article

Dental Erosion: Part 1. Aetiology and Prevalence of Dental Erosion

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Summary

Non-carious tooth tissue loss due to abrasion, attrition, abfraction and erosion has become a significant problem, occurring in up to 80 percent of children and in up to 43 percent of adults. Dental erosion is now recognised as an important cause of tooth tissue loss in both children and adults. It is caused by the presence of intrinsic or extrinsic acid of non-bacterial origin in the mouth. Intrinsic sources of acid include vomiting, regurgitation, gastro-oesophageal reflux or rumination. Extrinsic sources of acid are most commonly dietary acids. Medications, a patient's lifestyle choices and environment can also increase the risk of dental erosion. In this article we identify the prevalence of dental erosion and its main causative factors, and in Part II we will discuss the management of dental erosion.

INTRODUCTION

Tooth wear or non-carious loss of tooth tissue is a normal physiological process and occurs throughout life (Flint and Scully, 1988). Tooth wear is usually a multifactorial process which can be a result of erosion, attrition, abfraction or abrasion either alone or in various combinations (Milosevic, 1988). These terms are used to describe specific processes that are associated with the loss of dental hard tissues and their definitions are summarised in Table 1. Problems arise when the loss of hard dental tissue is substantial enough to cause either tooth sensitivity or aesthetic and functional problems for an individual.

It is not always possible to differentiate between erosion, attrition, and abrasion, as these conditions frequently occur in combination (Smith et al, 1997), but with differing proportional effects (Nunn et al, 1996). Erosion is a destructive process that renders the eroded tooth surface hypomineralised, making it more susceptible to the effects of abrasion and attrition (Imfeld, 1996; Jaeggi and Lussi, 1999). Erosion can lead to substantial loss of enamel and exposure of the softer dentine (Mahoney et al, 2000), which is more susceptible to abrasion and attrition; combined, these processes will accelerate tooth tissue loss. The clinical erosive lesion (Figures 1 and 2), is smooth, polished and rounded, with loss of tooth surface characteristics (Eccles, 1982; Imfeld, 1996), and any restorations present become proud (Deery et al, 2000) (Figure 3). The acids responsible for erosion are not products of the intraoral flora but stem from intrinsic, extrinsic or unknown sources (Imfeld 1996). Dental erosion, if allowed to progress, can lead to tooth sensitivity, poor aesthetics, loss of occlusal vertical dimension and functional problems.

TABLE I. Definitions of causes of tooth wear.

Term	Definition		
Abfraction	Loss of hard tissue from eccentric occlusal loads leading to compressive and tensile stresses at the cervical fulcrum area of the tooth. The tensile stresses weaken the cervical hydroxyapatite, which produces a special form of wedge-shaped defect with sharp rims at the cemento-enamel junction (Imfeld, 1996). (For review see Lyons, 2001)		
Abrasion	Pathological wearing away of dental hard tissue through abnormal mechanical processes, involving foreign objects or substances repeatedly introduced into the mouth and contacting teeth (Imfeld, 1996).		
Attrition	Physiological loss of tooth tissue as a result of tooth to-tooth contact with no foreign substances intervening (Imfeld, 1996). Attrition therefore primarily affects the occlusal and incisal surfaces of the teeth (Kelleher and Bishop, 1999).		
Erosion	The original definition of erosion is, the removal of hard tissue from the tooth surface by chemical means in the absence of dental plaque. Usually the agent is an acid but some tooth loss is possible through		



chelating action at near-normal pH (Eccles, 1982).

FIG 1 - Erosive lesions on posterior teeth in the permanent dentition.

Over the past five years, awareness of the deleterious effects of erosion has increased in both the adult and the paediatric population, as attested by the literature on this subject. Erosion can be a particular problem for young children, as the enamel and dentine layers of the primary dentition are much thinner than those of the permanent tectle (Figure 4). Once dentine becomes exposed, tissue-loss accelerates (Shaw and Smith, 1994). There is anocdotal evidence to support the suggestion that in children, erosion may be increasing (Harley, 1986; Shaw and Smith, 1994), although the reason for this is unknown.

In the management of dental erosion, determining its cause is the first step. Although for some patients this is obvious, determining the cause of an individual patient's dental erosion can be difficult, due to the variety of possible causes. A thorough understanding of the possible causes is therefore required. The aim of this article is to outline the prevalence and causes of dental erosion. The second article in this series will discuss its treatment.

