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Dental erosion. Definition, classification and links

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An overview of tooth wear, i.e. of non-carious destructive processes affecting the teeth including abrasion, demastication, attrition, abfraction, resorption and erosion is presented. The nomenclature and classification of dental erosion commonly used in the dental literature are summarized. They are based on etiology (extrinsic, intrinsic, idiopathic), on clinical severity (Classes I to III), on pathogenetic activity (manifest, latent) or on localization (perimolysis). Interactions between erosion and abrasion, demastication, attrition, and abfraction as well as caries and low salivary flow rate are highlighted.

Key words: abfraction; abrasion; attrition; demastication; dental erosion; non-carious lesions; resorption

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Non-carious destructive processes involved in tooth wear

Different forms of chronic destructive processes other than caries affecting the teeth and leading to an irreversible loss of tooth structure from the external surface are described in the literature. They are referred to as abrasion, demastication, attrition, abfraction, resorption and erosion. These processes can be viewed as physiological and/or pathological, but the literature does not give any clear definitions when to appropriately use which term. Non-carious loss of dental hard tissue, also generically termed tooth wear, is hardly ever caused by one of the above processes alone. According to current knowledge, tooth wear in any one individual is likely to be multifactorial.

Abrasion, derived from the Latin verb *abradere, abrasi, abrasum* (to scrape off), describes the wearing away of a substance or structure through mechanical processes, such as grinding, rubbing or scraping. The clinical term dental abrasion or *abrasio dentium* is used to describe the pathological wearing away of dental hard tissue through abnormal mechanical processes involving foreign objects or substances repeatedly introduced in the mouth and contacting the teeth. Depending on the etiology, the pattern of wear can be diffuse or localized. Based on the clinical observation of the frequent coincidence of smooth-surface and/or cervical abrasion and extensive oral hygiene, the latter has been incriminated to be a main etiological factor in dental abrasion (1). Both patient factors and material factors have been found to influence the prevalence of abrasion. Patient factors include brushing technique, frequency of brushing, time spent on brushing, force applied during brushing, where on the dental arch brushing is started, etc. Material factors refer to type of material, stiffness and end-rounding of toothbrush bristles, tuft-design of the brush, flexibility and length of the toothbrush grip, as well as abrasiveness, pH and amount of dentifrice used. Abrasion on proximal tooth surfaces may be caused by extensive use of interdental cleaning devices such as tooth picks or interdental brushes, especially when they are inserted along with toothpaste or toothpowder. Occupational abrasion, i.e. excessive tooth wear due to any professional cause such as abrasive dust at the work place, holding nails between the teeth, biting thread, etc. are only rarely seen today.

Demastication, derived from the Latin verb *mandere, mandi, mansum* (to chew), describes the wearing away of tooth substance during the mastication of food with the bolus intervening between opposing teeth. Wear is then influenced by the abrasiveness of the individual food. Demastication is normally a physiological process affecting primarily the occlusal and incisal surfaces, but may be termed pathological when occurring due to abnormal food consumption such as betel nut demastication or the like. Although the term demastication is used in the dental literature, this type of wear can also be looked at as a combination of abrasion and attrition.

The term *Attrition* is derived from the Latin verb

atterere, attrivi, attritum, describing the action of rubbing against something. The clinical term dental attrition or *attritio dentium* is used to describe the physiological wearing away of dental hard tissue as a result of tooth-to-tooth contact with no foreign substance intervening. Such contact occurs when grinding the teeth, e.g. during swallowing, speech, and when lifting heavy things, and the resulting wear involves the occlusal and incisal surfaces of teeth. Proximal surfaces are also worn by attrition during mastication. The individual degree of attrition is chiefly associated with age. Clinically, this loss of substance mostly leads to the formation of facets.

The term *Abfraction*, derived from the Latin verb *frangere, fregi, fractum* (to break), is used to describe a special form of wedge-shaped defect at the cementoenamel junction (CEJ) of a tooth (2). Such lesions observed on a single tooth or on non-adjacent teeth are hypothesized to be the result of eccentrically applied occlusal forces leading to tooth flexure rather than to be the result of abrasion alone. According to the tooth flexure theory, masticatory or parafunctional forces in areas of hyper- or malocclusion may expose one or several teeth to strong tensile, compressive or shearing stress. These forces are focussed on the CEJ, where they provoke microfractures in enamel and dentin. The microfractures are thought to propagate with time perpendicular to the long axis of the stressed teeth until enamel and dentin break away. The resulting wedge-shaped defects have sharp rims. As the scientific basis of the tooth flexure theory is not yet sufficiently explored, more research is needed for a better understanding of this process.

