Clinical insight

Occlusion and cervical lesions: a controversial connection
By Dr Gary Unterbrink

The problems at the cervical aspect of teeth are prevalent. Patients retain their teeth longer due to advancements in prevention and the increase in endodontic treatment. Most of us work in countries with an aging population. Sensitivity, stained restoration margins, aesthetic compromises; how many of your patients have these problems?

Introduction: Cervical lesions are multi-factorial
The loss of tooth substance at the necks of the teeth clearly has a variety of causes (1-5). No one questions the influence of the toothbrush and the brushing technique, acid erosion and caries as additional causes are also universally accepted. It is, however, nearly impossible to cause a cervical defect in enamel with a toothbrush alone, no matter which toothpaste is used (6-9). The interaction of exposure to acid and the time interval until brushing have been elegantly shown (10, 11). A toothbrush can indeed share responsibility for enamel loss, if used soon after the acid exposure.

In general, no significant correlation has been found in the cited studies in relation to the brushing technique, although interestingly, patients who brush their teeth several times per day have a greater tooth substance loss than those who brush only once per day (12). Everything in life can be done in excess!

The typical morphology of abrasion and erosion lesions has been described, and these guidelines are helpful when we are evaluating and advising our patients (13, 14). The clinical morphology does not always permit immediate determination of the aetiology, since combinations of the causal factors alter the form. Determination of the cause (or causes) is established during the conversation with the patient concerning habits and diet.

The influence of occlusion remains controversial. The first theoretical models were proposed more than 100 years ago, but are generally still regarded as theories. Here we will examine this relationship more closely.

Terminology
Many different expressions have been used to describe cervical defects. Some of them basically define the cause; cervical abrasion implies the toothbrush, cervical erosion implicates dietary acid. One frequently finds the word ‘idiopathic’, essentially meaning ‘but we are not really sure’.

Patients in California suffer from DCS: Dental Compression Syndrome. This sounds pretty serious, and probably requires expensive therapy (this does not detract from the excellent work of Dr McCoy). Some authors seem to call all defects abfractions, a word which points toward mechanical fracture, although it should be clear that many lesions have other causes. ‘NCCD’ is also seen frequently in the literature, a non-carious-cervical-defect. This is purely descriptive, and while it excludes caries, it is intentionally used in order to avoid designating an aetiology. ‘Angular cervical defects’ is also descriptive and defects with sharp borders in enamel and dentin do require closer examination.

Tooth anatomy
The cervix of the tooth has often been described
as a “locus minoris resistencia” (15). For example, there are clear micro-anatomical differences at the enamel-dentin interface if compared to cusp tips. Cervical enamel is poorly bonded to the dentin and breaks off fairly easily, a phenomenon which all dentists have observed when extracting teeth. The frequency of developmental defects is higher in cervical enamel, and the proportion of organic material is higher (16).

The hard and brittle enamel covers the relatively soft and flexible dentin. The deformation of dentin with fairly small forces is documented in countless studies (17-19). Laboratory investigations with cervical restorations in extracted teeth are interesting. If an occlusal load is applied, more gaps and higher microleakage is the result, a clear proof of deformation (20-23). It is also worthy of note that the elasticity of dentin varies with the position of the applied load; the elasticity modulus is approximately 14 GPa if the tooth is loaded mesially or distally, but only 9 GPa if bent in a buccal or lingual direction (24). A final comment on elasticity; the elasticity modulus of dentin is lower than that of maple wood (25). Look at the trees during the next storm and think about teeth. Enamel and dentin are anisotropic; i.e. their physical properties such as flexibility or strength change depending on the direction of load (26-30). Enamel can be fractured quite easily parallel with the prisms, but is much stronger if the load is perpendicular to them. Dentin also has a structure, in this case it can be fractured perpendicular to the tubules more easily than parallel with them.

