6 Physiology of Erosive Tooth Wear and Relationship with Dentine Hypersensitivity

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6.1 Introduction

Erosive tooth wear is defined as the chemo-mechanical removal of dental tissue in the absence of bacteria [1]. Although the term 'tooth wear' is commonly used in the United Kingdom, the term erosive tooth wear is increasing globally to recognise that, clinically severe wear rarely occurs without an underlying acid component acting alongside mechanical removal. Dietary acids and gastric acids are the most common underlying acid aetiologies in erosive tooth wear. Although we all consume dietary acids, it is when we consume them at an increased frequency or for prolonged durations that they can contribute to erosive tooth wear [2]. Although gastric acid erosion is less common, gastric acids are stronger acids (producing more protons in solution) and have been shown to bypass the protective salivary pellicle at a faster rate [3]. In common with extrinsic dietary acid exposures, it is the timing, periodicity and frequency of intraoral acid exposures which influence the clinical pattern and progression of tooth substrate loss.

Dentine hypersensitivity is characterised by short, sharp, transient pain in response to stimuli which cannot be attributed to any other form of pathology [4]. The hydrodynamic theory originally described by Gysi in the 1900's describes this pain to be a result from fluid movement within dentine tubules. Odontoblast bodies are located within the pulpal aspect of the dentine with the odontoblast processes extending a few microns into the tubule. The rest of the tubule is filled with fluid. Stimulating this fluid, either through thermal, tactile or osmotic stimuli will cause fluid movement. When this movement is large enough it will stimulate the A delta fibres in the pulp periphery, just below the odontoblast cell bodies, triggering sharp pain.

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A. Eder, M. Faigenblum (eds.), *Tooth Wear*, BDJ Clinician's Guides,
https://doi.org/10.1007/978-3-030-86110-0_6

Both erosive tooth wear and dentine hypersensitivity are increasing in prevalence, particularly amongst the younger populations [5, 6]. This chapter will cover the early wear process and give reasons why acid exposure or harsh mechanical wear can possibly lead to dentine hypersensitivity.

6.2 A Recap on Enamel Physiology with Respect to Tooth Wear

Enamel is one of the hardest structures in the human body. It has roughly 96% mineral content in the form of substituted calcium hydroxyapatite, 3% water and 1% organic tissue. Hydroxyapatite crystals are arranged in key-hole shaped prism structures which run perpendicular to the outermost layer of enamel. On tooth eruption, the outside layer of enamel, is a relatively disorganised structure, containing no prisms and hence is called the 'aprismatic layer' which is up to 100 μm deep. It has the highest mineral content, containing fluoride and phosphate in the form of fluorohydroxyapatite [7]. This layer has been shown to offer the greatest protection against both acid and mechanical challenges. The enamel prisms are separated by the inter-prismatic layer. The inter-prismatic layer is composed of less organised hydroxyapatite, but with a relatively high proportion of proteins and organic tissue.

Due to its high mineral content, enamel is very strong against mechanical challenges but weak against acidic challenges.

6.3 A Recap on Dentine Physiology with Respect to Wear

Dentine is a permeable structure composed of 75% mineral, 20% organic material and 5% water. Each odontoblast forms one tubule, laying down mineral tissue around the cell processes and body. The diameter of tubules varies between 2–4 μm with tubule density increasing towards the pulp [8]. Closest to enamel is the mantle dentine, which is roughly 15–30 μm thick. Mantle dentine, similar to the aprismatic layer in enamel, is disorganised and only has a few thin, curved tubules. The bulk of dentine consists of intertubular dentine, a type I collagen-rich structure. Each collagen fibril is 100–120 nm thick in diameter. This collagen network is moisture rich and elastic resulting in reduced hardness and higher susceptibility to wear, such as abrasion. Dentine apatite crystals are needle-like structures, 3–4 nm thick and located either along the surface of the collagen fibrils or filling the empty intracollagen spaces [8].

Peritubular dentin is formed within the lumen of the tubules. It is formed by a network of proteins and apatite crystals with no collagen fibrils. Peritubular dentin is highly mineralised making it more susceptible to an acid challenge.

Secondary dentine is laid down slowly within the pulp chamber after the tooth has erupted and often contains an irregular distribution of dentine tubules. 'Tertiary', 'reparative', or 'reactionary' dentine laid down in response to irritation, such as dental caries often contains no dentine tubules [8].

In order for dentine hypersensitivity to occur, dentine needs to be exposed, the tubules need to be patent and, more than likely, widened [9]. Erosive tooth wear can expose the dentine through removing enamel and the protective smear layer. Once the mineralised peritubular dentine is exposed, it can then widen the tubules. These are discussed in the following sections below.

