Medical College of Virginia
Virginia Commonwealth University

June 16, 1986

Doc. 268

Mr. Carl G. McMahon
Attorney and Counsellor at Law
410 Leader Building
East Sixth and Superior Avenue
Cleveland, Ohio 44114-1958

Dear Mr. McMahon,

I am replying to your letter of June 11, 1986 requesting my opinion about the validity of Dr. Kenneth R. Callahan's Dental report on Patricia Nelson as well as about whether her TMJ problem were caused by the automobile accident in which she was involved in April, 1983.

In respect to Dr. Callahan's report, my comments relate to two aspects: his evaluation of the patient and his discussion of temporomandibular joint disease. Regarding the latter, I am not in agreement with his statement that "current state-of-the-art thinking about TMJ disease is in overwhelming consensus that the primary cause of TMJ symptoms has to due (sic) with dental problems." Most patients with so-called TMJ problems actually have a muscular problem (Myofascial pain-dysfunction (MPD syndrome) and the condition is stress related and not caused by faulty occlusion, malocclusion, or excessive overbite or overjet. Patients with a true TMJ problem (degenerative joint disease) usually have this condition from longstanding MPD syndrome or trauma. Therefore I also cannot agree with his statement that "there are" no experts....who list-external trauma as being a primary causative factor in longstanding TM joint disease." Although I could not find the quotation from "Scientific Foundations for Dentistry" to which he refers, I did find a quotation on page 613 of the 1976 edition that states "It thus seems that purely dental factors, at least in the young and middle-aged groups, may be of comparatively little importance in the etiology of degenerative joint disease of the mandibular condyle."

In respect to the quotes attributable to me, Dr. Callahan has substituted the words "TMJ syndrome" for "MPD syndrome". The entire quotation was taken from a chapter dealing with myofascial pain-dysfuntion (MPD) syndrome, a muscle spasm problem and not a joint problem. He also adds "(such as an auto accident)" after "since spasm of traumatic origin" where I was referring to being a cause of degenerative changes in the joint and therefore do not "simply dismiss(es) external trauma out of hand".

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In respect to my opinion about Dr. Callahan's conclusions regarding the cause of Ms. Nelson's TMJ problem, I must first indicate that it is based on the letter from him as well as those from Drs. Stern, Langa, Winer, Curran, Fowler, Schneider, and Linsey that were furnished to me. Although I agree with all of these doctors, including Dr. Callahan, that she now has a TMJ problem, the question is whether the condition was pre-existing. Based on Dr. Langa's letter it would appear that she did have some prior involvement of her jaw joint. However, it is also very possible that the condition was made worse by the accident and therefore has a relationship to it. Only persons like Dr. Langa, Dr. Stern, or Dr. Linsey would be in a position to provide an accurate opinion on this matter since they all saw her prior to the accident.

I hope this information will be helpful to you in resolving this issue. Please fee; free to contact me if you wish ti, discuss the matter further.

Sincerely yours,

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DML/trf

SARNAT • LASKIN

TEMPOROMANDIBULAR JOINT

THIRD EDITION

THOMAS

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The scope and depth of this text have been greatly expanded for this Third Edition. It is almost an entirely new book. However, the high standards set by previous editions prevail, and the basic science approach to clinical practice continues to dominate the overall perspective.

With a Foreword by

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Third Edition

The Temporomandibular Joint

A Biological Basis for Clinical Practice

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CHAPTER 14

MYOFASCIAL PAIN-DYSFUNCTION SYNDROME: ETIOLOGY*

DANIEL M. LASKIN, D.D.S., M.S.

I. INTRODUCTION

ALTHOUGH THE CLINICAL symptoms associated with jaw dysfunction have long been recognized, only in the past twenty years has research shown that what previously was referred to by such names as *Costen's* syndrome or *temporomandibular joint* (TMJ) syndrome is neither a primary joint disorder, nor is it generally caused by occlusal abnormalities. Rather, studies have shown that masticatory muscle spasm is the primary factor responsible for the signs and symptoms and that psychological *stress* is the major etiologic agent. As a result, the term *myofascial pain-dysfunction* (MPD) syndrome is now used to describe the disorder more accurately, and the psychophysiologic theory has been proposed to explain its pathogenesis.[34] The evolution of this theory is described in Chapter 13.

* See Chapters 5, 6, 13, 15, 16, 18 and 19.

II. DIAGNOSIS OF THE MYOFASCIAL PAIN-DYSFUNCTION SYNDROME

Because of the confusing terminology used to describe MPD syndrome in the past and the differences of opinion regarding the principal signs and symptoms, it is important before discussing etiology to consider the essential findings that place a patient in the category of MPD syndrome. Pain of unilateral origin is the most common symptom.[26] This pain usually is described by the patient as a dull ache in the ear or preauricular region that frequently radiates to the temporal region, the angle of the mandible, or the lateral cervical or occipital regions. The pain can be relatively constant, but it more often is worse upon arising in the morning or is relatively mild or absent in the morning but gradually worsens as the day progresses. The pain is frequently exacerbated by use of the mandible.

