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Cervical Dentin Hypersensitivity: Etiology & Current Treatment

THOMAS A. COLEMAN, D.D.S.

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COURSE AUTHOR

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COURSE OBJECTIVES

A significant number of people are affected by CDH, a painful condition that is also somewhat enigmatic. This course addresses the origin of CDH and possible contributing etiologies. It also discusses pain pathways and the evaluative 'air indexing' method for diagnosis. Methods of treatment, including occlusal intervention, desensitizing dentifrices and restoration of CEJ hard tissue lesions, are also addressed.

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Cervical Dentin Hypersensitivity: Etiology & Current Treatment

Studies indicate that the phenomenon of cervical dentin hypersensitivity (CDH), a common chronic dental pain, affects between 4%-57% of our population. It is a pulp response to evaporative, thermal, tactile, osmotic, or chemical stimuli in the CEJ region of a tooth.

Studies have shown that regions of hypersensitive root dentin have more large and non-occluded tubules then non-sensitive regions. The condition of CDH has been found most frequently among 20 to 40-year-old populations involving one or two teeth. The number of patients has been found to decrease rapidly as the number of teeth per patient diagnosed with CDH increases. Studies seem indicated to evaluate the impact of CDH upon teeth, masticatory system function, and CNS response to this type of chronic pain.

THEORIES FOR THE CDH PAIN PATHWAY

Mechanoreceptor nerve endings extend from neural pulp tissue 150-200 microns into dentin tubules. The fluid-filled tubules extend from the pulp to their termination points either at the dentinoenamel junction or external root surfaces. Root surface dentin tubules have been found to be occluded by glycoprotein when exposed to saliva or by cementum for attached subgingival regions. Periodontal proprioceptive fibers attach between cementum and alveolus to detect root position. There is no direct communication between pulp and periodontal fibers. Pulp and periodontal neural stimulation are therefore to be considered as separate events. Dentin sensitivity cannot occur unless vital neural pulp tissue exists. Vital pulp tissue is defined herein as the integrity of neural response with capillary exchange of inflow and outflow of blood.

Brannstrom and Anderson et al introduced the "hydrodynamic theory" to explain the events of dentin sensitivity in the 1960s. According to this theory, mechanoreceptor stimulation occurs during the outward flow of dentin tubule fluids toward the root surface. This negative pressure gradient stimulates the Alpha-delta and C-fiber network to produce the painful response of CDH. This theory is difficult to prove or refute but remains as an accepted explanation for the CDH pain pathway.

The outward flow of dentin tubule fluids requires patent or open tubules to influence negative pressure from external dentin surfaces. This suggests that the smear or pellicle layers occluding tubules are partially absent. Known conditions that interrupt the integrity of these layers are: abfraction (microfracture of dentin, or more rarely, enamel), corrosion (acid), or abrasion (dental handpieces or toothbrush/dentifrice). The implication is that CDH results when the rate of physiologic salivary occlusion of dentin tubules is exceeded by the processes of abfraction, corrosion, or abrasion.

Pashley published a review of "neurogenic inflammation" in 1993. This pulp tissue response to chronic pain involves the release of substance P, neurokinin A (NKA), neurokinin B (NKB), and calcitonin gene-related peptide (CGRP). The presence of neurotransmitters may lower the threshold response of the C-fibers. Although the CDH event appears to be primarily a response of Alpha-delta fibers, the lowered threshold of C-fibers may excite this entire neural network in the pulp.

The CNS effect of neurotransmitter release by the pulp has not yet been studied. However, the central release of serotonin may result. Studies are indicated to determine the CNS effect of neurotransmitter release in the pulp during the chronic pain event of CDH.

DIAGNOSIS OF CDH

Until recently, a diagnosis of CDH was made in a varied manner by both subjective and objective means. Long duration air blasts add an evaporative component to test results. The use of thermal diagnostic protocols have been difficult to control for singular test teeth. Mechanical Yeaple or explorer probes produce unreliable data in the diagnosis of sensitive dentin. The introduction of osmotic reagents or acids as diagnostic tools does not appear to be practical for rapid and/or definitive results.

