Review of temporomandibular joint pathology. Part I: Classification, epidemiology and risk factors

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ABSTRACT

Pathology of the temporomandibular joint (TMJ) affects an important part of the population, though it is not viewed as a public health problem. Between 3-7% of the population seeks treatment for pain and dysfunction of the ATM or related structures. The literature reports great variability in the prevalence of the clinical symptoms (6-93%) and signs (0-93%), probably as a result of the different clinical criteria used. In imaging studies it is common to observe alterations that have no clinical expression of any kind. Radiographic changes corresponding to osteoarthrosis are observed in 14-44% of the population. Age is a risk factor, though with some particularities. In elderly patients there is an increased prevalence of clinical and radiological signs, though also a lesser prevalence of symptoms and of treatment demands than in younger adults. Approximately 7% of the population between 12 and 18 years of age is diagnosed with mandibular pain-dysfunction. Temporomandibular dysfunction (TMD) is more frequent in females. No clear relationship has been established between occlusal alterations and TMJ disease. Only disharmony between centric relation and maximum intercuspidation, and unilateral crossbite, have demonstrated a certain TMJ disease-predictive potential. Both local and systemic hyperlaxity has been postulated as a possible cause of TMD. Parafunctional habits and bruxism are considered risk factors of TMD with odds ratios (ORs) of up to 4.8. Psychophysiological theory holds stress as a determinant factor in myofascial pain. Genetic factors and orthodontic treatment have not been shown to cause TMD.

Key words: *TMJ, temporomandibular joint, TMD, temporomandibular dysfunction, epidemiology, risk factors.*

RESUMEN

La patología de la articulación temporomandibular (ATM) afecta a un colectivo importante de población aunque no se considere un problema de salud pública. Entre el 3 y el 7% de la población busca tratamiento a causa del dolor y la disfunción de sus ATMs o estructuras anexas. Los estudios encuentran una extraordinaria variabilidad en cuanto a prevalencia de síntomas (6-93%) y en cuanto a signos clínicos (0-93%), variación que está probablemente relacionada con los diferentes criterios clínicos utilizados. En los estudios de imagen es frecuente el hallazgo de signos sin que estos se traduzcan en sintomatología clínica alguna. Se observan cambios radiográficos de osteoartrosis entre el 14 y el 44% de la población. La edad constituye un factor de riesgo aunque con matices. En pacientes ancianos hay mayor prevalencia de signos clínicos y radiográficos, pero menor prevalencia de síntomas y de demanda de tratamiento que en pacientes de edad adulta. Alrededor del 7% de la población entre 12 y 18 años es diagnosticada de dolor-disfunción mandibular.

La DTM (disfunción temporomandibular), es mas frecuente en el sexo femenino. No se ha podido establecer relación inequívoca entre alteraciones de oclusión y patología de la ATM. Únicamente disarmonías entre relación céntrica y màxima intercuspìdación, y mordida cruzada unilateral han mostrado un cierto poder predictivo de patología de la ATM. La hiperlaxitud, tanto local como sistémica se ha postulado como posible causa de DTM. Los hábitos parafuncionales y el bruxismo se consideran factores de riesgo de DTM con odds ratio de hasta 4,8. El estrés es considerado por la teoría psicofisiológica como el factor determinante del dolor miofascial. Factores genéticos y tratamiento ortodóncico no se han mostrado como causantes de DTM.

Palabras clave: *ATM, articulación temporomandibular, DTM, disfunción temporomandibular, epidemiología, factores de riesgo.*

INTRODUCTION

Texts on pathology of the temporomandibular joint (TMJ) tend to consider the work of James Bray Costen as the reference point from which TMJ disease, and particularly its relationship to the dental apparatus, reached universal recognition – introducing the treatment of such problems in the professional setting of dentists. In 1934, this ear, nose and throat specialist described the process which is still known by some as the "Costen syndrome". He for the first time related the symptoms and signs of a qualitative nature (hearing loss, plugged ear sensation, vertigo, headache and trismus) to alterations in bite – specifically to vertical overocclusion and the loss of posterior dental support.

