Title
Does bruxism contribute to root canal treatment?

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Publication Date
2012

Peer reviewed|Thesis/dissertation

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by

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THESIS

Submitted in partial satisfaction of the requirements for the degree of

MASTER OF SCIENCE

in

Oral and Craniofacial Sciences

in the

GRADUATE DIVISION

of the

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

ACKNOWLEDGEMENT

I would like to thank my mentors Dr. Tri Huynh, Dr. Susan Hyde, Dr. Art Miller, Dr. Charles McNeill, and Dr. Jing Cheng for their guidance and support for my research project.

Sandy Wang

Does Bruxism Contribute to Root Canal Treatment?

By Sandy Wang, DDS

Introduction: Bruxism is an oral motor disorder characterized by awake or sleep-related grinding and/or clenching of the teeth, and, tooth wear is its most evident effect. Currently, there have not been any studies investigating the relationship between bruxism and root canal treatment. The aim of this study was to determine whether bruxism is a risk factor for root canal treatment**.**

Methods: This case-control study included 3240 patients, aged 25-85 years, treated at the University of California San Francisco (UCSF) School of Dentistry between 2005-2011, with and without clinical evidence of root canal treatment as documented in the AXIUM patient database. A logistic regression model was used to assess potential risk factors associated with having root canal treatment, including bruxism, caries risk, age and gender.

Results: After adjusting for age, gender, and caries risk, bruxism was associated with higher odds of having root canal treatment, odds ratio (OR): 1.30, 95% confidence interval (CI): 1.10-1.54. Females were more likely to have had root

canal treatment than males, OR: 1.19, 95% CI (1.01-1.41), and each year of increasing age increased the odds of having root canal treatment, OR:1.04 (1.03-1.04). Patients with moderate caries risk had higher odds of having root canal treatment than those with low caries risk, OR: 1.434 (1.21-1.70).

Conclusion: Bruxism is a movement disorder characterized by grinding and clenching of teeth that can cause cracks and fractures of teeth, which can lead to pulpal injury. There appears to be a significant association between bruxism and root canal treatment.

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Introduction

Bruxism is a movement disorder of the masticatory system that is characterized by teeth grinding and clenching which can pose a threat to the integrity of the oral dentition (1). Bruxism has a prevalence of 10% and has been regarded as one of the possible causative factors for temporomandibular joint pain, and tooth wear in the form of attrition and loss of teeth (2). Bruxism most often occurs during sleep although it may also occur while awake (3, 4), and it can be classified accordingly: awake bruxism, sleep bruxism, and combined bruxism (5).

Bruxism can be suspected when there is clinical evidence of wear facets that cannot be interpreted as the result of the masticatory function. The American Academy of Orofacial Pain (AAOP) has defined bruxism as diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth (10). A more operational definition, offered by the American Sleep Disorders Association (ASDA), refers to bruxism as a periodic, stereotyped

movement disorder of the masticatory system that involves tooth grinding or clenching during sleep (11). Typical signs of occlusal trauma are chipping and fracturing of teeth and prostheses, pain in the affected muscles and joints, and teeth sensitivity (12, 13). Bruxism can cause occlusal trauma and also lead to pulpal necrosis. Sleep bruxism produces 220N or 30% of maximum vertical bite force and clenching produces 35-100N or 5-11% maximum vertical bite force (6-9).

The following 3 cases are described to help illustrate the effects of bruxism. A 49 year- old male presented with a chief complaint of sensitivity on #25 with no reported history of trauma. Radiographs revealed extensive root resorption of #26 and radiolucency around the roots of #25 and 26. On clinical examination, the mandibular anterior teeth showed signs of incisal wear consistent with bruxism. Examination and diagnostic testing concluded a diagnosis of pulpal necrosis on #25 (Figure 1). A 75 year- old male presented with a chief complaint of “my teeth are sensitive.” A radiograph revealed radiolucency around the apex of #25, and examination and diagnostic testing confirmed the

diagnosis of pulpal necrosis #25 (Figure 2). A 55 year-old male presented with a chief complaint of swelling in the lower right quadrant (Figure 3). He reported a history of bruxism and did not wear an occlusal guard as had been recommended by his general dentist. A sinus tract was present and traced as seen in Figure 3. Occlusal wear was evident on clinical examination, and diagnostic testing confirmed the diagnosis of pulpal necrosis #30. The tooth was not restorable due to an occlusal fracture to the furcation.