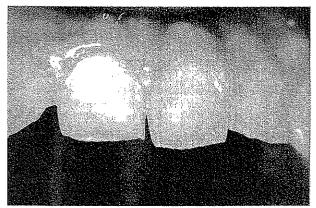


FIG 2 - Erosive lesions of anterior teeth.



PIG 3 - Erosive lesions of permanent teeth resulting in proud amalgam restorations.

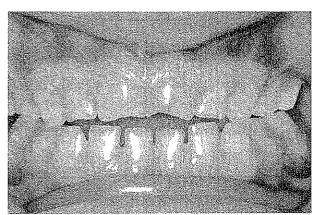


FIG 4 - Erosion of the primary dentition.

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PREVALENCE OF DENTAL EROSION

It is difficult to assess the prevalence of tooth wear in general, as it is often difficult to distinguish erosion from other forms of tooth wear. There have been many scales and indices suggested to measure tooth wear, and their very diversity suggest that not one of them is ideal (Shaw and Smith, 1998). Each scale used to determine the presence of tooth wear also grades the severity of the wear (for

example, whether the wear is contained in enamel, or extends so that dentine or even pulp is exposed), and these results are often used when determining the management of erosion. The mere presence of erosion does not always indicate a clinical problem.

Table II summarises the data available to date on the prevalence of tooth wear in adults. It would appear that between 5 and 43 percent of adults have some form of tooth wear, however it is uncommon for these studies to identify erosion specifically. All adult studies have shown that tooth wear increases with increasing age. This is not surprising, as tooth wear is a normal physiological process and occurs throughout life (Flint and Scully, 1988).

TABLE II. Summary of studies investigating the prevalence of tooth wear in the adult population.

Authors (and date)	Population	Number	Prevalence
Smith and Robb. 1996	15 to 654 yrs	1007	5.73 percent 15-26 years, 8.19 percent 56-65 years, and 8.84 percent 65 years+ bad tooth surfaces worn to an unacceptable degree.
Robb et al, 1991	16 yrs+ and skutts	884 151	19.9 percent of skulls had extensive tooth wear with 71 percent of this tooth wear due to crosion
Incolnen	Arlidt	106	5 nerrent

Xhonga 14-80 yrs 521 251 et al, 1972

However, in studies involving the paediatric population it would appear that erosion is often assessed more specifically, with signs being shown in up to 80 percent of all primary incisors (Millward et al, 1994b). Table III summarises the data available on the prevalence of erosion in children. In one study signs were found in nearly half of the 178 children between the age of four and five (Millward et al. 1994a). Ayers and colleagues (2002) reported in their study of 104 5-8 year-old pre-school children in Dunedin, New Zealand, that 82 percent showed some form of tooth wear in their primary molars or canines. These authors did not attempt to ascribe an actiology to the wear reported. The finding in the UK National Diet and Nutrition Survey that up to 10 percent of children as young as one and a half years old showed signs of crosion associated with the labial surfaces of their primary incisors (Hinds and Gregory, 1995), is interesting and very alarming. This amount of erosion can have long-term consequences such as pulp exposure if this rate of crosion continues in this young patient group.

TABLE III. Summary of studies investigating the prevalence of erosion in the child population.

Authors (and date)	Population	Number	Prevalence
Ayers et al,	5-8 yrs	104	82 percent of children 2002 exhibited tooth wear; study did not distinguish between erosion and other forms of tooth wear
Al-Malik et a 2002	1, 2-5 yrs	987	31 percent evidence of erosion 12 percent erosion through to dentine and/ or pulp
Al-Dlaigan et al. 2001a	14 yrs	418	48 percent low erosion; 51 percent moderate erosion; 1 percent severe erosion
Decry et al, 2000	11-13 yrs	129 in USA 125 in UK	41 percent of children in USA and 37 percent in the UK had erosion
Kunzel et al. 2000	12 yrs	1010	17.4 percent of children examined
Bartlett et al 1998	,11-14 yrs	210	57 percent had enamel erosion on 10+ teeth; 2 percent had incisal and palatal surfaces with exposure of dentine
Hinds and Gregory, 1995	1.5 – 4.5 yrs	1658	10 percent of children had erosion on buccal of incisors 19 percent had erosion affecting palatal surfaces
Millward et al. 1994a	4 – 5 yrs	178	> 50 percent showed signs of erosion

AETIOLOGY OF DENTAL EROSION Extrinsic erosion

Extrinsic erosion results from the action of exogenous acids, that is, acids entering the mouth from an external source. Traditionally, factors that may cause extrinsic erosion have been identified as originating from the environment, diet, medications and lifestyle choices of a patient.

