JS Rees M Addy

A cross-sectional study of buccal cervical sensitivity in UK general dental practice and a summary review of prevalence studies

Authors' affiliations:

JS Rees and M Addy, Department of Oral & Dental Science, Division of Restorative Dentistry, University of Bristol Dental School, Lower Maudlin St., Bristol BS1 2LY, UK

Correspondence to:

JS Rees

Department of Oral & Dental Science Division of Restorative Dentistry University of Bristol Dental School Lower Maudlin St. Bristol BS1 2LY, UK

Tel.: +44 117 928 4312 Fax: +44 117 928 4780 E-mail: j.s.rees@bristol.ac.uk

Dates:

Accepted 8 December 2003

To cite this article:

Int J Dent Hygiene 2, 2004; 64–69
Rees JS, Addy M:
A cross-sectional study of buccal cervical sensitivity in UK general dental practice and a summary review of prevalence studies

Copyright © Blackwell Munksgaard 2004

Abstract: Aim: The initial aim of this study was to establish the prevalence of dentine hypersensitivity (DH) in a cross-sectional study of patients visiting general dental practitioners in the UK over a period of one calendar month. Methods: Eighteen dental practitioners examined 5477 patients over a period of one calendar month, and patients who were diagnosed with DH were questioned further about their occupation and smoking habits. The amount of buccal gingival recession associated with the sensitive teeth was also recorded. as was the presence of periodontal disease. Results: One hundred and fifty-two patients were diagnosed as having DH, giving a prevalence figure of 2.8%. The commonest teeth affected were the first molars and premolars, and the commonest initiating factor was cold drinks. A tendency for a greater number of DH teeth was also found for patients with periodontal disease who also smoked. There was also a tendency for the patients with DH teeth to come from higher social groups. Conclusion: At the time of conducting this study, the European Federation of Periodontology had not recommended that sensitive teeth associated with periodontal disease and treatment be termed root sensitivity (RS). The title of this paper was therefore chosen to reflect this decision, and the data represent teeth both within and between subjects with DH and RS.

Key words: dentine; dentine hypersensitivity; epidemiology; gingival recession; smoking

Introduction

Dentine hypersensitivity (DH) may be defined as pain arising from exposed dentine, typically in response to chemical, thermal or osmotic stimuli that cannot be explained as arising from any other

Table 1. Summary of prevalence studies on DH

Authors	Country	Setting	Study type	n	Prevalence (%)
Jensen (1964)	USA	University	Clinical	3000	30
Graf and Glase (1977)	Switzerland	Practice	Clinical	351	15
Flynn et al. (1985)	UK	University	Clinical	369	18
Orchardson and Collins (1987)	UK	University	Clinical	109	74
Fischer <i>et al.</i> (1992)	Brazil	University	Clinical	635	17
Murray and Roberts (1994)	Indonesia	Not stated	Questionnaire	1000	27
Murray and Roberts (1994)	USA	Not stated	Questionnaire	1000	18
Murray and Roberts (1994)	Japan	Not stated	Questionnaire	1000	16
Murray and Roberts (1994)	France	Not stated	Questionnaire	1000	14
Murray and Roberts (1994)	Germany	Not stated	Questionnaire	1000	13
Murray and Roberts (1994)	Australia	Not stated	Questionnaire	1000	13
Chabanski et al. (1997)	UK	University	Clinical	51	73
Irwin and McCusker (1997)	UK	Practice	Questionnaire	250	57
Liu et al. (1998)	Taiwan	University	Clinical	780	32
Rees (2000)	UK	Practice	Clinical	3593	4
Taani and Awartani (2002)	Saudi Arabia	University	Clinical	295	42-60
Clayton et al. (2002)	UK	Air force	Questionnaire	228	50
Rees and Addy (2002)	UK	Practice	Clinical	4841	4
Rees et al. (2002)	Hong Kong	University	Clinical	226	68

form of dental defect or pathology (1). DH is a common problem found in many adult populations with prevalence figures varying widely (Table 1). This may be because of a number of factors, including the different study designs used to assess the condition, variation in the patients' oral hygiene habits and consumption of erosive foods and drinks, together with the type of setting where the study was carried out. For example, some studies have used a patient questionnaire that requires the self-reporting of DH that may well overestimate the magnitude of the problem because of sensitivity caused by other pathologies, such as caries. Studies carried out in a hospital or specialist practice setting tend to report higher prevalence values, presumably because of the greater risk of root exposure as a result of periodontal attachment loss and gingival recession following periodontal treatment (2). This has prompted Dababneh et al. (3) to suggest that the DH associated with periodontal disease may have a different aetiology, possibly related to the bacterial penetration of the dentinal tubules (4). In this respect, and after the present study was completed, the report from the 2002 European Federation of Periodontology meeting used the term root sensitivity (RS) to describe sensitivity associated with periodontal disease and treatment (5).

