



Cervical dentin hypersensitivity. Part II: Associations with abfraction lesions

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Objective: The purpose of this retrospective study was to evaluate the association between cervical dentin hypersensitivity and the presence of abfraction lesions. **Method and materials:** Written records and study casts for 250 active-care patients, selected alphabetically, were analyzed for the clinical detection of abfraction lesions and cervical dentin hypersensitivity from 1979 until 1996. Clinical diagnosis of abfraction lesions was made according to existing literature descriptions of these hard tissue lesions. Cervical dentin hypersensitivity was diagnosed when a verified positive threshold patient response was found during tooth evaluation by the air indexing method. Patient groups I and II were formed solely on the basis of the presence or absence, respectively, of a verified positive threshold patient response of cervical dentin to air.

Results: A significant association was found between air-indexed cervical hypersensitivity and the presence of abfraction lesions. The primary locations for both cervical hypersensitivity and abfraction lesions were the buccal surfaces of posterior teeth. **Conclusion:** This long-term retrospective study found a positive association between cervical dentin hypersensitivity and abfraction lesions. The correlative nature of this study suggests the need for further investigation. (*Quintessence Int* 2000;31:466-473)

Key words: abfraction lesion, air indexing method, Fluid Control Block, verified positive threshold patient response

CLINICAL RELEVANCE: Abnormal occlusal relationships may initiate cervical hypersensitivity, which could be a precursor to the formation of abfraction lesions.

In 1991, Grippo¹ introduced the term *abfraction* as a fourth classification of dental hard tissue lesions (Fig 1). This term was used to designate stress-induced lesions that result from hyperfunction and parafunction and can be further exacerbated by erosion-corrosion and toothbrush/dentifrice abrasion.^{2,3} Abfraction lesions develop over time as wedge-shaped hard tissue defects in the cervical region and more rarely as oval or crescent shapes within enamel (Fig 2). The work of numerous researchers⁴⁻¹³ suggests that horizontal loading of teeth results in tension, compression, and torsion stresses at the cervix. These stresses appear to cause a physical or physicochemical loss of cervical tooth

structure and may result in dentin hypersensitivity in these regions due to the progressive exposure of dentinal tubules. The literature contains no studies associating cervical dentin hypersensitivity to abfractions.

Responses to lateral occlusal loading forces on enamel and dentin suggest that resultant stress under hyperfunction or parafunction may promote disruption of apatite crystals in the cervical regions of teeth. Lee and Eakle¹¹ postulated that both tensile and shearing forces contribute to the loss of hydroxyapatite crystals at cervices during active lesion formation. Braem et al¹⁴ noted that the location and distribution of abfraction lesions support this tensile stress-induced biomechanical theory. Goel et al¹⁵ found that enamel and dentin have dissimilar mechanical responses to both compressive and shearing forces at the dentinoenamel junction in response to occlusal loading forces. Xhonga¹⁶ found a high correlation between patients with active bruxism and the development of abfraction lesions. Spranger⁸ found that laterotrusive (working-side) or mediotrusive (non-working-side) loading forces produce flexure that is 10 to 20 times greater than that from vertical loading forces. The abfraction process with active physical breakdown of hydroxyapatite crystals could be further accelerated by acidic fluids,^{2,3} provided that the pellicle glycoprotein layer or cementum is disrupted in the cervical region.¹²

