Refereed paper

Bulimia: implications for the practising dentist

F. J. T. Burke,[1] DDS, MDS, MSc, FDS, MGDS, T. J. Bell,[1] BDS, N. Ismail,[1] BDS, and P. Hartley,[2] PhD, AFBPsS, CPsychol

[1]University Dental Hospital of Manchester, Higher Cambridge Street, Manchester M15 6FH; [2]Centre for Health Studies, University College Salford, Frederick Road, Salford M6 6PU

The incidence of eating disorders appears to be increasing, with the dental practitioner potentially being the first healthcare worker to make a diagnosis, due to the characteristic dental signs of tooth substance loss. It is therefore important that members of the dental team are aware of the dental sequelae of anorexia and bulimia and are able to offer advice and treatment to sufferers.

Knowledge and awareness of eating disorders by dental practitioners is important, as they may be first to discuss the problem with the patient due to the prevalence of characteristic oral sequelae. It is also important that practitioners are aware of the potentially life threatening nature of these conditions. However, the diagnosis of the disease may present difficulties to the dental health care worker, because the affected individual may be reluctant to talk about their problem.

Definitions

Eating disorders have been described by the Eating Disorders Association as the 'outward expression of deep psychological and emotional turmoil, with sufferers turning to food and eating as a means of expressing their difficulties'.[1] There are two closely-linked disorders which comprise the majority of the group of disturbed eating patterns. These are anorexia and bulimia nervosa. Bulimia nervosa was first differentiated in 1979 by Gerald Russell who found in his study comparing a group of 28 females with bulimia and 29 females with anorexia nervosa that the 'bulimics' differed in terms of weight and amenorrhea on admission to hospital.[2] Anorexia nervosa, however, was first documented in 1689 by Richard Morton.[3] His account describes in excellent detail the features of eating disorders as they are today. There are details which are similar to those of bulimia: 'The Spring following by the prescription of some Emperick, she took a Vomit, and after that I know knot what Steel Medicines . . . whereupon consuming every day more and more, she was after three Months taken with a Fainting Fit and dyed.'

Anorexia nervosa literally means loss of appetite for nervous reasons, and has been defined as self-imposed starvation, together with an intense fear and pre-occupation with food and weight.[4] Bulimia nervosa, literally meaning ox-hunger, consists of recurrent episodes of compulsive binges (consumption of large amounts of food), with or without self-induced vomiting or purgation.[5]

Bulimic behaviour has long been recognised, having been mentioned in the 'Talmud' (a collection of Jewish laws several thousand years old) where it speaks of a person being permitted to eat on Yom Kippur, a traditional fasting day, only if the person is bulimic.[6] The disorder has also been termed, Binge–Purge Syndrome and Dietary Chaos Syndrome.[7]

The goal of the bulimic is to eat yet not gain weight. In this respect, bulimia sufferers are within 80% of the mean weight for age[8] but may be of normal weight or 5 to 15 lbs overweight.[9] Hilde Bruch postulated three symptoms of causal significance in anorexia nervosa in 1974 which are also descriptive of bulimia nervosa.[10] Bruch considered that these patients would manifest:[10]

1 Disturbance of delusional proportions of body image and body concept.
2 Disturbance in the accuracy of perception of stimuli arising in the body.
3 A paralysing sense of ineffectiveness.

Classification

It is the experience of most clinicians that the eating disordered population may be divided into sub-groups.[10,11] These are as follows:

Dent J 1996; **180**: 421–426
British Dental Journal 1996
ed for Publication 05.09.94
ed for Publication 23.10.95

1 Anorexia nervosa (restrictive subtype).
2 Anorexia nervosa (bulimic subtype).
3 Bulimia nervosa (with a history of anorexia).
4 Bulimia nervosa (without a history of anorexia).

It is suggested, therefore — as three of the four sub-groups will use vomiting as weight control — that dental problems will occur. Even restricting anorexics, binge-free, will often vomit to rid themselves of what little food they do eat. The American Psychiatric Association defined the disorders clearly,[12] as shown in Table I.

