

UK National Clinical Guidelines in Paediatric Dentistry*

Introduction

The ninth National Clinical Guideline in Paediatric Dentistry is published here. The process of guideline production began in 1994, resulting in first publication in 1997. Each guideline has a nominated main author but the content is not a personal view; it represents rather a consensus of opinion of current best clinical practice. Each guideline has been circulated to all consultants in Paediatric Dentistry in the UK, to Council of BSPD, and to people of related specialities recognised to have expertise in the subject. The final version of the guideline is produced from a combination of this input and thorough review of published literature. The intention is to encourage improvement in clinical practice and to stimulate research and clinical audit in areas where scientific evidence is inadequate. Evidence underlying recommendations is scored according to the SIGN classification and guidelines should be read in this context. For those wishing for further detail, the process of guideline production in the UK is described in *International Journal of Paediatric Dentistry* 1997; **7**: 267–268.

Diagnosis and prevention of dental erosion in children

LINDA SHAW & ELIZABETH O'SULLIVAN

Introduction

Tooth wear is becoming more commonly recognised in both adults and children, with recent studies suggesting a prevalence of 98% in adults [1]. However, unacceptable pathological levels of wear possibly requiring treatment, are of the order of 6% to 10%, depending on age. The triad of erosion, attrition and abrasion has been known for many years, but the contribution of erosion to tooth wear may be increasing. Dental erosion is the irreversible loss of dental hard tissue due to a chemical process not involving bacteria, and not directly associated with mechanical or traumatic factors, or with dental caries. It is, however, fair to say that erosion usually co-exists with attrition and/or abrasion, but that one of these three factors is often more significant than the other two. Although there are no longitudinal studies on the prevalence of dental

erosion, the UK Child Dental Health Survey of 1993 showed that 52% of 5-year-olds had significant erosion [2].

The following guidelines are intended to be of assistance to practitioners in the diagnosis and prevention of erosion. This may be complex and require interdisciplinary long term treatment and liaison with physicians. Further details are given under 'Explanatory Notes'.

1. Aetiology

It is essential that the aetiology of erosion is identified where possible as the clinical management of the patient based on management of the aetiological factors before definitive preventive measures or restorative care are undertaken. The pattern of tooth tissue loss may give some clues as to the most important of the aetiological factors. All acids, whether from within the body or from external sources, are capable of de-mineralizing tooth tissue and therefore of causing erosion.

*Copyright for these guidelines is held by the Faculty of Dental Surgery, Royal College of Surgeons.

1.1. *Intrinsic acidic sources*

These are essentially of gastric acid origin and are often associated with significant palatal dental erosion. The development of palatal erosion, although not definitive, may lead to a suspicion that the cause is from intrinsic acid sources of gastric origin which enter the mouth from gastric reflux, vomiting or rumination.

1.1.1. *Gastro Oesophagal Reflux (Grade B)*. Gastro-oesophageal reflux is more common than previously thought [3,4,5] (see Table 1). It is known to cause erosion in susceptible patients and should always be considered a possible cause for erosion, especially in the presence of such symptoms as indigestion, heart burn or epigastric pain [4]. However, dental erosion may not be as great a problem in children with gastro-oesophageal reflux as it is in adults. It may be that refluxing is limited to the oesophagus [6,7].

1.1.2. *Vomiting (Grade B)*. Vomiting may be spontaneous or self induced and may be associated with a variety of medical problems (see Table 2 for the principal causes). Current estimates suggest that the prevalence of anorexia and bulimia nervosa is increasing.

1.1.3. *Rumination (Grade C)*. This is an uncommon condition in which people deliberately induce reflux of a small amount of their gastric contents and chew this before re-swallowing. Several case reports have been published [8].

1.2. *Extrinsic acid sources*

1.2.1. *Environmental (Grade C)*. Various sources of contact with acids as part of work or leisure activities have been reported [9].

