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Short Textbook of ENDODONTICS



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Foreword
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JAYPEE

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CHAPTER 4

The Pulpal Reactions to Caries and Dental Procedures

This chapter explains how the reaction of pulp to various external stimuli is unique and describes in detail the reaction of pulp to dental caries and various dental procedures.

You must know

- How is the Response of Dental Pulp Unique and Different from Other Connective Tissues of the Body?
- Which are the Different External Stimuli that can Affect Dental Pulp?
- How does the Pulp React to Dental Caries?
- How does the Pulp React to Dental Procedures?
  - How does the Pulp React to some of the Diagnostic Procedures?
  - How does the Pulp React to Treatment Procedures?
- How does the Pulp React to Specific Dental Materials?

HOW IS THE RESPONSE OF DENTAL PULP UNIQUE AND DIFFERENT FROM OTHER CONNECTIVE TISSUES OF THE BODY?

Pulpal response to external stimuli is unique due to following reasons:

- Pulp is *encased* within the hard tissues, in an unyielding low-compliance environment
- Pulp has limited *space* to expand during inflammation
- Pulp has limited *portals of entry*
- Pulp is an organ of terminal and limited *circulation* with no efficient collateral circulation
- Pulp is more susceptible to *injury* and may have complicated regeneration due to scarcity of circulation
- Even the mature pulp resembles embryonic connective tissue. Therefore, it is relatively rich source of stem cells. It has the ability to form dentin throughout life
- Rich *neurovascular* supply within the pulp may promote the effect of inflammation and can lead to rapid degeneration and necrosis of pulp
- Various dental treatment procedures involve cleaning and shaping of enamel and dentin, causing further *irritation* of pulp
- Bacteria enter the pulp at a very late stage. Initially there is invasion of bacterial products and *toxins* rather than bacteria themselves
- Pulp organ lacks specific receptors for different external stimuli, the sensory response is always pain irrespective of the type of stimulus-heat, cold, pressure, touch, etc.

A mind-map to remember all unique features of dental pulp is given in Figure 4.1.

WHICH ARE THE DIFFERENT EXTERNAL STIMULI THAT CAN AFFECT THE DENTAL PULP?

Flow chart 4.1 gives the detailed list of external noxious stimuli that can affect the dental pulp causing its inflammation, necrosis and dystrophy beginning with the most frequent irritant: *Micro-organisms*. These have been discussed in detail in the next chapter under the possible causes of diseases of dental pulp.

HOW DOES THE PULP REACT TO DENTAL CARIES?

Dental caries is a polymicrobial disease affecting the dental pulp. Bacteria are mainly responsible for causing pulpal disease.

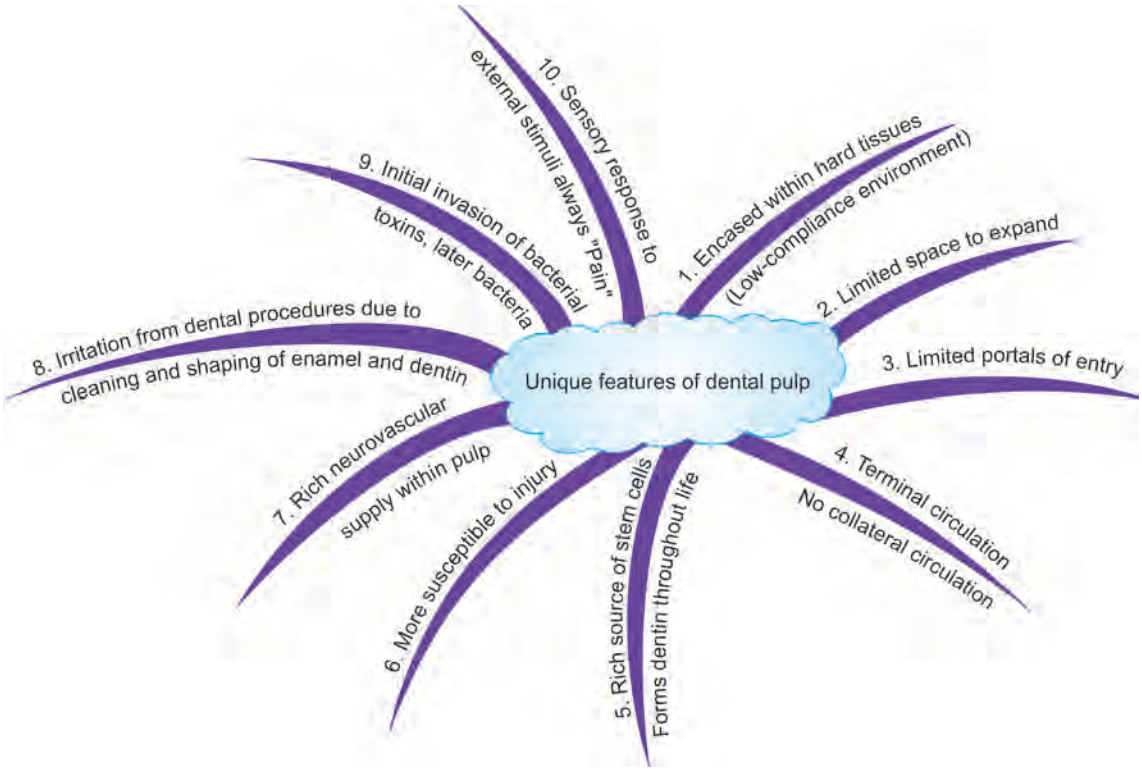


Fig. 4.1 Mind-map of unique features of dental pulp

Pathways of Bacterial Invasion of the Pulp

Figure 4.2 shows different pathways through which there is invasion of bacteria and toxins into the pulp.

Initially, there is invasion of bacterial toxins and invasion by bacteria themselves occurs at later stages of the carious process that clinically present as carious exposure. This is because toxins pass through enamel and dentin well ahead of bacteria themselves.

