*Opinion Paper**

Scientific Evidence of Occlusion and Craniomandibular Disorders

Pentti Kirveskari, Dr Odont

Pentti Alanen, Dr Odont, DSSc

Institute of Dentistry
University of Turku
SF-20520 Turku
Finland

Correspondence to Dr Kirveskari

Based on a paper presented by Dr Kirveskari at the First International Symposium of the American and European Academies of Craniomandibular Disorders in Chicago, February 10–11, 1992.

**Editor's note:* *This article has been termed an "opinion paper." It describes the authors' endeavors to explore the association of occlusal factors with CMD. The lack of a "classical controlled trial" is acknowledged, but sufficient information is presented to note that the "door is still ajar" (Storey AT. The door is still ajar [editorial]. J Craniomandib Disord Facial Oral Pain 1990;4:143–144). The acceptance for publication of such studies provides our readership with an exemplar for similar or contrary views, and such are invited for review and possible publication. An open mind is the linchpin of academic editorialism.*

The rejection of the old hypothesis concerning the role of occlusal factors in craniomandibular disorders has been suggested because of the lack of evidence supporting the hypothesis. However, it would be more helpful to seek tests that clearly show that the hypothesis is wrong. Most of the studies used to favor rejection of this hypothesis have incorporated designs precluding any causal conclusions on the role of occlusal factors. Time and resources have been wasted in repeated efforts to obtain what is only associative proof; studies designed to address the causal question are few. There are, as yet, no results clearly warranting the rejection of the hypothesis that occlusal factors are part of a causal complex of craniomandibular disorders.

J OROFACIAL PAIN 1993;7:235–240.

Studies on the etiologic role of occlusion in craniomandibular disorders (CMD) address the question of whether occlusion is a causal factor of CMD. A number of authors[1–4] have concluded that no scientific evidence for a causal relationship exists. Clinicians are reminded of this and of the ensuing responsibility to change their traditional views and practice accordingly.[5]

Some of the underlying theoretic problems in drawing causal conclusions from studies on occlusion and CMD have been discussed earlier,[6] and the conclusion has been drawn that occlusion cannot be excluded from the etiology of CMD on the basis of available evidence. The present paper purports to pinpoint some of the pitfalls in drawing causal conclusions from studies of occlusion and CMD and to demonstrate possible methods to put the causal hypothesis to a critical test.

Operational Definitions of Occlusion

Researchers make an educated guess when they decide which variables will be used and how these will be operationally defined for solving a scientific problem. The definitions reflect the researchers' concept of the nature of the problem. The better the educated guess, the more informative the results of the study are likely to be.

The variable in question, occlusion, has been given different operational definitions based on the relationship between joint- and tooth-guided contact positions. Interference is a collective name for occlusal anatomic or restored structures believed to prevent optimal occlusal function. Consider now the propositions that a centric slide of more than 1 mm is an interference, and that any slide in

centric position is an interference. These two propositions may not be metrically and biomechanically far apart, but in relation to sensory thresholds they are quite distant. Zero visible slide is much closer to the 0.01 to 0.03 mm sensory threshold than a 1-mm slide. If the sensory threshold is relevant to optimal occlusal function, studies using 1 mm as the operational limit may be insensitive to significant variation.

There are examples in the dental literature suggesting that metrically small interferences can be significant. For instance, in 1974, Dawson[7] drew attention to centric interferences in which no visible slide occurred. If forced by contacts on inclined planes, teeth can move in their sockets enough to be recognized as unfavorably loading by the sensory system. Riise and Ericsson[8] showed that there are about 8 to 10 actual contacts in light clenching and 16 to 18 contacts in hard clenching. This is essentially the same phenomenon referred to by Dawson. Recently, the number of light clenching contacts has been reported to show significant correlation with headache[9] and signs and symptoms of CMD.[10]

An examination of operational definitions of occlusal variables used leads to the conclusion that these studies, when they deny occlusion a role in the etiology of CMD, have not tested occlusal factors that are considered valid and significant by many clinicians. In fact, the testing of metrically small interferences is necessary, regardless of the clinical or intuitive stand taken as to their significance, because extremes of variation must be heavily represented in the analysis of a possible qualitative difference.