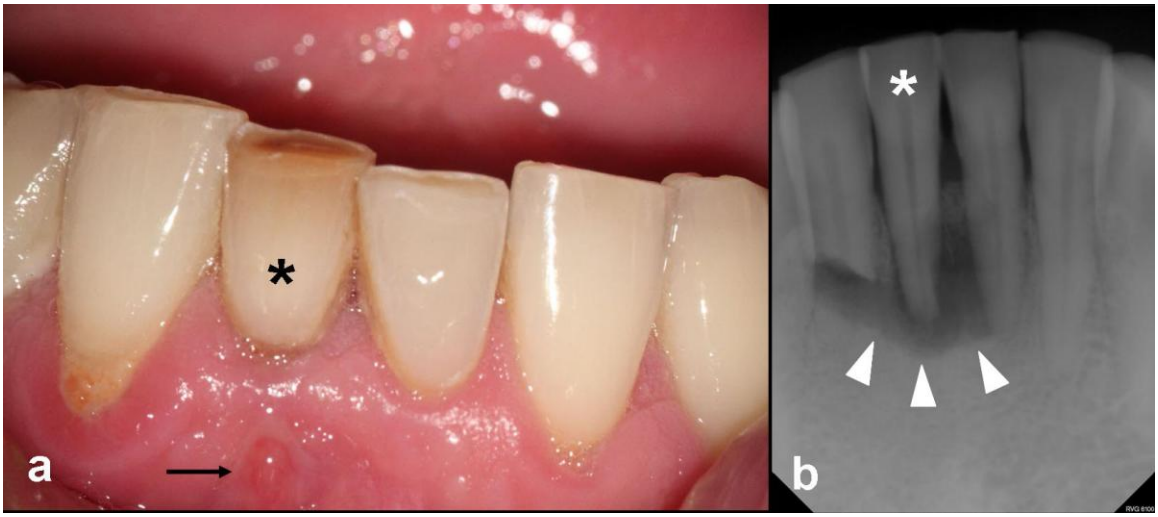

Figure 1. a) A 49-year-old male presented with discoloration, sinus tract (arrow) and signs of incisal wear. b) The radiograph revealed extensive root resorption of #26 and radiolucency (arrows) around apex of #25, 26.

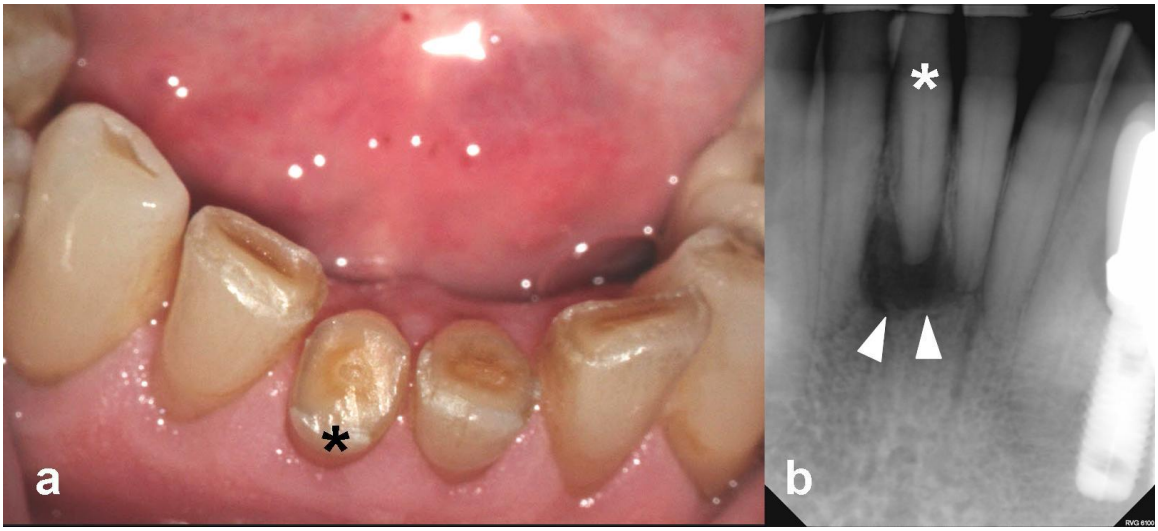

Figure 2. a) A 75 year-old male with a sign of severe incisal wear of #25. b) The radiograph revealed a radiolucency around apex of #25.

