



# Oral Findings in Anorexia Nervosa and Bulimia Nervosa with Special Reference to Salivary Changes

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**Summary:** Diagnosis of anorexia nervosa and bulimia nervosa is mainly based on self-reports and is complicated because patients, especially those in the initial stages of anorexia and bulimics, present a normal body appearance and have a predisposition to hide their disease and avoid professional help. Dental erosions caused by self-induced vomiting and/or intake of acidic food-stuffs are common in this patient group as well as major salivary gland enlargement. However, dental caries and gingivitis do not seem to be related to anorexia and bulimia *per se*. Paraclinical findings suggest that *Streptococcus sobrinus* and other aciduric microorganisms are possible candidate biomarkers of bulimia. Oral dryness, impaired salivary flow and compositional changes including increased activity of the salivary alpha-amylase isoenzymes may qualify as biomarkers of anorexia and bulimia. Additionally, body water, electrolyte- and acid-base disturbances of these patients may be mirrored in saliva.

**Conclusions:** The dentist may be the first clinician to suspect the presence of an eating disorder, as patients often attend the dental clinic for regular dental checks. Despite clinical signs, diagnosis is often complicated. Although there are no well established pathognomic changes in saliva that relate to anorexia and bulimia there is some evidence to suggest that saliva may play a role in the oral changes observed in eating disorders, or that a number of clinical and laboratory salivary findings may provide biomarkers of these disease entities. Future research will prove if some of these markers could be helpful as screening tools in the dental clinic.

**Key words:** eating disorders, salivary glands, xerostomia, amylase, dental erosion

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## INTRODUCTION

Although of low prevalence in the general population, eating disorders like anorexia and bulimia are among the most serious diseases seen in young women. Diagnosis is based on the Diagnostic and Statistical Manual of Mental Disorders (DMS-IV™) (American Psychiatric Association, 1994) or ICD-10 (World Health Organization, 1992), which include anorexia nervosa, bulimia nervosa and their subtypes (Table 1), as well as other eating disorders. The diagnosis of anorexia and bulimia is often complicated by the fact that these patients tend to hide their disease and avoid professional help.

Consequently, the prevalence of these eating disorders is often underreported. Additionally, the actual diagnosis is mainly based on self-reports, which further complicates the diagnostic process, especially in the early onset of the eating disorder.

Due to the methodological problems, assessment of incidence and prevalence of eating disorders is difficult (Hoek and Van Hoeken, 2003). In westernised countries some selected groups are known to have an increased risk of developing eating disorders. In particular these groups include female teenagers, and persons engaged in sports that emphasize thinness for performance or appearance, e.g. dance and elite athletics

**Table 1 Diagnostic criteria of anorexia nervosa and bulimia nervosa****Extract from American Psychiatric Association (1994)****Anorexia nervosa:**

- A. Refusal to maintain body weight at or above a minimally normal weight for age and height, e.g. weight loss leading to a body weight less than 85% of that expected.
- B. Intense fear of gaining weight or becoming fat, even though underweight.
- C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
- D. In postmenarcheal females, amenorrhoea, i.e. the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhoea if her periods occur only following estrogen administration).

*Specify type:*

**Restricting type:** during the current episode of anorexia nervosa, the person has not regularly engaged in binge eating or purging behaviour (i.e., self induced vomiting or the misuse of laxatives, diuretics, or enemas).

**Binge eating/Purging type:** during the current episode of anorexia nervosa, the person has regularly engaged in binge eating or purging behaviour (i.e., self induced vomiting, or the misuse of laxatives, diuretics, or enemas).

**Bulimia nervosa:**

- A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
  - 1) eating, in a discrete period of time (e.g. within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
  - 2) a sense of lack of control over eating during the episode (e.g. a feeling that one cannot stop eating or control what or how much one is eating).
- B. Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as: self-induced vomiting; misuse of laxatives, diuretics, enemas or other medications; fasting; or excessive exercise.
- C. The binge eating and inappropriate compensatory behaviours both occur on average, at least twice a week for 3 months.
- D. Self-evaluation is unduly influenced by body shape and weight.
- E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

*Specify type:*

**Purging type:** during the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

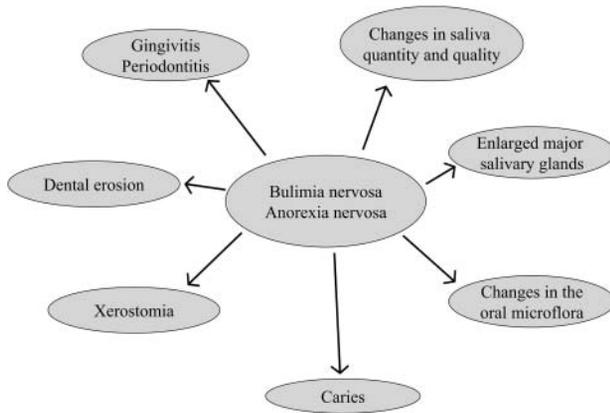
**Non-Purging type:** during the current episode of bulimia nervosa, the person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

**Extract from WHO (1992)****Anorexia nervosa:**

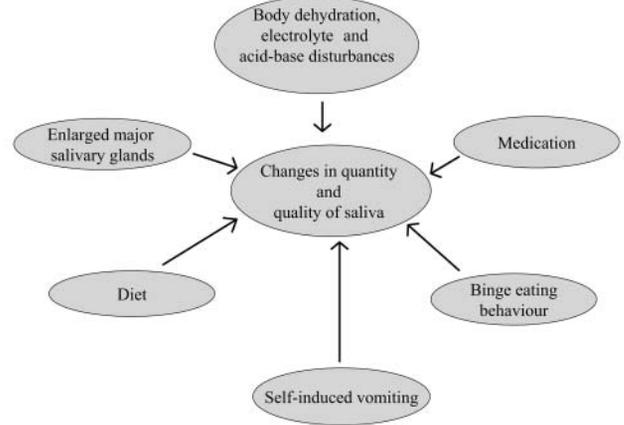
- A. There is weight loss or, in children, a lack of weight gain, leading to a body weight at least 15% below the normal or expected weight for age and height.
- B. The weight loss is self-induced by avoidance of 'fattening foods'.
- C. There is self-perception of being too fat, with an intrusive dread of fatness, which leads to a self-imposed low weight threshold.
- D. A widespread endocrine disorder involving the hypothalamic-pituitary-gonadal axis is manifest in women as amenorrhoea and in men as a loss of sexual interest and potency. (An apparent exception is the persistence on vaginal bleeds in anorexic women who are on replacement hormonal therapy, most commonly taken as a contraceptive pill.)
- E. The disorder does not meet criteria A and B for bulimia nervosa.

**Bulimia nervosa:**

- A. There are recurrent episodes of overeating (at least twice a week over a period of 3 months) in which large amounts of food are consumed in short periods of time.
- B. There is persistent preoccupation with eating, and a strong desire or a sense of compulsion to eat (craving).
- C. The patient attempts to counteract the 'fattening' effects of food by one or more of the following: 1) self-induced vomiting; 2) self-induced purging; 3) alternating periods of starvation; 4) use of drugs such as appetite suppressants, thyroid preparations, or diuretics; when bulimia occurs in diabetic patients they may choose to neglect their insulin treatment.
- D. There is self-perception of being too fat, with an intrusive dread of fatness (usually leading to underweight).



