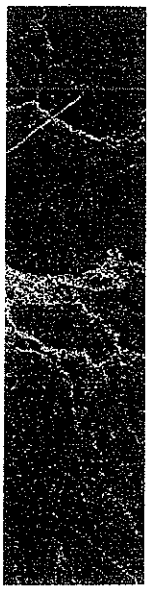

Critical Appraisal

CERVICAL HYPERSENSITIVITY

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Cervical dentin hypersensitivity affects a large proportion of the adult population. This Critical Appraisal reviews an article about the mechanism of dentin hypersensitivity and a series of articles on a novel method for assessing cervical hypersensitivity. The final review presents some interesting new information on a possible relationship between cervical dentin hypersensitivity and occlusal microtrauma.

MECHANISMS OF DENTIN SENSITIVITY

D.H. Pashley
Dental Clinics of North America 1990 (34:449–473)

ABSTRACT

Objective: This article presented a comprehensive review of the dentin sensitivity literature.

Summary: The "hydrodynamic theory" provides an explanation regarding the mechanism of dentin sensitivity. External stimuli cause a rapid outward flow of fluid in the dentinal tubules, activating mechanoreceptors at the pulp-dentin interface, thereby causing pain. Mechanoreceptor responses to stimuli are believed to result in the rapid myelinated A-delta fiber transmission of impulses to cranial pain centers. The flow of dentinal fluid is influenced by the linear dimensions between pulp and external dentin, the configuration of tubules, the tubular diameter, and the number of open tubules.

External dentin tubule stimuli can be tactile, osmotic, thermal, electric, or evaporative. A short-duration air stimulus was deemed evaporative in nature, whereas a longer air stimulus has both evaporative and thermal (cold) components. Pashley recommended standardization of the air blast stimulus for detection of sensitive dentin.

The phenomenon of "neurogenic inflammation" was presented as a condition that intensifies and sustains dentin sensitivity. Neurotransmitters such as substance P, calcitonin gene–related peptide, and neurokinins A and B are released in the pulp during the painful response of dentin sensitivity. These neurotransmitters, both peripherally within the pulp and centrally within cranial pain centers, were theorized to promulgate the reaction of dentin sensitivity in response to external dentin stimuli. Additional study of the role of neurogenic inflammation in dentin sensitivity was suggested.

Conclusions: Cervical dentin hypersensitivity is produced by mechanoreceptor stimulation. Neurogenic inflammation within pulp tissues may lower the threshold of this response.

COMMENTARY

Pashley's article on dentin sensitivity presents a comprehensive review, blending knowledge and a

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theory of the phenomenon. Dentin sensitivity seems to result from external stimulation of mechanoreceptors and is influenced by circulating neurotransmitters. The presence of chronic cervical dentin hypersensitivity (CDH) has been suggested to increase the response of neurogenic inflammation. This effect could be observed both with the pulp of teeth and within cranial pain centers. More study is indicated to clarify these relationships.

CDH occurs following external root exposure to various stimuli of open dentin tubules. Standardized detection and quantification of CDH seems important to evaluate changes of this condition. A finding of CDH is influenced by dentin anatomy, pulp vitality, subjective patient response to the stimulus means, operator skill in isolation of single test teeth, and the nature of the evaluating stimulus.

Pashley discussed various methods for detecting dentin sensitivity. The use of a standardized method of air stimulation to detect and quantify dentin sensitivity was recommended.

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CERVICAL DENTIN HYPERSENSITIVITY. PART I: THE AIR INDEXING METHOD

T.A. Coleman, K.E. Kinderknecht

Quintessence International 2000 (31:461–465)

ABSTRACT

Objective: This paper describes a novel means for detecting and quantifying CDH.

Technique: The "air indexing method" was designed to detect the sensitivity of dentin to air. Room temperature air was directed toward cervical regions of teeth at a 45° angle to the long-axis of teeth from a distance of 0.5 cm for 0.5 to 1 second. A diagnosis of a positive air index was recorded if the patient responded to air stimulation with a minor air stream or with a Fluid Control Block attachment to an air/water syringe. The Fluid Control Block device provides attenuated air emissions from an air/water syringe by selecting one of five volumes of air. A positive threshold patient response to air stimulation (positive air index) allowed an air index mapping of cervical dentin hypersensitivity over time and in response to treatment. Minor air streams at short durations of time were used for the air indexing method to minimize evaporative or temperature mechanoreceptor stimulation of cervical dentin.