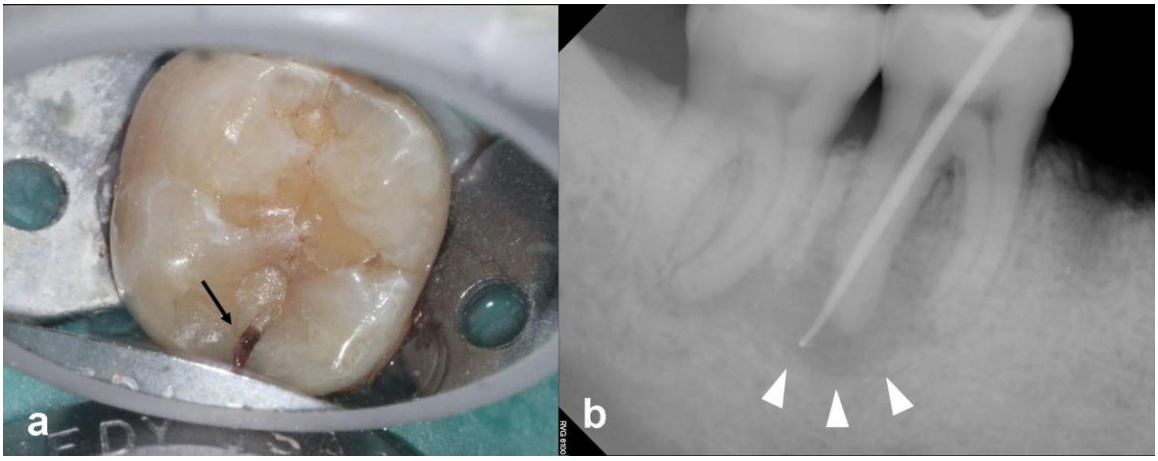

Figure 3. a) A 55-year-old male with mesial to distal occlusal fracture of tooth #31. B) The radiograph of the sinus tract traced and radiolucency around distal root of #31.

The dental pulp is densely innervated by nociceptive sensory neurons and pain is the dominant sensory modality elicited by stimulation of the dental pulp (14, 15). In the absence of any pathology, pulpal nociceptors are usually protected from mechanical stimuli by the hard tissues that surround them, the dentin and enamel. However with excessive sustained parafunction, such as found with bruxism, the nociceptors become activated by various stimuli. Currently, to the best of our knowledge, there have not been any studies looking at the association between bruxism and root canal treatment. However, there are studies that have investigated cracks and restorations and their association with pulpal pathology (16, 17). In reversible pulpitis the pulpal tissue is inflamed and removal of the stimulus will reverse the inflammation. Therefore the mechanisms mediating the pain symptoms in this clinical condition are likely primarily inflammatory in nature. In irreversible pulpitis, the inflammation has proceeded to the point of pulpal tissue degeneration, where the inflammatory process cannot be reversed. Abbott found that a crack or fissure involving dentin is a cause of pulp disease provided there are sufficient bacteria in the cracks (18). Krell and Rivera found 20% of patients diagnosed with reversible pulpitis from cracked teeth possibly related to

bruxism required root canal therapy within 6 months after the initial diagnosis (16).

The underlying hypothesis of this proposal is that patients with a history of bruxism are at higher risk for root canal treatment. The purpose of this case-control study is to evaluate the association of bruxism with root canal treatment in patients attending the UCSF School of Dentistry clinics.

Material and Methods

Subjects

The AXIUM database was searched between the years of 2005-2011 for cases who had a history of or received root canal treatment, and controls without root canal treatments, randomly selected. Case subjects had a history of root canal treatment or received root canal treatment at the UCSF School of Dentistry clinic during 2005-2011. Control subjects were those that had no history of nor received root canal treatment during the same time frame. The endodontic treatment status was confirmed in two ways: 1) the dental history form asking

whether the patient has had any history of endodontic treatment; and 2) odontogram charting of endodontic treatment as reported in the AXIUM (Las Vegas, NV) electronic dental records at the UCSF School of Dentistry. Male or female patients, aged 25-85 years, dentate, with moderate or low caries risk were included in the study. Those with a high or unrecorded caries risk, a history of TMJ disorders, a history of psychological disorders, and edentulous patients were excluded. Patients with a high caries risk were excluded from the study to minimize the potential confounder of root canal treatment arising from extensive caries. Patients with a history of TMJ disorders were excluded due to some controversy correlating bruxism and TMJ disorders and these patients may also have a psychological component (19). Bruxism can be induced by some psychological medications. Hence, patients with a history of psychiatric disorders were excluded from the study. Bruxism was measured by clinical signs only and self-report was not included due to the potential inaccuracy of that information. There were 1486 cases (53.8%) who had received root canal treatment and 1277 controls (46.2%) without root canal treatment.