Prevalence

There has been a gradual historical progression in diagnosis but as yet there is inconclusive evidence for epidemiological change. A study of the Aberdeen case-registers by Szmukler *et al.* found an increase of approximately 3.0 per 100,000 population between 1969 and 1982.[13] The prevalence of bulimia has variously been assessed as being 2% of women attending a UK Family Planning Clinic,[14] from 4.5% to 18% of female high school and college students, and 35% of college students in the USA.[15–17] No figures are available for changes in incidence over the past decade, but based on the numbers of individuals seeking treatment, bulimia appears to be increasing rapidly in incidence.[18]

Bulimia is found most often among young women of age from late teens to mid 30s but the disorder is also found in men and older women.[15,19] It has been considered that most bulimic individuals are white and from middle or upper-middle class backgrounds,[16] who are attractive, well-groomed, intelligent, high achievers, who mask their disorder with perfectionism.[18] However, beneath this facade is a hidden person with poor self-image and who is emotionally immature.[20]

Aetiology/predisposing factors

Numerous theories have been proposed as aetiological/predisposing factors to bulimia. Possible explanations include biological and psychological factors, but as yet no conclusive explanation exists. Garner and Bemis have suggested a number of distorted attitudes which may predispose an individual to develop and maintain an eating disorder:[21]

1 The striving for perfection.
2 Asceticism is superior to self-indulgence.
3 Thinness is admirable, fat is disgusting.
4 Weight gain means that one is bad or out of control.

Possible risk factors are therefore as follows:

1. *Cultural/social pressures.* Cultural fixation with thinness may reinforce the bulimic individual's thinking, as Garner *et al.* demonstrated when they found that Miss America winners over the past 25 years had a progressive decrease in bust and hip measurements although the population in general was increasing in weight.[21,22]

2. *Family factors.* Factors associated with the bulimia sufferer's family have also been implicated. It has been suggested that the bulimic patient may be part of a generally disturbed family.[17] However, there may also be a history of eating disorders with sufferers typically being from a high-achieving, high performance expectation family of higher socio-economic class.[23] A number of studies have also demonstrated an increased incidence of alcoholism in close relatives of anorexic and bulimic patients,[24] with Collins *et al.* finding alcoholism to be 2.5 times more common in families of bulimic patients than in the general population.[25]

3. *Individual factors.* Impaired psychological development has also been implicated,[18] while associations have also been noted between eating disorders and depression with Russell finding that, of 30 patients with bulimia, 87% had symptoms of depression.[2]

Table I Diagnostic criteria for anorexia nervosa and bulimia nervosa

Anorexia nervosa	Bulimia nervosa
a Refusal to maintain body weight over a minimal weight for age and height, eg weight loss leading to maintenance of body weight 15% below that expected; or failure to make expected weight gain during period of growth, leading to body weight 15% below that expected.	a Recurrent episodes of binge-eating (rapid normal consumption of a large amount of food in a discrete period of time).
b Intense fear of gaining weight or becoming fat, even though under weight.	b A feeling of lack of control over eating behaviour during the eating binges.
c Disturbance in the way in which one's body weight, size or shape is experienced, eg the person claims to 'feel fat' even when emaciated, believes that one area of the body is 'too fat' even when obviously under weight.	c The person regularly engages in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain.
d In females, the absence of at least three consecutive menstrual cycles when otherwise expected to occur (primary or secondary amenorrhea).	d A minimum average of two binge eating episodes a week for at least three months.
	e Persistent overconcern with body shape and weight.

