FOCUS ARTICLE

The Etiology of Temporomandibular Disorders: Implications for Treatment

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This article begins by reviewing the history of etiologic thinking in the field of temporomandibular disorders (TMD). I conclude from this review that not only are the old mechanistic etiologic concepts incorrect, but also that 2 of the most popular current concepts (biopsychosocial and multifactorial) are seriously flawed. Therefore, what we really have at the individual TMD patient level is nearly always an idiopathic situation—we simply do not know enough, or cannot measure enough, or cannot precisely determine why each patient has a TMD. In addition, we do not understand the host resistance factors that ultimately determine why one person gets sick while another does not. The issue of "why" (etiology) must be differentiated from the issue of "how" (pathophysiology), both semantically and intellectually, to discuss all of this properly. However, our current inability to precisely identify etiologies in TMD patients does not prevent us from providing sensible (and often successful) treatment for most of these patients. Many health conditions currently are treated by physicians and dentists with either incomplete or flawed understanding of their etiology, but the availability of empirical data about treatment outcomes permits some level of appropriate care to be given. Fortunately, a large number of comparative studies have been done in the field of TMD therapy, providing us with a basis for selecting initial therapies as well as for dealing with treatment failures. Even in the absence of a perfect understanding of etiology, we still can provide good conservative care, and we should avoid aggressive and irreversible treatments, especially when they are based on flawed concepts of etiology. The article concludes by discussing current basic science research activities in the field of TMD and orofacial pain. I propose that these ongoing studies of the molecular and cellular mechanisms of joint disease, muscle pain, and chronic pain are the most likely avenues to future progress in this field, as specific countermeasures are developed to become the basis for more precisely targeted therapies.

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Much has been written about the etiology of the various temporomandibular disorders (TMD), but where do we stand today as a result of all this discussion? One would hope that all the old theories had been either verified or discarded, and that current theories would adequately explain the onset of these disorders. However, an extensive analysis of the literature on TMD etiology reveals that neither of these objectives has been totally accomplished at this time.

Similarly, much has been written about the subject of treating the various TMD, but where do we stand today with regard to actually providing appropriate treatment? One would hope that all TMD patients were receiving rational therapy based on a combination of knowledge about their etiology and treatment outcome data from controlled studies. However, an extensive analysis of the literature on TMD therapy reveals that this is not what is occurring in most cases.

The above conclusions should not be interpreted as entirely negative statements, because in fact there has been significant progress, both in understanding etiologic factors in TMD and in rationally treating patients with these conditions. The problems lie in the assumptions that are frequently made about "finding" the etiology of an individual patient's problem, followed by "selection" of an appropriate anti-etiologic treatment strategy. It is the purpose of this article to deal with these issues by raising the following questions:

1. What do we actually know with some degree of certainty about the role of various putative etiologic factors in causing subgroups of TMD patients to develop clinical problems?
2. Can this knowledge be transferred from the group level and applied to the individual patient in a reliable manner?
3. Are currently recommended treatment protocols, even at the highest scientific level, based on suppressing or eliminating etiologic factors?
4. If not, then on what are these currently recommended protocols (most of which are quite successful for the vast majority of patients) actually based?

To limit the scope of this paper, the ensuing discussion will focus only on the 3 major categories of TMD: *(1)* myofascial pain and dysfunction involving the masticatory muscles and associated structures, *(2)* internal derangement phenomena that produce complaints of pain and dysfunction, and *(3)* temporomandibular joint (TMJ) inflammation and/or degeneration producing complaints of pain and dysfunction. The author recognizes that there are many systemic conditions that either affect the TMJ complex directly or are comorbid with TMD. Similarly, there are many organic pathologies that can occur locally in the tissues of the TMJ complex. However, none of these phenomena will be discussed here.

The Semantics of Etiology

Before I embark on a discussion about etiology, some common semantic problems need to be pointed out (Fig 1). Doctors often speak of finding the "cause" of a patient's pain, but they really mean the "source" (an anatomic structure producing the pain) as opposed to the "site" (an area of referred or heterotopic pain). Other common expressions include: "mechanism of pain," which really means the pathophysiologic process occurring in the anatomic structures; "pathogenesis," which refers to the origins and progression of a pathologic process; and, most confusing of all, "diagnosing the etiology," which usually describes a mental process within the clinician's brain. In this paper, the term *etiology* is defined in both the simplest and the strictest way: we want to know *why* a particular patient began to have both the biology and the perception of his pain (in the absence of frank trauma).

Historical Review of Etiologic Theories

I have written previously about the evolution of etiologic concepts in the field of TMD,[1,2] so only a brief review of some older and some more recent concepts will be offered here. As many people know, this field began as a lateral transfer from the otolaryngology profession in the early 1930s. While some papers had appeared in the dental literature before that time,[3–5] it was the pronouncements of Costen[6] that established the TMJ as a separate source of facial pain and of about 11 other symptoms (most of which turned out to be impossible to connect anatomically with the TMJ).[7,8] It is not important to belabor the details of Costen's concepts, but their main impact was to lay the foundation for 2 propositions that dominated the field for years to come: *(1)* These so-called TMJ problems were a result of structural malalignments between the mandible and the skull, and *(2)* only dentists could take care of TMJ problems because of the structural corrections that would be required. Terms such as *overclosed vertical dimension*,[9] *condylar malposition*,[10] *trapped mandibles*,[11] *occlusal disharmony*,[12] and *neuromuscular imbalance*[13] all were variants of this initial conceptual framework, and the treatments to correct all of them became part of the lexicon of dental therapies for many years. Whatever one may think of these concepts, it is clear that they were the basis for an etiologic viewpoint and that the related therapies were seen as being anti-

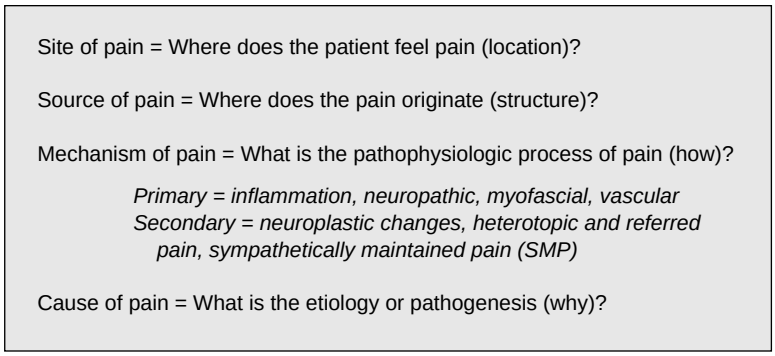

Fig 1 The semantics of orofacial pain "etiology."

etiologic; indeed, the word "definitive" often was used to describe the curative value of these treatment approaches.