Environmental Erosion

Environmental erosion occurs when patients are exposed to acids in their work-place or during leisure activities (Centerwall et al. 1986).

The most commonly-quoted examples of environmental erosion are those reported in factory workers and in long-term swimmers in gas-chlorinated swimming pools (ten Bruggen Cate, 1968; Centervall et al, 1986; Petersen and Gormsen, 1991; Tuomineñ et al, 1991; Filler and Lazarchik, 1994), ten Bruggen Cate (1968) investigated the amount of erosion in factory workers who were exposed to acid fumes

and aerosols, compared with a control population who were also factory workers but worked in acid-free environments. The battery factory workers were found to have the highest incidence of dental erosion. Petersen and Gormsen (1991) also investigated battery factory workers who were exposed to sulphuric acids. These authors noted that many workers in the factory reported having sharp, short, or fragile teeth. The prevalence of erosion amongst this group was 31 percent, however a large number of their teeth (average, 5.3 teeth per person) had been crowned previously, though which teeth were crowned was not reported.

More recently there have been reports in the literature of a link between wine tasting and tooth erosion. Wine has a pH of between 3.3 and 3.8, and has been reported to cause significant erosion when tasters are exposed to the liquid up to 30 times per day over many years (Chaundhy et al, 1997; Gray et al, 1998).

Medications

Many reports associate medications and over-the-counter dental products with erosion (Bhatti et al, 1994), as many medications have a very low pH (Lussi and Hellwig, 2001). Rytomaa and colleagues (1989) reported on their findings on the erosive potentials of mouthwashes. They found that the mouthwash Calcusan produced four times more surface erosion in vitro than did a sports drink known from previous investigations to be erosive. The authors stated that the EDTA (ethylenediaminetetracetic acid) contained in this mouthwash is a recognised demineralising agent, and suggested that its presence was responsible for the high erosive potential (Rytomaa et al, 1989). Medications in tablet form, for example aspirin and vitamin C, have also been associated with erosion (Sullivan and Kramer, 1983; Meurman and Murtomaa, 1986).

The pH of common asthma medications ranges from 4.31 (Bricanyl, powder form) to 9.30 (Ventolin, acrosol form) (O'Sullivan and Curzon, 1998b). The powder form of all these medications has a significantly lower pH than does the aerosol form. In the powder form, almost all commonly used drugs have a pH of less than 5.5. This could have significant effect on tooth erosion (O'Sullivan and Curzon. 1998b). Shaw and colleagues (2000) recently showed in their random sample of 14-year-old children that those with asthma had significantly more erosion than those who did not. The relationship between asthma and crosion has been supported by other studies (McDerra et al, 1998; Al-Dlaigan et al, 2002). The possibility that children with asthma have an increased incidence of gastro-oesophageal reflux has also been suggested as a co-factor in the actiology of dental crosion (Wilson et al, 1985; Jarvinen et al, 1991).

In a case-control study, 40 children with phenylketonuria and their siblings were examined for erosion (Kilpatrick et al, 1999). The children with phenylketonuria had a significantly higher incidence of tooth wear, of which erosive wear appeared to be the predominant process. The authors identified the amino-acid supplements used in the management of the condition as a potential cause of the erosion due to their low pH and high titratable acidity.

Lifestyle

Lifestyle habits, choices and conditions may have a direct effect on the amount of erosion experienced by individuals. This is highlighted in many case reports and studies. As early as 1977 there was a report of significant erosion in a man who held Coca-Cola in his mouth until all the dissolved gas had come out of solution (High, 1977).

A study carried out in 1994 found very little erosion in children from a low socio-economic background, compared with children from higher socio-economic groups (Millward et al, 1994a). The authors concluded that the differences might be due to their differing dietary habits or oral hygiene practices, which were not determined in that study. Davis and Winter (1980) reported that abrasion was accelerated by toothbrushing following demineralisation from dietary acid exposure. In contrast to the study by Millward and colleagues (1994a), Al-Dlaigan and colleagues found in a group of 14-year-old school children that there was significantly more erosion in children from low socio-economic groups (Al-Dlaigan et al, 2001a).