Subjective patient reports of improvements in the reduction of the painful symptoms of CDH following a treatment regimen cannot be considered as viable data for a number of reasons. The wide range for the incidence rate of CDH among modern populations has been suggested to be related to a weakness of detection methods. Studies demonstrate that patients 20 to 40 years of age have a significantly greater risk of CDH than younger or older populations. A diagnosis of CDH must be reliable, reproducible, cost-effective, not increase existing painful symptoms, and be easily performed by the clinician.

AIR INDEXING METHOD

An "air indexing method" was developed in the late 1970's to detect and quantify CDH. (In 2000, an introduction of the technique was published by Coleman et al.) The method was developed to diagnose CDH in a manner that minimizes thermal or evaporative stimuli to sensitive teeth. A minor puff of air from a standard air/water syringe was directed to the CEJ region at a 45-degree angle to the long axis of a test tooth at a distance of approximately one-half centimeter for a duration of one-half to one second. A "threshold patient response" was recorded as none (0), slight (1), moderate (2), or severe (3) for test zones of teeth. An "air index mapping" was

obtained by patient responses to the air stimulus beginning on the most distal upper right tooth, going toward the upper left, then mandibular left and so on for both buccal and lingual CEJ regions.

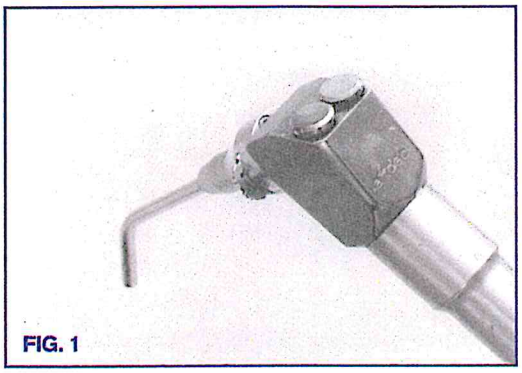


Fig. 1: Fluid control block with dial selections for one of five levels of air emissions.

A "Fluid Control Block" (Fig.1) was developed in 1994 to allow a choice of five distinct volumes of air emissions. This device was suitable for routine use of the standard air/water syringe. The dial selection was adjusted with the thumb to reduced air volumes when the operator desired to evaluate CDH. The Fluid Control Block attachment produced a standardized recording in the volume of the air stimulus whereas the attenuated "minor puff" delivery represented an auditory measure of emissions.

The air indexing method included an initial CDH detection with the air stimulus and a subsequent seven to 10 day verification of results prior to diagnosis. Air responses were evaluated at subsequent seven to 10 day intervals whereas only equivalent or enhanced CDH recordings for teeth were treated. This technique was easily accomplished by the clinician and yielded definitive results.

ORIGIN OF CDH

A long-term (17-year) retrospective study published in 2000 by Coleman et al associated CDH and abfractive lesions. A one-to-one relationship was found between total abfractive lesions and incidents of CDH for both location and distribution among teeth. The locations were found primarily on the buccal surfaces of molars and premolars. Evaluations did not find that CDH existed at all times for the abfractive lesion. Rather, CDH was found during the active or formative stage of abfractive lesion development. A literature review found that a primary etiologic factor for the development of abfractive lesions is chronic excessive horizontal functional or parafunctional forces upon teeth.

A third publication in 2003 of this same study found the resolution of CDH following conservative occlusal equilibration to these respective teeth. Patients diagnosed with CDH were found with a 300% risk factor for parafunctional activity (nocturnal bruxism) than those without. Non-working inclines for working excursions of positive CDH molars and premolars were most frequently equilibrated in this study. Equilibrations that resolved CDH were found to support the tenants of non-deflected CR, cuspid-rise lateral excursions, and anterior protected guidance for protrusion. A conclusion was that CDH represented a "sign" of excessive functional or parafunctional occlusal stress to teeth.