Understanding anisotropic behaviour is important when we try to interpret the scientific papers. Occlusion is a mechanical stress. The effect of this stress will depend on many factors; these include the anatomy of the root, the level of bony support, the force and angle of loading, etc. The ‘weakest link in the chain’ will suffer and will vary from patient to patient. Bone can be resorbed, often followed by gingival recession. The enamel can be abraded and we find wear facets. Teeth with narrow cervical cross-sections can bend more easily, perhaps this can explain the higher incidence of angular cervical defects in oriental populations. In any event, we know that teeth bend when we bite on them. How does this relate to cervical lesions?

Deformation and stress
Stress concentrations during deformation are often investigated with Finite Element Analysis (FEA).

FEA is a design tool of mechanical engineers and is routinely employed for the design of dams, skyscrapers, bridges, airplanes, etc.

In dental studies using FEA, upper premolars are generally modelled. These are the teeth with the highest incidence of cervical lesions. If the periodontal support is normal, we find maximum cervical stress just coronal to the CEJ, precisely the position of the enamel margins of angular cervical defects. If the bone level is reduced, the stress moves further apically (31, 32).

A plausible explanation for the location of cervical defects, on buccal surfaces rather than palatal or lingual, can also be found in FEA studies. Newer computer programmes permit modelling anisotropic behaviour and if the correct anatomy of the teeth has been observed, including the asymmetrical dentin, then the highest stress concentrations are always found in buccal cervical enamel (33, 34).

Despite the clear scientific evidence concerning the role of occlusion in initiation and progression of cervical defects, many dentists remain sceptical.”
Additional information concerning deformation is provided by laser interferometry. This method only shows surface deformation, but with extreme accuracy and can be done with real teeth. Here also, the major deformation is found at the buccal cervical area, irrelevant of load position (35).

**Stress corrosion**  
The phenomenon of stress corrosion can be found in every aspect of material science. Materials under load, in combination with a corrosive milieu, demonstrate accelerated fatigue and crack propagation. The best known example in dentistry is ceramic; the fracture resistance decreases significantly and continuously over time, until the critical limit is reached and the restoration breaks while the patient is eating a piece of bread (36).

Laboratory tests with extracted teeth show an increase in acid erosion or toothbrush abrasion if the teeth are subjected to occlusal stress simultaneously. Caries progression is also accelerated by occlusal force (37-40). This is particularly important for patients with inadequate hygiene (acid from plaque) or patients who consume acidic dietary products followed by intensive toothbrushing.

Here we find the explanation why a specific tooth has a large cervical defect and adjacent teeth have none:  
- Toothbrush alone = almost no enamel loss  
- Acid (from any source) + toothbrush = increased enamel loss  
- Acid (from any source) + toothbrush + occlusal force = significant enamel loss

Enamel cracks which progress to the DEJ followed by fracture of large sections of cervical enamel, as proposed by some authors, probably does not occur frequently clinically (41, 42). Whether tooth deformation causes micro-cracks or piezoelectric charges and hydrolysis is a subject we can leave for the scientists (43, 44).

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Epidemiological studies have repeatedly established an interesting correlation: teeth with mobility do not have cervical defects (45-47). A tooth that moves does not bend. Cervical defects can also be created in the laboratory with occlusal loading in an acidic environment, without a toothbrush, toothpaste or bacteria (48). There could hardly be clearer evidence that teeth are subject to stress corrosion.

**Bruxism and parafunction**  
Now we have arrived at the last source of confusion. Despite the clear scientific evidence concerning the role of occlusion in initiation and progression of caries, it is somewhat underestimated in clinical situations. The role of parafunction is therefore of particular interest. The term parafunction describes any occlusal activity that is not a normal functional activity of a particular part of the dentition.

The most common parafunctional activity is bruxism, which is defined as non-sleeping clenching, ground biting or grinding of the teeth. Bruxism is a common disorder, affecting up to 10% of the population. It is often associated with stress and anxiety and can lead to pain, wear of the teeth, and damage to the temporomandibular joint.

