

Eating disorders and oral health: A review of the literature

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Abstract

This article is a review of the recent literature pertaining to the oral sequelae of eating disorders (EDs). Dentists are recognized as being some of the first health care professionals to whom a previously undiagnosed eating disorder patient (EDP) may present. However, despite the prevalence (up to 4 per cent) of such conditions in teenage girls and young adult females, there is relatively little published in the recent literature regarding the oral sequelae of EDs. This compares unfavourably with the attention given recently in the dental literature to conditions such as diabetes mellitus, which have a similar prevalence in the adult population. The incidence of EDs is increasing and it would be expected that dentists who treat patients in the affected age groups would encounter more individuals exhibiting EDs. Most of the reports in the literature concentrate on the obvious clinical features of dental destruction (perimolysis), parotid swelling and biochemical abnormalities particularly related to salivary and pancreatic amylase. However, there is no consistency in explanation of the oral phenomena and epiphenomena seen in EDs. Many EDPs are nutritionally challenged; there is a relative lack of information pertaining to non-dental, oral lesions associated with nutritional deficiencies.

Key words: Eating disorders, oral health.

Abbreviations and acronyms: AN = anorexia nervosa; API = Approximal Plaque Index; BN = bulimia nervosa; CPITN = Community Periodontal Index of Treatment Needs; DFS = Decayed, Filled Surfaces; DMFT = Decayed Missing Filled Teeth; DS = Decayed Surfaces; DSM = Diagnostic and Statistical Manual; ED = eating disorder; EDNOS = eating disorders not otherwise specified; EDP = eating disorder patient; ICD = International Classification of Diseases.

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INTRODUCTION

What are eating disorders?

Eating disorders (EDs) are primarily psychological conditions, often with severe medical complications and share the core features of self-evaluation by shape

and weight perception and a desire to be thinner. The Diagnostic and Statistical Manual 4th edn¹ (DSM) classifies EDs as Anorexia Nervosa (AN), Bulimia Nervosa (BN) and Eating Disorders Not Otherwise Specified (EDNOS). The latter is a limited symptom variant of the complete syndrome; typically the individual meets most, but not all diagnostic criteria. The International Classification of Diseases² (ICD) includes AN, BN, atypical AN and BN (similar to EDNOS), vomiting associated with other psychological conditions and psychogenic loss of appetite. Both cite three similar AN diagnostic criteria for post-pubertal individuals: (1) body weight is maintained at less than 85 per cent expected for age; (2) a distorted body image; and (3) an endocrine disorder typified by amenorrhoea. ICD-10 includes a self-induced weight loss criteria achieved by food avoidance, vomiting, purging or exercise. DSM-IV includes an intense fear of weight gain, as well as stipulating that amenorrhoea is for a minimum of three consecutive menstrual cycles.

BN is typified by bingeing: eating large amounts of food and a subjective sense of loss of control. ICD-10 criteria include: (1) a persistent preoccupation with eating; (2) compensating for over-eating with behaviour to decrease calorie intake or expend calories, typically by self-induced vomiting, exercise and laxative abuse; and (3) a morbid dread of becoming fat.

DSM-IV criteria for BN are similar although requiring the recording of binge episodes. DSM-IV also includes the presence of compensatory behaviour and self-evaluation unduly based on shape and weight. In DSM-IV the presence of AN excludes a BN diagnosis.

EDs are often severe conditions with elevated standardized mortality ratios and marked impairment.³ Both AN and BN have a typical onset in late adolescence and early adulthood and are rare from a population perspective. However, they are more prevalent in certain groups. EDs are present in 1-4 per cent of schoolgirls and female university students,⁴ are the third most common chronic illness of teenage girls⁵ and are of increased prevalence in performing arts groups and elite athletes. Isolated EDs-like behaviour is very common, Flament *et al.*⁶ reported 86 per cent of French adolescent girls engaged in self-induced vomiting to control shape and weight at least once.

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Since 1990, a number of papers have been published linking EDs and oral health. Most of this research has been conducted by medical personnel and published in the medical literature. It has been pointed out by a number of authors^{7,8} that in many cases, dentists are one of the first healthcare providers who may come into contact with Eating Disorder Patients (EDPs). Most of the publications concentrate on the effects of EDs on teeth, salivary glands, saliva and serum amylase. There has been a paucity of publications on the latter subjects in the dental literature and comparatively little information is available on the impact of those disorders on other aspects of oral health especially the oral mucosa.

