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Effect of Lateral Excursive Movements on the Progression of Abfraction Lesions

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Clinical Relevance

The results of this randomized controlled trial have direct relevance to clinical practice and provide some evidence that occlusal adjustment to prevent further progression of abfraction lesions cannot be supported currently.

SUMMARY

The theory of abfraction suggests that tooth flexure arising from occlusal loads causes the formation and progression of abfraction lesions. The current study investigated whether reducing occlusal loading by adjusting the occlusion on a tooth during lateral excursive movements had any effect on the rate of progression of existing abfraction lesions. Recruited were 39 subjects who had two non-carious cervical lesions in the maxillary arch that did not need restoration and were in group function during lateral excursive movements of the mandible. One of the teeth was randomly selected to have the excursive occlusal contacts reduced by using a fine grain diamond bur. Centric occlusal contacts were not reduced. Impressions of the lesion were taken over a 30-month period to enable monitoring of the wear rate, and duplicate dies were poured into epoxy resin to allow for sectioning. The size of the lesions was measured using stereomicroscopic analysis of the sectioned epoxy resin dies, and the results were analyzed using an Independent *t*-test. No statistically significant difference in wear rates between the adjusted and non-adjusted teeth was found (p>0.05).

Within the limitations of the current study, it was concluded that occlusal adjustment does not appear to halt the progression of non-carious cervical lesions; consequently, this procedure cannot be recommended.

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INTRODUCTION

As an aging population retains its teeth longer, the issue of tooth wear or non-carious tooth tissue loss is becoming increasingly important to the dental profession. The phrase "non-carious cervical tooth surface loss" has arisen in an attempt to embrace these kinds of lesions, which occur at the neck of the tooth. Unfortunately, far more confusion arises from the use of other terminologies, such as erosions and abrasions, which have been used at different times and in different locations to describe similar lesions.

In 1908, Black, in his seminal work on Operative Dentistry, discussed the problematic etiology of what he termed "erosions" and stated that, "Our information regarding erosion is far from complete and much time may elapse before its investigation will give satisfactory results."[1] Black identified eight possible causes:

- Faults in the formation of teeth
- Friction from an abrasive toothpowder
- Action of an unknown acid
- Secretion of a diseased salivary gland
- Physiological resorption, as with deciduous teeth
- Acid associated with gouty diarethis
- Action of alkaline fluids on calcium salts
- Action of enzymes released by micro-organisms

After going through each hypothesis, in turn, finding fault with all, he concluded that he had no theory of his own to offer that did not have features that rendered it impossible.

Other researchers in the early part of the 20th century also considered these lesions. Miller, in 1907, looked at "wastings" and concluded that brushing with coarse toothpowder was the likely cause.[2] In 1931, Ferrier was unable to find a reasonable explanation and, in 1932, Kornfeld made the observation that, in all cases of cervical erosion, he noticed heavy wear facets on the articulating surfaces of the teeth involved and that the erosion tended to be at the opposite side of the tooth to the wear facet.[3-4]

The confusing use of the term erosion to describe a lesion that may actually be caused by mechanical abrasion is further compounded by the fact that, to a chemical engineer, the process described by dentists as erosion is known as corrosion.[5] Use of such imprecise terminology has contributed to both the difficulty of carrying out good quality research and making accurate diagnoses that enable appropriate treatments to be both recommended and provided.

Many practitioners have felt that over enthusiastic toothbrushing and the use of abrasive toothpastes were the primary causes of these lesions, but Lee and Eakle, in 1984, put forward the hypothesis that tensile stresses created in the tooth during occlusal loading may have a role in the etiology of cervical erosive lesions.[6] They described three types of stress placed on teeth during mastication and parafunction:

- Compressive—the resistance to compression
- Tensile—the resistance to stretching
- Shearing—the resistance to twisting or sliding

The authors were of the opinion that, in a "non-ideal" occlusion, large lateral forces could be created that would result in compressive stresses on the side being loaded and tensile stresses on the opposite side. As enamel is strong in compression but weak in tension, it was suggested that those areas in tension were prone to failure. The region of greatest stress is found at the fulcrum of the tooth. The characteristic lesion described was wedge-shaped, with sharp line angles and was situated at or near the fulcrum of the tooth. It was suggested that the direction of the lateral force governed the position of the lesion, while its size was related to the amount of force and its duration.