6.4 Defence Systems Against Dentine Hypersensitivity and How Erosive Tooth Wear Overcomes Them

6.4.1 First Layer of Defence Against Dentine Hypersensitivity: Enamel

Enamel is the first line of defence against erosive tooth wear. In the absence of acid challenges, enamel is the hardest structure in the body and can withstand a good degree of mechanical attrition and abrasion.

When teeth are exposed to an acidic environment, minerals are released from the surface causing softening of the outermost layer between 0.2 and 2 μm thick [10, 11]. When acid encounters a natural enamel surface, there is initial breakdown of the interface between the prism and interprismatic layer widening the prism [12]. Thereafter, the prism cores are richer in carbonate, making them more susceptible to erosion. Liquid can move through enamel prisms of the teeth causing subsurface softening [13]. In the absence of further erosive challenges or mechanical removal, there is possibility for minerals to form new ionic bonds in the acid softened enamel [14]. However, complete remineralisation of enamel after a severe acid challenge is very difficult to achieve in vivo and more recent research has suggested that proteins in saliva can interfere with remineralisation [15]. In one clinical study whereby teeth scheduled for extraction were acid etched for 2 min with 50% phosphoric acid, microscopic evidence of the etching was still present when the teeth were extracted 90 days later [16]. Enamel samples eroded with dietary acid challenges have also been found to not regain their original hardness after being left in the mouth undisturbed for 7 days [17].

As the acid challenge continues, layers of enamel become softened and are further eroded, resulting in bulk irreversible enamel loss. When softened, enamel is particularly susceptible to mechanical forces [10]. Toothbrushing, in the absence of an acid challenge, causes a negligible amount of enamel tooth structure loss when a low or medium abrasivity toothpaste is used [18]. However, in the presence of an acid challenge, minimal abrasive or mechanical wear can remove any softened enamel. Any attritive or abrasive wear will occur at a much faster rate in the presence of an acid, hence the reason why severe tooth wear rarely presents with a single aetiology.

The maximum bite forces generated by humans are roughly 700 N [19], roughly equivalent to the force on the ground generated by a 70 kg person. The typical forces seen when chewing or swallowing are surprisingly high at roughly 36–41% of the maximum force (forces of between 250–290 N were observed) [20]. Bruxism forces are estimated to be in the range of 30% of maximum (approximately 220 N) [21]. Enamel is remarkably well equipped to deal with these forces with many

laboratory studies finding negligible enamel wear in the absence of an acid challenge. However, aggressive wear can occur on acid softened enamel using relatively low forces [22].

A Quick Note on Abfraction

Abfraction has been defined as tooth wear along the cemento-enamel junction (CEJ) caused by eccentric loading of the tooth. The CEJ is a weak point on the tooth. The enamel is at its thinnest and the junction with cementum is a known mechanical weak point as observed by multiple studies in finite element analysis studies. It was a commonly held theory that repeated flexure of the tooth led to microfractures of the dental tissues causing non-carious cervical lesions. However, there is limited evidence to suggest that these lesions have an occlusal aetiology. They rarely occur on the lingual surfaces of teeth and are not correlated with those who have eccentric contacts or high levels of bruxism [23]. *These lesions are easily replicated in the laboratory through combinations of abrasion and erosion, whereas it is almost impossible to replicate them using physiological levels of eccentric loading or occlusal contacts. This has led to the international consensus that 'abfraction' is not an aetiological factor in erosive tooth wear* [24]. *Restoring these lesions is notoriously difficult and it is highly likely that occlusal factors play a role in the longevity of these restorations. However, occlusal factors are unlikely to play a role in the initiation of tooth wear lesions although they may exacerbate them. Instead, clinical examinations should focus on identifying erosive and abrasive aetiologies.*

Once enamel is worn away, dentine is exposed and the risk for dentine hypersensitivity increases. However, exposure of dentine does not always lead to dentine hypersensitivity. This is because fluid movement within the tubules is limited by both the smear layer on the surface of the dentine and secondary/tertiary dentine on the pulpal border of the dentine. Both are affected by erosive tooth wear and are discussed in the following two sections.

