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INCIDENCE OF PAIN OR DISCOMFORT FOLLOWING AN OPERATIVE TREATMEN--ETC(U)
AUG 82 P S GROVER, J HOLLINGER, L LORTON

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The incidence of dental pain or perioral pain after one-visit restorative dental procedures can be attributed to many possible origins. The presence or absence of pain in carious teeth probably depends on the rate of development of the cavity and on the extent to which an impermeable barrier is formed on the inner side of the dentin. Once the decay is removed, the cavity is repaired, the barrier is broken and there may be postoperative pain or discomfort. This paper presents a review on the possible sources of postoperative pain after one session of operative treatment.

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Incidence of pain or discomfort following an operative treatment: a review

P.S. Grover, B.D.S., D.M.D.,* J. Hollinger, D.D.S., Ph.D.,** and L. Lorton, D.M.D., M.S.***

U.S. Army Institute of Dental Research, Walter Reed Army Medical Center, Washington, DC 20012

The incidence of dental pain, or perioral pain after one-visit restorative dental procedures can be attributed to many possible origins. The presence or absence of pain in carious teeth probably depends on the rate of development of the cavity and on the extent to which an impermeable barrier is formed on the inner side of the dentin. Once the decay is removed, the cavity is repaired, the barrier is broken and there may be postoperative pain or discomfort. This article will present a review on the possible sources of postoperative discomfort or pain after one session of operative treatment.

REVIEW OF LITERATURE

A normal pain experience showed remarkable variation in different individuals. Chapman & Jones[1] proved that mental fatigue lowered the threshold and age raised it. Pain threshold was not constant for one individual to another or in one person from time to time. However, Sweet[2] considered it virtually impossible to separate the psychological component from the primary awareness of pain. Dworkin and Chen,[3] and Dworkin[4]

*Major, DC, Division of Clinical Operations
**Lieutenant Colonel, Division of Basic Sciences
***Lieutenant Colonel, Division of Clinical Operations

reported that the dental setting environment proved anxiety provoking and associated with a reduced tolerance of pain.

Tooth preparation for a restorative material generally involves both the enamel and the dentin. Enamel is free from the vital processes associated with the transmission of impulses that are interpreted as pain. In contrast to enamel is dentin, with its approximately 30,000 dentinal tubules per square millimeter containing the viable, sensitive protoplasmic extensions of the odontoblasts, called the Tomes' processes. These processes are continuous to the dentino-enamel and cemento-enamel junctions. There are numerous, intricate arborizations with terminal extensions of contiguous processes; therefore, a vast network of interconnected filaments course throughout the dentin and juxtaposed cementum. In addition to Tomes' processes, dentin is endowed with terminal branches of afferent sensory nerves (free nerve endings) that envelope odontoblasts and some of which may penetrate into the predentinal zone.[5]

Improper cooling during the use of high-speed rotary instrumentation can produce heat with subsequent dehydration of moisture from the dentinal tubules. The sensitive Tomes' processes may be aspirated or physically deformed. Such distortion and protoplasmic abuse can injure the processes and also deleteriously affect the afferent free nerve endings. Post-treatment discomfort following the termination of anesthesia would be expected in such circumstances. The hydrodynamic concept of Brännstrom[6] has been proposed to explain the situation previously described. However, it is not merely the excessive dry heat from high-speed cutting burs that can displace fluid from the

odontoblastic tubules. Dehydration of freshly prepared tubules by bursts of air from an air syringe can produce fluid movement that can adversely deform sensitive protoplasmic processes and result in post-treatment pain. Injudicious use of medicaments on freshly cut, vital dentin can be destructive or induce osmotic gradients with fluid displacement that engenders painful sequellae. Excessive pressure from dull cutting instruments (hand or rotary) may compel centripetal fluid movement that mechanically deforms fragile protoplasmic tissue within the tubules. Such procedures may produce painful post-treatment resu'ts.

Avery & Rapp[7] have shown by histochemical methods that cholinesterase is present in the odontoplastic processes throughout their entire length. This evidence supports the concept that the Tomes' processes, in some manner, are instrumental in conveying neuronal impulses from the dental organ to cognitive centers of the central nervous system. While this may be implication by association, it is nevertheless a viable concept because transmission of an impulse (action potential) requires not only a chemical mediator (i.e., acetylcholine), but also an enzyme for de-activating the mediator (i.e., acetylcholinesterase).

It should be obvious that extreme care must be taken by the dental operator to avoid disturbing the micro-environment of the odontoblast and their processes. Direct thermal, mechanical, chemical, or osmotic shock to vital dentinal tissue must also be avoided, because the sequellae of snythesis and release of such agents as histamines, bradykinins, cyclic nucleotides (AMP/CMP), and prostaglindins ($PGE_{1,2}$ & $PGF_{2\alpha}$ series) can, in turn, stimulate afferent free nerve endings and contiguous Tomes' processes.[8-11] The consequence of this stimulation

is pain cognition by the patient after dental treatment.