**Fig. 1** Possible oral findings in anorexia and bulimia.



**Fig. 2** Parameters and mechanisms possibly affecting the quantity and quality of saliva in persons with anorexia and bulimia.

(Dotti et al, 2002; Hulley and Hill, 2001; Ravaldi et al, 2003; Smolak et al, 2000). Estimates of the prevalence of bulimia nervosa are 1% for young women and 0.1% for young men, while the prevalence of anorexia nervosa in young females is equal to or below 1% and much below this figure in young men. Epidemiological studies have shown an increasing incidence of bulimia during the last 20-30 years, whereas the incidence of anorexia nervosa amongst women has been stable since the 1970s (Hoek and Van Hoeken, 2003).

Purging behaviours are common in persons with eating disorders (American Psychiatric Association, 1994; World Health Organization, 1992). This includes self-induced vomiting, which over longer periods of time, is known to cause dental erosion due to the acidity of repeated vomiting attacks. Unusual eating habits like excessive intake of acidic foodstuffs including fruits and juices also cause dental erosion (Hellström, 1977; Hurst et al, 1977; Rodger and Collyer, 1970). While the body appearance of the person with an eating disorder is often normal in the early onset of anorexia nervosa and in bulimia nervosa, oral complications may be early objective signs of an eating disorder (Fig. 1) (Altshuler et al, 1990; Herzog, 1982; House et al, 1981). It is therefore important for the dentist to recognize and diagnose the oral characteristics of eating disorders and plan treatment accordingly.

Because of its diluting, clearing and buffering abilities, saliva plays an important role in the maintenance of oral health, and protects the dental tissues against acidic exposure (Mandel, 1987; Sreebny, 2000). Any salivary imbalance caused by these eating disorders may have implications for the dentition.

Although several clinical studies show that salivary

gland hypofunction of various aetiologies may lead to serious oral sequelae (Papas et al, 1993; Longman et al, 1997; Young et al, 2001; for recent review see: Casolato and Turnbull, 2003), few studies have addressed the impact of anorexia and bulimia on salivary gland function and hence oral health. There is insufficient evidence as to whether salivary gland hypofunction and/or sialochemical changes should be considered common manifestations of anorexia nervosa and bulimia nervosa, and to what extent these changes affect oral health. This review focuses on the salivary glands, their secretions, and fluid composition and relates these to the oral findings in persons with anorexia nervosa and bulimia nervosa (Fig. 2). Their importance as a screening tool for eating disorders is reviewed.

## METHOD FOR LITERATURE SELECTION

The literature describing oral and salivary manifestations in anorexia and bulimia is diverse and includes numerous case reports and few case-control studies. Nonetheless, there is insufficient high-quality data for a systematic review (Moher et al, 1999). This review therefore describes the salivary and clinical oral manifestations of anorexia and bulimia and focuses on the role that saliva may have in these and explores its value as a candidate biomarker of bulimia or anorexia.

A literature search was conducted in MEDLINE using keywords in the title and abstract. Search words were: anorexia, anorexia nervosa, bulimia, bulimia nervosa, eating disorders, erosion, caries, saliva, and salivary glands. Additional studies were found from references in the retrieved papers. The papers included in Tables

3-5 are the case-control studies found in our literature search. Very few papers describe changes in the electrolyte composition of saliva in the eating-disturbed and case reports are therefore also included (Table 4). For evaluation of differences between the eating-disturbed and healthy controls we have relied on the significance of the differences obtained in the studies described ( $p < 0.05$ ).

## SALIVARY ALTERATIONS IN PERSONS WITH ANOREXIA AND BULIMIA

Both under normal and pathophysiological conditions, the quantity (Table 2) and quality of saliva are highly dependent on the degree of salivary gland stimulation. Unstimulated saliva is characteristically rather viscous and predominantly produced by the submandibular gland (mixed, but mainly serous type), while the stimulated secretion is much more watery and mainly produced by the parotid gland (strictly serous type). A clinical study describes increased risk of caries at unstimulated whole saliva flow rates below 0.16 ml/min (Navazesh et al, 1992). In addition, an increased number of saliva lactobacilli and an increased rate of demineralization of the tooth surface at similar flow rates has been demonstrated in an *in situ* caries model (Bardow et al, 2001). This implies that at such low secretion rates the risk of dental caries is considerably increased. At unstimulated whole saliva flow rates of 0.1 ml/min or less (hyposalivation) persons also have a five-fold greater risk of developing dental erosions (Järvinen et al, 1991) compared to those with secretion rates within the normal range (Table 2). Saliva in persons with anorexia and bulimia may alter, both in flow and composition.

## Xerostomia

Xerostomia, the subjective sensation of dry mouth, does not always correlate with the objective measure of salivary flow rate (Fox et al, 1987; Sreebny and Valdin, 1988). The prevalence of xerostomia in selective populations varies from 10-80%, with variations explained by differences related to age, sex, disease and pharmacotherapy. In a Swedish population sample, the prevalence of xerostomia among non-medicated 20-30-year-old men and women varied between 10-20% (Nederfors, 2000). Few studies have reported xerostomia in persons with eating disorders. Nevertheless xerostomia in persons with anorexia and/or bulimia caused by their intake of xerogenic medicines and body dehydration as a consequence of vomiting or other purging behaviors is common. Among bulimics the feeling of dry mouth is three times more common than that of controls (Rytömaa et al, 1998), and the prevalence of xerostomia in groups of persons with bulimia has been reported to range from 35% (Spigset, 1991) to 65% (Altshuler et al, 1990). The same pattern is seen in a group of anorexics, which shows different grades of body dehydration caused by starvation, laxatives, diuretics and/or vomiting. Vomiting anorexics had more complaints of thirst and dryness of the mouth (Hellström, 1977) (Table 3).

