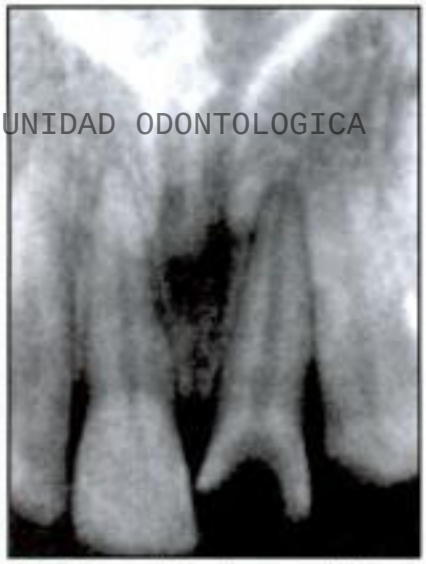


Fig. 20.2: Radiograph showing coronal destruction of tooth because of caries

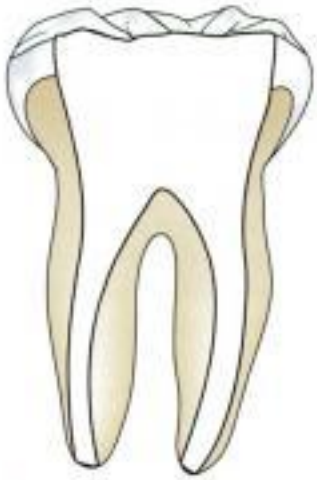


Fig. 20.3: Weakening of roots due to excessive removal of radicular dentin

Effect of Endodontic Treatment on Tooth

- ***Structural changes***
  Tooth weakening caused by :
  - Caries
  - Trauma
  - Access cavity preparation
  - Radicular preparation

  Compromised structural integrity.
- ***Changes in Dentin***
  - A reduced amount of moisture in nonvital teeth

Contd...

Contd...

- ***Aesthetic Considerations***
  - Loss of tooth structure
  - Change in appearance because of alteration in biochemical properties of dentin.
  - *Discoloration because of:*
    - Incomplete debridement
    - Accumulation of sealer or debris.

Changes in the Dentin Physical Characteristics

The endodontic treatment has shown to cause irreversible changes and weakening of the tooth. Physical chemistry of the dentinal structure also changes following the endodontic therapy. Radicular dentin possesses less of moisture content than the coronal part because of fewer tubules, more inorganic part and intertubular dentin. Moisture content further gets reduced because of aging, decrease in amount of organic content and increased in inorganic components. There are different opinions on whether endodontically treated teeth become more brittle because of moisture loss or caused by loss of pulpal tissue. Helfer et al proved that endodontically treated dog teeth have 9 percent less moisture than vital teeth. This should be noted that active retaining posts could induce mechanical stress during cementation and functional loading causing root fracture and failure of postendodontic restorations.

Aesthetic Consideration

Biochemical changes in dentin modify the refraction of light through the tooth and thus changes its appearance. The discoloration of tooth can result because of incomplete cleaning and shaping of the root canal system, the accumulation of sealer, debris or filling materials left in the chamber.

PLANNING POSTENDODONTIC RESTORATION PROCEDURE

Various studies of unsuccessful endodontic procedures have shown failures due to inadequate restoration of the teeth. Restorative treatment of tooth depends upon amount of remaining tooth structure, its functional need and need for the tooth as abutment. After caries is removed and access cavity is prepared, the postendodontic restoration can be planned following the complete visualization of the tooth.

The Restoration Plan Depends on:

- Remaining tooth structure
- Functional need
- Position of tooth in the arch
- Morphology of root canal.

As the remaining tooth structure decreases, and the functional need increases, greater restorative control is needed. Teeth with only little remaining tooth structure have increased risk of fracture. So good care is needed for restorative planning.

For Anterior Teeth

Not all endodontically treated teeth require posts.

1. Most teeth with healthy remaining tooth can be restored by direct filling of the access cavity, usually tooth composite resins or glass ionomer cements (Fig. 20.4).
2. For devitalized, discolored anterior teeth, where more than half of the coronal structure is intact, the preferred treatment should be bleaching or/and composite or porcelain laminate veneers rather than the full coverage crowns or post and core (Fig. 20.5).
3. But if there is doubt regarding the adequacy of resistance form of the coronal portion of the tooth for any restoration, then in such cases post and core is indicated (Fig. 20.6).

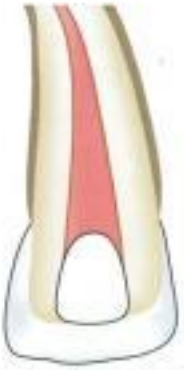


Fig. 20.4: Intact teeth can be restored using composite resins or GIC

Fig. 20.5: When more than half of coronal structure is intact, veneers can do

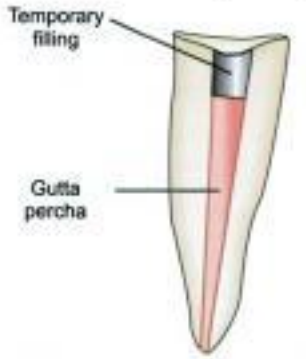


Fig. 20.6: When more coronal structure is lost, post and core is indicated

For Posterior Teeth

Since posterior teeth are subjected to greater loading than anterior teeth, these should be treated differently. Also the morphology of posterior teeth is such that the cusps can be wedged apart, which make them more susceptible to fracture.

1. If there are no proximal fillings, caries or unsupported cusps or strong facets, the access cavity of posterior teeth can be easily restored with amalgam or high strength posterior composites (Fig. 20.7).
2. If there is moderate damage of posterior teeth having at least minimum of one sound cusp, the choice of restoration can be:
   - *Amalgam:* Coronal-radicular core which is finally restored with cast restoration (Fig. 20.8).
   - Pin retained restorations
   - Onlay
   - Prosthetic crown

Fig. 20.7: Small access cavity of posterior teeth can be restored with amalgam or posterior composites

3. In case there is presence of severely damaged clinical crown with no remaining cusps (Fig. 20.9), the root canal is used as a space for intraradicular retention (Fig. 20.10).

Fig. 20.8: Corono-radicular restoration of amalgam

Fig. 20.9: Severely damaged crown

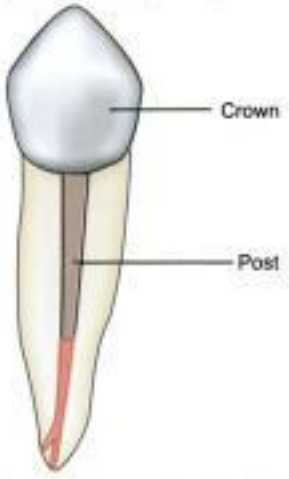


Fig. 20.10: Root can be used for intraradicular retention in case of severely damaged crown

Generally all the endodontically treated teeth should be restored using the crown. A post is indicated in the tooth when it is severely damaged, or it is to serve as an abutment for a removable partial denture. In such cases, the forces which act on teeth are not physiologic, thus a coronal reinforcement is indicated. Post is usually given in palatal canal of maxillary molars and distal canal of mandibular molars.

Features Evaluated before Going for Post and Core

- Restorability of the tooth
- Role of tooth in the month
- Periodontal considerations
- Functional loading

Requirements of a Tooth to Accept a Post and Core

- Optimal apical seal
- Absence of fistula or exudate
- Absence of active inflammation
- No sensitivity to percussion
- Absence of associated periodontal disease
- Sufficient bone support around the root.
- Sound tooth structure coronal to alveolar crest
- Absence of any fracture of root.

CONDITIONS WHERE POST SHOULD NOT BE GIVEN

1. Any sign of endodontic failures are evident, i.e. tooth exhibits
   - Poor apical seal and poor quality obturation (Fig. 20.11)
   - Active inflammation
   - Presence of fistula or sinus
   - Tender on percussion

Fig. 20.11: Endodontic failure due to poor quality obturation

2. If adequate retention of core can be achieved by natural undercuts of crown.
3. If there are horizontal cracks in the coronal portion of the teeth.

4. When tooth is subjected to excursive occlusal stresses such as when there is presence of lateral stresses of bruxism or heavy incisal guidance.

Post

It is relatively rigid restorative material placed in the root of a non vital tooth. It extends coronally to anchor the core material which supports the crown.

Core

Core is the supragingival portion which replaces the missing coronal tooth structure and forms the center of a new restoration. In other words it acts as a miniature crown (Fig. 20.12).

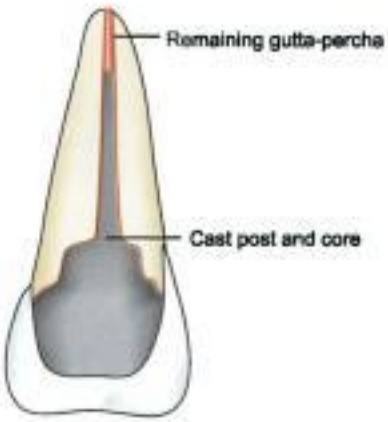


Fig. 20.12: Cast post and core system

Purpose of Use of Post and Core

Post mainly serves two functions:

- Helps in retaining the core
- Helps in favorable distribution of the stresses inside the radicular portion of teeth.

Earlier it was believed that posts strengthen or reinforce the teeth but it has been shown by various studies that posts actually weaken the tooth and increase the risk of root fracture. It has been suggested that endodontically treated teeth are more brittle and may fracture more easily than vital teeth. Subsequently post space preparation or placement of post can further weaken the root and may lead to root fracture. Therefore, a post should be used only when there is insufficient tooth structure remaining to support the final restoration. In other words the main function of post is retention of the core to support the coronal restoration.

Ideal Requirements of a Post

A Post should:

- Provide maximum protection of the root to resist root fractures
- Provide maximum retention of the core and crown
- Be easy to place
- Be less technique sensitive
- Have high strength and fatigue resistance
- Be visible radiographically
- Be biocompatible
- Be easily retrievable when required
- Be aesthetic
- Be easily available and not expensive.

CLASSIFICATION OF POSTS

A. *Posts can be classified as*

I. Prefabricated (Fig. 20.13)
II. Custom made (Fig. 20.14)

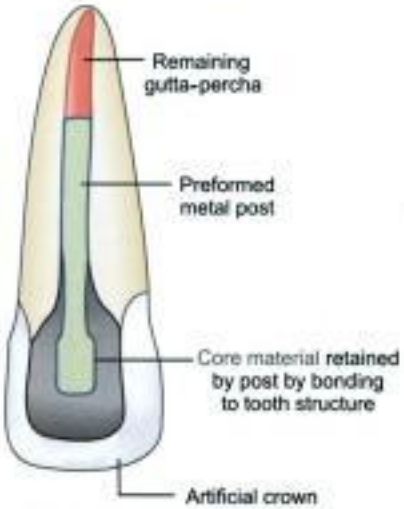


Fig. 20.13: Prefabricated post and core

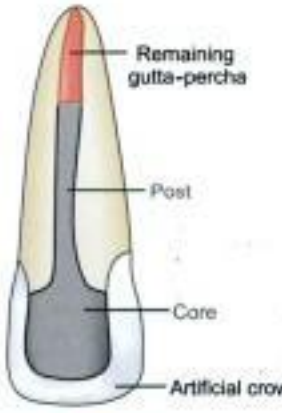


Fig. 20.14: Custom made post and core

I. Prefabricated Post

1. Metal prefabricated posts are made up of
   - Gold alloy
   - High platinum alloys
   - Co-Cr-Mo alloys
   - Stainless steel
   - Titanium and titanium alloys
2. Carbon fiber post
3. Quartz fiber post
4. Zirconia posts
5. Glass fiber post
6. Plastic posts.

II. Custom Made Posts

They can be cast from a direct pattern fabricated in patient's month or indirect pattern fabricated in the lab.

These can be of two types:

a. ***Custom cast metal post and core*** are usually made up of:
- Gold alloys
- Platinum-Palladium alloys
- Base metal alloys
- Co-Cr-Mo alloys
- Ni-Cr alloys.

b. ***Ceramic custom made posts*** are made up of all ceramic

B. ***Posts can also be classified as:***

1. ***Active Post:*** Active posts mechanically engage the canal walls. They are retentive in nature but can generate stresses during their placement and functional loading (Fig. 20.15).
2. ***Passive or Cemented Posts:*** Passive posts don't engage the canal walls. They are less retentive but also produce low stresses while placement and functional loading (Fig. 20.16).

C. ***According to Post Design***

They can be (Fig. 20.17):
- Smooth
- Parallel Sided
- Combination of above
- Serrated
- Tapered

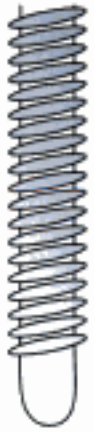


Fig. 20.15: Active post

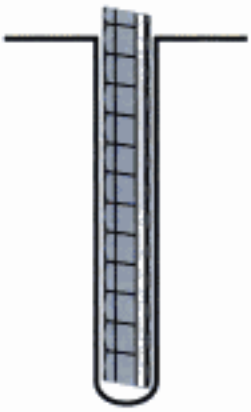


Fig. 20.16: Passive post

CUSTOM CAST METAL POST

The custom fabricated cast gold post and core has been used for decades as foundation restoration. Custom cast metal post is post of choice for single rooted teeth especially when remaining coronal tooth structure supporting the artificial crown is minimal. In such case, post must be capable of resisting the rotation which can be better achieved by custom cast posts.

Advantages

1. Adaptable to large irregularly shaped canals
2. Very strong
3. Better core retention because core is an inherent part of the post

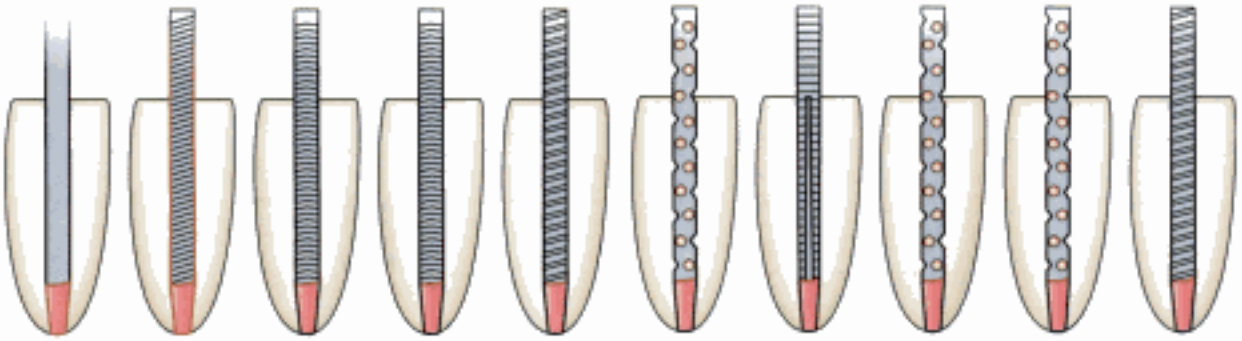


Fig. 20.17: Various post designed

CARBON FIBER POSTS

Carbon fiber posts were introduced by Duret et al in 1996 based on the carbon fiber reinforcement principle. Carbon fiber post consists of bundle of stretched carbon fibers embedded into an epoxy matrix. This was the first nonmetallic post introduced to the dentistry. The original form of carbon post was black and unaesthetic.

Advantages

1. Clinical procedure is less time consuming
2. Strong but low stiffness and strength than ceramic and metal posts
3. Easily retrievable
4. Less chair side time
5. Modulus of elasticity similar to dentin
6. Biocompatible
7. Good retention.

Disadvantages

1. Black in color, so unaesthetic
2. Radiolucent, so impossible to detect radiographically
3. Flexture strength decreases by 50 percent by moisture contamination
4. On repeated loading show reduced modulus of elasticity.

GLASS FIBER POST

It was introduced in 1992. It consists of unidirectional glass fibers embedded in a resin matrix which strengthens the dowel without compromising the modulus of elasticity.

Advantages

1. Aesthetically acceptable
2. Modulus of elasticity similar to dentin
3. Biocompatible
4. Distributes stresses over a broad surface area, thus increasing the load threshold.
5. Easy to handle and place
6. Less time consuming
7. Favorable retention in conjunction with adhesive bonding technique
8. High resistance to fracture
9. Easy retrieval.

Disadvantages

1. Poor radiographic visibility
2. Expensive
3. Technique sensitive

ZIRCONIA POST

These were introduced in dentistry in late 1980 by Christel et al. They are made from fine grained tetragonal zirconium polycrystals (TZP).

They possess high flexural strength and fracture toughness.

Advantages

1. For teeth with severe coronal destruction, zirconia posts provide adequate strength.
2. Smaller zirconia posts can be used for an all ceramic post and core construction for narrower canals.
3. Combination of glass ceramic and zirconia ceramic can be used because of their similarity in coefficient of thermal expansion.

Disadvantages

1. Adhesion to tooth and composite is compromised which becomes a problem for retreatment.
2. They are brittle with high modulus of elasticity.
3. When used with direct composite resin build up, high stresses and functional forces may lead to micro leakage and their deformation because of high polymerization shrinkage and high coefficient of thermal expansion of composites.
4. Expensive.

Factors to be Considered While Planning Posts:

- Retention and resistance form
- Preservation of tooth structure
- Ferrule effect
- Mode of failure
- Retrievability

FACTORS TO BE CONSIDERED WHILE PLANNING POST AND CORE

A. Retention and the Resistance Form

Post retention refers to the ability of post to resist vertical dislodging forces, Post resistance refers to the ability of the post and the tooth to with stand the lateral and rotational forces (Fig. 20.19).

Factors Affecting Post Retention

- Post length
- Post diameter
- Post taper and design
- Luting agent
- Luting method
- Canal shape
- Post position in dental arch

Factors Affecting Post Resistance
- Post length
- Rigidity
- Presence of antirotational features
- Presence of ferrule

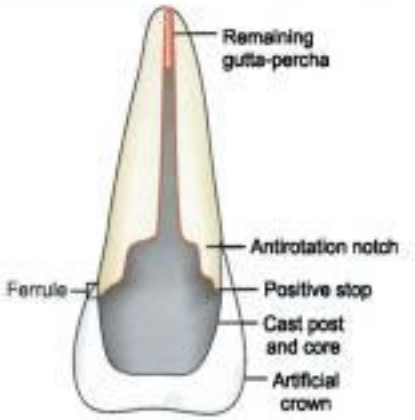


Fig. 20.19: Complete post and core system

POST LENGTH

There are many guidelines available as suggested by various authors regarding the post length. It is obvious that longer the post in the canal, more retentive it is. But increased length also increases risk of root fracture and perforation.

Generally, it is accepted that apical 3-6 mm of gutta-percha must be preserved to maintain the apical seal (Fig. 20.20).

Fig. 20.20: 3-6 mm of apical gutta-percha must be preserved to maintain apical seal

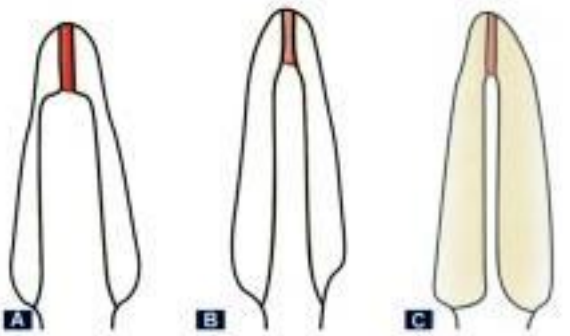
Accepted Guidelines for Determining Post Length Include

- Post should be equal to clinical crown length
- Post should be equal to one half to two thirds of the length of the remaining root.
- Post should end halfway between the crestal bone and the root apex.
- Post should be as long as possible without disturbing the apical seal.

Since root anatomy varies from tooth to tooth, so post space should be evaluated and planned accordingly.

POST DIAMETER

It has been seen that post diameter has little difference in the retention of post, but increase in post diameter increases the resistance form but it also increases the risk of root fracture (Fig. 20.21).



Figs 20.21A to C: **A.** Too wide diameter of post space **B.** Optimum diameter of post space **C.** Too narrow diameter of post space

Presently three different philosophies have been given regarding the post diameter. These are—

The Conservationist

It suggests the narrowest diameter that allows the fabrication of a post to the desired length. It allows minimal instrumentation of the canal for post space preparation (Fig. 20.22).

According to this, teeth with smaller dowels exhibit greater resistance to fracture.

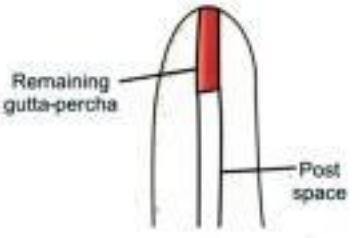


Fig. 20.22: Conservationist approach

The Preservationist

It advocates that at least 1mm of sound dentin should be maintained circumferentially to resist the fracture (Fig. 20.23).

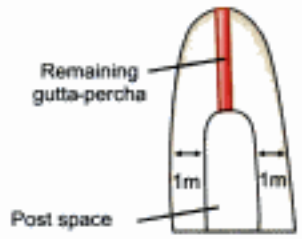


Fig. 20.23: Preservationist approach

The Proportionist

This advocates that post width should not exceed one third of the root width at its narrowest dimensions to resist fracture (Fig. 20.24). The guideline for determining appropriate diameter of post involves mesiodistal width of the roots.

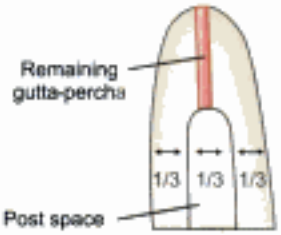


Fig. 20.24: Proportionist approach

POST DESIGN

Various types of post designs are available in the market (Fig. 20.25). The posts can be:

- Tapered, smooth sided—Least retentive.
- Tapered, serrated type

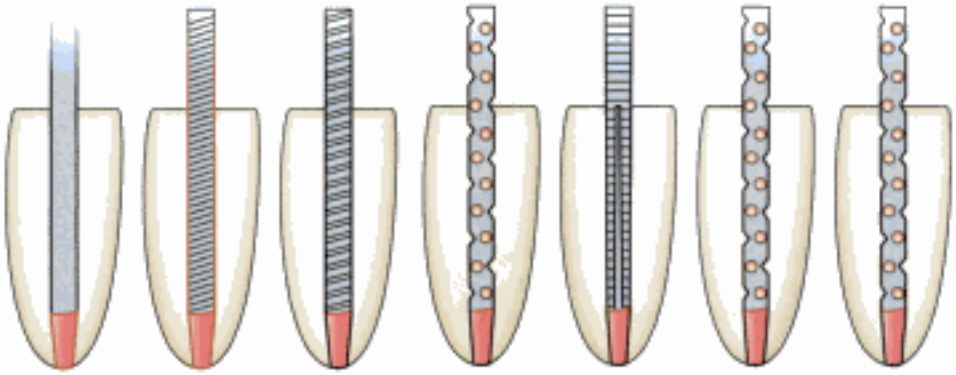


Fig. 20.25: Different post designs

- Parallel smooth sided
- Parallel serrated type
- Tapered notached
- Parallel threaded type
- Parallell notched type

**Generally parallel sided are more retentive than tapered ones. Threaded posts are more retentive than cemented ones.*

LUTING AGENTS

Commonly used dental cements for luting the posts are zinc phosphate (Fig. 20.26) , polycarboxylate, glass ionomer cement (Fig. 20.27) , resin based composite and hybrid of resin and ionomer.

Among these, zinc phosphate has shown the longest history of success. GIC is also one of the frequently used luting agent.

Fig. 20.26: Zinc phosphate cement used for luting post and core

(Fig. 20.36), over enlargement of the canal space (Fig. 20.37) and perforation (Fig. 20.38).

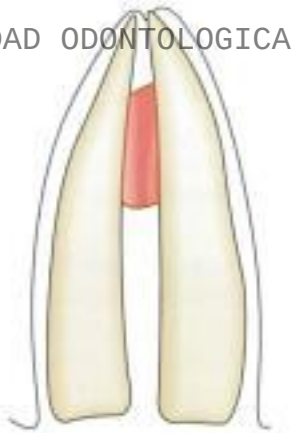


Fig. 20.36: Disturbance of apical seal

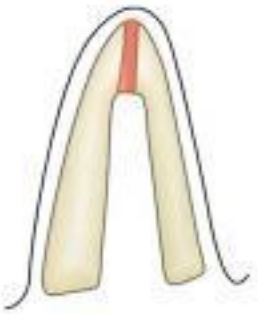


Fig. 20.37: Overenlargement of canal space

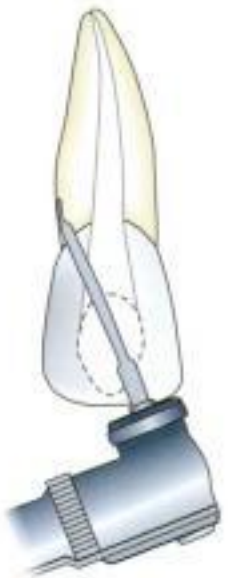


Fig. 20.38: Perforation caused by misdirection of bur

4. Following the preparation of canal space, preparation of coronal tooth structure should be prepared in the same manner as if an intact crown irrespective of the remaining tooth structure (Fig. 20.39).
5. Remove all the unsupported tooth structure (Fig. 20.40).

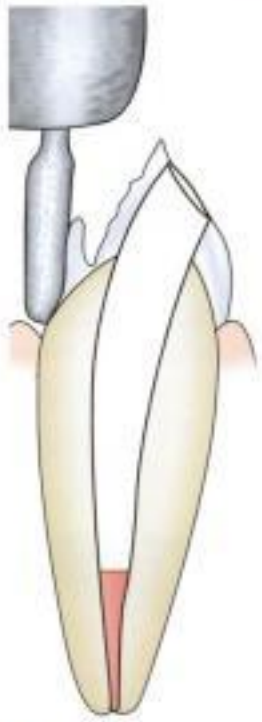


Fig. 20.39: Preparation of coronal surface

Fig. 20.40: Removal of unsupported tooth structure

6. Place an antirotational notch with the help of cylindrical diamond or carbide bur. This is done to provide the antirotational stability (Fig. 20.41).
7. Ferrule effect is provided thereafter. The remaining coronal tooth structure is sloped to buccal and lingual surfaces so as to provide a collar around the occlusal circumference of the preparation (Fig. 20.42). This gives rise to 360° ferrule effect. Ferrule ensures that the final restoration encircles the tooth apical to the core and rests on sound tooth structure. It also presents the vertical root fracture by posts.

Fig. 20.41: Placement of anti-rotational notch

Fig. 20.42: Establish a smooth finish line

8. Finally eliminate all the sharp angles, undercuts and establish a smooth finish line (Figs 20.43 to 20.45).

CORE

Core is the supragingival portion that replaces the missing coronal tooth structure and forms the center of new restoration. Basically it acts as a miniature crown.

Various Core Build Ups Materials Available are
- Dental amalgam
- Resin modified glass ionomers
- Composite resin
- Reinforced glass ionomers cement

Ideal Requirements for a Core Material
- Compressive strength to resist intraoral forces
- Biocompatibility
- Ease of manipulation
- Flexure strength to present core dislodgement
- Ability to bond to tooth structure and post.
- Coefficient of thermal expansion similar to dentin.
- Minimal water absorption
- Dimensionally stable
- No reaction with chemicals
- Low cost
- Easily available
- Contrasting color to tooth structure except when used for anterior teeth.

Fig. 20.43: Check the accuracy of final post and core

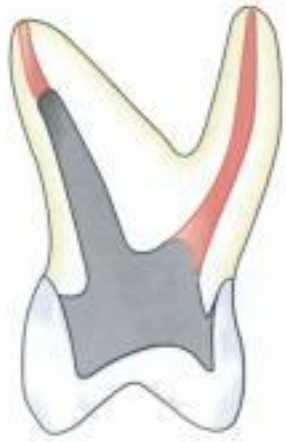


Fig. 20.44: In maxillary molars, palatal canal is selected for post placement

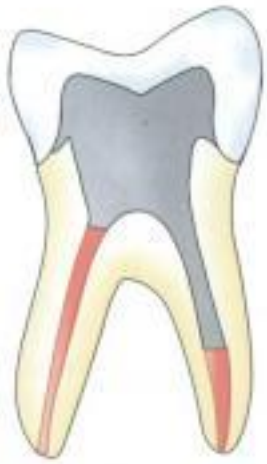


Fig. 20.45: In mandibular molars, distal canal is usually selected for post placement

BIOMECHANICAL CRITERIA FOR EVALUATION OF CORE MATERIALS

Bonding

Resin composites > Glass ionomers > Amalgam

Strength

Amalgam > Resin composite > Glass ionomers

Ease of Use

Resin composites > Amalgam > Glass ionomers

Setting Time

Resin composite > Glass ionomers > Amalgam

Dimensional Stability

Amalgam > Glass ionomers > Composite resins

Following the endodontic treatment, it is necessary to restore the original morphology and function of the tooth which can be achieved by restoration of the endodontically treated teeth. The restoration should begin at the earliest possible moment because tooth exposed to oral conditions without optimal restoration can not resist the occlusal forces and oral bacteria for a long period which can result in the treatment failure. Post endodontic restoration is an important treatment itself because successful treatment can not be achieved without adequate restoration after endodontic treatment. Proper restoration of endodontically treated tooth begins with understanding of their physical and biomechanical properties and anatomy. Though various new materials have become available for past many years, yet the basic concepts of restoring endodontically treated teeth remains the same.

Most post systems can be used successfully if basic principles are followed. After selection of the post system, finally it is the choice of core material and final restoration which increases the longevity of the treated tooth. The main function of post is retention of the core if insufficient tooth structure is present to support the coronal final restoration They don't strengthen the tooth, so posts should not be used habitually.

Various types of post systems are available with different strengths and weaknesses. Selection of post should be made by keeping in mind its strength, modulus of elasticity, biocompatibility, retrievability, aesthetics and cost. Though many new materials are available with their indications for use, but long term evaluations are needed. So care must be taken while selecting these materials.

21 Management of Traumatic Injuries

- Introduction
- Classification of Dentofacial Injuries
- Examination of Traumatic Injuries
- Management of Traumatic Injuries
  - Crown Infarction
  - Uncomplicated Crown Fractures
  - Complicated Crown Fractures
  - Crown Root Fractures
  - Root Fracture
  - Luxation Injuries
  - Avulsion
- Prevention of Traumatic Injuries

INTRODUCTION

It has been seen that dental traumatic injuries are increasing in their frequency of occurrence, though most of them usually consist of cracked and chipped teeth but it has been suggested that incidence of dental trauma will exceed that of dental caries and periodontal disease among children and young adults. A dentist must be prepared to treat not only the minor injuries but also more traumatic broken crowns, roots, bones, etc.

Though traumatic injuries can occur at any age but most commonly they are seen at the age of 2-5 years during which children are learning to walk. They fall commonly because their judgment and coordination are not fully developed. Another age at which dental injuries are common is 8-12 years when there is increased sports activity, and while learning bicycle, etc. Any one can be injured. Automobiles accidents, sports mishaps, bad fall may make any body patient of dental trauma.

Etiology of Traumatic Injuries

- **A**utomobile injury
- **B**attered child
- **C**hild abuse
- **D**rug abuse
- **E**pilepsy
- **F**alls from height
- **S**ports related injuries

40-60 percent of dental accidents occur at home. Prior to 1960's boys to girls ratio in traumatic injuries used to be 3:1 but because of more involvement of females in sports, it has reduced to 2:1.

Type and number of teeth injured in accident vary according to type of accident occurred, impact of force, resiliency of object hitting the tooth, shape of the hitting object and direction of the force. If bone is resilient, tooth will be displaced by trauma but if bone is thick and brittle tooth will fracture. Maxillary central incisor is most commonly affected tooth followed by maxillary lateral incisor and mandibular incisors (Fig. 21.1).

Fig. 21.1: Traumatized maxillary central incisor

Extent of trauma can be assessed by four factors (Hallet;1954)

- Energy of impact: As we know
  Energy = Mass × Velocity
  Thus, the hitting object with more mass or high velocity creates more impact.
- ***Direction of impacting force:*** Type of fracture depends on the direction.
- ***Shape of impacting object:*** Sharpness or bluntness of object also affect the impact.
- ***Resilience of impacting object:*** Hardness or softness of the object also affects the extent of the injury.

Repairing the injury is the immediate problem ensuring that the tooth will continue to thrive, understanding the impact of trauma on the pulp and importance of pulp vitality to sound tooth development is essential for optimum patient care Endodontics can offer a unique help to save a tooth.

The outcome of dental injury is influenced by patient age, severity, and treatment offered. In most of the cases, immature permanent teeth with injuries have better prognosis than fully formed teeth with same injuries.

Proper treatment of dental injuries can have a significant effect on outcome and prognosis especially in case of severe injury. For example timely replantation of an avulsed tooth followed by endodontic therapy can improve the prognosis of the tooth.

Follow-up evaluation is also important for example if root resorption is detected early, it can be arrested.

CLASSIFICATION OF DENTOFACIAL INJURIES

The purpose of classifying dental injuries is to provide description of specific condition allowing the clinician to identify and treat that condition using specific treatment remedies.

The currently recommended classification is one based on the WHO and modified by Andreasen and Andreasen. This classification is used by International Association of Dental Traumatology.

Soft Tissues

Lacerations	- 873.69
Contusion	- N 902.0
Abrasions	- N 910.00

Tooth Fractures

N873.60 Enamel fracture
N873.61 Crown-fractures-uncomplicated (no pulp exposure)
N873.62 Crown-fractures-complicated (with pulp exposure)
N873.64 Crown-root fractures
N873.63 Root fractures

Luxation Injuries:

873.66	- Tooth concussion
873.66	- Subluxation
873.66	- Extrusive luxation
873.66	- Lateral luxation
873.67	- Intrusive luxation
873.68	- Avulsion

Facial Skeletal Injuries:

Classification (Rabinowitch ;1956)

Class I: Enamel fracture
Class II: Enamel and dentin fracture
Class III: Enamel and dentin fracture along with pulpal exposure
Class IV: Root fracture
Class V: Communication
Class VI: Exarticulation

Classification Based on Endodontic Treatment (Ulton; 1985)

Class I: Fracture of enamel
Class II: Fracture of crown with indirect pulp exposure
Class III: Fracture of crown with direct pulp exposure

Ellis and Davey's Classification (1960)

- Class I - Simple fracture of the crown involving enamel.
- Class II - Extensive fracture of the crown, with considerable amount of dentin involved but no pulp exposure.
- Class III - Extensive fracture of the crown, with considerable amount of dentin involved, with pulp exposure.
- Class IV - Traumatized tooth becomes non vital

Contd...

Another important question to ask is whether treatment of any kind has been given elsewhere for injury before coming to dental office.

MEDICAL HISTORY

Patient should be asked for:

- Allergic reaction to medication
- Disorders like bleeding problems, diabetes, epilepsy etc.
- Any current medication patient is taking
- *Condition of Tetanus immunization*—In case of contaminated wound, booster dose should be given if more than 5 years have elapsed since last dose. But for clean wounds, no booster dose needed, if time elapsed between last dose is less than 10 years.

CLINICAL EXAMINATION

Extraoral examination should rule out any facial bone fracture and should include meticulous evaluation of the soft tissues. Soft tissues such as lips, tongue, cheek, floor of mouth ought to be examined. Lacerations of lips and intraoral soft tissues must be carefully evaluated for presence of any tooth fragments and/or other foreign bodies.

Occlusion and temporomandibular joints should also be examined carefully (Fig. 21.2). Abnormalities in occlusion can indicate fracture of jaws or alveolar process.

Teeth must be checked after proper cleaning the area. Enamel cracks can be visualized by changing the direction of light beam from side to side. Explore the extent of tooth fracture involvement, i.e. enamel, dentin, cementum and/or pulp. Evaluate the crowns of the teeth for presence of extent of fracture, pulp involvement or change in color. Root fracture can be felt by placing finger on mucosa over the tooth and moving the crown (Fig. 21.3).

Patient's periodontal status can influence the dentist's decision to treat that injury. Teeth and their supporting structures should be examined carefully not only the obviously injured tooth but the adjacent as well as opposite teeth as well.

Reaction to percussion is indicative of the damage to the periodontal ligament. Tooth can show response to percussion in the normal way or it may be tender on percussion when evaluation of periodontal ligament is being done (Fig. 21.4).

Examine mobility in all the directions. If adjacent teeth move along with the tooth being tested, suspect the alveolar fracture (Fig. 21.5). In crown fracture, the crown is mobile but tooth will remain in the position.

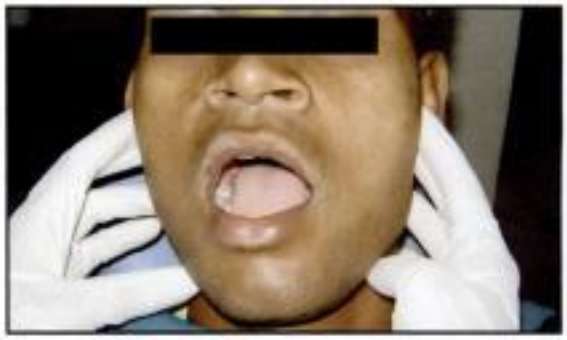


Fig. 21.2: Examination of TMJ

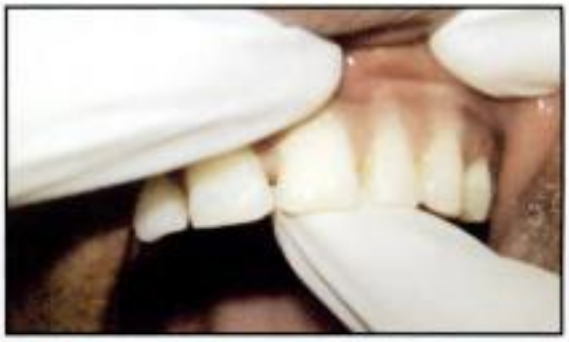


Fig. 21.3: Root fracture can be felt by placing finger on mucosa over tooth and moving the crown

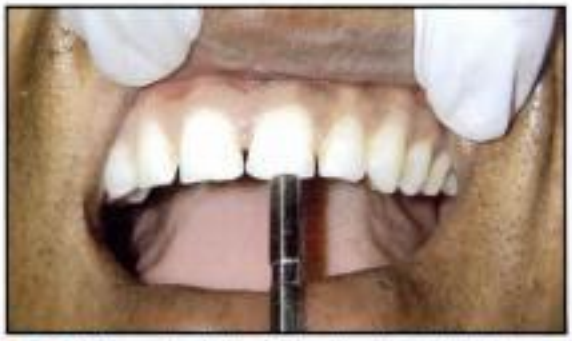


Fig. 21.4: Percussion to check integrity of periodontal ligament

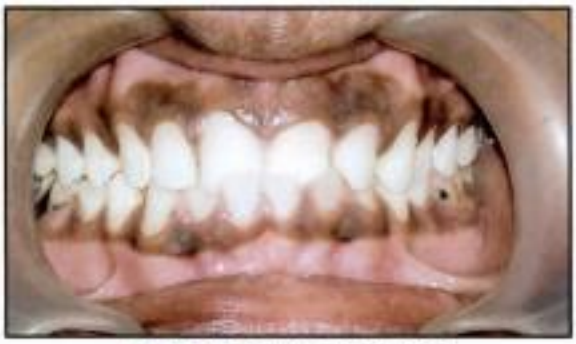


Fig. 21.5: Fractured central incisor

Fig. 21.8: Smoothening of rough edges by selective grinding of enamel **A.** Central incisor with ragged margins **B.** Smoothening of rough edges

- Repairing fractured tooth surface by composite if needed for cosmetic purposes.
- Regular pulp testing should be done and recorded for future reference.
- Follow-up of patient at 3, 6 and 12 months interval is done.

Prognosis

Prognosis is good for infarction cases.

CROWN FRACTURE

Uncomplicated Crown Fracture

Crown factures involving enamel and dentin and not the pulp are called as ***uncomplicated crown fractures*** (Fig. 21.9). These occur more frequently than the complicated crown fractures. Such fractures are usually not associated with pain and they don't require an urgent care.

Fig. 21.9: Uncomplicated crown fracture

Incidence

Incidence of these fractures varies from 26-92 percent of all the traumatic injuries of teeth.

Biological Consequences

Minimal consequences are seen if only enamel is fractured but if dentin is exposed, a direct pathway for various irritants to pass through dentinal tubules to underlying pulp is formed. Pulp may remain normal or may get chronically inflamed depending upon proximity of fracture to the pulp, size of dentinal tubules and time of the treatment provided.

Diagnosis

It could be easily revealed by clinical examination. If dentin is exposed, sensitivity to heat or cold may be present. Sometimes lip bruise or lacerations are also seen to be associated with injury.

Treatment

The main objective of the treatment is to protect the pulp by obliterating dentinal tubules.

In case of enamel fractures, selective grinding of incisal edges will be sufficient to remove the sharp edges to prevent injury to lips, tongue, etc.

For esthetic reasons, composite restorations can be placed after acid etching.

If there is involvement of both enamel and dentin: A restoration is needed to seal the dentinal tubules and to restore the aesthetics (Fig. 21.10).

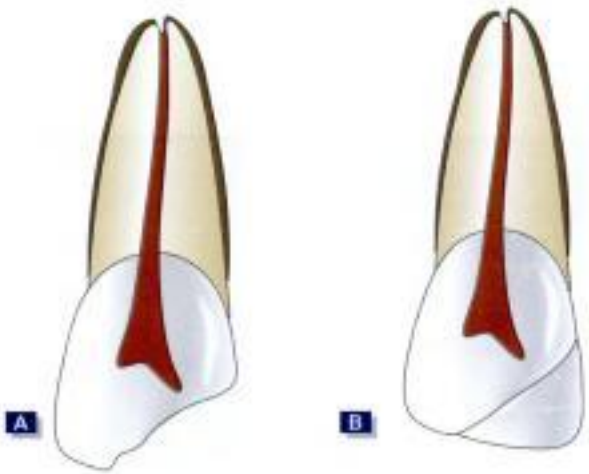


Fig. 21.10: Reattachment of broken fragment using bonding agent

Factors Affecting Pulpal Survival

Optimal blood circulation is necessary to nourish the pulp and keeps it healthy. The type of injury, the stage of root development and the degree of infection are factors that affect circulation to the injured area and pulp vitality. Bacteria may invade the pulp through crack that occurred because of trauma, causing inflammation and pulp necrosis.

Vitality testing will not be useful in determining the status of immature apex. Until apical closure occurs, teeth do not respond normally to pulp testing. Also, a traumatic injury sometimes temporarily alters the conduction potential of the nerve endings in the pulp leading to false readings. One must relate experience, radiographs, clinical signs or symptoms and knowledge of the healing process to assess pulp vitality.

Pulp Capping and Pulpotomy

Pulp capping and pulpotomy are the measures that permit apexogenesis to take place and may avoid the need for root canal therapy. The choice of treatment depends on the size of the exposure, the presence of hemorrhage and the length of time since the injury.

Pulp Capping

Pulp capping implies placing the dressing directly on to the pulp exposure (Figs 21.13A and B).

Indications

On a very recent exposure (< 24 hours) and probably on a mature, permanent tooth with a simple restorative plan.

Technique

After adequate anesthesia, a rubber dam is placed. Crown and exposed dentinal surface is thoroughly rinsed with saline followed by disinfection with 0.12 percent chlorhexidine or betadine. Pure calcium hydroxide mixed with anesthetic solution or saline is carefully placed over the exposed pulp and dentinal surface. The surrounding enamel is acid etched and bonded with composite resin.

Follow-up

Vitality tests, palpation tests, percussion tests and radiographs should be carried out for 3 weeks; 3, 6 and 12 months; and every twelve months subsequently. Continued root development of the immature root is evaluated during this periodic radiographic examination.

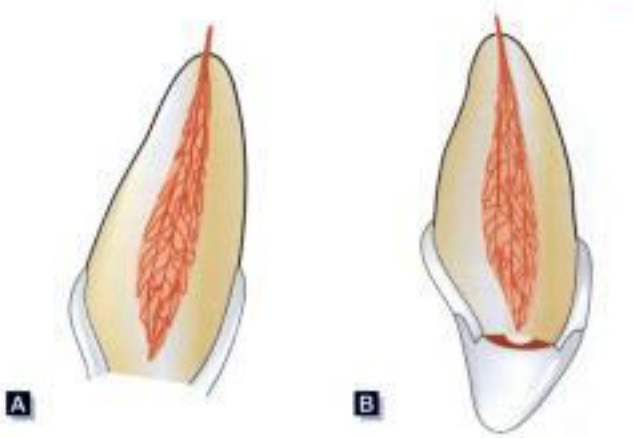


Fig. 21.13: Pulp capping of complicated crown fracture

Prognosis

It depends on ability of $Ca(OH)_2$ to disinfect the superficial pulp and dentin and to necrose the zone of superficially inflamed pulp. Along with it, quality of bacteria tight seal provided by restoration is an important factor. Prognosis is up to 80 percent

Pulpotomy

Two types:

- Partial pulpotomy
- Full (cervical) pulpotomy

Partial Pulpotomy

Partial pulpotomy also termed as "Cvek Pulpotomy", it implies removal of the coronal pulp tissue to the level of healthy pulp (Fig. 21.14).

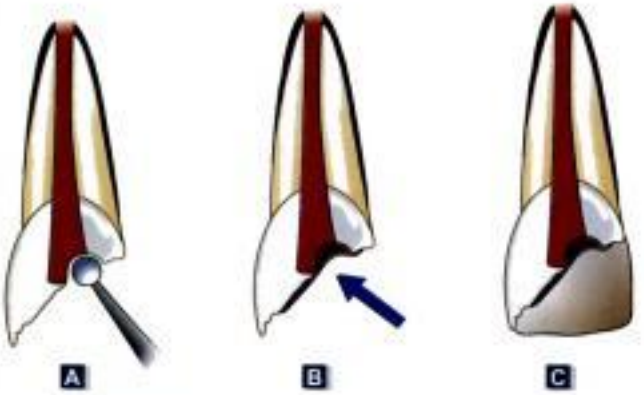


Fig. 21.14: Shallow pulpotomy of fractured tooth

vital pulp tissue remains in the root canal. If the pulp tissue is necrotic, apexification is the process which stimulates the formation of a calcified barrier across the apex (Fig. 21.16). Apexification is done to stimulate the hard tissue barrier. For this, initially all canals are disinfected with sodium hypochlorite solution to remove any debris and bacteria from the canal. Following this calcium hydroxide is packed against the apical soft tissue and later backfilling with calcium hydroxide is done to completely obturate the canal.

When completion of hard tissue is suspected (after 3-6 months), calcium hydroxide is removed and radiograph is taken. When found satisfactory, obturation of the canal using softened gutta percha techniques is done. One should avoid excessive lateral forces during obturation because of thin walls of the root.

Nowadays, MTA, i.e. mineral trioxide aggregate is also used in place of calcium hydroxide. Pulp response to this material is favorable. Since this material doesn't appear to disintegrate with time, it might not be necessary to replace the restoration after dentin bridge formation as it is done in case of calcium hydroxide (Fig. 21.17).

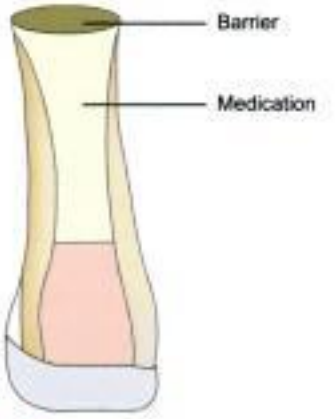


Fig. 21.16: Formation of hard tissue barrier

Fig. 21.17: Use of MTA for apexification

CROWN ROOT FRACTURES

Crown root fracture involves enamel, dentin and cementum with or without the involvement of pulp (Fig. 21.18). These fractures are usually oblique in nature involving both crown and root (Fig. 21.19). This type of injury is considered as more complex type of injury because of its more severity and involvement of the pulp (Fig. 21.20).

Incidence

It contributes 5 percent of total dental injuries. In anterior teeth, it usually occurs by direct trauma causing chisel type fracture which splits crown and root. In posterior teeth, fracture is rarely seen but it can occur because of indirect trauma like large sized restorations and high speed instrumentation, etc.

Fig. 21.18: Crown root fracture

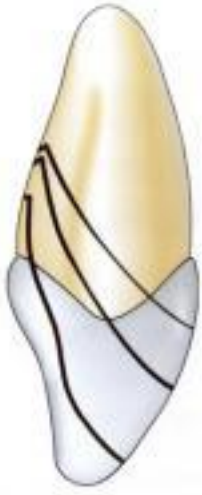


Fig. 21.19: Different levels of crown root fracture

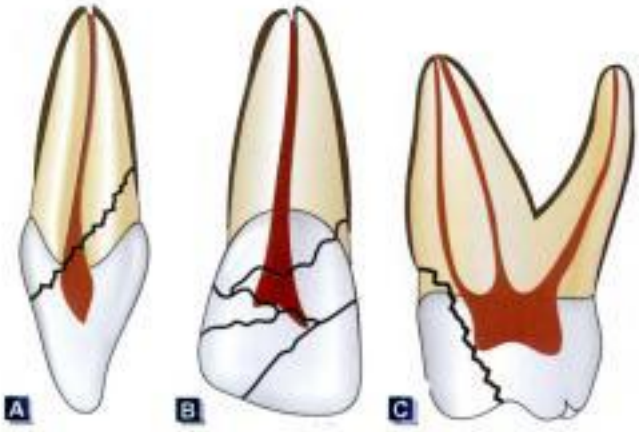


Fig. 21.20: **A.** Crown root fracture – chisel type, **B.** Shattering of crown by crown root fracture, **C.** Crown root fracture of posterior teeth

Biological Consequences

Biological consequences are similar to as that of complicated or uncomplicated fracture depending upon the pulp involvement. In addition to these, periodontal complications are also present because of encroachment of the attachment apparatus.

