A literature review on the causal relationship between Occlusal Factors (OF) and Temporomandibular Disorders (TMD) III: experimental studies with artificial Occlusal Interferences (OI)

Una revisión de la literatura sobre la relación causal entre los factores oclusales (FO) y los desórdenes temporomandibulares (DTM) III: estudios experimentales con interferencias oclusales (IO) artificiales

RODOLFO ACOSTA-ORTÍZ[1], *NELSON ROURA-LUGO*[2]

[1] Dentist from Universidad del Valle, Advanced Clinical Training in Temporomandibular Disorders and Orofacial Pain, Master of Science in Epidemiology. University of Minnesota, USA, Adjunct Professor, Department of Prosthodontics, College of Dental Medicine, Nova Southeastern University.

[2] Dentist of the University of Puerto Rico, certified in Prosthodontics and Master of Science in Restorative Dentistry from the University of Michigan, USA. Associate Professor in the Assistant Deanship of Research and Advanced Studies Program in Prosthodontics, School of Dental Medicine, University of Puerto Rico.

ABSTRACT

Introduction: *this is the third article in this series that aims to present a literature review about the causal relationship between occlusal factors (OF) and temporomandibular disorders (TMD).* ***Methods:*** *this article presents and analyzes experimental studies in which occlusal interferences (OI) were used according to parameters recommended by Mohl.* ***Results and conclusions:*** *the analysis reported in this article did not support any OF as causal in TMD development. Although some OI were associated to the development of TMD signs and symptoms, this was, in the best of the cases, transitory. Additionally, methodological aspects such as the short follow-up time, and lack of control groups in most of the studies made the information found questionable.*

Keywords: *occlusion, temporomandibular disorders, etiology, occlusal factors, temporomandibular joint, experimental studies, occlusal interferences, experimental interferences, artificial interferences.*

RESUMEN

Introducción: *este es el tercero de una serie de artículos que tienen como propósito presentar revisión de la literatura sobre la relación causal entre los factores oclusales (FO) y los desórdenes temporomandibulares (DTM).* ***Métodos:*** *en este artículo se presentaron y analizaron los estudios experimentales con interferencias oclusales (IO) artificiales, siguiendo los parámetros recomendados por Mohl.* ***Resultados y conclusiones:*** *el análisis de los estudios revisados en este artículo no soportó a ninguna de las IO como factor causal en el desarrollo de DTM. Aunque sí hubo asociación de ciertas IO con el desarrollo de signos o síntomas de DTM, este fue en el mejor de los casos de manera transitoria. Adicionalmente, aspectos metodológicos como el tiempo de seguimiento corto, la falta de grupos controles de la mayoría de los estudios, hace que la información arrojada por estos sea cuestionable.*

Palabras clave: *oclusión, desórdenes temporomandibulares, etiología, factores oclusales, articulación temporomandibular, estudios experimentales, interferencias oclusales, interferencias experimentales, interferencias artificiales.*

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INTRODUCTION

This article is the third in a series aiming to review the different existing epidemiological studies and their validity, in order to weigh the current scientific evidence that supports the possible relationship between TMDs and OFs. In the first two articles, descriptive and analytical observational epidemiological studies were analyzed.[1,2] In this third article, experimental studies[3] in which artificial OIs were used to evaluate changes in oral physiology and the presence of characteristics associated with TMDs will be presented and analyzed.

METHODS

The literature review was performed using different sources of information:

1. The standard database of medical information, Medline, specifically using the Medline Ovid library (from 1966 to 2007). The abstracts of the articles in English and whose title suggested the study of the relationship between occlusal factors and TMDs were reviewed. To narrow the search, the different occlusal factors, occlusal interferences, experimental interferences and relevant terms under the occlusion/malocclusion heading were used as keywords and cross-referenced with relevant terms under the TMD heading (temporomandibular joint (TMJ), temporomandibular joint disorders, TMJ dysfunction).

2. The bibliography of the articles initially found in the Ovid database search.

3. The bibliography of different books on the TMD and occlusion domain.

4. The bibliography of several literature reviews on this topic from the Ovid database.

The validity of the different studies was evaluated following the parameters recommended by Mohl.[4] These parameters were described in detail in both previous articles[1,2] and are presented in Table 1.

