Does Bruxism Cause Pain?

A “simple question” without a simple answer

And as mind (our mind or mind like ours) reappears to us in our study of Nature—Nature being everywhere formed after a method which we can well understand, and all the parts, to the most remote, allied or explicable—therefore our own organization is a perpetual key, and a well-ordered mind brings to the study of every new fact or class of facts a certain divination of that which it shall find.

This reduction to a few laws, to one law, is not a choice of the individual, it is the tyrannical instinct of the mind.

— Ralph Waldo Emerson[1]

The diseases of dentistry are largely understood by modern science. As a result, dentistry is spoiled by its science.

We know what factors are involved with periodontal disease; we understand decay. When patients are presented with treatment plans, rarely is "prognosis" considered or discussed because it's clear that if the patients play the limited role that is required of them, and if our treatment is well delivered, the treatment plan will be successful. If there are difficulties during the treatment process, both the dentist and the patient tend to attempt to determine where the "fault" lies. As a result, we expect to understand mechanisms of disease, we expect that all the disease processes are understood, and we feel responsible to cure our patients of those diseases and create ideal dental health.

Patients who receive quality dental care from quality dentists die with their teeth. Patients who receive quality medical care from quality physicians … still die.

Therefore, when considering odontogenic matters, dentistry is burdened by the expectation of perfection and success. This burden may in fact be largely responsible for dental burnout and consummate dissatisfaction within the profession.

The move from odontogenic dentistry

Over the years, dentistry has moved from attempting to separate the teeth and the supporting structures from the rest of the body to understanding that there are intricate relationships between these dental structures and other components of the cranial–cervicomandibular system.

It's not surprising that both function and dysfunction of these complicated systems are not quite as "finite" as the anatomy and physiology of the teeth and the periodontium. Unfortunately, dentistry tended to take the model of relative simplicity in terms of function and dysfunction involved in their odontogenic world into their understanding of these nonodontogenic structures. Dr. J.N. Campbell made this observation in 1957:

Time passed and it slowly dawned upon us that the problem of facial pain was bigger than we had thought, and that it could not be completely explained in terms of mechanics. Dentists have every reason to believe in their mechanical arts. They have developed a system of oral engineering of which they can be justly proud. However, their concentration on the

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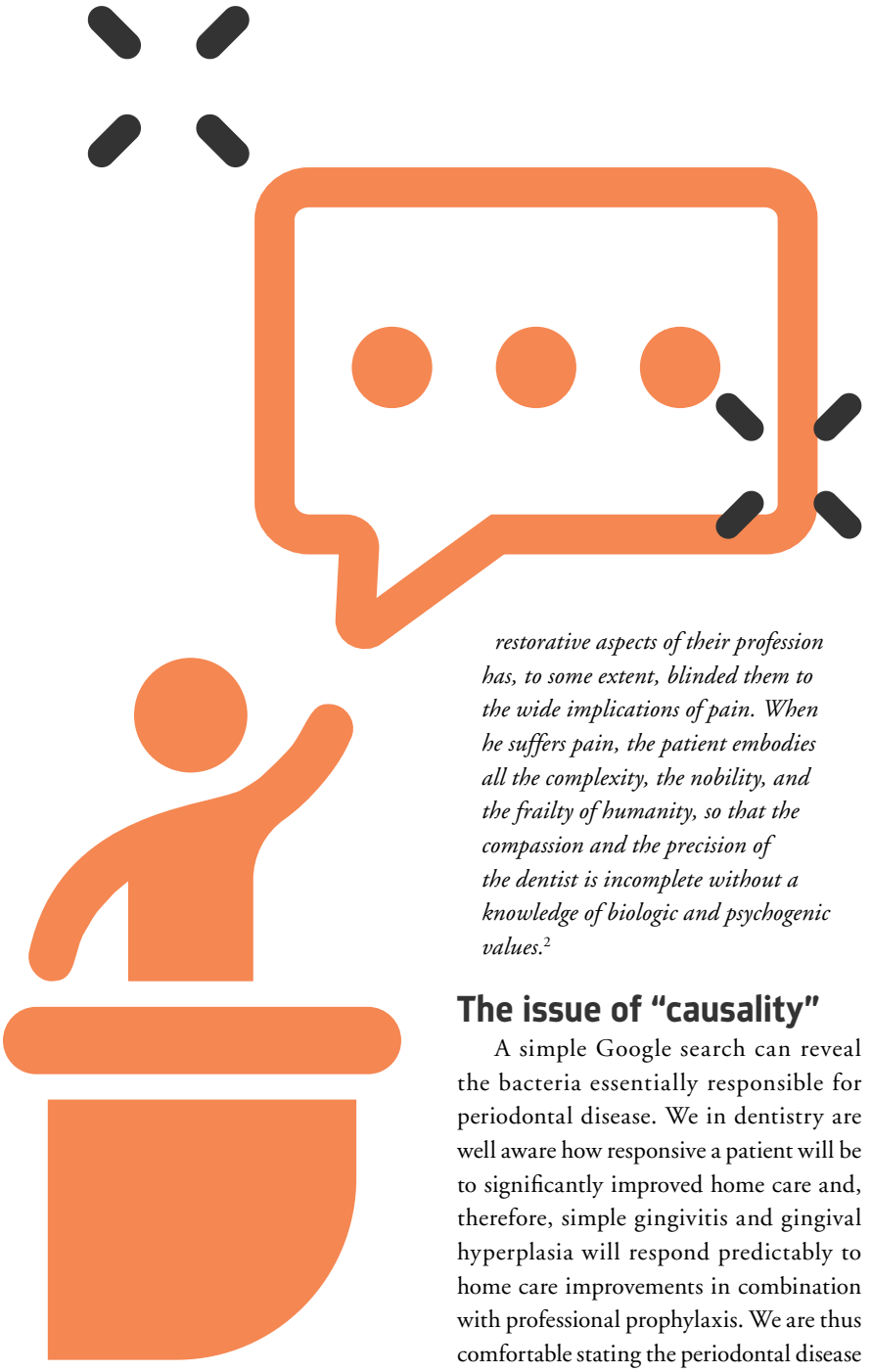

