AUTHOR'S RESPONSE TO CRITICAL COMMENTARIES

Charles S. Greene, DDS

The largely positive comments offered by my 3 distinguished colleagues are a source of great pride and satisfaction to me. At the same time, I appreciate their criticisms and suggestions, because they will enhance the discussion of this complex topic for both the readers and myself. In responding to their comments, I will attempt to clarify my positions on certain issues raised in my own article, as well as certain issues addressed primarily by them.

Perhaps the most important point to be made initially is that in discussing etiologic concepts, my article addresses mainly acute and short-term TMD. As Dr Carlsson[1] correctly states, such conditions sometimes will have clear antecedent events, such as trauma or even dental treatment. Yet, most clinicians dealing with these conditions have seen many new TMD patients who are completely mystified about how their troubles started. It is in these cases that clinicians begin to speculate on possible etiologies, usually based on their favorite theories of causation, and as Dr Clark[2] points out, the patient usually ends up with a treatment plan that is "matched" to the presumed etiology.

On the other hand, Drs Feinmann and Madland[3] correctly state that the situation is quite different for chronic facial pain patients. Not only is the diagnosis less clear or specific in these cases, but the etiology is more confusing, the prognosis for treatment is poorer, and the entire situation is likely to be confounded by psychosocial issues. Today we might speak of these problems in terms of *central sensitization* and *psychosocial distress*, but my favorite definition of chronic pain patients remains that offered years ago by Dr Benjamin Crue, who described them as ". . . those who complain chiefly of pain, but whose suffering is due either to unknown etiology and mechanism, or to trauma or disease that is considered too minor, or to have occurred so long ago, that it no longer can be regarded as a valid explanation for their symptoms."[4] Therefore, any meaningful discussion about the etiologies of TMD must be focused on the thousands of new patients who walk into dental offices around the world every day. It is for these patients that Clark's algorithm is primarily designed.

Dr Carlsson seems to feel that my article has not paid sufficient attention to the occlusal viewpoints and other structural concepts that have dominated the TMD field for so many years. This was a deliberate choice on my part, so that the article would not appear to be yet another "occlusionist versus nonocclusionist" debate. Therefore, I structured my review and critique of various etiologic concepts along purely chronological lines, so that the reader could follow the flow of competing ideas over the years. As Clark points out, in the end it is no better if somebody "mechanistically" applies either a biopsychosocial theory or a structural theory to a single patient's problem, because each will lead to a "specific" treatment concept that may be unwarranted.

I certainly agree with Carlsson that we all should acknowledge the importance of occlusion in restorative dentistry and prosthodontics—as a general dentist I recognize this every day in my practice. But I disagree with his conclusion that a friendlier or gentler approach to the occlusion/pain debate will make any significant difference in how that issue will ultimately be resolved. I also join him in recognizing that the impact of occlusion on temporomandibular health is not zero, but I do not see how we are supposed to "determine it in each individual case" unless the patient presents with a clear etiologic picture. I agree that one or more of the factors in the multifactorial TMD equation may sometimes be structural (joint anatomy, discal integrity, muscular tolerances, etc) as well as functional (occlusal relationships, parafunctions, oral habits, etc). However, my article argues that we do not currently have the tools to isolate and measure the etiologic significance of these factors in individual patients. Furthermore, since most correlational studies have shown only minimal associations between most of these factors and the presence of a TMD diagnosis, we should feel both intellectually and practically comfortable in the use of what Clark calls the "symptom-based escalation-of-treatment approach" until something better comes along.

Drs Feinmann and Madland have expressed their reservations about the meaningfulness of specific TMD diagnostic categories, as well as doubts about the future value of pathophysiologic solutions. Once again, they probably are correct in the case of chronic patients, where such distinctions become more blurred. But I think that acute forms of both myogenous and arthrogenous disorders throughout the body will someday be reversible to a great degree—and in the end, this is the best prevention against the development of chronicity.

The ultimate paradox of our current situation in the TMD field is this: We are getting much better at diagnosing and treating most of the patients, but we are not doing very well in persuading many of our clinical colleagues to abandon or modify their traditional (ie, dental) ways of thinking. Carlsson is correct in observing that this reluctance to change paradigms is a major obstacle to progress in our field, and in fact I have recently published a paper dealing with these issues.[5] But until we succeed in meeting that challenge, TMD patients will remain at risk for receiving vastly different therapies, determined primarily by who they consult rather than by the best scientific evidence.

References

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