Table 1. Parameters for assessing the validity of scientific studies

1. Definition of the gold standard.
2. Establishment of an acceptable system of diagnostic classification.
3. Use of clearly defined measures
4. Intra- and inter-examinators acceptable reliability
5. Use of suitable samples
6. Data collection by "blind" examiners.
7. Study replicability
8. Consideration of alternative hypotheses
9. Using matched groups
10. Random assignment of patients and subjects to the control group.

Source: by the authors

RESULTS

Among the experimental studies with artificial OIs there were a total of 32 reports, and these were presented in ascending sequence in relation to the year of publication (Table 2 in Appendix 2).[5-36]

Analysis of the factors guaranteeing the validity of the collected studies:

1. Definition of the gold standard. Most of the studies analyzed healthy subjects in whom the presence or absence of physiological changes or TMD signs or symptoms following the introduction of OIs was sought. The changes that were observed were usually presented in isolation and when present were not sufficient to diagnose the presence of a TMD. Perhaps this would explain why most of the studies did not try to make an analysis based on the presence or absence of signs or symptoms of the masticatory system that would lead to the diagnosis of TMD, but on the changes in the physiology of the masticatory system (electromyographic changes, speed of jaw movements, bite force, among others). In the studies by Le Bell (2002, 2006),[30, 35] in which patients with previous history of TMD were analyzed, the susceptibility of these subjects to present TMD signs or symptoms was always referenced. However, the susceptibility to develop a certain diagnostic category of TMD was not specified. In turn, Michelotti (2005, 2006)[33, 34] used the TMD research diagnostic criteria[37] to exclude subjects with TMD signs and symptom. This helped her determine with more clarity if the signs or symptoms developed by the subjects during the study were not present before the positioning of the artificial OIs.

2. Diagnostic classification system. Only one of the reviewed studies used a systematic diagnostic classification that would allow a differential diagnosis to be made between the different TMDs.[33]

3. Use of clearly defined measures. Several studies described the type and size of the interference, however, the type of material used to produce the interference varied between acrylics, resins and metals (usually gold alloys). Similarly, the system for measuring the changes produced by the OIs varied among the different studies. The clinical evaluation of TMD signs or symptoms, electromyographic recordings of some masticatory muscles, the presence of parafunctional movements, and occlusal force were the main measurement systems used in the different reports, among others. None of the studies reported the diagnostic validity of the methods used for the measurements. Some reports claimed that some type of calibration [6,8-10,12,15] of the instruments used had been performed, but none presented the reliability values.

4. Intra- and inter-examiner acceptable reliability. Most studies did not report the number of examiners, although it appeared that there was generally only one. Similarly, the different studies did not report any type of calibration or intra- or inter-examiner reliability.

5. Use of suitable samples. Samples varied widely in the number of subjects, although the limited number of participating subjects was a constant (average of 13 with a range of 3 to 30 subjects). It can be seen that the samples do not conduct to obtaining adequate representativeness to allow us to think in terms of the generalizability of the results obtained from these experimental reports. Some researchers studied only women and others only men, and they were often students of dentistry. Only in the reports by Le Belle (2002, 2006)[30, 35] subjects with a previous history of TMD were included.

6. Data collection by "blind" examiners. The vast majority of studies did not report taking this aspect into consideration and only a few reported some form of control for this variable of examiners bias. To try to control this aspect, at the time of the clinical evaluation, examiners were prevented from knowing whether or not the subject had an active OI or the group to which the participants belonged (experimental or control).[19, 30, 32-35]

7. Study replicability. Similar changes in the variables evaluated post-insertion of artificial OIs were reported in most of the studies. These changes were "replicated" among the different studies; however, the changes, magnitude and measurement systems were not necessarily similar among the different reports and therefore the validity of these "repetitions" in the changes evaluated cannot be reliably considered.

