UNIT 3 BIOLOGICAL RESPONSE OF PULP TO RESTORATIVE MATERIALS AND PROCEDURES

Structure

3.0 OBJECTIVES

After reading this unit, you should be able to:

- describe the physiology of pulp and dentin as related to response to external stimuli;
- define the terms used in the context of safety of dental materials;
- enumerate the factors influencing pulpal response to restorative materials;
- discuss the stages of pulpal inflammation; and
- describe pulp reaction with various restorative materials and other external stimuli.

3.1 INTRODUCTION

By now you have read about the development of tooth and physiology of pulp. That means you are aware of inside of tooth and its normal constituents. Let us go further...

Pulp, a specialized connective tissue, is very sensitive to external stimuli. In addition to the dental procedures that threaten the integrity of the pulp, injury may also result from irritation by a noxious agent brought into contact with the exposed dentin or the pulpal tissue. Any stimulus over the exposed dentinal tubules affects the pulp. The reaction of the pulp, most of the times, is physiologic; however, depending upon the intensity of the stimulus pathological changes do occur in pulp.

It is biologically evident that the contact of a dental material with cytoplasmic cell process in dentin can induce odontoblastic alterations and transient inflammatory reactions. Recently, it is established that leakage of bacteria and their products from the oral environment along the gaps in the cavity/restoration interface are more toxic than the material itself.

In this unit you will study the reaction of the pulp to the external stimuli in the form of caries ,restorative procedures .You will study the comparative toxicity of all materials which will help you to choose the materials that are kind to the pulp. It is also important for you to know the different measures undertaken to reduce the toxic effect of the materials. Further the effect of recently introduced cutting tools and other techniques utilized in restorative dentistry, on the pulp will also be evaluated to guide you to use the same cautiously.

3.2 COMMONLY USED TERMS

Some commonly used terms are explained below:

i) **Bio-compatibility**

The term ‘bio-compatible’ is defined as being harmonious with life and not having toxic or injurious effects on biological functions. The dental materials or technique or any external stimulus should not be harmful to the pulp and soft tissues. It should not contain toxic diffusible substance that can be absorbed into the circulation to cause a systemic toxic response. Further it should have no carcinogenic potential and also should be free of potentially sensitizing agents that could cause an allergic response.

ii) **Bio-material**

It can be defined as any substance, other than a drug, that can be used for any period as a part of a system that treats, augments or replaces any tissue, organ or function of a body. The host environment for dental bio-materials is especially complicated because of the presence of bacteria and debris in the oral cavity and the corrosive properties of saliva and other fluids.

iii) **Hazardous**

A material is considered to be hazardous if it has the potential to cause a problem when placed in the body tissues. Hazardous materials are generally identified through screening tests, which place them in direct contact with either cultured cells or animal tissues.

iv) **Risk**

Risk means that the material can cause sufficient damage. A material which is hazardous may not pose a significant risk to the body e.g. Zinc oxide eugenol may be hazardous but not risky. Risk may be reduced because of diffusion or dilution of the offending components, biological barriers, or a lack of sufficient time of contact between the offending components and susceptible tissues.

3.3 PULP DENTIN ORGAN

The dentin and the pulp must be considered as one organ (the pulp-dentin complex) because of the intimate relationship between the cellular tissue within the dentin and the peripheral pulp tissue. The embryonic dental papilla is responsible for the formation of this coupled tissue. Hence it is obvious that the response of the pulp to any restorative material will be influenced by its surrounding dentin also. The dentinal tubules occupy 20-39% of dentin, and the dentinal fluid within them represents about 22% of the total volume of dentin. Dentinal fluid in the tubules, which is continuous with the extra-cellular fluid of the pulp, serves as a medium for relaying injurious agents to the pulp to reduce an inflammatory response. Thus anything that contacts the living dentin can be carried into the pulp. Also, either positive hydrostatic pressure or negative osmotic pressure may move the fluid in the dentinal tubules, which may displace the odontoblastic process or nerve endings resulting in pain.

3.3.1 Physiology

As described earlier, the tissues of pulp and dentin are considered as one. The pulp is vascularized as are other organs of the body. A large arteriole passes through the root pulp to supply the coronal pulp. They branch as capillaries in the coronal sub-odontoblastic region. The blood capillaries have discontinuity in the endothelial walls. Such types of arrangements facilitate easy exchange of nutrients and the waste fluid. This exchange is important in case of pulp injury. The fluid flow from the pulp to exposed dentin is dependent on the hydraulic conductance of the dentinal fluid. Any reduction in this conductivity will reduce the dentin sensitivity.

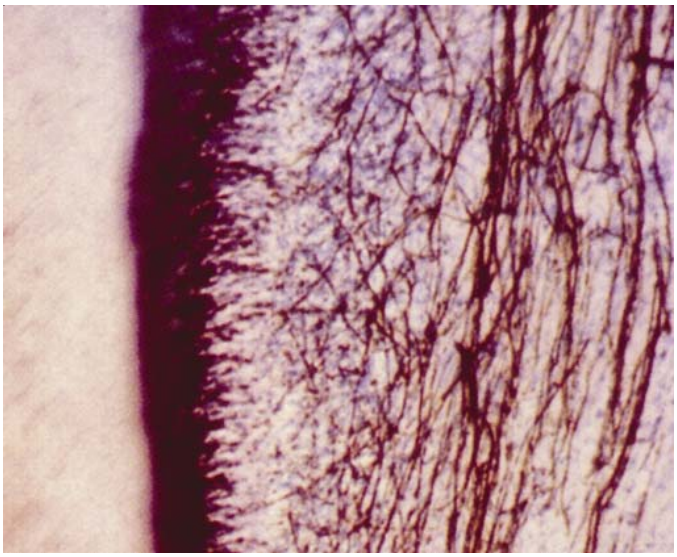

Fig. 3.1: Myelinated and un-myelinated nerves enter the pulp through apical foramen

Usually both the myelinated A fibers and un-myelinated C-fibers carry pain impulses. The sensory and the sympathetic nerve endings are activated at an early stage of the inflammatory process and are the initiator of vasodilatation. This is the start of the protective response to the injury by increasing blood volume and vascular permeability in the affected areas. Both the sympathetic and sensory nerve fibers have effect on the pulpal circulation. The number of nerve fibers decreases with age, which explains the reduced sensitivity in older adults.