6.4.2 Second Line of Defence Against Dentine Hypersensitivity: The Smear Layer

If the hydrodynamic theory of dentine hypersensitivity is correct, whereby pain is caused by rapid changes in fluid within the tubule, blocking or sealing the tubule should prevent pain. The naturally occurring smear layer, formed as a consequence of intraoral exposure, is a thin 'loose' layer of organic collagen and proteins coming from saliva and dentine particles which form an adherent matrix for mineralised tissue [25]. This smear layer is excellent at occluding dentinal tubules and limiting dentine hypersensitivity [26]. The smear layer is remarkably stable under normal oral conditions and several studies have shown that the smear layer is a better sealant than adhesive resins under normal oral conditions [27]. It is not removed by toothbrushing

with a normal force and replenishes in a non-aggressive environment [28]. However, the smear layer is highly susceptible to acid challenges [29]. In day-to-day clinical dentistry, we are used to removing the smear layer with acid for our bonding procedures. Laboratory studies have shown that dietary acid challenges can also dissolve this protective layer [30]. One clinical study observed that consuming a dietary acid within the previous hour was related to increased clinical dentine hypersensitivity on examination [31]. Once the smear layer is removed, the mineralised peri-tubular dentine is exposed. This means that continued acid challenges will widen the dentinal tubules, making the tubules more difficult to occlude [32]. The habit of consuming dietary acids over a long period of time has been associated with increased levels of dentine hypersensitivity compared to those who consumed dietary acids more frequently but over a short period of time [33]. Aggressive mechanical challenges have also been shown to remove the smear layer. Toothbrushing with a force of 400 g (normal brushing force is 100–300 g), has been shown to increase the number of patent dentine tubules [34]. In contrast to enamel, attritional forces alone have the capacity to remove exposed dentine. This again is worse on acid softened dentine [35]. To date, there have been no studies demonstrating that attritional forces can remove the smear layer, however this may also be a possibility.

A Quick Note on Saliva

The relationship between saliva (Chap. 4), erosive tooth wear and dentine hypersensitivity is not straightforward. The salivary pellicle is a protein layer that forms rapidly and spontaneously through protein adsorption to the tooth surface. Whilst it is essential for the subsequent colonisation of the tooth surface by bacteria to form a biofilm, it can also be considered to provide a protective barrier to the dental surface. The thickness and protein content of this pellicle has been shown to be related to the level of protection conferred which may explain variation between individuals [36]. *There is also evidence that saliva protects against dietary acids to a greater extent than stomach acids* [5]. *However, recent evidence suggests that saliva's ability to remineralise eroded dental tissue is limited. Both artificial and natural saliva have been observed to increase the surface hardness of enamel and dentine following an erosive challenge* [37–39]. *However, although some degree of rehardening occurs, recent papers are reporting that limited meaningful remineralisation occurs. Studies have shown no additional protective effect against abrasion with 2–4 h intraoral remineralisation time* [15, 40]. *Proteins within saliva can interfere with the remineralisation process, binding to enamel and acting as a barrier to remineralisation* [15, 41]. *Laboratory studies have demonstrated that saliva offers a protective role against erosive challenges resulting in less bulk loss but leaving behind a softer structure* [42]. *Those with reduced salivary flow or altered salivary composition have anecdotally reported to experience increased dentine hypersensitivity and this may be due to their reduced ability to form a sealing smear layer. Although further work is required in this area, the evidence would suggest that saliva has an important role acting as a barrier but a limited role in any reparative mechanism.*

6.4.3 Third Line of Defence Against Dentine Hypersensitivity: Secondary/Tertiary Dentine

During active abrasion, attrition or particularly erosion, dentine hypersensitivity is commonly reported [31, 33, 43]. However, it is interesting that dentine hypersensitivity is not the most common presenting complaint amongst those with severe erosive tooth wear [44]. Clinical cases with high levels of exposed dentine and near pulpal exposures can often be vital but not hypersensitive [44]. In the same respect, patients may present with dentine hypersensitivity and no tooth wear [33]. When there are normal rates of physiological tooth wear, this allows time for the pulp to lay down reparative or secondary dentine. This less-organised dentine has reduced fluid flow within the tubules and reduces dentine hypersensitivity as the dental tissue is gradually worn away [45]. This would explain how severely worn teeth with very little protective dentine can remain both vital and with normal or reduced levels of sensitivity. If erosive tooth wear progresses at a relatively stable rate, periapical pathology that is asymptomatic is commonly observed [44].

6.5 Prevention

6.5.1 Limiting Acid Exposure

Given that acids, either extrinsic from the diet or intrinsic from the stomach, remove protective tooth substance and the smear layer, it could be argued that limiting exposure to acids is the single most important aspect of preventing dentine hypersensitivity and erosive tooth wear. Duration of acid exposure [33] in addition to frequency of acid exposure [43] has been shown to be associated with dentine hypersensitivity and it is worth counselling patients on both of these aspects. It is important to check for sources of dietary acids that are uncommonly acknowledges by patients such as fruit teas, fruit-flavoured water and fruit-flavoured or acidic sweets. It is also important to check for fruit grazing habits such as chopping fruit into small pieces or snacking slowly on punnets of grapes/berries.

