REVIEW

Australian Dental Journal 2009; 54: 2–8

doi: 10.1111/j.1834-7819.2008.01080.x

Abfraction: separating fact from fiction

JA Michael,* GC Townsend,* LF Greenwood,* JA Kaidonis*

*School of Dentistry, The University of Adelaide, South Australia.

ABSTRACT

Non-carious cervical lesions involve loss of hard tissue and, in some instances, restorative material at the cervical third of the crown and subjacent root surface, through processes unrelated to caries. These non-carious processes may include abrasion, corrosion and possibly abfraction, acting alone or in combination. Abfraction is thought to take place when excessive cyclic, non-axial tooth loading leads to cusp flexure and stress concentration in the vulnerable cervical region of teeth. Such stress is then believed to directly or indirectly contribute to the loss of cervical tooth substance. This article critically reviews the literature for and against the concept of abfraction.

Although there is theoretical evidence in support of abfraction, predominantly from finite element analysis studies, caution is advised when interpreting results of these studies because of their limitations. In fact, there is only a small amount of experimental evidence for abfraction. Clinical studies have shown associations between abfraction lesions, bruxism and occlusal factors, such as premature contacts and wear facets, but these investigations do not confirm causal relationships. Importantly, abfraction lesions have not been reported in pre-contemporary populations.

It is important that oral health professionals understand that abfraction is still a theoretical concept, as it is not backed up by appropriate clinical evidence. It is recommended that destructive, irreversible treatments aimed at treating so-called abfraction lesions, such as occlusal adjustment, be avoided.

Key words: Abfraction, non-carious cervical lesions, stress, tooth wear.

Abbreviations and acronyms: FEA = finite element analysis; GICs = glass ionomer cements; NCCLs = non-carious cervical lesions; RMGICs = resin-modified GICs.

(Accepted for publication 24 July 2008.)

INTRODUCTION

Non-carious cervical lesions (NCCLs) involve loss of hard tissue, and in some instances, restorative material at the cervical third of the crown and subjacent root surface, through processes unrelated to caries.[1] Although it is accepted that NCCLs have a multifactorial aetiology, the relative contributions of the various processes remain unclear. Currently, the most widely accepted causes of NCCLs are abrasion and corrosion, although several alternative theories have been proposed.

Abfraction, a biomechanically-based theory, is one of the most discussed and controversial of these alternative theories. Grippo[2] first used the term abfraction to refer to a process of cervical tooth structure loss, based on work completed by McCoy[3] and Lee and Eakle.[4]

Recent reviews of this field lack detail in particular areas and have not focused on the current clinical implications of abfraction. As an increasing number of articles exploring the validity of abfraction are published, it is essential that clinicians understand where abfraction stands in current clinical practice. The aim of this article was to critically review the literature for and against abfraction.

The development of the concept of abfraction

Abfraction is said to take place when excessive cyclic, non-axial tooth loading leads to cusp flexure and concentration of stresses in the vulnerable cervical region of teeth. Such stresses are then believed to directly contribute to tooth structure loss, by overcoming bonds between hydroxyapatite crystals, or indirectly lead to tooth structure loss by making the tooth more susceptible to future breakdown via further abfraction and other processes (e.g., abrasion and corrosion).[4–6]

The concept that occlusal loading could cause cervical stress, with resultant loss of cervical tooth structure, began evolving in the late 1970s.[3,4,7] The process was eventually termed abfraction by Grippo[2] in 1991.

Common to all accounts of this hypothesis was a lack of sound evidence and a number of errors in logic.