Pulpal pain is characteristically pulsating, long lasting and of variable severity, very rarely excruciating. It is also affected by changes in blood pressure. The typical dentinal pain is short lasting, sharp and may be lancinating. The cold stimulus is considered more painful than the hot stimulus. In cold stimulus, there is outward fluid flow that results from shrinkage of the contents of the tubules. When heat is applied the contents of the tubules expand and an inward flow occurs, leading to comparatively less pain.

Check Your Progress 1

What is the origin of pulp? Why cold stimulus is more painful than hot one?

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3.3.2 Composition

Dentin has an average of 45,000-90,000 tubules/mm^2 near the pulp, 30,000-35,000 in the middle and 10,000-25,000 at the periphery. At the pulp surface, the diameter of the tubules ranges from 2.5-3.0 μm and at the periphery to less than 1.0 μm. Each tubule is about 1 μm in diameter and 2.0-3.0 mm in length. The occluded dentinal tubules referred to as dentin sclerosis, react differently to acid etching. The etching time here is to be modified to provide an adequate hybrid layer of collagen and resin.

The interface dentin with irregular often atubular dentin forms a barrier between the physiologic secondary and tertiary dentin. This barrier reduces the permeability of the affected dentin and may make it impermeable because the tubules from primary dentin do not cross the interface dentin. This phenomenon protects the pulp.

Generally, dentin directly under the dentino-enamel junction is only 1.0% permeable, but permeability increases to 7.6% halfway to the pulp and up to 22.0% at the pulp surface. The pulpal circulation maintains an intercellular hydraulic pressure of about 24 mm of Hg, which causes the fluid flow in the tubules to be directed outwards from the pulp to the dentino-enamel junction. Chemical and bacterial products when introduced into unprotected dentinal tubules diffuse against the pressure gradient towards the pulp. During cavity preparation by the operator, a layer is formed by the action of the cutting instrument on the calcified dentinal matrix called the ‘smear layer’. This mat of organic and inorganic particles obliterates the tubules to some extent but can be removed by acid etching and other means.

It is richly supplied by thin walled but wide capillaries. Bundles of myelinated nerve fibers accompany the blood vessels. Fibers predominantly present are collagenous and reticular fibers which lie in close proximity to the blood vessels along with many undifferentiated mesenchymal cells and histiocytes. These are believed to transform into secondary odontoblasts that form reparative dentin following the death of primary odontoblasts. When confronted with toxic bacterial or chemical products by way of dentinal tubules, the pulp usually responds initially by acute pulpitis. The response resolves naturally if the injurious agent is removed or its concentration lowered. If pulpitis does not resolve, it may spread to involve the pulp in liquefaction necrosis or chronic inflammation. Differentiation of secondary odontoblasts may eventually lead to formation of reparative dentin.

3.3.3 Factors Influencing Pulpal Response

The risks of pulpal response associated with any dental material depend to a large extent on its ability to diffuse through dentin and accumulate in the pulp. It has been established that materials, which may not be toxic at low concentrations become toxic at high concentrations. Pulp reactions to the same irritant may vary not only between different persons but also between different teeth in the same mouth, and between opposing teeth of the similar anatomic form. Factors influencing pulpal response are:

a) **Dentin Permeability**

The rate of permeation of substances through dentin depends upon a number of factors like:

i) **Location**: Dentin permeability varies in different areas of the same tooth e.g., it increases towards the pulpal side. This is because both the tubule diameter and the number increase towards the pulp chamber.

ii) **Dentin Diffusion Surface Area**: This is a product of tubule diameter and number, which directly influences the wetness and hence the hydrolytic dissolution of the restorative material.

iii) **Smear Layer**: The presence of the 1-5 μm thick smear layer reduces permeability.

iv) **Intratubular Contents**: The intratubular contents such as mineral deposits, collagen fibrils, proteins etc. may reduce permeability.

v) **The Concentration and Solubility of the Diffusing Solutes**: The substances with high molecular weight and size are less penetrating than the substance with low molecular size and weight. Solutes that are water soluble would show a rapid rate of penetration.

vi) **Patency of the Dentinal Tubules**: Sclerotic dentin is less permeable than the physiological tubular dentin.

vii) **Reparative Dentin**: In case where reparative dentin has been formed previously, the response of the pulp is less, because reparative dentin reduces the penetration and movement of the tubular contents.

viii) **Remaining Dentin Thickness**: Effective remaining dentin thickness of 2.0 mm provides an adequate insulating barrier against almost all the techniques and restorative materials.

Check Your Progress 2

1) Describe the factors which influence the permeability of dentin.

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2) What is the importance of remaining dentin?

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b) **Dentin Pretreatments**

Preconditioning of dentin by acids (etchants and conditioners) might increase dentin permeability by removing smear layer and enlarging tubule orifices. The effect depends upon the concentration and the duration for which the etchant/ conditioner is applied.

c) **Age of the patient**

It is usually accepted that the inflammatory response of the older persons is slightly more extensive and the secondary dentin formation is slightly less than in the younger person. The older pulps have a far less defensive capacity in resolving a lesion and resisting infection. However, certain authors have shown that age differences do not affect the pulpal response of human teeth.

Check Your Progress 3

Describe the features which influence the biocompatibility of any restorative material.

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3.3.4 Intensity of Pulpal Response

The response of pulp should be systematically established. There is a need to record not only the location but also the severity of pulpal responses. The pulpal reactions are categorized into following histopathological characteristics:

1) **Cellular Displacement**: It is characterized by movement of odontoblasts and leukocytes into the dentinal tubules. This occurs in the first few days and can persist well over 30 days. It provides excellent evidence of the acuteness of initial response.

2) **Infiltration of Inflammatory Cells in the Superficial and Deeper Tissues**: The infiltration of inflammatory cells into odontoblastic layer, zone of Weil and cell rich zone is arbitrarily graded from zero to three degrees merely on the basis of the number of displaced cells. When the response represents only one or two displaced cells, a graded value of one-half is given.

3) **Predominantly Inflammatory Cells**: The predominating inflammatory cells (polymorphonucleated leukocyte, lymphocyte, eosinophil, monocyte and plasma cells) are usually recorded as the intensity of the cellular inflammatory response is increased.

