Changes in understanding of painful temporomandibular disorders: the history of a transformation

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Objective: The understanding the etiology of painful temporomandibular disorders (TMD) has evolved over the last eight decades. Evidence-based systematic research had questioned historical concepts and abandoned preconceived dogmas based purely on mechanically based etiologies, transforming TMD into a complex musculoskeletal chronic pain model. Unfortunately, many of these old ideas persist in undergraduate education and the dental community. Revisiting the historical development and the way the etiology of painful TMD has changed over the years may be helpful to understand the complexities of TMD as a group of chronic pain pathologies. **Method and materials:** A literature search using the MeSH terms: "temporomandibular joint disorders," "TMD," "etiology," "causality," "history," and "evolution" using Medline and Scopus databases was conducted aiming to answer the focused question: "In what ways has etiologic understanding of temporomandibular disorders evolved?" A narrative review was performed with the selected studies, highlighting significant contributions that have transformed TMD from a purely mechanical-based phenomenon into a chronic pain biopsychosocial disease model.
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Key words: chronic pain, etiology, myofascial pain, occlusion, pain, temporomandibular disorders, temporomandibular joint

Painful temporomandibular disorders (TMD) have significantly changed over the last 80 years. Evidence-based dentistry (EBD) has shifted our core understanding of TMD from a purely mechanically based etiology to a complex multifactorial chronic pain model. Many historically held concepts have been questioned or have become obsolete. However, many of these ideas persist as preconceived dogmas in undergraduate education and as part of the dental community at large. Unfortunately, the lack of understanding regarding the etiology of TMD may lead to errors in diagnosis and treatment and may result in a significant increase concerning healthcare cost and impact for persistent pain in patients with TMD.

Dr Charles S. Greene realized this problem back in 1982 and described the experience of patients with TMD: "Whatever happens to patients with painful TMD when seeking professional help depends exclusively on which floor the elevator stops." Sadly, this predicament still affects most patients with TMD and continues to be neglected in undergraduate dental curricula.

In order to understand the complexities of TMD as a group of chronic pain pathologies, it might be helpful to revisit their earlier historical development and the way the etiology of TMD has changed over the years. This historical narrative review aims to explore etiologic concepts of TMD and how they have evolved over many decades.

Method and materials

A literature search was conducted in June 2018 aiming to identify relevant articles regarding historical changes in the etiology of TMD. MeSH terms included: "temporomandibular joint disorders," "TMD," "etiology," "causality," "history," and "evolution" using Medline and Scopus databases to search for relevant papers. The lead author (NPS) performed an initial search in English. The search was then expanded by all the authors using

the Google Scholar database, in order to find potential additional papers using the following MeSH terms: "temporomandibular joint disorders/etiology" OR "temporomandibular joint disorders/history," AND "temporomandibular joint dysfunction syndrome/etiology" OR "temporomandibular joint dysfunction syndrome/history." References for the studies included in the review were manually examined in order to identify additional studies and analyze their potential eligibility.

A focused question was used to determine study eligibility: "In what ways has etiologic understanding of TMD evolved?" All the studies, book chapters, and expert opinions describing historically relevant etiologic explanations or theories of TMD were considered for inclusion. Studies that did not meet the eligibility criteria or those considered irrelevant from a historical perspective were excluded from the present review. All authors participated in the study selection process. Study selection involved screening every title and abstract of all selected articles followed by analysis of the full text. The studies included in this narrative review were selected by all the authors. Disagreements concerning the relevance of selected studies were resolved by discussion among the authors until a consensus was reached.