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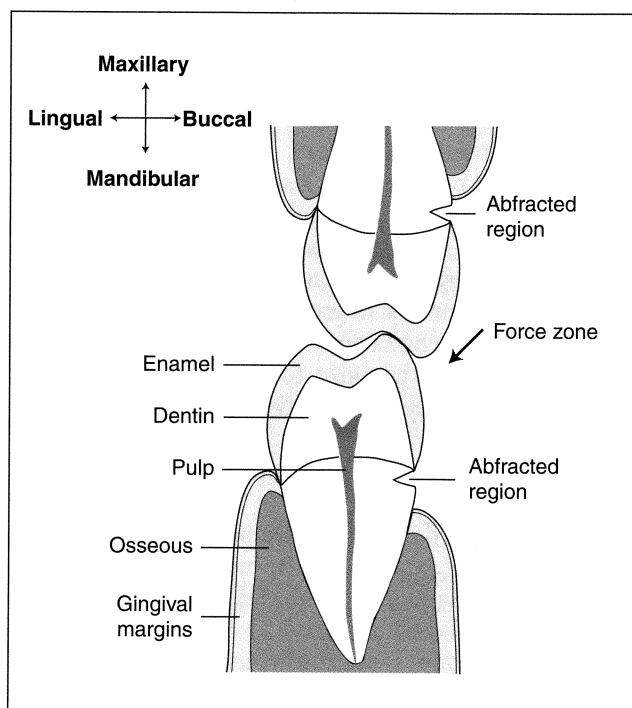


Fig 1 (left) Cross-sectional view of premolars in chronic hyperocclusion.

Fig 2 (below) Mature saucer- and wedge-shaped abfraction lesions in a 73-year-old man. The lesions are seemingly associated with previous Nesbitt wear of the mandibular left second premolar over more than 20 years. Note the light appearance of the actively forming abfraction lesion in the maxillary left lateral incisor, which is subjected to excessive lateroprotrusive occlusal forces, and the darkly stained lesions of adjacent teeth, which are without active excessive functional occlusal forces.



Cervical dentin hypersensitivity may be defined as a dentinopulpal response to air, cold, acid exposure, tactile stimulation, or any combination of these external stimuli.¹⁷⁻²² The hydrodynamic theory, proposed by Gysi²³ and developed by Brännström²⁴ and Anderson et al,²⁵ suggests that tactile, evaporative, thermal, and osmotic stimuli cause outward shifts of dentinal tubule fluids that, in turn, stimulate mechanoreceptors at the pulp-dentin interface. A literature review did not reveal a universal scientific method for determining and quantifying cervical dentin hypersensitivity in teeth, nor their response following various treatment modalities.

As a patient receives oral prophylaxis with rubber cup and pumice, thin layers of cementum and/or glycoprotein pellicle layer may be removed, resulting in exposure of some dentinal tubules. Such patients often experience sensitivity to cold and/or air.^{18,19} Pashley¹⁹ has stated that cervical hypersensitivity reverses after oral prophylaxis care in 3 to 5 days and will subsequently resolve during the next 2 weeks. Pellicle reformation occurs after several days, but acellular cementum does not regenerate. Tavares et al¹⁷ reported that the use of a fluoride-releasing resin seems to alleviate hypersensitivity symptoms by occluding dentinal tubules. Xhonga et al²⁶ found that the rate of abfraction lesion formation in the presence of bruxism does not decrease following topical fluoride application. The literature is devoid of any studies associating cervical hypersensitivity to occlusal hyperfunction or

abfraction lesion formation although these are common and concurrent clinical findings in the general population. A study is indicated to determine the role that excessive occlusal function or parafunction may play in the development of cervical dentin hypersensitivity and abfraction lesions.

Coleman and Kinderknecht²⁷ recently introduced the air indexing method. This technique, first developed in 1979, involved use of a restricted air stream of 0.5- to 1.0-second duration directed toward crevices at a 45-degree angle to the long axis of teeth to detect and quantify a "threshold patient response" to an air stimulus. The air indexing method was provided to general dental patients at ambient temperatures of 18°C to 25°C. The "minor puff" of air from the standard air-water syringe used at that time was not easily quantified, because the sound of air emission was the determinant for the amount of air.

In 1994, a Fluid Control Block^{28,29} was developed; it offered 5 distinct volumes or pressures for use with the air indexing method. This device and method generated comparative threshold patient responses that are useful for evaluating the changes in dentin sensitivity resulting from treatment or by observation over a period of time.²⁷ Furthermore, the method was developed in an attempt to detect cervical dentin hypersensitivity and to minimize the pulpal response from evaporation produced by a longer-term air blast, as has been reported in the literature.¹⁷

The purpose of this retrospective study was to evaluate the association between cervical dentin hypersensitivity and the presence of abfraction lesions.

