CRITICAL COMMENTARY 2

THE ETIOLOGY OF TEMPOROMANDIBULAR DISORDERS: IMPLICATIONS FOR TREATMENT

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Dr Greene suggests in his Focus Article[1] that, for the most part, the cause or etiology of a specific patient's seemingly spontaneous onset of a temporomandibular disorder (TMD) will not be discovered, even though the clinician asks many questions and makes many measurements of the patient. At first this suggestion seems counterintuitive to medical diagnostic process, but Greene points out that while the pursuit of an etiology is laudatory, it may be fruitless until we have better diagnostic tools. He states that recent molecular and cellular research information has already substantially improved our understanding of the mechanisms of joint disease, muscle pain, and chronic pain. He suggests that these basic science breakthroughs will improve treatment approaches, even though they do not solve the etiologic dilemma.

The second and equally important assertion that Greene makes is that to treat TMD patients successfully with the methods available, it is not necessary to know the original etiology if we have a reasonable understanding of the mechanism and use evidence-based treatment approaches. Given that our current treatments are mostly palliative physical medicine and short-term behavioral interventions, this second assertion seems logical. Greene is basically saying that since we cannot find a single clear-cut cause for a patient's problem, we should stop worrying about it and just treat the symptoms, since to a large degree these symptoms resolve with time. He points out that symptomatically focused treatment with reversible and minimally invasive therapies has been shown to be reasonably successful in many treatment-outcome studies on TMD. He delicately points out that sometimes the well-meaning clinician will perform many forms of "definitive" and usually irreversible treatment, which may or may not be needed, in pursuit of "treating the underlying etiology." Of course, irreversible therapies are not inherently bad, but they are usually more expensive and almost always have more morbidity associated with them. Also, they would be somewhat illogical given that conservative and reversible treatments are often sufficient.

One of the earliest and most persistent theories for causation of TMD is the concept that the problem arises from abnormal occlusal structure or "malalignment of the jaws." Other theories also exist, but the occlusal-structural model of TMD causation has received the most attention from researchers over the last 4 decades. This attention is largely because malocclusions can readily be visualized and quantitatively described. The presence of a malocclusion is frequently invoked as a justification for applying various forms of definitive occlusal therapy, such as orthodontics, orthognathic surgery, and occlusal adjustment and/or reconstruction, to TMD patients. The evidence for the widespread application of these therapies as a necessary and required treatment that can prevent the recurrence of the patient's TMD problem is absent.

Dr Greene does not limit his comments to the occlusal-structural theory and appropriately points out that the other 2 popular etiologic theories (biopsychosocial and multifactorial) have little evidence backing them as well. He suggests that in the absence of such evidence, whichever etiologic theory best fits a clinician's current belief model is most likely to be advocated. Moreover, the clinician will most likely select the matched treatment approach. For example, if a clinician favored the biopsychosocial model, he or she would advocate psychologic interviews, psychometric testing/assessment, stress management, biofeedback, self-hypnosis, and/or psychoactive medications. Such

treatment methods are not in the same "irreversible" category as occlusal-structural interventions, but nonetheless they require substantial behavioral and lifestyle changes on the patient's part and thus must be both evidence-based and competitively compared (eg, cost-benefit analysis) to other models of therapy.

The multifactorial model of disease is the other major player on the TMD etiology scene. This theory suggests that any single element—or more likely a combination of putative etiologies (eg, bruxism, joint hypermobility, arthritic disease susceptibility, daily oral habits, external traumatic events, developmental anomalies, disc malpositioning, acquired malocclusions, and of course stress and associated muscle tension)—could produce a TMD. Since this etiologic model has tremendous flexibility when it comes to determining the appropriate etiologic agents, the associated therapeutic methods selected are also highly variable. If the clinician favors bruxism as a causative agent, then this argues for occlusal splint therapy; if the problem is perceived to be an arthritic disorder, then anti-inflammatory methodologies will be used; if it is presumed secondary to a disc malposition, jaw realignment or discal surgery will be selected. Of course, the above pairings between etiology and treatment are not inherently logical, but Greene suggests that the underlying evidence that such etiologies can be established, and that these anti-etiology treatments can resolve the problem, is mostly non-existent or weak at best.