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**“Acid (from any source) + toothbrush + occlusal force = significant enamel loss”**

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*Figure 2: If the enamel was not missing, it would reach the gingival margin. Why is the defect on one tooth extensive, and quite small on the adjacent tooth?*

*Figure 3: A very small enamel lesion which certainly has nothing to do with a toothbrush. It correlates perfectly with the stress concentrations created by the position of the occlusal contacts.*

*Figure 4: Clearly both acid and the toothbrush are involved here. But why is the lesion on the first premolar so large and why is the cusp tip fractured?*
Numerous studies have examined the relationship between wear facets and cervical defects and have not found a correlation (49-51). Results of course depend on patient selection, a better but still not statistically significant correlation is seen if only defects with sharp borders are included. This lacking correlation is frequently cited as proof that occlusion does not play a major role. Once again, I feel we should examine this more closely.

Bruxism is classically divided into two types: grinding and clenching. The muscles responsible for closing and chewing, primarily the masseter and temporalis, are extremely strong in relation to the muscles responsible for opening and performing lateral movements, for example the lateral pterygoid. Every person in the world can press their teeth together hard enough that lateral movement is physically impossible, even if they try.

Clenching your teeth obviously will not cause wear facets, but at the same time creates significantly more stress and deformation. Many defects caused by parafunction are found on teeth without wear facets. In one clinical study the force of the occlusion was measured rather than just looking for wear facets, and in this study a significant correlation with cervical defects was found (52).

An exhaustive review of the literature concerning bruxism is beyond the scope of this short article, but it should not surprise anyone that wear facets do not correlate with TMJ problems. In fact, the opposite is true. Patients with extensive wear facets have a lower than average risk of developing craniomandibular dysfunction (53).

The high risk patients are those with cervical defects but minimal wear facets. TMJ problems, as well as chronic headaches or other symptoms, correlate with the contraction intensity of the temporalis during sleep (54). Here again, the studies often cited to negate the occlusal aetiology of cervical defects can also be used to confirm it.

Summary
Scientific publications clearly prove these facts:
• Teeth bend under load
• The maximum stress concentration from this deformation is found in the precise location of angular cervical defects
• Stress corrosion occurs in both dentin and enamel

Two clinical observations can be made:
• Neither the angles nor the form of many cervical defects can be explained with caries, acid erosion or toothbrush abrasion
• The distribution of the defects varies dramatically and single teeth are often affected while the adjacent teeth remain intact. (Does anyone really believe the patient’s oral hygiene is so strange that they ‘always’ or ‘never’ brush this single tooth?)

When the occlusion is included as a factor, these observations can be explained. The congruence of the force vectors of occlusal contacts and the angles of lesion borders cannot be accidental. The scientific evidence, at least in my opinion, is sufficient and conclusive.

Perhaps the occlusal aetiology of cervical defects will remain controversial. The extreme biological variation in anatomy, the even higher variability of human behaviour in relation to diet and oral hygiene and the overlapping causes of cervical lesions make things difficult. Occlusion itself is dynamic and changes occur both naturally through attrition and artificially with dental treatment. A new crown on an upper right molar can cause a lesion on the lower left canine, or remove the cause of an existing defect.
Parafinction does not correlate with occlusion, but it does correlate with psychological stress. This factor is also dynamic and a further complication for our clinical diagnosis and treatment planning. There is the old saying ‘You only see what you know’. When you begin to see, your clinical observations will continuously confirm the relationship between occlusion and cervical defects.

Literature

34. Palmara D. Effect of stress on acid dissolution of enamel. Dental Materials 2001;17(2);109-115
35. Staninec M, Nalla RK, Hilton JF, Ritchie RO,


I have chosen only representative papers from a large number of publications. My apologies to those whose work has not been cited.

Gary Unterbrink graduated from dental school before completing mandatory military service in an Army dental clinic in Germany. Gary then spent three years working in private practice in Regensburg, then a year at a government clinic in Austria. Fifteen years with Ivoclar-Vivadent followed, including positions as director of clinical research and later, director of professional services. In 2001, Gary joined a former department employee in private practice in Liechtenstein, while continuing to lecture. Gary has delivered more than 5,000 lectures in more than 60 different countries.

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