Diagnosis

Crown root fractures are complex injuries which are difficult both to diagnose as well as treat. The fracture line in such cases is usually single but multiple fractures can also occur, often originating from the primary fracture.

A tooth with crown root/fracture exhibits following features:

- Coronal fragment is mobile. Patient may complain of pain from mastication due to movement of the coronal portion.
- Inflammatory changes in pulp and periodontal ligament are seen due to plaque accumulation in the line of fracture.
- Patient may complain of sensitivity to hot and cold.

Radiographs are taken at different angles to assess the extent of fracture (Fig. 21.21). Indirect light and transillumination can also be used to diagnose this type of fracture.

Treatment

The primary goal of the treatment, initially is the elimination of pain which is mainly because of mobile crown fragment. It can be done by applying bonding agents to bond the loose fragments together, temporary crown placement or by using glass ionomer cement.

The main objective of the treatment is to:

a. Allow subgingival portion of the fracture to heal.
b. Restoration of the coronal portion.

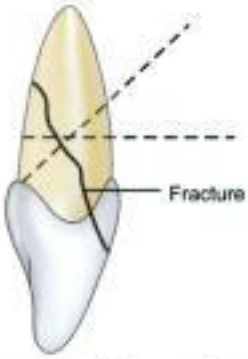


Fig. 21.21: Radiographic technique used for crown root fracture

Following should be considered while management of crown root fracture:

- If there is no pulp exposure, fragment can be treated by bonding alone or by removing the coronal structure and then restoring it with composites (Fig. 21.22).
- If pulp exposure has occurred, pulpotomy or root canal treatment is indicated depending upon condition of the tooth.
- When remaining tooth structure is adequate for retention, endodontic therapy and crown restoration are possible with the help of crown lengthening procedures.
- When root portion is long enough to accommodate a post retained crown, then surgically removal of the coronal fragment and surgical extrusion of the root segment is done (Fig. 21.23).
- To accommodate a post retained crown, after removal of the crown portion, orthodontic extrusion of root can also be done (Fig. 21.24).

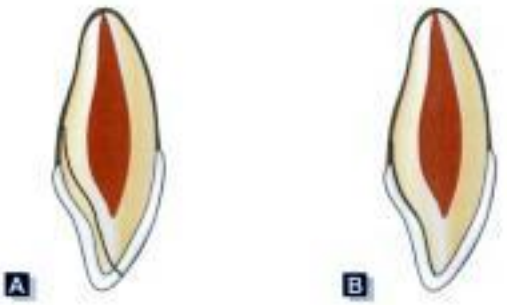


Fig. 21.22: Removal of coronal fragment and supragingival restoration

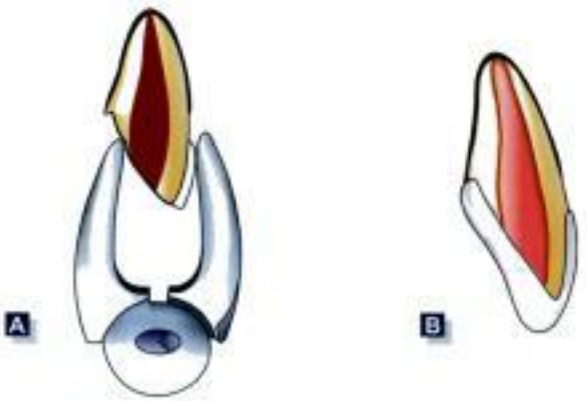


Fig. 21.23: Removal of coronal fragment and surgical extrusion of root

Fig. 21.24: Orthodontic extrusion of the root

- When the fracture extends below the alveolar crest level, the surgical repositioning of tissues by gingivectomy, osteotomy etc. should be done to expose the level of fracture and subsequently restore it.

Prognosis

Long term prognosis depends on quality of coronal restoration. Otherwise the prognosis is similar to complicated or uncomplicated fracture.

ROOT FRACTURE

These are uncommon injuries but represent a complex healing pattern due to involvement of dentin, cementum, pulp and periodontal ligament (Fig. 21.25) and (Fig. 21.26).

Incidence They form the 3 percent of the total dental injuries. These fractures commonly result from a horizontal impact. Root fractures are usually transverse to oblique in nature (Fig. 21.27). These fractures require radiographs at different angles foe their accurate identification.

Fig. 21.25: Root fracture of anterior teeth

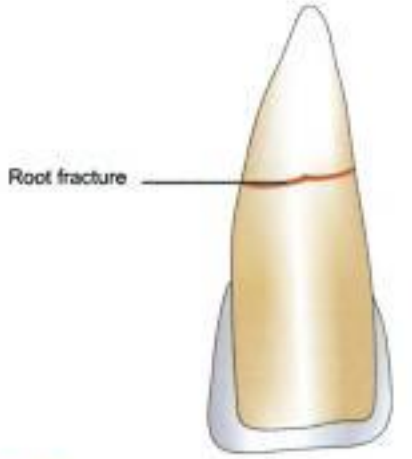


Fig. 21.26: Root fracture involving central incisor

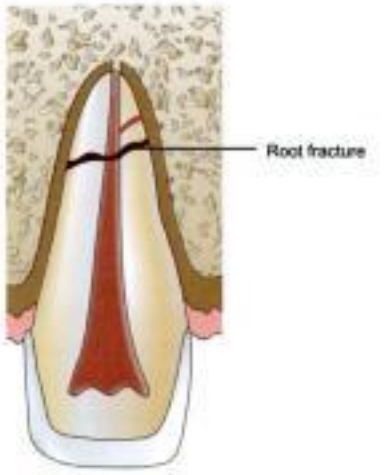


Fig. 21.27: Root fracture can be transverse or oblique in nature

Biological Consequences

When root fractures occur horizontally, coronal segment is displaced to varying degrees. If vasculature of apical segment is not affected, it rarely becomes necrotic.

Diagnosis

Displacement of coronal segment usually reflects the location of fracture (Fig. 21.28).

Radiographs at varying angles (usually at 45°, 90° and 110°) are mandatory for diagnosing root fractures (Fig. 21.29).

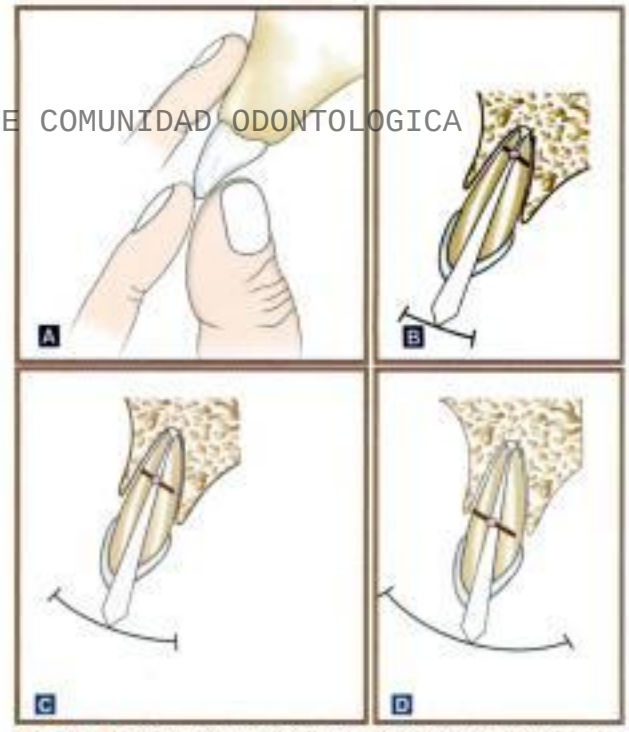


Fig. 21.28: Diagnosis of location of root fracture A. Palpating the facial mucosa with one finger and moving crown with other finger B. to D. Arc of mobility of incisal segment of tooth with root. As fracture moves incisally, arc of mobility increases

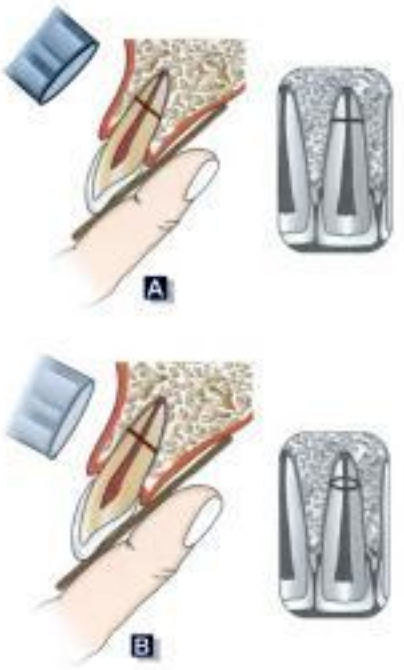


Fig. 21.29: A. Radiographic beam parallel to fracture B. Radiographic beam oblique to fracture

Treatment Options for Teeth with Root Fractures with Necrotic Pulp

1. Root canal treatment for both apical and coronal segment
2. Root canal treatment for coronal part and no treatment for apical segment.
3. Surgical removal of apical segment, while root canal treatment of coronal part.
4. Root canal treatment for coronal part with apexification procedure in apical part of this segment, but no treatment for apical portion of the teeth.
5. Use of splints.
6. Endodontic implant after extraction.
7. Root extrusion in teeth with fracture at the level of alveolar crest.

Treatment of Root Fractures

If there is no mobility of tooth and tooth is asymptomatic, only apical third fracture is suspected. In this case to facilitate pulpal and periodontal ligament healing, displaced coronal portion should be repositioned accurately (Fig. 21.30). It is stabilized by splinting for 2-3 weeks (Fig. 21.31).

By and large it is seen that apical segment of fractured root contains vital healing pulp whereas coronal pulp has become necrotic. In these cases, following treatment options are available.

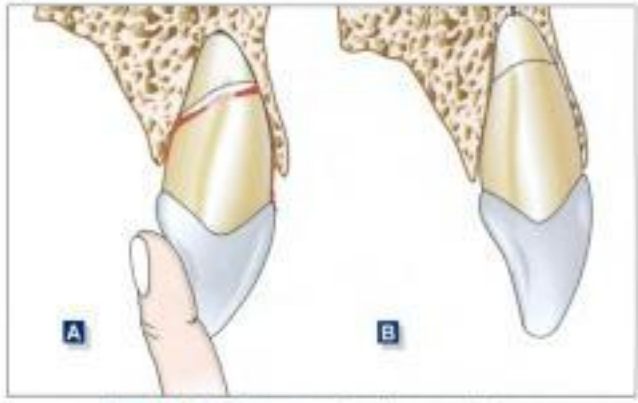


Fig. 21.30: Treatment of root fracture involves repositioning of tooth and splinting

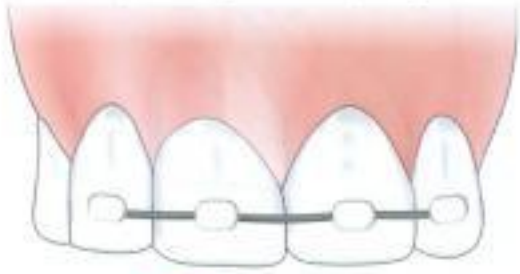


Fig. 21.31: Splinting of teeth

1. Root canal therapy for both coronal and apical segment, when they are not separated (Fig. 21.32).
2. Root canal therapy of coronal segment and no treatment of apical segment, which apical segment contains vital pulp (Fig. 21.33).
3. Root canal therapy for coronal segment and surgical removal of apical third (Fig. 21.34).
4. Apexification type procedure of coronal segment, i.e. inducing hard tissue barrier at exit of coronal root canal and no treatment of apical segment. This is most commonly used procedure nowadays (Fig. 21.35).
5. Intraradicular splint in which rigid type of post is used to stabilize the two root segments (Fig. 21.36).
6. Endodontic implants, in which the apical portion of implant replaces the surgically removed apical root segment (Fig. 21.37).
7. Root extrusion for teeth with fracture at or near alveolar crest. Here coronal segment is removed and apical segment is extruded to allow restoration of missing coronal tooth structure (Fig. 21.38).

Fig. 21.32: Endodontic treatment of crown and root

Fig. 21.33: Endodontic treatment of coronal segment only

Fig. 21.34: Endodontic treatment of coronal segment with surgical removal of apical part

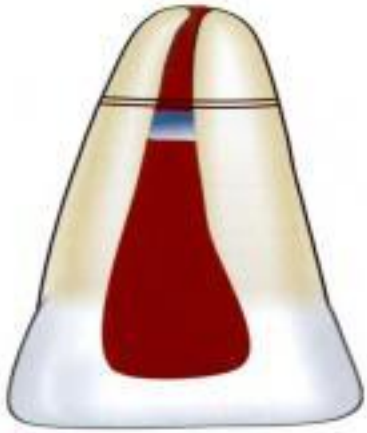


Fig. 21.35: Apexification of coronal segment

Fig. 21.36: Stabilization of root with intraradicular splint

Fig. 21.37: Endosseous implant for stablization of tooth

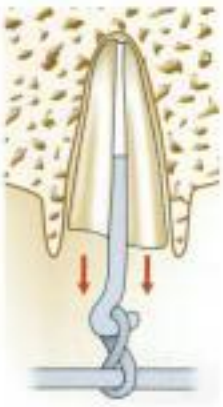


Fig. 21.38: Orthodontic extrusion of apical segment

PROGNOSIS DEPENDS ON

- ***Amount of dislocation and degree of mobility of coronal segment:*** More is the dislocation, poorer is the prognosis.
- Stage of tooth development : More immature the tooth, better the ability of pulp to recover from trauma.

According to the Andreasen and Hjorting—Hansen, root fracture can show healing in following ways:

1. Healing with calcified tissue in which fractured fragments are in close contact (Fig. 21.39).
2. Healing with interproximal connective tissue in which radiographically fragments appear separated by a radiolucent line (Fig. 21.40).
3. Healing with interproximal bone and connective tissues. Here fractured fragments are seen separated by a distinct bony bridge radiographically (Fig. 21.41).
4. Interproximal inflammatory tissue without healing, radiographically it shows widening of fracture line (Fig. 21.42).

Fig. 21.39: Interproximal inflammatory tissue seen in root fracture

Fig. 21.40: Healing of root fracture with calcified tissue

Fig. 21.41: Healing of root fracture by interproximal bone

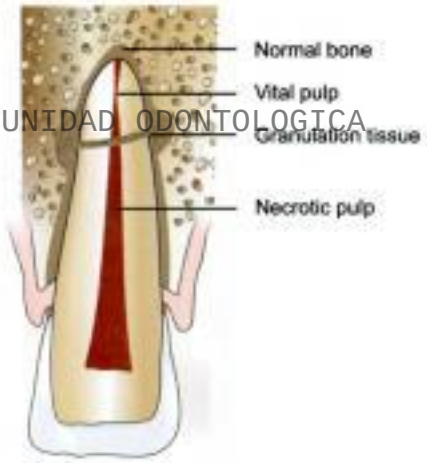


Fig. 21.42: Healing of root fracture by formation of connective tissue between the segments

Follow-up Procedure

- Pulp testing and radiographic examination should be performed at 3 weeks, 6 weeks, 6 and 12 months after the injury.
- Radiographs are taken to predict healing of root fracture. Resorption within the root canal originating at fracture line indicates healing following pulpal damage after trauma. But resorption within the bone at the level of fracture line indicates pulp necrosis which requires endodontic therapy.

LUXATION INJURIES

Luxation injuries cause trauma to supporting structures of teeth ranging from minor crushing of periodontal ligament and neurovascular supply of pulp to total displacement of the teeth.

They are usually caused by sudden impact such as blow, fall or striking a hard object.

Mainly Five Types of Luxation Injuries are seen:

- Concussion
- Subluxation
- Lateral luxation
- Extrusive luxation
- Intrusive luxation

Incidence

They form 30-40 percent of all the dental injuries.

CONCUSSION

In concussion (Fig. 21.43):

- Tooth is not displaced.
- Mobility is not present.
- Tooth is tender to percussion because of edema and hemorrhage in the periodontal ligament.
- Pulp may respond normal to testing.

SUBLUXATION

In subluxation (Fig. 21.44)

- Teeth are sensitive to percussion and have some mobility.
- Sulcular bleeding is seen showing damage and rupture of the periodontal ligament fibers.
- Pulp responds normal to testing.
- Tooth is not displaced (Fig. 21.45).

Fig. 21.43: Concussion

Fig. 21.44: Subluxation

Fig. 21.45: Subluxation showing injury to periodontium

Treatment of Concussion and Subluxation

- Rule out the root fracture by taking radiographs.
- Relief the occlusion by selective grinding of opposing teeth (Fig. 21.46).
- Immobilize the injured teeth.
- Endodontic therapy should not be carried out at first visit because both negative testing results and crown discoloration can be reversible.

Follow-up is done at 3 weeks, 3, 6 and 12 months.

Prognosis there is only a minimal risk of pulp necrosis and root resorption.

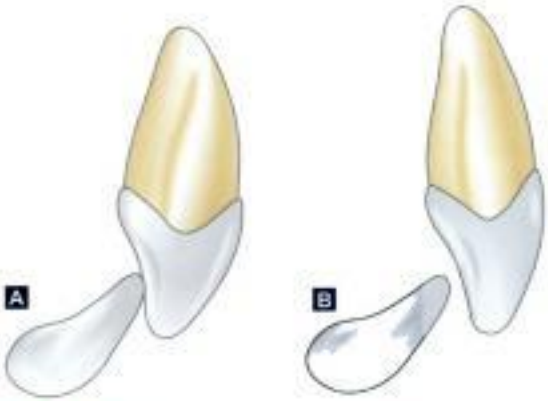


Fig. 21.46: Treatment of injury by selective grinding of tooth

LATERAL LUXATION

In lateral luxation:

- Trauma displaces the tooth lingually, buccally, mesially or distally, in other words out of its normal position away from its long axis (Fig. 21.47).

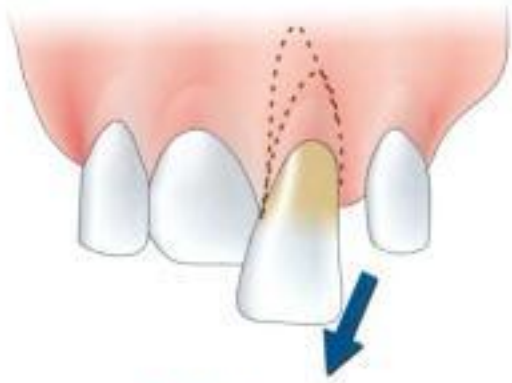


Fig. 21.47: Lateral luxation

- Sulcular bleeding is present indicating rupture of PDL fibres (Fig. 21.48).
- Tooth is sensitive to percussion
- Clinically, crown of laterally luxated tooth is usually displaced horizontally with tooth locked firmly in the new position. Here percussion may elicit metallic tone indicating that root has forced into the alveolar bone.

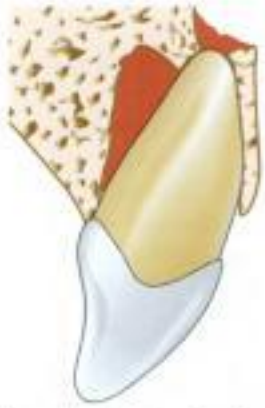


Fig. 21.48: Lateral luxation showing injury to periodontium

EXTRUSIVE LUXATION

In extrusive luxation

- Tooth is displaced from the socket along its long axis (Fig. 21.49)
- Tooth is very mobile
- Radiograph shows the displacement of tooth.

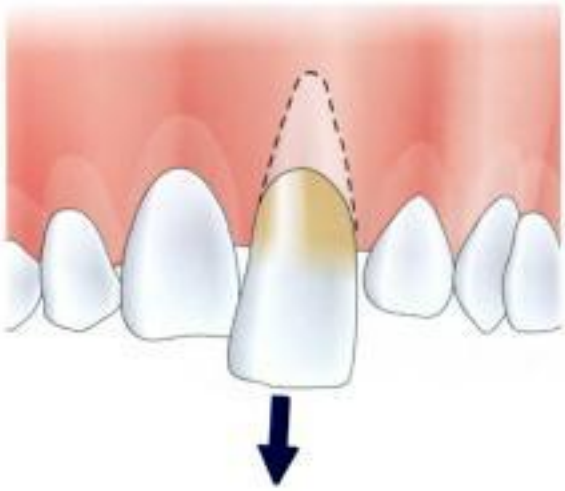


Fig. 21.49: Extrusive luxation

Treatment of Lateral and Extrusive Luxation

Treatments of these injuries consist of a traumatic repositioning and fixation of teeth which prevents excessive movement during healing.

Laterally luxated teeth are repositioned with minimal required force. Before repositioning laterally luxated teeth, anesthesia should be administrated. Tooth must be dislodged from the labial cortical plate by moving it coronally and then apically. Thus tooth is first moved coronally out of the buccal plate of bone and then fitted into its original position (Fig. 21.50).

For repositioning of extruded tooth, a slow and steady pressure is required to displace the coagulum formed between root apex and floor of the socket (Fig. 21.51). After this tooth is immobilized, stabilized and splinted for approximately 2 weeks. Local anesthesia is not needed while doing this.

Follow-up: Splint is removed 2 weeks after extrusion. If tooth has become nonvital, inflammatory root resorption can occur, requiring immediate endodontic therapy.

Pulp testing should be performed on regular intervals.

Prognosis

It depends on stage of root development at the time of injury. Commonly seen sequel of luxation injuries are pulp necrosis, root canal obliteration and root resorption.

INTRUSIVE LUXATION

In intrusive luxation

- Tooth is forced into its socket in an apical direction (Fig. 21.52).
- Maximum damage has occurred to pulp and the supporting structures (Fig. 21.53).
- When examined clinically, the tooth is in infraocclusion (Fig. 21.52).

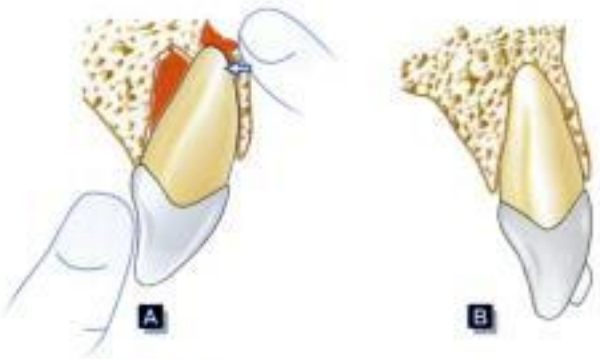


Fig. 21.50: Treatment of lateral luxation

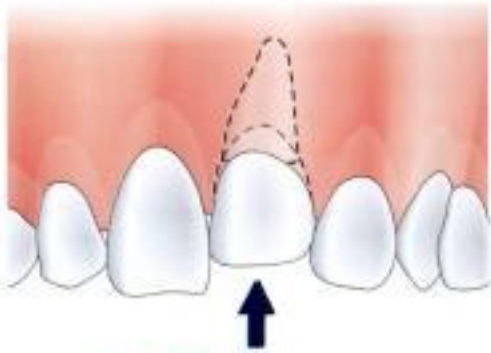


Fig. 21.52: Intrusive luxation

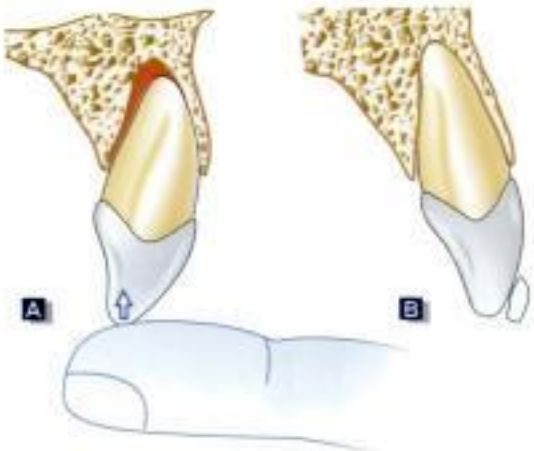


Fig. 21.51: Treatment of extrusive luxation

Fig. 21.53: Damage to periodontium by intrusive luxation

- Tooth presents with clinical presentation of ankylosis because of being firm in socket.
- On percussion metallic sound is heard.
- Intrusion should be diagnosed correctly as difficult as intrusion can mimic a tooth undergoing eruption.
- Radiographic evaluation is needed to know the position of tooth.

Treatment

Healing following the intrusive luxation is complicated because intensive injury to the PDL can lead to replacement resorption and further dentoalveolar ankylosis. Pulp is also affected by this type of injury. So the main objective of treatment is to reduce the extent of these complications. Treatment mainly depends upon stage of root development.

In immature teeth, spontaneous reeruption is seen. If reeruption stops before normal occlusion is attained, orthodontic movement is initiated before tooth gets ankylosed (Fig. 21.54).

Fig. 21.54: Orthodontic extrusion of intruded tooth

If tooth is severely intruded, surgical access is made to the tooth to attach orthodontic appliances and extrude the tooth.

Tooth can also be repositioned by loosening the tooth surgically and aligning it with the adjacent teeth.

Follow-up

Regular clinical and radiographic evaluation is needed in this case because of frequent occurrence of pulpal and periodontal healing complications.

AVULSION (EXARTICULATION)

Definition

It is defined as complete displacement of the tooth out of socket (Fig. 21.55). The common cause is a directed force sufficient to overcome the bond between the affected tooth and the periodontal ligament within the alveolar socket (Fig. 21.56).

Losing a tooth can be physically and emotionally demanding, as a result vacant place is not esthetically agreeable and is difficult to fill and replace. Long-term consequences include shifting of adjacent teeth resulting in misalignment and periodontal disease.

As early as 400 BCE, Hippocrates suggested that dislodged teeth should be replaced and fixed firmly to adjacent teeth with wire. Modern emergency techniques focus on reimplanting the tooth as soon as possible, minimizing periodontal damage, and preventing infection of the pulp tissue.

Incidence

- It usually occurs in age group of 7-10 years.
- 1-16 percent of all traumatic injuries occur to permanent dentition.

Sports, fall from height and automobile accidents are most frequent cause.

Fig. 21.55: Avulsion of tooth

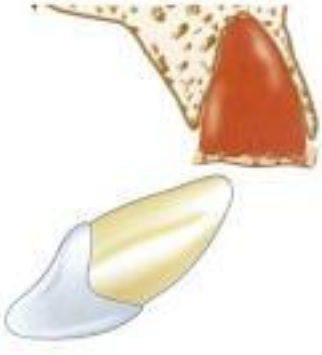


Fig. 21.56: Periodontium of avulsed tooth

the lamina dura and the root assumes a moth-eaten appearance as dentin is replaced by bone. Clinically the tooth will not show any sign of mobility and on percussion it sounds metallic or ankylotic. Replacement resorption in younger patients may interfere with the growth and development of alveolar process which subsequently results in infraocclusion of that tooth.

Biologic Consequences:

- Pulpal necrosis
- Surface resorption
- Inflammatory resorption
- Replacement resorption.

What to Do When a Patient Comes with Avulsed Tooth?

When a patient comes with the avulsed tooth, the main aim of the reimplantation is to preserve the maximal number of periodontal ligament cells which have capability to regenerate and repair the injured root surface. Most important factor in the success of the reimplanted tooth is the speed with which the tooth is reimplanted. The sooner an avulsed tooth is replanted, better is the prognosis. The periodontal ligament cells should be prevented from drying which can result in loss of their normal physiology and morphology. If it is not possible to maintain viable cells of PDL, the aim of the treatment should be directed to slow down the resorption process. If it is not possible to reimplant the tooth immediately, it should be placed in an adequate storage media. Make use of one of the following carrier media in order of preference:

Hank's Balanced Solution (Save-A-Tooth)

This pH-preserving fluid is best used with a trauma reducing suspension apparatus. The HBSS is biocompatible with the tooth periodontal ligament cells and can keep these cells viable for 24 hours because of its ideal pH and osmolality. Researches have shown that this fluid can rejuvenate degenerated ligament cells and maintain a success rate of over 90 percent if an avulsed tooth is soaked in it for 30 minutes prior to replantation.

Milk

Milk has shown to maintain vitality of periodontal ligament cells for 3 hours. Milk is relatively bacteria-free with pH and osmolality compatible with vital cells.

Saline

Saline is isotonic and sterile and thus can be used as tooth carrier solution.

Saliva

Saliva keeps the tooth moist, however, it is not ideal because of incompatible osmolality, pH, and presence of bacteria.

Water: This is the least desirable transport medium because it results in hypotonic rapid cell lysis.

Management Options for an Avulsed Tooth

- ***If the tooth has been out of its socket less than 15 minutes,*** take it by the crown, place it in a tooth-preservation solution (Hank's solution), wash out the socket with the same solution, reimplant the tooth firmly, have the patient bite down firmly on a piece of gauze to help stabilize the tooth and when possible secure it to adjacent teeth with wire, arch bars, or a temporary periodontal pack. Put the patient on a liquid diet, prescribe antibiotics preferably Penicillin VK and plan next dental appointment.
- ***If the tooth has been out 15 minutes to 2 hours,*** soak for 30 minutes to replenish nutrients. Local anesthesia will probably be needed before reimplanting as above.
- If the tooth was out over two hours, the periodontal ligament is dead, and should be removed, along with the pulp. The tooth should soak 30 minutes in 5 percent sodium hypochlorite and 5 minutes each in saturated citric acid, 1 percent stannous fluoride and 5 percent doxycycline before reimplanting. The dead tooth should ankylose into the alveolar bone of the socket like a dental implant.
- ***If the patient is between 6 and 10 years old,*** soak the tooth for 5 minutes in 5 percent doxycycline to kill bacteria which could enter the immature apex and form an abscess.

Precautions to be Taken While Handling the Avulsed Tooth

- Do not touch a viable root with hands, forceps, gauze or anything, or try to scrub or clean it to avoid injury to the periodontal ligament which on further, unable to re-vascularize the re-implanted tooth.
- Do not overlook fractures of teeth and alveolar ridges.
- Do not replace primary teeth, because loss of these teeth early does not hinder development of succedaneous teeth.

When loss of a primary tooth is early, eruption of permanent successors may be delayed.

Reimplanted primary teeth heal by ankylosis. Histologically, dentoalveolar ankylosis is characterized by direct contact between bone and cementum without separation by the periodontal ligament. Ankylosis of deciduous teeth will have the following consequences:

- It will result in cosmetic deformity since the area of ankylosis will not grow at the same rate as the rest of the dentofacial complex.
- Ankylosis can also interfere with the eruption of the permanent tooth.

Contraindications of Replantation

- Compromised medical status of the patient
- Extensive damage to supporting tissues of the tooth
- Child's stages of dental development in which there are chances of ankylosis are more.

Following replantation it should be kept in mind that the tooth should not be in hyperocclusion, because any premature contact can cause delayed recovery. So the necessary grinding of the tooth is done if needed. After evaluating the occlusion, stabilize the tooth if required. Ideal splint required for an avulsed tooth is a flexible splint. The commonly used flexible splints are made of Gortex, metallic mesh stripes, synthetic clothes or orthodontic wire. A thick strip of composite bonded to the avulsed and the adjacent teeth also act as a splint. The splint should engage several teeth around the avulsed tooth and it should be kept in place for not more than 7-10 days.

POST EMERGENCY TREATMENT

- The splint should be removed after 7 days unless the excessive mobility is present.
- Endodontic therapy should be started in 7-10 days except if tooth has an open apex.
- If tooth has closed apex or tooth with an open apex has got the infection, start the root canal treatment at the earliest. At this time, intracanal dressing of calcium hydroxide should be placed for at least four weeks.
- Recall the patient after one month, if radiograph is found to be satisfactory, obturate the tooth with gutta percha points. If lamina dura is not found to be intact or if there is the evidence of external resorption, the calcium hydroxide paste is removed and is replaced with the fresh paste.

ASSESSMENT OF TRAUMATIC INJURIES

Name of the Patient :
Age of the patient :
Sex of the patient :
Date :
Time :

Legal Consequences:

- Delaying reimplantation
- Improper handling and transportation of the tooth
- Reimplanting a primary tooth
- Not providing the tetanus prophylaxis
- Incomplete examination of the surrounding traumatized tissue for tooth fragments
- Failure to warn patients that any trauma to teeth may disrupt the neurovascular supply and lead to long-term pulp necrosis or root resorption.

Patient's History

a. Medical
b. Dental
c. Injury
   i. How injury occurred
   ii. Where injury occurred
   iii. When injury occurred

Check if Present and Describe

- Loss of consciousness
- Orientation to person, place and time
- Hemorrhage/bleeding from nose/ears/oral cavity
- Nausea
- Vomiting
- Headache
- Amnesia
- Spontaneous dental pain
- Pain on medication

Extraoral Examination

- Abrasions/contusions/lacerations/Ecchymosis
- Asymmetry
- Bones
   i. Mobility
   ii. Crepitus
   iii. Tenderness
- Swelling

The mouthguard, also referred to as gumshield or mouth protector is "a resilient device or appliance placed inside the mouth to reduce oral injuries, particularly to teeth and surrounding structures".

Mouthguard can be classified into 3 categories (given by the American Society for Testing and Materials).

1. **Type I**
   - Stock mouthguards are purchased over the counter.
   - Designed to use without any modification.
2. **Type II**
   - Mouth-formed, made from thermoplastic material adapted to the mouth by finger tongue and biting pressure after immersing the appliance in hot water.
   - Commonly used by athletes.
3. **Type III**
   - Custom-fabricated mouthguards.
   - Produced on a dental model by either vacuum forming or heat pressure lamination technique.
   - Should be fabricated for maxillary class I and II occlusions and mandibular class III occlusions.
   - Best in performance.

Functions of Mouthguard

1. Protect the lips and intraoral structures from bruising and laceration.
2. Act as cushion and distribute forces so that crown fractures, root fractures, luxation and avulsions are avoided.
3. Protect jaw from fracture and dislocation of the mandible.
4. Protect against neck injuries.
5. Provide support for edentulous space.
6. Prevent the teeth in opposing arches from violet contract.

22 Pulpal Response to Caries and Dental Procedure

- Introduction
- Pulpal Reaction to Dental Caries
- Response of Pulp to Tooth Preparation
- Response of Pulp to Local Anesthetics
- Effect of Chemical Irritants on Pulp
- Response of Pulp to Restorative Materials
- Effect of Radiations on Pulp
- Effect of Lasers on Pulp
- Defense Mechanism of Pulp
- How the Pulp Recovers

INTRODUCTION

By definition, pulp is a soft tissue of mesenchymal origin residing within the pulp chamber and root canals of teeth.

Embryological studies have shown that pulp is derived from the cephalic neural crest cells. These neural cells arise from the ectoderm along the lateral margins of neural plate and migrate extensively. Before going into details of various pulp responses first we should have a brief idea about the pulp histology.

The dental pulp is a connective tissue consisting of ground substances cells and fibres. Dental pulp can be divided into four **zones:**

1. ***Central Zones:*** Also called pulp proper and form a core of loose connective tissue with larger nerves and blood vessels that distribute to the peripheral area of pulp.
2. ***Cell-rich Zone:*** This zone is the central zone and is composed of fibroblast, undifferentiated mesenchymal cells (Fig. 22.1). This acts as reservoir for the replacement of the injured odontoblasts.

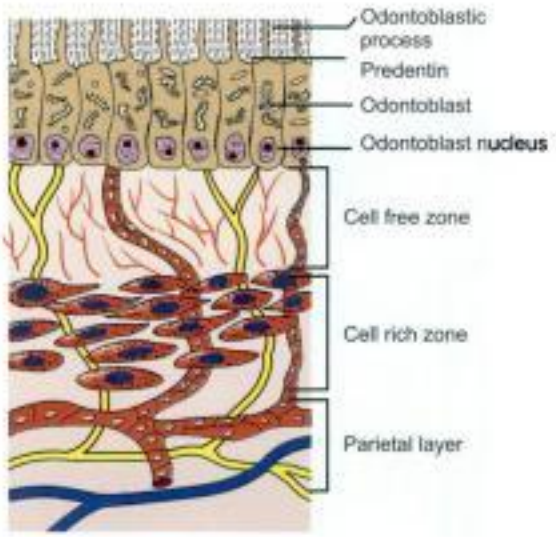


Fig. 22.1: Zones of pulp

3. ***Cell-poor Zone:*** Between the cell rich zone and the odontoblast zone, is a space of round 40 μm in width that is relatively free of cells also called the ***Zone of Weil.*** It contains mainly blood capillaries, unmylinated nerve fibres. It may not be apparent in young pulp or in other teeth where dentin production is rapid.
4. ***Odontoblastic Layer:*** It forms the outer ring of the pulp. It is located immediately beneath the predentin. The odontoblastic processes are embedded in the dentinal tubules and so this layer primarily consists of cell bodies of odontoblasts.

Components of the Pulp

- Nerves
- Vascular tissues
- Fibres
- Odontoblasts
- Fibroblasts
- Ground substance
- Interstitial fluid
- Minor cellular components.

Why Pulp is Unique?

- Enclosed by rigid mineralized dentin so a low compliance environment
- Lacks true collateral blood supply
- Ability to form dentin throughout life
- Potential for regeneration and repair diminishes with age

PULPAL REACTION TO DENTAL CARIES

Dental caries is the most common route for causing irritation to the pulp. Dental caries is localized, progressive, decay of the teeth characterized by demineralization of the tooth

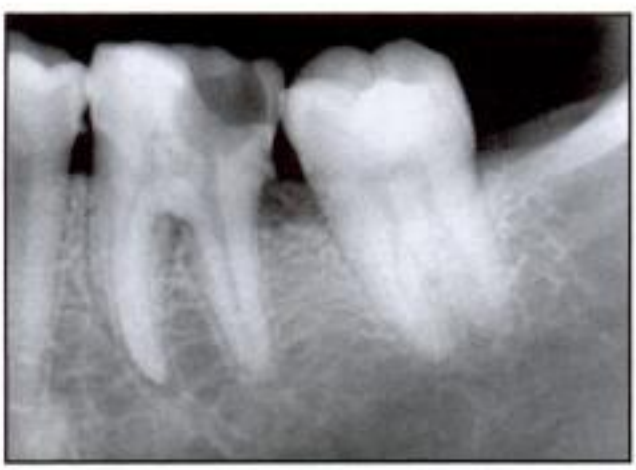


Fig. 22.2: Radiograph showing carious tooth

Fig. 22.3: Formation of irritation dentin in response to caries

surface by organic acids, produced by microorganisms. From the carious lesion, acids and other toxic substances penetrate through the dentinal tubules to reach the pulp (Fig. 22.2).

The following defense reactions take place in a carious tooth to protect the pulp:

1. Formation of reparative dentin.
2. Dentinal sclerosis, i.e. reduction in permeability of dentin by narrowing of dentinal tubules.
3. Inflammatory and immunological reactions.

The rate of reparative dentin formation is related to rate of carious attack. More reparative dentin is formed in response to slow chronic caries than to acute caries. For dentin sclerosis to take place, vital odontoblasts must be present within the tubules. In dentin sclerosis, the dentinal tubules are partially or fully filled with mineral deposits, thus reduce the permeability of dentin and hence act as a barrier for the ingress of bacteria and their product (Fig. 22.3).

INFLAMMATION UNDER CARIES

The bacterial toxins, enzymes, organic acids and the products of tissue destruction show inflammatory response in the pulp. The degree of pulpal inflammation beneath a carious lesion depends on closeness of carious lesions with pulp and permeability of underlying dentin.

The pulp underlying reparative dentin remains relatively normal until the carious process comes close to it. The bacteria are seldom seen in unexposed pulp. When the pulp is exposed, bacteria penetrate the infected dentinal tubule and cause beginning of inflammation of the pulp. The pulp does not become inflamed until the reparative dentin is invaded and wide areas of dentinal tubules are demineralized.

The diagnosis of extent of pulpal inflammation under the carious lesion is difficult. Many factors play an important

role in determining the nature of carious process in hard dental tissues. The rate of carious process is influenced by following factors:

a. Cariogenicity of the diet
b. Composition of the tooth
c. Salivary flow
d. Type of bacterial flora
e. Oral hygiene
f. Age of the person
g. Buffering capacity of the saliva
h. Antibacterial substances present in saliva.

The early evidence of pulpal reaction to caries is seen in underlying odontoblastic layer. There is reduction in number and size of odontoblast cells bodies, change in the shape of odontoblasts, i.e. from tall and columnar to flat and cuboidal before any inflammatory changes seen in pulp. Concomitant with the changes in odontoblastic layer, hyperchromatic line may develop along the pulpal margin of the dentin, which indicates disturbance in normal equilibrium of the odontoblasts. In addition to dentinal changes, antibodies are also produced by the pulp. These antibodies act against the antigenic component of dental caries. Immunoglobulins IgG, IgM, IgA, complement components etc. found in the odontoblasts and adjacent pulp cells are capable of reacting against the invading microorganisms. The presence of bacterial antigens and immunoglobulins emphasize the involvement of specific immunologic reactions during carious process. The persistence of dental caries provides a continuous stimulus for an inflammatory response in dental pulp. The pulp protects itself in many ways like by formation of sclerotic dentin and elaboration of reparative dentin etc.

In acute caries, the caries progress more rapidly than the formation of reparative dentin and chronic inflammatory cells become apparent in the pulp tissue. Initially they are small in number but as the carious lesion comes closer to pulp, more and more plasma cells, macrophages and lymphocytes are seen in pulp. Finally, the pulp gets exposed (Fig. 22.4).

The pulp reacts at site of exposure with infiltration of inflammatory cells. In the region of exposure, small abscess develops consisting of dead inflammatory cells and other cells. The remainder of the pulp may be uninflamed or if the exposure is present for long time, the pulp gets converted into granulation tissue. The chronic inflammation can be partial or complete, depending upon the extent and amount of pulp tissue involved.

As the exposure progresses, partial necrosis of pulp may be followed by total pulp necrosis (Fig. 22.5). The drainage is one of the important factor which determines whether partial or total necrosis of the pulp occurs. If pulp is open to oral fluids, the drainage occurs and apical pulp tissue remains uninflamed. But if the drainage is not possible, entire pulp may become necrotic (Fig. 22.6).

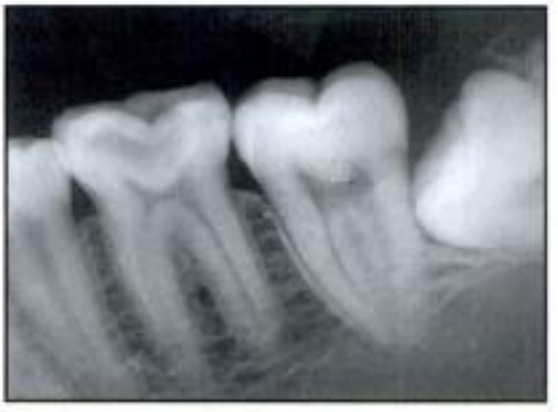


Fig. 22.5: Radiograph showing carious tooth

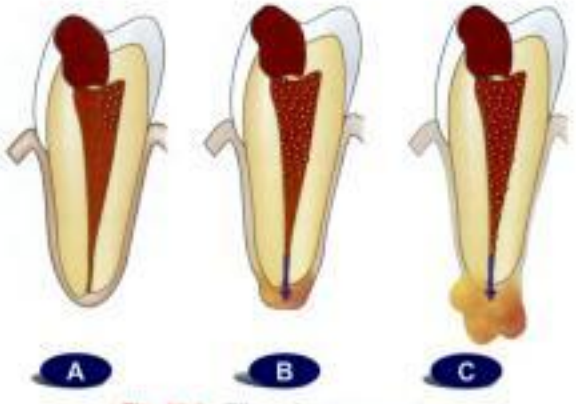


Fig. 22.4 : Effect of caries on pulp

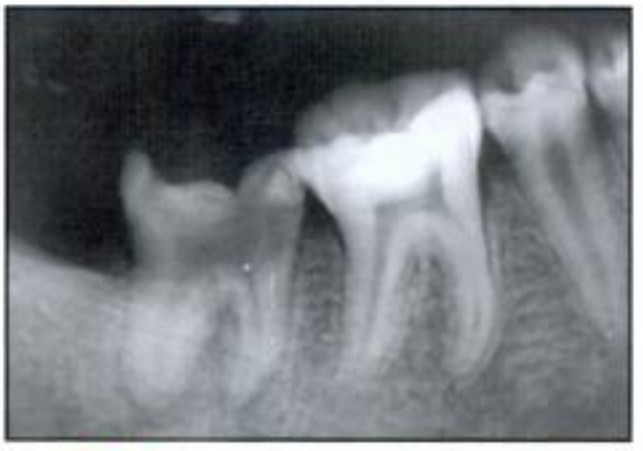
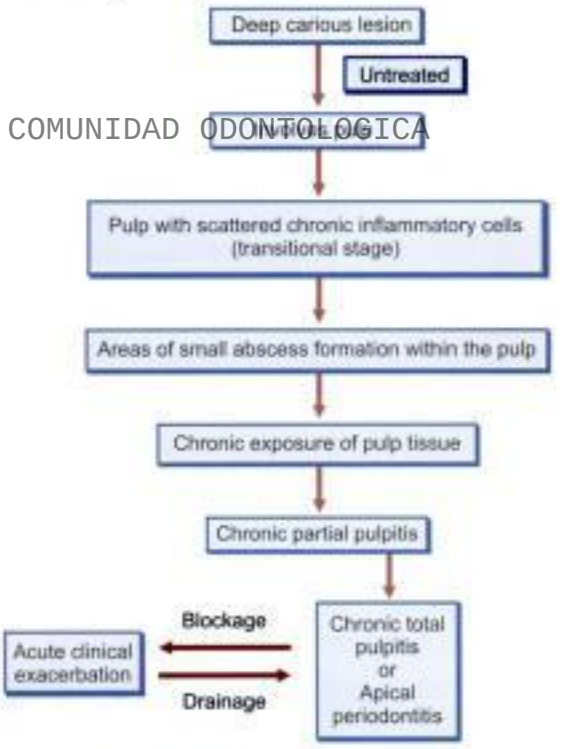


Fig. 22.6: Radiograph showing grossly carious tooth



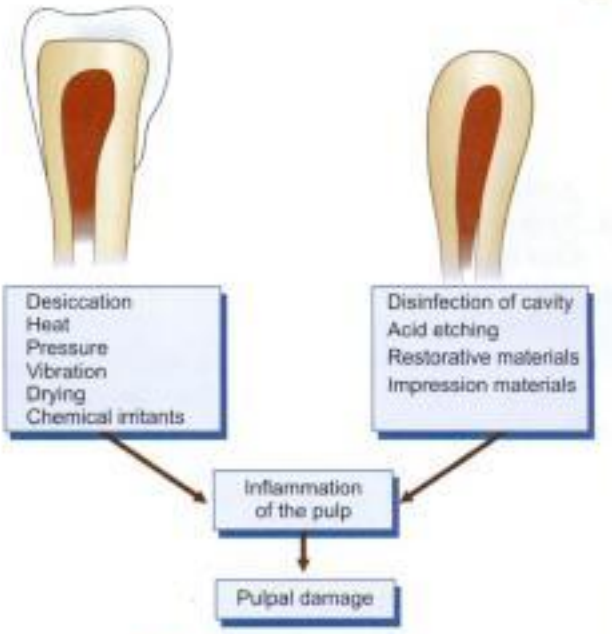


Fig. 22.7: Effect of Irritants on pulp

RESPONSE OF PULP TO TOOTH PREPARATION

The principle aim of operative dentistry is to preserve as much of healthy tissue as possible to enable pulp to respond to future episodes of disease or iatrogenic trauma. Pulpal inflammation resulting from the operative procedures is often termed as **Dentistogenic pulpitis**.

Factors Affecting Response of Pulp to Tooth Preparation:

- Pressure
- Heat
- Vibration
- Remaining dentin thickness
- Thermal and mechanical injury
- Speed
- Nature of cutting instruments.

Irritating Agents of Tooth Preparation

A tooth preparation introduces a number of irritating factors to the pulp (Fig. 22.7). The actual cutting of dentin, in as much as every square mm contains 30,000 to 45,000 dentinal tubules can irritate many millions of odontoblasts.

The ***pressure*** of instrumentation on exposed dentin characteristically causes the aspiration of the nuclei of the odontoblasts or the entire odontoblasts themselves or nerve endings from pulp tissues into the dentinal tubules. This will obviously stimulate odontoblasts, disturb their metabolism and may lead to their complete degeneration and disintegration. This can occur by excessive pressure of hand or rotary instruments, especially in decreased effective depths. Sometimes this pressure may move some microorganisms from infected cavity floor or wall into the pulp, leading to its irritation.