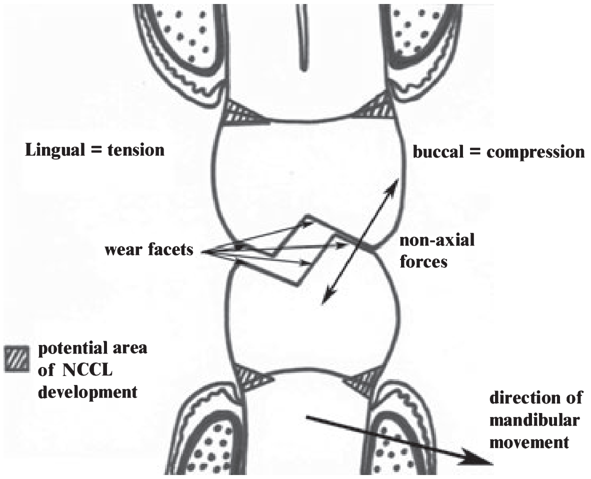

Fig 1. Diagrammatic representation of second premolars and their supporting structures engaged in tooth grinding. The non-axial forces produced as a result of tooth grinding may produce unfavourable stresses in the cervical region of the teeth, potentially leading to the development of NCCLs. Interestingly, logic suggests that with tooth grinding in a buccal direction, the buccal cervical regions would be in compression, while the lingual cervical regions would be in tension.

Although the loss of enamel was addressed, no explanation was given about how dentine could be lost during this process.[3,4,7] As dentine has a different structure from enamel and can withstand tensile stress better than enamel, this omission represents a major flaw in the concept of abfraction.[8]

Typically, abfraction is said to result from forces associated with mastication, swallowing and malocclusion. However, Gibbs *et al.*[9] found that occlusal forces during swallowing and mastication are only approximately 40 per cent of maximal bite force. Suit *et al.*[10] reported that tooth contact occurs on average for only 194 milliseconds during mastication and for 683 milliseconds during swallowing. Considering that the duration and magnitude of forces during bruxism are much greater than those during functional activity, it is more likely that parafunction would result in such a process rather than function.[11] Interestingly, logic suggests the non-axial forces that result from tooth grinding (Fig 1), would actually cause compression on the buccal cervical surfaces of the teeth involved. How would compression produce a breakdown in this tooth structure?

Terminology

Throughout the literature there is misunderstanding about the most appropriate terminology to use when discussing abfraction. For example, Miller *et al.*[12] state that NCCLs are also called abfractions. This statement is misleading because NCCLs have a variety of possible causes, of which abfraction is only one. In addition, the aetiology of NCCLs is multifactorial, so while abfraction may be the primary aetiological agent, it must be understood that other factors may also play a part. This statement applies to all the following aetiological classifications of NCCLs described in this article. The correct use of terminology is essential in this confusing area of dentistry.

While on the topic of confused terminology, it is important to address two other misconceptions. Traditionally, the dental profession has defined erosion as tooth wear resulting from prolonged exposure to low pH substances of non-bacterial origin. However, this process should be termed "corrosion" as it results from the action of a chemical (i.e., acid). In contrast, the term "erosion" refers to an abrasive process resulting from the dynamic contact of a solid, liquid or gas with a surface (e.g., water waves degrading a limestone coastline).[13] Erosion is a physical process, whereas corrosion is a chemical process.

It is also important to differentiate the term "stress-corrosion" from "abfraction". Stress-corrosion refers to the synergistic effects of stress and corrosion acting simultaneously.[13] For example, this may occur when a tooth is heavily loaded in an acidic environment. Currently, there is only limited research exploring the role of stress-corrosion in NCCL development.

Characteristics of abfraction lesions

Lee and Eakle[4] first described the characteristics of lesions that may result from tensile stresses. They concluded that an abfraction lesion should be located at or near the fulcrum in the region of greatest tensile stress concentration, be typically wedge-shaped, and display a size proportional to the magnitude and frequency of tensile force application (Fig 2). Interestingly, Lee and Eakle[4] proposed that the direction of the lateral force(s) acting on a tooth would determine the location of the lesion. For example, if there were two or more lateral forces the result would be an NCCL composed of two or more overlapping wedge-shaped NCCLs. They acknowledged that local factors, such as abrasion and corrosion, might modify the appearance of NCCLs related to tensile stresses but did not expand on this speculation. The descriptions of Lee and Eakle[4] have inherent limitations, as they were not based on

Fig 2. Proximal view of a mandibular central incisor demonstrating a wedge-shaped NCCL (located within the white box).

direct experimental or clinical evidence. Laboratory studies are needed to attempt to produce NCCLs through abfraction to validate such descriptions.