4) **Special Histopathologic Characteristics**: There are a number of histopathological characteristics that can be classified as given below:

   a) **Abscess Formation**: Occasionally, in a healthy pulp, there may occur certain abscess like conditions (dense accumulations of leukocytes between the odontoblastic layer and the predentin), which usually resolve; however, any technique that produces such conditions should be modified or eliminated. These characteristics are not necessarily localized.

   b) **Foci of Necrosis**: The dentinal burns and the lesions induced by toxic restorative materials or chemicals, lead to loss of cellular details, collapse of vascular channels and a paucity of inflammatory cells. Subsequently, these lesions are heavily infiltrated by inflammatory cells and might either resolve with granulation tissue replacement or undergo abscess formation.

   c) **Lesions of Delayed Healing**: Such lesions usually present dense infiltrations of chronic inflammatory cells and may develop abscess formation.

   d) **Regeneration of Odontoblasts**: With the resolution of a lesion, most or all the inflammatory cells may disappear and leave behind an atrophic or degenerate odontoblastic layer, even exhibiting foci completely lacking in primary odontoblasts (Fig. 3.2). In some instances, only regenerated odontoblasts are found and a distinction needs to be made between these two types of layers.

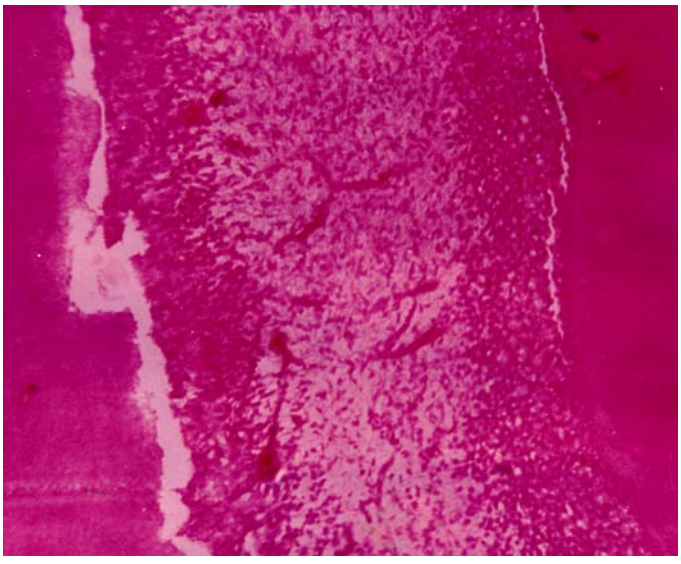

Fig. 3.2: Degenerated odontoblastic layer, exhibiting foci completely lacking in primary odontoblasts

e) **Reparative (tertiary) dentin formation**: The incidence of reparative dentin formation depends upon the initial irritation caused by cutting the tooth structure and placing the restorative material. With high-speed techniques used these days, a very low incidence of reparative dentin formation results, leaving many primary dentinal tubules patent for the subsequent seepage of toxic products into the pulp.

Reparative dentin seldom occurs in human pulp tissue sooner than 30 days. Many a times, the lesions persist along with inflammatory response and the differentiation of new odontoblasts is difficult.

Check Your Progress 4

1) Describe the 'Cell-free Zone' and its importance in restorative dentistry.

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2) How is reparative dentin formed?

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3.3.5 Stages of Pulpal Inflammation

The reaction of the pulp to external stimuli is reflected in two broad processes: Inflammatory changes and secondary dentin formation. Various authors have graded these changes for evaluating pulpal response. The inflammatory processes have been qualitatively divided into slight, moderate and severe. Slight reaction recognizes the increased number of cells in the cell free zone and in the adjacent pulpal tissue. These cells are similar to fibroblasts and undifferentiated cells. However, few inflammatory cells are also observed. The capillaries blood flow is also increased localized to the affected dentinal tubules. The irregularities in the odontoblastic layer are also observed (Fig. 3.3).

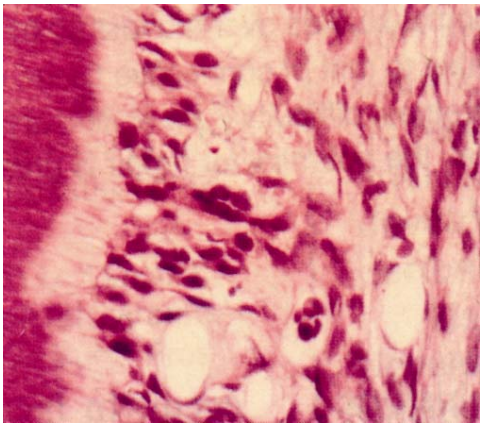

Fig. 3.3: Slight inflammatory reaction.Note the irregularities in the odontoblastic layer

Moderate reaction is characterized by increased number of cells around the injury site. The mononuclear leucocytes and the neutrophils invade the odontoblast-predentin area. Some odontoblastic nuclei can be seen in dentinal tubules. The number of capillaries is increased along with the blood flow. Occasional haemorrhage in odontoblastic or subodontoblastic zone is also observed (Fig.3.4).

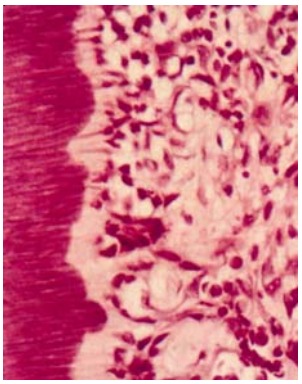

Fig. 3.4: Moderate reaction .Haemorrhage in subodontoblastic zone

Severe reaction is characterized by marked cellular infiltration, including abscess formation. The odontoblastic layer remains unidentified. This layer is either destroyed or greatly disrupted. The predentin is not formed. Numerous blood vessels are found in the tissues surrounding the cellular infiltration (Fig. 3.5).

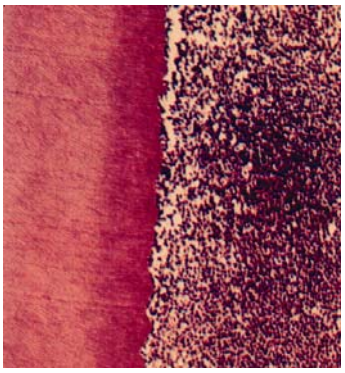

Fig. 3.5: Severe Reaction. Odontoblastic layer remains unidentified

Since the qualitative approach is subjective, attempts have been made to quantitatively designate pulpal reactions. The technique measures the cytoplasm-nucleus ratio of the odontoblasts along the area of pulpal reaction. Statistically, quantitative method is considered more accurate, however, at initial level, the qualitative approach is more appropriate.

Earlier plus sign was used to indicate the degree of infiltration of inflammatory cells in the odontoblastic layer and in the rest of the pulp. The plus sign was also used to indicate the different amounts of secondary dentin formation.

Later, the inflammation and secondary dentin formation was graded as I^0-I^3 and D^{-1} – D^3 respectively.