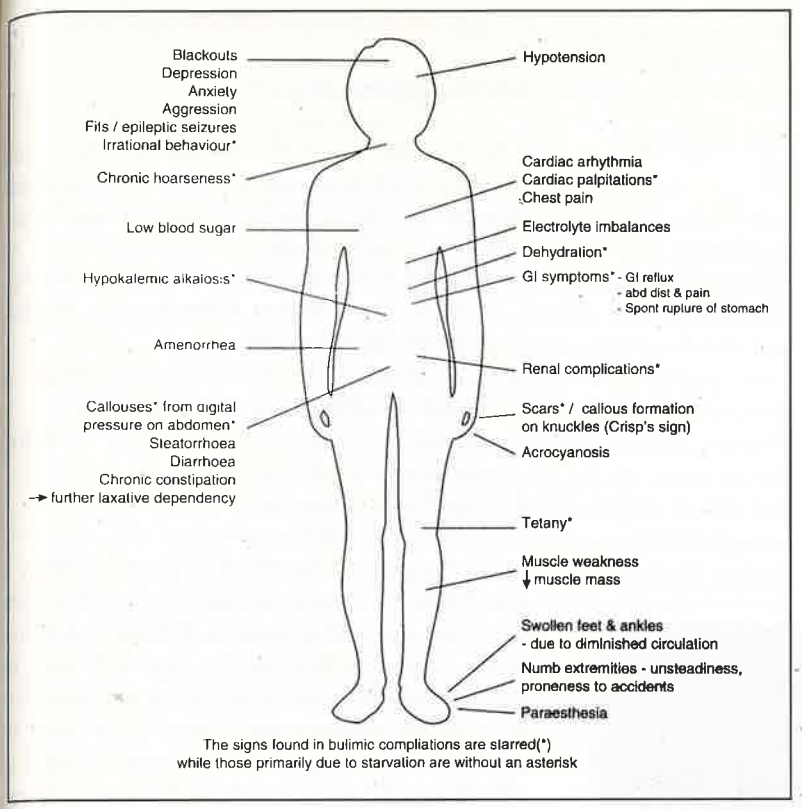

Fig. 1 Possible physical and emotional signs of anorexia nervosa and bulimia.

4. *Other factors*. Anorexia/bulimia may be triggered by an upsetting life event, such as the break-up of a relationship, moving away from home etc.

According to Hsu[4] once the pathological eating disturbances are established, they may then be perpetuated by both positive and negative reinforcers. Family dysfunction may also serve as a reinforcer, either positively (the illness increases a sense of solidarity) or negatively (the illness leads to avoidance or detour of the conflict). However, no conclusive evidence has been produced to demonstrate that family dysfunction causes eating disorders, although a strong body of evidence suggests that eating disorders may lead to family dysfunction. Cognitive and conceptual distortions also perpetuate the illness and may reflect a disturbance in self-structure and/or a dysfunctional parent-child interactions.

Hsu suggests that in the pathogenesis of eating disorders, genetic predisposition may be involved by affecting personality development. Slade also incorporates this perspective in his analysis of eating disorders.[26] In summary, bulimia and anorexia may represent multiple disorders which have a single final common pathway, with contributions to its development coming from multiple sources which vary from person to person.

Binges

In binges, large amounts of up to 20,000 kCals of food may be rapidly consumed in a short period of time.[27] The binges are often secretive and planned, consisting of high calorie 'forbidden' foods such as sweets, chocolate, bread and biscuits. Sufferers will explain how loss of control occurs during the binge, with feelings of guilt, self-deprecation and depression occurring after the binge.[17]

Purge

Regurgitation is the most frequent method of purgation following a binge, with the gag reflex being induced by placing fingers, a comb or toothbrush or other object in the throat. Fingers may develop callouses due to the chronic trauma from the dentition (Crisp's sign).[28] A number of individuals may develop the ability to vomit by contracting the abdominal muscles or by application of external pressure to the abdominal musculature.[11,29] Vomiting may lead to electrolyte loss, resulting in alkalosis with associated hypokalaemia, characterised by muscle weakness, constipation, abdominal pain and heart palpitations (fig. 1). Vitamin deficiencies may also occur as a result of the disruption to the diet. Other methods of purgation include the use of diuretics, laxatives, thyroid drugs and amphetamines, with laxatives being used in up to 50 times the recommended dosage.[14]

The dental evidence

The oral manifestations of bulimia are shown in Table II. In bulimic patients, erosion by regurgitation of acid gastric contents occurs initially on the palatal surfaces of the maxillary incisors, canines and first premolars while occlusal surfaces of molars, second premolars and mandibular premolars may occur as a result of more chronic vomiting.

This decalcification due to chronic regurgitation has been termed perimolysis and perimylolysis[30–32] (fig. 2). Typically the symptoms will be increased sensitivity of the teeth to hot and cold and acidic substances, the patient may complain of poor aesthetics because of rounding of tooth contour or jagged incisal edges following fracture of unsupported enamel. In cases where the occlusal surfaces are eroded, restorations may protrude above the occlusal surfaces, with the resultant effect having been described as 'fillings appearing as islands of metal'[33] (fig. 3). Perimolysis is not specifically diagnostic of bulimia as it may be seen in other medical conditions associated with gastric dysfunction such as hiatus hernia, gastric and duodenal ulcers, pregnancy, rumination and achlorhydria.[34] Erosion may also be caused by dietary substances, defined by Eccles as extrinsic erosion,[35] this differing by a more generalised loss of enamel, or associated with areas where the erosive beverages are held or swished.[36] The severity of the oral sequelae of bulimia are related to five factors:

Table II Oral manifestations of bulimia

Dental erosion with resulting:	Dentine exposure and hypersensitivity
	Pulpal exposure
	Diastemas
	Thinning/fracture of maxillary incisal edges
	Loss of vertical dimension
	Prominence of restorations
	Pseudo-open-bite
	Poor aesthetics
Possible increase in caries incidence[28,41]	
Glossodynia	
Periodontal disease	
Orthodontic tooth movement	
Parotid enlargement	
Halitosis	
Dysgeusia/taste impairment	
Sore throat/dysphagia	
Effects of nutritional deficiencies:	Glossitis
	Angular cheilitis
	Osteoporosis
	Mouth ulcers

Fig. 2 Perimolysis — 'loss of tooth substance associated with rumination or persistent vomiting'.[30] Gross tooth surface loss affecting palatal surfaces of upper teeth in a bulimic patient.

