A Difference in Perspective— The North American and European Interpretations of Tooth Wear

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Purpose: There is considerable interest in the European dental research literature about the problem of tooth wear and specifically about dental erosion, but this interest does not appear to be matched in North America based on the volume of the literature there. The purpose of this article is to consider the possible explanations for this difference. Materials and Methods: This article examines the reasons for this disparity and attempts to explain the difference by reviewing the North American and European literature on the etiology, pathogenesis, and prevalence of tooth wear. Results: It would appear from the literature that the reason for the difference in interest between the 2 continents is a reflection of how the appearance, etiology, and terminology are interpreted and used to define tooth wear, attrition, and erosion. Conclusion: Attrition is the wear of teeth against teeth; therefore, by definition any worn surface that does not contact the opposing tooth must have another etiology. An appropriate descriptive term is "tooth wear" when the etiology is multifactorial or cannot be determined. A search of the literature shows more studies in the European literature of the etiology and prevalence of tooth wear than in the North American literature. The thrust of the European studies supports the view that erosion is more important than attrition in the etiology of tooth wear. Int J Prosthodont 1999;12:401-408.

Min the European literature has recently been directed to erosion caused by dietary or gastric acids. On the other hand, research from North America has appeared to concentrate on attrition as the predominant factor, with little acknowledgment of the role of acid erosion. This difference will be explored by reviewing the etiology, prevalence, and appearance of erosion, attrition, and abrasion.

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Definition

"Tooth wear" is an all-embracing term used to describe the combined processes of erosion, attrition, and abrasion, or when the specific diagnosis cannot be determined.1 Erosion is defined as the chemical dissolution of teeth by acids other than those produced by bacteria, attrition is the wear of tooth against tooth, and abrasion is the wear of teeth by physical means other than opposing teeth.² The term tooth wear can be used as a generic description until a more specific diagnosis can be made; this is comparable to making the observation that a patient is pyretic and then moving on to a diagnosis of pneumonia and then investigating the cause of pneumonia. Some tooth wear continues as a slow process throughout life and is normal, but in some individuals the rate increases to such an extent that the longevity of the teeth is compromised. The term "pathologic tooth wear" has been used to describe the state when the destruction

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of the teeth has reached a level at which restorations are indicated.³

Erosion

There are 3 main sources of acid erosion of teeth: dietary, gastric, and industrial acids.

Dietary Erosion

Darby⁴ reported the role of acids in the diet as early as 1892 when he described fruit juices dissolving teeth. Since that time many in vitro studies have investigated the acidity of food and drinks and their ability to erode enamel and dentin,^{5–8} but the evidence from clinical studies remains poor.

Citric acid is found in citrus fruits and drinks and as an additive in carbonated drinks. ^{9,10} It is one of the most potent erosive agents found in foods because of its ability to chelate calcium in hydroxyapatite, increasing the rate of dissolution. This ability to chelate hydroxyapatite is believed to continue even after the pH increases at the tooth surface, thereby prolonging the erosion. ⁶ Erosion caused by citric acid in the form of vitamin C tablets and drinks has also been reported. ^{11,12}

Other important dietary organic acids are commonly found in fruits such as pineapples, grapefruits, apples, cranberries, and black currants. Other dietary additives have also been implicated in erosion; these include phosphoric acid (found in some drinks), carbonic acid (found in all carbonated drinks), and others. The pH of some of these products falls below 4.0, which is recognized from in vitro studies as the approximate threshold for erosion to occur. 7,13 The carbonation of the drink is probably less important than its pH because carbonic acid is weak.14 Carbonated water, therefore, is probably not particularly erosive, whereas carbonated lemon or cranberry juice would be more so. A popular drink in the United States is iced tea, which is normally served with a piece of lemon. While the tea itself would not be acidic, the impact of the lemon—especially if the fruit is sucked or chewed—would have a greater potential for erosion.

Frequent consumption and the type of acidic food or drinks will to some extent depend on where a person lives. Hotter climates necessitate a higher frequency and volume of fluid consumption and this may take the form of fruit juices or carbonated drinks. However, this ignores the influence of fashion and peer pressure on young people, which is associated with the consumption of carbonated drinks. Not only is the type of acidic food or drink important, but the way it is consumed may have an

impact on the teeth.¹⁴ Holding or "swilling" carbonated drinks within the palatal vault may cause significant erosion, especially on the palatal/lingual surfaces of the maxillary anterior teeth, although erosion in this area is usually associated with acid originating in the stomach.^{14,17}