During this same general time period, the orthodontic profession had come up with their own version of structural disharmony concepts and corrective treatments. Since I have previously written about these matters,[14,15] I will limit the current discussion about them here. However, it is important to note that the orthodontic viewpoint recently has converged considerably toward the traditional prosthodontic/occlusal viewpoint,[16] as more and more members of both groups have become devotees of mechanical and electronic instrumentation to analyze and treat their patients.[17,18] While this is arguably acceptable or not as a protocol for providing good orthodontic or prosthodontic care, such concepts and instrumentation have not been shown to be of specific value in diagnosing or treating TMD patients.[19–22]

Another structural concept of TMD etiology has been proposed by various physical therapists, chiropractors, and dentists[23,24] based on the notion of "bad" craniocervical postural relationships causing TMD. While this etiologic theory has enjoyed some popularity in the past (and is still popular in some regions of the world), several studies have demonstrated that there are no consistent postural findings that differentiate TMD patients from normal subjects.[25,26]

In more recent years, other etiologic factors besides structural ones have been recognized and discussed as a result of studies of large patient populations.[27,28] For example, trauma at both the macro and micro level[29] has been observed in the history of certain patients, with rather clear relationships to symptom onset in many of them. The most significant changes in etiologic theorizing, however, began in the 1950s and 1960s, when the Columbia University group (Schwartz, Marbach, and others[30–32]) as well as the University of Illinois group (Laskin, Greene, and Lupton[33–35]) proposed a psychophysiologic basis for many TMD, especially those involving myofascial pain and dysfunction (MPD). Again, these concepts arose from studies of large TMD patient populations using a variety of psychometric approaches for assessing personality characteristics as well as various state/trait variables in these patients.[36–38] In addition, a large number of experimental stress provocation studies showed that TMD/MPD patients differed from normal subjects in many of their responses, and several psychophysical measurement studies demonstrated other significant physiologic differences between them.[39–46] While Laskin's classic paper about the etiology of MPD[33] served as the basis for much of this experimental and analytic work, eventually his psychophysiologic theory proved to be incomplete as an etiologic explanation for developing myofascial pain. Based on our own research findings as well as those from other centers, the Illinois group in 1982 published an important paper expressing these reservations.[47] Today, the importance of psychologic factors in the onset, progression, treatment, and persistence of various TMD is well recognized as foundational knowledge in this field,[48] but the question of why some patients get TMD symptoms while others do not remains unanswered by that etiologic theory.

Finally, no historical review of etiologic concepts in the TMD field is complete without at least a brief consideration of the unorthodox and pseudoscientific theories that have been proposed. This is also a topic that I have written about previously,[49,50] so little needs to be said about it here. Many of these ideas originated outside of the dental profession (eg, craniosacral therapy from osteopathy, applied kinesiology or jaw malalignment theories from chiropractic, nutritional

theories), only to be warmly embraced by dentists.[50] Others originated within the profession, such as TMJ malalignment causing whole-body symptomatology,[51] or neuromuscular imbalance in the face causing widespread problems with other muscles or even in various organs.[52] These peculiar concepts have served only to muddy the TMD waters further, but fortunately each one seems to die a deserved natural death within some reasonable period of time.

Hybrid Theories

Most of the above etiologic concepts could be described as unicausal, implying a simplicity of cause-and-effect that became increasingly untenable as more was learned about TMD. In the past 25 years, we have seen the emergence of various hybrid concepts of etiologies. The earliest was the proposal by Ramfjord and Ash[53,54] that a combination of stress and occlusal disharmonies was responsible for the development of TMD symptoms in previously asymptomatic persons with "bad" occlusions. Rather than focus on the psychologic component, however, the authors advocated occlusal correction as their primary treatment. This type of lip service to the concepts of psychobiology has been repeated by many others who have claimed that they appreciate the importance of psychologic factors, but who emphasized mainly mechanical treatments in their clinical approaches to TMD patients. Most recently, the combination of a biologic and psychologic perspective in etiologic discussions about TMD has been given the name "biopsychosocial."[55,56] More will be said about this in the next few paragraphs.

Another approach to describing the complexity of etiologies is to invoke the word "multifactorial,"[58,59] thereby indicating an awareness that many extrinsic factors in the environment, as well as various intrinsic factors within the patient, might be involved in the development of symptomatic TMD. This is intellectually attractive in the sense that it suggests an appreciation of complexity, but does it indicate a deeper understanding of what is actually happening?

This may be an opportune time to consider the true meaning of the words "biopsychosocial" and "multifactorial" as expressions of etiologic thinking. In doing so, we also must consider their application at 2 levels of patient analysis: first, how do they help us understand *groups* of TMD patients; and second, how do they help us understand *individual* patients?

My answer to these questions is as follows, adapted from the discussion by Okeson.[56] The word "biopsychosocial" is actually a combination of 3 words, producing an excellent descriptor of the world that most pain patients (and especially chronic patients) are living in from day to day. They have a *biologic* problem (ie, activation of pain pathways, with or without demonstrable pathology), which may have *psychologic* antecedents as well as behavioral consequences. This situation exists in a *social* framework that includes interpersonal relationships with friends, families, and health providers, which almost always produces major negative experiences for the patients themselves. But how can we assess all of these variables at the individual patient level with the crude physical and psychometric tools that are currently available? I submit that we cannot do so, and therefore this concept is valuable only at the group level.

The word "multifactorial" is, in my view, even worse in these respects. Of course we know that a very complicated assortment of extrinsic physical and psychologic factors is acting on the variable host factors of physical and mental healthiness. Behavioral issues, including stress, anxiety, interpersonal relationships, and oral habits, are potentially significant in such an etiologic matrix, and physical issues of joint anatomy, loading, and pathology as well as muscle physiology are undeniably important—but how are we supposed to assess all of this in an individual patient? Once again, I submit that this concept is both correct and valuable at the group level, but it cannot be specifically applied to any single patient sitting in a pain clinic.