Masticatory muscle tenderness is the next most common finding.[26] Although usually not reported by the patient, this symptom can be elicited easily by the examiner. The most frequent areas of tenderness are found over the neck of *the* condyloid process, in the area distal and superior to the mandibular tuberosity, in the anterior temporal region, and over the mandibular angle and temporal crest. These sites of tenderness are presumed to be areas of spasm in the masticatory muscles.

A clicking or popping noise in the TMJ is another common symptom of MPD syndrome. However, patients with only this symptom initially should not be included in this category. Even though it is known that many patients with MPD syndrome report clicking or popping as their earliest symptom,[26] not all persons with this symptom develop MPD syndrome. Therefore, to establish the diagnosis, there must also be accompanying symptoms of pain, muscle tenderness, or both. It is also important to distinguish clicking and popping sounds, which in MPD syndrome are generally a result of asynchronous muscle action, from the grating or crepitant sounds that are due to actual joint pathosis.

Limitation of mandibular movement is the fourth cardinal symptom of the MPD syndrome. It may manifest itself either by inability to open the mouth as wide as usual, deviation of the mandible on opening, or both. Patients with limitation of opening rarely have clicking, although their past history frequently reveals that it had been present at one time.[26]

Along with having one or more of the four cardinal symptoms of pain, muscle tenderness, limitation of mouth opening and clicking, patients with MPD syndrome usually have an absence of clinical, radiographic, or biochemical evidence of organic changes in the TMJ. These negative characteristics are significant in establishing the diagnosis, since they indicate that the primary site of the problem is not in the articular structures. This distinction forms the essential basis for understanding the pathogenesis of the MPD syndrome.

Although MPD syndrome generally begins as a functional muscular disorder, it ultimately can lead *to* degenerative arthritis in the TMJ. The past history of the disorder is often helpful in determining whether the arthritic changes are primary or secondary.

There are a number of other conditions besides MPD syndrome that can produce similar symptoms of facial pain and jaw dysfunction. Some are nonarticular problems that merely mimic MPD syndrome; others are true pathologic conditions of the TMJ. All of these must be considered and eliminated before a final diagnosis of MPD syndrome is made (*see* Chaps. 18 and 19).

III. ETIOLOGY OF THE MYOFASCIAL PAIN-DYSFUNCTION SYNDROME: THE PSYCHOPHYSIOLOGIC THEORY

In 1955, Schwartz[52] reported that he was able *to* delineate from the large group of patients with supposed "temporomandibular joint (TMJ) syndrome" a more definitive group *of* individuals with painful, limited mandibular movement due to spasm in the muscles of mastication. He termed this condition *temporomandibular joint pain-dysfunction syndrome*. The subsequent studies of Schwartz and his co-workers[53] emphasized the psychological characterisrics of the patients and represented the first major shift away from the narrow mechanical concept of an occlusal etiology. These studies had a profound influence on the thinking of many individuals interested in TMJ problems and led *to* new areas of basic and clinical research. As a result of such investigations at the Temporomandibular Joint and Facial Pain Research Center of the University of Illinois, the psychophysiologic theory for explaining the etiology of MPD syn-

drome was introduced in 1969.[34] While differing somewhat from the concept proposed by Schwartz and his co-workers,[53] the two theories are not mutually exclusive.

As suggested by Schwartz, masticatory muscle spasm is considered the primary factor responsible for the signs and symptom of MPD syndrome (Fig. 14-1). This muscle spasm can be initiated in any of four ways: trauma, muscular overextension, muscular overcontraction, or muscular fatigue. Since spasm of traumatic origin is not difficult to diagnose and is usually a self-limiting episode of short duration, it is not considered in this discussion. Examples of some of the conditions that can cause overextension and spasm of the various masticatory muscles are overly contoured dental restorations or fixed and removable prostheses that encroach excessively upon the intermaxillary space. Overcontraction, on the other hand, can result from bilateral loss of posterior teeth or from the "settling" of partial dentures replacing them. Similarly, it can occur from excessive alveolar bone resorption in patients wearing complete dentures.

Although these kinds of adverse mechanical factors can cause MPD syndrome, most patients do not acquire the problem in this way. Rather, in the majority of cases the cause appears to be muscular fatigue produced by hyperactivity. This hyperactivity usually takes the form of chronic parafunctional habits, such as clenching or grinding the teeth. These habits sometimes can be initiated by a dental irritation, such as an improperly occluding restoration, an overhanging margin, or a tooth with periodontitis, Generally, however, they are an involuntary tension-relieving mechanism in response to psychological stress.