The purpose of this paper was to review current knowledge of ED signs and symptoms from a pan-oral perspective. The subtle changes in the mouth, which should be recognized by the general dental practitioner, when taken as a whole may be utilized as early indicators of a serious underlying psychiatric condition. Recognition of these signs should result in earlier diagnosis, referral and instigation of management of both the underlying condition and the secondary oral conditions in their early stages. Such early recognition should result in more favourable treatment outcomes.

A Medline search was conducted from 1990 to the present using recognized National Library of Congress MeSH headings: EDs, AN, BN and oral health. The search was limited to human studies and the English literature. In total 32 published studies have been reviewed. These included 12 cross-sectional and two prospective studies; nine case reports and nine review articles.

For the purpose of this review, oral health may be defined as the health of the oral and associated perioral structures. For example, the major salivary glands are not strictly oral structures but their secretions and the secretions of the minor salivary glands are vital to oral health. Any analysis of oral health must include alterations of salivary gland function and physiology and functional abnormalities of the mouth including mastication and deglutition. The health of support structures such as bone is also important in normal oral function. Conditions affecting bone may impact on the basal and specialized alveolar bone of the jaws.

Hard tissues

Most work pertaining to EDs and dentistry has been published on the effects on teeth. There is limited information regarding the effects of EDs on the supporting bone.

Dental Effects

Tooth erosion

There is agreement in the literature^{9,10} that enamel erosion is the most common and dramatic oral manifestation of chronic regurgitation typical of EDs (Fig 1). Three cross-sectional studies¹¹⁻¹³ have re-visited the topic of tooth erosion in relation to EDs. Robb



Fig 1. Vomiting induced erosion of the lingual surfaces of maxillary anterior teeth in a patient suffering from anorexia nervosa.

*et al.*¹² conducted a case controlled study of 122 EDPs and 122 age, sex and social class matched controls. The patients were divided into those with AN, with and without vomiting and those with BN. The Tooth Wear Index was recorded scoring cervical, buccal, occlusal and lingual surfaces of all standing permanent teeth on all subjects.

The study showed that subjects who suffered from AN but did not vomit had significantly more erosion than a control group but less than the vomiting groups and the erosion was predominantly on the buccal and occlusal surfaces of posterior teeth. This type of erosion had not previously been associated with EDs. No explanation was put forward to account for this phenomenon. The authors concluded that abstaining anorectic patients should be excluded as controls when looking at tooth wear due to their suffering more tooth wear than the general population.

No relationship was found between vomiting frequency, duration of vomiting, oral hygiene and erosion. This was attributed to different susceptibility to erosion between patients. Differences in buffering capacity of saliva, flow rate, pH and tooth surface composition were proposed. However, the reliability of patients self reporting vomiting may be questioned.

Öhrn *et al.*¹¹ investigated 81 patients with EDs, diagnosed according to DSM-III-R diagnostic criteria. Patient groups included those who suffered from AN, BN, concomitant AN-BN and EDNOS. Patients were compared to 52 age matched healthy volunteers. Tooth erosion was assessed from study models and photographs. Buccal, lingual, occlusal and incisal surfaces were scored. Data from all patients was pooled and analyzed together; there was no separation of ED subtypes. It was found that EDPs generally had more tooth erosion when compared to controls. However, there was no distinction between ED subtypes.

Philipp *et al.*¹³ looked at tooth erosion in 11 individuals with anorexia and 41 with bulimia and compared them to 50 age and sex matched controls. The authors concluded that all patients had more tooth erosion when compared to controls although it is not stated how this was determined. Finally Bidwell *et al.*¹⁴

presented a rare case report of bulimia-induced dental erosion in a 40-year-old male patient.

Several research limitations are noted. Some but not all studies refer to the diagnostic criteria by which EDs are diagnosed.¹²⁻¹⁴ Where the ED may be subdivided into recognizable clinical entities such sub classification is necessary. This is essential in those cases where an habitual feature is involved which has a direct bearing on the teeth. In EDs the distinction between vomiting and non-vomiting subtypes is required. Further, there is no consistency across the reports regarding methodology of dental examinations. Some reports¹¹⁻¹³ detail charting and scoring methods where as others¹⁴ do not. Standardized dental examinations are required for reproducibility and comparison of data.

Omissions such as those highlighted above illustrate the need for standardization in the approach to dental investigations in patients with oral health problems. There needs to be consistency in the diagnostic criteria applied to the systemic problem and there should be consistency in the approach to investigation of the oro-dental problems.

