Non-carious cervical lesions – can terminology influence our clinical assessment?

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Key points

- The most common cause of non-carious cervical lesions (NCCLs) is abrasion combined with erosion.
- Occlusal forces may play a role in NCCL progression but are unlikely to be an aetiological agent.
- Terming these lesions 'abfraction lesions' may be misleading.

Abstract

Introduction Abfraction is a theoretical term used that has been classified as a type of non-carious cervical lesion (NCCL) and characterised by the microstructural loss of hard dental tissue in areas of high stress concentration. There is a lack of consensus among researchers and clinicians as to whether occlusal loading, particularly interferences or eccentric loading, generates sufficient tensile stress to be an aetiological factor in the loss of hard dental tissue at the cemento-enamel junction (CEJ).

Aim This narrative review article assesses the evidence behind the theory of abfraction.

Results It is difficult to control all influencing factors in a clinical trial making it challenging to generate sufficient evidence to conclusively support the theory of abfraction. There is limited evidence occlusal forces are an aetiological agent in non-carious cervical lesion development. However, if occlusal forces do play a role, the term non-carious cervical lesion is more reflective of the limited role it may play and a multifactorial aetiology.

Conclusion The term 'abfraction lesion' remains misleading and could be removed from our diagnostic vocabulary.

The introduction of abfraction into tooth wear nomenclature

Non-carious cervical lesions (NCCLs) are common clinical conditions that negatively impact the structural integrity and pulpal vitality of the tooth, as well as the aesthetical features.[1] A practice-based study observed them to be the main reason, besides caries, for the placement of restorations on permanent tooth surfaces.[2] Non-carious cervical lesion formation has long been considered a consequence of toothbrush and dentifrice abrasion with or without an erosive component.[3] In 1984, Lee and Eakle proposed a hypothetical reason for cervical wear, later termed abfraction. They proposed, based on their opinion, the lesions were a result of the tensile stress from mastication and malocclusion formed along the cervical area.[4] Smooth and round lesions from abrasion and erosion, were distinguished from the wedge-shaped angled defects seen in the cervical region of teeth. The flexure of the tooth, as seen in Figure 1, was hypothesised to propagate microcracks, increasing the likelihood of further toothwear at the cervical region.[4] In this paper we investigate the evidence behind abfraction and question whether it should be considered as an aetiological agent in tooth wear.

The cervical area of teeth has long been established as a weak area of the tooth. It has a thin layer of enamel, lower mineral content, higher protein content and Lynch *et al.*[5] demonstrated the low density of Hunter-Schreger Bands in the cervical area. The inherent weakness of the cervical region of the tooth, coinciding with its role as a fulcrum under occlusal forces was seen as a contributing factor in the appearance of the morphologically distinct lesions. McCoy also questioned the contribution of toothbrush abrasion, and suggested that bruxism may be the predominant aetiological factor behind these characteristic angled lesions at the cemento-enamel junction (CEJ).[6]

Lee and Eakle presented three case reports to support their proposal that occlusal forces and enamel flexure are primary aetiological agents in lesion formation. Despite the clear selection bias, an absence of any data and low sample size from case reports, some view their underlying arguments as valid today. They argued that occlusal forces are at play as lesions can be localised and the appearance of sharp line angles can coincide with the direction of occlusal force. If abrasion was a primary aetiological agent single lesions would be unlikely. They also observed that subgingival lesions occur where toothbrush bristles would find it difficult to reach.

Grippo termed these lesions as abfraction lesions, from the Latin words *'ab'* and *'fractio'* which together mean 'breaking away' and defined them as loss of dental hard tissue caused

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by the consistent cyclic and non-axial loading of teeth that leads to biomechanical stress, particularly at the CEJ.[7] As this theory gained momentum, further clinical characterisation of these lesions by several other authors was published without supporting data to determine their true aetiology. Sarode and Sarode described the appearance of abfraction lesions as ranging from shallow grooves to large wedge-shaped lesions,[8] while acknowledging that the theory remained unproven.

Laboratory evidence investigating abfraction

Finite element analysis (FEA), an arithmetic technique that uses computer algorithms to analyse stresses and distortions, has been extensively used to explore the stress profile of teeth when applied with varying occlusal forces from a multitude of angles. For example by Rees who loaded a computerised model of a premolar at seven different positions.[9]

Rees reported that the sampling planes A and B experienced different stresses depending on the direction of the load. The highest principal stresses in the cervical section were observed on both the lingual and buccal sides when loading forces were generated on the slopes of the cusp tips. Vertical loading generated the lowest stress numbers.[9] This has been confirmed in several other more recent and advancing FEA studies[10,11] and demonstrates that altering the position of the occlusal load in a non-axial direction increases the stress in cervical enamel.