Bacterial byproducts and toxins include:

- Acids and proteinases that dissolve and digest enamel and dentin
- Lipopolysaccharides (LPS)
- Lipoteichoic acid (LTA).

Reaction of Pulp to Dental Caries

Pulp exhibits *inflammatory reaction* in response to bacteria and their byproducts and toxins.

- To protect *dental pulp* against caries, three basic *reactions* occur in the following sequence:
  - *Dentin sclerosis:* Decrease in dentin permeability
  - Formation of tertiary dentin
  - Inflammatory and immune reactions.

Dentin Sclerosis: Decrease in Dentin Permeability

First defense reaction to caries is dentin sclerosis.

This occurs by:

- Increased deposition of *intratubular dentin.* Transforming growth factor-beta 1 (TFG-beta 1) has been implicated to be mainly responsible for this reparative dentinogenesis.
- Direct deposition of precipitated *mineral crystals* into the narrowed dentinal tubules causing occlusion of tubules.

  As a result, there is effective *decrease in dentin permeability* occurring in a relatively short period of time.

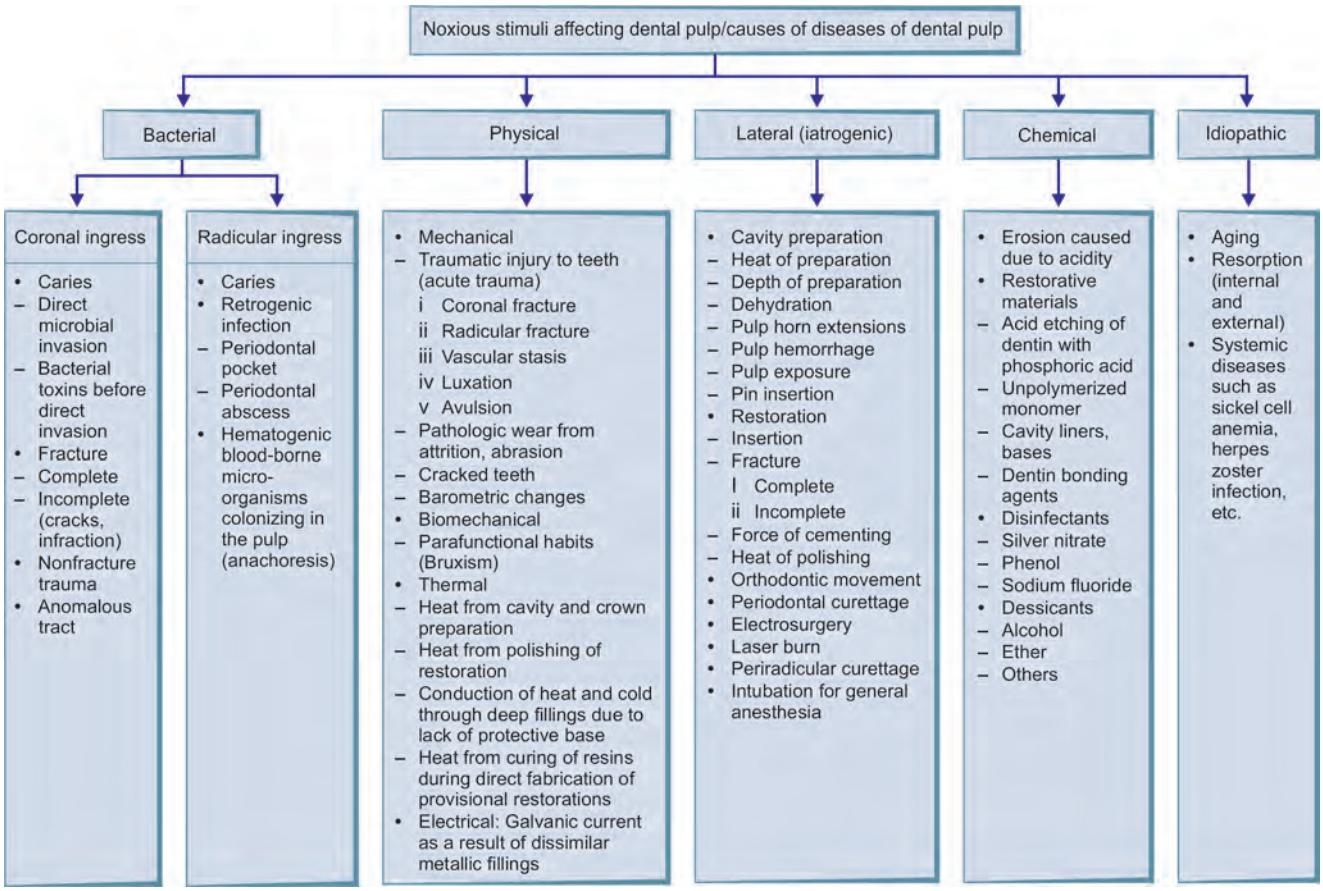
Formation of Tertiary Dentin

This occurs over a longer period of time as compared to sclerotic dentin.

Resultant character of tertiary dentin depends on the intensity of stimulus. Either *reactionary* or *reparative dentin* is formed (Fig. 4.3).

- In response to *mild stimulus,* resident quiescent odontoblasts are activated forming *reactionary dentin.* This mostly is seen in cases where dentin

Flow chart 4.1 Noxious stimuli affecting dental pulp/causes of diseases of dental pulp