A number of review articles have addressed the issue of tooth erosion.^{7,9,10,15-19} It is generally accepted in those reports that tooth erosion takes about two years to become clinically apparent,^{7,9,10,15,18} although one report states that it may be visible after only six months of vomiting behaviour.¹⁷

Vomiting habits and oral hygiene practices¹⁸ have been proposed to account for the difference seen in erosion in EDPs and controls. However, dental erosion is not a simple phenomenon and other factors may play a role in determining the rate of enamel erosion. Much emphasis has been placed on the role of vomitus as the causative agent of enamel erosion but no account has been taken of the dietary intake in EDPs. This is a surprising omission, as dietary factors are known risks for tooth related pathology.²⁰ It has been shown that the pattern of dental erosion is different depending on whether acids are of extrinsic or intrinsic nature. Patients with BN who consume low acid diets initially exhibit dental erosion confined to palatal and occlusal surfaces of maxillary teeth. However, when acidic drinks such as cola beverages are also consumed, the initial erosion lesions become confined to palatal and buccal surfaces of teeth. As such, the initial lesions of patients with BN cannot be distinguished from non-BN subjects who simply consume large amounts of acid containing beverages.²¹ However, Valena and Young²² have shown that when erosive lesions are found on the lingual aspects of mandibular anterior teeth, they can reliably distinguish between patients with BN or chronic gastroesophageal reflux and those with dental erosion due to extrinsic acids. It may be too simplistic to ascribe all dental effects as being secondary to vomiting when the effects of dietary substances, food and drink and the pattern of their consumption, which are major aetiological factors in pathogenesis of dental hard tissue lesions have not been studied.

Dental caries

Dental caries becomes a problem in individuals whose diet is rich in cariogenic food, who have poor oral hygiene and manifest salivary disturbances.²³ The issue of dental caries has been addressed in relation to EDs in a number of papers^{7,11,13,15,16,18,19,24} with somewhat conflicting conclusions.

In a cross-sectional study Touyz *et al.*²⁴ looked at 15 females with BN, 15 with AN and 15 controls. All subjects had Decayed Missing Filled Teeth (DMFT) indices recorded and the levels of known cariogenic micro-organisms *Streptococcus mutans* and *Lactobacillus*. No differences in the levels of *S. mutans* or *Lactobacillus* were found among the three groups and there was no difference in caries prevalence. This is in contrast with the results of two other cross-sectional studies by Philipp *et al.*¹³ and Öhrn *et al.*¹¹

In Philipp *et al.*¹³ case controlled study cross-sectional DMF values were recorded for all subjects. Statistically significant lower DMF values were reported in the EDPs when compared to controls, with AN patients scoring statistically significant lowest values. The authors attributed differences in DMF values to better oral hygiene in the EDPs due to their vomiting. There is no discussion of the mechanism whereby vomiting may improve oral hygiene. There was no report of the subtypes of AN involved and whether vomiting was a factor.

In contrast, in their case control study Öhrn *et al.*¹¹ conducted a dental examination including radiographs and Decayed, Filled Surfaces (DFS), DMFS and untreated, Decayed Surfaces (DS) scores. They found that the frequency of dental caries was significantly higher among the EDPs than controls. They also reported statistically significantly higher *Lactobacillus* and *S. mutans* counts among the EDPs.

There is a general consensus in the reviewed literature^{11,13,15,18,19} that caries incidence in EDPs is variable, but studies provide conflicting results which may be attributed to a number of factors. Results based on small sample sizes may be unrepresentative. In one study²⁴ there is no report on the methodology used to obtain bacteriological data. Discrepancies in reported caries incidence may be further attributed to incomplete consideration of all factors which may be involved in the highly complex aetiology of dental caries. It is essential when reporting dental caries in EDPs to distinguish between clinical subtypes and not assume similar caries predisposition between all EDPs.

Little detail has been reported regarding other influences on the cariogenic process: in particular concurrent medical conditions or medication that may influence salivary flow. Both salivary rates and salivary composition influence caries susceptibility.²³ Antidepressant treatment is a recognized management option for the psychiatric aspects of EDs.¹¹ Antidepressant medications are profound antisialagogues significantly increasing dental caries risk. Other medications, both prescribed and over the counter

preparations have antisialagogue effects. Appetite suppressants may also influence salivation. In EDs where there are known salivary problems, failure to address saliva and salivary factors may have led to conflicting results.^{11,24}

The presence or absence of vomiting is an important modifying factor in the caries experience of EDPs. The observed lower caries incidence in EDPs where vomiting is a feature,²⁵ has been explained by the observation that cariogenic *S. mutans* cease to metabolize at pH values below 4.2. However, Belli and Marquis²⁶ have shown that *S. mutans* can adapt, survive and still remain viable with pH as low as 3.14. Young²¹ has observed that EDPs tend towards higher and not lower caries incidence.