Grippo put forward a new classification of hard tissue lesions of teeth.[7] He defined four categories of tooth wear as follows:

- Attrition—the wearing away of tooth substance as a result of tooth-to-tooth contact during normal or parafunctional masticatory activity.
- Abrasion—the pathological wear of tooth substance through biomechanical frictional processes, such as toothbrushing.
- Erosion—the loss of tooth substance by acid dissolution of either an intrinsic or extrinsic origin; for example, gastric acid or dietary acids.
- Abfraction—the pathologic loss of tooth substance caused by biomechanical loading forces. It was postulated that these lesions were caused by flexure of the tooth during loading, leading to fatigue of the enamel and dentin at a location away from the point of loading. The word "abfraction" was derived from the Latin "to break away."

Grippo then further described five categories of abfraction:

- Hairline cracks
- Striations—horizontal bands of enamel breakdown
- Saucer shaped—a lesion entirely within enamel
- Semi-lunar shaped—a crescent shaped lesion entirely within enamel
- Cusp tip invagination—a depression on the cusp tip seen in molars and premolars

By 1994, Lambert and Lindenmuth considered that the profession should now consider occlusal stress as a primary factor in the creation and progression of cervical notch lesions and a considerable body of theoretical work had accumulated to support this hypothesis.[8] A comprehensive review of abfraction lesions has recently been published, which concluded that the etiology of these lesions is multifactorial but that occlusal factors are a primary etiological factor.[9]

This study investigated whether or not reducing excursive occlusal loading had any impact on the rate of progression of abfraction lesions *in vivo*. The test hypothesis (H_t) was that a reduction in lateral excursive loads would result in a reduction in the rate of abfraction lesion development. The null hypothesis (H_0) for this study was that there was no difference in the rate of abfraction lesion progression when excursive occlusal loads were reduced.

METHODS AND MATERIALS

Subject Selection

Following ethical approval obtained from the local research ethics committee (Leeds East REC, Leeds, UK), 39 patients were recruited into the current study. They ranged in age from 18 through 75 years and had two abfraction lesions that did not require operative intervention. In addition, the test teeth had to be in group function during lateral excursive movements, with no mobility of the test or antagonist teeth being detectable clinically. In contrast, subjects were excluded from the study if:

- The abfraction lesions were in teeth that had no antagonist
- There was canine guidance in lateral excursion
- There was more than 3 mm of pocketing
- The teeth were mobile
- There was poor oral hygiene
- Other restorative treatment was required of the teeth included in the current study
- The subject had poor general health

This study was designed as a randomized controlled trial, with each patient acting as his/her own control. In order to minimize operator bias, the tooth to be adjusted was allocated at random. This was achieved by recording each tooth according to FDI notation. For example, an upper right second premolar was recorded as 15 and an upper left first premolar as 24. Equal numbers of opaque envelopes were prepared with either the word "High" or "Low" in them. No identifying marks were present externally. The patient chose an envelope at random from within a box and opened it. If it contained the word "High," then the tooth with the higher notation, for example, 24, as in the example above, was adjusted. Conversely, if the envelope contained the word "Low," the tooth with the lower notation, that is, 15, was adjusted.

Operative Procedures

All subjects recently had full mouth examinations, including clinically appropriate radiographs and a periodontal screening, to check for attachment loss, gingival bleeding and mobility of the relevant teeth. The dynamic and static occlusal contacts of the test teeth were marked using red and blue Bausch 40 micron articulating paper (Dr Jean Bausch KG, Seefeld, Germany) respectively, after having dried the occlusal surfaces of the teeth with tissue held in Miller's forceps (Unodent, Witham, UK). The areas marked red were reduced using Hi-Di fine grain diamond finishing burs (Hi Di Burs, Dentsply, Weybridge, UK) in a water-cooled air turbine. Care was taken to leave the blue centric stops and the red point of maximum excursive contact intact to ensure occlusal stability. The adjusted area was lightly polished with Shofu abrasive cones (Shofu Dental GmbH, Ratingen, Germany) to reduce any surface roughness, and the occlusion was rechecked with articulating paper. Further adjustment, if required, was carried out as described above.

