The titles of the following essays were chosen by our guest contibutors to preface their responses to both the Interface papers and the Study Group Reports.

Critical Commentary on the Occlusal Interface

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The focus articles presented in the interface "Occlusion" explain the present condition and the future of occlusion. However, as pointed out by Sessle,[1] parts of these articles still tend to discuss the occlusal interface in the traditional manner, as a structural entity. The occlusal-structural model of TMD causation is a typical example. Unlike the internal organs of the body, malocclusion can readily be visualized and quantitatively described and is easily perceived by both patients and doctors; however, it is flawed by being directly tied up with signs and symptoms.

This is also true of reports that expressed negative views on occlusal factors of TMD. The work of Pullinger et al,[2] as cited by Alstergren,[3] is a clear example. In their research, they have merely analyzed with statistical instruments the classical variations from the habitual intercuspal position acquired by TMD patients, such as overlap of anterior teeth, the difference between centric occlusion (CO) and centric relation (CR), and crossbite. The basis for the analysis was the intercuspal position. But whether this intercuspal position was a healthy one or not is unknown.

Research[4,5] performed in my department has repeatedly shown that in a healthy subject, both mandibular condyles are in the center of their fossae and bilaterally symmetrical. In other words, the condyles of a healthy person are in a stable position. In cases where the condyles had been significantly displaced, the TMD symptoms went away completely when the displacement was treated with a splint or a treatment denture.

The intimate relationship between TMD symptoms and mandibular displacement has been clearly and accurately documented in recent research.[6–9] The 1999 annual review[10] of the American Academy of Restorative Dentistry featured research in which a positive relationship was observed between TMD symptoms and mandibular displacement. As for the causation of the symptoms or the disease-causing mechanism, it was suggested that venous plexus or nerve fibers (auricular, autonomic, and sensory fibers) dispersed around TMJ were being stimulated.[11,12]

Therefore, in order to specify the occlusal factors of TMD, analysis should be performed with the healthy stable mandibular position as the basis. As stated by Pullinger et al,[2] no matter how extensively the occlusal factors of acquired mandibular problems are analyzed, no clear and accurate answers can be expected. Regrettably, none of the 11 focus articles mentioned this problem. This basic problem should have been addressed, especially in the articles by Bryant,[13] Alstergren,[3] Nilner,[14] and Klineberg and Stohler,[15] who used Beyron's concepts[16] or CO-CR measurement as the basis for evaluating occlusion. Thus far, only animal experiments have shown that occlusal factors can cause mandibular displacement[17]; more detailed analysis is indispensable.

On the other hand, Dao[18] quoted evidence that suggested that perceived changes in occlusion may be the consequence of pain rather than its cause, or that the mechanistic occlusal view of the pathophysiology of TMD is being eclipsed. Occlusal change due to muscle pain was also emphasized in Svensson et al's review of muscle pain.[19] The grounds for these claims were experiments in which jaw muscle pain was deliberately induced and subsequent jaw movement traced with a gothic arch tracer. However, experiments with greater accuracy are needed to support such claims.

As for occlusal factors of TMD, Dao[18] pointed out that occlusal interferences are highly prevalent in both TMD patients and control subjects. This is because classic and large interferences can be controlled by jaw muscles using information obtained from receptors in the oral cavity, whereas the minute interferences that cannot be controlled have a greater effect. In fact, a small interference of less than 0.5 mm can well be adapted to, yet affect the masticatory muscles the most.[20]