Dental caries in EDPs is a complex, multifactorial and controversial subject. Investigations of this aspect of the oral sequelae of EDs should include analysis of diet and salivary factors as well as detailing vomiting behaviour.

Effect on bone

The main interest in looking at bone lies in the link between periodontal disease and osteopenia/osteoporosis. While controversy exists concerning the nature of this association, there is evidence to suggest a positive relationship with periodontal disease. It has been observed that patients with osteopenia/osteoporosis suffer from more severe periodontal disease than controls.²⁷⁻²⁹ The probable mechanism for accelerated progression of periodontal disease may be quite simple. Given the same bacterial infection and host response, alveolar bone loss will proceed at a much quicker rate if the density of structural bone being destroyed is suboptimal.

Osteoporosis is now recognized as a paediatric disease even though its manifestations may not become evident till later life.³⁰ Although the aetiology of osteoporosis is multifactorial, the peak bone mass constitutes one of the major determinants of risk of osteoporosis. Peak bone mass is attained at 30-35 years of age and is determined by genetic factors modulated by dietary and environmental factors. Insufficient calcium intake during childhood and adolescence can decrease peak bone mass and enhance postmenopausal and age related osteoporosis.³¹ The preferred consumption of caffeine containing beverages in place of milk has been linked, at least in part, to inadequate calcium intake.³²

EDPs are certainly at risk of low peak bone mass.³³ Dieting adolescent females who have amenorrhea but do not have an ED and those with EDNOS are at similar risk of low bone density as adolescent females with AN.³³ Lean body mass and timing of menarche are important factors in the aetiology of osteopenia. Of interest is the potential for EDPs who survive to become post menopausal to exhibit enhanced periodontal disease.

Eating disorders and oral soft tissues

It is well accepted that the oral mucosa is a good marker of underlying general health and that underlying systemic disease may manifest in oral tissues before becoming evident elsewhere in the body.

Effect on periodontal and gingival tissues

Inflammatory periodontal diseases related to the oral commensal microbiological biofilm (dental plaque) are the most prevalent bacterial diseases affecting mankind. Periodontal conditions may be subdivided into gingivitis and periodontitis. The aetiologies of periodontal conditions are not simple, all are multifactorial and complex. Any factors, which influence composition of the micro biota, host-defence mechanisms to the chronic bacterial insult or host soft-tissue repair mechanisms may have an influence both on the establishment and progression of periodontal conditions. On the whole it may be stated that gingivitis, which represents accumulation of supra-gingival plaque, occurs in any age group but periodontitis, except in uncommon manifestations, is a disease of adults. Unless EDPs manifest deficient anti-bacterial host-defence mechanisms it would be unlikely that periodontitis would be a feature of these conditions. It has been suggested that avitaminosis-C is an aetiological factor in periodontal disease in EDPs.^{8,16} However, the rapid, catastrophic periodontal destruction of scurvy cannot be equated with the insidiously slow progression of periodontitis. It would be expected that patients with hypovitaminosis-C would manifest other features of the deficiency.

Some studies^{18,24} suggest that EDPs do not exhibit more periodontal disease than controls although EDPs tend to have worse oral hygiene and increased prevalence of gingivitis. In a study²⁴ of periodontal factors using Community Periodontal Index of Treatment Needs (CPITN), and determining probing depth >3mm as indicative of the presence of periodontitis, no difference was found in the prevalence of periodontitis between the EDPs and controls. It is not surprising that even with the increased rates of gingivitis, advanced periodontal disease is not diagnosed, as the great majority of those patients are relatively young.

These results are challenged in a study¹³ which examined the loss of attachment associated with six representative teeth and scoring Approximal Plaque Index (API) values. Results show that EDPs had significantly reduced API values and gingival inflammation and that no loss of attachment was found among the patients. However, it is not stated whether controls had any attachment loss.

Methodology may account for the discrepancies in results. Both CPITN and use of representative teeth are screening procedures and are not as reliable as a full periodontal examination. In addition there has been considerable debate in the periodontal literature regarding the efficacy of using representative teeth to



Fig 2. Angular cheilitis.

index periodontitis, a disease which is highly variable in its site specificity. Lastly none of the articles discussed above mention whether any of the patients were smokers. Smoking is a well-recognized risk factor for periodontal disease.³⁴

Effect on the oral mucosa

Many oral mucosal lesions are related to nutritional deficiencies such as iron, B-group vitamins and folate. Such deficiencies may impair repair and the regenerative potential of the oral mucosa. Information pertaining to biochemical parameters associated with oral mucosal lesions is absent in the reviewed literature.

