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Reactions of Pulp to Various Restorative Procedures and Materials In Operative Dentistry: A Review.

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ABSTRACT

Dental pulp is a specialized connective tissue consisting of terminal nerve endings, blood vessels, ground substance, interstitial fluids, odontoblasts, fibroblasts and other minor cellular components. It is a multi functional tissue encased with a highly mineralised hard tissues dentin and enamel from outside. Pulp is highly sensitive to thermal stimuli and various other forms of stimulus which may occur during excavation and restoration of a deep carious lesion. It has got a high regenerative potential capable of producing tertiary or reparative dentin to any external injury. Therefore, this article enlightens the pulpal response or reactions towards various restorative procedures and materials and various restorative factors causing a pulpal injury.
Keywords: Dental pulp, Pulpal injury, Restorative factors, Restorative materials

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INTRODUCTION

Dental pulp is defined as a special organ with a unique environment of the unyielding dentin surrounding a resistant, resilient soft tissue of mesenchymal origin,S reinforced with a ground substance. It has a close relationship between its peripheral cells, the odontoblasts and dentin making it a functional entity. It is referred as the pulp-dentin complex. Pulp has soft gelatinous consistency and it is infiltrated by a network of blood vessels and nerve bundles emanating from the apical region. [1]

The elaboration of dentin to form the tooth and to protect against and to repair the effects of noxious stimuli is the primary function of the pulp. The elaboration of the dentin creates a special environment for the pulp. [2]

The encasement of the pulp in dentin creates an environment that allows only small amounts of intercellular accommodation of exudate during inflammatory reactions and this inability of the pulp to swell creates abnormally high pressure in the area of inflammation, with interruption of blood flow due to the collapse of the pulpal veins resulting in anoxia and localized necrosis. [3]

The dental pulp has been described as a unique organ with limited capacity to heal after it is subjected to repeated insults. The intact hard tissues of the tooth normally protect the pulp by acting as physical barriers to noxious irritants. Any pulpal injury can result in inflammation and its consequences, such as increased vascular permeability, vasodilatation, pain, hard tissue resorption and sometimes pulpal necrosis. Although irritants can be physical, thermal or chemical, microbes are considered to be the major cause of pulpal pathosis. [4]

DISCUSSION

The dental pulp is a very dynamic tissue that responds to external stimuli in a variety of ways.

Restorative factors contributing to pulpal injury [5]:

Effect of cavity preparation:

- Frictional heat.
- Desiccation.
- Exposure of dentinal tubules.
- Direct damage to odontoblast processes.
- Chemical treatment of exposed dentinal surface.

Factors associated with the restorative material and its placement:

- Material toxicity
- Insertion pressures
- Thermal effects
- Induced stresses

Effects subsequent to restoration:

- Marginal leakage
- Cuspal flexure

Effects of cavity preparation:

The pulp immediate reactions to high-speed preparation can be classified in three groups:

- Structural changes- the most important being the displacements of the odontoblastic nuclei into the dentinal tubules;

- Vascular reactions- dilatations, decrease of blood flow and/or hemorrhages on wide areas, interstitial edema, vascular stasis;
- Inflammatory reactions even in the absence of bacteria.

The increase of pressure in the pulp, as a result of the exudate and the interstitial edema, may cause self-aggression for the pulp elements and, thus, the amplification of inflammatory phenomena. Also, the high pulp pressure leads to compression of the venules, followed by the slowing down of the return circulation and venous stasis, determining the accumulation of toxic products from the metabolic processes. Histologic and clinical experience showed that, although the pulp has a good regenerative potential, and the inflammatory response will often be followed by healing, the induced modifications can become significant on the long run.