The type of cutting instruments used has variable irritating factors. Sharp hand-cutting instruments in which the energy used with them is completely dissipated in the actual cutting are the most biologically acceptable cutting instruments.

Rotary cutting instruments are also biologically acceptable, if used over effective depths of 2 mm and more, and with proper coolants. This is true with carbide than with steel burs, as the former are more cool-cutting than the latter. Rotary abrasive instruments are not recommended for cutting in vital dentin, as their abrasive action elevates the temperature of surrounding dentin. This is because the energy used to abrade is more than that used to cut. Rotary abrasive instruments may also crush vital dentin much more than any other cutting instruments. Therefore, abrasive actions should be confined to the enamel and the superficial 1 mm of the dentin, as much as possible.

The depth of the cavity is the most disadvantageous exasperating factor to the pulp. Most important is the thickness of the dentin bridge between the floor of the cavity

and the roof of the pulp chamber, also termed as the effective depth. Lesser is the effective depth, more destructive the pulpal response will be:

Heat production is the second most damaging factor. If the pulp temperature is elevated by 11°F, destructive reaction will occur even in a normal, vital periodontal organ. Lesser temperatures can precipitate similar responses in already irritated organs. That *"heat"* is a function of;

a. ***RPM,*** i.e. more the RPM more is the heat production. The most deleterious speed is from 3,000 to 30,000 rpm. In deep penetrations of dentin without using coolants, e.g. pin holes, the cutting speed must not exceed 3,000 rpm.
b. ***Pressure*** is directly proportional to heat generation. Whenever, the RPM's are increased, pressure must be correspondingly reduced. Instrumentation pressure should not be more than four ounce when using high speed and twelve ounce when using low speed.
c. ***Surface area of contact***, which is related to the size and shape of the revolving tool. The more the contact between the tooth structures and revolving tool, the more is the heat generation. Heat creates destruction in the pulp tissues, coagulate protoplasm, and burn dentin if the temperature is amply elevated.
d. ***Desiccation***, if occurring in vital dentin such that water in the protoplasm of Tome's fibers is eliminated, can cause aspiration of the odontoblasts into the tubules (Fig. 22.8). The subsequent disturbances in their metabolism may lead to the complete degeneration of odontoblasts. Desiccation increases the permeability of the vital dentin to irritants like microorganisms or restorative materials. So care must be taken to keep a prepared tooth moist during preparation. If air alone is applied, remove only debris and extra moisture from the operative field, and not the dentin's own moisture. A spray of air and water is satisfactory coolant to dislodge attached debris. Coolant sprays should be used even in non-vital or devitalized tooth structures, since the heat will burn the tooth structures, and these burnt areas will be sequestrated later leaving a space around the restoration where failures can occur.

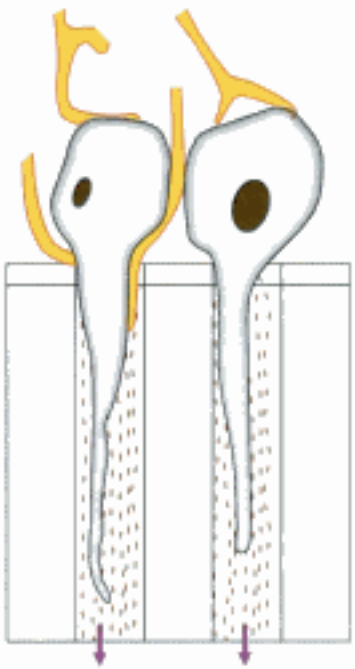


Fig. 22.8: Aspiration of odontoblasts into tubules due to desiccation

Vibrations which are measured by their amplitude or their capacity and frequency (the number / unit time) are an indication of eccentricity in rotary instruments. The higher the amplitude, the more destructive may be the response of the pulp. The reaction is termed as the Rebound Response which is due to the effect of the ultrasonic energy induced. It is characterized by

1. Disruption of the odontoblasts in the opposite side of the pulp chamber from where the cavity is prepared.
2. Edema
3. Fibrosis of pulp tissues proper.
4. Changes in ground substance.

In addition to affecting the pulp tissues, vibration can create microcracks in enamel and dentin. These cracks may transmit and coalesce, directly joining the oral environment with pulp and periodontal tissues. Vibrations also increase the permeability of the dentin and enamel. The extensiveness of preparation or preparation time is directly related to the extensiveness of the reaction of the periodontal organ, for example a crown preparation will cause more response in the pulp than a Class I preparation. Not all persons will have the same reaction towards the same irritant. Also the individual variations in pulpal response can occur that is teeth in the same person can react in a different way towards the identical irritant. This is because of multiple factors like cellularity and vascularity of the pulp, age, heredity, and other unknown factors. The ultimate effect on periodontal organ is due to a cumulative effect of decay, cavity preparation, instrumentation, placement of the restorative materials and finishing procedures. So during a tooth preparation it should always be remembered that the periodontal organ has been already irritated before the

instrumentation and it is going to be more irritated by the restoration procedures.

Factors Affecting the Response of Pulp to Irritants:

- Cellularity of the pulp
- Vascularity of the pulp
- Age
- Heredity
- Unknown factors.

REMAINING DENTIN THICKNESS

Remaining dentin thickness (RDT) between the floor of the cavity preparation and the pulp chamber is one of the most important factor in determining the pulpal response. This measurement differs from the depth of cavity preparation since the pulpal floor in deeper cavities on larger teeth may be far from the pulp than that in shallow cavities on smaller teeth.

Remaining Dentin Thickness (RDT)

- In human teeth dentin is approximately 3 mm thick
- Dentin permeability increases with decreasing RDT
- RDT of 2 mm or more effectively precludes restorative damage to the pulp
- At RDT of 0.75 mm, effects of bacterial invasion are seen
- When RDT is 0.25 mm odontoblastic cell death is seen.

The amount of remaining dentin underneath the cavity preparation plays the most important role in the incidence of a pulp response. Generally, 2 mm of dentin thickness between the floor of the cavity preparation and the pulp will provide an adequate insulting barrier against irritants (Fig. 22.9). As the dentin thickness decreases the pulp response increases. It is seen that response of cutting occurs only in areas beneath freshly cut dentinal tubules not lined with reparative or irregular dentin. In presence of reparative dentin only minimal response will occur.

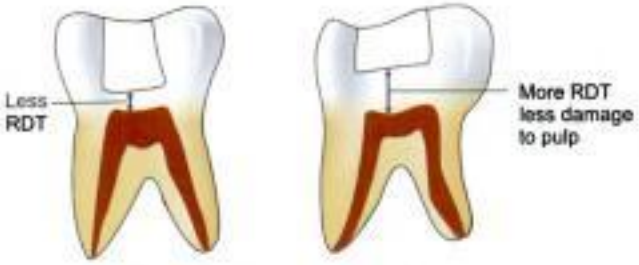


Fig. 22.9: As dentin thickness decreases, the pulp response increases

THERMAL AND MECHANICAL INJURY

Cutting of dentin with bur or stone always produces some amount of heat which is determined by several factors such as:

i. Size and shape of the cutting instrument.
ii. Speed of rotation.
iii. Length of time, the instrument is in contact with dentin.
iv. Amount of pressure exerted.

To reduce the effect of heat on the pulp, proper cooling should be done along with cutting. As the thermal conductivity of dentin is relatively low, there are less chances of injury to the pulp in deep cavity preparation than shallow cavity preparation. Production of heat is most severe stress for pulp. If higher temperature is reached during cutting procedures, there are chances of severe damage of pulp. If damage is extensive, cell rich zone would be destroyed and chances of reparative dentin formation become less.

Blushing of teeth during or after cavity or crown preparation has been seen in teeth after cutting. After dentin is cut, the coronal dentin develops pinkish hue and this hue is due to vascular stasis in the sub-odontoblastic layer.

Speed of Rotation

Ultra high speed should be used for removal of enamel and superficial dentin. A speed of 3,000 to 30,000 rpm without coolant can cause pulpal damage. It should be kept in mind that without the use of coolant there is no safe speed. High speed cutting is disadvantageous when burs are countersunk into the dentin, since water is excluded in a confined region. High speed without coolant can produce burning of dentin, which in turn affect the integrity of the pulp.

Nature of Cutting Instrument

Thermal damage to the pulp was greater with steel burs than with carbide burs, because of greater heat produced by steel burs. Uncooled carbide burs and diamond instruments produce severe damage to the dental pulp. Diamond burs cause most damage to pulp due to its abrasive action and need for increased pressure. Larger size burs cause greater damage due to increased heat generation, cutting of larger area and reduced effectiveness of the coolants. Improper use of handpiece, use of old, broken down and damaged handpiece can cause pulpal damage from eccentric bur rotation and heavy cutting force necessitated by poor torque characteristic.

Basic Rules for Use of Diamond Instruments

- Should be used at high speed
- Apply light pressure
- Coolants should be used
- Use of dragging motion rather than pushing.

Use of Coolants

In deep cavities air blast should not be used to dry the cavity, instead cotton pellets should be used. Air blast can cause desiccation of dentin which can damage the odontoblasts. Water spray is considered as the ideal coolant. Studies have shown that immediate damage was less in water cooled teeth and also repair responses were better when compared with other coolants.

Coolants are Most Effective Method to Reduce the Thermal Damage. Some of the Commonly Used Coolants are:

- Air spray
- Air and Water
- Water
- Water through hollow bur
- Water jet.

Requisites of water coolant:

a. It should have sufficient pressure.
b. It should be directed at bur tooth junction.
c. Ideally water should be delivered from both sides of instrument.

RESPONSE OF PULP TO LOCAL ANESTHETICS

Vasoconstrictors are added to local anesthesia for the purpose of prolonging anesthetic effect by reducing the blood flow in the area in which anesthetics is administered. The most commonly used vasoconstrictor is epinephrine. It has been shown that epinephrine causes decrease in pulpal blood flow. The length of flow cessation and the concentration of vasoconstrictor are directly related to each other. Low oxygen consumption in the pulp helps the healthy pulp to withstand a period of low blood flow when a vasoconstrictor is administered to it. However, the prolonged reduction in oxygen transport could interfere with cellular metabolism and alter response of pulp to injury.

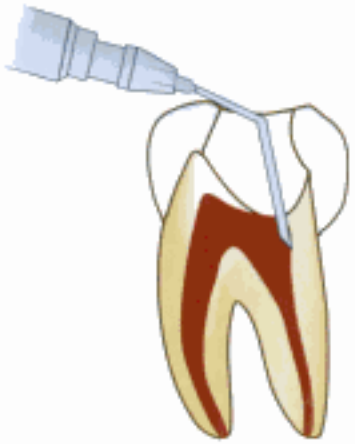


Fig. 22.10: Intrapulpal anesthesia: achieved by injecting the anesthetic into pulp tissue under pressure

Effects of Local Anesthetics on the Pulp

- Vasoconstrictor of local anesthetic (LA) potentiates and prolongs anesthetic effect by reducing blood flow in the area.
- Local anesthetic with epinephrine can significantly decrease pulpal blood flow
- Reduction in blood flow during a restorative procedure could lead to an increase in concentration of irritants accumulating within the pulp.

The intrapulpal anesthesia is achieved by injecting the anesthetic into the pulp tissue under pressure (Fig. 22.10). The resultant anesthesia is attributed to:

- Pharmacologic action of anesthetic on nerve cell membrane
- Circulatory interference from the mechanical pressure of injection.

Though pulp can withstand decrease blood flow but when blood flow is completely arrested or decreased for prolong time, the accumulation of the vasoactive agents occurs into the extracellular compartment of pulp. The accumulation of these substances and other metabolic waste products can cause permanent damage to pulp.

EFFECT OF CHEMICAL IRRITANTS ON PULP

The pulp is subjected frequently to chemical irritation from materials generally used in dentistry. Various filling materials produce some irritation ranging from mild to severe, as do various medicaments used for desensitization or dehydration of the dentin.

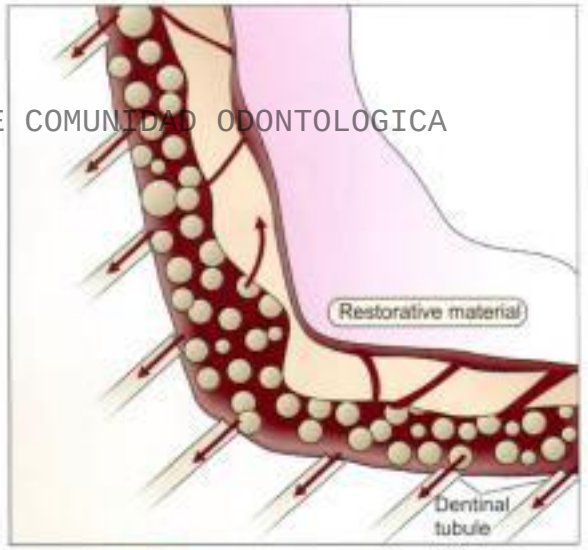


Fig. 22.11: Pathways of marginal leakage

Properties of a material that could cause pulpal injury are its cytotoxic nature, acidity, heat evolved during setting and marginal leakage (Fig. 22.11).

Factors Influencing the Effect of Restorative Materials on Pulp:

- Acidity
- Absorption of water from dentin during setting
- Heat generated during setting
- Poor marginal adaptation leads to bacterial penetration
- Cytotoxicity of material

DENTIN STERILIZING AGENTS

1. ***Silver Nitrate***: Silver salts diffuse rapidly through the dentinal tubules and apart from the depth of the cavity, ultimately reach the pulp tissue, causing an inflammatory reaction in the pulp. Also, the silver nitrate can penetrate into dead tracts, irregular dentin, sclerosed dentin and calcific barriers.
2. ***Phenol***: Phenol is known for its cytotoxic effect though it has been used widely in the past. It has shown by various studies that phenol causes increase in dentin permeability which may further result in greater pulp damage. Its use has declined now because of its poor sterilizing ability and greater irritating qualities.
3. ***Camphorated parachlorophenol and penicillin***: A combination of parachlorophenol and penicillin was found to be an effective sterilizing agent for deep cavities. But studies have shown that this combination produces the pulpal inflammation. Also the local use of penicillin is not advocated because of its potential for sensitization of the patient.

CAVITY LINER AND VARNISHES

The use of cavity liner is advocated under restorative material to reduce the sensitivity of freshly cut dentin and to protect pulp (Fig. 22.12). Unlined cavities restored with composite resins have been shown to contain dense accumulation of bacteria, probably from contraction of the restorations. Toxic products from such bacteria may be instrumental in causing inflammation of the pulp.

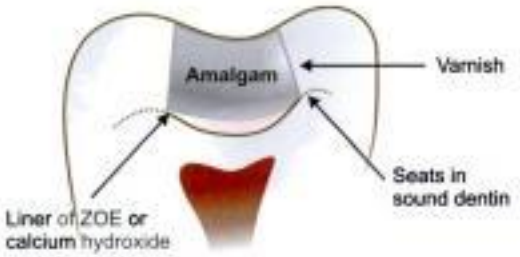


Fig. 22.12: Use of liner and varnish under restoration

RESPONSE OF PULP TO RESTORATIVE MATERIALS

Calcium Hydroxide

Calcium hydroxide has been used in dentistry for past many years because of its following features:

1. It causes dentin mineralization by activating the enzyme ATPase.
2. It stimulates proliferation of pulp fibroblasts.
3. It stimulates reparative dentin formation.
4. It forms a mechanical barrier, when applied to dentin.
5. Because of high pH, it neutralizes acidity of silicate and zinc phosphate cements.
6. Calcium hydroxide dissociates into Ca^{2+} and OH^- ions, the OH^- ions neutralize the (H^+) hydrogen ions from acids of cement.

In extremely deep cavities with microscopic exposure, it is recommended that calcium hydroxide should be applied followed by zinc oxide eugenol or zinc phosphate under the restoration (Fig. 22.13). But, when pulp exposure does not exist, zinc oxide eugenol is preferred over calcium hydroxide because of its least irritating, most palliative and anodyne effect. Calcium hydroxide does not significantly depress the nerve impulse activity and thus should not be used to treat painful pulpitis.

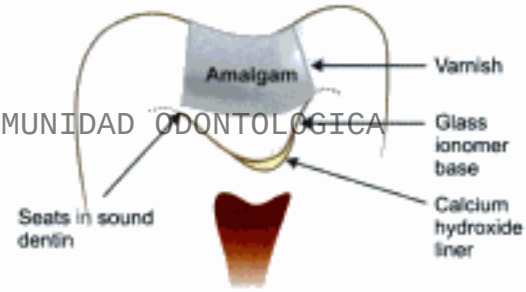


Fig. 22.13: Use of base to protect pulp

Zinc Oxide Eugenol

Zinc oxide eugenol is temporary filling material that is also used for provisional and permanent cementation of crowns, bridges, inlays and as liner and base. Of all the filling materials, it has always been considered the safest from biological aspect. Zinc oxide eugenol has been used as an anodyne for pulpal pain. The sedative effects are apparently because of ability of eugenol to block or reduce nerve impulse activity. This effect is obtained only when a reasonably thin mix of ZOE is used. Another advantage of ZOE is that there is no heat rise during setting.

Its disadvantages as a temporary filling material are:

a. Its softness,
b. Long setting time
c. The ease with which it may be displaced by biting stress before setting.

Zinc Phosphate

Zinc phosphate cement can cause severe pulpal damage because of its irritating properties. Toxicity is more pronounced when the cement is placed in deep cavity preparations. In deep cavities, zinc phosphate cement should not be used without an intervening liner of zinc oxide eugenol or calcium hydroxide. Thick mixes should be used to minimize pulp irritation and marginal leakage. The pulp may be affected by the components of the material, the heat that is liberated in setting, and the marginal leakage that permits the ingress of irritants from saliva. The pulpal injury from the cement is mainly due to marginal leakage rather than to its toxic chemical properties.

Zinc Phosphate: Effect of zinc phosphate on pulp are due to:
- Components of zinc phosphate
- Acidic nature
- Heat produced during setting
- Marginal leakage.

Routes of Microleakage
- Within or via the smear layer
- Between the smear layer and the cavity varnish / cement
- Between the cavity varnish/cement and restorative material

Zinc Polycarboxylate Cements

Zinc Polycarboxylate Cement contains modified zinc oxide powder and an aqueous solution of polyacrylic acid. It chemically bonds to enamel and dentin and has antibacterial properties. Polycarboxylate cement is well tolerated by the pulp, being roughly equivalent to Zinc Oxide Eugenol cements in this respect.

Glass Ionomer Cement

It possess anticariogenic properties and is well tolerated by the pulp. Toxicity diminishes with setting time. Its pH at mixing is 2.33 and after 24 hours it is 5.67.

Amalgam

Amalgam has been used in dentistry since ages. It is considered one of the safest filling materials with least irritating properties. Even if varnish is not employed, within a period of few weeks, marginal seal develops between the tooth and the restoration due to its corrosion products. It has been shown to produce discomfort due to its high thermal conductivity. So liners or bases are necessary to provide thermal insulation.

Effects of Amalgam on Pulp:
- Mild to moderate inflammation in deep caries
- Harmful effects due to corrosion products
- Inhibition of reparative dentin formation due to damage to odontoblasts
- Copper in high copper alloy is toxic
- High mercury content exerts cytotoxic effects on pulp
- Post-operative thermal sensitivity due to high thermal conductivity.

Precautions to be taken while using Amalgam as a Restorative Material

1. Use of cavity liner or base under the silver-amalgam restoration (Fig. 22.13).
2. Use of varnish restoration and at the margins (Fig. 22.14).

Fig. 22.14: Use of varnish beneath amalgam restoration

Post-operative sensitivity of amalgam occurs because of expansion and contraction of fluid present in the gap between amalgam and the cavity wall. This fluid communicates with fluid in subjacent dentinal tubule. Any variation in temperature will cause axial movement of fluid in the tubules which further stimulates the nerve fibres, thus causing pain.

RESTORATIVE RESINS

Restorative resins have been used in dentistry for past many years. Despite of having several advantages, they are not considered best materials because of their high coefficient of thermal expansion and polymerization shrinkage, which results in marginal leakage, subsequently the recurrent caries and ultimately the pulp damage. Monomer present in composite resins also acts as an irritant to the pulp. Though marginal seal can be improved by acid etching of beveled enamel and the use of bonding agent or primer but studies have shown that initial marginal leakage tends to deteriorate as the etched composite restoration ages.

Newer composite materials, filler systems, catalysts and methods of curing, have shown improvement in polymerization characteristics and lower coefficient of thermal expansion but still many researches have shown that all composite resins irritate pulp though to different degrees. Some have been found more irritating than the others . It has been seen that unlined composite resins are harmful to the pulp because of bacterial contamination beneath the restoration so the use of cavity liner is advocated under composite restoration (Fig. 22.15).

Liners containing calcium hydroxide have shown to provide good protection against bacteria. Zinc oxide eugenol liners should not be used with composite resins since they interfere with polymerization of composites.

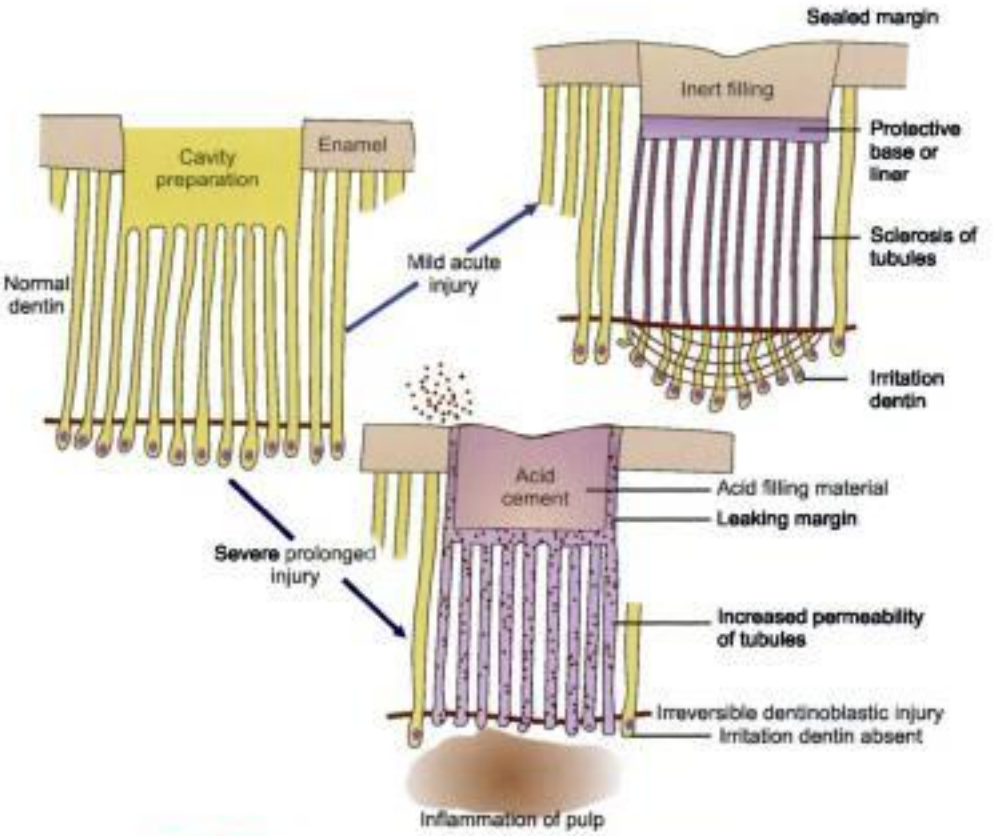


Fig. 22.15: Response of pulpodentinal complex to mild and severe injury

Acid Etchants

Acid etching is an important step in the placement of composite restorations. Commonly used acid etchant is 37 percent phosphoric acid. It has been shown that acid etching does not cause pulpal injury. Etching results in opening of the dentinal tubules and increases dentin permeability. On the contrary acid etching also enhances bacterial penetration of dentinal tubules.

EFFECTS OF PIN INSERTION

Pins are used in amalgam restoration for building up badly broken tooth or to support amalgam restoration. The insertion of pin results in:

i. Dentinal fractures and unnoticed pulp exposure (Fig. 22.16).
ii. Increases the pulp irritation of already stressed pulp (inflammation of pulp directly proportioned to depth, extensiveness of decay).
iii. Cements used for pins add more irritation to the pulp.

To reduce continued irritation from the pins, use of calcium hydroxide is recommended.

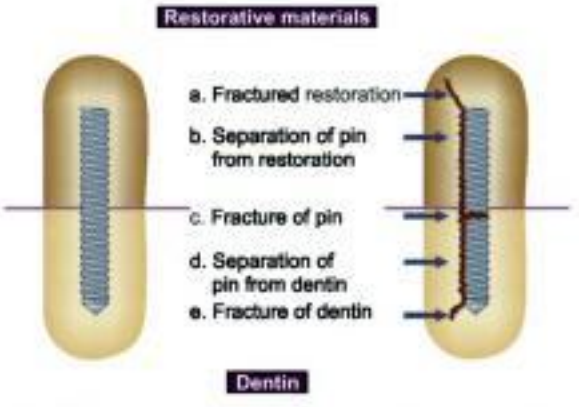


Fig. 22.16: Five possible locations of pin retained restorations

IMPRESSION MATERIAL

The taking of impressions for inlay and crown fabrication also exposes the pulp to serious hazards. Seltzer et al showed that pulpal trauma can occur when more pressure is applied while taking impression. When the modeling compound is applied to the cavity or full crown preparation, a pressure is exerted on pulp. Also the negative pressure created in removing an impression may cause odontoblastic aspiration.

EFFECTS OF RADIATIONS ON PULP

The basic cellular effect of ionizing radiation is interference with cell division. Radiation damage to teeth depends on dose, source, and type of radiation, exposure factors, and stage of tooth development at the time of irradiation. In developing human teeth, the extent of damage depends on the amount of radiation and the stage of tooth development at the time of irradiation. Heavy doses at the earliest stage of development can cause complete failure of the tooth to develop; mild doses can result in root end distortions and dilacerations. Circulatory disturbances in the tooth germ are also manifested by the presence of dilated vessels, hemorrhages and endothelial cells swelling. The odontoblasts fail to function normally and may elaborate abnormal dentin, and amelogenesis is retarded or ceases. In the later stages, fibrosis or atrophy of the pulp may occur. The pulps of fully formed human teeth may be affected in patients who are exposed to radiation therapy. Relative dosage, mature odontoblasts appear to be extremely radio-resistant. However, in time, the pulp cells exposed to the ionizing radiation may become necrotic. The effects appear to be related to vascular damage and interference with mitosis of cells. The salivary glands are also affected.

Effects of Radiations:

- Interference with cell division
- Mild dose can cause root end distortions and dilacerations
- Heavy doses at the earliest stage of development can cause complete failure of tooth to develop
- Abnormal dentin formation is seen in some cases
- Retardation or cessation of enamel formation
- Fibrosis or atrophy of the pulp
- Salivary glands are also affected.

EFFECT OF HEAT FROM ELECTROSURGERY

Heat may be delivered to the pulp by electrosurgical gingivoplasty. In a study it was seen that when the electrode tip contacted Class V amalgam restoration electrosurgical current was delivered for not more than one second with a fully rectified unit, the pulps became severely damaged. But no damage to the pulp was noticed from the application of current to unrestored teeth. The contact of the activated electrode with the gingival restorations to no more than 0.2 to 0.4 seconds would be more compatible with clinical usage. However, longer periods of exposure to the electrosurgical currents produced severe pulpal damage. Even the placement of the calcium hydroxide base, covered

23 Pharmacology in Endodontics

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- Infection Control
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- Guidlines for Antibiotic Prophylaxis
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INTRODUCTION

In field of endodontics, dentist commonly faces the problems regarding pain, anxiety and infection. Accordingly clinician has to be well prepared to use drugs, which help to solve these problems. Pain and anxiety though are entirely different problems, yet they are closely related. In endodontics, pain control often proves to be more of a difficult problem than management of anxiety, but because of this difficulty in achieving effective pain control, patients often anticipate the experience with great deal of anxiety.

Pain management is an integral part of endodontics. Pain from a pulpitis or apical periodontitis that produces the need for endodontic therapy may be unbearable without analgesic. Also a similar degree of pain may follow appointments in which canals are enlarged or filled, particularly when the apical constriction is violated or following a surgical procedure. Analgesics may be necessary before an emergency appointment. Practice of endodontics requires a thorough understanding of pain mechanisms and management. In addition to pharmacological effectiveness of drugs many people have a positive psychological reaction to the prescription.

Because of common belief that root canal treatment is very complex in nature and there may be severe pain as nerve removal will take place from root canal, patient may suffer from mild to severe apprehension. Also, dental incident or the stories told by other persons about their dental experience causes further dental fear in patient's mind. When this reaches a degree that produced alteration of physiologic and psychological functions, preoperative sedation is required usually in the form of sedatives or tranquilizers. In the present day pharmacological drugs are available that work directly against anxiety without much of side effects, making dental treatment easier for the patient and the practitioner, enabling more patients to receive optimal dental care.

ANXIETY CONTROL

Certain patients enter the office in such a state of nervousness or agitation that they even find taking of

radiographs almost unbearable. Some of them who outwardly appear normal may also be suffering from severe inner apprehension. A kind, supportive and understanding attitude together with suggestion for control of such feelings will be greatly appreciated and usually yield acceptable response.

When an apprehensive patient is rested in the dental chair, clinician should try to recognize the cause of anxiety. Doctor should spend some time at each visit speaking with the patient, anxiety may be expressed at that time.

A variety of techniques for management of anxiety are available. Together these techniques are termed as spectrum of pain and anxiety control. They represent a wide range from non drug technique to general anesthesia.

On the whole there are two major types of sedation, first, requiring administration of the drugs (pharmacosedation) and second , requiring no administration of drugs (latrosedation).

Pharmacosedation

Sedatives and tranquilizers are drugs that are CNS depressants and decrease cortical excitability. Both have similar actions reducing abnormal and excessive response to environmental situations that produce agitation, tension and anxiety.

Sedative is a drug that subdues excitement and calms the subject without inducing sleep but drowsiness may be produced. Sedation refers to decreased responsiveness to any level of stimulation and is associated with some decrease in motor activity and sedation. At higher doses sleep may occur.

Tranquilizers do not produce sleep and serves effectively to block intolerant and overly aggressive reactions. With tranquilizer use, no attendant is required to the clinic as driving is not contraindicated with tranquilizers. It also eliminates more objectionable types of patient defense reactions and produce acceptable relaxation. Ambulatory patient for whom sedatives are prescribed must be warned against driving at the completion of appointment. Tranquilizer themselves do not produce sleep but may relax the patient to such a degree that extreme drowsiness will develop. This is particularly true where stress or lack of sleep is caused by some emotional factors. Therefore, sedatives and tranquilizers both should be prescribed in lower dosages for patient without prior experience of ingestion until some indication as to the degree of relaxation produced by a given dose is determined.

Short acting barbiturates and their substitutes are excellent for use with endodontic therapy. Initial dose should be given the night before the appointment to ensure restful night, with another taken 30 min before the patient is seated for the treatment. Patients requiring sedatives should be seen in the morning.

Barbiturates

Barbiturates depress all areas of CNS but reticular activating system is most sensitive, its depression is primarily responsible for inability to maintain wakefulness. Barbiturates don't have selective antianxiety action. They can impair learning, short term memory and judgment.

Short acting barbiturates are used in endodontics which are:

a. Butobarbitone
b. Secobarbitone
c. Pentobarbitone

Barbiturates are well absorbed from gastrointestinal tract. The rate and entry of the drug in CNS depends on lipid solubility.

Drugs	Dosage
A. Pentobarbital (Nembutal)	a. For relaxed patient → 30 mg night before appointment and 30-60 mg 30 min. before appointment b. For heavier sedation → 30 mg night before appointment and 90 mg 30 min before appointment.
B. Secobarbital (Seconal)	50 mg night before appointment and 50 mg before appointment
C. Ethinamate (Valmid)	1-2 tab night before appointment and 1 tab. 20-30 min before appointment.

Contraindications of use of Barbiturates

1. Acute Intermittent porphyria → By inducing microsomal enzyme (8 aminolevulinic acid synthetase) and increases porphyrin synthesis.
2. Liver and kidney disease.
3. Severe pulmonary insufficiency.
4. Obstructive sleep aponea.

Benzodiazepines

Antianxiety benzodiazepines are:

- Diazepam
- Chlordiazepoxides
- Oxazepam
- Lorazepam
- Alprazolam

Benzodiazepines have a high therapeutic index. Benzodiazepines acts preferentially on midbrain ascending reticular formation and on limbic system. Muscle relaxation is produced by a primary central site of action.

Benzodiazepines have different oral bioavailability and intramuscular bioavailability. It depends on lipid solubility and plasma protein binding capacity.

Diazepam

The active metabolites of diazepam are desmethyl diazepam and oxazepam.

With prolonged use, its accumulation occurs which results in anxiolytic effects. Its withdrawal phenomena are mild.

Doses

Oral Route – 5, 10 mg tab. night before appointment and 1 tab. before appointment.
i.m. Route – 10 mg/ 2 ml syringe before appointment.

Triazolam

Half life of about 3 hours, so popular for dental procedures.

- Very potent, peak effect occurs in 1 hour
- Does not accumulate on repeated use
- Higher doses can alter sleep architecture, produce anta grade amnesia and anxiety the following day.

Midazolam

- Extremely rapid absorption.
- Peak in 20 min.
- Also used as an IV anesthetic.

Chlordiazepoxide

5 mg capsule therapy to start day before appointment, 1 capsule 3 times daily and 1 capsule morning of appointment.

Iatrosedation

It is a non-drug technique of causing sedation. A relaxed and pleasant doctor patient relationship has favorable influence on action of the sedative drugs. A patient, who is comfortable with doctor, responds well to the drugs than to patient who are anxious about the doctor and treatment to be done. An effective effort should be done by all the members of dental clinic to help allay the anxiety of patients.

PAIN CONTROL

Odontogenic pain is usually caused by either noxious physical stimuli or the release of inflammatory mediators that stimulate receptors located on terminal endings of nociceptive afferent nerve fibers.

There are two type of nociceptors:

a. C – fibers
b. A – delta fibers

In human dental pulp C fibers are found 8–10 times more than A – delta fibers. There are unmyelinated fibers. C fibers are thought to have a predominant role for encoding inflammatory pain arising from dental pulp and periradicular tissues. After activation C and A–delta fibers transmit nociceptive signals primarily via trigeminal nerves to the trigeminal nucleus caudalis located in Medulla. Hyperalgesia is caused by both peripheral and central mechanisms.

Pain control in endodontics though is not very difficult but sometimes it becomes almost impossible to control pain. In endodontics procedures the control of pain becomes difficult mostly at the first appointment. Once the pulp has been extirpated, the pain comes under control. But before initiating the procedure, to avoid discomfort or pain and for soft tissue anesthesia for rubber dam application, local anesthesia injection is needed.

Analgesic can be taken either while local anesthesia is still effective or if no anesthesia was used, as soon as possible after treatment. When a local anesthesia is used and analgesic is prescribed the dentist must calculate the anticipated duration of remaining anesthesia effectiveness. The analgesic should be ingested 30-60 minutes prior to the wearing off of anesthetic so the drug will have sufficient opportunity to take effect. Highly nervous or fidgety patients may metabolize rapidly, and thus the anesthesia lasts for less than the anticipated duration.

Pain control can be achieved through:

- Opioid drugs
- Non-opioid drugs.

Opioid Drugs

Generally narcotic (opioid) analgesics are used to relieve acute, severe pain and slight to moderate pain. Clinician's therapeutic judgment determines which analgesic should be prescribed. He must decide the strength of the drug, whether it is to be used alone or in compound form, the frequency of use and side effects associated with it. The drugs used most often are the mild, non-opioid analgesics.

The opioid receptors are located at several important sites in brain, and their activation inhibits the transmission of nociceptive signals from trigeminal nucleus to higher brain centers. Opioids also activate peripheral opioid receptors.

Classification

a. Natural opium alkaloids:
   - Morphine
   - Codeine

b. Semisynthetic opiates:
   - Diacetyl morphine (Heroin)
   - Pholcodeine

c. Synthetic opioids:
   - Pethidine (Meperidine)
   - Fentanyl
   - Methadone
   - Dextro propoxyphene
   - Tramadol
   - Etoheptazine

Others – Alfentanil, Sufentanil, Lenorphanel, Dextromoranride.
Generally Codeine, Morphine, Tramadol, Propoxyphene, Hydrocodeine and Oxycodeine are used in endodontic pain management.

Codeine

- It is methyl morphine
- Occurs naturally in opium, partly converted into morphine in body
- Good activity by oral route
- It is less potent than morphine and also less efficacious. Codeine is 1/6 to 1/10 as analgesic to morphine
- Comparative to aspirin it is more potent.
  60 mg Codeine ≥ 600 mg Aspirin.
  Side effects – Constipation.

Morphine

- It has site specific depressant and stimulant actions in CNS
- Degree of analgesia increases with dose
- Dull poorly localized visceral pain is relieved better than sharply defined somatic pain
- Calming effects on mood, inability to concentrate
- Depress respiratory centers, death in poisoning is due to respiratory failure
- Oral bioavailability averages ¼ of parenterally administered drug
- About 30% bound to plasma protein and high first pass metabolism present
- Plasma t½ → 2 – 3 hours
- Morphine is non cumulative.

Doses – 10 – 15 mg I.M. or s.c.

Side Effects

- Sedation
- Constipation
- Respiratory depression
- Blurring of vision
- Urinary retention
- Nausea and vomiting
- Blood pressure fall in hypovoluemic patients
- Urticaria, itching and swelling of lips

Antidote – Naloxone 0.4 – 0.8 mg IV repeated every 2 – 3 min till respiration picks up; used in acute morphine poisoning.

Dextropropoxyphene

- Half as potent as codeine
- Has a low oral : parenteral activity ratio
- t½ → 4 – 12 hours
- Abuse liability is lower than codeine
- Mild oral analgesic
- Combination with aspirin and paracetamol is supraadditive

Doses—60 – 120 mg three times a day.

Tramadol

- Centrally acting analgesic; relieves pain by opioid as well as additional mechanism
- Injected IV 100 mg tramadol is equianalgesic to 10 mg morphine
- Oral bioavailability is good (oral : parentral ratio is 1:2)
- t½ is 3 – 5 hours, effects last 4 – 6 hours
- Indicated for medium intensity short lasting pain due to diagnostic procedures, injury, surgery etc, as well as for chronic pain including cancer pain
- Abuse potential is low.

Side effects— Dizziness, nausea, sleepiness, dry mouth, sweating

- It inhibits reuptake of serotonin and norepinephine, a monoamine, hence, concomitant administration with monoamine oxidase inhibitor drugs is not recommended.
- Narcotics can cause addiction with characteristics unique from other types of addiction. Both physical and psychological addiction occurs. Narcotics are central nervous system, depressants and work synergistically with all other CNS depressants. Alcohol is contra-indicated with narcotics. Narcotics patient must not drive or operate machinery. Narcotics are combined with acetaminophen or aspirin or an NSAID to make them more effective without excessive narcotic side effects.

Non-opioid Drug

- Weaker analgesic
- They act primarily on peripheral pain mechanism also in CNS to raise pain threshold.

Non-opioid analgesics interfere with membrane phospholipid metabolism. Mild analgesics interfere with cyclooxygenase pathway and reduce synthesis of prostaglandin and result is the reduction or elimination of pain. More frequently used non-narcotic analgesics are aspirin, acetaminophen, diflunisal, naproxen and ibuprofen, etc.

Classification:

a. *Analgesic and anti-inflammatory*
   1. Salicylates → Aspirin
   2. Pyrazolone derivatives → Phenylbutazone, Oxyphenbutazone
3. Indole derivatives → Indomethacin
4. Propionic acid derivatives → Ibuprofen, Naproxen
5. Anthranilic acid derivatives → Mephenamic acid
6. Aryl-acetic acid derivatives → Diclofenac
7. Oxicam derivatives → Piroxicam, Meloxicam
8. Pyrrolo-pyrole derivative → Ketorolac
9. Sulfonanilide derivative → Nimesulide
10. Alkanones → Nabumetone.

b. *Analgesic but poor anti-inflammatory:*
   1. Paraaminophenol derivatives → Paracetamol (Acetaminophen)
   2. Pyrazolone derivative → Metamizol
   3. Benzoxazocine derivative → Nefopam.

Selective COX-2 Inhibitors:

- Rofecoxib
- Celecoxib
- Etocoxib, etc.

NSAIDS are very effective for managing pain of inflammatory origin and by virtue of binding to plasma proteins actually exhibits increased delivery to inflamed tissue.

Aspirin

- Rapidly converted in the body to salicylic acid which is responsible for most of actions
- Aspirin inhibits COX irreversibly. Return of COX activity depends on synthesis of fresh enzymes
- Analgesic action is mainly due to obtunding of peripheral pain receptors and prevention of prostaglandin mediated sensitization of nerve endings
- It has antipyretic action by promoting heat loss
- Absorbed from stomach and small intestines
- Plasma t½ = 15-20 min
  t½ of anti-inflammatory dose – 8-12 hours.
- Analgesic dose – 600 mg three times a day
- Anti-inflammatory dose – 3-6 g / day or 100 mg / kg / day.

Side Effects

- Gastric upset
- Irreversibly inhibits TXA_2 synthesis by platelets thus it interferes with platelet aggregation and prolong bleeding time
- Hypersensitivity and Idiosyncrasy.

Contraindications

- Peptic ulcer patient
- Bleeding disorders
- Chronic liver disease
- Pregnancy.

Precaution

Aspirin should be stopped 1 week before elective surgery.

Aspirin can be buffered with chemicals such as magnesium, calcium or aluminum compounds to decrease stomach complaint.

Ibuprofen

- Better tolerated alternative to aspirin
- Side effects are milder than aspirin
- Gastric discomfort, nausea and vomiting are less than aspirin.

Contraindications

- Pregnancy
- Peptic ulcer

Dose—400–800 mg TDS.

Piroxicam

- It is a long acting potent NSAID
- Rapidly and completely absorbed, 99 percent plasma protein bound
- Plasma t½ → 2 days
- Suitable for use as short term analgesic as well as long term anti-inflammatory drug
- Single daily administration is sufficient.

Dose—20 mg BD

Diclofenac Sodium

- Well absorbed orally
- Plasma t½ → 2 hours
- Epigastric pain, nausea, headache, dizziness, rashes are side effects
- Gastric ulceration and bleeding are less common
- Used in postoperative inflammatory condition.

Dose—50 mg eight hourly

Nimesulides

- Selective COX-2 inhibitor
- Weak inhibitory action on prostaglandin synthesis
- Used primarily for short lasting painful inflammatory conditions like sports injury, sinusitis, dental surgery, post operative pain
- Almost completely absorbed orally

Dose → 100 mg BD.

PARA AMINO PHENOL DERIVATIVE (PARACETAMOL OR ACETAMINOPHEN)

- Central analgesic action, it raises pain threshold
- Weak peripheral anti-inflammatory component
- Poor ability to inhibit COX in the presence of peroxides which are generated at site of inflammation
- Well absorbed orally
- Plasma t½ →2-3 hours
- Paracetamol is one of the most commonly used "over the counter" analgesia where anti-inflammatory action is not required
- One of the best drugs to be used as antipyretic
- Much safer analgesic

Dose – 0.5-1 mg TDS

- Should be used cautiously in patients with liver disease or chronic alcohol use.

Choice of NSAIDs

a. Mild to Moderate pain with little inflammation – Paracetamol or low dose ibuprofen.
b. Acute musculoskeletal / injury associated inflammation – Diclofenac or piroxicam, ibuprofen.
c. Short lasting painful condition with minimal inflammation – Ketorolac, nefopam.
d. Exacerbation of acute pain – High dose aspirin, indomethacin, piroxicam.
e. Severe pain → Aspirin, or combination with narcotic drugs.

INFECTION CONTROL

In the usual picture, pulpal invasion begins with the mixed infection of aerobes and anaerobes. As the infection increases, flora changes to obligate anaerobes and facultative organisms because of oxygen depletion. One of the primary goals of endodontic therapy is to eliminate a habitat of microorganisms in canal space. Thus thorough sterilization is needed starting from the pulpal debridement up to the step of obturation. It has been seen many times that chronic infection persists in periapical area, following root canal therapy. Studies have shown presence of bacteria in chronic asymptomatic periapical radiolucent lesions. Though antibiotics are considered as tool for fighting infection, endodontic therapy mainly emphasizes the importance of thorough debridement of canal apace.

When drainage from root canal system becomes difficult to obtain or when host resistance is low or when virulence of attacker is high, antibiotics are needed.

ANTIBIOTICS

These drugs attack cell structure and metabolic paths, unique to bacteria and not shared with the human cells. Since ***Flemming*** produced the first antibiotic over 60 years ago, dealing with infection, has changed.

Definition

These are substances which are produced by micro-organisms, suppress or kill other microorganisms at very low concentrations.

These drugs attack cell structure and metabolic pathways of bacteria but not the human cells. Nowadays, oral and systemic antibiotics are most frequently used so, the thorough understanding about their pharmacologic profile is necessary. In this topic, we will discus the indications, uses and side effects of most commonly antibiotics.

Classification of Antibiotics

Classification

I. Based on spectrum of activity
   a. Narrow spectrum
      i. Penicillin G
      ii. Streptomycin
      iii. Erythromycin
   b. Broad spectrum
      i. Tetracyclines
      ii. Chloramphenicol

II. Type of action
   1. Bactericidal
      a. Penicillin and cephalosporins
      b. Metronidazole
      c. Fluoroquinolone
         i. Nalidixic acid
         ii. Ciprofloxacin
         iii. Ofloxacin
         iv. Sparfloxacin
         v. Gatifloxacin
      d. Aminoglycosides
         i. Streptomycin
         ii. Amikacin
   2. Primarily bacteriostatic
      a. Sulfonamides
      b. Tetracycline
      c. Clindamycin
      d. Erythromycin

III. On the basis of family
   Pencillins
   Cephalosporins
   Sulfonamides
   Tetracyclines
   Aminoglycosides
   Macrolides

Factors Determining the Efficiency of Antimicrobial Agents

- Host defense
- Source of infection
- Tissue affected
- Margin of safety
- Bacterial susceptibility / resistance to agent being used.

Mechanism of Action of Antimicrobial Agents

Action	Antimicrobial Agents
1. Inhibition of cell wall synthesis	Pencillins, cephalosporins vancomycin
2. Inhibition of protein synthesis	Aminoglycosides, tetracycline chloramphenicol, lincomycin
3. Interference in transcription/translation of genetic information	Ciprofloxacin, ofloxacin gatifloxacin, metronidazole
4. Antimetabolite actions	Sulfonamides, trimethoprim

FACTORS AFFECTING SELECTION OF ANTIBIOTICS

Selection of Antibiotic

Selection of antibiotic depends upon several factors like:

- Clinical diagnosis
- Identification of causative organism
- Age
- Severity of the disease
- Drug resistance and toxicity
- Drug allergic reactions
- Cost of the therapy

- *Clinical diagnosis*: A clinical diagnosis should be made before initiating antimicrobial therapy.
- *Age of the patient*: Drug should be given according to age of the patient for example-Chloramphenicol is not given to infants.
- *Severity of the disease*: In general, antimicrobial therapy should be considered for patients with established orofacial infections. In mixed infections, a combination of antimicrobial should be used.
- *Pregnancy*: Many antimicrobial agents should be used with caution as these drugs cross placental barrier.

Drugs which can be given in these conditions are:

1. Pencillins
2. Cephalosporins
3. Erythromycin
4. Lincomycin
5. Clindamycin
6. Azithromycin.

Drug Resistance and Toxicity

Drug resistance should also be kept in mind while prescribing antibiotic as some microorganisms are resistant to some antibiotics.

For example: Some strains of staphylococci are resistant to penicillin, then an alternative should be prescribed. Drug toxicity usually occurs when there is defect in excretion of drug. It is usually seen in hepatic and renal damage. Usually, the drugs are cleared by two mechanisms.

1. By renal excretion
2. Non-renal mechanism.

The mechanism of clearance of some commonly prescribed antibiotics is described in tabulated form as follows:

Mechanism of Clearance

Renal Excretion	Non-renal
1. Penicillins	1. Erythromycin
2. Tetracyclines	2. Doxycycline
3. *Cephalosporins	3. Chloramphenicol
4. Aminoglycosides	

(* *Cephalosporin excluding cefoparazore*)

Drug Allergic Reactions

Previous allergic reactions if any, to antimicrobials should be noted and alternative should be prescribed.

***For example*:** If a person is allergic to penicillin and its products, he/she should be prescribed erythromycin.

Cost Therapy

Presently many drugs are available in the market. Newer drugs are added from time-to-time which are usually more expensive but not necessarily more effective so proper selection of antibiotic is necessary.