## Enlargement of the Major Salivary Glands

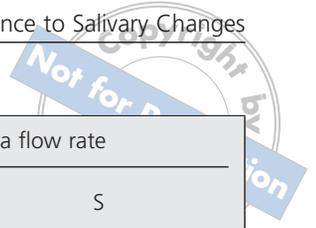
Several studies have estimated glandular enlargement by inspection and palpation and reported benign bilateral painless parotid enlargement in patients with bulimia nervosa (Table 3). The submandibular and sublingual glands can also be affected, but to a lesser extent. There is no consensus as to whether these clinical findings are specific to persons with bulimia nervosa (Kinzi et al, 1993; Levin et al, 1980; Liew et al, 1991),

**Table 2 Saliva flow, xerostomia and hyposalivation – definitions and normal mean values**

Hyposalivation: cut-off values for saliva flow rates measured by sialometry. It can be caused by e.g. medication intake, and Sjögren's syndrome.				
Xerostomia: the subjective sensation of dry mouth. People with xerostomia do not necessarily suffer from hyposalivation.				
	Normal – mean values (ml/min)		Hyposalivation (ml/min)	
	Unstimulated	Stimulated	Unstimulated	Stimulated
Whole saliva <sup>1</sup>	0.3	1.5	≤0.1	≤0.7
Parotid saliva, single gland <sup>2</sup>	0.05	0.50	<0.02	<0.20

<sup>1</sup>Bertram, 1967; Heintze et al, 1983; Navazesh and Christensen, 1982; Sreebny, 2000

<sup>2</sup>Kalk et al, 2001


**Table 3 Saliva gland function in persons with anorexia and bulimia – case control studies**

	Sample size	Xerostomia	Salivary gland enlargement	Saliva flow rate	
				NS	S
<b>Bulimia nervosa</b>					
Altshuler et al (1990)	40 BN 40 controls	65% (26/40) <sup>1</sup>	8% (3/40)		
Rytömaa et al (1998)	35 BN 105 controls	34% (12/35) p<0.001			
Scheutzel and Gerlach (1991)	20 BN 30 controls		25% (5/20)		UWS↑ SWS↑
Philipp et al (1991)	41 BN 50 controls		66% (27/41)		
Metzger et al (1999)	17 BN 21 controls		2		
Milosevic and Dawson (1996)	19 BN 10 controls				SWS↓
Öhrn et al (1999)	46 BN 7 AN+BN 52 controls			SWS→	UWS↓
Riad et al (1991)	28 BN 30 controls				UPS↓ SPS↓ <sup>3</sup>
Howat et al (1990)	10 BN 10 controls			SWS↓	
Rytömaa et al (1998)	35 BN 105 controls			SWS→	UWS↓
Touyz et al (1993)	15 BN 15 controls			SWS→	
<b>Anorexia nervosa</b>					
Philipp et al (1991)	11 AN 50 controls		0% (0/11)		
Liew et al (1991)	15 AN (restricting) 15 controls		0% (0/15)	SWS→	
Scheutzel and Gerlach (1991)	12 AN (restricting) 13 AN (bulimic) 30 controls		17% (2/12) <sup>4</sup> 54% (7/13) <sup>5</sup>		UWS↓ SWS↑
Öhrn et al (1999)	3 AN 7 AN+BN 52 controls			SWS→	UWS↓
Touyz et al (1993)	15 AN 15 controls			SWS→	

NS: non-significant; S: significant; AN: anorexia nervosa; BN: bulimia nervosa; SWS: stimulated whole saliva; UWS: unstimulated whole saliva; SPS: stimulated parotid saliva; UPS: unstimulated parotid saliva; ↑: increased in AN or BN compared to control; →: no difference between the groups; ↓: reduced in AN or BN compared to controls.

<sup>1</sup> Xerostomia was observed exclusively in the bulimia nervosa group

<sup>2</sup> Size estimation by ultrasound shows a 36% increase in parotid gland volume of bulimics compared to healthy controls

<sup>3</sup> SPS was only reduced in the bulimia patients who developed sialadenosis

<sup>4</sup> 'restricting anorexics' without any significant history of vomiting

<sup>5</sup> bulimic anorexics

or if anorexia nervosa is also associated with enlargement of the major salivary glands (Roberts and Li, 1987; Walsh et al, 1981).

Assessment of the size of both submandibular and parotid salivary glands by ultrasound show that persons with bulimia nervosa have a 36% increase in parotid gland volume compared to healthy controls, whereas the submandibular gland enlargement was less significant (Metzger et al, 1999). The parotid enlargement usually develops two to six days after a bulimic episode that includes binge eating and vomiting. Glandular enlargements are generally intermittent with complete or near complete regression of gland size between bulimic episodes (Brady, 1985; Levin et al, 1980; Ogren et al, 1987) or when nutritional balance is normalized (Dawson and Jones, 1977). Although most parotid enlargements are painless there are case reports describing pain and fullness at the angle of the jaw bilaterally (Brady, 1985) and occasionally ache (Wilson and Price, 2003).

The pathogenesis of the salivary gland enlargement is not yet clarified. Several studies show that both frequency of self-induced vomiting and frequency of binge eating are correlated with size of both parotid and submandibular glands (Brady, 1985; Levin et al, 1980; Metzger et al, 1999). However, it has been difficult to test whether salivary gland enlargement is more closely associated with binge eating or purging behaviours, since they mostly occur together (Metzger et al, 1999). Excessive starch ingestion and work hypertrophy has also been related to salivary gland enlargement. A case report describes bilateral parotid enlargement in a person, on adequate nutritional intake, who ingested as much as 200 grams of laundry starch every day. A biopsy of the right parotid gland showed cells depleted of zymogen granules. The authors suggest that the parotid enlargements were due to chronic starch ingestion as a result of work hypertrophy (Silverman and Perkins, 1966); Merkatz (1961) reported a similar finding. The salivary enlargements may be related to the rapid and excessive stimulation of the glands during binge eating (Roberts and Li, 1987). It has also been proposed that the repeated stimulation of the glands during vomiting caused by stretching of the stomach, via the vagal nerve, centrally activates salivary reflexes causing increased salivary flow (Pedersen et al, 2002).

Few studies have characterized the changes of the enlarged glands histopathologically. In general, inflammatory changes are absent (Brady, 1985; Levin et al, 1980; Vavrina et al, 1994; Walsh et al, 1981) and the morphological appearance of the parotid

gland tissue is normal (Levin et al, 1980; Walsh et al, 1981; Wilson and Price, 2003). However, a case report describes a biopsy of an enlarged parotid gland of a bulimic man that shows swollen serous acinar cells with a subsequent slight compression of the ducts. In addition, the densely packed mature enzyme granules in the cytoplasm of the acinar cells displace the nuclei towards the basal part of the cells (Vavrina et al, 1994). Similar light and electron microscopic findings are described in a bulimic woman, who had parotidectomies for cosmetic reasons (Coleman et al, 1998).

Interestingly, Ogren et al (1987) found that within a group of bulimics only those with parotid gland hypertrophy have dental abnormalities, principally enamel erosion and excessive caries, whereas those with hypertrophy of the submandibular glands did not exhibit such dental findings.

### **Saliva Flow**

Persons with eating disorders are commonly treated with anti-depressants (Hellström, 1977; Walsh et al, 2000; Krüger and Kennedy, 2000), which are known to reduce salivary flow (Clemmesen, 1988; Sreebny and Schwartz, 1997). In addition, the practises of inappropriate purging-behaviours like self-induced vomiting, misuse of laxatives, diuretics, and excessive exercise (Table 1) may result in body dehydration (Hellström, 1977) and thereby exert a negative impact on salivary flow rate (Fig. 2).

Few studies have investigated the salivary flow rate in persons with anorexia or bulimia (Table 3). The majority of these studies demonstrates that these patients have reduced unstimulated whole saliva and parotid saliva flow rates when compared to healthy controls (Öhrn et al, 1999; Riad et al, 1991; Rytömaa et al, 1998) or defined normal secretion values (Hellström, 1977). In the stimulated state, inconsistent results are reported when compared with healthy control persons:

- reduced stimulated whole saliva (Milosevic and Dawson, 1996)
- reduced parotid saliva (Altshuler et al, 1990; Riad et al, 1991)
- normal stimulated whole saliva flow rate (Howat et al, 1990; Öhrn et al, 1999; Touyz et al, 1993)
- an increased stimulated whole saliva flow rate (Scheutzel and Gerlach, 1991).