The Idiopathic Concept of Etiology

The central thesis of this article is that we currently have a set of disorders affecting the stomatognathic system that we know a lot about, thanks to nearly 40 years of systematic research, but we do not know much about their etiology at the *individual patient* level. In fact, as the old joke goes, we used to know a lot more about their etiology before we submitted them to so much systematic research! Some doctors seem to feel sad that these negative conclusions about etiology leave us with nothing solid to hang our hat on, and so we end up with the term "idiopathic" (defined as a disease of unknown origin or for which no cause is known) to describe our current state of knowledge about most TMD.[59–63] However, I would argue

Table 1 Relationships Between Diagnosis, Etiology, and Treatment in Temporomandibular Disorders

	Diagnosis	Etiology	Treatment
Ideal*	Clear and correct Measurable Demonstrable	Specific Measurable Treatable	Anti-etiologic Definitive Successful
Acceptable†	Presumptive Probably correct Categorical labels	Unclear Complex Reversible	Empirically validated Matched to diagnosis Conservative
Wrong/bad‡	Parochial specialty labeling Technological diagnosis Possibly correct	Favorite theory Morphofunctional analysis Mechanical concept	Prolonged splint wear Bite-changing procedures Jaw repositioning
Outrageous§	Misdiagnosis of pain Neglect of serious pathologies Neglect of chronicity	Guru/cult concepts Quackery concepts Parochial specialty concepts	Whole-body procedures Quackery procedures Extreme dental procedures

*Not achievable at this time.
†Frequently achievable; represents best current practice.
‡Most common current practice, despite lack of scientific foundation.
§All too common; represents fringe of current practice.

that any sadness about our relative ignorance regarding specific etiologies should be greatly mitigated by the enormous amount of specific knowledge we do have, thanks again to the extensive research of the past 40 years. In looking at the traditional triad of diagnosis, etiology, and treatment, it should be apparent that we have vastly improved our ability to recognize and classify patients with TMD, and also that we have developed logical, sensible, and successful methods for treating most of these patients. This conclusion is strongly supported by the widely reported treatment success rates of 75% to 90% from around the world.[64–74]

In the remainder of this article, the case will be made for treating TMD entirely on the basis of the application of research-based treatment protocols to specific TMD diagnostic categories—an approach that requires little or no attention to individual etiologic factors. While some people may disagree, I believe that this is what every clinician *really* is doing while providing treatment for most TMD patients, regardless of protestations to the contrary (Table 1).

A Low-tech, High-prudence Therapeutic Approach

The title of this section is taken directly from the title of one of the finest papers written to date on the subject of treating TMD, by Stohler and Zarb.[75] After extensive review and discussion of the large number of incredibly diverse treatments that have been proposed over the years, they conclude that patient safety should be the top criterion for selection of appropriate therapies. They argue that diagnosis must be primarily symptom-based until credible biomarkers of various TMD are discovered, and so they caution against "overly ambitious data-gathering" (eg, excessive imaging, electronic assessments) unless it contributes to better choices of treatment options. Regarding currently available therapies, Stohler and Zarb make 2 important points (emphasis mine):

1. "*No* compelling data has been presented with respect to any kind of treatment possessing either disease-modifying or even curative properties," and
2. "The superiority of invasive procedures over conservative therapies has *not* been demonstrated by any kind of systematic research."[75p259]

In light of these facts, they conclude that a reasonable clinician must choose low-tech and high-prudence treatment approaches, and fortunately these work for many patients. The objective of these approaches can be summarized quite simply as *reducing* pain, inflammation, and psychologic effects, while *increasing* muscle strength, range of motion, and bite comfort.

In addressing the subject of etiology, Stohler and Zarb remind us that one cannot use the word "cure" in the absence of knowledge of causal mechanisms, and especially without the availability of biotechnologies that interfere with those mechanisms. Near the end of their paper, they offer these important conclusions:

> Two compelling realities underscore our conviction about the significance of a prudent, low-tech management of TMD. The first is that the etiology and pathophysiology of the disorders are poorly understood . . . Furthermore, neither dental nor psychologic factors per se have been shown to cause TMD, although they can be associated with the onset of symptoms. The second reality recognizes the collective clinical experience that most patients with TMD can be efficaciously managed at a primary care level. This is quite similar to the predicament of most patients with other types of musculoskeletal disorders. Therefore, a stepwise, patient-centered approach to management, based on symptom severity, is advocated.[75p260]

Primary Care for TMD Patients (Phase I)

Almost all of the current authoritative guidelines for treating TMD suggest a conservative, reversible approach to initial therapy.[57,76–79] This approach includes the use of well-known and widely accepted treatment modalities, including various medications, oral appliances, physical therapies, and home care procedures. Unfortunately, some people have created a false dichotomy between this initial "Phase I" therapy and a so-called "Phase II" regimen that requires irreversible dental and skeletal changes to be made.[54,80,81] Under this concept, the use of oral splints to reposition the mandible in Phase I often produces an unavoidable Phase II.[82] Alternatively, some people have argued that Phase I is merely a palliative approach that is sufficient if it works, but one must be prepared to "escalate" to more aggressive treatments in many cases.[83,84] Under this concept, treatment failure becomes an excuse to perform invasive therapies, rather than signaling the possibility of non-responding chronic pain (with all its psychologic implications).