Full-arch impressions were taken using a polyether impression material (Impregum, 3M ESPE, Seefeld,

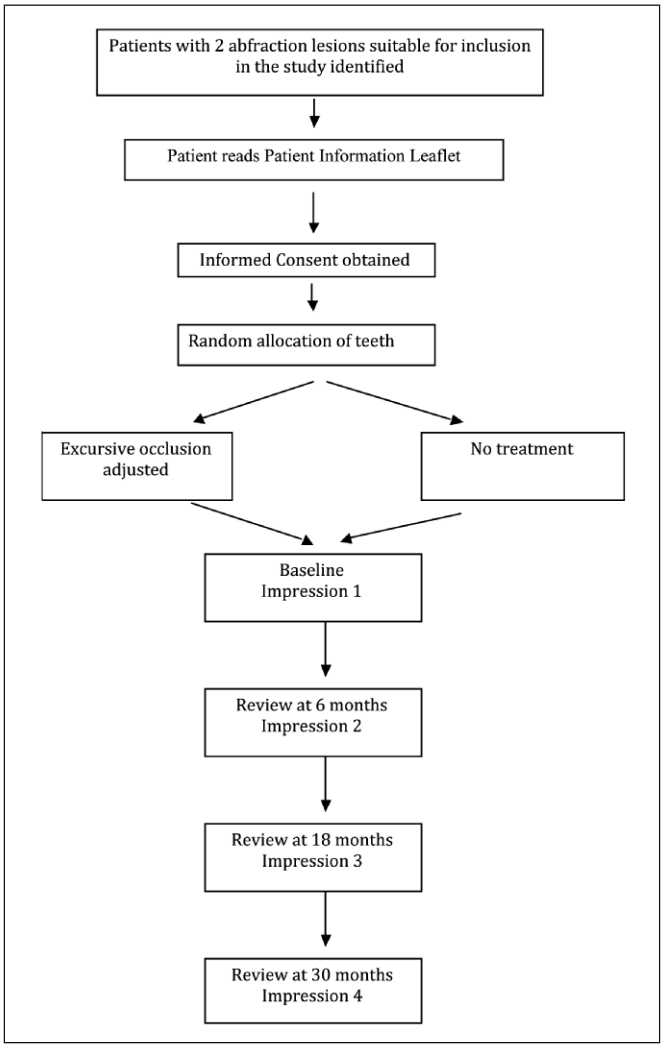

Figure 1: *Flow chart summarizing investigative procedures.*

Germany) in polycarboxylate disposable trays (Polytrays, Dentsply). The impressions were rinsed, disinfected and cast up immediately in blue die stone (Skillstone, Whip Mix Corporation, Louisville, KY, USA). The investigative procedures are summarized in Figure 1.

Follow-Up

The subjects were followed-up at six, 18 and 30 months after baseline. At the follow-up appointments, routine full mouth examinations were carried out, along with appropriate radiographs and periodontal screening. The occlusion of the test teeth was marked again with red and blue articulating paper to check whether there had been any change. If the dynamic markings were heavy or if new dynamic markings were identified, these were adjusted as before and repolished. At each visit, impressions were also taken as described previously.

Model Analysis

To minimize operator error in the processing and measuring of the models, all the models were measured at the same time. In order to keep the original models intact, the test teeth were duplicated using a polyvinylsiloxane putty and wash (Provil Novo, Heraeus Kulzer GmbH, Hanau, Germany) and replica dies were cast in epoxy resin (Exacto-Form Model Resin, Bredent, Senden, Germany). Three points were marked on each model (Figure 2) as follows:

- Tip of the cusp
- Midpoint of the lesion in the long axis of the tooth
- Soft tissues along the same axis

A scalpel blade was used to make the marks, as it is thinner than a pencil.

