EFFECT OF DIFFERENT DESENSITIZING AGENTS ON LEAKAGE OF CROWNS LUTED WITH TWO ADHESIVE CEMENTS

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Dedication

*To my great **Father**, who was the reason for where I am today, and without his support and encouragement, this work would have not been possible.*

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Introduction

The dentin sensitivity is a well known clinical problem **(*Shillingburg et al., 1981; Richardson et al., 1991)***. Epidemiological researches suggested that 8%-35% of our patients will experience dentin sensitivity ***(Brannstrom et al., 1963)*** after tooth preparation.

It was reported that sensitivity is higher among people in their 30`s &40`s, ranging from 45% to 52%. Researches has reported that after tooth preparation bacteria & it`s toxins can penetrate into the dentinal tubules leading to clinically detectable symptoms ***(Bergenholtz et al., 1982; Weber, 1974; Addy, 2005)***. Also, provisional restorations usually demonstrate microleakage ***(Pashley, 1989)***, thus allow bacterial ingression & fluid movement which will elict pain(***Bergenholtz et al.,1982; Addy,2005)***.

Dentin sensitivity can be defined as ;short , sharp pain arising from exposed dentin surfaces ,due to external chemical ,thermal or osmotic stimuli ***(Shillingburg et al.,1981)***dentin sensitivity may be caused by enamel loss [from attrition ,abrasion , erosion ,or tooth preparation]. The therapeutic goals of dentin sensitivity treatment are either to interrupt pulpal neural response or to block the sensitive mechanism via occlusion of dentinal tubules, using resins, varnishes & dentin bonding agent ***(Pashley, 1984; Langland, 1965)***.

The principal mechanism of dentine hypersensitivity is explained by the hydrodynamic theory of Brännström., which states that a stimulus on

the dentine surface creates a current in the dentinal tubule. A cold stimulus and airflow creates a current directed to the surface of the dentinal tubule, whereas a warm stimulus creates the opposite effect ***(Brännström, 1986).***

He also postulated that this occurred as a result of fluid movement in the dentinal tubules which stimulates odontoblasts & in turn elicited a response by nerve fibers and resulted in pain ***(Weber, 1974; Addy, 2005)***. Usually, exposed dentinal tubules will be closed as a result of reactive sclerosis and deposition of secondary and tertiary dentine ***(Weber, 1974)***.

Reactive sclerosis occurs through hypermineralisation of peritubular dentine and/or the precipitation of minerals from the saliva or fluid within the tubules ***(Addy, 2005)*** Unfortunately, This form of blockage can be removed through tooth brushing, eating and drinking acidic nutrients ***(Pashley, 1989).*** It has been also shown that the dentinal tubules in hypersensitive teeth are eight times more open and two times wider than dentine tubules in control teeth ***(Oyama & Matsumoto, 1991; Absi et al., 1987; Absi et al., 1989) .***

Some luting cements like G.I. may be accompanied by post cementation hypersensitivity, their acidic nature ***(Oyama & Matsumoto, 1991)*** can remove part of the smear layer ***(. Pashley, 1984)***. Due to prolonged low pH of cement ***(Smith &Ruse, 1986)*** during setting and/or hydrostatic pressure that enabled cement to enter dentinal tubules ***(Pashley, 1984; Oyama & Matsumoto, 1991)***. Therefore, desensitizing agents could be used after tooth preparation & before final cementation to reduce sensitivity.

Application of desensitizing agents is gaining popularity, but unfortunately debates about their effect on leakage of crown are prevailing. And so, further studies are needed to clarify their actual effects plus offering the optimum performance

Review of Literature

➢ Dentin Hypersensitivity:

Dentinal hypersensitivity is defined as a sharp, sudden, painful reaction of short duration arising from exposed dentin in response to various stimuli ***(Duran &Sengun, 2004***).Various theories have been cited to explain the mechanism involved in dentinal hypersensitivity, but the hydrodynamic theory is widely accepted. This theory is based on that when the tooth is subjected to temperature changes or physical osmotic changes, displacement of dentinal fluid occurs resulting in deformation of the nerve fibers in the dentinal tubules ***(Patricia, 2005).*** Some luting agents are also accompanied by Post-operative hypersensitivity, to overcome this problem desensitizing agents could be used after tooth preparation & before final cementation to reduce sensitivity. However bonding to desensitized dentin surfaces and it`s effect on leakage is still a matter of controversy between authors.

Dowell & Addy, in 1983, studied the dentine hypersensitivity it`s etiology, symptoms and theories of pain production. Management of the condition tends to be empirical due to the lack of knowledge concerning the mechanism of pain transmission through dentine. The literature reviews indicated that, at most, nerve fibers only penetrate a limited distance along some dentinal tubules. The theories that either the odontoblasts and their processes act as dentinal receptors or the nerves in the pulp are the pain receptors were discussed. Evidence for the stimulation of pulp nerve fibers by a hydrodynamic mechanism was the

most likely mechanism. Nevertheless, whichever theory proves to be correct, occlusion of dentinal tubules would appear an essential request for an effective desensitising agent.

Absi et al., in 1987, discussed the Dentine hypersensitivity they studied the patency of dentinal tubules in sensitive and non-sensitive dentine (*based* on the hydrodynamic theory for stimulus transmission across dentine). Hypersensitive teeth group showed highly significantly increased numbers of tubules per unit area (approximately 8 X) compared with non-sensitive teeth group. Tubule diameters were significantly wider (approximately 2 X) in hypersensitive compared to non-sensitive ones. The number of teeth showing the penetration of methylene blue through the zone of exposed cervical dentine was larger and the depth of penetration was greater in hypersensitive teeth compared to non-sensitive teeth. These results provide further evidence that stimulus transmission across dentine in hypersensitive teeth is mediated by a hydrodynamic mechanism.

Nqassapa DN, in 1996, performed a comparison of functional characteristics of intradental A- and C-nerve fibers in dental pain .The sensory nerve fibres in the pulp consist of myelinated A- and unmyelinated C-fibres which conduct nerve impulses. The A-fibres are larger in diameter and fast conducting, Most of them are in the A-delta group, but also the very fast A-beta fibres has been demonstrated. C-fibres are small and slow conducting. When natural stimuli such as heat, cold, drilling or drying of dentine with air blasts are applied on the tooth, the only sensation perceived is pain. On the other hand perception of pain symptoms in clinical situations varies from sharp and piercing to dull and poorly