The Degree to Which Attrition Characterizes Differentiated Patient Groups of Temporomandibular Disorders

Andrew G. Pullinger, DDS, MSc
Associate Professor
Section of Orofacial Pain and Occlusion
and Dental Research Institute
UCLA School of Dentistry
Los Angeles, California

Donald A. Seligman, DDS
Adjunct Assistant Professor
Section of Orofacial Pain and Occlusion
UCLA School of Dentistry
Los Angeles, California

Correspondence to:
Dr Andrew Pullinger
Center for the Health Sciences/Dentistry
Room 43-009
University of California Los Angeles
Los Angeles, California 90024

Dental attrition severity as the cumulative record of parafunctional and functional wear was graded from study cast analysis using established methodology. Attrition severity was compared in anterior, posterior mediotrusive, and posterior laterotrusive segments. Attrition scores in 48 female and 100 male totally asymptomatic controls were compared to 239 female and 31 male patients differentiated into five patient groups of temporomandibular disorders: (1) disc displacement with reduction, (2) disc displacement without reduction, (3) osteoarthrosis with a history of prior derangement, (4) osteoarthrosis without a history of prior derangement, and (5) myalgia only. All the male patients were in the myalgia-only group. Age was controlled in the analysis to control for functional wear. Comparisons between patients and controls were made according to 10-year age intervals. Analysis included ANCOVA confirmed by a Games-Howell post-hoc test, with $P < .01$ interpreted as a significant difference in the attrition score. Only 1 of 112 ANCOVAs showed a significant difference, with younger men from 20 to 29 years of age in the myalgia-only group having lower mediotrusive attrition than the male controls. It would therefore be difficult if not impossible to differentiate patients from nonpatients based on the severity of dental attrition. Consequently, a major peripheral occlusal etiologic role for attrition in TMD is questioned. Some clinical implications are elaborated.

J OROFACIAL PAIN 1993;7:196–208.

The in vivo dental attrition rates described for functional wear[1] are not sufficient to explain the advanced attrition seen in young adults in today's society. This suggests that notable attrition in younger populations is more the result of bruxism than functional wear.

Clinically it is common to postulate a connection between attrition and temporomandibular disorder (TMD) symptoms in presenting patients and for occlusal signs of bruxism to be part of TMD etiology. However, this popularly hypothesized association is questioned in certain TMD patient studies[2,3] and in an earlier study of young adult nonpatients by the authors.[4] The present study continues this investigation in a symptomatic patient population.

Several articles have stated that bruxism is associated with occlusal interferences[5–9] and with some occlusal variations.[6] Nevertheless, the possible differences between acute experimental interference models and chronic occlusal variation need to be studied since it is the chronic occlusal model that must be examined for TMD etiology.

There is experimental evidence that TMD symptoms can be induced by acute placement of supracontacts equivalent to placement of a high restoration.[10–12] However, adaptation to acute interferences over time is also reported.[10,13] In contrast, an increasing body of research suggests that experimental deflective occlusal contacts may actually inhibit bruxism activity.[14–16] Although some recent EMG studies report increased symptom levels after placement of acute interferences, they involve too few subjects for adequate statistical analysis.[17,18] The equivocal data on acute experimental interferences have not prevented the belief that bruxism is induced by chronic occlusal interferences or occlusal variation in the general population and can be etiologic for TMD.

The authors contend that the extrapolation of information from acute experimental interferences to a chronic occlusal morphologic etiologic model is suspect, and the association between chronic occlusal interferences and bruxism is unclear. The bruxism-occlusal interference association is questioned by several studies.[14,19–22] With respect to the presence of TMD symptoms, few studies report any association to attrition levels,[9,23,24] and others report none.[2–4,25–30] EMG studies[15,31–34] and information from attrition in dental casts[4] indicate that bruxism is a universal habit.

Temporomandibular joint remodeling or degeneration has also been associated with bruxism[35] and thereby indirectly to occlusion. The basis for this hypothesis comes from skull studies or clinical impressions,[35] but no published studies were found that actually tested for attrition levels in patients with osteoarthrosis. The findings from various skull studies are not, however, in agreement. In one sample of Australian aboriginal skulls with osteoarthrosis, more severe posterior attrition was noted with arthrosis present,[36] but in another sample this association was reported only in mandibular teeth and not in the opposing dentition.[37] Canine wear has also been associated with flattening of the articular eminence in native American skulls.[38] In contrast, osteoarthrosis could not be predicted according to attrition severity in 18th century Londoner,[39] Romano-British,[40] or contemporary American[41] skulls. Findings from skull studies are difficult to interpret because they usually cannot reliably control for age, diet (related to functional wear), or gender. A recently published skull study that was able to control for age found no more osteoarthrosis in skulls with more severe attrition than those without.[42] The absence of the overlying articular soft tissue data makes in vivo comparisons to actual joint mechanics in living populations purely speculative.[43,44]

In contrast to the morphologic, structural, and peripheral etiologic concepts of bruxism, TMD symptoms may instead be related more to centrally induced hyperactivity. Several studies point to bruxism as a central nervous system disorder[6,45–47] originating in the limbic system and modulated in the cortex.[48] It has been emphasized that masticatory movements are driven by central pattern generators.[49,50]

Bruxism has also been described as a sleep disorder, related to emotional states.[15] There is evidence that bruxism movements relate to changes in sleep stages and are concurrent with generalized body movements[31] as well as with REM sleep.[51] It is evident that general body movements cannot be explained by an occlusal etiology.[31] Similarly, Rugh et al[52] point out that bruxism in symptomatic individuals predominantly occurs during REM sleep and can be differentiated from bruxism occurring in asymptomatic subjects, which is usually limited to non-REM sleep.

Only two studies were found that examined dental attrition levels in defined patient populations, but they were contradictory. Attrition severity in one study did not differentiate patients with arthrographically confirmed disc displacement from patients without derangement.[53] In contrast, greater attrition severity was reported in patients with reciprocal clicking who progressed to nonreducing disc displacement over a 3-year period than in patients whose condition remained stationary.[54]

The purpose of this study was to investigate any relationship of the cumulative record of dental attrition measured on dental casts to differentiated TMD patient groups. The null hypothesis was that differentiated groups of TMD patients could not be distinguished from matched asymptomatic subjects according to dental attrition. Age was controlled to control for the effects of functional wear.

