The Etiology and Pathogenesis of Tooth Wear

PART 1

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Historically, the most common reason for tooth loss and dental hard tissue loss has been dental caries. Since the introduction of fluoride, the prevalence, incidence and severity of caries has declined and the dental life expectancy has increased. One of the most common problems associated with this prolonged dental life expectancy is tooth wear. Tooth wear is an irreversible, non carious, destructive process, which results in a functional loss of dental hard tissue. It can manifest as abrasion, attrition, abfraction and erosion.[1] This article will describe the etiology of pathogenesis of tooth wear.

ETIOLOGY

Tooth wear can manifest as abrasion, attrition, abfraction and erosion. The distinct definitions of the patterns of dental wear tend to reinforce the traditional view that these processes occur independently. However, a combination of etiologies probably reflects the true clinical situation.[2] Identification of the etiology of tooth wear is essential for its successful management.

ABRASION

The term abrasion is derived from the Latin verb abradere (to scrape off).[1] It describes the pathological wearing away of dental hard tissue through abnormal mechanical processes involving foreign objects or substances repeatedly introduced in the mouth. Abrasion patterns can be diffuse or localized, depending on the etiology. Extensive oral hygiene has been incriminated as a main etiologic factor in dental abrasion. Both patient and material factors influence the prevalence of abrasion. Patient factors include brushing technique, frequency of brushing, time and force applied while brushing. Material factors refer to type of material, stiffness of toothbrush bristles, abrasiveness, pH and

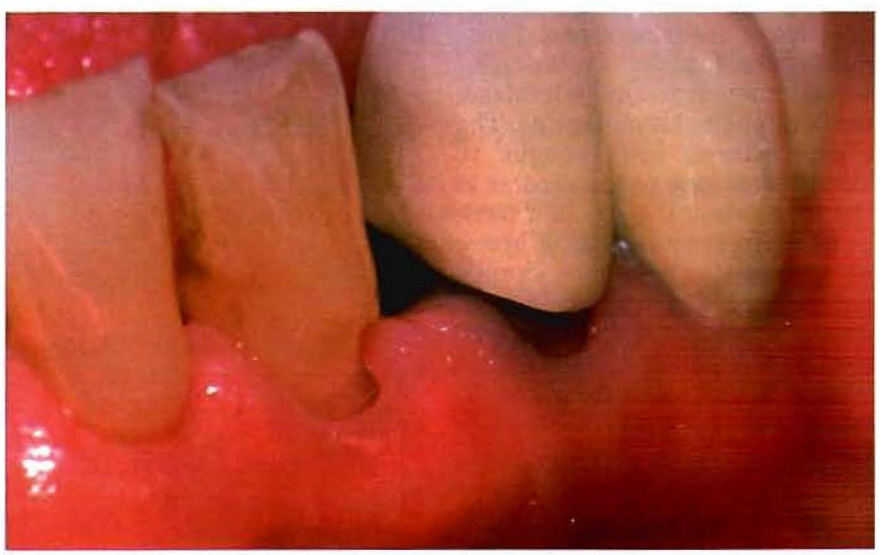

FIGURE 1 Dental abrasion due to horizontal brushing technique

amount of dentifrice used.[3] The most commonly cited effect of abrasion is the V-shaped defect, which usually is ascribed to the use of an intensive horizontal brushing technique (Figure 1). Cervical areas are susceptible to toothbrush abrasion, particularly cuspids and first premolars, where thin buccal plates, gingival recession and exposed root surfaces predispose cervical notching. Habits involving other intraoral objects (e.g., pipe smoking, toothpick use, threadbiting) can cause defects on the incisal and occlusal surfaces.[4] Dietary abrasion is not very prominent in modern days, as the typical western diet tends to be very soft, as opposed to primitive man's diet which was more abrasive, and thus contributed greatly to tooth wear.

