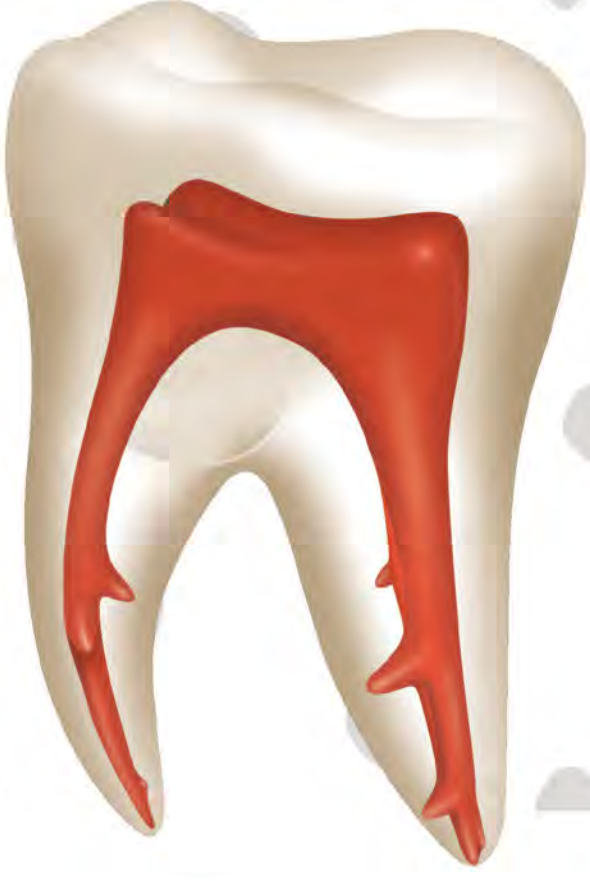
Complimentary Online Resource

Textbook of Endodontics



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5th Edition

JAYPEE

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3 Pathologies of Pulp and Periapex

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INTRODUCTION

Dental pulp consists of vascular connective tissue contained within the rigid dentin walls. It is the principal source of pain in oral cavity and also a major site of attention in endodontics and restorative procedures. Thus, knowledge of pulp is essential not only for providing dental treatment but also to know the rationale behind the treatment provided.

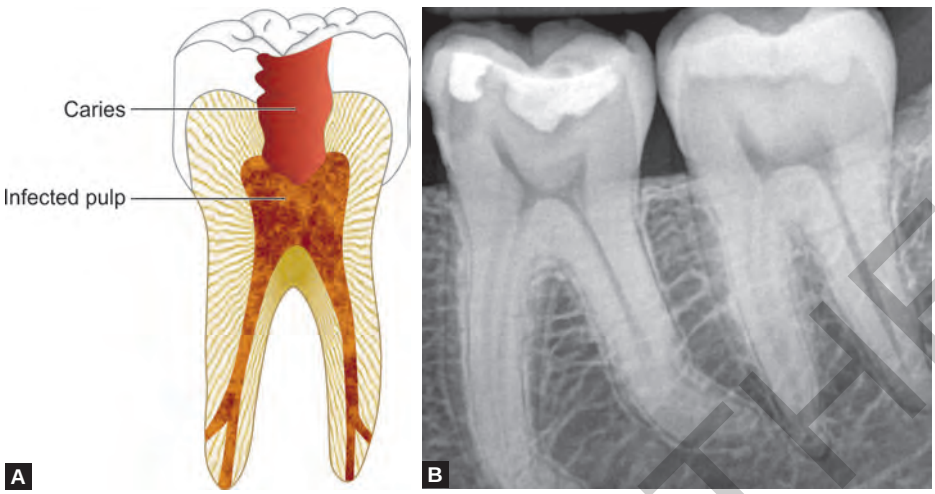
Important features of pulp:

- ❑ It is a coherent soft tissue, surrounded by dentin which limits the area for expansion and restricts its ability to tolerate edema.
- ❑ Odontoblasts present in pulp have ability to form dentin in response to caries and irritants.
- ❑ Pulp has almost total lack of collateral circulation. This limits its ability to cope with bacteria, necrotic tissue, and inflammation.
- ❑ It gives radiographic appearance as radiolucent line.

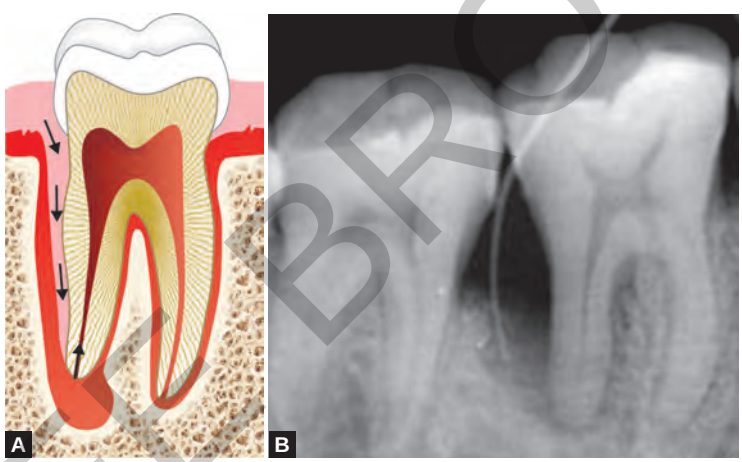
ETIOLOGY OF PULPAL DISEASES

❖ Classification of etiology according to *WEINE* beginning with the most common irritant:

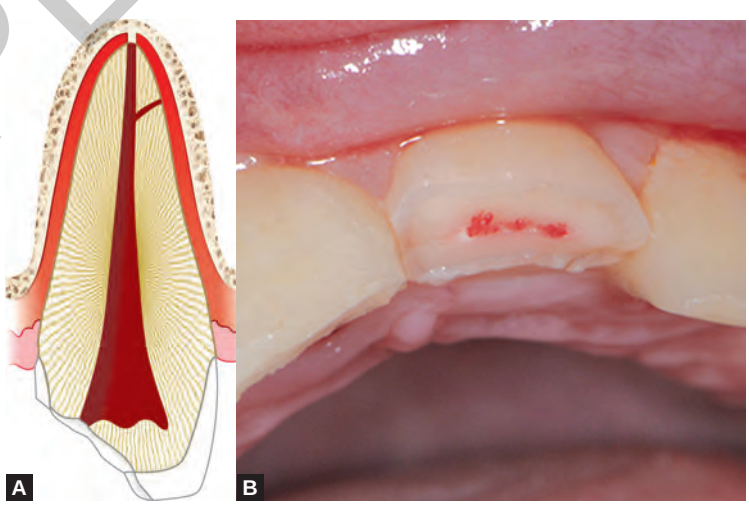
- **Bacterial:** Most common cause of pulpal injury is bacteria or their by-products which may enter the pulp through a break in dentin from:
  - Caries **(Figs. 3.1A and B).**
  - Accidental exposure.
  - Fracture.
  - Percolation around a restoration.
  - Extension of infection from gingival sulcus.
  - Periodontal pocket and abscess **(Figs. 3.2A and B)**.
  - Anachoresis (process by which microorganisms get carried by the bloodstream from another source and localize on inflamed tissue).
- **Traumatic:**
  - Acute trauma like fracture, luxation, or avulsion of tooth **(Figs. 3.3A and B)**.
  - Chronic trauma including parafunctional habits like bruxism.
- **Iatrogenic:** Pulp inflammation resulting from clinician's own procedures is referred to as dentistogenic pulpitis. Iatrogenic causes of pulp pathologies can be:
  - *Thermal changes* caused during tooth preparation, restoration, bleaching of enamel, electrosurgical procedures, etc.
    - There occurs increase in 20 degree in temperature during dry tooth preparation



Figs. 3.1A and B: (A) Schematic representation of deep caries; (B) Radiograph showing deep caries approximating pulp.