Resorption, derived from the Latin verb *resorbere* (to suck back), describes the process of biological degradation and assimilation of substances or structures previously produced by the body. The clinical terms dental resorption, root resorption, tooth resorption and the like describe the biological removal of dental hard tissue by cementoclastic, dentinoclastic and ameloclastic activity. This can be either a physiological process, as in the case of root resorption of deciduous teeth, or a pathological process such as resorption owing to trauma, cysts or neoplasms. Resorption is of no relevance in the context of erosion, but for the sake of completeness cannot be omitted from the list of non-carious destructive processes.

Erosion, derived from the Latin verb *erodere, erosi, erosum* (to gnaw, to corrode), describes the process of gradual destruction of the surface of something, usually by electrolytic or chemical processes. The clinical term dental erosion or *erosio dentium* is used to describe the physical result of a pathologic, chronic, localized, painless loss of dental hard tissue chemically etched away from the tooth surface by acid and/or chelation without bacterial involvement (3–5). The acids responsible for erosion are not products of the intraoral flora; they stem from dietary, occupational or intrinsic sources.

The described forms of non-carious loss of tooth substance are summarized in Table 1.

Nomenclature and classification of erosion

Since the early reports on tooth erosion published in 1892 by DARBY (6), in 1907 by MILLER (7) and in 1923 by PICKERILL (8) many differing nomenclatures and classifications have been used in the dental literature, describing erosive lesions of the teeth. The variety came about because different authors chose different approaches. Thus, nomenclature and classification were based on 1) etiology, 2) clinical severity, 3) activity of progression and 4) localization of erosion.

Classification based on etiology

Tooth erosion is termed either *extrinsic, intrinsic* or *idiopathic*, implying that according to the anamnesis (case history taking) the acids producing tooth destruction may be of exogenous, endogenous or unknown origin.

Extrinsic erosion is the result of exogenous acids. These can be airborne acidic contaminates of the working environment, sometimes referred to as industrial acids (9–12), or acidic water of swimming pools, a side effect of chlorination using chlorine gas that reacts with water to form hydrochloric acid (13, 14). Severe cases of extrinsic erosion have also been reported owing to the oral administration of medicaments such as iron tonics, acid replacements for patients suffering from achlorhydria, or acid to dissolve small renal calculi (15–

Table 1

Survey of non-carious chronic destructive processes leading to tooth wear, i.e. a loss of tooth substance from the external surface.

Terminology	Cause of loss of hard tissue
Abrasion	Mechanical process involving foreign objects or substances
Demastication	Mechanical interaction between food and teeth
Attrition	Mechanical process involving tooth-to-tooth contact
Abfraction	Mechanical process involving tooth flexure by eccentric occlusal forces
Erosion	Chemical etching and dissolution
Resorption	Biological degradation

19). Dietary acids, however, undoubtedly are the principal causative factor for extrinsic tooth erosion (20–23). The most frequently consumed erosive acids are fruit acids and phosphoric acid contained in fresh fruits, fruit juices and soft drinks (24–29). More recently ascorbic acid (vitamin C) contained in all sorts of drinks, sports drinks and candies has been identified as a significant cause of extrinsic erosion (20, 30–36).

Intrinsic erosion is the result of endogenous acid. This is gastric acid contacting the teeth during recurrent vomiting, regurgitation or reflux. Eating disorders of psychosomatic origin, such as nervous vomiting, *anorexia nervosa* or *bulimia* (37–42) are often the cause of regurgitation or vomiting which in these cases is self-induced. Causes of somatic origin include pregnancy, alcoholism (43), antabus therapy for alcohol abuse (44) and gastrointestinal disorders, such as gastric dysfunction (45), chronic obstipation (46), hiatus hernia (47), duodenal and peptic ulcer (48) and gastroesophageal reflux disease (49).

Idiopathic erosion is the result of acids of unknown origin, i.e. an erosion-like pathology where neither tests nor anamnesis are capable of providing an etiologic explanation.

Classification based on clinical severity

Different classifications of tooth wear have been proposed, based on visual examination of the tooth surfaces and on ascribing scores to them according to the extent of the lesions. Few indices, however, have explicitly aimed to assess erosion.

In 1979 ECCLES (50) classified erosion as follows:

Class I: Superficial lesion, involving enamel only;

Class II: Localized lesion, <1/3 of surface involving dentin;

Class III: Generalized lesion, >1/3 of surface involving dentin.

Based on these three classes, LUSSI and co-workers (51) have recently published a similar, more detailed index of erosion for epidemiologic use.