Trauma to the mucosa, particularly to pharynx and soft palate is almost universally recognized^{9,10,15,19} as a sequela of EDs. Such trauma is believed to arise as a result of inserting foreign objects into the oral cavity to induce vomiting. This is illustrated by a case report³⁵ of a bulimic patient presenting with bleeding from the oral cavity, specifically from a laceration of the posterior third of the tongue sustained when placing a spoon in her mouth to induce vomiting after an episode of binge eating.

Angular cheilitis (Fig 2) has been reported in EDPs.^{10,15,18,19} Such reports have implicated non-specified nutritional deficiencies and trauma as aetiological factors. It is well established in the oral pathological literature that angular cheilitis is a manifestation of chronic infection either solely by the fungus *Candida albicans* or sometimes by concomitant candidal and staphylococcal flora.³⁶ Angular cheilitis should be regarded and managed as a form of chronic oral candidiasis. Oral candidiasis is associated with both nutritional deficiencies and salivary dysfunction.

Mucosal lesions such as those induced by trauma are frequently secondarily infected by *Candida* organisms and of diagnostic importance is the fact that many of the manifestations of chronic oral candidiasis present as atrophic, erythematous lesions of the oral mucosa (Fig 3). Such lesions may be misdiagnosed as being of traumatic aetiology. Candidal micro-organisms and candidal lesions of the oral mucosa have warranted little investigation in the recent literature. Current knowledge of oral candidiasis suggests that the



Fig 3. Median rhomboid glossitis.

infections may present either with superficial fungal infections of oral epithelium or lesions where the micro-organisms have infiltrated deeper layers of epithelium. All forms of oral candidiasis are associated with nutritional deficiencies: EDPs are nutritionally challenged individuals and would be expected to exhibit candidal lesions of the oral mucosa. Oral candidiasis is an early clinical marker of systemic conditions as diverse as AIDS, diabetes and sideropenia. It may be expected that in nutritionally deficient individuals such as EDPs, oral candidiasis may be a clinical marker of suspicion, especially as chronic oral candidiasis is not a feature of healthy adolescents.

Candida species are commensal organisms in the mouth of about 50 per cent of the population. Isolation of *Candida* from the mouth is only of clinical importance when associated with mucosal lesions.

Other oral mucosal manifestations^{7,15} of EDs receive brief mention in the literature and include oral ulceration (Fig 4) and glossitis (Fig 5). These are also known to be related to hypovitaminosis B-12 as well as folate and iron deficiency states. Such deficiency states should alert the clinician to a potentially serious underlying problem.

Eating disorders and salivary factors

The effects of EDs on salivary glands, saliva and serum salivary amylase levels constitute the most



Fig 4. Oral ulceration.



Fig 5. Subtle glossitis in a patient suffering from anorexia nervosa.

studied topics^{13,37-40} The relationship between serum salivary amylase and ED in EDPs has merited particular attention as it has been suggested as a means for monitoring patient behaviour and treatment response in BN.^{37,38}

Effects on salivary glands

Sialadenosis, salivary gland swelling, in patients with EDs, has been investigated by numerous authors.^{13,39,41-44} It is generally agreed that sialadenosis is a feature of some but not all patients with BN.

Philip *et al.*¹³ investigated sialadenosis. Bilateral facial swelling was observed in 27 of 41 patients with BN. However, the results are questionable as the parotid glands were examined only by palpation and it is not known how many investigators performed this examination. The sample sizes were relatively small and ED subtype was not assessed.

Metzger *et al.*³⁹ investigated 17 females in outpatient treatment for BN and compared them to 21 healthy female controls. All subjects had parotid and submandibular gland dimensions estimated by ultrasonography. In comparison to controls, BN had a 36 per cent increase in parotid gland volume and also a 27 per cent non statistically significant enlargement in submandibular gland size. Self reported frequency of binge eating and self-induced vomiting was correlated significantly with both the parotid and submandibular gland size.

Numerous case reports⁴¹⁻⁴⁴ have also been published on the subject. Mendel *et al.*⁴² presented a case of painless, bilateral, symmetrical parotid swelling in a 27-year-old female with BN (criteria not specified). Parotid swelling was assessed with the aid of a CT scan. Sialadenosis was also reported and confirmed by histopathology. Sialadenosis may be the only presenting sign in BN as has been reported by Coleman *et al.*⁴⁴ in a 21-year-old female. Sialadenosis was again confirmed

by histopathology. Vavrina *et al.*⁴¹ and Buchanan *et al.*⁴³ also reported bilateral parotid enlargement, this time in two males with BN. Sialadenosis in all of the above was confirmed by histopathology following either FNA or open parotid biopsy. Mignogna *et al.*⁴⁵ presented a first case report of anorexia/bulimia related sialadenosis of palatal minor salivary glands. This highlights the need for comprehensive examination of all oral tissues.