It should be reiterated that not all wedge-shaped NCCLs necessarily result from abfraction. Abrasion, which results from prolonged dynamic contact of a tooth with an exogenous substance, such as a toothbrush with dentifrice, is also a widely accepted cause of wedge-shaped NCCLs.[14] There is also the possibility that other, yet to be discovered, processes contribute to the formation of wedge-shaped NCCLs. Sognnaes *et al.*[15] observed extracted teeth that exhibited wedge-shaped NCCLs within silicate and amalgam restorations. This phenomenon could result from abrasion, but is unlikely to be explained by abfraction because the properties of amalgam and silicate restorations are notably different to those of dental hard tissues.

NCCLs in pre-contemporary populations

An important epidemiological finding relating to abfraction is the lack of NCCLs observed in pre-contemporary populations. NCCLs have not been observed in studies investigating their prevalence in ancient American skulls or prehistoric and historic skeletal remains from the south of France.[16,17] Interproximal grooving has been observed in the cervical regions of teeth in pre-contemporary Australian Aboriginals but the lesions result from task activity (passing animal tendon interproximally between teeth) and are not observed in contemporary populations.[18] NCCLs unrelated to task activity have not been observed in pre-contemporary Australian Aboriginals.[19]

There is a high prevalence of occlusal wear faceting observed in pre-contemporary Australian Aboriginals.[20] Many of these wear facets can only be matched between opposing teeth in extreme mandibular positions, supporting a parafunctional aetiology.[20] Heavy occlusal loading of teeth in Australian Aboriginals resulted in non-axial forces large enough to cause the movement of adjacent teeth against one another, leading to interproximal tooth wear.[21] However, despite the presence of occlusal forces that one would expect might lead to abfraction, no lesions have been found. Absence of abfraction lesions in these circumstances, particularly in an environment where there was also no toothbrush abrasion and little corrosion, provides strong evidence against the concept that abfraction can be caused by occlusal loading alone.

Theoretical evidence of abfraction

Finite element analysis (FEA) is a numerically-based computer modelling method that can be used to improve understanding of complicated mechanical problems, such as the stresses involved with tooth loading. FEA involves breaking down a complex problem into numerous simple elements which, using appropriate formulae and data, are solved individually. Individual solutions of each element are combined to enable an overall model to be developed. FEAs can be either two- or three-dimensional.[18] A number of authors have used FEAs to investigate the validity of abfraction and Table 1 summarizes the outcomes of several of these studies. When considering the results of this research, the limitations of FEA as a means of investigating abfraction should be considered.

Table 1. Summary of the outcomes of FEA studies related to abfraction

Reference(s)	Conclusions
Rees[23]	Eccentrically loaded, restored premolars demonstrated higher stresses in the cervical region compared with similarly loaded unrestored premolars.
Palamara *et al.*[30]	Non-axial tooth loading resulted in potentially damaging cervical strains. The direction and magnitude of loading had a strong influence on the nature of cervical strains.
Rees *et al.*[28]	Peak cervical stresses upon tooth loading were highest for the maxillary incisor, intermediate for the maxillary premolar and lowest for the maxillary canine.
Rees[24] Lee *et al.*[25] Palamara *et al.*[31]	Non-axial tooth loading increases the magnitude of cervical stress values.
Borcic *et al.*[32]	Teeth in malocclusion generated larger tensile stresses in the cervical region compared to teeth in normal occlusion upon tooth loading.

Rees[23] correctly acknowledged the limitations of using two-dimensional FEA to investigate three-dimensional objects such as teeth. An important advantage of three-dimensional FEA, as pointed out by Rees,[24] is that torsional stress can be measured. As such, three-dimensional FEAs allow more realistic simulations to be developed. However, many of the FEAs investigating abfraction have used two-dimensional models (Table 2).

Researchers have also used different force magnitudes in their FEA models, ranging from 100 to 500 newtons, thereby making comparisons between FEA studies problematic (Table 2).

