

# Etiology of dental erosion – intrinsic factors

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Dental erosion due to intrinsic factors is caused by gastric acid reaching the oral cavity and the teeth as a result of vomiting or gastroesophageal reflux. Since clinical manifestation of dental erosion does not occur until gastric acid has acted on the dental hard tissues regularly over a period of several years, dental erosion caused by intrinsic factors has been observed only in those diseases which are associated with chronic vomiting or persistent gastroesophageal reflux over a long period. Examples of such conditions include disorders of the upper alimentary tract, specific metabolic and endocrine disorders, cases of medication side-effects and drug abuse, and certain psychosomatic disorders, e.g. stress-induced psychosomatic vomiting, anorexia and bulimia nervosa or rumination. Based on a review of the medical and dental literature, the main symptoms of all disorders which must be taken into account as possible intrinsic etiological factors of dental erosion are thoroughly discussed with respect to the clinical picture, prevalence and risk of erosion.

Dental erosion is caused not only by exogenous acids but also by the effect of gastric acids or acidic gastric contents reaching the oral cavity and the teeth as a result of vomiting, regurgitation, gastroesophageal reflux or rumination.

JÄRVINEN *et al.* (1) found the risk of erosion quadrupled by weekly regurgitation symptoms and up to eighteen times higher in patients with chronic vomiting compared with non-vomiting patients. This is conceivable in view of the fact that the pH value of gastric acid is 1–1.5 and thus far below the 5.5 level critical for the dissolving of dental enamel.

The theory of a causal connection between vomiting, regurgitation or gastroesophageal reflux and dental erosion was initially postulated in the dental literature by BARGEN & AUSTIN (2) in 1937 and HOLST & LANGE (3) in 1939. Internal specialists, too, had recognized the detrimental effect of gastric acid on the teeth by that time. JORES (4), for example, wrote in his textbook of internal medicine published in 1935: "Through regurgitation and vomiting, acidic gastric contents which attack the dental enamel gain access to the oral cavity".

The prevalence of dental erosion resulting from the action of gastric acid on the teeth, i.e. from vomiting, regurgitation or gastroesophageal reflux and rumination, is unknown. However, studies by SMITH & KNIGHT (5) and by JÄRVINEN *et al.* (6)

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suggest that internal factors, i.e. disorders with vomiting, regurgitation or reflux of gastric contents, have been crucial in approximately one-quarter of all cases of dental erosion.

*Vomiting*, which is the forceful expulsion of gastric contents through the mouth, is a common manifestation of many organic and psychosomatic disorders (7, 8) (Table 1). *Regurgitation* is distinguished from vomiting by the lack of abdominal diaphragmatic muscular contraction and the relatively small quantity of material ejected (8). *Regurgitation* and *reflux of gastric contents* into the mouth are generally seen with gastroesophageal sphincter incompetence, with increased gastric pressure, or with increased gastric volume (7–10) (Table 2). One special form of regurgitation is *rumination*, where the gastric contents are regurgitated, rechewed and reswallowed.

In theory, all disorders associated with vomiting, regurgitation or gastroesophageal reflux and rumination may result in erosion of the dental hard tissues due to the effect of gastric acid on the teeth. Clinical manifestation of dental erosion does not, however, usually occur until gastric acid has acted on the dental hard tissues regularly several times a week for a period of at least 1–2 yr (11–13).

Therefore dental erosion induced by gastric acid has been observed to date only in disorders associated with chronic vomiting, i.e. regular vomiting

occurring at least once a week for several years, with persistent regurgitation or gastroesophageal reflux or with protracted rumination, such as some disorders of the upper gastrointestinal tract, specific metabolic and endocrine disorders, cases of medication side-effects and drug abuse as well as certain psychosomatic disorders (stress-induced psychogenic vomiting, *anorexia* and *bulimia nervosa*, rumination).

Whether or not dental erosion occurs as an outcome of these disorders depends, however, just as their severity and progression, not only on the frequency and duration of vomiting or regurgitation but also on the oral hygiene habits of patients after their teeth have been exposed to gastric acid and on constitutional factors such as the nature of the dental hard tissues (degree of mineralization, fluoride content) and the quantity and quality of secreted saliva (1, 14, 15, 16).

#### Dental erosion as an outcome of upper gastrointestinal disorders

Dental erosion in upper gastrointestinal disorders may be either the result of frequent vomiting or persistent gastroesophageal reflux or regurgitation (3, 17–24).

#### Upper gastrointestinal disorders accompanied by chronic vomiting, persistent regurgitation or gastroesophageal reflux

In peptic ulcer and chronic gastritis, *vomiting* may be either spontaneous or self-induced and leads to a temporary amelioration in symptoms, particularly if antral or pyloric endema has resulted in gastric outlet obstruction (8).

Vomiting is also a regular occurrence in patients with disordered gastrointestinal motility, including postvagotomy, diabetic or idiopathic gastroparesis, other gastric dysrhythmias resulting from abnormal myoelectric activity, and intestinal pseudo-obstruction due to abnormal intestinal myogenic or neurogenic function (8). Other intestinal obstructions, e.g. adhesions, malignancy, hernia or volvulus, are also accompanied by vomiting (Table 1).