The origin of CDH appears to be multi-factorial. The presence of acidic diet, GERD, bulimia, or environmental acid exposure are cited as contributing etiologies. One pulp expression of irritation from poorly set base or liner could be CDH. Any condition which produces pulp hyperemia may predispose a finding of CDH. Excessive toothbrush-dentrifice abrasion has been suggested to result in CDH symptoms. The absence of adequate tooth brushing with the subsequent acid influence from plaque has also been theorized as an etiology. The verification in findings of CDH at seven to 10 days is suggested as a valuable protocol prior to its diagnosis. Additional study is indicated to determine a more precise model for the primary and secondary etiologies of CDH.

OCCLUSAL TREATMENT

A differential evaluation toward diagnosis seems indicated once CDH has been detected.

- Is the etiology due to abrasion, corrosion, abfraction, or combinations thereof?
- Is a transient pulp hyperemia present?
- Has recent dental treatment been provided that may have altered functional occlusion?
- Has periodontal stability recently changed?
- Are there recent changes in systemic health or medications whose side effect reduced salivary flow?
- Are there dietary events that have increased acid exposure or produced incidental tooth trauma?

A thorough differential evaluation to establish an etiology for the presence of CDH seems indicated to maximize oral or systemic health and masticatory system function.

The treatment of teeth with a verified diagnosis of CDH by occlusal equilibration should not occur unless the clinician has comprehensive knowledge in the field of occlusion. A publication in 1997 by Christensen called for expanded knowledge in the effects of occlusion upon masticatory system function. Programs offered by the Dawson Center for Advanced Dental Study, L.D. Pankey Institute, or other occlusion/TMD programs may enhance knowledge in this arena of treatment.

If a diagnosis has been made which suggests

occlusal intervention for the resolution of CDH, then it is suggested that additional pre-treatment information be reviewed. An accepted starting and end point is a non-deflected CR with the condyle/disc assembly in an antero-superior position within the fossa. Internal joint integrity must exist and the clinician aware of any masticatory muscle symptoms. Specific masticatory muscle symptoms may assist the clinician in a more complete analysis of mandibular function.

- Masseter muscle stress on one side may suggest the inability of the patient to accommodate to a centric occlusal prematurity on that side.
- Bilateral masseter muscle pain to palpation suggests clenching or nocturnal parafunction.
- Unilateral temporalis muscle symptoms imply an excessive attempt by the patient to avoid a lateral incline occlusal deflection.

The interpretation of muscular pain may vary among clinicians, but occluso-muscular information enhances the objective of arriving at a more complete diagnosis. Mounted study casts may assist the clinician to evaluate excursive and protrusive movements of the mandible. A basic decision to treat by appliance therapy, equilibration, orthodontics or other modality may be made once basic occlusal, joint and muscle information has been obtained.

Operator knowledge, skill, patient preference and cost/benefit ratio are considerations in the selection for the direction of treatment. Conservative occlusal equilibration to enamel or restorative may be indicated to resolve CDH only after a complete assessment of information has been made.

DESENSITIZING DENTRIFICE TREATMENT

The use of desensitizing dentrifices to obtund CDH symptoms is suggested for a variety of circumstances. Any non-avoidable reduction in salivary flow and therefore lowered PH i.e. anti-hypertensives or diuretics may produce CDH. Environmental acids that cannot be avoided by the patient may also result in CDH. If the dental professional or patient do not elect equilibration as a treatment for occlusal deflections, then desensitizing methods may be selected to treat this painful symptom. Desensitizing dentrifices offer the patient and operator a choice in the treatment for the sign or symptom of CDH. The application of fluoride, strontium, oxalate, phosphate, carbonate or resin desensitizing formulations are intended to occlude dentin tubules. The action of nitrates is to inhibit neural conductivity.