Fig. 3 Amalgam restoration standing proud of occlusal surface in the lower second molar of a bulimic patient. The patient's first molar teeth had been crowned 3 years previously because of gross tooth surface loss.

1 Frequency of purging incidents.
2 Duration of habit — it has been considered that enamel will not erode until regurgitation has occurred for 2 years.[37]
3 Oral hygiene habits, especially immediately following the vomiting incident.
4 Degree of acid dilution by water rinsing or drinking (eg milk) following vomiting.
5 Timing and technique of toothbrushing.

Management

The management of the bulimic patient should include members of a number of medical specialities. Among the forms of treatment which a bulimic patient is likely to require are:

1 Medical treatment — to rule out an underlying medical cause and to determine the possible sequelae from the habit.
2 Nutritional advice/dietary counselling, as nutritional deficiencies may be present.
3 Treatment from a psychologist, including attendance at self-help groups.
4 Psychiatric advice and help.
5 Dental advice and treatment.

As the major component in overall therapy is psychological, the psychologist is often the person of choice to coordinate both medical and dental needs. This interdisciplinary approach may be used to best advantage by setting up special centres for sufferers where comprehensive care may be provided, including a dental surgery, in order to co-ordinate the dental needs of the patient alongside the other members of the healthcare team.

Dental management

Dentists may be the first healthcare worker to suspect bulimia in a patient because of the prominence of oral signs and symptoms with their role being the differential diagnosis from other forms of erosion and appropriate early referral. The provision of a sympathetic, informative and non-judgmental approach is essential if the dentist is to gain the confidence of the patient and successfully complete treatment.[23] Rather than asking direct questions, the dentist should ask, in a kind, caring manner, leading questions related to the amount of food eaten and gastrointestinal problems. It is essential to make patients aware of their dental condition and to demonstrate this using a mirror or video camera. Subsequent to this, the sufferer may impart further information regarding the cause of these findings. Dental management of the bulimic patient may involve the following:

1 Emergency care.
2 Patient education.
3 Pre-restorative care.
4 Restorative care.
5 Maintenance and review.

Emergency care

Reasons for bulimia patients to attend a dentist, other than for routine review, are because of pain or because of deteriorating dental aesthetics. If the patient attends in pain, diagnosis and treatment of this is the first step in management. The patient may complain of sensitivity, and this may be treat-

ed, initially by protection of the exposed dentine by glass-ionomer material, desensitisation using fluoride varnishes, or endodontic treatment as appropriate.

Patient education/pre-restorative care

Bulimic patients who have poor oral hygiene may present with evidence of extensive carious attack as a result of the intake of large quantities of high carbohydrate foods. Such patients will require oral hygiene instruction alongside dietary advice. For those sufferers whose teeth have not suffered carious attack, the main thrust of patient education should be directed towards increasing the patient's awareness of their problem and the extent of the damage to their dentition. The patient should also be made aware of the need for acid neutralisation by use of antacid mouth rinses such as sodium bicarbonate or magnesium hydroxide after vomiting. In the absence of these, the mouth should be washed out with water. Also, the daily use of neutral sodium fluoride mouthrinses (0.05%), daily brushing or application of a stannous fluoride gel (0.4%) have been suggested as being useful in reducing erosion and the associated sensitivity.[28,38]

The use of a plastic splint lined with magnesium hydroxide has also been used to protect and buffer the teeth during periods of vomiting.[39] Normal oral hygiene measures appear adequate for the maintenance of a satisfactory periodontal condition in bulimic patients, with the periodontal condition of bulimic patients having been considered to be close to normal.[23,30] However, patients should be advised to avoid toothbrushing immediately after purging, as the presence of acid on the tooth surface may predispose to toothbrush abrasion. In patients who have reduced salivary flow, the use of sugar-free chewing gum may be advised.