Materials and Methods

Control Sample (Asymptomatic Normals)

One hundred men and 48 women without any symptoms, signs, or history of TMD were screened using an established protocol from a previously described population of dental and dental hygiene students[55] supplemented by consecutive male and female patients across a wider age range from a general dental practice.

Experimental Sample (TMD Patients)

Consecutive patients at the University of California–Los Angeles Pain Management Center,

Orofacial Pain Clinic and from a private practice setting were assigned to five patient groups with common diagnostic characteristics. These groups have been previously distinguished as independent by gender and age,[56] as well as by range of mandibular opening.[57]

Group 1: Disc Displacement With Reduction. This group (40 women) was defined by clinical signs of TMJ reciprocal clicking, whereby the joint sound corresponded to different positions on the mandibular movement pathways, and the clicks were reduced through chairside trial mandibular repositioning. There was an absence of clinical signs (crepitation) or radiographic signs (condylar erosion, regressive or proliferative changes) associated with arthrosis.

Group 2: Disc Displacement Without Reduction. This group (26 women) was defined by a history of TMJ clicking prior to closed lock and showed no clinical or radiographic signs of arthrosis.

Group 3: TMJ Osteoarthrosis With a Prior History of TMJ Derangement. This group (50 women) was defined by the clinical presence of crepitation and radiographic signs of significant intracapsular osseous changes. The patients reported a history of prior mandibular movement restriction associated with an intracapsular TMJ interference due to clicking or locking.

Group 4: TMJ Osteoarthrosis Without a Prior History of Derangement (Primary Osteoarthrosis). This group (39 women) was defined as in group 3, but without any known earlier history of mandibular movement interference due to TMJ clicking or locking.

Group 5: Myalgia Only. This group (42 women and 31 men) was defined by the presence of four or more sites of moderate to severe tenderness in the masticatory musculature on palpation and the absence of any symptom or sign of an intracapsular TMJ disorder.

All samples were examined according to the same criteria.[55] The clinical groups with TMJ dysfunction were also examined by serial tomographic imaging in the sagittal and frontal planes of both TMJs. An oral radiologist interpreted the results. The numbers of men in groups 1 (n = 10); 2 (n = 2); 3 (n = 4); and 4 (n = 4) were too few to analyze.

Evaluation of Attrition

Dental casts were graded for severity and location of wear facets (Fig 1). All scoring was performed by a calibrated observer after consensus evaluation by both authors of 10 casts selected at random.

The severity scoring was a contraction of the method of Richards and Brown[36] abbreviated to fewer groups because of the inability to absolutely identify dentin exposure on dental casts. All scoring was performed by one examiner according to the following five-point scale: 0 = no wear; 1 = minimal wear; 2 = noticeable flattening parallel to the occluding planes; 3 = flattening of cusps or grooves; and 4 = total loss of contour and/or dentinal exposure when identifiable.

The facets were graded in seven zones: incisor; right and left canine; right and left premolar and molar laterotrusion (Fig 1, a, c); and right and left premolar and molar mediotrusion (Fig 1, b). The worst-score finding was recorded in each zone.

The maximum possible laterotrusion score for the posterior dentition was 2 premolar and 2 molar zones × 2 facet locations × maximum severity score of 4 = 32. The maximum possible mediotrusion score for the posterior dentition was 2 premolar and 2 molar zones × 1 facet location × maximum severity score of 4 = 16. The maximum possible anterior score was 1 incisor and 2 canine zones × 1 facet location × maximum severity score of 4 = 12.

Analysis

It was important to control for functional wear, supposedly occurring with age, so the study examined the differences in mean scores between each patient group and the controls according to age.

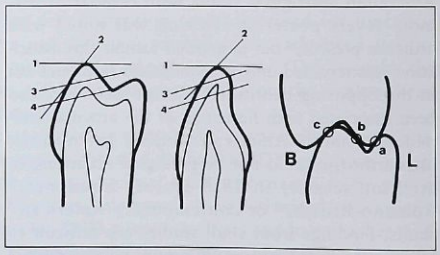

Fig 1 Attrition score criteria *(left and center)*: 1 = minimal wear of cusp or incisal tips; 2 = facets parallel to the normal planes of contour; 3 = noticeable flattening of cusps or incisal edges; 4 = total loss of contour and dentinal exposure when identifiable. Locations a and c correspond to contact in laterotrusion and location b to mediotrusion posterior tooth facets (B = buccal; L = lingual).

To increase the power through the use of sufficient numbers of subjects and reduce beta-type errors (defined as a statistical determination of no difference when one actually exists), 10-year age intervals were used in the analysis (Figs 2 to 5). To enable a valid prediction at a confidence level of $P < .01$, 2.5 standard errors (SE) in the differences between the means were used to isolate absolute differences of the patient groups from the asymptomatic controls. Analysis of covariance for age and patient group (type III sum of squares, SS) was used to predict differences in attrition scores (SuperANOVA, Abacus Concepts, Berkeley, California).

An assumption was made that the sample size and the range of the attrition scores (0 to 60) permitted the data to be treated as a continuous scale. This was sustained by a strong correlation of median to mean scores ($r = .89$). Individual differences from the mean were plotted on a scatter chart for each patient group to test for residuals and confirm normal distributions. A Games-Howell post-hoc test for differences in the mean attrition scores was performed to confirm differences between patient groups found with the ANCOVA. To control for chance relationships (alpha errors) in an extensive analysis involving 224 paired comparison tests, a difference of $P < .01$ was interpreted to be a significant difference in attrition rather than $P < .05$.

Age versus attrition effects within patient groups and intergroup patient comparisons were not considered in this study. Analysis of male patients was only conducted in a myalgia-only group because of the low male prevalence within the other diagnostic groups.

Results

The mean difference in attrition scores of the asymptomatic controls from each patient group (disease group score minus the asymptomatic controls score) at each age range is shown in Figs 2 to 5. The results are divided into scores for the anterior, laterotrusion, and mediotrusion segments. A positive mean score difference represents greater attrition in the patient group, and a negative mean score difference means greater attrition in the controls. The brackets around each score difference represent ±2.5 SE. A clinically useful predictor of a difference in the scores ($P < .01$: equivalent to high specificity) could only be assumed when the bracket was entirely above or below the zero line.

Anterior Attrition (Table 1, Figs 2 and 5)

Women older than age 59 in the specific group with primary osteoarthrosis (group 4) showed a trend for less attrition in the anterior dentition than the controls ($P = .0379$). No other differences were found in anterior attrition scores for the osteoarthrosis groups (groups 3 and 4), and no differences were found at any age for the derangement and myalgia groups (groups 1, 2, and 5) compared to the controls.

