



Oral Parafunction, Pain, and the Dental Occlusion

Bruxism is nowadays generally considered an oral parafunction, but the etiology of bruxism and the relationship of bruxism to the dental occlusion and to craniofacial pain, including temporomandibular disorders (TMD), have been topics of considerable debate and controversy in dentistry. Until the past one to two decades, particular emphasis was placed on occlusal factors as being important in the etiology of bruxism, but there has been a gradual shift to concepts, supported by increasing scientific evidence, that central neural mechanisms (eg, “psychological” factors) play the major role in mechanisms underlying the development and maintenance of bruxism. Nonetheless, the possibility that occlusal factors may be of etiological significance in bruxism and that oral parafunctions such as bruxism may be at least risk factors for TMD continues to generate considerable research interest. Indeed, this issue of the *Journal of Orofacial Pain* carries several articles bearing on these matters.

In the case of occlusal factors, the articles by Manfredini et al,¹ Michelotti et al,² and Glaros and Williams³ collectively first draw attention to conflicting findings in the literature on their possible role in bruxism and craniofacial pain. Manfredini et al¹ evaluated the potential contribution of a variety of clinically assessed occlusal features to see if they could identify bruxers based on their self reports of daytime or nighttime clenching and/or grinding of the teeth. There was no clear evidence that occlusal features could differentiate these bruxers from non-bruxers, thus adding to the growing evidence of the apparently minor contribution of occlusal factors in the etiology and pathogenesis of bruxism.

To address the possible role of the occlusion, particularly in the development of craniofacial pain and its association with oral parafunction, Michelotti et al² introduced occlusal interferences and tested if their effects differed between subjects reporting a low or high frequency of waketime oral parafunction. They found evidence that a molar occlusal interference was associated with craniofacial pain in the subjects with a high frequency of parafunction, suggesting that the presence of an existing oral parafunction may increase the risk that an occlusal interference will produce craniofacial pain.

Nonetheless, these articles point to several “caveats” that bear not only on these two studies but

also on much of the earlier literature on the possible association between the dental occlusion, bruxism, and craniofacial pain. Associations may vary (and indeed confounds introduced) depending on whether the bruxism is daytime or nighttime and how the oral parafunction is defined and diagnosed. For example, the current “gold standard” for diagnosing sleep bruxism is a diagnosis based on polysomnographic recordings, but this may not be a practical or economic approach for everyday clinical use in the dental clinic. As Manfredini et al¹ note, epidemiological and most clinical studies have had to rely on self reports and questionnaire data to diagnose bruxism, but these introduce potential bias and confounds and have low specificity. The presence of wear facets on a patient’s teeth as a diagnostic criterion for bruxism in clinical studies also is problematic (eg, the wear may not necessarily reflect recent bruxing episodes by the patient, or bruxism per se.)^{4,5} Other variables that may influence the outcome of studies of bruxism vis-à-vis an altered occlusion is the type and location of the occlusal interference, and the operational definition of a parafunction in terms of grinding, clenching, and tooth contact; the latter is addressed in the article by Glaros and Williams³ in this issue.

The diagnosis of TMD and other craniofacial pain conditions is also not without its problems, given, for example, the various subgroups of TMD and headache conditions that exist and the variety of approaches advocated to delineate them.^{6,7} In the case of TMD, there is now considerable evidence, as with bruxism, that central neural factors may be more important than local (eg, occlusal) factors in the etiology and pathogenesis of most TMD conditions. The adaptability (or not) of the subject to stress and other psychosocial factors appears to be important in whether the subject develops such a condition. Indeed, two papers in this issue provide evidence for the role of stress and adaptive coping in the expression of craniofacial pain.^{8,9}

The findings of these various articles collectively point to the role of the brain in determining the ability of subjects to adapt and cope to alterations in the oral cavity or in their psychosocial circumstances. Of note in this regard is the emerging evidence of the neuroplasticity of brain regions controlling orofacial sensory and motor function, revealing how these brain circuits can undergo neuroplastic changes in

humans and laboratory animals following not only alterations in other brain regions but also following certain alterations to the dental occlusion or to sensory inputs to the brain as a result of nerve trauma or pain.^{6,10} Further research into brain neuroplasticity and its relationship to oral sensory and motor function and dysfunction and adaptation and coping promises to provide important new insights into oral parafunctions, craniofacial pain, and the role of the dental occlusion.



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