There has been considerable interest in the link between dietary acids and dental erosion, but the evidence from studies that have compared the severity of erosion with the consumption of acidic food and drinks is not conclusive. Most research on the role of dietary acids in erosion has been based in the laboratory. 18,19 However, care should be taken when applying in vitro experimental methods to clinical problems. It appears from in vitro reports that dietary acids have the capacity to cause erosion of dentin and enamel, 7,18,19 but there are few published clinical trials on the effect of acidic drinks on teeth. A recent controlled clinical study by West et al²⁰ compared the amount of erosion caused by orange juice to water in volunteers from the staff of a dental hospital. Not surprisingly, the orange juice was significantly more erosive than water. Thomas²¹ also investigated the effect of dietary acids on volunteers and noted erosion in most subjects. Interestingly, some of the subjects who consumed high quantities of acids appeared to have marked erosion after 6 weeks while others with a similar diet did not. Thomas²¹ suggested that other factors such as drinking habit, the buffering capacity of the saliva, and the physical state of the teeth might have caused the difference in the degree of erosion observed in the subjects. Neither study used subjects with preexisting erosion, who might have been more susceptible to acids in the diet.

There is some epidemiologic evidence to support the hypothesis that diet is implicated in erosion.²² Millward et al,23 in an investigation of 100 schoolchildren, identified significant correlations between the reported consumption of acidic food and drinks and the prevalence of erosion. However, in a study of 210 schoolchildren in southeastern London, Bartlett et al 24 did not report statistical correlations between the prevalence of erosion and the reported consumption of acidic food and drinks. The data for this study were collected during the summer and the correlations reported were in spite of the fact that the mean consumption of carbonated drinks was 3 to 4 cans per day. A problem with all of these studies is that data on diets relies on questionnaires, which can be notoriously inaccurate, especially in children.

The difference in appearance between eroded and noneroded enamel surfaces can be difficult to detect, especially in the early stages of erosion. Most prevalence studies on erosion have reported significant levels of enamel erosion and lower levels of more severe

erosion involving dentin.^{23–25} However, most of the statistical correlations related to the diet are based on minor enamel changes and these changes are especially difficult to differentiate from the appearance of uneroded tooth surfaces. Conversely, dentin exposure is much easier to diagnose but there is little evidence from the literature that erosion of dentin is correlated to the diet. Therefore, while it may be reasonable to conclude that erosion is sometimes caused by acidic food and drinks, the association with severe erosion has not been readily established.

Erosion Caused by Gastric Juice

Gastric juice has a pH of around 1 and if it enters the oral cavity regularly the damage to teeth can be catastrophic. One of the most powerful effects of gastric acid has been shown on the teeth of patients suffering from the eating disorders of anorexia and bulimia nervosa. Typically, affected patients may induce vomiting to purge themselves of food or drink. The distinction between anorexia and bulimia can be unclear as some patients pass through episodes characteristic of both diseases. Frequent vomiting during pregnancy can have a similar effect.

The most important constituent of stomach juice is hydrochloric acid formed in the parietal cells lining the stomach walls and secreted in response to food. The erosive potential of gastric acid is related to the time of consumption of food. Recently swallowed food enters a highly acidic environment necessary for protein digestion. After some time the ingested food buffers the hydrochloric acid, reducing the erosive potential of the gastric contents. ²⁷ If vomiting or regurgitation occurs soon after consuming food the gastric juice will have its greatest erosive potential, but if food is vomited later the gastric juice becomes less erosive. Acidic food and drinks are known to provoke reflux and therefore have a greater potential to cause regurgitation erosion. ²⁸

Regurgitation is an involuntary phenomenon whereby gastric acid passes into the mouth without conscious control; unlike vomiting, it is not coordinated by the autonomic nervous system.²⁹ The regurgitation of gastric acid into the mouth is not normal and is associated with the condition known as gastroesophageal reflux (GER).²⁹ Heartburn is the most common symptom of GER and is caused by gastric acid that has leaked from the stomach, irritating the mucosal lining of the distal esophagus. In most people any leakage of gastric acid into the esophagus is temporary and the acid is quickly returned to the stomach by peristalsis. In others the acid remains for longer periods and symptoms become chronic and prolonged and patients require treatment. Other

evidence that regurgitated gastric juice causes palatal erosion has been derived from research in chronic alcoholics and ruminants, in whom pathologic GER may also be involved.^{30,31}