Table 1 Significant Trends Between Differentiated Patient Groups and Controls (ANCOVA)*

Group	Age	Factor	*df*	SS	$\bar{x}^2$	f	*P*
Anterior attrition score							
Women	60+	Disease	1	22.412	22.412	5.909	.038
Osteoarthrosis –		Residuals	9	34.133	3.793		
Posterior laterotrusive attrition score							
Women	20 to 29	Disease	1	107.789	107.789	6.032	.019
Osteoarthrosis +		Residuals	36	643.263	17.868		
Women	20 to 29	Disease	1	104.021	104.021	6.657	.017
Osteoarthrosis –		Residuals	22	343.937	15.633		
Posterior mediotrusive attrition score							
Women	40+	Disease	1	26.535	26.535	4.864	.038
Myalgia		Residuals	23	125.465	5.455		

*Remainder of NS tests available on request to the authors.

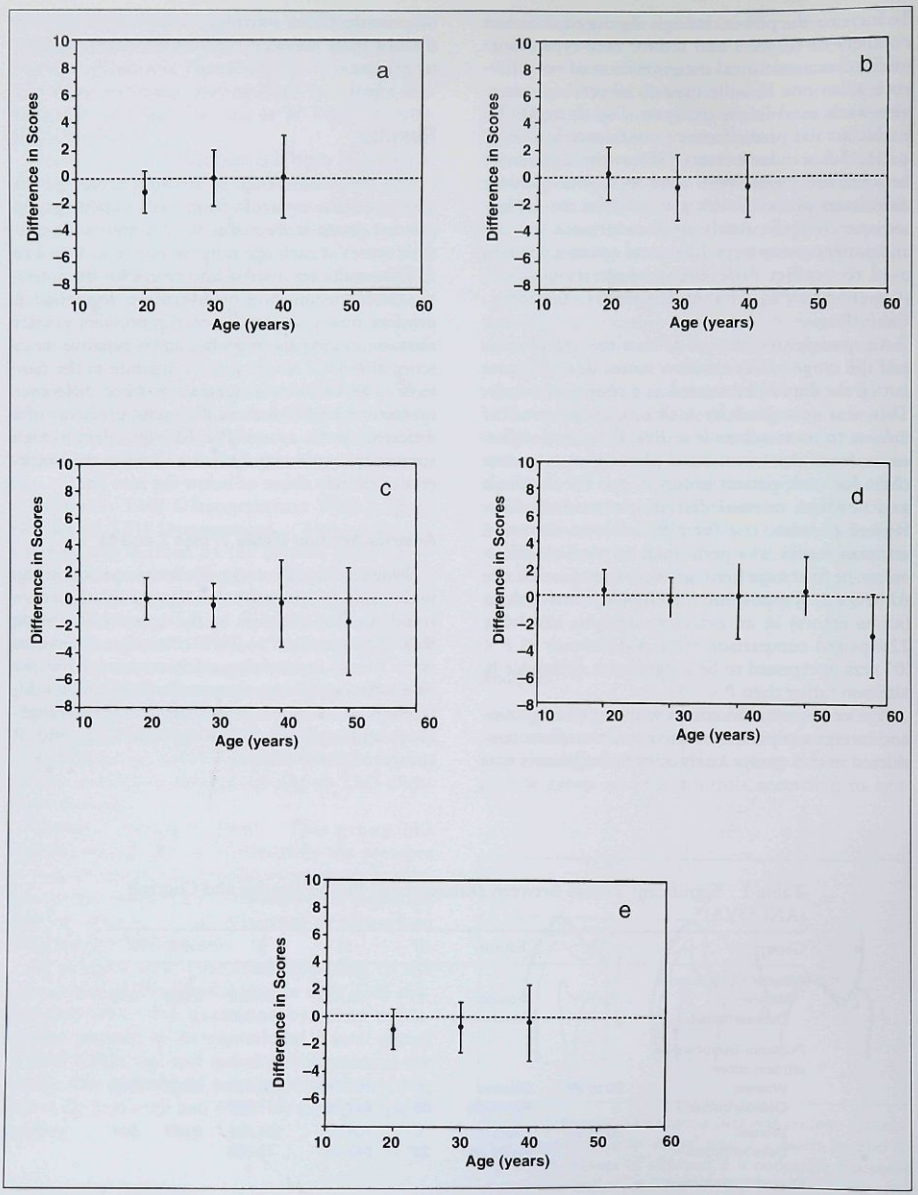

Fig 2 Female anterior attrition scores. Mean score difference between each diagnosis and the asymptomatic female control group for each 10-year age range. Brackets represent 2.5 × the SE. The 0-line represents no difference. (a) disc displacement with reduction; (b) disc displacement without reduction; (c) osteoarthrosis with a history of prior derangement; (d) primary osteoarthrosis; (e) myalgia only.