Figs. 3.2A and B: (A) Schematic representation showing how periodontal pocket or abscess can cause pulpal inflammation via portals of communication like apical foramen or lateral canals; (B) Radiograph showing endodontic periodontal lesion.



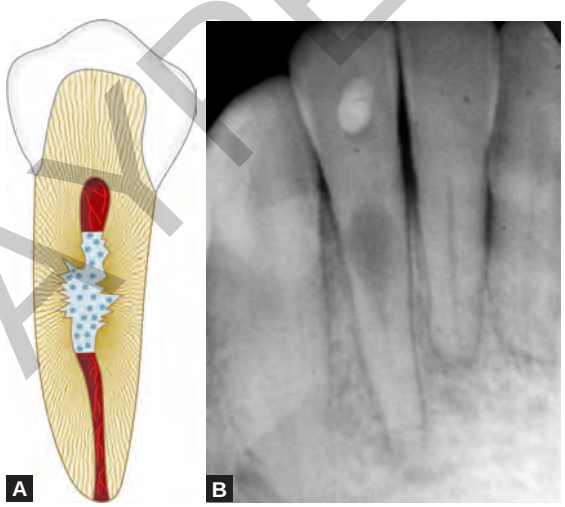
Figs. 3.3A and B: (A) Schematic representation of tooth fracture approximating pulp; (B) Clinic photograph showing tooth fracture involving dental pulp.

1 mm from pulp and 30 degree increase 0.5 mm from pulp. This heat generated can result in irreversible pulp damage.
Therefore, continuous use of water or air water spray is recommended during tooth preparation.
- During tooth preparation and placing a restoration, the remaining dentin thickness (RDT), determinants the extent of pulp damage due to heat generation by these procedures.

- Orthodontic movement.
- Periodontal curettage.
- Periapical curettage.
- Use of chemicals like temporary and permanent restorations, liners, bases, and use of cavity desiccants such as alcohol.

- **Idiopathic:**
  - Aging.
  - Resorption; internal or external **(Figs. 3.4A and B)**

Radiation injury to pulp
- Pulp cells exposed to ionizing radiation may become necrotic, show vascular damage and the interference in mitosis of cells.
- Irradiation affects salivary glands resulting in decreased salivary flow, thereby increased predisposition to dental caries and pulpal involvement.
- Effects of radiation damage to teeth depend on dose, source, type of radiation, exposure factor, and stage of tooth development at the time of irradiation.



Figs. 3.4A and B: (A) Line diagram showing internal resorption of tooth; (B) Radiograph showing internal resorption.

PROGRESSION OF PULPAL PATHOLOGIES

Pulp reacts to above mentioned irritants same as other connective tissues. A normal pulp gives mild-to-moderate response to pulp tests and this response subsides on removal of stimulus. Degree of inflammation is proportional to the intensity and severity of the tissue damage. For example, slight irritation like incipient caries or shallow tooth preparation causes little or no pulpal inflammation, whereas extensive operative procedures may lead to severe pulpal inflammation.

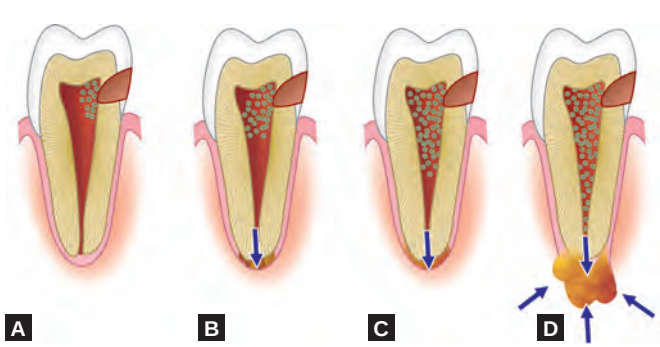
Depending on condition of pulp, severity and duration of irritant, host response, pulp may respond from mild inflammation to pulp necrosis. These changes may not be accompanied by pain and thus may proceed unnoticed.

Pulpal reaction to microbial irritation (Figs. 3.5A to D)

Microorganisms present in carious enamel and dentin
↓
Penetration of microorganisms in deeper layers of carious dentin
↓
Pulp is affected before actual invasion of bacteria via their toxic by-products
↓
By-products cause local chronic cell infiltration
↓
When actual pulp exposure occurs, pulp tissue gets locally infiltrated by PMNs 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

Pulp Inflammation and its Sequelae

Traditional theory which explained pulpal inflammation and its sequelae was referred to as strangulation theory. Strangulation theory is no longer accepted and a current theory explains the sequelae of pulpal inflammation.



Figs. 3.5A to D: Schematic representation of gradual pulpal response to dental caries.

Strangulation Theory

It says that on irritation, there is local inflammation in pulp, which results in vasodilation, increased capillary pressure and permeability. These result in increased filtration from capillaries into tissues, thus increased tissue pressure. By this, thin vessel walls get compressed resulting in decreased blood flow and increased venous pressure. This results in vicious cycle, because increase in venous pressure further increases capillary pressure. Consequently, choking/strangulation of pulpal blood vessels occur because of increased tissue pressure. This results in ischemia and further necrosis.

Current Theory

Many studies have shown that increase of pressure in one area does not affect the other areas of pulp. Therefore, local inflammation in pulp results in increased tissue pressure in inflamed area and not the entire pulp cavity.

It is seen that injury to coronal pulp results in local disturbance, but if injury is severe, it results in complete stasis of blood vessels in and near the injured area. Net absorption of fluid into capillaries in adjacent uninflamed area results in increased lymphatic drainage thus keeping the pulpal volume almost constant.

Limited increase in pressure within affected pulpal area is explained by the following mechanism:

- ❑ Increased pressure in inflamed area favors net absorption of interstitial fluids from adjacent capillaries in uninflamed tissues.
- ❑ Increased interstitial tissue pressure lowers the transcapillary hydrostatic pressure difference, thus opposes further filtration.
- ❑ Increased interstitial fluid pressure increases lymphatic drainage.
- ❑ Break in endothelium of pulpal capillaries facilitate exchange mechanism.

