Dental erosion patterns from intrinsic acid regurgitation and vomiting

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Abstract

Background: The distribution of lesions from dental erosion due to intrinsic acid regurgitation and vomiting may be different from patterns of dental erosion due to extrinsic acids. To date studies have failed to validate this assumption. This study described the sites and nature of lesions from dental erosion in cases of intrinsic acid regurgitation, and compared them with the distribution of lesions occurring in age and sex matched controls, whose lesions are due to extrinsic acids.

Methods: The University of Queensland tooth wear clinic patients were screened to select 30 cases, 21 self-identified bulimics and nine medically diagnosed chronic gastric acid regurgitators, and 30 controls. Epoxy resin models of the subjects’ dentition were examined under stereoscopic light microscope at magnification 16 to 40. The patterns and sites of tooth wear were recorded for teeth representative of 20 tooth sites in every subject.

Results: While the incisal edges of maxillary and mandibular anterior teeth of acid regurgitators were more frequently affected by erosion, incisal attrition was more common on controls’ teeth. Cervical lesions were more commonly found in association with incisal attrition in the controls, and in association with incisal erosion in the cases. In 10 per cent of sites in case subjects, cervical lesions associated with incisal erosion were found on the lingual aspects of their mandibular incisors, canines and premolars. These lesions were almost exclusive to the case subjects.

Conclusions: These results validate that lingual cervical lesions associated with incisal erosion on the mandibular anterior teeth are strong discriminating between tooth wear in patients with bulimia nervosa or chronic gastro-oesophageal reflux and those whose dental erosion is due to extrinsic acids.

Key Words: Bulimia nervosa, dental erosion, gastro-oesophageal reflux, tooth wear.

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INTRODUCTION

While diet is considered by some to be the most important cause of dental erosion, the risk of erosion due to the effect of intrinsic acids has long been recognized and studied.1 Theoretically, all disorders associated with gastric acid reaching the oral cavity, may result in demineralization of the hard dental tissues. However, the clinical manifestations of dental erosion by intrinsic acids appear to occur only after several years of repeated exposure.1 Consequently, dental erosion induced by intrinsic factors has only been observed to date in disorders associated with persistent acid regurgitation and chronic vomiting. These include gastro-oesophageal reflux disease (GORD),1 chronic alcoholism,2 and bulimia nervosa.1,3

Gastro-oesophageal reflux disease is probably the most common gastrointestinal disorder with approximately 40 per cent of the general population complaining of reflux symptoms at some time.4 In GORD, the gastric contents reflux into the oesophagus, in most cases as a consequence of an incompetent lower oesophageal sphincter.5 Severe reflux that reaches the pharynx and the oral cavity is distinguished from vomiting by the relatively small amount of material ejected, and by the lack of diaphragmatic muscular contraction.1 The major symptoms of GORD include waterbrash (a filling of the mouth with saliva), epigastric pain and heartburn.5 Aside from these symptoms, GORD has been implicated as a likely contributor to tooth wear.6 In particular, the relationship between palatal dental erosion and severe GORD has been repeatedly described in dental literature.7,8 A possible mechanism suggested to explain the high susceptibility of the palatal surfaces is, that the force of regurgitation passing from the pharynx into the mouth propels the gastric juice forward and causes damage to the palatal surfaces of the maxillary teeth more commonly.4 In addition, it is argued, the palatal surfaces are also relatively remote from the major salivary glands and the tongue is involved by maintaining contact of the gastric acid against the palatal surfaces.4 In contrast, the lower teeth are not affected in the early stages of the erosive process as the tongue provides protection.5 Despite the high
prevalence of GORD, in many individuals the condition remains undiagnosed and under treated for several years. This is because the majority of people experience only mild reflux symptoms, which are commonly tolerated, or experience no GORD symptoms at all. Bartlett et al. introduced the term ‘silent refluxers’ to describe such patients, and have suggested that, in the absence of reflux symptoms, the oral manifestations of GORD may be the only clinical sign that pathological reflux is occurring in these patients.