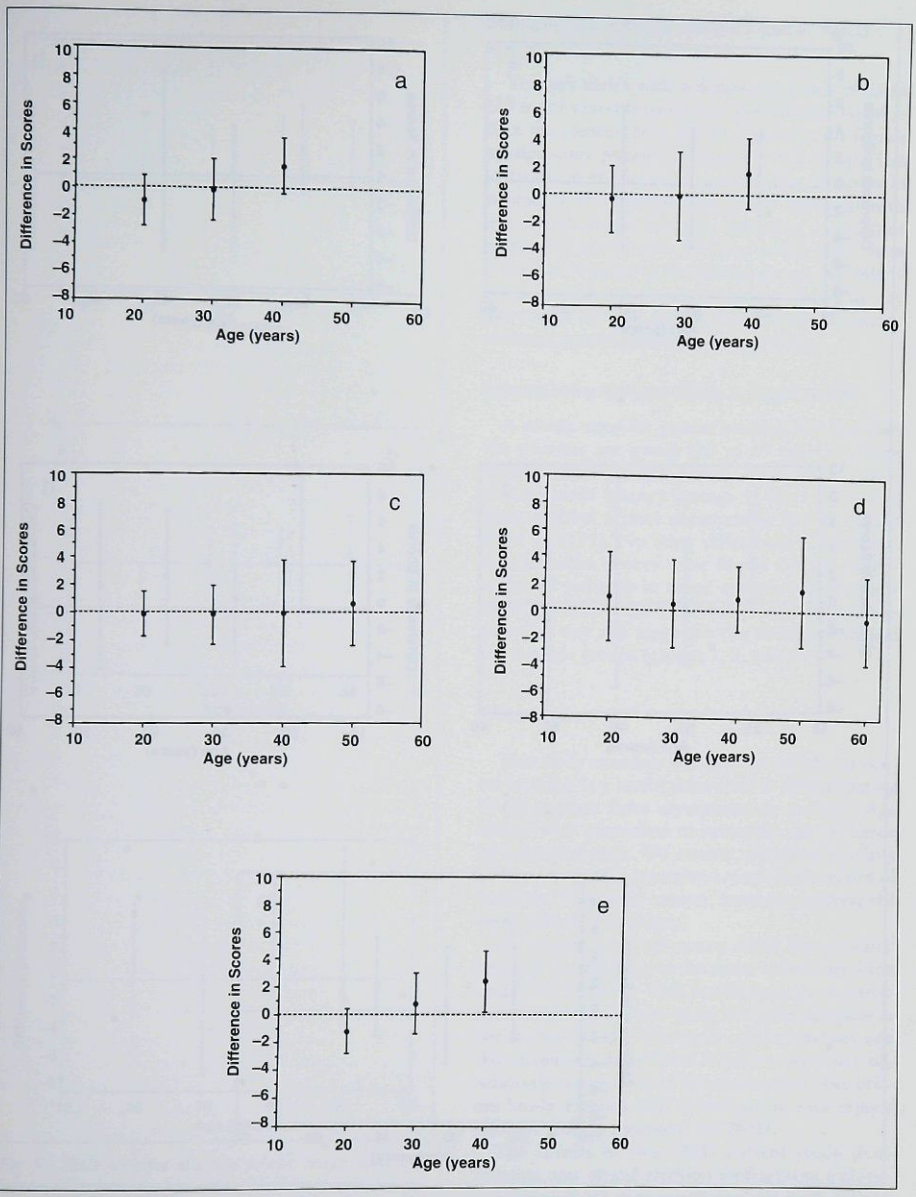

Fig 3 Female mediotrusive attrition scores. Mean score difference between each diagnosis and the asymptomatic female control group for each 10-year age range. Brackets represent 2.5 × the SE. The 0-line represents no difference. (a) disc displacement with reduction; (b) disc displacement without reduction; (c) osteoarthrosis with a history of prior derangement; (d) primary osteoarthrosis; (e) myalgia only.

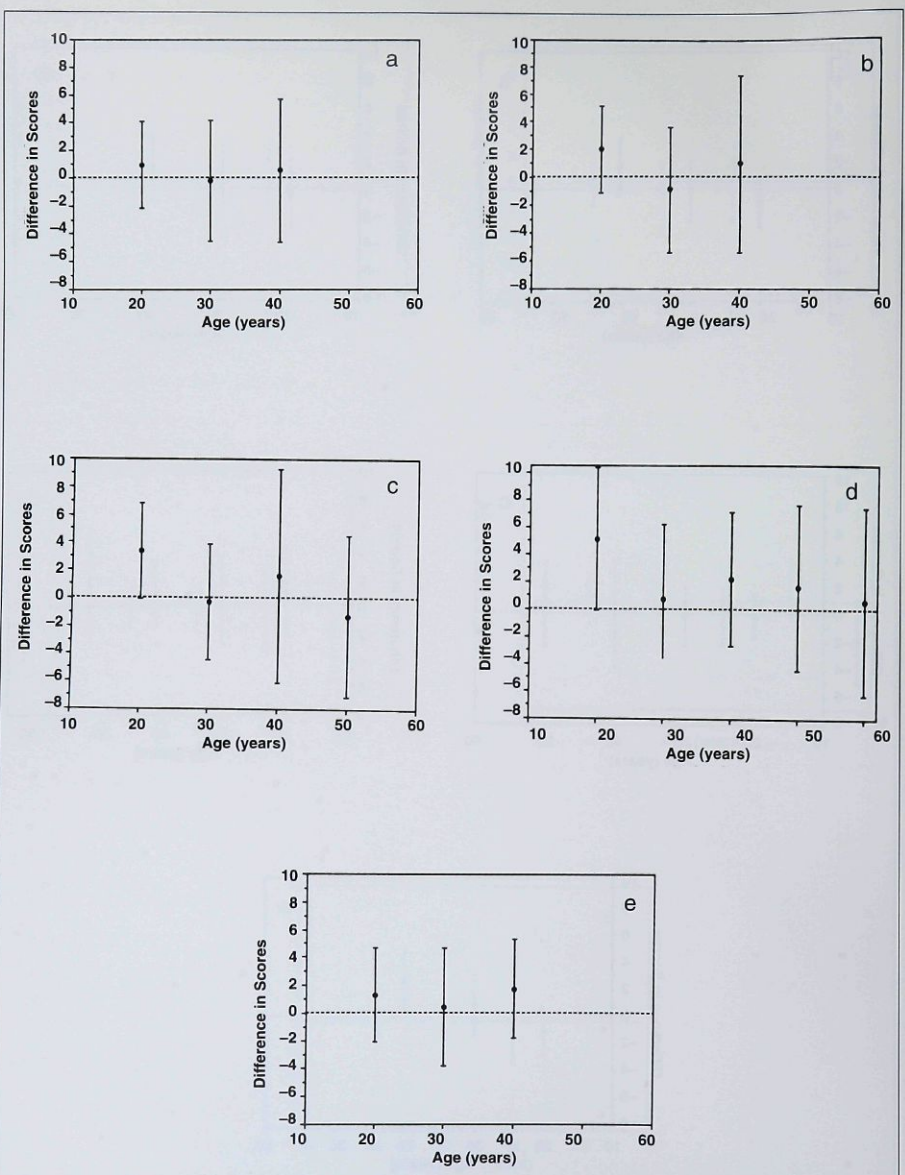

Fig 4 Female laterotrusive attrition scores. Mean score difference between each diagnosis and the asymptomatic female control group for each 10-year age range. Brackets represent 2.5 × the SE. The 0-line represents no difference. (a) disc displacement with reduction; (b) disc displacement without reduction; (c) osteoarthrosis with a history of prior derangement; (d) primary osteoarthrosis; (e) myalgia only.