Conclusions: The air indexing method offers a convenient and standardized way to detect and quantify CDH. Thus, the effectiveness of treatments for this common oral condition can be monitored over time.

COMMENTARY

Several authors have recognized the use of air stimulation for detecting CDH. The use of air blasts for extended periods introduces both evaporative and thermal stimulation of dentin. The air indexing method offers a convenient and patient-specific way to detect and quantify CDH. The Fluid Control Block allows standardization of air streams to reproduce results for both clinicians and researchers for either treatment or study.

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CERVICAL DENTIN HYPERSENSITIVITY. PART II: ASSOCIATIONS WITH ABFRACTIVE LESIONS

T.A. Coleman, J.O. Grippo, K.E. Kinderknecht

Quintessence International 2000 (31:466–473)

ABSTRACT

Objective: A retrospective study was completed to evaluate associations between CDH and abfractive lesions.

Materials and Methods: Retrospective records for 250 active-care patients treated between 1979 and 1996 were analyzed for the presence of CDH and abfractive lesions. CDH was diagnosed following the detection of a positive threshold patient response to air stimulation by the air indexing method. Groups I (verified positive air index) and II (no verified air index) were formed according to the presence or absence of a verified positive air index over the study period of 17 years. A diagnosis of abfractive lesions was made based on the presence of hard tissue lesions that fit existing literature descriptions of abfraction. Abfractive lesions have been reported as elliptic in enamel and wedge shaped in cervical dentin. A diagnosis of an abfractive lesion was not made when a lesion of dentin was rounded, such as would appear following chronic toothbrush abrasion or generalized among teeth (eg, with erosion). The distribution and location were analyzed for associations between CDH and abfractive lesions.

Results: The mean study periods for groups I and II were 10 and 9 years, respectively. Significant associations were found in group I between the total distribution and location of verified CDH and the presence of abfractive lesions. Comparisons for both group I and II abfractive lesion locations and distributions yielded similar findings. This long-term retrospective study found predominantly buccal locations for both CDH and abfractive lesions in molars and premolars. Seventy-six percent of group I patients and only 39% of group II patients had abfractive lesions. Over three times the risk factor for active bruxism was found in group I than in group II.

Conclusions: This retrospective study revealed a positive association between CDH and abfractive lesions. The results suggest that CDH and abfractive lesions share a common etiology of chronic excessive horizontal functional or parafunctional force. Additional study is indicated to support or refute these correlative study results.

COMMENTARY

The phenomenon of CDH appears to coincide with the microfracture of enamel/dentin associated with chronic abfraction. Neurogenic inflammation in the pulp has been suggested to result in excitation of mechanoreceptors. Further research is indicated to identify whether CDH occurs from neurogenic inflammation or from the event of dentin tubule exposure resulting from microfractures during abfraction.

The associations between CDH and abfractive lesion location, distribution, and frequency of occurrence within group I suggest a common etiology. The similar location and distribution of abfractive lesions for both groups I and II suggest that CDH is not necessarily concurrent with the clinical detection of abfractive lesions. Rather, CDH seems to appear during the early or formative stage of abfraction. Additional study is needed to verify this hypothesis.

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Coleman TA, Grippo JO, Kinderknecht KE. Cervical dentin hypersensitivity. Part III: resolution following occlusal equilibration. Quintessence Int 2003; 34:427–434.

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CERVICAL DENTIN HYPERSENSITIVITY. PART III: RESOLUTION FOLLOWING OCCLUSAL EQUILIBRATION

T.A. Coleman, J.O. Grippo, K.E. Kinderknecht

Quintessence International 2003 (34:427–434)

ABSTRACT

Objective: This retrospective study was designed to determine whether CDH resolves following occlusal equilibration of the involved teeth. The null hypothesis was that the etiology of CDH is not related to excessive functional or parafunctional occlusal stress.