Prior to the mentioned work of this author, there already were many references in the literature to TMJ pathology, and some of them even pointed to the possible relationship between dental alterations and TMJ problems by suggesting that certain cranial, facial, hearing and mandibular symptoms could be a consequence of atrophy of the meniscus, of the skull and of the glenoid cavity, and that these processes would take place following loss of the posterior teeth.

In the mid-1950s one of the central references of the theories interpreting TMJ pathology came under questioning: its relationship to occlusion. In effect, it was postulated that emotional tension constitutes a primary etiological factor - an idea that constituted a radical change from an "ideal structure" concept to a more physiological concept based on joint biomechanics and muscle physiology. Logically, the approach to treatment also underwent radical change, with the suggestion of medical management for TMJ problems. In this sense, Laskin in 1969 suggested that muscle spasm and fatigue produced by chronic oral habits are responsible for the symptoms of mandibular pain-dysfunction. This paved the way to the idea of a multifactorial nature of TMJ disorders - a concept that remains fully applicable today.

A parallel and sometimes confronting change was also taking place, based on the newly developed imaging techniques. Although for a long time alterations in the position of the joint meniscus had been suspected, it was not until introduction of arthrography, and posteriorly of magnetic resonance imaging (MRI), when the possibility of internal joint derangement was seriously considered as the primary cause of the observed signs and symptoms. From this perspective, it was suggested that an ideal intraarticular structural relationship is needed, which in treatment terms led to attempts to reposition the structures in their theoretical ideal location and – ultimately – to substitute or eliminate the altered structures.

Recent advances in neurophysiology have introduced the concept of central nervous system (CNS) "plasticity" (neuroplasticity) and behavioral plasticity, which would account for the persistence of pain despite disappearance of the initial lesion. Neuroplasticity refers to reorganization of the nervous system based on a mechanism that influences synaptic efficacy and connectivity to all levels of the brain and CNS.

Turk and Rudy (1) established the similarity between the manifestations of chronic pain of the TMJ and other forms of chronic pain, stressing the importance of pain management from the perspective of CNS plasticity, and from a psychosocial and behavioral viewpoint.

This interpretation of TMJ pathology has been supported by longitudinal studies which have concluded that most of the disorders follow a natural course independently of treatment, and that there are structural alterations with respect to the purported "ideal anatomy" in approximately 30 % of all subjects.

At present, it is emphasized that the role of the clinician should be to provide pain management and patient support, including self-care measures, and avoiding treatment concepts that center on recovering purported ideal anatomical structures.

CLASSIFICATION OF THE DISEASES

In 1972, Farrar proposed a classification that contemplates eight dimensions within the global concept of dysfunction: hyperactivity of the masticatory muscles, capsulitis and synovitis, rupture or distension of the capsular ligaments, anterior disc displacement, muscle incoordination, and reduction of the mandibular movement range secondary to degenerative joint disease. The system has some deficiencies, however, such as the fact that painful muscle disorders are obviated entirely.

In 1980, Block proposed a classification based on neurological and orthopedic models of pain and dysfunction. Its main contribution is classification from a strictly medical perspective (fundamentally neurological and rheumatological), and the establishment of a clinical parallelism between myofascial pain-dysfunction and the observations in other parts of the body.

In 1986, Welden E. Bell developed a classification based on an orthopedic-mechanical model. The system differentiates the following major categories of temporomandibular disorders (TMD): masticatory pain, restriction of mandibular movements, joint interference during mandibular movements, and acute malocclusion. The classification identifies the following muscular processes: myositis, muscle spasm, myofascial pain, late-onset muscle irritation and protective co-contraction or protective stiffness. This author and his disciple, Jeffrey P. Okeson, have been and remain an obligate reference in TMJ pathology.

In 1990, the American Academy of Craniomandibular Disorders (AACD) proposed a taxonomic system integrated within the classification project of the International Headache Society (IHS). Category 11 of this classification corresponds to the taxonomic proposal of the AACD. The principal contributions on one hand comprise the distinction between two major categories - one for joint disorders and the other for muscle disorders - and on the other hand the possibility of establishing multiple diagnoses.

The classification developed by Edmond L. Truelove et al., known as the Clinical Diagnostic Criteria for Temporomandibular Disorders, for the first time contemplates defined diagnostic criteria for each clinical category. The classification system moreover allows for multiple diagnoses. It distinguishes between muscle alterations (myalgia and myofascial pain), internal joint alterations (disc displacement with or without reduction, capsulitis/synovitis and disc perforation), and degenerative disorders. This classification, in our opinion, is an excellent aid for the management of temporomandibular disorders.