Table 1. Principal Causes of Gastro-Oesophageal Reflux

Sphincter incompetence	Oesophagitis–alcohol Hiatus hernia Pregnancy Diet Drugs, e.g. Diazepam Neuromuscular, e.g. Cerebral Palsy
Increased Gastric Pressure	Obesity Pregnancy Ascites
Increased Gastric volume	After meals Obstruction Spasm

Table 2. Principal causes of Vomiting

Psychosomatic	Stress induced psychogenic vomiting Eating disorders Bulimia nervosa Anorexia nervosa
Metabolic and Endocrine	Uraemia Diabetes Pregnancy
Gastro-intestinal disorders	Peptic ulcer, gastritis Obstruction Nervous system disorders Encephalitis Cerebral palsy
Drug Induced	Primary, e.g. Cytotoxics Secondary to gastric irritation e.g. alcohol, aspirin, non steroidal anti-inflammatory drugs

1.2.2. *Dietary (Grade B)*. Much emphasis has been placed on healthy food and drink in recent years and there is now good evidence that dietary practices and habits have changed [10]. This is particularly the case in relation to the consumption of soft drinks [11] with a considerable increase in quantity and change in age distribution [11,12,13,14,15]. Some alcoholic drinks, such as dry wines and alcopops are also acidic. However, alcohol consumption is linked with gastro-oesophageal reflux and erosion may be from both intrinsic and extrinsic sources.

It is not just the total consumption of acidic dietary substances that is important but also the periodicity and relationship to tooth brushing practices (see Explanatory notes) [15,16,17].

1.2.3. *Medication and oral hygiene products (Grade C)*. A number of common medications, such as Vitamin C tablets, aspirin and iron preparations are very acidic as well as some proprietary mouth-washes [18,19].

1.2.4. *Lifestyle (Grade C)*. Changes in general lifestyle have increased exposure to extrinsic acidic sources [20,21].

1.3. *Predisposing factors*

Although the aetiology of erosion is acidic substances from a variety of sources, there are some individual factors that may predispose to erosion, or indeed be protective. For example, there is some evidence that salivary mucins can all modify the development of erosion. There have also been

reports of differing clearance rates from various parts of the mouth which may modify the distribution of erosion.

2. Management

Early diagnosis is important so that possible aetiological factors can be identified and preventive measures can be taken to halt further progression. Once the diagnosis has been made then it is essential to record accurately the severity and extent. This will enable any subsequent progression to be observed, and the effect of preventive measures assessed.

2.1 Recording erosion

The most useful diagnostic index for epidemiological studies is the Tooth Wear Index (T.W.I) of Smith and Knight [22] and variations on this. Study casts are essential and good clinical photographs helpful (Grade B).

Teeth should be thoroughly examined with particular attention paid to the pattern and appearance of the worn teeth. Erosion is probably the most important cause of tooth wear where contact does not occur between opposing surfaces and where abrasion is unlikely. All potential causes of dental erosion should be considered before any definitive diagnosis is made.

2.2 Intrinsic acid sources: Gastro-oesophageal reflux and vomiting

2.2.1. If there is evidence or suspicion of reflux or eating disorders then referral to the General Medical Practitioner and onwards to a Gastroenterologist or Psychiatrist may be required (see Explanatory notes) (Grade B).

2.2.2. Anti reflux medication may be helpful. This should be prescribed in liaison with the General Medical Practitioner and/or Gastroenterologist. A proton pump inhibitor may be useful.

2.2.3. Following reflux, rinsing the mouth with water and sodium bicarbonate helps to neutralise the oral environment (Grade C).

2.2.4. An occlusal guard containing sodium bicarbonate can be used at night if there is significant reflux at that time (see Explanatory Notes) (Grade C).

2.3. Dietary analysis (Grade B)

Record at least a 3 day detailed diet history, preferably 4 consecutive days, two of which should be at a weekend.

2.4. Dietary counselling (Grade B)

This must be tailored to the individual.

2.4.1. Limit acidic foods and drinks to mealtimes.

2.4.2. Reduce frequency [23].

2.4.3. Finish meals with alkaline foods [24].

2.4.4. Avoid acid foods and drinks last thing at night [14].

2.4.5. Avoid habits such as prolonged sipping and holding acidic beverages in the palatal vault and 'frothing' prior to swallowing.

2.4.6. Avoid tooth brushing after acidic substances.

2.4.7. Check the pH of medication, mouthwashes, etc.

2.4.8. Chewing gum has been shown to stimulate salivary flow but may also cause increased gastric secretions [25].

2.5. Desensitisation

2.5.1. Fluoride mouth rinses and varnish

2.5.2. High fluoride level toothpaste [26] (Caution in children under 6 years) (Grade B).

2.5.3. Low abrasive toothpaste

2.5.4. Sugar free chewing gum.

2.5.5. Dentine bonding agents

2.6. Restorative treatment

The indications for interventive treatment are:

2.6.1. Intractable sensitivity which cannot be controlled by the methods listed in 2.5. This is rare and is usually associated with bulimia nervosa. The most

effective treatment is to extirpate the pulps of the most severely affected teeth.