The properties allocated to the materials under investigation are critical to the validity of FEAs, since each element is assigned specific values that affect the results. Different researchers have assumed markedly different physical properties of dental tissues, such as enamel.[22] Some have considered enamel to be an isotropic material in which properties are similar in all directions (Table 2).[24,25] However, Spears[26] provided evidence that enamel should be considered to be anisotropic, rather than isotropic, as it is suspected of having different physical properties in different directions. This is an important issue because when

Table 2. Summary of the outcomes of FEA studies related to abfraction

Reference	Force (newtons)	Tooth class(es) investigated	Assumed behaviour of enamel
Rees[23]	100	Mandibular second premolars	Anisotropic
Palamara *et al.*[30]	100	Mandibular second premolars	Not specified
Lee *et al.*[25]	170	Maxillary second premolars	Isotropic
Rees[24]	500	Mandibular second premolars	Isotropic
Rees *et al.*[28]	500	Maxillary central incisors, canines and first premolars	Anisotropic
Rees and Hammadeh[33]	100	Maxillary central incisors, canines and first premolars	Anisotropic
Borcic *et al.*[32]	200	Maxillary first premolars	Isotropic
Palamara *et al.*[31]	100	Mandibular second premolars and lower central incisors	Isotropic

enamel is considered to be anisotropic, the tooth seems to be better able to cope with loading. Not only are the resultant stresses of lower magnitude, they are also preferably transferred into dentine which tolerates tensile stress better than enamel.[27] Therefore, the work of those authors who have considered enamel to be isotropic should be interpreted with caution (Table 2).

Another flaw of FEA is its inability to simulate accurately the biological dynamics of the tooth and its supporting structures. For example, with non-carious cervical tooth structure loss there are also changes in the structure of dentine as it becomes exposed to the oral environment. It is very difficult to develop a predictive model for the complex structure of tertiary dentine, which is formed in response to a stimulus such as tooth wear.

Perhaps the most important issue in using FEA to investigate abfraction is that this approach has highlighted an anomaly. A common finding of FEAs has been that the magnitudes of facial and lingual stresses are similar in response to loading. However, this does not match the clinical picture of NCCLs that present much more commonly on the facial, rather than the palatal or lingual surfaces of teeth.[14] Rees *et al.*[28] propose that stress-corrosion may provide an explanation for this anomaly. They refer to the work of Lecomte and Dawes[29] who found that erosive fluids, such as fruit juice, are cleared from palatal sites six times faster than from facial sites.

Experimental evidence of abfraction

There is only a small amount of experimental evidence supporting the concept of abfraction and, unfortunately, there are many limitations to the studies reported so far. Palamara *et al.*[34] found that teeth exposed to 500 newton loading over 200 000 to 500 000 cycles, while immersed in water, demonstrated microfractures and small areas of enamel loss when examined under scanning electron microscopy at 200× to 1200× magnification. However, this research did not completely replicate a clinical situation. For example, although the teeth were subjected to cyclic, non-axial loading, a 20 newton pre-load was maintained. Maintaining a certain load, no matter the magnitude, limits the value of the results, as this is not what occurs clincally.

Litonjua *et al.*[35] conducted an experiment to determine the effect of axial and non-axial forces on the initiation and progression of NCCLs in teeth that were also subjected to toothbrush abrasion. They found that axially loaded teeth that were subjected to toothbrush abrasion exhibited significantly less cervical tooth substance loss than control teeth that were not loaded. Non-axially loaded teeth that were simultaneously subjected to toothbrush abrasion showed similar amounts of cervical wear to control teeth that were not loaded. However, this experiment had several flaws. For example, teeth were loaded while being simultaneously subjected to toothbrush abrasion, a situation unlikely to occur clinically. In addition, the teeth were subjected to static loads. This does not represent the true clinical picture of tooth loading which is dynamic and variable in nature. The results of this study should therefore be interpreted with caution.

Staninec *et al.*[36] investigated the magnitude and location of tooth structure loss using sectioned segments of tooth structure that were subjected to cyclic mechanical loading. Despite the obvious limitation of using sectioned segments of tooth structure, they found that there was a greater loss of material in high-stress areas, supporting the possibility of abfraction.