Hydoroxyapetite is the basic building block for dentin or enamel hard tissues. The action of stannous or sodium fluoride preparations is to replace the calcium cation in the hydroxyapetite molecule with the fluoride ion. This substitution reduces the solubility of hydroxyapetite to acids. This process encourages smear layer production by offering a stable substrate upon which to build the glycoprotein layers that occlude dentin tubules. There is indication from the literature that the presence of plaque may enhance the uptake of fluoride by dentin.

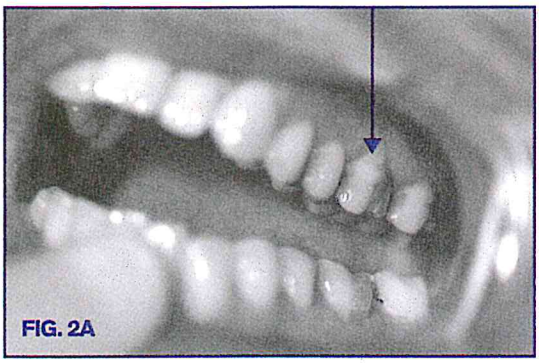
Strontium chloride, potassium oxalate, calcium chloride, potassium phosphate, ferric oxylate, amorphous calcium phosphate, and potassium carbonate react with dentin to produce a crystalline precipitate which occludes dentinal tubules. All of these active ingredients in dentrifice formulations appear to be successful in reducing CDH symptoms for the short-term. Randomized double-blind studies to compare the effectiveness of one active ingredient to another have been difficult to assess due to study design and the historical lack of reliable objective detection of CDH.

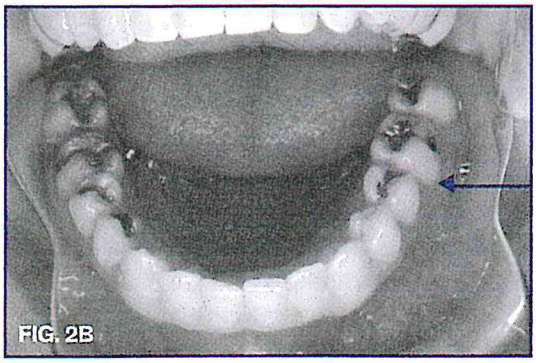
Resin-based dentrifice formulations have an action of occluding tubules. The development of bonding resins had been given much attention in recent years. New product research has produced dentrifices of improved resin/dentin bond strength over the past several years. Resins are less soluble then other dentin tubule occluding agents, but adhesion to root dentin had been an obstacle of long-term success. Microabrasion may enhance the dentin-resin bond by increasing the effective surface area and enhancing the removal of contaminants. Care must be given to avoid gingival bleeding which may interrupt the bond. Gingival irritation may result if these resins are applied to soft tissue.

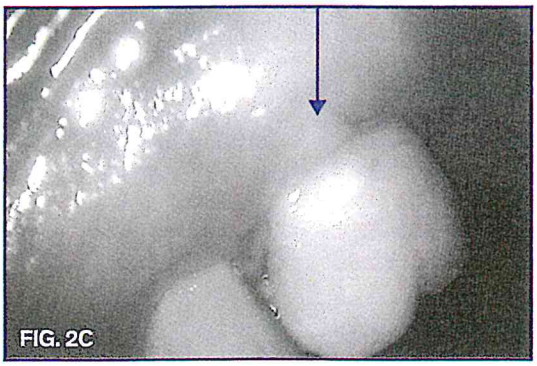
The action of *potassium nitrate dentrifices* is to depolarize neural elements following the penetration of open dentin tubules. This action inhibits neural response to stimuli. A reduction of neural conductivity requires deep penetration of this active ingredient. Occluded tubules do not allow this necessary diffusion. Existing study results are difficult to interpret but potassium nitrate dentrifices are recognized by the profession for the reduction of CDH symptoms.

The choice exits for the operator or patient to choose a dentrifice that occludes tubules or one whose action is to depolarize neural elements at the pulp/dentin interface. It may be important for the operator to recognize this difference in action when making treatment recommendations to the patient for a reduction of CDH symptoms.