Salivary gland swelling is extensively covered in the review literature.^{7,9,17-19} There is general agreement that swelling predominantly effects the parotid glands, either uni or bilaterally and a prevalence figure of 10-50 per cent among bulimic patients is quoted.^{9,18,19} Other major salivary glands may also be affected. Salivary gland swelling seems to occur only in individuals who purge by vomiting and not in individuals who purge by other methods.¹⁸ The onset of swelling usually follows a binge purge episode by 2-6 days¹⁸ and at least in the early stages is intermittent and reversible.^{17,18} The affected glands are reported to be soft to palpation and usually painless^{17,19} although painful swellings have also been reported.¹⁷ Painful swellings are rare and likely to have a different aetiology.

Histopathology generally reveals absence of inflammation, increased acinar size, increased secretory granules, fatty infiltration and non-inflammatory fibrosis.^{17,18} Aetiology of the salivary gland swelling is subject to debate.^{9,10,17-19} Numerous factors have been proposed with the most likely explanations including functional hypertrophy^{17,18} and increased cholinergic stimulation associated with vomiting.^{9,18,19}

Other proposed mechanisms include gastric juice irritation of the opening and lining of salivary gland ducts^{17,19} autonomic stimulation of the glands by activation of taste buds,^{9,18} pancreatic proteolytic enzymes brought into the mouth during vomiting stimulating taste receptors which in turn increase autonomic stimulation to the salivary glands,¹⁹ humoral interaction between pancreas and parotid^{17,19} nutritional deficiencies,¹⁷ excessive starch consumption¹⁷ and re-feeding after starvation.¹⁷ There is no discussion of the exact mechanisms involved and most are difficult to explain on the basis of anatomy or physiology.

Sialadenosis is not the only salivary gland pathology associated with BN. Schoning *et al.*⁴⁶ presented two case reports of necrotizing sialometaplasia in patients with BN. In both cases diagnosis was confirmed by biopsy. Necrotizing sialometaplasia is associated with traumatic injury to minor salivary glands⁴⁷ and Schoning proposed that insertion of fingers and foreign objects into the mouth in order to induce vomiting may be the aetiological factor. However, his association of gastric juice irritation of the opening of salivary gland ducts and necrotizing sialometaplasia is questionable.

Effects on saliva

The effects of ED on saliva^{10,11,13,18,19,24,48-50} may be quantitative, resulting in altered salivary flow rates or

qualitative resulting in altered salivary chemistry or both. Study results are conflicting. This at least in part may be attributed to limitations in study design.

Salivary flow rates

Salivary flow rates have been investigated in EDPs by numerous authors.^{11,24,40,48} Decreased salivary flow rates are associated with increased caries risk, susceptibility to other oral infections and taste disturbances.

Riad *et al.*⁴⁸ cannulated parotid ducts to investigate parotid salivary secretory pattern in 28 patients with BN (two male) compared to 30 healthy volunteers. Both stimulated and unstimulated salivary flow rates were measured. Four females with BN had sialadenosis and were investigated separately. Riad *et al.*⁴⁸ showed that BN patients had a reduced resting salivary flow rate. Resting salivary flow rates were even further decreased in patients with sialadenosis whereas the stimulated flow rate was only reduced in the sialadenosis group. Riad *et al.*⁴⁸ concluded that the basic functional disorder of salivary glands in BN patients, leading to decreased salivary flow rates, lies in the acinar cells and their innervation.

Öhrn *et al.*¹¹ demonstrated similar results. However, in this investigation authors apparently measured whole saliva. Stimulated and unstimulated salivary flow rates were investigated in 79 women and two men with ED and compared with 52 healthy volunteers. The authors demonstrated low unstimulated salivary flow rates among the EDPs. Milosevic *et al.*⁵⁰ also demonstrated significantly lower mean stimulated whole salivary flow rates among 19 BN patients compared with 10 controls.

Roberts *et al.*⁴⁰ sampling both parotid and submandibular secretions and Touyz *et al.*²⁴ apparently measuring whole saliva failed to demonstrate any differences between stimulated and unstimulated salivary flow rates among cases and controls.

