Occlusal Factors Are Not Related to Self-Reported Bruxism

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Aims: *To estimate the contribution of various occlusal features of the natural dentition that may identify self-reported bruxers compared to nonbruxers.* ***Methods:*** *Two age- and sex-matched groups of self-reported bruxers (n = 67) and self-reported nonbruxers (n = 75) took part in the study. For each patient, the following occlusal features were clinically assessed: retruded contact position (RCP) to intercuspal contact position (ICP) slide length (< 2 mm was considered normal), vertical overlap (< 0 mm was considered an anterior open bite; > 4 mm, a deep bite), horizontal overlap (> 4 mm was considered a large horizontal overlap), incisor dental midline discrepancy (< 2 mm was considered normal), and the presence of a unilateral posterior crossbite, mediotrusive interferences, and laterotrusive interferences. A multiple logistic regression model was used to identify the significant associations between the assessed occlusal features (independent variables) and self-reported bruxism (dependent variable).* ***Results:*** *Accuracy values to predict self-reported bruxism were unacceptable for all occlusal variables. The only variable remaining in the final regression model was laterotrusive interferences (*P = *.030). The percentage of explained variance for bruxism by the final multiple regression model was 4.6%. This model including only one occlusal factor showed low positive (58.1%) and negative predictive values (59.7%), thus showing a poor accuracy to predict the presence of self-reported bruxism (59.2%).* ***Conclusion:*** *This investigation suggested that the contribution of occlusion to the differentiation between bruxers and nonbruxers is negligible. This finding supports theories that advocate a much diminished role for peripheral anatomical-structural factors in the pathogenesis of bruxism.*
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Key words: bruxism, occlusal features, predictive value

The etiology of bruxism is one of the most debated issues in dentistry. Past theories on the purported role of dental occlusion abnormalities in the etiology of bruxism have never been proven, and they have progressively lost importance in favor of theories supporting the role of other factors of central origin, eg, psychosocial, neurobiological, and genetic factors.[1] In general, the recent literature suggests a shift from occlusal- to pyschological-based hypotheses and from peripheral to central regulation hypotheses.[1–4] Notwithstanding these shifts, the hypothesis that certain occlusal features may be related to bruxism onset has not been completely abandoned and is occasionally revisited.[5–7]

For a causal relationship between occlusion and bruxism to be present, a compelling prerequisite is that the two variables are associated, viz, the prevalence of the disorder should be significantly

higher in subjects presenting a certain risk factor.[8,9] Only then can hypothesis-driven studies test the existence of a causal link on a rational basis. Past studies on the issue showed that an association between bruxism and occlusal features of the natural dentition could be ruled out[10,11] and, in general, comprehensive reviews on the argument suggested that bruxism and the bite are likely unrelated.[12] Nonetheless, since the quality of the available literature on the argument is not optimal, studies adopting multivariate analyses of the various occlusal risk factors are needed to depict biological models. Therefore, the aim of this investigation was to estimate the contribution of various occlusal features of the natural dentition that may identify self-reported bruxers compared to nonbruxers.

Materials and Methods

Subjects and Study Design

A total of 142 subjects (52.8% females; mean age 25.1 ± 4.4 years) participated in this study. The study was performed according to a case-control design, with age- and sex-matched groups of self-reported bruxers (n = 67) and self-reported nonbruxers (n = 75), consecutively recruited among 20- to 30-year-old patients attending the Dental School, University of Padova, Italy, for conservative care. Subjects were included on the basis of the presence of all permanent teeth, except for third molars. The presence of bruxism was anamnestically investigated based on self-reported clenching and/or grinding of the teeth during the day and/or the night. The study was approved by the institution's Medical Ethics Committee, and all subjects signed a consent form prior to the start of the study.

The following occlusal features were recorded for each patient: retruded contact position (RCP) to intercuspal contact position (ICP) slide length (< 2 mm was considered normal), vertical overlap (< 0 mm was considered an anterior open bite; > 4 mm, a deep bite), horizontal overlap (> 4 mm was considered a large horizontal overlap), incisor dental midline discrepancy (< 2 mm was considered normal), and the presence of a unilateral posterior crossbite, mediotrusive interferences, and laterotrusive interferences. The clinical examination was made by the same trained operator.

Statistical Analysis

The prevalence of the assessed occlusal features in self-reported bruxers and in nonbruxers was compared by means of single regression analysis. Values of sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV), and accuracy to detect self-reported bruxism were assessed on the basis of 2 × 2 contingency tables (rows: occlusal features; columns: bruxism). PPV and NPV were calculated on the basis of the bruxism prevalence in this study's group, while accuracy was defined as the percentage of subjects who were correctly classified by the presence of each single occlusal feature.

Subsequently, a multiple logistic regression model was used to identify the significant associations between the assessed occlusal features (independent variables) and self-reported bruxism (dependent variable). Only those factors that were significant at $P < .10$ in the single regression analysis were included in the initial multiple regression model. Then, the variable with the weakest association with "recovery" was removed from the multiple regression model. This was repeated in a backward stepwise manner until all variables that were retained in the model showed a P value ≤ .05. The odds ratios (OR) for bruxism were assessed for each occlusal variable, while simultaneously controlling for the other variables in the model. OR values higher than 2 are commonly considered significant from a clinical viewpoint.

