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Tooth Hypersensivity after Fixed Partial Prosthesis An enigma – A Review

Dr.Nitu Chaubey (MDS)[1], Dr. Rajul Vivek (MDS,Phd)[2]
***Designation* & *First* Author**[1]- *Senior Lecturer in Department of Conservative Dentistry &Endodontics, Purvanchal institute of Dental Sciences, Gorakhpur*
Designation* & *Corresponding Author[2] - *Reader in Department of Prosthodontics and Crown & Bridge, Purvanchal institute of Dental Sciences, Gorakhpur*

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ABSTRACT –Hypersensitivity after cementation of a crown with glass-ionomer cement is often attributed to an adverse effect on the pulp by the luting agent. Most permanent restorative materials in common use today do not tend to irritate the pulp; the main cause of pulpal damage is infection, the bacteria originating in the smear layer or deep in the dental tubules, inaccessible to caries-excavating procedures. A poorly fitting provisional crown may expose cut dentin to the oral fluids, and mechanical trauma caused by frictional heat during preparation may also damage the pulp.
Key words - Hypersensivity, Pulpal Damage, Crown, Cementation

I. INTRODUCTION

The Dentine and pulp are placed in sequence to arrangement the pulpo-dentine multifaceted. The multifaceted characterizes a rangeamong intratubular dentinal fluid and pulpal fluid.[1] A straightconsequence of this range is the effect of restorative dentistry on the health of the dental pulp, as showed by the pulpal necrosis rate of 1% year for vital crowned teeth.[2,3]Sympathetic the biomechanics of the multifaceted is vital not only in amplification the pulpal necrosis rate, but also in allowingapproaches to be devised in an effort to reduce pulpal damage caused by monotonous restorative dentistry. This review articleconsequently aims to discuss the dynamics of the liquid range and pulpal pressures in relation to forces and pressures of cementation and theperception of dentine sealing.

Construction of dentine

Dentine is poised of almost 50% (v/v) mineral, 30% (v/v) organic matter, and the remainder is fluid. As a living tissue, there are four elements that make up the hydrated composite of mineral and organic matter: (i) dentinal tubules, surrounded by (ii) a peritubular zone, embedded in (iii) an intertubular matrix, and perfused by(iv) dentinal fluid.[4]

Dentine is consequently much like anatomic sponge filled with tubules in connection with the pulpal microvasculature via both the intratubular dentinal fluid and pulpal fluid.

Pulpal end ± the fluid source

At the pulpal end, there is richly vascularized pulpal tissue. The pulpal microvasculature serves as a source to replenish the outward flow of fluid through the exposed tubules. Matthews et al.[5]stated that, presumably, the water lost from the peripheral (enamel) ends of tubules is immediately replaced by movement of water from pulpal blood vessels out into pulpal interstitial fluid and thence into the dentinal tubules. Such replacement maintains a net water content of dentine that probably does not really change. The notion of `dehydrated' dentine as a result of overdriving with an air syringe needs to be reconsidered although the loss of tubule fluid may lead to a delay in refilling the tubule, and thereby challenge the pulpal tissue. The hemodynamicsgoverning such fluid movement in the pulpal microvasculature is discussed below in the section on pulpal pressures.

The intratubular course

Dentinal tubules can be regarded as a semi-permeable biological barrier. Solvent and/or solute movement across the biological barrier involves two mechanisms of permeation: (i) convective transport, and (ii) diffusive transport .[6] Anatomically, the two processes use the same channels for transport. Mechanistically, there are some differences. Which are discussed below.

Convective transport. T

This is a mechanism for solute and solvent transport across dentine; it is also known as bulk fluid movement or fluid filtration. One of the

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earliest pieces of evidence for convective transport came from Fish .[7]The intrapulpal placement of India ink in vital dog teeth led to the peripheral movement of ink particles into and along dentinal tubules in a matter of hours. As the particles moved over long distances in a short time, bulk fluid movement was suggested.

The peripheral end

The enamel end of the exposed tubules represents an air-dentinal fluid interface. However, the interface is initially established at the junction of a pool of dentinal fluid or water coolant lying on top of the wet dentine. As evaporation occurs, the interface is shifted closer to the tubule orifice until it reaches the smear layer. This layer, being very porous to water vapour, may permit air to penetrate it to set up an interface at the tubular orifice.

Two processes are at work at this interface: (i) the expression of capillary forces when an air±liquid interface is established at the orifice of an exposed tubule (the capillary forces can allow sudden outward movement of dentinal fluid); (ii) the evaporation of fluid from dentine, which sustains the outward fluid movement.

Pulpal pressures

Pulpal haemodynamicsThe pulpal microvasculature serves as a fluid source to replenish the outward flow of fluid through the exposed tubules. Such fluid replenishment, which is extracellular to the pulpal capillaries and venules, is regulated by three key mechanisms.

Two opposing sets of Starling forces (1896).

The forces promoting extravascular filtration are the capillary hydrostatic pressure (Pc) and the interstitial tissue osmotic pressure (pi). Both interstitial fluid pressure (IFP) and plasma osmotic pressure (pp) oppose the movement of fluid out of the capillary. The net effect of these forces along an average capillary at transmicrovascular fluid exchange equilibrium.

Hydrostatic buffering.

It is highest at the arteriolar end of the capillaries and lowest at the venular end. Under some conditions, slightly more fluid is filtered than is reabsorbed on the venous side. This net fluid filtration can cause an increased interstitial fluid volume.

Pulpal lymphatics

The preservation of a low colloid osmotic pressure in interstitial fluid is also required to prevent an increasing . In high submission tissues, this is achieved by increasing net filtration for dilution or osmotic buffering.