- I^0 – Absence of inflammation and no disturbance of the odontoblastic layer.
- I^1 – Involvement of the odontoblastic layer only, including aspiration of the nuclear debris.
- I^2 – Extension of inflammation to the subodontoblastic layer.

- I^3 – Involvement of the central pulp.
- D^{-1} – Absence of secondary dentin formation and destruction of odontoblasts.
- D^0 – A normal appearance of odontoblastic layer with a normal width of predentin (20-30 μm).
- D^1 – A slight amount of secondary dentin formation (35-60 μm)
- D^2 – A moderate amount of secondary dentin formation (60-90 μm)
- D^3 – A considerable amount of secondary dentin formation (90 and >90 μm)

Check Your Progress 5

1) Describe stages of pulpal inflammation.

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2) How will you grade the secondary dentin formation?

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3.4 TESTS FOR EVALUATION OF BIO-COMPATIBILITY

The material should pass all the three levels of bio-compatibility testing before it is approved for final usage in the human oral cavity. The three levels are as follows:

Primary tests:

- Cytotoxicity tests
- Genotoxicity tests.

Secondary tests:

- Systemic toxicity testing
- Inhalation toxicity testing
- Skin irritation and sensitization tests
- Implantation tests.

Preclinical usage tests:

- Pulp and dentin usage tests
- Pulp capping and pulpotomy usage tests
- Endodontic usage tests.

Let us learn more about these tests:

3.4.1 Primary Tests

These are the most rapid and economical and include:

- *Cytotoxic testing* includes the placement of a dental material directly on to the tissue culture cells. The cells react metabolically depending upon the components that leach from these dental materials. The toxic response can be quantified by measurement of some biologic function such as DNA synthesis, protein synthesis, or the concentration of components that cause a decrease in cellular activity by 50% i.e. TC 50 concentration. TC 50 concentration is inversely proportional to the potency of the cytotoxic response i.e., lower the TC 50 concentration higher is the potency. These tests do not measure whether materials biodegrade over long periods of time as a result of the physical and chemical environment.

- *Genotoxicity tests* mammalian or non-mammalian cells, bacteria, yeasts or fungi are used to determine specifically whether gene mutations, changes in chromosome structure, or other DNA or genetic changes are caused by the test materials, devices and extracts from materials, their extract and other devices, if any.

3.4.2 Secondary Tests

Only those materials that have passed the primary tests are subjected to secondary testing. These are slightly expensive and include:

- *Oral Systemic toxicity tests* — The test sample is administered daily to rats for 14 days either by oral lavage or by dietary inclusion. If 50% of the animals survive, the product is considered safe.

- *Inhalation systemic toxicity tests* — These tests are performed on rats, rabbits, or guinea pigs in an exposure chamber with aerosol preparations. The spray material is released around the head and upper trunk of the animals for 30 seconds, each at 30 minutes intervals. After ten consecutive exposures, the animals are observed for four days. If any animal dies within this period, the agent is considered toxic, otherwise safe.

- *Skin Irritation and Sensitization tests* — The test material is held in contact with the shaved skin of albino rats for periods ranging from 24 hrs (one exposure) to 90 days (with repeated exposures) for evaluating irritation effects.

  However, for establishing allergic contact sensitization, the guinea pig is the preferred animal. An allergen is defined as a substance that is not primarily irritating on the first exposure but produces reactions on subsequent exposures of similar concentrations. The stain reactions are evaluated after subsequent application of the test material.

- *Implantation tests* — During the implantation techniques, the various physical properties of the product such as form, density, hardness and surface finish, which influence the character of the tissue response is tested. The animals are selected according to the duration of the test. The implant material is packed in plastic tubes and put into subcutaneous tissues or directly into bones in drilled holes. The reactions are evaluated.

3.4.3 Pre-Clinical Usage Tests

These are the most expensive and include:

- *Pulp and dentin usage test* — The test is designed to assess the biocompatibility of dental materials placed in dentin adjacent to the dental pulp of non-rodent mammals like dogs, ferrets etc. The material is placed in contact with open dentin 1.0 mm from the pulp and evaluated after given period of time. The specimens are examined regarding inflammatory response, reparative dentin formation and the number of micro-organisms entrapped in the surrounding cavity walls and cut dentinal tubules. If an inflammatory response is produced, the time required for its disappearance is also measured.
- *Pulp capping and pulpotomy usage tests* — The testing procedures are similar to the previous ones, except that the pulp is exposed. The animals are evaluated for dentin bridge formation, its quality and structure.
- *Endodontic usage tests* — For this test, the pulp is completely removed from the pulp chamber and roots and replaced by the obturating test material and control material. The degree of inflammation is evaluated in the periapical tissues.

3.5 THE RESTORATIVE MATERIALS AND PULP REACTIONS

There are various restorative materials used for 'normal health' of tooth. The pulp, like a pampared child, reacts to these restorative materials in different manners. Let us take these commonly used materials and see the pulp reactions.

3.5.1 Zinc Oxide Eugenol

Zinc oxide eugenol is frequently used as a material in dentistry. It is generally considered bland or even therapeutic to the pulp and is routinely used as the non-toxic control in most in-vivo tooth tests of pulpal toxicity. It is highly cytotoxic in all tissue culture test systems that lack a dentin barrier. If it comes in direct contact with bone, the pain could be extremely severe so much so if extruded into the mandibular canal, it may damage the mandibular nerve.

Zinc oxide when mixed with eugenol lead to formation of zinc eugenolate matrix. The zinc eugenolate units are held together by vander- waal forces and particle interlocking. When exposed to aqueous media such as saliva or dentinal fluid, hydrolysis of zinc eugenolate occurs yielding eugenol and zinc hydroxide. Eugenol liberated from zinc eugenolate can diffuse through dentin and into the saliva. It has been established that concentration of 10-2 mol/L of eugenol is observed below the fillings in dentin and 10-4 mol/L in the pulp. These concentrations were maintained for more than 1 week. Calcium in the dentinal tubules chelates eugenol, limiting its ability to diffuse through dentin. Eugenol also binds with the organic matrix of dentin, especially collagen, which slows the diffusion rate. Modified ZOE cements have demonstrated less release of eugenol and fewer cytotoxic effects. It has been observed that acid etched dentin may facilitate diffusion of potentially toxic amounts of eugenol to the pulp.