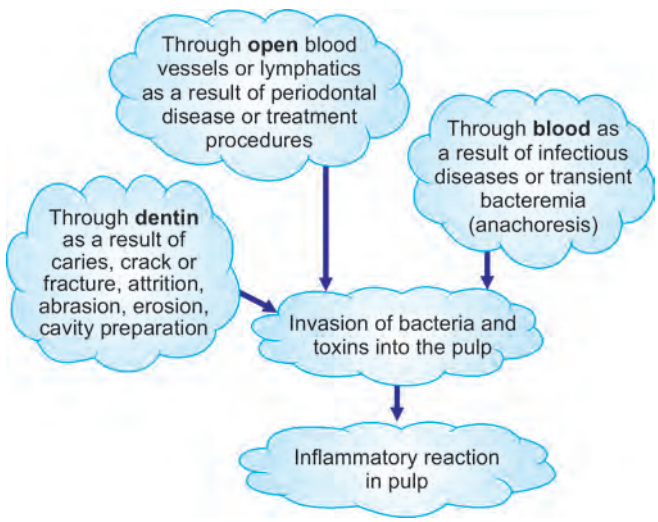


Fig. 4.2 Pathways of bacterial invasion into the pulp

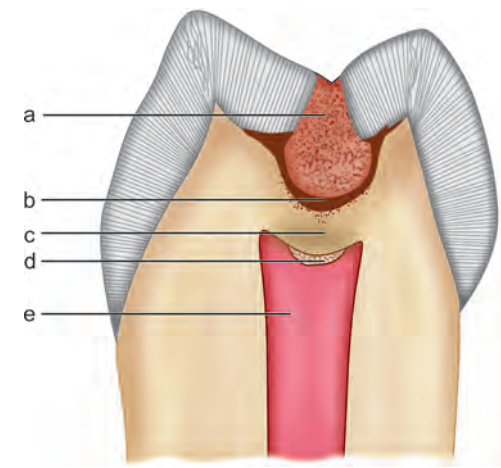


Fig. 4.3 Diagram showing effect of caries on pulp. a: Bacterial plaque; b: Outer carious dentin; c: Transparent inner carious dentin; d: Reparative dentin; e: Pulp

demineralization has occurred beneath the noncavitated enamel lesion.

- In response to *aggressive stimulus,* the subjacent odontoblasts die and there is disruption of odontoblast layer. As a result, there is differentiation of secretory cells to cause repopulation of the disrupted odontoblast layer either by
  - Organized tubular *reparative dentin* or
  - Disorganized irregular *fibrodentin.*

  Sclerotic and tertiary dentin formed provides a *physical barrier* to noxious stimuli.

Inflammatory and Immune Reactions

Inflammation

- *Acute inflammation:* Polymorphonuclear (PMN) leukocytes reach the area of bacterial involvement to prevent the further dissemination of bacteria deeper into the pulp.

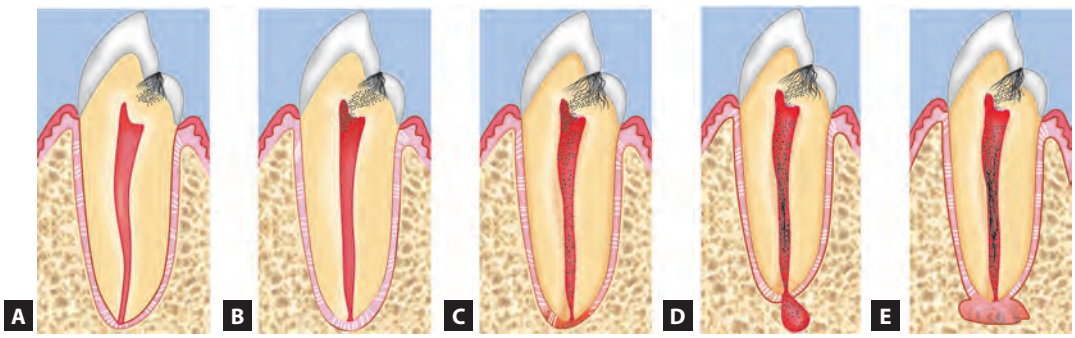
  In case of *severe* inflammatory process, symptoms of acute reaction are manifested.
  - There is accumulation of inflammatory exudates, causing *pain* from pressure on nerve endings.
  - As the PMN leukocytes die, there is formation of *pus* which further irritates the nerve cells.
- *Chronic inflammation:* In case of *less severe* inflammatory process, PMN leukocytes are replaced by lymphocytes and plasma cells. The inflammatory reaction is confined to the surface of the pulp.
  - Microorganisms may penetrate deeper causing an *acute exacerbation* manifested by clinical flare-up.
  - Microorganisms may cause *reaction* in the *periapical* tissue by means of their metabolic products.

  Chronic inflammation due to deep dental caries involving the pulp appears radiolucent on intraoral periapical radiograph (IOPA) as seen in Figure 4.4.
- *Chronic hyperplastic pulpitis:* In case of primary and immature permanent teeth, pulp exposure can result in a proliferative pulpal response called chronic hyperplastic pulpitis characterized by proliferation of exuberant inflammatory pulp tissue through the exposure forming *pulp polyp*.
- *Pulp necrosis:* In case of persistent inflammation, tissue pressure is increased, *stasis* occurs resulting in *pulp necrosis*. Whether partial or total necrosis of pulp occurs is determined by possibility of drainage. In case of pulp open to oral fluids, drainage is possible. So, partial necrosis is likely to occur. Apical pulp tissue remains uninflammed. But if drainage is not possible, total necrosis of pulp may occur.

  Figure 4.5 shows diagrammatic representation of sequel of dental caries, how it affects the dental pulp and the periradicular area demonstrating caries in enamel and dentin becomes deeper and causes inflammation in



Fig. 4.4 Radiograph showing mandibular first molar with extensive caries involving the pulp (*Courtesy* of Dr Chetan Shah)



Figs 4.5A to E Diagrammatic representation of sequel of dental caries: (A) Caries in enamel and dentin; (B) Deep caries causing pulpitis; (C) Widening of the periodontal ligament space; (D) Periapical granuloma or abscess; (E) Necrotic pulp and periapical abscess

the pulp. There is widening of the PDL space. Persistent inflammation causes necrosis of the pulp. Less virulent microorganisms may result in formation of chronic abscess. Low grade irritation can result in the formation of periapical granuloma or cyst.

Immune Reactions

- Humoral and cellular pulpal immune response occurs to invading microorganisms.
- Immunoglobulins such as IgG, IgM and IgA have been found within the odontoblasts of carious dentin.
- The three antigen-presenting cell types of pulp include:
  - Macrophages
  - Dendritic cells
  - B-lymphocytes.