Operational Definitions of CMD

The rapid development of diagnostic methods, in particular the introduction of magnetic resonance imaging (MRI), has disclosed that our traditional clinical diagnosis is inaccurate, even without regard to interexaminer and intraexaminer errors. Parallel to the improvement of diagnostic accuracy, the validity of the concept of CMD and its synonyms has been questioned. Lumping together signs and symptoms that in clinical experience are associated with CMD involves the risk of including irrelevant elements in the variable that is supposed to represent the effect of a given causal complex.

Palpation tenderness of the masticatory muscles and clicking of the TMJ are commonly considered to be clinical signs of CMD. Greene and Marbach[1] maintain that these signs mainly represent normal variation. This interesting critique appears to follow from at least two assumptions. The first pertains to a prevalence that is considered too high to be characteristic of abnormality. While the predominant condition in a population is readily conceived as normal, such is not always the case in biology. We do not consider caries normal with respect to health, even if we do not call people with caries abnormal. Biologic normalcy cannot be defined on the basis of prevalence. The other assumption could be called the concept of fluctuation. Muscles occasionally become sore or painful and then return to the painless state without treatment. Therefore, palpation tenderness is considered to be within normal variation. This assumption rests on the presumed nonprogressive character of the condition. But, even if the condition were nonprogressive, it does not result in normalcy. There are a lot of conditions, such as headaches and common colds, that come and go even without treatment. The conditions are hardly commensurate with good health, and a great deal of money and resources are spent on fighting them.

An incorrect operational definition of CMD would seem to result in lack of association or in associations independent of the causal hypothesis studied. Observed irrelevant elements (such as normal variation) have the tendency to weaken or eliminate the association even if the operational definition also encompasses relevant elements.

Causal Inference

Much, if not most, of the confusion about the role of occlusion is deeply rooted in a lack of appreciation of the problems in causal inference. Overlooking the logic of causal inference is by no means limited to dental epidemiology, but is also widespread in medical literature. A recent review by Susser[11] gives useful advice that is applicable to an evaluation of the credibility of the conclusions drawn on the causal role of occlusion.

When specific causal agents are studied, four types of causality should be considered:

1. Sufficient and necessary
2. Sufficient but not necessary
3. Necessary but not sufficient
4. Neither necessary nor sufficient

Hypotheses of sufficient causes are usually easy to test. However, these types of causal problems are rarely encountered in etiologic epidemiology. A necessary but not sufficient causal factor is of more interest in studies of occlusion. Testing a hypothe-

sis of this type is theoretically straightforward. An observed effect without the factor is sufficient for rejecting the hypothesis. If the factor is very common, as is occlusal interference, testing may prove problematic in practice. The study design must include observation of the presumed effect in the absence as well as in the presence of the suspected causal factor. Effects observed in the absence of the factor will be at odds with the hypothesis.

Causal factors sought in epidemiology are usually neither sufficient nor necessary. They are parts of sufficient composite causes[12] or effective causal complexes. As a rule, chronic disorders can be caused by more than one constellation of factors, each forming an effective causal complex. Most of the component parts of the complexes are usually unknown.

The theoretic aspects of causality are by no means generally agreed on. According to Susser,[11] establishing causality requires the minimum of three attributes: association, time order, and direction.

Time order and direction in studies of occlusion and CMD can be dismissed with a brief discussion, because they are hardly touched on in the current literature. Time order simply means that the cause must precede the effect. Direction means that the relationship between cause and effect is asymmetric. Change in outcome must be a consequence of change in an antecedent factor. Cross-sectional studies are by design incapable of determining time order and direction.

Thus, the current problems in causal inference from studies of occlusion and CMD hinge on association. It is not unusual among researchers to suspect others of accepting association as proof of causality. The fact that weak or uncertain association does not exclude causal relationship appears less well known.