Mechanical Stimuli during Cavity Preparation:

The pulp tissue reaction to cavity preparation may vary from a mild inflammatory response associated with slight tissue disorganization to partial pulp necrosis or complete pulpal breakdown. Several studies have reported on the release of enzymes and other immunoreactive substances in the dental pulp during mechanical tooth preparation procedures. The release of these substances may be due to temperature increase to the mechanical stimuli of tooth preparation, or to both. Many enzymes and other immunoreactive substances are normally present in the pulp under non-stimulated conditions. When the pulp is stimulated mechanically, these substances are released by physiologic mechanisms (eg, exocytosis) or by disruption of cellular membranes. An early study in monkey teeth examined the effect of cavity preparation on pulpal enzyme release (alkaline acid phosphatases and others). Tooth preparation by air turbine and adequate water cooling did not affect enzyme activity. When calcium hydroxide ($Ca(OH)_2$) was applied to the cavity floor, however, enzyme activity was increased after 24 hours in the odontoblastic and subodontoblastic cell layers adjacent to the $Ca(OH)_2$ covered dentin. Fifteen days later, slight dentinal formation was found, possibly indicating a role for these enzymes in stimulated hard tissue formation. [6]

Frictional heat:

The low thermal diffusivity of dentin tends to minimize direct elevation of intrapulpal temperature unless the preparation is cut deep in dentin without effective cooling. There appears to be a "critical range" for intrapulpal temperature, with an increase of approximately 6°C required before irreversible pulpal injury occurs. This extent of temperature increase occurs after about 25 seconds of dry cutting with either high- or low speed burs. In contrast, the use of water cooling led to a transient decrease in intrapulpal temperature of 6°C to 7°C.

Early studies on pulpal reactions to thermal challenges were performed by *Zach and Cohen* using rather crude instruments. They were concerned about heat generated during cavity preparation or finishing procedures. Their histopathologic assessment of the subsequent pulpal reactions to application of heat to enamel in intact teeth indicated that more progressively severe pulpal necrosis caused by increase in pulpal temperature upto 5 °C to 17°C. [7]

High speed engines and carbide burs may reduce operating time, but they are used without a coolant. The heat generated may be sufficient to cause irreparable pulp damage. The sound human teeth in which cavity preparations were done at 50,000 rpm or higher with an air turbine, with adequate water cooling of tooth showed less injury to the pulp tissue than when cavities were prepared at speeds between 6,000 and 20,000 rpm without a coolant. Damage and abscess formation of the pulp occurred when a water spray was not used and when a cavity is prepared with an air turbine and water spray, the pulp shows little or no response to the cutting. The pulpal damage is repaired more rapidly when cavity preparation is done under water spray. In evaluation of the effect of high speed on dental pulp, *Zander* stated that all research on pulp reaction to high speed instrumentation has been conducted on sound teeth. Any injury produced by high speed is superimposed upon already existing alterations in pulp. Enough heat may also be generated during polishing of a filling or during setting of cement to cause at least transient pulp injury. [5, 6]

Many authors have assumed that 5 °C to 10°C elevations in external root temperatures would produce damage to the periodontal tissues similar to that produced in pulpal tissues. *Zach and Cohen* demonstrated that cavity preparations with high-speed handpieces using air-water spray actually lowered

pulpal temperature because the water spray was cooler than the temperature of the pulp and because of the high heat capacity of water. [7]

Desiccation and direct damage to odontoblast processes:

Desiccation of the exposed dentinal surface, for example, from a stream of air used to dry the cavity preparation, leads to an outward fluid flow. If the stimulus is severe enough, injury to the odontoblast layer may result, causing disruption of junctional complexes and displacement of odontoblast cell bodies into the dentinal tubules. Direct trauma to odontoblast processes will occur with deep cutting of intact dentin. In mature dentin, odontoblast processes extend into dentinal tubules a distance of 0.1 to 1.0 mm. The odontoblast cell numbers are unaffected by cavity preparations as close as 0.5 mm to the pulp, indicating absence of an irreversible level of injury where as deeper cutting (less than 0.3 mm from the pulp) resulted in direct odontoblast injury and cell death.[8]

MarylandEA et al(1970) [9] done repair in the human dental pulp following cavity preparation, found that immediate damage to the dental pulp was greater in air-cooled than water-cooled specimens and this greater degree of injury was evident up to 15 days post-operatively. Up to 7 weeks, repair processes were more advanced when cavities were prepared with water coolant. After longer periods of time, however, the pulps of water-cooled and air-cooled specimens showed no significant differences. When repair occurred, new odontoblasts were differentiated from the less-specialized cells of the pulp. The young, uninfected, dental pulp has a very considerable potential for repair following injury.