Many previous investigations of DH (Table 1) have examined a sample of patients referred to a university hospital or specialist practice. Limited information is available on a sample of patients attending a general dental practice. The aim of the present study was to carry out a cross-sectional study of buccal cervical sensitivity in a group of patients treated in a general dental practice setting. In hindsight, given the recommendations of the European Federation of Periodontology, the data collected would include patients with DH, RS or both.

Materials and methods

All 18 general dental practitioners, who were undertaking a postgraduate distance-learning course on toothwear, run by the University of Bristol Dental Postgraduate Department, were recruited to participate in this study, and all the participants completed the study. Prior to the start of the study, the practitioners met with the authors to finalise details of the study protocol. In addition to this, they were also asked to study a module that gave an overview of the topic of DH (6) prior to the commencement of the study. This module included a number of review articles on the topic (1, 7, 8), and the practitioner's understanding of the topic was checked using a series of short answer questions that were evaluated by the authors. Feedback to the practitioner was given to iron out any misapprehensions. Throughout the study protocol meeting and in the module, it was emphasised that in order to make the diagnosis of dentine hypersensitive, other pathologies, such as caries, must be ruled out (7).

The study ran from 1 June-30 June 2001, and all the patients seen by each dentist during the trial period were screened for sensitive teeth. If the dentist received a positive response, the diagnosis was confirmed using a blast of air from a triple syringe and by ruling out other causes of sensitivity. Tactile sensitivity using a probe applied to the cervical region was not assessed as Chabanski et al. (2) found no difference in the ability of tactile and evaporative stimuli to elicit a subjective response of sensitivity. Periodontal disease was assessed using a Basic Periodontal Examination (BPE). Periodontal disease was diagnosed by a BPE score more than 3, and/or loss of attachment measured from the cemento-enamel junction by clinical and/or radiographic examination.

Where a diagnosis of DH was made, a study form was completed. This included details of the patients' age, gender and occupation, smoking habits, teeth affected and any factor known to initiate the sensitivity. In addition to this, each dentist was asked to measure any buccal gingival recession associated with the sensitive teeth. Measurements were made using a 1 mm graduated periodontal probe from the amelo-cemental junction to the free gingival margin. They were also asked to record the total number of patients seen during the trial period.

Results

Eighteen general dental practitioners took part in the study, and the total number of patients seen during the trial period was 5477. A total of 1054 teeth were diagnosed as having DH in 152 patients, giving an overall prevalence figure for DH of 2.8%. The average age of these patients was 42.9 years, with a range of 15–80 years. Sixty-one patients were males and 91 were females, giving an overall male:female ratio of 1:1.5. A histogram showing the age distribution of the patients with hypersensitive dentine is given in Fig. 1. By far, the highest number of patients with DH lay within the 4th decades (30-40 years).

The number of sensitive teeth classified by tooth type (Fig. 2) shows that the upper first molars were most commonly affected, closely followed by the first premolars, canines and then the second molars.

The mean number of sensitive teeth per patient by age group (Fig. 3) shows a peak of 7.3 sensitive teeth for the 11–20-year age

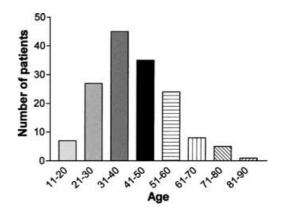


Fig 1. Age distribution of patients with sensitive teeth.

group and a second smaller peak of 5.8 sensitive teeth for the 41-50-year age group.

The amount of gingival recession associated with the sensitive teeth (Fig. 4) shows that overall 985 of the 1054 sensitive teeth (93%) had some associated buccal gingival recession, the majority (91%) in the range of 1–3 mm.

The mean amount of gingival recession per tooth that was associated with the sensitive teeth was recorded (Fig. 5). These data were further classified according to whether the patients were smokers or non-smokers, and whether they had periodontal disease or not (Table 2). Within the cohort with sensitive teeth, 52 patients (34%) smoked. As can be seen, patients who had periodontal disease had more gingival recession regardless of whether they smoked or not. ANOVA also showed that the amount of mean gingival recession found in the groups with periodontal disease was statistically significantly different (P < 0.001) from the other groups.

