Royal College of Surgeons of England

ADVANCING SURGICAL CARE

Clinical guidelines for dental erosion

Diagnosis, prevention and management of dental erosion

2021

Contents

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Introduction

Toothwear is recognised as a major dental problem in both children and adults. The triad of erosion, attrition and abrasion has been known for many years, and the contribution of erosion to toothwear is increasing.[1] Dental erosion is the irreversible softening and subsequent loss of dental hard tissue due to a chemical process of acid dissolution but not involving bacterial plaque acid, and not directly associated with mechanical or traumatic factors, or with dental caries. Attrition may be defined as direct tooth-to-tooth contact wear while external foreign agents moving across and contacting the tooth surface results in abrasion. Erosion usually coexists with attrition and/or abrasion but one of these factors may be more significant than the others, making differential diagnosis difficult.

Erosive wear is now deemed a common global phenomenon of developed countries,[2] and its financial burden on patients and public health services is significant.[3] More importantly, the morbidity associated with erosive wear may be high, and can include pain/sensitivity, dissatisfaction with aesthetics and reduced function. While the ultimate causative factors of dental erosion are non-bacterial acids, it is a multifactorial condition, and the progression of the lesions are driven by a complex interplay between nutritional and patient related factors.

Owing to its progressive nature, preventative measures implemented (ideally, in the early stages of erosive wear) can be effective in reducing the need for dental intervention and symptoms experienced by patients. Operative measures will still be necessary for those with advanced erosion progression in order to eliminate pain, and to restore function and aesthetics. Additionally, interventive treatment may be warranted to preserve the structural integrity of the reduced and remaining tooth structure, and to protect against further tissue loss.

This guideline aims to assist the dentist to diagnose, prevent and manage erosion in children, adolescents and adults. This may be complex, and can require interdisciplinary long-term management and liaison with medical colleagues.

Intended audience

This guideline provides information for the whole dental team who are involved in the diagnosis, prevention and management of dental erosion. It would also be useful for other healthcare providers who give dietary advice to patients. It is intended that by using the guideline informed and evidence-based discussions can be carried out with patients.

Statement of conflict of interest

The Faculty of Dental Surgery is funded by its fellows and members, and no contributors or reviewers were paid for their work on this guideline.

Aims and Objectives

The aims and objectives of this guideline are to describe and explain the diagnosis, prevention and management of dental erosion in children and adults using evidenced based literature, so that clear information and discussions can be carried out with patients and carers.

Development and evidence base

This guideline was developed from existing guidelines by a small working group with members from the specialties of Paediatric Dentistry and Restorative Dentistry and the final draft was approved by each specialist societies clinical effectiveness group. At the beginning of development of the guideline the evidence base was established by each member carrying out a rapid review of the literature to optimise the use of the most current and relevant literature. The guideline development group employed a modified Delphi methodology (the guideline draft is circulated to the group, feedback is collated, before another draft is produced and circulated again for appraisal. In order for a recommendation to be included in the final guideline at least 90% of the working group were required to provide approval. Other stakeholders were then involved including the Dental Faculty RCS England Guideline Committee and the Delivering Better Oral Health Working Group. Final approval was sought from the British Society of Paediatric Dentistry and the British Society of Restorative Dentistry.

Prevalence

Epidemiological studies over the past 20 years both in the UK and abroad have highlighted the prevalence for dental erosion.[2,4,5] In recent reviews on the global prevalence of erosive wear, it has been deemed a common phenomenon of the general population of developed countries, with a mean prevalence in deciduous teeth of between 30% and 50%, and in permanent teeth of between 20% and 45%.[2,6] Such wide variation in global prevalence rates is a reflection of the heterogeneity of erosive toothwear studies. This heterogeneity relates to factors such as differences in the toothwear indices used, sample size, study populations chosen and lack of standardisation in the quantification of dimensions of tissue loss.

Prevalence data from cross-sectional UK studies indicate that dental erosion increases between different age cohorts of young people over time (Table 1).[7–20] Prevalence of overall toothwear in dentate English adults increased from 66% to 76% between 1998 and 2008; moderate wear that has exposed a large area of dentine on any surface also increased from 11% (1998) to 15% (2009).[21] This increase, however, is not uniform across age groups, with the greatest increases in adults seen in the younger age groups. A systematic review reportedthat the prevalence of toothwear in adults increases with age.[22] While data could not indicate whether this increase simply reflected the aging process, it is probable that the increase in moderate toothwear is small in surveys conducted in England. Wear in younger adults is likely to be important clinically and is suggestive of more rapid toothwear attributable to factors other than age.

Sex differences in the prevalence of toothwear are not clearly confirmed in the literature. Some studies have observed no association with sex, with others reporting a higher prevalence in one of the sexes. Overall, where a sex association has been found, the majority of studies favour an increased prevalence seen in male patients.[2]

Primary teeth have been shown to be more prone to erosive toothwear, which occurs more rapidly than in the permanent dentition,[23] with the prevalence and severity of erosive toothwear increasing in the preschool population.[24] Those patients who exhibit signs of erosive toothwear in the primary dentition have been shown to have an increased prevalence of erosion in the permanent dentition.[25]

Table 1 Prevalence studies

Author	Year of publication*	Age	Sample size	% with dentine exposed	% with palatal / occlusal / labial dentine exposed	Teeth	Surfaces
O'Brien[7]	1994	5 years 12/14 years	Total 17,061		24% 2%	24% 2%	U1" incisors U2" incisors
Millward[8]	1994	4 years	178	48%		All 1" teeth	All
Milosevic[9]	1994	14 years	1,035	30%	8%	8%	All 2" teeth
Jones[10]	1995	3 years	135	17%		U1" incisors	Labial/palatal
Hinds[11]	1995	1½–4½ years	1,496		8%	U1" incisors	Labial/palatal
Smith[12]	1996	<26–>65 years	1,007	26% with extensive toothwear		All 2" teeth	All
Bartlett[13]	1998	11–14 years	210		2%	All 2" teeth	All
Williams[14]	1999	14 years	525	11%	1%	U2" incisors	Labial/palatal
Walker[15]	2000	4–6 years 7–10 years 11–14 years 15–18 years	363 500 518 345		19% 18% 3% 5%	U1" or 2" incisors First 1" or 2" molars	Labial/palatal Occlusal
Al-Dlaigan[16]	2001	14 years	418	52%		All 2" teeth	All
Bardsley[17]	2004	14 years	2,351	53%	10%	All 12 anterior teeth First molars	Labial, incisal, palatal/lingual Occlusal
Chadwick[18]	2004	5 years 12/15 years	Total 12,698		22%	U1" incisors U2" incisors	Labial/palata Labial/palata
Dugmore[19]	2004	12 years	1,753	3%		Incisors and first molars	Labial/palatal Buccal/occlusal/ lingual
Pitts[2]	2015	5 years 12 years 12 years 15 years 15 year	2,549 2,532 2,532 2,418 2,418		16%/4% 2%/– 1% 4%/1% 3%	U1" incisors U2" incisors First 2" molars U2" incisors First 2" molars	Lingual/buccal Lingual/buccal Occlusal Lingual/buccal Occlusal

*not year survey conducted

Aetiology

Dental erosion involves the softening of the tooth surface by acidic substances of intrinsic or extrinsic origin, or a combination of both.[26] Repeated and prolonged contacts with acidic substances not only cause softening of the tooth surface but can also lead to overall loss of tooth structure.[6]

The softened tooth structure is further left susceptible to mechanical impact.[27] Erosion, therefore, rarely has an isolated effect on toothwear but interacts with other wear mechanisms (abrasion, attrition) to potentiate their effect.[28,29] The combination of chemical-mechanical tooth surface loss caused by the processes of dental erosion followed by abrasive and/or attritive wear is referred to as erosive toothwear.[6,26] This term highlights the co-action between the three commonly recognised toothwear processes.

As stated, the principal aetiological agents of erosive wear are intrinsic and extrinsic non-bacterial acids. Ideally, the source of the acidic substances should be identified prior to patient management. This is not always possible because of the difficulty in gaining an accurate and contemporaneous relevant history or because the patient may withhold important information regarding lifestyle or behaviour. Nevertheless, the identification and reduction of risk factors will improve the success of management. It is consequently important to question each patient about their medical history, medication, dietary content and habits, lifestyle and occupation.

The dietary intake of acidic foodstuffs may be quite high in certain cases, and careful questioning on the intake of specific items of food and drink is necessary (Table 2). Dietary associations with erosion are present but weak. Future research may establish causal relationships and the influence of co-factors in the erosive process. In vitro studies have identified dietary factors with erosive potential but further research is needed to fully understand causal relationships and co-factors such as risky behaviours that increase the risk of erosion.

Table 2 Dietary items with erosive potential

Beverages
• Carbonated or fizzy drinks excluding ordinary unflavoured sparkling water
• Pure fruit juice, smoothies and cordials
• Certain alcoholic drinks (eg cider, white wine)
• Herbal teas
Foods
• Fruits (especially citrus, grapes, sour apples)
• Sauces (eg ketchup, brown sauce)
• Snack foods (eg salt and vinegar crisps)
• Vinegar and pickled foods

In vitro studies show promise with respect to modification of drinks to reduce erosive potential.[30,31] Continuing acid exposure not only results in a clinically detectable defect but also softens the tooth surface, making it more prone to mechanical impact. The primary causative factor is not always apparent; however, it is clear, for example, that while enamel is scarcely abraded by normal toothbrushing, it is rendered more susceptible to wear following an acid challenge.[27]

Intrinsic acidic sources

The singular source of intrinsic acid related to dental erosion is gastric juice. Gastric acid may enter the mouth secondary to gastro-oesophageal reflux, vomiting or rumination and can lead to significant palatal erosion.