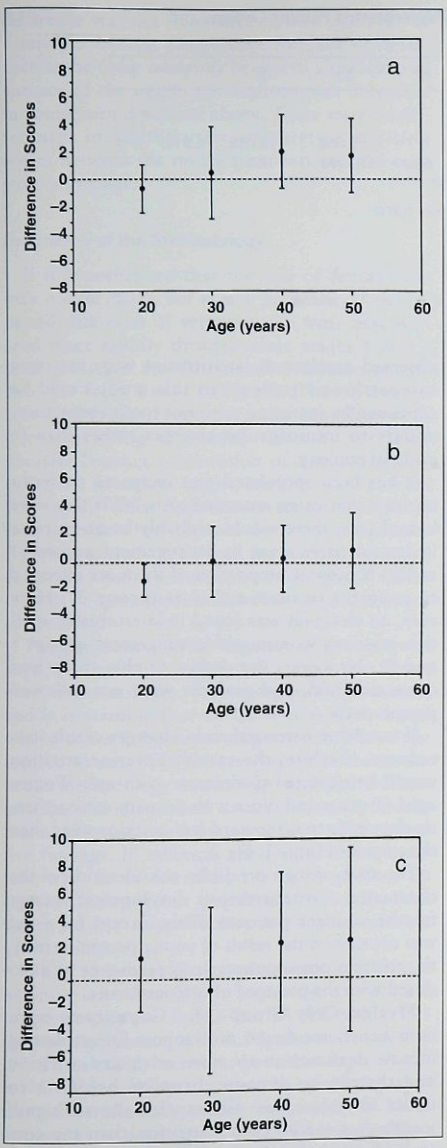

Fig 5 Male anterior attrition scores. Mean score differences between myalgia-only patients and male asymptomatic controls in (a) anterior; (b) mediotrusive; and (c) laterotrusive locations for each 10-year age range. Brackets represent 2.5 × the SE. The 0-line represents no difference.

Mediotrusive Attrition (Tables 1 and 2, Figs 3 and 5)

Younger males with a diagnosis of only myalgia (20 to 29 years) showed less mediotrusive attrition than the controls ($P = .0105$). Similarly, the female myalgia-only patients showed a trend toward less attrition in the younger group. The attrition in women in the myalgia-only group increased with age relative to the controls, becoming a statistical trend after age 40 ($P = .0377$) at which point there is greater attrition in the disease group. No differences in mediotrusive attrition were found at any age range for the derangement and the osteoarthrosis groups (groups 1, 2, 3, and 4).

Laterotrusive Attrition (Table 1, Figs 4 and 5)

A strong trend for greater attrition was found in the younger age group (20 to 29 years) in both osteoarthrosis groups, namely those with a prior derangement history (group 3) ($P = .019$) and those without a prior derangement history (group 4) ($P = .0171$). No other differences in laterotrusive attrition scores were found for the osteoarthrosis patients in other age groups. Furthermore, no differences in laterotrusive attrition were found at any age range for the derangement and the myalgia groups (groups 1, 2, and 5).

Discussion

This study examines whether the severity of dental attrition is a useful parameter in differentiating TMD patients from asymptomatic controls. Age effects were controlled to neutralize the influence of functional wear. We assume, therefore, that any differences mainly represent a cumulative record of bruxism. This study cannot, however, address the static effects of clenching.

An earlier study of young adult nonpatients[4] showed no association between dental attrition and TMJ clicking, TMJ tenderness, or masticatory muscle tenderness using the same protocol as the present study. Inherently, osteoarthrosis and disc displacement without reduction were not significantly represented in that population. The present study extends that investigation into typical patients seeking treatment for TMD.

The results of this TMD patient study demonstrate that dental attrition representing a chronic bruxism habit does not distinguish these patients from asymptomatic subjects. This suggests that the effects of dynamic bruxism have been overstated as a peripheral cause for TMD.

Table 2 Significant Differences Between Differentiated Patient Groups and Controls (ANCOVA)*

Group	Age	Factor	*df*	SS	$\bar{x}^2$	f	*P*
Posterior mediotrusive attrition score							
Men	20 to 29	Disease	1	15.058	15.058	6.918	.011
Myalgia		Residuals	69	150.182	2.177		

*Remainder of NS tests available on request to the authors.
Games–Howell post-hoc test: Difference: 1.273 (Critical difference 0.971).

The controls were compared with patients differentially diagnosed according to common clinical characteristics. Although the classifications were clinically defined, the selected patient groups had been previously differentiated according to age, gender, and range of motion[56,57] and were considered valid independent groups. Earlier studies analyzed less specifically defined TMD patients or combined all TMD patients into a single patient group.[58] The authors believe that real differences may be more evident when populations are more specifically defined as in the present study.

Examination of attrition in males was restricted to subjects with myalgia only (group 5) due to the lower prevalence of male patients in certain disease categories seeking treatment.[56]

Attrition in TMD Patient Groups

Derangement Groups (1 and 2). Because the attrition scores for women with disc displacement with or without reduction could not be differentiated from those in asymptomatic controls, the use of attrition as a clinical predictor of derangement pathology would have unacceptably low specificity.

Arthrosis Groups (3 and 4). Greater laterotrusive posterior attrition was found in younger patients (20 to 29 years) with TMJ osteoarthrosis than in the controls (Table 1, Fig 4). Laterotrusive attrition could not, however, differentiate older patients. This supports earlier speculation that younger patients with osteoarthrosis may have to be classified separately from older populations.[56]

It is speculated that the increased laterotrusive attrition seen in younger osteoarthrosis patients is a consequence of condylar changes associated with rapidly developing arthrosis. We have previously attributed the prevalence of reduced overbite[59] and anterior open bite[60] in osteoarthrosis patients to intracapsular osseous changes. Similarly, the observed accelerated laterotrusive wear in young osteoarthrosis patients in this study could be explained by increased posterior tooth contact secondary to intracapsular changes rather than to occlusal etiology.

It has been speculated that increased posterior attrition can cause osteoarthrosis.[35–38] If this were indeed true, there would probably be an increase in laterotrusive wear in derangement groups (1 and 2) because a proportion of them are expected to progress to osteoarthrosis (group 3). However, no elevation was found in laterotrusive attrition in young women with derangement (groups 1 and 2). However, the design of this study was cross-sectional, and patients were not followed prospectively.

If condylar osteoarthrosis changes result in a reduced overbite, the rate of anterior attrition would be expected to decrease with age. Women aged 60 years and older with primary osteoarthrosis showed a trend toward less anterior wear than the controls (Table 1, Fig 2).