METHOD AND MATERIALS

Retrospective database

A base of 250 active-care patients older than 20 years of age was selected alphabetically from a general dental practice. Active care was defined as compliance with preventive visits and completion of recommended routine oral health care. The data for this retrospective analysis excluded patients receiving less than 1 year of treatment, those lacking the completion of regular care, and those whose last treatment date was prior to 1994. Retrospective analysis included examination of written records and study casts from 1979 to 1996 for all 250 patients who satisfied the aforementioned criteria.

Cervical dentin hypersensitivity and abfraction lesion detection

Abfraction lesions were identified in this study as angular notches appearing in the cervical regions or as ovate or crescent-shaped lesions in the enamel.^{1,2,11-15,27} Detection was made visually and by the use of an explorer.

Cervical dentin hypersensitivity was detected by patient response to the air indexing method.²⁷ From 1979 until 1993, a minor air stream from an air-water syringe served as the stimulus for initiating patient responses in the cervical regions. The Fluid Control Block was introduced after 1993 to regulate and quantify the amounts of air delivered (see Fig 1 of Part I).^{28,29} Air delivered for 0.5 to 1.0 second at a distance of approximately 5 mm from the cervixes of teeth was used to produce a single-tooth threshold patient response.²⁷

Except for emergency care, no treatment was rendered for 7 to 10 days after the recording of a positive threshold patient response to the air stimulus. From 1979 to 1996, all patients were encouraged not to use desensitizing dentifrices except for fluoride toothpastes. Verification of a positive threshold patient response was established 7 to 10 days following the initial detection of cervical dentin hypersensitivity. The data were recorded during regularly scheduled appointments for general dental care.

Groupings

Patient information gained by analysis of records dated 1979 to 1996 was assigned to 1 of 2 groups.

Patients were assigned to group I if treatment records indicated a verified positive patient response to air for 1 or more teeth. Group II included those patients without a positive or verified response to air during this 17-year treatment period. Patients with a history of recent oral prophylaxis (less than 7 days), masticatory tooth trauma, periodontal surgery (less than 14 days), or tooth extraction (less than 7 days) in a zone of cervical air hypersensitivity were assigned to group II. Patient assignment to group I or II was made based only on the presence or absence of a verified positive threshold patient response to air, irrespective of the presence or absence of abfraction lesions.

Data collection

Analysis of records included the patient age, the Angle classification of occlusion, the number of treatment years, and the number of teeth. Data were analyzed for the presence of verified cervical hypersensitivity and abfraction lesions, resulting in assignment to either group I or II. The diagnosis of active bruxism was based on the patient's report of symptoms of occlusal or muscular pain on awakening in the absence of sinusitis, signs of occlusal wear facets, or by report from a household member. Data included palpable detection of morning tenderness in elevator muscles of mastication that diminished during nonsleeping hours.

Retrospective data were processed on a Pentium computer using Microsoft Excel 5.0 in Windows 95.

RESULTS

Table 1 presents patient age, Angle's classification, average number of teeth, signs and symptoms of active bruxism, and years in treatment for both groups I and II. Routine clinical visits over the years of general dental care found 149 patients (group II) without verified cervical hypersensitivity and 101 patients (group I) with verified air-indexed cervical sensitivity.

The average time in treatment for group I was 10 years, compared to 9 years for group II. Age characteristics at the initiation of care were similar in groups I and II. Comparison of Angle's classification revealed greater distribution of Class II, division 2, patients in group I, whereas group II contained more Class I individuals. The average number of teeth present in groups I and II were 26 and 24, respectively. Although not shown in Table 1, the distribution of age found more 60- to 69-year-old patients in group II than in group I. In group II, the average number of teeth in patients in this age range was 19.3. The comparisons in Table 1 are intended to provide baseline information for patient samples in groups I and II.