Gastro-oesophageal reflux disease

Reflux is the passive or effortless movement of regurgitated acid into the mouth and when this occurs regularly over a prolonged period, it is referred to as gastro-oesophageal reflux disease (GORD). It is this more persistent form of reflux that may lead to pathological dental erosion. GORD is considered relatively prevalent and in a 2014 systematic review, between 8.8% and 25.9% of adults in Europe were reported to be affected by this condition.[32] The causes of GORD are summarised in Table 3.

Table 3 Principal causes of gastro-oesophageal reflux

Sphincter incompetence
• Oesophagitis
• Alcohol
• Hiatus hernia
• Pregnancy
• Diet (eg spicy/fatty foods)
• Drugs (eg diazepam)
• Neuromuscular (eg cerebral palsy)
Increased gastric pressure
• Obesity
• Pregnancy
• Ascites
Increased gastric volume
• After meals
• Obstruction
• Spasm

Signs and symptoms associated with reflux comprise heartburn, retrosternal discomfort, epigastric pain and hoarseness or asthma-like symptoms although symptoms are not reliable indicators of the presence or absence of GORD. Patients may be symptom free despite continuation of reflux. These patients are described as 'silent refluxers' and can remain undiagnosed.

GORD is known to cause dental erosion and should always be considered a possible cause in the presence of indigestion, heartburn or epigastric pain.[33,34] Extra-oesophageal symptoms (including dental erosion, chronic cough, asthma and laryngitis) have significant correlations with GORD.[35] Furthermore, reflux disease patients with frequent respiratory symptoms appear to have a greater prevalence of dental erosion than those without reflux associated respiratory disorders.[36] Clinicians should be mindful, however, that nearly 25% of adult patients presenting with extensive palatal erosion had pathological GORD diagnosed by standard criteria but did not have any symptoms of reflux.[33] In silent reflux, dental erosion may therefore be the only clinical sign that reflux is occurring.

Dental erosion in relation to GORD is less of a problem in children. This may be due to a shorter history of GORD or because refluxing is limited to the oesophagus.[37] The exception is seen in neurologically impaired children, where significantly higher levels of gastric reflux are seen compared with healthy children, with over 70% of children with cerebral palsy having abnormal reflux activity.[38–40]

Vomiting

Vomiting involves a host of physiological events, coordinated in the medulla, resulting in the forceful propulsion of stomach and upper intestinal contents towards the mouth. Vomiting may be spontaneous or self-induced and is often associated with an underlying medical condition. Spontaneous or self-induced vomiting needs to be persistent for dental erosion to occur, and the medical conditions associated most frequently with persistent vomiting include eating disorders, pregnancy, alcoholism and cyclic vomiting syndrome.[41]

Eating disorders can be divided into anorexia nervosa, bulimia nervosa, self-induced vomiting and eating disorders not otherwise specified.[2] The prevalence of eating disorders in England in those aged 16 years and older is estimated at 7.8% and the disorder typically develops in younger females in their late teens to mid-twenties.[42] Traditionally, it has been considered that teenage girls are particularly prone to abnormal eating behaviours; nevertheless, the number of affected males is felt to be underrepresented in the literature.[43] Self-induced vomiting is a common feature of the eating disorders of anorexia and especially bulimia nervosa. Athletes including professional jockeys have also been reported to engage in this habit. Patients who self-induce vomiting have been shown to have higher risk of tooth erosion than peers who do not vomit.[44]

Eating disorders associated with vomiting are associated with an increased occurrence, severity and risk of dental erosion.[2] Interestingly, the frequency, duration and total number of self-induced vomiting episodes in those with eating disorders are not seen to be linearly associated with the severity or number of eroded teeth, indicating that other factors are at play.

Spontaneous vomiting may occur frequently during the first trimester of pregnancy.

Seen in children, cyclic vomiting syndrome is recognised to be linked with irritable bowel syndrome, motion sickness, migraine and epilepsy.[41] Prolonged bouts (weeks) of vomiting can begin in preschool children, occur throughout child development and reduce in frequency by adulthood. The condition is therefore self-limiting.

Alcohol dependence may be associated with both regular vomiting and reflux, significantly increasing the risk for dental erosion. There is conflicting evidence in the literature regarding the relationship between alcohol dependence and erosive defects. However, a trend for both increased severity and prevalence of erosion is broadly seen in those with chronic alcoholism.[45]

Rumination
The ability to relax the lower oesophageal sphincter, reflux gastric contents into the mouth and re-swallow is uncommon but has been reported.[46] This naturally poses a risk for dental erosion.

Extrinsic acidic sources

There are multiple exogenous acids that have been discussed in relation to dental erosion. These can be categorised into those from dietary, pharmacological and environmental/lifestyle sources.

Dietary extrinsic acids
An acidic diet is widely associated with dental erosion, and careful questioning on the intake of specific items of food and drink is necessary (Table 2). A low pH and a high buffer capacity (the resistance of a dietary product to being neutralised by saliva) are deemed the major risk factors for erosive potential, with the calcium concentration in foods and drinks regarded as the major protective factor.[26] It is important to consider all these factors together in understanding the erosive potential of foods and drinks. For instance, yoghurt has a low pH of 4 but a very high calcium concentration and therefore has a low erosive risk overall. Additionally, the consistency as well as the content of foods and drinks are important; a consistency that increases adherence to the tooth surface will clearly prolong the erosive challenge.

While the erosive potential of specific dietary acids is important to consider, it is also necessary to review the overall pattern of consumption. Frequency of dietary acid intake is a primary risk factor for erosive toothwear progression.[47,48] Four or more nutritional acidic intakes per day, in the presence of other risk factors (such as low buffering capacity of stimulated saliva and use of a hard bristled toothbrush), is associated with higher risk for the development and progression of erosion.[49] Moreover, an increased frequency of dietary acids that are also consumed between meals represents a further increased risk for erosive toothwear.[50]

The above said, it should be noted that the relationship between dietary acids and erosion is not straightforward, with some who consume dietary acids developing erosive toothwear and others not.[47] It is accepted that this a reflection of other co-factors being highly influential (eg salivary flow rates, buffering capacity and salivary composition). Future research is needed to fully understand causal relationships and the influence of co-factors in the erosive process.

Drinks
Carbonated soft drinks (including their sugar free counterparts) are considered to have a high erosive potential. Evidence linking dental erosion with soft drink consumption is available in the literature.[51] Fruit juices, including smoothies, may also have high acidity and potential for dental erosion due to the fruit content and other additives (see Foods).

For carbonated soft drinks, fruit juices and smoothies, the risk to children and adolescents is concerning given that consumption rates are high and increasing among this age group.[6,52,53] Indeed, soft drinks have been reported to provide as much as a fifth of the added sugars in

the diet of 11–12-year-old children, and 42% of fruit drinks are consumed by children aged between 2 and 9 years.[54] Drinks delivered from a feeding bottle, used as a comforter, may be particularly harmful to infants, with reported extreme dental destruction resulting from abuse of fruit juices.[55] Preventative measures aimed at dietary extrinsic acids for children and young adults are therefore a vital strategy in reducing prevalence of erosive toothwear, especially as dietary patterns established in formative years can carry on into adulthood.

The addition of fruit (particularly slices of lemons or limes) or fruit flavourings (eg cordials) to drinks has increased and it is safe to assume that any added fruit flavourings of this kind will render the beverage acidic.[47]

Fruit flavoured teas such as ginger and lemon, berry and rosehip are also considered acidic.[47,56] There is some evidence that warming beverages may increase their erosive potential; this may be especially relevant to the consumption of fruit flavoured teas and hot cordials.[57]

Sports drinks are also considered to have a high erosive potential and are increasingly consumed owing to the promotion of active, healthy lifestyles.

Some alcoholic drinks, such as dry wine, cider and alcopops, are also acidic.[58–60] Alcohol consumption, as mentioned, is also linked with gastric reflux, and may consequently be both an intrinsic and extrinsic agent of erosion.[61]

As a general rule when simplifying the message for patients, most drinks that are not water, plain carbonated mineral water (sparkling water) or milk are acidic.[47] Beverages with erosive potential are listed in Table 2.

Part of the erosive potential of a given beverage is also the manner in which it is consumed. In a recent study, participants who admitted to constant sipping, swishing, swilling or holding the drinks in the mouth prior to swallowing had approximately ten times increased odds of developing erosive toothwear.[50] This finding has been confirmed in other studies. Where advanced erosive toothwear is present in someone with seemingly limited acid exposure, the presence of a habit that increases the duration of contact between the acidic component and the tooth surface should be considered.

Calcium and/or phosphate compounds have been added to beverages to test their effect in reducing the erosive potential. In vitro studies show promise with respect to modification of drinks to reduce erosive potential.[30,31]

Foods

Fresh fruit and in particular citrus fruit[62] have erosive potential, as do chillies and tomatoes.[47] Foods pickled in vinegar and vinegars can be highly acidic; their consumption may be prevalent in certain international diets and are being increasingly promoted as part of a healthy diet. Less well known is the influence of covert acids in foodstuffs that have been associated with erosion in teenagers (eg brown sauce, crisps, ketchup and vinaigrette).[63] Flavoured (acid-based, sugar containing) chewing gums are also considered to have potential for dental erosion. Foods with erosive potential are listed in Table 2.