According to Stanley,[12] approximately 2 mm of sound dentin is required to provide for insulation between a restoration and the pulp. In contrast, Langeland and Langeland[13] maintain that the thickness of remaining dentin is not a primary factor in mitigating against pain following dental treatment. They believe that adverse pulpal reactions can take place even in shallow preparations.

Pain following routine restorative dental treatment can result from an undetected pulpal exposure[14] or from the use of a cavity liner or base.[15] A metal matrix for confining amalgam that is forcibly pressed apically can sever the epithelial attachment, exposing cementum to the oral environment. Such exposure can result in post-treatment pain.[16] Complex and extensive pin amalgam restorations can fracture the crown or root. Ritchey[17] and others described incomplete root fractures and the associated symptoms of discomfort during mastication and sensitivity to cold. Cytotoxic effects of luting agents on vital dentin can be potential causes of pain following the cementation of a fixed prosthesis. Improperly contoured interproximal restorations can predispose to painful food impaction. Plunger cusps may also be an etiologic factor associated with pain, even if the contact areas appear to be sound. Supra occlusion following the insertion of a fixed prosthesis or restorative material is a common cause for pain and discomfort. Prudent occlusal equilibration will more then likely obviate uncomfortable post-treatment sequellae.

Injudicious use of rubber dam clamps and forceful interproximal insertion of wedges can painfully traumatize the sensitive periodontal

ligament. Occasionally, a piece of rubber dam, restorative or impression materials are unknowingly left in the gingival sulcus following treatment. These foreign bodies are potential sources of post-treatment pain. Overly aggressive placement of gingival retraction cord within the sulcus can injury tissue and produce discomfort for the patient. The magnitude of damage further depends upon presence or absence of existing inflammation of the gingival tissue.

Partial or full separation of the epithelial attachment from the tooth is unavoidable during subgingival matrix adaptation, placement of a clamp for rubber dam, and placement of a wedge or tooth separator. Such separation also takes place during curettage and root planing, but the junctional epithelium will become reattached to the tooth surface again within a week, provided it is free from deposits and the gingiva was healthy prior to the procedure, and the injury was minimum.[18]

Voids at the tooth-restoration interface and open margins of a fixed prosthesis are common sources for not only bacterial ingress, but for direct communication between sensitive nerve terminals and Tomes' processes. Oral thermal gradients from hot and cold foods and beverages, and osmotic gradients caused by sugars and salts, predispose to the neuronal transmission of impulses perceived as pain.

Perioral structures are often sites of post-treatment dental pain. The tongue that was anesthesized for an inferior alveolar nerve block during treatment may have been unintentionally traumatized by the dentist or it may have been bitten by the patient. When the anesthesia subsides, the patient is terribly uncomfortable. Soreness at an injection site may be intense and even painful, and difficulty in

opening and closing the mouth may be encountered. Hydrostatic pressure from an anesthetic solution being deposited too rapidly within the tissue can produce such sequellae. An aphthous ulcer may appear at or around the injection site. This pathosis is uncomfortable and potentially painful.[19]

In brief, this review presented some typically encountered painful sequellae from routine restorative dental treatment. The hydrodynamic theory of Brannstrom and a brief description of the importance of Tomes' processes and free nerve endings were mentioned. While this review is not meant to be all inclusive, it serves as a background for a clinical study concerning post-treatment dental pain that will be described in a subsequent issue.

Reprint requests to:

Major P. S. Grover
Research Dental Officer
USAIDR
%LAIR
Presidio of San Francisco, CA 94129

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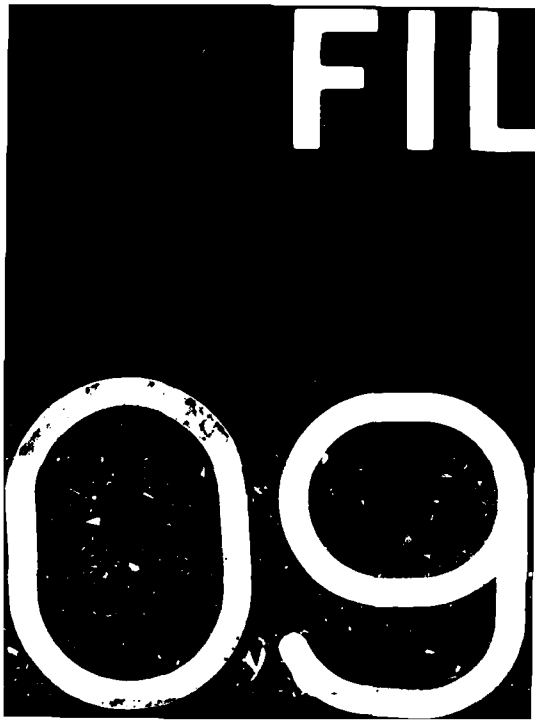
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