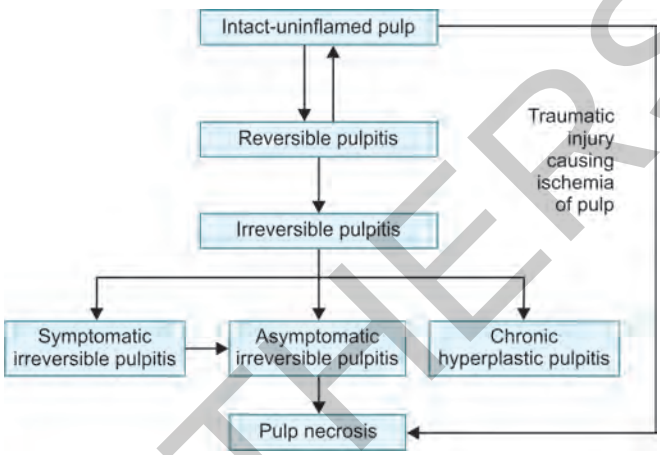
Points to Remember

Degree and nature of inflammatory response caused by microbial irritants depends upon:

- ❑ Host resistance.
- ❑ Virulence of microorganisms.
- ❑ Duration of exposure.
- ❑ Lymphatic drainage.
- ❑ Amount of circulation in affected area.
- ❑ Opportunity of release of inflammatory fluids.

Infectious sequelae of pulpitis include apical periodontitis, periapical abscess/cellulitis, and osteomyelitis of the jaw **(Flowchart 3.1)**. Spread from maxillary teeth may cause purulent sinusitis, meningitis, brain abscess, orbital cellulitis, and cavernous sinus thrombosis. Spread from mandibular teeth may cause Ludwig's angina, parapharyngeal abscess, mediastinitis, pericarditis, and empyema.

Flowchart 3.1: Sequel of pulpal inflammation.



DIAGNOSIS OF PULPAL PATHOLOGY

- ❖ **Subjective symptoms:** Most common symptom is pain.
- ❖ **Objective symptoms:**
  - Visual and tactile inspection—3Cs.
    - Color.
    - Contour.
    - Consistency.
  - Thermal tests:
    - *Heat tests*: Use of
      - Warm air.
      - Hot water.
      - Hot burnisher.
      - Hot gutta-percha stick.
    - *Cold tests*:
      - Ethyl chloride spray.
      - Ice pencils.
      - CO_2 snow (temperature –78°C).
  - Electrical pulp testing.
  - Radiographs.
  - Anesthetic tests.
  - Test cavity.

Recent advances in diagnostic aids for pulp pathologies include:

- ❖ Laser Doppler flowmetry.
- ❖ Liquid crystal testing.
- ❖ Hughes Probeye camera.
- ❖ Infrared thermography.
- ❖ Thermocouples.
- ❖ Pulse oximetry.

- Dual wavelength spectrophotometry.
- Plethysmography.
- Xenon-133 radioisotopes.

CLASSIFICATION OF PULPAL PATHOLOGIES

Grossman's Clinical Classification

I. **Inflammatory disease of dental pulp:**
   1. *Reversible pulpitis:*
      a. Acute reversible pulpitis.
      b. Chronic reversible pulpitis.
   2. *Irreversible pulpitis:*
      a. Symptomatic irreversible pulpitis (previously known as acute irreversible pulpitis).
      b. Asymptomatic irreversible pulpitis (previously known as chronic irreversible pulpitis).
      c. Chronic hyperplastic pulpitis (pulpal hyperplasia).
      d. Internal resorption.

II. **Pulp degeneration:**
   1. Calcific degeneration (radiographic diagnosis).
   2. Fibrous degeneration.

III. **Pulp necrosis:**
   1. Pulp necrobiosis (part of pulp is necrotic and infected, rest is irreversibly inflamed).
   2. Pulp necrosis with no signs of infection.
   3. Necrotic and infected pulp.

Baume's Classification

Based on clinical symptoms:

- Asymptomatic, vital pulp which has been injured or involved by deep caries for which pulp capping may be done.
- Pulp with history of pain which is amenable to pharmacotherapy.
- Pulp indicated for extirpation and immediate root filling.
- Necrosed pulp involving infection of radicular dentin accessible to antiseptic root canal therapy.

Seltzer and Bender's Classification

Based on clinical tests of pulp and histological diagnosis:

Treatable without Pulp Extirpation and Endodontic Treatment

- Intact uninflamed pulp.
- Transition stage.
- Atrophic pulp.
- Acute pulpitis.
- Chronic partial pulpitis without necrosis.

Untreatable without Pulp Extirpation and Endodontic Treatment

- Chronic partial pulpitis with necrosis.
- Chronic total pulpitis.
- Total pulp necrosis.

Ingle's Classification

Inflammatory Changes

- Hyper-reactive pulpalgia:
  - Hypersensitivity.
  - Hyperemia.
- Acute pulpalgia.
  - Incipient (may be reversible).
  - Moderate (may be referred).
  - Advanced (relieved by cold).
- Chronic pulpalgia.
- Hyperplastic pulpitis.
- Pulp necrosis.

Retrogressive Changes

- Atrophic pulposis.
- Calcific pulposis.

BARODONTALGIA/AERODONTALGIA

It is pain experienced in a recently restored tooth during low atmospheric pressure. Pain is experienced either during ascent or descent. Chronic pulpitis which appears asymptomatic in normal conditions may manifests as pain at high altitude because of low pressure. It is generally seen in altitude over 5,000 ft but more likely to be observed in 10,000 ft and above.

Rauch classified barodontalgia according to chief complaint:

- **Class I:** In acute pulpitis, sharp pain occurs for a moment on ascent.
- **Class II:** In chronic pulpitis, dull throbbing pain occurs on ascent.
- **Class III:** In necrotic pulp, dull throbbing pain occurs on descent but it is asymptomatic on ascent.
- **Class IV:** In periapical cyst or abscess, severe and persistent pain occurs with both ascent and descent.

Possible mechanism of barodontalgia:

- Direct ischemia resulting from inflammation itself.
- Indirect ischemia resulting from increased intrapulpal pressure due to vasodilatation and fluid diffusion to the tissue.
- Due to expansion of intrapulpal gas which is a by-product of acids, bases, and enzymes of inflamed tissues.
- Due to leakage of gas through vessels because of decreased gas solubility.

REVERSIBLE PULPITIS/HYPEREMIA/ HYPERACTIVE PULPALGIA

This is the first stage of pulpitis giving sharp hypersensitive response to cold, but pain subsides on removal of the stimulus. Patient may describe symptoms of momentary pain and is unable to locate the source of pain. This stage can last for months or years.

Definition

Reversible pulpitis is mild-to-moderate inflammatory condition of pulp caused by noxious stimuli in which the pulp is capable of returning to inflamed state following removal of stimuli.