Improvement in comparative study design and acceptance of the air indexing method for the detection of CDH may lead to improved studies of dentrifices.







Three stages of abfractive lesions on the mesial buccal root of a first molar. Fig. 2a: immature. Fig. 2b: moderately mature. Fig. 2c: mature.

RESTORATION OF ABFRACTIVE LESIONS WITH RESIN OR GLASS IONOMER

The restoration of Class V abfractive lesions (Figs. 2a, 2b, 2c) with resin or glass ionomer has been favored in recent years over amalgam due to esthetic considerations of these primarily buccal or labial hard tissue defects. Numerous authors have reported premature deterioration of restorations for these lesions. Recommendations have been made by prominent clinicians to provide mechanical undercuts for enhancing the longevity of restoratives. Improvements of dentin bond strength have been made with new generations of products.

The action of resins and glass ionomers is to occlude tubules. Resins offer greater esthetics than do ionomers, but ionomers are less irritating to exposed dentin. Calcium hydroxide bases or glass ionomer liners may reduce the irritation of cervical dentin to resins.

Premature deterioration of the abfractive lesion restoration has been found to begin at the apical extent of the material (Fig. 3). This may be a resultant of continued occlusal stress during the process of abfraction. It is not possible to determine the progressive nature of the abfractive lesion at an initial clinical examination unless CDH is diagnosed. Either time in observation of the lesion or occlusal equilibration to resolve the CDH sign of excessive occlusal stress may assist the clinician in the determination of active abfractive forces upon a tooth. It seems prudent to resolve this question prior to restoration placement.

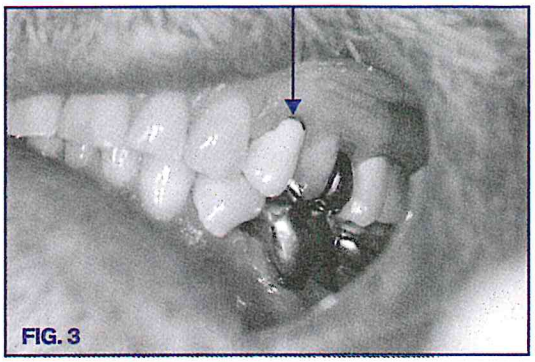


Fig. 3: Premature apical extent deterioration of Class V restoration of premolar. Note that a full coverage crown and root composite exist.

CONCLUSIONS

An objective diagnostic method for CDH appears to be critical in the assessment for this common oral condition. The air indexing method offers the clinician an objective protocol that is specific for the respective patient to detect and quantify CDH.

A primary etiology of CDH appears to chronic occlusal stress. The presence of endogenous or exogenous acids appears to be a significant co-factor in the development of CDH. Toothbrush/dentrifice abrasion or coarse diets may perpetuate CDH symptoms.

The treatment options for the presence of CDH are occlusal equilibration, desensitizing dentrifices, restoration of CEJ hard tissue lesions, or combinations thereof. Further study seems indicated to establish a more precise model for primary and secondary etiologies of CDH, comparisons of desensitizing dentrifices, and oral or systemic health sequellae during this chronic pain event.

Self-Test

1. **A verification of CDH with the air indexing method occurs after ___ days.**
   a. 2-3
   b. 5-7
   c. 1-2
   d. 7-10

2. **The use of ___ is intended to occlude dentin tubules for the treatment of CDH.**
   a. nitrates
   b. glass ionomers
   c. oxylates
   d. b and c
   e. a and b

3. **Neurogenic inflammation involves pulp production of ___.**
   a. acids
   b. neurotransmitters
   c. amylase
   d. prostaglandin

4. **A ___ may produce CDH.**
   a. heart attack
   b. stroke
   c. diabetic coma
   d. hypertensive medication

5. **Abfractive lesions have a primary etiologic factor of ___.**
   a. air
   b. water
   c. occlusal trauma
   d. abrasive foods