These different results could be attributed to different methods of salivary stimulation and collection, and also to unreported variations in the patients' medicine intake. Duration of disease, type of eating disorders (i.e. anorexia restricting and binge eating/purging type and

bulimia purging and non-purging type) and frequency of vomiting vary, and could all affect salivary flow and result in inconsistencies. In a group of anorexics the lowest unstimulated whole saliva flow rates were demonstrated in the persons with the highest frequencies of self-induced vomiting (Hellström, 1977), whereas Altshuler et al (1990) found no relation between duration and frequency of self-induced vomiting on paraffin-stimulated parotid secretion or feeling of oral dryness in a group of bulimics.

### **Saliva Composition: pH and Buffer Systems**

The maintenance of a non-harmful pH in the mouth during the time it takes to clear acids introduced via acidic foodstuffs or vomit is dependent on the buffering capacity of saliva. When the acidic exposure is substantial, the buffering capacity (which is measured in mmol H<sup>+</sup>/(litre saliva · pH unit) in a certain pH interval) is not always sufficient to resist the acid load. Under such conditions demineralization of tooth substance is likely to occur when saliva at a given pH is undersaturated with respect to hydroxyapatite. The pH of saliva, which in healthy individuals varies between 6.0 and 7.5, is highly dependent on the flow rate, with the most alkaline values observed at high flow rates. Also, the saliva buffering capacity, to which three major buffer systems contributes, i.e. the bicarbonate, the phosphate and the protein systems (Bardow et al, 2000), is higher in stimulated compared to unstimulated saliva. The contribution of each of the three buffer systems is highly pH-dependent. The bicarbonate system is predominantly in the pH range 5-7 and in stimulated whole saliva, because the concentration of bicarbonate rises with flow-rate increases this results in a more alkaline pH and a high buffering capacity. However, in unstimulated whole saliva, in the same pH range, the buffering capacity is equally due to the phosphate and the bicarbonate system, while the buffering capacity of the protein system is most pronounced at pH values below 5 in both stimulated and unstimulated saliva (Bardow et al, 2000).

Since the saliva flow rate seems to be less in persons with anorexia and bulimia (Altshuler et al, 1990; Hellström, 1977; Milosevic and Dawson, 1996; Öhrn et al, 1999; Riad et al, 1991; Rytömaa et al, 1998), a lower pH is expected as well as a decreased contribution by the bicarbonate-system (Bardow et al, 2000), possibly causing a lower buffering capacity and thereby an increased risk of tooth demineralization in persons with anorexia and bulimia than in a healthy controls. Studies measuring pH and buffering

capacity of saliva of persons with anorexia and bulimia are sparse, and the few results are contradictory (Table 4).

Anorexics and bulimics appear to have lower pH in their stimulated whole saliva than healthy controls (Liew et al, 1991; Howat et al, 1990; Touyz et al, 1993) although normal saliva pH has also been reported (Milosevic and Dawson, 1996).

Low buffering effect (final pH) (Öhrn et al, 1999) and low bicarbonate concentration of stimulated whole saliva (Milosevic and Dawson, 1996) is described in persons with anorexia and bulimia. However, when measuring the combined buffering effect by the chair-side test, the Dentobuff kit<sup>TM</sup>, Milosevic and Slade (1989) found no difference in buffering effect between persons with anorexia and bulimia and a group of age-matched controls.

It is very likely that variations in the methods of collecting and measuring pH and buffering capacity of saliva contribute to the diversity of results (Table 4) because the equilibrium of the bicarbonate buffer system is rarely taken into consideration. The bicarbonate buffer system is based on the equilibrium:



where CO<sub>2</sub> denotes carbon dioxide dissolved in saliva, H<sub>2</sub>CO<sub>3</sub> denotes carbonic acid, and HCO<sub>3</sub><sup>-</sup> denotes bicarbonate. When saliva is exposed to the atmosphere during sampling and/or measurements some CO<sub>2</sub> evaporates, thereby increasing pH in the alkaline direction (Schmidt-Nielsen, 1946), and consequently affect the buffering capacity. Accordingly, adequate saliva collection and measurement of salivary pH and buffering capacity should be performed in closed systems, thereby avoiding loss of CO<sub>2</sub> to ensure correct measures that resemble the original values present in the oral cavity (Bardow et al, 2000).

### **Saliva Composition: Electrolytes**

The process of saliva formation is dynamic. Thus, the composition of blood and interstitial fluid, the metabolic state of the gland, as well as the activity of electrolyte transporters in the cell membrane of both acinar and ductal segments are all parameters that have, or may have an influence on the final saliva. Consequently, the saliva secretion represents an aggregate of all mechanisms involved in the formation processes throughout the glandular tissue.

In rat studies the glandular tissue can adapt to general metabolic changes, e.g. metabolic acidosis and alkalosis, respectively, by regulation of the activity and number of specific membrane transporters. Thus, changes in secretion of bicarbonate are described as

**Table 4 Saliva composition in persons with anorexia and bulimia – case-control studies and selected case reports**

	Sample size	Inorganic components		Organic components	
		NS	S	NS	S
<b>Bulimia nervosa</b>					
Tylenda et al (1991)	15 BN 15 controls	K <sup>+</sup> , Cl <sup>-</sup> , Calcium→			
Philipp et al (1991)	41 BN 50 controls	K <sup>+</sup> , Cl <sup>-</sup> , Na <sup>+</sup> , Calcium →	pH↓		Amylase↑ <sup>1</sup>
Milosevic and Dawson (1996)	19 BN 10 controls	pH→	HCO <sub>3</sub> <sup>-</sup> ↓		
Öhrn et al (1999)	46 BN 7 AN+BN 52 controls	pH→	Buffering effect ↓		
Riad et al (1991)	28 BN 30 controls	→ <sup>2</sup>			Amylase↑ <sup>1</sup>
Howat et al (1990)	10 BN 10 controls		pH↓		
Rytömaa et al (1998)	35 BN 105 controls	Buffering effect ↓			
Touyz et al (1993)	15 BN 15 controls		pH↓		
Milosevic and Slade (1989)	33 BN (vomiting) 7 BN (restricting) 50 controls	Buffering effect →			
Metzger et al (1999)	17 BN 21 controls				Amylase↑ <sup>1</sup>
Gwirtsman et al (1989)	23 BN 31 controls				Amylase↑ <sup>1</sup>
Walsh et al (1990)	40 BN 25 controls			Pancreas- amylase→ <sup>1</sup>	Amylase↑ <sup>1</sup> Saliva-amylase↑ <sup>1</sup>
Kinzl et al (1993)	50 BN 50 AN				Amylase↑ <sup>1+3</sup> Saliva-amylase↑ <sup>1+3</sup>
Scheutzel and Gerlach (1991)	20 BN 30 controls				Amylase↑ <sup>1+4</sup> Saliva-amylase ↑ <sup>1</sup> Pancreas-amylase↑ <sup>1</sup>
<b>Anorexia nervosa</b>					
Philipp et al (1991)	11 AN 50 controls	K <sup>+</sup> , Cl <sup>-</sup> , Na <sup>+</sup> , Calcium →	pH↓		Amylase↑ <sup>1</sup>
Liew et al (1991)	15 AN (restricting) 15 controls		pH↓		
Öhrn et al (1999)	3 AN 7 AN+BN 52 controls	pH→	Buffering effect ↓		
Touyz et al (1993)	15 AN 15 controls		pH↓		