Eugenol is bactericidal at relatively high concentration of 10-2 to 10-3 mol/L. Brief exposure to 10-2 mol/L and prolonged exposure to 10-3 mol/L of eugenol

can kill mammalian cells. Even lower concentrations can inhibit cell respiration and cell division. Inhibitory eugenol concentrations are significantly higher than the one required for anti-inflammatory effects. The zinc oxide eugenol placed in direct contact with the pulp tissue result in chronic inflammation and necrosis.

However, when placed against dentin, the cytotoxic effects are nil. Various mechanisms that explain the cytotoxicity of eugenol are:

- Eugenol can be oxidized by peroxidase enzyme; the product formed is toxic to hepatocytes.
- It has a high affinity to plasma membranes because of its lipid solubility, which can cause cell damage.
- Eugenol has shown to uncouple oxidative phosphorylation in mitochondria.

At concentration levels of 10-4 mol/L or just below it, eugenol has been shown to inhibit prostaglandin synthesis and sensory nerve excitability. At low concentrations the intradental nerve activity is blocked reversibly just like a local anaesthetic, whereas at high concentrations of eugenol, nerve conduction is irreversibly blocked, indicating a neurotoxic effect. It exerts an anti-inflammatory effect by the following mechanisms:

- Protects tissue from damage by inhibiting neutrophil function and chemotaxis.
- Inhibits prostaglandin and leukotriene synthesis, which are important mediators of inflammation by increasing blood flow and vascular permeability and lowering pain threshold.
- Eugenol causes vasodilatation and decreases the response of these vessels to nor epinephrine and histamine. Vasodilatation would result in prevention of toxic accumulations and rapid removal of irritants.

Effects of Eugenol

Toxic(High Dose)	Beneficial(Low Dose)
– Induces cell death	– Inhibits white cell chemotaxis
– Unknown vascular effect	– Inhibits prostaglandins synthesis
– Inhibits cell growth and respiration	– Inhibits nerve activity

Thus, it can be seen that the effect of eugenol is dose dependent.

3.5.2 Zinc Phosphate Cement

Zinc phosphate cement has been the most widely used dental cement. It is used as a luting agent for all indirect restorations and appliances. In deep cavities, it is used as a base as a substitute of lost dentin, because the thermal conductivity is approximately equal to that of enamel and considerably less than that of amalgam and gold.

When used as a thick base, it has a low toxicity level but when used as a luting agent in a thin state, it can be quite toxic. A young tooth or deep preparations with wide open tubules are more susceptible to intense inflammatory response to zinc phosphate cement, than an older tooth, which has produced a considerable amount of sclerotic and reparative dentin that blocks the dentinal tubules and

prevents acids from reaching the pulp. Zinc phosphate cement is irritating because of its low pH and the rapid penetration of its lower molecular weight phosphoric acid into the dentinal tubules and pulp tissues. The hydraulic forces, which are induced during the seating of the restoration or during functional movements, cause phosphoric acid in large quantities to be forced into the dentinal tubules. Since dentin can be penetrated by phosphoric acid to a depth of more than 1.0 mm, especially in luting procedures, insulating materials are indicated for deep preparations with narrow remaining dentin thickness. In deep preparations, a moderate to severe localized pulp damage is produced within the first three days probably because the cement has an initially low pH on setting. The pH of the set cement approximates neutrality only at 48 hours. Resolution of inflammation occurs by 5 to 8 weeks.

When zinc phosphate is used as a luting agent, there occurs an intense diffuse infiltration of neutrophils throughout the pulp tissue. The initial pH of luting mix ranges from 2.0 - 3.3, which changes to 3.0 - 4.2 after one hour. It has been established that such low pH induced vascular thrombosis and necrosis in rodent pulp when the duration of exposure is prolonged over thin dentin. The pH for zinc phosphate, polycarboxylate and glass ionomer cements rise during the first 15 minutes. The rise in pH was faster for the zinc phosphate and polycarboxylate cements but slower for the glass ionomer cements. When the cement is used as a base with a remaining dentin as 1.0 mm, there occurs lifting of the odontoblastic layer and also the inflammatory cells become countable. In case the remaining dentin thickness is 1.5 mm, there occurs only moderate reaction with segmented odontoblastic layer and some reparative dentin formation.

3.5.3 Silicate Cements

Silicate cements, though widely used earlier, are rarely used these days. Its adverse effects are mainly because of the prolonged acidity due to phosphoric acid, even 24 hours after the setting of the cement; and to some extent because of the release of fluoride. The pH of silicate cement at the time of insertion into the cavity is less than three and it remains below seven even after seven months. Fluoride ion concentrations of 15-25 gm/ml are also known to reduce cell growth.

3.5.4 Polycarboxylate Cements

Polycarboxylate cements are a combination of aqueous polyacrylic acid and zinc oxide. They have an excellent biocompatibility with the pulp and are almost equivalent to zinc oxide eugenol cements. The pH of the cement liquid is approximately 1.7. The pH of the mix rises rapidly as the setting reaction proceeds. Despite the initial acidic nature of the polycarboxylate cements, these products produce minimal irritation to the pulp probably because the larger size of the polyacrylic acid molecule limits its diffusion through the dentinal tubules. At all times, the pH of the polycarboxylate cement is higher than the pH of the phosphate cements. Also in the set cement the acrylic acid ions bind the metallic ions so tightly that they are not easily leached out from the set cement.

3.5.5 Pulpal Reaction to Composites

The earlier resin bonded materials, developed as tooth coloured materials, were detrimental to the pulp. The free monomer of these self cured or chemically cured materials was injurious to the health of the pulp. Over the years, with the development of composites and also the tremendous improvements in the material aspect of these resins, the pulp effects are minimum but not absent.

The bond strength of these materials is linked to the etching of the normal mineralized tissue. The etching, since being carried out for 10-15 seconds only, its effect on pulp is minimum or negligible. (Fig. 3.6)

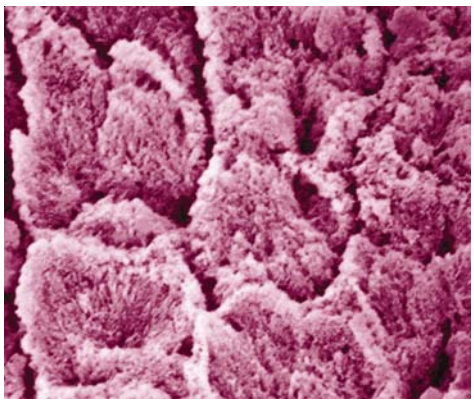

Fig. 3.6: Etched surface

To achieve optimal bonding to dentin, the adhesive material (bonding agents) must penetrate the demineralized dentin; enter the dentinal tubules and their branches. (Fig. 3.7a,b)