The study design precludes consideration of the chronicity of osteoarthrosis development, except for the younger patients. Thus, except for what was considered the result of young person's arthritis, attrition does not appear to predict or be associated with the presence of osteoarthrosis.

Myalgia-Only Group (5). Contrary to common belief, we found no support for attributing muscle dysfunction to more advanced attrition and thereby to dynamic bruxism habits. Men under 30 years of age with myalgia showed significantly less mediotrusive attrition than the controls (Table 2, Fig 5). Similarly, less mediotrusive attrition was observed in young women, but there was no statistically significant difference. Although there was a slightly elevated trend for mediotrusive attrition in older female patients (40 to 50 years) with myalgia (Table 1, Fig 3), this

difference was not duplicated in men and may be a random finding. Conversely, the lack of difference in the older men may be due to a natural persistence of the significant mediotrusion difference in young men described above. There were no differences in laterotrusive or anterior attrition scores between the myalgia patients and the controls at any age.

Evaluation of the Methodology

It is hypothesized that the rate of dental wear may not be linear, but it may be phasic.[1] It is suspected that rates of vertical tooth wear may proceed more rapidly through score stages 1 and 2 until pressures are distributed over increasingly broader facet areas (score 3). Then particles released from irregular wear together with microfracturing of enamel rods may act as an abrasive causing acceleration of dental attrition (score 4). At certain stages, attrition may proceed very rapidly when dissimilar surfaces come in contact, such as when exposed dentin meets enamel. The present study deals with the cumulative record of attrition and is not sensitive to the phasic nature of wear or symptoms. It also does not address static clenching.

The predominance of negative differences was supported mainly by adequate sample size. However, some samples were marginally restricted, and it is possible that no differences were found where some differences might exist (beta-type errors). Nonetheless, the consistency of the results and a methodology employing strict and uniform criteria, including blind data collected by a calibrated examiner, support acceptance of the negative findings. In addition, the results were consistent with those found by the same authors on another independent population of nonpatient young adults,[4] which further supports acceptance of the results.

While some significant relationships to dental attrition were found, it is important to maintain proper perspective: only 5 out of 112 ANCOVAs showed significance at $P < .05$, and only 1 at $P < .01$. These could, therefore, be within expectations for random findings. It must be concluded that there is no clinically useful relationship of TMD symptoms to attrition levels. Only one patient group (young men with myalgia) showed a difference in attrition compared to the controls, but the absolute mean score difference of −1.0 was actually very small. In clinical terms, the small differences discussed above are unlikely to be discernible to the examining clinician.

Conclusions

The results of this study have the following impact on clinical decision making:

1. Dental attrition as an isolated variable should not be considered as predisposing to TMD problems.
2. Conversely, dental attrition should not be considered a consequence of an existing TMD disease, except possibly for laterotrusive attrition in young adult osteoarthritis (or any rapidly progressing arthritis).
3. Chronic mediotrusive and laterotrusive attrition contacts appear to be common and have little or no association to the presence of TMD symptoms or disease. However, the present study cannot address the effects of a sudden imposition of occlusal interference through reconstruction or orthodontics, which is equivalent to an acute experimental interference.
4. The lack of any evident attrition relationship to TMD symptoms or disease when generalized to the population suggests that this component of any occlusal peripheral etiology has been overstated. It seems more likely that attrition-bruxism is an epiphenomenon of centrally mediated neurologic activity, which has a variable relationship to producing symptoms.
5. Restorative treatment for attrition is defendable if the integrity of a tooth is compromised, but the belief in restoring the structural effects of attrition to treat current TMD problems or to prevent anticipated TMD problems does not appear to be supportable.
6. Because the controls showed similar attrition scores to the patients, dentists should recognize the potential for bruxism in all patients and plan their standard restorative and periodontal treatments accordingly where tooth integrity and alveolar support is compromised.
7. Temporary control of an active bruxism habit may still be desirable to relieve and gain control over acutely inflamed and painful tissues in dysfunctional patients. However, short-term control of a bruxism habit does not imply an occlusal etiology or an indication for occlusal treatment.

References

1. Lambrechts P, Braem M, Vuylsteke-Wauters M, Van Herle G. Quantitative in vivo wear of human enamel. J Dent Res 1989;68:1752–1754.