A study by **Nyborg and Brannstrom (1968) [10]** applied heat to the dentinal floor of Class 5 cavities in human volunteers. They applied a 150°C stimulus for 30 seconds to dentin that had remaining dentinal thickness (RDT) of 0.5 mm. Histologic examination of the pulps of these teeth showed loss of odontoblasts on the side of the pulp containing the cavity. After 1 month, the pulpal reaction beneath the heated dentin exhibited excessive collagen matrix formation that occasionally contained cells and capillaries but did not mineralize.

Exposure to dentinal tubules:

Dentin cutting exposes dentinal tubules. If cavity preparations are limited to the extent of affected dentin, the pulpal effects of restorative procedures would probably be minimal: tubules occluded by mineral deposition are less permeable to bacterial products and diffusible components of restorative materials; the hydrodynamic response to cutting, heating, and drying is likely to be reduced and the risk of mechanical damage to odontoblast processes, which recede toward the pulp as tubules are occluded peripherally, is minimized. Despite the more conservative cavity preparations recommended in contemporary restorative dentistry, cutting of intact unaffected dentin is still necessary in instances such as full-crown preparations.

Effects of lasers on pulp:

Effects depend on

- Thickness of the dentin: If remaining dentin thickness is less than 1 mm, pulp is inflammed on application of laser.
- Pulse per second rate: High frequency lasers such as ND-YAG lasers with greater penetration may cause pulpal injury.
- Time of exposure: If exposed for more than 15 seconds on a particular area-significant structural damage is seen.

Yamaguchi H et al (2005) [11] concluded that Thermal pain perception thresholds for each pulp were significantly increased immediately following Nd: YAG laser irradiation. After one week, the increased thresholds values of each tooth returned to the pretreatment values. It is suggested that Nd: YAG laser irradiation has anesthetic effects on human pulp.

Krmek SJ et al (2009) [12] concluded that Cavity preparation with an Er: YAG laser did not cause significant increases in temperature in the pulp chamber of human molars.

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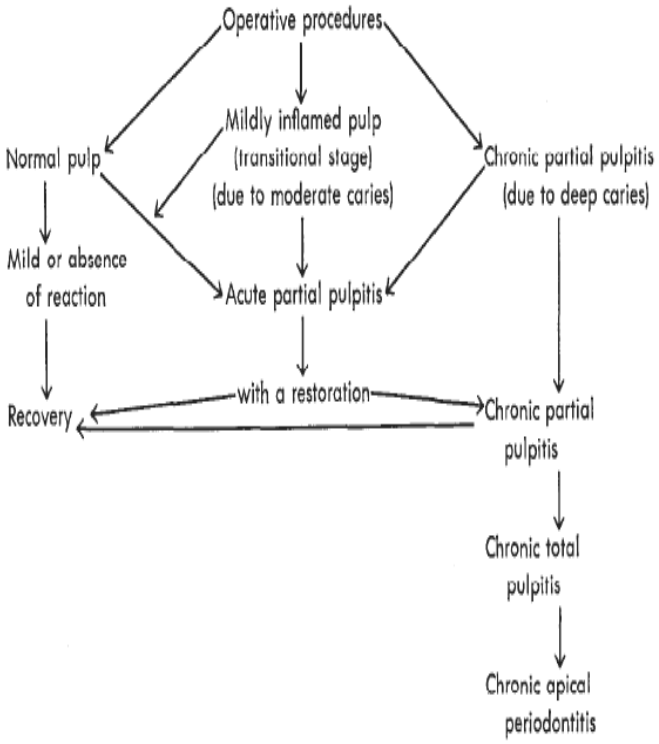
Pulpal reactions to mechanical, thermal and chemical irritants [13]:

Fig-1: Pulpal reactions to mechanical, thermal and chemical irritants



Factors associated with the restorative material and its placement:

Toxicity of restorative materials:

Along with replacing lost or damaged tissue and restoring function, a restorative material should be biocompatible, i.e., it should not elicit an adverse reaction either in the tissues with which it comes into contact or systemically. [13] Traditionally, the ideal material is "biologically neutral," which implies that it is insoluble and inert. Current trends in restorative materials include attempts to incorporate therapeutic agents that may promote pulpal healing. The International Standards Organization (ISO) in 1997 promulgated ISO standard 7405 to regulate the preclinical evaluation of dental material biocompatibility under standardized conditions. Three levels of testing are prescribed: “cytotoxicity”, using cell culture techniques; “tissue toxicity” assessed by implantation in subcutaneous tissue or muscle and "usage tests." Excellent reviews of systemic effects and cytotoxicity of dental materials are available and need not be considered here. For restorative materials, the usage test involves material placement in prepared cavities of permanent teeth for intervals of up to 3 months. The ISO standard 7405 stipulates that materials should be tested in cervical (Class 5) cavity preparations. [14]

Effects of material placement:

Amalgam is condensed under sufficient pressure to cause measurable strain in cusps. A transient neutrophil infiltration between the odontoblast layer and predentin following amalgam placement was attributed to condensation pressures. Crown cementation also involves substantial pulpal pressures to the extent that components of luting cements and bacterial toxins can be forced into the pulp. Heat generated during polymerization of resin composite materials may cause hydrodynamic effects (inward fluid movement), while polymerization shrinkage results in permanent stresses in the tooth, accompanied by postoperative sensitivity. The long term consequences of such stresses in teeth are not well understood.

Effects subsequent to restoration:

Microleakage:

The significance of an inadequate marginal seal between the restorative material and cavity wall, permitting bacterial ingress, is well recognized. Investigators have concluded that microleakage rather than material toxicity is the primary factor in an inflammatory pulpal response. The primacy of microleakage in promoting pulpal inflammation unless leakage is extreme and they associated with a defective restoration predisposing to secondary caries. Dentinal permeability decreases with time, and the pulp is capable of mounting a protective response. The severity of pulpal inflammation associated with microleakage also decreases with time and pulpal recovery with hard tissue repair is the most commonly encountered outcome.

Cuspal flexure:

The extent to which cusps are weakened (or reinforced) by restorative procedures can be expressed in terms of cuspal stiffness or cuspal flexure. Cuspal flexure is known to occur during occlusal function, although the extent of flexure tends to be small in the intact tooth. Cavity preparations increase markedly the extent of cuspal flexure under occlusal load. Depending on the type of restorative material used, this increased cuspal flexure may persist following restoration of the tooth. *Lee and Eakle* proposed that the entire tooth flexes during normal occlusal function and this idea serves as the basis for the "abfraction" theory of cervical noncaries lesions. The effects of cuspal or tooth flexure on dentinal fluid flow have not been measured experimentally but are likely to be great, particularly if the tubules are exposed because of cavity preparation. The magnitude of cuspal flexure in response to clinically realistic occlusal loads under experimental conditions (up to 25 µm) is sufficient to result in marginal leakage if the restoration does not protect the cusps.

REACTIONS OF PULP TO VARIOUS RESTORATIVE MATERIALS [*13, 15, 16]:*

Glass ionomer materials [17]:

When glass ionomer cements were first introduced, pulpal responses were classified as bland, moderate, and less irritating than with other cements or composite resins. Glass ionomer fillings were reported to be non-toxic to pulp tissue if bacterial penetration was avoided. Clinical studies shown that cements may cause early inflammatory reactions on newly prepared dentin, which resolve within a few days. Screening tests in cell cultures indicate that glass ionomers can be cytotoxic and therefore, protective calcium hydroxide liners are recommended when working near the pulp and when the thickness of remaining dentin is not certain. Still, it is thought that the high molecular weight of the polymer liquid, as well as other aspects of its composition (e.g., the use of weaker acids and less toxic monomers), help guard against permeation of the material through the dentinal tubules to the pulp.