8. Consideration of alternative hypotheses. Finding certain changes in the participating subjects post-insertion of artificial OIs does not necessarily indicate that these are the causal factor of TMD. Especially because many of the changes were temporary and because the results in the controlled studies were inconclusive. Based on the results of the publications analyzed, one could alternatively ask: Does the elimination of these OIs in asymptomatic subjects make them less prone to the development of TMD? Does the elimination of these OIs in patients with TMD improve or eliminate the condition? Is there any proportional relationship between the size or dimension of the artificial OI and the severity of the changes found?

9. Using matched control groups. Most of the studies used a single-group experimental design with pre- and post-test measurements. Only a few studies used control groups [19, 30, 32-35] and only in one an attempt was made to pair the subjects.[19]

10. Random assignment of patients and subjects to the control group. A limited number of studies reported random assignment of participants to the experimental or control group.[19, 30, 35]

Table 2. Experimental studies with occlusal interferences (OI)

Main author and year	Sample (♀ / ♂)	Average age (range)	OI type	IO size	OI material	Study duration	Measurement type	Results
Jankelson, 1955	10 (NR) subjects	NR (4-72)	Premature contacts	NR	Acrylic cement	One hour	Observation of grinding movements	Grinding movements began immediately after OI was introduced and persisted until they were removed by dental grinding.
Anderson, 1958	4 (NR) subjects	NR	Premature contacts	0.5 mm	Gold crowns	NR	Occlusal force	Increased occlusal force
Graf, 1963	5 (2/3) subjects	NR	NR	NR	Acrylic resin and metal	Same day	Occlusal contacts and film	Tooth contacts occurred during mastication in the intercuspal position, but not in the retruded position
Schaerer, 1966	4 (NR) subjects	NR	Non-working	NR	Gold	2 days	Occlusal contacts during mastication and symptoms	Although initially more frequent dental contacts were reported, there was immediate adaptation and no changes in the chewing pattern. No symptoms were reported.
Schaerer, 1967	3 (NR) subjects	NR	Non-working	NR	Gold	2 days	EMG activity and contacts during mastication	During mastication there were no differences in immediate EMG response as the number of eccentric contacts increased, although the number of inhibitory pauses increased.
De Boever, 1969	4	NR	Non-working	NR	Gold fillings	8 days	EMG activity	No significant changes were reported during the time of the experiment
Kloprogge, 1976	8 (5/3) patients 3 (NR) subjects	NR	Premature contacts	NR	Metals	1 week	EMG activity	Abnormal EMG traces when putting premature contacts on restored teeth of patients
Randow, 1976	8 (1/7) subjects	NR (22-26)	Premature contacts	0.05 mm	Gold fillings within amalgams	14 days	Signs and symptoms and EMG activity	During the study, subjects reported dental sensitivity, TM (temporomandibular) muscle tension, changes in chewing patterns, looking for a more comfortable occlusion, sound in TMJ (TM joint).
Funakoshi, 1976	3 (NR) subjects	NR	Premature contact	0.3 mm	Metal	1 week	Balanced EMG activity	Introduction of OI caused imbalance in EMG activity, which disappeared by eliminating OI
Bakke, 1980	4 (1/3) subjects	NR (20-35)	Premature contact	0.5 mm to 2 mm	Metal sheets	Same day	EMG activity while clenching	Introduction of OI caused asymmetry in EMG, more marked in temporal muscles than masseters
Hannam, 1981	5 subjects	NR	Working	NR	Composites	Same day	EMG activity while clenching and jaw movement	Introduction of OI caused small impact, but there was no consistency among all subjects in EMG or movement
Riise, 1982; Shekholesman, 1983 & Riise, 1984	11 (♂) subjects	NR (24-32)	Premature contact	0.5 mm	Amalgam	1 week	Symptoms and EMG activity while resting, clenching and chewing	Introduction of OI caused temporary pain, tenderness and fatigue mostly in elevator muscles. There was also increased EMG activity at rest, reduced EMG activity when clenching, and change in muscle EMG coordination during mastication. The pattern of muscle symmetry was also altered. Symptoms and EMG activity returned to normal one week after OI removal
Magnuson, 1984	12 (♀) experimental subjects 12 (♀) control subjects	22 (16-33) 23 (18-34)	Non-working simulated OI	NR	Composites	2 weeks	Symptoms	Although both groups reported symptoms, the OI group reported a much higher percentage of subjects with symptoms than the simulated OI group. Symptoms disappeared in all subjects (except two) within one week of OI removal. It took 6 weeks for the two subjects (one from each group) to return to their previous state.
Rugh, 1984	10 (5/5)	NR (26-41)	Premature contact	Designated to produce deflection (lateral and forward) from 0.5 to 1 mm from CR	Gold crowns	10 to 21 days	Unilateral cumulative EMG activity of the masseter during sleep, and symptoms	5 out of 10 subjects showed significant reduction, 4 did not change, and one showed significant increase in EMG activity. 4 out of 10 developed mild symptoms (pain in TMJ, dental mobility, and muscle sensitivity)
Belser, 1985	12 (3/9)	31 (23-45)	Working (canine and molar guide) and non-working	NR	Gold fillings	Same day	EMG activity and mandibular displacement, in various parafunctional and masticatory activities	Placement of OI to cause canine guidance or non-working interference showed changes in EMG activity when clenching in eccentric. The effect of working interference was minor. EMG activity when chewing was not significantly altered by any of the OI
Shiau, 1989	14 (8/6)	NR (22-30)	Working	1.5 mm	Metal	4 weeks	Amplitude and duration of muscle contractions and speed of opening and closing movements	The speed of opening and closing movements, chewing patterns and muscle activity were mild and temporarily affected. These were recovered or maintained without causing any symptoms. Most subjects (84%) adapted to OI on the same day of placement. Only 2 subjects (14%) reported mild pain