However, FEA studies have several limitations. Firstly, they are based on computerised models, and despite technological advances, they are not clear representatives of teeth *in vivo*. FEA studies rely on the fact that the occlusal loading research is repetitive and static, whereas in reality, this is a dynamic occurrence. Secondly, most FEA studies for abfraction use two-dimensional models, whereas a three-dimensional model would be more appropriate as it can measure torsional stress too.[9,11] Thirdly, researchers have used varying force magnitudes and have allocated different properties to the materials they explore using this computerised technique. For example, some researchers have considered enamel to be an isotropic material,[9] whereas others have stated that enamel should be considered to be anisotropic.[13] When enamel is regarded as anisotropic, greater tolerance to tensile stress and occlusal loading is evident.[13]

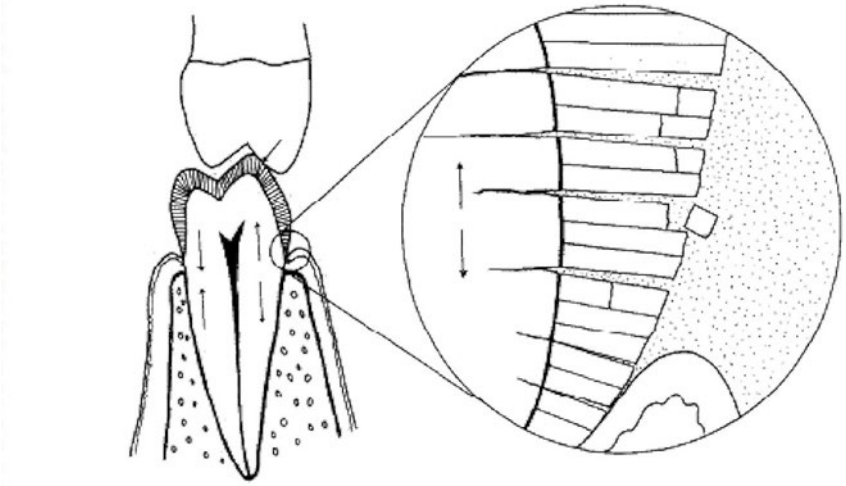

Fig. 1 Diagrammatic representation of lateral forces causing tension at the cervical region of the tooth. The magnified sector displays the disruption of the chemical bonds enamel rods. (Republished with permission of Elsevier Science and Technology Journals, from Lee W C, Eakle W S. Possible role of tensile stress in the aetiology of cervical erosive lesions of teeth. *J Prosthet Dent* 1984; 52: 374–380[4]; permission conveyed through Copyright Clearance Center, Inc.)

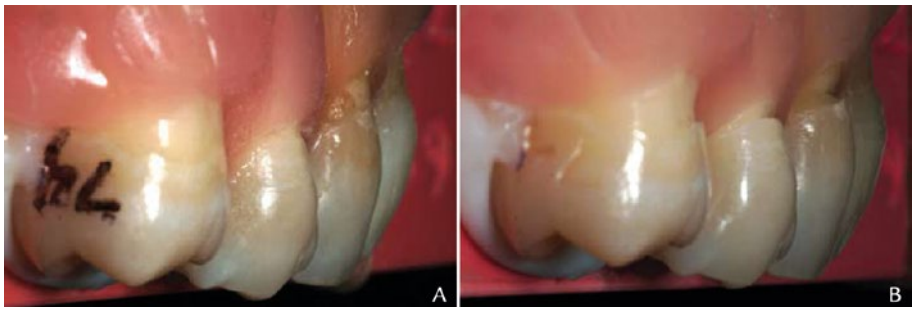

Fig. 2 Simulated 7 years of a horizontal toothbrushing technique with no occlusal loading. (Republished with permission of Elsevier Science and Technology Journals, from Dzakovich J J, Oslak R R, *In vitro* reproduction of noncarious cervical lesions. *J Prosthet Dent* 2008; 100: 1–10[16]; permission conveyed through Copyright Clearance Center, Inc.)

There is a universal problem when using computerised models to replicate physiological function. Models tend to focus on the enamel, despite the enamel layer in that area being very thin. The increased organic material in dentine would suggest that dentine can withstand occlusal loading more effectively than enamel.[14] However, one FEA study demonstrates that as the lesion advanced in depth, the stress was increased at the deepest part of the lesion,[10] potentially having implications for bonding to this region.