restorative aspects of their profession has, to some extent, blinded them to the wide implications of pain. When he suffers pain, the patient embodies all the complexity, the nobility, and the frailty of humanity, so that the compassion and the precision of the dentist is incomplete without a knowledge of biologic and psychogenic values.[2]

The issue of "causality"

A simple Google search can reveal the bacteria essentially responsible for periodontal disease. We in dentistry are well aware how responsive a patient will be to significantly improved home care and, therefore, simple gingivitis and gingival hyperplasia will respond predictably to home care improvements in combination with professional prophylaxis. We are thus comfortable stating the periodontal disease is caused by poor home care. In the same vein, we are also comfortable and accurate in stating the "cause" of dental decay, and we are well aware that increasing the patient's adaptive capacity to decay with fluoride, along with good home care, will essentially resolve the decay process.

Al Fonder coined the term *dental distress system* (DDS) when in 1961 he related dental structure in terms of posture of the mandible and the upper cervical spine to altered disorders of nerve root compression and more. This structural concept suggests that when the malocclusions and altered jaw positions are improved, symptoms resolve, thus proving that the altered structure was the "cause" of the symptoms:

> *DDS patients complain of headache, dizziness, hearing loss, depression, worrying, nervousness, forgetfulness, suicidal tendencies, insomnia, sinusitis, fatigue, indigestion, constipation, ulcers, dermatitis, allergies, frequent urination, kidney and bladder complications, cold hands and feet, body pains and numbness, and a host of sexual failures and gynecological problems. Elimination of the DDS reverses these chronic problems, the body chemistry and blood picture normalize. Even backward students when treated rapidly advance in classroom productivity, often becoming honor students.*[3]

It was further suggested that DDS caused Parkinson's disease and epilepsy.[4]

An internationally known physician named A.B. Leads who treated Roosevelt, Eisenhower and Stalin, who worked with the late dentist Willie B. May, said, "When this treatment is fully researched and understood, it will be capable of revising every diagnosis, treatment procedure and prognosis in the medical world."[5]

Is it any wonder, then, that without evidence-based principles at work, empirical evidence suggesting causality continued to dominate the nonodontogenic world of dentistry?

Occlusion, bruxism and pain

The role of occlusion in pain can be traced from Fonder to Costen, Guichet, Gelb, Dawson and Jankelson, and to the rise of the "TMJ camps," suggesting a direct relationship between occlusion and joint position and pain and dysfunction. Each of

these pioneers suggested a direct relationship amid occlusion, jaw position and pain. While there was disagreement on what was "normal," there was the general agreement of a direct relationship between their definition of "abnormal" and pain or dysfunction.

Costen was an otolaryngologist who reported in 1934 that loss of vertical dimension led to ear pain and dysfunction.[6] Guichet, a dentist, promoted the need to alter the occlusion to result in a condylar position that was both distal and superior, and thus "repeatable."[7] Gelb used the anatomy of the disk, condyle and glenoid fossae to insist on a condylar position that was down and forward for improved health.[8] Dawson suggested a use of mandibular repositioning based on relaxed muscle and manipulation techniques ("romancing a mandible") that resulted in a somewhat different position than Gelb's 4/7 positioning.[9,10] Jenkelson founded the neuromuscular camp that recommended the tensing of muscle to result in "relaxed" musculature, leading to potentially different condylar positions for ultimate health.[10] Interestingly, all of these "camps" reported some success with patients with the same various signs and symptoms of the upper quarter.

Of course, the assumption was being made that when the jaw position was changed and resulted in a symptomatic improvement, the symptoms were therefore caused by the "improper" jaw position. And as in the past, causation was assumed and statements of causality again were being made between structure and headache, jaw pain and more.

Evidence-based scientific principles were ignored and anecdotal reporting with assumptions of mechanism reigned. Confirmation bias using all of the techniques resulted in each jaw position, no matter how different, being claimed to be responsible

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for overwhelming successful symptomatic resolution, including everything from headaches to internal derangements of the temporomandibular joint.