*Gastroesophageal reflux*, i.e. the reflux of gastric contents into the esophagus without retching or vomiting, is due in most cases to incompetence of the gastroesophageal sphincter, which may occur primarily as the result of a functional disorder or secondarily as the result of various organic disorders, listed in Table 2 (7–10, 25). Also in cases of normal gastroesophageal sphincter function there may be gastroesophageal reflux or regurgitation when the intraabdominal pressure is increased, for

Table 1  
Causes of vomiting (7, 8)

<i>Disorders of the alimentary tract</i>
• peptic ulcer, chronic gastritis
• disordered gastrointestinal motility (postvagotomy, diabetic, idiopathic gastroparesis)
• intestinal obstruction (e.g. adhesions, malignancy, hernia, volvulus)
• infections of the intestinal tract (gastroenteritis, pancreatitis, hepatitis, cholecystitis, cholangitis)
<i>Central nervous system disorders with increased intracranial pressure</i> (e.g. encephalitis, neoplasms, hydrocephalus)
<i>Neurological disorders</i>
• migraine headaches
• diabetic crisis
• diabetic or alcoholic polyneuropathy
• disorders of the labyrinthine apparatus (e.g. Méniere's disease, benign recurrent vertigo)
<i>Metabolic and endocrine disorders</i>
• uremia
• diabetic ketoacidosis
• hypo-, hyperparathyroidism
• hyperthyroid crisis
• adrenal insufficiency
• pregnancy (hyperemesis gravidarum)
<i>Side-effect of drugs</i>
• central emetic effect (e.g. digitalis, estrogens, chemotherapeutic agents, emetine, histamine, beta blockers, tetracycline, levodopa, opioids)
• gastric irritation with secondary effect of vomiting (e.g. alcohol, salicylates, aminophylline, ipecacuanha, ferrous sulfate, potassium chloride, diuretics)
<i>Psychosomatic disorders</i>
• stress induced psychogenic vomiting
• eating disorders (anorexia nervosa, bulimia nervosa)

Table 2

*Causes of gastroesophageal reflux and regurgitation (7-10)*

<i>Incompetence of the gastroesophageal sphincter</i>
Primary
• idiopathic with or without hiatus hernia secondary
Secondary
• impairment of the gastroesophageal sphincter by progressive systemic sclerosis, mixed connective tissue disease, and neurogenic disorders (e.g. diabetic and alcoholic polyneuropathia)
• destruction of the sphincter by surgical resection, myotomy, balloon dilatation or esophagitis
• neurohumoral induced decrease of gastroesophageal sphincter pressure by drugs (e.g. beta-adrenergics, serotonin, cholecystokinin, diazepam, glukagon), increased estrogen and progesterone (luteal phase of menstrual cycle, pregnancy, intake of oral contraceptives), diet (fatty meals, peppermint, chocolate, coffee, alcohol) or smoking
<i>Increased intraabdominal pressure</i>
• obesity
• pregnancy
• ascites
<i>Increased intragastric volume</i>
• after meals
• pyloric spasm
• obstruction due to peptic ulcer, gastroparesis
• gastric stasis syndrome

example due to pregnancy or obesity. Another reason for gastroesophageal reflux or regurgitation might be an increased intragastric volume, for instance after large meals, or obstruction due to peptic ulcer or pyloric spasm (7-10).

Prevalence data on gastroesophageal reflux disease have been difficult to obtain due to wide variability of its clinical presentation (26). The classic symptoms of heartburn and regurgitation are well known to the majority of the population. A large survey of healthy hospital staff members, carried out by NEBEL *et al.* (27), indicated that approximately 7% of normal individuals can be expected to experience reflux symptoms at least once a month. Most individuals with reflux symptoms are never seen by physicians, although silent chronic gastroesophageal reflux may be implicated in a variety of pulmonary diseases (25). Also in children, silent reflux may be an important contributing factor in intractable asthma and chronic lung disease (28). It is estimated that up to three quarters of children with recurrent apnoeas have pathological gastroesophageal reflux (28).

Furthermore, function of the gastroesophageal sphincter is influenced by various neurohumoral factors, including diet, alcohol, and smoking (7-10, 25) (Table 2).

#### **Frequency and location of dental erosion in patients with disorders of the upper gastrointestinal tract**

In 1939, HOLST & LANGE (3) were the first authors to suggest a connection between dental erosion and vomiting induced by a gastric disorder. They described 6 cases in which "gastric dysfunction

with periodical or continuous vomiting and pyrosis for a lengthy period" had led to "uniform tooth wasting from non-mechanical causes - perimyolysis". Further case reports were published by GUERNSEY (19) (a patient with "duodenal cancer" who had vomited "quite frequently for a period of years"), by ALLAN (17) (a patient with persistent vomiting, on average once per day, for 8 yrs due to a *duodenal ulcer*), by HOWDEN (20) (a patient with a history of frequent vomiting after meals for the previous 2 yr as the result of a *hiatus hernia*) and by MYLLÄRNIEMI & SAARIO (23) (four patients with *sliding hiatus hernia* suffering from "long-lasting and severe regurgitation").

In all quoted cases, it was only the palatal surfaces of the upper incisors, followed later by the palatal and occlusal surfaces of the upper premolars and molars, which were affected by erosion, with the lower teeth remaining largely undamaged.