Interestingly, FEA studies show equal stress on both buccal and lingual sides which seems to contradict clinical evidence which indicates that NCCLs are more prevalent buccally[8,15] and lingual lesions are not commonly found.[3]

It is also worth noting that abfraction-like lesions on enamel/dentine or whole tooth samples have never been replicated under laboratory conditions using occlusal loading. In contrast, the distinct clinical appearance of sharp line angles mimicking these lesions has been replicated *in vitro* by several authors using erosion and abrasion challenges. Dzakovich replicated sharp line angles not using any occlusal forces but using abrasive dentrifices and repeated horizontal tooth brushing movements (Fig. 2).[16]

Recent studies have observed that increased tissue loss was dependent on the abrasivity of the toothpaste and the stiffness of the toothbrush.[17,18] It has also long been established in periodontology that toothbrushing can reach subgingival locations and remove plaque.[19]

Thus, the formation of lesions in subgingival areas can be expected, particularly if an underlying acidic aetiological component is also involved. The localisation of lesions, which can sometimes be limited to a single tooth in

the arch, is an interesting phenomenon. It may be due to inherent weaknesses in that individual tooth but it is also an argument for the role of eccentric occlusal forces in the aetiology of these lesions. Laboratory evidence will be biased by the fact that it is easier to investigate the interplay between abrasion and erosion, than the interplay between occlusal force and abrasion on a cervical surface. Only one small laboratory study has investigated this interplay,[20] placing either a 45 kg continuous axial load or a 45 kg intermittent non-axial load on the buccal cusp of the premolars in addition to performing abrasion. Results indicated that axial loading was associated with decreased wear at the cervical area which the researchers attributed to compressive effects of occlusal loading. No evidence of microcracking was observed. However, this was a small sample size and teeth were loaded while simultaneously being subjected to toothbrush abrasion – a situation that is unlikely to occur in reality. This area could be further investigated *in vitro*.

Finally, there is very limited evidence of microfractures when these lesions were examined under a microscope. Walter *et al.* used SEM and confocal scanning laser microscopy to examine 42 teeth with 19 'wedge-shaped' lesions and 23 'saucer-shaped' lesions. Not a single microfracture was observed, however those teeth with wedge-shaped lesions were more likely to have attritional wear. Signs of abrasion, large amounts of sclerotic dentine and dead tracts were observed in all lesions.[21]

Clinical evidence investigating abfraction

Logically bruxists and particularly those with eccentric or interfering contacts should have a higher incidence of abfraction lesions. However, this is not consistent in epidemiological studies. Studies that have found relationships between bruxism and NCCLs rarely control for abrasion or erosion factors.[22,23] A recent case control study on 280 dental students observed bruxism, protrusive interferences and non-working side interferences to be associated with NCCL formation.[24] However, the study recognised that this model did not fully predict NCCL formation and concluded that 'occlusal factors alone do not appear to be sufficient to explain the presence of NCCLs'. While toothbrushing and dietary data were collected they unfortunately did not control for it in their multivariate regression analysis.[24] One case report digitally analysed wear over 14 years and observed a strong correlation between occlusal wear and cervical wear. However, correlation is not causation and suggest similar risk factors for both sites.[25] Studies investigating all variables have tended to observe equal odds ratios or risk for excessive toothbrushing habits and occlusal factors.[26,27] For example, a case-control study on 264 participants noted an odds ratio of 8.79 (95% CI 2.87–26.77) for brushing three times a day and 4.23 (95% CI 1.52–11.70) for self-reported bruxism to being in the NCCL group. There have also been studies which have shown no association between bruxism and NCCL formation,[28] but have shown association with occlusal factors such as occlusal contact area.[29] In contrast, a pan-European study on over 3,000 participants observed buccal and lingual lesion formation to be associated with an acidic diet, not toothbrushing habits.[30] They did not investigate the role of occlusal factors. Two systematic reviews, one with quite a broad review[31] and the other with more stringent inclusion criteria,[32] explored the association between occlusal factors and NCCL formation and concluded that the current scientific evidence does not support the association between occlusal factors and NCCL formation. However, both reviews did draw attention to the presence of bias and the lack of control for confounders.

Perhaps the most interesting clinical fact is that anthropological researchers examining archeological dentitions in skulls consider non-carious cervical lesion formation as a 'modern day' pathology.[33] Prior to the introduction of modern day toothbrush and dentifrice use, non-carious cervical lesions were absent on skulls despite the presence of heavy occlusal wear. This has led anthropological researchers to question the role of occlusal forces in the aetiology of these lesions.[34,35,36,37]

Conclusion

There is little doubt that NCCLs are multifactorial in origin. The varying clinical presentation of NCCLs has been replicated in multiple laboratory studies by a combination of abrasion and erosion. Occlusal forces, particularly, eccentric occlusal loading, may play a role in the development of these lesions. However, there is good evidence to suggest that it is not a substantive role and the term 'abfraction lesion' is misleading. If the definition of attrition is expanded to include any wear resulting from tooth to tooth contact, there may be little need to include abfraction in erosive tooth wear terminology. More clinical evidence, ideally a large multi-centre study consisting of a comprehensive functional occlusal examination, comprehensive brushing assessment (including dentifrice abrasivity) and a comprehensive dietary assessment, is needed to help to determine the relative influence of each component.