Lifestyle changes have undoubtedly increased the acidity content of modern diets. A greater emphasis on a healthy diet has resulted in an increase in fruit and vegetable consumption for

some individuals. National campaigns for healthy eating have also emphasised the importance of eating at least five portions of fresh fruit or vegetables per day, and the health benefits of consuming fruit and vegetables regularly should be encouraged as part of a balanced diet. (See the NHS Eatwell Guide – www.nhs.uk/live-well/eat-well/the-eatwell-guide.)

More people are becoming vegetarian and this tends to be a more acidic diet. However, very few studies confirm a correlation with dental erosion and overall, the results are contraindicatory. Lacto-vegetarians were reported to have significant dental erosion although the study has not been repeated in order to confirm this association.[64] Raw food diets, which include consuming only non-processed foods, have been shown in a small sample study to increase the risk of dental erosion versus consumption of a conventional diet.[65]

Given the importance of fruit and non-starch vegetables for general health, it is important that people are not advised to reduce their fruit consumption since as a society, we do not consume enough for health. Instead, only people for whom it is a clear identified risk factor should be advised to modify their diet to perhaps swap a citrus fruit for vegetables, banana or avocado between meals to ensure that they maintain a healthy diet and consume at least five a day.

As with acidic beverages, it is not just the total exposure to acidic foods that appears to have increased in recent years. There have also been changes in dietary habits/patterns. The frequency of food intake is changing; greater numbers of snacks are being consumed and long periods of snacking are occurring, commonly known as ‘grazing’.

In a multicultural society, there will be different habits and varieties of food not necessarily indigenous to the UK as well as different methods of food preparation. Little is known about these influences on dental erosion. Slaking palm with lime juice, betel nut chewing and crunching of chicken bones to savour the bone marrow have all been reported to increase the risk of toothwear and erosion.[66] Although not usual among Western cultures, these habits will be common among people from other cultures who live in the Western world. Clinicians should be aware of these cultural differences and question patients about any habits that may increase the risk of wear.

Pharmacological extrinsic acids

A number of medications and dietary supplements (such as vitamin C, aspirin and some iron preparations) are acidic and potentially erosive if they are in the form of chewable tablets or effervescent drinks.[26,67] It is unlikely, however, that these are in widespread use among children and adult population groups. Asthma medications have been speculated to increase dental erosion but the results are inconclusive.[68,69]

Many medications or medical treatments induce a dry mouth (eg antihistamines, anti-emetics, antidepressants, anti-Parkinson, diuretic medications, and head and neck targetted radiotherapy). The increased risk of erosion in these patients occurs because of the loss of salivary protective factors with reduced salivary flow. Furthermore, xerostomic patients may have a tendency to suck acidic sweets to alleviate their symptoms, which can further compound their risk of erosion. Some medications induce nausea and vomiting. The potential comorbidities of dry mouth and vomiting caused by some medications on dental erosion has not been investigated widely.

Destruction of dental hard tissue can be found in drug dependent persons. Nevertheless, attritional toothwear is seen more commonly than erosive toothwear owing to drug induced muscle hyperactivity.[70]

Lifestyle/occupational extrinsic acids
Active lifestyles and leisure trends can be associated with a greater risk of erosion,[71,72] and in a systematic review of the oral health of elite athletes, prevalence values for dental erosion of between 36% and 85% were recorded.[73] Professional athletes and swimmers are reported to be at increased risk of dental erosion due to exposure to sports drinks and swimming pool water. Well buffered, pH controlled chlorinated swimming pools, however, mitigate the risk of dental erosion and highly frequent swimming activities are therefore considered an uncommon risk factor for erosion.[2]

Work related exposure to acids in the form of liquids or vapours can result in dental erosion.[74] Wine tasters, battery manufacture workers, and those working in galvanising and plating factories may be considered at increased risk for dental erosion. This is generally considered a rare cause of erosive toothwear and most industries have to adhere to occupational health legislations limiting exposure.[26,71,74–76]

The use of mood enhancing drugs such as ecstasy increases the risk of dental erosion/ toothwear.[70,77–79]

Presentation and diagnosis

A diagnosis of erosive wear should be made as early as possible so that preventative strategies may be implemented quickly to limit further progression. Careful examination should consequently take place under good lighting with dry teeth to facilitate recognition of even the subtlest signs of dental erosion.

In general, erosive toothwear is characterised by the loss of tooth surface morphology and contour. Few studies have investigated the site specificity of dental erosion but the literature indicates that the palatal and labial aspects of maxillary teeth and occlusal surfaces of the mandibular teeth are commonly affected.[80] It is further reported that extrinsic acids contributing to dental erosion will predominantly affect the buccal cervical surfaces of the maxillary teeth and the occlusal surfaces of the mandibular posterior dentition. In cases of dental erosion caused by intrinsic acids, toothwear affects the palatal surfaces of the maxillary dentition, with protection of the lower anterior lingual surfaces by the tongue.[81]

On the occlusal surface, cusp tips may be cupped with flattening of occlusal contours and a less defined groove–fossa system may be observed. In advanced stages, the entire occlusal morphology may disappear with the presence of hollows, concavities and exposed areas of dentine. Concavities are typically wider than they are deep. Restorations may stand proud of the adjacent occlusal surface. Incisally, edges may become grooved or chipped and increased translucency may be seen, resulting in a bluish appearance. Exposure of dentine increases as erosion progresses.

On smooth surfaces, there is flattening of the entire surface. Loss of the perikymata and cingulum may also be noted. As enamel becomes thinner, chamfered ridges or ledges in enamel are visible and can be felt with a probe. A thin band of enamel at the gingival margin may be observed on the labial and palatal aspect of maxillary incisors. Teeth may appear darker as the enamel thins and the yellow dentine increasingly shines through/is exposed.

On both occlusal and smooth surfaces, the enamel acquires a rounded and glossy, shiny characteristic.

A diagnosis of dental erosion is made more difficult because of the triad of toothwear mechanisms and their complex interplay. While it is accepted that more than one type of toothwear mechanism is often in existence, identifying the principal aetiology will favour successful management. Occlusal erosive toothwear can be distinguished from attritive lesions, which are typically sharp and flat with matching facets. Smooth surface erosive toothwear can be distinguished from abrasive lesions, which are often wedge shaped, located at the cervical margin with sharp edges at right angles to the enamel surface. An accurate history will also help to elucidate the principal aetiology of toothwear defects.

Radiographically, occlusal surfaces may appear eroded with loss of enamel and dentine thickness.

Patients complain of poor aesthetics once a significant volume of enamel and dentine becomes lost, resulting in shortened anterior teeth and/or dentinal exposure. This is the common complaint on presentation rather than sensitivity or any functional difficulty.

Useful means of estimating the current activity of erosive wear include reports of dentine hypersensitivity and the absence of staining. These indicators suggest progression of erosive toothwear.

Management: preventative treatment

As dental erosion is progressive, preventative measures delivered when there are only early signs of erosive toothwear can be particularly effective in reducing the morbidity and dental intervention experienced by patients. The main objectives of prevention are to reduce modifiable factors (extrinsic acids), to appropriately manage non-modifiable factors (intrinsic acids), and to record and monitor the erosion.

An ‘active monitoring’ philosophy is recommended, especially if there are no patient reported concerns regarding pain/sensitivity, function or aesthetics. Clinically, if this approach is to be adopted, it must also be ensured that the remaining tooth structure will remain favourable to maintaining a comfortable, functional and aesthetic dentition in the long term, and that further loss of structure would not preclude or complicate restoration if this becomes necessary.

Patient information leaflets

These are very useful and allow patients to consider risk factors, behaviours etc in their own time. Patient information leaflets are produced by some companies or they can be made ‘in house’.

Recording and monitoring erosion

Regular monitoring of dental erosion should be established on an individual basis and will help to ascertain whether there is arrest or progression of the pathology. Records can also provide information on the efficacy of the preventative measures implemented.

In children and adults, good quality study casts and photographs aid the monitoring of dental erosion. While photographs can be stored digitally, safe storage of study casts can be problematic unless scanning and digital models are available. In adults, a silicone putty impression of the worst affected area is more readily stored with the patient notes and may be a helpful tool to assess progression. At a subsequent recall appointment, the putty index is sectioned labiopalatally and placed over the teeth. Any gap between the putty index and the tooth surface indicates progress of the erosion/wear and possible poor compliance with lifestyle changes. In children, growth and dentoalveolar development will preclude accurate seating of a putty index at review.

Many different clinical indices have been proposed and experts in the field have recommended the use of the Basic Erosive Wear Examination.[82] As per the Basic Periodontal Examination, the mouth is divided into sextants and the most severely worn tooth is graded according to a grading system (0 = no erosion; 1 = initial loss of enamel tooth surface texture; 2 = distinct defect, hard tissue loss <50%; 3 = hard tissue loss ≥50%). The scores for each sextant are added and the cumulative total is linked to a recommended management plan. As with any other clinical index, it is subjective and discrimination between the various grades can be difficult.[83] It has been argued that the ability to achieve several goals effectively with one index (monitor and clinical treatment need) is questionable.[84,85] Nevertheless, it does introduce some level of standardisation of recording erosion progression in the clinical notes.