Over the past few years an increasing amount of literature has identified other, less-common lifestyle choices that may be linked to dental erosion and tooth wear. Milosevic and colleagues reported in 1999 that 60 percent of people using ecstasy (3,4 methylenedioxymethamphetamine) had tooth wear involving dentine, in comparison to only 11 percent of non-users. The main risk factors identified in this study were dry mouth, tooth grinding and increased consumption of carbonated beverages, all of which are seen in this type of drug use. Although the authors did not draw conclusions as to the aetiological factor causing the tooth wear, the common practice of ingestion of acidic beverages to alleviate the dry mouth in this group of people is an obvious behaviour-related risk factor (Milosevic et al, 1999). Richards and Brofeldt (2000) report that the preferred route of administration of ecstasy can affect the tooth wear seen. Patients who "snorted" ecstasy had significantly more tooth wear on their anterior teeth than did those who injected it.

Acidic Drinks

Many in vivo and in vitro studies link acidic drinks and erosion (Zero, 1996; O'Sullivan and Curzon, 2000; Al-Dlaigan et al, 2001b). Table IV summarises a number of studies in the literature from 1990. The acids most frequently found in drinks are citric, malic, phosphoric and carbonic (Milosevic, 1988), but the most frequentlyconsumed erosive acids are the fruit acids and phosphoric acids contained in fresh fruit, fruit juices and soft drinks (Al-Dlaigan et al, 2001b; Allan, 1967; Asher and Read, 1987; Levine and Rowles, 1973; Lussi et al, 1993). The pH of most fruit juices, sports drinks and soft drinks varies between 2.1 and 4.46 (Eccles, 1982; Eccles and Jenkins, 1974; Smith and Shaw, 1987; Milosevic, 1997); this is well below the critical pH value for enamel demineralisation (Smith and Shaw, 1987; Milosevic, 1997). O'Sullivan and Curzon, (2000) found in their case-control study that young patients with erosion consumed a significantly larger quantity of carbonated beverages and cordials than did controls. They also found that 43 percent of patients presenting with erosion had swishing or sucking habits while drinking. This was in stark contrast to the 3 percent of controls with similar habits (O'Sullivan and Curzon, 2000). This study shows that the erosive process is influenced by the way the beverage is consumed, as well as by the quantity consumed.

There have been at least two reports of alcoholic soft drinks having a high erosive potential (Rees et al, 1998; O'Sullivan and Curzon, 1998a). Rees and co-workers in 1998 demonstrated in vitro that, depending on the flavour, the pH of alcoholic lemonades ranged from 2.57 to 3.54.

The authors concluded that alcoholic lemonades had a greater erosive potential than orange juice. After immersion in the product for one hour, the enamel surface loss of extracted teeth was between 1.8 and 3.28 μ m, which (apart from the blackcurrant flavoured lemonade) was greater than that caused by orange juice.

Acidic foods.

Much less research exists on the potential erosive effects of acidic foods; the available literature is summarised in Table V. Jarvinen and colleagues showed that people who consumed citrus fruits more than twice a day had a risk of erosion 37 times greater than that of individuals who consumed citrus fruit less often (Jarvinen et al, 1991). This finding is reinforced by many other reports (Eccles and Jenkins, 1974; Smith and Shaw, 1987; Grobler et al, 1989; Milosevic, 1997; Rees et al, 1998; O'Sullivan and Curzon, 1998a; O'Sullivan and Curzon, 2000).

Acids are present in foods such as tomato sauces and baked beans, as well as in vinegar (acetic acid), which can be used as an accompaniment to food or as an ingredient in cooking (Milosevic et al, 1994). The erosive potential from these foods is probably dependant on individual eating habits (Milosevic, 1988). In one study, individuals who consumed a lactovegetarian diet were shown to have a higher frequency of erosion than the general population (Linkosalo and Markkanen, 1985). This is not surprising, as lactovegetarian diets contain plenty of coarse fresh foods, citrus fruits, acidic drinks and honey, which may increase the frequency of erosion. The subjects also had a much higher frequency of ingestion of acidic berries, vinegars, vinegar conserves and acidic drinks (Linkosalo and Markkanen, 1985). A more recent study involving a group of 14-year-old children found that although dental erosion is common in this age group, there was no significant difference in this regard between vegetarian and nonvegetarian children (Al-Diaigan et al, 2001c).

Intrinsic Erosion

The term intrinsic erosion implies the presence of gastric acid in the oral cavity (Milosevic, 1988). Vomiting, regurgitation, gastro-oesophageal reflux and rumination can result in the presence of gastric acids in the oral cavity.