Variations in results may in part be attributed to limitations in study design. In some studies the sample sizes are very small.^{40,48} The use of medication is not taken into account^{24,48} and there are discrepancies in methods of saliva collection. Many medications (e.g., antidepressants) have antisialagogue effects and affect both the qualitative and quantitative properties of saliva. Salivary flow rates vary diurnally. Any studies of salivary flow rate should be conducted at the same time of day for all subjects.^{24,40,50}

Salivary composition

Qualitative salivary composition has been investigated in patients with BN^{40,48-50} Riad *et al.*⁴⁸ analyzed stimulated and unstimulated parotid salivary samples for salivary immunoglobulins, total salivary protein content and salivary electrolyte content. It was concluded that BN patients had significantly greater amylase levels in both resting and stimulated conditions. No significant differences in immunoglobulin or electrolyte levels were detected

between patients and controls. However, Roberts *et al.*⁴⁰ failed to demonstrate any difference between amylase concentrations in 13 women with BN compared to 13 age and sex matched controls.

Tylenda *et al.*⁴⁹ in a blind study investigated salivary composition in 15 patients with BN and 15 age and race matched controls. Stimulated and unstimulated parotid and submandibular saliva was collected and analyzed for the concentration of potassium, chloride, calcium, urea nitrogen and salivary albumin. All salivary collections were performed between 8am and noon and at least two hours after eating or brushing. Their results indicated that BN and control subjects had similar values for salivary composition; there was no significant difference in the salivary concentration of potassium, chloride, calcium, urea nitrogen or albumin in both stimulated and unstimulated saliva. This is in agreement with Riad *et al.*⁴⁸ Tylenda *et al.*⁴⁹ concluded that since there was no difference in salivary chemistry between BN and control subjects, increased fermentable carbohydrates in the diet and binge-purging were the principal causes of oral tissue changes associated with BN and not physiological changes in saliva. However, the nature of the oral soft tissue changes alluded to was not clarified nor was the role of dietary fermentable carbohydrates and binge-purging explained.

The authors further proposed that during inflammation of salivary glands, chloride and albumin concentrations rise due to a breach in salivary-blood barriers and disruption of the transport system. For the same reasons potassium levels fall. Thus an inflammatory process could be ruled out as cause of parotid gland swelling since no alteration in salivary chemistry can be detected between BN and controls.

Milosevic *et al.*⁵⁰ looked at salivary factors in 19 BN patients who vomited with and without pathological tooth wear and at 10 healthy volunteers. Saliva collections were made between 11am and 3pm. Subjects had not eaten one hour prior to collection. Stimulated saliva was collected. Saliva was analyzed for flow rate, bicarbonate concentration and viscosity. In order to assess the calcium dissolved from surface enamel and therefore its susceptibility to acid erosion, an enamel biopsy was performed. Each subject had two enamel biopsies, one with and one without the pellicle. Controls were not subjected to enamel biopsies. The mean bicarbonate concentration in both BN groups was significantly less than in the control group. The mean salivary viscosity was significantly greater in the group with pathological tooth wear present group than in the tooth wear absent group and the control group. The dissolved calcium in the tooth wear present group was significantly lower than in the tooth wear absent group. This finding has been attributed to enamel having less available calcium, possibly due to the fact that some erosion may have taken place and protein had been deposited in porous enamel protecting it from further acid attack or that remineralization by

fluorapatite reduced enamel dissolution. Enamel biopsy was only performed on BN patients and not on controls and saliva was collected over a wide period throughout the day. However, this is one of few studies which took into account medication taken by the EDPs under investigation.

Salivary pH

Salivary pH is of interest as low pH predisposes patients to demineralization of enamel and promotes the growth of aciduric micro-organisms including *S. mutans*, *Lactobacillus spp* and *Candida spp*.

Milosevic *et al.*⁵⁰ failed to demonstrate any difference in salivary pH between BN patients and controls. However, Touyz *et al.*²⁴ showed that the mean salivary pH of patients with AN and BN was lower than that of controls. This was in agreement with the results published by Phillip *et al.*¹³ who also reported that EDPs had reduced salivary pH levels.

Salivary pH is dependant on bicarbonate concentration and an increase in bicarbonate concentration results in an increase in pH. Salivary bicarbonate content is in turn critically dependant on salivary flow rate.⁵¹ Bicarbonate concentration varies from 1mmol/l in unstimulated saliva to almost 60mmol/l at high flow rates. Bicarbonate is an important buffering system in saliva but only at high flow rates. In unstimulated saliva, the level of bicarbonate ions is too low to be effective and as such, unstimulated saliva is poorly buffered.⁵¹ The discrepancies in salivary pH reported in the literature may reflect non-standardized approaches to saliva collection.