2.6.2. Appearance which is sufficiently changed to concern the patient significantly. Treatment is not justified on the grounds that the patient is concerned that the appearance might deteriorate. The longer treatment can be deferred the fewer replacements will be necessary in a normal lifetime.

2.6.3. Progression of uncontrolled erosion which cannot be prevented and where the teeth are becoming thinner/shorter to the point where crowns would become technically difficult if the erosion was allowed to continue.

Explanatory notes

1. Aetiology

1.1. *Intrinsic acidic sources*

1.1.1. *Gastro-Oesophageal Reflux (Grade A)*. Gastro-oesophageal reflux (GOR) is the uncontrolled movement of gastric juice through the lower oesophageal sphincter into the distal oesophagus. In some patients the reflux continues past the upper oesophageal sphincter to reach the mouth when it is called regurgitation. There is an important distinction between regurgitation and vomiting. Vomiting is the propulsion of the stomach contents, coordinated by a centre in the brain, and triggered by a well recognised pattern of physiological mechanisms [27]. Regurgitation, on the other hand, is the passive movement of gastric juice from the stomach into the mouth.

For most people GOR is a temporary phenomenon, which needs no recourse to medical treatment, for others the symptoms persist, and pain relief is necessary. The distinguishing feature between physiological or transient GOR and pathological GOR is that the response to the refluxate is both prolonged and excessive. Gastro-oesophageal reflux is involved in the pathophysiology of anorexia and bulimia nervosa, rumination and chronic alcoholism. It is common in the Western World and has been stated to affect 7% of the adult population on a daily basis and one third every few days [3].

Symptoms are not reliable indicators to the presence or absence of GOR. Patients with long standing pathological GOR are described as 'silent

refluxers' and may be symptom free despite continuing to reflux. These patients can remain undiagnosed. One study observed that nearly 25% of patients presenting with extensive palatal erosion had pathological GOR diagnosed by standard criteria but did not have any symptoms of reflux [4]. In silent refluxers dental erosion may be the only clinical sign that pathological GOR is occurring. Conversely, there are patients with severe symptoms of reflux, who have normal pH studies, who tend to be very sensitive to small amounts of acid in the oesophagus.

Signs and symptoms associated with reflux are heartburn, retrosternal discomfort, epigastric pain, and dysphagia (including odynophagia—pain on swallowing, particularly of hot liquids). Other symptoms such as chronic cough, globus (a feeling of a 'lump in the throat'), hoarseness, chronic laryngitis and asthma may be present and could indicate that the refluxate has entered the upper respiratory passages and investigations should be considered if they are particularly severe [28].

Hiatus hernia is also associated with gastrooesophageal reflux. It has been shown that raised intra abdominal pressure plays an important part in the genesis of hiatus hernia. This occurs particularly in pregnancy, but also in athletes such as weightlifters, body builders and long distance runners [29]. Although there have been recent concerns expressed linking sports drinks to the development of erosion in athletes [30,31], it may be that gastric regurgitation is a significant aetiological factor.

It has been found that neurologically impaired children have significantly higher levels of gastro-oesophageal reflux than 'normal' children. Indeed, over 70% of children with cerebral palsy have been found to have abnormal reflux activity [5].

1.1.1.1. *Factors known to provoke Gastro-oesophageal reflux*. Excessive food or alcohol consumption can cause temporary symptomatic reflux especially if taken late at night or shortly before sleep [32,33]. In patients suffering from pathological GOR these symptoms become prolonged and uncomfortable. Other dietary components known to provoke GOR are chocolate, coffee, peppermint, spicy and fatty foods and carbonated drinks [34]. Heartburn may be worse at night in some patients and can interrupt sleep patterns, although GOR most commonly occurs in patients during the day or when upright [4].

1.1.1.2. *Indications for Gastroenterologist or specialist referral.*

- if the signs and symptoms associated with reflux (as above) are interfering with the daily quality of life of the patient.
- if there have been previous investigations for gastro-oesophageal reflux and the tests were either inconclusive or borderline and the erosion is noted to be particularly severe.
- if, after the elimination of dietary factors and a period of review and monitoring, the erosion continues.
- when there is no other obvious cause of severe erosion.
- in cases of severe erosion of palatal surfaces of the upper incisor teeth or posterior teeth with exposure of secondary dentine or pulp and loss of form and function of the teeth.