TABLE 1 Comparisons between patients with verified cervical dentin hypersensitivity (group I) and patients with no verified cervical dentin hypersensitivity (group II)

	Group I			Group II		
	All	Female	Male	All	Female	Male
No. of participants	101	55	46	149	77	72
Average years in treatment	10	10	11	9	9	9
Average no. of teeth	26	26	26	24	24	24
Age at beginning of dental care						
Mean (y)	38	36	42	40	39	42
Median (y)	35	33	39	38	35	41
SD (y)	12	11	12	17	19	15
Maximum (y)	73	73	70	82	82	73
Minimum (y)	20	20	21	7	7	10
Occlusal classification (Angle)						
Occlusal Class I (%)	54	23	32	66	30	36
Occlusal Class II, div 1 (%)	6	5	1	5	5	0
Occlusal Class II, div 2 (%)	35	25	10	17	13	3
Occlusal Class III (%)	5	2	3	11	3	9
Patients with abfraction lesions						
No.	77	37	40	58	28	30
%	76	37	40	39	19	20
Patients with signs/symptoms of bruxism (%)						
	50	30	20	15	8	7

All percentages are rounded to the nearest percent.

Analysis revealed that 76% of patients in group I were found to have abfraction lesions whereas only 39% in group II were identified with this type of hard tissue lesion. Data were not collected to identify the ratio of wedge-shaped dentin lesions to coronal enamel lesions, because it was noted that ovate and crescent-shaped coronal lesions were rarely detected in either group. Fifty percent of the patients in group I were found to have active episodes of bruxism during the years of treatment, whereas only 15% of the patients in group II had such episodes.

Figures 3a and 3b compare the incidence of abfraction lesions to age for groups I and II. The number of lesions detected per person was greater in group I than in group II. The group II abfraction lesion peak was found among 60- to 69-year-old patients, who had the fewest teeth present for any age range of either group. The presence of abfraction lesions among 20- to 29-year-old patients was strongly associated to group I (patients with verified cervical threshold air sensitivity). The group I results reveal that a verified positive threshold patient response and the association with an abfraction lesion occurs primarily in patients younger than 50 years.

Figure 4 illustrates the distribution of teeth found with a positive verified cervical threshold patient response to air for group I patients. The data reveal a predominant buccal location of cervical hypersensitivity. The incidence of cervical hypersensitivity was greatest in molars (65%) and premolars (34%) (Table 2).

Figure 5 compares the distribution of abfraction lesions by tooth and surface location for molars and premolars in groups I and II. This reveals a predominance of buccal abfraction lesions for both groups. Table 3 shows distribution of abfraction lesions by tooth type for both groups. Unlike cervical hypersensitivity, abfraction lesions in group I were more evenly distributed at the cervices of premolars (42%) and molars (47%). Patients in group II experienced 10% more abfraction lesions in anterior teeth than did those in group I. Although not reflected in these 2 illustrations, the more aged patients in both groups were found to have a greater number of abfraction lesions in anterior teeth.

Figure 6 plots positive verified responses to air, by tooth number, to abfraction lesions detected in group I patients. The nearly coincident lines suggest an association between cervical hypersensitivity and abfraction lesions.

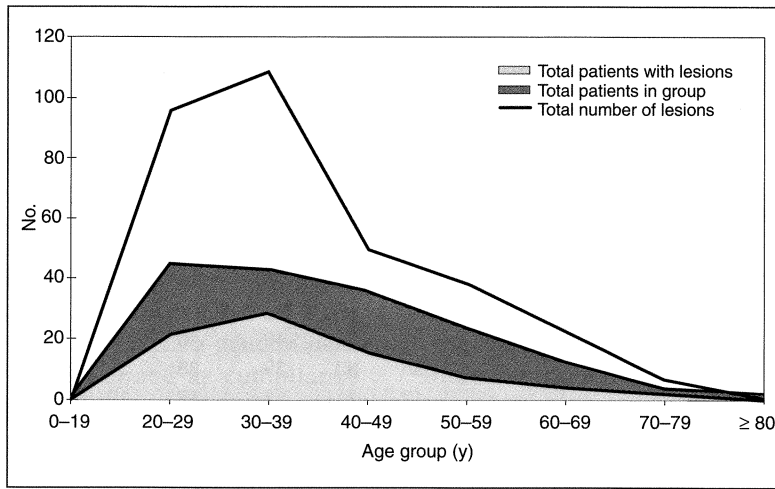


Fig 3a Group I. Distribution of abfraction lesions by age (101 patients).