It should be emphasized at this point that my use of the word "irreversible" does not refer exclusively (or even primarily) to TMJ surgical procedures. While many medical treatment arguments may revolve around a dichotomous choice between medicine and surgery, in dentistry we have a third choice of irreversible therapies that often are applied to TMD patients. These therapies range from simple bite-adjustment procedures to complex jaw-repositioning therapies, often involving the use of sophisticated-looking electronic devices, articulators, and imaging (especially magnetic resonance imaging). Inevitably, these complex treatment programs conclude with full-mouth bite-changing procedures such as orthodontics, orthognathic surgery, and reconstructive dentistry. Indeed, it has been argued that such therapies for TMD patients can be more invasive and more irreversible than most TMJ surgical procedures.[85]

In my own 1992 paper on initial therapy,[85] I argued that good primary care is not a first phase—it is the actual treatment program that most TMD patients require and will be quite successful for many of them. In that paper, I pointed out the dangers of various irreversible treatment approaches and improper escalations of therapy, so I will not repeat them here. Many other prominent researchers in this field have proposed a similar viewpoint,[57,76–79] and the long-term research on clinical outcomes from around the world supports the use of conservative and reversible treatments as the only approach for the vast majority of TMD patients.[64–74] This important conclusion is represented most clearly and concisely in the *Guidelines for Assessment, Diagnosis, and Management of Orofacial Pain* from the American Academy of Orofacial Pain (AAOP).[57]

Clark's Treatment Algorithm for TMD

This article includes several citations from the work of Glenn Clark, who with his colleagues at the University of California at Los Angeles has established a sensible foundation for the diagnosis and treatment of orofacial pain patients. In particular, the logical treatment algorithm for treating TMD patients that was designed by Clark more than 20 years ago is still one of the best summaries of this subject, and a recent updated version of his algorithm flow chart[86] is reproduced here in its entirety (Fig 2). The astute reader will quickly notice that the driving force behind the selection of treatments for each patient is the presumptive diagnosis—not the etiology. With limited jaw opening as a pivotal dividing criterion, Clark recommends treatments whose outcomes help the clinician to verify or discard the original diagnosis. The conservative treatment modalities presented are strategically arranged so that either success will

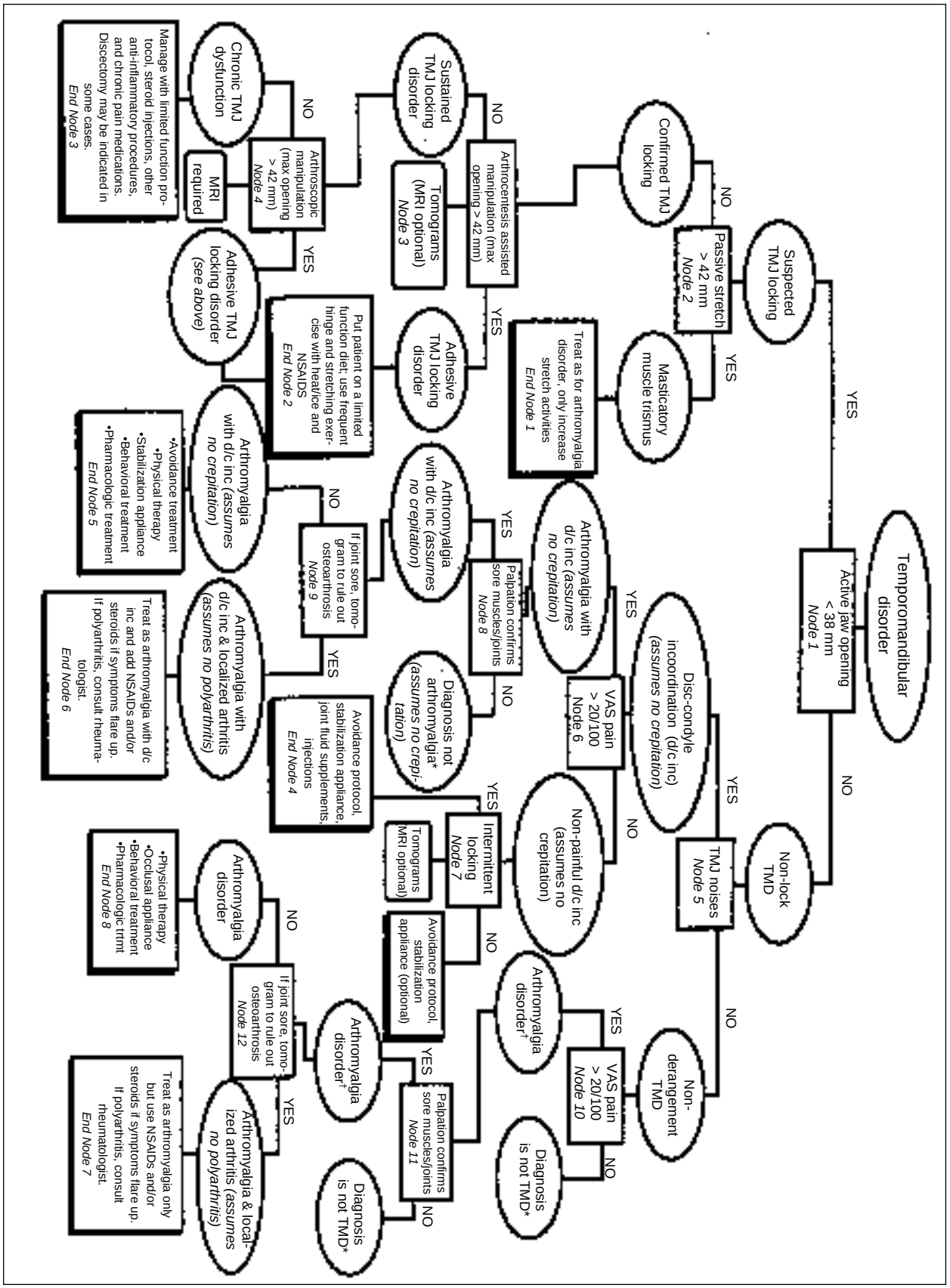

Fig 2 Clark's algorithm for treating temporomandibular disorders.[85] *Continue workup; †Verify via palpation to rule out osteoarthritis. VAS = visual analog scale; MRI = magnetic resonance imaging; NSAIDs = non-steroidal anti-inflammatory drugs. Reprinted with permission from the Japanese Prosthodontic Society.

occur, or a new logical choice can be tried next. Both positive and negative outcomes turn out to be valuable in making the next decisions, and every treatment "node" ends with some reasonable management strategies. Even complete treatment failures are addressed in a manner that avoids improper escalations to inappropriate therapies.

It is no coincidence that a similar situation has existed for many years in the field of back pain, another complex area of musculoskeletal pain and dysfunction that is filled with analogous arguments about structural defects, functional disabilities, and psychologic overlays. Indeed, the following paragraph written by Flor and Turk in 1984[87] could be applied today in our field, simply by changing the words "back pain" to "temporomandibular disorders":

> If one tries to delineate the possible causes of back pain, one must resort to speculation with little empirical basis. As the research reviewed shows, neither degenerative, nor structural or muscular, nor occupational factors seem to have any clear-cut relationship to lower back pain. They may contribute to back pain, but they do not appear to be sufficient causes for most cases of lower back pain. This conclusion is especially troubling in light of the number of treatment approaches offered, which often are based on questionable assumptions about the pathophysiology of these chronic pain conditions.[87]

Therefore, clinicians responsible for treating back pain have had to employ decision matrices that are not unlike Clark's algorithm so that they can care for their patients logically and effectively.