Karlsson, 1992	12 (5/7)	22 (NR)	Non-working	NR	Composites	1 week	Symptoms, signs and masticatory movements	Almost no symptoms were reported during the experiment; there was a lot of variability in OI perception. Initially, mildly sensitive muscles were palpated and changes (some of them significant) in the magnitude of opening and speed of movement were measured; then, there was adaptation
Kobayashi, 1992	7 (NR)	NR (22-23)	Premature contact	0.1 mm	NR	Same day	Maximum opening and closing speed during mastication	The introduction of this OI could cause decrease and instability of the maximum speed of masticatory movements
Shiau, 1995	27 (12 - 15) people with and without bruxism	NR (18-31)	Working	1.5 mm	Composites	1 month	EMG activity (duration and masticatory movements, bruxism reports	Temporary lengthening of muscle contraction, reduction of jaw velocity in closing and narrowing of the occlusal phase of the masticatory cycle immediately after placing the OI
Christensen & Rassouli, 1995	12 (3/9) subjects	26 (NR) (d. e. ± 5)	Rigid premature contact; unilateral: 6 right and 6 left	0.24 (0.05 to 0.75) mm	Acrylic resins	Same day	EMG activity and intercuspal space, maximum clenching effort	OI placement caused significant distortion of amplitude but not duration of bilateral EMG activity when clenching (increased on the same side of the OI and decreased on the other); caused mandibular rotation creating interocclusal versus lateral space of 0.32 (0.05 to 0.55), usually greater (not significant) than interference
Rassouli &, Christensen 1995	12 (3/9) subjects	26 (NR) (d. e. ± 5)	Rigid premature contact; unilateral: 6 right and 6 left	0.24 (0.05 to 0.75) mm	Acrylic resins	Same day	EMG activity and mandibular rotation, maximum clenching effort	Frontal plane: all showed rotation that raised the contralateral condyle; deceleration of the descent of the ipsilateral condyle and acceleration of the sitting of the contralateral condyle. Frontal plane: there were inconsistencies, but it tended to rotate backwards ipsilaterally. They speculated about generation of negative hydrostatic pressures that could suck the disc into the temporal articular surface.
Baba, 1996	12 (♂) subjects	NR (25-28)	Working, non-working, high contact in canine	NR	Metal	Same day	EMG activity when clenching	After placing non-working OI, high canine contact and working OI, EMG activity increased, changed very little and decreased respectively
Baba, 2001	12 (♂) subjects	26,7 (25-28)	Working, non-working, high contact in canine	NR	Self-curing resins	Same day	Mandibular rotation on clenching	Mandibular displacement was reported after placing each OI that was used
Le Bell, 2002	26 (♂) subjects 21 (♀) patients with a history of TMD	24 (19-33) 31,5 (23-42)	Premature contact, non-working	NR (it opened 0,3 mm between incisors)	Composites	2 weeks	TMD signs and symptoms	Although reduction, increase or no change in signs and symptoms was observed in both groups, the group with a history of TMD did not adapt as well to OI as the group without a history of TMD, especially regarding signs and symptoms.
Okano, 2002	20 (15/5) subjects	26,5 (NR)	Different OI patterns	NR	Gold-palladium	Same day	EMG activity and mandibular rotation when clenching	More rotation with group function and semi-function occurred and significantly lower rotation with canine protection and balancing contact. Canine protection caused the lowest EMG activity of all occlusal patterns
Ferrario, 2003	30 (15/15) subjects	24,4 (20-33)	Premature contact	0,2 mm		Same day	Muscle asymmetry on clenching	Symmetrical muscle pattern of the sternocleidomastoid muscle became asymmetrical after placing the OI
Michelotti, 2005	11 (♀)	19,7 (16-23)	Premature contact	0,25 mm	Metallic gold sheets	6 weeks	EMG activity and TMD signs and symptoms	OI placement decreased the usual EMG activity of the masseters in the natural environment. None of the subjects developed TMD or TMD signs or symptoms
Michelotti, 2006	11 (♀)	19,7 (16-23)	Premature contact	0,25 mm	Metallic gold sheets	6 weeks	Muscle sensitivity measured with algometer pressure	OI placement did not influence the pain threshold to pressure in the masseter and temporal muscles
Le Bell, 2006	26 (♂) subjects 21 (♀) patients with a history of TMD	24 (19-33) 31,5 (23-42)	Premature contact, non-working	NR (it opened 0,3 mm between incisors)	Composites	2 weeks	TMD signs and symptoms	Subjects with no history of TMD showed fewer symptoms and better adaptation than subjects with a history of TMD. There are differences in vulnerability to OI between the two groups
Learreta, 2007	50 (NR)	NR (14-36)	Premature contact	0,4 mm	Thermoplastic material	Same day	EMG activity and electronic pressoreceptor system for occlusal contacts	Premature contacts cause changes in the EMG activity of masticatory muscles