A more severe pulp response has been reported with the powder-liquid ratios used for the luting cement. Both the proximity of the pulp and treatment of the bacterial layer covering the tooth will affect this response. Pulp response studies of resin-modified glass ionomers have shown conflicting results. In one study, almost no effects were observed in the pulp tissue below resin-modified glass- ionomer fillings and a transient inflammatory response was followed by dentin bridge formation in pulps directly exposed to the material. The overall conclusion was that resin-modified glass ionomers showed acceptable biological behavior toward both exposed and non-exposed pulps. In another study, a resin-modified glass ionomer was compared with calcium hydroxide as the pulp capping material. The resin-modified glass ionomer caused a moderate to intense, persistent inflammatory response in the pulp, together with the formation of a large necrotic zone.

Adhesive restorative resins [18]:

The pulpal effects of these materials can be divided into two scenarios: the immediate effects of cavity preparation and acid etching, and the prolonged effects of leachables from the restorative materials. Acid etch and bonding materials applied to the dentin caused vasodilatation in pulpal tissue and changes in pulp microcirculation may induce nerve signalling responses. The effect was more pronounced after acid etch and bonding than after bonding alone. Placement of dental adhesives on intentionally exposed human pulp

has resulted in inflammation of variable severity. Inflammation was also observed in the pulp after placement of dental adhesives in deep cavities where only a thin dentin wall separated the pulp and the adhesives.

Composites [19]:

Incomplete polymerization is an inert problem with resin based composites and it predisposes the material to degradation and leaching into adjacent tissue. It is important to obtain as complete polymerization as possible through the entire restoration in order to minimize pulpal responses. The level of pulpal responses to composite resins is intensified especially in deep cavity preparations when an incomplete curing of the resin permits an even higher concentration of residual unpolymerized monomer to leach into the pulp.

During the past 20 years, pulp and dentin reactions to composite materials have been related more too bacterial leakage than to the toxicity of the material. Leakage, adverse pulp reactions and the development of recurrent caries are associated with polymerization shrinkage of composites and imperfect adhesive bonding of the material to the tooth cavity. Thermal stress also increases marginal leakage around composite restorations, as does the use of composites with higher viscosity and lower water-sorption values. Although there is less leakage with heat and light-treated composite inlays, the problems associated with marginal gaps have not been solved completely.

Dental amalgam:

If amalgam restoration is done in unlined deep cavities, there may be slight to moderate pulpal inflammation which gradually reduces and diminishes within a week, as formation of reparative dentin takes place under the filling. Concerns regarding toxicity are directly related to systemic effects of mercury rather than to direct effects on dental pulp.

Post operative thermal sensitivity may result after insertion of amalgam in a cavity. Sensitivity is due to expansion and contraction of fluids which is present between amalgam and cavity wall. As a result of heat or fluid movement, nerve fibres of the underlying pulp are stimulated, and thus may give rise to pain. Use of base or cavity varnish seals the dentinal tubules, thus preventing pain. High-copper amalgam, placed in deep cavities of human teeth (RDT 0.15 to 0.5 mm) in conjunction with a zinc oxide eugenol outer seal, showed no inflammation or only slight inflammatory cell infiltration.' Amalgam undergoes corrosion in the mouth, and the accumulation of corrosion products in marginal spaces between the cavity wall and the restoration is considered responsible for a progressive reduction in marginal leakage.

Mercury from amalgam restorations does not penetrate dentin, while zinc and tin ions have been found in high concentrations in dentin beneath amalgam restorations. These metals do not appear to exert an effect on the pulp, although the inflammation accompanying direct placement of amalgam over exposed pulps was tentatively attributed to zinc toxity. Approximately one third of bonded amalgam restorations had pulpal inflammation regardless of the presence or absence of bacterial leakage.