Treating TMD in a Biopsychosocial Framework

Several researchers in the TMD field have observed that the primary symptom that determines treatment-seeking is the facial and head pain experienced by these patients. Since it is well known that both acute and chronic pain have psychologic associations, a responsible clinician must take that fact into account while treating all TMD patients. The literature supporting that conclusion and endorsing that approach to TMD treatment goes back over 30 years, beginning with the early studies of Schwartz[30,88] and Moulton[32,89] and including those of Laskin, Greene, Lupton, and others.[33–36,47] These ideas were advanced greatly by the work of Rugh and Solberg in the 1970s and 1980s,[37,90] as well as the work of Turk, Rudy, and associates in the past 15 years.[91,92] Perhaps the strongest focus on psychologic issues has come from the diverse investigations directed by Dworkin and LeResche for many years,[93–95] and their use of the term "biopsychosocial" has become widely recognized as a most appropriate label for the TMD patient population. Along with Turk et al,[91] they have recommended the use of a cognitive-behavioral approach to the education and treatment of TMD patients, and they have clearly demonstrated its effectiveness.[93–95] This approach offers the dual benefit of teaching the patient how to self-manage many of his symptoms, while enhancing the feeling of empowerment (locus of control) that comes from such skills.

As pointed out earlier, this is not an etiologic issue but rather a tactical one. Good clinicians need to be sensitive to the psychologic ramifications of pain in both acute and chronic TMD patients, and they must expect to encounter significant psychologic issues such as anxiety and depression more frequently in the latter group. Only by developing this kind of awareness can they avoid the mistakes of escalation either to surgeries or to major dental treatments, instead of referring their non-responding patients for the kind of complex chronic pain management that is much more likely to be appropriate. In the end, it is this awareness that defines the biopsychosocial approach to the diagnosis and treatment of TMD patients.

The False Dichotomy: Palliative Versus Definitive Treatments

The arguments in medical circles about palliative versus definitive treatments is as old as Greek and Roman times, when healers debated bloodletting and amputations as alternatives to poultices, herbs, and incantations. The implication always has been the same: A good doctor must not settle for mere palliation when a definitive cure is available. But as Stohler and Zarb[75] pointed out in the passage cited earlier, one cannot use the word "cure" in the absence of knowledge of causal mechanisms, and especially without the availability of biotechnologies that can reverse those mechanisms. Therefore, this becomes a false dichotomy in those conditions where it cannot be properly applied, for example, low back pain, headaches, and TMD.

In the TMD field, this argument is best understood by consideration of the Phase I/Phase II controversy.[96–99] Several important papers addressing

this controversy have appeared in the TMD literature during the past 25 years, and all of these have reached similar conclusions: TMD patients treated with conservative and reversible modalities on the whole respond as well as patients treated with more aggressive regimens, in both short-term and long-term assessments of outcomes.[64–74] In other words, Phase I therapy alone, or in combination with Phase II therapy, produces similar overall results in large populations of TMD patients. Since Phase II treatment by definition involves irreversible procedures, these outcomes strongly suggest that Phase II represents unnecessary overtreatment in most cases.[82,85,100]

It should be apparent from these findings that the distinction between "palliative" and "definitive" has little meaning in the treatment of these benign musculoskeletal disorders. Instead, we can now speak more rationally about TMD treatment in terms of 3 clinically and intellectually important considerations:

1. The natural history of each of the major TMD, especially the intracapsular diseases and derangements, is now fairly well understood. In general, the course of these conditions is characterized by positive tissue adaptations and recoveries from episodes of pain and dysfunction.
2. The objective of TMD treatments generally should be to make patients more comfortable (palliation) as the above adaptations are occurring, while also enhancing the amount of recovery as much as possible. These treatments should be selected on the basis of the clinical subdiagnoses of myogenous and/or arthrogenous conditions, as defined by the AAOP Guidelines[57] and by Dworkin and LeResche in the Research Diagnostic Criteria for TMD.[101] The psychologic distress associated with being sick as well as worry about one's illness also must be addressed, which means that simple behavioral management techniques should be incorporated into every treatment protocol.
3. Successful TMD treatment, therefore, should be defined as a return to a more normal biopsychosocial existence, with pain either greatly diminished or gone, and with the patient educated to self-manage most recurrences (if any) of the problem.

These conclusions about treatment are further supported by the official Science Information Statement published by the American Association of Dental Research in 1996[102]:

> Based on the evidence from clinical trials [of TMD], . . . it is strongly recommended that, unless there are specific and justifiable indications to the contrary, treatment be based on the use of conservative and reversible therapeutic modalities. While no specific therapies have been proven to be uniformly effective, *many of the conservative modalities have provided at least palliative relief from symptoms without producing harm* (emphasis added).

Future Perspectives

Most papers like this one end with the author urging scientists to continue searching for more answers to the main topical issue—in this case, to learn more about the etiology of TMD so that better treatments can be provided. While I am happy to endorse that sentiment, I actually do not expect much progress to come from such endeavors. Instead, I believe most of the future progress in this musculoskeletal pain field will come from intensive studies of the pathophysiologic mechanisms underlying all kinds of muscle and joint pain, as well the phenomena of neuroplasticity leading to chronic pain. Some of these kinds of studies are already either completed or under way, and eventually they should provide the scientific basis for developing tissue-targeted therapies that will reverse the pathologic processes, rather than merely palliating the associated symptoms. In the remainder of this paper, a brief summary of some current work in these areas will be presented.

At this point in time, the study of joint tissue changes at both microscopic and molecular levels is far ahead of similar studies of muscle tissues.[103–109] Several important features of cartilage degradation, bone catabolism, and inflammatory biochemicals have been elucidated, with a strong focus on proteoglycan synthesis or degradation. Chemicals in the cytokine and metalloproteinase groups are known to be significant mediators of this activity. Experiments in laboratories as well as in clinical populations continue to demonstrate the details of these pathologic processes, and researchers in this area are optimistic about their ability to reverse them in the near future.