The fact that even the teeth of the primary dentition may be damaged by regurgitation or gastroesophageal reflux is illustrated by the case reports published by GALLO & RANDEL (18) and by TAYLOR *et al.* (24). One report dealt with a 3.5-yr-old girl suffering from *myodystrophia fetalis deformans (arthrogryposis multiplex congenita)*, in whom feedings of milk or cereals made with milk usually resulted in regurgitation, whereas semisolid food meals without milk were vomited (18). The other report dealt with an 8-yr-old non-handicapped girl with a long history of asymptomatic gastroesophageal reflux, especially at night (24). As a result of the frequent exposure of the teeth to gastric content, the

maxillary and mandibular anterior teeth as well as the upper and lower premolars in both children were damaged by erosion with, in contrast to adult patients, the mandibular teeth being most affected by erosion.

Apart from these case reports, only two studies (21, 22) investigating the occurrence of dental erosion in a large patient group with disorders of the upper gastrointestinal tract have been published to date in the dental literature. Therefore, no exact data are available on the frequency of dental erosion in the different disorders of the upper gastrointestinal tract associated with vomiting or gastroesophageal reflux.

JÄRVINEN *et al.* (21) examined 109 patients suffering from upper gastrointestinal symptoms, with esophageal reflux being diagnosed in 20, duodenal ulcer in 24, and gastric ulcer in 17 cases. The remaining 48 patients had undergone cholecystectomy. Dental erosions were recorded in only 7 of these 109 patients: in 4 with esophageal reflux and in 3 with duodenal ulcer. The affected patients were between 32 and 59 yr old and had been suffering from the disorders for 5 to 40 yr. The erosions were located mainly on the palatal and occlusal surfaces of the upper teeth, with the greatest loss of substance being recorded on the incisors. No direct correlation was found between the severity of the erosion and the duration of gastrointestinal symptoms or the frequency of gastroesophageal reflux or regurgitation, although the most severe erosions were registered in those patients with disorders dating back at least 10 yr.

In a study of 107 patients with reflux disease, MEURMAN *et al.* (22) recorded dental erosion in 28 patients (26%). Patients in which erosion occurred were older on average than those without erosion (54 versus 49 yr) and had been suffering longer from reflux disease (17 versus 11 yr). The severity of the reflux disease was also more marked than in those without dental erosion.

In addition, the fact that the number of patients with a low salivary buffering capacity was higher among those patients with erosion than among those without (22) and the observations by JÄRVINEN *et al.* (21), who found a reduced flow rate of resting saliva in 3 of their 7 patients with erosion, suggest that the risk of dental erosion in patients with gastroesophageal reflux is also influenced by salivation. This hypothesis was confirmed by studies carried out by JÄRVINEN *et al.* (1) on the risk factors in dental erosion. These authors found that patients with an unstimulated salivary flow rate of 0.1 ml/min or less had a five-fold greater risk of erosion than patients with a higher flow rate.

#### Dental erosion in connection with metabolic and endocrine disorders

Nausea and vomiting commonly accompany several metabolic and endocrine disorders, including uremia, diabetic ketoacidosis, hypo- and hyperparathyroidism, hyperthyroid crisis, and adrenal insufficiency (8). The morning sickness of pregnancy is another instance of vomiting possibly related to hormonal changes. Such vomiting occurring very frequently over a period of several weeks is referred to as *hyperemesis gravidarum*.

Reports on dental erosion in connection with metabolic or endocrine disorders are rare in the dental literature. The erosions described by FINCH (29) in patients with *diabetes insipidus* and by XHONGA & VAN HERLE (30) in patients with *hyperthyroidism* were located solely on the vestibular surfaces of the teeth and were thus not due to chronic vomiting. The cause of erosion in the patients with *diabetes insipidus* is more likely to have been the illness-induced excessive intake of acidic juices and soft drinks (29), whereas the limited salivary flow rate (the average stimulated salivary flow rate was 0.67 ml/min) can be assumed to have been a crucial cause of erosion in the patients with *hyperthyroidism* (30).

SAMPSON & MEISTER (31) reported the case of a 30-yr-old woman who had been suffering for 15 yr from *chronic renal failure* due to glomerulonephritis and had to undergo hemodialysis three times a week. As the outcome of frequent vomiting during dialysis over a period of several years, the patient displayed an excessive loss of tooth substance due to erosion. Besides the palatal surfaces of the maxillary teeth, the occlusal surfaces of the maxillary and mandibular posterior teeth were also affected, resulting in a marked loss of vertical dimension.

Although vomiting is a common occurrence during *pregnancy*, it rarely leads to detectable dental erosions because the period of pregnancy is limited. Only when *hyperemesis gravidarum* with frequent vomiting has occurred in a number of successive pregnancies may dental erosion become apparent (13).

#### Dental erosion as an indirect side-effect of drugs

Dental erosion as a side-effect of medication is observed not only as a direct outcome of the frequent intake of acidic drugs retained in the patient's mouth for a sustained period but may also be triggered indirectly by drug intake leading to vomiting. There are many drugs whose side-effects include vomiting (32, 33) (Table 3). A distinction can be made between those with a central emetic effect,

Table 3

List of drugs that may cause vomiting as a side-effect (based on 25, 26)