Study Design

This retrospective chart review of patients with and without root canal treatment was approved by the Institutional Review Board of the University of California, San Francisco.

Data Collection/ Statistical Analysis

Data for bruxism was collected for patients with clinical signs of bruxism as reported by the clinician. Assuming that the proportion of bruxism is 30%, a total sample size of 2884 (1442/group) would have a 80% power to detect an odds ratio of 1.25 with Type I error of 0.05. Means, standard deviations and percentages were used to summarize the data for cases and controls. A logistic regression model was applied to assess the association of bruxism with having root canal treatment while controlling for age, gender and caries risk. Associations were considered statistically significant at $p< 0.05$. Odds ratios (OR) and 95% confidence intervals (95% CI) are presented. All analyses were performed using SPSS software version 20.0.0.

RESULTS

A total of 3240 patient records were obtained from AXIUM of which 2763 records had completed data for age, gender, caries risk, the status of bruxism and root canal treatment. Descriptive statistics of the study population are provided in Table 1. There were 1486 cases (53.8%) who had received root canal treatment and 1277 controls (46.2%) without root canal treatment. The cases were significantly older than the controls (58.2 ± 14.2 years vs. 49.1 ± 16.3 years). The mean age of the study population was 54 years old. Fifty-two percent (1426) of the subjects were female and forty-eight percent (1337) of the subjects were male. There was no statistical difference in gender distribution between cases and controls. Significantly more cases had a moderate caries risk, (68.0%) than did the control group, (56.8%), $p<0.0001$. Clinical signs of bruxism were significantly more apparent among cases (55.4%) than among controls (43.9%), $p<0.0001$. Logistics regression (Table 2) revealed significant associations between root canal treatment and bruxism (OR, 1.30; 95% CI, 1.10-1.54) as well as with increasing age for each year (OR, 1.04; 95% CI, 1.03-1.04), female gender (OR, 1.19; 95% CI, 1.01-1.41), and moderate caries risk (vs. low caries risk) (OR, 1.43, 95% CI 1.21-1.70).

Table 1: Patients' characteristics (N=2763)

Variable	Cases n=1486 No. 53.7%%		Controls n=1277 No. 46.2%	p-value*
Age (years) Mean ± SD	58.2 ± 14.2		49.1 ± 16.3	<0.0001
Gender				0.11
Female	788 (53.0%)		638 (50.0%)	
Male	698 (47.0%)		639 (50.0%)	
Caries Risk				<0.0001
Low	475 (32.0%)		552 (43.2%)	
Moderate	1011 (68.0%)		725 (56.8%)	
Bruxism				<0.0001
Yes	823 (55.4%)		560 (43.9%)	
No	537 (36.1%)		615 (48.2%)	

*Comparison between cases and controls based on t test for age and χ^2 test for gender, caries risk and bruxism.

Table 2. Logistics Regression Analysis of the Association of the Independent Variables of Age, Gender, Caries Risk, and Bruxism with Root Canal Treatment.

Variables	Coefficient	P value*	OR* (95% CI)
Age (each increasing year)	.035	.000	1.04 (1.03, 1.04)
Gender (female vs male)	0.176	.036	1.19 (1.01, 1.41)
Caries Risk (moderate vs low)	.360	.000	1.43 (1.21, 1.70)
Bruxism	.261	.002	1.30 (1.10, 1.54)

* Adjusted p values and odds ratios from the logistic regression model with age, gender, caries risk and bruxism in the model

Discussion

Bruxism seems to be primarily regulated centrally (pathophysiological and psychological), rather than peripherally (morphological) (20). Morphological factors include dental discrepancies and deviations in the anatomy of the bony

structures of the orofacial region (1); the pathophysiological mechanism is where bruxism is linked to sleep disturbances (20). Psychological roles are still unclear but Olkinuora found a higher incidence of bruxism in patients with psychological disorders (21). It has been found that several neurochemicals (e.g., serotonin, dopamine, gamma aminobutyric acid, and noradrenaline) are involved in the genesis of rhythmic jaw movements, bruxism, and the modulation of muscle tone during sleep (22).