Dental erosion has been identified in patients participating in upper gastrointestinal investigations for GER,³² and GER has also been diagnosed in patients presenting with dental erosion. 33,34 It appears that the 2 conditions are related and that regurgitation caused by GER is responsible for erosion in some patients. There is also evidence that regurgitation erosion causes more extensive damage than dietary erosion.¹ However, a history of heartburn and other symptoms may not always indicate the presence of GER. In an investigation of 36 patients presenting with dental erosion 26 were diagnosed with GER using criteria developed by gastroenterologists.²⁹ Interestingly, 9 of these patients did not have symptoms of GER; they are called "silent refluxers." Therefore, the presence of dental erosion in these patients appeared to be a better indicator of GER than the disclosure of symptoms. Gastroesophageal reflux is an important cause of dental erosion but is difficult to diagnose in some patients because of the lack of symptoms. Since the diagnosis of GER requires gastroenterologic investigations and the influence of dietary acids requires only a questionnaire, the latter tends to receive more blame.

Industrial Erosion

The work environment has been reported to cause dental erosion. Historically, car battery workers, those exposed to industrial electrolytic processes, and others working with acids that are present as vapor in the air have suffered from dental erosion.³⁵ Exposure of the teeth to acid continuously through the contaminated air produced erosive lesions similar in pattern to those caused by dietary acids. Improvements in occupational health and safety have made this type of erosion rare.³⁶

Attrition

The constant rubbing of teeth against one another during routine function and during parafunction must be regarded as normal and the resulting wear as part of the aging process.³⁷ The amount of contact between teeth that normally occurs while eating a modern diet is probably insufficient to cause significant attrition even over a lifetime. However, frequent clenching or grinding of teeth for purposes other than mastication (bruxism) may produce flattened cusps.^{38,39} Bruxism can occur during the waking hours or during sleep and, although common, may be regarded as abnormal if it damages teeth or produces symptoms.¹⁵

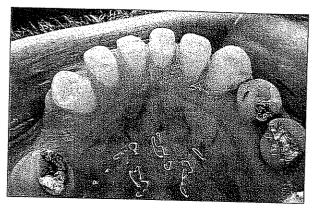


Fig 1 Wear of anterior teeth caused by a combination of erosion and attrition. The "cupped out" incisal surface of the canine could not have been caused solely by attrition as the center of the worn area cannot contact the opposing tooth.

Attrition produces wear facets on the occluding surfaces of teeth, including the incisal edges. 40 This commonly begins soon after eruption or may start later in life. Eventually the cusps become flattened, the incisal edges are shortened, and dentin is exposed. Once dentin is exposed the wear resistance of the tooth is reduced and rate of wear is increased. 10 The distinction between damage caused by bruxism and by the normal aging process can be difficult to determine, especially in middle to late life, except in extreme cases.

The cause of bruxism is unknown, but Ramfjord and Ash⁴¹ described 2 possibilities. They suggested that the act of bruxism in some way relieves stress, eliciting some gratification; this could occur while asleep or awake. Alternatively, the process could be more mechanical. An occlusal interference may trigger a bruxing habit, producing attrition. To test this hypothesis Ramfjord⁴² experimentally provoked bruxism in 10 Rhesus monkeys by placing high occlusal amalgam restorations in their mandibular molars. The bruxing began immediately and continued until the amalgam was no longer high. The author considered that the cause of the bruxism was a subconscious effort by the monkeys to remove the high spots. Therefore, in the author's opinion the occlusal interference triggered the bruxism.

However, not all researchers agree with this concept. Rugh et al⁴³ placed crowns with deflective contacts in 10 human subjects and reported that the interferences did not stimulate bruxism. The evidence based on these studies remains unclear as the reported studies used low numbers of subjects. There is some evidence to suggest that bruxism is a condition related to daytime stress or the anticipation of stress. However, a clear relationship between the 2 has yet to be proved. Clinically, occlusal interferences are

identified in patients with bruxism, but it is often difficult to determine whether the interference caused the bruxism or whether the interference was produced as a result of the bruxism. Whichever is true, bruxism can cause considerable damage to teeth that may be made worse if teeth are restored with abrasive materials such as unglazed porcelain.

However, a close inspection of worn incisal edges often reveals areas of exposed dentin where the opposing teeth do not contact in any excursions (Fig 1). This is common, especially in older patients, and cannot be entirely caused by attrition. Therefore, there must be some other explanation (this will be discussed later). A useful diagnostic observation is whether the maxillary and mandibular occluding surfaces are wearing at the same rate. If they are, this suggests attrition; if not, something else—usually erosion—must be accelerating the wear in one arch more than the other.