Nagelkerke's R-square (R^2) was obtained as an estimation of the total variance explained by the occlusal factors included in the model. If R^2 is > 0.75, the regression model is considered capable of predicting the presence of disease at a very good level. The model's ability to predict disease is considered good if R^2 is between 0.50 and 0.75, fair if R^2 is between 0.25 and 0.50, and poor for a R^2 of 0.25 or less.[13] The accuracy of the final logistic regression model to predict bruxer (sensitivity) or nonbruxer (specificity) status as well as PPV and NPV was determined from a 2 × 2 classification table.

All statistical procedures were elaborated with the Statistical Package for the Social Sciences (SPSS version 19.0; SPSS).

Results

A comparison of the prevalence of the assessed occlusal features in self-reported bruxers and nonbruxers was performed by means of single regression analysis to build a multiple regression. A significant association was revealed of self-reported bruxism with laterotrusive ($P = .030$) and mediotrusive interferences ($P = .037$). A slide ≥ 2 mm ($P = .083$) was also selected for inclusion in the multiple regression

Table 1 Comparison of the Prevalence of the Occlusal Features in Self-Reported Bruxers and Nonbruxers, and Significance in the Single Regression Analysis

Variable	Bruxers	Non-bruxers	Significance	OR (95% CI)
Laterotrusive interferences	19/67	10/75	.030	2.57 (1.09–6.03)
Mediotrusive intereferences	31/67	22/75	.037	2.07 (1.03–4.14)
Anterior open-bite	1/4	3/75	.367	0.36 (0.03–3.58)
Unilateral crossbite	18/67	17/75	.562	0.79 (0.37–1.71)
Large horizontal overlap	4/67	9/75	.214	0.46 (0.13–1.58)
Dental midline discrepancy	26/67	33/75	.531	1.23 (0.63–2.42)
Deep bite	20/67	17/75	.330	1.45 (0.68–3.08)
Slide RCP-ICP > 2 mm	23/67	16/75	.083	0.51 (0.24–1.09)

CI = confidence intervals.

Table 2 Accuracy, Specificity, Sensitivity, PPV, and NPV of Single Occlusal Features to Predict Self-Reported Bruxism

Variable	Accuracy	Specificity	Sensitivity	PPV	NPV
Laterotrusive interferences	59.1%	89.3%	28.3%	65.5%	57.5%
Mediotrusive intereferences	59.1%	70.6%	46.2%	58.4%	59.5%
Anterior open bite	51.4%	96%	1.5%	25%	52.1%
Unilateral crossbite	53.5%	77.3%	26.8%	51.4%	54.2%
Large horizontal overlap	49.2%	88%	5.9%	30.7%	51.1%
Dental midline discrepancy	47.8%	56%	38.8%	44.1%	50.6%
Deep bite	54.9%	77.3%	29.8%	54%	55.2%
Slide RCP-ICP > 2 mm	57.7%	78.6%	34.3%	58.9%	56.7%

Table 3 Significant Variables Remaining in the Final Logistic Regression Model and Model's Accuracy, Specificity, Sensitivity, PPV, NPV, and Total R^2 to Predict Self-Reported Bruxism

Variable in the final logistic regression model	Significance	OR (95% CI)	Model's accuracy	Model's specificity	Model's sensitivity	Model's PPV	Model's NPV	Total R^2
Laterotrusive interferences	.030	2.57 (1.09–6.03)	59.2%	69.3%	47.7%	58.1%	59.7%	4.6%

analysis, while the presence of self-reported bruxism was not significantly associated with anterior open-bite (P = .367), deep-bite (P = .330), large horizontal overlap (P = .214), dental midline discrepancy (P = .531), and unilateral posterior cross-bite (P = .562) (Table 1). Accuracy values to predict self-reported bruxism were unacceptable for all occlusal variables, if considered singularly (Table 2).

The three variables showing a P < .10 (slide ≥ 2 mm; mediotrusive interferences; laterotrusive interferences) were entered in the multiple regression model, and the variable remaining in the final model was laterotrusive interferences (Table 3). This means that the data on the mediotrusive interferences and slide did not add any information to the regression model including laterotrusive interferences (P =.030). Laterotrusive interferences showed an OR for self-reported bruxism of about 2.6.

The percentage of explained variance for bruxism by the final multiple regression model was 4.6% (Nagelkerke's R^2 = 0.046). This model including only one occlusal factor showed unacceptable PPV

(58.1%) and NPV (59.7%), thus showing a poor accuracy to predict the presence of self-reported bruxism (59.2%).