2. De Kanter RJAM, Van Bladel AJWP, Partman SRK, Van 'T Hof MA. Mandibular dysfunction and attrition, age and sex influences [abstract 209]. J Dent Res 1989;68,4 (special issue):635.
3. Lederman KH, Clayton JA. Restored occlusions. Part II: The relationship of clinical and subject symptoms to varying degrees of TMJ dysfunction. J Prosthet Dent 1982;47: 303–309.
4. Seligman DA, Pullinger AG, Solberg WK. The prevalence of dental attrition and its association with factors of age, gender, occlusion, and TMJ symptomatology. J Dent Res 1988;67:1323–1333.
5. Graf H. Bruxism. Dent Clin North Am 1969;13:359–365.
6. Kopp S. Pain and functional disturbances of the masticatory system: A review of etiology and principles of treatment. Swed Dent J 1982;6:49–60.
7. Krough-Poulson WG, Olsson A. Occlusal disharmonies and dysfunction of the masticatory system. Dent Clin North Am 1966; 11:627–635.
8. De Boever JA. Functional disturbances of the temporomandibular joint. In: Zarb G, Carlsson GE (eds). Temporomandibular Joint Function and Dysfunction. Copenhagen: Munksgaard, 1979:193–214.
9. De Laat A, van Steenberghe D, Lesaffre E. Occlusal relationships and temporomandibular joint dysfunction. Part II: Correlations between occlusal and articular parameters and symptoms of TMJ dysfunction by means of stepwise logistic regression. J Prosthet Dent 1986;55:116–121.
10. Christiansen J. Effect of occlusion-raising procedure on the chewing system. Dent Pract 1970;20:233–238.
11. Riise C. Rational performance of occlusal adjustment. J Prosthet Dent 1982;48:319–327.
12. Randow K, Carlsson K, Edlund J, Öberg T. The effect of an occlusal interference on the masticatory system. Odontol Rev 1976;27:245–256.
13. Joo HY, Kim KN. Influence of artificial occlusal interference on A. Temporalis and Masseter activity [abstracts 0–18]. J Dent Res 1989;68,4(special issue):689
14. Schaerer P, Stallard MS, Zander HA. Occlusal interferences and mastication: An electromyographic study. J Prosthet Dent 1967:17:438–449.
15. Rugh JD, Barghi N, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxism. J Prosthet Dent 1984; 51:548–553.
16. Ingergall B, Mohlin B, Thilander B. Prevalence of symptoms of functional disturbances of the masticatory system in young Swedish men. J Oral Rehabil 1980; 7:185–197.
17. Kobayashi Y, Hansson TL. Auswirkung der okklusion auf den menschlichen koerper. Phillip J 1988;5:255–263.
18. Takeda Y, Ishihara H, Kobayashi Y. Influence of occlusal interference on nocturnal sleep and masseter muscle activity [abstract 561]. J Dent Res 1989;68,4(special issue):936.
19. Hannum AG, De Cou RE, Scott JD, Wood WW. The relationship between dental occlusion, muscle activity and associated jaw movement in man. Arch Oral Biol 1977; 22:25–32.
20. Cachiotti D, Bianchi P, McNeill C, Plesh O. TMJ disorders: Comparing signs and symptoms in two populations [abstract 621]. J Dent Res 1989;68,4(special issue):259.
21. Egermark-Eriksson I, Carlsson GE, Magnussen T. A long-term epidemiologic study of the relationship between occlusal factors and mandibular dysfunction in children and adolescents. J Dent Res 1987;66:67–71.
22. Kotran M, Haber B, Markovska N, Stripaj M. Bruxism and other parafunctions in the symptomatology of arthropathies of the temporomandibular joint. Cesk Stomatol 1983;83: 136–142.
23. Runge ME, Sadowsky C, Sakols EL, BeGole EA. The relationship between temporomandibular joint sounds and malocclusion. Am J Orthod 1989;96:36–42.
24. Lieberman MA, Gazit E, Fuchs C, Lilos P. Mandibular dysfunction in 10–18 year old schoolchildren as related to morphological malocclusion. J Oral Rehabil 1985;12: 209–214.
25. Droukas B, Lindee C, Carlsson GE. Relationship between occlusal factors and signs and symptoms of mandibular dysfunction. Acta Odontol Scand 1984;42:277–283.
26. Sigaroudi K, Knap FJ. Analysis of jaw movements in patients with temporomandibular joint click. J Prosthet Dent 1983;50:245–250.
27. Seligman DA, Pullinger AG. Association of occlusal variables among refined TM patient diagnostic groups. J Craniomandib Disord Facial Oral Pain 1989;3:227–236.
28. De Boever JA, van dem Berghe L. Longitudinal study of functional conditions in the masticatory system in Flemish children. Community Dent Oral Epidemiol 1989;15:100–103.
29. Meng HP, Dibbets JMH, van der Weele LT, Boering G. Symptoms of temporomandibular joint dysfunction and predisposing factors. J Prosthet Dent 1987;57:215–222.
30. Reuling N. Comparative study of clinical examination, occlusal analysis and new radiological imaging procedures in patients with functional TMJ disorders. J Oral Rehabil 1987;14:165–174.
31. Fuchs P. Muscular activity of the chewing apparatus during sleep. J Oral Rehabil 1975;2:35–48.
32. Clark ST, Beemsterboer PL, Rugh JD. Nocturnal masseter muscle activity and the symptoms of masticatory dysfunction. J Oral Rehabil 1981;8:279–286.
33. Clark NG, Townsend GC, Carey SE. Bruxing patterns in man during sleep. J Oral Rehabil 1984;11:123–127.
34. Kydd WL, Dayley C. Duration of nocturnal tooth contacts during bruxism. J Prosthet Dent 1985;53:717–721.
35. Keith DA. Etiology and diagnosis of temporomandibular pain dysfunction: Organic pathology (other than arthritis). In: Laskin D et al (eds). Report of the President's Conference on the Examination, Diagnosis, and Management of Temporomandibular Disorders. Chicago: American Dental Association, 1983:118–122.
36. Richards LC, Brown T. Dental attrition and degenerative arthritis of the temporomandibular joint. J Oral Rehabil 1981;8:293–307.
37. Richards LC. Degenerative changes in the temporomandibular joint in two Australian aboriginal populations. J Dent Res 1988;67:1529–1533.
38. Granados J. The influence of the loss of teeth and attrition on the articular eminence. J Prosthet Dent 1979;42:78–85.
39. Whittaker DK. Surface and form changes in the temporomandibular joints of 18th century Londoners [abstract 1211]. J Dent Res 1989;68,4(special issue).
40. Whittaker DK, Davies G, Brown M. Tooth loss attrition, and temporomandibular joint changes in a Romano-British population. J Oral Rehabit 1985;12:407–419.
41. Eversole LR, Pappas JR, Graham R. Dental occlusal wear and degenerative disease of the temporomandibular joint: A correlational study utilizing skeletal material from a contemporary population. J Oral Rehabil 1985;12: 401–406.
42. Whittaker DK, Jones JW, Eduards PW, Molleson T. Studies on the temporomandibular joint of an eighteenth-century London population (Spitalfields). J Oral Rehabil 1990;17:89–97.