In spite of the increasingly popular subscription to the multifactorial etiologic theory of CMD, data are frequently interpreted in a way that is only valid for sufficient causes. Sufficient causes can legitimately be expected to show strong correlations with effects, but other types of causal factors cannot. It is a serious methodologic error to dismiss causal hypotheses solely on the basis of weak or uncertain statistical association. For the same reason, the so-called explanatory power of association, or *r* value, is not a valid measure of the qualitative question of association. The same methodologic problem has been discussed also in connection with caries risk assessment.[13,14]

De Kanter[15] concludes from his cross-sectional epidemiologic study that occlusion and CMD are unassociated. He dismisses the associations found in some studies because these have failed to use multifactorial statistical analyses. However, multifactorial analyses tell us whether a given association can be totally or in part explained by variation in other factors. The analysis says nothing of the causal relationships between the factors included in the analysis. For example, human stature and length of hair are inversely associated; entering gender and various cultural factors into the analysis discloses variation explaining the association. It is for the analyzer to decide which of the associations are worth a causal analysis. The fact that variation in somatization and general joint disease explained the association between occlusion and CMD in De Kanter's study warrants no causal conclusions.

In causal analyses, association sought from cross-sectional data is a qualitative question. A study sample evenly drawn from the whole range of variation is not only unnecessary, but may be misleading. Only extremes of variation are needed. Solberg et al[16] found a significant association between asymmetric centric slide and CMD when applying this two-point design. Discussions concerning the causal role of occlusion miss the point when the doubtfulness, strength, and explanatory power of associations are the main factors determining the stand taken.

Artificial Interferences

It has been reported that signs and symptoms indicative of CMD arise after the introduction of an artificial interference.[17,18] Suggestive as the results are, they do not test the hypothesis that naturally occurring interferences could be causative of CMD. In addition to the need for adequate controls, at least two major problems are encountered. The experimental subjects should not be, or even have been, exposed to the suspected causal factor prior to testing. If they are, or have been, the subjects are selected for resistance to or tolerance of the causal factor.

The other major problem is the sudden introduction of interference. Naturally occurring interferences need not behave in the same way. Even if an artificial interference in a fully controlled study is shown to elicit CMD, the result can only be suggestive with respect to natural interferences.

Elimination of Interferences

Since patients who are totally interference-free according to stringent clinical criteria are extremely rare, eliminating interferences in a study group

is, in practice, the only way to form a group free from the suspected causal factor. Observing the emergence of CMD in such a group and in another group exposed to the factor for a sufficiently long period is, in principle, a rigorous test of causality. If significantly less CMD emerges in the unexposed group than in the exposed group, the causal hypothesis has survived.

A study[19] conducted at our Institute attempted to follow the above principles. The observed increase in both symptoms and signs of CMD was significantly greater in the exposed than in the unexposed group, in which interferences had been eliminated at the beginning of the study. However, the study fell short of a classical controlled trial in at least three respects:

1. Interferences have a tendency to reappear.[20] Because occlusion was adjusted only at the beginning of the 2-year study period, some reappearance of interferences took place before the final examinations. This shortcoming is likely to decrease the difference between the groups, not increase it. In other words, the risk of a false-negative result increases.
2. The study sample was not unbiased with respect to the problem. As the emergence of CMD was to be observed, those already having it had to be excluded. The remaining subjects were thus selected for good resistance to or tolerance of interferences. Again, this bias is likely to result in a false-negative outcome.
3. Craniomandibular disorders ought to have been totally absent in both groups at the beginning. The fact that some mild signs were present leaves open the possibility that factors involved in the genesis of CMD cannot be distinguished from factors affecting the course of it. This methodologic shortcoming makes causal conclusions uncertain.

We have conducted an extensive intervention study in children, eliminating interferences annually in half of them. Limited time per child and long intervals between adjustments, growth, and dental development were the presumed reasons that interferences could be kept eliminated in only a very small number of children. Nevertheless, that was sufficient to disclose a consistent association between the number of occlusal interferences and signs of CMD.[21,22]

Our method and result have been rejected by Seligman and Pullinger[3] because a statistical difference could only be found when "the groups were manipulated and data rearranged." The logic of this statement is not understood. It is a venerable scientific strategy to simplify the conditions of observation by design.[11] Our design was chosen to elicit the valid relations between cause and effect.