Stress can also generate masticatory muscle hyperactivity directly without the clinical manifestations of clenching or grinding.", [18, S, 44] In these instances, such actions as excessive gum chewing, prolonged opening of the mouth, or chewing hard foods can add to the preexisting muscle hyperactivity and fatigue and thereby trigger the syndrome. This accounts for most of the patients who deny pernicious oral habits and relate the onset of their problem to a specific episode.

Once spasm develops in the muscles of mastication, regardless of whether it was caused by fatigue, overcontraction, or overextension, the patient has pain and limitation of mandibular movement, i.e. MPD syndrome. At this stage, it is still a functional disorder. However, if the condition persists, it can ultimately lead to organic changes in the dentition, muscles and TMJ (Fig. 11-1).

Unilateral spasm of one or more of the muscles of mastication, particularly the lateral pterygoid, can produce a slight shift in jaw position so that the teeth do not occlude properly. This has been referred to as

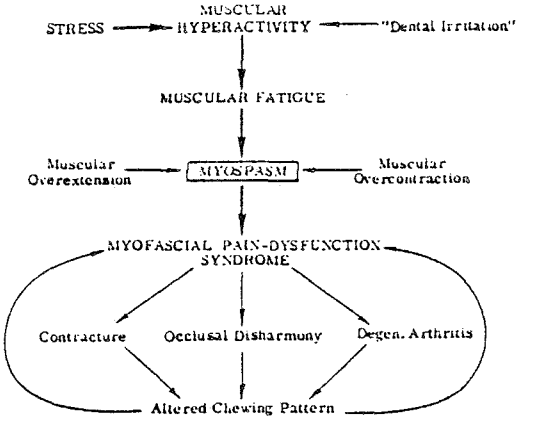


Figure 14-1. Etiology of the myofascial pain-dysfunction syndrome. Although three means of entry into the syndrome are shown, the *broader arrows* indicate the most common path. The mechanism whereby stress leads to myospasm is termed the *psychophysiologic theory* of MPD syndrome. (Modified from D. M. Laskin, Etiology of the pain-dysfunction syndrome. *JADA, 79:*147-153, 1969.)

terial invasion of the synovium with purulent joint fluid showing microorganisms both on smear and culture. These infections may be the result of either direct invasion by bacteria as in otitis media (Fig. 20-5) and certain kinds of trauma, or they niay be the result of bacteremia from a systemic infection or a local sire resulting in pyogenic joint infection (Fig. 20-7j. The latter rarely affects the TMJs, although it has been reported, especially in association with gonococcal infections. Septic arthritis of the TMJ lends itself to specific antibiotic therapy.

The incidence of gonorrhea in the United States is over 300 per 100,000 population; 5 to 10 percent of these patients may develop arthritis. It is the most common type of infectious arthritis at the present time. Fourteen percent of the patients with gonococcal arthritis develop TMJ involvement.[1,5] This must be distinguished from other types of acute inflammatory TMJ arthritis, including rheumatoid, the rheumatoid variants, and that resulting from gout, trauma, and, of course, other infections.

Gonococcal arthritis is a most important diagnosis to consider, since early recognition of this disorder can prevent irreparable damage of the joint. The diagnostic clues are a high index of suspicion, a history of exposure to gonorrhea, and frequently the clinical features of arthritis affecting the other joints of the body. The definitive diagnosis is made by aspisation of fluid either from the TMJ or from other affected joints and the demonstration of gram-negative diplococci within the white blood cells, and the subsequent culture of *Neisseria gonorrhea* on appropriate culture media, such as chocolate agar.

Chronic infections of the TMJ, including tuberculous and fungal arthritis, have been rare. Today, however, patients with chronic diseases, such as diabetes mellitus, cancer, and collagen vascular diseases, live longer. Many of them are receiving immunosuppressive or corticosteroid therapy. This population of compromised hosts are at great risk for the development of opportunistic infections, including chronic granulomatous disorders. Thus, there must be renewed awareness of the possible development of chronic tuberculous or fungal arthritis of the TMJ. Diagnosis in these instances is made not only by appropriate history, physical examination, and blood and radiographic studies, but also by biopsy of the synovium.

C. Degenerative (Osteoarthritis)

Degenerative joint disease (DJD) affects various joints (Figs. 13-71 and 17-4). It is the *most* common disease of the TMJ and usually occurs after the fourth decade, but it may occur at any age secondary to trauma.[12] As is true in other joints, this condition is irequently asymptomatic. When symptoms do occur, they usually begin with pain in the TMJ often described by the patient as an earache. Sometimes the pain resembles a toothache in the maxillary molar region. The pain is usually unilateral. When bilateral, it is seldom equally severe. Pain is worse during mandibular function, such as during chewing or yawning, or at the extreme range of motion.