<i>Anorectics</i>	<i>Drugs acting on the peripheral circulation</i>	<i>Opioid analgesics</i>
fenfluramine	buphenine	alfentanil
amfepramone	co-dergocrine	buprenorphine
piracetam	isoxsuprine	butorphanol
phendimetrazine		ciramadol
mazindol		conorfone
<i>Antiallergic drugs and antitussives</i>	<i>Drugs affecting autonomic function or the extrapyramidal system</i>	cyclazocine
clofedanol	amantadine	dezocine
cranoglycate disodium	carbidopa	nalbuphine
letosteine	dopamine	naloxone
<i>Antibiotics</i>	ergometrine	naltrexone
tetracyclines	ergotamine	pentazocine
<i>Anticonvulsants</i>	mesulergine	sufentanil
progabide	piribedil	tramadol
$\gamma$ -vinyl-GABA	serotonin	
zonisamide	tyrosine	<i>Opioid agonists</i>
<i>Antifungal drugs</i>		alfentanil
nystatin	amantadine	buprenorphine
<i>Antihypertensive drugs</i>		butorphanol
nitroprusside	cannabis nutmeg	ciramadol
clonidine	lysergide tetrahydrocannabinol	
<i>Anti-inflammatory analgesics and drugs used in gout</i>		<i>Positive inotropic drugs and drugs used in dysrhythmias</i>
ibuprofen	salazosulfapyridine	aprinidine
indometacin	mercaptamine	bretylium
phenylbutazone	pentagastrin	digitalis glycosides
piroxicam	loperamide	flecainide
<i>Antiprotozoal drugs</i>		lorcainide
iodoquinol	cyclopropane	tocainide
emetine	isoflurane	
<i>Antipyretic analgesics</i>		<i>Prostaglandins</i>
acetylsalicylic acid and related compounds	benzodiazepines	Sex hormones
<i>Central nervous system stimulants</i>	chloralhydrate	estrogens
caffeine	ethylchlorynol	tamoxifen
theophylline and related substances	methaqualone	
doxapram		<i>Stimulant and anorectic agents</i>
euprophylline		cocaine
lobeline	Immunemodulating agents	methylphenidate
nikethamide	picibanil	
propoxyphynline	lithium	<i>Tricyclic antidepressants</i>
<i>Cytostatic and immunosuppressive drugs</i>		fluoxetine
<i>Diuretics</i>	Metals	fluvoxamine
spironolactone	gallium nitrate	tryptophan
thiazide diuretics	gold salts	viloxazine
triamterene	iron salts	
	selenium	
	zinc	
	<i>Metal antagonists</i>	
	dimercaprol	

e.g. digitalis, estrogen, chemotherapeutic agents, emetine, histamine, beta-blockers, tetracycline, levodopa and opioids, and others which induce vomiting as a secondary effect through drug-induced gastric irritation, e.g. alcohol, salicylates, amorphophylline, ipecacuanha, ferrous sulfate, potassium chloride and diuretics (8).

Dental erosion is to be expected, however, only if these drugs have to be taken over a long period, i.e. for several years, or in cases of drug abuse, e.g. alcoholism (34–36) or opioid dependency (37).

Another medication side-effect playing an important role in the development and progression of dental erosion is drug-induced xerostomia. The

importance of unstimulated salivary flow rate in determining the occurrence and progression of dental erosion has been pointed out by various authors including JÄRVINEN *et al.* (1), HELLSTRÖM (10) and WÖLTGENS *et al.* (32). Besides other etiological factors, such as radiation-induced damage to the salivary glands or irreversible glandular changes due to autoimmune diseases, drug-induced decrease of salivary secretion is a common cause of xerostomia, especially in the elderly (38, 39). In a study conducted on 1148 70-yr-old individuals ÖSTERBERG *et al.* (38) observed that 16% of the men and 25% of the women complained of dry mouth. The prevalence of xerostomia was positive-

ly correlated with the consumption of anticholinergic drugs, antihistamines, sedatives, hypnotics and phenothiazines. PARVINEN (40) who measured the rate of flow of whole, paraffin stimulated saliva in 463 medicated and 642 nonmedicated persons aged between 30 and 70 yrs found in 17% of the entire group mean flow rates of less than 1 ml/min; 5.8% showed flow rates of less than 0.7 ml/min. The medicated subjects had a lower mean flow rate than the unmedicated. However, this difference was statistically significant only in those persons on antihypertensive drugs, tricyclic antidepressants and neuroleptic medicaments. A thorough reference guide to drugs and dry mouth has been published recently by SREEBNY *et al.* (41) who listed more than 400 drugs that may cause xerostomia.

#### Dental erosion in chronic alcoholism

Alcoholism is a very common condition thought to affect about 10% of the adult population (43, 44). The lifetime risk for primary alcoholism, i.e. for alcoholism not resulting from psychiatric illness, in most western countries is about 10 % for men and 3–5% for women (45). Besides numerous pathological changes in other organic systems, acute alcohol intake can result in reflux of gastric contents with inflammation of the esophagus and stomach, followed by chronic esophagitis and gastritis with persistent gastroesophageal reflux.