Seligman and Pullinger[3] also state the following: "The implication that interferences are universal and at the same time pathologic is most disturbing because this suggests universal prophylactic occlusal adjustments even in the absence of symptoms." As long as the causal role of interferences has not been disproven, it is advisable to call them probable risk factors. Only if interferences were a sufficient cause of CMD could they be called pathologic, because their presence would invariably lead to CMD. A mole on the skin is part of an effective causal complex of melanoma, but it is not a sufficient causal factor. The risk per single mole is very small. Nevertheless, people often have them prophylactically removed. A skin mole is an example of a very common risk factor. The decision to undergo prophylactic treatment is quite obviously determined by several factors other than just the knowledge of the causal relationship.

Prophylactic occlusal adjustment is considered contraindicated or unwise by many authors and clinicians. However, we are aware of no study showing harmful effects of a properly conducted occlusal adjustment. There seems to be no reason to ban it before it is critically tested. Prophylactic occlusal adjustment may or may not prove useful in future tests. We can see no logical way in which our studies,[21,22] showing that an association and a causal relationship between interferences and CMD cannot be excluded, could lead to the conclusion drawn by Seligman and Pullinger.[3]

Critique of studies on occlusion and CMD tends to focus attention on methodologic detail, with little or no bearing on the basic causal problem. Categorization of continuous variation, less-than-perfect sensitivity and specificity of diagnosis, poor representation of the population at large in the study sample, and weak explanatory power of associations are examples of "methodologic flaws" that are by no means critical in solving the qualitative problem of association, provided that the study design is otherwise adequate.

It is, in principle, possible to test the hypothesis that occlusal interferences are a nonsufficient causal factor using data from studies such as ours.[22] If interferences are a necessary causal factor, no new cases of CMD are to be expected in cases where interferences are and remain absent. The weight of evidence against the hypothesis is clearly dependent on diagnostic accuracy. The data presented in Table 1 are taken from repeated cross-sectional examinations without an individual

Table 1 Occurrence of Signs of CMD in Groups of Children Free From Interferences at Annual Examination

Group 1			Group 2		
Age (y)	No. without signs	No. with signs	Age (y)	No. without signs	No. with signs
5	2	0	10	1	0
6	3	0	11	0	0
7	4	1	12	1	0
8	3	0	13	2	1
9	2	0	14	6	0
10	3	1	15	6	0

follow-up analysis. The data yield too few interference-free subjects for a really effective statistical test, but one can hardly suggest outright rejection of the hypothesis, either.

Conclusion

It may be concluded, based on the literature and our own studies of occlusion and CMD, that the causal role of occlusion has not yet been adequately tested, and that the available data relevant to the causal question do not permit the exclusion of occlusal factors from effective causal complexes of CMD.