Early concepts in the etiology of TMD

Temporomandibular problems were first mentioned years before Costen's syndromic description. Prentiss was among the first researchers to describe TMD and atrophy of the temporomandibular joint (TMJ) after multiple tooth extractions.[1] Decades later, the first syndromic description that gained acceptance among the dental community came from the medical field.[2] Costen, an otorhinolaryngologist, reported, in a case series of 11 patients in the 1930s, that TMJ problems were responsible for otologic symptoms and also an important source of pain in the face and ear. He also stated that many of those patients improved after correcting some dental problems, such as dental malalignment or prosthetic malalignment, pronounced overbite, tooth loss, and loss of vertical dimension. Costen theorized that the occlusal factors mentioned above – especially overclosure of the mandible – were responsible for the auric symptoms due to impaction of the mandibular condyle against the glenoid fossa, thus compressing and irritating the tympanic plaque, the Eustachian tube, and the auriculotemporal nerve.[2] Almost 10 years later, Costen published a series of 500 cases naming the clinical features described in his first publication as "mandibular neuralgia" or "Costen's syndrome."[3,4]

Although Costen's postulates were discredited and proven anatomically implausible,[5-7] his claims found solid ground within the dental community and galvanized dental professionals into treating patients with TMD and orofacial pain.[8] Moreover, these ideas encouraged clinicians in the following decades to perform a variety of dental procedures aiming at resolving painful TMD and setting the theoretical foundations of mechanistic theories on the etiopathology of TMD.[9] As a result, some dental specialties have embraced concepts such as temporomandibular alignment, ideal occlusal schemes, muscular balance and harmony, condyle-glenoid fossa positioning, or achieving the correct vertical dimension as a therapeutic goal, and even prophylactic measures to treat or prevent TMD.[10]

Mechanically based etiologic theories of TMD

Orthodontics was one of the pioneering dental disciplines to embrace mechanistic TMD etiologic models.[11] In 1941, Thompson was one of the first authors to suggest that an adequate condyle-glenoid rest postural position was imperative to allow the mandible freedom of movement.[12] Thompson believed that malocclusions interfere with such correct condylar position and jaw closure, producing pain and discomfort.[13] In 1949, Moyers suggested that malocclusion was responsible for generating aberrant muscular patterns that could be corrected with orthodontic treatment.[14,15] Later, Ricketts described condylar morphologic changes in cephalometric laminography studies when the mandible was not in the rest position described by Thompson.[16] In a series of cases in 1956, Perry reported that mandibular misalignment and malocclusion were responsible for generating an electromyographic imbalance of the masticatory muscles, producing metabolic depletion and muscle spasms.[17,18]

Likewise, Jarabak supported the ideas of Thompson, Moyers, and Perry and published that patients with malocclusions and temporomandibular disturbances had higher electromyographic muscle masticatory activity compared to healthy individuals, assuming that occlusal interferences and inadequate inter-arch occlusal relationships were responsible for muscle spasms.[19,20]

Gnathologists also adopted these concepts in prosthodontics. McCollum and Stuart proposed that several occlusal gnathologic features formulated for edentulous patients were required, refined, and later applied to fixed prosthodontics.[21] In 1950, Stuart suggested that a coincidental position between

centric relation (CR) and centric maximum intercuspation (MI) was necessary to avoid tooth wear and joint pain in the natural dentition.[22,23]

In 1961, D'Amico suggested that canine guidance and coincidence between CR/MI was crucial in order to avoid periodontal and TMJ trauma.[24] That very same year, Ramfjord published two of the most iconic and significant studies in mechanistic theories of TMD. Based on experimental findings with Rhesus monkeys, Ramfjord selected 32 patients with TMD with signs and symptoms of what he believed to be severe bruxism. After an electromyography (EMG) assessment and determining the discrepancy between CR/MI in his patients, Ramfjord performed occlusal equilibrations for consecutive weeks and assessed EMG response after treatment. In his studies, Ramfjord found a decrease in EMG activity and concluded that occlusal equilibration was effective in treating and preventing bruxism, reestablishing muscular balance, and avoiding TMD.[25,26]