The models were then sectioned using a low-speed sectioning machine (Model 650, South Bay Technology Inc, San Clemente, CA, USA) with a water-cooled diamond wheel saw (76.2 x 0.4 mm, Saint Gobian Abrasive, Gloucester, UK) according to the sectioning plan. The two sides were labeled A and B. This enabled both sides of the slice to be measured in order to provide an average value for the size of the lesion. The sections were then ready to be measured (Figure 3).

Abfraction Area Measurement

The sections were washed under running water to remove any debris from the sectioning process. They were then examined by light-reflecting microscopy using a stereomicroscope (Type S Wild M3Z, Wild Heerburgg, Heerburgg, Switzerland) at 10x magnification. Each section was fixed to a glass slide and positioned under the microscope in such a way that the entire cross section of the abfraction area could be viewed and examined at right angles to the microscope lens. Live images from the microscope were transferred to a computer (Dell Latitude D510, Dell, Inc, Round Rock, TX, USA) by a built-in digital video camera (PEC 3010, Pulmix, Basingstoke, England). A digital micrograph was obtained from each section. The abfraction area measurement was carried out using image analysis with SigmaScan Software (SigmaScan Pro 5.0.0, Aspire Software International, Leesburg, VA, USA). The software counts the number of pixels enclosed within a defined boundary. In order to do this, the operator must define those boundaries around the lesion using a mouse and pointer on the computer screen. To minimize potential error, sequential lesions were measured at the same time, and two readings were taken from each side of the sample in an effort to get consistent values. The mean of the two measurements of abfraction area from each section was calculated. The mean of the two measurements from side A and side B was then worked out and recorded. In order to blind the operator, no means of identifying which tooth had been adjusted was made available at the time of analysis.

Data Analysis

Data was input into SPSS and the results were analyzed with a paired-samples *t*-test with the level of significance set at 0.05.

RESULTS

In total, 39 subjects were recruited into the study. They ranged in age from 35 years to 70 years, with a mean of 51 years and 3 months, sd ± 9.1 years. Two subjects failed to return and one patient was eliminated from the study because one of the test teeth sustained a cusp fracture and had to be restored. The distribution of the lesions is summarized in Table 1. The abfraction area from models for five patients could not be measured for several reasons; therefore, data were obtained from a total number of 31 patients.

Statistical analysis revealed that there was no statistically significant difference between the rate of lesion progression for teeth that had been adjusted and those that had not at the six-, 18- and 30-month

Figure 2.

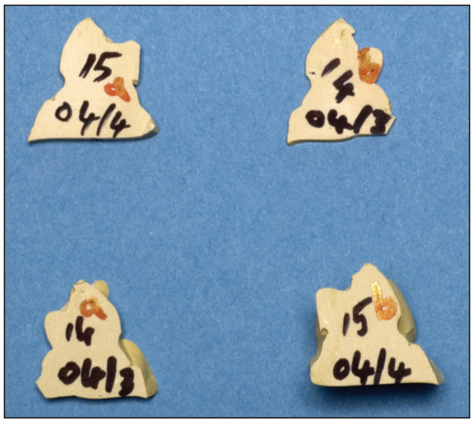

Figure 3.

reviews (p=0.510, p=0.682 and p=0.669, respectively). The results of the current study are summarized in Tables 2 and 3. The test hypothesis that, reducing the occlusal load in lateral excursion would reduce the rate of progression of abfraction lesions, was therefore rejected.

DISCUSSION

The current study primarily investigated the hypothesis that reducing excursive occlusal loads on a tooth in lateral excursion would lead to a reduction in the rate of progression of abfraction lesions. The study was carried out in a primary care setting, in this case, a predominantly private general dental practice, with most of the subjects regularly attending the practice. It is often stated that, "real world research will lead to real world findings." It was envisioned that, carrying out research in practice would make it easier to follow patients over a longer period of time than it is in a hospital setting, let alone test the feasibility of using this intervention in primary dental care to produce findings of clinical application in everyday practice. The relatively low dropout rate in this study is generally supportive of this principal.