The situation appears to be much more complicated in regard to muscular pain. In a 40-page review with over 400 references that was written in 1993,[110] Mense offers many hypotheses to explain the pathophysiology of muscle pain, including a variety of cellular and molecular mechanisms, but no clear answer emerges. Stohler has

summarized current theories about painful muscular TMD in a recent article[111] that discusses the role of both peripheral and central sensitization factors in muscle pain.[112–114] The chemical mediators released from damaged tissue cells, mast cells, and platelets can either activate or sensitize free nerve endings in muscle tissue. Also, sympathetic stimulation of these endings in muscle can occur following injury or inflammation. Stohler's main focus, however, is on the role that nerve growth factor can play as a mediator in persistent muscle pain,[115] and also on the role that estrogen can play in that process.[116] These findings may offer a scientific basis for understanding the increased persistence and severity of muscle pain in women, and inevitably some advances in treatment will come from such investigations.

Finally, we must consider the enormous impact that neuroplasticity has on the pain experiences reported by our patients. The research by Dubner,[117] Sessle,[112,118] and many others in this field has demonstrated how normal peripheral sensitization of receptors can lead to an amplification and persistence of pain that goes far beyond "normal."[119–122] Long-term changes in nerve cell activity at the level of the spinal cord and higher centers in the brain (central sensitization) are also a frequent result of nerve excitation or injury. The mechanisms that determine whether the body suppresses this sensitization or facilitates it are still largely unknown, but significant work is proceeding in those areas; already, certain neurochemicals have been identified that may be manipulated for the management of persistent pain.[123,124]

Hopefully, these diverse scientific investigations will lead ultimately to specific therapies for each kind of TMD. Even chronic facial pain patients can look forward to more specific treatments as the perpetuating mechanisms for chronic pain become better understood. Until then, we must recognize the limits of our knowledge about both etiologies and mechanisms. Fortunately, we already have enough scientific information to enable us to provide the majority of our TMD patients with what they want most: relief from pain, return to more normal function, and avoidance of iatrogenic harm.

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CRITICAL COMMENTARY 1

THE ETIOLOGY OF TEMPOROMANDIBULAR DISORDERS: IMPLICATIONS FOR TREATMENT

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Dr Greene has provided in his Focus Article a well-written review of the etiology of temporomandibular disorders (TMD).[1] However, it may seem provocative to many people. Its conclusion that all old etiologic concepts are incorrect is probably discouraging to many of those who have been working in the TMD field for several decades. The unicausal etiologic concept—that TMD is one disorder with one cause—has long since been abandoned. It has been widely accepted that TMD are a set of disorders affecting the stomatognathic system; this has led to new explanatory etiologic models, eg, the multifactorial and biopsychosocial concepts. The goal for much of the more recent research has been to clarify the etiology of the various types of disorders that most people currently agree constitute TMD.[2,3] Greene not only rejects the unicausal concept, but he also considers the multifactorial and biopsychosocial concepts seriously flawed, even if he submits that they may be correct at the group level. His conclusion is that at the individual TMD patient level, there is nearly always an idiopathic situation, ie, we do not know why the individual patient has a TMD. However, he gives some comfort by saying that even in the absence of a perfect understanding of etiology, we still can provide good conservative care that may be helpful for the majority of TMD patients. The argumentation for his statements is strong and convincing, but it deserves some critical comments.

Are Temporomandibular Disorders Idiopathic?

Greene can find support in a recent proposal for a new classification system of idiopathic orofacial pain.[4] This Focus Article, by Woda and Pionchon, included as idiopathic not only so-called atypical types of pain but also facial arthromyalgia, a term that seems to correspond to TMD. The inclusion of all types of TMD in this group probably surprised many readers, and it was also questioned, especially in one of the commentaries to the article.[5] In a similar way, Greene's suggestion that TMD etiology is unknown at the individual patient level may be too pessimistic. Acute muscle and TMJ pain can often be identified as caused by trauma to the face or an inflammatory process in the TMJ. Many TMD have been studied extensively and well described, and this knowledge should of course be used not only at the group level but also in managing an individual patient who fits a known diagnosis with reasonable certainty. However, we have to agree that in many patients with chronic orofacial pain, the etiology is unknown. For such conditions, the "idiopathic" label may be adequate. The implications for treatment of patients with such conditions are well described in Greene's article. Escalation of the therapeutic attempts can lead to overtreatment and should be avoided.

Occlusion and Temporomandibular Disorders

Greene has written very little about occlusion and TMD in his article, which is surprising given the enormous earlier interest in this topic. Greene refers to some of his previous papers in which he has repudiated occlusal and other mechanical and structural factors in TMD etiology. Nevertheless, a brief but clear discussion of the importance of occlusion would have been desirable. A majority

of dentists in the United States and probably in several other countries still believe that occlusal factors are of great importance for development of TMD, and consequently they also consider occlusal treatment to be essential in the management of TMD patients.[6,7] Dentists with such a belief will most probably regard a review of TMD without discussion of the role of occlusion as inadequate. Even if most "TMD experts" agree today that the role of occlusion is minor in TMD etiology,[8–10] there are groups of colleagues who still maintain that occlusion has a great influence on TMD.[11] Such statements are certainly based more often on emotions than on evidence. However, there are researchers who have had serious ambitions to further evaluate this issue through the use of well-designed, long-term, controlled clinical studies. Based on results of a 4-year comparison of real and mock occlusal adjustment, Kirveskari et al concluded that elimination of the presumed structural risk from the dental occlusion appeared to significantly reduce the incidence of TMD in a select group of young subjects.[12] As a consequence, they also suggested the need for further clinical studies.

The continuing divergence of opinions indicates that this issue deserves further discussion, based on the best possible evidence. It is difficult to convince colleagues who believe in a great influence of occlusion on TMD development that it is nonexistent or very small. The discussion must be better structured than it usually has been between "occlusionists" and "nonocclusionists." To start by agreeing that occlusion is of great importance in restorative dentistry and prosthodontics may be helpful. The next step—discussion of the relationship between TMD and occlusion—may then be easier. The most extreme standpoints should also be avoided. A recent review concluded that occlusion does not play a major role in the etiology of TMD; however, the impact of occlusion is not zero, and should be determined in each individual patient.[9,10] To ease the reluctance to abandon nonsupported dogmas on occlusion, it may be appropriate to acknowledge that initial simple occlusal adjustment may be as effective as any of the nondental low-tech therapeutic approaches available.[13] At that point, it might be easier to gain acceptance of the fact that there is no evidence to support repeated or extensive occlusal therapy as a meaningful TMD treatment.