  Macrophages express type II major histocompatibility complex (MHC) molecules on stimulation of bacteria or cytokines.

  Dendritic cells form network around blood vessels within the pulp and the odontoblast layer and constantly express MHC molecules without provocation.

  B-lymphocytes secrete antibodies during specific immune response and express MHC molecules.

  Initially, antibodies accumulate in the odontoblast layer. But as the lesion progresses, they may be found in dentinal tubules.
- The first cells to encounter an antigen diffusing along the dentinal tubules are the odontoblasts. The odontoblasts are stationary and do not directly participate in the activation of T-cells, but may have a role in activating dendritic cells.
- *Calcification and internal resorption:*

  Formation of pulp stones and calcific metamorphosis has been found to occur in response to caries in chronic long standing cases.

  Calcific metamorphosis
  - A pulpal response to traumatic injury characterized by deposition of hard tissue within the root canal space is called calcific metamorphosis.
  - Calcific metamorphosis commonly occurs in the anterior teeth that get affected by trauma.
  - A tooth with calcific metamorphosis appears darker in hue than the adjacent teeth and has dark yellow color due to greater thickness of dentin.
  - There is reduction in size of both the coronal and radicular pulp spaces, sometimes resulting in partial or total obliteration of pulp canal when seen on radiograph. PDL space is normal and lamina dura is intact.
  - Calcific metamorphosis progresses from coronal part of the tooth to the apical canal.

  An alternative sequela to trauma may be idiopathic internal resorption.
- *Repair and regeneration:* When inflammatory and immune reactions are effective, the foreign material may be neutralized and removed, initiating repair and regeneration.
- There is formation of *reactionary* (tertiary) *dentin*, when no odontoblasts are killed.
- There is formation of *reparative* (tertiary) *dentin*, when odontoblasts are killed.

  However, normally organized pulp may not reform.

  Pulp polyp is considered as a chronic inflammation in which injury and repair are going on at the same time. Pulp polyp contains epithelial cells from oral mucosa, large lymphocytes and also fibrous tissue.

  Figure 4.6 shows a photograph of pulp polyp in a primary second molar.

  Lymphocytes represent cells of inflammation.

  Fibrous tissue represents an attempt to 'wall off' the diseased tissue, i.e. *repair*.

  A mind-map to remember all points of reaction of pulp to dental caries (Fig. 4.7).

HOW DOES THE PULP REACT TO DENTAL PROCEDURES?

Flow chart 4.2 shows different diagnostic and dental treatment procedures.



Fig. 4.6 Photograph showing pulp polyp in a primary second molar (*Courtesy* of Dr CR Suvarna)

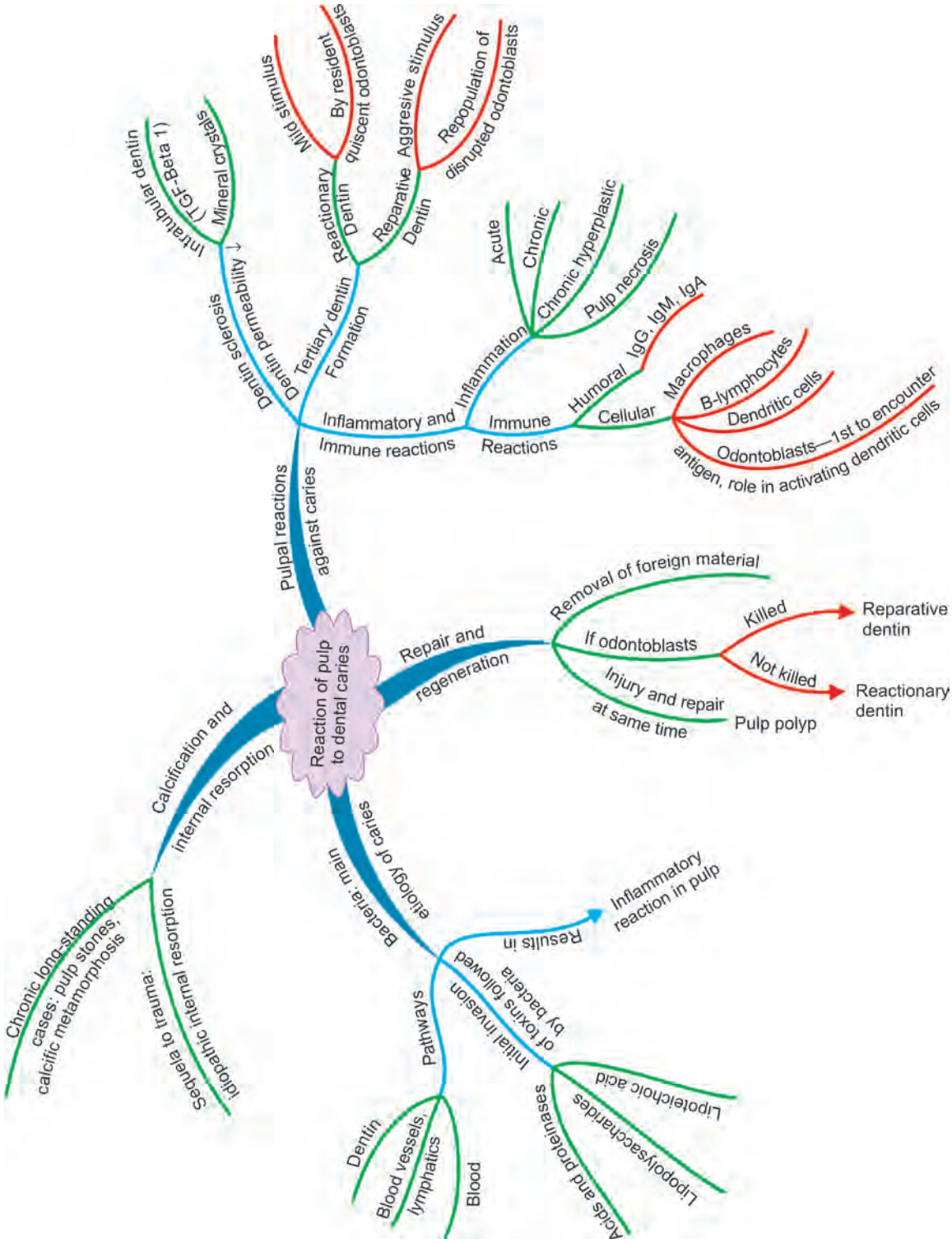
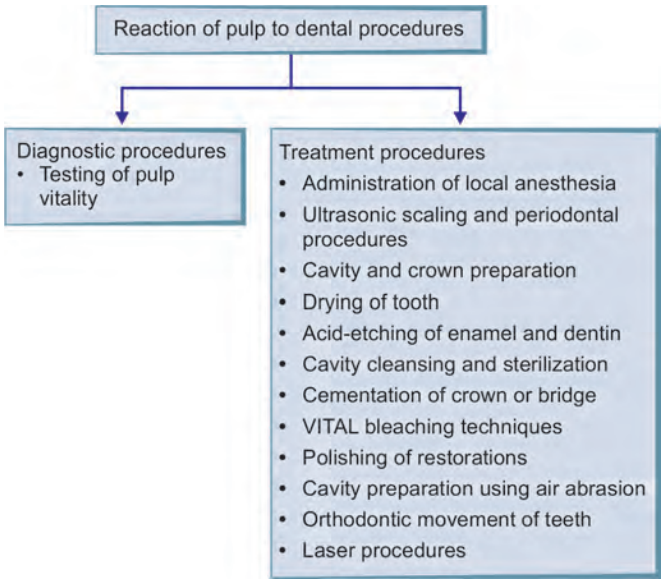