Table 4 Continued

	Sample size	Inorganic components		Organic components	
		NS	S	NS	S
Milosevic and Slade (1989)	18 AN 50 controls		Buffering effect →		
Rodger and Collyer (1970)	Case report		K <sup>+</sup> ↓		
Scheutzel and Gerlach (1991)	12 AN (restricting) 13 AN (bulimic) 30 controls				Amylase↑ <sup>1+5</sup> Saliva-amylase ↑ <sup>1</sup> Pancreas-amylase ↑ <sup>1</sup>
Gwirtsman et al (1989)	31 AN (restricting) 25 AN (bulimic) 31 controls				Amylase → <sup>1+6</sup>
Jensen et al (1987)	Case report		Calcium↑ Phosphate↑		

NS: non-significant; S: significant; AN: anorexia nervosa; BN: bulimia nervosa; ↑: increased in AN or BN compared to controls; →: no difference between the groups; ↓: reduced in AN or BN compared to controls.

<sup>1</sup> Measured in serum

<sup>2</sup> The authors do not state which electrolytes were measured

<sup>3</sup> Patients with bulimia nervosa were compared to patients with anorexia nervosa

<sup>4</sup> Measured in saliva – increased amylase activity in both stimulated and unstimulated parotid saliva

<sup>5</sup> Total amylase was measured in both serum and saliva. The proportions of pancreatic- and salivary alpha-amylase isoenzyme in serum were within the normal range for patients with restrictive anorexia nervosa but increased in the bulimic anorexia nervosa group

<sup>6</sup> Serum amylase was elevated in anorectic patients who engaged in binge eating and purging

a result of the ductal epithelial response to acid-base changes in blood leading to abolishment of ductal bicarbonate during metabolic acidosis, while bicarbonate is increased during metabolic alkalosis (Knauf et al, 1975). Furthermore, experimentally induced acute and chronic metabolic acidosis in rats causes a redistribution of H<sup>+</sup>-ATPase especially in the striated and granular ductal epithelium, suggesting that this membrane-transporter is involved in regulation of acid-base homeostasis (Roussa et al, 1998).

Conditions of metabolic alkalosis (Mitchell et al, 1983; Warren and Steinberg, 1979) or acidosis (Dawson and Jones, 1977; Mitchell et al, 1987; Pines et al, 1985) are frequently described in persons with anorexia and bulimia. Besides changes in blood pH and bicarbonate, general changes include altered serum levels where potassium (Crow et al, 1997; Dawson and Jones, 1977; Koh et al, 1989; Mars et al, 1982; Ogren et al, 1987; Wolfe et al, 2001), sodium (Mitchell et al, 1983), chloride (Geliebter et al, 1992; Mitchell et al, 1983; Wolfe et al, 2001), magnesium (Hall et al, 1988), zinc and phosphate (Bonne et al, 1995) are among the most distinct (Antonson et al, 1999). Accordingly, Mitchell et al (1983) describe a group of 168 bulimics of which half of them present with electrolyte abnormali-

ties. It is suggested that serious serum abnormalities are more prevalent in person with low weight in addition to frequent vomiting and laxative abuse (Peeters and Meijboom, 2000). Thus, it is very likely, that the changes in the levels of serum electrolytes can be reflected in the electrolyte composition of saliva.

Few studies (Table 4) have performed measurements of electrolyte composition of saliva in patients with eating disorders. Two studies comparing the composition of parotid and submandibular saliva of bulimics and healthy individuals reveal no differences in the electrolyte levels (Riad et al, 1991; Tylenda et al, 1991), which is supported by a study evaluating the electrolyte levels in whole saliva (Phillip et al, 1991). However, a case report presenting an anorexic woman with hypokalemic alkalosis demonstrates low salivary potassium without stating details about flow rate or type of saliva (Rodger and Collyer, 1970).

In an experimental study of women of normal weight subjected to an eight-day fasting diet, flow rate, phosphate and sialic acid concentrations in stimulated whole saliva are significantly decreased after seven days of fasting. A less pronounced reduction in potassium and calcium concentrations was also observed. All changes normalized to baseline level shortly

after the fasting period. The authors suggest that more extreme and longer lasting conditions such as eating disorders might give longer-lasting effects on saliva composition (Johansson et al, 1984). These results support the assumption that electrolyte levels of saliva in persons with bulimia and/or anorexia may be disturbed.

### **Saliva Composition: Organic Components**

The total protein concentration in whole saliva is on average 2.0 mg/ml saliva, which is almost 40 times lower than the protein concentration in plasma. The concentration of protein in saliva is dependent on both the flow rate and the duration of stimulation, as long periods of stimulation and high flow rates result in high total protein concentration (Dawes, 1969).

The multiple functions of the salivary proteins in relation to oral health have not been studied in detail. Individual protein composition of saliva may play a role in resistance against tooth demineralization (Lenander-Lumikari and Loimaranta, 2000), antimicrobial functions, and the texture and viscosity of saliva. The functions of some salivary proteins are well defined, and in persons with eating disorders the alpha-amylase protein, a digestive enzyme involved in the breakdown of starch to simple hexoses, has been studied.

**Amylase:** The breakdown of starch occurs in two phases starting in the oral cavity. It is initiated by salivary amylase, and continues in the intestine with pancreatic amylase. The family of amylase isoenzymes in saliva has a mass (molecular weight) around 55-60 kDa depending on glycosylation and is secreted from the serous acinar cell type, particularly from the parotid gland. Measurement of amylase activity in persons with eating disorders has mainly been carried out in plasma samples, and as amylase is also secreted from the pancreatic gland, discrimination of the amylase origins must take place. This is performed using electrophoretic separation procedures (Gwirtsman et al, 1986; Kronvall et al, 1992; Walsh et al, 1990) or a variety of enzymatic assays (Humphries et al, 1987; Metzger et al, 1999).

The amylase activity in plasma – be it of salivary or pancreatic origin – is frequently increased in persons with anorexia and bulimia (Levin et al, 1980; Metzger et al, 1999; Mitchell et al, 1983; Scheutzel and Gerlach, 1991), and several studies have demonstrated that this hyperamylasemia is of salivary origin (Humphries et al, 1987; Kaplan, 1987; Kinzl et al, 1993; Walsh et al, 1990). The hyperamylasemia is most frequent in persons with bulimia nervosa, but in-

creased levels of amylase are also described in patients with anorexia nervosa (Gwirtsman et al, 1989; Scheutzel and Gerlach, 1991) (Table 4). The pathogenesis of the hyperamylasemia is widely discussed. Consensus exists about the fact that binge eating and vomiting behaviour is related to hyperamylasemia, but whether it is the binge eating episodes, the frequent management of self-induced vomiting or other conditions that are the main causes are not clear.