A confounding factor to consider when examining the formation of abfraction lesions is the vulnerability of cervical tooth structure to breakdown.[24] Cervical enamel has been shown to be more brittle compared with occlusal enamel.[37] The DEJ in the cervical region of teeth has poorly developed scalloping, which means the strength of this junction is compromised.[38] The hardness modulus and elastic modulus of enamel also decrease as the DEJ is approached.[39]

In summary, the small amount of experimental evidence published for abfraction has limitations. What has been shown experimentally is that cervical tooth structure may be more vulnerable to stress-induced breakdown. Future experimental studies that replicate the clinical situation as closely as possible are needed to provide further evidence for or against abfraction.

Abfraction lesions and occlusal factors

There have been strong associations reported between occlusal wear facets and NCCLs. Since occlusal wear facets are indicative of past tooth grinding it has been proposed that this is evidence for abfraction.[12,40–42] However, not all teeth with NCCLs exhibit wear facets and not all teeth with wear facets exhibit NCCLs.[42] As indicated previously, a cross-sectional study looking at facet frequencies caused by tooth grinding in an Australian Aboriginal population indicated a high prevalence of tooth grinding and no evidence of NCCLs.[20]

One must recognize that a limitation of using wear facets as indicators of bruxism is that not all wear results from bruxism-related attrition. It may also result from corrosion and abrasion (e.g., with mastication of coarse substances). Previous researchers have apparently ignored this fact and, therefore, their results should be interpreted with caution. Khan *et al.*[43] tried to overcome this issue by determining whether occlusal wear lesions had resulted from erosion or attrition. They found a 96 per cent association between non-carious occlusal and cervical lesions but no statistically significant association was found between the number of wedge-shaped NCCLs associated with occlusal erosion or attrition lesions.[43] Future studies must take into account the multifactorial aetiology of wear facets.

As malocclusion may predispose to non-axial forces, there has been research investigating the associations between malocclusion and NCCLs. Strong associations have been found between NCCLs and group function.[12,41] Piotrowski *et al.*[44] found that all 10 teeth with premature contacts, which may have predisposed these teeth to excessive, non-axial loading, had wedge-shaped lesions.

Analysing the occlusion has its limitations because the occlusal relationships noted at the time of an examination may change as NCCLs progress. In addition, bruxism involves both clenching and grinding of teeth. Clenching does not result in the formation of wear facets, yet it can still lead to tooth flexure. There is no simple, accurate way of recording or reporting clenching. Bruxism is thus underestimated if wear facets are the only measures of its activity.

Management of abfraction lesions

As evident from the previous text, there is currently a lack of sound evidence to confirm whether abfraction is a real clinical phenomenon. However, a variety of management strategies for abfraction lesions have been proposed. Some strategies are tailored for abfraction lesions and others are used for NCCLs of all aetiologies.

Monitoring abfraction lesions

As abfraction is still yet to be supported by appropriate evidence, it is advisable to monitor suspected abfraction lesions where appropriate. This avoids unnecessary treatment and, over time, a more obvious cause(s) (e.g., toothbrush abrasion or corrosion) may present itself. As with all forms of tooth wear, it is vital to take into consideration the age of the individual and the predicted rate of tooth wear. Wear can be considered physiological in older individuals when the tooth in question is not causing clinical consequences and would be seen to last the patient's lifetime without operative intervention. However, if the tooth wear is likely to compromise the long-term prognosis of the tooth then operative intervention may be required.[19]

When possible abfraction lesions are not causing clinical consequences and/or they are only shallow in depth (less than 1 mm), one may elect to simply monitor them at regular intervals (e.g., six-monthly). Standardized intra-oral photographs, study models and measuring lesion dimensions are all potential approaches. However, these methods are generally only useful for long periods of time, such as months and years.

A novel method of determining the activity of abfraction lesions over time is to undertake a scratch test. A number 12 scalpel blade is used to superficially scratch the tooth surface. Visual observation of the scratch will give an indication of the rate of the tooth structure loss. Loss of scratch definititon or loss of the scratch altogether signifies active tooth structure loss.[19]

Restoration of abfraction lesions

It should be noted that when restoring abfraction lesions clinicians are not treating the aetiology but are merely replacing what has been lost. There are no generally accepted, specific guidelines in the literature stating when abfraction lesions should be restored. Logic and good clinical judgement would suggest that they should be restored when clinical consequences (e.g., dentine hypersensivity) have developed or are likely to develop in the near future. Aesthetic demands of the patient may also influence the decision to restore these lesions. One must conduct a risk-benefit analysis when considering restoring these lesions. Cervical restorations may contribute to increased plaque accumulation potentially leading to caries and periodontal disease.