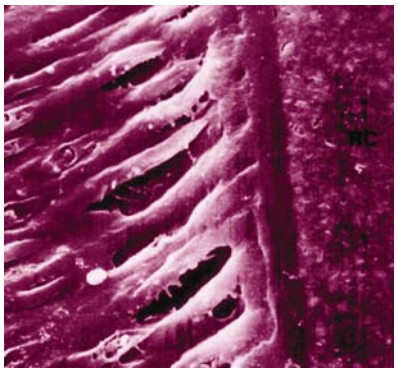

Fig. 3.7a: Demineralised dentin

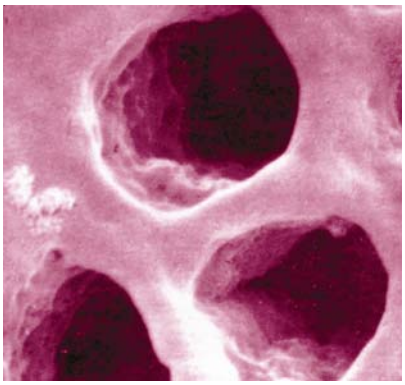

Fig. 3.7b: Dentinal tubules

The resin monomer enters the collagen fibers to completely infiltrate the demineralized dentin forming the hybrid layer and over this the composite is filled. (Fig. 3.8).

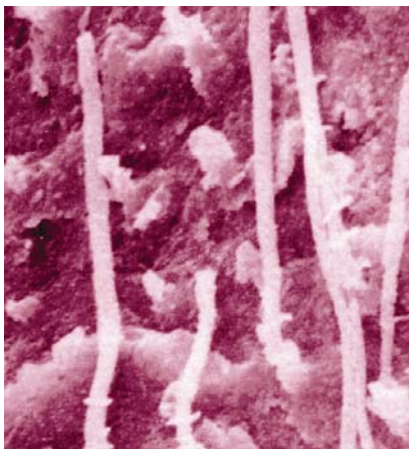

Fig. 3.8: Infiltration of resin monomer

The bonding materials have been tried as 'pulp capping agents' with reasonably good results. It imparts healing process. However, the polymerization shrinkage of the composite creating vacuum in between the remaining dentin and the restoration might create problems for the pulp. It has been established that even after curing, the monomer is leached from composites. However, the monomer is usually neutralized by the bonding agents and the remaining dentin thickness. In case the dentin thickness below the composites is less, the leaching monomer can affect the underlying pulp.

Check Your Progress 6

What are the pulp reactions to etchants and adhesives?

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3.5.6 Pulpal Reaction to Glass-Ionomer Cements

The glass-ionomer cement is a combination of fluoro-alumino silicate and polyacrylic acid and water. The set material is composed of an inorganic-organic complex with high molecular weight. The material is considered biocompatible, since it is indicated in cavity bases and liners. The bonding of glass-ionomer material to dentin involves chemical and mechanical bonds. The chemical bond is based on exchange of ions between carboxyl group of substrate and calcium ions. The mechanical interlocking is based on the demineralization of exposed dentin by polyacrylic acid.

Various modified forms of glass ionomers are also available. These are resin modified glass ionomer cements and compomers. These materials achieve advantages of both the glass ionomers and composites. After one week of placement of glass ionomer cement, the odontoblastic layer is disrupted and dilated blood vessels seen in pulp area (Fig. 3.9). Bacterial penetration into tubules is also observed. After about a month the pulp tissue recovered and displayed a normal appearance. The disruption of odontoblasts becomes normal.

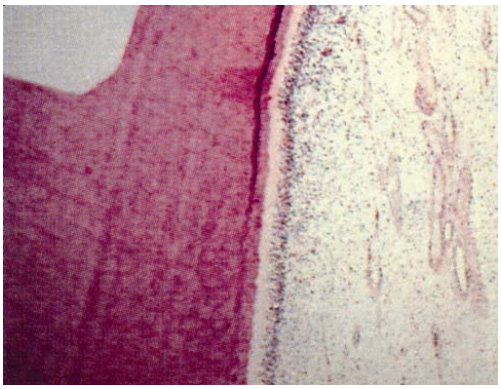

Fig. 3.9: Dilated blood vessels in the pulp

Check Your Progress 7

Are Glass-ionomer cements conducive to pulp?

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3.6 PULPAL REACTION TO TOOTH PREPARATION

In the recent past, high speed cutting instruments have largely been employed in cutting cavities and preparing teeth for crowns. Adequate cooling of the burs is essential to prevent injury to dentin and also to underlying odontoblastic region of the pulp. The water spray should reach at the site of cutting and bur. Light pressure with intermittent cooling can minimize temperature increase.

When the cooling is inadequate, the injury leads to displacement of odontoblastic nuclei into the dentinal tubules (Fig. 3.10). The odontoblasts appear disorganized. The cooling, if missing, or excessive pressure is utilized constantly the burning of dentin becomes evident (Fig. 3.11). Many a times, the smear layer may obturate the opening of the tubules and reduce the extrusion of contents of dentinal fluids. It is established that such flow of fluid, later, is helpful in formation of peritubular dentin. The disturbance and redistribution of the cellular constituents, if continued, leads to degeneration of odontoblastic processes. This might form the base for formation of 'dead tracts' and subsequently formation of tertiary dentin.

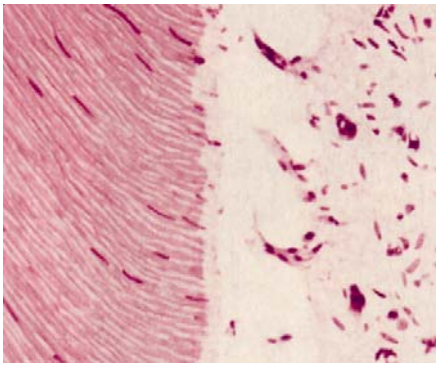

Fig. 3.10: Displacement of odontoblastic nuclei

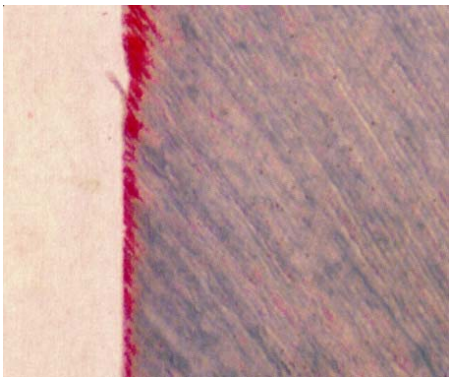

Fig. 3.11: Burning of dentin due to inadequate cooling or blunt cutting instrument, during tooth preparation

Gentle grinding over the dentin leads to increased blood flow. Grinding halfway into the dentin causes much more increase in blood flow. However, grinding in the inner half leads to decrease in blood flow. The blood flow has been seen as decreasing after crown preparation, since the inner thickness of dentin might not be more than 1.0 mm.