Examination of adipose patients (no information of weight or body-mass-index) with a binge eating syndrome (without vomiting) does not reveal elevated levels of serum amylase after consumption of a large meal, whereas in a group of bulimics most cases show a close positive correlation between the frequency of vomiting and the levels of total serum amylase. This suggests that hyperamylasemia is largely conditioned by vomiting (Kinzl et al, 1993), which is supported by a study describing hyperamylasemia of salivary origin in bulimic persons and women who suffer from excessive vomiting during pregnancy (Robertson and Millar, 1999). On the other hand, Blinder and Hagman (1986) report that increased salivary amylase values are related only to the presence of binge eating as found in bulimics, while in anorexics there are reduced levels of salivary isoamylase. However, significant correlations between serum amylase concentration and both frequency of binge eating and frequency of self-induced vomiting are also reported (Mitchell et al, 1983). Additionally, parotid and submandibular gland size and serum amylase concentration correlates positively in persons with bulimia nervosa (Metzger et al, 1999), while parotid swelling seen in persons with other diseases than anorexia and bulimia, i.e. diabetes, liver cirrhosis, chronic alcoholic liver damage, and hypertension, does not affect serum isoamylase levels (Ueda et al, 1993).

An analysis of the parotid saliva of persons with bulimia nervosa shows that the principal change in the salivary constitution is an increase in salivary amylase levels in the parotid saliva under both unstimulated and stimulated conditions. This was seen together with a reduction in both unstimulated and stimulated parotid flow rates. The findings that amylase activity and protein concentration increases concomitantly with reduced salivary flow leads the authors to suggest that bulimics have increased activity of the sympathetic branch of the autonomic nervous system (Riad et al, 1991). Whole saliva of persons with anorexia and bulimia reveals an increase in salivary amylase activity (Scheutzel and Gerlach, 1991).

Further exploration of the salivary amylase-isoenzymes in persons with anorexia and bulimia shows a distinct pattern with a decreased activity of the  $S_1$ -,  $S_2$ - and  $S_5$ -fractions of salivary amylase-isoenzymes and an increased activity of the  $S_3$ - and  $S_4$ -fractions in both serum and whole saliva when compared to healthy controls (Scheutzel and Gerlach, 1991).

The suitability of amylase assays as a routine or monitoring test in persons presumed to have an eating disorder has been investigated. One study describes that, at a serum amylase criterion level of 60 IU/l, it is possible to distinguish normal-weight persons with bulimia nervosa from healthy controls with a specificity of 97% and a sensitivity of 52% (Gwirtsman et al, 1989). However, this is questioned by Walsh et al (1990) who find that the sensitivity is only 31% when the criterion level is used in a population with much lower binge frequency (7.9 binges/week compared with 32 binges/week in the study by Gwirtsman et al, 1989). Additionally, a study monitoring persons with bulimia nervosa during an 8-week treatment trial leads to the conclusion that the results do not justify the use of amylase assays as a routine diagnostic or monitoring test, but may instead serve as a useful laboratory tool in certain subgroups of eating disorder patients (Kronvall et al, 1992).

In summary, it seems that the hyperamylasemia commonly found in persons with eating disorders is due to an increase in the amount, and thus activity of the salivary isoenzymes. However, since there is no clear relationship between bulimic behaviours and variation in amylase levels, the value of amylase assay as a diagnostic or screening tool may presently be considered as questionable. However, future investigations of the salivary alpha-amylase isoenzymes may elucidate more concise associations with purging behaviours as implied by Scheutzel and Gerlach (1991). Additionally, the relation between food intake and the amylase level should be taken into consideration, since experimental studies in rats show that both parotid and serum amylase levels are consequences of food intake (Nagy et al, 2001). In humans, salivary flow exhibits diurnal variations (Dawes, 1974) which also account for salivary components like amylase (Jenzano et al, 1987). Evaluation of amylase in saliva therefore requires standardized sampling.

**Salivary viscosity:** The salivary viscosity, which is dependent on the organic composition of saliva, is rarely described in persons with eating disorders. However, one study describes significantly greater viscosity of whole saliva in bulimics with tooth wear

compared to those without, and those in a non-bulimic control group (Milosevic and Dawson, 1996). Thus, a more detailed investigation of the protein and glyco-protein composition of saliva in persons with eating disorders seems relevant. When considering the viscosity of whole saliva one should take into account that a high viscosity mainly arises from the mucin-rich fluid produced by the submandibular and minor salivary glands. Since the parotid gland secretes a much more watery fluid a reduced parotid secretion may also contribute to the increased viscosity described above.

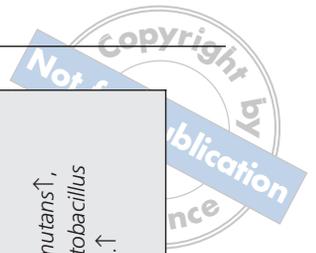
How the salivary alterations may have an impact on oral findings in persons with anorexia and bulimia is described below.

## ORAL FINDINGS IN PERSONS WITH ANOREXIA AND BULIMIA

### *Dental Erosion*

Dental erosion, which by definition is the chemical dissolution of the dental hard tissue in a process not including bacteria, is the most common oral complication observed in persons with anorexia and bulimia (Table 5), especially in those who practise self-induced vomiting (Altshuler et al, 1990; Hellström, 1977). The aetiology is characterized as exogenous, endogenous or a combination of these. Exogenous factors causing dental erosions in patients with eating disorders are related to diet. Large amounts of acidic foodstuffs such as fruits and juice are found to be common ingredients in the diet of some persons with anorexia and bulimia (Hellström, 1977; Hurst et al, 1977; Rodger and Collyer, 1970). In persons with eating disorders the exposure to endogenous acids in the oral cavity is a result of recurrent self-induced vomiting. However, the loss of enamel and eventually dentine is often first clinically recognisable when vomiting has been practised for a period of at least six months (Altshuler et al, 1990). Additionally, if the person is a low secretor (unstimulated whole saliva flow  $\leq 0.1$  ml/min) the hyposalivation *per se* increases the risk of dental erosion by five times as compared to persons with normal flow rates (Järvinen et al, 1991).