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

Etiology

In normal circumstances, enamel and cementum act as impermeable barrier to block the patency of dentinal tubules. When a stimulus interrupts this natural barrier, dentinal tubules become permeable, causing inflammation of pulp. Etiological factors can be:

- Dental caries.
- **Trauma:** Acute or chronic occlusal trauma.
- **Thermal injury:**
  - Tooth preparation with dull bur without coolant.
  - Overheating during polishing of a restoration.
  - Keeping bur in contact with teeth too long.
- Chemical stimulus—like sweet or sour foodstuff.

Symptoms

- Characterized by sharp momentary pain, commonly caused by cold stimuli.
- Pain does not occur spontaneously and does not continue after removal of irritant.
- Following insertion of a deep restoration **(Fig. 3.6)**, patient may complain mild sensitivity to temperature changes, especially cold. Such sensitivity may last for a week or longer but gradually, it subsides. This sensitivity is symptom of reversible pulpitis.

Histopathology

- It shows hyperemia to mild-to-moderate inflammatory changes.
- Evidence of disruption of odontoblastic layer.
- Formation of reparative dentin.
- Dilated blood vessels.
- Extravasation of edema fluid.

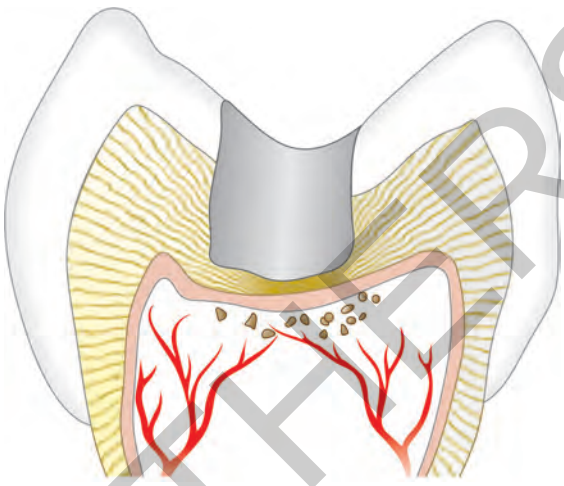


Fig. 3.6: Insertion of deep restoration causing pulp inflammation.

- Presence of immunologically competent chronic inflammatory and occasionally acute cells.

Diagnosis

- **Pain:** It is sharp but of short duration, usually caused by cold, sweet, and sour stimuli. Pain ceases after removal of stimulus.
- **Visual examination and history:** It may reveal caries, recent restoration, traumatic occlusion, and undetected fracture.
- **Radiographic examination:**
  - Shows normal PDL and lamina dura, i.e., normal periapical tissue.
  - Presence of deep dental caries or restoration **(Fig. 3.7)**.
- **Percussion test:** Tooth is normal to percussion and palpation without any mobility.
- **Vitality test:** Pulp responds readily to cold stimuli.

Differential Diagnosis

- In reversible pulpitis, pain disappears on removal of stimuli, whereas in irreversible pulpitis, pain stays longer even after removal of stimulus.
- Patient's description of pain, character, and duration leads to the diagnosis.

Treatment

- The best treatment of reversible pulpitis is prevention.
- Usually, a sedative dressing is placed, followed by permanent restoration when symptoms completely subside.
- Periodic care to prevent caries, desensitization of hypersensitive teeth, and proper pulp protection

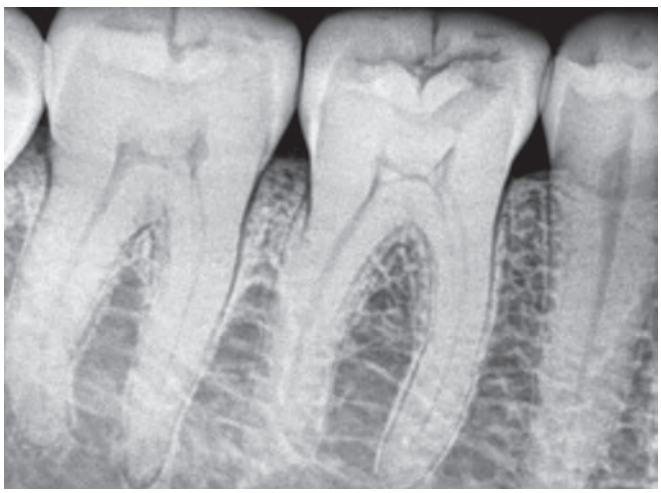


Fig. 3.7: Mandibular left first molar had deep occlusal caries and the patient complained of sensitivity to sweets and cold liquids. There was no discomfort on biting or percussion. The tooth was hyper-responsive to cold with no lingering pain. ***Diagnosis: reversible pulpitis; normal apical tissues.*** Treatment provided was excavation of caries followed by placement of a permanent restoration.

by using cavity varnish or base before placement of restoration is recommended.

- If pain persists despite of proper treatment, pulpal inflammation should be considered as irreversible and it should be treated by pulp extirpation.

Prognosis

Prognosis for pulp is favorable if irritant is removed at early stage otherwise it may result in irreversible pulpitis.

Points to Remember

Threshold to pain decreases in reversible pulpitis because of:

- Release of mediators (endogenous allogenic agents) which initiate or lower the threshold of excitability.
- Release of neuropeptides from unmyelinated C-fibers mediate neurogenic inflammation causing hyper excitability of nerve endings.

IRREVERSIBLE PULPITIS

Definition

It is a persistent inflammatory condition of the pulp, symptomatic or asymptomatic in nature with pulp becoming incapable of healing.

Etiology

Irreversible pulpal inflammation can result from:

- Dental caries (most common cause).
- Chemical, thermal, mechanical injuries of pulp.
- Untreated reversible pulpitis.

Types

- Asymptomatic irreversible pulpitis.
- Symptomatic irreversible pulpitis.

Symptoms

- Rapid onset of pain, 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 from standard to supine.
- Pain is so severe that it keeps the patient awake in night.
- Presence of referred pain.
- In later stages, pain is severe, boring, and throbbing in nature which increases with hot stimulus. Pain is relieved by using cold water. Sometimes pain is so severe that patient may report dental clinic with jar of ice cold water.
- Apical periodontitis develops in later stages when inflammation extends to periodontal ligament.