Taking this classification as reference, Samuel Dworkin and Linda LeResche (2) proposed a new system known as the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD), with the aim of establishing standardized criteria for research, based on the available knowledge on TMJ pathology (Table 1). The diagnostic criteria are proposed for both clinical research and for epidemiological work. The objective of the authors was to maximize the reliability and minimize the variability of the examination methods and clinical judgment. The system comprises two classification axes. The first axis (clinical aspects of temporomandibular disorders) contemplates three groups: 1.- muscle diagnosis; 2.- disc displacement; and 3.- arthralgia, arthritis and arthrosis. The second axis in turn contemplates disabilities related to pain and the psychological condition of the patient. The classification criteria include:

• Intensity of pain and degree of disability (according to the severity of chronic pain grading scale).

• Depression (according to the SCL-90R; depression and vegetative symptoms scale).

• Limitations related to mandibular function.

This system is a relevant contribution, since for the first time psychological factors are included in the diagnosis, and are evaluated by means of reliable and reproducible instruments.

Table 1. Classification of temporomandibular joint disorders. Axis I. (Dworkin and LeResche, 1992).

Group I: Muscle disorders
I.a. Myofascial pain
I.b. Myofascial pain with limitations in aperture
Group II: Disc displacement
II.a. Disc displacement with reduction
II.b. Disc displacement without reduction and no limitations in aperture
II.c. Disc displacement without reduction and with limitations in aperture
Group III: Arthralgia, arthritis, arthrosis
III.a. Arthralgia
III.b. Osteoarthritis of the TMJ
III.c. Osteoarthrosis of the TMJ

Many studies have been made to validate the RDC/TMD. In our setting, Bermejo in 1995 (3) proposed a classification based on the "temporomandibular joint complex" – a concept that postulates the existence of two clearly differentiated joints: meniscocondylar and temporomeniscal. Two large diagnostic groups are established. The first comprises alterations of the masticatory muscles, while the second corresponds to disorders of the temporomandibular joint complex. Both include functional disorders, traumatisms, inflammatory disorders, degenerative processes and hereditary and developmental alterations. In the second group, each of these alterations can affect both the meniscocondylar joint and the temporomeniscal joint.

EPIDEMIOLOGY

In the National Oral Health Survey conducted in Spain in 1994 (4), in accordance with the criteria for epidemiological studies on oral health auspiced by the World Health Organization (WHO), it was seen that at 12 years of age 6.3% of the population presented clicks – a figure that increased to 9.4% in those aged 15 years, 14.70% in the 35-44 years age range, and 23% in the 65-74 years age group. Limitation of oral aperture was seen to affect 2.2% at 12 years of age, 4.5% in the 35-44 years interval, and 3.5% in the 65-74 years age group.

Pain in turn affected 0.2% of the population aged 15 years, 3.4% of those in the 35-44 years age group, and 1.3% of the subjects aged 65-74 years.

In the following survey carried out at national level in the year 2000 (5), it was seen that 17.6% of the population aged 35-44 years presented clicks, while 1.8% suffered pain in response to palpation, and 1.8% had limited mobility. Symptoms were detected in 10.8% of the population. In the 65-74 years group, clicks were present in 15.5% of subjects, pain in response to palpation in 2.5%, and reduced mobility in 2.9%. Symptoms were present in 11.2% of the

population.
In the studies of prevalence of the disease, the variability is extreme – ranging from 6% to 93% when based on patient-contributed information, and from 0% to 93% when based on clinical evaluation (6). It is very unlikely that such discrepancies are due to variations in the populations studied. A much more plausible explanation is to be found in the clinical criteria used in the mentioned studies to define TMD (temporomandibular dysfunction).
The epidemiological studies of TMJ alterations based on imaging analyses likewise have been unable to define a standardized pattern in the distribution of the disease. Radiographic changes corresponding to osteoarthrosis appear in 14-44% of the individuals – a figure far from the 1-24% of patients who show crepitants in response to palpation or to auscultation of the TMJ (crepitation being considered a clinical sign of osteoarthrosis) (7). In contrast to what might be expected, there is a poor correlation between the magnetic resonance imaging (MRI) findings in relation to the alterations of the intraarticular meniscus and the corresponding clinical findings. A considerable proportion of healthy individuals show disc dislocation in the imaging studies.
One way to address the problem is quantification according to the population demands for treatment. Based on this criterion, 3-7% of the population seeks treatment for pain and/or dysfunction of the TMJ or related structures. From this perspective it is understood that those patients who do not seek treatment do not consider such alterations to be a relevant problem, and are able to lead a normal life despite the symptoms (8).