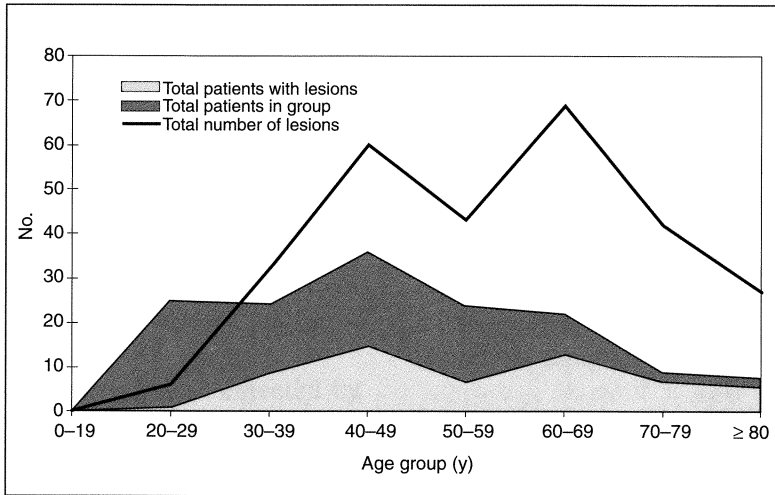


Fig 3b Group II. Distribution of abfraction lesions by age (149 patients).

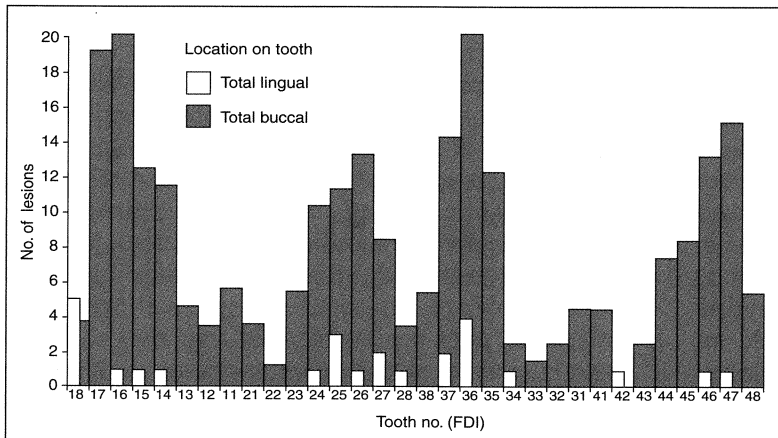


Fig 4 Group I. Locations of cervical dentin hypersensitivity.

TABLE 2 Locations of cervical dentin hypersensitivity

Teeth	%
Anterior	
Lingual and buccal	0
Molars	
Lingual only	6
Buccal only	57
Lingual and buccal	2
Premolars	
Lingual only	2
Buccal only	31
Lingual and buccal	1