COMMONLY USED ANTIBIOTICS

Beta-lactam Antibiotics

1. Penicillins
2. Cephalosporins.

Mechanism of Action

- Inhibition of cell wall synthesis.
- Bacteria possess a cell wall, which is absent in mammalian cells
- Bacterial cell contents are usually under high osmotic pressure and the viability of the bacteria depends upon the integrity of this peptidoglycan lattice in the cell wall
- These drugs bind to bacterial cell surface receptors which are actually enzymes involved in the transpeptidation reaction
- They also cause the inactivation of the cell well hibitor of autolytic enzymes in the cell well which results in the enzymatic cell lysis.

Classification of Penicillins

- Benzyl penicillin (penicillin G), penicillin V
- Penicillinase resistant penicillins: Methicillin, cloxacillin
- Broadspectrum penicillins: Ampicillin, amoxycillin
- Antipseudomonal penicillin: Carbencillin

Benzyl Penicillin (Penicillin G)

- Not very effective orally, therefore used i.m. or i.v.
- Easily destroyed by gastric acids
- Bactericidal active against gram-positive organisms.

(Available in different form-crystalline penicillin, procaine penicillin, benzathine penicillin)

- Adverse reaction: Anaphylaxis.

Penicillin V (Phenoxymethyl penicillin)

- This is not destroyed by gastric acid.
- Well absorbed orally.
- Antimicrobial spectrum-similar to penicillin G.

Penicillinase Resistant Penicillins

Penicillinase can inactivate beta-lactam antibiotics.Chemical modifications in the beta-lactamase ring has led to the development of penicillinase resistant penicillins such as methicillin and cloxacillin.

Broad-spectrum Penicillins (Ampicillin, Amoxycillin)

- Effective against gram-positive and gram-negative bacteria.
- They are destroyed by beta-lactamase enzyme but are acid stable
- Can be given orally
- Amoxycillin is better absorbed orally than ampicillin, has lower incidence of diarrhea and has a similar antibacterial spectrum.

Cephalosporins

- Broad-spectrum of activity, effective against gram-positive and gram-negative organisms
- Adverse reaction – allergy.

(Usually patients sensitive to penicillins are allergic to cephalosporins also)

- These drugs are classified according to their antibacterial spectrum into first, second, third and fourth generation cephalosporins.

Generation	Examples	Spectrum of activity
First generation cephalosporins	Cephalexin Cefadroxil	Effective against Gram+ve organisms
Second generation	Cefuroxime	Greater activity against

Contd...

Contd...

Cephalosporins	Cefaclor	Gram +ve organisms *Klebsiella, H. influ-enzae, E.coli*
Third generation cephalosporins	Cefotaxime Ceftriaxone Ceftazidime	Less activity against Gram +ve organisms *Pseudomonas, Enterobacteria Gonococci*
Fourth generation cephalosporins	Cefepime	*Enterococci, Gonococci*

Erythromycin

- Can be used in treating patients who are allergic to penicillin
- Effective against gram-positive cocci, streptococci, staphylococci
- Bacteriostatic in nature
- This drug is penicillinase resistant and thus can be used against staphylococcal infections
- Well absorbed orally.

Tetracycline

Mechanism of Action

- Inhibit bacterial protein synthesis
- Bacteriostatic in nature
- Broad-spectrum antibiotics
- Effective against gram-positive, gram-negative organisms, Mycoplasma and Rickettsia
- Problem of bacterial resistance
- Absorption of tetracycline is inhibited by chelation with milk
- Deposited into growing teeth and bones causing hypoplasia and staining. It should be avoided in children under 12 years of age and in pregnancy.

Metronidazole

- Main indication is for anaerobic infections.
- Bactericidal
- Effective in orofacial infections
- Can be given orally or parentrally
- Adverse reactions: nausea and metallic taste.

Ciprofloxacin

- Inhibition of DNA replication
- It inhibitis the enzyme DNA gyrase which prevents the supercoiling of the bacterial chromosome
- Bactericidal in nature
- Effective in treating gram-negative and gram-positive infections.

GUIDELINES FOR ANTIBIOTIC PROPHYLAXIS

The American heart association and the American dental association recently modified protocols for antibiotic prophylaxis against bacterial endocaditis. These changes show improvements in understanding of these disease processes and changing attitude towards the use of antibiotics.

Dental Procedures and Antibiotic Prophylaxis

Antibiotic prophylaxis recommended for

- Dental extraction
- Periodontal procedures including surgery, scaling and root planning, probing
- Dental implant placement
- Root canal instrumentation beyond apex
- Initial placement of orthodontic bands but not brackets
- Intraligamentary local anesthetic injections.

Antibiotic prophylaxis not recommended for

- Restorative dentistry (operative and prosthodontic) with or without retraction cord
- Local anesthetic injections (nonintraligamentary)
- Intracanal endodontic treatment; post placement and build-up
- Placement of rubber dams
- Postoperative suture removal
- Placement of removable prosthodontic or orthodontic appliances
- Taking of oral impressions
- Orthodontic appliance adjustment.

Guidelines for Antibiotic Prophylaxis for Dental Procedures

Condition	Drug	Dose
a. General prophylaxis	Amoxicillin	2000 mg given orally 1 hr before procedure
	Ampicillin	2000 mg given IM or IV 30 min. before procedure
b. Allergy to penicillin	Cephalexin	2000 mg given orally 1 hr before procedure
	Clindamycin	600 mg given orally 1 hr before procedure

contd...

contd...

	or IV 30 min before procedure
Azithromycin	500 mg given orally 1 hr before procedure

Cardiac Conditions Associated with Endocarditis

Prophylaxis recommended		*Prophylaxis not recommended*
High risk	*Moderate risk*	*Negligible risk*
Prosthetic heart valves	Rheumatic heart disease	Surgical repair of atrial septal defect
Previous bacterial endocarditis	Congenital cardiac diseases	Previous coronary bypass graft surgery
Complex cyanotic heart disease	Cadiomyopathy	MVP without valvular regurgitation
	Mitral valve prolapse (MVP)	Cardiac pacemakers and implanted defibrillators

REASONS FOR FAILURE OF ANTIBIOTIC THERAPY

- Inappropriate choice of antibiotics
- Patient failure to take antibiotics
- Impaired host defense
- Poor penetration to infected site
- Slow microbial growth
- Failure to eradicate source of infection
- Unfavorable local factors
- Limited vascularity
- Presence of resistant microorganisms.

24 Bleaching of Discolored Teeth

- Introduction
- Classification of Discoloration
- Bleaching
- Contraindications for Bleaching
- Medicaments Used as Bleaching Agents
- Mechanism of Bleaching
- Effects of Bleaching Agents on Tooth
- Home Bleaching Technique
- In Office Bleaching
- Bleaching of Non-vital Teeth
- Intra-coronal Bleach/Walking Bleach Procedure
- Inside/Outside Bleaching Technique
- Closed Chamber Bleach
- Microbrasion

INTRODUCTION

In the pursuit of looking good man has always tried to beautify his face. Since the alignment and appearance of teeth influence the personality they have received considerable attention.

Tooth discoloration varies in etiology, appearance, localization, severity and adherence to tooth structure. It may be classified as intrinsic, extrinsic and combination of both. Intrinsic discoloration is caused by incorporation of chromatogenic material into dentin and enamel during odontogenesis or after eruption. Exposure to high levels of fluoride, tetracycline administration, inherited developmental disorders and trauma to the developing tooth may result in pre-eruptive discoloration. After eruption of the tooth, aging, pulp necrosis are the main causes of intrinsic discoloration.

Coffee, tea, red wine, carrots and tobacco give rise to extrinsic stains. Wear of the tooth structure, deposition of secondary dentin due to aging or as a consequence of pulp inflammation and dentin sclerosis affect the light-transmitting properties of teeth, resulting in a gradual darkening of the teeth.

Scaling and polishing of the teeth remove many extrinsic stains. For more stubborn extrinsic discoloration and intrinsic stain, a variety of tooth whitening options are available today, the include over the counter whitening systems, whitening tooth pastes and the latest high tech option-laser tooth whitening.

Current available tooth whitening option are:

1. Office bleaching procedures
2. At home bleaching kits
3. Composite veneers
4. Porcelain veneers
5. Whitening tooth pastes

Among these procedures, bleaching procedures are more conservative than restorative methods, simple to perform and less expensive.

This chapter revises discoloration and its correction. Following aspects of discoloration and bleaching procedures are discussed in this chapter:

1. Etiology and types of discoloration.
2. Commonly used medicaments for bleaching.
3. External bleaching technique, i.e. bleaching in teeth with vital pulp.
4. Internal bleaching technique, i.e. usually perform in non vital teeth.
5. Efficacy and performance of each procedure.
6. Possible complications and safety of various procedures.

Before discussing bleaching of the discolored teeth, we should be familiar with the color of natural healthy teeth. Teeth are polychromatic, color varies among the gingival, incisal and cervical areas according to the thickness, reflections of different colors and translucency of enamel and dentin (Fig. 24.1). Color of healthy teeth is primarily determined by the translucency and color of dentine and is modified by:

- Color of enamel covering the crown
- Translucency of enamel which varies with different degrees of calcification.
- Thickness of enamel which is greater at the occlusal/incisal edge of the tooth and thinner at the cervical third. That is why teeth are more darker on cervical one-third than at middle or incisal one-third.

Normal color of primary teeth is bluish white whereas color of permanent teeth is grayish yellow, grayish white or yellowish white.

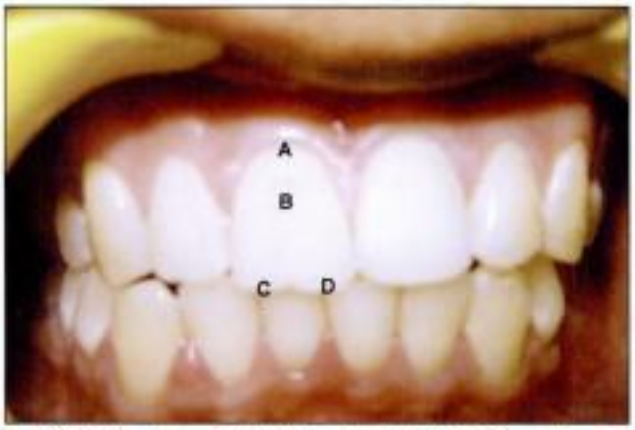


Fig. 24.1: Normal anatomical landmarks of tooth (A) Cervical margin (B) Body of tooth (C) Incisal edge (D) Translucency of enamel

CLASSIFICATION OF DISCOLORATION

Tooth discoloration varies with etiology, appearance, localization, severity and adherence to the tooth structure. It may be classified as extrinsic or intrinsic discoloration or combination. Feinman et al 1987, describes ***extrinsic discoloration*** as that occurring when an agent stains or damages the enamel surface of the teeth. Extrinsic staining can be easily removed by a normal prophylactic cleaning ***Intrinsic staining*** is defined as endogenous staining that has been incorporated into the tooth matrix and thus can not be removed by prophylaxis. Combination of both is multifactorial in nature e.g. nicotine staining.

Classification of discoloration

- Extrinsic discoloration
- Intrinsic discoloration
- Combination of both

Etiology of Tooth Discoloration

Intrinsic stains:

A. Pre eruptive causes
   a. Disease:
      I. Alkaptonurea
      II. Hematological diseases
      III. Disease of enamel and dentine
      IV. Liver disease
   b. Medication
      I. Tetracycline stains and other antibiotic use
      II. Fluorosis stain
B. Post eruptive causes of discoloration
   1. Pulpal changes
   2. Trauma
   3. Dentin hypercalcification
   4. Dental caries
   5. Restorative materials and operative procedures
   6. Aging
   7. Functional and parafunctional changes

Extrinsic stains:

A. Daily acquired stains
   1. Plaque
   2. Food and beverages
   3. Tobacco use
   4. Poor oral hygiene
   5. Swimmer's calculus
   6. Gingival haemorrhage
B. Chemicals
   1. Chlorhexidine
   2. Metallic stains

Stains During Odontogenesis (Pre-eruptive)

These are incorporated into the deeper layers of enamel and dentine during odontogenesis and alter the development and appearance of the enamel and dentine.

1. ***Alkaptonurea***: Dark brown pigmentation of primary teeth is commonly seen in alkaptonurea. It is an autosomal recessive disorder resulting into complete [illegible] of tyrosine and phenylalanine causing increases level of homogenitisic acid.

II. ***Hematological Disorders***
   a. *Erythroblastosis fetalis*: It is a blood disorder of neonates due to Rh incompatibility. In this stain does not involve teeth or portions of teeth developing after cessation of hemolysis shortly after birth. Stain is usually green, brown or bluish in color.
   b. *Congenital porphyria*: It is an inborn error of porphyrin metabolism, characterized by overproduction of Uroporphyrin. Deciduous and permanent teeth may show a red or brownish discoloration. Under ultra violet light teeth show red fluorescence.
   c. *Sickle cell anemia*: It is inherited blood dyscrasia characterized by increased hemolysis of red blood cells. In sickle cell anemia infrequently the stains of the teeth are similar to those of erythroblastosis fetalis, but discoloration is more severe, involves both dentitions and does not resolve with time.

III. ***Disease of Enamel and Dentine***

Developmental defects in enamel formation
- Amelogenesis imperfecta
- Endemic fluorosis
- Vitamin and mineral deficiency
- Chromosomal anomalies
- Inherited diseases
- Tetracycline
- Childhood illness
- Malnutrition
- Metabolic disorders

Amelogenesis Imperfecta (AI)

It comprises of a group of conditions, that demonstrate developmental alteration in the structure of the enamel in the absence of a systemic disorders. Amelogenesis Imperfecta (AI) has been classified mainly into Hypoplastic, hypocalcified and hypomaturation type (Fig. 24.2). In AI both deciduous and permanent dentition are affected differently.

Fluorosis

In fluorosis staining is due to excessive fluoride uptake during development of enamel. Excess Fluoride induces a metabolic change in ameloblast and the resultant enamel has a defective matrix and an irregular, hypomineralized structure (Fig. 24.3).

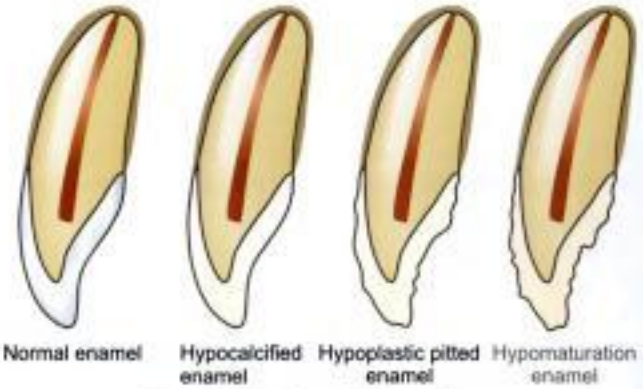


Fig. 24.2: Amelogenesis imperfecta

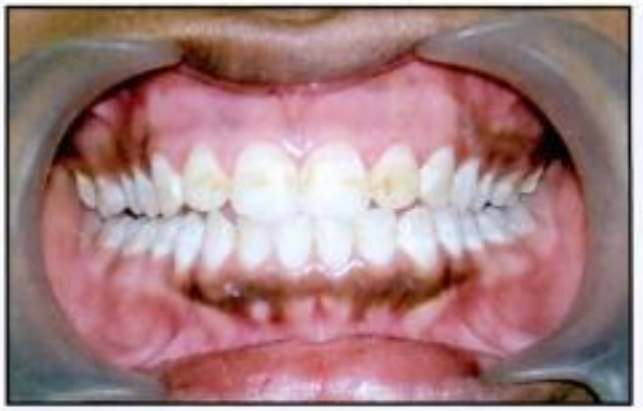


Fig. 24.3: Fluorosis of teeth

Staining Manifests as:
- Grey or white opaque flecks or areas on teeth.
- Yellow to Brown discoloration on a smooth enamel surface.
- Moderate and severe changes showing pitting and brownish discoloration of surface.
- Severely corroded appearance with dark brown discoloration and loss of most of enamel.

Enamel hypoplasia and hypocalcification due to other causes (Fig. 24.4).
- Vitamin D deficiency results in characteristic white patch hypoplasia in teeth.
- Vitamin C deficiency together with vitamin A deficiency during formative periods of dentition results in pitting type appearance of teeth.

Defective and Leaky Restoration

Defective and leaky restorations are not good candidate for bleaching. Because:

a. ***Discoloration and metallic salts particularly silver amalgam***: The dentinal tubules of the tooth become virtually saturated with alloys and no amount of bleaching with available products will significantly improve the shade.
b. ***Defective obturation***: If root canal is not well condensed, then refilling must be done before attempting bleaching.

Contraindication of Bleaching

1. Poor patient and case selection
   a. Psychological or emotional patient.
   b. Not fit for bleaching.
2. Dentin hypersensitivity
3. Extensive resorted tooth
4. Teeth with hypoplastic marks and cracks
5. Defective and Weakly restoration
   a. Defective obturation
   b. Discoloration from metallic salts as mercury

VARIOUS MEDICAMENTS USED AS BLEACHING AGENTS

An Ideal Bleaching Agent should:

- be easy to apply on the teeth.
- have a neutral pH
- lighten the teeth efficiently
- remain in contact with oral soft tissues for short periods.
- be required in minimum quantity to achieve desired results.
- not irritate or dehydrate the oral tissues.
- not cause damage to the teeth
- be well controlled by the dentist to customize the treatment to the patient's need.

Tooth bleaching today is based upon hydrogen peroxide as an active agent. Hydrogen peroxide may be applied directly or produced in a chemical reaction from sodium perborate or carbamide peroxide. Hydrogen peroxide acts as a strong oxidizing agent through the formation of free radicals, reactive oxygen molecules and hydrogen peroxide anions. These reactive molecules attack the long chained, the dark-colored chromophore molecules and split them into smaller, less colored and more diffusible molecules.

Carbamide peroxide also yields urea which is further decomposed to CO_2 and ammonia. It is the high pH of ammonia which facilitates the bleaching procedure.

This can be explained by the fact that in basic solution, lower activation energy is required for formation of free radicals from hydrogen peroxide and the reaction rate is higher, resulting in improved yield rate compared with an acidic environment.

The outcome of bleaching procedure depends mainly on the concentration of bleaching agents, the ability of the agents to reach the chromophore molecules and the duration and number of times the agent is in contact with chromophore molecules.

Constitution of Bleaching Gels

Constituents of the Bleaching Gels

- Carbamide peroxide
- Hydrogen peroxide and sodium hydroxide (Li, 1998)
- Sodium perborate
- Thickening agent-carbopol or polyx
- Urea
- Vehicle-glycerine, dentifrice, glycol
- Surfactant and pigment dispersants
- Preservatives
- Flavors
- Fluoride and 3 percent potassium nitrate

Carbamide Peroxide: ($CH_6N_2O_3$)

It is a bi-functional derivative of carbonic acid. It is available as:

1. Home Bleaching
   a. 5 percent carbamide peroxide
   b. 10 percent carbamide peroxide
   c. 15 percent carbamide peroxide
   d. 20 percent carbamide peroxide
2. In office bleaching
   a. 35 percent solution or gel of carbamide peroxide

Hydrogen Peroxide (H_2O_2)

H_2O_2 breaks down to water and nascent oxygen. It also forms free radical perhydroxyl (HO_2) which is responsible for bleaching action.

Sodium Perborate

It comes as monohydrate, trihydrate, tetrahydrate. It contains 95 percent perborate, providing 10 percent available oxygen.

Thickening agents

Carbopol (Carboxy polymethylene): Addition of cabopol in bleaching gels causes:

1. Slow release of oxygen
2. Increased viscosity of bleaching material, which further helps in longer retention of material in tray and need of less material.

3. Delayed effervescence – Thicker products stay on the teeth for longer time to provides necessary time for the carbamide peroxide to diffuse into the tooth.
4. The slow diffusion into enamel may also allow tooth to be bleached more effectively.

Urea – It is added in bleaching solutions to:
- Stabilise the H_2O_2
- Elevate the pH of solution.
- Anticariogenic effects

Surfactants
Surfactant acts as surface wetting agent which allows the hydrogen peroxide to pass across gel tooth boundary.

Preservatives
Commonly used preservatives are phosphoric acids, citric acid or sodium stannate. They sequestrate metals such as Fe, Cu, Mg accelerate breakdown of H_2O_2 and give gels better durability and stability.

Vehicle
1. Glycerine: It is used to increase viscosity of preparation and ease of manipulation.
2. Dentrifrice

Flavorings
They are added to improve patient acceptability.

MECHANISM OF BLEACHING

Mechanism of bleaching is mainly linked to degradation of high molecular weight complex organic molecules that reflect a specific wavelength of light that is responsible for color of stain. The resulting degradation products are of lower molecular weight and composed of less complex molecules that reflect less light, resulting in a reduction or elimination of discoloration.

Rate of Color Change is Affected by:
- Frequency that solutions are changed.
- Amount of time, the bleach is in contact with tooth
- Viscosity of material
- Rate of oxygen release
- Original shade and condition of the tooth
- Location and depth of discoloration
- Degradation rate of material

EFFECTS OF BLEACHING AGENTS ON TOOTH

1. ***Tooth hypersensitivity***: Tooth sensitivity is common side effect of external tooth bleaching. Higher incidence of tooth sensitivity (67-78%) is seen after in office bleaching with hydrogen peroxide in combination with heat. The mechanism responsible for external tooth bleaching though is not fully established, but it has been shown that peroxide penetrated enamel, dentin and pulp. This penetration was more in restored teeth than that of intact teeth.
2. ***Effects on enamel***: Studies have shown that 10 percent carbamide peroxide significantly decreased enamel hardness. But application of fluoride showed improved reminera-lization after bleaching.
3. ***Effects on dentine***: Bleaching has shown to cause uniform change in color through dentine.
4. ***Effects on pulp***: Penetration of bleaching agent into pulp through enamel and dentine occur resulting in tooth sensitivity. Studies have shown that 3 percent solution of H_2O_2 can cause:
   - Transient reduction in pulpal blood flow
   - Occlusion of pulpal blood vessels.
5. ***Effects on cementum***: Recent studies have shown that cementum is not affected by materials used for home bleaching. But cervical resorption and external root resorption in teeth has been seen in teeth treated by intracoronal bleaching using 30-35% H_2O_2.
6. ***Effects on restorative materials***:
   A. Application of bleaching on composites has shown following changes:
      - Increased surface hardness
      - Surface roughening and etching
      - Decrease in tensile strength
      - Increased microleakage
      - No significant color change of composite material itself other than the removal of extrinsic stains around existing restoration.

   B. Effect of bleaching agents on other materials:
      - No effect on gold restorations
      - Microstructural changes in amalgam
      - Alteration in the matrix of glass ionomers
      - IRM on exposure to H_2O_2 becomes cracked and swollen.
      - Provisional crowns made from Methyl Methacrylate discolor and turn orange.
7. ***Mucosal irritation***: A high concentration of hydrogen peroxide (30-35%) is caustic to mucous membrane and may causes burns and bleaching of the gingiva. So the bleaching tray must be designed to prevent gingival exposure by use of firmly fitted that has contact only with teeth.

8. ***Genotoxicity and carcinogenicity***: Hydrogen peroxide shows genotoxic effect as free radicals released from hydrogen peroxide (hydroxy radicals, perhydroxyl ions and superoxide anions) are capable of attacking DNA.
9. ***Toxicity***: The acute effects of hydrogen peroxide ingestion are dependent on the amount and the concentration of hydrogen peroxide solution ingested. The effects are more severe, when higher concentrations.

Signs and symptoms usually seen are ulceration of the buccal mucosa, esophagus and stomach, nausea, vomiting, abdominal distention and sore throat. It is therefore important to keep syringes with bleaching agents out of reach of children to prevent any possible accident.

Effects of Bleaching Agents on Tooth
- Tooth Sensitivity
- Alteration of Enamel Surface
- Effects on Dentine
- Effects of bleaching on pulp
- Effects on cementum
- Effects on Restorative materials
- Mucosal Irritation
- Genotoxicity and Carcinogenicity
- Toxicity

HOME BLEACHING TECHNIQUE/ NIGHT GUARD VITAL BLEACHING

Advantages
- Simple method for patients to use
- Simple for dentists to monitor
- Less chair time and cost effective
- Patient can bleach their teeth at their convenience

Disadvantages
- Patient compliance is mandatory
- Color change is dependent on amount of time the trays are worn.
- Chances of abuse by using excessive amount of bleach for too many hours per day.

Indications for Use
- Mild generalized staining
- Age related discolorations
- Mild tetracycline staining
- Mild fluorosis
- Acquired superficial staining
- Stains from smoking tobacco
- Color changes related to pulpal trauma or necrosis.

Contraindications
- Teeth with insufficient enamel for bleaching
- Teeth with deep and surface cracks and fracture lines
- Teeth with inadequate or defective restorations
- Discolorations in the adolescent patients with large pulp chamber
- Severe fluorosis and severe pitting hypoplasia
- Non compliant patients
- Pregnant or lactating patients
- Teeth with large anterior restorations
- Severe tetracycline staining
- Fractured or malaligned teeth
- Teeth exhibiting extreme sensitivity to heat, cold or sweets
- Teeth with opaque white spots
- Suspected or confirmed bulimia.

Factors that Guard the Prognosis for Home Bleaching
- History or presence of sensitive teeth
- Extremely dark gingival third of tooth visible during smiling
- Extensive white spots
- Translucent teeth
- Excessive gingival recession and exposed root surfaces.

Thickness of Tray
- Standard thickness of tray is 0.035 inch.
- Thicker tray, i.e. 0.05 inch is indicated in patients with breaking habit.
- Thinner tray, i.e. 0.02 inch thick is indicated in patients who gag.

Nature of material used for fabrication of bleaching tray is flexible plastic. Most common tray material used is ethyl vinyl acetate.

Steps of Tray Fabrication
- Take the impression and make a stone model
- Trim the model
- Place the stock out resin and cure it
- Apply separator / cold mould seal
- Choose the tray sheet material
- Cast the plastic in vacuum tray forming machines
- Trim and polish the tray
- Checking the tray for correct fit, retention and over extension
- Demonstrate the amount of bleaching material to be placed

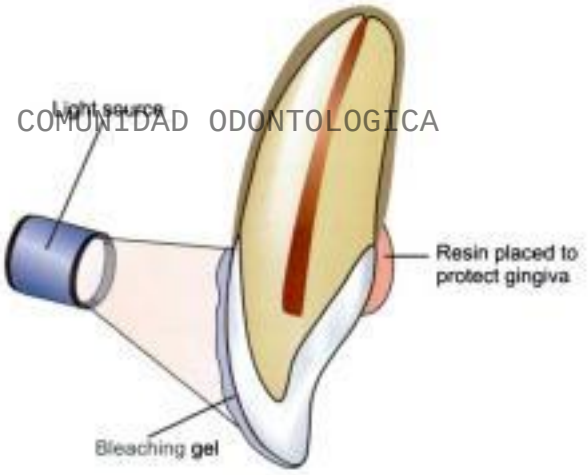


Fig. 24.7: Power bleaching technique

BLEACHING OF NONVITAL TEETH

1. Intracoronal bleaching/walking bleach procedure
2. Open chamber bleaching
   Inside and outside technique
3. Closed chamber bleaching-extracoronal
   - Power bleaching using 35% H_2O_2
   - Nightguard bleaching (10%, 15% or 20 % Carbamide Peroxide)

INTRACORONAL BLEACHING/WALKING BLEACH OF NONVITAL TEETH

It involves use of chemical agents within the coronal portion of an endodontically treated tooth to remove tooth discoloration.

Indications of Intracoronal Bleaching
- Discolorations of pulp chamber origin
- Moderate to severe tetracycline staining
- Dentine discoloration
- Discolorations not agreeable to extracoronal bleaching

Contraindications of Intracoronal Bleaching
- Superficial enamel discoloration
- Defective enamel formation
- Presence of caries
- Unpredictable prognosis of tooth

Steps

1. Take the radiographs to assess the quality of obturation. If found unsatisfactory, retreatment should be done.
2. Evaluate the quality and shade of restoration if present. If restoration is defective, replace it.
3. Evaluate tooth color with shade guide.
4. Isolate the tooth with rubber dam.
5. Prepare the access cavity, remove the coronal gutta percha, expose the dentine and refine the cavity (Fig. 24.8).
6. Place mechanical barriers of 2 mm thick, preferably of glass ionomer cement, zinc phosphate, IRM, polycarboxylate cement or cavity on root canal filling material (Fig. 24.9). The coronal height of barrier should protect the dentinal tubules and conform to the external epithelial attachment.

Fig. 24.8: Removal of coronal gutta-percha using rotary instrument

Fig. 24.9: Placement of protective barrier over gutta-percha

7. Now mix sodium perborate with an inert liquid (local anesthetic, saline or water) and place this paste into pulp chamber (Fig. 24.10)
8. After removing the excess bleaching paste, place a temporary restoration over it
9. Recall the patient after 1-2 weeks, repeat the treatment until desired shade is achieved
10. Restore access cavity with composite after 2 weeks.

Fig. 24.10: Placement of bleaching mixture into pulp chamber and sealing of cavity using temporary restoration

Complications of Intracoronal Bleaching
- External root resorption
- Chemical burns if using 30-35 percent H_2O_2
- Decrease bond strength of composite

Precautions to be taken for Safer Non-vital Bleaching
1. Isolate tooth effectively
2. Protect oral mucosa
3. Verify adequate endodontic obturation
4. Use protective barriers
5. Avoid acid etching
6. Avoid strong oxidizers
7. Avoid heat
8. Recall periodically

INSIDE/OUTSIDE BLEACHING TECHNIQUE

Synonyms: Internal/External Bleaching, Modified Walking Bleach Technique

This technique involves intracoronal bleaching technique along with home bleaching techniques. This is done to make the bleaching program more effective. This combination of bleaching treatment is helpful in treating difficult stains, for specific problems like single dark vital or nonvital tooth and to treat stains of different origin present on the same tooth.

Procedure

1. Assess the obturation by taking radiographs.
2. Isolate the tooth and prepare the access cavity by removing gutta percha 2-3 mm below the cemento-enamel junction.
3. Place the mechanical barrier, clean the access cavity and place a cotton pellet in the chamber to avoid food packing into it.
4. Evaluate the shade of tooth.
5. Check the fitting of bleaching tray and advise the patient to remove the cotton pellet before bleaching.
6. Instructions for home bleaching. Bleaching syringe can be directly placed into chamber before seating the tray or extrableaching material can be placed into the tray space corresponding to tooth with open chamber (Fig. 24.11).
7. After bleaching tooth is irrigated with water, cleaned and again a cotton pellet is placed in the empty space.
8. Re-assessment of shade is done after 4-7 days.
9. When the desired shade is achieved seal the access cavity initially with temporary restoration and finally with composite restoration after at least two weeks.

Advantages
- More surface area for bleach to penetrate
- Treatment time in days rather than weeks
- Decreases the incidence of cervical resorption
- Uses lower concentration of carbamide peroxide

Disadvantages
- Non compliant patients
- Over-bleaching by over zealous application
- Chances for cervical resorption is reduced but still exists

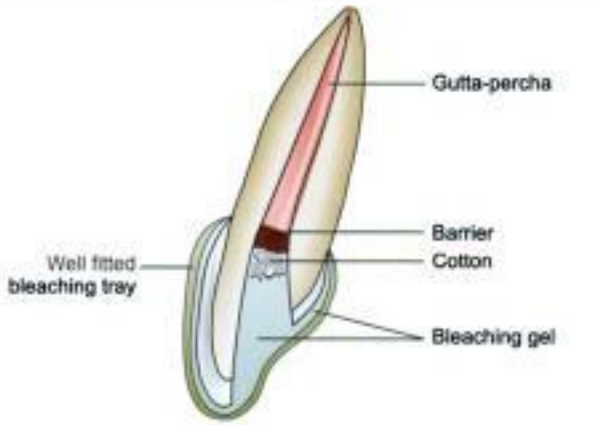


Fig. 24.11: Inside/outside technique. In this tray is seated over an open internal access opening, with a cotton pellet placed in open access cavity

25

Tooth Resorption

- Introduction
- Definition
- Classification of Resorption
- Cells Involved in Tooth Resorption
- Mechanism of Tooth Resorption
- Factors Regulating Tooth Resorption
- Internal Resorption
- External Resorption
- Cervical Root Resorption
- Transient Apical Breakdown
- Conclusion

INTRODUCTION

Pulp and periodontal tissues are unique in many aspects. A number of researches have been conducted to study the reparative, formative and protective reactions of the pulp and the periodontal tissues. Tooth resorption is one such reactive mechanism of these tissues to various forms of injuries. Roots of teeth undergo resorption under many circumstances. But unlike bone, which undergoes resorption and apposition as a part of continual remodeling process, the roots of permanent teeth are not normally resorbed.

If resorption occurs, it is because of some pathologic reasons but deciduous teeth show physiologic resorption before they are shed off.

DEFINITION

According to the American Association of Endodontics in 1944, (Glossary – Contemporary Terminology for Endodontics) ***resorption is defined as*** "A condition associated with either a physiologic or a pathologic process resulting in the loss of dentin, cementum or bone."

Root-resorption is the resorption affecting the cementum or dentin of the root of tooth.

Resorption is a perplexing problem for all dental practitioners. The etiologic factors are vague, diagnosis are the educated guesses and often the treatment does not prevent the rapid resorption of dental tissues.

The occurrence of resorption can not be predicted, it can be identified radiographically. But even this diagnostic tool has limitation because resorption on buccal or lingual surface of tooth usually can not be seen until 20 – 40% of the tooth structure has been dematerialized. Since the etiological factors, diagnosis, treatment and prognosis differ for the various types of resorption defects, the practitioners must be able to diagnose resorption radiographically or clinically, distinguish internal from external resorption and instigate appropriate treatment to stop the progress of the resorption process.

CLASSIFICATION OF RESORPTION

The area of root resorption is poorly understood and confusing. Many authors have used their own terminology to classify resorptive area.

Classification

- ***Physiologic tooth resorption*** is seen in deciduous teeth during eruption of permanent teeth
- ***Pathologic tooth resorption*** is seen in both deciduous as well as permanent teeth due to underlying pathology.
  a. ***Internal Resorption***
     - Root canal replacement resorption
     - Internal inflammatory resorption
  b. ***External Resorption***
     - Surface Resorption
     - Inflammatory Resorption
     - Replacement Resorption
     - Dentoalveolar Ankylosis

Classification of Resorption:

- ***Inflammatory***
  a. Internal
  b. External
- ***Non- inflammatory***
  a. Transient
  b. Pressure
  c. Replacement

CELLS INVOLED IN TOOTH RESORPTION

Clast Cells

Odonoclasts, dentinoclasts, osteoclasts and cementoclasts, all these cells belong to the group of clast cells and they have a common origin. They are derived from the circulating monocytes which form macrophages. When the inflammation gets out of control of the monocytes\ macrophages, they join together to form ***giant cells*** (Fig. 25.1). Osteoclasts have a life span of approximately 2 week. They are highly vacuolated and contain numerous mitochondria. Majority of odontoclasts have 10 or fewer nuclei, i.e. 96 percent are multinucleated and rest 4 percent are mononucleated.

Oligonuclear odontoclasts are cells with fewer than 5 nuclei. They resorb more dentin per nucleus when compared with cells with higher number of nuclei. Osteoclasts usually have 20 to 30 nuclei. Clear zone gives indication of the resorption activity.

Monocytes and macrophages: Monocytes and macrophages along with osteoclasts, play an important role in bone and

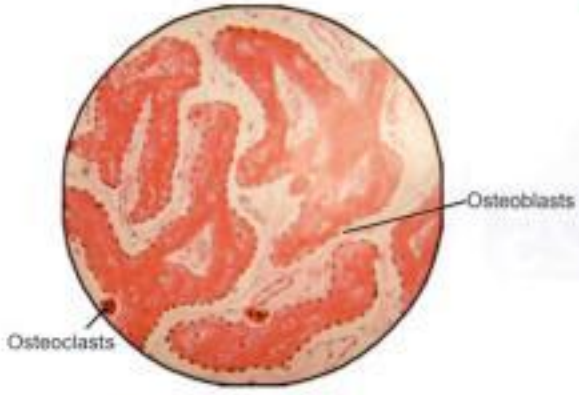


Fig. 25.1: Structure of bone showing osteoclasts and osteoblasts

tooth resorption. They are found in tissue surfaces adjacent to bone for example in resorpting surfaces of rheumatoid arthritis, periodontal diseases, periradicular granulomas, cysts, and in metastatic bone tumors. Although macrophages have a structure (Fig. 25.2) similar to that of osteoclasts, and like osteoclasts, can also become multinucleated giant cells, but macrophages lack a ruffled border that is attached to hard tissue substrates during resorption and do not create lacunae on the dentinal surface.

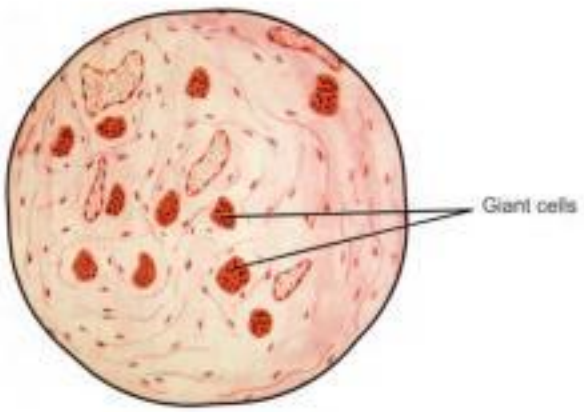


Fig. 25.2: Diagram showing giant cells

When there is an irritation of some kind, the tissue responds by the process of inflammation in which, the blood supply to the area is increased. There will be migration of monocytes to the site of inflammation, where they differentiate into macrophages. These processes are regulated by chemotactic factors like c-AMP and calcium.

Fig. 25.3: Internal resorption

According to ***Shafer***, "internal resorption is an unusual form of tooth resorption that begins centrally within the tooth, apparently initiated in most cases by a peculiar inflammation of the pulp". It is characterized by oval shaped enlargement of root canal space (Fig. 25.3). It is usually asymptomatic and discovered on routine radiographs. Internal resorption may progress slowly, rapidly or intermittently with period of activity and inactivity.

Etiology

Internal resorption is pulpally related problem that triggers resorption of the dentin from the pulp outward (Fig. 25.4). The tooth often has a history of trauma or pulp cap. Discoloration may or may not be present. Pulp tests may indicate vitality or necrosis. The abnormal pulpal response results in ***dentinoclastic*** activity that generates an increase in the size of the chamber and canal space.

Internal resorption may also be caused while doing restorative procedure like preparation of tooth for crown, deep restorative procedures, application of heat over the pulp or pulpotomy using calcium hydroxide, i.e. iatrogenic in origin. Internal resorption can also be idiopathic in origin.

Fig. 25.4: Internal resorption with root perforation

Etiology

- Long standing chronic inflammation of the pulp
- Caries related pulpits
- Traumatic injuries
  a. Luxation injuries
- Iatrogenic injuries
  a. Preparation of tooth for crown
  b. Deep restorative procedures
  c. Application of heat over the pulp
  d. Pulpotomy using $Ca(OH)_2$
- Idiopathic

Clinical Features

The pulp usually remains vital and asymptomatic until root has been perforated and become necrotic. Patient may present pain when the lesion perforates and tissue is exposed to oral fluids (Fig. 25.4).

It is commonly seen in maxillary central incisors, but any tooth of the each can be affected (Fig. 25.5).

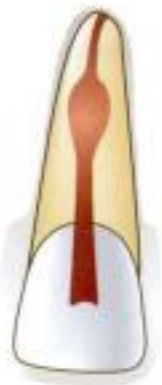


Fig. 25.5: Internal resorption

Internal resorption is usually unilateral; Solomon CS (1986) reported the occurrence of bilateral internal resorption in maxillary premolars.

Clinical Features of Internal Resorption

- Usually asymptomatic until it perforates the root and communicates with the periodontium
- Common in maxillary central, but can affect any tooth
- Spreads rapidly in primary teeth
- Pathognomic feature is pink spot appearance of tooth which represents the hyperplasic vascular pulp tissue showing off through crown of tooth.

Fig. 25.8: Placement of $Ca(OH)_2$ dressing in canal

and at the next visit, obturation can be done after flushing the calcium hydroxide and tissue debris from the canal.

Canal Obturation

Because of the size, irregularity and in accessibility of the resorption defects, obturation of the canal may be technically difficult.

The canal apical to the defect is filled with solid gutta-percha while the resorptive area is usually filled with material that will flow in the irregularities. The warm gutta-percha technique, thermoplasticized gutta-percha technique and use of chemically plasticized gutta-percha are methods of obturation to be used.

Management of Perforating Internal Resorption

When the internal root resorption has progressed through the tooth into the periodontium, there are additional problem of periodontal bleeding, pain and difficulty in obturation. Presence of a perforation can not be determined radiographically unless a lateral radiolucent lesion is present adjacent to the lesion. Clinically in some cases a sinus tract may be present and there will be continued hemorrhage in the canal after the pulp is removed.

a. Non-surgical Repair

Indications: Non-surgical repair is indicated in following cases:

i. When the defect is not extensive.
ii. When defect is apical to epithelial attachment.
iii. When hemorrhage can be controlled.

In this technique, after thorough cleaning and shaping of the canal, the intracanal calcium hydroxide dressing is placed and over it a temporary filling is placed to prevent interappointment leakage.

Patient is recalled after three months for replacement of calcium hydroxide dressing and for radiographic confirmation of the barrier formation at the perforation site. Afterwards two months recall visits are scheduled until there is a radiographic barrier at resorption defect. After the barrier is formed, the canal is obturated with gutta-percha as in the non perforating internal resorption.

b. Surgical Repair

Indication of surgical repair:

i. Surgical flap
ii. Root resections
iii. Intentional replantation

If the calcium hydroxide treatment is unsuccessful or not feasible, surgical repair of the defect should be considered.

1. ***Surgical flap*:** Here the defect is exposed to allow good access. The resorptive defect is curetted, cleaned and restored. The restoration of the defect can be done using an alloy, composite, glass ionomer cement, super EBA or more recently MTA. Finally the obturation is done using gutta-percha.
2. ***Root Resection*:** If the resorbed area is located in the radicular third, root may be resected coronal to the defect and apical segment is removed afterwards. Following root resection, retrofilling is done.

   If one root of a multirooted tooth is affected, root resection may be considered based on anatomical, periodontal and restorative parameters.
3. ***Intentional replantation*:** If the perforating resorption with minimal root damage occurs in an inaccessible area, intentional replantation may be considered.

EXTERNAL ROOT RESORPTION

External root resorption is initiated in the periodontium and it affects the external or lateral surface of the root (Fig. 25.9).

Classification

According to Rita F. Ne, Guttman et al as given in Quintessence International 1999, classification of ***External resorption*** is of 3 types by ***clinical and histologic manifestation:***

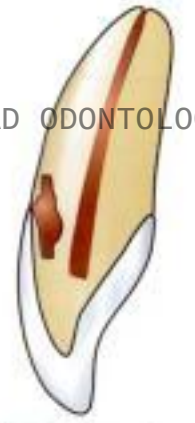


Fig. 25.9: External resorption

- Surface resorption
- External inflammatory root resorption
- Replacement resorption

According to ***Cohen***, depending ***on the basis of location***, 3 types of ***external resorption*** could be:

- Cervical
- Lateral
- Apical

External Resorption may be Found in Following Conditions:

- Periodontal disease
- Luxation injuries
- Hypoparathyroidism
- Hyperparathyroidism
- Turner's syndrome
- Paget's disease
- Gaucher's disease
- Radiation therapy

Surface Resorption

Surface resorption is common occurrence in which root surface shows superficial resorption lacunae repaired with new cementum. These lacunae are called ***surface resorption*** (Fig. 25.10). It is associated with trauma to teeth in which the injury damages cementum and cementoblasts. It is least destructive type of resorption.

Surface resorption can be transient or progressive. In the transient variety, the tooth has a vital, healthy pulp that has recovered from traumatic event. In such cases, the resorbed area will be restored completely to normal surface contour by deposition of new cementum.

In the progressive type, the surface resorption is the beginning of more destructive resorption, either inflammatory resorption or replacement resorption.

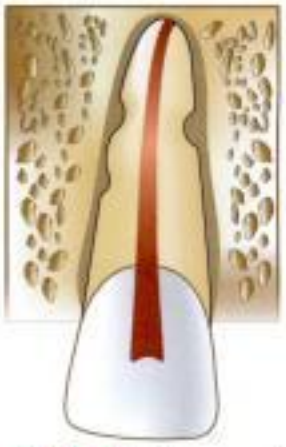


Fig. 25.10: Surface resorption

Because surface resorption is not detectable radiographically and only can be observed histologically, it follows that no clinical treatment is available.

External Inflammatory Root Resorption

It is most common and most destructive type of resorption and is thought to be caused by presence of infected or necrotic pulp tissue in the root canal (Fig. 25.11).

Fig. 25.11: External inflammatory resorption

Etiopathology

- Injury or irritation to the periodontal tissues where the inflammation is beyond repair
- Trauma leads to pulpal necrosis which may further cause periodontal inflammation due to the passage of the toxins and microorganisms from the infected pulp, lateral canals, apical foramen, accessory canals, dentinal tubules where there is a discontinuity of cementum

It has also been observed in primary mandibular molars. Ankylosis may be transient or progressive. In the transient type, less than 20 percent of the root surface becomes ankylosed. In such cases, spontaneous cure occur, resulting in reestablishment of a periodontal ligament connection between tooth and bone.

In the progressive type, tooth structure is gradually resorbed and replaced with the bone.

Etiopathogenesis

Replacement resorption usually occurs after a severe dental injuries like intrusive luxation or avulsion injuries resulting in drying and death of periodontal ligament cells. An inflammatory process removes the necrotic debris from the root surface. After initial inflammatory response to remove debris from injury, root surface devoid of cementum results. To compensate this, cell in vicinity of root surface try to repopulate it. It is seen that cells which form bone move across the socket wall and colonize the damaged root wall. Because of this, the bone comes in direct contact with root without an intermediate attachment apparatus. This results in dentoalveolar ankylosis.

Histological Examination

It shows a direct fusion between dentin and bone without separating cemental or periodontal ligament layer. Active resorption lacunae with osteoclasts are seen in conjunction with apposition of normal bone laid by osteoblasts.

Clinically

Replacement resorption is usually asymptomatic. Infra occlusion, incomplete alveolar process development (if the patient is young), and prevention of normal mesial drift are commonly seen. Pathognomic feature is immobility of affected tooth and a distinctive metallic sound on percussion.

Diagnosis

Diagnosis can be made from clinical evaluation and radiographic observation. Lack of mobility and high pitched metallic sound on percussion of tooth are often the characteristic features of ankylosis.

Radiographically, the loss of periodontal ligament space with replacement by bone in association with an uneven contour of root is indicative of ankylosis.

Treatment

Currently there is no treatment offered for replacement resorption. It may be possible to slow the resorptive process by treating the root surface with fluoride solution prior to replantation if it is known that extraoral time for tooth was more than two hours and it was not kept moist to protect the periodontal ligament. One should not view replacement resorption as total failure. A replanted tooth undergoing replacement resorption can see many years before root is fully resorbed.

Recently Introduced Enamel Product "Emdogain"

Emdogain was developed in 1992 with the purpose of regenerating lost periodontium. When it is applied, amelogenin rich protein matrix precipitate out of the solution and forms and insoluble layer on the root surface promoting the attachment of mesenchymal cells. These cells then produce new matrix component and growth factors, which play major role in formation of new periodontal attachment. Emdogain also inhibits epithelial cell growth which could interfere the regenerative process. It promotes both proliferation and differentiation of osteoblastic cells and inhibits the formation of osteoclasts by stimulating the effect of osteoprotegerive (OPG).

Emdogain may also act as growth factor which prolongs the osteoblast growth and maintain their morphology.

There are ***two schools*** of thoughts of its use as an adjunct in treatment of replacement resorption.

One school says the main cause of replacement resorption is damage to periodontal tissue during replantation. Emdogain accumulates in the cell at the root surface and promotes regeneration of PDL tissues.

However ***other school*** of thought does not support is use. In a clinical study, avulsed and previously ankylosed teeth replanted with Emdogain developed the subsequent ankylosis after six months leading to conclusion that Emdogain could not prevent or cure ankylosis.

Further studies are needed to prove the efficacy of Emdogain in prevention of replacement resorption.

Dentoalveolar Ankylosis

Dentoalveolar ankylosis is the union of tooth and bone with no intervening connective tissue.

Etiopathology

- Traumatic injuries to teeth—In cases of intrusive luxation and reimplantation of avulsed tooth, especially with long extraoral dry time
- The rate of progression of ankylosis is directly related to—the amount of damage to the root surface
- Progression of the ankylosis is very rapid in young individuals as compared to adults.

Clinical Features of Cervical Root Resorption

- Initially asymptomatic
- Pulp vital in most cases
- Tooth mobility is lost
- Long standing cases give pink spot appearance Clinically, misdiagnosed as internal resorption but confirmed radiographically
- In due course, it spreads laterally along the root, i.e. apical and coronal direction "enveloping" the root canal.

Theories of Cervical Root Resorption

i. Some procedures and events (bleaching, trauma, orthodontic treatment) cause alteration in the organic and inorganic cementum, finally making it more inorganic. This makes the cementum less resistant to resorption.

ii. Immunological system senses the altered root surface, as a different tissue, attacks as a foreign body.