Vomiting and Eating Disorders

Vomiting has many causes, including pregnancy and the side-effects of some drugs, disorders of the alimentary tract, as well as neurological, metabolic and endocrine disorders, and a large group of psychosomatic disorders including eating disorders and stress-induced psychogenic disorders (Scheutzel, 1996). The effects of vomiting are welldocumented in patients suffering from bulimia nervosa, as individuals with this condition may vomit many times per day over a long period of time (Stege et al, 1982; Robb et al, 1995; Burke et al, 1996). Rytomaa and colleagues (1998) found that the incidence of tooth erosion, abrasion and attrition was 1.5 to 6 times more common among bulimics than among controls. The most common sign of this condition is the presence of perimolysis-erosive lesions localised on the palatal aspect of the maxillary teeth (Rytomaa et al, 1998).

Patients suffering from bulimia often binge on foods with high energy and sugar content, as well as foods which may also have a high erosive potential (Rytomaa et al, 1998). Bidwell and colleagues describe a male adult patient with

TABLE IV. Summary of studies investigating the erosive potential of beverages.

Author and date	Beverage/s Studied	Method of investigation	Results
Al-Dlaigan et al, 2001b	Soft drinks, carbonated beverages, alcoholic drinks	In vivo epidemiological study	Statistically significant association
Brunton and Hussain, 2001	Black and herbal tea	In vitro profilometry	Both types of tea caused loss of enamel Time period immersed very long (14days)
Larsen and Nyvad, 1999	18 soft drinks, mineral waters and juices	In vitro, microradiograph	Soft drinks erosion> mineral waters
Rees et al, 1998	Alcoholic lemonade 'Hooch'	In vitro profilometry	Considerable erosive potential
West et al, 1998	Orange juice with water as control	In vivo and in vitro surfometry	Orange juice significant erosive potential
Johansson et al, 1997	Association of erosion with consumption of soft drinks	In vivo epidemiological study	Significant correlation of soft drink consumption and dental erosion
Jussi et al, 1995	Various juices carbonated and uncarbonated	In vitro surface microhardness	Apple juice, Schweppes> orange soft drink> grapefruit juice; all produced significant decreases of surface microhardness
Mistry and Grenby, 1993	Apple juice, still orange juice and carbonated orange	In vivo digital image analysis on rats teeth	Intact surface remaining after 6 week exposure was 47 percent for apple juice, 27 percent on still orange juice and only 6 percent on carbonated orange
arvinen et al, 1991	Soft drinks and sports drinks	In vivo epidemiologica) study	Considerable risk of dental erosion if soft drinks were consumed daily or if sports drinks were consumed weekly
Grobler et al, 1990	Orange and apple juice, Pepsi and Diet Pepsi cola	In vitro, calcium dissolution	Highly significant increase in calcium concentration of all beverages at 4 to 40 minutes immersion of enamel in drinks. Orange juice> apple juice> Pepsi> Diet Pepsi

extensive erosion that they associated with the patient's self-reported bulimia disorder (Bidwell et al, 1999). However, these authors made no mention of possible confounding factors such as the large quantities of alcohol consumed daily by this patient, which undoubtedly increased the severity of the erosion. Other authors have noted the relationship between chronic alcoholism and erosion (Simmons and Thompson, 1987; Smith and Robb, 1989).

Regurgitation and Gastro-oesophageal Reflux

Gastro-oesophageal reflux is the involuntary passing of gastric contents into the oesophagus. This condition is due to laxity of the lower oesophageal sphincter, which allows the contents of the stomach into the oesophagus. Gastrooesophageal reflux is a frequent finding in patients with cerebral palsy, bronchitis and other respiratory disorders, hiatus hernia, and pregnancy, and it has also been reported to occur two to three times more commonly in children with asthma than in the general population (Kumar and Clark, 1990; Mansfield 1992). Gastric acidity ranges from pH 1 to pH 3 and therefore if the contents of the stomach reach the oral cavity there is no doubt that these acids have the potential to cause dental erosion. Some dietary components such as chocolate, fat and coffee can also cause reflux, as can smoking, chronic alcoholism and certain types of exercise (Rytomaa et al, 1998; Kumar and Clark, 1990; Bishop et al, 1997).

Although not all patients with reflux disease develop erosion (Meurman et al, 1994), regurgitation of gastric acid

into the oral cavity has been associated with tooth erosion in several studies (Howden 1971; Jarvinen et al, 1991; Bartlett et al, 1996). Bartlett and colleagues (1996) demonstrated a strong association between gastro-oesophageal reflux and erosion of the palatal surface of maxillary teeth. Patients with erosion but with no previous history of gastrooesophageal reflux were tested for gastro-oesophageal reflux. This well-designed study continuously measured the pH change over a 16 to 23-hour period at three positions, the distal oesophagus, the proximal oesophagus and the oral cavity (palate). Of the patients with palatal erosion, 64 percent (n=36) had pathological gastro-oesophageal reflux, indicated by distal oesophagus pH measurements over a 16 to 23-hour period. From the results of the distal oesophagus pH measurements, the authors concluded that palatal erosion was strongly associated with gastro-oesophageal reflux, although there was no such relationship between reflux and the observed pH change intraorally.