As gnathology evolved through the years, various occlusal features were theorized and included in gnathologic principles, leading to a construct of principles for an "organic" (ie, well-organized) occlusion.[27] Soon enough, these concepts that had initially been intended for oral rehabilitation led to analyzing every patient's anatomical and physiologic discrepancies according to gnathologic principles, and served to justify extreme bite rearrangement procedures.[28-33] Even today, gnathologic principles support dental occlusion philosophies and craniocervical alignment postural theories.[34] Over the years, systematic research has still failed to prove a consistent association between morphologic variables and TMD, which have probably more normal variations within the population than pathogenic maladaptive anatomical characteristics.[35,36]

Change of perspective

During the 1930s and 1940s, the etiology of TMD was mostly understood as a mechanical/occlusal-based problem. It was not until 1955, when Dr Laszlo L. Schwartz from Columbia University wrote about "temporomandibular joint pain and dysfunction syndrome," that etiopathology of TMD moved away from mechanics towards a biopsychosocial model.[37]

In his syndromic description, Schwartz reported that signs and symptoms of TMD often manifested in circumstances of emotional stress. He also identified several predisposing emotional factors (psychological, temperamental, and constitutional), contributing factors (malocclusions), precipitating factors (muscular imbalance), and aggravating factors (alarmism and previous trauma).[38,39]

Thus, the introduction of psychosocial factors as possible contributors to TMD etiology[40] was pivotal in shifting the focus of dental researchers away from occlusion into broader concepts such as psychophysiology, neuromuscular physiology, and TMJ biomechanics.

Transformation of painful TMD into a chronic pain model

The transformation of painful TMD into a chronic pain model was progressive and linked to the changes in the understanding of TMD etiology. The growing research interest on this field led to the elaboration of diagnostic taxonomies, the emergence of newer etiologic theories, and the development of instruments to assess signs and symptoms.

Weinmann and Sicher, in 1951, proposed one of the first etiology-based taxonomies, differentiating TMJ problems produced by vitamin deficiencies, endocrine disorders, and arthritis.[41] A few years later, Dr Welden E. Bell led the first attempt at devising a classification system without using syndromic descriptions. Bell divided TMD into six distinctive subgroups differentiating joint problems from masticatory muscle problems.[42]

During the 1960s, advances in craniocervical pain neurophysiology had a great impact on TMD, highlighting the neurophysiologic base of their etiologies.[43-45] Moreover, several controlled studies reported that placebo treatments and conservative approaches had similar outcomes to nonconservative treatments.[46,47]

Based on a series of clinical studies, Dr Daniel M. Laskin described the "pain-dysfunction syndrome" in 1969 after finding sound inconsistencies in mechanistic models.[48] Laskin claimed that the most significant source of temporomandibular symptoms and signs were found in the masticatory musculature. In general terms, the syndrome began as a functional problem, which was the consequence of psychophysiologic factors that manifested as tension, oral habits, or dental irritation. The most common result of these oral habits was muscle fatigue, which later initiated a muscle spasm.[48]

With the support of epidemiologic, radiologic, psychologic, physiologic, and biomechanical studies, Laskin hypothesized that, in time, muscle spasms turned into organic problems, such as myofascial pain syndrome, muscle contractures, occlusal disharmonies, and degenerative joint diseases. Several studies supported the role of psychosocial factors in the etiology of TMD, changing the focus of the scientific community from narrow mechanical concepts to a much broader multifactorial vision.[48,49]

In the 1970s and 1980s, the implementation of systematic evaluation systems, such as dysfunction and temporomandibular indices, was fundamental to potentiate systematic research and develop important epidemiologic and etiologic studies.[50,51] During this period, the idea that facial pain and TMD was understood as an umbrella term for different conditions started to gain acceptance in the scientific community.[52,53]

In 1980, the American Academy of Craniomandibular Disorders published its first position paper on etiology of TMD, which proposed the term "craniomandibular disorders" to describe an amalgamation of conditions that might contribute to facial and mandibular pain. It also stated that etiologic factors were multifactorial and complex. Further, the workshop classified etiology into precipitating, predisposing, or perpetuating risk factors.[54]