Clinical Features

- Initially the cervical root resorption is asymptomatic in nature
- Pulp if present will be vital in most cases of cervical root resorption. The pulp responds normal to sensitivity tests
- Long standing cervical root resorption cases show extensive loss of tooth structure replaced by granulation tissues which undermines the enamel in due course giving rise to Pink spot appearance clinically. It is misdiagnosed as internal resorption, but confirmed with a radiograph. It initially starts as a small lesion, progress and reaches the predentin. Predentin is more resistant to resorption, spreads laterally in apical and coronal direction "enveloping" the root canal.

Rarely, it perforates into the tooth causing secondary involvement of pulp. In most of the cases, it occurs at the immediate subgingival level.

Franks's Classification of Cervical Root Resorption

- ***Supraosseous*** — Coronal to the level of alveolar bone (Fig. 25.14).
- ***Intraosseous*** — Not accompanied by periodontal breakdown (Fig. 25.15).
- ***Crestal*** — At the level of alveolar bone (Fig. 25.16).

Fig. 25.14: Supraosseous extra canal invasive resorption

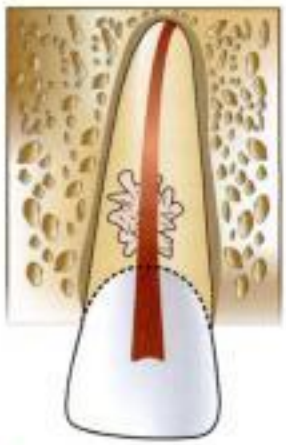


Fig. 25.15: Intraosseous extra canal invasive resorption

Fig. 25.16: Crestal extra canal invasive resorption

Radiographic Features

Radiographically one can see the moth-eaten appearance with the intact outline of the canal. Because bone is often involved, resorption may give the appearance of an infrabony pocket.

Treatment

The ***main aim*** of the treatment is to restore the lost tooth structure and to disrupt the resorptive process. A traditional approach is to treat the tooth endodontically first, followed by repair of the resorbed area either from an internal approach or an external one.

A combination of internal restoration followed by a surgical approach to smoothen and finish the surface of filling material where it exits through the original resorptive entry, may provide acceptable results.

Another treatment approach has been recommended because the Pulp is often vital in a tooth with invasive resorption, the repair of resorbed area may be accomplished without removing the pulp. The clinical procedure consists of surgically exploring the resorbed lacuna and curetting the soft tissue from the defect, which can then be the prepared for restoration. The advantage of such a non endodontic approach is that, it is more conservative than the more common approach of including root canal therapy in the treatment. If pulpal symptoms develop later, root canal therapy can be done when needed. Other treatment options are intentional replantation or root amputation of affected tooth.

There is not known method for prevention of invasive resorption, early detection will allow more conservative treatment. The prognosis after treatment is uncertain because clinical experience has shown that, even after restoration of resorptive defects, new foci of resorption just apical to the previous lacunae may recur.

TRANSIENT APICAL BREAKDOWN

It is a temporary phenomenon in which the apex of the tooth displays radiographic appearance of resorption—followed by surface resorption and/or obliteration of the pulp canal (Fig. 25.17).

- Repair takes place within a year. It is commonly seen in mature teeth with completely closed apex
- Usually it occurs after moderate injuries to the tooth for example—subluxation, extrusion, lateral luxation
- Infections, orthodontic treatment, trauma from occlusion etc.
- No treatment is recommended.

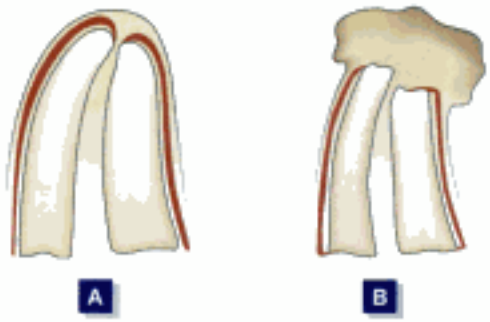


Fig. 25.17: **A.** Normal root apex **B.** Apical root resorption

CONCLUSION

Tooth resorption is a perplexing problem where the etiologic factors are vague and less clearly defined. For the best treatment outcome, the clinician should have a very good knowledge of the etiopathology of resorptive lesions. Early diagnosis and prompt treatment in such cases are the key factors which determine the success of the treatment. More clinical studies and research with animal models are required to explain more about this phenomenon scientifically.

26

Tooth Hypersensitivity

- Introduction
- Historic Review
- Neurophysiology of Teeth
- Mechanism of Tooth Hypersensitivity
- Dentin Hypersensitivity
  - Incidence and Distribution
  - Etiology and Predisposing Factors
  - Treatment Strategies
  - Management of Sensitive Lesions

INTRODUCTION

Clinical experience suggests that dentine hypersensitivity is a relatively common cause of pain associated with the teeth. Despite this, the condition has been described as "an enigma" being frequently encountered but ill understood.

The term tooth hypersensitivity, dentinal sensitivity or hypersensitivity often used intermittently to describe clinically condition of an exaggerated response to an exogenous stimulus.

The exogenous stimuli may include thermal, tactile or osmotic changes. While extreme stimuli can make all the teeth hurt, the term ***hypersensitivity*** means painful response to stimuli not normally associated with pain. The response to a stimulus varies from person to person due to difference in pain tolerance, environment factors and psychology of patient. Tooth hypersensitivity can fit the criteria of several pain terms described by Merskey (1979), for the International Association for the study of pain (IASP). Pain is described "as an unpleasant sensory and emotional experience associated with actual or potential tissue damage."

To standardize the description of the condition, *dentin hypersensitivity is defined as "sharp, short pain arising from exposed dentine in response to stimuli typically thermal, chemical, tactile or osmotic and which cannot be ascribed to any other form of dental defect or pathology. (Holland et al, 1997).*

Tooth hypersensitivity is not associated with actual tissue damage in the acute sense but can involve potential tissue damage with constant erosion of the enamel or cementum along with the concomitant pulpal response.

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

Such conditions include;

- Chipped teeth
- Fractured restoration
- Restorative treatments
- Dental caries

- Cracked tooth syndrome
- Other enamel invaginations.

Thus, a careful history (to exclude all the above conditions) together with a thorough clinical and radiographic examination, is necessary before arriving at a definitive diagnosis of dentine hypersensitivity. However, the problem may be made difficult when two or more conditions coexist.

Tooth hypersensitivity differs from dentinal or pulpal pain in that in case of dentine hypersensitivity, patient's ability to locate the source of pain is very good, whereas in case of dentinal pain, it is poor and in pulpal pain, it is very poor. The character of the pain does not outlast the stimulus; the pain is intensified by thermal changes, sweet and sour. Intensity of pain is usually mild to moderate. The pain can be duplicated by hot or cold application or by scratching the dentin. The pulpal pain is explosive, intermittent and throbbing and can be affected by hot or cold.

HISTORIC REVIEW

1. Leeuwenhoek (1678) who shortly after his invention of the microscope described "tooth canals in dentin."
2. JD White (1855) made a presentation, which proposed that dentinal pain was caused by movement of fluid in dentinal tubules.
3. In 1900, Alfred Gysi, published a report that dental canaliculi are devoid of nervous substances, but at the inner border of the dentine around the odontoblast there is an "abundant network of finest nerve fibers".
4. In 1915, Alfred Gysi reported that fluid in the tubule was incompressible, thus stimulus induces a wave like motion which is transmitted to the pulp.
5. Louis I Grossman (1935) described hypersensitiveness in dentin as uncommon sensitivity or painful response of the exposed dentin to an irritation.
6. McGhee (1936) reported that pain was not continuous but was felt only as long as the irritant was present.
7. Lukomsky (1941) advocated sodium fluoride as a desensitizing obtundent.
8. Brannstrom (1962) summarized the *"hydrodynamic theory"* of dentinal pain excitation that is pain is induced by the movement of tubular fluid in response to externally applied stimuli.
9. Kleinberg (1986) summarized the different approaches that have been used to treat hypersensitive dentin as follows:
   i. Remineralization by salivary deposits of calcium phosphate complexes within dentinal tubules.
   ii. Formation of secondary dentine, which may occur naturally or can be stimulated by brushing.
   iii. Calcium hydroxide facilitates calcium phosphate deposition from dentinal fluid and saliva.
   iv. Sodium fluoride promotes the deposition of less soluble fluoroapatite.
   v. Silver nitrate precipitates proteins within dentinal tubules.
   vi. Strontium chloride forms strontium hydroxyapatite and strontium phosphate within dentinal tubules.
   vii. Resins seal the outer ends of dentinal tubules.
   viii. Dentifrices may provide one of the active ingredients above or function by occluding tubular orifices.

1678	Leeuwenhoek	Made first scientific observation for better understanding of dentinal hypersensitivity
1941	Lukomsky	Advocated sodium fluoride as desensitizing agent
1962	Brannstrom	Hydrodynamic theory of dentinal pain
1986	Kleinberg	Summarized the different approaches that are used to treat hypersensitivity.

THE NEUROPHYSIOLOGY OF TEETH

The dental pulp is richly innervated. On their way from the apex to the coronal pulp, the nerves branch and finally form a dense network of the fine fibers at the pulp dentin border area.

Both myelinated and unmyelinated axons innervate the pulp. According to conduction velocities, the nerve units can be classified into A group—having the conduction velocity more than 2 m/s, and C group—with conduction velocity less than 2 m/s.

Most of the A fibers have their conduction velocities within the Aδ range (6-30 m/s). The sharp, better localized pain is mediated by Aδ fibers, whereas C fibers activation seems to be connected with the dull radiating pain sensation. Myelinated A fiber seems to be responsible for dentin sensitivity.

It is well known that even the most peripheral part of dentin can be sensitive. However, varying opinions have been presented on the mechanism of intradental nerve activation in response to external irritation.

Byer (1984) in a recent neuroanatomic studies have shown that only the inner 100 to 200 μm of dentin is innervated, indicating that the pain sensations induced by

stimulation of superficial dentin cannot be a result of direct irritation of nerve endings.

MECHANISM OF DENTIN SENSITIVITY

Theories of Dentin Sensitivity
- Neural theory
- Odontoblastic transduction theory
- Hydrodynamic theory
- Modulation theory

Theories of Dentine Sensitivity

A. Neural Theory

The neural theory attributes activation to an initial excitation of those nerves ending within the dentinal tubules. These nerve signals are then conducted along the parent primary afferent nerve fibers in the pulp, into the dental nerve branches, and then into the brain (Fig. 26.1). Neural theory considered that entire length of tubule contains free nerve endings. However, the study of Byer (1984) shown that only 100-200 µm of peripheral dentin contains nerve endings.

B. Odontoblastic Transduction Theory

The theory assumed that odontoblast extended to the periphery. The stimuli initially excite the process or body of the odontoblast. The membrane of odontoblasts may come into close apposition with that of nerve endings in the pulp or in the dentinal tubule, and the odontoblast transmits the excitation of these associated nerve endings. However, in the most recent study; Thomas (1984) indicated that the odontoblastic process is restricted to the inner third of the dentinal tubules. Accordingly it seems that the outer part of the dentinal tubules does not contain any cellular elements but is only filled with dentinal fluid.

C. The Hydrodynamic Theory

This theory proposes that a stimulus causes displacement of the fluid that exists in the dentinal tubules. The displacement occurs in either an outward or inward direction, and this mechanical disturbance activates the nerve endings present in the dentin or pulp.

***Fish* (1927)** observed the interstitial fluid of the dentin and pulp, referring to it as the "dental lymph".

***Brannstrom* (1962)** suggested that the displacement of the tubule contents is rapid enough to deform nerve fiber in pulp or predentin or damage odontoblast cell. Both of these effects appear capable of producing pain.

Currently most investigators accept that dentin sensitivity is due to the hydrodynamic fluid shift, which occur across exposed dentin with open tubules. This rapid fluid movement in turn activates the mechanoreceptor nerves of A group in the pulp (Fig. 26.2).

***Mathews et al* (1994)** noted that stimuli such as cold causes fluid flow away from the pulp, produces more rapid and greater pulp nerve responses than those such as heat, which causes an inward flow. This certainly would explain the rapid and severe response to cold stimuli compared to the slow dull response to heat.

The dehydration of dentin by air blasts or absorbent paper causes outward fluid movement and stimulates the

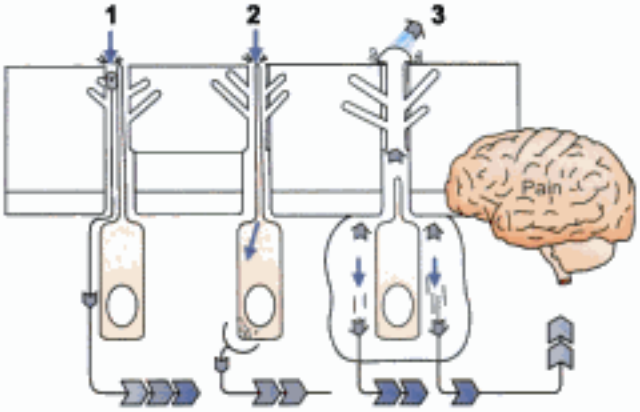


Fig. 26.1: Theories of dentin hypersensitivity (1) Neural theory: Stimulus applied to dentin causes direct excitation of the nerve fibers (2) Odontoblastic transduction theory: Stimulus is transmitted along the odontoblast and passes to the sensory nerve endings through synapse (3) Hydrodynamic theory: Stimulus causes displacement of fluid present in dentinal tubules which further excite nerve fibers

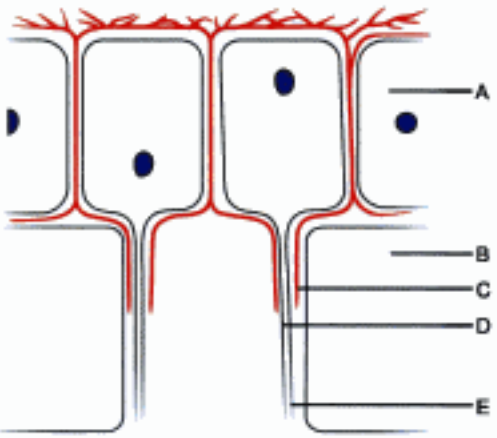


Fig. 26.2: Hydrodynamic theory (A) Odontoblast (B) Dentin (C) A-S nerve fiber (D) Odontoblastic process (E) Stimulation of A-S nerve fiber from fluid movement

mechanoreceptor of the odontoblast, causing pain. Prolonged air blast causes formation of protein plug into the dentinal tubules, reducing the fluid movement and thus decreasing pain (Fig. 26.3a).

The pain produced when sugar or salt solutions are placed in contact with exposed dentin can also be explained by dentinal fluid movement. Dentinal fluid is of relatively low osmolarity, which have tendency to flow towards solution of higher osmolarity, i.e. salt or sugar solution (Fig. 26.3b).

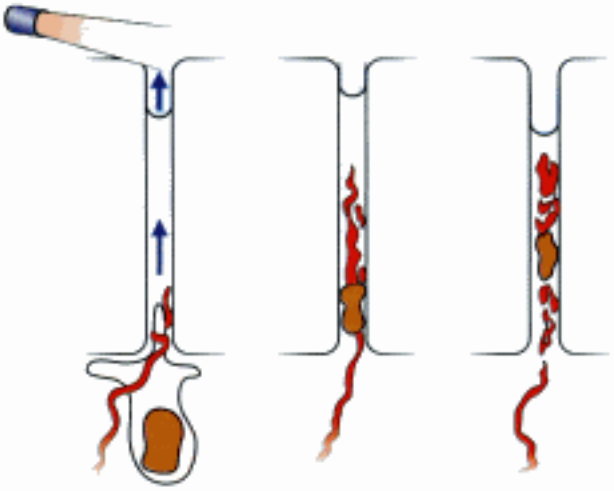


Fig. 26.3a: Effect of air blast on dentin

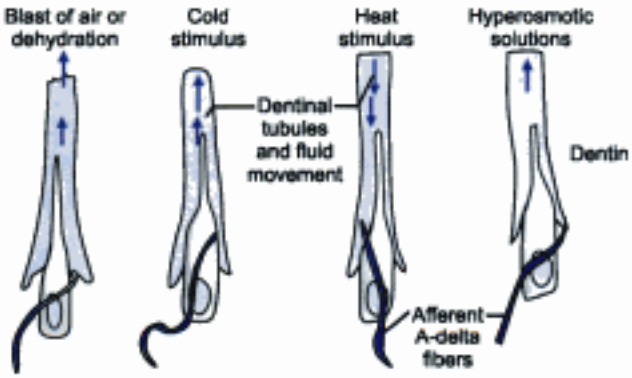


Fig. 26.3b: Pain produced by different stimuli

D. Modulation Theory

***Kroeger* (1968)** implicated number of polypeptides as regulators of neural transmission of plasma kinins. These substances may selectively alter the permeability of the odontoblastic cell membrane, so that pulp neurons are more prone to discharge upon receipt of subsequent stimuli. Kroeger believed that plasma kinins were formed in the pulp when kallikinins were released as result of nerve stimulation. Under normal circumstances, pulp tissue contains enzymes capable of inactivating plasma kinins. These substances may modulate associated nerve fiber action potentials by increasing neuronal cyclic AMP levels through cell membrane adenylate cyclase receptors.

Incidence and Distribution of Dentine Hypersensitivity

The prevalence studies for dentine hypersensitivity are limited in number. The available prevalence data vary considerably, and dentine hypersensitivity has been stated to range from 8 to 30 percent of adult population.

- Most sufferers range in range from 20-40 years and a peak occurrence is found at the end of the third decade.
- In general, a slightly higher incidence of dentine hypersensitivity is reported in females than in males.
- The reduced incidence of dentin hypersensitivity in older individuals reflects age changes in dentin and the dental pulp. Sclerosis of dentin, the laying down of secondary dentin, and fibrosis of the pulp would all interfere with the hydrodynamic transmission of stimuli through exposed dentin.

Intra-oral Distribution

- Hypersensitivity is most commonly noted on buccal cervical zones of permanent teeth. Although all tooth type may be affected, canines and premolars in either jaw are the most frequently involved.
- Regarding the side of mouth, in right handed tooth brushers the dentine hypersensitivity is greater on the left sided teeth compared with the equivalent contra-lateral teeth.

Etiology and Predisposing Factors

The primary underlying cause for dentin hypersensitivity is exposed dentin tubules. Dentin may become exposed by two processes; either by loss of covering periodontal structures (gingival recession), or by loss of enamel.

The most common clinical cause for exposed dentinal tubules is gingival recession (Fig. 26.4). Various factors which can cause recession are inadequate attached gingiva, improper brushing technique, periodontal surgery, overzealous tooth cleansing habits, oral habits etc.

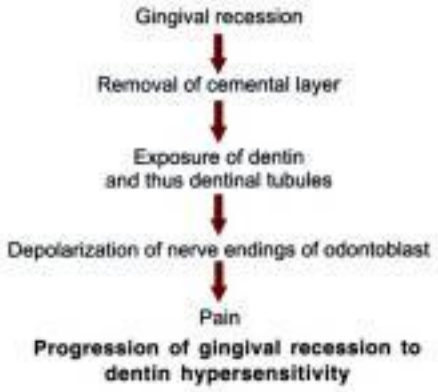
Fig. 26.4: Recession of gingiva

Common Reasons for Gingival Recession

- Inadequate attached gingiva
- Prominent roots
- Toothbrush abrasion
- Pocket reduction periodontal surgery
- Oral habits resulting in gingiva laceration, i.e. traumatic tooth picking, eating hard foods
- Excessive tooth cleaning
- Excessive flossing
- Gingival secondary to specific diseases, i.e. NUG, periodontitis, herpetic gingivostomatitis
- Crown preparation.

The recession may or may not be associated with bone loss. If bone loss occurs, more dentinal tubules get exposed. When gingival recession occurs, the outer protective layer of root dentin, i.e. cementum gets abraded or eroded away (Fig. 26.5).

Fig. 26.5: Erosion of cementum



Progression of gingival recession to dentin hypersensitivity

This leaves the exposed underlying dentin, which consists of protoplasmic projections of odontoblasts within the pulp chamber (Fig. 26.6). These cells contain nerve endings and when disturbed, they depolarize and this is interpreted as pain (Fig. 26.7).

Fig. 26.6: Exposure of dentinal tubules

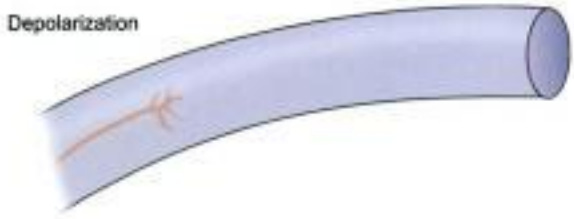


Fig. 26.7: Depolarization of nerve ending causing pain

We have seen earlier that most accepted theory of pain is Brannstrom's hydrodynamic theory of dentin hypersensitivity. Once the dentinal tubules are exposed, there are oral processes which keep them exposed. These include poor plaque control, enamel wear, improper oral hygiene technique, cervical erosions, enamel wear and exposure to acids.

Reasons for Continued Dentinal Tubular Exposure
- Poor plaque control, i.e. acidic bacterial byproducts.
- Excess oral acids, i.e. soda, fruit juice, swimming pool chlorine, bulimia
- Cervical decay
- Toothbrush abrasion
- Tartar control toothpaste

The other reason for ***exposure of dentinal tubules is due to loss of enamel***. Loss of enamel can occur by:

i. Poor occlusal functions and exaggerated para-functional habits leading to attrition.
ii. Abrasion caused by improper brushing technique and dietary factors.
iii. Erosion which is associated with exposure to acidic environment and dietary components.

Causes of loss of enamel:
- Attrition by exaggerated occlusal functions like bruxism
- Abrasion from dietary components or improper brushing technique
- Erosion associated with environmental or dietary components particularly acids.

Since dentinal tubules get sclerosed of their own and plug themselves up in the oral environment, treatment should focus on eliminating factors associated with continued dentinal exposure.

TREATMENT STRATEGIES

Hypersensitivity can resolve without the treatment or may require several weeks of desensitizing agents before improvement is seen. Treatment of dentin hypersensitivity is challenging for both patient and the clinician mainly for two main reasons:

1. It is difficult to measure or compare pain among different patients.
2. It is difficult for patient to change the habits that initially caused the problem.

Two main principal treatment options are:

1. Plug the dentinal tubules preventing the fluid flow.
2. Desensitize the nerve, making it less responsive to stimulation.

All the current modalities address these two options.

Treatment of Dentine Hypersensitivity can be Divided into:
a. Home care with dentifrices
b. In office treatment procedure
c. Patient education

Management of Dentine Hypersensitivity

1. *Home care with dentifrices*:
   a. Strontium chloride dentifrices
   b. Potassium nitrate dentifrices
   c. Fluoride dentifrices
2. *In office treatment procedure*:
   a. Cavity varnishes
   b. Anti-inflammatory agents.
   c. Treatments that partially obturate dentinal tubules.
      - Burnishing of dentin
      - Silver nitrate
      - Zinc chloride—potassium ferrocyanide
      - Formalin
      - Calcium compounds—Calcium hydroxide
        Dibasic calcium phosphate
      - Fluoride compounds—Sodium fluoride
        Sodium silicofluoride
        Stannous fluoride
      - Iontophoresis
      - Strontium chloride
      - Potassium oxalate
   d. Tubule sealant
      - Restorative resins
      - Dentin bonding agents
   e. Miscellaneous
      - Laser
3. Patient education
   a. Dietary counseling
   b. Tooth brushing technique
   c. Plaque control

Management of Tooth Hypersensitivity

It is well known that hypersensitivity often resolves without treatment. This is probably related to the fact that dentin permeability decrease spontaneously—because of occurrence of natural processes in the oral cavity.

Natural Process Contributing to Desensitization Include the:
1. Formation of reparative dentin by the pulp
2. Obturation of tubules by the formation of mineral deposits (Dental sclerosis)
3. Calculus formation on the surface of the dentin.

A. Home care with dentifrices

Dentifrice has been defined as a substance used with a toothbrush to aid in cleaning the accessible surfaces of the teeth. Dentifrice components include abrasive, surfactant,

50 percent gum camphor was found to be effective in preventing postoperative thermal sensitivity.

The use of corticosteroids is based, on the assumption that dentinal pain is of inflammatory origin; hence, more information is needed regarding the relationship between these two conditions.

Partial Obliteration of Dentinal Tubules

Burnishing of Dentin

Burnishing of dentin with a toothpick or orange wood stick results in the formation of a smear layer which, partially occludes the dentinal tubules, and thus resulting in reduction in hypersensitivity.

Formation of Insoluble Precipitates to Block Tubules

Certain soluble salts react with ions in tooth structure to form crystals on the surface of the dentin. To be effective, crystallization should occur in 1-2 minutes and the crystals should be small enough to enter the tubules and must also be large enough to partially obturate the tubules.

- ***Calcium oxalate dihydrate crystals*** are formed when potassium oxalate is applied to dentin; these crystals are very effective in reducing permeability.
- ***Silver nitrate ($AgNO_3$)*** has ability to precipitate protein constituents of odontoblast processes, thereby partially blocking the tubules.
- ***Zinc chloride – Potassium ferrocyanide*** when applied forms precipitate, which is highly crystalline and covers the dentine surface.
- ***Formalin 40 percent*** is topically applied by means of cotton pellets or orangewood sticks on teeth. It had been proposed by ***Grossman in 1935*** as the desensitizing agent of choice in treating anterior tooth because, unlike $AgNO_3$, it does not produce stain.
- ***Calcium compounds*** have been popular agent for many years for the treatment of hypersensitivity. The exact mechanism of action is unknown but evidence suggests that
  a. It may block dentinal tubules
  b. May promote peritubular dentin formation.
  c. On increasing the concentration of calcium ions around nerve fibers, may results in decreased nerve excitability. So, calcium hydroxide might be capable of suppressing nerve activity.
  - A paste of $Ca(OH)_2$ and sterile distilled water applied on exposed root surface and allowed to remained for 3-5 minutes, can give immediate relief in 75 percent of cases.
  - Dibasic calcium phosphate when burnished with round tooth pick forms mineral deposits near the surface of the tubules and found to be effective in 93 percent of patients.
- ***Fluoride compounds***: Lukomsky (1941) was the first to propose sodium fluoride as desensitizing agent. Because dentinal fluid is saturated with respect to calcium and phosphate ions, application of NaF leads to precipitation of calcium fluoride crystals, thus, reducing the functional radius of the dentinal tubules.
  - *Acidulated sodium fluoride*
    Concentration of fluoride in dentin treated with acidulated sodium fluoride is found to be significantly higher than dentin treated with sodium fluoride.
  - *Sodium silicofluoride*
    Silicic acid forms a gel with the calcium of the tooth, thus produces an insulating barrier. Thus application of 0.6 percent sodium silicofluoride is much more potent than 2 percent solution of sodium fluoride as desensitizing agent.
  - *Stannous fluoride*
    10 percent solution of stannous fluoride forms dense layer of tin and fluoride containing globular particles blocking the dentinal tubules.
    0.4 percent stannous fluoride is also an effective agent however requires prolonged use (up to 4 weeks) to achieve satisfactory results.

Fluoride Iontophoresis-Iontophoresis is a term applied to the use of an electrical potential to transfer ions into the body for therapeutic purposes. The objective of fluoride iontophoresis is to drive fluoride ions more deeply in to the dentinal tubules than can not be achieved with topical application of fluoride alone.

- ***Strontium chloride***: Studies have shown that topical application of concentrated strontium chloride on an abraded dentin surface produces a deposit of strontium that penetrates dentin to a depth of approximately 10-20μm and extend into the dentinal tubules.
- ***Oxalates***: Oxalates are relatively inexpensive, easy to apply and well tolerated by patients. Potassium oxalate and ferric oxalate solution make available oxalate ions that can

react with calcium ions in the dentine fluid to form insoluble calcium oxalate crystals that are deposited in the apertures of the dentinal tubules.

Dental Resins and Adhesives

The objective in employing resins and adhesives is to seal the dentinal tubules to prevent pain producing stimuli from reaching the pulp. Several investigators have demonstrated immediate and enduring relief of pain for periods of up to 18 months following treatment. Although not intended for treatment of generalized areas of root sensitivity, this can be an effective method of treatment when other forms of therapy have failed.

a. Wycoff (1982) advocates the use of adhesives for severe cases of hypersensitivity that do not respond to other therapy. He prefers a glass-ionomer cement because it is hydrophilic acid and conditioning is not required, the material adheres well and it is esthetically pleasing.
b. Copeland (1985) found that application of Scotchbond produced immediate and lasting relief from hypersensitivity. Clinically superior results were obtained by covering the resin with a drop of dilute restorative material.
c. GLUMA is a dentin bonding system that includes gluteraldehyde primer and 35 percent HEMA (hydroxyethyl methacrylate). It provides an attachment to dentin that is immediate and strong. GLUMA has been found to be highly effective when other methods of treatment failed to provide relief (Fig. 26.9).

Fig. 26.9: GLUMA desensitizing solution

Patient Education

Dietary Counseling

Dietary acids are capable of causing erosive loss of tooth structure, thereby removing cementum and resulting in opening of the dentinal tubules. Consequently, dietary counseling should focus on the quantity and frequency of acid intake, and intake occurring in relation to tooth brushing. Any treatment may fail if these factors are not controlled. A written diet history should be obtained on patients with dentinal hypersensitivity in order to advise those concerning eating habits.

Because of the presence of a smear layer on dentin, teeth are not usually sensitive immediately following scaling and root planning. However, removal of the smear layer may result from exposure to certain components of the diet. Studies have shown that citrus fruit juices, apple juice and yoghurt are capable of dissolving the smear layer.

Because loss of dentin is greatly increased when brushing is performed immediately after exposure of the tooth surface to dietary acids, patients should be cautioned against brushing their teeth soon after ingestion of citrus food.

Tooth Brushing Technique

Because incorrect tooth brushing appears to be an etiologic factor in dentin hypersensitivity, instruction about proper brushing techniques can prevent further loss of dentin and the resulting hypersensitivity.

Plaque Control

Saliva contains calcium and phosphate ions and is therefore able to contribute to the formation of mineral deposits with in exposed dentinal tubules. The presence of plaque may interfere with this process, as plaque bacteria, by producing acid, are capable of dissolving any mineral precipitates that form, thus opening tubules.

Recent Developments

Treatment of Dentine Hypersensitivity by Lasers

Kimura Y et al (2000) reviewed treatment of dentine hypersensitivity by lasers. The lasers used for the treatment of dentine hypersensitivity are divided into two groups:

1. Low output power (low level) lasers: helium-neon [He-Ne], and gallium/aluminum/arsenide (GaAlAs) [diode] lasers.

2. Middle output power lasers: Nd:YAG and CO_2 lasers.

Laser effects are considered to be due to the effects of sealing of dentinal tubules, nerve analgesia or placebo effect. The sealing effect is considered to be durable, whereas nerve analgesia or a placebo effects are not.

SUMMARY

Professional interest in the causes and treatment of dentinal hypersensitivity has been evident in the dental literature for approximately 150 years or more. Dentinal hypersensitivity satisfies all the criteria to be classified as a true pain syndrome. Myelinated A fibers mostly Aδ type seems to be responsible for the sensitivity of dentine. Of the various theories proposed, the hydrodynamic theory is the most accepted explanation for the mechanism of dentine hypersensitivity. Management of the condition requires determination of etiologic factors and predisposing influences. Desensitizing tooth pastes containing potassium nitrate, strontium chloride and sodium monofluorophosphate have proven to be effective in the management of hypersensitivity. Partial obturation of open tubules is the most widely practiced in-office treatment of dentinal hypersensitivity.

27

Lasers in Endodontics

- Introduction
- History and Development
- Classification of Laser
- Laser Physics
- Types of Lasers
- Laser Interaction with Biological Tissues
- Laser Safety in Dental Practice
- Soft and Hard Tissue Applications of Lasers in Dentistry

INTRODUCTION

Laser is an acronym for **"Light Amplification by Stimulated Emission of Radiations."** The application of lasers is almost in every field of human endeavor from medicine, science and technology to business and entertainment over the past few years. There has been a strong emergence of lasers in the field of dentistry. When used efficaciously and ethically, they are an exceptional modality of treatment for clinical conditions that dentists treat on a daily basis.

HISTORY AND DEVELOPMENT

Although the history of lasers dates back to 1400BC and the centuries when light was used as a therapeutic agent by ancient Greeks, Chinese, Indians and Egyptians.

1960	Miaman	Developed laser or maser
1961	Snitzer	Neodymium laser
1965	Leon Goldman	Exposure of vital tooth to laser
1965	Taylor et al	Studied histological effects on pulp
1966	Lobene et al	Use of CO_2 lasers in dentistry
1974	Yamamota et al	Nd:YAG in prevention of caries
1977	Lenz et al	First application in oral and maxillofacial surgery
1985	Pick et al	First in periodontal surgery.

The first ***laser or maser*** as it was initially called developed by Theodore H. Maiman in 1960. Maser like laser is an acronym for 'Microwave amplification by stimulated emission of radiation." This laser constructed by Miaman was a pulsed ruby laser.

The second laser to be developed was the neodymium laser by ***Snitzer*** in 1961. The first report of laser exposure to a vital human tooth was given in 1965 by ***Leon Goldman MD.*** The first laser patient experienced no pain with only superficial damage to the crown. Surprisingly the first laser dentist was a physician and the dental laser patient was a dentist. In 1965, ***Taylor and associates*** reported the histologic effect of ruby laser on the dental pulp. From the 1960's to the early 1980's, dental researchers continued to search for other type of lasers. ***Lobene et al*** in 1966 researched more about the CO_2 lasers. Because its wavelength of 10.6 micrometer is well absorbed by enamel, it was thought that the CO_2 laser might be suitable for sealing of pits and fissures, welding of ceramics to enamel or prevention of dental caries.

The first report of dental application of neodymium laser to vital oral tissues in experimental animals was given by Yamamoto et al. They found that Nd:YAG laser was an effective tool for inhibition of progression of incipient caries both *in vitro* and *in vivo*. The first application of laser in maxillofacial surgery was by lenz (lenz et al 1977) who used the argon laser to create a nasoantral window.

The advantages of CO_2 laser was first applied to periodontal surgery by Pick in 1985. He developed a technique for a nearby bloodless gingevictomy in patients with bleeding disorders.

Sufficient research exists to predict that current laser systems such as Erbium:YAG, Holmium:YAG, Nd:YAG and excirmer have the potential to replace the dental drill for a number of uses (Fig. 27.1).

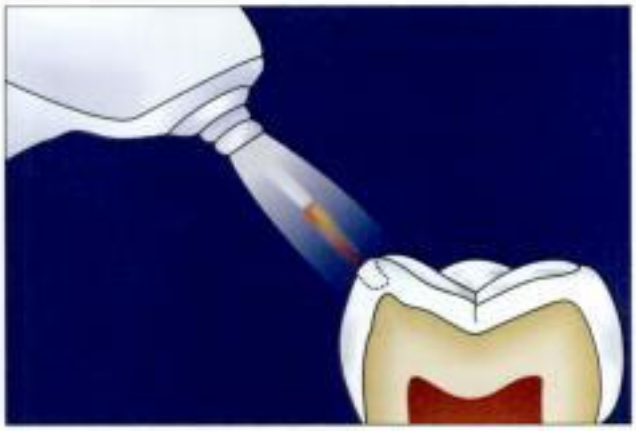


Fig. 27.1: Laser beam produces precised and clean cavity cutting with minimal tooth loss

CLASSIFICATION OF LASER

1. ***According to ANSI and OHSA standards lasers are classified as***:

Class I

These are low powered lasers that are safe to use, e.g. Laser beam pointer

Class II

Low powered visible lasers that are hazardous only when viewed directly for longer than 1000 seconds, e.g. He –Ne lasers.

Class II_b

Low powered visible lasers that are hazardous when viewed for more than 0.25 seconds.

Class III_a

Medium powered lasers that are normally hazardous if viewed for less than 0.25 seconds without magnifying optics.

Class III_b

Medium powered lasers hat can be hazardous if viewed directly.

Class IV

These are high powered lasers (>0.5W) that produce ocular skin and fire hazards.

2. ***Based on the wavelength of the beam***:
   i. **Ultraviolet rays** –140 to 400 nm
   ii. **Visible light** – 400 to 700 nm
   iii. **Infrared** – 700 to microwave spectrum
3. ***Based on penetration power of beam***:
   i. **Hard**: increased penetration power
   For example, Nd: YAG, Argon.
   ii. **Soft lasers**: Decreased penetration power.
   For example, diode, Gallium-Sa, He-Ne lasers.
4. ***Based on pulsing***:
   i. **Pulsed**: The beam is not continuous, i.e. is of short durations.
   ii. **Non pulsed**: The beam is continuous and of fixed duration.
5. ***According to type of laser material used***:
   i. **Gas lasers**: CO_2 lasers, Argon lasers, He-Ne lasers
   ii. **Liquid lasers**: Ions of rare earth or organic fluorescent dyes are dissolved in a liquid, e.g. Dye lasers.
   iii. **Solid state lasers**:
      – Ruby lasers
      – Nd: YAG lasers.
   iv. **Semiconductor lasers**:
      – Gallium
      – Arsenide.

LASER PHYSICS

The basic units or quanta of light are called photons. Photons behave like a tiny wavelets similar to sound wave pulses. A quantum of light can be depicted as an electromagnetic wave with an electric field oscillating up and down. There is also a magnetic field associated with the photon that moves in and out of the page.

The common principles on which all lasers work is the generation of monochromatic, coherent and collimated radiation by a suitable laser medium in an optical resonator (Fig. 27.2).

Common Principles of Laser

- Monochromatic
- Coherence
- Collimation.

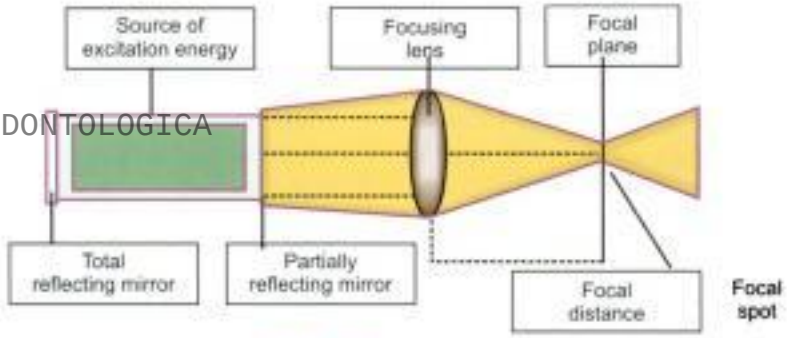


Fig. 27.2: Laser physics

Monochromatic means that the light produced by a particular laser will be of a characteristic wavelength. If the light produced is in the visible spectrum (400 nm to 750 nm), it will be seen as a beam of intense color. It is important to have this property to attain high spectral power density of the laser (Fig. 27.3).

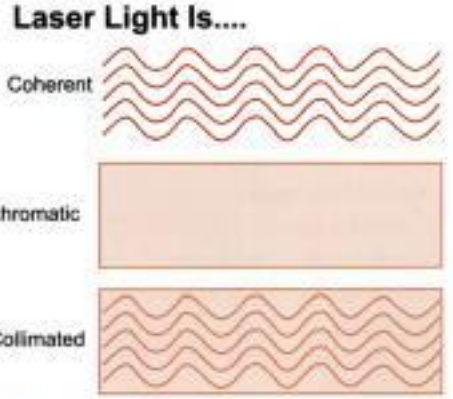


Fig. 27.3: Common principles on which all lasers. Work is generation of monochromatic, coherent and collimated beam

Coherence means that the light is all perfectly in phase as they leave the laser. That means that unlike a normal light source, their individual contributions are summated and reinforce each other. In an ordinary light source, much of the energy is lost as out of phase waves cancel each other.

Collimation means that the laser light beam is perfectly parallel when leaving the laser aperture (Fig. 27.4). This property is important for good transmission through delivery systems.

The main differentiating characteristic of lasers is wave length which depends on the laser medium and excitation diode, i.e. continuous wave or pulsed mode. The different wavelengths can be classified into three groups.

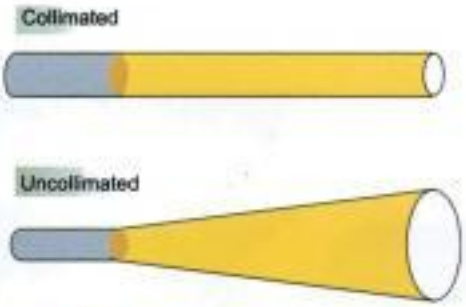


Fig. 27.4: Collimated and uncollimated beam

Ultraviolet (UV range) approx 140-400 µm
Visible light (VIS range) approx 400-700 µm
Infra red (IR range) approx 700-microwave spectrum
The shorter the wavelength, more energetic is the light.

Light Absorption and Emission

When light encounters matter, it can be deflected (reflected or scattered) absorbed (Fig. 27.5). If a photon is absorbed,

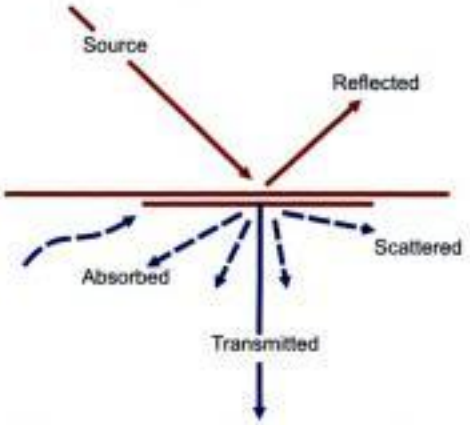


Fig. 27.5: When light encounters matter, it can be reflected, scattered, absorbed or transmitted

its energy is not destroyed, but rather used to increase the energy level of the absorbing atom. The photon then ceases to exist and an electron within the atom jumps to an higher energy level. This is known as pumping up to an excited state from the resting ground state. In the excited state, the atom is unstable and will soon spontaneously decay back to the ground state, releasing stored energy in the form of an emitted photon. This process is called spontaneous emission. The spontaneously emitted photon has a longer wavelength and less energy than the absorbed photon. The difference in the energy is usually turned into heat.

Light Amplification by Stimulated Emission of Radiation

The process of lasing occurs when an excited atom can be stimulated to emit a photon before the process occurs spontaneously. When a photon of exactly the right energy enters the electromagnetic field of an excited atom, the incident photon triggers the decay of the excited electron to a lower energy state. This is accompanied by release of the stored energy in the form of a second photon. The first photon is not absorbed but continues to encounter another excited atom. Stimulated emission can only take place when the incident photon of the identical wavelength traveling in the same direction.

If a collection of atoms is more that are pumped up into the excited state than remain in the resting state, the spontaneous emission of a photon of one atom will stimulate the release of a second photon in a second atom and these two photons will trigger the release of two more photons. These four then yield eight, eight yields sixteen and the cascading reaction follows to produce a brief intense flash of a monochromatic and coherent light

Beam Profile and Spot Geometry

The projection of the beam on the target is called the spot. A cross-section of the beam is called the beam profile. The diameter of the spot is called the spot size.

Power Density

Power density is simply the concentration of photons in a unit area. Photons concentration is measured in watts and area in square cm

Therefore PD = w/cm^2

$= w/\pi r^2$ (r = beam diameter/2)

From the beam profile, we know that the power density in the center of the spot is higher and that at the edge of the spot, it approaches zero.

Power density can be increased significantly by placing a lens in the beam path because the light is monochromatic and collimated.

Power density can be increased by the wattage but increasing the power by 10 changes the power density by 10. But decreasing area by 10 increases the power density by 100.

The size and shape of the lens determine the focal length and the spot size at the focal length.

The term focused and defocused refers to the position of the focal point in relation to the tissue plane. The laser beam can be focused through a lens to achieve a converging beam, which increases in intensity to form a focal spot or hot spot, the most intense part of the beam. Past the focal spot, the beam diverges and the power decreases (Fig. 27.6).

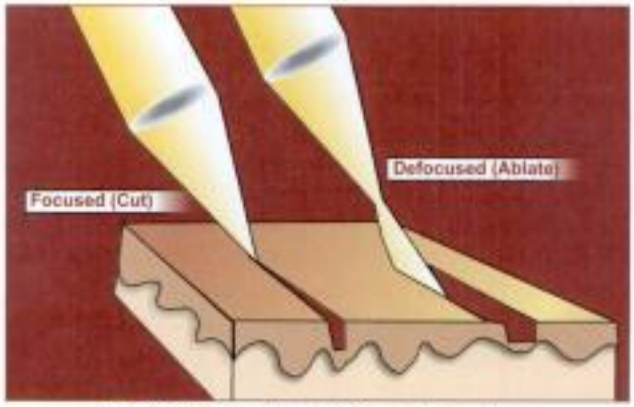


Fig. 27.6: Focused and defocused laser beam

When working on tissue, the laser should always be used either with the focal point positioned at the tissue surface or above the tissue surface. The laser should never be positioned with the focal spot deep or within tissue as this can lead to deep thermal damage and tissue effects.

TYPES OF LASERS

Carbon Dioxide Lasers

It was developed by Patel et al in 1964. It has a wavelength of 10.6 microns and falls into infrared range on the spectrum. In the United States, the CO_2 laser was the first laser approved by the food and drug administration for dental soft- tissue surgical procedures. CO_2 laser energy is readily absorbed by tissues high in water content.

When CO_2 laser is focused to a fine point, its most energy dense state, it can perform fine dissection. As the beam is defocused and widened its effect on the tissue changes. Instead of a definitive cutting action, the laser ablates the

shockwaves can lead to high pressures so that adjacent tissue will be destroyed or damaged.

Chemical Effects

The basis of the photochemical effect is the absorption of laser light without any thermal effect which leads to an alteration in the chemical and physical properties of the irradiated tissue.

Laser Effects on the Dental Pulp

Characteristically pulp tissue can not survive on environment of elevated temperature for protracted periods when tooth structure is irradiated with lasers.

The use of combination of air and water spray before during or immediately after laser irradiation to enamel and dentin may be a more effective method for temperature control and reduction of heat transfer to the pulp and other vital structure surrounding the teeth.

Precooling by air and water spray prior to lasing may be used with laser systems such as CO_2, Holmium, Erbium-which are more readily absorbed by water. Lasers with limited transmission through enamel and dentin may also be effectively cooled with the application of an air water spray immediately after lasing.

LASER SAFETY IN DENTAL PRACTICE

The surgical lasers currently used in dentistry generally fall in class IV category which is considered the most hazardous group of lasers. The types of hazards that may be encountered within the clinical practice of dentistry may be grouped:

1. Ocular injury
2. Tissue damage
3. Respiratory hazards
4. Fire and explosion
5. Electrical shock.

1. ***Ocular Hazards***: Injury to the eye can occur either by direct emission from the laser or by reflection from a mirror like surface. Dental instruments have been capable of producing reflections that may result in tissue damage in both operator and patients.

   Irreversible retinal burns can occur by conversion of incident radiation to heat energy within a fraction of a second resulting in permanent radiation.
2. ***Tissue Hazards***: Laser induced damage to skin and other non-target tissue can result from the thermal interaction of the energy with the tissue proteins .Temperature elevations of 21°C above normal body temperature can produce destruction by denaturation of cellular enzymes and structural proteins which interrupt basic metabolic processes.
3. ***Environmental Hazards***: These secondary hazards belong to a group of potential laser hazards referred to as non-beam hazards. Most surgical lasers used in dentistry are capable of producing smoke, toxic gases and chemicals.

   The generation of smoke during surgery can be a danger to both operator and patient. Inhalation of toxic or infectious matter in the form of aerosols and particles has been found to be potentially damaging to the respiratory system. The greatest producers of smoke are CO_2 and Erbium lasers followed by Nd:YAG lasers.
4. ***Combustion Hazards***:
   Flammable solids, liquids, gases used within the surgical setting can be easily ignited if exposed to the laser beam.
5. ***Electrical Hazards***: Because class IV surgical lasers often use very high currents and high voltage power supplies, there are several associated hazards that may be potentially lethal. Electrical hazards can be in form of electric shock, fire or explosion.

Fire and Electrical Control Measures

To avoid an electrical hazard, the operatory must be kept dry. The control panel and its electrical power unit should be protected from any kind of splashing.

Personal Protective Equipment

Eye Protection

Light produced by all class IV lasers by definition presents a potential hazards for ocular damage by either direct viewing or reflection of the beam. Therefore all people must wear adequate eye protection, including the patient.

When selecting appropriate eye wear several factors should be considered:

1. Wavelength permissible emission
2. Restriction of peripheral vision
3. Maximum permissible exposure limits
4. Degradation of the absorbing media
5. Optical density of the eye wears
6. Need for corrective lenses
7. Comfort and fit.

Control of Air Borne Contamination

Airborne contamination must be controlled by ventilation, evacuation or other method of respiratory protection.

The 308 nm excimer laser is the only system that offers precise ablation of tissue, fiber delivery, bactericidal effects. Good transmission through water and tumefied contact conditioning in one system.