1.1.1.3. *Process for investigation.* Clinical histories should be taken in a quiet surgery and in confidence. Patients may be secretive about their symptoms, especially if there is a tendency towards an eating disorder or alcohol abuse. It may be helpful to repeat the history at a subsequent appointment when the patient may have had sufficient time to reflect upon their symptoms.

If the patient has been referred to a gastroenterologist then manometry and oesophageal pH measurement may be undertaken. Manometry measures the pressure around the lower oesophageal sphincter and allows accurate and reproducible location of the oesophageal pH electrodes. Manometry is performed just before the pH measurement and can last for up to one hour. A pressure of less than 6mmHg around the lower oesophageal sphincter is considered pathological according to internationally recognised guidelines [35,36] (Grade A).

The motility of the oesophagus is a measure of its capacity to clear refluxed acid. It is measured by monitoring the response of the body of the oesophagus to a series of water swallows and assessing whether peristalsis has occurred. If non-peristaltic movement of water occurs for greater than 25% of the total number of swallows then a pathological abnormality exists [35,36,37].

Gastro-oesophageal reflux is diagnosed by 24 hour ambulatory pH measurement. This remains the gold standard for the investigation of reflux disease. The position of the distal pH electrode is standardised throughout the world at 5cm above the lower oesophageal sphincter, the position of

which is determined by manometry. The catheter may contain two electrodes; one situated at the tip and the other 15 cm above the first, so that pH can also be recorded near to the proximal oesophageal sphincter. The catheter is connected to an ambulatory recorder which is worn around the patient's waist and data recorded for 24 hours. The data from the recorder is downloaded to a computer which analyses the pH and the results are compared to internationally recognised norms [32,33] (Grade A).

Gastroenterologists calculate the percentage time that the oesophageal pH remains below pH 4 in the distal oesophagus. If the percentage time in the distal oesophagus is below pH 4 for greater than 5.78% of the total time of the investigation the acid levels are considered pathological [3,4]. This time can be divided into the time spent upright (upright time) and the time spent lying down (supine time). Pathological pH levels are also recognised for these periods, for supine time it is 3% and upright 8% of the time in each position.

1.1.1.4. *Medical management of Gastric reflux.*

Patients with symptoms and pathological GOR diagnosed by 24 hour pH monitoring may be offered medication such as a proton pump inhibitor (e.g. Omeprazole) or H₂ blocker to increase gastric pH to potentially reduce the erosion. However, the most likely reason for medication would be to reduce symptoms of reflux rather than to control dental erosion. The responsibility for prescription is with the patient's medical practitioner but the dental surgeon may wish to be involved with the decision (Grade C).

Patients without symptoms of reflux and with pathological GOR, diagnosed by 24 hour pH measurement, may be offered medication to control the dental erosion but a degree of reluctance is possible. There is no scientific research to directly support the control of dental erosion with anti-reflux medication and research is needed to validate this hypothesis.

Patients without symptoms and normal results from 24 hour pH measurement would not normally be offered medication. However, reflux disease is known to be a cyclical disorder with periods of activity and inactivity and it is possible that the disease was quiescent on the day of the test. Therefore, where doubt persists as to the cause of erosion, a repeat test could be considered after a period of review which shows that the erosion is continuing.

1.1.1.5. *Dental management of Gastroesophageal reflux.*

Dental erosion, like GOR, is cyclical in nature with periods of activity and inactivity. The management of erosion should include strenuous efforts to make a diagnosis and prevent further damage by preventive advice. Monitoring of dental erosion is clinically achieved by assessing serial study casts of the patients teeth, sometimes continuing for many years. Where the condition is particularly aggressive, investigation of GOR in a asymptomatic patient would be indicated.

Restorative treatment should be considered in patients where the erosion is shown to be rapidly progressive, as seen from changes on the teeth or study casts (see Explanatory Notes 6).

1.1.2. *Vomiting.* Vomiting may be spontaneous or self induced and may be associated with a variety of medical problems (see Table 2 for the principal causes). More emphasis has recently been placed on the eating disorders of anorexia and bulimia nervosa (the self-induced vomiting in bulimia nervosa may cause extensive erosion of the palatal aspects of upper incisors and molar teeth).

Current estimates suggest that the prevalence of these disorders is increasing. Bulimia nervosa affects between 1 and 4% of white females between 18 and 30 years in the USA. Most bulimics either approximate to a normal weight range for their height and age or are grossly overweight. Anorexia is less common, affecting between 0.5 and 1% of white females of 11–18 years of age. Most anorexics are well below their ideal body weight, this is achieved by starving and abstinence and/or vomiting.