ETIOLOGY: RISK FACTORS

Age
The estimated prevalence of TMD in children and adolescents varies from 6-68%, depending (as has already been commented) on the different diagnostic criteria used and on the differences in clinical examination. In a study published by List et al. (9) in adolescents between 12 and 18 years of age, 7% were diagnosed with temporomandibular pain-dysfunction, the prevalence being significantly higher in females than in males. Clicks were recorded 11% of the study population, with stiffness and mandibular fatigue in 3% and limitations in aperture in 1%. Schmitter et al. (10) reported that geriatric patients experience joint sounds in 38% of the cases and muscle pain in 12%, though without resting pain or joint pain. This contrasts with the group of young patients – with joint sounds in only 7% of cases, but with a much higher incidence of symptoms: facial pain in 7%, joint pain in 16%, and muscle pain in 25%.
Genetic factors
Michalowicz et al. (11) evaluated the hypothesis that signs and symptoms of TMD may be hereditary. To this effect they collected information by means of a questionnaire administered to a group of 494 monozygous and dizygous twins. The monozygous twins showed no greater similarities than in the case of the dizygous twins, and the homozygous twins that grew up together showed no greater similarities than those that grew up separately. The authors concluded that genetic factors and the family environment exert no relevant effect upon the presence of symptoms and signs of the TMJ.
Sex
Epidemiological studies generally document a greater frequency and severity of TMD in females than in males. In effect, TMD is seen to be up to four time more frequent in women, and these tend to seek treatment for their TMJ problems three times more often than males. Attempts have been made to explain these differences in terms of behavioral, psychosocial, hormonal and constitutional differences, though no conclusive results have been drawn to date.
It has been suggested that the presence of estrogen receptors in the TMJ of women modulates metabolic functions in relation to laxity of the ligaments, and this could be relevant in TMD. Estrogens would act by increasing vigilance in relation to pain stimuli, modulating the activity of the limbic system neurons. Although not all authors coincide, studies in humans have shown that the appearance of pain in the context of TMD increases approximately 30% in patients receiving hormone replacement therapy (HRT) in postmenopause (estrogens), and approximately 20% among women who use oral contraceptives (12).
Occlusion
Alterations in occlusion such as Angle malocclusions, crossbite, open bite, occlusal interferences, prominent overjet and overbite, crowding, midline discrepancies and missing teeth have been identified in different studies as predisposing, triggering or perpetuating factors. However, on one hand a relatively weak association is observed between occlusal factors and TMD, and on the other hand most studies published in the literature are of a cross-sectional design; as a result, few firm conclusions can be drawn regarding a possible causal relationship.
Donald Selligman and Andrew Pullinger, of the University of California, are probably the authors who have shown the greatest rigor in studying the relationship between occlusion and TMD. In their study published in the year 2000 (13) comparisons were made of a group of women with internal TMJ derangement versus asymptomatic control women. The patients with disc displacement were mainly characterized by unilateral posterior crossbite and long displacement of centric relation to the position of maximum intercuspidation. The patients with osteoarthrosis in turn associated an increased distance between centric relation and maximum intercuspidation, greater overjet and a reduction in overbite. The authors concluded that occlusal alterations may act as cofactors in the identification of patients with TMD, and that some occlusal variables may be a consequence rather than a cause of TMD.
The results of this study are partially refuted by Hirsch et al. (14), who after studying 3033 subjects concluded that greater or lesser overjet or overbite – even at extreme values – does not constitute a risk factor for the appearance of joint sounds (reciprocal clicks and crepitation).