The displacement of odontoblastic nuclei into the dentinal tubules has been established. Earlier, 'aspiration of odontoblastic nuclei' was considered the phenomenon, but recently the accepted phenomenon is found to be the 'displacement of odontoblasts'. The cavity depth or the remaining dentin thickness has always been the key factor in such reactions. A number of morphologic changes occur including intracellular disorganization of the odontoblasts. This can lead to disruption of odontoblastic layer. The exact mechanism of such movements might not be fully understood, however, mechanical distortion of the dentin can be the cause.

The alternative means of cavity preparation such as air abrasion and laser cavity preparation also affects the pulp. The generation of heat with the use of lasers is more detrimental than air abrasion technique.

Check Your Progress 8

Describe the reaction of pulp during tooth preparation.

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3.7 PULPAL REACTION TO CARIES

The dental caries may be in any form or at any site, affects the underlying pulp. The initiation of pulpal inflammation also stimulates the repair process. A balance usually occurs between repair and injury and deviation from the balance lead to changes in the dentin-pulp complex.

The cellular injury associated with caries arises from acidic environment created by diffusion of bacterial acids. The ability of the cells to withstand this acid environment depends upon hydrogen ion concentration. Local lymphatic drainage in the pulp may contribute to clearance of acid but the pulpal lymphatic system is not well developed. The odontoblasts are significantly reduced in size beneath the active enamel lesions whereas at the site of arrested lesions such reduction is not exhibited. Before the carious lesion reaches dentino-enamel junction, a significant reduction in cytoplasm to nucleus ratio of odontoblasts and also reduction in predentin thickness is observed. At ultrastructural level increase in activity of phosphate enzyme is reported in odontoblasts and in predentin beneath the carious lesions.

The terms 'slight', 'moderate' and 'severe' have the subjectivity and also the varied interpretation of the histopathological results make it difficult to standardize the classification of pulpal reactions.

The onset of pulpal reactions to caries starts early, but its effect has not been documented properly. Pulpal reactions have been established even with the formation of white-spot lesions. There are marked changes in dentin with 'acute' or rapidly progressing caries than with ‘arrested’ or slow progressing caries. The odontoblasts in active lesion were significantly smaller than were, odontoblasts in other lesions. The cellular proliferation of the cell free zone is also observed in active lesion only. Changes in the sub-odontoblastic region also occur early and these changes might include early onset of neurogenic inflammatory reactions.

The active lesions do not show any marked changes in dentin mineralization. Slowly the demineralization of the affected dentin starts. Soon after the demineralization, the evidence of tertiary dentin can be noted at the pulp dentin border usually defined as reactionary dentin. The pulp adjacent to deep caries show the presence of chronic inflammatory exudates, including lymphocytes, macrophages and plasma cells. The localized increase in dentin thickness is often accompanied by reduced odonotoblastic layer in the affected area. The tertiary dentin can be seen at the affected site. The accumulation of inflammatory cells is particularly great whenever the bacteria associated with caries process reach the tertiary dentin. This stage corresponds to severe pulpal inflammation with minimum chances of healing. It may lead to pulp necrosis at a later stage.

In case of rampant caries, the breakdown of affected enamel and dentin will occur within months. This leads to destruction of odontoblasts and lack of tertiary dentin formation. If the odontoblasts are destroyed slowly, there is formation of hard tissue, which is initially atubular with some cellular inclusions, defined as fibrodentin and interface dentin. The progress of caries, if halted, lead to formation of reparative dentin with differentiation of new secondary odontoblast like cells. If the lesion is allowed to progress, pulp necrosis may follow.

The immune system helps in defence mechanism of pulp. In association with carious dentin, accumulation of immuno-competent cells has been demonstrated. During rapid lesion progression, accumulation of immuno-competent cells is observed in conjunction with reduced number of primary odontoblasts. In case the tertiary dentin develops, it is formed as an atubular dentin.

3.8 PULPAL REACTIONS TO TRAUMA

Physical trauma in the form of accidents is very common, especially during adolescent period. Trauma can be with wear, erosion, orthodontic movements and other occlusal traumas. Many a times only quick orthodontic movements cause changes in the pulp dentin complex.

The extensive trauma involves avulsion or displacement of the jaw bones. The blood supply to the pulp is severed off. Either the pulp is necrosed with time or characterized by formation of hard tissues in the pulp chamber. These hard tissues have been labelled as ostodentin, because it has cellular infiltration similar to bone. In case of reimplantation, there occur degeneration of odontoblast and simultaneous loss of the adjacent cell free zone. This degeneration is accompanied by reduction of the width of predentin. In teeth with open apices, the odontoblasts can survive and produce reactionary dentin. Atypical interface dentin is formed separating the primary dentin and reparative dentin. The mechanism of cellular differentiation is similar to the avulsed and implanted teeth. The stimulation leads to differentiation of odontoblasts or odontoblast like cells.

With the orthodontic movements of teeth putting pressure more than required, there occurs increased blood flow in the pulp. Hyperemia has been shown to be more marked in teeth adjacent to the one where force is applied (Fig.3.12).

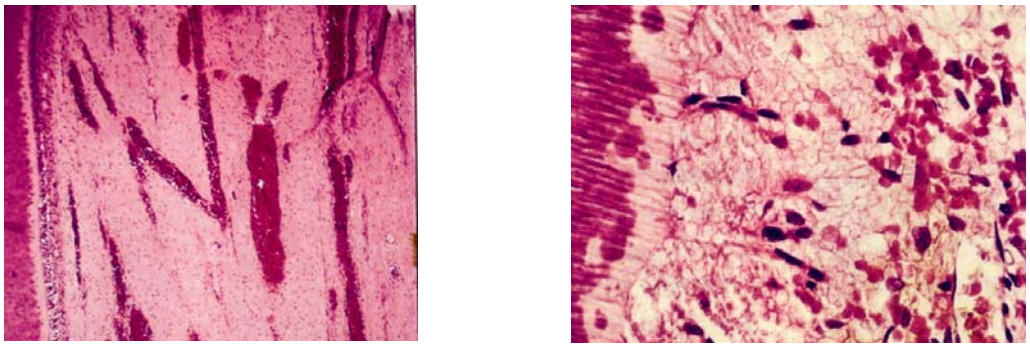

Fig. 3.12: Hyperemia of the pulp in tooth undergoing orthodontic treatment

The orthodontic treatment is becoming more common in adults these days with the root fully formed. These teeth are likely to have more pulpal changes than the teeth in young individuals. The vacuolization of odontoblastic layer in the pulpal tissue is often the result when teeth are subjected to heavy forces. These changes are suggestive of degeneration of pulp. The impaired predentin formation is also suggestive of degenerative changes. After the force is removed, the predentin formation starts.