Bruxism can lead to craze lines, fractured cusps, and cracked teeth. The American Association of Endodontics categorized longitudinal tooth fractures into 5 major categories: craze line, fractured cusp, cracked tooth, split tooth, and vertical root fractures (23, 24). While the etiology of cracks is difficult to ascertain, trauma from parafunctional activity has been cited as a common reason (25). Cracks can be found in 28% of non-restored teeth (26). A crack in a tooth, caused by bruxism, for example, is a potential cause of pulpal and periradicular diseases in much the same manner that caries and breakdown of restoration margins can cause diseases by allowing bacteria to enter the tooth and produce endotoxins that can reach the pulp. This will only occur if a

sufficient number of bacteria are present in the crack and of the particular bacteria are virulent enough to cause pulp and periradicular disease (16).

The aim of our study was to evaluate the association of bruxism with root canal treatment. Our results showed positive associations between bruxism, moderate caries risk, female gender, and age with root canal treatment. Our findings for the association with moderate caries risk was in agreement with Kirkevang where he found the presence of coronal restorations increased the risk for apical periodontitis (17). There are no studies relating age and gender to root canal treatment, but there are studies relating bruxism and cracks pertaining to age and gender. According to Hublin, there is a higher incidence of bruxism in females (27). Rosen found a higher prevalence of cracks after middle age (28) but Roh and Lee found the highest prevalence in the population aged 30-50 (29).

To our knowledge, this is the first case-control study to describe the association of bruxism with root canal treatment. Our findings suggest that females, increasing age, a moderate caries risk and bruxism may increase the risk for root

canal treatment. Previous studies have investigated other risk factors for root canal treatment. Segura-Egea investigated the relationship between smoking and endodontic variables in hypertensive patients (30). The author reported the prevalence of apical periodontitis and root canal treatment was significantly higher in hypertensive patients who smoked compared to nonsmoking subjects. Krall *et al,* also investigated smoking as a risk factor for root canal treatment. The authors concluded that there is a dose response relationship between cigarette smoking and the risk for root canal treatment (31). Kirakozova and Caplan found that the total extent of coronal tooth structure was related to a higher risk for root canal treatment (32).

The limitations of this study include misclassification bias arising from a retrospective chart review, including the possibility of either under-reporting or over-reporting of root canal status or the clinical signs of bruxism and missing data. Also, a case-control study does not confirm a causal effect, and the sample selection is subject to bias. Since data were collected from a single academic institution, the generalizability of this study is limited. Future research

could conduct a large prospective cohort study to assess the relationship between bruxism, age, gender, and caries risk for root canal treatment.

Conclusions

This study has found a positive association between bruxism and risk for root canal treatment. Patients who had clinical signs of bruxism and/or exhibit clinical signs of occlusal trauma and receive an accurate diagnosis of bruxism can be treated conservatively with the use of occlusal guards to protect their dentition from further trauma which may lead to pulpal and periradicular pathosis.

References

1. Kato T, Thie NM, Huynh N, Miyawaki S, Lavigne GJ. Topical review: sleep bruxism and the role of peripheral sensory influences. J Orofac Pain 2003;17(3):191-213.

2. Lobbezoo F, Van Der Zaag J, Naeije M. Bruxism: its multiple causes and its effects on dental implants - an updated review. J Oral Rehabil 2006;33(4):293-300.

3. Glaros AG, Rao SM. Bruxism: a critical review. Psychol Bull 1977;84(4):767-781.

4. Bader GG, Kampe T, Tagdae T, Karlsson S, Blomqvist M. Descriptive physiological data on a sleep bruxism population. Sleep 1997; 20(11):982-990.

5. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil 2008; 35(7):476-494.

6. Ferrario VF, Sforza C, Zanotti G, Tartaglia GM. Maximal bite forces in healthy young adults as predicted by surface electromyography. J Dent 2004; 32(6):451-457.

7. Nishigawa K, Bando E, Nakano M. Quantitative study of bite force during sleep associated bruxism. J Oral Rehabil 2001;28(5):485-491.