References

1. Greene CD, Marbach JJ. Epidemiologic studies of mandibular dysfunction: A critical review. J Prosthet Dent 1982;48:184–190.
2. Mohl ND, Zarb GA, Carlsson GE, Rugh JD (eds). A Textbook of Occlusion. Chicago: Quintessence, 1988.
3. Seligman DA, Pullinger AG. The role of functional occlusal relationships in temporomandibular disorders: A review. J Craniomandib Disord Facial Oral Pain 1991; 5:265–279.
4. Clark GT. Etiologic theory and the prevention of temporomandibular disorders. Adv Dent Res 1991;5:60–66.
5. Mohl ND, Ohrbach R. The dilemma of scientific knowledge versus clinical management of temporomandibular disorders. J Prosthet Dent 1992;67:113–120.
6. Alanen PJ, Kirveskari PK. Disorders in TMJ research. J Craniomandib Disord Facial Oral Pain 1990;4:223–227.
7. Dawson PE. Evaluation, Diagnosis and Treatment of Occlusal Problems. St Louis: Mosby, 1974.
8. Riise C, Ericsson SG. A clinical study of the distribution of occlusal tooth contacts in the intercuspal position at light and hard pressures in adults. J Oral Rehabil 1983;10: 473–480.
9. Wänman A, Agerberg G. Recurrent headaches and craniomandibular disorders in adolescents. J Craniomandib Disord Facial Oral Pain 1987;1:229–236.
10. Wänman A, Agerberg G. Etiology of craniomandibular disorders: Evaluation of some occlusal and psychosocial factors in 19-year-olds. J Craniomandib Disord Facial Oral Pain 1991;5:35–44.
11. Susser M. What is a cause and how do we know one? A grammar for pragmatic epidemiology. Am J Epidemiol 1991;132:635–648.
12. Rothman KJ. Modern Epidemiology. Boston: Little Brown, 1986.
13. Stamm JW, Stewart PW, Bohannan HM, Disney JA, Graves RC, Abernathy JR. Risk assessment for oral diseases. Adv Dent Res 1991;5:4–17.
14. Disney JA, Graves RC, Stamm JW, Bohannan HM, Abernathy JR, Zack DD. The University of North Carolina caries risk assessment study: Further developments in caries risk prediction. Community Dent Oral Epidemiol 1992;20:64–75.
15. De Kanter RJAM. Prevalence and Etiology of Craniomandibular Dysfunction. An Epidemiological Study of the Dutch Adult Population [Thesis]. University of Nijmegen. Winssen, RP Scientifics, 1990.
16. Solberg WK, Woo MW, Houston JB. Prevalence of mandibular dysfunction in young adults. J Am Dent Assoc 1979;98:25–34.
17. Randow K, Carlsson K, Edlund J, Öberg T. The effect of an occlusal interference on the masticatory system. Odontol Revy 1976;27:245–256.
18. Takeda Y, Ishihara H, Kobayashi Y. Influences of occlusal interference on nocturnal sleep and masseter muscle activity. J Dent Res 1989;68:936.
19. Kirveskari P, Le Bell Y, Salonen M, Forssell H, Grans L. Effect of elimination of occlusal interferences on signs and symptoms of craniomandibular disorder in young adults. J Oral Rehabil 1989;16:21–26.
20. Hellsing G. Occlusal adjustment and occlusal stability. J Prosthet Dent 1988;59:696–702.
21. Kirveskari P, Alanen P, Jämsä T. Association between craniomandibular disorders and occlusal interferences. J Prosthet Dent 1989;62:66–69.
22. Kirveskari P, Alanen P, Jämsä T. Association between craniomandibular disorders and occlusal interferences in children. J Prosthet Dent 1992;67:692–696.

Resumen

Correlación de la Evidencia Científica de la Oclusión y los Desórdenes Craneomandibulares

La falta de evidencia concerniente al papel de los factores oclusales en la etiología de los desórdenes craneomandibulares ha sido nuevamente considerada puesto que no se ha llegado a acuerdos contundentes. Sin embargo, sería mas provechoso buscar pruebas que demuestren claramene que la hipótesis está errada. La mayoría de los estudios que solían rechazar esta hipótesis, han incorporado diseños que impiden las conclusiones causa-efecto sobre el papel de los factores oclusales. Se ha desperdiciado el tiempo y los recursos en esfuerzos repetidos para obtener lo que sólo es una prueba asociativa; los estudios diseñados para atender la etiología, son pocos. No existen todavía, resultados que justifiquen claramente el rechazo de la hipótesis de que los factores oclusales son parte de un complejo etiológico de desórdenes craneomandibulares.

Zusammenfassung

Wissenschaftliche Erkenntnisse über Okklusion und Myoarthropathien des Kausystems

Verschiedene Autoren verwerfen die Hypothese, dass okklusale Faktoren in kausalern Zusammenhang mit Myoarthropathien des Kausystems (MAP) stehen mangels schlüssiger Beweise. Es wäre nützlicher, Tests zu fordern, die ein Verwerfen der Hypothese tatsächlich untermauern, zumal die meisten dieser Studien einen kausalen Zusammenhang zum vornherein ausschliessen. Viel Aufwand wurde betrieben, um ein Vorliegen von Zusammenhägen zu finden - selten nur wurde die Frage nach der Kausalität gestellt. Nach Sichtung der Literatur und aus eigenen Arbeiten ziehen die Autoren den Schluss, dass die verfügbaren Resultate den Ausschluss der Okklusion als einen kausalen Faktor in der Entstehung der MAP nicht zulassen.