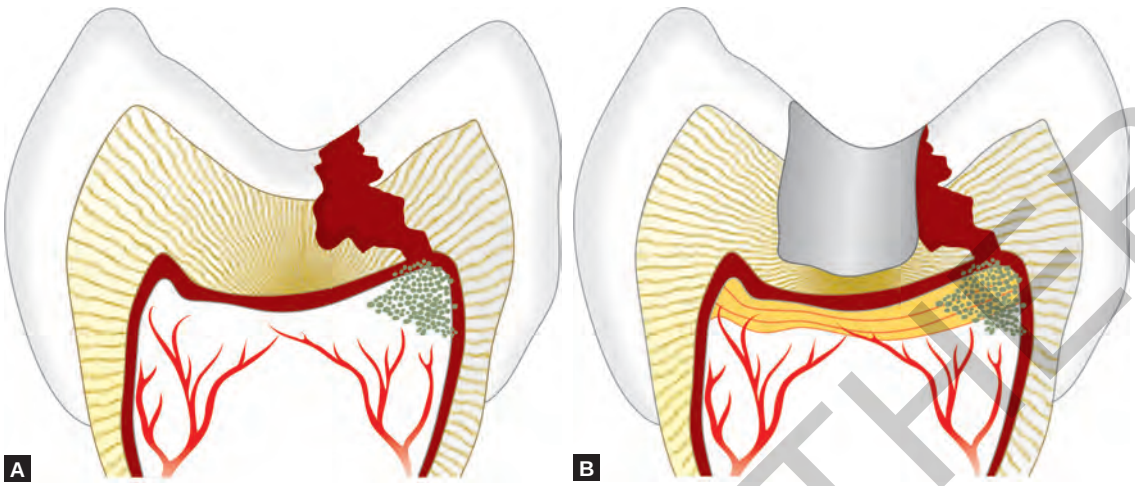
Histopathology

Pulp shows acute and chronic inflammatory changes such as:

- Vascular dilatation and edema.
- Granular cell infiltration.
- Odontoblasts are destroyed.
- Formation of minute abscess formation.
- In later stages, pulp undergoes liquefaction and necrosis.

Diagnosis

- **Visual examination and history:** One may find deep cavity involving pulp **(Fig. 3.8A)** or secondary caries under restorations **(Fig. 3.8B)**.
- **Radiographic findings:**
  - Shows depth and extent of caries **(Fig. 3.9)**.
  - Periapical area shows normal appearance but a slight widening may be evident in advanced stages of pulpitis.



Figs. 3.8A and B: (A) Deep dental caries causing exposure of pulp; (B) Secondary caries beneath restoration causing pulpal exposure.

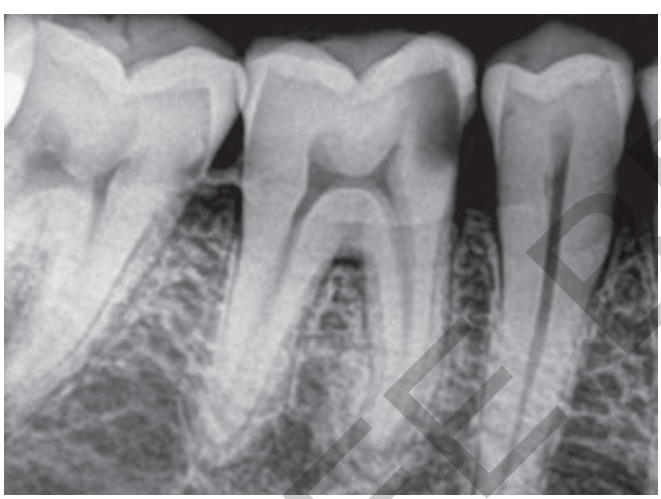


Fig. 3.9: Mandibular left first molar had deep mesio-occlusal caries. Patient complained of sensitivity to hot and cold liquids initially but later pain became spontaneous. EPT test showed lingering pain even after removal of the stimuli. Response to both percussion and palpation was normal. ***Diagnosis: symptomatic irreversible pulpitis.*** Treatment given was nonsurgical endodontic treatment followed by a permanent restoration.

- **Percussion:** Sometimes tooth is tender on percussion because of increased intrapulpal pressure due to exudative inflammatory tissue **(Fig. 3.10)**.
- **Vitality tests:**
  - *Thermal test:* Hyperalgesic pulp responds more readily to cold stimulation than for normal tooth, pain may persist even after removal of irritant. As pulpal inflammation progresses, heat intensifies the response because of its expansible effect on blood vessels. Cold tends to relieve pain because of its contractile effect on vessels, thereby reducing the intrapulpal pressure.

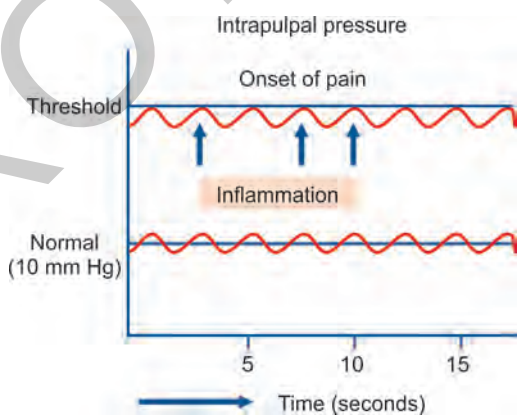


Fig. 3.10: Increased intrapulpal pressure causing pulpal pain.

  - *Electric test:* Less current is required in initial stages. As tissue becomes more necrotic, more current is required to generate the response.

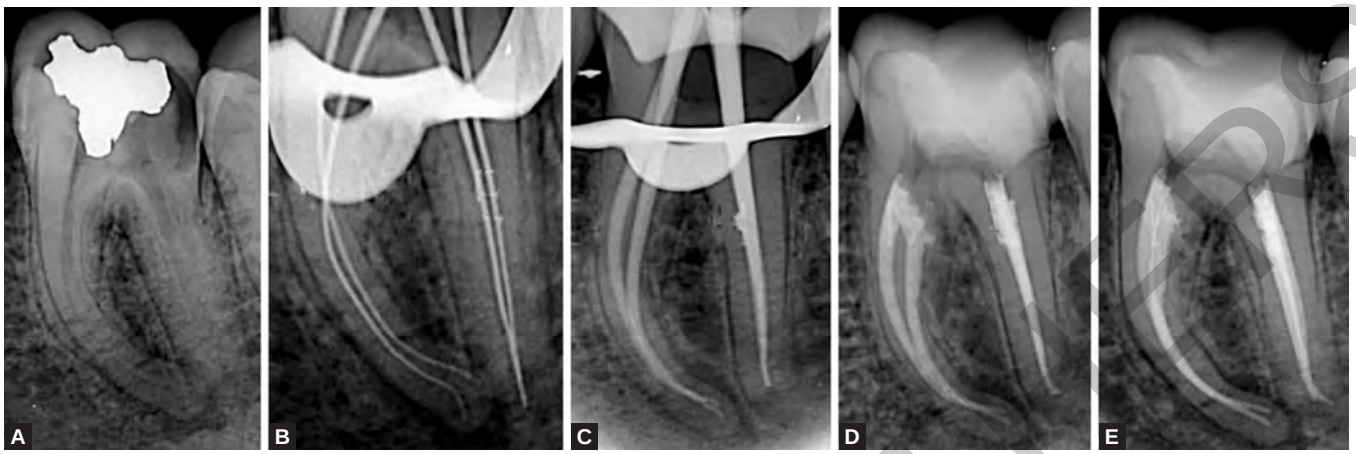
Treatment

Pulpectomy, i.e., root canal treatment **(Figs. 3.11 and 3.12)**.