Discussion

Despite the number of etiology theories proposed over the years to explain bruxism, most authors agree on a multifactorial etiology, in which both peripheral and central factors can co-occur.[4] Among the peripheral factors, occlusal abnormalities were thought in the past to have a central role in bruxism etiology.[14] At present, the role of occlusal-anatomical features is believed to be much smaller,[15] but unfortunately, as often happens with evidence suggesting a diminished role of dental occlusion abnormalities in daily practice, such a conceptual shift from peripheral to central regulation is not easy to transfer to the clinical setting. This may be related to the fact that it is actually a true paradigm shift that requires clinicians to adopt a different way of thinking in the diagnosis and management of bruxism.[16] Also, the issue sometimes causes confusion because the etiopathogenetic role of natural occlusion is not discriminated from the role of iatrogenically altered occlusion, such as, for example, acute dental interferences due to high spots on dental restorations. Indeed, the bruxism-like effects of acute changes causing premature occlusal contacts or altering the vertical dimension of occlusion have been studied by means of several investigations in human models as well as in animal models.[17–19] They have led to the hypothesis of occlusal hypervigilance, according to which acute changes may play a worsening role on preexisting parafunctions in some subjects prone to react to external stimuli.[15] So, findings from such experimental studies, which were designed to test a specific cause-and-effect link between artificially modified occlusal features and their consequences, cannot be extrapolated to the natural occlusion, which is a target for parafunctional activities rather than an etiological factor.

Regarding the role of naturally existing occlusal disturbances as risk factors for bruxism, the best method to assess the existence of associations between bruxism and certain occlusal features is a multifactorial design, which better represents biological models than a univariate analysis and has been used to identify subpopulations of temporomandibular disorder patients.[20,21] In the present investigation, single-factor regression analysis was used to identify the occlusal variables to be included in the multiple regression analysis, and the accuracy of those single occlusal features to identify bruxism was poor, viz, less than 60%. Subsequently, the final multiple regression model included only the presence of laterotrusive interferences as a significant predictor, thus suggesting that knowing the presence of multiple occlusal features did not add any significant information. The estimated amount of variance in the presence of self-reported bruxism accounted for by the significant occlusal features was only about 4.6%. From a statistical viewpoint, such a value is commonly considered low, because it is far from depicting the full spectrum of predictors, and it is in line with values from studies showing the diminished role of occlusal features in the etiology of temporomandibular disorders.[20,21] From a clinical viewpoint, it should be noted that laterotrusive interferences reached an OR value higher than 2, which is commonly considered clinically relevant.[22] However, the multiple regression model showed that the occlusal characteristics have a poor value to discriminate bruxers from nonbruxers in a case-control design. The accuracy, sensitivity, specificity, and predictive values of the final model were unacceptable, ranging from 47.7% to 69.3%. Thus, these findings do not provide support to the association between occlusal features and bruxism.

The present investigation supported findings from an early study performed with a clinical diagnosis of bruxism,[11] thus suggesting that, in contrast with what happens in other fields of the bruxism literature,[23] the poor predictive value of occlusal features to detect bruxism is not influenced by the strategy adopted to diagnose bruxism. This observation is important if one considers the uncertainties characterizing the diagnostic approach to this phenomenon,[24] which are often a source of bias affecting the bruxism literature. For instance, self-reported bruxism studies have described associations with temporomandibular disorders that could not be replicated in studies adopting more controlled quantitative assessments. Notwithstanding that, it can be assumed that, even when studies employing more controlled strategies to diagnose bruxism are performed to increase the external validity of the present findings on bruxism and occlusion, it is unlikely that the lack of association between bruxism and any of the investigated occlusal features would be dismantled.

A major shortcoming of the bruxism literature is the poor specificity with respect to the different motor activities characterizing bruxism, viz, clenching and grinding. Hence, the generic umbrella term "bruxism" groups together phenomena featuring different types of muscle contractions. In addition, the bruxism subtypes "clenching" and "grinding" have different frequencies in relation to the circadian rhythm, as well as a different etiology and

clinical consequences. Clearly, there is a compelling need to design studies on the specific issue of, for example, sleep or awake bruxism. The present investigation makes no exception in presenting such a shortcoming, since it adopted a self-report approach to bruxism diagnosis and was not able to provide any information on the different motor activities and their relation with the circadian rhythm. Self-report/questionnaire-diagnosed bruxism, which still remains the most suitable approach to gather large-sample data for epidemiological reasons, is poorly specific and may introduce potential bias and confounders at the diagnostic level, among others, due to the preconceived idea by the patients and/or the interviewing clinicians that pain in the morning is a criterion for bruxism self-recognition. At present, reference criteria for a bruxism diagnosis exist only for polysomnographic recordings in sleep bruxers,[25] while debate is still open on the best suitable approach for an awake bruxism diagnosis and on the best way to discriminate between motor activities (ie, clenching or grinding). So, the various pros and cons of adopting a self-report approach to bruxism diagnosis in the present investigation were weighted in the present study design phase, and it seemed that no options were available at this time for performing a large-sample investigation that could validly discriminate between sleep and awake bruxism or between clenching and grinding. Based on the above, it is recommended that strategies for the assessment of ongoing bruxism activity and its neuromuscular features be better defined in order to obtain deeper insights into the etiology of bruxism and its clinical consequences.

Conclusions

This investigation has suggested that the contribution of occlusion to the differentiation between bruxers and nonbruxers is negligible. This finding supports theories that advocate a much diminished role for peripheral anatomical-structural factors in the pathogenesis of bruxism activities.

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