Fig. 4.7 Mind-map of reaction of pulp to dental caries

Flow chart 4.2 Different diagnostic and dental treatment procedures



How does the Pulp React to Some of the Diagnostic Procedures?

Pulp Vitality Testing

Thermal tests: Both heat and cold tests if performed within normal clinical parameters do not cause any damage to dental pulp.

Heat Test

Heated gutta-percha or tip of heat-testing device such as system B reaches a temperature of 200°C, just before the smoke point and is applied to the tooth surface for 5–10 seconds.

The maximum temperature at the pulp-dentin junction (PDJ) was recorded using a thermistor and found to be about 39.9°C for 10 second application of heat to tooth. This temperature does not reach the threshold at which damage to the tissue can occur.

Thus, within normal clinical parameters (temperature and time) heat test is relatively safe and does not cause any pulpal damage.

Cold Test

Carbon dioxide snow has an inherent temperature of –78°C that may be applied to tooth surface for 5 seconds to 5 minutes.

The maximum temperature at the pulp-dentin junction (PDJ) was recorded using a thermistor and found to be about 9.5°C for 5-minute application. When carbon dioxide snow was applied to the tooth for 20 minutes, the pulp suffered damage and there was necrosis of odontoblasts.

Thus, within normal clinical parameters (temperature and time) cold test is relatively safe and does not cause any pulpal damage.

It has been found that value of 10°C above body temperature is considered as threshold to cause irreversible damage to vital tissues. Higher temperatures and greater time of application leads to greater damage.

How does the Pulp React to Dental Treatment Procedures?

How does the Pulp React to Local Anesthetics?

- Vasoconstrictors, such as epinephrine, which are added to local anesthetics to enhance its duration of action, cause reduction in pulpal blood flow (Fig. 4.8).
- Reduction in pulpal blood flow can have a negative effect on the health of dental pulp as it reduces the clearance of large molecular weight toxins or waste products that may result in irreversible pulpal pathosis. This effect is more likely particularly if the pulp is inflamed preoperatively.
- During cavity preparation in an anesthetized tooth, the response of pulp is suboptimal. If the pulp is already severely inflamed, local anesthesia compromises its ability to recover from inflammation.
- Supplemental injections (such as intraosseous, intra-ligamentary and intrapulpal) cause more severe reduction or even transient cessation of pulpal blood flow. This has been concluded from animal studies.
- During Endodontic access preparation, if in spite of nerve block and repeated supplemental injections, if pain persists, then intrapulpal injection is indicated. It is achieved by injecting the anesthetic into pulp tissue under pressure. The pharmacologic action of anesthetic solution occurs on nerve cell membrane. Mechanical pressure of injection causes circulatory interference. If anesthetic solution is deposited passively into the pulp chamber, the solution will not diffuse throughout the pulp and hence is ineffective.

How does the Pulp React to Ultrasonic Scaling and Periodontal Procedures?

Heat is generated during ultrasonic scaling which has the potential to cause pulpal damage if adequate cooling with water coolant is not used (Fig. 4.9).

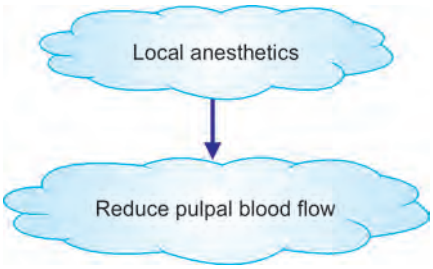


Fig. 4.8 Reaction of pulp to local anesthetics

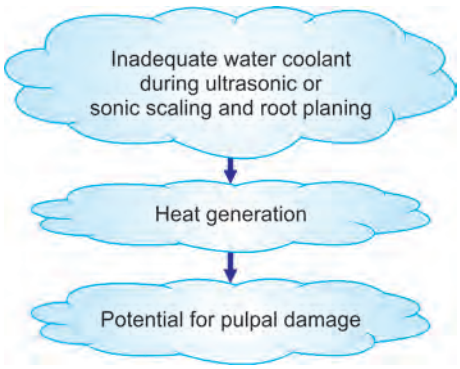


Fig. 4.9 Reaction of pulp to inadequate water during periodontal procedures

There is greater amount of heat generated with heavy load of force exerted by the ultrasonic tip against the tooth.