8. Hannam AG. In C McNell (Ed). Science and practice of occlusion. Chicago: Quintessence Pub. Co.; 1997: 41-49

9. Waltimo A, Kononen M. A novel bite force recorder and maximal isometric bite force values for healthy young adults. Scand J Dent Res 1993; 101(3):171-175.

10. Reny DL. Orofacial pain: guidelines for assessment, diagnosis, and management. 4th ed. Chicago: Quintessence; 2008.

11. Thorpy M. The international classification of sleep disorders: Diagnostic and coding manual. Rochester, MN: Allen Press; 1990: 142-185

12. Rugh WK, Ohrbach R. Occlusal parafunction**.** In: Mohl N, Zarb GA, C arlsson GE, Rugh JD (eds) A textbook of occlusion. *Chicago:* Quintessence Publishing Company. 1988; 252

13. Greene CS, Mohl ND, McNeill C, Clark GT, Truelove EL. Temporomandibular disorders and science: a response to the critics. Am J Orthod Dentofacial Orthop 1999;116 (4):430-431.

14. Mumford JM, Newton AV. Spatial summation of pre-pain and pain in human teeth. Pain 1985;22(3):320-321.

15. Narhi MV. The characteristics of intradental sensory units and their responses to stimulation. J Dent Res 1985;64 Spec No:564-571.

16. Krell KV, Rivera EM. A six year evaluation of cracked teeth diagnosed with reversible pulpitis: treatment and prognosis. J Endod 2007;33(12):1405-1407.

17. Kirkevang LL, Vaeth M, Wenzel A. Tooth-specific risk indicators for apical periodontitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;97(6):739-744.

18. Abbott P, Leow N. Predictable management of cracked teeth with reversible pulpitis. Aust Dent J 2009;54(4):306-315.

19. Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? J Orofac Pain 1997;11(1):15-23.

20. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil 2001; 28(12):1085-1091.

21. Olkinuora M. Psychosocial aspects in a series of bruxists compared with

a group of non-bruxists. Proc Finn Dent Soc 1972;68(4):200-208.

22. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med 2003;14(1):30-46.

23. Berman LH, Kuttler S. Fracture necrosis: diagnosis, prognosis assessment, and treatment recommendations. J Endod 2010;36(3):442-446.

24. Endodontists. Endodontics: Colleagues for Excellence-Cracking the Cracked Tooth Code. American Association of Endodontists 2008

25. Abou-Rass M. Crack lines: the precursors of tooth fractures - their diagnosis and treatment. Quintessence Int Dent Dig 1983;14(4):437-447.

26. Seo DG, Yi YA, Shin SJ, Park JW. Analysis of factors associated with cracked teeth. J Endod 2012;38(3):288-292.

27. Hublin C, Kaprio J, Partinen M, Koskenvuo M. Sleep bruxism based on self-report in a nationwide twin cohort. J Sleep Res 1998;7(1):61-67.

28. Udoye CI, Jafarzadeh H. Cracked tooth syndrome: characteristics and distribution among adults in a Nigerian teaching hospital. J Endod 2009;35(3):334-336.

29. Roh BD, Lee YE. Analysis of 154 cases of teeth with cracks. Dent

Traumatol 2006;22(3):118-123.

30. Segura-Egea JJ, Castellanos-Cosano L, Velasco-Ortega E, Rios-Santos JV, Llamas-Carreras JM, Machuca G, et al. Relationship between smoking and endodontic variables in hypertensive patients. J Endod 2011;37(6):764-767.

31. Krall EA, Abreu Sosa C, Garcia C, Nunn ME, Caplan DJ, Garcia RI. Cigarette smoking increases the risk of root canal treatment. J Dent Res 2006;85(4):313-317.

32. Kirakozova A, Caplan DJ. Predictors of root canal treatment in teeth with full coverage restorations. J Endod 2006;32(8):727-730.

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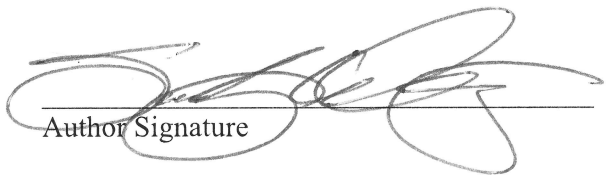

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