NR = not reported, EMG = electromyography, TMJ = temporomandibular joint, TMD = temporomandibular disorders.

DISCUSSION

The possible relationship between OFs and TMDs is not easy to analyze and interpret. When analyzing Table 2, it is observed that in most of the studies, changes in the masticatory system were reported after the experimental insertion of artificial OIs. However, these changes occurred temporarily, and the severity of these changes was not clearly determined by factors such as intensity, duration or frequency of the changes assessed, but merely in terms of the presence or absence of these. Moreover, the changes observed after the introduction of the OI do not necessarily resemble the signs or symptoms that characterize TMD patient. Most likely, this may be the reason why none of the research studies used a clinical diagnosis of TMD and only evaluated the presence of signs and symptoms that do not really represent a disease state with the clinical features of TMD patients.

Besides, the fact that the analysis results of these studies show serious methodological problems in most of them (the size of the samples, the absence of intra- and inter-examiner reliability values, the non-use of a diagnostic classification system, etc.) make the validity of any conclusions questionable. The findings confirm the results of recent literature reviews in which they concluded, with the data from the studies analyzed, that there is no evidence to associate OIs as causal for TMDs.[38-40] However, Le Bell (2002, 2006)[30, 35] argued that one of the reasons why these investigations did not yield clear data to establish the causal relationship between OIs and TMDs was the fact that there might be a hidden selection bias in most of these investigations because the vast majority of them had used normal subjects. Le Bell (2002, 2006)[30, 35] also argues that using "normal" subjects perhaps means that these subjects have adequately adapted to the self-inferences that occur naturally in individuals. Therefore, excluding subjects who are possibly not well adapted to the presence of the OI (such as patients with a history of TMD) suggests that the results of the investigations could be false negatives.