It is useful to use lasers as adjuncts to conventional treatment, but it is not possible to use lasers alone for treatment.

4. ***Treatment of incomplete fracture***:
Lasers are using in repairing incomplete vertical fractures by causing fusion of the fracture.
5. ***Apicoectomy***:
If laser is used for surgery, a bloodless surgical field should be easier to achieve. If the cut surface is irradiated, it gets sterilized and sealed.

Clinically the use of Er;YAG laser resulted in improved healing and diminished postoperative discomfort.

6. ***Treatment of dental hypersensitivity***:
The lasers used for the treatment of dental hypersensitivity are divided into two groups:
   i. Low output power lasers (He-Ne and Ga, Al, As lasers)
   ii. Middle output power lasers-Nd:YAG and CO_2 lasers.

The mechanism causing a reduction in hypersensitivity is most unknown but is thought that the mechanism for each laser is different.

In case of low output power lasers, a small fraction of the lasers energy is transmitted through enamel or dentin to reach the pulp tissue. He:Ne laser affects the peripheral A delta or C fiber nociceptor.

Laser energy of Nd:YAG are indicating thermally mediated effects and pulpal analgesia. Using CO_2 lasers mainly seal the dentinal tubules as well as reduce the permeability.

7. ***Sterilization of instruments***:
Argon, CO_2 and nd:YAG lasers have been used successfully to sterilize dental instruments.

Bleaching

The whitening effect of the laser is achieved by a chemical oxidation process. Once the laser energy is applied H_2O_2 breaks down to H_2O and free O_2 radical which combines with and thus remove stain molecules.

The energy of CO_2 laser is emitted in the form of heat. This energy can enhance the effect of the whitening after initial argon laser process.

CONCLUSION

Laser advantages such as a bloodless operative and postoperative course, usually requiring no suturing, minimal to absent postoperative pain, and high patient acceptance help make lasers a highly advantageous alternative to conventional treatment modalities such as the scalpel or electrosurgery. As more and more clinicians and researchers discover the advantages lasers have to offer, the presence of lasers in the dental office will become increasingly common.

Some clinicians are still doubtful of entering this exciting field because of the size and cost of equipment. Lasers will continue to get smaller in size and less costly, but this is true of all technology—consider the history of computers and pocket calculators. The original lasers were not only large but had six-figure price tags. Today's dental lasers are smaller, lightweight, highly portable and more reasonably priced. The future of dental lasers is bright. When used ethically and efficaciously, it results in increase on income which comes from greater patient acceptance of certain treatments. Patients experience minimal discomfort post-procedure, leading to increased referrals.

28 Pediatric Endodontics

- Introduction
- Importance of Pulp Therapy
- Anatomic Differences between Primary and Permanent Teeth
- Pulp Therapy Procedures
- Indirect Pulp Capping
- Direct Pulp Capping
- Pulpotomy
- Pulpectomy for Primary Teeth
- Apexification

INTRODUCTION

Preservation of dental arch and its functions is main motive behind pediatric dentistry. The retention of the primary teeth is needed until they are naturally exfoliated. There are several advantages of preserving the natural primary teeth (Fig. 28.1). Primary teeth help in preserving the arch length, play an important role in mastication, esthetics, appearance, speech and act as space maintainers for permanent teeth.

Importance of Pulp Therapy

- Maintains arch length
- Prevents abnormal habits
- Maintains esthetics
- Helps in mastication
- Prevents infection
- Prevents speech problems
- Helps in timely eruption of permanent tooth

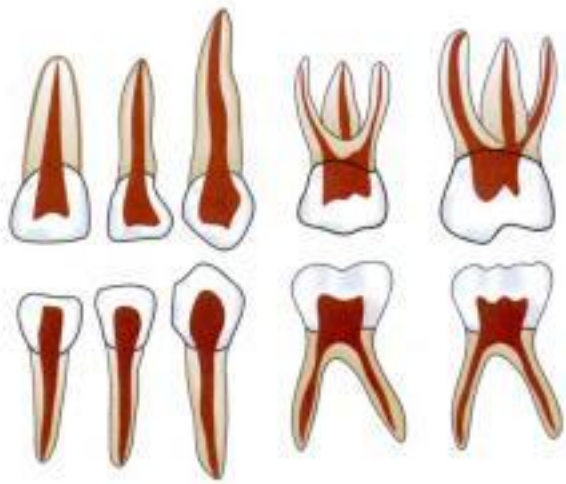


Fig. 28.1: Pulp anatomy of primary teeth

Why Endodontic Treatment of Primary Teeth is Challenging?

Endodontic therapy of primary teeth is considered more challenging and difficult than adults teeth due to various reasons

1. Lesser patient co-operation
2. Reduced month opening
3. More chances of injury to permanent tooth bud
4. Behavioral management
5. Anatomic differences between primary and permanent teeth.

ANATOMY OF PRIMARY TEETH

In primary teeth enamel and dentine are thinner with pulp horns closer to the cusps when compared with permanent teeth (Fig. 28.2). Floor of the pulp chamber is thin and there are more number of accessory canals in primary teeth than in permanent teeth. Roots of the primary teeth are long, slender and flared apically (Fig. 28.3). Roots are in close relation with permanent successor and undergo physiologic resorption during the exfoliation phase.

Anatomic Differences between Primary and Permanent Teeth

1. Pulp horns are closer to cusps in primary teeth than in the permanent tooth particularly mesial pulp horn
2. Enamel and dentin thickness is less in deciduous teeth, thus increases the risk of exposure.
3. Pulp chamber anatomy in primary teeth resembles outer surface of crown and pulp volume is relatively larger than permanent teeth.
4. Roots of primary tooth are longer and slender.
5. Number of accessory canals are more in primary teeth .
6. Roots of primary teeth are more flared apically than permanent teeth.

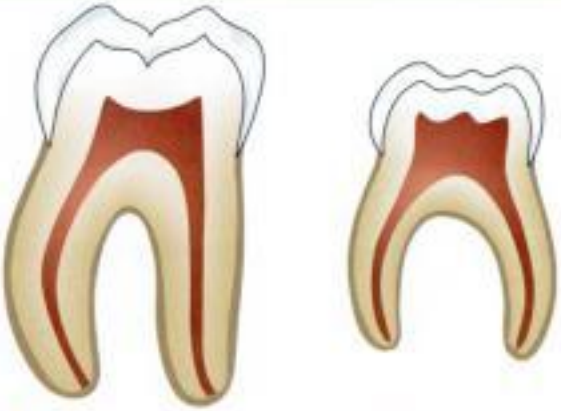


Fig. 28.2: Difference between anatomy of permanent and primary teeth

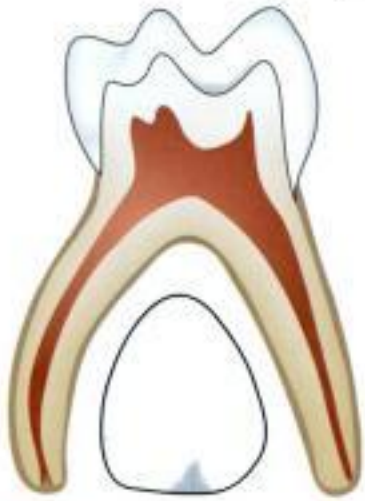


Fig. 28.3: Roots of primary teeth are long, slender and are flared apically

PULP TREATMENT PROCEDURES

Indirect Pulp Capping

Indirect pulp capping is a procedure performed in a tooth with deep carious lesion adjacent to the pulp (Fig. 28.4). In this procedure, caries near the pulp is left in place to avoid pulp exposure and is covered with a biocompatible material.

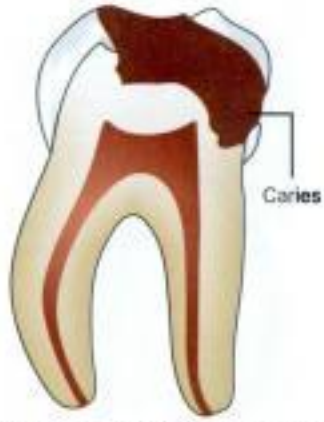


Fig. 28.4: Tooth showing deep carious lesion adjacent to pulp

Indications

1. Deep carious lesion near the pulp tissue but not involving it.
2. No mobility of tooth
3. No history of spontaneous toothache
4. No tenderness to percussion

10. After this, remove the paper point and fill the canal with zinc oxide eugenol cement (Fig. 28.16). Thereafter, tooth is restored with stainless steel crown (Fig. 28.17).

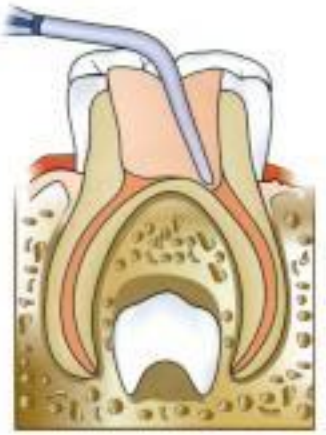


Fig. 28.16: Placement of ZOE cement in the canal

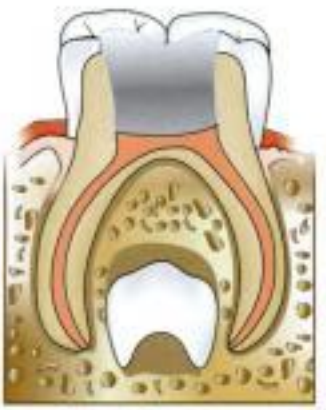


Fig. 28.17: Complete restoration of the tooth

Commonly used material for filling the canals are-

1. Zinc oxide eugenol
2. Iodoform paste
3. $Ca(OH)_2$ and zinc oxide paste.

The main criteria of filling material to be used in deciduous teeth is that it should be resorbable so that it is resorbed along with the roots, so does not interfere with the eruption of the permanent teeth.

The root canal filling material is carried into the pulp chamber and then into the canal with the help of pluggers or lentulospirals. Care should be taken to avoid overfilling of the root canals.

Follow-up after Pulpectomy

Deciduous teeth with pulpectomy should be checked for the success of the treatment. The treatment would be considered successful if:

a. Tooth is asymptomatic.
b. There is absence of pain, sinus, mobility or swelling associated with tooth.
c. Preoperative radiographs show success.
d. There is simultaneous resorption of filling material and the deciduous roots.
e. There is normal radiographic appearance.

APEXIFICATION

Apexification is the process of inducing the development of the root and the apical closure in an immature pulpless tooth with an open apex. It is different from apexogenesis in that in latter root development occurs by physiological process.

In young permanent teeth with nonvital pulp, apexification is advantageous over the conventional root canal treatment because—

a. Apex is funnel shaped with apical part wider than canal.
b. Canal walls are thin and fragile.
c. Absolute dryness of canal is difficult to achieve.

Indications

Young permanent teeth with blunderbass canal and necrotic pulp (Fig. 28.18).

Materials used for Apexification

1. Calcium hydroxide

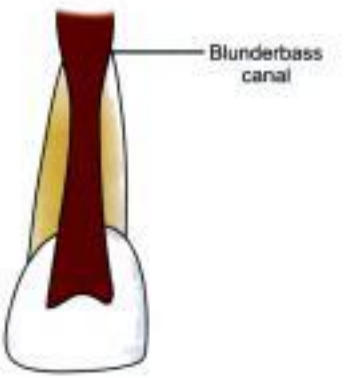


Fig. 28.18: Blunderbass canal

Fig. 28.21: Root-end development in normal pattern

Fig. 28.22: Apex closes but wider at apical end

Fig. 28.23: Development of calcific bridge coronal to apex

Fig. 28.24: Formation of thin barrier close to apex

c. Absence or decrease in mobility.
d. Evidence of firm stop both clinically as well radiographically.

Obturation in such teeth using lateral condensation is not advocated because the lateral pressure during compaction of gutta-percha may fracture the teeth. In such teeth vertical compaction method of obturation is preferred.

Since the dentinal walls are weak in such cases, restoration should be designed to strengthen the tooth. To strengthen the root, gutta-percha should be removed below the alveolar crest, the dentin is acid etched and then composite resin is placed. Placement of posts in such cases should be avoided as far as possible.

MTA a recently introduced material is also used in the apexification procedure. MTA is considered choice of material for apexification because it creates a permanent apical plug at the outset of treatment (Fig. 28.25) to place MTA in the canal isolate the tooth, mix MTA and compact it to the apex of the tooth, creating a 2 mm thickness of plug. Wait for it to set; then fill in the canal with cement and gutta-percha.

Fig. 28.25: Use of MTA in apexification

Basically the rationale behind the pediatric endodontic therapy is to maintain the integrity of dental arch. A successful pediatric outcome should fulfill the following aims:

a. Re-establishment of healthy periodontium tissue
b. Maintaining the primary teeth free of infection and acting as space maintainers for their permanent successors.
c. Maintaining the vitality of pulp in young permanent teeth and thus enhancing the root dentin formation.
d. Freedom from pathologic root resorption.

So we can say that pediatric endodontics may prove helpful in providing the health benefits to the child.

29

Ethics in Endodontics

- Introduction
- Principles of Ethics
- Root Canal Ethics
- Informed Consent
- Dental Negligence
- Malpractice and the Standard of Care
- Abandonment
- Malpractice Cases

INTRODUCTION

Ethics is a moral concept which has been considered worthly of major contemplation since the begining of human life on the earth.

The word "ethics" is derived from a Greek word "Ethos" meaning custom or character.

Nature of Ethics

1. It is related with evaluation of human conduct and standards for judging whether actions performed are right or wrong.
2. It is philosophy of human conduct, away from stating and evaluating principles by which problems of behavior can be solved.
3. It is an attempt to determine the goals of living.

PRINCIPLES OF ETHICS

The principles of ethics for dental profession should be considered as guidelines for the dentist in treating patients. The dentist has obligation to work on some principles for providing service to the patient, community and his profession.

Related to Patient

Primary duty of dentist is to provide proper care to patients irrespective of nationality, socioeconomic status or race, etc.

Dentist should not hesitate in referring specialist for treatment of patient.

Dentist should tell all the possible treatment options available to patient.

Proposed treatment plan and option should be explained to the patient before starting the procedure.

Any complication which may occur during or after the dental procedure should be explained to the patient.

Related to Community

1. Dentist should provide knowledge about prevention, prophylaxis and treatment of dental diseases.
2. Dentists may advance their reputation through professional services to patients and to society and assume a responsible role in the community.

Related to Profession

1. Dentist should update his knowledge and skill by continuing education.
2. Dentist should maintain honor, morality and integrity of profession and should avoid any misconduct.
3. Dentist should have obligation to support advancement of their profession through membership at scientific and professional organization.

ROOT CANAL ETHICS

In present situation, patients really want to know what the problems are, and their solutions. Before commencing a treatment, the dentist should take treatment records as well as informed consent of the patient. These two, treatment records and informed consent are most important tools in prevention and/or defense of dental malpractice claim.

Treatment Records

Each dentist should have standardized protocol for diagnosis and management of pulpal and periapical diseases. In addition to established standardized protocol, the endodontist should have the habit of recording written documentation of treatment provided. It include the following procedure:

1. A detailed written medical history should be taken if medical consultation is required, then consultants remarks should be recorded in the file.
2. The chief complaint of patient should be recorded in his or her own words and treatment should be planned according to that.
3. The dental history of the patient should also be recorded. If any treatment previously given, affects the present outcome of the treatment, it should be explained to the patient. It should also be recorded in the performa.
4. The extra oral and intra oral examination should be conducted and recorded in the performa.
5. An important part of peforma, i.e. examination of affected tooth/teeth should be done thoroughly. Both subjective and objective tests related to diagnosis and treatment should be done and recorded in the performa. If required, a dental specialist can be referred. Radiographs of good diagnostic quality should be made and interpreted. The dentist should record the findings of radiographs in the performa.
6. A detailed pulpal and periodontal examination should be done and recorded in the performa.
7. A proposed treatment plan and provisional diagnosis should be presented to the patient. It should be recorded in the performa also.
8. The medication if prescribed should also be recorded in the dental performa.
9. The informed consent regarding the treatment outcomes should be recorded and included in the performa.
10. The dentist should always sign the performa.

INFORMED CONSENT

As a general rule, the information presented to a patient must be presented in a terminology that is easily understood by the patient. The dentist should tell advantages, risks, and cost related to patient's problems. Informed consent should also be duly signed by the patient and date should also be recorded. Failure to provide the adequate information to the patient is also breach in the code. In the written informed consent should include the following:

1. The diagnosis for each affected tooth should be recorded.
2. The treatment plan should be recorded in brief.
3. The date in which consent is taken, should be recorded in consent form.
4. The potential complication which may occur during or after the treatment should be written in consent form.
5. The success rate of treatment should also be mentioned in the consent form.
6. Alternative treatment options such as tooth extraction or no treatment should be told to patient and it should also be mentioned in the consent form.
7. The patient or his/her guardians should sign the consent form along with date.

No specific form should be used in every case. In endodontics, the incidence of complications is relatively low if done by specialist. The endodontist should tell the patient about the following facts:

1. Despite best efforts by endodontist few cases of root canal failure are reported.
2. Sometimes overextensions occur in root canal therapy. If it is minor, then no treatment is required in these cases because these cases heal well and remain asympto-matic.
3. Slight to moderate pain may occur after root canal therapy.
4. A file may break in canal during root canal therapy then, patient should be informed about this occurence.
5. Perforation may also occur during root canal therapy. Tell the patient about the perforation and explain him/her that it can be repaired with newer materials.

DENTAL NEGLIGENCE

Dental negligence is defined as a violation of the standards of care. Malpractice is lay term for negligence. Dental negligence occur mainly due to two reasons either a clinician does not possess a required qualification or despite of qualification he or she acts carelessly.

Related to Local Anesthesia

There are certain problems which can occur while injecting local anesthesia in the patient. Some cases which can give rise to allegations of negligence are:

1. Syncope (fainting)
2. Fracture of the needle in site.
3. Hematoma
4. Trismus
5. Drug allergy
6. Injection of incorrect solution
7. Infection
8. Injection of expired solution

Syncope (fainting) usually occurs in the dental opd. But this can be reduced if proper counseling of the patient is done before initiating treatment.

Doctor should explain patient about each and every step of dental procedure which he is doing on the patient.

If at all it happens even after taking proper care, the clinician and his assistant should be ready to manage the situation effectively.

Fracture of the Needle *In Situ*

Incidence of fracture of the needle has been reduced in modern era because of wider availability of disposable needles and syringes. In past, reuse of the needles and syringes was the main reason for this problem.

Several other conditions such as hematoma, trismus and drug allergy, may also make the conditions worse for dentist in the dental clinic. So, good communication and rapport between the practitioner and the patient is key in these circumstances to prevent the allegation of negligence.

The injection of an incorrect or expired solution causing harm is considered as an indefensible action. Such occurences should be avoided in the dental office or extra care should be taken during injection of local anesthetic.

Thermal or Chemical Burns

Both thermal or chemical burns are also part of dental negligence.

Thermal Burns

Thermal burns can occur due to overheated instruments such as handpieces or when instruments are insufficiently cooled after sterilization. These can cause burns on the lips, oral mucosa and the lips. To prevent or minimize such occurences.

1. All instruments such as handpiece should be properly maintained and oiling of the handpiece should be done regularly.
2. Burs used in these handpieces should be new and sharp.
3. Excessive pressure should not be applied during cutting.
4. Irrigation with normal saline should be done all the way during cutting of bone.

Any instrument which appears warm to the operator's hands is likely to retain some heat which can cause problem when applied to oral structure immediately. It is usually found that claims based on these findings are difficult to defend. So, these circumstances should be avoided.

Chemical Burns

Chemical burns are also common in the dentist's clinic. These can be avoided by following steps:

1. Provide proper training of dental assistants.
2. Avoid use of strong chemicals in the oral cavity.
3. Avoid overuse of chemicals.
4. Avoid carrying the chemicals over patient's face.
5. Accidental ingestion or inhalation.

Sometimes incidents such as accidental ingestion or inhalation of certain objects may occur for example:

- A portion of tooth.
- Burs
- Endodontic instruments such as file or reamer.
- Bridge

It is on dentist's part to make all provisions so that no instrument or object is ingested or inhaled. To prevent this dentist should take following precautions:

1. Use of rubber dam
2. Use of floss to tie endodontic instruments and rubber dam clamps.

If claims are made for these negligence, heavy compensation has to paid because these cases are truly a case of negligence on the part of dentist.

he/she can be exposed to liability on the basis of abandonment. A dentist/endodontist if wants to ends his or her treatment obligation may have several reasons like patient:

1. Failed to keep appointments.
2. Failed to cooperate.
3. Failed to follow home care instructions.
4. Failed to give payment at time.

To avoid abandonment claim, several prophylactic measures are:

1. No law can force the dentist to do all patients despite severe pain, infection or any other emergency condition. A dentist can do the emergency treatment, if patient and dentist both are interested but dentist should write clearly in the patient's record that he has given emergency treatment only.
2. Reasonable notice should be given to patient if patient is willing to seek endodontic treatment from somewhere else. The dentist should provide copies of treatment record and radiographs.
3. Once treatment is complete and any complication or emergency situation develops not related to the treatment given by the dentist, then there is no law which can force dentist to continue treatment.

Regardless of the justification given for treatment cessation, a dentist/endodontist who fails to follow the proper procedures may incur liability on the ground of abdonment. For prevention of abandonment claim, reasonable notice should be given to the patient. Reasonable notice would be considered valid only when no immediate threat to patient's medical and dental health is found evident. The following points should be taken care of while preparing a notice:

1. Notify the patient that he/she plans to terminate the treatment.
2. Give in detail the reason for not continuing the treatment for example, if patient is not following instruction properly, the notice should include instruction in writing.
3. Give reasonable time to patient to locate a new dentist/endodontist. Time given is usually one month. In rural areas, time limit may be prolonged due to lesser number of dentists available.
4. Provide all details about the treatment, i.e. treatment records and diagnostic radiographs.
5. Dentist should provide emergency care during the intermediate time.
6. A patient can contact any time regarding previous treatment given by dentist.
7. The notice should be certified by the dentist himself mentioning the date and signature.

MALPRACTICE CASES

Injury from Slips of the Drill

A slip of the drill is usually the result of operator's error. It can cause injury to tongue oral mucosa and lips. This injury is like a slip of tongue. To avoid malpractice claim the dentist should follow these steps:

1. Inform the patient about incident and explain that he/she feels sorry for this incident.
2. Refer the patient to an oral and maxillofacial surgeon or plastic surgeon.
3. Dentist should bear the expenditure.
4. Call the patient for periodic check-up.

Inhalation or Ingestion of Endodontic Instruments

Rubber dam should be used in every conditions and its use is mandatory for endodontic work. It not only reduces the chances of aspirating or swallowing endodontic instruments but also reduces the microbial contamination. If patient swallows or aspirates dental instrument, it is operator's fault. He should follow the following steps.

1. Inform the patient about the incident and should regret what has happened.
2. Refer the patient immediately for medical care.
3. Pay all the bills of patient.

Broken File

These incidents usually occur in routine endodontic practice. But to avoid malpractice claims, you have to follow some guidelines. Before going into discussion about these guidelines consider some facts about broken or separated instruments:

1. Multiple use can result in fatigue of the instruments which further lead to failure of the these instruments.
2. Failure to follow the manufactures's instructions regarding use of the instruments can lead to failure.
3. Manufacturing defect may also lead to failure.
4. Teeth with separated files may remain asymptomatic and functional for years.

When an instrument gets separated in a tooth, dentist should follow some guidelines which are as follows.

1. Explain the patient about the incident.
2. Show the remaining part of endodontic instruments to the patient and assure that tooth will remain asymptomatic.
3. Dental assistant should place the part of endodontic instrument and radiographs in the treatment record for future reference.
4. Dentist should reassure the patient that he/she would follow this case closely.

Perforations

Any dentist who is performing endodontic treatment can cause perforation. It usually occurs in or around furcal floor. Despite getting panic at the time of incident dentist should follow some basic steps:

1. Explain the patient about the incident that despite of best effort, perforation has occured.
2. Record the findings in treatment records of the patient.
3. Assure that it can be quickly repaired with newer materials.
4. Follow-up the case regularly.

Overextensions

Overextensions usually happen to every dentist. The irony about overextensions is that no one agree on exactly where the overextensions begin. Does it begin at the apex? one mm beyond the apex or 2 mm?

Rather than going into controversial discussion, we should follow some basic steps which are as follows:

1. Explain the incident to the patient mention the patient that some of the biocompatible material is gone beyond the end of the root.
2. There can be little more soreness for few days.
3. Mostly these cases heal asymptomatically.
4. Follow-up the case closely.

Further Reading

1. Aanderud-Larsen K, Brodin P, Aars H, Skjelbred P: Laser Doppler flowmetry in the assessment of tooth vitality after Le Fort I osteotomy, J Craniomaxillofac Surg 23:391, 1995.
2. Abou-Rass M: The stressed pulp condition: an endodontic-restorative diagnostic concept, J Prosthet Dent 48:264, 1982.
3. Andreasen JO: Atlas of replantation and transplantation of teeth, Philadelphia, 1992, WB Saunders.
4. Augsburger RA, Peters DD: *In vitro* effects of ice, skin refrigerant, and CO_2 snow on intrapulpal temperature, J Endod 7:110, 1981.
5. Baumgartner JC: Treatment of infections and associated lesions of endodontic origin, J Endod 17:418, 1991.
6. Bender IB, Seltzer S: The effect of periodontal disease on the pulp, J Oral Surg 33:458, 1972.
7. Bender IB, Seltzer S: Roentgenographic and direct observation of experimental lesions in bone (part 1), J Am Dent Assoc 62:152, 1961.
8. Bernick S: Effect of aging on the nerve supply to human teeth, J Dent Res 46:694, 1967.
9. Bhaskar SN, Rappaport HM: Dental vitality tests and pulp status, J Am Dent Assoc 86:409, 1973.
10. Cameron CE: Cracked tooth syndrome, J Am Dent Assoc 68:930, 1964.
11. Dal Santl FB, Throckmorton GS, Ellis E III: Reproducibility of data from a hand-held digital pulp tester used on teeth and oral soft tissue, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 72:103, 1992.
12. Degering CI: Radiography of dental fractures, J Oral Surg 30:213, 1970.
13. Drinnan AL: Differential diagnosis of orofacial pain, Dent Clin North Am 31:627, 1987.
14. Ebihara A, Tokita Y, Izawa T, Suda H: Pulpal blood flow assessed by laser Doppler flowmetry in a tooth with a horizontal root fracture, Oral Surg Oral Med Oral Pathol Oral Radiol Endod 81:229, 1996.
15. Fogel HM, Peikoff MD, Christie WH: Canal configuration in the mesiobuccal root of the maxillary first molar: a clinical study, J Endod 20:135, 1994.
16. Fuss Z et al: Assessment of reliability of electrical and thermal pulp testing agents, J Endod 12:301, 1986.
17. Gazelius B, Olgart L, Edwall B, Edwall L: Non-invasive recording of blood flow in human dental pulp, Endod Dent Traumatal 2:219, 1986.
18. Gelfand M, Sunderman EJ, Goldman M: Reliability of radiographical interpretations, J Endod 9:71, 1983.
19. Goho C: Pulse oximetry evaluation of vitality in primary and immature permanent teeth, Pediatr Dent 21:125,1999.
20. Goldman M, Pearson A, Darzenta N: Reliability of radiographic interpretations, J Oral Surg 32:287, 1974.
21. Grossman LI: Endodontic practice, ed 10, Philadelphia, 1981, Lea and Febiger.
22. Hatton JF, Ferrillo PJ, Wagner G, Stewart GP: The effect of condensation pressure on the apical seal, J Endod 14:305, 1988.
23. Hutter JW: Facial space infections of odontogenic origin, J Endod 1-A22, 1991.
24. Ingram TA, Peters DD: Evaluation of the effects of carbon dioxide used as a pulpal test. I. *In vivo* effect on canine enamel and pulpal tissues, J Endod 9:296, 1983.
25. Jacobsen I, Kerekes K: Long-term prognosis of traumatized permanent anterior teeth showing calcifying processes in the pulp cavity, Scand JDent Res 85:588, 1977.
26. Kaffe I, Gratt BM: Variations in the radiographic interpretation of the periapical dental region, J Endod 14:330, 1988.
27. Kaplan AS: History and examination of the orofacial pain patient, Dent Clin North Am 41:155, 1997.
28. Khocht A, Zohn H, Deasy M, Chang KM: Screening for periodontal disease: radiographs vs. PSR, J Am Dent Assoc 127: 749, 1996.
29. Klein H: Pulp response to electrical pulp stimulator in the developing permanent dentition, J Dent Child 45:199, 1978.
30. Kulild JC, Peters DD: Incidence and configuration of canal systems in the mesiobuccal root of the maxillary first and second molars, J Endod 16:311, 1990.
31. Maxwell EH, Braly BV, Eakle WS: Incompletely fractured teeth: a survey of endodontists, Oral Surg Oral Med Oral Pathol 61:113, 1986.
32. Mesaros SV, Trope M: Revascularization of traumatized teeth assessed by laser Doppler flowmetry: case report, Endod Dent Traumatol 13:24, 1997.
33. Miller CA, Leonelli FM, Latham E: Selective interference with pacemaker activity by electrical dental devices, Oral Surg Oral Med Oral Pathol Oral Radial Endod 85:33,1998.
34. Musselwhite JM, Klitzman B, Maixner W, Burkes EJ: Laser Doppler flowmetry: a clinical test of pulpal vitality, Oral Surg Oral Med Oral Pathol Oral Radio Endod 84:411, 1997.
35. Myers J: Demonstration of a possible source of error with an electric pulp tester, J Endod 24:199, 1998.
36. Natkin E, Harrington GW, Mandel MA: Anginal pain referred to the teeth. Report of a case, Oral Surg Oral Med Oral Pathol Oral Radio Endod 40:678, 1975.
37. Neaverth EJ, Kotler LM, Kaltenbach RF: Clinical investigation (*In vitro*) of endodontically treated maxillary first molars, J Endod 13:506, 1987.
38. Oikarinen K, Kopola H, Makiniemi M, Herrala E: Detection of pulse in oral mucosa and dental pulp by means of optical reflection method, Endod Dent Traumatol 12:54, 1996.
39. Okeson JP, Bell WE: Bell's orofacial pains, ed 5, St Louis, 1995, Mosby.
40. Peters DD, Baumgartner JC, Lorton L: Adult pulpal diagnosis. I. Evaluation of the positive and negative responses to cold and electrical pulp tests, J Endod 20:506, 1994.

41. Peters DD et al: Evaluation of the effects of carbon dioxide used as a pulpal test. I. *In vitro* effect on human enamel, J Endod 9:219, 1983.
42. Piazzini LF: Periodontal screening and recording (PSR) application in children and adolescent, J Clin Pediatr Dent 18:165, 1994.
43. Ramadan AE, Mitchell DF: A roentgenographic study of experimental bone destruction, J Oral Surg 15:934, 1962.
44. Ramsay DS, Artun J, Martinten SS: Reliability of pulpal blood-flow measurements utilizing laser Doppler flowmetry, J Den! Res 70:1427, 1991.
45. Rickoff B et al: Effects of thermal vitality tests on human dental pulp, J Endod 14:482, 1988.
46. Robertson A, Andreasen FM, Bergenholtz G, Andreasen JO, Noren JG: Incidence of pulp necrosis subsequent to pulp canal obliteration from trauma of permanent incisors, J Endod 22:557, 1996.
47. Roeykens H, Van Maele G, De Moor R, Martens L: Reliability of laser Doppler flowmetry in a 2-probe assessment of pulpal blood flow, Oral Surg Oral Med Oral Pathol Oral Radial Endod 87:742, 1999.
48. Rubach WE, Mitchell DF: Periodontal disease, age and pulp status, J Oral Surg 19:482, 1965.
49. Rugh JD: Psychological components of pain, Dent Clin North Am 31:579, 1987.
50. Schneider JM, Wallace JA: Pulse oximetry as a diagnostic tool of pulpal vitality, J Endod 17:488, 1991.
51. Schwartz SF, Foster JK: Roentgenographic interpretation of experimentally produced bony lesions (part 1), J Oral Surg 32:606, 1971.
52. Seltzer S, Bender IB, Ziontz M: The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp, Oral Surg Oral Med Oral Pathol 16:846, 1963.
53. Seltzer S: Classification of pulpal pathosis, Oral Surg Oral Med Oral Pathol 34:269, 1972.
54. Smith ON, Walton RE: Periodontal ligament injection: distribution of injected solutions, J Oral Surg 55:232, 1983.
55. Welander U, McDavid WD, Higgins NM, Morris CR: TheB effect of viewing conditions on the perceptibility of radiographic details, Oral Surg Oral Med Oral Pathol Oral Radiol^t Endod 56:651, 1983.
56. Zachrisson BU, Jacobsen I: Long-term prognosis of 66 permanent anterior teeth with root fractures, Scand J Dent Res 83:345, 1975.
57. Aars H, Brodin P, Anderson E: A study of cholinergic and β-adrenergic components in the regulation of blood flow in the tooth pulp and gingiva of man, Acta Physiol Scand 148:441, 1993.
58. Ahlberg K, Brannstrom M, Edwall L: The diameter and number of dentinal tubules in rat, cat, dog and monkey: a comparative scanning electronic microscopic study, Acta Odontol Scand 33:243, 1975.
59. Avery JK, Cox CF, Chiego DJ Jr: Presence and location of adrenergic nerve endings in the dental pulps of mouse molars, Anat Rec 198:59, 1980.
60. Bender IB et al: The optimum placement-site of the electrode in electric pulp testing of the 12 anterior teeth, J Am Dent Assoc 118:305, 1989.
61. Bernick S, Nedelman C: Effect of aging on the human pulp, J Endodl-M, 1975.
62. Bishop MA, Malhotra MP: A investigation of lymphatic vessels in the feline dental pulp, Am JAnat 187:247, 1990.
63. Bishop MA, Malhotra M, Yoshida S: Interodontoblastic collagen (von Korff fibers) and circumpulpal dentin formation: an ultrathin serial section study in the cat, Am JAnat 191:67, 1991.
64. Brannstrom M: Communication between the oral cavity and the dental pulp associated with restorative treatment, Oper Dent 9:57, 1984.
65. Brannstrom M, Astrom A: A study of the mechanism of pain elicited from the dentin, J Dent Res 43:619, 1964.
66. Byers MR, Schatteman GC, Bothwell MA: Multiple functions for NGF-receptor in developing, aging and injured rat teeth are suggested by epithelial, mesenchymal and neural immunoreactivity, Development 109:461, 1990.
67. Byers MR, Sugaya A: Odontoblast process in dentin revealed by fluorescent Di-I, JHistochem Cytochem 43:159, 1995.
68. Byers MR, Taylor PE: Effect of sensory denervation on the response of rat molar pulp to exposure injury, J Dent Res 72: 613, 1993.
69. Kim S: Neurovascular interactions in the dental pulp in health and inflammation, J Endod 14:48, 1990.
70. Kim S, Schuessler G, Chien S: Measurement of blood flow in the dental pulp of dogs with the 133 xenon washout method, Arch Oral Biol 28:501, 1983.
71. Kim S et al: Effects of local anesthetics on pulpal blood flow in dogs, J Dent Res 63:650, 1984.
72. Langeland K, Langeland LK: Histologic study of 155 impacted teeth, Odontol Tidskr 73:527, 1965.
73. Lantelme RL, Handleman SL, Herbison RJ: Dentin formation in periodontally diseased teeth, J Dent Res 55:48, 1976.
74. Lesot H, Osman M, Ruch JV: Immunofluorescent localization of collagens, fibronectin and laminin during terminal differentiation of odontoblasts, Dev Biol 82:371, 1981.
75. Lilja J: Innervation of different parts of the predentin and dentin in a young human premolar, Acta Odontol Scand 37:339, 1979.
76. Lilja J, Noredenvall K-J, Brannstrom M: Dentin sensitivity, odontoblasts and nerves under desiccated or infected experimental cavities, Swed Dent J 6:93, 1982.
77. Linde A: The extracellular matrix of the dental pulp and dentin, J Dent Res 64(special issue):523, 1985.
78. Luthman J, Luthman D, Hokfelt T: Occurrence and distribution of different neurochemical markers in the human dental pulp, Arch Oral Biol 37:193, 1992.
79. Madison S et al: Effect of leukotriene B_4 on intradental nerves, J Dent Res 68(special issue)243:494, 1989.
80. Mangkornkarn C, Steiner JC: *In vivo* and *in vitro* glycosaminoglycans from human dental pulp, J Endod 18:327, 1992.
81. Meyer MW, Path MG: Blood flow in the dental pulp of dogs determined by hydrogen polarography and radioactive microsphere methods, Arch Oral Biol 24:601, 1979.

125. Bystrom A, Claeson R, Sundqvist G: The antibacterial effect of camphorated paramonochlrophenol, camphorated phenol, and calcium hydroxide in the treatment of infected root canals phenol, Endod Dent Traumatol 1:170, 1985.
126. Bystrom A, Happonen RP, Sjogren U, Sundqvist G: Healing of periapical lesions of pulpless teeth after endodontic treatment with controlled asepsis, Endod Dent Taumatol 3:58, 1987.
127. Bystrom A, Sundqvist G: Bacteriological evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy, Scand JDent Res 89:321, 1981.
128. Carlsson J, Frolander F, Sundqvist G: Oxygen tolerance of anaerobic bacteria isolated from necrotic dental pulps, Acta Odontol Scand 35:139, 1977.
129. Christiansen OO: Observations on lesions produced in arteries of dogs by injection of lipids, Arch Pathol 27:1011, 1939.
130. Cohen S, Bigazzi PE, Yoshida T: Similarities of T cell function in cell-mediated immunity and antibody production, Cell Immunol 12:150, 1974.
131. Coleman DL, King RN, Andrade JD: The foreign body reaction: a chronic inflammatory response, J Biomed Mater Res 8:199, 1974.
132. Cotti E, Torabinejad M: Detection of leukotriene C_4 in human periradicular lesions, Int Endod J 27:82, 1994.
133. Cymerman JJ, Cymerman DH, Walters J, Nevins AJ: Human T-lymphocyte subpopulations in chronic periapical lesions, J Endod 10:9, 1984.
134. Dahle UR, Tronstad L, Olsen I: Observation of an unusually large spirochete in endodontic infection, Oral Microbiol Immunol 8:251, 1993.
135. Dahle UR, Tronstad L, Olsen I: Characterization of new periodontal and endodontic isolates of spirochetes, Eur J Oral Sci 104:41, 1996.
136. Dahlen G, Bergenholtz G: Endotoxic activity in teeth with necrotic pulps, J Dent Res 59:1033, 1980.
137. Dahlen G, Fabricius L, Hey den G, Holm SE, Moller AJR: Apical periodontitis induced by selected bacterial strains in root canals of immunized and non-immunized monkeys, Scand J Dent Res 90:207, 1982.
138. Dahlen G, Fabricius L, Holm SE, Moller AJR: Circulating antibodies after experimental chronic infection in the root canal of teeth in monkeys, Scand J Dent Res 90:338, 1982.
139. Hockett RN, Loesche WJ, Sodeman TM: Bacteraemia in asymptomatic human subjects, Arch Oral Biol 22:91, 1977.
140. Holland R et al: Tissue reactions following apical plugging of the root canal with infected dentin chips, Oral Surg Oral Med Oral Pathol 49:366, 1980.
141. Horiba N, Maekawa Y, Matsumoto T, Nakamura H: A study of the detection of endotoxin in the dental wall of infected root canals, J Endod 16:331, 1990.
142. Hughes RA: Focal infection revisited, Br J Rheumatol 33: 370, 1994.
143. Hungate RE: The anaerobic mesophilic cellulolytic bacteria, Bacterial Rev 14:1, 1950.
144. Hunter W: Oral sepsis as a cause of disease, Br Med J 2:215, 1900.
145. Hylton RP, Samules HS, Oatis GW: Actinomycosis: is it really rare? Oral Surg Oral Med Oral Pathol 29:138, 1970.
146. Iwu C, MacFarlane TW, MacKenzie D, Stenhouse D: The microbiology of periapical granulomas, Oral Surg Oral Med Oral Pathol 69:502, 1990.
147. Jones OJ, Lally ET: Biosynthesis of immunoglobulin isotopes in human periapical lesions, J Endod 8:672, 1980.
148. Kakehashi S, Stanley HR, Fitzgerald RJ: The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats, Oral Surg Oral Med Oral Pathol 20:340, 1965.
149. Kantz WE, Henry CA: Isolation and classification of anaerobic bacteria from intact pulp chambers of non vital teeth in man, Arch Oral Biol 19:91, 1974.
150. Kapsimalis P, Garrington GE: Actinomycosis of the periapical tissues, Oral Surg Oral Med Oral Pathol 26:374, 1968.
151. Kerekes K, Tronstad L: Long-term results of endodontic treatment performed with standardized technique, J Endod 5:83, 1979.
152. Killian M: Degradation of human immunoglobulins A_1, A_2 and G by suspected principal periodontal pathogens, Infect Immun 34:57, 1981.
153. Klevant FJH, Eggink CO: The effect of canal preparation on periapical disease, Int Endod J 16:68, 1983.
154. Kopp W, Schwarting R: Differentiation of T-lymphocyte subpopulations, macrophages, HLA-DR-restricted cells of apical granulation tissue, J Endod 15:72, 1989.
155. Koppang HS, Koppang R, Solheim T, Aarneals H, Stolen S: Cellulose fibers from endodontic paper points as an etiologic factor in postendodontic periapical granulomas and cysts, J Endod 15:369, 1989.
156. Koppang HS, Koppang R, Solheim T, Aarnes H, Stolen S: Identification of cellulose fibers in oral biopsis, Scand J Dent Res 95:165, 1987.
157. Koppang HS, Koppang R, Stolen S: Identification of common foreign material in postendodontic granulomas and cysts, J Dent Assoc S Afr 47:210, 1992.
158. Kuntz DD, Genco RJ, Guttuso J, Natiella JR: Localization of immunoglobulins and the third component of complement in dental periapical lesions, J Endod 3:68, 1977.
159. Lalonde ER: A new rationale for the management of periapical granulomas and cysts. An evaluation of histopathological and radiographic findings, J Am Dent Assoc 80:1056, 1970.
160. Lalonde ER, Luebke RG: The frequency and distribution of periapical cysts and granulomas, Oral Surg Oral Med Oral Pathol 25:861, 1968.
161. Langeland MA, Block RM, Grossman LI: A histopathologic and histobacteriologic study of 35 periapical endodontic surgical specimens, J Endod 3:8, 1977.
162. Langworth BF: Fusobacterium necrophorum: its characteristics and role as an animal pathogen, Bacterial Rev 41:373, 1977.
163. Laux M, Abbott P, Pajarola G, Nair PNR: Apical inflammatory root resorption: a correlative radiographic and histological assessement, Int Endod J 33:483, 2000.

164. Lerner UH: Regulation of bone metabolism by the kallikrein-kinin system, the coagulation cascade, and acute phase reactions, Oral Surg Oral Med Oral Pathol 78:481, 1994.
165. Lew M, Keudel KC, Milford AF: Succinate as a growth factor for Bacteroides melaninogenicus, J Bacteriol 108:175, 1971.
166. Lim CG, Torabinejad M, Kettering J, Linkhardt TA, Finkelman RD: Interleukin Ib in symptomatic and asymptomatic human periradicular lesions, J Endod 20:225, 1994.
167. Lin LM, Pascon EA, Skribner J, Gangler P, Langeland K: Clinical, radiographic, and histologic study of endodontic treatment failures, Oral Surg Oral Med Oral Pathol 71:603, 1991.
168. Linenberg WB, Waldron CA, DeLaune GF: A clinical roentgenographic and histopathologic evaluation of periapical lesions, Oral Surg Oral Med Oral Pathol 17:467,1964.
169. Listgarten MA: Structure of the microflora associated with periodontal health and disease in man. A light and electron microscopic study, J Periodontol 47:1, 1976.
170. Nair PNR, Schmid-Meier E: An apical granuloma with epithelial integument, Oral Surg Oral Med Oral Pathol 62: 698, 1986.
171. Nair PNR, Schroeder HE: Periapical actinomycosis, J Endod 10:567, 1984.
172. Nair PNR, Schroeder HE: Epithelial attachment at diseased human tooth-apex, J Periodontal Res 20:293, 1985.
173. Nair PNR, Sjogren U, Kahnberg KE, Krey G, Sundqvist G: Intraradicular bacteria and fungi in root-filled, asymptomatic human teeth with therapy-resistant periapical lesions: a long-term light and electron microscopic follow-up study, J Endod 16:580, 1990.
174. Nair PNR, Sjogren U, Krey G, Sundqvist G: Therapy-resistant foreign-body giant cell granuloma at the periapex of a root-filled human tooth, J Endod 16:589, 1990.
175. Nair PNR, Sjogren U, Schumacher E, Sundqvist G: Radicular cyst affecting a root-filled human tooth: A long-term post-treatment follow-up, Int Endod J 26:225, 1993.
176. Nair PNR, Sjogren U, Sundqvist G: Cholesterol crystals as an etiological factor in non-resolving chronic inflammation: an experimental study in guinea pigs, Eur J Oral Sci 106:644, 1998.
177. Newman HN: Focal infection, J Dent Res 75:1912, 1996.
178. Nilsen R, Johannessen A, Skaug N, Matre R: In situ characterization of mononuclear cells in human dental periapical lesions using monoclonal antibodies, Oral Surg Oral Med Oral Pathol 58:160, 1984.
179. Nobuhara WK, Del Rio CE: Incidence of periradicular pathoses in endodontic treatment failures, J Endod 19:315,1993.
180. O'Grady JF, Reade PC: Periapical actinomycosis involving Actinomyces israelii, J Endod 14:147, 1988.
181. Offenbacher S et al: Potential pathogenic mechanisms of periodontitis-associated pregnancy complications, Ann Periodontol 3:233, 1998.
182. Okiji T, Morita I, Sunada I, Murota S: The role of leukotriene B_4 in neutrophil infiltration in experimentally induced inflammation of rat tooth pulp, J Dent Res 70:34, 1991.
183. Oppenheimer S, Miller GS, Knopf K, Blechman H: Periapical actinomycosis, Oral Surg Oral Med Oral Pathol 46:101, 1978.
184. Papadimitriou JM, Ashman RB: Macrophages: current views on their differentiation, structure and function, Ultrastruct Pathol 13:343, 1989.
185. Patterson SS, Shafer WG, Healey HJ: Periapical lesions associated with endodontically treated teeth, J Am Dent Assoc 68: 191, 1964.
186. Perez F, Galas P, de Falguerolles A, Maurette A: Migration of a Streptococcus sanguis through the root dentinal tubules, J Endod 19:297, 1993.
187. Piattelli A, Artese L, Rosini S, Quarenta M, Musiani P: Immune cells in periapical granuloma: morphological and immunohistochemical characterization, J Endod 17:26,1991.
188. Pitt-Ford TR: The effects of the periapical tissues of bacterial contamination of the filled root canal, Endod J 15:16, 1982.
189. Priebe WA, Lazansky JP, Wuehrmann AH: The value of the roentgenographic film in the differential diagnosis of periapical lesions, Oral Surg Oral Med Oral Pathol 7:979, 1954.
190. Pulver WH, Taubman MA, Smith DJ: Immune components in human dental periapical lesions, Arch OralBiol 23:435,1978.
191. Rietschel ET, Brude H: Bacterial endotoxins, Sci Am 267:54, 1992.
192. Robinson HBG, Boling LR: The anachoretic effect in pulpitis. Bacteriologic studies, J Am Dent Assoc 28:268, 1941.
193. Rosebury T, Reynolds JB: Continuous anaerobiosis for cultivation of spirochetes, Proc Soc Exp Biol Med 117:813, 1964.
194. Rosenow EC: The relation of dental infection to systemic disease, Dent Cosmos 59:485, 1917.
195. Ryan GB, Majno G: Acute inflammation, Am J Pathol 86: 185,1977.
196. Safavi KE, Rossomando ER: Tumor necrosis factor identified in periapical tissue exudates of teeth with apical periodontitis, J Endod 17:12, 1991.
197. Sahara N et al: Odontoclastic resorption of the superficial nonmineralized layer of predentine in the shedding of human deciduous teeth, Cell Tissue Res 277:19, 1994.
198. Sakellariou PL: Periapical actinomycosis: report of a case and review of the literature, Endod Dent Traumatol 12:151, 1996.
199. Samanta A, Malik CP, Aikat BW: Periapical actinomycosis, Oral Surg Oral Med Oral Pathol 39:458, 1975.
200. Samuelsson B: Leukotrienes: mediators of immediate hypersensitivity reactions and inflammation, Science 220:268, 1983.
201. Schein B, Schilder H: Endotoxin content in endodontically involved teeth, J Endod 1:19, 1975.
202. Sedgley CM, Messer H: Long-term retention of a paper-point in the periapical tissues: a case report, Endod Dent Traumatol 9:120, 1993.
203. Sundqvist G, Figdor D, Persson S, Sjogren U: Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative re-treatment, Oral Surg Oral Med Oral Pathol 85:86, 1998.
204. Sundqvist G, Johansson E, Sjogren U: Prevalence of black pigmented Bacteroides species in root canal infections, J Endod 15:13, 1989.
205. Sundqvist G, Reuterving CO: Isolation of Actinomyces israelii from periapical lesion, J Endod 6:602, 1980.