The knee-jerk reaction that opposes this thought process suggests that occlusion is not at all related to orofacial pain patterns. This concept is problematic for the general dental population that has personally witnessed many occasions of both odontogenic and nonodontogenic pain relieved with simple occlusal adjustments.

The nature of causality thus becomes critically important and needs to be examined carefully.

This discussion is extremely timely and long overdue, and a current TMJ thread on Dentaltown's message board is titled "The End of Occlusion in TMD. A Major Crisis Is on the Horizon." [*Editor's note: A link to this message board is embedded in the digital version of this article online at* **dentaltown.com/magazine**.] The concern is that there are those who have applied for a specialty status of pain management who question any role of occlusion in pain.

Where does the truth lie? Not surprisingly, perhaps somewhere in between.

TMD and occlusion

The use of the term "TMD" in any discussion is problematic[11] because it's an umbrella "diagnosis" that is not specific and includes a host of very different conditions. Nevertheless, the predominance of the evidence suggests no direct relationship between any specific pain pattern and any specific scheme of tooth-to-tooth contact.

Yet, as dentists, we are well aware that changes in occlusion have resulted in either initiation of pain or resolution of pain before and after restorative therapy.

Despite the fact that dentistry tends to "stipulate" occlusion, the fact remains that studies have suggested that teeth are actually in contact less than 20 minutes a day in the absence of parafunction.[12]

In dental terminology, occlusion (a noun) refers to the relationship of the dental scheme when the elevators contract and bring the teeth into contact in maximum intercuspation. Occluding (a verb) refers to the action of dental contact. It would seem obvious, then, that occluding and the consequential forces that result are at issue when it comes to possible damage to the components of the cranial–cervicomandibular complex.

But is it that simple? Of course not.

Is bruxism caused by occlusion?

It has been assumed and generally thought by most all of dentistry that bruxism causes internal derangements and orofacial pain as well as headaches. In 1961, Ramfjord and Ash wrote, without evidence, that bruxism was caused by "interferences" and thus malocclusion.[13] Dawson's teachings were clearly geared toward putting the condyle in centric relation and eliminating all interferences to that position. When this type of equilibration had been completed and the patient's symptoms had improved, it was then assumed that the bruxism had caused the pain and that the equilibration had stopped the bruxism.[14]

The suggestion that the equilibration has stopped the bruxism has never been proven. In fact, Goodman and Greene demonstrated that "mock equilibrations" were as effective in symptom reduction as fully performed equilibrations.[15] Michelotti demonstrated that when she added interferences (gold foil) into the occlusal scheme of healthy females, not only did it not produce symptoms, but also the masseter EMG levels decreased.[16]

Does bruxism cause pain?

Raphael has demonstrated that people with pain do not necessarily brux more

than people without pain. She goes further to suggest that there are those with pain who don't brux, and those who brux and don't have pain. In fact, there are those who brux significantly in terms of frequency and duration and do not have any pain or dysfunction.[17]

While this is irrefutable, Raphael concludes not only that bruxism does not cause pain, but that therefore treatment towards parafunctional control was "misguided."[18] After conducting an excellent study that used EMGs in polysomnograms to determine the existence of bruxism in patients with and without pain (as opposed to many past studies, which used unreliable questionnaires) Rafael notes the lack of the direct relationship and concludes that because most patients with pain did not exhibit sleep bruxism (SB), therefore "the common belief that SB is a sufficient explanation for myofascial TMD should be abandoned." She further notes that pain does not, therefore, justify SB treatment.[18]

A discussion about causation and potential contributing mechanisms becomes essential and takes us back to both Emerson's and Campbell's quotes at the beginning of this paper. Emerson suggested that man's tendency was to attempt to simplify cause and effect; Campbell's quote made it clear that understanding pain is not simple. Pain is a combination of not only the degree of negative stimulus to the organism but also a complex physiology that is not totally understood and cannot be simply measured. Pain is not directly related to the painful event but is a concept with many compounding, not readily measured or always understood, factors.

Every dentist has adjusted an occlusion and noted a change in a patient's dental—and, often, nondental—symptoms. Success. Every dentist has then repeated that adjustment for another patient, only to be surprised by a total lack of response. Failure!

It seems clear that there is no direct relationship between bruxism and pain, and yet certainly incorrect to suggest that altering the forces during the bruxism event in terms of magnitude and direction by changing the occlusal scheme can't result in altered symptoms.

It therefore follows that it would be incorrect to suggest that when occlusion is altered and the symptoms resolve that the occlusal scheme and bruxism were the "cause" of the pain pattern. It would be more appropriate and accurate to suggest that the occlusal scheme and bruxism was certainly a contributing factor, and that the alteration of that scheme with that particular patient's adaptive capacity considered, helped resolve their pain or dysfunction.

It is misguided, therefore, to suggest that the lack of a direct relationship between bruxism and pain would suggest that treatment aimed at parafunctional control and thus altering the forces of bruxism is "misguided." ■

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