The adaptability of the masticatory system to OIs has also been suggested in other studies carried out in young adults with nonworking contacts.41 In the spirit of overcoming this potential methodological problem of bias, Le Bell (2002, 2006)[30, 35] reported the first randomized clinical study with artificial OIs using subjects with and without a history of TMD. Their results showed that there were significant differences after insertion of artificial or placebo OIs in the signs or symptoms in subjects with no history of TMD. However, in the group with a history of TMD, subjects who were fitted with the artificial OIs reported a greater number of signs and symptoms when compared to subjects in whom the artificial placebo OIs were fitted. This author concluded that the differences found between patients with and without a history of TMD suggest that the role of the OI in TMD etiology may not have been properly investigated in previous studies with artificial OIs. However, although the results are encouraging and the reasons for the research and methodology were acceptable, none of the subjects with a history of TMD were reported to resort to the same previous clinical features that motivated them to seek treatment for muscular-type TMDs. Therefore, the results presented in this research are not sufficient to argue that artificial OIs are causal in the

development of TMDs, and most likely other factors must be involved in their development. Michelotti (2006)[34] provides a possible explanation by arguing that patients with TMD hold their teeth together more frequently than asymptomatic control subjects and therefore this would cause patients with TMD to report greater discomfort in the presence of occlusal interference. For his part, in a controlled two-week study, Magnusson (1984)[19] reported that placement of interferences on the non-working side bilaterally in young adult subjects resulted, although not in all, in the appearance of signs and symptoms in several of the subjects. Interestingly, signs and symptoms were also reported in the control group to which false OIs had been fitted. Two subjects, one in the experimental group and one in the control group, who had developed symptoms, took six weeks to become completely asymptomatic again. In this study it was concluded that local factors are important in TMD etiology but that this relationship is not simple or "obligatory" and perhaps other factors other than interferences, possibly psychological ones, were involved in TMD etiology. Recently, it has been shown that psychological factors play a significant role in the symptomatic response of subjects to the presence of artificial OIs.[42]

Similarly, as presented in the two articles (parts I and II) on descriptive[1] and analytical observational[2] studies, the criteria for identifying TMD signs and symptoms were not entirely similar among different studies, and the techniques used during clinical examination were also different. Neither did the studies report examiner training or levels of intra- and inter-examiner reliability.

The subjects in the control group were chosen from different populations (students, dental patients, non-randomized general population), therefore, being able to generalize or extrapolate the results to the general population is problematic. In addition, the TMD levels of severity (or their signs or symptoms in regard to intensity, frequency, or duration) were not reported in most of the studies. When it came to establishing the severity of TMD cases, it was done using the Helkimo index[43] and, as mentioned above, the diagnostic validity of this index is doubtful. Additionally, many of the parameters used to evaluate changes in the subjects (EMG recordings, bite force, jaw movement speed, among others) are not currently considered valid diagnostic tools to evaluate TMD.[39]

CONCLUSIONS

The analysis of the experimental studies with artificial OIs reported in this article did not support the idea of any of the OIs as a causal factor in TMD development. Although there was an association between OIs and the generation of certain changes in oral physiology or with the isolated presence of TMD signs and symptoms, this was temporary at best. In addition, none of the experimentally induced OIs could be associated with the development of a TMD (nor with any diagnostic category among TMDs), nor could they be used as predictors of the need for treatment. With the analysis of the information obtained from the experimental studies presented in this article, it cannot be concluded that artificially induced OIs are the

cause of TMD development. It seems obvious to consider that there is no single etiological factor in the development of TMD, and perhaps other factors such as psychological factors may play an important role in the relationship between the subjects' individual response to OIs and the development of masticatory system symptoms. Perhaps the improvement of research designs, which also include other variables in the analysis, such as the aforementioned psychological factors, will make it possible to obtain new data that could lead to a different interpretation, which could be used to establish how, realistically, OIs affect individuals. This would improve predictions of the need for, and effectiveness of specific treatments aimed at eliminating OI.

CONFLICT OF INTEREST

The authors state that they have no conflict of interest.

CORRESPONDING AUTHOR

Doctor Rodolfo Acosta Ortiz
Department of Prosthodontics
College of Dental Medicine
Nova Southeastern University
3200 South University Drive
Fort Lauderdale, Florida 33328
Office Phone: 954 262 73 43. Fax: 954 262 17 82
USA
Email: acostaor@nova.edu

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