Abrasion

Abrasion is physical wear caused by materials other than teeth. Abrasion has been assumed to be associated with over-zealous toothbrushing, especially along the cervical margins of the canines and premolars. Toothbrushing may or may not be the main factor as these lesions also appear on the lingual surfaces of molars and on lingually displaced teeth, where access by a brush is difficult. There is considerable debate but little scientific evidence on the etiology of V- and saucer-shaped cervical wear lesions and the less common vertical defects on the facial surfaces of posterior teeth.

Svinnseth et al⁴⁵ and Redmalm⁴⁶ believe that the mechanical action during toothbrushing combined with the abrasive in toothpaste causes abrasion. Lewis and Smith⁴⁷ argue that the process is more likely to be multifactorial, with erosion as the major influence. Acids present in the diet or originating from the stomach could weaken the tooth, making it more susceptible to abrasion.⁴⁷

Abfraction is another popular concept. The wear is thought to originate from occlusal loads acting on teeth to produce minute stress concentrations around the cervical margin. Continual loading of the tooth will produce stress fractures called abfractions, which make the area more susceptible to erosion and abrasion. Abfraction remains a controversial theory with little evidence to support its existence. A combination of factors seems the most likely explanation of these lesions.

The texture of food is thought to have caused abrasion in the past, although the abrasive influence of the modern Western diet is unlikely to be a significant

factor. Bread containing grit from the millstone is thought to have produced abrasion/attrition. Today, in certain parts of the world, for example the Aborigines in Australia, people continue to have a high rate of tooth wear and abrasion; food is thought to be the cause. ^{40,49}

Relative Importance of Erosion, Attrition, and Abrasion in Etiology of Wear

In 1984 Smith and Knight¹ reported that 35 of 100 consecutive patients with tooth wear referred to a dental hospital for diagnosis and treatment planning suffered from erosion, while only 11 suffered from attrition. Jarvinen et al⁵⁰ reported similar findings in 1989 in a study on Scandinavian patients. Davis and Winter¹⁰ reported that a combination of erosion and attrition increased the rate of tooth wear to a greater degree than if either factor was operating independently. Dental erosion in Europe is acknowledged to be the most common cause of tooth wear, either independently as in eating disorders, or in combination with attrition or abrasion. The appearance of "cupped out" dentin on the incisal edges of anterior teeth or on the cusps of posterior teeth that do not contact the opposing teeth suggests a combination of erosion and attrition.

Dietary acids probably cause some erosion, especially in the young.^{23,25} The influence of the diet is probably more important in the early stages of erosion than in more severe cases. This association is supported by the common use of acidic food and drinks in the Western diet.^{6,23} Dietary acids are less likely to cause severe erosion; regurgitated gastric acid—with its lower pH and established association with eating disorders—GER, and other conditions are more likely to be the main etiologic factor.^{24,51}

There have been few studies to investigate the effect of other factors on the etiology of tooth wear. Gudmundsson et al⁵² suggested that salivary buffering capacity might be important in dental erosion. Further support for the role of saliva in erosion was reported by Milosevic and Dawson⁵³ when they compared flow rate and bicarbonate concentration in 9 bulimics. Interestingly, a few years earlier one of the same authors reported no association between saliva and patients with dental erosion and an eating disorder.54 Some researchers have postulated that the abrasive surface of the tongue lying against the palatal surfaces of the maxillary incisors may contribute to erosion.²⁹ Milosevic and Dawson⁵³ investigated the susceptibility of different tooth surfaces to erosion with an enamel biopsy technique. The results suggested that tooth surfaces have differing susceptibilities to erosion. Surprisingly, the enamel of the group with unworn teeth released higher concentrations of calcium than the worn teeth. It is possible, therefore, that factors other than acids or attrition may be important in tooth wear, but as of yet research is not available to identify the effects of these factors.

Importance of Site and Pattern of Wear

The site of tooth wear can be used to some extent to suggest the source of the acid. Typically, but not always, acid originating from the stomach strikes the palatal surfaces of the maxillary incisors, eroding enamel and in due course dentin. Possibly, in the early stages of the process the tongue protects the other surfaces of the teeth. As the erosive action persists the protective mechanisms of the mouth are overwhelmed and a more generalized pattern of erosion emerges, commonly affecting the occlusal surfaces of the mandibular molars, followed by the maxillary occlusal and posterior palatal surfaces.²⁹ The lingual surfaces of the mandibular teeth are seldom affected.

It has been suggested that sipping an acidic beverage will erode the buccal or facial surfaces of the maxillary incisors, and after prolonged use a more generalized pattern of erosion develops.¹³ However, palatal erosion may also result if the beverage is held in the palatal vault or "swilled" around the mouth prior to swallowing.¹⁷ Therefore, although the pattern of erosion may be useful in identifying the source of acid it is by no means definitive.