6. **The placement of ___ may increase the longevity of Class V restorations.**
   a. a liner
   b. mechanical retention
   c. a base
   d. a desensitizing dentrifice

7. **The air indexing method minimizes the ___ effect of this stimulus for CDH.**
   a. osmotic
   b. evaporative
   c. mechanical
   d. acidic

8. **The microfracture of ___ results in CDH or abfractive lesions.**
   a. dentin
   b. cementum
   c. periodontal fibers
   d. pulp

9. **Alternatives to occlusal equilibration include the use of ___ for the resolution of CDH.**
   a. microabrasion
   b. acidic foods or drinks
   c. appliance therapy
   d. dietary supplements

10. **The etiology of both CDH and abfractive lesions appears to be ___.**
    a. linear
    b. from toothbrushing
    c. multifactorial
    d. from acidic diets

11. **Abfractive lesions occur primarily on the ___ surface of a tooth.**
    a. buccal
    b. lingual
    c. incisal
    d. distal

12. **The presence of CDH is a ___ of excessive occlusal stress to a tooth.**
    a. result
    b. sign
    c. symptom
    d. all of the above

13. **Which active ingredient in dentrifices interrupts neural conductivity?**
    a. nitrates
    b. oxalates
    c. phosphates
    d. resins

14. **Nerve endings may extend from the pulp in to dentin tubules by ___ .**
    a. 150-200 microns
    b. 1500-2000 microns
    c. One millimeter
    d. Two millimeters

15. **The duration of the air stimulus leading to a diagnosis of CDH with the air indexing method is ___ seconds.**
    a. ½ - 1
    b. Several
    c. Less than ½
    d. One to two

16. Both CDH and abfractive lesions seem to result from ___ movements of the mandible.

a. protrusive
b. saggital
c. excursive
d. sudden

17. The event of CDH is best characterized as a ___ pain.

a. spontaneous
b. chronic
c. self-limiting
d. periodontal

18. The resolution of CDH by occlusal equilibration is best provided ___ restoration of an abfractive lesion.

a. before
b. during
c. after

19. Premature loss or deterioration of Class V restorations may be due to ___.

a. gingival bleeding during placement of restoration
b. active abfractive stress upon the tooth
c. contamination by water or saliva during placement
d. all of the above

20. Stimuli that help to diagnose CDH include___.

a. air
b. water
c. explorer
d. a, b, and c
e. b and c

21. The direction of the air stimulus is at a ___ degree angle to the long axis of the tooth.

a. 60
b. 45
c. 90
d. 30

22. Most frequently, ___ teeth have been diagnosed for patients with CDH symptoms.

a. 1 to 2
b. 3 to 4
c. 5 to 6
d. Greater than 6

23. Environmental ___ may cause CDH.

a. pathogens
b. carcinogens
c. ozone
d. acids

24. The air indexing method introduces this stimulus at a distance of ___ for the detection of CDH.

a. 1cm
b. 1mm
c. 5mm
d. 5cm

25. The painful event of CDH is a result of a neurologic signal from ___.

a. the pulp
b. the periodontum
c. cementum
d. all of the above

26. Cervical dentin hypersensitivity most frequently occurs among ___ year-old groups.

a. greater than 50
b. 10 to 20
c. 20 to 40
d. greater than 60

27. The use of ___ may react with dentin to produce a more stable substance for the deposition of salivary glycoproteins.

a. oxylates
b. fluorides
c. nitrates
d. resins

28. A diagnosis of CDH is most frequently found on the ___ surface of a tooth.

a. buccal
b. lingual
c. distal
d. mesial

29. The Fluid Control Block attachment allows ___ adjustments of air volumes.

a. five
b. two
c. one
d. three

30. The event of CDH occurs when dentin tubules are ___.

a. solidified
b. closed
c. covered
d. open

Cervical Dentin Hypersensitivity: Etiology & Current Treatment

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