Classification based on pathogenetic activity

MANNERBERG (52, 53) distinguished two types of erosion according to activity, namely manifest and latent erosion. A manifest erosion, i.e. an actively progressing erosion, is clinically diagnosed by its enamel border zones. These are thin where they meet the exposed dentin. In the scanning electron microscope (SEM), they show a honeycomb enamel prism pattern, resembling that seen in acid-etched enamel. Latent or inactive erosions which, through a change in the etiologic factor, are no longer subject to further decalcification, have prominent thick enamel borders and do not show a honeycomb enamel prism structure in shadowed replicas observed in the SEM (20).

Terminology based on localization

Chronic regurgitation, be it of somatic or of psychosomatic origin, often leads to a typical distribution of erosion within the dental arches and on the teeth. This clinical finding has been termed *perimolysis* or *perimylolysis* (44, 45, 54). Corresponding with the path of the regurgitated hydrochloric gastric acid over the dorsum of the tongue, along the palatal surfaces of the maxillary teeth, and over the occlusal surfaces into the mandibular vestibulum, perimolysis affects the maxillary and mandibular teeth in different ways. While affecting the palatal and occlusal surfaces of all teeth in the maxilla, the erosion is confined to the buccal and occlusal surfaces of premolars and molars only in the mandible. The buccal surfaces of the maxillary dentition are not contacted by the acid and are further protected by the neutralizing effect of the parotid saliva. The lingual surfaces of the mandibular teeth are covered by the tongue and are thus also spared from the acid. Additionally they are bathed in the pooled oral fluid from the submandibular and sublingual glands. A further feature of perimolysis is that restorations remain intact and project above the tooth surfaces. A recent study on risk factors in dental erosion (21) was, however, rather guarded about any distinct intraoral distribution of erosion in relation to etiology.

The nomenclature and classification of erosion are summarized in Table 2.

Multifactorial etiology of tooth wear

Abrasion, demastication, attrition and abfraction

The surface of erosive lesions is hypomineralized. Although enamel softening is not clinically detectable, erosion decreases the wear-resistance of dental hard tissue (55), thus rendering both enamel and dentin more susceptible to the effects of mechanical abrasion (56). As a consequence, erosion is *in vivo* frequently exacerbated by mechanical abrasion, such as toothbrushing immediately following an acid challenge. Erosive lesions may also be aggravated by demastication (57), especially in lactovegetarian patients (58), by attrition of incisal edges and cusps or possibly by abfraction at the cementoenamel junction. A clinical *ex-post facto* differential diagnosis between chemical and mechanical etiology is consequently often difficult, as the pathogenesis of any individual case may well be multifactorial.

Table 2

Survey of nomenclature and classification of erosion used in the literature.

Basis	Nomenclature or Classification	Etiology	Clinical finding
Source of acid	extrinsic	diet, medication, Swimming pool, industrial	Class I to III, predominantly labial and occlusal
	intrinsic	eating disorders, gastrointestinal disorders, pregnancy, alcoholism	Class I to III, predominantly perimolysis
	idiopathic	unknown	Class I to III
Clinical severity	Class I	all possible	enamel only
	Class II	all possible	<1/3 of lesion in dentin
	Class III	all possible	>1/3 of lesion in dentin
Pathogenetic activity	manifest	all possible	actively progressing; thin enamel border zones, loss of lustre
	latent	all possible	inactive, stopped; thick enamel borders, lustre
Localization	perimolysis/perimylolysis	intrinsic	predominantly palatal and occlusal surfaces in upper arch, buccal and occlusal surfaces of lower premolars and molars

Caries

Another link of erosion is the association with caries, more frequently observed since the increased usage of acid- and sugar-containing sport drinks during exercise when, due to mouthbreathing, there is less salivary clearance of acids and carbohydrates. In this case, erosions advancing along with poor oral hygiene are often characterized by the presence of chalky spots and streak-shaped marks in the thin enamel surrounding the lesions. The chalky spots are a result of carious decalcification and are not found with good oral hygiene.

Saliva

Erosion has been studied in animals and *in vitro*. For obvious reasons, only clinical observations but no clinical trials have been made in humans. The effect of erosive agents in the human mouth may depend on many factors not yet elucidated, but erosion is certainly in some way influenced by salivary flow rate and buffer capacity as well as by the pellicle formed by salivary mucins. People with low unstimulated salivary flow rates showed a much higher risk for erosion (21), and patients suffering from idiopathic erosion had lower salivary flow rates than the controls (59). Erosion seems to be aggravated by insufficient salivary flow and/or buffer capacity. More research is, however, needed to elucidate the interactions between saliva and erosion.

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