Early enamel erosion is seen as smoothing of the minor enamel irregularities to produce a shiny, unstained surface. Once the dentin is exposed it erodes more quickly because it is less mineralized. As the dentin is exposed the tooth color changes from the creamy white of enamel to the vellower color of dentin. In extreme cases erosion will result in the teeth becoming short and/or thin, and they may become level with the gingival margin. Ultimately the form of the tooth is lost and the function of the teeth impaired. Hypersensitivity to temperature changes may be a problem with rapidly progressing erosion as the dentinal tubules are exposed to the oral cavity. However, if the wear is slow enough the pulp responds by forming secondary dentin and sensitivity is not a common problem even in severe cases, particularly in older patients.

Prevalence of Tooth Wear

Tooth wear is acknowledged to be an almost universal condition and part of the aging process. In a study of 1,007 adult patients attending dental practices in southeastern England, Smith and Robb⁵⁵

reported that almost 98% had evidence of some tooth wear but only 3% to 5% of tooth surfaces in the younger age group had levels of wear that might necessitate treatment. However, this rose to 8% to 9% in patients over age 56.55 Robb et al,56 in a controlled study on the prevalence of tooth wear in 151 Roman and Saxon skulls in Britain, reported that the condition is not a modern phenomenon; 30 skulls (nearly 20%) showed extensive tooth wear. Other European research by Jarvinen et al57 reported that erosion was present on all teeth of 106 patients referred to a dental hospital. A better indication of the prevalence of tooth wear was reported by Linkosalo and Markkanen⁵⁸ in a random sample of Swiss adult patients. The amount of tooth wear varied according to site and age, mostly occurring on the facial and occlusal surfaces, while the least commonly affected surface was the palatal surface of the maxillary incisors. The largest published survey on the prevalence of erosion was on 10,000 extracted teeth from southern California and showed that 18% of the teeth had evidence of erosion.59

No clinical epidemiologic studies on dental erosion were found in the North American literature, but studies of the prevalence of erosion have been conducted on children in Europe. In 1992 the UK National Child Dental Health Survey of 17,000 children reported the presence of erosion in 5 to 15 year olds. 25 The results showed that over half of the 5 and 6 year olds had evidence of erosion in the deciduous dentition and in nearly a quarter of these dentin was involved. Palatal erosion was the most commonly affected site with 52% of incisors affected, while only 18% presented with buccal/facial erosion. In the permanent dentition dentin exposure was found in 2% of affected 13 to 15 year olds. Milosevic et al,60 reporting on 1,035 14-year-old children, observed that 30% had exposed dentin, mainly on the incisal surfaces. Millward et al²³ and Bartlett et al,²⁴ in studies of 178 and 210 children, respectively, observed levels of erosion similar to those found in the UK National Child Dental Health Survey. As in the larger study the most commonly affected surface was the palatal surface of the maxillary incisors.

There is evidence in the literature to support these findings from North America, but unlike the European studies the incidence of attrition is reported. Seligman et al¹⁵ reported the severity of attrition on the occlusal surfaces of study casts in 222 young adults by studying the appearance of the casts. Like Smith and Robb, ⁵⁵ Seligman et al observed that more than 90% of the subjects had evidence of wear, but a direct comparison between the 2 studies is impossible because each used a different method of assessment. Silness et al⁶¹ and Pintado et al⁶² observed the

progression of tooth wear using indices measuring occlusal wear.

Criteria for Assessing Prevalence of Tooth Wear

One of the problems in attempting to record the prevalence of tooth wear is how the condition is defined and what parameters are used to measure it. The evidence from the literature is confusing and difficult to compare. Some researchers have reported the prevalence of erosion, ^{24,25} some have reported the prevalence of tooth wear, ^{1,55,63} and to complicate the matter further, others have reported the prevalence of attrition. ^{15,64} The use of different terminology also confuses the interpretation of studies from the 2 continents and from different European countries.