These phenomena can be sufficiently explained by clinical research performed in my department.[21] A small experimental occlusal interference with a thickness of 100 μm (0.1 mm) that would have no traumatic effect on the periodontal membrane was placed on the occlusal surface of a mandibular first molar in a carefully selected normal subject. Prior to the placement of the experimental interference, the week of the placement of the experimental interference, and a week after removal of the experimental

interference, biological phenomena, clinical signs, and psychoendocrine secretions were recorded during sleep throughout the night employing a wireless telemeter system. The results confirmed that the interference itself stimulated the brain and increased bruxism from 15 minutes per night (ie, a healthy level) to 40 minutes per night (ie, an abnormal level) with strong biting force. Furthermore, the bruxism persisted. In addition, tooth movement, mandibular displacement, the tensing of the related muscles, and the appearance of symptoms similar to TMD were observed. At the same time, changes in the function of the autonomic nervous system, including the appearance and augmentation of central sleep apnea and hyperactivity of sympathetic nerve, sleep disorders, and emotional stress were all observed. These phenomena were caused by stimulation of the nerve fibers dispersed around the TMJ, as stated earlier. They could be thought of as being caused by the effect of oral sensation on the brain. That is to say, minute, continuous stimulation of the pulp and periodontal membrane that leads to corresponding pain or discomfort will inhibit respiration.[22,23]

This research showed that a small occlusal interference in intercuspal position cannot be adapted to completely and will augment bruxism to an abnormal level. Such an interference will be a promoting factor for sustaining bruxism and will eventually lead to TMD and muscle pain. In fact, the hypothesis that bruxism during sleep leads to TMD and muscle pain is well supported by other research.[24,25]

Koyano et al,[26] citing an article by Rugh et al,[19] stated that experimental occlusal interference during sleep decreases muscular activity. However, in the research by Rugh et al,[19] no record of sleep was made, and the fact that sleep changes its form greatly as one gets older makes it difficult to discuss sleep bruxism in a group of subjects with such a wide range of ages (26 to 41 years old). Furthermore, since a 0.5- to 1.0-mm-thick occlusal interference that covered the entire occlusal surface was placed in CR, it is very possible that the interference acted as a pivoting splint that suppressed the muscular activity. Koyano et al[26] also cited Nishigawa et al's research[28] on the mean amplitude and duration of bruxism events. However, Nishigawa et al used intraoral equipment similar to a splint for measurement, which would definitely suppress the occluding force. Thus, the whole experiment was performed inadvertently in an environment wherein the occluding force was suppressed.[29] In these examples, even in the experiment in which a polygraph was used to record bruxism, most experiments were performed using a wired system that would have a large effect on the procedures being recorded. The process of selecting a recording day, which can have a significant effect on the results in sleep laboratory studies, was not consistent either. Furthermore, there is some inconsistency among researchers in setting the standard for bruxism, and at the present it is not known why healthy people also exhibit bruxism.

On the other hand, in TMD patients, a positive correlation between the identification of bruxism and sleep apnea, plus a positive correlation between the onset of bruxism and a decrease in REM sleep duration was recognized.[30,31]

Therefore, occlusion should be able to cope with bruxism during sleep, with its longer duration and stronger biting force than the occlusal contacts made during normal functions. Additionally, an extensive study of the mechanisms of bruxism during sleep will be indispensable.

Walther[32] noted that a shortened dental arch is functionally useful. However, loss of molars may affect spatial memory and neurologic changes of the brain and central nervous system.[33] When all of these observations are considered, it becomes clear that detailed neurophysiological studies of the body as a whole are essential. Only this will ensure a correct understanding of occlusion.