The age range of the patients recruited, 31 to 70, with an average age of 51, tends to support the findings of Levitch and others, namely, that the prevalence of abfraction lesions increases with age.[10] In contrast to the findings of Pegoraro, no paired lesions were identified in patients under the age of 30.[11] That is not to say that lesions were not present in small numbers, but, if they were, they may not have been in group function in lateral excursion, which would have excluded them from the study.[11]

The data in Table 1 is not, of course, strictly an accurate indicator of lesion distribution, as the mouths of the subjects were examined from the right side first, when initially screened, and, if lesions were identified in that quadrant, the left side was not included in the study. The data does, however, give some indication of the spread between premolar and molar lesions, which is slightly at variance with reports by Pegoraro and others and Radentz and others, which found maxillary molars to be the most frequently affected teeth.[11-12] It is, of course, possible that this slightly different distribution, when compared with the published literature, has arisen, because only teeth that were in group function during lateral excursion were eligible for inclusion in the current study.

During the recruitment period, it was interesting to note how many lesions were seen that were ineligible for inclusion, because they were either not in group function in lateral excursion or not at all in occlusion. Several patients had classic wedge-shaped lesions on teeth that had never been in occlusion, for example, anterior open bites caused by skeletal discrepancies, which would indicate that occlusal forces could have played no part in their etiology. This calls into question Kornfeld's observation that all teeth with cervical erosions had heavy occlusal wear facets and similarly throws into doubt Lee and Eakle's theory about the possible role of tensile stress in the etiology of NCCL.[4,6] It does, however, tend to support the theory that, not only abfraction lesions are multifactorial in etiology, but that clinically identical lesions may have different etiologies. Much like Black, the authors of the current study still

Table 1: *Distribution of Lesions*

Tooth	Number	Percentage
Right maxillary premolar	34	55
Left maxillary premolar	16	26
Right maxillary molar	7	11
Left maxillary molar	5	08
Total	62	100

Table 2: *Change in Mean Values of Area of Abfraction Lesions From Baseline*

	Increase in Abfraction Area After Six Months	Increase in Abfraction After 18 Months	Increase in Abfraction Area After 30 Months
	Mean (sd)		
Not adjusted	0.06 (± 0.069)	0.142 (± 0.107)	0.202 (± 0.140)
Adjusted	0.08 (± 0.134)	0.158 (± 0.162)	0.225 (± 0.224)
(*p*-value)	0.510	0.682	0.669
Significance	†	†	†

KEY: †, not significant (p>0.05); sd, standard deviation.

do not have an explanation that satisfactorily explains the etiology of all these lesions without having some feature that renders it impossible.[1]

Within the design of the current study, the centric occlusal stop and the stop in the position of maximum excursion were both maintained to prevent occlusal instability. These markings were checked at each review appointment and adjusted, if necessary.

It was noted that some patients had re-established full sliding contacts by the review visit, requiring further occlusal adjustment. This would have had the effect of reintroducing occlusal stresses into their lateral excursive movements and lessening the effect of adjustments done in the current study. This might explain the results obtained from the current study. As the centric stop was maintained in all cases of distortion under vertical loading, for example, barreling effects, as described by Goel and others, would still be active, so that the influence of occlusal forces cannot be ruled out completely.[13] It is possible that reducing the occlusal loads in lateral excursion by re-establishing canine guidance as suggested by Murray and others would be a more effective intervention.[14]

A further factor not taken into account by the current study was the restorative condition of the teeth being monitored. It has been established by Rees that the presence of occlusal restorations in lower premolars significantly increases the cervical area stresses that occur during excursive loading, and it is not unreasonable to assume that the same effect would be found in maxillary teeth and could have had an influence on wear rates.[15]

It was interesting to note that both sets of lesions continued to increase in size. This indicates that the measuring technique was subtle and effective enough to detect small changes and illustrates the progres-