Restorative care

It has previously been considered that restorative treatment should not be provided for the bulimic patient until the patient has controlled the habit.[40] However, it may be considered that there may be definite psychological benefits in improving dental comfort and also appearance, by providing definitive treatment before the patient has achieved full control of the binge–purge behaviour. The decision to commence treatment should be made in consultation with the patient's other carers after weighing the advantages and risks. Nevertheless, it is appropriate to inform the patient that treatment prognosis will be prejudiced if the bulimic habit is not controlled.

Formulation of a treatment plan

Following oral examination, assessment of radiographs and the results of special tests, a treatment plan may be formulated. Study casts should be examined to assess occlusal relationships, but may also be utilised to monitor future wear. Depending on the severity of the tooth surface loss, treatment may range from the provision of simple restorations, to complex oral rehabilitation for patients in whom there has been severe destruction and/or loss of vertical dimension. Treatment will, in general, be directed towards coverage of exposed, worn dentine to reduce sensitivity and prevent further wear. While full crown restorations may be considered to be a traditional well-tested approach, newer restorations using an adhesive approach have been suggested, such as palatal porcelain veneers,[10,41] etched metal onlays or oxidised gold veneers.[43] A major difficulty facing the restorative dentist may be overclosure following the loss of tooth substance, and, in this respect, the use of minimal preparation, dentine-bonded, all-ceramic crowns may be appropriate.[44] The placement of crown/veneer margins subgingivally may be appropriate for patients whose teeth are still likely to be subjected to acid attack, but this must be weighed against the effect on the periodontal tissues and the difficulties in maintaining a dry field in such areas if adhesive restoration are being placed.

Conclusions

Worldwide, the incidence of eating disorders appears to be increasing, and, accordingly, the general dental practitioner, given the preponderance of dental signs and symptoms, may be likely to be the first healthcare worker to make a diagnosis and to provide appropriate advice and treatment. It is therefore essential that the dentist is aware of these signs and symptoms and is thereby able to manage, with other health care workers, the holistic care of the patient, given the potentially life threatening nature of eating disorders.

References

1 *Eating disorders*. Norwich: The Eating Disorders Association, 1989.
2 Russell G F M. Bulimia nervosa: an ominous variant of anorexia nervosa. *Psychol Med* 1979; **9:** 429–448.
3 Morton R. Phthisiologia, seu. *Exercitationes de Phthisi* 1689. Smith, London.
4 Halmi K A. Anorexia nervosa: recent investigations. *Annu Rev Med* 1978; **29:** 137–148.
5 Cowan R D, Sabates C R, Gross K B W, Elledge D A. Integrating dental and medical care for the chronic bulimia nervosa patient: a case report. *Quintessence Int* 1991; **22:** 553–557.
6 Blackman P (ed). *Mishnayoth Order Moed*. Vol. 2. p 310. New York: Judaice Press Inc., 1965.
7 Miles D A, Gregg B E, Glass B J, Van Dis M L. Bulimic erosion. Dental management and report of cases. *J Canad Dent Assoc* 1985; **10:** 757–760.
8 Milsevic A, Slade P D. The orodental status of anorexics and bulimics. *Br Dent J* 1989; **167:** 66–70.
9 Bailey S. Diagnosing bulimia. *Am Fam Physician* 1984; **29:** 161–164.