Chronic alcoholism is another condition that reportedly produces a similar erosive pattern. A significantly higher incidence of erosive tooth wear was reported in chronic alcoholics compared to the general population, specifically on the palatal surfaces of the maxillary anterior teeth. Hede found associations between the severity of dental erosion in chronic alcoholics and the duration of alcoholic abuse. In general, regurgitation of gastric juice into the oral cavity is believed to be the underlying cause of dental erosion in alcoholics, as long-term alcohol abuse leads to chronic gastritis, which in turn can cause recurrent vomiting and/or gastro-oesophageal reflux. Bulimia nervosa is a psychosomatic disorder characterized by the patient’s inability to exert continuous control over their voluntary food intake. Patients suffering from bulimia nervosa often indulge in binge eating, followed by various compensatory mechanisms such as purging, vigorous exercise or fasting. The most common form of purging is self-induced vomiting. Overall, the reported oral health of patients with diagnosed eating disorders is poorer than that of the general population, with higher frequencies of carious lesions and erosive tooth wear. Some prevalence studies suggest that up to 90 per cent of patients with bulimia nervosa are affected by dental erosion. Stege et al. reported extensive lingual erosion on both maxillary and mandibular incisors as the most significant intra-oral pathology in their case study of a 24-year-old patient with bulimic anorexia. Specifically, the ‘polished’ appearance of the wear facets was attributed to a combination of chemical and mechanical action of the gastric acid retained in the filiform papillae of the tongue, which mechanically polished the teeth. Occlusal erosion on the posterior teeth and ‘dished out’ erosion lesions on the mid-facial surfaces of both anterior and posterior teeth have also been reported as common in bulimic patients.

Predisposing or causal factors such as diminished salivary flow rate, decreased buffering capacity of stimulated saliva and xerostomia are also common to bulimia nervosa, chronic alcoholism and GORD patients. In bulimia nervosa the misuse of laxatives by the patient and the medication used in the treatment of eating disorders can potentially cause electrolyte disturbances and a decreased total volume of saliva. In some GORD patients, gastric contents are regurgitated at night when salivary flow is at its lowest. In addition, GORD and gastric acid regurgitation commonly occur in older patients who may have drug-induced xerostomia, or in alcoholics, in whom the dehydrating action of alcohol reduces salivary flow. In this context, the importance of salivary protection against gastric acid must be taken into account when considering the site specificity and the severity of erosive lesions found in gastric acid regurgitators.

As outlined above, it is widely accepted that persistent GORD, chronic alcoholism and bulimia nervosa produce similar erosive patterns at similar locations. However, in a report on a group of 19 cases of erosion, Eccles noted that erosion from extrinsic sources also affect the palatal surfaces of the anterior maxillary teeth, leading to flat, hollowed-out surfaces and thin incisal edges. Similar erosion patterns were reported by Smith and Shaw describing the erosive effect of baby fruit juices, Asher and Read, who studied erosion due to acidic soft drinks; and Fuller and Johnson, who described palatal tooth wear in a dedicated tequila drinker. To date, dental research has failed to validate the assumption that the distribution and the nature of lesions from dental erosion due to intrinsic acids are significantly different from patterns of dental erosion that result as a consequence of extrinsic acids in the diet.

The ability to differentiate between the lesions resulting from intrinsic and extrinsic acids undoubtedly has significant application in dental practice. The ability to predict the cause of dental erosion reliably from the nature and the distribution of the lesions would serve as a valuable diagnostic tool for both bulimia nervosa and gastro-oesophageal reflux. Such an objective diagnostic tool would be especially important in those cases, where the patient is unaware of the symptoms, or unwilling to disclose them. By increasing the precision with which a diagnosis of intrinsic acid regurgitation can be made, on the basis of tooth wear alone, appropriate preventive measures could then be employed that would improve both the oral and general health of these patients.

It was therefore the aim of this study to describe the sites and nature of lesions from dental erosion in subjects known to be the cases of bulimia and of intrinsic acid regurgitation, and to compare these cases with the distribution of lesions occurring in age and sex matched control subjects, whose lesions are due to extrinsic acids.

**METHODS AND MATERIALS**

Both case and control subjects were identified from the 400 patients referred to The University of Queensland Tooth Wear Clinic between the years 1988 and 1999 by dental practitioners for the investigation of tooth-wear. The structured interview and examination of each patient allowed positive or negative responses to be established for items of history and examination pertaining to:

- History, signs and symptoms of chronic acid regurgitation.
• History, signs and symptoms of bulimia nervosa with chronic vomiting.

History of gastric acid regurgitation was ascertained by positive responses to the following questions: do you suffer from indigestion, heartburn, glossodynia, sour mouthfuls or a ‘vomit-like’ taste in your mouth on waking? Have you ever had gastritis or hiatus hernia investigated by endoscopic or radiologic assessment? Do you take any medication for these complaints? Cases were not selected on the basis of alcohol consumption.