Patients with eating disorders frequently brush their teeth after each vomiting episode—which may increase abrasion. Although it is often relatively easy for dental personnel to recognise these disorders, initiation of medical help is a sensitive undertaking.

Other conditions causing vomiting may also occur in conjunction with gastric reflux to compound the problem. Alcohol will cause both reflux and vomiting and additionally may itself be acidic giving both intrinsic and extrinsic acidic challenge to the teeth. Obviously all these conditions need to be operant over a lengthy period of time to potentially cause erosive problems.

1.2 *Extrinsic acidic sources*

1.2.1. *Environmental.* Extrinsic sources include

environmental causes such as contact with acids as part of work or leisure activities. Battery, dynamite and fertilizer factory workers, laboratory technicians, professional wine tasters and competitive swimmers have all been reported as having significant dental erosion [9].

1.2.2. *Dietary.* Much emphasis has been placed on healthy food and drink in recent years and dietary habits are apparently changing.

Consumption of soft drinks has increased dramatically since the 1950s when they were first associated with tooth erosion in children. In 1950, 100 million litres of soft drinks were sold in the UK: this had increased sevenfold by 1990. This trend shows no indication of levelling off, indeed, the consumption in 1991 of 151 litres per head of population in the UK is still a long way behind the figure in the USA (263 litres in the same year) [11].

Mean consumption figures can hide important facts. Soft drink intake is much higher in younger age groups: soft drinks have been reported to provide as much as one-fifth of the added sugars in the diet of 11–12-year-old children [12] and 42% of fruit drinks are consumed by children aged between 2 and 9 years. Frequency, rather than total, intake may be critical in the erosive process. It is also the titratable acidity of the drinks that is more important in causing erosion than just the actual pH.

Erosion may be particularly harmful to infants if drinks are taken over a prolonged period from a feeding bottle used as a comforter. There have been reports of extreme destruction resulting from abuse of fruit juices [13].

It is apparent therefore, that those most likely to show the affects of erosion in the dental tissues from excessive fruit drink intake are children. However, young people are taking the habit of having soft drinks with them into adult life rather than drinking tea or coffee. In 1995 it was projected that 12–25-year-olds would be drinking 50% more soft drinks by the year 2000.

A significant association between soft drink consumption and dental erosion was shown in an in-depth study of 101 children, mean age 8–9 years involving clinical examinations, dietary analysis and salivary flow and pH monitoring [14]. There was also an association between bed-time consumption of fruit based drinks and the prevalence of dental erosion.

A survey of U.K pre-school children [15], involving a large diet and nutrition enquiry as well as oral

health, also showed a weak relationship between the frequent consumption of sweetened drinks and carbonated beverages and dental erosion. It also confirmed the bed-time drink association.

It has already been mentioned that alcohol consumption is related to gastroesophageal reflux, but it may also be acidic itself. Dental erosion has been noted in patients with chronic alcoholism [38].

Another important consideration is that dental erosion is frequently associated with individuals with high standards of oral hygiene [16] and it is likely that the bed-time drink may have occurred just before bed-time tooth brushing. The influence of oral hygiene practices is a significant complicating feature in the distribution of erosion. The effects of acid, whether from the diet or from the stomach, on demineralising the enamel and dentine may be compounded and modified by tooth brushing. If demineralized tissue is brushed [17], even with a brush and water, abrasion accelerates until the demineralized layers are removed. The effects of repeatedly consuming acidic foods and drinks followed by tooth brushing are probably very important as far as erosion is concerned.

Erosion on its own causes much greater loss of tooth substance than abrasion alone, but the two in combination produce more destruction than can be accounted for by simply summing the effects [17].

Although there is increasing evidence of the role of soft drinks in the development of erosion, it is not just drinks that contain acid. There are also other potential dietary sources such as fresh fruit, pickles and sauces, lactovegetarian foods and yoghurt [14].

1.2.3. *Medication and oral hygiene products.* A number of common medications including vitamin C tablets and iron preparations are also very acidic.

Vitamin C has been produced in a chewable tablet form which has been associated with extensive destruction when used to excess [18].

Some mouthwashes and saliva substitutes have also been found to be very acidic, but now that the problem has been identified the mouthwashes have largely been reformulated [19]. Saliva substitutes with a low pH should be avoided in patients with natural teeth although there is little scientific evidence to support or refute that they have an influence on erosion.