Two years later, the American Dental Association recognized the importance of establishing a rational, organized, unbiased approach to examining, diagnosing, and treating these disorders. The workshop also concluded that using broad, nonspecific categories such as "TMJ dysfunction" had to be discouraged and replaced with more specific classification systems, separating disorders of masticatory muscles from those disorders affecting the TMJ. Finally, these disorders were named TMD and organized similarly into the system proposed by Bell.[55]

Likewise, TMD started to gain recognition as a quintessential source of pain in the head and face in the 1986 Chronic Pain Classification of the International Association for the Study of Pain and the International Classification of Headache Disorders in 1988.[56,57]

In the 1990s, changes in the etiology of TMD were due to the appearance of two growing fields in research: neurobiology of the trigeminal nerve system and psychopathology.[58-62] Also, research on trigeminal pain perception and psychopathology steered this area towards the integration of physical illness and psychiatric diseases.[62,63]

After analyzing the publication of the Diagnostic and Statistical Manual for Mental Disorders, Dr Samuel Dworkin realized that diagnosis and etiology in TMD should use a similar descriptive model to aid clinical research and remove any subjectivity. Dworkin suggested the need to assess TMD, including physical manifestations using TMD subtypes and psychosocial profiles, based on the biopsychosocial of diseases proposed by Engel.[62,64] This premise was essential in the development of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) and molded its underlying principles, which are: painful TMD as a biopsychosocial pain model; the inclusion of epidemiologic data; the use of a dual axis system for physical and psychosocial profiles; and accurate operational specifications and protocols for standardization.[65]

Since the introduction of RDC/TMD, scientific research in TMD has greatly increased and at the time of writing this article, RDC/TMD has been cited over 2,250 times and has more than 6,000 hits on Google Scholar, making it one of the most widely cited publications in the dental literature. By the late 1990s, the majority of the peer-reviewed scientific research related to TMD used RDC/TMD-based protocols.[66] Concurrently, the creation of postgraduate university programs and the increasing number of books, guidelines, seminars, lectures, and articles dealing with the topic discredited many mechanistic theories of the past.

Painful TMD as a chronic musculoskeletal pain model

In the early 2000s, it became clear that occlusal and other mechanical or structural etiologies had played a minor or nonexisting role in the etiology of TMD. Despite this issue, reluctance to accept this reality still lingers on in dental practice to this day.[9]

On the other hand, scientists and evidence-based dentistry have directed their attention towards two etiologic models that are in fact complementary and consistent with current pain models: biopsychosocial and multifactorial. The biopsychosocial pain model combines three different words: "bio" refers to a biologic problem; "psycho" refers to pain conduct, suffering, and behavioral changes; and "social" refers to the social framework of the patient.[67] The multifactorial model encompasses a mix of intrinsic and extrinsic factors that interact and contribute to the etiology of TMD.[68]

In a critical review on TMD etiology in 2000, Dr Charles Greene considered the term "idiopathic" as a more accurate way to describe TMD etiology.[10] Nevertheless, Greene's proposal still matches several chronic orofacial pain disorders. In the last two decades, several efforts have been made to improve the understanding of painful TMD and their relationships. The increasingly RDC/TMD-based research and interest in the field has led to a series of international consortiums and workgroups that have focused on assessing the validity and reliability of diagnoses of TMD and improving their taxonomy for research and clinical purposes.[69-74]

Alongside these efforts, an extensive, National Institute of Dental and Craniofacial Research (NIDCR)-funded, multicenter prospective study (Orofacial Pain: Prospective Evaluation and Risk Assessment [OPPERA]) investigated the underlying factors in the development of first-onset TMD and their transition to chronic, painful conditions. Collectively, the OPPERA group has

published more than 35 studies investigating over 2,924 genetic variants representing 358 genes and more than 200 phenotypic risk factors, including sociodemographics, general health status, clinical orofacial characteristics, pain sensitivity, cardiac autonomic activity, and psychological characteristics.[75] The OPPERA study reaffirmed that TMD have an overwhelmingly complex and multifactorial etiology that consistently fits with the biopsychosocial aspects of illness. Also, the study made important contributions in identifying putative risk factors for first-onset TMD and profiling clinically applicable clusters based on individual risk characterization.[76,77]