Digital scanning may also be employed effectively to record erosive toothwear, entirely eliminating issues with physical storage of measurement indices.[86] This method of recording erosive toothwear may become more commonplace with further adoption and development of this technology. Novel research into optical coherence tomography may prove useful in the measurement of erosive tooth loss in the future.[87]

If other oral risk factors are low, recall interval of one year for adults and six months for children is reasonable.

Dietary analysis

Patients should be instructed to record a minimum three-day diet history (including a weekend). All food, drink and medication consumption should be recorded with timings. Patients should be encouraged to complete their diet history sheets as accurately and honestly as possible by emphasising that their resulting tailored management plan will be more effective. On review of the completed dietary history sheet, all acidic components should be highlighted and particular note should be made of acidic components consumed between meals.

In addition to asking patients to complete a diet history sheet, the following questions will highlight habits with an associated increase in erosive damage:[47]

- Do you like to ‘graze’ on acidic foods/sip acidic drinks over long periods?
- Do you swish, hold or swill acidic drinks prior to swallowing?
- Do you enjoy heated acidic beverages (eg hot cordials, fruit teas, hot water with lemons)?

Dietary counselling

While there is some evidence that one-to-one dietary interventions in the dental setting can change a patient's behaviour, there is no robust evidence relating to the effectiveness of different strategies to apply when providing dietary advice for the prevention of dental erosion.[88,89] Counselling, however, must be tailored to the individual and is only possible after the diet has been assessed thoroughly. Realistic, clear goals should be established to eliminate or reduce erosive challenge. The benefits of a balanced healthy diet should be emphasised and advice on limiting foods such as acidic fruits and vegetables, which have recognised health benefits, should only be given to those individuals where excess consumption has been identified as a risk factor.

Prior to giving specific dietary advice, a clear explanation of the difference between erosion and caries is advisable as the public confuse these terms and believe them to be synonymous pathologies. This is especially relevant to artificially sweetened diet drinks, which patients may feel are a better alternative to full fat varieties owing to their low sugar, not realising that their highly acidic component will contribute to the erosive process. Specific dietary advice to emphasise to those susceptible to erosive toothwear includes:

- Reduce the frequency of dietary acid intake (if relevant).
- Limit acidic foods and drinks to mealtimes (if relevant) as this is the time of maximum salivary flow and increased buffering capacity. For the same reason, acidic intake should be avoided at bedtime and during the night.
- Avoid dietary habits that extend the length of time of contact between the dietary acid and tooth surface (eg grazing on fruit, sipping/swishing/holding of acidic beverages). It is therefore advisable that drinks are consumed quickly or if consumed slowly, placing a wide bore straw towards the back of the mouth is advisable to reduce contact of acidic fluid with the teeth.[90,91]
- Avoid hot acidic beverages or wait for these to cool before drinking.[57]
- Consumption of water, plain sparkling water or milk should be promoted as alternatives to acidic beverages.
- Chew sugar free gum after an acid exposure. Chewing sugar free gum increases salivary flow and encourages tooth remineralisation[92] but this may not be the case for acid containing gums.[93]
- Finishing a meal or following an acid exposure with cheese or milk will neutralise intraoral acid.[94]
- Where possible, chewable or liquid formulations of acidic medications should be changed. Liaison with the GP will be needed to ensure a safe switch to an alternative preparation.
- Avoid mechanical toothbrushing immediately after exposure to acid (see Oral Hygiene).

GORD, vomiting and rumination

Patients should be questioned regarding symptoms of GORD (see Gastro-oesophageal reflux disease page 8) although it should be remembered that a proportion of patients suffer with silent reflux. Many patients with GORD will present having self-medicated with over-the-counter medicines. Dentists should refer (with the patient's permission) to the patient's GP where GORD is suspected as an aetiological factor for presenting with erosive tooth loss.

Referral to gastroenterology is made in the following instances:

- if symptoms interfere with daily life;
- if previous tests for GORD were either inconclusive or equivocal;
- if after elimination of dietary factors and after a period of review, erosion progresses;
- if there is no other obvious cause of erosion;
- if severe erosion is present, which may be unilateral and affecting the buccal surface.

Where GORD is suspected or has been confirmed, preventative lifestyle modification should also be encouraged to help avoid reflux provoking foods/drinks, such as alcohol, vinegar, fatty foods, coffee, black tea, peppermint, carbonated drinks, chocolate and meals before bedtime. Chewing sugar free gum may also be recommended in such patients with the benefits of increasing salivary flow and swallowing frequency.[75]

Patients with a medical disorder that has a sequela of repeated vomiting (see Vomiting page 9) may be under the care of a medical professional already but this needs to be confirmed and where medical intervention has not been sought, a referral to the patient's GP is appropriate. Dental professionals may be the first to suspect signs of an eating disorder. Patients with an eating disorder may benefit from psychological counselling, arranged through the patient's GP. This is a sensitive undertaking and care is needed regarding the maintenance of confidentiality in teenage patients. Those who suffer from frequent vomiting should be advised to rinse afterwards with a fluoride containing mouthrinse, or milk or water when this is unavailable.

Rumination syndrome is considered a learnt behaviour and management may involve elements of behavioural psychology. Referral to the patient's GP is advised in these cases.

Oral hygiene, remineralisation and desensitisation

Oral hygiene
An appropriate non-traumatic toothbrushing technique designed to effectively remove plaque in combination with a low abrasive toothpaste should be recommended for patients with dental erosion.[95] Advising patients to not brush their teeth after an acidic episode to allow remineralisation of the softened enamel and prevent further tooth loss is a matter of dispute among researchers.[26,50,75] Historically, it was advocated to not brush after an acid challenge as the acid softened tooth surfaces would be more susceptible to abrasion.[96,97] More recently, it has been argued that saliva does not remineralise softened enamel quickly and as such, tooth surface loss will occur regardless of whether toothbrushing is performed soon after an acidic exposure. It is further suggested that not brushing after an acidic exposure denies the opportunity of exposure to fluoride, which is helpful to aid remineralisation and prevent caries.[26]

Based on a recent observational cross-sectional study[98] and case controlled study,[50] it may be appropriate to advise patients with erosive toothwear to not brush immediately (within 10 minutes) after an acidic episode and a fluoride containing mouthwash may be used as an alternative.

Remineralisation
Fluoride agents (especially those containing stannous fluoride or stannous chloride) should be used as part of the preventative management of erosive toothwear. These include fluoride mouthrinses and toothpastes.[26] Alternatively, conventional high fluoride toothpastes and mouthwashes may be prescribed. High fluoride preparations need to be used in caution in those under six years old.[99]

The mechanism by which conventional fluorides such as sodium fluoride and amine fluoride prevent erosive demineralisation is through the formation of a calcium fluoride layer.[100] This acts as a physical barrier to the acids, and as a reservoir of calcium and fluoride ions that can be released during an acid attack.[6] Conventional fluoride products can therefore potentially slow the progression of toothwear. It is thought, however, that under the conditions of an acidic attack, the protection of a calcium fluoride layer is short lived. More recently, research into the addition of polyvalent metal ions (such as stannous or titanium) to fluoride toothpastes has yielded promising results in preventing erosive demineralisation.[6] These metal cations have been shown to make the tooth surface more resistant to dissolution during an acid exposure.

Toothpaste application prior to an erosive challenge seems to be favourable compared with tooth cleaning after exposure. In practice, it may seem unlikely that patients prone to erosive toothwear will execute a fluoride regime before an acid attack such as vomiting although application prior to sleeping and overnight reflux may consequently be of benefit.[101] Nevertheless, the effectiveness of fluoride in typical toothpaste concentrations may be dependent on the acid attack.[102]

Some newer 'enamel care' dentifrices have been shown to provide enhanced resistance of enamel and dentine to acid challenge, and may aid remineralisation although prospective randomised clinical trials have not been reported to date.[103–105]

Fluoride varnishes in patients who have a high frequency of acid exposure are likely to offer protection for a short duration and be cost ineffective.[83] Furthermore, the 'protective' effect offered by these varnishes is more likely related to a mechanical, physical effect than due to remineralisation.[6]

Sugar free chewing gum increases salivary flow and will also aid remineralisation.[92]

Desensitisation
Patients with significant erosion and dentine exposure may complain of tooth sensitivity. This may also be an indication that the erosion is still active. The use of fluoride mouthrinses and varnishes is beneficial but they must be used frequently and regularly. A high fluoride toothpaste may be helpful as long as it is not also highly abrasive. Other products such as specially formulated toothpastes for sensitive teeth or GC Tooth Mousse® may also be useful. Application of a resin sealant or dentine bonding agent may also reduce sensitivity temporarily.[26]

Other/novel preventative strategies

Dentine bonding agents applied to areas of exposed dentine have been shown to reduce further erosive tooth loss.[106] In a small scale trial, the application of fissure sealant to the palatal surface of worn maxillary anterior teeth reduced erosion progression by up to nine months.[107]

Recent in vitro studies suggest that the modification of the enamel pellicle (also referred to as acquired pellicle engineering) may play a role in the prevention of erosion.[108] In broad terms, this process is based on the principle that the dental pellicle, rich in proteins, is known to offer some form of protection to the tooth surface by erosive attacks. For this reason, modification of this layer may be a useful strategy in preventing erosion.

Management: restorative treatment

Restorative, operative intervention for erosive wear is necessary to rehabilitate function and aesthetics as well as to eliminate painful symptoms. Additionally, interventive treatment may be indicated to preserve the remaining structural integrity of the eroded dentition and circumvent more complicated intervention at a later date. Importantly, restorative management of erosive toothwear should be preceded by or used in conjunction with the aforementioned preventative strategies.