Interstitial fluid pressure

(IFP) serves as an effective means of monitoring pulpal microcirculatory status the early measuring techniques for IFP were invasive, requiring direct exposure of pulpal tissue.

Crown cementation

A wide range of cementation forces have been used in many studies, ranging from a minimum of 22.5 N[9] to a maximum of 700 N (Moore et al. 1985).[10] With such an extensive range of forces documented, it is not certain what constitutes a relevant force clinically. There seems to be some consensus for 100 N to be used in laboratory studies.[11]An average force of 90 N was obtained over a period of 1 min during which a crown was cemented using a model bite pad. The range of forces measured was between 15 and 230 N. On the other hand, it has been shown more recently that the typical force used for crown cementation was initially 60 N for the first few seconds, followed by a constant force of 20±30 N.[12] Forces used in cementation can generate intracoronal hydraulic pressure. This cementation pressure has been successfully measured in vitro using brass and stainless steel dies.[12] The pressure has been postulated to be sufficient to precipitate pulpal necrosis. Jorgensen (1960)[13] noted that as pressure was exerted on dental cement, filtration of cement constituents into a solid and a less viscous (most reactive) liquid phase occurs. During cementation, the cut dentinal tubules provide a pulpward route for the less viscous cement constituents that are potentially toxic.

The clinical situation is more complex, in that there would be an opposing outflow of fluid from the pulp, although it has been shown that the use of local anaesthetic with adrenaline vasoconstrictor reduces pulpal pressure to very low values. It is plausible that the pulpward movement of potentially toxic constituents would also be facilitated by a pulpward mass transport via the tubules, driven by the cementation pressure pulse. The centrifugal pulpal fluid movement through fluid spaces as a result of evaporative forces can set up disruptive forces. Such forces can cause tissue damage as the fluid streams across small tissue spaces .[5] Similarly, it is plausible to speculate the reverse. The pulpward pressure pulse could cause centripetal movement of pulpal fluid. The shear forces associated with the pressurized fluid movement through pulpal tissue spaces could also result in permanent tissue damage.

Dentine surface treatment

Dentine acts as a buffer zone to obnoxious stimuli external to the pulp. The hydroxyapatite in dentine can buffer the H+ ions of strong acids.[14]The hydroxyapatite in whole dentine is more effective in H+ ion buffering because of the additional effects of calcium phosphate, protein and/or other macromolecular components. Hydroxide (OH) ions are also buffered, but less so than for H+ ions, by displacing the less electronegative phosphate ions from hydroxyapatite . Other factors also offer protection. Remaining dentine thickness (RDT) of 1 mm or more has been shown to be effective in counteracting the toxicity of both zinc phosphate and glass ionomer cements. The positive interstitial pulpal pressure against the walls of the pulp chamber also offers some resistance to pulpward ingress of toxins.

Strategies of dentine surface treatment

There are two aims to dentine surface treatment postoperatively: to modify or remove smear layer in order to improve the quality and quantity of dentine substrate for optimizing adhesive bond strength; and to occlude the dentinal tubules exposed following operative procedures. Restoratively, the smear layer is an unsatisfactory intermediary layer between tooth and restoration. Various strategies have been devised to either modify or remove it, in an attempt to achieve optimal adhesive bond strength.[15]It has also been condemned as a depot of bacteria and toxins when produced under septic conditions. The strategies devised in dealing with the smear layer are (i) modification to produce a resin impregnated smear layer, (ii) partial removal to preserve the smear plugs and create only a limited resin-impregnated dentine layer, and (iii) complete removal and decalcification of the dentine top layer to produce a resin-impregnated hybridlayer. Biologically, the smear layer can be viewed as a useful barrier to external stimuli that are noxious to the pulp. It is responsible for as much as 86% of the total resistance to fluid flow across dentine into the pulp.

Dentine sealing in fixed prosthodontics

The concept of sealing dentinal tubules has also been applied to postoperative sensitivity associated with teeth prepared for crowns. Braennstroem (1996)[16] recommended the use of Tubulicid cleansing agent (Dental Therapeutics AB, Nacka, Sweden) followed by Tubulitec Lining System (Dental Therapeutics AB, Nacka, Sweden). The cleansing agent contains 1% q 1999 Blackwell Science Ltd International Endodontic Journal, 32, 249±256, 1999 Lamb & Wilson Crown cementation and pulpal health 253 sodium fluoride, which precipitates calcium fluoride to reduce dentine permeability. Another precipitation technique uses oxalates.[17] However, the oxalate crystals may interfere with subsequent attempts to bond cements or adhesive resins (Pashley et al. 1993)[5] to the treated surfaces. More recently, dentine bonding agents have been recommended in prophylactically sealing the dentinal tubules of crown-prepared teeth. The aim is to reduce postoperative problems associated with crown-prepared teeth. Various studies on dentine sealing of teeth prepared for crown have been conducted in extracted human teeth using silver nitrate penetration, fluid filtration rates and liquid chromatography.[18]

II. CONCLUSIONS

A direct result of the liquid continuum within the pulpo-dentine complex is the potential for restorative dentistry to affect the health of the dental pulp. Flow of fluid in dentinal tubules has been demonstrated, both in vitro and in vivo, and may be a mechanism for pulpal damage. This review of the literature has produced laboratory evidence that dentine bonding agents can reduce fluid flow through tubules, both prior to direct restorations and during cementation. It is proposed that sealing of dentine before crown cementation would be a useful clinical procedure that may be beneficial and which is unlikely to be harmful.

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