History of bulimia nervosa and of frequent vomiting was ascertained by positive responses to questions: have you ever had an illness that was accompanied by persistent vomiting? Was this vomiting self-induced? Do you know what bulimia nervosa is? Have you ever suffered from this illness?

Further, the medical history investigated other systemic conditions that could be used as a guide in the clinical diagnosis of bulimia nervosa. The nutritional status of the patients and their past and present weight were recorded. The incidence of amenorrhoea in female patients and excessive sensitivity to hypothermia were also noted. Further enquiries investigated the presence of xerostomia or of medications affecting salivation. Details of situations and times of day when dry mouth was experienced were also elicited. Psychological evaluation of each patient was performed during which a history of anxiety, depression and the use of medications for these complaints were specifically addressed. During the physical examination, in addition to the examination of the teeth, signs of oral candidiasis, salivary gland enlargement, firmness or tenderness and of facial lanugo were recorded.

Based on the information obtained from the structured interview and examination, a group of 30 subjects was identified. Of these, nine subjects were self-identified bulimics and 21 were medically-diagnosed chronic regurgitators of gastric acid. In the bulimic group, all subjects were female. Their ages ranged from 19 to 51 years old, with an average age of 32. In the group of acid regurgitators, there were seven female and 14 male subjects. Their ages ranged from 30 to 75 years with an average age of 55. For each subject, a control was selected from the same population of tooth wear patients. All controls were sex-matched and their ages were approximated to the ages of the subjects (± two years). All subjects in the study and the reference groups had been examined and interviewed by the same clinician (WGY). Where possible, it was also ensured that the structured interview and examination was performed on both the subject and the control within a time frame of two years. None of the controls had positive responses to the questions pertaining to the history, signs and symptoms of chronic acid regurgitation or bulimia, neither was there any systemic indications of these conditions. None of the controls reported being alcoholic or having a history of chronic alcoholism. Both case and control subjects were also investigated for other aetiologies of dental erosion. In particular, the level of exposure to dietary acids and the possibility of salivary gland hypofunction due to occupation or sport’s induced dehydration were determined. In most control subjects, excess consumption of dietary acids in soft drinks, under dehydrating climatic conditions, was clearly identified as the cause of dental erosion.

For all case and control subjects, silicone elastomeric impressions were taken during physical examination and their replicas were poured in clear epoxy-resin. With no knowledge of clinical information or the case/control group, the epoxy resin models of the dentitions of all subjects and controls were examined by one of the authors (VV) under a stereoscopic light microscope at magnifications between 16 and 40. The patterns and sites of tooth wear were recorded for teeth representative of 20 tooth sites in every subject. The teeth chosen for recording were the permanent maxillary and mandibular central incisors, canines, first and second premolars and first molars. To validate these detailed observations, selected type-specimens of tooth erosion patterns were examined using the scanning electron microscope at magnification of 20-23.

Examination of the replicas enabled discriminations to be made between wear facets due to attrition, and tooth-tissue loss due to erosion, on the basis of the contours of enamel to dentine interfaces on the occlusal and incisal surfaces of the examined teeth. Attritional tooth wear was characterized by planar enamel wear and shallow dentine wear. Attritional facets on anterior teeth were characterized by flat planes of wear with well-defined enamel margins. On the posterior teeth, the wear planes were often contoured in the direction of relative tooth-to-tooth movement in accordance with the Greaves effect, with equivalent facets being found on all opposing teeth. In contrast, tooth wear due to erosion was characterized by deeply cupped dentine loss on incisal or occlusal surfaces with sharp and chipped enamel edges. The enamel/dentine interfaces were generally not contoured in accordance with the Greaves effect. The absence of tooth wear into dentine on the occlusal and incisal surfaces of the examined teeth was classified as ‘Nil’.

The presence of non-carious cervical lesions, in association with either occlusal/incisal attrition or erosion, was recorded as C+, whereas the absence of a cervical lesion was recorded as C-. As previously described, these cervical lesions showed variable degrees of severity; shallow, grooved or wedge-shaped, or were restored. Lesions, which involved generalized loss of lingual enamel and dentine that merged with occlusal pathology, were included (D lesions).

Restored teeth were included only in those cases where the restorations did not obscure the occlusal pathology. Several teeth with full coverage of occlusal and/or cervical surfaces were excluded. In this study the associations between occlusal/incisal wear pattern and cervical lesions were recorded for a total of 1952 tooth sites. Results describing the patterns and sites of tooth sites.
wear for each case and control subject were recorded by data spreadsheet and subjected to chi-square analysis for case-control comparisons.