1.2.4. *Lifestyle.* It is not just the total exposure to acidic substances that appears to have increased in

recent years; there have also been changes in habits and general lifestyle. Undoubtedly there has been increased emphasis on a healthy diet and this involves a necessary increase in fruit and vegetable consumption. More people are becoming vegetarians, and this tends to be a more acidic diet.

The frequency of intake of food is changing with greater numbers of snacks being consumed and a reduction in the number of meals eaten at home. This is commonly known as 'grazing'. A habit of 'frothing' up carbonated beverages in the mouth has also developed along with constant sipping from canned drinks.

Encouragement to take regular exercise plainly is of benefit to general health but excessive and frequent consumption of acidic sports drinks in some athletes has been shown to be a problem [20,30,31].

Conversely, there are unhealthy lifestyles that may be implicated in dental erosion. The use of the drug 'Ecstasy' (3-4. Methylenedioxy-methamphetamine) reduces salivary flow. The dry mouth combined with dehydration from vigorous exercise and excessive consumption of low pH drinks has also been linked to dental erosion [21].

2. Management

2.1. *Recording erosion*

The most useful diagnostic index is the TWI of Smith and Knight [22]. However, it is not sufficiently sensitive to be used for recording small changes in erosion in individual patients. For this good clinical photographs are helpful and study casts are essential. These should be accurate and produced in stone. When the patient returns for review a localised silicone rubber index is taken of the study cast in the areas of most concern. This can be cut through with a sharp scalpel over the area in question the transferred to be fit onto the patient. If there is any gap between the silicone impression and the surface of the tooth then there has been further tooth wear. It is possible to use Scanning Electron Microscopy from replicas of the teeth but this is really a research procedure and not very practical clinically.

2.2. *Intrinsic acid sources: Gastro-oesophageal Reflux and Vomiting (Grade B)*

If there is evidence, or suspicion, of gastric reflux or

vomiting activity then contact should be made with the patient's General Medical Practitioner, outlining the problem. Referral to a Gastroenterologist for investigation and treatment may be desirable [4], and those suffering from eating disorders usually need psychiatric help (see Explanatory notes Aetiology, section 1.1.).

If acid from the stomach is repeatedly entering the patient's mouth they should be advised to rinse out with water or sodium bicarbonate and avoid teeth cleaning at this time.

A hard acrylic occlusal guard may be used at night if there is evidence of parafunctional activity causing attrition combined with reflux activity at night. A small amount of sodium bicarbonate can be placed in the guard. Evidence for the effectiveness of this is extremely limited and great caution should be exercised in the use of this device in case the buffer should leak out, and regurgitated acid become trapped beneath the guard overlying the teeth.

2.3. Dietary analysis

A current, detailed diet history is vital. Also, a retrospective long term dietary history is sometimes revealing. For example, people living in hot countries may have increased fruit juice consumption or there may have been previous episodes of alcohol abuse.

A current dietary history can be obtained by getting the patient to keep an accurate 3 day diet diary but stressing that the time over which consumption occurred should also be recorded. Specific questioning about such habits as sipping, swishing, frothing and holding drinks in the mouth should be undertaken. Erosion caused by an excess of acidic food and drink commonly affects the labial and palatal surfaces of upper anterior teeth. These surfaces (sites where food and drink take longest to clear) are particularly at risk, whereas the lower incisor region (which is subjected to increased salivary washing and buffering) clears relatively quickly [39]. Factors such as how the drink is consumed may influence erosion: drinks taken from a glass take longer to clear from the mouth than when a straw or a child's feeder cup is used (Grade B).

Apart from food and drink, any other substances that pass through the mouth should be inquired about. Medication and oral hygiene products and practices are particularly relevant [18,19].

2.4. Dietary counselling

Counselling can only be given after the diet has been thoroughly assessed. It must be tailored to the individual on a positive basis to maximise compliance (Grade B).

Specific points to emphasise are the limitation of acidic foods and drinks to mealtimes. This is the time of maximum salivary flow and increased buffering capacity.

Although there is huge individual variation in these parameters it is really not practicable to increase salivary flow and buffering capacity by drug control. It has been suggested, and there is some scientific evidence for this [25], that the use of chewing gum is helpful in increasing salivary flow and encouraging enamel remineralisation following early caries. Finishing a meal with something to neutralise any acids, for example with a little cheese or milk, is also useful [24].