ABFRACTION

The term abfraction, derived from the Latin verb frangere (to break), describes a wedge shaped defect at the cementoenamel junction of a tooth.[5] These lesions are sometimes located subgingivally, beyond the influence of toothbrush abrasion and 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 expose one or several teeth to strong tensile, compressive or shearing stress. These forces are focused at the CEJ where they provoke microfractures in enamel and dentine. The microfractures are thought to slowly propagate perpendicular to the long axis of the stressed teeth until enamel and dentine break away resulting in wedge shaped defects with sharp rims. The scientific basis of the tooth flexure theory has not yet sufficiently been explored and it is often difficult to differentiate between abrasion and abfraction lesions.[1]

ATTRITION

Attrition is the term used to describe the physiological wearing away of dental hard tissue as a result of tooth to tooth contact with no foreign substance intervening. It is derived from the Latin verb atterere, which is defined as the action of rubbing against something.[1] Such contact occurs with tooth grinding, such as with parafunction, mastication, swallowing and speech. A typical presentation of attrition is the presence of extensively demarcated facets which usually match the opposing arch facets in excursive contact positions (Fig-

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Table 1
Sources of extrinsic acids

Environmental	Diet	Medicaments
atmospheric sulfuric acids	citrus fruit juices	vitamin C
HCL in gas-chlorinated swimming pools	acidic carbonated beverages	aspirin
	acidic uncarbonated beverages	acidic oral hygiene products
	wines	acidic saliva substitutes
	citrus fruits	

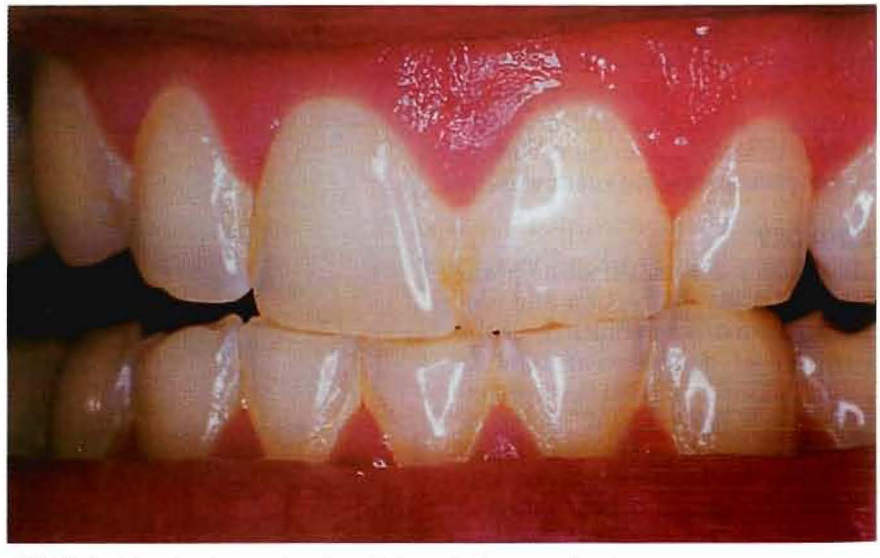


FIGURE 2 Attrition due to parafunction. Note matching wear facets.