RESULTS

The percentages of sites affected by associations of occlusal and cervical tooth wear on the buccal aspects of maxillary and mandibular incisors, canine, premolar and molar teeth in acid regurgitating subjects (black) and their age- and sex-matched controls (grey). Tooth wear patterns were classified as 'C- Attrition' for occlusal attrition in the absence of cervical lesions; 'C+ Attrition' for occlusal attrition associated with cervical lesions; 'C- Erosion' for occlusal erosion in the absence of cervical lesions; 'C+ Erosion' for occlusal erosion associated with cervical lesions and 'Nil' for no occlusal wear with cervical lesions. Degrees of significance between subjects and controls determined by chi-square analysis were indicated by * for p<0.05 and ** for p<0.01.

In general, incisal attrition, characterized by flat planes of wear with well-defined enamel margins and complementary facets on opposing teeth, was found more commonly on control subjects’ teeth. Specifically, shallow, grooved or wedge-shaped cervical lesions, or restorations, were more commonly found in association with incisal attrition in the control subjects, especially on the palatal surfaces of maxillary canine and premolar teeth, and on the lingual aspects of mandibular incisors, canine and premolar teeth (Fig 2).

Maxillary teeth

Maxillary central incisors and canines in case subjects were significantly more affected by incisal erosion in the absence of cervical lesions than were those of the controls (p<0.01). In those upper incisors affected by erosion associated with cervical wear either facially or palatally, no significant differences were found between cases and controls. However, palatal cervical lesions associated with incisal erosion were much more common on maxillary canines and premolars of the cases than of the controls (p<0.01). Seventy per cent of the lesions found on the palatal surfaces of cases’ maxillary canines were generalized loss of lingual enamel and dentine involving the incisal edge. The majority of lesions found on the palatal surfaces of cases’ maxillary premolars were shallow cervical lesions.

In contrast, incisal attrition on maxillary incisors and canines with associated buccal and palatal cervical lesions was significantly more common in the control subjects (p<0.05). The majority of these cervical lesions were shallow cervical lesions.

In the absence of occlusal pathology, only two differences were found in the sites where the numbers
of cervical lesions were different between case and control subjects. Firstly, significantly more cervical lesions without incisal pathology were found on the palatal aspects of controls’ maxillary incisors (p<0.01). Secondly, 5 per cent more cervical lesions in the absence of occlusal pathology were found on the palatal aspects of case subjects’ maxillary molar teeth (p<0.05). These differences were almost exclusively due to the numbers of palatal restorations found at these sites.

Mandibular teeth

Mandibular central incisors, canine and premolar teeth of control subjects were found to have significantly more examples of incisal attrition in the absence of cervical wear (p<0.05). In contrast, 3 per cent more cervical lesions in the absence of occlusal pathology were found on the palatal aspects of case subjects’ maxillary molar teeth (p<0.05). These differences were almost exclusively due to the numbers of palatal restorations found at these sites.

Lingual cervical lesions associated with incisal erosion were almost exclusively found on the mandibular incisors, canine and premolar teeth of the case subjects (p<0.01). All of the lesions found on the lingual surfaces of cases’ mandibular incisors involved generalized loss of lingual enamel and dentine, while 40 per cent had shallow cervical lesions (Fig 4). The majority (56 per cent) of sites affected on the lingual surfaces of case subject’s mandibular premolars were shallow cervical lesions. No differences between case and control subjects were found in the distribution of buccal cervical lesions on any of the mandibular teeth.

Figure 5 illustrates a wear facet on the incisal edge of a mandibular incisor with evidence that erosion has destroyed the lingual enamel edge of the facet, and has exposed the dentine on the lingual proximal edge. Examples of such lingual extension of occlusal pathology were found almost exclusively on the mandibular incisors, canine and premolar teeth of bulimic subjects. In cases of chronic reflux, the involvement of entire lingual surfaces of mandibular incisors and canine teeth was found more commonly (Fig 6).