Evidence-based Dentistry

With the increased emphasis on evidence-based care, the efficient transfer of knowledge from scientific results to the clinical practice is essential. Researchers and educators have obviously, to a large extent, failed to transfer the evidence-based knowledge available in the TMD field to general practitioners. It takes time to change opinions. To shift the occlusion paradigm that was so predominant for so long in the TMD field will require generations of dentists. So many dentists have been "indoctrinated" with what at the time of their dental training was considered the "definitive truth" on occlusion and TMD, and some of them will keep their conviction forever. The opposition against abandonment of occlusal etiology has been strong, as seen in many conferences focusing on occlusion and TMD. The turbulence at one of these events has been described as "a clash of cultures—between that of the researcher and that of the practitioner."[14] The discussion continues on a quite aggressive level, the anger of the clinicians is obvious, and much of the research on which the "TMD experts" base their conclusions is called into question.[15] If the evidence provided so far is not convincing enough, more high-quality clinical research should be performed. To solve this discrepancy in opinions in a longer perspective, it is necessary that dental education at all levels be permeated by a strong emphasis on evidence-based principles. Students must also be taught the need for continuing education and perusal of the relevant literature.

The discrepancy between evidence-based knowledge and clinical practice is not unique to the TMD field. The example cited by Greene—a paper from 1984 regarding back pain—is still relevant in the year 2000 according to Cochrane Reviews available as abstracts on the Internet (http://www.update-software.com/ccweb/cochrane/revabstr/g05index.htm). Similar situations are evident in other areas of dentistry. The etiology of dental caries and periodontal diseases is well understood, and efficient methods of preventing and controlling these diseases have been developed.[16] Nevertheless, these diseases are far from eradicated, because the knowledge has not been generally implemented.[17] Caries prevalence has, however, decreased in most industrialized countries during the last 2 decades, and the prognosis for further improvement through the use of the available knowledge is good.[18]

In the TMD field there is, as Greene notes, an enormous amount of specific knowledge, thanks to

the extensive research of the past 40 years. We have learned to manage successfully most TMD patients by using a "low-tech, high prudence therapeutic approach."[19] In Scandinavia, a similar approach has been applied for decades, emphasizing simple methods in diagnosis and management of TMD.[20]

Future Perspectives

Greene believes more in basic than in clinical research for future development of more precisely targeted therapies. The basic methods will of course be of great importance, as they have already been for pain and related research. A problem has been the difficulty of having the new knowledge disseminated and understood at the clinical level. Therefore, I think that well-planned and performed clinical studies continue to be necessary to test, apply, and control results of laboratory research. The ultimate goal is of course to reduce as much as possible the impression that the etiology of TMD is idiopathic.

Conclusions

Greene has provided a thought-provoking article that deserves to be read and discussed by all interested in TMD. I think that the idiopathic label should be used as little as possible, but it has to be admitted that the etiology and pathophysiology are poorly understood in many TMD patients, especially in those with chronic problems. There is a need for more good clinical studies in the TMD field. The prolonged controversy surrounding the relationship between TMD and occlusion can only be settled by acceptance of evidence based on high-quality research.

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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.

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CRITICAL COMMENTARY 3

THE ETIOLOGY OF TEMPOROMANDIBULAR DISORDERS: IMPLICATIONS FOR TREATMENT

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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]

Undue attention has undoubtedly been given in the past to subclassifying disorders affecting the temporomandibular joint (TMJ)—the lumbar spine of the face. Concentration on clicks, locks, and noise leads the practitioner further and further into irreversible treatments, a route without much evidence-based support, since there is a high prevalence of joint sounds in the general population[5] and a huge range of mandibular mobility.[6] Confusion of non-pathologic and pathologic problems in the joint is probably unhelpful also. Of course, the etiologies of TMD are not clearly understood, but as Greene reminds us, this does not mean therapeutic disaster or therapeutic nihilism.

Rather than adopt a restricted view of pain, we should view TMD as one of an ill-understood group of chronic pain conditions that may involve the whole of the mouth and face. Unfortunately, descriptions of disorders and treatment tend to be influenced by the background of the specialist assessing the patient. Greene acknowledges this. While Greene has doubts about the application of biopsychosocial and multifactorial variables to the individual on the grounds that these aspects cannot all be assessed in an individual patient, this should move us to adopt better psychometric instruments, as in other pain conditions. A multidisciplinary clinic allows all aspects of a patient's problem to be assessed. Patients who see maxillofacial surgeons have symptoms described in terms of clicking, sticking, and locking of the TMJ and pain in the associated musculature. Ear-nose-throat surgeons may retain Costen's outdated notion that the pain results from missing molar teeth and may refer the patient to maxillofacial surgeons or restorative dental specialists. Greene reminds us that, despite advice from the NIH that "there is no evidence linking occlusal abnormalities with pain," patients' occlusions continue to be adjusted by ill-informed practitioners, often leading to more problems for patients, well described by Clark and clearly illustrated by Forssell et al in their review of occlusal treatment.[7]

Participants in the NIH conference in 1996[2] reviewed the issues regarding the management of orofacial pain, concluding that significant problems hampered present diagnostic classifications and treatment disorders. More than 4 years later, there is no greater clarity in classification. The American Academy of Orofacial Pain has recently recommended that such pains be diagnosed and treated in a manner consistent with the diagnosis and treatment of any system of joints and muscles in the body, ie, as "head and neck management" rather than "TMJ management." In addition, it seems sensible to separate acute and chronic (over 6 months) TMD, particularly as they show major psychologic differences.[8]