Indeed, these attempts have guided new pathways to develop newer taxonomies (eg, Diagnostic Criteria for Temporomandibular Disorders [DC/TMD] and the Expanded DC/TMD Taxonomy)[78,79] and led to the inclusion of TMD in international chronic pain classifications such as the World Health Organization (WHO) International Classification of Diseases (ICD-11)[80,81] as well as the taxonomy of the Analgesic, Anesthetic, and Addiction Clinical Trial Translations, Innovations, Opportunities, and Networks and the American Pain Society (ACTTION-APS).[82]

Co-morbid pain conditions and TMD

Studying the etiology of TMD has uncovered the heterogeneity of these disorders, making it decreasingly plausible to explain the complexity of the disease by simply establishing single etiologic factors.[83] Therefore, it is likely that the etiology of painful TMD depends on complex interactions of multiple environmental, phenotypic, and genetic variables. Hence, it is not surprising that the underlying mechanisms of persistent painful TMD are still poorly understood and may rely upon future prospective observational studies to report on how these factors interact.[84]

Epidemiologic studies have shown that painful TMD share some common features with other chronic pain conditions such as chronic migraine, tension-type headache, lower back pain, and fibromyalgia.[85] Similar to other chronic pain conditions, recent research has also highlighted that persistent orofacial pain patients experienced significant financial impact and had greater healthcare utilization than healthy individuals.[86,87]

By establishing putative risk factors on painful TMD, the OPPERA study is undoubtedly one of the most comprehensive etiologic prospective studies performed to date. Based on a heuristic model of disease, the OPPERA study measured eight different dimensions: sociodemographic and sociocultural characteristics, general health status, orofacial characteristics, psychologic profile, pain perception, autonomic function, and genetic susceptibility.[76] From these dimensions, co-morbid health conditions, particularly nonpainful ones, were the most significant putative risk factor contributing to TMD. Also, their results indicated that poor general health is a major predictive factor in the incidence of painful TMD. Another intriguing finding was the relationship between painful TMD, sleep quality, and sleep breathing disorders. Patients who developed TMD showed progressive deterioration of their sleep quality until onset of TMD. Cardinal symptoms of obstructive sleep apnea, such as loud snoring, daytime sleepiness, witnessed apnea events, and hypertension were also predictive factors in the onset of TMD. Nonspecific orofacial symptoms (TMJ noises), as well as density and frequency of oral behaviors, were significant predictors of incidence of TMD.[77]

In order to create a clinically meaningful model to assess successfully which individuals are predisposed to develop painful TMD, researchers have developed a supervised cluster methodology to identify risk factors and health profiles and their possible outcomes. Three clusters ("adaptive," "pain-sensitivity," and "global symptoms") were identified using a centroid model with four variables. Two of the three subgroups showed a strong risk association in developing first-onset TMD, had increased odds of developing chronic TMD, and reported more severe and painful TMD. The "adaptive" cluster represented the highest proportions of controls, whereas "pain sensitivity" (higher evoked pain pressure sensitivity) and "global symptoms" represented the majority of TMD cases. The TMD cases of the "global symptoms" cluster showed an increased risk and severity of pain as well as more physical and psychologic dysfunction. Also, the "global symptoms" cluster group presented a higher prevalence of co-morbid pain conditions than the other two groups.[88]

Conclusions

It is unquestionable that the etiologic understanding of TMD and their complexities have undergone important conceptual changes in the last 80 years. Awareness of these changes has not fully extended into the whole dental community. It is therefore paramount to revisit how these concepts have evolved and changed. In this review, the aim was to highlight the important contributions that have transformed TMD from a purely mechanical-based phenomenon into a chronic pain biopsychosocial disease model.

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