Smith and Knight³ described a method for measuring tooth wear using an index to record the loss of tooth tissue on the lingual/palatal, buccal, cervical, and occlusal/incisal surfaces of teeth. The index graded the degree of wear from 0 to 4 depending on its severity; 0 is defined as no wear, while 4 represents severe wear or pulpal exposure. This index was used to report the prevalence of tooth wear in 1,007 patients examined in general practice in southeastern England as described earlier.⁶³ In the UK National Child Dental Health Survey²⁵ the authors used the Smith and Knight index but reduced the grading to 3 levels, making a comparison to the results of other studies on children and adults difficult.^{65,66}

In several studies a diagnosis has been made and then an index used to record its prevalence, for example, the prevalence of attrition on the incisal or occlusal surfaces of teeth. 61,64 Identifying the prevalence of tooth wear by specifying the etiology is hazardous unless individual clinical histories of each patient are thoroughly investigated. This is not always achieved. In 1988 Seligman et al¹⁵ reported the prevalence of attrition recorded from study casts taken from 222 young adults and reported that nearly 92% had evidence of dentin exposure on their incisal or occlusal surfaces. Hugoson et al⁶⁷ reported similarly high levels of incisal or occlusal wear in 527 children. Although some of these authors used the term tooth wear in the title of their articles, they used an occlusal score to record the severity of the wear, which ignores by definition all other surfaces. This type of index excludes palatal/lingual wear, which is most commonly associated with erosion.

It would appear that at least one group of North American researchers reported on the prevalence of erosion in their study of 10,000 extracted teeth.⁵⁹ However, on close inspection of the methodology the authors appear to classify cervical wear lesions as erosive in origin. The appearance of the cervical wear lesion has traditionally been associated with abrasion

and erosion rather than just erosion, an etiology that tends to be at some variance to the literature.

Early Tooth Wear as a Predictor of Later Susceptibility?

An important factor in measuring tooth wear is the extent of damage. If dentin is exposed on the incisal edges of the maxillary anterior teeth in a 15 year old and the situation remains relatively stable for the next 40 years it is unlikely to be a significant problem. However, if the wear continues and ultimately compromises the appearance and function of the teeth there is a problem. Most of the studies on the prevalence of erosion, attrition, or tooth wear report that early wear is common and severe wear, by comparison, is not.

There is some evidence that wear in the young may predict wear later in life. Knight et al⁶⁴ measured occlusal/incisal wear in the pretreatment and posttreatment records of 223 patients treated orthodontically. The authors reported that there were statistically significant associations with wear on the mandibular deciduous incisors and in the entire mouth of the same person as an adult. Pintado et al⁶² measured wear over 2 years on 18 adults and also reported that attrition had progressed. The possibility that wear is episodic is supported by evidence that children pass through active phases of wear between the ages of 3 and 12 years and then the wear slows later in life.⁶⁸ If so, some of the high levels of erosion reported in children in the UK may later become inactive in adult life. Until a method is developed to measure wear, and in particular dental erosion, accurately this question will remain unanswered.

Conclusion

Fundamental to interpreting the information from studies on the etiology and prevalence of tooth wear are the differences between erosion, attrition, and abrasion. It would appear from the European literature that the most common and destructive cause of tooth wear is erosion. Conversely, most of the prevalence studies in North America have concentrated on attrition rather than tooth wear or erosion. It is only through a comprehensive history, studying the appearance of a wear lesion, and considering the etiology that a specific diagnosis can be properly made. If a specific diagnosis of predominantly erosion or attrition cannot be made despite a thorough investigation, then the descriptive term tooth wear should be used.

Perhaps the reason there is more interest in tooth wear, and in particular dental erosion, in Europe is that there has been more research in Europe on the etiology and prevalence, much of it on children.