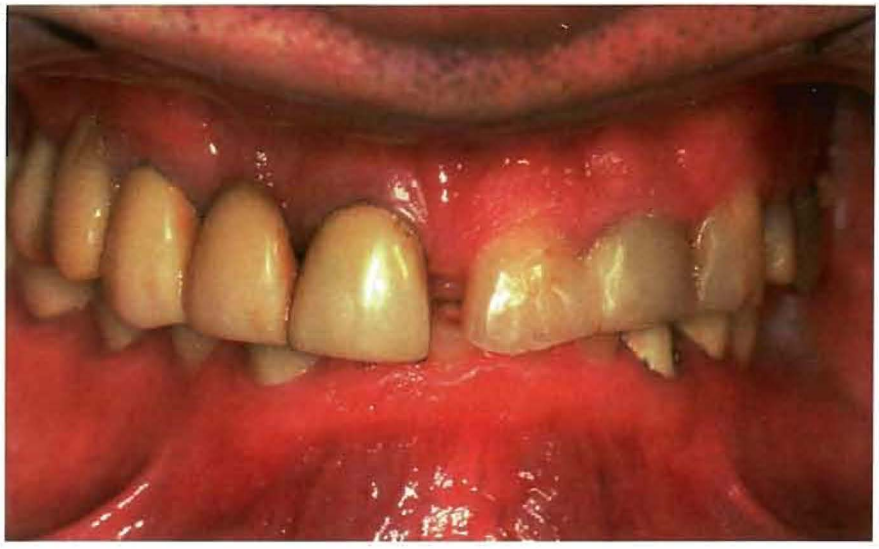


FIGURE 3 Sixty year-old male with advanced tooth wear; **A** deep vertical overbite.

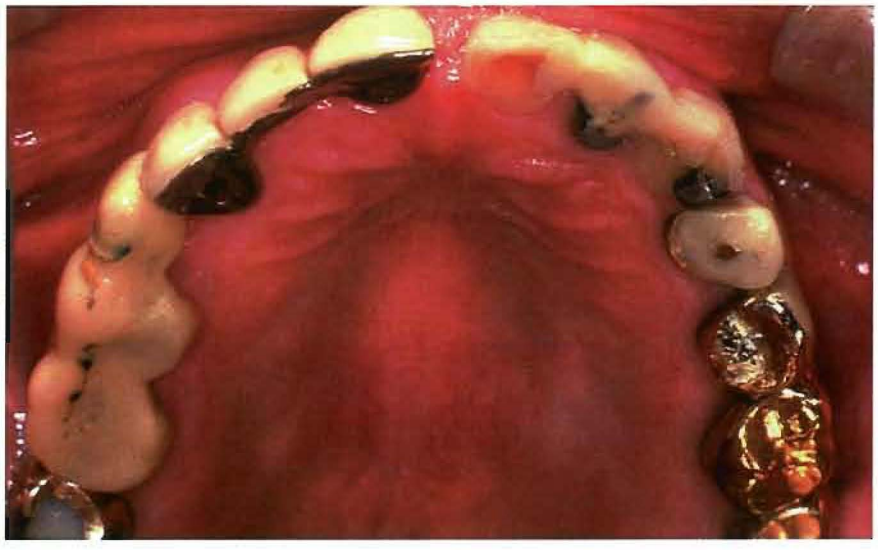


FIGURE 3 **B** wear at palatal of maxillary anteriors.

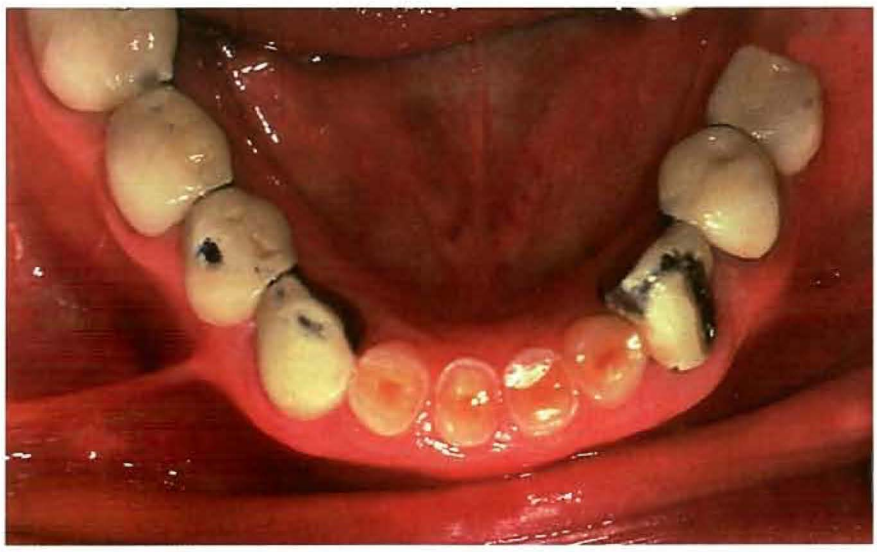


FIGURE 3 **C** wear of mandibular incisors.

ure 2). Attrition occurs almost entirely on occlusal and incisal surfaces, although it may also affect the buccal and palatal surfaces of the maxillary and mandibular anterior teeth in deep vertical overlap relationships[6] (Figures 3A, B, C).