DISCUSSION

Subjects involved in this study were not a random sample from the general population but patients specifically referred to the clinic for investigation of tooth wear. During case selection for this study, it was ensured that only those patients who self-identified as bulimic or had a medically confirmed diagnosis of an upper gastro-intestinal disorder were included. Moreover, patients who denied being bulimic or acid
regurgitators, and yet presented with systemic signs or symptoms indicative of bulimia, GORD or chronic alcoholism were excluded from the control group. While the choice of these criteria ensured a low possibility of selecting a case subject without bulimia or GORD, it also introduced a selection bias to the study. It may be hard to identify cases of bulimia nervosa within a sample of tooth wear patients, mostly because the social stigma associated with eating disorders in Western culture makes disclosure difficult for the patient. Moreover, denial of the illness and negative attitudes towards somatic and psychiatric treatment are underlying characteristics in bulimia nervosa. The above factors make self-identification a very restrictive selection criterion for a study involving bulimic cases. This may have the tendency to underestimate the size of the case group among tooth wear patients. Because of the relatively small size of the case group it was impossible to draw conclusions about possible differences in associations between incisal/occlusal and cervical wear pathology in bulimic patients and in patients with chronic GORD.

Three subjects with medically diagnosed GORD self-identified as being chronic alcoholics. As this study was
not concerned with the effects of chronic alcoholism *per se*, and because, similar to bulimia, disclosure of chronic alcoholism is difficult for the patient, it was not possible to determine whether case subjects suffering from bulimia nervosa or GORD were also alcoholics. Similarly, no information was elicited concerning the type of alcohol consumed by the three case subjects. It is also important to emphasize, that clinical signs of both bulimia nervosa and gastro-oesophageal reflux disease are relatively subtle and non-specific, and both disorders can often progress undiagnosed and untreated for several years. As a consequence, it is possible that some control subjects within this study were in fact bulimic, chronic alcoholics or experienced ‘silent’ gastro-oesophageal reflux, without being aware of their condition or choosing not to disclose it. Further studies on the site specificity of mandibular lingual lesions should therefore be extended to differentiate bulimic from alcoholic subjects, and to examine the influence of different types of alcohol and salivary gland function on dental erosion.

Figure 7 compares, in summary, the significant differences in associations between incisal/occlusal and cervical wear pathology, in cases of intrinsic acid regurgitation and in control subjects, whose dental erosion is due to extrinsic acids. This comparison shows that while the incisal edges of both maxillary and mandibular anterior teeth of acid regurgitators and bulimics were more frequently affected by erosion, incisal attrition was much more common on control subjects’ teeth. This suggests that in cases, where gastric acid regurgitation or vomiting occur regularly over many years, the lesions of dental erosion become the characteristic type of hard dental tissue loss. In contrast, in controls, it is attritional wear of the hard tissues softened by extrinsic acid that is the predominant presenting occlusal pathology. Perhaps extrinsic acids, such as citric and orthophosphoric acids from soft drinks predispose to attrition and erosion, whereas gastric hydrochloric acid more commonly produces erosion; for non-carious cervical lesions and restorations were found in association with incisal attrition in the control subjects, and in association with incisal erosion in the cases.

One specific site where the results showed no significant differences between cases and controls, in terms of erosion associated with cervical lesions, was the palatal aspect of maxillary incisors. This erosive pattern was found to be relatively common to both cases and controls, affecting approximately 20 per cent of maxillary palatal surfaces of anterior teeth surveyed in this study. Other studies, documenting the influence of gastric disturbances on hard dental tissues, have suggested a progressive pattern of erosion, in which the palatal surfaces of the maxillary anterior teeth are usually first affected, followed by the involvement of occlusal surfaces of molars and premolars and of labial surfaces of maxillary incisors in more advanced cases. However Jarvinen et al. have shown that while this pattern of erosion is prevalent, it is unrelated to etiological factors. As the present study found no significant differences between cases and controls, in terms of cervical wear either facially or palatally, palatal erosion on maxillary incisors is not a reliable indicator of intrinsic acid regurgitation. However, significant differences of erosion associated with palatal cervical wear were found further along the maxillary arch, on both the canine and premolar teeth between cases and controls. In the cases, the majority of lesions that were found on the palatal surfaces of maxillary
canines involved their entire lingual surfaces. Moreover, the majority of lesions found on the palatal surfaces of maxillary premolars were shallow cervical lesions. These differences found between the extent of lesions on canine and premolar teeth suggest a pattern of progression of erosive tooth wear from anterior to posterior regions. It may also reflect greater protection of premolar and molar palatal surfaces by parotid saliva.