References

- Smith BGN, Knight JK. A comparison of patterns of tooth wear with aetiological factors. Br Dent J 1984;157:16–19.
- 2. The glossary of prosthodontic terms, ed 6. J Prosthet Dent 1994; 71:43–112.
- Smith BGN, Knight JK. An index for measuring the wear of teeth. Br Dent J 1984;156:435–438.
- Darby ET. Dental erosion and gouty diathesis: Are they usually associated? Dent Cosmos 1892;34:629–640.
- Grenby TH, Mistry M, Desai T. Potential dental effects of infant's fruit drinks studied in vitro. Br J Nutrition 1990;64:273–283.
- Grenby TH, Phillips A, Desai T, Mistry M. Laboratory studies of dental properties of soft drinks. Br J Nutr 1989;62:451–464.
- Rytomaa I, Meurman JH, Koskinen J, Laakso T, Gharazi L, Turunen R. In vitro erosion of bovine enamel caused by acidic drinks and other foodstuffs. Scand J Dent Res 1988;96:324–333.
- Meurman JH, Harkonen M, Naver H, Koskinen J, Torkko H, Rytomaa I, et al. Experimental sport drinks with minimal dental erosive effect. Scand J Dent Res 1990;98:120–128.
- Smith AJ, Shaw L. Baby fruit juices and tooth erosion. Br Dent J 1987;162:65–67.
- Davis WB, Winter PJ. Dietary erosion of adult dentine and enamel. Br Dent I 1977;143:116–119.
- Asher C, Reid MJF. Early enamel erosion in children associated with the excessive consumption of citric acid. Br Dent J 1987; 162:384–387.
- Giunta JL. Dental erosion resulting from chewable vitamin C tablets. J Am Dent Assoc 1983;107:253–256.
- Eccles JD, Jenkins WG. Dental erosion and diet. J Dent 1974; 2:153–159.
- 14. Milosevic A. Toothwear: Aetiology and presentation. Dent Update 1998;25:6–11.
- Seligman DA, Pullinger AG, Solberg WK. The prevalence of dental attrition and its association with factors of age, gender, occlusion, and TMJ symptomatology. J Dent Res 1988;67:1,323–1,333.
- Kelleher M, Bishop K. The aetiology and clinical appearance of tooth wear. Eur J Prosthodont Restorative Dent 1998;5:157–160.
- 17. Bartlett DW. The causes of dental erosion. Oral Dis 1997; 3:209-211.
- Lussi A, Jaeggi T, Jaeggi-Scharer S. Prediction of the erosive potential of some beverages. Caries Res 1995;29:349–354.
- Lussi A, Jaeggi T, Jaeggi-Scharer S. The influence of different factors on in vitro enamel erosion. Caries Res 1993;27:387–393.
- West NX, Maxwell A, Hughes JA, Parker DM, Newcombe RG, Addy M. A method to measure clinical erosion: The effect of orange juice consumption on erosion of enamel. J Dent 1998; 26:329–335.
- 21. Thomas AK. Further observations on the influence of citrus fruit juices on human teeth. N Y State Dent J 1957;23:424–430.
- Johansson A-K, Johansson A, Birkhed D, Omar R, Baghdadi S, Carlsson G. Dental erosion, soft-drink intake, and oral health in young Saudi men, and the development of a system for assessing erosive anterior tooth wear. Acta Odontol Scand 1996;54:369–378.
- Millward A, Shaw L, Smith AJ, Rippin JW, Harrington E. The distribution and severity of tooth wear and the relationship between erosion and dietary constituents in a group of children. Int J Paedtr Dent 1994;4:151–157.
- Bartlett DW, Coward PY, Nikkah C, Wilson RF. The prevalence of tooth wear in a cluster sample of adolescent schoolchildren and its relationship with potential explanatory factors. Br Dent J 1998;184:125–129.
- O'Brien M. Children's Dental Health in the United Kingdom 1993. London: Office of Population Censuses and Surveys, 1993.
- Hellstrom I. Oral complications in anorexía nervosa. Scand J Dent Res 1977;85:71–86.

- Bartlett DW, Evans DF, Smith BGN. Oral regurgitation after reflux-provoking meals: A possible cause of dental erosion? J Oral Rehabil 1997;24:102–108.
- Bartlett DW, Evans DF, Smith BGN. Review: The relationship between gastro-esophageal reflux disease and dental erosion. J Oral Rehabil 1996;23:289–297.
- Robb ND, Smith BGN. Dental erosion in patients with chronic alcoholism. J Dent 1989;17:219–221.
- 31. Gilmour AG, Beckett HA. The voluntary reflux phenomenon. Br Dent J 1994;175:368–372.
- Jarvinen V, Meurman JH, Hyvarinen H, Rytomaa I, Murtomaa H. Dental erosion and upper gastrointestinal disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1988;65:298–303.
- Schroeder PL, Filler SJ, Ramirez B, Lazarchik DA, Vaezi MF, Richter JE. Dental erosion and acid reflux disease. Ann Intern Med 1995;122:809–815.
- 34. Bartlett DW, Evans DF, Anggiansah A, Smith BGN. A study of the association between gastro-esophageal reflux and palatal dental erosion. Br Dent J 1996;181:125–132.
- Skogedal O, Silness J, Tangerud T, Laegried O, Gilhuus-Moe O. Pilot study on dental erosion in a Norwegian zinc factory. Community Dent Oral Epidemiol 1977;5:248–251.
- Petersen PE, Gormsen C. Oral conditions among German battery factory workers. Community Dent Oral Epidemiol 1991; 19:104–106.
- 37. Berry DC, Poole DFG. Masticatory function and oral rehabilitation. J Oral Rehabil 1974;1:191–205.
- Woda A, Gourdon AM, Faraj M. Occlusal contacts and tooth wear. J Prosthet Dent 1987;57:85–93.
- Xhonga FA. Bruxism and its effect on the teeth. J Oral Rehabil 1977;4:65–76.
- Molnar S, Mckee JK, Molnar IM, Przybeck TR. Tooth wear rates among contemporary Australian aborigines. J Dent Res 1983;62:562–565.
- 41. Ramfjord S, Ash M. Occlusion, ed 3. London: WB Saunders, 1983:179–182.
- Ramfjord SP. Bruxism, a clinical and EMG study. J Am Dent Assoc 1961;62:21–44.
- Rugh JD, Barghi H, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxism. J Prosthet Dent 1982;51:548–553.
- Rugh JD. Psychological factors in the etiology of masticatory pain and dysfunction. Chicago: American Dental Association, 1983:85–94.
- 45. Svinnseth PN, Gjerdet NR, Lie T. Abrasivity of toothpastes. Acta Odontol Scand 1987;45:195–202.
- Redmalm G. Dentifrice abrasivity. The use of laser light for determination of the abrasive properties of different silicas. An in vitro study. Swed Dent J 1986;10:243–250.
- 47. Lewis KJ, Smith BGN. The relationship of erosion and attrition in extensive tooth tissue loss. Br Dent J 1973;135:400–404.
- Lee WC, Eakle WS. Possible role of tensile stress in the aetiology of cervical erosive lesions of teeth. J Prosthet Dent 1984;52: 374–379.