Thus, proper water cooling of ultrasonic scalers is a must to control the excessive heat production and prevent any damage to pulp.

Also, periodontal scaling and root planing may result in removal of thin cementum that exposes the dentin to the oral cavity resulting in dentin hypersensitivity.

Thus, periodontal *disease* that causes attachment loss and exposes root surface to oral cavity as well as periodontal *treatment* such as scaling and root planning can be associated with pulpal pathosis particularly if large lateral or accessory canals are exposed (Fig. 4.10).

How does the Pulp React to Cavity and Crown Preparation?

The physical irritation of the pulp from heat, desiccation or vibration while performing restorative procedures may affect the pulp adversely.

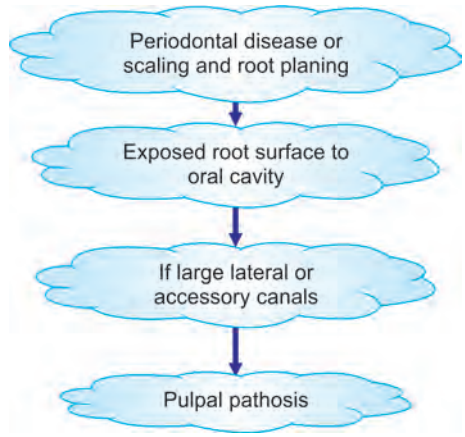


Fig. 4.10 Reaction of pulp to ultrasonic scaling of some teeth

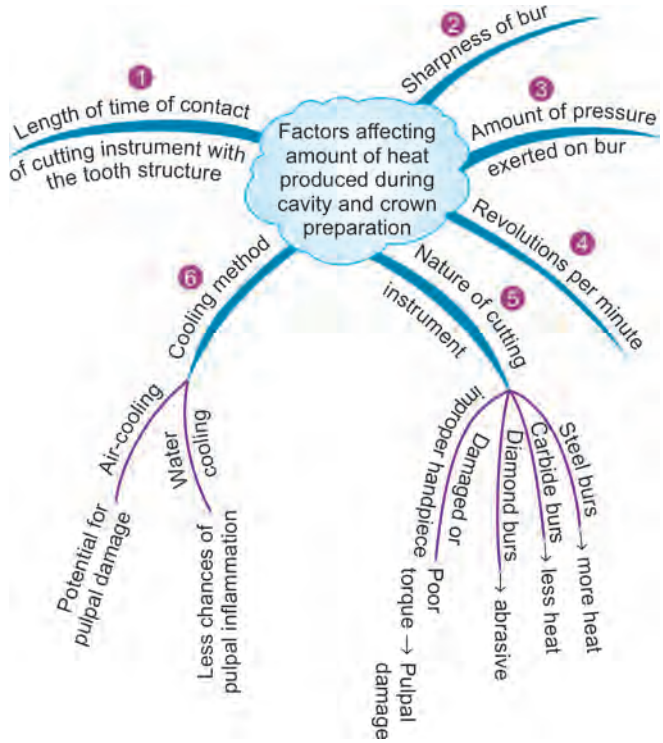


Fig. 4.11 Factors affecting amount of heat produced during cavity and crown preparation

Factors Affecting Amount of Heat Produced during Cavity and Crown Preparation (Fig. 4.11)

i. Length of time of contact of cutting instrument with the tooth structure, if more, then can cause lots of heat production. Intermittent cutting is recommended.

ii. *Sharpness of bur:* Old blunt burs may require application of lots of pressure for cutting.
iii. *Amount of pressure exerted on bur:* Light pressure is recommended. More pressure can cause more heat production.
iv. Revolutions per minute.
v. *Nature of cutting instrument:* Steel burs produce more heat as compared to carbide burs causing more thermal damage to the pulp. Diamond burs cause abrasive action and pressure may need to be exerted. Damaged or improper handpiece with poor torque characteristics can cause pulpal damage due to eccentric bur rotation.
vi. *Cooling method:* When only air is used as coolant, more potential for pulpal damage. When water is used as coolant, less chances of pulpal inflammation.

If only air is used as coolant at 2,00,000 rpm, it has been found that it takes just 11 seconds of continuous preparation at this speed to burn the pulp.

The density of dentinal tubules at DEJ is 65,000/mm^2 and the density of dentinal tubules at the pulp is 15,000/mm^2.

As the remaining dentinal thickness (RDT) decreases, the response of pulp to restorative procedures increases.

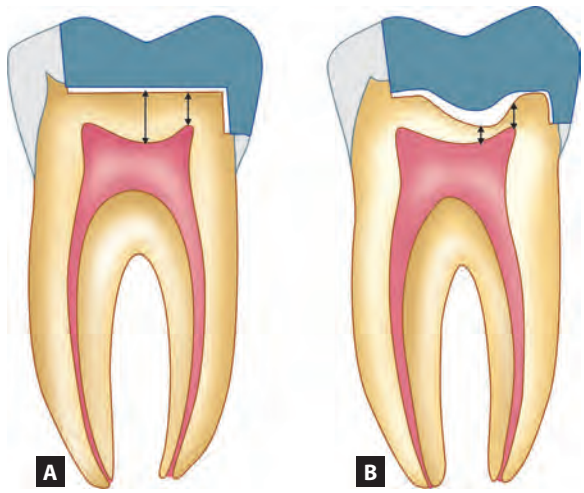
Remaining Dentinal Thickness

- It is the thickness of dentin present between the floor of cavity preparation and the roof of the pulp chamber.
- It is an important factor in determining the pulpal response to various noxious stimuli.
- Dentin thickness of about 2 mm between the floor of cavity preparation and the pulp is considered adequate to serve as an insulating barrier against irritants.
- Permeability of dentin increases with lesser remaining dentinal thickness (RDT). It has been found that when RDT is about 0.75 mm, effects of bacterial invasion are seen and when RDT is about 0.25 mm, odontoblastic cell death is seen

Figures 4.12A and B show diagram of cavity preparation is madibular molar with more RDT in (A), less possibility of damage to pulp and lesser RDT in (B) where there will be increased pulpal response.