Along with restriction of acidic substances to meal times there should be advice to reduce the total amount and frequency of consumption. It has been shown that there is a rapid intra-oral drop in pH at the surface of the enamel following an acidic challenge and this comes back to resting pH levels only slowly [23]. Therefore, for example, frequent sips of an acidic drink will maintain the pH at a low level over a long period of time. The majority of the scientific evidence in support of this relates to caries rather than erosion.

Patients should be advised to avoid acidic food and drink between meals and particularly last thing at night. There is a marked circadian rhythm to salivary flow and the flow rate is negligible at night during sleep.

Avoidance of tooth cleaning immediately after an acid challenge should also be advised [17]. It would be more sensible to clean the teeth with a fluoride toothpaste before that meal or to delay tooth brushing.

2.5. Desensitization

Patients with significant erosion and dentine exposure may complain of tooth sensitivity although this is surprisingly uncommon. This can be a serious problem to control. The use of fluoride mouth rinses and fluoride varnishes is helpful but they must be used frequently and regularly. A high fluoride content toothpaste, 1450 p.p.m, is also sensible as

long as it is not also highly abrasive [26]. The use of a sugar free chewing gum has already been mentioned (Grade B).

In patients with refractory sensitivity, dentine bonding agents can help to alleviate the symptoms. Although glass ionomer restorations, with high levels of leachable fluoride, may seem very applicable, their acid solubility is also high and retention may therefore be a problem. Compomers may be a useful alternative. In extreme cases pulp extirpation is necessary.

2.6. Restorative Treatment

Without doubt it is important to identify the problems first and try to address the aetiological factors before proceeding with complex restorative techniques. Covering eroded teeth without addressing the basic cause merely disguises the situation, but leaving surfaces unrestored may lead to further erosion. It may be impossible to completely eliminate some aetiological factors and withholding active therapy in some patients may lead to more complex restorative treatment having to be carried out later.

Preventive programmes must remain the cornerstone of management of dental erosion.

References

- Smith BGN, Robb ND. The prevalence of tooth wear in 1007 dental patients. *Journal of Oral Rehabilitation* 1996; **23**: 232–239.
- O'Brien M. Children's Dental Health in the United Kingdom 1993 Office of Population Censuses and Surveys. HMSO London. 1994.
- Colin-Jones DG. Gastro-oesophageal reflux disease. *Prescribers Journal* 1996; **36.2**: 66–72.
- Bartlett DW, Evans DF, Anggiansah A, Smith BGN. A study of the association between gastro-oesophageal reflux and palatal dental erosion. *Br. Dent. J.* 1996; **181**: 125–132.
- Reyes AL, Cash AJ, Green SH, Booth IW. Gastro-oesophageal reflux in children with cerebral palsy. *Child: Care, Health and Development* 1993; **19**: 109–118.
- O'Sullivan EA, Curzon ME, Roberts GJ, Milla PJ, Stringer MD. Gastroesophageal reflux in children and its relationship to erosion of primary and permanent teeth. *European Journal of Oral Sciences* 1998; **106(3)**: 765–769.
- Shaw L, Weatherill S, Smith AJ. Tooth wear in children: an investigation of etiological factors in children with cerebral palsy and gastroesophageal reflux. *ASDC Journal of Dentistry for Children* 1998 **65**: 439, 484–486.
- Gilmour AG, Becket HA. The voluntary reflux phenomenon. *Br. Dent. J* 1993; **175**: 368–372.
- Centerwall BS, Armstrong CW, Funkhouser LS, Elzay RP. Erosion of dental enamel among competitive swimmers at a gas-chlorinated swimming pool. *American Journal of Epidemiology* 1986; **123**: 641–647.
- Gofton L, Ness M. Twin trends: health and convenience in food change or who killed the lazy housewife? *Br. Food J.* 1992; **93**: 17–23.
- British Soft Drinks Association. Report of Seminar in Heidelberg. Factsheet number 1991; 9–7.91.
- Rugg-Gunn AJ, Lennon MA, Brown JG. Sugar consumption in the United Kingdom. *British Dent Journal* 1987; **167**: 339–364.
- Smith AJ, Shaw L. Baby fruit juice and tooth erosion. *British Dent Journal* 1987; **162**: 65–67.
- 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. *International Journal of Paediatric Dentistry* 1994; **4**: 151–157.
- Hinds K, Gregory JR. National diet and nutrition survey: children aged 1½ to 4½ years Volume 2: Report of the Dental Survey Office of Population Consensus and Surveys. HMSO London. 1995.
- Millward A, Shaw L, Smith A. Dental erosion in four year old children from differing socio-economic backgrounds. *Journal of Dent Child* 1994; **61**: 263–266.
- Davis WB, Winter PB. The effect of abrasion on enamel and dentine after exposure to dietary acid. *British Dent Journal* 1980; **148**: 253–256.
- Giunta JL. Dental erosion resulting from chewable vitamin C tablets. *J. Am. Dent. Assoc.* 1983; **107**: 253–256.
- Bhatti SA, Walsh TF, Douglas CWI. Ethanol and pH levels of proprietary mouth rinses. *Comm. Dent. Health.* 1994; **11**: 71–74.
- Sorvari R. Effects of various sport drink modifications on dental caries and erosion in rats with controlled eating and drinking pattern. *Proc. Fimm. Dent. Soc.* 1989; **85**: 13–20.
- Duxbury AJ. Ecstasy – Dental implications. *Br. Dent. J.* 1993; **175**: 38.
- Smith BGN, Knight JK. An index for measuring the wear of teeth. *Br. Dent. J.* 1984; **156**: 435–438.
- Millward A, Shaw L, Harrington E, Smith AJ. Continuous monitoring of salivary flow rate and pH at the surface of the dentition following consumption of acidic beverages. *Caries Research* 1997; **31**: 44–49.
- Gedalia I, Ionat-Bendat D, Ben-Mosheh S, Shapira L. Tooth enamel softening with a cola type drink and re-hardening with hard cheese or stimulated saliva in situ. *Journal of Oral Rehab* 1991; **18**: 501–506.
- Jenkins GN, Edgar WM. The effect of daily chewing gum on salivary flow rates in man. *Journal of Dent. Research* 1989; **68**: 786–790.
- Bartlett DW, Smith BGN, Wilson RF. Comparison of the effect of fluoride and non-fluoride toothpaste on tooth wear in vitro and the influence of enamel fluoride concentration and hardness of enamel. *Br. Dent. J.* 1994; **176**: 346–348.
- Bartlett DW, Anggiansah A, Owen WJ, Evans DF, Smith BGN. Dental erosion: a presenting feature of gastro-oesophageal reflux disease. *European J. Gastroenterology & Hepatology* 1994; **6**: 895–900.
- Weiner GJ, Koufman JA, Wu WC, Cooper JB, Richter JE, Castell DO. Chronic hoarseness secondary to gastro-oesophageal reflux disease: documentation within 24-hour ambulatory pH monitoring. *Am J. Gastroenterol.* 1989; **84**: 1503–1508.
- Dickerman KD, MacConathy WJ, Smith AB. Can pressure overload cause sliding hiatal hernia? A case report and review of the literature. *J. Clinical Gasroenterology* 1989; **25**: 352–353.