6.5.2 Limiting Aggressive Mechanical Exposure

The amount of dentine removed has been shown to be related to the frequency of brushing, the abrasivity of the dentifrice and the force of brushing. Although there are difficulties in assessing toothbrushing habits, evidence suggests that the first toothbrushing site receives the most brushing time and the last site receives the least. Due to difficulties with accurately measuring wear and comparing dentine hypersensitivity in vivo, no high quality randomised controlled trials have been performed assessing the impact of these variables. Laboratory and in situ

evidence would suggest that choosing a low-medium abrasivity toothpaste and using a low toothbrushing force would minimise dentine hypersensitivity. Brushing immediately after an acid challenge does increase the tooth structure removed. However, little is known and about whether this tooth structure would not be removed under normal physiological functioning. Regardless of the timing of when you brush, it is important to choose less aggressive brushing techniques [46].

The role of bruxism and clenching in dentine hypersensitivity is underinvestigated. The additional tactile stimulation may stimulate the A delta fibres or continued low grade stimulation may cause a lower firing threshold. Some epidemiological studies have observed parafunctional behaviour to be associated with dentine hypersensitivity [47, 48]. From a clinical perspective, it would be prudent to attempt to control parafunctional activity to prevent erosive tooth wear. This may in turn impact on dentine hypersensitivity.

6.5.3 The Role of Fluoride

Although fluorapatite is less soluble than hydroxyapatite, fluoride incorporation into the dental structure has been observed to have a relatively weak protective effect against acid erosion compared with caries [49]. Although the presence of available fluoride ions does have a protective effect [49], the presence of calcium fluoride deposits may also be limited with fluoride covering no more than 40% of an enamel surface even under optimum conditions [50]. Furthermore, retention of fluoride precipitates is unlikely during repeated or severe erosive challenges, even when high fluoride varnish is used [51, 52]. It is likely that fluoride alone will not prevent erosive tooth wear and dentine hypersensitivity in aggressive acidic environments but remains an essential part of a holistic prevention plan.

6.5.4 The Role of Tubule Occluding Agents

Tubule occluding agents, predominantly found in dentifrices, can be very effective at occluding dentinal tubules. However, the stability of these agents is not ideal, particularly with erosion and abrasion challenges. Frequent application is often necessary with symptoms returning when use ceases. A recent systematic review and meta-analysis supported the use of toothpastes containing potassium, stannous fluoride, potassium and strontium, potassium and stannous fluoride, calcium sodium phosphosilicate, arginine, and nanohydroxyapatite to relieve dentine hypersensitivity [53]. There was limited evidence to suggest that strontium and amorphous calcium phosphate were effective tubule occluding agents [53]. There is a growing community of clinicians advocating for the use of lasers to occlude tubules. However, this is expensive and the evidence is limited to suggest that lasers offer any advantage over conventional topical desensitising agents [54].

6.5.5 The Role of Dentine Bonding Agents

The use of dentine bonding agents to prevent dentine hypersensitivity and tooth wear has also been tested. Although the etch and rinse technique removes the smear layer and opens the tubules, it then attempts to seal them by hybridising with the inter-tubular dentine to form strong retentive plugs. These plugs would theoretically prevent fluid movement thus stopping any pain. However, dentine adhesive resins are not effective at entirely reducing dentine permeability, particularly when compared with the smear layer [27]. They do not last for a long period of time when exposed intraorally, and are not sufficient to reduce erosive tooth wear progression [55]. Once the dentine bonding agent is lost, it will leave behind a widened tubule with decreased peritubular dentine, increasing the sensitivity. Acid etching the surface does erode a significant amount of tooth structure and there is also difficulty in deciding which surface to cover with dentine bonding agent. Etching only the affected area is difficult, potentially painful for the patient and you risk damaging the surrounding dental tissue. Therefore, dentine bonding agents can only be advocated when all other preventive techniques have failed.

6.6 Conclusion

Both erosive tooth wear and dentine hypersensitivity are complex, multi-factorial conditions. However, the underlying processes for both conditions are inter-related with overlapping aetiologies. There is increasing evidence that presenting with dentine hypersensitivity is a sign of active erosive wear. If a patient presents with dentine hypersensitivity in early life, it would be prudent to thoroughly examine their diet, medical history and oral hygiene habits to rule out risk factors for erosive tooth wear. Documenting their current clinical wear status at this stage, either through a clinical index such as the Basic Erosive Wear Examination (BEWE), an intraoral scan, study models or intraoral photographs may aid in patient education and the early detection of erosive tooth wear progression.

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