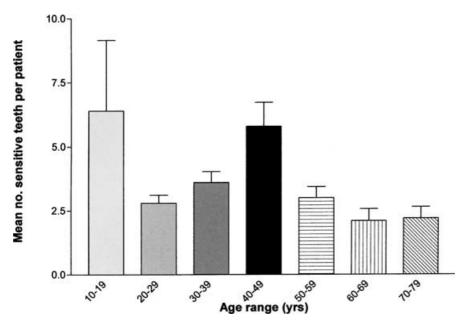


Fig 2. Mean number of sensitive teeth by patient.

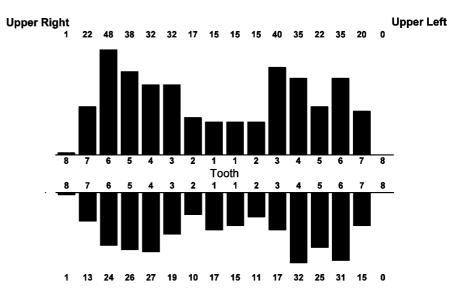


Fig3. Sensitive teeth classified by tooth type.



Lower Left

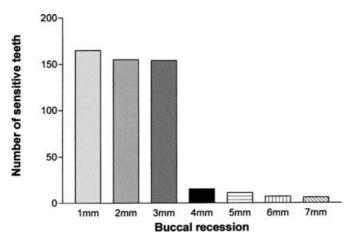


Fig 4. Amount of buccal gingival recession.

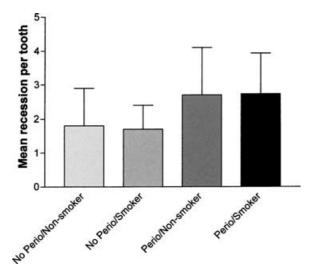


Fig 5. Mean amount of gingival recession per sensitive tooth.

Table 2. Relationship between sensitive teeth, smoking and periodontal disease

Group	Mean number sensitive teeth per patient	SD	Range
Non-smoker/no periodontal disease Non-smoker/periodontal disease Smoker/no periodontal disease Smoker/periodontal disease	3.3 3.7 4.0 5.8	3.6 3.4 3.6 5.0	1–15 1–17 1–17 2–22

The relationship between DH and social class was examined (Fig. 6) using the Registrar General's Classification of Occupations as used by Treasure et al. (9) in the UK Adult Dental Health Survey. This divides occupation into a series of six groups using the following classification:

- I: Professional (e.g. doctor, dentist, lawyer).
- II: Managerial and lower professional (e.g. manager, nurse, school teacher).
- IIIN: Skilled, non-manual (e.g. clerk, cashier).
- IIIM: Skilled, manual (e.g. carpenter, brick layer, coal face worker).
- IV: Semi-skilled, manual (e.g. postman, agricultural worker).
- V: Unskilled, manual (e.g. porter, ticket collector, general labourer).

As can be seen (Fig. 6), there were a greater number of sensitive teeth in the higher social groups I and II (46%) as compared to group III (31%) and the lower groups IV and V (23%).

Discussion

The overall prevalence figure found in this study was 2.8%, which is much lower than the prevalence figures reported in many

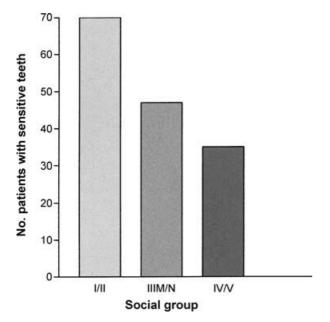


Fig 6. Sensitivity and social group.

previous studies (Table 1). The reasons for these differences may be because of a number of factors. Firstly, many earlier studies have used a questionnaire approach, with the patient 'self-reporting' sensitivity with no subsequent clinical examination. It is very likely that this approach will grossly overestimate the prevalence figure, as the sensitivity reported could be a result of a number of different pathologies (7). Furthermore, only four previous studies have attempted to estimate the prevalence of DH within a general practice setting (10-13). The study of Graf and Galasse (10) reported on a relatively small sample (n = 351), while Irwin and McCusker (11) used a self-reporting questionnaire design with no clinical examination. Rees (12), and Rees and Addy (13) used a similar methodology as the current study and reported prevalence values of 4-4.1%. It is interesting to note that the current study found a slightly lower prevalence value of 2.8% while employing a totally different cohort of dentists and patients.