Zinc oxide eugenol:

Zinc oxide eugenol is a temporary filling material that is also used as a liner under other restorative materials. It has been found to be an irritant to the pulp and also considered bland or even therapeutic to the pulp. The type of eugenol used in the mixture may be responsible for some of the irritating effects. Eugenol, a phenol derivative, is known to be toxic, and it is capable of producing thrombosis of blood vessels when applied directly to pulp tissue. Greater the amount of free eugenol present, higher the chances of pulpal irritation. The zinc oxide eugenol placed in direct contact with the pulp tissue result in chronic inflammation and necrosis. The histologic response of a normal pulp to ZOE placed in standard cavity depths is a mild chronic inflammatory cell infiltration.

On the cavity side, the toxicity of eugenol is advantageous as it has antibacterial properties, whereas the much lower concentration reaching across dentin to the pulp may serve as a mild analgesic and anodyne to that tissue. It has also been used as an anodyne for pulpal pain. The sedative effects are apparently due to eugenol's ability to block or reduce nerve impulse activity. It provides a better marginal seal than zinc

phosphate. It is an effective insulating material and prevents galvanic action of the amalgam, thus inhibiting corrosion. Also there is no heat rise during setting.

Zander and Glass(1949) [20] found that ZOE, in direct contact with the pulp tissue, produced chronic inflammation, a lack of calcific barrier and an end result of necrosis.

Zinc phosphate cement:

Zinc phosphate cement can cause severe pulpal damage because of its inherent irritating properties. Phosphoric acid found to cause stasis of red blood cells eventually resulting in autolysis when applied to the capillaries. Toxicity is more pronounced when placed in deep cavities. The pulp may be affected by components of the cement, the heat that is liberated in setting and the marginal leakage that permits the ingress of irritants from saliva. The chemical toxicity of these materials may not completely explain the harmful effects on the pulp, because it has been seen that in germ-free rats no chronic inflammation or focal necrosis is induced but a dentin bridge is formed. These observations indicate that bacterial leakage would be an important factor for inducing pulpal damage around such restorations.

Silicate cements [21]:

The silicate cements are markedly cytotoxic and show severe pulpal reactions. Its adverse effects are mainly because of the prolonged acidity due to phosphoric acid even 24 hours after setting of the cement. In standard depth cavities, the initial response of the pulp to unlined silicates is a noticeable degree of acute inflammatory cell infiltration with disruption of the odontoblastic layer within one or three days after placement. It has been demonstrated that microleakage around the silicate may also be an important factor in inducing pulp response.

Polycarboxylate cements:

They have an excellent biocompatibility with the pulp and are almost equivalent to zinc oxide eugenol cements. Despite the initial acidic nature, 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. The pulpal response to polycarboxylate cements is slight to moderate inflammation at three days and only mild chronic inflammation at five weeks. When these cements are placed directly on pulpal exposure varying degrees of inflammation have been noted. Almost all reports have mentioned mild pulpal reactions with polycarboxylate cements as a base material on dentin or as luting agent. However, pulp necrosis has also been reported.

Gold inlay [21]:

Gold inlays are potentially damaging to the pulp, not because of irritation inherent in the gold itself but several other factors are involved. The first is the thinner mixture of $ZnPO_4$ cement with which inlay is inserted, acts as an irritant due to the acidity and heat resulting from the setting of the cement. The second factor is the large amount of pressure, generated in seating the inlay, brought to bear on the dentinal tubules during cementation injures the odontoblastic layer and is very deleterious to the pulp. A third factor is the predisposition of cast gold restorations to marginal leakage due to poorly adapted margins or excessive use of cement. The end result is the dissolution of the cement, recurrent caries and pulp involvement.

Gold restorations: [22]

The insertion of gold foil may result in pulpal reactions, but these are generally thought to be caused by cavity preparation, dehydration of the cavity, the forces of condensation, thermal conductivity and micro leakage. Pulpal inflammation, destruction of odontoblasts, and hemorrhage were attributable to the toxicity of gold.

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CONCLUSION

Thus, every dentist must be aware of the pulpal morphology and its reactions to various stimuli, so that he or she can use appropriate technique and materials to prevent pulpal injury.

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