EROSION

The term erosion describes the process of gradual destruction of the surface of something, usually by electrolytic or chemical processes. It is derived from the Latin verb eroder (to corrode).[1] Dental erosion is the result of a pathologic, chronic, painless loss of dental hard tissue chemically etched away from the tooth surface by acid and/or chelation without bacterial involvement.[7] The acids responsible for erosion are not products of the intraoral flora; they stem from extrinsic or intrinsic sources.

Extrinsic sources of acid:

Extrinsic acids may stem from environmental sources, diet and medication (Table 1).[8] Dental erosion has been reported in battery factory workers exposed to atmospheric sulfuric acids.[9] There have also been reports of competitive swimmers suffering dental erosion from swimming in gas-chlorinated pools. Large swimming pools generally use gas chlorination, which results in the formation of hydrochloric acid that requires neutralization and buffering to maintain the recommended pH range of 7.2-8.0. Therefore, inadequate monitoring of pool pH has been associated with dental erosion.[10] Though extrinsic environmental sources of acids exist, improved industrial safety regulations have gradually diminished the extent of environmental dental health hazards.[8]

The role of diet in the etiology of erosion has received the most attention.[11] Certain products, especially citrus fruits, exhibit a low pH, and when consumed frequently and excessively, may lead to dental erosion. It appears that dietary substances with a pH above 4.5 have a low potential to

cause dental erosion. However, foods and beverages containing acids with calcium chelating properties, such as citrate, may cause tooth damage at higher pH levels. Several reports have associated medicaments and oral health products (rinses) with erosion.[12,13,14,15] Many such products exhibit a low pH and may be erosive when used frequently. In most cases, the risk associated with a product could be reduced by either product modification (such as encapsulation of acidic medicaments), or altering consumption habits. Special attention should be given to saliva substitutes aimed at patients with reduced salivary secretion or xerostomia. These substitutes often have a low pH and may be detrimental to patients whose lack of saliva leads to prolonged clearance times. Factors to be considered with exposure to extrinsic sources of acid include: the duration of contact with the teeth (which is influenced by swallowing habits, motions of the lips and cheeks, saliva), frequency of ingestion, amount ingested, buffering capacity of saliva, the chemical and physical properties of enamel.[8]

Intrinsic sources of acid:
Dental erosion due to intrinsic factors is caused by gastric acid reaching the oral cavity and the teeth as a result of vomiting, persistent gastroesophageal reflux, regurgitation or rumination. Since the 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 conditions which are associated with chronic vomiting or persistent gastroesophageal reflux. Examples of such conditions are listed in Tables 2 and 3.

Bulimic eating disorder is the underlying cause in most cases of dental erosion due to chronic vomiting. Recent studies suggest that approximately 90% of bulimic patients are affected by dental erosion.[16] 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 (Table 2).