However, this prevalence value must be interpreted with a certain amount of caution. Firstly, the practices chosen for inclusion in this study were not chosen randomly, but were selfselected because of the dentist's participation in the postgraduate programme, and this could introduce a certain element of bias. Secondly, it is almost impossible to assess whether the patients attending the chosen practices could be considered to be representative of all patients in general practice within the UK. However, the practices were well distributed geographically throughout the UK, and they also served as a mixture of rural and inner-city areas. It is likely that the social profile of the patients attending these dental practices differed, which could contribute to the variation in the individual prevalence data recorded. Finally, as a binary index was used for the diagnosis of sensitivity, the chances of an α -error because of examiner bias is highly unlikely.

This study found that DH was commonest in two age groups: 10-19 and 40-49 years. However, the first peak in the 10-19-year age group is probably artifactual as one patient was recorded as having eight sensitive teeth within a group with a very small sample size (n = 3). The second peak at 40–49 years is probably more realistic and concurs with the findings of Fischer et al. (14). Orchardson and Collins (15) showed a peak prevalence between 20 and 25 years, Graf and Galasse (10) between 25 and 29 years and Absi et al. (16) between 20 and 40 years.

The teeth most often affected by DH were the upper molars, followed by the premolars/canines, with the lower incisors being the least sensitive. This is similar to the findings of Chabanski et al. (2) and Rees et al. (17). However, many other workers have reported that the canine/premolar regions were the commonest sites for sensitivity (12, 15-19). It is possible that the reports of greater sensitivity in the canine/premolar region could relate to a sample with a greater prevalence of periodontal disease compared to the general practice-based sample in this study. As molars are lost more readily because of periodontal disease compared to canines/premolars, it is possible that patients with periodontal disease would have greater sensitivity related to these teeth. Thus, in the present study, it is almost certain that the total cohort comprised patients with sensitivity, which was consistent with the agreed definition of DH (20).

Most of the sensitive teeth included in the study also had some degree of buccal gingival recession (Fig. 4). Most teeth had at least 1-3 mm of gingival recession, which is similar to the average recession of 2.5 mm reported by Addy et al. (21). In this study, some teeth were sensitive but had no obvious buccal recession. Two explanations can be propounded to explain this apparent anomaly. Firstly, the minimum measurement of buccal recession was less than 1 mm and this may have been recorded as 0 mm. Secondly, in cases of early recession, there is a high failure rate to detect recession clinically (22).

It has been established for some years that smoking is a major risk factor for periodontal disease and attachment loss (23). Because of attachment loss, root surfaces become exposed potentially leading to sensitivity. Therefore, it was decided to investigate whether there was any difference in the number of sensitive teeth per patient when the patient was a smoker or non-smoker, and if they did or did not have periodontal disease. There seemed to be little difference in the number of sensitive teeth when the patient was a non-smoker with and without periodontal disease (3.3 vs. 3.7). Similarly, patients who had no periodontal disease but who were smokers had a similar number of sensitive teeth (4.0). However, patients who had periodontal disease and also smoked had a higher number of sensitive teeth (5.8). However, these differences were not statistically significant when examined using ANOVA.

The mean amount of gingival recession per tooth in each of these four groups was also calculated (Fig. 5). As can be seen, the amount of recession was slightly higher in patients who smoked, regardless of whether they had periodontal disease or not. However, this difference was not statistically different. This would also seem to agree with the work of Müller et al. (24), who found that smoking did not seem to influence gingival recession.

It is the authors' clinical impression, supported by some data (13, 16), that DH is more prevalent in patients with clean mouths who have good oral hygiene, as tends to be the case in higher social groups (25). To investigate this further, the patients with DH were divided into social groups using the Registrar Generals Classification of Occupations as used in the recent UK Adult Dental Health Survey (9). This demonstrated (Fig. 6) that DH was more prevalent in the higher social groups, with 46% of the sensitive teeth being found in the top two social groups. However, these data must also be interpreted with a certain amount of caution as this sample may be further biased, as a number of regular attenders at dental surgeries are also known to belong to the higher social groups.