References

1. Sessle BJ. Biological adaptation and normative values. Int J Prosthodont 2003;16(suppl):72–73.
2. Pullinger AG, Seligman DA, Gornbein JA. A multiple logistic regression analysis of the risk and relative odds of temporomandibular disorders as a function of common occlusal features. J Dent Res 1993;72:968–979.
3. Alstergren P. Determinants of a healthy aging dentition: Freedom in the retrusive range of occlusal contacts and multidimensional freedom for functional tooth contact. Int J Prosthodont 2003;16(suppl):79–80.
4. Ishikawa T, Kobayashi Y. Clinical studies on diagnosis of condylar position using lateral radiographs of temporomandibular joint [in Japanese]. Shigaku 1984;72:493–540.
5. Yanagida S, Kobayashi Y. The effect of treatment denture with cuspless teeth on restoring mandibular displacement [in Japanese]. Shigaku 1992;80:840–864.
6. Ekberg EC, Sabet ME, Petersson A, Nilner M. Occlusal appliance therapy in a short-term perspective in patients with temporomandibular disorders correlated to condyle position. Int J Prosthodont 1998;11:263–268.
7. Bonilla-Aragon H, Tallents RH, Katzberg RW, Kyrkanides S, Moss ME. Condyle position as a predictor of temporomandibular joint internal derangement. J Prosthet Dent 1999;82:205–208.
8. Pullinger AG, Seligman DA, John MT, Harkins S. Multifactorial comparison of disk displacement with and without reduction to normals according to temporomandibular joint hard tissue anatomic relationships. J Prosthet Dent 2002;87:298–310.
9. Pullinger AG, Seligman DA, John MT, Harkins S. Multifactorial modeling of temporomandibular anatomic and orthopedic relationships in normal versus undifferentiated disk displacement joints. J Prosthet Dent 2002;87:289–297.
10. Allen EP, Bayne SC, Becker IM, Donovan TE, Hume WR, Kois JC. Annual review of selected dental literature: Report of the Committee on Scientific Investigation of the American Academy of Restorative Dentistry. J Prosthet Dent 2000;84:59–92.
11. Tahmasebi-Sarvestani A, Tedman RA, Goss A. Neural structures within the sheep temporomandibular joint. J Orofac Pain 1996;10:217–231.
12. Kobayashi Y, Matsumoto T, Ishigami K, Hirai T. The relationship between occlusion and the body function. J Jpn Prosthodont Soc 1996;40:1–23.
13. Bryant SR. The rationale for management of morphologic variations and nonphysiologic occlusion in the young dentition. Int J Prosthodont 2003;16(suppl):75–77.
14. Nilner M. Musculoskeletal disorders and the occlusal interface: II. Int J Prosthodont 2003;16(suppl):85–87.
15. Klineberg I, Stohler C. Occlusion study group report and discussion. Int J Prosthodont 2003;16(suppl):89–90.
16. Beyron H. Occlusion: Point of significant in planning restorative procedures. J Prosthet Dent 1973;30:641–652.
17. Aoki S. Effect of occlusal support, dietary and nutrition on osteoporotic changes of old rat's condyle. J Jpn Prosthodont Soc 1998;42:374–382.
18. Dao T. Musculoskeletal disorders and the occlusal interface: I. Int J Prosthodont 2003;16(suppl):83–84.
19. Svensson P, Graven-Nielsen T. Craniofacial muscle pain: Review of mechanisms and clinical manifestations. J Orofac Pain 2001;15:117–145.
20. Riise C, Sheikholeslam A. Influence of experimental interfering occlusal contacts on the activity of the anterior temporal and masseter muscles during mastication. J Oral Rehabil 1984;11:325–333.
21. Kobayashi Y. Influences of occlusal interferences on human body. J Oral Rehabil 1988;15:196.
22. Lobbezoo-Scholte AM, Lobbezoo F, Steenks MH, De Leeuw JRJ, Bosman F. Diagnostic subgroups of craniomandibular disorders. Part _: Symptom profiles. J Orofac Pain 1995;9:37–43.