Table 3: *Change in Area of Abfraction Lesions in Square Millimeters From Baseline: Distribution of Lesions*

ID	Adjust	Abfr 6 m	Abfr 18 m	Abfr 30 m
		Abfraction Area Increase (mm^2)		
0114	1	0.15	0.22	0.33
0115	2	0.13	0.17	0.17
0324	1	0	0.02	0.27
0325	2	0.01	0.18	0.41
0414	2	*	0.07	0.17
0415	1	*	0.16	0.23
0524	1	0.07	0.18	*
0525	2	0.19	0.29	*
0615	1	0.09	0.11	0.31
0616	2	0.05	0.15	0.33
0924	2	0.62	0.79	0.87
0926	1	0	0.09	0.12
1114	2	0.28	0.33	0.40
1115	1	0.09	0.32	0.39
1324	2	0	*	0.07
1316	1	0.02	*	0.13
1514	1	0.05	0.09	0.09
1516	2	0.03	0.03	0.04
1624	1	0	0.07	0.08
1625	2	0.01	0.06	0.11
1715	1	0.02	0.03	0.07
1724	2	0.02	0.10	0.19
1914	1	0.07	0.14	0.23
1915	2	0.18	0.27	0.37
2114	2	*	0.24	0.85
2115	1	*	0.19	0.50
2214	2	0.03	0.07	*
2217	1	0.01	0.31	*
2314	1	0.26	0.27	0.32
2315	2	0.11	0.17	0.23
2415	1	0	0.15	0.21
2416	2	0	0.06	0.10
2514	1	0.05	0.13	*
2515	2	0.01	0.03	*
2726	1	*	*	0.30
2727	2	*	*	0.02
2814	1	0.24	0.43	0.55
2816	2	0.01	0.02	0.06
2914	2	*	0.02	*
2915	1	*	0.02	*
3014	1	0.01	0.01	0.03
3015	2	0.03	0.03	0.05
3114	2	0.13	0.22	0.28
3126	1	0.03	0.20	0.20
3214	1	0.09	0.12	0.12
3216	2	0.02	0.05	0.06
3314	2	0.01	0.03	0.10
3315	1	0.02	0.03	0.08

Table 3: *Change in Area of Abfraction Lesions in Square Millimeters From Baseline: Distribution of Lesions (cont.)*

ID	Adjust	Abfr 6 m	Abfr 18 m	Abfr 30 m
		Abfraction Area Increase (mm²)		
3424	2	*	0.21	0.28
3425	1	*	0.14	0.20
3624	1	0.05	*	*
3625	2	0.01	*	*
3714	2	0.02	0.15	0.18
3715	1	0.09	0.10	0.12
3824	2	0.01	*	0.10
3825	1	0.02	*	0.03
4014	1	*	*	0.24
4026	2	*	*	0.01
4114	1	0.05	*	0.10
4124	2	0.01	*	0.05
4214	2	0.07	0.21	0.35
4215	1	0.01	0.02	0.02

*KEYS: ID: case ID ($X_1X_2X_3X_4$) (**X_1X_2**, patient ID; **X_3X_4**, tooth ID).*
***Adjust**: Occlusal adjustment (**1**, no adjustment; **2**, adjusted).*
***Abfr 6 m**: increase of size of abfraction area after six months.*
***Abfr 18 m**: increase of size of abfraction area after 18 months.*
***Abfr 30 m**: increase of size of abfraction area after 30 months.*
** Subject did not attend for this recall appointment.*

sive nature of the lesions being measured. As adjusting the excursive occlusal load did not affect the rate of progression, other factors, such as toothbrush abrasion and or acid erosion, may be contributing to this wear. Further research is still needed to improve diagnosis and allow for differentiation between the different etiologies, which may result in the formation clinically of very similar lesions, let alone allow for the investigation of interventions designed to stop the formation and progression of abfraction lesions.

CONCLUSIONS

Within the limitations of the current study, the results do not support removal of lateral excursive contacts by occlusal adjustment to reduce the rate of progression of abfraction lesions in maxillary teeth.

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