3.9 PULPAL REACTION TO VITAL BLEACHING

The vital tooth bleaching incorporates 10% carbamide solution in different modifications. Initial reaction of the pulp to bleaching includes less distinction of cell free zone in areas on facial surface of the teeth; the cells migrate to these areas. Scattered leucocytes are observed along with irregularities in the pseudostratified odontoblastic layer. Only slight pulp reaction is observed. In any case even high concentration of the carbamide etc. does not produce moderate or severe reactions. Few authors are of the view that using higher concentration of the hydrogen peroxide and the heat is potentially harmful to pulp. The changes in pulp tissue are reversible after or within two weeks time. Therefore, two weeks of treatment with 10% carbamide peroxide used for nightguard vital bleaching is considered safe for the pulp.

Check Your Progress 9

Does bleaching agents affect pulp?

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3.10 LET US SUM UP

The dental pulp is a special organ of the tooth .It is designed in a very meticulous manner with a 'hand to mouth living' .It has its own reparative mechanism, but is compromised with vascularity because of 'bottle-neck' apical foramen. you know that enamel does not regenerate and so, the tooth has to be repaired for it's normal functioning. The repair of tooth requires the restorative procedures and materials.

The understanding of the restorative materials and procedures make you learn as to what is palatable to tooth and it's associated structures and what is not! You need to apply it in your clinical practice to get positive reactions from the pulp.

The candidate after reading this unit should be able to answer the following questions.

3.11 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) Pulp is a connective tissue, which is derived from the mesenchyme, the dental papilla. Cold stimulus is considered more painful than the hot; as in cold stimulus there is an outward fluid flow that results from shrinkage of the contents of tubule.

Check Your Progress 2

1) Factors influencing dentin permeability are:

   i) location: dentin permeability increases towards pulpal side. This is because both tubule diameter and number increases towards pulp chamber.

   ii) dentin diffusional surface area.

   iii) smear layer: presence of smear layer reduces permeability.

   iv) intratubular material such as mineral deposit, collagen fibrils, proteins etc. may reduce permeability.

   v) concentration and solubility of diffusing solutes.

   vi) patency of dentinal tunules.

   vii) reparative dentin formation.

   viii) remaining dentin thickness.

2) Remaining dentin thickness is significant as the length of tubule increase the concentration of solute reaching the pulp decreases. It has been shown that 0.5 mm thickness of dentin reduces the toxicity level of a material to 75% and an 1 mm thickness over 90%. Effective remaining thicknes of 2 mm provides an adequate insulating barrier against almost all techniques and restorative materials.

Check Your Progress 3

1) The risks of pulpal response associated with any dental material depend to a large extend on their ability to diffuse through dentin and accumulate in pulp. Factors influencing pulpal response are:

   a) Dentin permeability which varies according to location, smear layer, intratubular material, patency of dentinal tubule, reparative dentin etc.

   b) Dentin pretreatments — by acids might increase dentin permeability.

   c) Age of the patient.

Check Your Progress 4

1) 'Cell poor zone' or 'Weil's layer' lies beneath the odontoblastic zone of pulp and is composed principally of young fibroblasts.

   This zone is important for operative dentistry in the formation of reparative dentin. If the intensity of stimulus (caries, operative procedures etc.) is enough to induce the differentiation of odontoblasts from undifferentiated mesenchymal cells lying in proximity to blood vessels in pulp, reparative dentin is formed thereby obliterating the underlying cell poor zone by movement of odontoblasts towards pulpal side.

   In mild to moderate inflammation, this zone is infilterated by inflammatory cells.

2) The greater the degree of initial response due to irritation caused by cutting and placing of a restorative material results in differentiation of secondary odontoblast which may eventually lay down reparative dentin. Reparative dentin seldom occurs in human pulp tissue sooner than 30 days.

Check Your Progress 5

1) Stages of pulpal inflammation have been divided into:

   a) Slight reaction; increased number of cells which are similar to fibroblasts and undifferentiated cells. Few inflammatory cells and increased number of capillaries.

   b) Moderate reaction: mononuclear leucocytes and neutrophils invade odontoblast — predentin area. Increased number of capillaries and occasional haemorrhage in odontoblastic zones.

   c) Severe reaction: marked by cellular infiltration including abscess formation. Odontoblastic layer is either destroyed or greatly disrupted. Predentin is not formed. Numerous blood vessels are found in the tissues.

2) Gradation of secondary dentin formation:

   D-1- absence of secondary dentin formation and destruction of odontoblasts.

   D0- a normal appearance of odontoblastic layer with a normal width of predentin (20-30um)

   D1- a slight amount of secondary dentin formation (35-60um)

   D2 - a moderate amount of secondary dentin formation (60-90um)

   D3- a considerable amount of secondary dentin formation (90um)

Check Your Progress 6

1) Pulpal reaction with etching is minimal or negligible since the process is carried out for 10-15 seconds only. Adhesives (bonding agents) penetrates the demineralized dentin, enter the dentinal tubules and their branches forming hybrid layer with no or insignificant pulpal effect.

Check Your Progress 7

1) GIC occasionally is mentioned being beneficial over pulp may be considered ancedotal and cannot be recommended for clinical practice. Calcium hydroxide is the most recommended pulp protection agent under GIC.

Check Your Progress 8

1) When cooling during cavity preparation is inadequate:

   i) Injury leads to displacement of odontoblastic nuclei into the dentinal tubules.

   ii) odontoblasts appears disorganized.

Check Your Progress 9

1) Bleaching agents does not effect pulp with only slight pulpal reactions with 10% carbamide solution in different modifications used in vital tooth bleaching.

3.12 FURTHER READINGS

Endodontics (5th Edition) by Ingle.

Pathways of Pulp (8th Edition) by Stephen.

Endodontic Practice (11th Edition) by Gross man.

Endodontics (5th Edition) by Weine.