The palatal surfaces of maxillary teeth, in particular the incisors, are the most obvious locations of dental erosion in persons with anorexia and bulimia due to the exposure to gastric acid. However, eroded surfaces are found throughout the dentition. Thus, Hurst et al (1997) distinguish between three types of erosions in persons with anorexia nervosa: a) severe ero-



**Table 5 Oral findings in persons with anorexia and bulimia – case-control studies**

	Sample size	Erosion		Caries		Gingivitis, periodontitis, plaque		Microflora
		NS	S	NS	S	NS	S	
<b>Bulimia nervosa</b>								
Altshuler et al (1990)	40 BN 40 controls		↑	DMFS→	DS↑	GI→ PDI→ PI→		
Bretz et al (1989)	11 BN 3 AN 25 controls			DMFS→			Lactobacillus spp.↑	S. sobrinus↑, S. mutans↑
Howat et al (1990)	10 BN 10 controls		Erosion-index→			PI→		
Jones and Cleaton-Jones (1989)	11 BN 22 controls		↑	DMFS↑				
Kühnl et al (1990)	28 BN 35 controls				DMFS↑			PBI↓
Milosevic and Slade (1989)	33 BN (vomiting) 7 BN (restricting) 50 controls		↑	DMFS→		GI→ PI→		
Philipp et al (1991)	41 BN 50 controls		↑		DMFT↓			
Rytömaa et al (1998)	35 BN 105 controls		↑	DMFS↑ DS↑	Buccal-lingual and approximal caries↑		S. mutans→, Lactobacillus spp.→	
Touyz et al (1993)	15 BN 15 controls		↑	DMFT→		Pockets≅ 4 mm →	Bleeding sites and gingival recession↑	S. mutans→, Lactobacillus spp.→
Öhrn et al (1999) <sup>3</sup>	46 BN 7 AN+BN 3 AN 52 controls		↑	DS↑	DMFT↑ DFST			S. mutans↑, Lactobacillus spp.↑



**Table 5 Continued**

	Sample size	Erosion		Caries		Gingivitis, periodontitis, plaque		Microflora	
		NS	S	NS	S	NS	S	NS	S
<b>Anorexia nervosa</b>									
Bretz et al (1989)	11 BN 3 AN 25 controls			DMFS→				<i>Lactobacillus</i> spp. ↑	<i>S. sobrinus</i> ↑, <i>S. mutans</i> ↑
Liew et al (1991)	15 AN (restricting) 15 controls			DMFT→		Pockets≡ 4 mm →	Bleeding sites and gingival recession↑	<i>S. mutans</i> →, <i>Lactobacillus</i> spp.→	
Milosevic and Slade (1989)	18 AN 50 controls		↑	DMFS→		GI→ PI→			
Philipp et al (1991)	11 AN 50 controls		↑				DMFT↓		
Touyz et al (1993)	15 AN 15 controls		↑	DMFT→		Pockets≡ 4 mm →	Bleeding sites and gingival recession↑	<i>S. mutans</i> →, <i>Lactobacillus</i> spp.→	
Öhrn et al (1999) <sup>1</sup>	3 AN 7 AN+BN 46 BN 52 controls		↑	DS↑			DMFT↑ DFS↑		<i>S. mutans</i> ↑, <i>Lactobacillus</i> spp.↑

NS: non-significant; S: significant; AN: anorexia nervosa; BN: bulimia nervosa; DMFS: decayed missing filled surfaces; DS: decayed surfaces; GI: gingival index; PDI: periodontal index; PI: plaque index; PBI: bleeding index of the papilla; DMFT: decayed missing filled teeth; ↑: increased in AN or BN compared to controls; →: no difference between the groups; ↓: reduced in AN or BN compared to controls.

<sup>1</sup> The patient group consisted of both anorexics and bulimics without differentiating between the subgroups

sions primarily affecting the palatal surfaces of the incisor, canine and premolar teeth; b) erosions mainly confined to the labial surfaces of the incisor teeth; and c) a generalised but minimal loss of enamel on the occlusal surfaces of molars and premolars, reflected clinically by unnaturally highly polished amalgam restorations. Additionally, a marked difference in the localization of the dental erosions in vomiting and non-vomiting anorexics is described in a study: lingual erosions occur only when self-induced vomiting is practised, while the buccal type of erosion appears, albeit rarely, in non-vomiting persons (Hellström, 1977). However, a study describing the localization of dental erosions concurrent with identification of the dietary and gastric causes by a questionnaire indicates that the cause of dental erosion cannot reliably be identified by location of the erosion (Järvinen et al, 1992). History taking is therefore important when evaluating the causes of erosion.

Dental erosion is commonly found in both anorexics and bulimics, but not all persons with an eating disorder show pathological tooth wear. Liew et al (1991) examined 15 persons with anorexia nervosa classified as 'restricting anorexics without significant vomiting habits', and only one showed any erosion on enamel surfaces of the teeth. However, it has also been reported that non-vomiting anorexics have a higher level of dental erosion than a control population (Robb et al, 1995). In such cases this could be explained by an excessive intake of acidic foodstuffs. Differentiating between erosion, abrasion and attrition is not common in studies on oral complications in eating disorders. One study distinguishes between these types of tooth wear showing that erosion, abrasion and attrition is 1.5-6 times more frequent among bulimics than controls, with erosion being the most distinct and prevalent type (Rytömaa et al, 1998).

Although the degree of tooth wear is significantly greater in groups with eating disorders than in the normal population (Altshuler et al, 1990; Milosevic and Slade, 1989; Robb et al, 1995), and the most severe cases of erosion are seen in persons with a long history of intense vomiting (Hellström, 1977), no direct linear relationship between vomiting experience and tooth wear is described (Altshuler et al, 1990; Milosevic and Slade, 1989; Robb et al, 1995). This suggests that other parameters than vomiting frequency and duration may contribute to severity and progression of the erosions seen in persons with eating disorders. Low saliva flow could be such a parameter. Thus, Öhrn et al (2000), who followed 35 persons with eating disorders for one year and showed significant differences at both baseline and

after one year in stimulated salivary flow rates between persons with progression of erosive tooth wear and those without, suggest that salivary flow rate may serve as an indicator of risk of progression of erosive tooth wear in persons with eating disorders.

A study describing the oral hygiene procedures that persons with anorexia and bulimia practise in relation to vomiting did not show any differences in oral hygiene practises between persons with and without severe erosion (Milosevic et al, 1997). However, the record of hygiene practise could be of importance when explaining differences in the progression of dental erosions in persons with eating disorders. Likewise, dietary records with special emphasis on acidic foodstuffs should be obtained.

### **Caries**

Persons with high caries activity harbour more acidogenic and aciduric bacteria, i.e. bacteria able to produce acid (acidogenic) that grow and survive in an aciduric environment (aciduric), which upon exposure to sugar nutrients produces acid that reduces the plaque pH and makes it more acidogenic (Aranibar Quiroz, 2003). Saliva contains proteins that take part in pellicle formation and binding of these caries associated bacteria (Liljemark and Bloomquist, 1996). However, saliva also plays an important role in protection against caries, since saliva via its flow and buffering capacity can eliminate the sugar nutrients for acid producing bacteria and the acid formed by the bacteria in the dental plaque (Mandel, 1997). Therefore, a higher caries experience/progression in persons with anorexia and bulimia may theoretically be expected, since oral sugar clearance is reduced by the low unstimulated whole saliva flow rate (Dawes, 1983). Furthermore, changes towards lower pH in the oral cavity because of repeated self-induced vomiting could occur as a change in normal microflora towards a more aciduric type also associated with increased caries activity (Hoffer et al, 2004). However, reports of the caries experience in persons with eating disorders are conflicting (Table 5).