This often symptom-free reflux and the early-morning vomiting frequently occurring in chronic heavy drinkers are the causes of the dental erosion found in many alcoholics (34–36). Alcohol abuse as an etiological factor for erosion often remains undetected, as chronic alcoholism is a condition which still carries a large social stigma and most patients are secretive about it. SMITH & KNIGHT (5), who investigated the etiological factors of tooth wear in 100 patients, found chronic alcoholism to be the cause in 9 of the 18 patients with erosion resulting from vomiting and regurgitation. In contrast, ROBB & SMITH (34) recorded signs of pathological tooth wear of an erosive nature in 34 cases (92%) among 37 alcoholics, with those who drank regularly displaying a more marked loss of dental hard substance than those who binge drank, i.e. drank large amounts of alcohol in phases lasting up to 1–2 wk, having dry periods in between. As the previously published case reports by SIMMONS & THOMPSON (35) and by SMITH & ROBB (36) show, it is the palatal surfaces of the upper anterior teeth which are most severely affected by erosion in alcoholics. At an advanced stage of erosion, a severe loss of substance has also occurred on the palatal and occlusal surfaces of the maxillary posterior teeth and at the incisal edges of the anterior

teeth (Fig. 1), whereas the mandibular teeth and the buccal surfaces of the maxillary teeth are hardly affected by erosion.

#### Stress-induced psychogenic vomiting as a cause of dental erosion

Psychogenic vomiting appears to be an exacerbation of a long-standing pattern of episodic stress-related vomiting.

WRUBLE *et al.* (46) summarized the clinical features of psychogenic vomiting as follows (36): (1) usually chronic and episodic; (2) typically occurs soon after meal has begun or just after it has been completed. Can be suppressed if necessary; (3) commonly occurs in the absence of nausea – “the food just seems to come back up”; (4) the vomiting act is often self-induced; (5) rarely associated with retching; (6) weight loss is not significant; (7) vomiting is often of relatively little concern to the patients – of more concern to the family; (8) no definite “personality type”.

Based on these characteristics, psychogenic vomiting can be easily distinguished from the likewise self-induced vomiting of patients with *anorexia* and *bulimia nervosa*, although some cases may overlap in view of the fact that stress is a triggering factor for binge eating and subsequent vomiting in patients with *bulimia nervosa* in particular (see below).

Some case reports (2, 47, 48) describing dental erosion as the outcome of many years' chronic vomiting are found in the dental literature. However, closer scrutiny of the case histories suggests that the psychogenic vomiting in some of these patients was not an independent disorder but rather a symptom of an undetected bulimic eating disorder.

#### Dental erosion as a symptom of anorexia and bulimia nervosa

Among the different disorders that may cause dental erosion in young patients, bulimic eating disorders are of special interest, as the prevalence of these psychosomatic disorders, especially among young women aged between 20 and 30 yrs, is very high at about 5% in the Western industrial countries and still increasing (49, 50). In patients with an eating disorder it must be differentiated between those suffering from *anorexia nervosa* and those suffering from *bulimia nervosa*.

#### Anorexia and bulimia nervosa: clinical pictures

The primary features of *anorexia nervosa* are profound weight loss due to self starvation and body image distortion (49). Depending on whether ano-



Fig. 1. Severe erosion of the palatal and occlusal or incisal surfaces in maxillary teeth of a 56-yr-old woman with a 6-yr history of alcoholism and regular vomiting. A) Occlusal view of the maxillary teeth. B) Frontal view.

anorectic patients manage to lose weight through ascetic fasting alone or by self-induced vomiting and laxative abuse as additional means, a distinction is made between '*restrictive*' and '*bulimic*' *anorexia nervosa* (50). *Bulimia nervosa* is characterized by

binge-eating and self-purging of ingested food by vomiting and laxative abuse (49). In contrast to the underweight anorectic patients, the body weight of most *bulimia nervosa* patients remains within normal range (49, 52).