The reader may be asking, "In the absence of an etiologic-driven approach to TMD treatment, what is appropriate?" The most common model of therapy when etiology is not known is what has been described as the escalation-of-treatment approach. This approach rank-orders the available treatments that have demonstrated efficacy, weighs the efficacy with the cost-benefit data, and applies each treatment, one at a time, beginning with the least invasive and moving to the most invasive. While this approach would be suitable for a single disease entity, TMD are multiple diseases; therefore, the escalation-of-treatment model needs to be modified. This modification involves matching the symptom to the treatment. For example, limiting jaw opening and modifying the diet to avoid temporomandibular joint clicking would be best matched with patients who have this symptom as a chief complaint. I am pleased that Greene has seen fit to endorse the latest version of the therapeutic algorithm that I have published in various forms over the years.[2,3] As Greene appropriately points out, this algorithm does not require specific etiologic knowledge, but instead tries to match treatments with symptoms while advocating a cautious escalation-of-treatment model.

In apparent contrast to the direction taken by Greene are comments from the 1996 U.S. National Institutes of Health (NIH) Technology Assessment Conference on TMD.[4] Specifically, the distinguished scientists empanelled by the NIH evaluated evidence submitted by various experts at this conference and published the following thoughts about TMD etiology: "There are significant problems with present diagnostic classifications of TMD, because these classifications appear to be based on signs and symptoms rather than on etiology."[4p183] Also, they suggested that "studies need to be conducted that will elucidate the relationship between signs and symptoms, and etiology."[4p182] I interpret these sentences to suggest that pursuit of etiologic understanding is a worthwhile and important endeavor for both scientists and clinicians. Unfortunately, the NIH conclusions are similar to the recent Florida Supreme Court's decision to allow a recount of the U.S. presidential ballots. The Supreme Court did not specify the rules for conducting this recount, and without established rules, the potential for bias becomes highly probable. Similarly, the pronouncements from the NIH panel suggest that the pursuit of etiology is appropriate, but they do not describe how it is to be done. To properly determine etiology, evidence is needed that data collected on interview, by questionnaire, by examination, or through diagnostic testing will allow a clinician to clearly distinguish one etiologic agent from another. Moreover, data are needed to demonstrate that an etiology has a causal relationship between the putative agent and the patient's current disease status. Greene has correctly concluded that this evidence is not present and is not likely to appear on the horizon in the near future, and without it, bias toward a clinician's favorite etiologic theory becomes highly probable.

Finally, as a dental educator-clinician, I have long endorsed the idea that it is our job to seek out the etiology to the disorders we are trying to diagnose and manage, although there are times when it will be elusive. In fact, the motto I have used for years in the syllabus for my third-year DDS class on TMD is, "Our job is to find the clues which point to the correct diagnosis and management method, but if they are elusive, we must do no harm, as time and symptom management are often the best therapy of all for chronic musculoskeletal-based disorders." As I read Greene's article, I find that this motto is wholly in keeping with his thoughts.

Conclusions

In general, Dr Greene raises a viewpoint that many will find controversial, but it is extremely important to discuss. The wholesale pursuit of an etiologic-based therapeutic approach for TMD is both premature and difficult to justify, since such treatments often have greater expense, risk, and morbidity than a symptom-based escalation-of-care model. When one looks closely (as Greene has), the data proving that a specific agent or even a combination of etiologic agents can be differentiated from other possible agents do not exist. The data showing a clear-cut causal link between 1 or more etiologic factors and a specific TMD are lacking (with some obvious exceptions, such as macro-trauma and autoimmune-based polyarthritic diseases). There is no proof that individual anti-etiologic therapies actually can stop or reduce the etiologic agent they are supposed to affect. If I were asked whether Greene's commentary will affect how I actually practice and teach, I would respond that there is no conflict between my educational philosophy and approach to patients and his commentary. I have long recognized that typically our treatment will be symptom-driven and evidence-based, not etiology-driven. In addition, although it may be difficult to achieve, I will still search for an etiology every time I sit down with a patient, since I believe this is my job as a diagnostician. If and when I discover evidence that suggests that etiology is unequivocally linked to symptomatology, this information will influence but not obviate the logic I use to select suitable treatment, since all care provided must be evidence-based and appropriately sequenced.

References

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CRITICAL COMMENTARY 3

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Charles Greene's article[1] is a useful summary of current understanding of temporomandibular disorders (TMD). It complements the U.S. National Institutes of Health (NIH) 1996 statement on the management of these disorders[2] and the European literature. For instance, Molins' "From bite to mind" is a very similar look back at a career in temporomandibular pain.[3] Dr Greene draws attention to the similarity between back pain and TMD, endorsing the recent treatment approach in back pain, ie, early but conservative intervention in an attempt to prevent chronicity.[4]