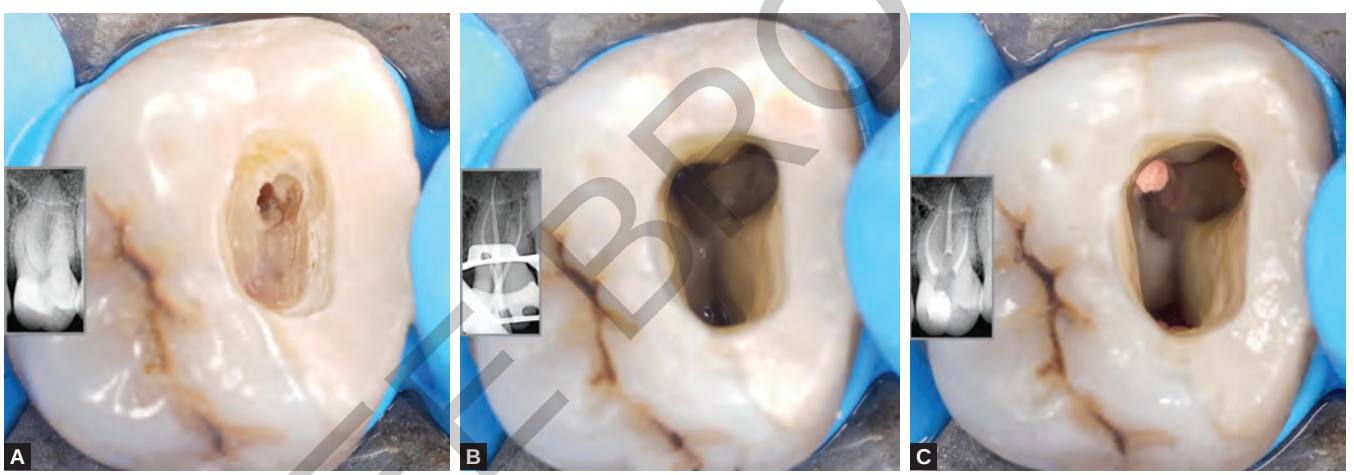
Points to Remember

Clinical guide given by Carrotte in 2003 to determine the status of dental pulp in irreversible pulpitis:

- 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 later stages, heat causes pain and cold relieves the pain.
- Radiating pain in initial stages but localizes later due to involvement of periodontal ligament.
- Tooth may become tender to percussion, once the inflammation spreads to periodontal ligament.
- Radiograph may reveal widening of periodontal ligament space.



Figs. 3.11A to E: (A) Preoperative radiograph showing fractured restoration with deep caries approximating pulp; (B) Working length radiograph; (C) Master cone radiograph; (D and E) Postobturation radiograph.
Courtesy: Ahmed Hameed.



Figs. 3.12A to C: (A) Preoperative radiograph and photograph showing access opening; (B) Master cone radiograph and photograph showing cleaned and shaped canals; (C) Postobturation radiograph and photographs.

CHRONIC HYPERPLASTIC PULPITIS/PULP POLYP/PULPAL HYPERPLASIA

Definition

Chronic hyperplastic pulpitis is a productive pulpal inflammation due to extensive carious exposure of a young pulp.

It shows development of granulomatous tissue into carious cavity resulting from long-standing low-grade infection **(Fig. 3.13)**.

Etiology

Hyperplastic form of chronic pulpitis is commonly seen in teeth of children and adolescents because in these pulp tissue has high resistance and large carious lesion permits free proliferation of hyperplastic tissue.

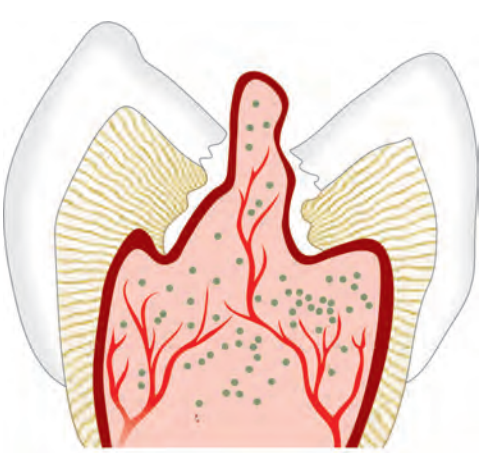


Fig. 3.13: Schematic representation of hyperplastic form of chronic pulpitis.

Differential diagnosis of reversible and irreversible pulpitis		
Features	***Reversible pulpitis***	***Irreversible pulpitis***
Pain type	Sharp and momentary pain which ceases after stimulus is removed	Intense, continuous, and prolonged pain due to pressure of secondary irritants
Stimulus	External stimulus, for example, heat, cold, and sugar	• Spontaneous in nature • Heat acts as stimulant • Dead or injured pulp tissue acts as secondary stimulant
Pain at night/postural	No	Yes
Pain localization	Only with applied cold stimulus or PDL inflammation	Only with applied heat stimulus or PDL inflammation
Referred pain	Usually not found	Common finding
History	History of recent dental procedure Sometimes cervical erosion/abrasion	History of: • Deep caries • Trauma • Extensive restoration
Percussion/occlusion	If due to occlusion, percussion test is positive, otherwise normal	If PDL is involved, percussion test is positive, otherwise normal
Pulp tests • EPT • Cold • Heat	 • Normal response • Exaggerated response • Normal—exaggerated response	 • Normal to elevated response • Pain relieved by cold occasionally • Acute pain
Color change	No	Yes
Radiograph	Caries, defective restoration without pulp protection	Caries, defective restorations, PDL space enlargement
Treatment	Removal of decay, repair of defect, sedative restoration, occlusal adjustment	Endodontic treatment

Signs and Symptoms

- It is usually asymptomatic.
- Fleshy pulpal tissue fills the pulp chamber. It is less sensitive than normal pulp but bleeds easily due to rich network of blood vessels.
- Sometimes this pulpal growth interferes with chewing.

Histopathology

- Tissue of pulp chamber is transferred into granulation tissue which projects out from pulp chamber.
- Granulation tissue contains PMNs, lymphocytes, and plasma cells.
- Surface of pulp polyp is usually covered by stratified squamous epithelium which may be derived from gingiva, desquamated epithelial cells of mucosa and tongue.
- Nerve fibers may be present in the epithelial layer.

Diagnosis

- **Pain:** It is usually absent.
- Hyperplastic form shows a fleshy, reddish pulpal mass which fills most of pulp chamber or cavity **(Fig. 3.14)**.