- 49. Molnar S, Richards L, McKee J, Molnar I. Tooth wear in Australian populations from the River Murray Valley. Am J Phys Anthropol 1989;79:185–196.
- Jarvinen V, Rytomaa II, Heinonen OP. Risk factors in dental erosion. J Dent Res 1991;70:942–947.
- 51. Bartlett DW, Smith BGN. The dental impact of eating disorders. Dent Update 1995;21:404–407.
- Gudmundsson K, Kristleifsson G, Theodors A, Holbrook WP. Tooth erosion, gastroesophageal reflux, and salivary buffer capacity. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995; 79:185–189.
- Milosevic A, Dawson LJ. Salivary factors in vomiting bulimics with and without pathological tooth wear. Caries Res 1996;30: 361–366.
- Milosevic A, Slade PD. The orodental status of anorexics and bulimics. Br Dent J 1989;167:66–70.
- 55. Smith BGN, Robb ND. The prevalence of tooth wear in 1007 dental patients. J Oral Rehabil 1996;23:232–239.
- 56. Robb ND, Cruwys E, Smith BGN. Regurgitation erosion as a possible cause of tooth wear in ancient British populations. Arch Oral Biol 1991;36:595–602.
- 57. Jarvinen V, Rytomaa I, Meurman JH. Location of dental erosion in a referred population. Caries Res 1992;26:391–396.
- Linkosalo E, Markkanen H. Dental erosions in relation to lactovegetarian diet. Scand J Dent Res 1985;93:436–441.
- Sognnaes RF, Wolcott RB, Xhonga FA. Erosion-like patterns occurring in association with other dental conditions. J Am Dent Assoc 1972;84:571–576.
- Milosevic A, Young PJ, Lennon MA. The prevalence of tooth wear in 14-year-old school children in Liverpool. Community Dent Health 1994;11:83–86.
- Silness J, Berge M, Johannessen M. Longitudinal study of incisal tooth wear in children and adolescents. Eur J Oral Sci 1995; 103:90–94.
- 62. Pintado MR, Anderson GC, DeLong R, Douglas WH. Variation in tooth wear in young adults over a two-year period. J Prosthet Dent 1997;77:313–320.
- Smith BGN, Bartlett DW, Robb ND. The prevalence, etiology and management of tooth wear in the United Kingdom. J Prosthet Dent 1997;78:367–372.
- Knight DJ, Leroux BG, Zhu C, Almond J, Ramsey DS. A longitudinal study of tooth wear in orthodontically treated patients. Am J Orthod Dentofac Orthop 1997;112:194–202.
- Millward A, Shaw L, Smith AJ. Dental erosion in four-year-old children from differing socioeconomic backgrounds. J Dent Child 1995;61:263–266.
- Lussi A, Schaffner M, Holtz P, Suter P. Dental erosion in a population of Swiss adults. Community Dent Oral Epidemiol 1991; 19:286–290.
- 67. Hugoson A, Bergendal T, Ekfeldt A, Helkimo M. Prevalence and severity of incisal and occlusal tooth wear in an adult Swedish population. Acta Odontol Scand 1988;46:255–265.
- 68. Cash RG. Bruxism in children: Review of the literature. J Pedod 1988;12:107–127.