286. Yoshida M et al: Correlation between clinical symptoms and microorganisms islolated from root canals of teeth with periapical pathosis, J Endod 13(1):24, 1987.
287. Yun MW, Hwang CE, Lui CC: Cavernous sinus thrombus following odontogenic and cervicofacial infection, Eur Arch Othorhinolarygol 248:422, 1991.
288. Rass M, Bogen G: Microorganisms in closed periapical lesions, Int EndodoJ 31:39, 1998.
289. Absi EG, Addy M, Adams D: Dentine hypersensitivity. a study of the patency of dentinal tubules in sensitive and nonsensitive cervical dentine, J Clin Periodontol 14:280, 1987.
290. Addy M: Etiology and clinical implications of dentin hypersensitivity, Dent Clin North Am 34:503, 1990.
291. Aderhold L, Konthe H, Frenkel G: The bacteriology of dentigerous pyogenic infections, Oral Surg Oral Med Oral Pathol 52:583, 1981.
292. Ahlquist M, Franzen O, Coffey J, Pashley D: Dental pain evoked by hydrostatic pressures applied to exposed dentin in man: a test of the hydrodynamic theory of dentin sensitivity, JEndod 20:130, 1994.
293. Ahlquist M, Franzen O: Pulpal ischemia in man: effects on detection threshold, A-delta neural response and sharp dentinal pain, Endod Dent Traumatol 15:6, 1999.
294. Ardekian L et al: Burkitts lymphoma mimicking an acute dentoalveolar abscess, J Endod 22:697, 1996.
295. Balaban FS, Skidmore AE, Griffin JA: Acute exacerbations following initial treatment of necrotic pulps, J Endod 10:78, 1984.
296. Barr CE: Practical considerations in the treatment of the HIV-infected patient, Dent Clin North Am 38:403, 1994.
297. Battrum DE and Guttman JL: Phantom tooth pain: a diagnosis of exclusion, Int Endod J 29:190, 1996.
298. Baumgartner JC: Treatment of infections and associated lesions of endodontic origin, J Endod 17:418, 1991.
299. Gatewood RS, Himel VT, Dorn SO: Treatment of the endodontic emergency: a decade later, J Endod 16:284, 1990.
300. Gatot A, Arbelle J, Leiberman A, Yanai-Inbar I: Effects of sodium hypochlorite on soft tissues after its inadvertent injection beyond the root apex, J Endod 17: 573, 1991.
301. Georgopoulou M, Anastassiadis P, Sykaras S: Pain after chemiomechanical preparation, Int Endod J 19:309, 1986.
302. Georgopoulou M, Kontakiotis E, Nakou M: *In vitro* evaluation of the effectiveness of calcium hydroxide and paramonochlorophenol on anaerobic bacteria from the root canal, Endod Dent Traumatol 9:249, 1993.
303. Gilad JZ et al: Development of a clindamycin-impregnated fiber as an intracanal medication in endodontic therapy, J Endod 25:722, 1999.
304. Glassman G et al: A prospective randomized double-blind trial on efficacy of dexamethasone for endodontic interappointment pain in teeth with asymptomatic inflamed pulps, Oral Surg Oral Med Oral Pathol 67:96, 1989.
305. Goerig AC, Michelich RJ, Schulz HH: Instrumentation of root canals in molars using the step-down technique, J Endod 8:550, 1982.
306. Goon WWY, Jacobsen PL: Prodromal odontalgia and multiple devitalized teeth caused by a herpes zoster infection of the trigeminal nerve: report of case, J Am Dent Assoc 116: 500, 1988.
307. Green BL, Green ML, Mcfall WT: Calcium hydroxide and potassium nitrate as desensitizing agents for hypersensitive root surfaces, JPeriodontol 48:667, 1977.
308. Hargreaves KM, Troullos ES, Dionne RA: Pharmacologic rationale for the treatment of acute pain, Dent Clin North Am 31:675, 1987.
309. Harrington GW, Natkin E: Midtreatment flare-ups, Dent Clin North Am 36:409, 1992.
310. Harrison JW, Baumgartner JC, Svec TA: Incidence of pain associated with clinical factors during and after root canal therapy. II. Postobturation pain, J Endod 9:434, 1983.
311. Harrison JW: The appropriate use of antibiotics in dentistry: endodontic indications, Quintessence Int 28:827, 1997.
312. Harrison JW, Svec TA: The beginning of the end of the antibiotic era? I. The problem: abuse of the "miracle drugs," Quintessence Int 29:151, 1998.
313. Hasselgren G, Olsson B, Cvek M: Effects of calcium hydroxide and sodium hypochlorite on the dissolution of necrotic porcine muscle tissue, J Endod 14:125, 1988.
314. Hiatt WH: Incomplete crown root fracture and pulpal—periodontal disease, J Periodontol 44:4, 1975.
315. Hirata T et al: Dentinal fluid movement associated with loading of restorations, J Dent Res 70:975, 1991.
316. Holland GR: Steroids reduce the periapical inflammatory and neural changes after pulpectomy, J Endod 22:455, 1996.
317. Holmes-Johnson E, Geboy M, Getka EJ: Behavior considerations, Dent Clin North Am 30:391, 1986.
318. Hong D, Byers MR, Oswald RJ: Dexamethasone treatment reduces sensory neuropeptides and nerve sprouting reactions in injured teeth, Pain 55:171, 1993.
319. Hutter JW: Facial space infections of odontogenic origin, JEndod 17:422, 1991.
320. Ikeda H, Suda H: subjective sensation and objective neural discharges recorded from clinically nonvital intact teeth, JEndod 24:552, 1998.
321. Imura N, Zuolo ML: Factors associated with endodontic flare-ups: a prospective study, Int Endod J 28:261, 1995.
322. Ishikawa K et al: Occlusion of dentinal tubules with calcium phosphate using acidic calcium phosphate solution followed by neutralization, J Dent Res 73:1197, 1994.
323. Isidor F, Brodum K, Raunholt G: The influence of post length and crown ferrule length on the resistance to cyclic loads of bovine teeth with prefabricated titanium posts, Int J Oral Prosthodont 12:78, 1999.
324. Jackson DL, Moore PA, Hargreaves KM: Postoperative nonsteroidal anti-inflammatory medication for the prevention of postoperative dental pain, J Am DentAssoc 119:641, 1989.
325. Javid B, Barkhorder RA, Bhinda SV: Cyanacrylate—a new treatment for hypersensitive dentin and cementum, J Am Dent Assoc 114:486, 1987.
326. Jeansonne MJ, White RR: A comparison of 2.0% chlorhexidine gluconate and 5.25% sodium hypochlorite as antimicrobial endodontic irrigants, J Endod 20:276, 1994.

327. Jyvasjarvi E, Kniffki KD: Cold stimulation of teeth: a comparison between the responses of cat intradental A and C fibers and human sensations, J Physiol 391:193, 1987.
328. Katebzadeh N, Hupp J, Trope M: Histological repair after obturation of infected root canals in dogs, J Endod 25:364, 1999.
329. Kaufman E et al: Intraligamentary injection of slow-release methylprednisolone for the prevention of pain after endodontic treatment, Oral Surg Oral Med Oral Pathol 77:651, 1994.
330. Kim S: Microcirculation of the dental pulp in health and disease, J Endod 11:465, 1985.
331. Kim S: Neurovascular interactions in the dental pulp in health and inflammation, J Endod 16:48, 1990.
332. Klepac RK et al: Reports of pain after dental treatment, electrical tooth stimulation and cutaneous shock, J Am DentAssoc 100:692, 1980.
333. Kontakiotis E, Nakou M, Georgopoulou M: *In vitro* study of the indirect action of calcium hydroxide on the anaerobic flora of the root canal system, Int Endod J 28:2%5, 1995.
334. Krasner P, Jackson E: Management of posttreatment endodontic pain with oral dexamethasone: a double-blind study, Oral Surg Oral Med Oral Pathol 62:187, 1986.
335. Laskin DM: Anatomic considerations in diagnosis and treatment of odontogenic infections, J Am Dent Assoc 69:38, 1964.
336. Law AS, Lily JP: Trigeminal neuralgia mimicking odontogenic pain, Oral Surg Oral Med Oral Pathol 80:96, 1995.
337. Li X, Tronstad L, Olsen I: Brain abscess caused by oral infection, Endod Dent Traumatol 15:95, 1999.
338. Lilly JP, Law AS: Atypical odontalgia misdiagnosed as odontogenic pain: a case report and discussion of treatment, J Endod 23:337, 1997.
339. Reeh ES, Messer HH: Long-term paresthesia following inadvertent forcing of sodium hypochlorite through perforation in maxillary incisor, Endod Dent Traumatol 5:200, 1989.
340. Rees RS, Harris M: Atypical odontalgia, BrJOral Maxillofac Surg 16:212, 1979.
341. Rehman K, Saunders WP, Foye RH, Sharkley W: Calcium ion diffusion from calcium hydroxide-containing materials in endodontically treated teeth: an *in vitro* study, Int Endod J 29:271, 1996.
342. Roane JB, Dryden JA, Grimes EW: Incidence of postoperative pain after single- and multiple-visit endodontic procedures, Oral Surg Oral Med Oral Pathol 55: 68, 1983.
343. Rogers MJ, Johnson BR, Remeikis NA, BeGole EA: Comparison of the effect of intracanal use of ketorlac tromethamine and dexamethasone with oral ibuprofen on post treatment endodontic pain, J Endod 25:381, 1999.
344. Rosenberg PA, Babick PJ, Schertzer L, Leung A: The effect of occlusal reduction on pain after endodontic instrumentation, J Endod 24:492, 1998.
345. Rugh JD: Psychological components of pain, Dent Clin North Am 31:579, 1987.
346. Ruiz-Hubbard EE, Guttman JL, Wagner, MJ: A quantitative assessment of canal debris forced periapically during root canal instrumentation using two different techniques, J Endod 13:554, 1987.
347. Sadeghein A, Shahidi N, Dehpour AR: A comparison of ketorlac tromethamine and acetaminophen codeine in the management of acute apical periodontitis, J Endod 25:257, 1999.
348. Safavi KE, Nichols FC: Alteration of biological properties of bacterial lipopolysaccharide by calcium hydroxide treatment, J Endod 20:127, 1994.
349. Safavi KE, Spangberg LSW, Langeland K: Root canal tubule disinfection, J Endod 16:207, 1990.
350. Salzgeber RM, Brilliant JD: An *in vivo* evaluation of the penetration of an irrigating solution in root canals, J Endod 3:394, 1977.
351. Sandler NA, Ziccardi V, Ochs M: Differential diagnosis of jaw pain in the elderly, J Am Dent Assoc 126:1263, 1995.
352. Scholz A et al: Complex blockade of TTX-resistant NA+ currents by lidocaine and bupivicaine reduce firing frequency in DRG neurons, J Neurophysiol 79:1746, 1998.
353. Schwartz S, Cohen S: The difficult differential diagnosis, Dent Clin North Am 36:279, 1992.
354. Segura JJ et al: Calcium hydroxide inhibits substrate adherence capacity of macrophages, J Endod 23:444, 1997.
355. Seka W et al: Light deposition in dentinal hard tissue and simulated thermal response, J Dent Res 74:1086, 1995.
356. Selbst AG: Understanding informed consent and its relationship to the incidence of adverse treatment events in conventional endodontic therapy, J Endod 16:387, 1990.
357. Selden HS: The endoantral syndrome, J Endod 3:462, 1977.
358. Selden HS, Manhoff PT, Hatges NA, Michel RC: Metastatic carcinoma to the mandible that mimicked pulpal/periodontal disease, J Endod 24:267, 1998.
359. Seltzer S: Long-term radiographic and histological observations of endodontically treated teeth, J Endod 25:818, 1999.
360. Seltzer S, Bender IB, Zsontz M: The dynamics of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp, Oral Surg Oral Med Oral Pathol 16:846, 1963.
361. Seltzer S, Boston D: Hypersensitivity and pain induced by operative procedures and the cracked tooth syndrome, General Dentistry 45:148, 1997.
362. Seltzer S, Naidorf IJ: Flare-ups in endodontics. I. Etiological factors, J Endod 11:472, 1985.
363. Sen BH, Safavi KE, Spangberg LSW: Antifungal effects of sodium hypochlorite and chlorhexidine in root canals, J Endod, 25:235, 1999.
364. Sessle BJ: Neurophysiology of orofacial pain, Dent Clin North Am 31:595, 1987.
365. Sigurdsson A, Jacoway JR: Herpes zoster infection presenting as an acute pulpitis, Oral Surg Oral Med Oral Pathol 80:92, 1995.
366. Sigurdsson A, Stancill R, Madison S: Intracanal placement of $Ca(OH)_2$: a comparison of techniques, J Endod 18:367, 1992.
367. Siqueira JF Jr, De Uzeda M: Disinfection by calcium hydroxide pastes of dentinal tubules infected with two obligate and one facultative anaerobic bacteria, J Endod 22:674, 1996.
368. Sjogren U, Figdor D, Spangberg L, Sundqvist G: The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing, Int Endod J24:19, 1991.

369. Sjogren U, Figdor D, Persson S, Sundqvist G: Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis, Int Endod J 30:297, 1997.
370. Sodium A et al: Toxicity of camphorated phenol and camphorated parachlorophenol in dental pulp cell culture, J Endod 22:284, 1996.
371. Stabholz A et al: Efficacy of XeCl 308 nm excimer laser in reducing dye penetration through coronal dentinal tubules, J Endod 21:266, 1995.
372. Staehle HJ, Thoma C, Muller HP: Comparative *in vitro* investigation of different methods for temporary root canal filling with aqueous suspensions of calcium hydroxide, Endod Dent Traumatol, 13:106, 1997.
373. Sundqvist G: Ecology of the root canal flora, J Endod 18:427, 1992.
374. Sundqvist G et al: Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative retreatment, Oral Surg Oral Med Oral Pathol 85:86, 1998.
375. Sundqvist G, Johansson E, Sjogren U: Prevalence of black-pigmented bacteroides species in root canal infections, J Endod 15:13, 1989.
376. Takahashi K: Changes in the pulp vasculature during inflammation, J Endod 16:92, 1990.
377. Takahashi K, Macdonald FD, Kinane DF: Detection of IgA subclasses and J chain mRNA bearing plasma cells in human dental periapical lesions by in situ hybridization, J Endod 23:513,1997.
378. Tamse A, Fuss Z, Lustig J, Kaplavi J: An evaluation of endodontically treated vertically fractured teeth, J Endod 25: 506, 1999.
379. Tarbet W, Silverman G, Fraterangelo PA, Kanapfa JA: Home treatment for dentinal hypersensitivity: a comparative study, J Am Dent Assoc 105:227, 1982.
380. Wright EF, Gullickson DC: Identifying acute pulpalgia as a factor in TMD pain, J Am Dent Assoc 127:773, 1996.
381. Yang SF, Rivera EM, Baumgardner KR, Walton RE, Stanford C: Anaerobic tissue-dissolving abilities of calcium hydroxide and sodium hypochlorite, J Endod 21:613, 1995.
382. Yoshiyama M et al: Treatment of dentin hypersensitivity: effect of a light-curing resin liner on tubule occlusion, J Jpn Conserv Dent 34:76, 1991.
383. Zhang C et al: Effects of CO_2 laser in treatment of cervical dentinal hypersensitivity, J Endod 24:595, 1998.
384. Yang SF, Rivera EM, Baumgardner KR, Walton RE, Stanoot ford C: Anaerobic tissue-dissolving abilities of calcium hydroxide and sodium hypochlorite, J Endod 21:63, 1995.
385. Zhang C et al: Effects of CO_2 laser in treatment of cervical dentinal hypersensitivity, J Endod 24:595, 1998.
386. Bender IB, Naidorf IJ, Garvey GJ: Bacterial endocarditis: a consideration for physician and dentist, J Am Dent Assoc 109: 415, 1984.
387. Bystrom A, Sundqvist G: The antibacterial action of sodium hypochlorite and EDTA in 60 cases of endodontic therapy, Int Endod J 35, 1985.
388. Bystrom A, Sundquist G: Bacteriologic evaluation of the effect of 0.5 % sodium hypochlorite in endodontic therapy, Oral Surg Oral Med Oral Pathol 55:307, 1983.
389. Fava LRG: One appointment root canal treatment: incidence of postoperative pain using a modified double-flapped technique, Int EndodJ 24:258, 1991.
390. Freeman JP, Brand JW: Radiation doses of commonly used dental radiographic surveys, Oral Surg Oral Med Oral Pathol 77:285, 1994.
391. Hasselgren G, Reit C: Emergency pulpotomy: pain relieving effect with and without the use of sedative dressings, J Endod 15:254, 1989.
392. Imura N, Zuolo ML: Factors associated with endodontic flare-ups: a prospective study, Int Endod J, 28:261, 1995.
393. Little JW, Palace DA, Miller CS, Rhodus NL: Dental management of the medically compromised patient, ed 5, St Louis, 1997, Mosby.
394. Matsumoto T et al: Factors affecting successful prognosis of root canal treatment, J Endod 13:239, 1987.
395. Mole RH: Radiation effects on prenatal development and their radiological significance, Br J Radiol 52:89, 1979.
396. Natkin E, Oswald RJ, Games LI: The relationship of lesion size to diagnosis, incidence and treatment of periapical cysts and granulomas, Oral Surg Oral Med Oral Pathol 51:82, 1984.
397. Pallasch TJ: Antibiotic prophylaxis: theory and reality, J Calif Dent Assoc 17:27, 1989.
398. Pekruhn BP: Single-visit endodontic therapy: a preliminary clinical study, J Am Dent Assoc 103:875, 1981.
399. Roane JB, Dryden JA, Grimes EW: Incidence of postoperative pain after single and multiple visit endodontic procedures, Oral Surg OralMed Oral Pathol 55:68, 1983.
400. Rosenberg PA, Babick PJ, Schertzer L, Leung D: The effect of occlusal reduction on pain after endodontic instrumentation, J Endod 24:492, 1998.
401. Rosenberg RJ, Goodis HE: Endodontic case selection to treat or to refer, J Am Dent Assoc 123:57, 1992.
402. Seltzer S, Bender IB, Turkenkopf S: Factors affecting successful repair after root canal therapy, J Am Dent Assoc 67:651, 1962.
403. Sequeira JF, Batista MM, Fraga RC, de Uzeda M: Antibacterial effects of endodontic irrigants on black pigmented gram-negative anaerobes and falcultative bacteria, J Endod 24:414, 1998.
404. Serman NJ, Singer S: Exposure of the pregnant patient to ionizing radiation, Ann Dent 53:13, 1994.
405. Shrout MK et al: Treating the pregnant dental patient: four basic rules addressed, J Am Dent Assoc 123:75, 1992.
406. Abou-Rass M, Patonai FJ: The effects of decreasing surface tension on the flow of irrigating solutions in narrow root canals, Oral Surg Oral Med Oral Pathol 53:524, 1982.
407. Abou-Rass M, Piccinino MV: The effectiveness of four clinical irrigation methods on the removal of root canal debris, Oral Surg Oral Med Oral Pathol 54:323, 1982.
408. Ahmad M: An analysis of breakage of ultrasonic files during root canal instrumentation, Endod Dent Traumatol 5:78, 1989.
409. Ahmad M: Effect of ultrasonic instrumentation on Bacteroides intermedius, EndodDent Traumatol 5:83, 1989.
410. Ahmad M, Pitt-Ford TR: A comparison using macroradiography of canal shapes in teeth instrumented ultrasonically and by hand, J Endod 15:339,1989.

411. Ahmad M, Pitt-Ford TR, Crum LA: Ultrasonic debridement of root canals: acoustic streaming and its possible role, J Endod 13:490,1987.
412. Ahmad M, Pitt-Ford TR, Crum LA: Ultrasonic debridement of root canals: an insight into the mechanisms involved, J Endod 13:93, 1987.
413. Ahmad M, Pitt-Ford TR, Crum LA, Walton AJ: Ultrasonic debridement of root canals: acoustic cavitation and its relevance, J Endod 14:486, 1988.
414. Ahmad M, Pitt-Ford TR, Crum LA, Wilson RF: Effectiveness of ultrasonic files in the disruption of root canal bacteria, Oral Surg Oral Med Oral Pathol 70:328, 1990.
415. Ahmad M, Roy RA, Ghanikamarudin AG: Observations of acoustic streaming fields around an oscillating ultrasonic file, Endod Dent Traumatol 8:189,1992.
416. Ahmad M, Roy RA, Ghanikamarudin A, Safar M: The vibratory pattern of ultrasonic files driven piezoelectrically, Int Endod J 26:120,1992.
417. Anderson RW, Powell BJ, Pashley DH: Microleakage of three temporary endodontic restorations, J Endod 14:497, 1988.
418. Anic I, Matsumoto K: Dentinal heat transmission induced by a laser-softened gutta-percha obturation technique, J Endod 21:470,1995.
419. Anthony DR, Gordon TM, del Rio CE: The effect of three vehicles on the pH of calcium hydroxide, Oral Surg Oral Med Oral Pathol 54:560, 1982.
420. Araki K, Isaka H, Ishii T, Suda H: Excretion of 14C-formaldehyde distributed systematically through root canal following pulpectomy, Endod Dent Traumatol 9:196, 1993.
421. Archer R, Reader A, Nist R, Beck M, Meyers WJ: An *in vivo* evaluation of the efficacy of ultrasound after step-back preparation in mandibular molars, J Endod 18:549, 1992.
422. Bahcall J, Howard P, Miserendino L, Walia H: Preliminary investigation of the histological effects of laser endodontic treatment on the periradicular tissues in dogs, J Endod 18:47, 1992.
423. Baker NA, Eleazer PD, Averbach RE, Seltzer S: Scanning electron microscopic study of the efficacy of various irrigating solutions, J Endod 1:127,1975.
424. Barbosa SV, Burkard DH, Spangberg LSW: Cytotoxic effect of gutta-percha solvents, J Endod 20:6,1994.
425. Ellerbruch ES, Murphy RA: Antimicrobial activity of root canal medicament vapors, JEndod 3:189, 1977.
426. England MC, West NM, Safavi K, Green DB: Tissue lead levels in dogs with RC-2B root canal fillings, J Endod 6:728, 1980.
427. Engstrom B, Spangberg L: Studies on root canal medicaments. I. Cytotoxic effect of root canal antiseptics, Acta OdontolScand 25:77, 1967.
428. Eriksson AR, Albrektsson T: Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit, JProsth Dent 50:101, 1983.
429. Feldmann G, Nyborg H: Tissue reaction to root filling materials. I. Comparison between gutta-percha and silver amalgam implanted in rabbit, OdontolRevy 13:1, 1962.
430. Felt RA, Moser JB, Heuer MA: Flute design of endodontic instruments: its influence on cutting efficiency, J Endod 8:253, 1982.
431. Finne K, Nord PG, Persson G, Lennartsson B: Retrograde root filling with amalgam and cavit, Oral Surg Oral Med Oral Pathol 43:62, 1977.
432. Fouad AF: The use of electronic apex locators in endodontic therapy, Int Endod J 26:13, 1993.
433. Fouad AF et al: The effects of selected electronic dental instruments on patients with cardiac pacemakers, J Endod 16: 188, 1990.
434. Fouad AF, Krell KV: An *in vitro* comparison of five root canal length measuring instruments, J Endod 15:573, 1989.
435. Fouad AF et al: A clinical evaluation of five electronic root canal length measuring instruments, J Endod 16:446, 1990.
436. Fouad AF, Rivera EM, Krell KV: Accuracy of the endex with variations in canal irrigants and foramen size, J Endod 19:63, 1993.
437. Friedman S, Rotstein I, Mahamid A: *In vivo* efficacy of various retrofills and of CO_2 laser in apical surgery, Endod Dent Traumatol 7:l9, 1991.
438. Gani O, Visvisian C: Apical canal diameter in the first upper molar at various ages, J Endod 25:689, 1999.
439. Gazelius B, Olgart L, Edwall B: Restored vitality in luxated teeth assessed by laser Doppler flowmetry, Endod Dent Traumatol 4:265, 1988.
440. Gazelius B, Olgart L, Edwall B, Edwall L: Noninvasive recording of blood flow in human dental pulp, Endod Dent Traumatal 2:219, 1986.
441. Goldberg F: Relation between corroded silver points and endodontic failures, J Endod 7:224, 1981.
442. Goldberg F, Abramovich A: Analysis of the effect of EDTAC on the dentinal walls of the root canal, J Endod 3:10, 1977.
443. Goldberg F, Spielberg C: The effect of EDTAC on the variation of its working time analyzed with scanning electron microscopy, Oral Surg Oral Med Oral Pathol 53:74, 1982.
444. Goldman M et al: The efficacy of several endodontic irrigation solutions: a scanning electron microscopic study: part 2, J Endod 8:487, 1982.
445. Goldman M et al: New method of irrigation during endodontic treatment, J Endod 2:257, 1976.
446. Goodman A, Schilder H, Aldrich W: The thermomechanical properties of gutta-percha. II. The history and molecular chemistry of gutta-percha, Oral Surg Oral Med Oral Pathol 37:954, 1974.
447. Goodman A, Schilder H, Aldrich W: The thermomechanical properties of gutta-percha. Part IV. A thermal profile of the warm gutta-percha packing procedure, Oral Surg Oral Med Oral Pathol 51:544, 1981.
448. Grassi MD, Plazek DJ, Michanowicz AE, Chay I-C: Changes in the physical properties of the ultrafill low-temperature (70° C) thermoplasticized gutta-percha system, J Endod 15:517, 1989.
449. Grieve AR, Parkholm JDD: The sealing properties of root filling cements, further studies, Br Dent J 135:327, 1973.
450. Grossman LI: The effect of pH of rosin on setting time of root canal cements, J Endod 8:326, 1982.

492. Powell SE, Simon JHS, Maze B: A comparison of the effect of modified and nonmodified instrument tips on apical canal configuration, J Endod 12:293, 1986.
493. Powell SE, Wong PD, Simon JHS: A comparison of the effect of modified and nonmodified instrument tips on apical canal configuration. Part II, J Endod 14:224,1988.
494. Pratten DH, McDonald NJ: Comparison of radiographic and electronic working lengths, J Endod 22:173, 1996.
495. Ram Z: Effectiveness of root canal Irrigation, Oral Surg Oral Med Oral Pathol 44:306, 1977.
496. Ramsay DS, Artun J, Martinen SS: Reliability of pulpal blood-flow measurements utilizing laser Doppler flowmetry, J Dent Res 70:1427, 1991.
497. Read RP, Baumgartner JC, Clark SM: Effects of a carbon dioxide laser on human root dentin, J Endod 21:4, 1995.
498. Richman MJ: The use of ultrasonic in root canal therapy and root resection, J Dent Med 12:12, 1957.
499. Rickoff B et al: Effects of thermal vitality tests on human dental pulp, J Endod 14:482, 1988.
500. Roane JB, Sabala CL, Duncanson MG: The "balanced force" concept for instrumentation of curved canals, J Endod 11: 203, 1985.
501. Rome WJ, Doran JE, Walker WA: The effectiveness of glyoxide and sodium hypochlorite in preventing smear layer formation, J Endod 11:281,1985.
502. Rutberg M, Spangberg E, Spangberg L: Evaluation of enhanced vascular permeability of endodontic medicaments *in vivo*, J Endod 3:347, 1977.
503. Sabala CL, Powell SE: Sodium hypochlorite injection into periapical tissues, J Endod 15:490, 1989.
504. Sabala CL, Roane JB, Southard LZ: Instrumentation of curved canals using a modified tipped instrument: a comparison study, J Endod 14:59, 1988.
505. Spangberg L, Rutberg M, Rydinge E: Biological effects of endodontic antimicrobial agents, JEndod 5:166, 1979.
506. Spangberg LSW, Barbosa SV, Lavigne GD: AH26 releases formaldehyde, J Endod 19:596, 1993.
507. Stabholz A et al: Sealing of human dentinal tubules by XeCl 308 nm excimer laser, J Endod 19:267, 1993.
508. Staehle HJ, Spiess V, Heinecke A, Müller H-P: Effect of root canal filling materials containing calcium hydroxide and the alkalinity of root dentin, Endod Dent Traumatol 11:163, 1995.
509. Stamos DE, Sadeghi EM, Haasch GC, Gerstein H: An *in vitro* comparison study to quantitate the debridement ability of hand, sonic, and ultrasonic instrumentation, J Endod 13:434, 1987.
510. Stenman E, Spangberg LSW: Machining efficiency of endodontic K files and Hedstrom files, J Endod 16:375, 1990.
511. Stenman E, Spangberg LSW: Machining efficiency of Flex-R, K-Flex, Trio-Cut, and S Files, J Endod 16:575, 1990.
512. Stenman E, Spangberg L: Root canal instruments are poorly standardized, J Endod 19:327, 1993.
513. Sundada I: New method for measuring the length of the root canal, J Dent Res 41:375, 1962.
514. Sunzel B et al: The effect of zinc oxide on Staphylococcus aureus and polymorphonuclear cells in a tissue cage model, Scand J Plast Reconstr Surg 24:31, 1990.
515. Svec TA, Harrison JW: The effect of effervescence on debridement of the apical regions of root canals in single-rooted teeth, J Endod 7:335, 1981.
516. Tagger M: Use of thermo-mechanical compactors as an adjunct to lateral condensation, Quintessence Int 15:27,1984.
517. Tagger M, Tagger E, Kfir A: Release of calcium and hydroxyl ions from set endodontic sealers containing calcium hydroxide, J Endod 14:588, 1988.
518. Tani Y, Kawada H: Effects of laser irradiation on dentin. I. Effect on smear layer, J Dent Mater 6:127, 1987.
519. Tronstad L, Barnett F, Flax M: Solubility and biocompatability of calcium hydroxide-containing root canal sealers, Endod Dent Traumatol 4:152, 1988.
520. van der Fehr FR, Nygaard-Østby B: Effect of EDTAC and sulfuric acid on root canal dentine, Oral Surg Oral Med Oral Pathol 16:199, 1963.
521. Vessey RA: The effect of filing versus reaming on the shape of the prepared root canal, Oral Surg Oral Med Oral Pathol 27:543, 1969.
522. Villalobos RL, Moser JB, Heuer MA: A method to determine the cutting efficiency of root canal instruments in rotary motion, J Endod 6:667, 1980
523. Walia H, Brantley WA, Gerstein H: An initial investigation of the bending and torsional properties of nitinol root canal files, J Endod 14:246, 1988.
524. Walker TL, del Rio CE: Histological evaluation of ultrasonic and sonic instrumentation of curved root canals, J Endod 15: 49, 1989.
525. Walker TL, del Rio CE: Histological evaluation of ultrasonic debridement comparing sodium hypochlorite and water, J Endod 17:66, 1991.
526. Webber J, Moser JB, Heuer MA: A method to determine the cutting efficiency of root canal instruments in linear motion, J Endod 6:829, 1980.
527. Weine FS, Kelly RF, Lio PS: The effect of preparation procedures on original canal shape and apical foramen shape, J Endod 1:255, 1975.
528. Weinreb MM, Meier E: The relative efficiency of EDTA, sulfuric acid, and mechanical instrumentation in the enlargement of root canals, Oral Surg Oral Med Oral Pathol 19:247,1965.
529. Weller RN, Brady JM, Bernier WE: Efficacy of ultrasonic cleaning, J Endod 6:740, 1980.
530. Wennberg A, Bergdahl M, Spangberg L: Biologic effect of polyisobutylene on HeLa cells and on subcutaneous tissue in guinea pigs, Scand J Dent Res 82:613, 1974.
531. Wennberg A, Örstavik D: Adhesion of root canal sealers to bovine dentine and gutta-percha, Int Endod J 23:13, 1990.
532. West NM, England MC, Safavi K, Green DB: Levels of lead in blood of dogs with RC-2B root canal fillings, J Endod 6:598, 1980.
533. Wolcott JF, Himel VT, Hicks ML: Thermafil retreatment using a new system B technique or a solvent, J Endod 25:761, 1999.

575. Kazemi RB, Stenman E, Spangberg LSW: The endodontic file is a disposable instrument, J Endod 21(9):451, 1995.
576. Keir DM, Senia SE, Montgomery S: Effectiveness of a brush in removing post-instrumentation canal debris, J Endod 16(7):327, 1990.
577. Kennedy WA, Walker WA III, Gough RW: Smear layer removal effects on apical leakage, J Endod 12:21, 1986.
578. Kessler JR, Peters DD, Lorton L: Comparison of the relative risk of molar root perforations using various endodontic instrumentation techniques, J Endod 9:439, 1983.
579. Kois J, Spear FM: Periodontal prosthesis: creating successful restorations, J Am Dent Assoc 10:123, 1992.
580. Kosa DA, Marshall G, Baumgartner JC: An analysis of canal centering using mechanical instrumentation techniques, J Endod 25(6):441, 1999.
581. Koskinen KP, Meurman JH, Stenvall LH: Appearance of chemically treated root canal walls in the scanning electron microscope, Scand J Dent Res 88:397, 1980.
582. Kovacevic M, Tamarut T: Influence of the concentration of ions and foramen diameter on the accuracy of electronic root canal length measurement — an experimental study, J Endod 24(5):346, 1998.
583. Lenchner NH: Restoring endodontically treated teeth: ferrule effect and biologic width, Pract Periodontics Aesthet Dent 1:19, 1989.
584. Levin H: Access cavities, Dent Clin North Am 701, November, 1967.
585. Luiten DJ, Morgan LA, Baumgartner JC, Marshall JG: A comparison of four instrumentation techniques on apical canal transportation, J Endod 21(1):26, 1995.
586. Mandel E, Machtou P, Friedman S: Scanning electron microscope observation of canal cleanliness, J Endod 16(6):279, 1990.
587. McCann JT, Keller DL, LaBounty GL: Remaining dentin/cementum thickness after hand or ultrasonic instrumentation, J Endod 16(3): 109, 1990.
588. McComb D, Smith DC: A preliminary scanning electron microscopic study of root canals after endodontic procedures, J Endod 1:238, 1975.
589. McDonald NJ: The electronic determination of working length, Dent Clin North Am 36:293, 1992.
590. Miserendino LJ, Moser JB, Heuer MA, Osetek EM: Cutting efficiency of endodontic instruments. II. Analysis of tip design, J Endod 12(1):8, 1986.
591. Montgomery S: Root canal wall thickness of mandibular molars after biomechanical preparation, J Endod 11(6):257, 1988.
592. Morgan LF, Montgomery S: An evaluation of the crown-down pressureless technique, J Endod 10(10):491, 1984.
593. Pagavino G, Pace R, Baccetti T: A SEM study of *in vivo* accuracy of the Root ZX electronic apex locator, J Endod 24(6): 438, 1998.
594. Pedicord D, El Deeb ME, Messer HH: Hand versus ultrasonic instrumentation: its effect on canal shape and instrumentation time, J Endod 12(9):375, 1986.
595. Pineda F, Kuttler Y: Mesiodistal and buccolingual roentgenographic investigation of 7275 root canals, Oral Surg 33:101, 1972.
596. Piskin B, Turkun M: Stability of various sodium hypochlorite solutions, J Endod 2l(5):253, 1995.
597. Pruett JP, Clement DJ, Carnes DL Jr: Cyclic fatigue testing of nickel-titanium endodontic instruments, J Endod 23 (2): 77, 1997.
598. Raiden G et al: Residual thickness of root in first maxillary premolars with post space preparation, J Endod 25(7):502, 1998.
599. Reddy SA, Hicks ML: Apical extrusion of debris using two hand and two rotary instrumentation techniques, J Endod 24:180, 1998.
600. Reynolds MA et al: An *in vitro* histological comparison of the step-back, sonic, and ultrasonic instrumentation techniques in small, curved root canals, J Endod 13(7):307, 1987.
601. Roane JB, Sabala CL: Clockwise or counterclockwise, J Endod 10:349, 1984.
602. Roane JB, Sabala CL, Duncanson MG: The "balanced force" concept for instrumentation of curved canals, J Endod 11(5):203, 1985.
603. Rosenberg PA, Babick PJ, Schertzer L, Leung A: The effect of occlusal reduction on pain after endodontic instrumentation, J Endod 24(1)A92, 1998.
604. Rowan MB, Nicholls JI, Steiner J: Torsional properties of stainless steel and nickel-titanium endodontic files, J Endod 22(7):341, 1996.
605. Royal JR, Donnelly JC: A comparison of maintenance of canal curvature using balanced-force instrumentation with three different file types, J Endod 21(6):300, 1995.
606. Ruddle CJ: Microendodontic nonsurgical retreatment, Microscopes in Endodontics, Dent Clin of North Am 41(3):429, WB
607. Walton RE: Histologic evaluation of different methods of enlarging the pulp canal space, J Endod 2:304, 1976.
608. Weine FS: The use of non-ISO tapered instruments for canal flaring, Compend Contin Educ Dent 17:651, 1996.
609. Weine FS, Kelly RF, Lio PJ: The effect of preparation procedures on original canal shape and on apical foramen shape, J Endod 1(8):255, 1975.
610. Yee RDJ et al: The effect of canal preparation on the formation and leakage characteristics of the apical dentin plug, J Endod 10:308, 1984.
611. Yoshida T et al: Clinical evaluation of the efficacy of EDTA solution as an endodontic irrigant,J Endod 21(12): 592,1995.
612. Baisden MD, Kulild JC, Weller RN: Root canal configuration of the mandibular first premolar, J Endod 18:505, 1992.
613. Benjamin KA, Dowson J: Incidence of two root canals in human mandibular incisor teeth, J Oral Surg 38:122, 1974.
614. Booth JM: The longest tooth Aust Endod News 13:17, 1988.
615. Bram SM, Fleisher R: Endodontic therapy in a mandibular second bicuspid with four canals, J Endod 17:513, 1991.
616. Buchanan LS: Management of the curved root canal: predictably treating the most common endodontic complexity, J Calif Dent Assoc 17:40,1989.
617. Cams EJ, Skidmore AE: Configuration and deviation of root canals of maxillary first premolars, J Oral Surg 36:880, 1973.
618. Clements RE, Gilboe DB: Labial endodontic access opening for mandibular incisors: Endodontic and restorative considerations, J Can Dent Assoc 57:587, 1991.

660. Hatton EH: Changes produced in the pulp and periapical regions, and their relationship to pulp-canal treatment and to systemic disease, Dent Cosmos 66:1183, 1924.
661. Hatton JF, Ferrillo PJ, Wagner G, Stewart GP: The effect of condensation pressure on the apical seal, J Endod 14:305, 1988.

662. Heling I, Chandler NP: The antimicrobial effect within dentinal tubules of four root canal sealers, J Endod'22:251, 1996.
663. Holcomb J, Pitts D, Nicholls J: Further investigation of spreader loads required to cause vertical root fracture during lateral condensation, J Endod 13:277, 1987.
664. Holland GR: Periapical response to apical plugs of dentin and calcium hydroxide in ferret canines, J Endod 10:71, 1984.
665. Holland R, de Souza V: Ability of a new calcium hydroxide root canal filling material to induce hard tissue formation, J Endod 11:535, 1985.
666. Holland R et al: Tissue reactions following apical plugging of the root canal with infected dentin chips, Oral Surg Oral Med Oral Pathol Oral Radial Endod 49:366, 1980.
667. Hunter W: The role of sepsis and of antisepsis in medicine, Lancer 1:79, 1911.
668. Ingle JI: A standardized endodontic technique using newly designed instruments and filling materials, Oral Surg Oral Med Oral Pathol Oral Radial Endod 14:83, 1961.
669. Ingle JI, Zeldow BJ: An evaluation of mechanical instrumentation and the negative culture in endodontic therapy, J Am Dent Assoc 57:471, 1958.
670. Jacobsen EL: Clinical aid: adapting the master gutta-percha cone for apical snugness, J Endod 10:274, 1984.
671. Jerome CE: Warm vertical gutta-percha obturation: a technique update, J Endod 20:97, 1994.
672. Johnson WB: A new gutta-percha technique, J Endod 4:184, 1978.
673. Kaplowitz GJ: Evaluation of gutta-percha solvents, J Endod 16:539, 1990.
674. Karagoz-Kiicukay I, Bayirli G: An apical leakage study in the presence and absence of the smear layer, Int Endod J 27:87, 1994.
675. Keane K, Harrington GW: The use of a chloroform-softened gutta-percha master cone and its effect on the apical seal, J Endod 10:57, 1984.
676. Kersten HW, Fransman R, Thoden van Velzen SK: Thermomechanical compaction of gutta-percha. I. A comparison of several compaction procedures, Int Endod J 19:125, 1986.
677. Kersten HW, Wesselink PR, Thoden van Velzen SK: The diagnostic reliability of the buccal radiograph after root canal filling, Int Endod J 20:20, 1987.
678. Kontakiotis E, Panopoulos P: pH of root canal sealers containing calcium hydroxide, Int Endod J 29:202, 1996.
679. Kytridou V, Gutmann JL, Nunn MH: Adaptation and scalability of two contemporary obturation techniques in the absence of the dentinal smear layer, Int Endod J32:464,1999.
680. Langeland K: Root canal sealants and pastes, Dent Clin North Am 18:309, 1974.
681. Leonard JE, Gutmann JL, Guo IY: Apical and coronal seal of roots obturated with a dentine bonding agent and resin, Int Endod J 29:16, 1996.
682. Love RM, Chandler NP, Jenkinson HF: Penetration of smeared or nonsmeared dentine by Streptococcus gordonii, Int Endod J 29:2, 1996.
683. Lugassy AA, Yee F: Root canal obturation with gutta-percha: a scanning electron microscope comparison of vertical compaction and automated thermatic condensation, J Endod 8:120, 1982.
684. Mader CL, Baumgartner JC, Peters DD: Scanning electron microscopic investigation of the smeared layer on root canal walls, J Endod 10:477, 1984.
685. Marais JT, van der Vyver PJ: Sizing gutta-percha points with a gauge to ensure optimal lateral condensation, JDent Assoc South Afr 51:403, 1996.
686. Martin H, Fischer E: Photoelastic stress comparison of warm (Endotec) versus cold lateral condensation techniques, Oral Surg Oral Med Oral Pathol Oral Radial Endod 70:325, 1990.
687. Saunders WP, Saunders EM: Coronal leakage as a cause of failure in root canal therapy: a review, Endod Dent Traumatol 10:105, 1994.
688. Saunders WP, Saunders EM, Gutmann JL, Gutmann ML: An assessment of the plastic Thermafil obturation technique. III. The effect of post space preparation on the apical seal, Int Endod J26:IS4, 1993.
689. Saw L-P, Messer HH: Root strains associated with different obturation techniques, JEndod 21:314, 1995.
690. Schilder H: Filling root canals in three dimensions, Dent Clin North Am 11:723, 1967.
691. Schilder H, Goodman A, Aldrich W: The thermomechanical properties of gutta-percha. I. The compressibility of gutta-percha, Oral Surg Oral Med Oral Pathol Oral Radial Endod 37:946, 1974.
692. Scott AC, Vire DE: An evaluation of the ability of a dentin plug to control extrusion of thermoplasticized gutta-percha, J Endod 18:52-57, 1992.
693. Seltzer S, Bender IB, Turkenkopf S: Factors affecting successful repair after root canal therapy, J Am Dent Assoc 67: 651, 1963.
694. Seltzer S, Naidorf I: Flare-ups in endodontics. II. Therapeutic measures, J Endod 11:559, 1985.
695. Sen BH, Piskin B, Baran N: The effect of tubular penetration of root canal sealers on dye microleakage, Int Endod 729:23, 1996.
696. Sen BH, Wesselink PR, Tiirkun M: The smear layer: a phenomenon in root canal therapy, Int Endod J 28:141, 1995.
697. Sjogren U, Figdor D, Persson S, Sundqvist G: Influence of infection at the time of root filling in the outcome of endodontic treatment of teeth with apical periodontitis. Int Endod J3: 297, 1997.
698. Sjogren U, Hagglund B, Sundqvist G, Wing K: Factors affecting the long-term results of endodontic treatment, J Endod 16: 498, 1990.
699. Sjogren U, Sundqvist G, Nair PNR: Tissue reaction to gutta-percha particles of various sizes when implanted subcutaneously in guinea pigs, Eur J Oral Sci 103:313, 1995.
700. Skillen WG: Why root canals should be filled to the dentinocemental junction, J Am Dent Assoc 17:2082, 1930.

740. Stanley HR, Going RE, Chauncey HH: Human pulp response to acid pretreatment of dentin and to composite restoration, J Am Dent Assoc 91:817, 1975.
741. Stanley HR et al: The detection and prevalence of reactive and physiologic alterations of dentin, especially dentin and dead tracts beneath various types of dental lesions according to tooth surface and age, J Pathol 12:257, 1983.
742. Swerdlow H, Stanley HR: Reaction of human dental pulp to cavity preparation. I. Effect of water spray at 20,000 RPM, J Am Dent Assoc 56:317, 1958.
743. Swerdlow H, Stanley HR: Reaction of human dental pulp to cavity preparation, J Prosthet Dent 9:121, 1959.
744. Taylor PE, Byers MR, Redd PE: Sprouting of CGRP nerve fibers in response to dentin injury in rat molars, Brain Res 461: 371, 1988.
745. Tender K, Kvinnsland I: Micropuncture measurement of interstitial tissue pressure in normal and inflamed dental pulp in cuts, J Endod 9:105, 1983.
746. Trowbridge HO: Pathogenesis of pulpitis resulting from dental caries, J Endod 7:52, 1981.
747. Trowbridge HO, Edwall L, Panopoulos P: Effect of zinc oxide and eugenol and calcium hydroxide on intradental nerve activity, J Endod 8:403, 1982.
748. Trowbridge HO, Franks M, Korostoff E, Emling R: Sensory response to thermal stimulation in human teeth, J Endod 6(1): 405, 1980.
749. Abbott AA et al: A prospective randomized trial on efficacy of antibiotic prophylaxis in asymptomatic teeth with pulpal necrosis and associated periapical pathosis, Oral Surg 66:722, 1988.
750. Abbott PV: Factors associated with continuing pain in endodontics, Aust Dent J 39:151, 1994.
751. Albashaireh ZS, Alnegrish AS: Postobturation pain after single and multiple-visit endodontic therapy. A prospective study, J Dent 26:227,1998
752. Bacsik C, Swift J, Hargreaves KM: Toxic systemic reactions of bupivacaine and etidocaine: review of the literature, Oral Surg. Oral Med Oral Paro 79:18, 1995.
753. Balaban FS, Skidmore AE, Griffin JA: Acute exacerbations following initial treatment of necrotic pulps, J Endod 10:78, 1984.
754. Barnett F, Tronstad L: The incidence of flare-ups following endodontic treatment, J Dent Res 68(special issue): 1253, 1989.
755. Basbaum A, Fields H: Endogenous pain control systems: brainstem spinal pathways and endorphin circuitry, Ann Rev Neurosci 7:309, 1984.
756. Beaver W: Mild analgesics. A review of their clinical pharmacology, Am J Med Sci 251:576, 1966.
757. Bisgaard H, Kristensen J: Leukotriene B4 produces hyperalgesia in humans, Prostaglandins 30:791, 1985.
758. Breivik E, Barkvoll P, Skovlund E: Combining diclofenac with acetaminophen or acetaminophen-codeine after oral surgery: a randomized, double-blind, single oral dose study, Clin Pharmacol Ther 54:(in press), 2000.
759. Brown A et al: Spatial summation of pre-pain and pain in human teeth, Pain 21:1,1985.
760. Gracely R et al: Placebo and naloxone can alter post-surgical pain by separate mechanisms, Nature 306:264, 1983.
761. Hargreaves KM et al: Naloxone, fentanyl and diazepam modify plasma beta-endorphin levels during surgery, Clin Pharmacol Ther 40:165, 1986.
762. Hargreaves KM, Joris J: The peripheral analgesic effects of opioids, JAm Pain Soc 2:51, 1993.
763. Hargreaves KM et al: Pharmacology of peripheral neuropeptide and inflammatory mediator release, Oral Surg Oral Med OralPathol 78:503, 1994.
764. Hargreaves KM, Troullos E, Dionne R, Pharmacologic rationale for the treatment of acute pain, Dent Clin North Am 31:675, 1987.
765. Harrison JW, Bellizzi R, Osetek EM: The clinical toxicity of endodontic medicaments, J Endod 5:42, 1979.
766. Harrison JW, Baumgartner CJ, Zielke DR: Analysis of interappointment pain associated with the combined use of endodontic irrigants and medicaments, J Endod 7:272, 1981.
767. Harrison JW, Baumgartner JC, Svec TA: Incidence of pain associated with clinical factors during and after root canal therapy. I. Interappointment pain, J Endod 9:384, 1983.
768. Harrison JW, Baumgartner JC, Svec TA: Incidence of pain associated with clinical factors during and after root canal therapy. II. Postobturation pain, J Endod 9:434, 1983.
769. Hasselgren G, Reit C: Emergency pulpotomy: pain relieving effect with and without the use of sedative dressings, J Endod 15:254, 1989.
770. Hylden J, Nahin R, Traub R, Dubner R: Expansion of receptorfields of spinal lamina I projection neurons in rats with unilateral adjuvant-induced inflammation, Pain 37:229, 1989.
771. Imura N, Zuolo ML: Factors associated with endodontic flare-ups: aprospective study, Int Endod J 28:261, 1995.
772. Jackson D, Moore P, Hargreaves KM: Preoperative nonsteroidal anti-inflammatory medication for the prevention of postoperative dental pain, J Am Dent Assoc 19:641, 1989.
773. Jebeles J et al: Tonsillectomy and adenoidectomy pain reduction by local bupivacaine infiltration in children, Int J Pediatr Otorhinolaryngol 25:149, 1993.
774. Johnson D, Harshbarger J, Rymer H: Quantitative assessment of neural development in human premolars, Anat Rec 205: 421, 1983.
775. Jostes JL, Holland GR: The effect of occlusal reduction after canal preparation on patient comfort, J Endod 10:34, 1984.
776. Kaufman E et al: Intraligamentary injection of slow-release methylprednisolone for the prevention of pain after endodontic treatment, Oral Surg Oral Med Oral Pathol 77:651, 1994.
777. Kimberly C, Byers M: Inflammation of rat molar pulp and periodontium causes increased calcitonin gene related peptide and axonal sprouting, Anat Rec 222:289, 1988.
778. Krasner P, Jackson E: Management of posttreatmerit endodontic pain with oral dexamethasone: a double-blind study, Oral Surg Oral Med Oral Pathol 62:187, 1986.
779. Kumazawa T, Mizumura K: Thin-fiber receptors responding to mechanical, chemical and thermal stimulation in the skeletal muscle of the dog, Am J Physiol 273:179, 1977.
780. Levine J, Moskowitz M, Basbaum A: The contribution of neurogenic inflammation in experimental arthritis, JImmunol 135:843, 1985.