Ideally, in both children and adults, aetiological factors should be identified first and brought under control. This may involve a period of monitoring as outlined previously before definitive restorative treatment is commenced. Clearly, the patient's desire to improve appearance and/or reduce sensitivity may hasten the start of interventional treatment.

Primary dentition

In the primary dentition, if the child is not experiencing any symptoms, restorative treatment is not indicated. If teeth are sensitive, small areas of erosion may be covered with composite resin. Larger areas may require placement of composite crowns on anterior teeth and stainless steel crowns on posterior teeth. For severe symptoms, extraction of the offending teeth may be necessary.

Mixed dentition

In the mixed dentition stage, the permanent dentition should be treated conservatively by either long-term monitoring or the addition of dental composite resin to eroded surfaces. Dentine bonding agents (without dental composite addition) can offer short-lived dentine protection (up to 3 months) while unfilled fissure sealant in combination with a compatible, self-etching, single stage adhesive has been shown to confer protection to the palatal surface of maxillary anterior teeth from toothwear for periods of up to nine months.[109,110] The adaptive capacity of the stomatognathic system during growth may be greater than in adulthood and so restoration of the eroded occlusion including guiding surfaces has not resulted in reports of postoperative problems.

Minimal space is required to bond composite resin without increasing the occlusal vertical dimension (OVD). Cupped and grooved surfaces can be restored to the enamel rim, which does not usually involve an increase in the OVD. Dentine surfaces should be cleaned with pumice/water or slow speed rose head burs prior to etching in order to remove the salivary pellicle and enhance bonding resin infiltration/penetration of sclerotic dentine. The polyalkenoates or glass ionomers are themselves susceptible to acid erosion/dissolution and have no application in the eroding dentition.[111] There is also weak evidence that the surface hardness of microfilled composite and resin modified glass ionomer is significantly reduced following short immersion in acidic beverages although this has not been examined in vivo.[112]

Permanent dentition

The management of erosion in the permanent dentition follows the guidance in the previous section.

The treatment of erosive wear in the permanent dentition can range from relatively simple and localised interventions to more complex treatment planning including full mouth rehabilitation. Previous restorative approaches used for rehabilitation of the eroded permanent dentition commonly involved extensive conventional crown and bridgework or removable overdentures. While these treatment modalities may still have a place in the management of erosion, the advancement of adhesive technology has meant that less destructive options for restoration are now available and used widely in general dental practice. These more conservative approaches can be applied successfully and predictably, and should be considered in the first instance.

Regardless of the level of restorative intervention, the patient naturally needs to be made aware of the cost and time implications, in addition to the potential future maintenance burden. It is also clearly necessary that definitive restoration of the eroded permanent dentition requires prior elimination of all primary disease as well as the preventative measures outlined above.

Decision making and treatment strategies in erosive toothwear
Assessment of the space in the intercuspal position (ICP) is the first step in decision making. As the erosive wear process often happens gradually, dentoalveolar compensation occurs to re-establish ICP contacts and maintain masticatory function, resulting in maintenance of the OVD and facial proportions. This leads to a lack of interocclusal space for restoration.

If the toothwear process has been rapid and has exceeded the physiological mechanisms of tooth eruption, then interocclusal space may be present in the ICP as dentoalveolar compensation has not had time to occur. Where space is present for restoration, restorations may be placed comparatively easily by conforming to the patient's existing ICP. This scenario is relatively uncommon and often there will be no space for restoration.

Localised toothwear presents frequently with an OVD within normal limits and a lack of interocclusal space for restoration. In generalised toothwear, two situations can occur: dentoalveolar compensation has occurred, and there is maintenance of the OVD and facial proportions; or there is no compensation with an increase in freeway space, loss of OVD and mandibular overclosure.[113] This latter situation is seen more often in partially dentate patients presenting with loss of the posterior dentition.[114]

Localised erosion
Labial/buccal wear and small occlusal cupped lesions
Where wear is localised to the labial or buccal surfaces only, restoration can be achieved with direct or indirect composite restorations or veneers. Where the labial wear has been more significant (and ideally, where a ring of enamel remains), 'no prep' adhesively bonded feldspathic or reinforced glass-based ceramics can be considered.

Localised cupped occlusal sites may be restored with occlusal composite. These sites are prone to further erosive attacks owing to potential pooling of acids, and must therefore be monitored along with ongoing preventative measures and advice.

Palatal wear of maxillary incisors

The well established management of palatal wear of the maxillary incisors has been to utilise a 'Dahl appliance'.[115] This is, in effect, an anterior bite platform that takes the posterior teeth out of occlusion. It promotes relative axial movement of anterior and posterior teeth resulting in creation of space for the restoration of eroded anterior teeth. The interocclusal space created occurs through a process of combined intrusion (40%) and extrusion (or 'compensatory eruption') (60%). There is also some suggestion of mandibular repositioning occurring concomitantly.[111] It is thought that re-establishment of the separated occlusal contacts occurs in most cases within 4–6 months but in some cases, this may take up to a period of 18–24 months.[116] Localised increases in the OVD are extremely well tolerated and have become entirely predictable as a treatment modality. Good patient understanding of the treatment, however, is a prerequisite for success of this technique.

The first iteration of the Dahl appliance was a removable cobalt-chrome anterior bite platform.[115] Subsequently, a fixed anterior bite plane has been utilised,[117] in place of a removable appliance as described originally, which reduced the need for patient compliance with respect to the time the appliance was in place; as such, it produced more predictable results in a shorter period. Clinical studies now support the concept of restoring the worn upper anterior teeth at an increased OVD without the interim stage of a removable, or fixed, Dahl appliance as the restorations themselves have a 'Dahl effect'.[118] Once space has been gained, then restoration of the anterior teeth may be carried out by a variety of means.

In this clinical scenario, palatal veneers in metal, or direct or indirect composite resin restorations can provide an acceptable functional and aesthetic outcome while being cost effective, minimally abrasive to antagonists, minimally invasive and repairable.[109] Direct composite application can be freehand or with the use of customised matrices. Various techniques have been described that are largely dependent on operator preference and the degree of laboratory support available. Contemporaneous summaries describing these methods are referenced for further support.[109,110]

There is good evidence that both direct and indirect palatal composite restorations placed according to manufacturers' instructions at an increased OVD perform favourably in combination with dentine bonding although opinion suggests that these should be placed in relatively thick sections to maximise their longevity and where there is sufficient posterior support.[111,119–121] In general, major failure is uncommon in the first five years after placement although minor wear, marginal discolouration and marginal fracture are likely. A careful explanation is therefore necessary of the trade-off between the significant biological benefit with regard to the far less destructive nature of these adhesive restorations and their maintenance compared with traditional restorative methods.[111,118,122,123]

Where palatal erosive wear is severe or occurring in combination with attrition, a more durable material may be indicated. Palatal gold and non-precious metal veneers have been described as favourable materials in this clinical scenario.[109,124–126] In these cases, the palatal metal veneer is bonded and used as a matrix to guide build-up of the labial and incisal aspects to the desired form if an increase in incisal length is also desired.[126] No preparation of the palatal surface is required other than smoothing any sharp enamel margins. Bonding should be completed with an opaque adhesive resin cement.

Regardless of the material used for restoration of maxillary palatal erosive defects, even contacts with restorations should typically be obtained on the six anterior teeth, with smooth, evenly distributed anterior guidance in protrusion lateral excursions where possible.

Incisal/labial wear of mandibular incisors
Incisal wear of mandibular incisors can be challenging to manage as their diminutive nature presents a reduced surface area for bonding. Indirect full coverage restorations also present increased risk of loss of pulpal vitality in these teeth due to the amount of tissue removal required relative to the volume of the unprepared tooth. For this reason, it is generally accepted that minor incisal wear should be monitored and preventative measures employed.

In more advanced mandibular incisal wear, where there is inadequate space in the ICP, direct composite restorations at an increased OVD as part of the Dahl approach have been shown to have good survival rates.[119,127] Dentine bonded crowns involving minimal cervical and incisal preparation may also be considered in more severe cases or in cases where composite resin has failed repeatedly. Caution should be exercised over their use in patients with parafunction and it is reported that their success is predicated on an 'enamel ring' being present.[119]

Posterior occlusal wear
Posterior occlusal wear may be addressed in a similar fashion to anterior palatal wear by placing restorations in a supraocclusion and employing the Dahl concept.[113,128] Should anterior contacts not re-establish, consideration may need to be given to providing further restorations on the remaining anterior teeth to re-establish even and balanced contacts throughout the arch. This would also apply to anterior restorations placed at an increased OVD, as described previously (see Palatal wear of maxillary incisors).

Indirect restorations including partial coverage restorations and onlays may be indicated in the clinical scenario of posterior occlusal wear. Few studies to date have examined the use of composite restorations for the restorative management of worn posterior teeth. While an early study reported a high failure rate (fracture or complete loss of restoration), a microfilled composite material was used and a hard acrylic stabilisation splint was not provided.[129] Later case reports using a hybrid composite resin and full coverage, heat cured acrylic resin splints for ongoing supportive care may explain a far greater level of success, albeit on limited numbers of patients.[130]

It has also been argued that application of a composite resin provides the clinical advantage of allowing the patient to accommodate to the occlusal changes and enabling the clinician to make any adjustments. The composite resin may then be readily replaced with a restorative material with increased durability if needed.[128]

Where signs of early posterior tooth surface loss are observed and there is an absence of disclusion in lateral excursions, metal palatal veneers on the canines acting as 'canine risers' can be used to re-establish a mutually protective occlusion and protect the posterior teeth from further wear.