Thus, RDT of less than 0.5 mm, can cause severe pulpal reaction and more chance of pulp undergoing irreversible pulpal pathosis. Dentin is a good insulator, damage to pulp does not occur unless RDT is less than 1 mm.

In case of large and wide cavity preparation and extensive preparation for crown, more dentinal tubules are exposed to external microbial or chemical irritation. If adequate water coolants are not used during such preparations, there can be marked reduction in pulpal blood flow.



Figs 4.12A and B (A) More RDT; (B) Lesser RDT
Abbreviation: RDT, remaining dential thickness

The diameter of dentinal tubules at DEJ is 0.6–0.8 μm and the diameter of dentinal tubules at pulp is 3 μm. Bacterial cells have diameter of 0.5–1 μm and can migrate through remaining dentin into pulp.

To minimize the liberation of heat during preparation:

- Use ultrahigh speeds of rotation for Enamel and superficial dentin.
- Use efficient water cooling system.
- Use light pressure. When using high speed, instrumentation pressure should not be more than four ounce and when using low speed, instrumentation pressure should not be more than twelve ounce.
- Do intermittent cutting.
  Remember that the bur-dentin interface must always be *wet.*
- Finish the crown/cavity preparation using low-speed cutting and hand instruments.

How does the Pulp React to Drying of Tooth?

Rapid, prolonged blast of air causes rapid outward flow of dentinal fluid into patent dentinal tubules which stimulates the nociceptors in dental pulp producing pain (Fig. 4.13). Also, odontoblasts are aspirated into the tubules. These displaced cells undergo autolysis and disappear within a short-time. They are replaced by new odontoblasts from stem cells deeper in the pulp which are capable of producing tertiary dentin.

Some harsh chemicals such as lipid solvents-like acetone or ether have been used in the past to clean cavity floors. But they were found to produce strong hydrodynamic forces in dentinal tubules causing odontoblast displacement.

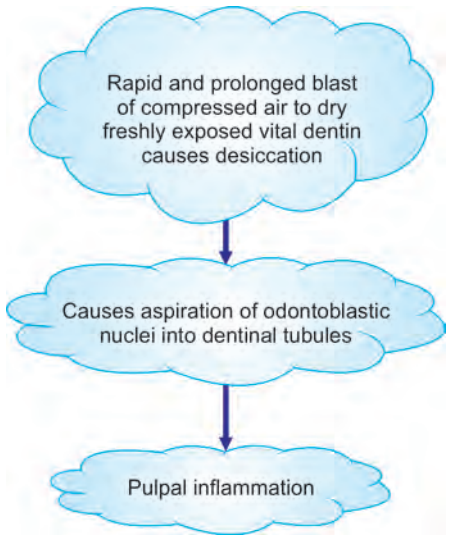


Fig. 4.13 Reaction of pulp to drying of tooth

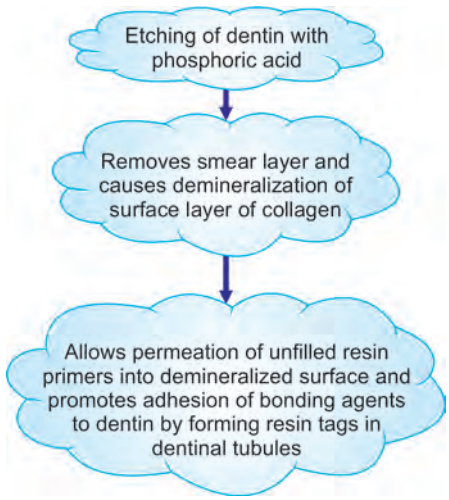


Fig. 4.14 Reaction of pulp to acid-etching of dentin

It is recommended that cavity floors be dried with cotton pellets and short blasts of air rather than harsh chemicals. In deep cavities, air blast can cause lots of discomfort to the patient. Cotton pellets should be used.

Also, the tooth must be kept moist during preparation and while drying the cavity preparation, care must be taken just to remove extra moisture from the operative field and not remove dentin's natural moisture (desiccation).

How does the Pulp React to Cavity Cleansing and Sterilization?

Certain caustic chemicals such as hydrogen peroxide, sodium hypochlorite, calcium hydroxide, etc. were used in the past for cavity disinfection to get rid of residual microbial contamination of the cavity preparation. But these chemicals may be potentially toxic to the pulp and so they are not used these days.

When dentin is exposed, there is outward flow of dentinal fluid, so the inward flow of any noxious agents is minimized. As a result, the irritation from residual microbial contamination in dentinal tubules is reduced.

How Does the Pulp React to Acid Etching of Enamel and Dentin?

When dentin is cut, smear layer is produced containing fragments of microscopic mineral crystals and organic matrix. Smear layer blocks the orifices of dentinal tubules and reduces the permeability of dentin. Etching of dentin with phosphoric acid removes the smear layer and causes demineralization of surface layer of collagen (Fig. 4.14).

If the open dentinal tubules are left unsealed, the irritants can diffuse into the pulp and may intensify and prolong the severity of pulpal reactions.

In superficial cavities, diameter of dentinal tubules is narrow and density of peripheral dentin is low, so etching of dentin followed by adequate sealing with restorative resin will not cause any detrimental effects on pulp.

In case of deep cavities or presence of exposure, phosphoric acid etching can be detrimental.

Also, in cases where phosphoric acid etching is done and bacteria are also present, severe pulpal inflammation and necrosis tends to occur.

Due to such effects with total etch systems, self-etching systems have become popular these days which eliminate etching with phosphoric acid. They do not remove the smear layer but this smear layer is incorporated within the restoration. But the only drawback of self-etch system is that it may form a relatively poor bond due to weaker acidity of acidic primers of self-etch system than total etch system.

How does the Pulp React to Cementation of Crown or Bridge?

When cementation of crowns, inlays or bridges is done on vital teeth, *hydraulic* forces may be exerted on the pulp as cement compresses the fluid in dentinal tubules.