Another possible etiologic intrinsic factor of dental erosion is persistent gastroesophageal reflux (GOR). GOR is the movement of stomach acids through the lower esophageal sphincter. In healthy individuals, small amounts of gastric acids reflux into the esophagus. This physiological GOR usually occurs after eating and may be associated with eructation. In healthy people, most of the refluxate is returned to the stomach by

Table 2
Potential causes of vomiting[20,21]

Disorders of the alimentary tract:
- chronic gastritis
- peptic ulcer
- intestinal obstruction

Neurologic disorders:
- migraine headaches
- benign recurrent vertigo
- diabetic or alcoholic polyneuropathia

Metabolic or Endocrine disorders:
- uremia
- hyperparathyroidism
- diabetic ketoacidosis
- adrenal insufficiency
- hypo-hyperparathyroidism

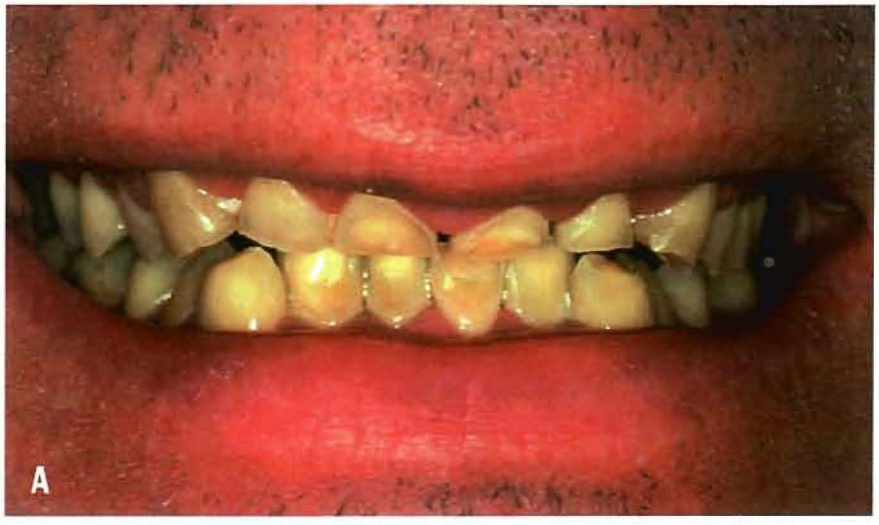
Psychosomatic disorders:
- eating disorders (bulimia, anorexia)
- stress induced psychogenic vomiting

Table 3
Causes of oastroesophaaeal reflux and reaurclitation[20,21,22,23]

Incompetence of the gastroesophageal sphincter.
- Idiopathic
- impairment of sphincter
- neurohumoral induced decrease of gastroesophageal sphincter pressure
- destruction of sphincter by surgical resection

Increased intraabdominal pressure:
- obesity
- pregnancy

Increased Intragastric volume:
- after meals
- pyloric spasm
- obstruction due to peptic ulcer
- gastric stasis syndrome



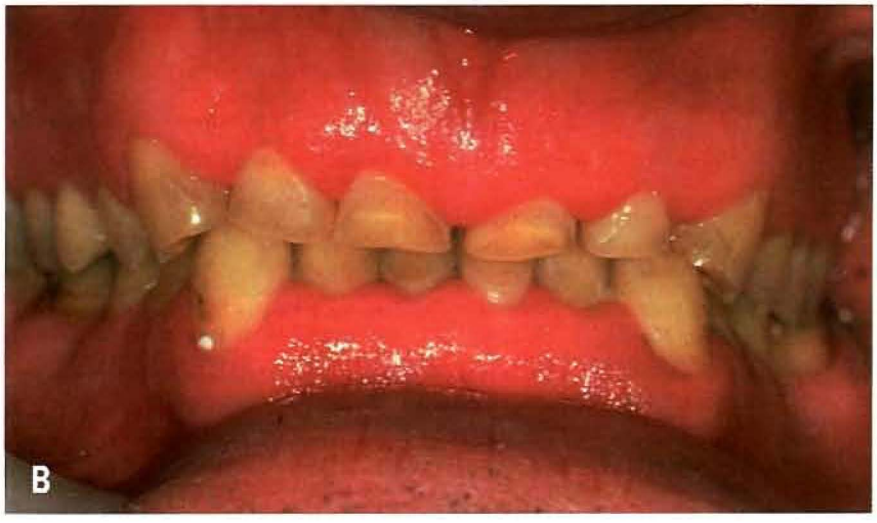


FIGURE 4 Forty six-year-old male with Gastroesophageal reflux; **A & B** Dental erosion of maxillary and mandibular anteriors;