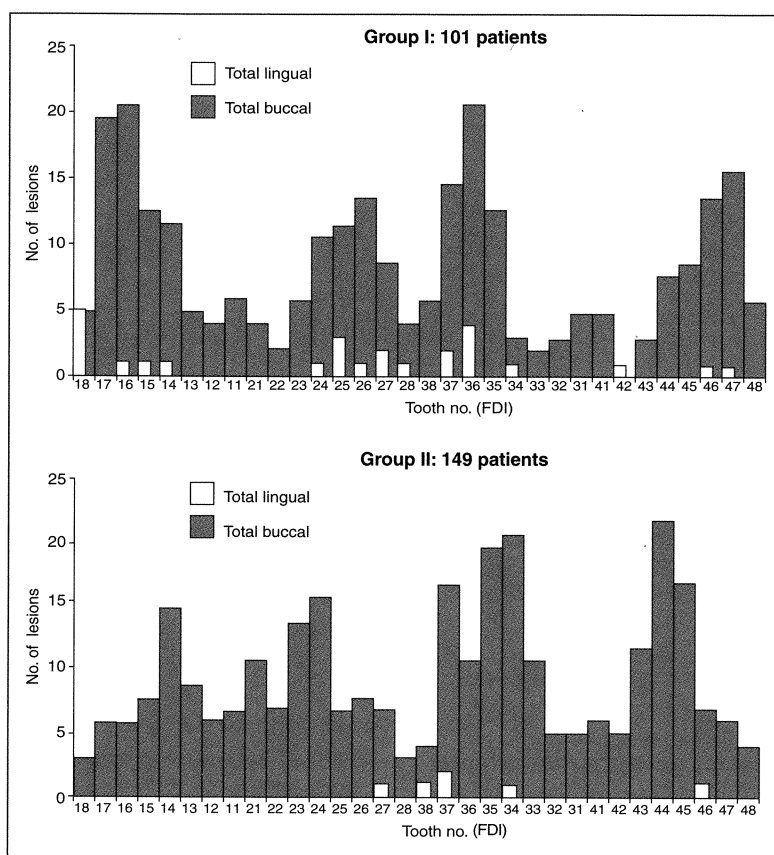


Fig 5 Distribution of abfraction lesions by location.

TABLE 3 Distribution of abfraction lesions by tooth type			
Teeth	%	Teeth	%
<i>Group I</i>		<i>Group II</i>	
Anterior		Anterior	
Buccal only	21	Buccal only	31
Lingual and buccal	0	Lingual and buccal	0
Premolars		Premolars	
Lingual only	0	Lingual only	0
Buccal only	42	Buccal only	42
Lingual and buccal	0		
Molars		Molars	
Lingual only	15	Lingual only	2
Buccal only	32	Buccal only	25
Lingual and buccal	0	Lingual and buccal	0

DISCUSSION

The data revealed associations in the presence of verified cervical dentin hypersensitivity, abfraction lesions, and signs/symptoms of active bruxism. Abfraction lesion formation has been associated with excessive functional or parafunctional forces. This long-term retrospective study found a 76% association of group I

abfraction lesions to teeth with positive verified threshold patient responses to the air stimulus. Further study is encouraged to corroborate these correlations and to firmly establish the relationship between cervical dentin hypersensitivity and abfraction lesions. In addition, the associations of abfraction lesions, cervical dentin hypersensitivity, and bruxism warrant clinical study related to the efficacy of occlusal therapy.

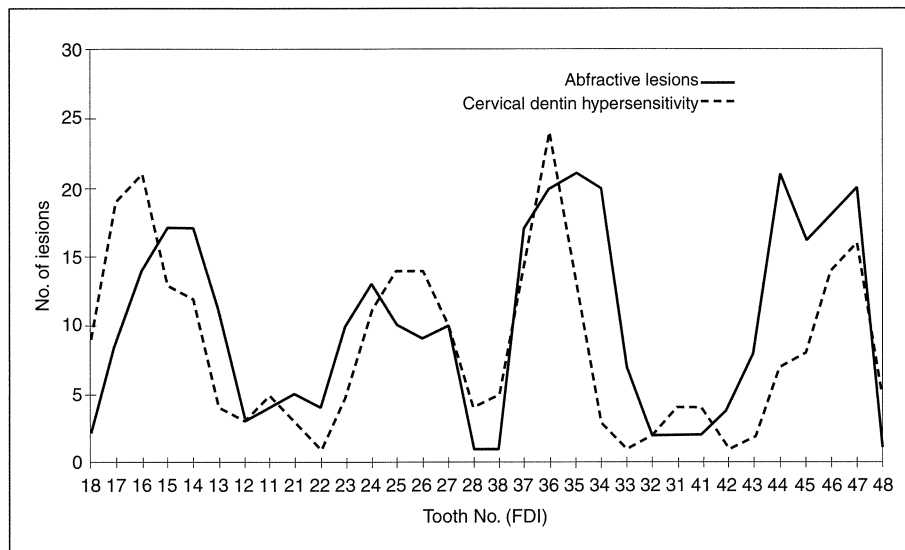


Fig 6 Group I. Association between total abfraction lesions and cervical dentin hypersensitivity by tooth.