23. Arima T, Svensson P, Arendt-Nielsen L. Experimental grinding in healthy subjects: A model for postexercise jaw muscle soreness? J Orofac Pain 1999;13:104–114.
24. Moruzzi G, Magoun HW. Brain stem reticular formation and activation of EEG. Electroenceph Clin Neurophysiol 1949;1:455–473.
25. Yamamura K, Shimada K, Yamada Y. Effect of periodontal stimulation on respiratory activity. Jpn J Oral Biol 1994;36:526–535.
26. Koyano K, Tsukiyama Y, Ichiki R. Local factors associated with parafunction and prosthodontics. Int J Prosthodont 2003;16(suppl):82–83.
27. Rugh JD, Barghi N, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxism. J Prosthet Dent 1984;51:548–553.
28. Nishigawa K, Bando E, Nakano M. Quantitative study of bite force during sleep associated bruxism. J Oral Rehabil 2001;28:485–491.
29. Ishihara H, Kobayashi Y. Studies of bruxism during sleep, Effect of Michigan type full coverage bite plane [in Japanese]. Shigaku 1987;75:907–952.
30. Kobayashi Y, Shiga H, Yokoyama M. Sleep apnea and bruxism in TMD patients during nocturnal sleep. J Dent Res 2002;81(special issue A):297.
31. Kobayashi Y, Shiga H, Yokoyama M. REM Sleep and bruxism in TMD patients. J Oral Rehabil 2004;31:885.
32. Walther W. Determinants of a healthy aging dentition: Maximum number of bilateral centric stops and optimum vertical dimension of occlusion. Int J Prosthodont 2003;16(suppl):77–79.
33. Chiba A. Effects of molar tooth loss on central nervous system with a behavioral and histological study in mice. J Jpn Prosthodont Soc 1999;43:299–11.

The Interface of Occlusion as a Reflection of Conflicts Within Prosthodontics

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It was interesting to read the papers of the occlusal interface section with the eyes of both a clinical teacher and researcher. The conclusions that these 2 groups would draw from the same papers would most likely be diametric opposites. The clinical teacher would be left with a lot of unanswered clinical questions and probably would think, despite all the skepticism of the academicians, that most of the therapies he uses in daily practice are efficacious in the vast majority of cases, provided that certain basic biological rules that are a common denominator for most occlusal philosophies are taken into consideration. Thus, he would consider the interface of little value, as it does not solve his daily problems. The clinical academic, on the other hand, would conclude that we do not know very much about the importance of the occlusal interface for the long-lasting health of the stomatognathic system or the patient's quality of life. This dualism well represents the gap that exists between the university and the clinical settings, and I fully agree with the statement "there is a gap between the clinician who performs prosthodontic treatment as a matter of routine using (mostly) reliable conventional techniques and the scientist who is preoccupied with problems that do not occur in the dental office."[1] If we want to progress in prosthodontics, it is of utmost importance that this gap disappears. Clinicians must be involved in the formulation of clinical questions, the dental office must be considered by academics as a resource for clinical knowledge, and the university must not be regarded any longer by dental practitioners as the "ivory tower." Thus, a better way of communicating between those who perform dental care and those who perform dental research is necessary.

Occlusal Interface Pivotal?

In the study group report and discussion, Klineberg and Stohler pointed out that "the occlusal interface is pivotal to successful prosthodontic treatment."[2] Although as a prosthodontist I would like to agree with them fully, I think that, in light of the knowledge we have today, the sentence must be rephrased as follows: "Occlusion is an important aspect of prosthodontics in its broadest context. However, its significance on treatment outcome and the longevity of the patient's health and quality of life is still far from clear." Indeed, except for technical failures, the long-term success of at least fixed prostheses depends in the first instance upon the prevention of infection and thus upon the maintenance of oral health.[3] Occlusal features also seem to be of little or no importance in regard to the long-term success of implant-supported reconstructions,[4] and this is the case for complete dentures too, as pointed out in an extensive literature review conducted almost 10 years ago whose conclusions are still valid: "Occlusion is an important aspect of the technical process of denture fabrication, as it is closely related to the physical aspect of load distribution, denture retention, and stability. Despite its biomechanical importance, occlusion, as well as the technical quality of the denture, plays only a minor role in determining success or failure of a denture treatment. A number of psychosocial factors are likely to be more important than the prosthodontic factors for a positive outcome."[5]