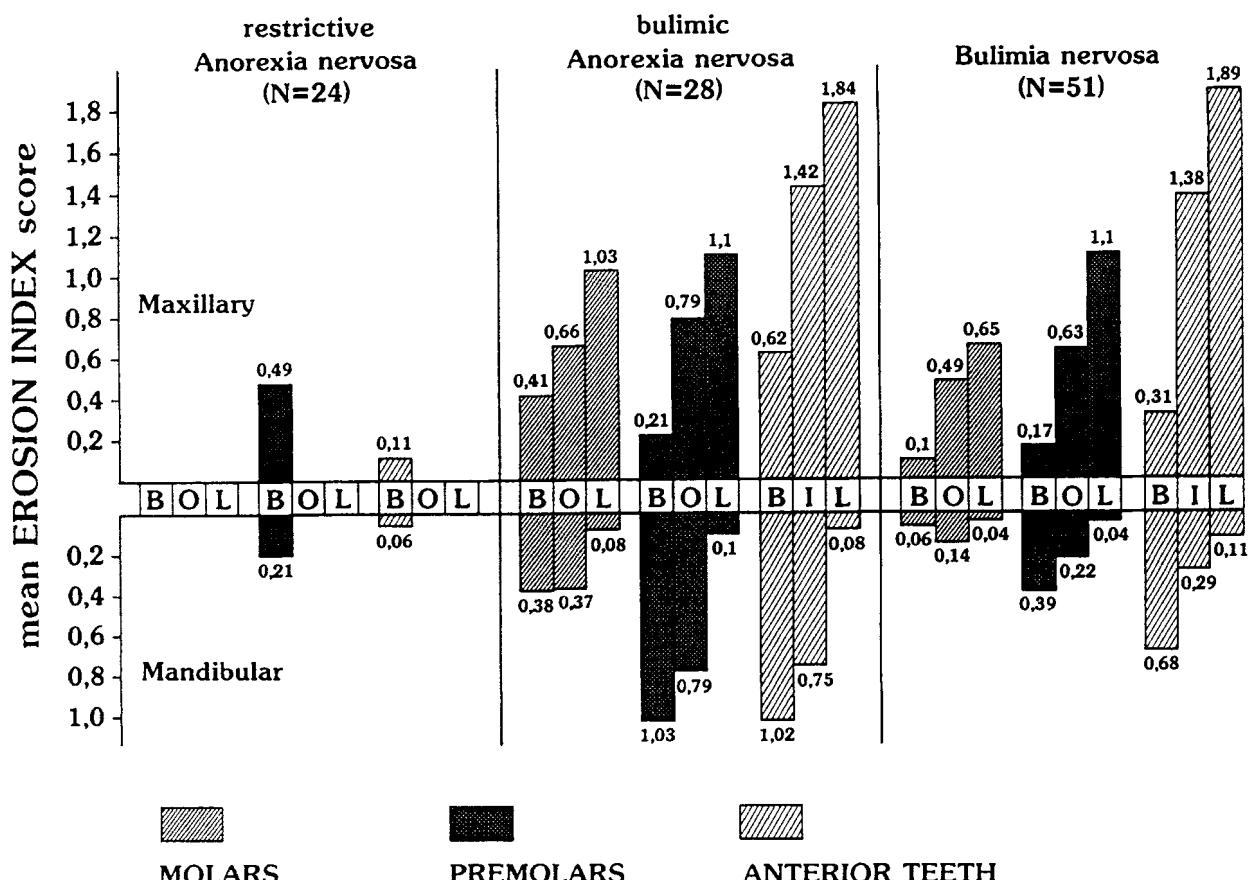


Fig. 2. Mean Erosion Index score (based on the *Tooth Wear Index* of SMITH & KNIGHT (66)) of the buccal (B), occlusal (O), incisal (I) and lingual (L) surfaces of the maxillary and mandibular molars, premolars and front teeth in patients with eating disorders (58).

Both anorexia nervosa and bulimia nervosa are psychosomatic disorders with an addictive character. Both diseases represent a vain attempt to cope with, or at least to suppress, personal conflicts and problems through preoccupation with food intake and body weight (53). Women are more often affected by anorexia and bulimia nervosa, while male patients account for less than 10% (54). The results of epidemiological studies carried out in recent years put the proportion of Western European and North American women aged between 18 and 35 yrs suffering from bulimia nervosa at 5% (50). The majority of patients with anorexia nervosa are aged between 12 and 20 yr, with a prevalence in this age group of about 2% (49). In both eating disorders, the disturbed eating pattern and the associated malnutrition lead to numerous somatic complications, some of them very severe with a potentially fatal outcome (55, 56).

Early diagnosis of these eating disorders is important if they are to be treated successfully. This normally presents no problem in severely underweight patients with anorexia nervosa. Because bulimia nervosa patients retain their normal weight, however, it generally takes many years for their disorder to be correctly diagnosed. Their medical symptoms are highly non-specific and the patients are unwilling to reveal their abnormal eating behaviour, a source of great shame to them, to either the family or the doctor. For this reason it is often the dentist who first diagnoses a bulimic eating disorder based on the dental erosion induced by chronic vomiting (11, 12, 57, 58).

#### **Frequency and location of dental erosion in patients with anorexia nervosa and bulimia nervosa**

Erosions on teeth of patients with abnormal eating patterns and chronic vomiting were reported in the dental literature (2, 13) when the clinical picture of anorexia nervosa was still relatively unknown and bulimia nervosa had yet to be defined as a separate syndrome. BARGEN & AUSTIN (2), for example, reported in 1937 the case of a 26-yr-old teacher with pronounced erosions on the palatal and occlusal surfaces of her maxillary teeth. The patient, described as a 'walking skeleton', used laxatives regularly and vomited after each meal without any organic explanation being found. According to current expertise, the patient was undoubtedly suffering from bulimic anorexia nervosa which was, however, not diagnosed at that time.

From 1980 onwards, the dental literature provides numerous case reports (47, 57, 59–61) describing irreversible dental damage due to erosion in patients with anorexia or bulimia nervosa. The

prevalence of dental erosion in anorexia and bulimia nervosa has been investigated in many studies involving large collectives of patients with eating disorders (11, 12, 52, 62–65), but only a few authors (11, 12, 52, 65) have made a clear distinction between the various groups of eating disorders. These studies suggest that approximately 20% of patients with restrictive anorexia nervosa, and more than 90% of those with bulimic anorexia nervosa and with bulimia nervosa, are affected by dental erosion, with clear-cut differences being recorded between the different groups of patients with respect to location and severity of the erosions (Fig. 2) (11, 12, 52, 65).

In patients with restrictive anorexia nervosa, the erosions which are caused exclusively by extrinsic dietary factors are confined to the vestibular tooth surfaces (Fig. 2) (11, 12, 52), whereas in patients with bulimic anorexia nervosa or bulimia nervosa, it is essentially the palatal surfaces of the maxillary teeth, in particular of the incisors, that are damaged by erosion (Fig. 2) (11, 12, 52, 62, 63, 65). Based on a detailed history of the dietary habits and eating disorder patterns of more than 100 bulimic patients, HELLSTRÖM (11) and SCHEUTZEL (52, 58) confirmed a relationship between the location of erosions and the underlying cause in bulimic eating disorders: Only in those bulimic patients consuming larger quantities of acidic foods, especially cola beverages, were the palatal erosions supplemented by vestibular erosions without any association between the palatal and buccal erosions (Fig. 3). In contrast, in bulimic patients with only a low consumption of acidic foods, the erosive lesions are initially confined to the palatal and occlusal surfaces of maxillary teeth (Fig. 4). Only when regular vomiting persisted over a period of 5 yrs or longer the labial and buccal surfaces of the teeth were also affected by erosion (Fig. 5).