References

- 1 Addy M, Urquart E. Dentine hypersensitivity: its prevalence, aetiology and clinical management. Dent Update 1995; 22: 407-12.
- 2 Chabanski MB, Gillam DG, Bulman JS, Newman HN. Clinical evaluation of cervical dentine sensitivity in a population of patients referred to a specialist periodontology department: a pilot study. J Oral Rehabil 1997; 24: 666-72.
- 3 Dababneh RH, Khouri AT, Addy M. Dentine hypersensitivity an enigma? A review of terminology, epidemiology, mechanisms, aetiology and management. Br Dent J 1997; 187: 603-7.
- 4 Adriaens PA, de Boever JA, Loesche WJ. Bacterial invasion in root, cementum and radicular dentine of periodontally diseased teeth in humans – a reservoir of periodontopathic bacteria. J Periodontol 1988; **59**: 222–30.
- 5 Von Troil B, Needleman M, Sanz M. A systematic review of the prevalence of root sensitivity following periodontal therapy. J Clin Periodontol 2002; 29 (Suppl. 3): 173-7.

- 6 Addy LD Dentine Hypersensitivity. Diagnosis and Clinical Management. Bristol: Bristol University Open Learning for Dentists Course, Dental Postgraduate Department. University of Bristol, 2000.
- 7 Dowell PD, Addy M, Dummer PMH. Dentine hypersensitivity. Aetiology, differential diagnosis and management. Br Dent J 1985; 158: 92-6.
- 8 Addy M, Pearce NX. Aetiological, predisposing and environmental factors in dentine hypersensitivity. Arch Oral Biol 1994; 39 (Suppl.) 33S-8S.
- 9 Treasure E, Kelly M, Nuttall N et al. Factors associated with oral health: a multivariate analysis of results from the 1998 Adult Dental Health Survey. Br Dent J 2001; 190: 60-8.
- 10 Graf H, Galasse R. Morbidity, prevalence and intraoral distribution of hypersensitive teeth. J Dent Res 1997; 76: A162 (Abstract no. 479).
- 11 Irwin CR, McCusker P. Prevalence of dentine hypersensitivity in a general dental population. J Ir Dent Assoc 1993; 43: 7-9.
- 12 Rees JS. The prevalence of dentine hypersensitivity in general dental practice in the UK. J Clin Periodontol 2000; 27: 860-5.
- 13 Rees JS, Addy M. A cross-sectional study of dentine hypersensitivity. J Clin Periodontol 2002; 29: 997-1003.
- 14 Fischer C, Fischer RG, Wennberg A. Prevalence and distribution of cervical dentine hypersensitivity in a population in Rio de Janeiro, Brazil. J Dent 1992; 20: 272-6.
- 15 Orchardson R, Collins WJN. Clinical features of hypersensitive teeth. Br Dent J 1987; 162: 253-6.
- 16 Absi E, Addy M, Adams D. Dentine hypersensitivity. The patency of dentinal tubules in non-sensitive and sensitive dentine. J Clin Periodontol 1987; 14: 280-4.
- 17 Rees JS, Jin LJ, Lam S, Kudanowska I, Vowles R. The prevalence of dentine hypersensitivity in a hospital clinic population in Hong Kong. J Dent 2003; 31: 453-61.
- 18 Flynn J, Galloway R, Orchardson R. The incidence of 'hypersensitive' teeth in the West of Scotland. J Dent 1985; 13: 230-6.
- 19 Liu H-S, Lan W-H, Hsieh C-C. Prevalence and distribution of cervical dentine hypersensitivity in a population in Taipei, Taiwan. J Endodont 1998; 24: 45-7.
- 20 Holland GR, Narhi MN, Addy M. Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. J Periodontol 1997; **24**: 808–13.
- 21 Addy M, Absi ED, Adams D. Dentine hypersensitivity: the effects of acids and dietary compounds on burred and planed dentine. J Clin Periodontol 1987; 14: 274-9.
- 22 Bevenius J, Lindskog S, Hultenby K. The micromorphology in vivo of the buccocervical region of premolar teeth in young adults. A replica study by scanning electron microscopy. Acta Odontol Scand 1994; 52: 323-34.
- 23 Harber J, Wattles J, Crowley P. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol 1993; 64: 16-23.
- 24 Müller H-P, Stadermann S, Achim Heinecke A. Gingival recession in smokers and non-smokers with minimal periodontal disease. J Clin Periodontol 2002; 29: 129-36.
- 25 Dummer PMH, Addy M, Hicks R et al. The effect of social class on the prevalence of caries plaque, gingivitis and pocketing in 11-12-year-old children in South Wales. J Dent 1987; 15: 185-90.

Copyright of International Journal of Dental Hygiene is the property of Blackwell Publishing Limited and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.

Copyright of International Journal of Dental Hygiene is the property of Wiley-Blackwell and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.