Although most studies have evaluated reflux and erosion in adults, a similar relationship in children has been suggested (Taylor et al, 1992), but this relationship may be less clear (Gudmundsson et al, 1995; Dodds and King, 1997). O'Sullivan and colleagues (1998c) investigated the amount of erosion in the primary and permanent teeth of children with previously confirmed moderate to severe gastro-oesophageal reflux (using 24-hour pH monitoring). This study did not have a control group of patients, but used the rate of erosion reported in the 1993 UK Child Dental Health Survey (O'Brien, 1994) to compare the level of

TABLE V. Summary of studies investigating the crosive potential of foodstuffs.

Author and date	nor and date Food studied Method of investigation		Results
Al-Dlaigan et al, 2001b	Fresh fruit, Vitamin C tablets and other acidic foodstuffs	<i>In vivo</i> epidemiological study	Statistically significant association between these foods and erosion in random sample of 14-year-old children
Kunzel et al, 2000	Oranges	Case report	17.4 percent of children examined had erosion
O'Sullivan et al, 2000	Fresh fruit	In vivo case control study	Consumption of fresh fruit at least twice per day caused statistically significant more erosion than controls
Dodds et al, 1997a	Mexican citric acid snacks	In vitro microhardness	Statistically significant reduction in Vickers hardness number for snacks
Grobler et al, 1994	Honey	Scanning electron microscopy and in vitro microhardness	No erosion noted with either method
Jarvinen et al, 1991	Citrus fruits	<i>In vivo</i> epidemiological study	Considerable risk of dental erosion if citrus fruits consumed more than twice a day

erosion with that of a control child population. Although the authors admitted that a number of the children investigated were being treated before the study with anti-reflux medicine, they did not attempt to compare the erosion in patients who had been on preventative medicine with that found in those who had not.

They did conclude that dental erosion might not be as great a problem in children as it is in adults suffering from gastro-oesophageal reflux, due to the low occurrence of erosion seen in their potentially at-risk population (O'Sullivan et al, 1998c).

Rumination

Rumination is the voluntary regurgitation of gastric contents into the mouth. It is more commonly seen in institutionalised mentally-disabled children and adults, particularly those suffering from stress or significant psychological disturbances (Amarnath et al, 1986; Reis, 1994). The contents of the stomach are often held in the mouth, leading to erosion on the palatal surfaces of the teeth as well as on the buccal surfaces of the posterior teeth (Gilmour and Beckett, 1993). It is difficult to confirm a diagnosis of rumination from the oral condition as no clear clinical picture is evident (Scheutzel, 1996). In general, there is little known about the rumination syndrome and the prevalence of erosion associated with it (Milosevic, 1988).

Salivary Gland Hypo-function / Dehydration

There is increasing evidence from the literature that salivary flow rates and the quality of a patient's saliva may be important in the aetiology of dental erosion, although no causal relationship has been shown (Young, 2001; Khan et al, 2001; O'Sullivan and Curzon, 2000). Young and colleagues have shown through numerous case reports that dehydration is a risk factor for dental erosion, especially in younger patients (Young, 2001; Khan et al, 2001). Young and colleagues have also investigated the distribution and severity of erosive lesions (lower first and second permanent molars having cupped erosive lesions more commonly than do maxillary molars), and concluded that the differences reflect the ability of saliva to protect specific teeth in different parts of the mouth (Khan et al, 2001).

Idiopathic Erosion

Idiopathic erosion is caused by acids of unknown origin, where no tests are capable of providing an aetiology (Imfeld, 1996).

CONCLUSION

Dental erosion has come to be recognised as occurring in a significant proportion of the population, although it does not always indicate a clinical problem needing immediate treatment. It can result in tooth sensitivity, poor aesthetics, loss of occlusal vertical dimension, and functional problems such as impaired mastication. Clinicians must have a thorough understanding of the causes of dental erosion, as identification of the cause is the first step in its management. Determining the actiology for an individual patient can be difficult, but must be addressed by all clinicians before and during treatment. Part II of this article will discuss the management of dental erosion.

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