43. Baldioceda F, Pullinger AG, Bibb CA. Relationship of condylar bone profiles and dental factors to articular soft tissue thickness. J Craniomandib Disord Facial Oral Pain 1990;4:71–79.
44. Pullinger AG, Baldioceda F, Bibb CA. Relationship of TMJ articular tissue to underlying bone in young adult condyles. J Dent Res 1990;69:1512–1518.
45. Yemm R. Causes and effects of hyperactivity of the jaw muscles. In: Bryant P et al (eds). Oral Motor Behavior: Impact on Oral Conditions and Dental Treatment. Washington, DC, NIH Publication 1979a, 79-12845: 138–156.
46. Yemm R. Neurophysiologic studies of temporomandibular dysfunction. In: Zarb G, Carlsson GE (eds). Temporomandibular Joint Function and Dysfunction. Copenhagen: Munksgaard, 1979:231.
47. Holmgren K, Sheiksholeslam A. Long-term study of the effects of an occlusal splint in patients with parafunctional disorders [abstract 1068]. J Dent Res 1978;57(special issue).
48. Schaerer P. Bruxism. In: Ferguson DB (ed). Frontiers of Oral Physiology, vol 3. Basel, Switzerland: Karger, 1981: 293–322.
49. Lund JP, Dellow PG. The influence of interactive stimuli on rhythmical masticatory movements in rabbits. Arch Oral Biol 1971;16:215–223.
50. Dellow PG, Lund JP. Evidence for central timing of rhythmical mastication. J Physiol 1971;215:1–13.
51. Okeson JP, Phillips BA, Berry DTR, Cook Y, Paesani D, Galante J. Nocturnal bruxing events in healthy geriatric subjects. J Oral Rehabil 1990;17:411–418.
52. Rugh JD, Ware JC. Polysomnographic comparison of nocturnal bruxists with and without pain [abstract 97]. J Dent Res 1986;65(special issue).
53. Roberts CA, Tallents RH, Katzberg RW, Sanchez-Woodworth RE, Espeland MA, Handelman SL. Comparison of internal derangements of the TMJ with occlusal findings. Oral Surg Oral Med Oral Pathol 1987;63: 645–650.
54. Lundh H, Westesson P-L, Kopp S. A three-year follow-up of patients with reciprocal temporomandibular joint clicking. Oral Surg Oral Med Oral Pathol 1987;65:530–533.
55. Pullinger AG, Seligman DA, Solberg WK. Temporomandibular disorders: Part 1. Functional status, dentomorphologic features, and sex differences in a non-patient population. J Prosthet Dent 1988;59:228–235.
56. Pullinger AG, Seligman DA. TMJ osteoarthrosis: A differentiation of diagnostic subgroups by symptom history and demographics. J Craniomandib Disord Facial Oral Pain 1987;1:251–256.
57. Seligman DA, Pullinger AG. TMJ derangements and osteoarthrosis subgroups differentiated according to active range of mandibular opening. J Craniomandib Disord Facial Oral Pain 1988;2:35–40.
58. Rasmussen OC. Description of population and progression of symptoms in a longitudinal study of temporomandibular arthropathy. Scand J Dent Res 1981;89:196–203.
59. Pullinger AG, Seligman DA. Overbite and overjet characteristics of refined diagnostic groups of temporomandibular patients. Am J Orthod Dentofac Orthop. In press.
60. Seligman DA, Pullinger AG. Association of occlusal variables among refined TM patient diagnostic groups. J Craniomandib Disord Facial Oral Pain 1989;3:227–236.

Resumen

El grado en que la atrición caracteriza a grupos diferenciados de pacientes con desórdenes temporomandibulares

Se clasificó la severidad de la atrición dental como el registro cumulativo de los desgastes parafuncionales y funcionales, por medio del análisis de modelos de estudio utilizando una metodologia establecida. Se comparó la severidad de la atrición en los siguientes segmentos: anterior, mediotrusión posterior, y laterotrusión posterior. Los puntajes de atrición en el grupo totalmente asintomático (control) compuesto por 48 mujeres y 100 hombres fueron comparados a los de 239 mujeres y 31 hombres diferenciados en 5 grupos de pacientes afectados por desórdenes temporomandibulares. Tales grupos fueron los siguientes: (1) desplazamiento del disco con reducción, (2) desplazamiento del disco sin reducción, (3) osteoartrosis con historia de malfuncionamiento interno previo, (4) osteoartrosis sin historia de malfuncionamiento interno previo, y (5) mialgia solamente. Todos los pacientes del sexo masculino pertenecian al grupo afectado por mialgia. Las edades fueron agrupadas en el análisis de control del desgaste funcional. Las comparaciones entre los pacientes y los controles fueron hechos conforme a intervalos de edad de 10 años. Los análisis realizados incluyeron el ANCOVA (Análisis de Covarianza) el cual se confirmó por medio de una prueba de Games–Howell, después, con un $P < .01$ interpretado como una diferencia significativa en el puntaje de atrición. Sólo 1 de 112 ANCOVAS demostraron una diferencia significativa, en cuanto que los varones jóvenes de 20 a 29 años de edad que sufrian de mialgias y quienes solamente presentaban atrición, en mediotrusión inferior en comparación a los varones que servian de control. Por lo tanto seria dificil si no imposible el diferenciar entre los pacientes y los que no calificaban como pacientes, basados en la severidad de la atrición dental. Por consiquiente, el papel oclusal periférico etiológico de la atrición es cuestionable en los desórdenes temporomandibulares. Se elaboran algunas implicaciones clinicas.

Zusammenfassung

Kann die Abrasion Patienten mit unterschiedlichen Formen von Myoarthropathien des Kausystems unterscheiden?

Die Schwere der dentalen Abrasion, die das kumulative Resultat von parafunktioneller und funktioneller Abnutzung ist, wurde durch Modellanalysen mittels einer etablierten Methodik quantifiziert. Die Abrasion wurde jeweils im Frontzahnbereich und an Mediotrusionssowie Laterotrusionsfacetten im Seitenzahngebiet verglichen. Der aus 48 Frauen und 100 Männern bestehenden völlig symptomfreien Kontrollgruppe wurden 239 weibliche und 31 männliche Patienten gegenübergestellt. Letztere waren in 5 Gruppen eingeteielt: (1) Diskusverlagerung mit Reposition, (2) Diskusverlagerung ohne Reposition, (3) Arthrose mit vorausgegangener Diskusverlagerung, (4) Arthrose ohne vorausgegangene Diskusverlagerung und (5) Tendomyopathie. Alle männlichen Patienten gehörten zur Tendomyopathie-Gruppe. In der Analyse wurde das Patientenalter berücksichtigt, um die funktionelle Verschleisskomponente miteinzubeziehen. Die

Vergleiche zwischen den Patienten und den Kontrollpersonen wurden nach Einteiung in Altersgruppen mit 10-Jahresintervallen vorgenommen. Die Daten wurden statistisch mit dem ANCOVA und dem Games-Howell post-hoc Test auf eine Signifikanz von $P < .01$ untersucht. Nur 1 von 112 ANCOVAs zeigte einen signifikanten Unterschied zwischen Patienten und Kontrollpersonen. Junge Männer zwischen 20 und 29 Jahren aus der Tendomyopathie-Gruppe wiesen weniger Abrasion an unteren Mediotrusionsfacetten auf als Kontrollpersonen. Es ist daher schwierig, wenn nicht sogar unmöglich, anhand des Abrasionsgrades Patienten von asymptomatischen Personen zu unterscheiden. Folglich ist es fragwürdig, ob die Abrasion als okklusaler peripherer Faktor eine wichtige Rolle bei der Entstehung von Myoarthropathien spielt.