Quantitative rather than qualitative changes affect saliva^{10,15,18,19} in EDs. It is generally agreed^{10,15,18} that this can be attributed to the side effects of commonly used psychoactive medications including antidepressants and antipsychotic medication. In EDPs, it would be expected that hyposalivation with resultant associated decrease in buffering capacity would promote the growth of aciduric micro-organisms such as *Candida spp*. Abuse of laxatives and diuretics¹⁸ may further compound the problem. It has also been stated^{15,16,18} that salivary disturbances may be associated with salivary gland enlargement. It is unlikely that the salivary gland enlargement is the cause of hyposalivation. Tissue damage has not been noted histologically although the presence of fatty infiltration and non-inflammatory fibrosis has been reported.^{17,18} Furthermore, salivary gland enlargement tends to affect the parotid glands which only contribute about 20 per cent of the total volume of unstimulated saliva and the symptoms of hyposalivation are usually noted when the salivary flow rate falls below 40-50 per cent.⁵²

Effect on serum amylase

It has been reported that 25-60 per cent^{17,52} of BN patients, have elevated levels of serum amylase. While it is generally accepted that the rise in serum amylase is

due to the salivary and not the pancreatic isoenzyme^{13,17,37-39,53} there is no consensus on the mechanism underlying this finding. Serum amylase levels have excited considerable research^{13,17,19,37-40,52} as they have been suggested as both a means of diagnosis and monitoring stability in BN.^{37,38}

Despite this ascertainment there is conflicting evidence on the clinical usefulness of the parameter. Walsh *et al.*³⁷ were not able to discriminate between patients with BN and controls on the basis of hyperamylasemia and Kronvall *et al.*³⁸ found no correlation between the severity of symptoms and salivary amylase levels in BN. However, Kinz *et al.*⁵³ reported a close positive correlation between the frequency of vomiting and levels of total serum amylase in patients with BN and suggested that serum amylase may act as a marker both for the presence of BN and degree of therapeutic control.

Gwirtsman *et al.*⁵⁴ noted inherent limitations in using serum amylase levels to diagnose or monitor EDs. Following a binge, amylase levels can undergo a two to four fold increase. As the plasma half life of the enzyme is 10-15 hours, isolated high levels may represent abnormal eating behaviour within the previous 24 hours and hence may give a misleading impression regarding the patient's condition. They concluded that dramatic fluctuations in salivary isoenzyme levels over short periods of time may be more useful in monitoring compliance of inpatients during periods of leave from their institution.

This interesting parameter is unlikely to have any bearing on the oro-dental management of EDPs.

Eating disorders and oral function

Oral function comprises: ingestion, mastication, deglutition, communication and speech. In the reviewed literature, deglutition and gustatory impairment have been studied. Roberts *et al.*⁴⁰ investigated swallowing patterns in 13 patients with BN comparing them to 13 healthy female controls. Real time ultrasound scanning and barium swallow studies were used. However, these techniques were not applied to all patients and controls. The presence or absence of pharyngeal and velar gag reflexes were also ascertained. All of the normal controls had both gag reflexes. The pharyngeal gag reflex was absent in nine out of 13 BN patients and a velar gag reflex could be elicited in only one. It was postulated that BN patients learn to inhibit all but the most forceful self-induced tactile pharyngeal stimulation. This may also arise as desensitization resulting from years of gastric purging. All of the patients with BN were found to have abnormal oropharyngeal swallow patterns and an increased duration of dry swallow. These changes were found to be difficult to explain.

Taste impairment (hypogeusia) associated with BN is mentioned in the review literature.^{7,17} Poor taste sensitivity, in particular to sucrose, hydrochloric acid, sodium chloride and quinine hydrochloride stimuli, has

been associated with the development of trace metal deficiencies, in particular the functional availability of zinc.⁵⁵ Abnormal eating patterns, unusual food choices and frequent vomiting have also been proposed as possible etiological factors for taste disturbances.¹⁷

CONCLUSIONS

Despite the vast literature published on the oral sequelae of EDs, relatively little is known on the oral medicine aspects of the subject. Dental destruction, parotid swelling and biochemical abnormalities, particularly related to amylase, constitute the focus of research. In contrast, limited data is available on the oral presentation of nutritional deficiencies in EDPs. This is both surprising and of concern as symptomatology of intraoral manifestations of deficiency states may present early in the clinical course of the ED therefore leading to an earlier diagnosis of a potentially life threatening disease.

Dentists are recognized as some of the first health care professionals to whom a previously undiagnosed EDP may present, and as such need to be aware of oral symptomatology other than dental erosion, which takes months to years to become apparent.

It is the strong opinion of the authors that focus of future research in this area should turn to the oral mucosa. Manifestations of nutritional deficiencies such as oral candidiasis, including angular cheilitis, glossitis and oral ulceration of non-traumatic origin should be more closely investigated in EDPs.

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