Whereas some authors dispute any relation between the underlying cause and the site specificity of erosive lesions,19 this study found a significant relationship between chronic vomiting or persistent gastro-oesophageal reflux and lingual erosion on the mandibular anterior teeth. The most impressive finding of this study was that incisal erosion associated with cervical lesions on the lingual aspects of mandibular incisors, canine and premolar teeth, was found exclusively in 10 per cent of the sites of cases. This finding is contrary to other studies describing the patterns of erosion in acid regurgitators, which reported the lower teeth were largely undamaged.1

Similarly to palatal cervical lesions associated with occlusal erosion in the maxilla, mandibular cervical lesions were also found to range in extent from generalized loss of lingual enamel and dentine (D lesions) on incisors to shallow cervical lesions on canines and premolars. Such lingual cervical lesions were almost exclusive to the case subjects, as only 1 to 2 per cent of the lingual surfaces of premolars and molars in controls were affected. The only example of generalized loss of lingual enamel and dentine found on lingual aspects of mandibular incisors in a control subject, was a 59-year-old female who gave a history of daily consumption of freshly-squeezed lemon juice for health reasons. However, in this instance, her cervical lesions were associated with incisal attrition rather than erosion as is common with the cervical lesions of cases. These results indicate that, where found, lingual cervical lesions associated with incisal erosion on mandibular anterior teeth are strong discriminators between the tooth wear pattern of patients with bulimia nervosa and GORD and those whose dental erosion is due to extrinsic acids.

There has been speculation on the factors responsible for the site specificity observed in dental erosion. The principle sites of contact of the erosive agent, protection of some sites by the tongue and the adverse effect of

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**Fig 7.** A comparison of the significant differences in associations between incisor and cervical wear pathology found in cases of intrinsic acid regurgitation versus control subjects with extrinsic acid erosion. The lowest degrees of significance found between subjects and controls were indicated by * for p<0.05 and ** for p<0.01.
anatomical structures contacting the teeth have all been cited.17,18 Salivary secretion is also thought of central importance in influencing both the severity and site-specificity of erosive lesions. The importance of unstimulated salivary flow rate in determining the occurrence and progression of dental erosion has been pointed out by various authors, including Hellström15 and Jarvinen et al.16 These authors found the flow rate of unstimulated saliva was significantly lowered in erosion patients suffering from upper gastrointestinal disorders and eating disorders. The buffering capacity of saliva may also be an important variable that differs from patient to patient. The mean bicarbonate concentration for stimulated saliva has been found to be significantly lower in bulimic patients with dental erosion, when compared to bulimics without tooth tissue loss.18 In addition, the variation in the thickness of salivary pellicle around the dental arches in normal subjects has been shown to be of importance in determining the sites and severity of dental erosion.20 In their study, Amaechi et al.21 showed that in five normal subjects the thickest pellicle occurred at the posterior and anterior lingual surfaces mandibular teeth, while the thinnest pellicle was formed at the upper anterior palatal surface. Further, an inverse relationship was found between the depth of erosion and the thickness of the pellicle in vitro. The degrees of erosion reported corresponded inversely to the pellicle thickness at these sites, with the levels of erosion in the anterior palatal region of the maxilla being significantly greater than the level of erosion found in the lingual region of mandibular anteriors. This suggests that the thickness of the acquired pellicle is an important factor for the site specificity of dental erosion, and explains the minimal levels of erosion found on the lingual aspects of mandibular incisors in the control subjects of this study.

In addition, these findings raise questions about other properties of saliva, serous versus mucous, that might influence the protective effect of pellicle against tooth demineralization in bulimic patients, in whom 10 per cent of the lingual aspects of mandibular incisors are affected by serious erosion. In this context, it is interesting to postulate that clinical changes that occur in the salivary glands of long-term bulimics could result in reduced salivary protection of their teeth. Reportedly, serous acini of the parotid gland are more susceptible to metabolic disturbances than the mucinous acini of the submandibular, sublingual and minor salivary glands and episodic bilateral parotid gland enlargement has been reported in bulimic patients.22 Reduced parotid gland function in bulimia nervosa may manifest as reduced serous output and a reduced bicarbonate contribution to whole saliva. These, in turn, may affect the protective properties of acquired salivary pellicle.

In conclusion, where found, lingual cervical lesions associated with incisal erosion on the mandibular anterior teeth are strong discriminators between the tooth wear pattern of patients with bulimia nervosa and persistent gastro-oesophageal reflux from those whose dental erosion is due to extrinsic acids. It appears that both the sources and type of acid and the degree of saliva protection of the sites are responsible for this most rare form of dental erosion found in bulimics and chronic gastric acid regurgitators.

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