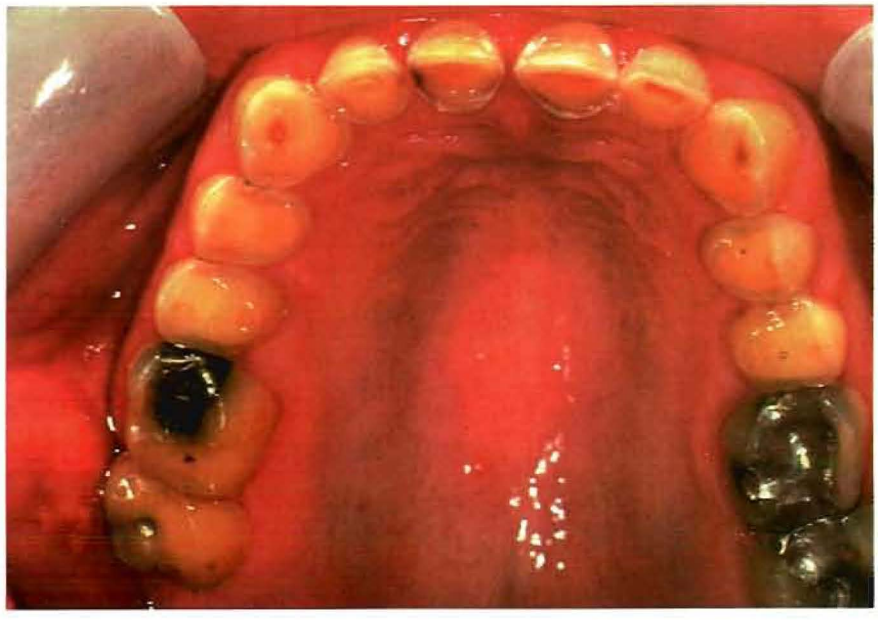


FIGURE 4 C Occlusal view of palatal erosion.

the peristalsis stimulated by swallowing.[17] It is estimated that 60% of the population suffer from this phenomenon at some stage of their lives. If the clearance mechanisms cannot return the refluxate to the stomach and the symptoms become chronic, the condition is known as pathological GOR or GOR disease (GORD). In some patients, the refluxate breaks through the lower and upper esophageal sphincter and oral regurgitation occurs. Oral regurgitation may cause severe damage to the dentition[18] (Figures 4A, B). Causes of gastroesophageal reflux and regurgitation are listed in Table 3. Often the erosion is most severe on palatal tooth surfaces, but other surfaces may also be affected when the gastric contents are chewed or kept in the buccal sulci before reswallowing.

PATHOGENESIS

Since the critical pH of dental enamel is approximately 5.5, any solution with a lower pH value may cause erosion, particularly if the attack is of long duration and repeated over time. Saliva and the salivary pellicle counteract the acid attacks, but if the challenge is severe, a total destruction of the tooth tissue follows. Erosive lesions are seen as characteristic demineralization patterns within the enamel. In dentine, the first area to be affected is the peritubular dentine. With progressing lesions, the dentinal tubules become enlarged but disruption is also seen in the intertubular areas. If the erosion process is rapid, increased sensitivity of the teeth is the presenting symptom. However, in cases with slower progression, the patient may remain asymptomatic even though the whole dentition may become severely damaged.[19]

CONCLUSION

The interrelationship of the four modes of tooth wear and individual susceptibility influence the degree of tooth wear. Recognition of the multifactorial nature of tooth wear is the first step in man-

agement, as failure to appreciate this may lead to inappropriate management and ultimate failure of restorative therapy. The second part of this publication will discuss the management of tooth wear. Treatment planning strategies, as well as case presentations will be presented.

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Oral Health welcomes this original article.

Part II will appear in our November 1999 issue of Oral Health.

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