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Correction to: CariesCare practice guide: consensus on evidence into practice

The original article can be found online at https://doi.org/10.1038/s41415-019-0678-8.

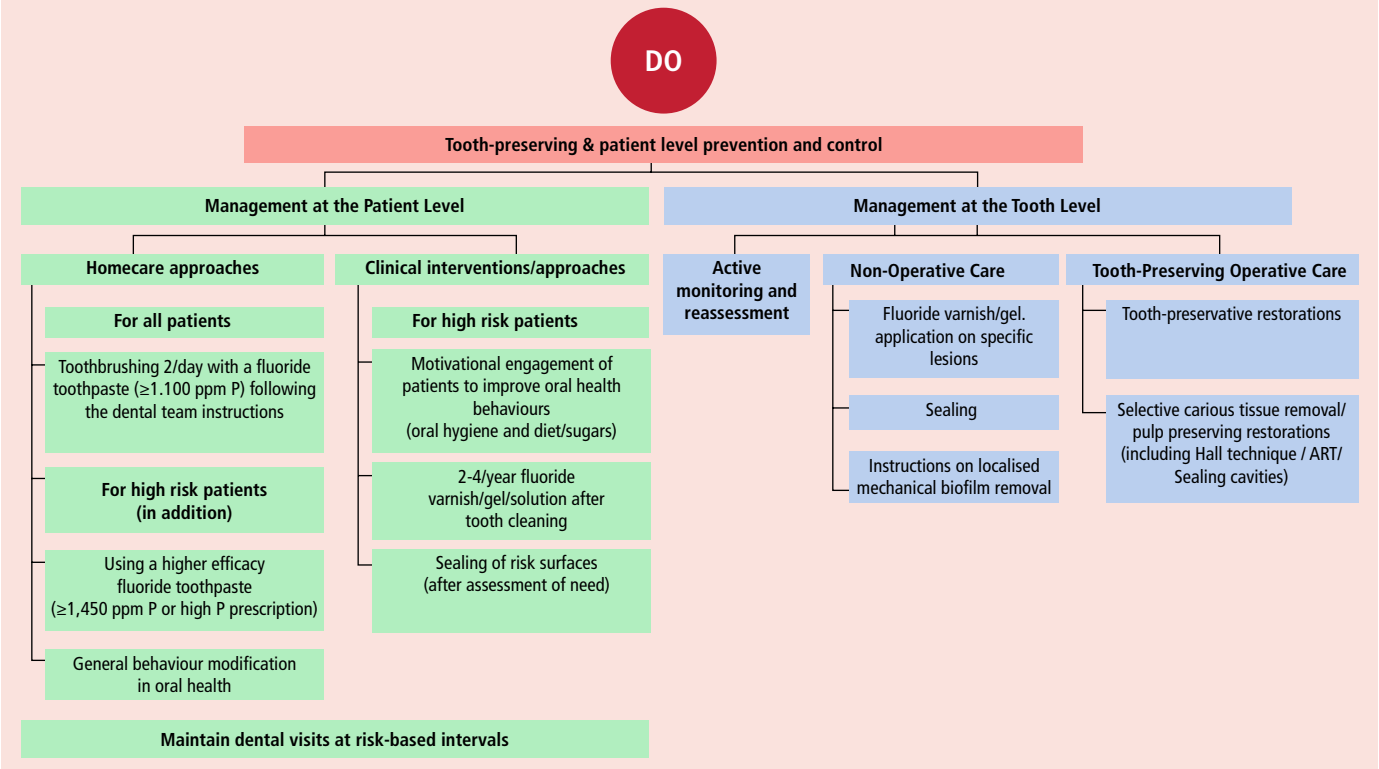
Author's correction note: Clinical article *Br Dent J* 2019; **227:** 353–362.

When this article was initially published, one of the boxes in Figure 6 had been duplicated. The correct figure is shown below:

Also, Matteo Basso's affiliation in the Acknowledgements section was incorrect. It should have read 'Matteo Basso, University of Milano, Italy'.

The authors apologise for any confusion caused by these errors.

Fig. 6 Tooth-preserving and patient-level prevention and control fowchart



Note: this figure is an illustration, not a specific prescription.