781. Lewin G, Rueff A, Mendell L: Peripheral and central mechanisms of NGF-induced hyperalgesia, Eur J Neurosci 6:1903, 1994.
782. Liesinger A, Marshall F, Marshall J: Effect of variable doses of dexamethasone on postreatment endodontic pain, J Endod 19:35, 1993.
783. Lindahl O: Pain—a chemical explanation, Acta Rheumatol Scand 8:161, 1962.
784. Maddox D, Walton R, Davis C: Influence of posttreatment endodontic pain related to medicaments and other factors, J Endod 3:447, 1977.
785. Madison S et al: Sensitizing effects of leukotriene B_4 on intradental primary afferents, Pain 49:99, 1992.
786. Maixner W et al: Responses of monkey medullary dorsal horn neurons during the detection of noxious heat stimuli, J Neurophysiol 62:437, 1989.
787. Malmberg A, Yaksh T: Antinociceptive actions of spinal nonsteroidal anti-inflammatory agents on the formalin test in rats, J Pharmacol Exp Ther 263:136, 1992.
788. Marshall J, Liesinger A: Factors associated with endodontic posttreatment pain, J Endod 19:573, 1993.
789. Marshall J, Walton R: The effect of intramuscular injection of steroid on posttreatment endodontic pain, J Endod 10:584, 1984.
790. Marshall J, Walton R: The effect of intramuscular injection of steroid on posttreatment endodontic pain, J Endod 19:573, 1993.
791. Sessle BJ: Recent developments in pain research: central mechanisms of orofacial pain and its control, J Endod 12:435, 1986.
792. Soltanoff W: A comparative study of the single-visit and the multiple-visit endodontic procedure, J Endod 4:278, 1978.
793. Stambaugh J, Drew J: The combination of ibuprofen and oxycodone/acetaminophen in the management of chronic cancer pain, Clin Pharmacol Ther 44:665, 1988.
794. Sugimoto T, Bennett G, Kajander K: Transsynaptic degeneration in the superficial dorsal horn after sciatic nerve injury: effects of a chronic constriction injury, transection and styrchnine, Pain 42:205, 1990.
795. Sunshine A et al: Analgesic efficacy of a hydrocodone with ibuprofen combination compared with ibuprofen alone for the treatment of acute postoperative pain, J Clin Pharmacol 37: 908, 1997.
796. Torabinejad M et al: Factors associated with endodontic interappointment emergencies of teeth with necrotic pulps, JEndod 14:261, 1988.
797. Torabinejad M et al: Effectiveness of various medications on postoperative pain following complete instrumentation, J Endod 20:345, 1994.
798. Torabinejad M et al: Effectiveness of various medications on postoperative pain following root canal obturation, J Endod 20:427, 1994.
799. Trope M: Relationship of intracanal medicaments to endodontic flare-ups, Endod Dent Traumatol 6:226, 1990.
800. Trope M: Flare-up rate of single-visit endodontics, Int Endod. 724:24, 1991.
801. Troullos E, Freeman R, Dionne R: The scientific basis for analgesic use in dentistry, Anesth Prog 33:123, 1986.
802. Trowbridge H: Review of derital pain—histology and physiology, J Endod 12:445, 1986.
803. Wagner R, Myers R: Endoneurial injection of TNF-alpha produces neuropathic pain behaviors, Neuroreport 7:2897,1996.
804. Wallace J: Selective COX-2 inhibitors: is the water becoming muddy? Trends Pharmacol Sci 20:4, 1999.
805. Walton R, Fouad A: Endodontic interappointment flare-ups: a prospective study of incidence and related factors, J Endod 18:172, 1992.
806. Walton R, Chiappinelli J: Prophylactic penicillin: effect on posttreatment symptoms following root canal treatment of asymptomatic periapical pathosis, J Endod 19:466, 1993.
807. Wideman G et al: Analgesic efficacy of a combination of hydrocodone with ibuprofen in postoperative pain, Clin Pharm Therap 65:66, 1999.
808. Woblf C: Evidence for a central component of post-injury pain hypersensitivity, Nature 306:686, 1983.
809. WoolfC: Windup and central sensitization are not equivalent, Pain 66:105, 1996.
810. Wright C et al: Ibuprofen and acetaminophen kinetics when taken concurrently, Clin Pharm Therap 34:707, 1983.
811. Akinosi JO: A new approach to the mandibular nerve block, Br J Oral Surg 15:83, 1977.
812. Cohen F et al: Occupational disease in dentistry and chronic exposure to trace anesthetic gases, J Am Dent Assoc 101:21, 1980.
813. Corah N: Development of dental anxiety scale, J Dent Res 48: 596, 1969.
814. Council on Dental Education, American Dental Association: Guidelines for teaching the comprehensive control of pain and anxiety in dentistry, JDent Educ 36:62, 1972.
815. Covino BJ: Physiology and pharmacology of local anesthetic agents, Anesth Prog 28:98, 1981.
816. Dormauer D, Aston R: Update: midazolam maleate, a new water-soluble benzodiazepine, J Am Dent Assoc 106:650, 1983.
817. Fast TB, Martin MD, Ellis TM: Emergency preparedness: a survey of dental practitioners, J Am Dent Assoc 112:499, 1986.
818. Friedman MJ, Hochman MN: The AMSA injection: a new concept for local anesthesia of maxillary teeth using a computer-controlled injection system, Quintessence Int 29(5):297, 1998.
819. Frommer J, Mele FA, Monroe CW: The possible role of the mylohyoid nerve in mandibular posterior tooth sensation, J Am Dent Assoc 85:113, 1972.
820. Gale E: Fears of the dental situation, J Dent Res 51:964, 1972.
821. Gow-Gates GAE: Mandibular conduction anesthesia: a new technique using extraoral landmarks, OralSurg OralMed Oral Pathol 36:321, 1973.
822. Jastak JT, Malamed SF: Nitrous oxide and sexual phenomena, J Am Dent Assoc 101:38, 1980.
823. Leonard M: The efficacy of an intraosseous injection system of delivering local anesthetic, J Am Dent Assoc 126(1):81, 1995.
824. Malamed SF: The recreational abuse of nitrous oxide by health professionals, J Calif Dent Assoc 8:38, 1980.

825. Malamed SF: The Gow-Gates mandibular block: evaluation after 4275 cases, Oral Surg Oral Med Oral Pathol 51:463, 1981.
826. Malamed SF: The periodontal ligament injection: an alternative to mandibular block, Oral Surg Oral Med Oral Pathol 53: 118,1982.
827. Malamed SF, Quinn CL, Hatch HG: Pediatric sedation with intramuscular and intravenous midazolam, Anesth Prog 36: 155, 1989.
828. Malamed SF, Reggiardo P: Pediatric oral conscious sedation: changes to come, J Calif Dent Assoc, 1999 (in press).
829. Malamed SF, Trieger NT: Intraoral maxillary nerve block: an anatomical and clinical study, Anesth Prog 30:44, 1983.
830. Matsuura H: Analysis of systemic complications and deaths during dental treatment in Japan, Anesth Prog 36:219, 1990.
831. Barkhordar R et al: Cyanoacrylate as a retrofilling material, Oral Surg 65:468, 1988.
832. Bates C, Carnes DL, del Rio CE: Longitudinal sealing ability of mineral trioxide aggregate as root-end filling material, J Endod 22:515, 1996.
833. Blackman R, Gross M, Seltzer S: An evaluation of the biocompatibility of a glass ionomer-silver cement in rat connective tissue, J Endod 15(2):76, 1989.
834. Brynoff I: A histological and roentgenological study of the periapical region of human upper incisors, Odontol Revy 18:1, 1967.
835. Cambruzzi J, Marshall F: Molar endodontic surgery, J Can Dent Assoc 1:61, 1983.
836. Carr G: Common errors in periradicular surgery, Endod Rep 8:12, 1993.
837. Carr G: Ultrasonic root end preparation, Dent Clin North Am 41:541, 1997.
838. Chivian N: Surgical endodontics: conservative approach, JNJ State Dent Soc 40:234, 1969.
839. Cutright DE, Hunsuck EE: Microcirculatixm of the perioral regions in the Macaca rhesus: part 1, Oral Surg 29:776,1970.
840. Dajani AS et al: Prevention of bacterial endocarditis. Recommendations by the American Heart Association, JAMA 277(22): 1794, 1997.
841. Dionne R et al: Suppression of postoperative administration of ibuprofen in comparison to placebo, acetaminophen and acetaminophen plus codeine, J Clinic Pharmacol 23:37, 1983.
842. Dorn SO, Gartner AH: Retrograde filling materials; a retrospective success-failure study of amalgam, EBA and IRM, J Endod 8:391, 1990.
843. Ehrich DG et al: Comparison of triazolam, diazepam and placebo as outpatient oral premedication for endodontic patients, J Endod 23(3):181, 1997.
844. Engle T, Steiman H: Preliminary investigation of ultrasonic root end preparation, J Endod 21:443, 1995.
845. Evans B: Local hemostatic agents, N Y State Dent J 47:109, 1977.
846. Flanders D, James G, Burch B, Dockum N: Comparative histopathologic study of zinc-free amalgam and Cavit in connective tissue of the rat, J Endod 1:56, 1975.
847. Forte SG, Hauser MJ, Hahn C, Hartwell GR: Microleakage of super-EBA with and without finishing as determined by the fluid filtration method, J Endod 24(12):799, 1998.
848. Frank A, Click D, Patterson S, Weine F: Long-term evaluation of surgically placed amalgam fillings, J Endod 18(8):391, 1992.
849. Gartner AH, Dorn SO: Advances in endodontic surgery, Dent Clin North Am 36:357, 1992.
850. Gilheany P, Figdor D, Tyas M: Apical dentin permeability and microleakage associated with root end resection and retrograde filling, J Endod 20:22, 1994.
851. Green D: Double canals in single roots, Oral Surg 35:689, 1973.
852. Grossman L: Intentional replantation of teeth: a clinical evaluation, J Am Dent Assoc 104:633, 1966.
853. Harrison J, Jurosky K: Wound healing in the tissues of the periodontium following periradicular surgery. II. The dissectional wound, J Endod 17:544, 1991.
854. Hersh EV et al: Single dose and multidose analgesic study of ibuprofen and meclofenamate sodium after third molar surgery, Oral Surg Oral Med Oral Pathol 76(6):680, 1993.
855. Hirsh J et al: Periapical surgery, Int J Oral Surg 8:173, 1979.
856. Hsu YY, Kim S: The resected root surface. The issue of canal isthmuses, Dent Clin North Am 41(3):529, 1997.
857. Johnson J, Anderson R, Pashley D: Evaluation of the seal of various amalgam products used for root-end fillings, J Endod 21:505, 1995.
858. Jou Y, Pert! C: Is there a best retrograde filling material? Dent Clin North Am 41:555, 1997.
859. Kim S: Principles of endodontic surgery, Dent Clin North Am 41(3):481, 1997.
860. Kim S, Edwall L, Trowbridge H, Chien S: Effects of local anesthetics on pulpal blood flow, J Dent Res 63:650, 1984.
861. Kim S, Rethnam S: Hemostasis in endodontic microsurgery, Dent Clin North Am 41(3):499, 1997.
862. Kohn MW, Chase DC, Marciani RD: Surgical misadventures, Dent Clin North Am 17:533, 1973.
863. Lemon R, Steel P, Jeansonne B: Ferric sulfate hemostasis: effect on osseous wound healing: I. Left in situ for maximum exposure, J Endod 19:170, 1993.
864. Lilly G, Amstrong J, Cutcher J: Reaction of oral tissues to suture materials. Part III. Oral Surg 28:432, 1969.
865. Lilly G et al: Reaction of oral tissues to suture materials. II. Oral Surg 26:592, 1968.
866. Lilly G et al: Reaction of oral tissues to suture materials. Part IV, Oral Surg 13:52, 1972.
867. McDonald N, Dumsha T: A comparative retrofill leakage study utilizing a dentin bonding material, J Endod 13:224, 1987.
868. Nelson L, Mahler D: Factors influencing the sealing behavior of retrograde amalgam fillings, Oral Surg 69:356, 1990.
869. Whal M: Myths of dental surgery in patients receiving anticoagulant therapy, JAm Dent Assoc 131:77, 2000.
870. Witherspoon DE, Gutmann JL: Hemostasis in periradicular surgery, Int Endod J 29:135, 1996.
871. Yagiela J: Vasoconstrictor agents for local anesthesia, Anesth Progr 42:116, 1995.

916. Weine FS, Rice RT: Handling previously treated silver point cases: removal, retreatment, and tooth retention, Compend Contin Educ Dent 7:9, 1986.
917. Weller RN, Niemczyk SP, Kim S: Incidence and position of the canal isthmus. Part II. Mesiobuccal root of the maxillary first molar, J Endod 1:380, 1995.
918. Wilcox LR: Endodontic retreatment: Ultrasonics and chloroform as the final step in reinstrumentation, J Endod 15(3): 125, 1989.
919. Wilcox LR: Endodontic retreatment with halothane versus chloroform solvent, J Endod 21(6):305, 1995.
920. Wilcox LR, Krell KV, Madison S, Rittman B: Endodontic retreatment: Evaluation of gutta-percha and sealer removal and canal reinstrumentation, J Endod 13:9, 1987.
921. Winter R: Visualizing natural teeth, J Esthet Dent 5:3, 1993.
922. Wright WE: Prosthetic management of the periodontally compromised dentition, J Calif Dent Assoc 17:9, 1989.
923. Yoshida T, Gomyo S, Itoh T, Shibata T, Sekine I: An experimental study of the removal of cemented dowel-retained cast cores by ultrasonic vibration, J Endod 23:4, 1997.
924. Diem CR, Bower GM, Ferrigno PD, Fedi PF Jr: Regeneration of the attachment apparatus on pulpless teeth denuded of cementum in Rhesus money, J Periodontol 45:18, 1974.
925. Duggins I, Clay J, Himel V, Dean J: A combined endodontic retrofill and periodontal guided tissue regeneration for the repair of molar endodontic furcation perforations: Report of a case, Quintessence Int 25:109, 1994.
926. Ehnevid H, Jansson L, Lindskog S, Blomlof L: Endodontic pathogens: propagation of infection through patent dentinal tubules in traumatized monkey teeth, Endod Dent Traumatal 11:229, 1995.
927. Erpenstein H: A three year study of hemisections molars, J Clin Periodontol 10:1, 1983.
928. Everett FG, Kramer GM: The disto-lingual groove in the maxillary lateral incisor: a periodontal hazard, J Periodontol 443: 352, 1972.
929. Filipowicz F, Umstott P, England M: Vital root resection in maxillary molar teeth: a longitudinal study, J Endod 10:264, 1984.
930. Garrett S: Periodontal regeneration around natural teeth, Ann Periodontol 1:621, 1996.
931. Gerstein K: The role of vital root resection in periodontics, J Periodontol 48,478, 1977.
932. Gher ME, Dunlap RM, Anderson MH, Kuhl LV: Clinical survey of fractured teeth, J Am Dent Assoc 114:174, 1987.
933. Green EN: Hemisection and root amputation, J Am Dent Assoc 112:511, 1986.
934. Guldener PH: The relationship between periodontal and pulpal disease, Int Endod J 18:41, 1985.
935. Gutmann JL: Prevalence, location and patency of accessory canals in the furcation region of permanent molars, J Periodontol 49:21, 1978.
936. Hany JM, Nilveus RE, McMillan PJ, Wikesjo UME: Periodontal repair in dogs: expanded polytetrafluoroethylene barrier membranes support would stabilization and enhance bone regeneration, J Periodontol 64:883, 1993.
937. Haskell E: Vital root resection: a case report of long-term follow-up, Int J Periodontics Restorative Dent 4(6):57, 1984.
938. Hou GL, Tsai C: Relationship between periodontal furcation involvement and molar cervical enamel projections, J Periodontol 58:715, 1987.
939. Jansson LE, Ehnevid H: The influence of endodontic infection on periodontal status in mandibular molars, J Periodontol 69: 1392, 1998.
940. Kellert M, Chalfin H, Solomon C: Guided tissue regeneration: an adjunct to endodontic surgery, J Am Dent Assoc 125:1229, 1994.
941. Kirchoff DA, Gerstein H: Presurgical occlusal contouring for root amputation procedures, Oral Surg 27:379, 1969.
942. Kirkham DB: The location and incidence of accessory pulpal canals in periodontal pockets, J Am Dent Assoc 91:353, 1975.
943. Klavan B: Clinical observation following root amputation in maxillary molar teeth, J Periodontol 46:105, 1975.
944. Koenigs JF, Brilliant JD, Foreman DW: Preliminary scanning electron microscope investigations of accessory foramina in the furcation areas of human molar teeth, Oral Surg Oral Med OralPathol 38:773,1974.
945. Kramer IRH: The vascular architecture of the human dental pulp, Arch Oral Biol 2:177, 1960.
946. Langeland K, Rodrigues H, Dowden W: Periodontal disease, bacteria and pulpal histopathology, Oral Surg Oral Med Oral Pathol Oral Radial Ended 37:257, 1974.
947. Lantelme RL, Handelman SL, Herbison RJ: Dentin formation in periodontally diseased teeth, J Dent Res 55:48, 1976.
948. Lee KW, Lee EC, Poon KY: Palato-gingival grooves in maxillary incisors, Br Dent J 124:14, 1968.
949. Lowman JV, Burke RS, Pelleu GB: Patent accessory canals: incidence in molar furcation region, Oral Surg Oral Med Oral Pathol 36:580, 1973.
950. Mandi FA: Histological study of the pulp changes caused by periodontal disease, J Br Endod Soc 6:80, 1972.
951. Masters DH, Hoskins SW: Projection of cervical enamel into molar furcations, J Periodontol 35:49, 1964.
952. Mazur B, Massler M: Influence of periodontal disease on the dental pulp, Oral Surg Oral Med Oral Pathol Oral Radial Endod 17:592, 1964.
953. Miyashita H, Bergenholtz G, Grondahl K, Wennstrom JL: Impact of endodontic conditions on marginal bone loss, J Periodontol 69:158, 1998.
954. Obermayr G, Walton RE, Leary JM, Krell KV: Vertical root fracture and relative deformation during obturation and post cementation, J Prosthet Dent 66:181, 1991.
955. Pecora G, Baek SH, Rethnam S, Kim S: Barrier membrane techniques in endodontic microsurgery, Dent Clin North Amer 41:585, 1997.
956. Pecora G, Kim S, Celleti R, Davarpanah M: The guided tissue regeneration principle in endodontic surgery: one year postoperative results of large periapical lesions, Int Endod J 7:76, 1995.
957. Perlmutter S, Tagger M, Tagger E, Abram M: Effect of the endodontic status of the tooth on experimental periodontal reattachment in baboons: a preliminary investigation, Oral Surg Oral Med Oral Pathol 63:232, 1987.

999. Lewenstein I, Hirschfield Z, Stabholz A, Rotstein I: Effect of hydrogen peroxide and sodium perborate on the microhardness of human enamel and dentin, J Endod 20(2):6\, 1994.
1000. Li Y, Noblitt T, Zhang A, Origel A, Kaftaway A, Tookey K: Effects of long-term exposure to a tooth whitener, J Dent Res 0(Spec Iss):325 (abstract).
1001. Madison S, Walton RE: Cervical root resorption following bleaching of endodontically treated teeth, J Endod 16:570, 1990.
1002. McCloskey RJ: A technique for removal of fluorosis stain, Am Dent Assoc 109:64, 1984.
1003. McEvoy S: Chemical agents for removing intrinsic stains from vital teeth, Quintessence Int 20:323, 1589.
1004. McGuikin RS, Babin JF, Meyer BJ: Alterations in human enamel surface morphology following vital bleaching, J Prosthet Dent 68:7 54, 1992.
1005. Montgomery S: External cervical resorption after bleaching a pulpless tooth, Oral Surg 57:203, 1984.
1006. Muller CJF, Van Wyk CW: The amelocemental junction, J Dent Assoc 39: 799, 1984.
1007. Nathoo SA: The chemistry and mechanisms of extrinsic and intrinsic discoloration, J ADA 128:68, 1997.
1008. Reise-Schmidt T: Trends in dentistry. Longer, whiter, brighter: trends in tooth-whitening products and procedures, Dental Products Report, July 1996.
1009. Rotstein I et al: *In vitro* efficacy of sodium perborate preparations used for intracoronal bleaching of discolored non-vital teeth, Endod Dent Traumatol 7:177,1991.
1010. Rotstein I, Danker E, Goldman A, Heling I, Stabholz A, Zalkind M: Histochemical analysis of hard tissues following bleaching, J Endod 22::23, 1996.
1011. Rotstein I, Lehr T, Gedalia I: Effects of bleaching agents on the inorganic components of human dentine and cementum, J Endod 18:290, 1992.
1012. Rotstein I, Lewenstein I, Zuwabi O, Stabholz A, Friedman M: Effect of cervical coating of ethyl cellulose polymer and metacrylic acid copolymer on the radicular penetration of hydrogen peroxide during bleaching, Endod Dent Traumatol 8:202, 1992.
1013. Rotstein I, Torek Y, Lewinstein I: Effect of bleaching time and temperature on the radicular penetration of hydrogen peroxide, Endod Dent Traumatol 4:32, 1988.
1014. Rotstein I, Torek Y, Misgav R: Effect of cementum defects on radicular penetration of 30% hydrogen peroxide during intracoronal bleaching, J Endod 17:230, 1991.
1015. Schroeder HE, Scherle WF: Cemento-enamel junction revisited, J Periodontal Res 23:53, 1988.
1016. Shannon H, Spencer P, Gross K, Tira D: Characterization of enamel exposed to 10% carbamide peroxide bleaching agents, Quintessence Int 24:39, 1993.
1017. Swift EJ: A method for bleaching discolored teeth, Quintessence Int 19:607, 1988.
1018. Swift EJ: Restorative considerations with vital tooth bleaching, JADA 128:608, 1997.
1019. Swift EJ Jr, Perdigai J: Effect of bleaching on teeth and restorations, Compendium 19(8):815, 1998.
1020. Titley K, Torneck CD, Ruse ND: The effect of carbamide-peroxide gel on the shear bond strength of a microfil resin to bovine enamel, J Dent Res 71:20, 1992.
1021. Titley K, Torneck CD, Ruse ND, Krmec D: Adhesion of resin composite to bleached and unbleached enamel, J Endod 19: 112,1993.
1022. Abedi HR, Torabinejad M, Pitt-Ford TR, Bakland LK: The use of mineral tri-oxide aggregate cement (MTA) as a direct pulp-capping agent, J Endod 22:199, 1996 (abstract).
1023. Akimoto N et al: Biocompatability of Clearfil linear bond 2 and Clearfil AP-X system on nonexposed and exposed primate teeth, Quintessence Int 22:177, 1998.
1024. Alacam A: Long-term effects of primary teeth pulpotomies with formocresol, glutaraldehyde-calcium hydroxide and glutaraldehyde-zinc oxide-eugenol on succedaneous teeth, J Peridontol 13:307, 1989.
1025. Andrew P: The treatment of infected pulps in deciduous teeth, Br Dent J 98:122, 1955.
1026. Anthony DR: Apexification during active orthodontic movement, J Endod 12:419, 1986.
1027. Aponte AJ, Hartsook JT, Crowley MC: Indirect pulp capping success verified, J Dent Child 33:164, 1966.
1028. Armstrong RL et al: Comparison of Dycal and formocresol pulpotomies in young permanent teeth in monkeys, Oral Surg 48:160, 1979.
1029. Attala MN, Noujaim AA: Role of calcium hydroxide in the formation of reparative dentin, J Can Dent Assoc 35:267, 1969.
1030. Avram DC, Pulver F: Pulpotomy medicaments for vital primary teeth: surveys to determine use and attitudes in pediatric dental practice and in dental schools throughout the world, J Dent Child 56:426, 1989.
1031. Bevelander G, Benzer D: Morphology and incidence in secondary dentin in human teeth, J Am Dent Assoc 30:1079, 1943.
1032. Block RM et al: Cell-mediated immune response to dog pulp tissue altered by formocresol within the root canal, J Endod 3:424, 1977.
1033. Brian JD et al: Reaction of rat connective tissue to unfixed and formaldehyde-fixed autogenous implants enclosed in tubes, J Endod 6:628,1980.
1034. Chosack A, Cleaton-Jones P: A histological and quantitative histomorphometric study of apexification of nonvital permanent incisors of vervet monkeys after repeated root fillings with a calcium hydroxide paste, Endod Dent Traumatol 13: 211,1997.
1035. Coll JA, Sadrian R: Predicting pulpectomy success and its relationship to exfoliation and succedaneous dentition, Pediatr Dent 18:51,1996.
1036. Coviello J, Brilliant JD: A preliminary clinical study of the use of tricalcium phosphate as an apical barrier, J Endod 5:6, 1979.
1037. Guidelines for pulp therapy for primary and young permanent teeth: American Academy of Pediatric Dentistry reference manual, Pediatr Dent 18:44, 1996.
1038. Guthrie TJ, McDonald RE, Mitchell DF: Dental hemogram, J Dent Res 44:678, 1965.

1039. Gwinnett AJ, Tay FR: Early and intermediate time response of the dental pulp to an acid etch technique *in vivo*, Am J Dent 11:835, 1998 (special issue).
1040. Ham JW, Patterson SS, Mitchell DF: Induced apical closure of [illegible] pulpless teeth in monkeys, Oral Surg 33:438,1972.
1041. Hata G et al: Systemic distribution of 14C-labeled formaldehyde applied in the root canal following pulpectomy, JEndod 15:539, 1989.
1042. Hawes RR, Dimaggio JJ, Sayegh F: Evaluation of direct and indirect pulp capping, J Dent Res 43:808, 1964 (abstract).
1043. Hayashi Y: infrastructure of initial calcification in wound healing following pulpotomy, J Oral Pathol 11:174, 1982.
1044. Hebling J, Giro EMA, deSouza Costa CA: Biocompatability of an adhesive system applied to exposed human dental pulp, J Endod25:61'6, 1999.
1045. Heide S: Pulp reactions to exposure for 4, 24 and 168 hours, J Dent Res 59:1910, 1980.
1046. Heide S, Kerekes K: Delayed partial pulpotomy in permanent incisors of monkeys, Int Endod J 19:78, 1986.
1047. Hernandez R, Bader S, Boston D, Trope M: Resistance to fracture of endodontically treated premolars restored with new generation dentin bonding systems, Int Endod J 27:281,1994.
1048. Hibbard ED, Ireland RL: Morphology of the root canals of the primary molar teeth, J Dent Child 24:250, 1957.
1049. Hill S et al: Comparison of antimicrobial and cytotoxic effects of glutaraldehyde and formocresol, Oral Surg Oral Med Oral Pathol 11:89, 1991.
1050. Holan G, Fuks AB: A comparison of pulpectomies using ZOE and KRI paste in primary molars: a retrospective study, Pediatr Dent 15:403, 1993.
1051. Holan G, Topf J, Fuks AB: Effect of root canal infection and treatment of traumatized primary incisors on their permanent successors, Endod Dent Traumatol 8:12, 1992.
1052. Ireland RL: Secondary dentin formation in deciduous teeth, J Am Dent Assoc 28:1626, 1941.
1053. Jeng HW, Feigal RJ, Messer HH: Comparison of the cytotoxicity of formocresol, formaldehyde, cresol, and glutaraldehyde using human pulp fibroblast cultures, Pediatr Dent 9:295, 1987.
1054. Junn DJ, McMillan P, Bakland LK, Torabinejad M: Quantitative assessment of dentin bridge formation following pulp-capping with mineral trioxide aggregate (MTA), J Endod 24:278, 1998 (abstract).
1055. Kaiser JH: Management of wide-open canals with calcium hydroxide. Paper presented at the meeting of the American Association of Endodontics, Washington, DC, April 17,1964. Cited by Steiner JC, Dow PR, Cathey GM: Inducing root end closure of nonvital permanent teeth, J Dent Child 35:47,1968.
1056. Kakehashi S, Stanley HR, Fitzgerald RT: The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats, Oral Surg 20:340, 1965.
1057. Kalnins V, Frisbie HE: Effect of dentin fragments on the healing of the exposed pulp, Arch Oral Biol 2:96, 1960.
1058. Kanka J III: An alternative hypothesis to the cause of pulpal inflammation in teeth treated with phosphoric acid on the dentin, Quintessence Int 21:83, 1990.
1059. Karp WB, Korb P, Pashley D: The oxidation of glutaraldehyde by rat tissues, Pediatr Dent 9:301, 1987.
1060. Katebzadeh N, Dalton BC, Trope M: Strengthening immature teeth during and after apexification, J Endod 24:256, 1998.
1061. Kato S, Fusayama T: Recalcification of artificially decalcified dentin *in vivo*, J Dent Res 49:1060, 1970.
1062. Kelley MA, Bugg JL, Skjonsby HS: Histologic evaluation of formocresol and oxpara pulpotomies in rhesus monkeys, J Am Dent Assoc 86:123, 1973.
1063. Kennedy DB et al: Formocresol pulpotomy in teeth of dogs with induced pulpal and periapical pathosis, J Dent Child 40:44, 1973.
1064. Kerkhove BC et al: A clinical and television densitometric evaluation of the indirect pulp capping technique, J Dent Child 34:192, 1967.
1065. King JB, Crawford JJ, Lindahl RL: Indirect pulp capping: a bacteriologic study of deep carious dentine in human teeth, Oral Surg 20:663, 1965.
1066. Kitasako Y, Inokoshi S, Tagami J: Effects of direct resin pulp capping techniques on short-term response of mechanically exposed pulps, J Dent 27:257, 1999.
1067. Kleier DJ, Barr ES: A study of endodontically apexified teeth, Endod Dent Traumatol 7:112,1991.
1068. Klein H et al: Partial pulpotomy following complicated crown fracture in permanent incisors: a clinical and radiographic study, JPedodontol 9:142, 1985.
1069. Koenigs JF et al: Induced apical closure of permanent teeth in adult primates using a resorbable form of tricalcium phosphate ceramic, J Endod 1:102, 1975.
1070. Kopel HM et al: The effects of glutaraldehyde on primary pulp tissue following coronal amputation: an *in vivo* histologic study, J Dent Child 47:425, 1980.
1071. Kuboki Y, Ohgushi K, Fusayama T: Collagen biochemistry of the two layers of carious dentin, J Dent Res 56:1233, 1977.
1072. Langeland K: Management of the inflamed pulp associated with deep carious lesion, J Endod 7:169, 1981.
1073. Langeland K et al: Human pulp changes of iatrogenic origin, Oral Surg 32:943, 1971.
1074. Laws AJ: Pulpotomy by electro-coagulation, N Z Dent J 53:68, 1957.
1075. Lekka M, Hume WR, Wolinsky LE: Comparison between formaldehyde and glutaraldehyde diffusion through the root tissues of pulpotomy-treated teeth, JPedodontol 8:185, 1984.
1076. Liu J et al: Laser pulpotomy of primary teeth, J Pediatr Dent 21:128, 1999.
1077. Lloyd JM, Scale NS, Wilson CFG: The effects of various concentrations and lengths of application of glutaraldehyde on monkey pulp tissue, Pediatr Dent 10:115, 1988.
1078. Longwill DG, Marshall FJ, Creamer HR: Reactivity of human lymphocytes to pulp antigens, J Endod 8:27, 1982.
1079. Loos PJ, Han SS: An enzyme histochemical study of the effect of various concentrations of formocresol on connective tissues, Oral Surg 3:571, 1971.
1080. Rabinowitch BZ: Pulp management in primary teeth, Oral Surg 6:542, 1953.

1081. Ranley DM: Glutaraldehyde purity and stability: implications for preparation, storage, and use as a pulpotomy agent, Pediatr Dent 6:83, 1984.
1082. Ranley DM: Assessment of the systemic distribution and toxicity of formaldehyde following pulpotomy treatment. I, JDent Child 52:431, 1985.
1083. Ranley DM: Pulpotomy therapy in primary teeth: new modalities for old rationals, Pediatr Dent 6:403, 1994.
1084. Ranley DM, Garcia-Godoy F, Horn D: Time, concentration, and pH parameters for the use of glutaraldehyde as a pulpotomy agent: an *in vivo* study, Pediatr Dent 9:199, 1987.
1085. Ranley DM, Horn D, Zislis T: The effect of alternatives to formocresol on antigencity of protein, J Dent Res 64:1225, 1985.
1086. Ranley DM, Amstutz L, Horn D: Subcellular localization of glutaraldehyde, Endod Dent Traumatol 6:251, 1990.
1087. Ranley DM, Horn D: Distribution, metabolism, and excretin of (HC) glutaraldehyde, J Endod 16:135, 1990.
1088. Rickman GA, Elbadrawy HE: Effect of premature loss of primary incisors on speech, Pediatr Dent 7:119, 1985.
1089. Rimondini L, Baroni C: Morphologic criteria for root canal treatment of primary molars undergoing resorption, Endod Dent Traumatol 11:136, 1995.
1090. Roberts SC Jr, Brilliant JD: Tricalcium phosphate as an adjunct to apical closure in pulpless permanent teeth, J Endod 1:263, 1975.
1091. Rolling I, Poulsen S: Formocresol pulpotomy of primary teeth and occurrence of enamel defects on the permanent successors, Acta Odontol Scand 36:243, 1978.
1092. Ruemping DR, Morton TH Jr, Anderson MW: Electrosurgical pulpotomy in primates—a comparison with formocresol pulpotomy, Pediatr Dent 5:14, 1983.
1093. Sadrian R, Coll JA: A long-term follow-up on the retention of zinc oxide eugenol filler after primary tooth pulpectomy, Pediatr Dent 15:249, 1993.
1094. Savage NW et al: A histological study of cystic lesions following pulp therapy in deciduous molars, Oral Pathol 15: 209, 1986.
1095. Sayegh FS: Qualitative and quantitative evaluation of new dentin in pulp capped teeth, J Dent Child 35:7, 1968.
1096. Sayegh FS: The dentinal bridge in pulp-involved teeth. I, Oral Surg 28:579, 1969.
1097. Schumacher JW, Rutledge RE: An alternative to apexification,J Endod 19:529, 1993.
1098. Sciaky I, Pisanti S: Localization of calcium placed over amputated pulps in dogs' teeth, J Dent Res 39:1128, 1960.
1099. Shabahang S et al: A comparative study of root-end induction using osteogenic protein-I, calcium hydroxide, and mineral trioxide aggregate in dogs, J Endod 25: 1999.
1100. Shaw DW et al: Electrosurgical pulpotomy—a 6-month study in primates, J Endod 13:500, 1987.
1101. Sheller B, Morton TH Jr: Electrosurgical pulpotomy: a pilot study in humans, J Endod 13:69, 1987.
1102. Shoji S, Nakamura M, Horiuchi H: Histopathological changes in dental pulps irradiated by CO_2 laser: a preliminary report on laser pulpotomy, J Endod 11:379, 1985.
1103. Shovelton DS: A study of deep carious dentin, Int Dent J 18:392, 1968.
1104. Simon M, van Mullem PJ, Lamers AC: Formocresol: no allergic effect after root canal disinfection in non-presensitized guinea pigs, J Endod 8:269, 1982.
1105. Snuggs HM, Cox CF, Powell CF et al: Pulp healing and dentinal bridge formation in an acidic environment, Quintessence Int 24:501, 1993.
1106. Spedding RH: The one-appointment formocresol pulpotomy for primary teeth, J Tenn DentAssoc 48:263, 1968.
1107. Stanley HR, Lundy T: Dycal therapy for pulp exposure, Oral Surg 34:818, 1972.
1108. Stanley HR, White CL, McCray L: The rate of tertiary (reparative) dentine formation in the human tooth, Oral Surg 21: 180, 1966.
1109. Stanton WG: The non-vital deciduous tooth, Int J Orthod 21: 181,1935.
1110. Starkey PE: Methods of preserving primary teeth which have exposed pulps, J Dent Child 30:219, 1963.
1111. Steiner JC, Dow PR, Cathey GM: Inducing root end closure of nonvital permanent teeth, J Dent Child 35:47, 1968.
1112. Steiner JC, Van Hassel HJ: Experimental root apexification in primates, Oral Surg 31:409, 1971.
1113. Straffon LH, Han SS: Effects of varying concentrations of formocresol on RNA synthesis of connective tissue in sponge implants, Oral Surg 29:915, 1970.
1114. Sweet CA: Procedure for the treatment of exposed and pulpless deciduous teeth, J Am DentAssoc 17:1150, 1930.
1115. Tagger M, Tagger E: Pulp capping in monkeys with Reolite and Life, two calcium hydroxide bases with different pH, J Endod 11:394, 1985.
1116. Tagger E, Tagger M, Sarnat H: Pulpal reaction for glutaraldehyde and paraformaldehyde pulpotomy dressings in monkey primary teeth, Endod Dent Traumatol 2:237, 1986.
1117. Tatsumi T et al: Remineralization of etched dentin, J Prosthet Dent 67:617, 1992.
1118. Teplitsky PE: Formocresol pulpotomies on posterior permanent teeth, J Can Dent Assoc 50:623, 1984.
1119. Thoden van Velzen SK, Feltkamp-Vroom TM: Immunologic consequences of formaldehyde fixation of autologous tissue implants, J Endod 3:179,1977.
1120. Abbott PV, Hume WR, Heithersay GS: Effects of combining Ledermix and calcium hydroxide pastes on the diffusion of corticosteroid and tetracycline through human roots *in vitro*, Endod Dent Traumatal 5:188, 1989.
1121. Andersson L, Friskopp J, Blomlof L: Fiber-glass splinting of traumatized teeth, J Dent Child 3:21, 1983.
1122. Andreasen FM: Transient apical breakdown and its relation to color and sensibility changes after luxation injuries to teeth, Endod Dent Traumatol 2:9, 1986.
1123. Andreasen FM, Andreasen JO: Resorption and mineralization processes following root fracture of permanent incisors, Endod Dent Traumatol 4:202, 1988.
1124. Andreasen FM, Andreasen JO, Bayer T: Prognosis of root-fractured permanent incisors—prediction of healing modalities, Endod Dent Traumatol 5:11, 1989.

1125. Andreasen FM, Pedersen BV: Prognosis of luxated permanent teeth and the development of pulp necrosis, Endod Dent Traumatol 1:207, 1985.
1126. Andreasen FM, Zhijie Y, Thomson BL, Andersen PK: The occurrence of pulp canal obliteration after luxation injuries in the permanent dentition, Endod Dent Traumatol 3:103,1987.
1127. Andreasen JO: Luxation of permanent teeth due to trauma. A clinical and radiographic follow-up study of 189 injured teeth, Scand JDent Res 78:273,1970.
1128. Andreasen JO: Etiology and pathogenesis of traumatic dental injuries, Scand J Dent Res 78:329,1970.
1129. Andreasen JO: Periodontal healing after replantation of traumatically avulsed human teeth: assessment by mobility testing and radiography, Acta Odontol Scand 33: 325, 1975.
1130. Andreasen JO: The effect of extra-alveolar period and storage media upon periodontal and pulpal healing after replantation of mature permanent incisors in monkeys, Int J Oral Surg 10: 43, 1981.
1131. Andreasen JO, Hjorting-Hansen E: Replantation of teeth. I. Radiographic and clinical study of 110 human teeth replanted after accidental loss, Acta Odontol Scand 24:263, 1966.
1132. Andreasen JO, Hjorting-Hansen E: Intra-alveolar root fractures: radiographic and histologic study of 50 cases, J Oral Surg 25:414, 1967.
1133. Andreasen JO, Kristersson L: The effect of limited drying or removal of the periodontal ligament: periodontal healing after replantation of mature permanent incisors in monkeys, Acta Odontol Scand 39:1,1981.
1134. Andreasen JO, Ravn JJ: Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample, Int J Oral Surg 1:235, 1972.
1135. Antrim DD, Hicks ML, Altaras DE: Treatment of subosseous resorption: a case report, J Endod 8:567, 1982.
1136. Bergenholtz G: Microorganisms from necrotic pulp of traumatized teeth, Odont Rev 25:247,1974.
1137. Bergenholtz G, Reit C: Pulp reactions on microbial provocation of calcium hydroxide treated dentin, Scand J Dent Res 88:187,1980.
1138. Bhaskar SN, Rappaport HM: Dental vitality tests and pulp status, J Am DentAssoc 86:409, 1973.
1139. Binnie WH, Rowe AHR: A histological study of the periapical tissues of incompletely formed pulpless teeth filled with calcium hydroxide, J Dent Res 52:1110, 1973.
1140. Bjorvatn K, Selvig KA, Klinge B: Effect of tetracycline and SnF_2 on root resorption in replanted incisors in dogs, Scand J Dent Res 97:477, 1989.
1141. Blomlof L et al: Storage of experimentally avulsed teeth in milk prior to replantation, J Dent Res 62:912, 1983.
1142. Borum MK, Andreasen JO: Sequelae of trauma to primary maxillary incisors. I. Complications to the primary dentition. Endod Dent Traumatol 14:33, 1998.
1143. Braham RL, Roberts MW, Morris ME: Management of dental trauma in children and adolescents, J Trauma 17:857, 1977.
1144. Brannstrom M: Observations on exposed dentine and corresponding pulp tissue. A preliminary study with replica and routine histology, Odont Revy 13:253,1952.
1145. Heithersay GS: Calcium hydroxide in the treatment of pulpless teeth with associated pathology, J Br Endod Soc 8:74, 1962.
1146. Heide S, Mjor IA: Pulp reactions to experimental exposures in young permanent teeth, Int Endod J 16:11, 1983.
1147. Heller AL et al: Direct pulp capping of permanent teeth in primates using resorbable form of tricalcium phosphate ceramics, J Endod 1:95, 1975.
1148. Hernandez R, Bader S, Boston D, and Trope M: Resistance to fracture of endodontically treated premolars restored with new generation dentin bonding systems, Int Endod J 27:281, 1994.
1149. Hill FJ, Picton JF: Fractured incisor fragment in the tongue: a case report, Pediatr Dent 3:337, 1981.
1150. Hiltz J, Trope M: Vitality of human lip fibroblasts in milk, Hanks Balanced Salt Solution and Viaspan storage media, Endod Dent Traumatoll 69, 1991.
1151. Jacobsen I: Root fractures in permanent anterior teeth with incomplete root formation, Scand J Dent Res 84:210, 1976.
1152. Jacobsen I: Clinical follow-up study of permanent incisors with intrusive luxation after acute trauma, J Dent Res 62:4, 1983.
1153. Jacobsen I, Kerekes K: Long-term prognosis of traumatized permanent anterior teeth showing calcific processes in the pulp cavity, Scand J Dent Res 85:588, 1977.
1154. Jacobsen I, Kerekes K: Diagnosis and treatment of pulp necrosis in permanent anterior teeth with root fracture, Scand J Dent Res 88:370, 1980.
1155. Jacobsen I, Zachrisson BU: Repair characteristics of root fractures in permanent anterior teeth, Scand J Dent Res 83: 355, 1975.
1156. Johnston T, Messer LB: An *in vitro* study of the efficacy of mouthguard protection for dentoalveolar injuries in deciduous and mixed dentitions, Endod Dent Traumatol 12:277, 1996.
1157. Jarvinen S: Incisal overject and traumatic injuries to upper permanent incisors: a retrospective study, Acta Odontol Scand 36:359, 1978.
1158. Jarvinen S: Fractured and avulsed permanent incisors in Finnish children: a retrospective study, Acta Odontol Scand 37:47, 1979.
1159. Kakehashi S, Stanley HR, Fitzgerald RJ: The effect of surgical exposures on dental pulps in germ-free and conventional laboratory rats, Oral Surg 20:340, 1965.
1160. Katebzadeh N, Dalton C, Trope M: Strengthening immature teeth during and after apexification, J Endod 11:256, 1998.
1161. Kerekes K, Heide S, Jacobsen I: Follow-up examination of endodontic treatment in traumatized juvenile incisors, J Endod 6:744, 1980.
1162. Kerr LI: Mouth guards of the prevention of injuries in contact sports, Sports Med 3:415,1986.
1163. Kopel HM, Johnson R: Examination and neurologic assessment of children with oro-facial trauma, Endod Dent Traumatol 1:155, 1985.
1164. Lado EA, Stanley HR, Weissman MI: Cervical resorption in bleached teeth, Oral Surg 55:78, 1983.

1209. Alexander RE: Eleven myths of dentoalveolar surgery, JADA 129:1271, 1998.
1210. Aqrabawi J: Sealing ability of a Super EBA & MTA when used as a retrograde filling material, Br Dent J 11:266, 2000.
1211. Baldissara P, Baldissara S, Scotti R: Reliability of tactile perception using sharp and dull explorers in marginal opening identification, Int J Prosthodont 11:591, 1998.
1212. Berhelsen CL, Stilley KR: Automated personal health inventory for dentistry: a pilot study, JADA 131:59, 2000.
1213. Berry J: Physicians report boost in use of world wide web, ADA News 31(1): 3, January 10, 2000.
1214. Brown R, Hadley JN, Chambers DW: An evaluation of Ektaspeed Plus film versus. Ultraspeed film for endodontic working length determination, J Endod 24:54, 1998.
1215. California court upholds ban on AIDS discrimination, ADA News 21(3): 8, February 5, 1990.
1216. Cheung G: Endodontic failures, Int Dent J 146:131, 1996.
1217. Dajani AS et al: Prevention of bacterial endocarditis. Recommendations by the American Heart Association, JADA 128(8): 1142,1997.
1218. Davis M: Dentistry and AIDS: an ethical opinion, J Am Dent Assoc 119:(suppl 9-5) 95, 1989.
1219. Dorn S, Gartner A: Retrograde filling materials: a retrospective study of amalgam, EBA, and IRM, J Endod 16:391, 1990.
1220. 21a.Epstein JB, Chong S, Le ND: A survey of antibiotic use in dentistry, J Am Dent 131, 1600, 2000.
1221. Gagliani M, Taschieri S, Molinari R: Ultrasonic root end preparation: influence of cutting angle on the apical seal, J Endod 24:726, 1998.
1222. Goldman RP: Your duty to refer, ADA Legal Adviser 2(9):6, 1998.
1223. Simonsen R: Greed and gravy train: is this success? J Esthetic Dent 11:287, 1999.
1224. Terezhalmy G, Bottomley W: General legal aspects of diagnostic dental radiography, Oral Surg 48:486, 1979.
1225. Tsang A, Sweet D, Wood RE: Potential for fraudulent use of dental radiography, JADA 130:1325, 1999.
1226. Walton RE: News: Antibiotics not always necessary, JADA 130:782, 1999.
1227. Weichman J: Malpractice prevention and defense, J Calif Dent Assoc 3(8):58, 1975.
1228. Wensing M, Van der Weijden T, Grol R: Implementing guidelines and innovations in general practice: which interventions are effective? Br J Gen Pract 48:991, 1998.
1229. Yatsushiro JD, Baumgartner JC, Tinkle JS: Longitudinal study of microleakage of two root end filling materials using a fluid conductive system, J Endod 24:716, 1998.
1230. Zeider S, Ruttimann U, Webber R: Efficacy in the assessment of intraosseous lesions of the face and jaws in asymptomatic patients, Radiology 162:691, 1987.

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