Problems with restoring NCCLs include difficulty in obtaining moisture control, gaining access to subgingival margins and high failure rates.[45–47] Rubber dam clamps, gingival retraction cord and periodontal surgery are methods that can be used to retract and control the gingival tissues and thus facilitate access and control moisture. Reasons for restoration failure include loss of retention, secondary caries, marginal defects, discolouration and sensitivity.

It is evident from the recent literature that there is no place for metallic materials such as amalgam and gold in the modern day restoration of NCCLs.[48,49] Glass ionomer cements (GICs), resin-modified GICs (RMGICs), a GIC/RMGIC liner/base laminated with a resin composite, and resin composite in combination with a dentine bonding agent are all restorative options.

Tyas[48] recommended that RMGIC should be the first preference for restoration of NCCLs or, in aesthetically demanding cases, a RMGIC/GIC liner/base be laminated with resin composite. Vandelwalle and Vigil[49] recommend NCCLs suspected of being caused primarily by abfraction should be restored with a microfilled resin composite that has a low modulus of elasticity, as it will thus flex with the tooth and not compromise retention. However, a seven-year study found no statistically significant difference between failure rates of three resin composites of different stiffness used to restore NCCLs.[50]

Occlusal adjustment

As a result of the reported associations between occlusal interferences and abfraction lesions, and loading direction (influenced by cusp inclines) and unfavourable tensile stresses, occlusal adjustment has been advocated to prevent their initiation and progression and to minimize failure of cervical restorations.[44,51] Occlusal adjustments may involve altering cuspal inclines, reducing heavy contacts and removing premature contacts. Expensive devices claiming to assist the clinician in adjusting the occlusion and therefore managing abfraction should be considered with caution.

It is not recommended that occlusal adjustments be carried out at this time in an effort to manage abfraction because the effectiveness of such treatment is not supported by evidence. In fact, inappropriate occlusal adjustments may increase the risk of certain conditions such as caries, occlusal tooth wear and dentine hypersensitivity.

Occlusal splints

Occlusal splints, aimed at reducing the amount of nocturnal bruxism and non-axial tooth forces, have been recommended to prevent the initiation and progression of abfraction lesions.[44] However, it should be noted that the use of occlusal splints to reduce bruxism is still a controversial topic. Some studies support their efficacy, while others do not.[52] Occlusal splints have the potential to reduce non-axial tooth loading when constructed appropriately. Although they provide a conservative treatment option for managing suspected abfraction lesions, there is no evidence base to support their use.

CONCLUSIONS

There is still much to be discovered about NCCLs in general and the possible role of abfraction remains controversial and frequently misunderstood. The findings from anthropological, epidemiological, theoretical, experimental and clinical studies suggest that abfraction should still be considered to be a theoretical concept rather than a genuine contributor to NCCL formation. If abfraction is suspected to be a dominant factor in the aetiology of NCCLs, then any decision to carry out destructive, irreversible treatment, such as occlusal adjustment, should be considered very carefully.