#### **Severity and progression of dental erosion in patients with anorexia and bulimia nervosa**

A statistically significant correlation between the severity of dental erosion in patients with bulimic anorexia nervosa or bulimia nervosa and the duration of illness was confirmed by SCHEUTZEL (15, 52). In the same study, a relationship between the severity of erosions induced by chronic vomiting and the post-vomiting oral hygiene habits of bulimia nervosa patients was detected. With regard to the duration of illness, it was noticeable that those bulimia nervosa patients who regularly brush their teeth shortly after vomiting have more severe erosions than those who only rinse their mouths with water or observe no oral hygiene after

vomiting. These differences were statistically significant and support the *in vitro* results reported by SCHWEIZER-HIRT *et al.* (67) who have shown that even brushing without toothpaste immediately after exposure of a tooth surface to an acid attack leads to a substantial loss of enamel. On the other hand, a recently published study by ROBB *et al.* (65) failed to confirm these results, as the authors detected no relationship between post-vomiting oral hygiene and level of erosion in patients with bulimic eating disorders. Different methodological approaches were, however, used in the two studies. In contrast to ROBB *et al.* (65), who chose the frequency of vomiting as the control-variable, SCHEUTZEL (15) took the individual duration of eating disorder as the basis of analysis. The latter seems to be more realistic, as the case histories of bulimic patients usually show a high fluctuation in frequency and duration of vomiting (52). It must also be borne in mind that many patients do not tell the truth about their vomiting habit, because they feel ashamed. Further research is needed in this area to give a satisfactory answer to the question of the influence of different oral hygiene habits and of the time factor in particular on the progression of dental erosion.

#### Dental erosion as an outcome of rumination

The term 'rumination' derives from the Latin *ruminare* and means 'cud chewing up to the throat [ruma]'. Human *rumination* or *mercycism* implies, with reference to the rumination of certain herbivores, the regurgitation of food from the stomach back into the mouth, where it is subsequently re-chewed and then reswallowed. This phenomenon usually begins 15 to 30 min after meals, lasts for 30 min to 1 h, with 15 to 20 regurgitations within this period (67, 68).

The mechanisms underlying human rumination are not well understood, but since reverse peristalsis does not appear to occur in humans, regurgitation is accomplished by a combination of relaxation of the upper esophageal sphincter, tightening of the abdominal muscles, and squeezing contraction of the gastric musculature (67).

In infants rumination is a common feeding disorder which typically develops between 3 and 6 months of age and is believed to be a psychosomatic illness, resulting from a poor mother-infant relationship (69). In the mentally healthy adult, rumination occurs both as an independent clinical picture and in combination with anorexia and bulimia nervosa (70). The causes of rumination in the adult are open to controversy. In many adult patients, the ruminating behavior has existed since childhood and is seen as a somatic

projection of psychic conflicts without any specific psychiatric disorder being present (71). It may also be stress-induced. Precise data are not available on the prevalence of rumination, as most patients are highly secretive about this socially unacceptable behavior. However, the pilot retrospect by PARRY-JONES (68) and more recent studies indicate that the prevalence of rumination is certainly higher than previously assumed. For example the AMERICAN PSYCHIATRIC ASSOCIATION speaks in its 'Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R)' of an "apparently very rare" disorder (49). Men suffer more frequently from rumination than women (68, 71). MAYES *et al.* (71) recorded a male : female ratio of 5:1 in 66 patients examined.

#### Frequency and location of dental erosion in ruminating patients

The dental literature reports 11 cases of rumination, with men forming the majority with 7 cases. The patients, most of them academics, were between 26 and 58 yr old. In all cases the teeth were markedly affected by erosion, the factor which initially drew attention to the presence of ruminating behavior.

In 1940, LANGE (72) described the case of a 36-yr-old man who had been suffering for some 2 yr from rumination; he regurgitated his gastric contents after a meal, keeping them in his mouth for some time while pressing his tongue against his palate and the palatal surfaces of the upper incisors before reswallowing. This procedure was repeated several times, generally until it was impossible to bring up any more, which means about ten times within 30 to 45 min after the intake of the meal. Measurements taken during rumination revealed a sharp fall in the pH value on the tongue ( $\text{pH}=3.7$ ), whereas the pH value in the vestibulum was virtually normal ( $\text{pH}=6.5$ ). As an outcome of the frequent contact with the acidic gastric contents, the palatal surfaces of the upper incisors were eroded concavely almost to the pulp. A less pronounced but also clearly defined loss of substance was also recorded on the palatal and occlusal surfaces of the upper premolars. We also found a similar erosion pattern in an 18-yr-old ruminating patient (Fig. 6) who, as in the case reported by LANGE (78), regurgitated mainly liquid food and pressed it against the palatal surfaces of the upper incisors. The fact that this erosion location is not necessarily typical of all ruminating patients is reflected in the results published by GILMOUR & BECKETT (73), who examined 10 patients suffering from rumination. They not only found erosion in all cases on the palatal surfaces of the upper incisors but also pronounced loss of dental hard tissue

on the occlusal surfaces of the upper and lower molars and premolars due to erosion resulting from the patients rechewing the gastric contents before reswallowing (Fig. 7). In addition, some patients had erosions on the buccal surfaces of the lower premolar and canine teeth resulting from their depositing the gastric contents in the buccal sulci before reswallowing (73).