10 Bruch, H. *Eating disorders. Obesity, anorexia nervosa and the person within*. London: Routledge and Kegan Paul, 1974.
11 Hsu G. *Eating disorders*. New York: The Guildford Press, 1990.
12 American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 3rd Ed. Washington: American Psychiatric Association, 1987.
13 Szmukler G, McCance C, McCrone L, Hunter D. Anorexia nervosa: a psychiatric case register study from Aberdeen. *Psychol Med* 1986; **16:** 49–58.
14 Cooper P J, Fairburn C G. Binge-eating and self-induced vomiting in the community: a preliminary study. *Br J Psychiatry* 1983; **142:** 139–144.
15 Pyle R, Mitchell J, Eckert E, Halverson P, Neumann P, Goff G. The incidence of bulimia in freshman college students. *Int J Eating Disorders* 1983; **2:** 75–85.
16 Herzog D, Copeland P. Eating disorders. *N Engl J Med* 1985; **313:** 295–302.
17 Halmi K A, Folk J R, Schwartz E. Binge-eating and vomiting: a survey of a college population. *Psychol Med* 1981; **11:** 697–706.
18 Roff J C, Koch M O, Perkins S. Bulimia: dentomedical complications. *Gen Dent* 1992; **40:** 22–25.
19 Johnson C, Larson R. Bulimia: an analysis of moods and behaviour. *Psychosom Med* 1982; **44:** 341.
20 Brisman J, Siegel M. The bulimia workshop: a unique integration of group treatment approaches. *Int J Group Psychother* 1985; **35:** 585–681.
21 Garner D, Bemis K. A cognitive-behavioural approach to anorexia nervosa. *Cognitive Ther* 1982; **6:** 123–150.
22 Garner D, Garfunkel P, Schwartz D, Thompson M. Cultural expectations of thinness in women. *Psychol Rep* 1980; **47:** 483–491.
23 Roberts M W, Li S-H. Oral findings in anorexia nervosa and bulimia nervosa: a study of 47 cases. *J Am Dent Assoc* 1987; **115:** 407–410.
24 Hudson J, Pope H, Jonas J. Family history study of anorexia nervosa and bulimia. *Br J Psychiatry* 1983; **142:** 133–138.
25 Collins G, Katz M, Janasz J, Messina M, Ferguson C. Alcoholism in the families of bulimic anorexics. *Cleve Clin Q* 1985; **52:** 65–67.
26 Slade P D. Towards a functional analysis of anorexia nervosa and bulimia nervosa. *Br J Clin Psychol* 1982; **21:** 167–179.
27 Abrams R, Roff J. Oral signs and symptoms in the diagnosis of bulimia. *J Am Dent Assoc* 1986; **113:** 761–764.
28 Hurst P S, Lacey J H, Crisp A M. Teeth, vomiting and diet: a study of the dental characteristics of seventeen anorexia nervosa patients. *Postgrad Med J* 1977; **53:** 298–305.
29 Gandour M. Bulimia: clinical description, assessment, etiology and treatment. *Int J Eating Disorders* 1984; **3:** 3–38.
30 Burket L W. *Oral medicine*. 6th Ed. pp 195–202. Philadelphia: J B Lippincott Co, 1971.
31 Hellstron I. Oral complications in anorexia nervosa. *Scand J Dent Res* 1977; **85:** 71–86.
32 Holst J J, Lange F. Perimylolysis. *Acta Odontol Scand* 1939; **1:** 36–48.
33 Bargen J A, Austin L T. Decalcification of teeth as a result of obstipation with long continued vomiting. Report of a case. *J Am Dent Assoc* 1937; 24: 1271–1273.
34 Kleier D J, Aragon S B, Averbach R E. Dental management of the chronic vomiting patient. *J Am Dent Assoc* 1984; **108:** 618–621.
35 Eccles J D, Jenkins W. Dental erosion and the diet. *J Dent* 1974; **2:** 153–159.
36 Harrison J L, Roeder L B. Dental erosion caused by cola beverages. *Gen Dent* 1991; **39:** 23–24.
37 Stafne E C, Lovestedt S A. Dissolution of tooth substances by lemon juice, acid beverages and acids from other sources. *J Am Dent Assoc* 1947; **34:** 586–592.
38 Barkmeier W W, Peterson D S, Wood L W. Anorexia nervosa: recognition and management. *J Oral Med* 1982; **37:** 33–36.
39 Rowe A H. A palliative treatment for severe enamel erosion. *Br Dent J* 1972; **133:** 435–436.
40 Wolcutt R B, Yager J, Gordon G. Dental sequelae to the binge-purge syndrome (bulimia): report of cases. *J Am Dent Assoc* 1984; **109:** 723–725.
41 Milosevic A. The use of porcelain veneers to restore palatal tooth loss. *Restor Dent* 1990; **6:** 15–18.
42 Boksman L, Gratton D R, Burgoyne A R, Poltzke O B. The treatment of perimolysis using resin bonded etched metal onlays. *Quintessence Int* 1986; **17:** 69–74.
43 Darber U R. The treatment of palatal erosive wear by using oxidized gold veneers: a case report. *Quintessence Int* 1994; **25:** 195–197.
44 Burke F J T, Qualtrough A J E, Hale R W. The dentine-bonded all-ceramic crown: an ideal restoration. *Br Dent J* 1995; **179:** 58–63.

Guest Leaders

Guest leaders in the BDJ are there to provide an opportunity for anyone involved in dentistry (including patients) to write an appropriate comment for publication. These are published to accompany the usual Leader from the Editor in each issue of the Journal.

Submissions must be between 200 and 500 words, typed and double-spaced. Name, address and telephone number should be supplied, as well as position in the dental world. For further help and guidance please contact: The Editor, British Dental Journal, 64 Wimpole Street, London W1M 8AL (071-935 0875).