Although Greene deliberately restricts his discussion to TMD, it is worth considering the various other widely recognized pain problems that affect the mouth and face, and often coexist with TMD. *Atypical* or *chronic facial pain* refers to pain in the non-joint, non-muscular part of the face, often described as a dull ache and frequently crossing the midline. The pain is deep, and treatment provided by clinicians is almost invariably conservative. As the International Association for the Study of Pain (IASP) does not recognize atypical facial pain as a diagnosis, the term *chronic facial pain* may be more useful. The IASP does recognize *atypical odontalgia,* a toothache without a demonstrable cause, which should probably be called *chronic odontalgia.*[9] There are also odd burning pains in the tongue and gums, referred to as *oral dysesthesia* or *burning mouth syndrome,* which describe disturbances in oral sensation unrelated to any pathology. Up to 70% of the general population have orofacial symptoms, but only 5% seek treatment, the majority of whom are women.[10]

The association between the various facial pain problems is not clear, but patients frequently complain of more than 1 symptom, and it may be that patients start with joint symptoms and progress to more generalized pain. Patients with pain in the TMJ are generally younger than those with more generalized pain. It is unclear whether there is any clinical value in distinguishing between these pain problems, as treatment is identical for all. Furthermore, facial pain is rarely an exclusive problem; the majority of patients complain of many other bodily symptoms, such as irritable bowel, backache, and headache.[10] Some patients have multiple sensitivities and dizziness. About 50% of patients with chronic facial pain also complain of chronic fatigue, and about 50% to 70% of pain patients suffer from sleep disturbance.[10] In general, those with long-standing pain can continue normal activities, despite the pain being a daily or near daily occurrence.[11]

Initiation and Maintenance

Many chronic facial pain patients specifically relate the onset of their symptoms to dental treatment itself. This is vital to recognize, since so many problems are related to excessive dental treatment. Other reported precipitating factors include infections, toxins, and life stress, such as

that associated with bereavement. However, once the pain is initiated, the patient may inadvertently exacerbate and thereby maintain the pain problem through his or her own actions. For example, some patients completely avoid movement of the jaw, which eventually results in muscular atrophy and greater joint stiffness. Others compulsively stretch and hyperextend the jaw numerous times each day, provoking local irritation. Frequent prodding and touching the painful areas of the face, teeth, or gums are also common in facial pain patients and are also likely to irritate already sensitive muscles and nerves. Underpinning these behaviors is the patient's mood state. High levels of anxiety, related to concerns about whether the pain might worsen, a possible undetected malignant cause for the pain, and so on, increase the perception of pain, as does depressed mood. Aside from the biochemical associations between pain and depression, depressive symptoms, such as loss of interest in daily activities and fatigue, are critical factors in developing and maintaining a preoccupation with physical symptoms. It is essential for the clinician to adopt an understanding of the whole patient to avoid harmful mechanical therapy.

Greene believes that we should aim to treat specific TMD diagnostic categories, regardless of individual etiologic factors. However, this assumes diagnostic categories to be inherently meaningful, which is currently unproven. Despite his advocation of behavioral management techniques to combat the stress of illness, he risks propagating a mechanistic approach to TMD in ignoring individual reactions to pain as a stressor.[12]

Central Pain Mechanism

Greene sees the future in basic science research. It is possible that several discrete stimuli initiate chronic facial pain by a common final pathway that involves the generation of a central pain state through the sensitization of second-order nociceptive neurons. Derbyshire et al[13] have shown increased activity in the cingulate cortex on positron emission tomographic scans of chronic facial pain patients, compared to patients with postextraction pain or pain-free patients. This is similar to the areas reported to show increased activity in fibromyalgia. Since the cingulate is the area concerned with attention, this may be the cerebral basis of somatization.

Chronic Symptoms

Chronic symptoms and syndromes pose a major challenge to medicine, as well as dentistry. They are common and frequently persistent, and are associated with significant distress, disability, and unnecessary expenditure of medical resources. In primary care in the UK, somatic symptoms and syndromes account for 20% of consultations. Among medical outpatients, somatic complaints accounted for 35% of new referrals in a UK study.[14] The prevalence of emotional distress and disorder in patients who attend hospital with unexplained syndromes (such as irritable bowel syndrome) is higher than in patients with comparable medical conditions (such as inflammatory bowel disease), and many such patients are severely disabled.[15]

Antidepressant drugs and psychologic treatments are helpful, and response to treatment is similar in all diagnostic groups. Wessely et al suggest that patients seek help from doctors for symptoms, and doctors diagnose diseases to explain them.[14] Greene would recognize this. Wessely and colleagues postulated that "the existence of specific somatic syndromes is largely an artefact of medical specialization. That is to say that the differentiation of specific syndromes reflects the tendency of specialists to focus on only those symptoms pertinent to their specialty, rather than any real differences between patients."[14p936] Atypical facial pain and temporomandibular pain are in the dental domain, but chronic facial pain should be seen as part of a whole body disorder.

Prognosis and Impact

Treatment is most likely to be effective when the patient's pain is of recent onset.[10] Successful treatment of facial pain of many years' duration is a much greater challenge. There is little understanding of prognosis in these patients, but there is growing evidence for psychologic distress as a consequence rather than a cause of pain,[15,16] and this distress is likely to contribute to the persistence of symptoms. Such an approach frees Greene from his concern regarding the terms "biopsychosocial" and "multifactorial." Some patients improve quickly with conservative management, including physical therapy, occlusal splints, and antidepressants; others respond to cognitive therapy, hypnosis, and other forms of treatment.[2] However, improvement is sustained only when an attempt is made to resolve psychologic problems. Greene

correctly asserts that it is not clear who responds to what treatment or, indeed, what problems actually need to be treated. Assessment of disability may lead to more precise treatment guidelines. Only recently has facial pain been examined in terms of disability. Facial pain patients report that pain and fatigue adversely affect their quality of life. Typical problems, such as difficulties in mouth opening, affect their capacity to eat in public and enjoy a full social life. Disability, in terms of impact on mood, speech, self-image, taste, and digestion, has been shown to predict a significant proportion of associated psychologic distress.[11] Educational programs are an important part of the care of chronic pain patients.

Conclusions

There is a huge overlap between the symptoms of temporomandibular disorders, chronic pain, and other chronic disorders such as fibromyalgia,[17] and patients rarely fit into rigid diagnostic categories. Greene wisely encourages further research, but states his faith in pathophysiology. While this may unearth the holy grail of etiology, such an endeavor must not be allowed to blight the clinical care of current sufferers, and as no single speciality receives the training required for the differential diagnosis and management of these pain disorders, there is a real need for a multidisciplinary examination of the condition.[8]

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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.

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