Invited Commentary:
Why do Orofacial Movement Disorders Cause me to Brux?

The prosthodontic management of patients with tardive dyskinesia (TD) has taught me that there is much that we health professionals still need to learn about this condition. When I first sought relevant information by "Googling" the condition, I was referred to many websites that described the problem and talked about its history, prognosis, and causation. The available information changed dramatically in the ensuing years and I was alarmed to note the number of legal websites geared specifically towards those suffering from TD, as well as read about the numerous resulting medical malpractice awards.

TD is an iatrogenic involuntary movement disorder caused by the use of neuroleptic drugs. Readers are urged to view the 2005 publication by Blanchet et al[1] for an excellent introduction to the topic. This issue's paper by Katz et al[2] emphasizes the large variation in the reported prevalence of TD, one that can be arguably attributed to the dual difficulties of diagnosis and terminology.

In the early 1970s, a few papers described a small number of edentulous dyskinesia patients who were reportedly "cured" with dental treatment. Repeated references to these publications resulted in a diagnostic category called "Edentulous Dyskinesia," although it is quite clear the causation of attendant signs and symptoms are extremely difficult to prove in the absence of long-term prospective studies. The causation challenge is not unlike the one that confronted smoking research, where a large body of cross-sectional studies with methodologic rigor and compelling numbers finally led to an explicit relationship. It is therefore difficult at this stage to accept the premise that edentulism causes dyskinesia. It is far more likely that the presence of TD accentuates the symptoms due to the increase in the mandible's tracking length that results from a collapsed vertical dimension. Consequently, the recovery of a compromised vertical dimension of occlusion cannot be presumed to cure the condition although it may minimize tracking length, leading to a reduction in the overt nature of the involuntary movement.

Two recent publications[3,4] proposed a classification of movement disorders, but Blanchet et al's[1] earlier taxonomic inclusion of bruxism remains preferred. Bruxism is arguably the one movement disorder that is most readily diagnosed by the dentist, given its unpredictable potential for damaging both natural as well as restored dental tissues. It may be regarded as part of a broad spectrum of parafunctional habits that includes sleep bruxism, daytime bruxism, and clenching. A near knee-jerk reaction to its diagnosis and attempted management is to prescribe a nightguard, or so-called stabilization appliance. It is therefore interesting to note that numerous nightguard designs have failed to provide any robust evidence as to which works best. Perhaps we would all be better off accepting the fact that it is the interdental presence of a disposable piece of—consider inserting your predilected material here—that prevents damage to teeth. The technique has certainly not proven to be predictably effective as a parafunctional behavior-altering therapy.

Bruxers will typically present with severe dental wear and healthy bone with no periodontal destruction. However, it seems illogical that bruxers do not seem to have periodontal disease. If the latter is the case, typical wear patterns from parafunctional habits may tend to go unnoticed, particularly when teeth are absent. Besides the obvious medically mediated variables, how often do we ask these patients if they had a history of bruxing or, more importantly, look for the presence of other movement disorders? This concern is likely to become even more critical as we see more patients' teeth restored with implant-retained restorations. While there have been conflicting reports of bruxing damage to implants, this is an area that certainly needs rigorous study, as opposed to current reliance on informed anecdote. It should be remembered that even patients with severe dental wear will frequently deny a history of bruxism. In those with total tooth loss and numerous destroyed prostheses, we have to rely on patient recall data that is largely unreliable.

Given its prevalence, it is understandable that we dentists devote significantly more time to bruxism than to other movement disorders. However, an aging patient population with an attendant increase in pharmacologic interventions and the accompanying risks of adverse reaction and interaction issues will inevitably affect the occurance of other movement disorders. Can we therefore expect the neuromuscularly compromised patient, especially one with a movement disorder, to demonstrate similar tissue damage to what is so frequently encountered in our bruxism patients?

Edentulism is by definition a pathologic condition. The dentate patient presents with clinically determined reference positions: centric relation, physiologic rest position, occlusal vertical dimension, and the maximum intercuspal position; the totally edentulous patient offers only a centric relation and a physiologic rest position. If we look at the guidelines for the restoration of the totally edentulous patient, whether with implants or removable prostheses, we have anatomical guidelines to follow: the buccinator bulge, the lateral border of the tongue, the retromolar pads, and esthetics. We also have physiologic guidelines: breathing, speech, mastication, deglutition, as well as esthetic considerations. Again, in the neuromuscularly compromised patient, virtually all of the latter guidelines become unreliable.

I recently fabricated a new maxillary complete denture for a 69-year-old woman who had almost recovered fully from a stroke. Her mandibular restoration consisted of an implant-supported metal-ceramic restoration and her 5-year-old opposing denture demonstrated severe wear and chipping of the artificial teeth. She presented with no interocclusal gap and at rest, her mentalis and masseter muscles were visibly hypertonic. When I tried to record her physiologic rest position without her denture in place, her mandibular teeth came into contact with the maxillary residual ridge and her masticatory muscles were still taut. But this was a moment in time. Was she always a clencher or was this the result of the stroke? Would my newly fabricated denture end up showing the wear of the previous one? Probably yes, but what would occur if I had restored her with an implant-retained restoration in the maxilla?

The real issue here is not the correct diagnosis, which is ultimately a medically mandated one, but the dental management of the patient with these conditions. I am currently responsible for a patient who suffers from severe tremors of her entire body. She can hardly walk or write. The orofacial tremors make taking a radiograph nearly impossible, let alone preparing a tooth or establishing an intermaxillary record. Her physician has diagnosed her with a Central Nervous System disorder. She is not cognitively impaired and is reluctant to accept a "benign neglect" strategy as a treatment option. What do I do?

Given the current state of knowledge, I would not treat patients with one of the various movement disorders any differently. However, many clinical questions remain unanswered: Should one treat them similar to a patient with a history of bruxism? How does one handle the nonreproducible centric relation or the clearly reduced occlusal vertical dimension with little or no interocclusal gap? How much interocclusal gap is necessary for these patients? Should one treat the fully dentate patient and edentulous patient in a similar manner? Does the placement of implants alter one's approach to the occlusal vertical dimension, interocclusal gap, and centric relation issues of this patient population?

It is essential that our specialist discipline concedes that there is so much we still need to know regarding the management of this special patient group with movement disorders. Waiting for long-term randomized controlled trials to guide our clinical decisions is simply unrealistic given this patient group's immediate needs. What the discipline needs are scrupulously documented case series on the early diagnosis and treatment of these patients, as well as their prosthodontic management. This will ensure that a sense of collective clinical wisdom evolves in our ongoing commitment to professional and humanitarian care.

Gary Goldstein, DDS
Professor, Department of Prosthodontics
New York University College of Dentistry, New York
Email: gary.goldstein@nyu.edu

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