- 30 Millosevic A. Sports drinks hazard to teeth. *Br. J. Sports Medicine* 1997; **31**: 28–30.
- 31 Milosevic A, Kelly MK, McLean AN. Sports supplement drinks and dental health in competitive swimmers and cyclists. *Br. Dent. J.* 1997; **182**: 303–308.
- 32 Kjellen G, Tibbling L. Influence of body position, dry and water swallows, smoking and alcohol on esophageal acid clearing. *Scand. J. Gastroenterol.* 1978; **13**: 283–288.
- 33 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.
- 34 Kitchen LI, Castell DO. Rationale and efficacy of conservative therapy for gastroesophageal reflux disease. *Arch. Intern. Med.* 1991; **151**: 448–454..
- 35 Johnson LF, DeMeester TR. Twenty four hour pH monitoring of the distal oesophagus. *Am. J. Gastroenterol.* 1974; **62**: 325–332.
- 36 Richter JE, Wu WC, Johns DN, Blackwell JN, Nelson JL3, Castell JA, Castell DO. Oesophageal manometry in 95 healthy adult volunteers: variability of pressures with age and frequency of ‘abnormal’ contractions. *Digestive Diseases & Sciences* 1987; **32**: 583–592.
- 37 Weiner GJ, Morgan TM, Copper JB, Castell DO, Sinclair JW, Richter JE. Ambulatory 24-hour esophageal pH monitoring. *Digestive Diseases & Sciences* 1998; **33**: 1127–1133.
- 38 Robb ND, Smith BGN. Dental erosion in patients with chronic alcoholism. *J. Dent.* 1989; **17**: 219–221.
- 39 Smith AJ, Shaw L. Comparison of rates of clearance of glucose from various sites following drinking with a glass, feeder cup and straw. *Medicine Science Research* 1993; **21**: 617–619.