REFERENCES

1. Bader JD, McClure F, Scurria MS, Shugars DA, Heymann HO. Case-control study of non-carious cervical lesions. Community Dent Oral Epidemiol 1996;24:286–291.
2. Grippo JO. Abfractions: a new classification of hard tissue lesions of teeth. J Esthet Dent 1991;3:14–19.
3. McCoy G. The etiology of gingival erosion. J Oral Implantol 1982;10:361–362.
4. Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. J Prosthet Dent 1984;52:374–380.
5. Rees JS. A review of the biomechanics of abfraction. Eur J Prosthodont Restor Dent 2000;8:139–144.
6. Grippo JO, Simring M, Schreiner S. Attrition, abrasion, corrosion and abfraction revisited: a new perspective on tooth surface lesions. J Am Dent Assoc 2004;135:1109–1118.
7. Brady JM, Woody RD. Scanning microscopy of cervical erosion. J Am Dent Assoc 1977;94:726–729.
8. Xu HH, Smith DT, Jahanmir S, *et al*. Indentation damage and mechanical properties of human enamel and dentin. J Dent Res 1998;77:472–480.
9. Gibbs CH, Mahan PE, Lundeen HC, Brehnan K, Walsh EK, Holbrook WB. Occlusal forces during chewing and swallowing as measured by sound transmission. J Prosthet Dent 1981;46:443–449.
10. Suit SR, Gibbs CH, Benz ST. Study of gliding contacts during mastication. J Periodontol 1976;47:331–334.
11. Okeson JP. Causes of functional disturbances in the masticatory system. In: Okeson JP, ed. Management of temporomandibular disorders and occlusion. 5th edn. St. Louis: Mosby, 2003:149–189.
12. Miller N, Penaud J, Ambrosini P, Bisson-Boutelliez C, Briancon S. Analysis of etiologic factors and periodontal conditions involved with 309 abfractions. J Clin Periodontol 2003;30:828–832.
13. Grippo JO, Simring M. Dental 'erosion' revisited. J Am Dent Assoc 1995;126:619–620.
14. Bartlett DW, Shah P. A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. J Dent Res 2006;85:306–312.
15. Sognnaes RF, Wolcott RB, Xhonga FA. Dental erosion. I. Erosion-like patterns occurring in association with other dental conditions. J Am Dent Assoc 1972;84:571–576.