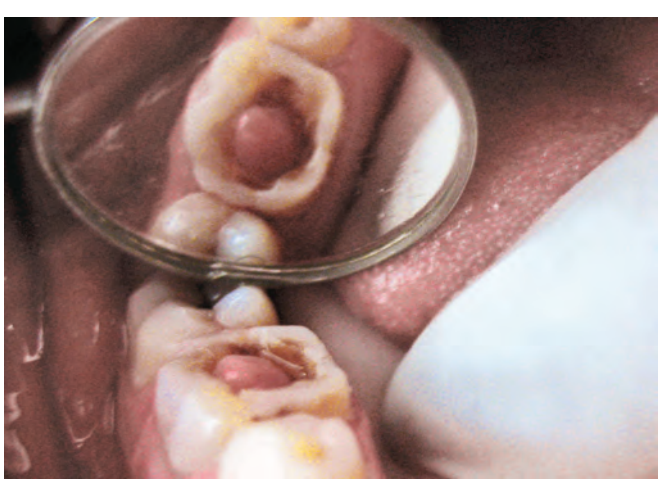


Fig. 3.14: Hyperplastic form of pulpitis showing fleshy reddish pulpal mass filling the pulp chamber.

It is less sensitive than normal pulp but bleeds easily when probed. When it is cut, it does not produce pain but pain can result due to pressure transmission to apical part.

- **Vitality tests:**
  - Tooth may respond feebly or not at all to thermal test, unless one uses extreme cold.

- More than normal current is required to elicit the 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, that is, pulp chamber.

Treatment

❖ In case of hyperplastic pulpitis, removal of polypoid tissue using periodontal curette or spoon excavator followed by root canal treatment.
❖ If tooth is at nonrestorable stage, it should be extracted.

INTERNAL RESORPTION/PINK TOOTH OF MUMMERY

Internal resorption was first reported by Bell in 1830. It is known as Pink tooth of Mummery because of pink discoloration of the crown and named after the anatomist Mummery.

Definition

Resorption is defined as condition associated with either physiologic or pathologic process resulting in loss of dentin, cementum or bone.

Internal resorption is an idiopathic slow or fast progressive resorption occurring in dentin of the pulp chamber or root canals of the teeth **(Fig. 3.15)**.

Etiology

Exact etiology is unknown. Patient may present history of trauma or persistent chronic pulpitis, or history of pulpotomy.

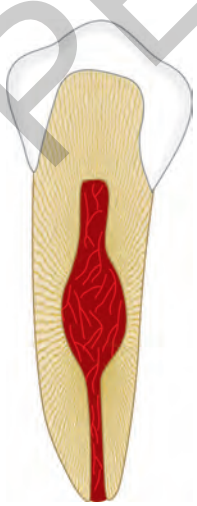


Fig. 3.15: Schematic representation of ballooning of pulpal cavity showing internal resorption of the tooth.

Mechanism of resorption
Pulp inflammation due to infection
↓
Alteration or loss of predentin 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 if resorption perforates the root **(Fig. 3.16)**.
❖ "Pink tooth" is the pathognomonic feature of internal root resorption.
❖ Pulp shows either partial or complete necrosis. In actively progressive lesion, pulp is partially vital and may show symptoms of pulpitis.
❖ In anterior teeth, it is typically seen in middle of the tooth in mesiodistal direction but in multirooted teeth, it can be present mesial, distal, or center.

Diagnosis

❖ **Clinically:** "Pink tooth" appearance.
❖ Radiographic changes **(Fig. 3.17)**:
- Classical description of internal resorption, that is, clearly well-defined radiolucency of uniform density which balloons out of root canal was given by Gartner el al.
- Original root canal outline distorted.

Fig. 3.16: Internal resorption of tooth causing perforation of root.

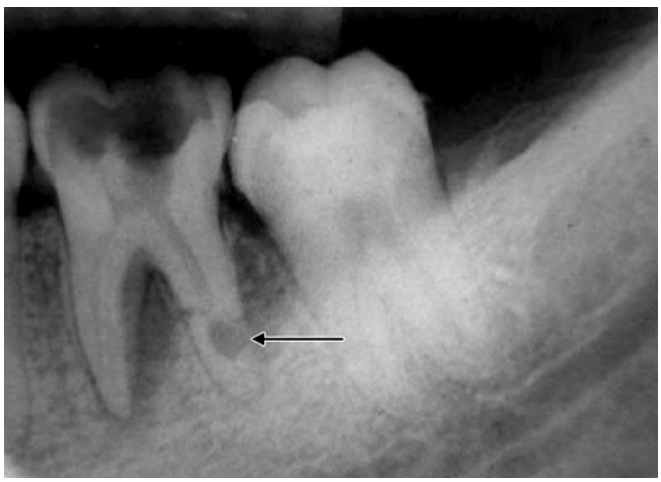


Fig. 3.17: Radiograph showing internal resorption in distal root of mandibular first molar.

- 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.

Differential Diagnosis

It is difficult to differentiate internal resorption from external resorption when it progresses to periodontal space causing root perforation.

It can be differentiated by:

❖ **History:** Giving history of trauma, pulpotomy, etc.
❖ Pink tooth appearance.
❖ Taking radiographs at different angles; radiolucency does not move when radiograph is taken at different angle, whereas in external resorption, radiolucent lesion changes position on changing angle.
- Uniform ballooning of root canal is seen in internal resorption, whereas irregular border with alteration of adjacent bone is seen in external resorption.

Treatment

❖ Pulp extirpation stops internal root resorption.
❖ Surgical treatment is indicated if conventional treatment fails.

PULP DEGENERATION

Pulp degeneration is generally present in old age. In young age, it may result from persistent mild irritation. Common causes of pulp degeneration are attrition, abrasion, erosion, operative procedures, caries, pulp capping, and reversible pulpitis. Forms of pulp degeneration:

Calcific Degeneration

In calcific degeneration, part of pulp tissue is replaced by calcific material. Mainly three types of calcifications are seen in pulp:

1. Dystrophic calcifications.
2. Diffuse calcifications.
3. Denticles/pulp stones.

Dystrophic Calcifications (Figs. 3.18 and 3.19)

❖ They occur by deposition of calcium salts in dead or degenerated tissue. 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.

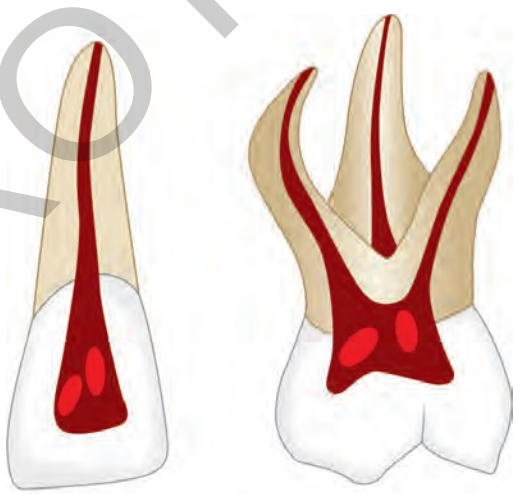


Fig. 3.18: Line diagram showing pulp stones present in pulp chamber of teeth.