- Gentle and careful cementation must be done in case of vital teeth
- Vents are provided in the casting that allow the cement to escape and facilitate seating.

How does the Pulp React to Vital Bleaching Techniques?

Strong oxidizing agents such as 10% carbamide peroxide and hydrogen peroxide are used to bleach enamel of vital teeth. These chemicals are in contact with teeth for long duration, i.e. overnight and they may cause mild pulpitis that usually gets reversed within 2 weeks. This occurs particularly if dentin with open tubules or cracks is present.

The symptoms usually are reversible and can be prevented by treating the teeth with fluorides and by doing the necessary restorative treatment preoperatively.

How does the Pulp React to Polishing of Restorations?

- When glass ionomer and composite restorations are polished, no significant increase in temperature at the pulp-dentin interface has been found.
- But polishing of amalgam restorations, especially with high speed, high contact pressure and no coolant can cause increased temperatures that may be damaging.

If water coolant is not to be used, it is recommended that speed should not exceed 4000 rpm.

It is safer to use a coolant, apply light pressure and intermittent contact during polishing of amalgam restoration to prevent any likely pulpal damage.

How does the Pulp React to Cavity Preparation using Air Abrasion?

Air abrasion is a conservative, nonpainful cavity preparation technique that forces aluminum oxide particles in a rapid stream onto tooth structure and is simultaneously evacuated from the field.

If higher pressure and small particle size is used, there are fewer pulpal effects as compared to high-speed burs.

Air-abrasion technique can be used in case of shallow caries and in pediatric patients.

How does the Pulp React to Orthodontic Tooth Movement?

Orthodontic forces cause changes in pulpal blood flow not only of teeth undergoing active movement but also of adjacent teeth (Fig. 4.15).

Thus, the pulpal response to orthodontic forces is hemodynamic.

Heavy forces used to reposition malaligned teeth especially impacted canines may lead to pulp necrosis or calcific metamorphosis.

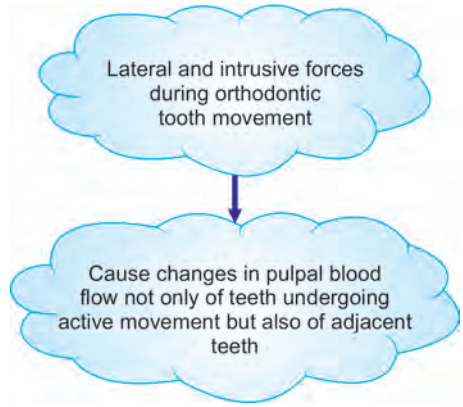


Fig. 4.15 Reaction of pulp to orthodontic tooth movement

Some of the methods used for removal of orthodontic brackets at the completion of treatment have been found to cause injury to pulp.

How does the Pulp React to Laser Procedures?

- Commonly used lasers in dentistry include Er:YAG, Nd:YAG and CO_2 lasers
- Er:YAG laser is used for removal of caries and cutting dentin. Lower energy delivered through low power settings with Er:YAG laser is found to produce favorable results
- CO_2 laser can be used for pulpotomy procedure in primary teeth, pulpotomy procedures in permanent teeth with immature apex and in exposed pulps due to fracture
- Nd:YAG laser can be used for pulp and in surgery
- The pulpal responses to Nd:YAG and CO_2 lasers are not found to be favorable. They may cause charring and significant inflammation in pulp compared to Er:YAG laser.

How does the Pulp React to Specific Dental Materials?

The effects of dental materials on pulp may be related to the permeability of associated dentin which in turn may be affected by the thickness of dentin remaining (RDT) between the cavity preparation and the pulp.

Zinc Oxide Eugenol

Zinc oxide eugenol (ZOE) has anesthetic and antiseptic properties. When ZOE is applied to deep cavities, it blocks transmission of action potentials in nerve fibers and

suppresses nerve excitability in the pulp. It adapts well to dentin and inhibits bacterial growth on cavity walls.

Zinc oxide eugenol is suitable as temporary filling but not long-term restoration as it has been found to leak over a period of time.

Zinc Phosphate Cement

The phosphoric acid liquid phase may be harmful to the pulp. But studies have shown that it is relatively safe. Zinc phosphate cement has been used as base beneath amalgam restorations for years.

Zinc phosphate is also well-tolerated by pulp when used as a luting agent.

Glass Ionomer Cement

When glass ionomer cement is used as liner in deep cavities with RDT of 0.5–0.25 μm, it results in deposition of tertiary dentin but at a slower rate than calcium hydroxide.

When glass ionomer cement is placed over exposed healthy pulp, there is severe pulpal inflammation or necrosis similar to that for calcium hydroxide.

When glass ionomer cement is used as luting agent in vital teeth, for sometime after luting, there is postcementation sensitivity which subsides over a period of time.

Silver Amalgam

Amalgam cannot be placed directly over the cavity preparation, it has to be placed over a liner/base such as zinc phosphate cement to protect the pulp.

During setting, amalgam shrinks, which results in microleakage. But over a period of time, marginal seal improves as the corrosion products accumulate between restoration and the cavity walls.

Restorative Resins

Placement of resin restoration is technique sensitive. Improper technique can result in faulty bond to tooth structure causing dentin hypersensitivity, recurrent disease and pulpal inflammation or necrosis.

Etching with phosphoric acid dissolves the highly mineralized peritubular dentin leaving free collagen fibrils and opening dentinal tubules. The resin in the form of bonding agent has to infiltrate the exposed collagen mesh and seal the open dentinal tubules. If it does not do so then there is nanoleakage.

Pulpal irritation from resin placement can be due to irritants such as unpolymerized monomer and polymerization shrinkage. The components of unpolymerized monomer may leach directly into pulp in case of deep cavities and cause chemical irritation. Polymerization shrinkage of composites induce internal stresses on dentin and create voids causing microleakage. This can be minimized by incremental placement and curing of composites and starting the restoration with flowable resins.

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