Thus, in contrast to patients with anorexia or bulimia nervosa practising self-induced vomiting, no typical erosion pattern can be detected in ruminating patients. The fact is rather that the location of dental erosion depends on the individual ruminating behaviour.

#### **Saliva as a contributing factor in the etiology of dental erosion**

As MEURMAN & TEN CATE (16) pointed out in their review on the pathology of dental erosion, salivary secretion is probably of central importance in the progression of erosion. However, up to now there have been only a few studies on the etiology and pathology of dental erosion where salivary secretion was taken into account besides other factors. The importance of unstimulated salivary flow rate in determining the prevalence and progression of dental erosion was pointed out by JÄRVINEN *et al.* (21), HELLSTRÖM (11), and WÖLTGENS *et al.* (42). These authors found the flow rate of unstimulated saliva significantly lowered in erosion patients suffering from upper gastrointestinal disorders or an anorectic disorder. In contrast, MEURMAN *et al.* (22) failed to confirm a statistically significant difference in salivary flow rates of unstimulated or stimulated saliva between 117 reflux disease patients with or without dental erosion, although the number of patients with low salivary buffering capacity was higher among those patients with erosions than in those without. GUDMUNDSSON *et al.* (75) also found a low salivary buffering capacity in most of their erosion patients with gastroesophageal reflux. In a comprehensive study of dental characteristics and laboratory findings in 103 patients with anorexia and bulimia nervosa SCHEUTZEL (15, 52) found a significantly lower mean flow rate of unstimulated and stimulated whole saliva in those patients with bulimic anorexia nervosa having more severe erosions than in the patients with bulimia nervosa and the same duration of eating disorder. The salivary buffer capacity in bulimic anorexia nervosa patients was, however, somewhat higher than in bulimia nervosa patients. This can be explained by the different dietary patterns in these two eating disorders (52, 75, 76). The question of whether the flow

rate of unstimulated or stimulated saliva or both and/or the salivary buffer capacity is the most important factor influencing the progression of erosion is not yet answered.

#### **Conclusions**

In contrast to extrinsic factors where several different types of exogenous organic and inorganic acids must be taken into consideration as possible etiological factors, dental erosion due to intrinsic factors is caused in all cases by gastric acid acting regularly on the dental hard tissues over a period of several years as the result of chronic vomiting or persistent gastroesophageal reflux, regurgitation or rumination.

A review of the dental literature revealed that a bulimic eating disorder is the underlying cause in most cases of dental erosion due to chronic vomiting. The prevalence of these psychosomatic disorders especially among young women between 20 and 30 yrs is very high at about 5% in the western industrial countries and still increasing. Recent studies suggest that approximately 90% of bulimic patients are affected by dental erosion. Other possible causes of long-term regular vomiting resulting in dental erosion are disorders of the alimentary tract, metabolic and endocrine disorders or medication side effects. Another, very common disorder which must be taken into account as a possible etiological intrinsic factor of dental erosion is persistent gastroesophageal reflux. Recent studies indicate that about 70% of healthy individuals can be expected to experience reflux symptoms daily. Although many studies have confirmed a relationship between chronic reflux disease and dental erosion, further research is needed to establish this relationship scientifically in terms of frequency and duration. Oral hygiene habits and constitutional factors such as the nature of dental hard tissues and the salivary secretion seem to play an important role in the pathology of dental erosion. A relationship between the severity of erosion and the post-vomiting oral hygiene habits was found in only one study dealing with bulimic patients. Further exploration is needed to assess the influence of different oral hygiene habits and of the time factor on the progression of erosion. Another question which must be clarified in further epidemiological studies is the influence of the etiology on the location of the erosive lesions. Whereas some authors dispute any relationship between the underlying cause and the localisation of dental erosion, others have found a significant correlation between chronic vomiting or persistent gastroesophageal reflux and palatal and occlusal erosion.



*Fig. 3.* Patient suffering from bulimic anorexia nervosa with daily vomiting and additional high consumption of fruit juice and cola beverages. A) Palatal erosion due to daily vomiting during the past 4 years. B) Additional buccal erosion due to frequent exposure to dietary acids.

*Fig. 4.* A, B) Erosion confined to the palatal surfaces of the maxillary teeth, the result of chronic vomiting in an anorectic patient with a low intake of dietary acids.

*Fig. 5.* A, B) Total loss of the enamel on the maxillary incisors and profound erosions on the palatal and occlusal surfaces of the maxillary posterior teeth as a result of bulimic anorexia nervosa with daily vomiting for at least 6 yr.

*Fig. 6.* Concave erosion of palatal surfaces of maxillary incisors and canines in an 18-yr-old man with rumination for 3 yr, caused by the regurgitated acid food being pressed against these tooth surfaces.

*Fig. 7.* Pronounced loss of dental hard tissue on the occlusal surfaces of mandibular premolars and molars in a 32-yr-old ruminating patient who rechewed the regurgitated gastric contents before reswallowing them.

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