Some studies describing caries experience in persons with eating disorders (both anorexics and bulimics) and age-matched controls find no significant difference in various caries indices between the groups (Bretz et al, 1989; Liew et al, 1991; Milosevic and Slade, 1989; Roberts and Li, 1987; Touyz et al, 1993). Other studies describe the opposite and find persons with eating disorders with a higher caries index (Öhrn et al, 1999; Kühnl et al, 1990; Altshuler et al, 1990), and approximal and buccal-lingual caries lesions (Rytömaa et al,

1998). Furthermore, a study even describes a lower Decayed Missing Filled Tooth (DMFT) index in persons with eating disorders compared to controls (Philipp et al, 1991). However, it is still debatable whether the caries activity in vomiters is higher than in non-vomiters. Hurst et al (1977) find that both anorexic 'regurgitators' and especially 'vomiters' have a higher frequency of caries compared to anorexic 'non-vomiters', while Hellström (1977) find no differences in caries experience between vomiting and non-vomiting anorexics.

It remains unclear whether caries experience and activity of persons with eating disorders is greater than in the normal population. Future investigations should take the relationship between caries and caries-related variables such as salivary flow rate and composition, oral microflora, oral hygiene, intake of medication and dietary habits into greater consideration.

### **Plaque, Gingivitis and Periodontitis**

As most persons with anorexia and bulimia are relatively young it is not surprising that severe periodontal disease is rarely present (Liew et al, 1991; Rytömaa et al, 1998) (Table 5). The incidence of gingivitis, as evidenced by gingival bleeding (Liew et al, 1991; Touyz et al, 1993) and plaque level (Touyz et al, 1993), is described to be worse among anorexics and bulimics compared with controls, while other studies find no differences in plaque levels and gingival conditions (Altshuler et al, 1990; Milosevic and Slade, 1989). When not including healthy controls, Hurst et al (1997) find that the prevalence of periodontal inflammation is significantly more common in 'non-vomiting' compared to 'vomiting' or 'regurgitating' anorexics, while a comparison of anorexics with bulimics revealed no significant differences in either plaque index or gingival indices (Roberts and Li, 1987). While it does not seem that gingival and periodontal health problems are more prevalent in persons with eating disorders than in healthy individuals, the presence of severe nutrient deficiency, which could be seen in a person with anorexia and bulimia, may contribute to diminished gingival and periodontal health (Petti et al, 2000; Sawyer and Nwoku, 1985).

### **Oral Microflora**

It has been hypothesised that persons with anorexia and bulimia, because of self-induced vomiting resulting in acid regurgitation, have an oral microbial flora with a high proportion of aciduric and cariogenic organisms such as lactobacilli and oral streptococci,

especially mutans streptococci (*S. mutans*) (Bretz et al, 1989). Additionally, a low saliva flow rate with its low pH favours an aciduric oral microflora (Hofer et al, 2004).

Cultivation and measurement of the level of aciduric organisms in stimulated whole saliva in persons with both anorexia nervosa and bulimia nervosa shows that the persons with eating disorders have higher levels of *S. mutans* and *Streptococcus sobrinus* (*S. sobrinus*) than subjects in a control group. Within the group of eating disordered these findings are not associated with active caries lesions. The investigators therefore suggest that if the finding of an elevated salivary level of *S. sobrinus* is consistent in bulimics, consequently, a salivary culture for *S. sobrinus* could be a relevant tool in diagnosing this eating disorder (Bretz et al, 1989). The cause of an elevated salivary level of *S. sobrinus* in persons with bulimia has not yet been clarified, but it has been suggested that in persons not suffering from eating disorders the presence of *S. sobrinus* may be an indicator of a change to a more carbohydrate-rich diet (Huis in't Veld et al, 1982), while the acid regurgitation in children with gastroesophageal reflux *per se* does not have an influence on the prevalence of *S. sobrinus* (Holttä et al, 1997).

Öhrn et al (1999) support the finding of high salivary levels of *S. mutans* in persons with eating disorders, which are found in addition to a high level of lactobacilli. However, in this study the number of decayed tooth surfaces tended to be higher in the persons with eating disorders (both anorexics and bulimics) than in the reference group, especially in the 21-30-year-old age group.

By contrast, other studies find no significant differences in the salivary levels of either *S. mutans* or lactobacilli species between anorexics and their controls (Liew et al, 1991; Rytömaa et al, 1998), and between anorexics, bulimics and controls (Touyz et al, 1993). The latter study does not state whether it is plaque or saliva that is assessed (Table 5).

## **CONCLUSION AND PERSPECTIVES**

### **Does Saliva make a Difference?**

Some persons with anorexia and/or bulimia present with reduced salivary flow, reduced buffering capacity and salivary pH, which are factors that increase the risk for dental caries and erosion. Enamel and dentine erosions are common oral findings in the eating disordered due to the practise of self-induced vomiting

and intake of acidic foodstuffs. Dental caries, however, does not seem to be a common oral finding in these persons and may be explained by the maintenance of good oral hygiene.

### **Salivary Biomarkers of Anorexia and Bulimia**

A number of salivary findings may provide oral biomarkers of anorexia and bulimia. In addition to the clinical finding of enlarged major salivary glands, mainly the parotid glands, an elevated salivary level of *S. sobrinus*, which is also an indicator of a carbohydrate-rich diet, has been suggested as a biomarker of bulimia. Thus, further research is needed in order to describe this or any other differences in the oral microflora of persons with anorexia and bulimia compared to healthy controls.

Alterations in salivary flow and composition may act as oral biomarkers of anorexia and bulimia, i.e. reduced flow rate and xerostomia, as well as increased activity of salivary alpha-amylase isoenzymes. Additionally, possible changes in the electrolyte composition of saliva due to electrolyte and acid-base disturbances in plasma should be considered in the search for oral biomarkers of anorexia and bulimia. In future research, emphasis should be placed on factors that have or may have an effect on saliva flow rate and saliva composition, including: medication intake, duration and severity of disease, purging behaviour, and eating pattern. The heterogeneity of these groups of patients must be taken into consideration. Thus, the positive findings of saliva biomarkers should always be evaluated in relation to other physiological findings, as well as by interview and appearance of the person examined.

### **Perspective**

During routine clinical examination of young female patients the possibility of an eating disorder should be considered when dentists notice any dental erosion, perhaps combined with salivary gland enlargement, and where the patient's history excludes other explanations, i.e. excessive intake of acidic foodstuffs and a history of gastrointestinal reflux. This may lead to further oral investigations such as saliva sampling in preparation for analysis of flow rate, alpha-amylase activity and possibly electrolyte composition, which together will enhance the possibility of early diagnosis and thereby early treatment of the disease by referral to a psychiatric specialist. Additionally, timely initiation of dental prophylaxis and treatment can occur and improve dental prognosis. Early diagnosis is a prerequisite not only for dental prophylaxis and treatment but also

for treatment by other relevant health care workers, thereby allowing for improvement of both general health and dental prognosis.

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