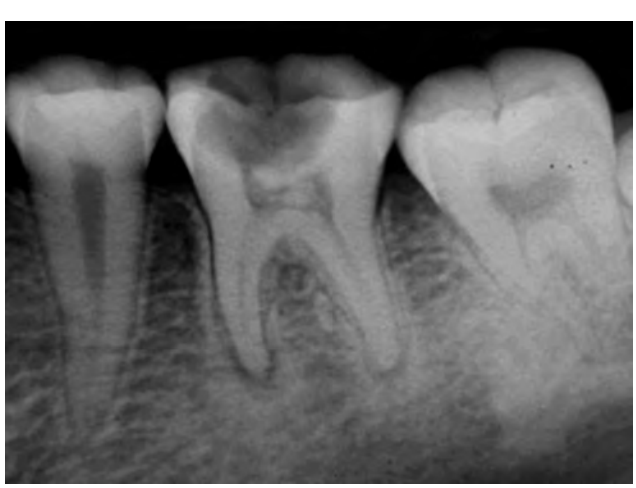


Fig. 3.19: Radiograph showing pulp stone in pulp chamber of mandibular first molar.

- They 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 Stones

These are usually seen in pulp chamber.

Classification of Pulp Stones

According to location (Fig. 3.20):

- Free.
- Embedded.
- Attached.

According to structure:

- True.
- False.

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

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

Atrophic Degeneration

- It is wasting away or decrease in size which occurs slowly as tooth grows old **(Fig. 3.21)**.
- Here fewer stellate cells are found and pulp is less sensitive than normal.

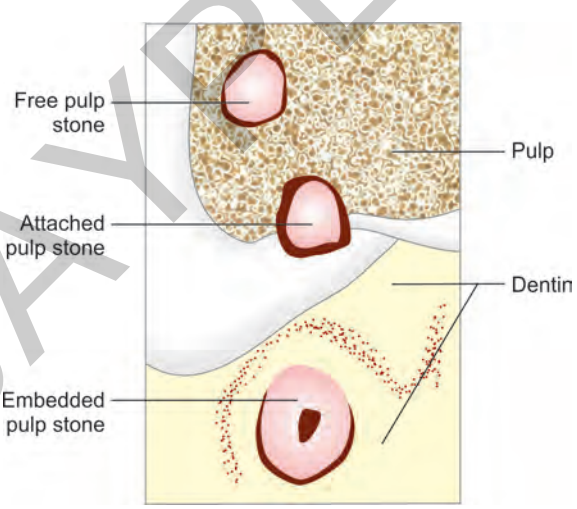


Fig. 3.20: Types of pulp stones according to location.

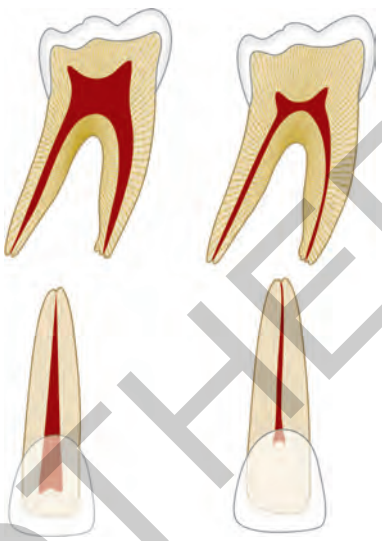


Fig. 3.21: Line diagram showing decrease in size of pulp cavity of anterior and posterior teeth due to secondary dentin deposition with age.

Fibrosis Degeneration

- There is gradual shift in ratio and quality of tissue elements. Here, the number of collagen fibers/unit area increases leading to fibrosis. Pulp has a characteristic leathery appearance **(Fig. 3.22)**.
- Number and size of cells decrease so cells appear as "shrunken solid particles in a sea of dense fibers".
- Fibroblastic processes are lost, cells have round and pyknotic nuclei.
- Dentinoblasts decrease in length, appear cuboidal or flattened.

Fig. 3.22: Vital and degenerated pulp extirpated from different teeth.

Textbook of Endodontics

Salient Features

- **Comprehensive coverage:** This textbook encompasses all essential topics in endodontics offering the latest techniques and updates crucial for undergraduates, postgraduates, and general practitioners.
- **Syllabus alignment:** Aligned with the prescribed syllabus by the Dental Council of India and other international universities, the text ensures comprehensive learning.
- **Rich visuals:** Enriched with clinical photographs, radiographic images, line diagrams, flowcharts, text boxes, and tables, enhancing the clarity and understanding of core concepts.
- **39 informative chapters:** The book includes 39 chapters that provide the latest clinical information, covering procedures from basic to intricate, catering to a wide spectrum of readers.
- **Examination ready:** Each chapter concludes with examiner's choice questions from various universities, facilitating effective last-minute preparation for examinations.
- **Updated content:** Stay ahead with the latest definitions and concepts including advancements in diagnostic methods, rotary instruments, materials techniques, bioceramics, and vital pulp therapy.
- **Universally applicable:** The content is designed to be useful for both undergraduate and postgraduate students, serving as a valuable resource for examination preparation.

In essence, this textbook is a comprehensive, visually enriched guide that aligns with academic requirements, offering the latest insights and aiding in effective examination preparation for dental students and practitioners.

Nisha Garg, a distinguished dental professional, earned her MDS from Government Dental College, Patiala, Punjab, India, and achieved excellence throughout her academic journey. Her consistent merit as a Gold Medalist during BDS earned her the title of the best graduate, securing the highest total marks in graduation. Currently serving as a Professor and PG Guide at Bhojia Dental College, Baddi, Himachal Pradesh, India, her expertise extends her tenure as an Ex-Resident at the esteemed Postgraduate Institute of Medical Education and Research (PGIMER) in Chandigarh, India. With a prolific record, she has published numerous national and international papers, contributing significantly to the dental field.

Amit Garg, holding an MDS from Government Dental College in Rohtak, Haryana, India, brings valuable experience as an Ex-Resident at Pandit Bhagwat Dayal Sharma Post Graduate Institute of Medical Sciences (PGIMS), Rohtak. His keen interest in research is reflected in numerous publications across various journals.

Dr Nisha and Dr Amit Garg manage a successful dental practice at "Perfect Teeth Dental Clinic and Facial Aesthetics" in Mohali, Punjab, India.

Together, they guide students for post-BDS competitive examinations (INBDE, AFK, ADC, etc.) at Dr Nisha Garg Dental Academy.

Their collaborative efforts also extend to authoring globally acknowledged and appreciated textbooks including the *Textbook of Operative Dentistry* and *Textbook of Preclinical Conservative Dentistry*, establishing them as influential figures in the dental education landscape.

Printed in India

Available at all medical bookstores or buy online at www.jaypeebrothers.com

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Medical Publishers (P) Ltd.
EMCA House, 23/23-B, Ansari Road,
Daryaganj, New Delhi - 110 002, INDIA
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ISBN 978-93-5696-464-8