The more aged patients in group II had greater numbers of abfraction lesions per person (see Fig 3b). Cervical dentin hypersensitivity was never detected in any endodontically treated teeth for group I or group II during this 9-year study. Abfraction lesions in older adults may not correlate well with cervical dentin hypersensitivity, because such individuals have reduced dentin permeability³⁰ and receded pulpal tissues. Forty-seven percent of 20- to 29-year-old patients with a verified positive threshold patient response to air had associated abfraction lesions in group I. In this age range in group II, without cervical dentin hypersensitivity, 1 abfraction lesion per 25 patients was found. Verified cervical dentin hypersensitivity detected by patients younger than 50 years correlated well with the presence of abfraction lesions (see Fig 3a). Patients in this age range who were found to have abfraction lesions exhibited cervical dentin hypersensitivity less frequently as age increased. Data interpretation suggest that cervical dentin hypersensitivity may be an indicator for active hyperfunction or parafunction (see Fig 4 and Table 1).

Figure 5 reveals that cervical dentin hypersensitivity was found predominantly on the buccal surfaces of molar and premolar teeth. In group I, abfraction lesions were also detected primarily on the buccal surfaces of the premolars and molars. Furthermore, Figs 5 and 6 show similar relationships between cervical dentin hypersensitivity and abfractions in both maxillary and mandibular posterior teeth.

The greatest incidence of cervical dentin hypersensitivity was found in molars, whereas abfraction lesions

were found more evenly in premolars and molars (see Tables 2 and 3). We hypothesize that the larger crowns of the molars may reduce flexure and resultant formation of abfractions.

Buccal distribution of abfraction lesions and positive verified threshold patient responses to air seemed to confirm that excessive masticatory or parafunctional stresses may be greater for working than nonworking excursions of the mandible during function (see Figs 4 and 5, Tables 2 and 3). Lucas and Spranger's *in vitro* study⁶ of flexural reactions in the cervical region demonstrated that horizontal forces on teeth produce greater stress than do vertical forces. Analysis of mandibular motion during mastication and contraction force generated by elevator muscles during working excursions may explain the dynamics of buccal locations and abfraction lesions/cervical dentin hypersensitivity.

The correlative data suggested that the teeth in group I patients with abfraction lesions also had concurrent air sensitivity (Fig 6). Cervical dentin hypersensitivity is not always present in an abfraction lesion, because the sensitivity may wax or wane over time. A verified positive threshold patient response appears to be related to excessive functional or eccentric loading and periods of active bruxism. The initiation and progression of cervical dentin hypersensitivity is also dependent on variables such as diet, masticatory accommodation patterns, and iatrogenic changes of occlusal contacts. Changes in the dynamics of functional or parafunctional occlusal forces would consequently vary resultant stresses to teeth over time, thus

altering the degree of hypersensitivity. The presence of hypersensitivity may be a precursor to or an active period in the formation of abfraction lesions. The air indexing method could generate information evaluating the effect of occlusal therapy on cervical dentin hypersensitivity. Part III of this article will present data relative to occlusal therapy and its effects on the resolution of cervical dentin hypersensitivity.

CONCLUSION

The results of this long-term retrospective study of 250 patients disclosed positive associations between cervical dentin hypersensitivity, as detected by a verified positive threshold patient response to a stimulus of air and abfraction lesions. Because the literature now supports the role of occlusal hyperfunction, eccentric loading, and parafunction as cofactors in the etiology of abfractions, these activities could also be etiologic factors in the development of cervical dentin hypersensitivity. The correlative nature of this study does not offer proof but indicates that a relationship between the findings does exist, both related to the dynamics of excessive occlusal loading. Further study is encouraged to corroborate these findings and to confirm the conclusion.

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