Other approaches to localised toothwear
Adhesive, additive measures employed as part of the Dahl approach, as outlined above, are the restorative strategies of choice in localised toothwear, their distinct advantage being that they are biologically conservative of tooth structure, which is especially desirable in a scenario where existing tooth tissue has already been lost. The conventional approach employs occlusal/incisal reduction to make space for a full or partial coverage indirect restoration. This increases the risk of pulpal necrosis[131] and reduces the structural integrity of the already worn tooth.

Generalised erosion

The treatment strategies for restorative management in cases of generalised erosion principally include planning for an increase in the OVD and reorganising the occlusal scheme in the retruded axis position or for space creation through occlusal equilibration (if a discrepancy exists between the retruded contact position and ICP). The latter approach rarely provides sufficient space alone and involves preparation of already compromised teeth. Its use as a restorative approach is consequently limited.

Traditional conventional strategies for generalised toothwear include tooth reduction to achieve appropriate space for restoration with full or partial coverage indirect restorations, as described previously for localised wear. The advent of reliable adhesive materials means more destructive and invasive options are indicated less often, and more conservative designs (such as indirect onlays) can be provided utilising adhesive luting cements.

Increases in the OVD are well tolerated by dentate patients owing to the proprioception through the periodontal ligament and associated neuromusculature.[113] Clinical studies in adults have also not reported any long-term increase in temporomandibular joint dysfunction or dental problems.[102,103,132,133] Occlusal splints are therefore not necessary to simply test an increase in the OVD. However, they may still be indicated in this cohort for other reasons, such as pre-restorative stabilisation to ensure a reproducible jaw relationship, to protect worn teeth/new restorations if there is an attritive co-factor and in the management of temporomandibular joint dysfunction.[113] Prescribing increases in the OVD requires careful planning and is supported by the use of appropriately mounted study casts, diagnostic wax-ups at the intended vertical relation and diagnostic previews with clinical ‘mock try-ins’.

There are limited data to support the use of specific materials or techniques in generalised toothwear.[113] Of the few studies available, one has compared the use of indirect materials with resin composites in cases of severe generalised toothwear, reporting ten-year survival rates of 74.5% and 62.0% respectively.[134] This difference was not statistically significant. Furthermore, biological failures were more highly associated with indirect restorations and mechanical failures with composite resin. These latter failures were recognised as being more straightforward to manage and amenable to repair. Gold restorations were associated with the fewest failures overall. In a more recent study, low failure rates have also been reported in the use of direct hybrid composites for cases of generalised toothwear.[135]

In selecting appropriate restorations, a prudent approach is to conduct a tooth-by-tooth assessment and prescribe appropriate restorations based on the structural integrity with any restoration or core removed. Parafunctional habits may also influence selection of materials. A combination of indirect or direct composite restorations anteriorly with the use of partial coverage adhesive gold, metal or reinforced glass ceramics posteriorly could be considered a conservative approach in such cases. As further studies are conducted, posterior direct or indirect composite restorations may also become an evidence-based treatment strategy for full mouth reconstructions in generalised wear.

When the generalised toothwear is deemed very advanced, it can be more appropriate to reduce the teeth further and provide overdentures.[114] By maintaining overdenture abutments (versus extraction), the advantages include continued proprioceptive feedback, a reduction in ridge resorption with a resulting increase in denture stability and support, and the potential to use precision attachments such as magnets or studs to increase retention. Conversely, caries and periodontal disease associated with abutment teeth is increased.[136] Onlay or overlay

References

1. Jaeggi T, Lussi A. Prevalence, Incidence and Distribution of Erosion. In Lussi A, Ganss C. *Erosive Tooth Wear: From Diagnosis to Therapy*. 2nd edn. Basel: Karger; 2014. pp55–74.
2. Schlueter N, Luka B. Erosive tooth wear – a review on global prevalence and on its prevalence in risk groups. *Br Dent J* 2018; **224**: 364–370.
3. O'Toole S, Pennington M, Varna S, Bartlett DW. The treatment need and associated cost of erosive tooth wear rehabilitation – a service evaluation within an NHS dental hospital. *Br Dent J* 2018; **224**: 957–961.
4. Murray JJ, Vernazza CR, Holmes RD. Forty years of national surveys: an overview of children's dental health from 1973–2013. *Br Dent J* 2015; **219**: 281–285.
5. Murakami C, Tello G, Abanto J et al. Trends in the prevalence of erosive tooth wear in Brazilian preschool children. *Int J Paediatr Dent* 2016; **26**: 60–65.
6. Lussi A, Buzalaf MA, Duangthip D et al. The use of fluoride for the prevention of dental erosion and erosive tooth wear in children and adolescents. *Eur Arch Paed Dent* 2019; **20**: 517–527.
7. O'Brien M. *Children's Dental Health in the United Kingdom 199*3. London: HMSO; 1994.
8. Millward A, Shaw L, Smith A. Dental erosion in four-year-old children from differing socioeconomic backgrounds. *ASDC J Dent Child* 1994; **61**: 263–266.
9. 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.
10. Jones SG, Nunn JH. The dental health of 3-year-old children in East Cumbria 1993. *Community Dent Health* 1995; **12**: 161–166.
11. Hinds K, Gregory JR. *National Diet and Nutrition Survey: Children Aged 1½ to 4½ Years. Volume 2: Report of the Dental Survey*. London: HMSO; 1995.
12. Smith BG, Robb ND. The prevalence of toothwear in 1007 dental patients. *J Oral Rehabil* 1996; **23**: 232–239.
13. 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.
14. Williams D, Croucher R, Marcenes W, O'Farrell M. The prevalence of dental erosion in the maxillary incisors of 14-year-old schoolchildren living in Tower Hamlets and Hackney, London, UK. *Int Dent J* 1999; **49**: 211–216.
15. Walker A, Gregory J, Bradnock G et al. *National Diet and Nutrition Survey: Young People Aged 4 To 18 Years. Volume 2: Report of the Oral Health Survey*. London: TSO; 2000.
16. Al-Dlaigan YH, Shaw L, Smith A. Dental erosion in a group of British 14-year-old, school children. Part I: Prevalence and influence of differing socioeconomic backgrounds. *Br Dent J* 2001; **190**: 145–149.
17. Bardsley PF, Taylor S, Milosevic A. Epidemiological studies of tooth wear and dental erosion in 14-year-old children in North West England. Part 1: The relationship with water fluoridation and social deprivation. *Br Dent J* 2004; **197**: 413–416.
18. Chadwick B, Pendry L. *Children's Dental Health in the United Kingdom, 2003: Non-carious Dental Conditions*. London: ONS; 2004.
19. Dugmore CR, Rock WP. The prevalence of tooth erosion in 12-year-old children. *Br Dent J* 2004; **196**: 279–282.
20. Pitts N, Chadwick B, Anderson T. *Children's Dental Health Survey 2013 Report 2: Dental Disease and Damage in Children – England, Wales and Northern Ireland*. Leeds: HSCIC; 2015.
21. Health and Social Care Information Centre. *2: Disease and Related Disorders – A Report from the Adult Dental Health Survey 2009*. Leeds: HSCIC; 2011.
22. Van't Spijker A, Rodriguez JM, Kreulen CM et al. Prevalence of tooth wear in adults. *Int J Prosthodont* 2009; **22**: 35–42.