Physical examination reveals tenderness of the mandibular condyle, particularly when this structure is palpated through the external auditory meatus. There is rarely evidrnce of effusion, but frequently there is considerable crepitus. Radiographic examination may reveal osteophyte formation on the anterosuperior aspect of the mandibular condyle (Fig. 12-21). Secondary symptoms from spasms affecting the muscles of mastication and neck muscles frequently accompany DJD. Indeed the patient may be more troubled by pain in the muscles than by the actual pain in the joint (*see* Chap. 14).

D. Traumatic

The term *traumatic arthritis* is best used

52. DEGENERATIVE DISEASE OF THE MANDIBULAR JOINT

P. A. TOLLER AND L. E. GLYNN

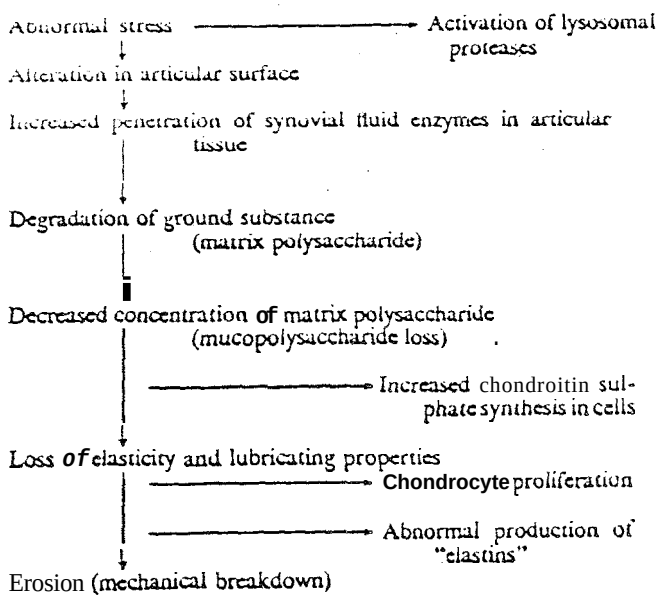
While rheumatoid arthritis is primarily a disease of synovial tissue, osteoarthrosis is considered to be a disease of articular cartilage. In recent years it has generally been agreed that the disease is of a degenerative nature, but dispuce continues with regard to the precise cause. A diagnostically important, and scientifically perplexing feature of degenerative disease of the mandibular condyle is that both its clinical and its histopathological characteristics differ from those seen in other diarthrodial joints.

CHARACTERISTICS OF MANDIBULAR OSTEOARTHROSIS

A recent clinical survey (Toller, 1973) was based on a series of 150 cases derived from a study of about 2,000 cases of temporomandibular joint lesions seen over the past 20 years, and this suggested that osteoarthrotic cases constitute about 8 per Cent of all the temporomandibular joint lesions which have been studied in one clinic during this period. Salient clinical findings from this study are summarized in Tables 1 and 2 and fig. 1. None of these patients had an abnormally raised sedimentation rate, and none was seropositive for rheumatoid factor. Nineteen cases of confirmed rheumatoid arthritis affecting the temporomandibular joints were found among the whole series of joint cases during this period, that is, a 1 per cent incidence of rheumatoid arthritis in this clinic compared with 8 per a n t incidence of degenerative joint disease over the whole period. This is reasonably similar to a series of 400 temporomandibular joint cases which included 14 per cent arthrosis deformans from ail causes, in Holland.

No case of bilateral active disease was seen, but in six cases the painful and degenerative disease subsequently started in the condyle opposite to the one in which the disease process had been seen to resolve and become symptomless. Ten other cases were noted where healed lesions were apparently present in the opposite condyle. Clinical polyarthritis was not manifest in any of this series, but complete clinical examinations were not carried out on all patients, and data regarding the occurrence of osteoarthrotic lesions in other joints were not recorded.

TABLE 1



Dental State

Dental state was recorded only as good, fair, or badly kept teeth, in addition to adequately or inadequately supported occlusion, the latter when three or more functional molars were missing or improperly restored, or where the freeway space in full denture cases exceeded 5 mm. The general standard of dental health and care among these patients was fairly high and only about one in three was judged to have inadequately restored dentitions. Although impaired dental efficiency has been said to bear a relationship to the occurrence of the pain-dysfunction syndrome, no such relationship was obvious in this series of osteoarthrosis patients. Striking clinical improvement did nut follow appropriate attention to the dental sate. However, about one-half had a history of pain-dysfunction systems ut some previous time in the affected joint and this suggests a relationship between the occurrence of untreated