16. Aaron GM. The prevalence of non-carious cervical lesions in modern and ancient American skulls: lack of evidence for an occlusal aetiology. Florida: The University of Florida, 2004. M.S. thesis.
17. Aubry M, Mafart B, Donat B, Brau JJ. Brief communication: study of noncarious cervical tooth lesions in samples of prehistoric, historic, and modern populations from the South of France. Am J Phys Anthropol 2003;121:10–14.
18. Brown T, Molnar S. Interproximal grooving and task activity in Australia. Am J Phys Anthropol 1990;81:545–553.
19. Kaidonis JA. Tooth wear: the view of the anthropologist. Clin Oral Investig 2008;12(Suppl 1):S21–S26.
20. Kaidonis JA, Townsend GC, Richards LC. Nature and frequency of dental wear facets in an Australian Aboriginal population. J Oral Rehabil 1993;20:333–340.
21. Kaidonis JA, Townsend GC, Richards LC. Brief communication: interproximal tooth wear a new observation. Am J Phys Anthropol 1992;88:105–107.
22. Geng JP, Tan KB, Liu GR. Application of finite element analysis in implant dentistry: a review of the literature. J Prosthet Dent 2001;85:585–598.
23. Rees JS. The role of cuspal flexure in the development of abfraction lesions: a finite element study. Eur J Oral Sci 1998; 106:1028–1032.
24. Rees JS. The effect of variation in occlusal loading on the development of abfraction lesions: a finite element study. J Oral Rehabil 2002;29:188–193.
25. Lee HE, Lin CL, Wang CH, Cheng CH, Chang CH. Stresses at the cervical lesion of maxillary premolar – a finite element investigation. J Dent 2002;30:283–290.
26. Spears IR. A three-dimensional finite element model of prismatic enamel: a re-appraisal of the data on the Young's modulus of enamel. J Dent Res 1997;76:1690–1697.
27. Spears IR, van Noort R, Crompton RH, Cardew GE, Howard IC. The effects of enamel anisotropy on the distribution of stress in a tooth. J Dent Res 1993;72:1526–1531.
28. Rees JS, Hammadeh M, Jagger DC. Abfraction lesion formation in maxillary incisors, canines and premolars: a finite element study. Eur J Oral Sci 2003;111:149–154.
29. Lecomte P, Dawes C. The influence of salivary flow rate on diffusion of potassium chloride from artificial plaque at different sites in the mouth. J Dent Res 1987;66:1614–1618.
30. Palamara D, Palamara JE, Tyas MJ, Messer HH. Strain patterns in cervical enamel of teeth subjected to occlusal loading. Dent Mater 2000;16:412–419.
31. Palamara JE, Palamara D, Messer HH, Tyas MJ. Tooth morphology and characteristics of non-carious cervical lesions. J Dent 2006;34:185–194.
32. Borcic J, Anic I, Smojver I, Catic A, Miletic I, Rubaric SP. 3D finite element model and cervical lesion formation in normal occlusion and in malocclusion. J Oral Rehabil 2005;32:504–510.
33. Rees JS, Hammadeh M. Undermining enamel as a mechanism of abfraction lesion formation: a finite element study. Eur J Oral Sci 2004;112:347–352.
34. Palamara D, Palamara JE, Tyas MJ, Pintado M, Messer HH. Effect of stress on acid dissolution of enamel. Dent Mater 2001;17:109–115.
35. Litonjua LA, Bush PJ, Andreana S, Tobias TS, Cohen RE. Effects of occlusal load on cervical lesions. J Oral Rehabil 2004;31:225–232.
36. Staninec M, Nalla RK, Hilton JF, *et al.* Dentin erosion simulation by cantilever beam fatigue and pH change. J Dent Res 2005; 84:371–375.
37. Hassan R, Caputo AA, Bunshah RF. Fracture toughness of human enamel. J Dent Res 1981;60:820–827.
38. Rees JS. The biomechanics of abfraction. Proc Inst Mech Eng [H] 2006;220:69–80.
39. Meredith N, Sherriff M, Setchell DJ, Swanson SA. Measurement of the microhardness and Young's modulus of human enamel and dentine using an indentation technique. Arch Oral Biol 1996;41:539–545.
40. Xhonga FA. Bruxism and its effect on the teeth. J Oral Rehabil 1977;4:65–76.
41. Mayhew RB, Jessee SA, Martin RE. Association of occlusal, periodontal, and dietary factors with the presence of non-carious cervical dental lesions. Am J Dent 1998;11:29–32.
42. Pegoraro LF, Scolaro JM, Conti PC, Telles D, Pegoraro TA. Noncarious cervical lesions in adults: prevalence and occlusal aspects. J Am Dent Assoc 2005;136:1694–1700.
43. Khan F, Young WG, Shahabi S, Daley TJ. Dental cervical lesions associated with occlusal erosion and attrition. Aust Dent J 1999; 44:176–186.
44. Piotrowski BT, Gillette WB, Hancock EB. Examining the prevalence and characteristics of abfractionlike cervical lesions in a population of US veterans. J Am Dent Assoc 2001;132:1694–1701.
45. Ibsen RL. Non-operative treatment for gingival erosion. Dent Surv 1972;48:22–24.
46. Brackett WW, Dib A, Brackett MG, Reyes AA, Estrada BE. Two-year clinical performance of class V resin-modified glass-ionomer and resin composite restorations. Oper Dent 2003; 28:477–481.
47. Heymann HO, Sturdevant JR, Bayne S, Wilder AD, Sluder TB, Brunson WD. Examining tooth flexure effects on cervical restorations: a two-year clinical study. J Am Dent Assoc 1991;122: 41–47.
48. Tyas MJ. The Class V lesion – aetiology and restoration. Aust Dent J 1995;40:167–170.
49. Vandewalle KS, Vigil G. Guidelines for the restoration of Class V lesions. Gen Dent 1997;45:254–260.
50. Peumans M, De Munck J, Van Landuyt KL, *et al.* Restoring cervical lesions with flexible composites. Dent Mater 2007; 23:749–754.
51. Ichim IP, Schmidlin PR, Li Q, Kieser JA, Swain MV. Restoration of non-carious cervical lesions Part II. Restorative material selection to minimise fracture. Dent Mater 2007;23: 1562–1569.
52. Kreiner M, Betancor E, Clark GT. Occlusal stabilization appliances. Evidence for their efficacy. J Am Dent Assoc 2001; 132:770–777.

Address for correspondence:
Professor Grant Townsend
School of Dentistry
The University of Adelaide
Adelaide, South Australia 5005
Email: grant.townsend@adelaide.edu.au