23. Assunção CM, Schlueter N, Rodrigues JA et al. Do fluoride toothpastes have similar preventive effect in permanent and primary teeth against erosive tooth wear? *Int J Paediatr Dent* 2019; **29**: 228–236.
24. Tschammler C, Müller-Pflanz C, Attin T et al. Prevalence and risk factors of erosive tooth wear in 3–6 year old German kindergarten children – a comparison between 2004/05 and 2014/15. *J Dent* 2016; **52**: 45–49.
25. Ganss C, Klimek J, Giese K. Dental erosion in children and adolescents – a cross-sectional and longitudinal investigation using study models. *Community Dent Oral Epidemiol* 2001; **29**: 264–271.
26. Carvalho TS, Colon P, Ganss C et al. Consensus report of the European Federation of Conservative Dentistry: erosive tooth wear – diagnosis and management. *Clin Oral Investig* 2015; **19**: 1557–1561.
27. Ashcroft AT, Joiner A. Tooth cleaning and tooth wear: a review. *Proc Inst Mech Eng J* 2010; **224**: 539–549.
28. Addy M, Shellis RP. Interaction between attrition, abrasion and erosion in tooth wear. *Monogr Oral Sci* 2006; **20**: 17–31.
29. El Aidi H, Bronkhorst EM, Huysmans MC, Truin GJ. Multifactorial analysis of factors associated with the incidence and progression of erosive tooth wear. *Caries Res* 2011; **45**: 303–312.
30. Manton DJ, Cai F, Yuan Y et al. Effect of casein phosphopeptide-amorphous calcium phosphate added to acidic beverages on enamel erosion in vitro. *Aust Dent J* 2010; **55**: 275–279.
31. Wegehaupt F, Günthart N, Sener B, Attin T. Prevention of erosive/abrasive enamel wear due to orange juice modified with dietary supplements. *Oral Dis* 2011; **17**: 508–514.
32. El-Serag HB, Sweet S, Winchester CC, Dent J. Update on the epidemiology of gastro-oesophageal reflux disease: a systematic review. *Gut* 2014; **63**: 871–880.
33. Bartlett DW, Evans DF, Anggiansah A, Smith BG. A study of the association between gastro-oesophageal reflux and palatal dental erosion. *Br Dent J* 1996; **181**: 125–132.
34. Bartlett DW, Fares J, Shirodaria S et al. The association of tooth wear, diet and dietary habits in adults aged 18–30 years old. *J Dent* 2011; **39**: 811–816.
35. Vakil N, van Zanten SV, Kahrilas P et al. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. *Am J Gastroenterol* 2006; **101**: 1900–1920.
36. Wang GR, Zhang H, Wang ZG et al. Relationship between dental erosion and respiratory symptoms in patients with gastro-oesohageal reflux disease. *J Dent* 2010; **38**: 892–898.
37. O'Sullivan EA, Curzon ME, Roberts GJ et al. Gastroesophageal reflux in children and its relationship to erosion of primary and permanent teeth. *Eur J Oral Sci* 1998; **106**: 765–769.
38. Reyes AL Cash AJ, Green SH, Booth IW. Gastrooesophageal reflux in children with cerebral palsy. *Child Care Health Dev* 1993; **19**: 109–118.
39. Shaw L, Weatherill S, Smith A. Tooth wear in children: an investigation of etiological factors in children with cerebral palsy and gastroesophageal reflux. *ASDC J Dent Child* 1998; **65**: 484–486.
40. Pace F, Pallotta S, Tonini M et al. Systematic review: gastro-oesophageal reflux disease and dental lesions. *Aliment Pharmacol Ther* 2008; **27**: 1179–1186.
41. Proceedings of the International Scientific Symposium on Cyclic Vomiting Syndrome. *J Pediatr Gastroenterol Nutr* 1995; **21**: S1–S62.
42. National Institute for Health and Care Excellence. *Resource Impact Report: Eating Disorders; Recognition and Treatment (NG69)*. London: NICE; 2017.
43. Mitchison D, Mond J. Epidemiology of eating disorders, eating disordered behaviour, and body image disturbance in males: a narrative review. *J Eat Disord* 2015; **3**: 20.
44. Hermont AP, Oliveira PA, Martins CC et al. Tooth erosion and eating disorders: a systematic review and meta-analysis. *PLoS* One 2014; **9**: e111123.

45. Schlueter N, Tveit AB. Prevalence of erosive tooth wear in risk groups. *Monogr Oral Sci* 2014; **25**: 74–98.
46. Gilmour AG, Beckett HA. The voluntary reflux phenomenon. *Br Dent J* 1993; **175**: 368–372.
47. O'Toole S, Mullan F. The role of the diet in tooth wear. *Br Dent J* 2018; **224**: 379–383.
48. Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. *Caries Res* 2000; **34**: 182–187.
49. Lussi A, Hellwig E. Risk assessment and preventive measures. *Monogr Oral Sci* 2006; **20**: 190–199.
50. O'Toole S, Bernabé E, Moazzez R, Bartlett D. Timing of dietary acid intake and erosive tooth wear: a case–control study. *J Dent* 2017: **56**: 99–104.
51. Dugmore CR, Rock WP. A multifactorial analysis of factors associated with dental erosion. *Br Dent J* 2004; **196**: 283–286.
52. 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 Res* 1997; **31**: 44–49.
53. Millward A, Shaw L, Smith AJ et al. The distribution and severity of tooth wear and the relationship between erosion and dietary constituents in a group of children. *Int J Paediatr Dent* 1994; **4**: 151–157.
54. Rugg-Gunn AJ, Lennon MA, Brown JG. Sugar consumption in the United Kingdom. *Br Dent J* 1986; **161**: 359–364.
55. Smith AJ, Shaw L. Baby fruit juices and tooth erosion. *Br Dent J* 1987; **162**: 65–67.
56. Lussi A, Megert B, Shellis RP, Wang X. Analysis of the erosive effect of different dietary substances and medications. *Br J Nutr* 2012; **107**: 252–262.
57. Barbour ME, Finke M, Parker DM et al. The relationship between enamel softening and erosion caused by soft drinks at a range of temperatures. *J Dent* 2006; **34**: 207–213.
58. Rees J, Griffiths J, Innes C. An in vitro assessment of the erosive potential of some white wines. *Eur J Prosthodont Rest Dent* 2002; **10**: 37–42.
59. Rees JS, Griffiths J. An in vitro assessment of the erosive potential of conventional and white ciders. *Eur J Prosthodont Rest Dent* 2002; **10**: 167–171.
60. O'Sullivan EA, Curzon ME. Dental erosion associated with the use of 'alcopop' – a case report. *Br Dent J* 1998; **184**: 594–596.
61. Smith BG, Robb ND. Dental erosion in patients with chronic alcoholism. *J Dent* 1989;*17*: 219–221.
62. Järvinen VK, Rytömaa II, Heinonen OP. Risk factors in dental erosion. *J Dent Res* 1991; **70**: 942–947.
63. Milosevic A, Bardsley PF, Taylor S. Epidemiological studies of tooth wear and dental erosion in 14-year old children in North West England. Part 2: The association of diet and habits. *Br Dent J* 2004; **197**: 479–483.
64. Linkosalo E, Markkanen H. Dental erosions in relation to lactovegetarian diet. *Scand J Dent Res* 1985; **93**: 436–441.
65. Ganss C, Schlechtriemen M, Klimek J. Dental erosions in subjects living on a raw food diet. *Caries Res* 1999; **33**: 74–80.
66. O'Sullivan E, Milosevic A. UK National Clinical Guidelines in Paediatric Dentistry: diagnosis, prevention and management of dental erosion. *Int J Paediatr Dent* 2008; **18(Suppl 1)**: 29–38.
67. Giunta JL. Dental erosion resulting from chewable vitamin C tablets. *J Am Dent Assoc* 1983; **107**: 253–256.
68. Dugmore CR, Rock WP. Asthma and tooth erosion; is there an association? *Int J Paediatr Dent* 2003; **13**: 417–424.
69. Al-Dlaigan YH, Shaw L, Smith AJ. Is there a relationship between asthma and dental erosion? A case control study. *Int J Paediatr Dent* 2002; **12**: 189–200.

70. Milosevic A, Agrawal N, Redfearn P, Mair L. The occurrence of toothwear in users of Ecstasy (3,4-methylenedioxymethamphetamine). *Community Dent Oral Epidemiol* 1999; **27**: 283–287.
71. Centerwall BS, Armstrong CW, Funkhouser LS, Elzay RP. Erosion of dental enamel among competitive swimmers at a gas-chlorinated swimming pool. *Am J Epidemiol* 1986; **123**: 641–647.
72. Milosevic A. Sports drinks hazard to teeth. *Br J Sports Med* 1997; **31**: 28–30.
73. Ashley P, Di Iorio A, Tanday A, Needleman I. Oral health of elite athletes and association with performance; a systematic review. *Br J Sports Med* 2015; **49**: 14–19.
74. Petersen PE, Gormsen C. Oral conditions among German battery factory workers. *Community Dent Oral Epidemiol* 1991; **19**: 104–106.
75. Buzalaf MA, Magalhães AC, Rios D. Prevention of erosive tooth wear: targeting nutritional and patient-related risk factors. *Br Dent J* 2018; **224**: 371–378.
76. Tuominen M, Tuominen R, Ranta K, Ranta H. Association between acid fumes in the work environment and dental erosion. *Scand J Work Environ Health* 1989; **15**: 335–338.
77. Nixon PJ, Youngson CC, Beese A. Tooth surface loss: does recreational drug use contribute? *Clin Oral Investig* 2002; **6**: 128–130.
78. Brand HS, Dun SN, Nieuw Amerongen AV. Ecstasy (MDMA) and oral health. *Br Dent J* 2008; **204**: 77–81.
79. Duxbury AJ. Ecstasy – dental implications. *Br Dent J* 1993; **175**: 38.
80. Ganss C, Lussi A. Diagnosis of erosive tooth wear. *Monogr Oral Sci* 2014; **25**: 22–31.
81. Hemmings K, Truman A, Shah S, Chauhan R. Tooth wear guidelines for the BSRD Part 1: Aetiology, diagnosis and prevention. *Dent Update* 2018; **45**: 483–495.
82. Bartlett D, Dattani S, Mills I et al. Monitoring erosive toothwear: BEWE, a simple tool to protect patients and the profession. *Br Dent J* 2019; **226**: 930–932.
83. Carvalho JC, Scaramucci T, Aimée NR et al. Early diagnosis and daily practice management of erosive tooth wear lesions. *Br Dent J* 2018; **224**: 311–318.
84. Fares J, Shirodaria S, Chiu K et al. A new index of tooth wear. *Caries Res* 2009; **43**: 119–125.
85. Bartlett D, Ganss C, Lussi A. Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. *Clin Oral Investig* 2008; **12(Suppl 1)**: S65–S68.
86. Ahmed KE, Whitters J, Ju X et al. Clinical monitoring of tooth wear progression in patients over a period of one year using CAD/CAM. *Int J Prosthodont* 2017; **30**: 153–155.
87. Wilder-Smith CH, Wilder-Smith P, Kawakami-Wong H et al. Quantification of dental erosions in patients with GERD using optical coherence tomography before and after double-blind, randomized treatment with esomeprazole or placebo. *Am J Gastroenterol* 2009; **104**: 2788–2795.
88. Harris R, Gamboa A, Dailey Y, Ashcroft A. One-to-one dietary interventions undertaken in a dental setting to change dietary behaviour. *Cochrane Database Syst Rev* 2012; **3**: CD006540.
89. Fox C. Evidence summary: how can dietary advice to prevent dental erosion be effectively delivered in UK general dental practice? *Br Dent J* 2010; **208**: 217–218.
90. O'Sullivan EA, Curzon ME. A comparison of acidic dietary factors in children with and without dental erosion. *ASDC J Dent Child* 2000; **67**: 186–192.
91. Edwards M, Ashwood RA, Littlewood SJ et al. A videofluoroscopic comparison of straw and cup drinking; the potential influence on dental erosion. *Br Dent J* 1998; **185**: 244–249.
92. Jenkins GN, Edgar WM. The effect of daily gum-chewing on salivary flow rates in man. *J Dent Res* 1989; **68**: 786–790.
93. Paice EM, Vowles RW, West NX, Hooper SM. The erosive effects of saliva following chewing gum on enamel and dentine: an ex vivo study. *Br Dent J* 2011; **210**: E3.
94. Gedalia I, Ionat□Bendat D, Ben□Mosheh S, Shapira L. Tooth enamel softening with a cola type drink and rehardening with hard cheese or stimulated saliva in situ. *J Oral Rehabil* 1991; **18**: 501–506.