Materials and Methods: Treatment records for 250 randomly selected active patients were analyzed for the presence or absence of verified positive CDH. Patients were assigned to group A ("treatment"), group B ("delayed treatment"), or group C (no verified CDH) for this retrospective study period of 17 years using the air indexing method. Profile comparisons were made between these three groups. Positive air indexed teeth for groups A and B received occlusal equilibration. If teeth were sensitive owing to recent vital pulp exposure or periodontal surgery, they were not equilibrated. Patients also were excluded from occlusal treatment when a restoration lacked a protective base or liner, or when acidic/environmental influences or esophageal reflux were present. These excluded patients were placed in group C, even though a positive threshold patient response to air existed at the cementoenamel junction of teeth. The resolution of CDH following occlusal equilibration to teeth in groups A and B was measured by the loss of a positive air index. Data were analyzed to determine if CDH was resolved by occlusal equilibration.

Results: The resolution of CDH following occlusal equilibration was similar whether this treatment occurred at an average of 7 days (group A) or 92 days (group B) following the initial detection of a positive threshold patient response to air (positive air index). The elimination of CDH in response to occlusal equilibration was found to be long term. Positive associations were found in groups A and B between CDH, hyperfunction, parafunction, and abfractive lesions. Group C patients had fewer abfractive lesions and less parafunction than did patients in groups A and B.

Conclusions: The null hypothesis was rejected because CDH resolved following occlusal equilibration.

COMMENTARY

The results of this study suggest an occlusal equilibration approach to teeth with CDH to reduce flexural stress and to prevent abfractive lesion formation. Cervical dentin hypersensitivity appears to represent an early or active sign/symptom detected for vital teeth undergoing abfraction. Conservative occlusal equilibration should not be performed for teeth diagnosed with CDH without use of the air indexing method, verification of a positive air index, and knowledge of current diagnostic/treatment protocols related to occlusal treatment. Alternatives to occlusal equilibration for teeth with a verified positive air index include establishing a cuspid rise laterotrusion by lengthening cuspid incisals, appliance therapy such as a night guard, or the application of desensitizing dentifrices or professionally applied materials. Although the application of desensitizing dentifrices is an alternative treatment to reduce the presence of CDH, their use may mask this possible sign and symptom of chronic occlusal prematurity. These agents are most useful in the presence of acidic diet, esophageal reflux, or reduced salivary flow.

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Coleman TA, Grippo JO, Kinderknecht KE. Cervical dentin hypersensitivity. Part II: associations with abfractive lesions. Quintessence Int 2000: 31:466–473.

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Editor's Note: We welcome readers' suggestions for topics and contributors to Critical Appraisal. Please address your suggestions to the associate editor:

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THE BOTTOM LINE

CDH results from mechanoreceptor stimulation during the outward flow of tubule fluid. Neurotransmitter release in the pulp during this event may intensify and promulgate the response (neurogenic inflammation). An air indexing method has been developed to detect and quantify CDH in a scientific and user-friendly technique. A long-term retrospective study found a positive association between CDH and abfractive lesion location and distribution. Abfractive lesions are hard tissue defects that develop over time from occlusal microtrauma during hyperfunction and/or parafunction. These defects are further degraded by toothbrush-related abrasion and erosion. Results from the study found the resolution of CDH following occlusal equilibration. The presence of an abfractive lesion did not correspond to the concurrent detection of CDH in this region of a tooth; rather, CDH appeared to be present in the early or active abfractive process.

The use of desensitizing dentifrices or professionally applied tubule blocking agents may mask this symptom of microtrauma to teeth during excessive functional or parafunctional loading force. Occlusal equilibration to teeth diagnosed with CDH may prevent abfractive lesion formation or enlargement. The central effect of neurogenic inflammation in the pulp needs study. These articles suggest studies to determine whether occlusal equilibration of teeth diagnosed with verified CDH reduces bruxism and masticatory muscle tension during periods of microtrauma to teeth. Future studies are needed to determine whether CDH treatment with occlusal equilibration or appliance therapy reduces the potential for cusp fractures and/or signs of periodontal occlusal trauma.

SUMMARY

The clinical detection of CDH may alert the operator to the presence of occlusal microtrauma to these teeth. The preventive nature of early detection and treatment has broad implications for oral health.