95. Ganss C, Klimek J, Schäffer U, Spall T. Effectiveness of two fluoridation measures on erosion progression in human enamel and dentine in vitro. *Caries Res* 2001; **35**: 325–330.
96. Davis WB, Winter PJ. The effect of abrasion on enamel and dentine and exposure to dietary acid. *Br Dent J* 1980; **148**: 253–256.
97. Attin T, Knöfel S, Buchalla W, Tütüncü R. In situ evaluation of different remineralization periods to decrease brushing abrasion of demineralized enamel. *Caries Res* 2001; **35**: 216–222.
98. Bartlett DW, Lussi A, West NX et al. Prevalence of tooth wear on buccal and lingual surfaces and possible risk factors in young European adults. *J Dent* 2013; **41**: 1007–1013.
99. Public Health England. *Delivering Better Oral Health: An Evidence-based Toolkit for Prevention*. 4th edn. London: PHE; 2021. https://www.gov.uk/government/publications/delivering-better-oral-health-an-evidence-based-toolkit-for-prevention.
100. Ganss C, Schlueter N, Hardt M et al. Effect of fluoride compounds on enamel erosion in vitro: a comparison of amine, sodium and stannous fluoride. *Caries Res* 2008; **42**: 2–7.
101. Lussi A, Megert B, Eggenberger D, Jaeggi T. Impact of different toothpastes on the prevention of erosion. *Caries Res* 2008; **42**: 62–67.
102. Lussi A. Dental erosion – novel remineralizing agents in prevention or repair. *Adv Dent Res* 2009; **21**: 13–16.
103. Rees J, Loyn T, Chadwick B. Pronamel and tooth mousse: an initial assessment of erosion prevention in vitro. *J Dent* 2007; **35**: 355–357.
104. Hooper SM, Newcombe RG, Faller R et al. The protective effects of toothpaste against erosion by orange juice: studies in situ and in vitro. *J Dent* 2007; **35**: 476–481.
105. Rahiotis C, Vougiouklakis G. Effect of a CPP-ACP agent on the demineralization and remineralization of dentine in vitro. *J Dent* 2007; **35**: 695–698.
106. Azzopardi A, Bartlett DW, Watson TF, Sherriff M. The surface effects of erosion and abrasion on dentine with and without a protective layer. *Br Dent J* 2004; **196**: 351–354.
107. Bartlett D, Sundaram G, Moazzez R. Trial of protective effect of fissure sealants, in vivo, on the palatal surfaces of anterior teeth, in patients suffering from erosion. *J Dent* 2011; **39**: 26–29.
108. Cheaib Z, Lussi A. Impact of acquired enamel pellicle modification on initial dental erosion. *Caries Res* 2011; **45**: 107–112.
109. Mehta SB, Banerji S, Millar BJ, Suarez-Feito JM. Current concepts on the management of tooth wear: part 4. An overview of the restorative techniques and dental materials commonly applied for the management of tooth wear. *Br Dent J* 2012; **212**: 169–177.
110. Satterthwaite JD. Tooth surface loss: tools and tips for management. *Dent Update* 2012; **39**: 86–96.
111. Hemmings KW, Darbar UR, Vaughan S. Tooth wear treated with direct composite restorations at an increased vertical dimension: results at 30 months. *J Prosthet Dent* 2000; **83**: 287–293.
112. Carlsson GE, Ingervall B, Kocak G. Effect of increasing vertical dimension on the masticatory system in subjects with natural teeth. *J Prosthet Dent* 1979; **41**: 284–289.
113. Hemmings K, Truman A, Shah S, Chauhan R. Tooth wear guidelines for the BSRD Part 2: Fixed management of tooth wear. *Dent Update* 2018; **45**: 590–600.
114. Hemmings K, Truman A, Shah S, Chauhan R. Tooth wear guidelines for the BSRD Part 3: Removable management of tooth wear. *Dent Update* 2018; **45**: 687–696.
115. Dahl BL, Krogstad O. The effect of a partial bite-raising splint on the inclination of upper and lower front teeth. *Acta Odontol Scand* 1983; **41**: 311–314.
116. Poyser NJ, Porter RW, Briggs PF et al. The Dahl concept: past, present and future. *Br Dent J* 2005; **198**: 669–676.
117. Gough MB, Setchell DJ. A retrospective study of 50 treatments using an appliance to produce localised occlusal space by relative axial tooth movement. *Br Dent J* 1999; **187**: 134–139.

118. Redman CD, Hemmings KW, Good JA. The survival and clinical performance of resin–based composite restorations used to treat localised anterior tooth wear. *Br Dent J* 2003; **194**: 566–572.
119. Poyser NJ, Briggs PF, Chana HS et al. The evaluation of direct composite restorations for the worn mandibular anterior dentition – clinical performance and patient satisfaction. *J Oral Rehabil* 2007; **34**: 361–376.
120. Milosevic A, Burnside G. The survival of direct composite restorations in the management of severe tooth wear including attrition and erosion: a prospective 8-year study. *J Dent* 2016; **44**: 13–19.
121. Gow AM, Hemmings KW. The treatment of localised anterior tooth wear with indirect Artglass restorations at an increased occlusal vertical dimension. Results after two years. *Eur J Prosthodont Rest Dent* 2002; **10**: 101–105.
122. Gulamali AB, Hemmings KW, Tredwin CJ, Petrie A. Survival analysis of composite Dahl restorations provided to manage localised anterior tooth wear (ten year follow-up). *Br Dent J* 2011; **211**: E9.
123. Varma S, Preiskel A, Bartlett D. The management of tooth wear with crowns and indirect restorations. *Br Dent J* 2018; **224**: 343–347.
124. Nohl FS, King PA, Harley KE, Ibbetson RJ. Retrospective survey of resin-retained cast-metal palatal veneers for the treatment of anterior palatal tooth wear. *Quintessence Int* 1997; **28**: 7–14.
125. Chana H, Kelleher M, Briggs P, Hooper R. Clinical evaluation of resin-bonded gold alloy veneers. *J Prosthet Dent* 2000; **83**: 294–300.
126. Eliyas S, Martin N. The management of anterior tooth wear using gold palatal veneers in canine guidance. *Br Dent J* 2013; **214**: 291–297.
127. Al-Khayatt AS, Ray-Chaudhuri A, Poyser NJ et al. Direct composite restorations for the worn mandibular anterior dentition: a 7-year follow-up of a prospective randomised controlled split-mouth clinical trial. *J Oral Rehabil* 2013; **40**: 389–401.
128. Mehta SB, Banerji S, Millar BJ, Suarez-Feito JM. Current concepts on the management of tooth wear: part 2. Active restorative care 1: the management of localised tooth wear. *Br Dent J* 2012; **212**: 73–82.
129. Bartlett D, Sundaram G. An up to 3-year randomized clinical study comparing indirect and direct resin composites used to restore worn posterior teeth. *Int J Prosthodont* 2006; **19**: 613–617.
130. Schmidlin PR, Filli T, Imfeld C et al. Three-year evaluation of posterior vertical bite reconstruction using direct resin composite – a case series. *Oper Dent* 2009; **34**: 102–108.
131. Saunders WP, Saunders EM. Prevalence of periradicular periodontitis associated with crowned teeth in an adult Scottish subpopulation. *Br Dent J* 1998; **185**: 137–140.
132. Slagsvold O, Karlsen K. The control mechanism of tooth eruption. An experimental study in adult monkeys. *Eur J Orthod* 1981; **3**: 263–271.
133. Rivera-Morales WC, Mohl ND. Relationship of occlusal vertical dimension to the health of the masticatory system. *J Prosthet Dent* 1991; **65**: 547–553.
134. Smales RJ, Berekally TL. Long-term survival of direct and indirect restorations placed for the treatment of advanced tooth wear. *Eur J Prosthodont Rest Dent* 2007; **15**: 2–6.
135. Hamburger JT, Opdam NJ, Bronckhorst EM et al. Clinical performance of direct composite restorations for treatment of severe tooth wear. *J Adhes Dent* 2011; **13**: 585–593.
136. Ettinger RL, Taylor TD, Scandrett FR. Treatment needs of overdenture patients in a longitudinal study: five-year results. *J Prosthet Dent* 1984; **52**: 532–537.

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Published: September 2021