In the work published by Magnusson et al. (15), involving the follow-up of 402 patients during 20 years, it was concluded that occlusal factors are weakly associated to TMD, though forced laterality between centric relation and maximum intercuspidation, and unilateral crossbite deserve consideration as possible local risk factors in the appearance of TMD.

In view of the information provided by the literature, the precise role of occlusion in TMJ pathology does not seem to be clearly defined. In contrast, and as has been pointed out by Koh et al.(16) in an analysis of the published randomized and quasi-randomized trials on the subject, there appears to be no evidence that occlusal fit treats or prevents TMD, and that it therefore cannot be recommended for the management or prevention of such disorders.

Hyperlaxity (Figure 1)

Kavuncu et al. (17) evaluated the risk of TMD in patients with systemic and TMJ hypermobility. Local hypermobility was diagnosed in the presence of condylar subluxation, while systemic hypermobility was assessed by means of the Beighton test. The authors found that both local and general hypermobility are more frequently detected in patients with TMD than in the controls, and that the risk of TMJ dysfunction is greater if the patient presents both alterations simultaneously. The investigators concluded that both situations may play a role in the etiology of TMD.

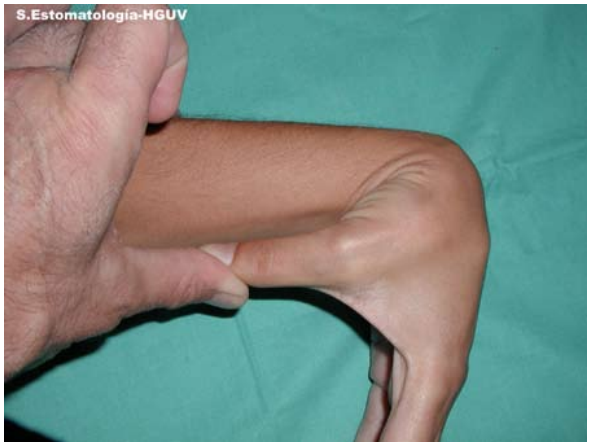

Fig. 1. Hiperlaxitud ligamentosa.

The study by de Coster et al. (18) likewise supports the hypothesis that hyperlaxity could cause TMD, since in a series of 31 subjects with Ehrler-Danlos disease, all presented symptoms of temporomandibular dysfunction and suffered recurrent temporomandibular dislocations. These results are in contrast to those previously reported by Conti et al. (19), who compared a group of 60 patients with mandibular sounds, pain or block versus a group of 60 asymptomatic patients. No association was found between the intraarticular disorders and systemic hyperlaxity, or between TMJ mobility and systemic hypermobility.

Antecedents of acute trauma

The possibility that acute trauma may induce histological alterations of the TMJ has been evidenced by studies in rats in which joint synovitis was generated by forcing condylar mobility. Improvement in synovitis or its total disappearance 20 weeks later was also observed.

There are no conclusive results regarding whether acute trauma (whiplash in traffic accidents being the most extensively studied example) acts as a triggering factor of chronic TMD.

Klobas et al. (20) found that patients with antecedents of whiplash showed significant differences versus patients without such antecedents, with more frequent severe TMJ symptoms (89% versus 18%) and also more clinical signs. Likewise, maximum oral aperture was smaller (54 mm versus 48 mm). Pain in response to the palpation of muscles and joints was more common, as was pain in response to mobilization. The authors concluded that the prevalence of TMD is greater among individuals with chronic whiplash injury than in the controls, and that neck injuries can affect TMJ function.

Different results have been published by Probert et al. in a retrospective study in Australia, involving 20,673 traffic accident victims. They documented 28 patients with TMD, and only one of the 237 patients that suffered mandibular fracture required posterior treatment for TMD. They concluded that the incidence of TMD after whiplash is very small, and that this mechanism of trauma alone is unable to account for TMD. Ferrari et al. postulated that a series of cultural and psychosocial factors could in fact be more relevant than whiplash in explaining why some patients in certain societies refer chronic symptoms (21).

Parafunctional habits

Dorland's Medical dictionary defines parafunction as disorderly or perverted function. Although the relationship between parafunction and muscle pain is biologically plausible, and there is some evidence to suggest a chronological relationship between the two, the fact is that controversy exists regarding this purported causal relationship.

Chewing gum has been used in a number of studies to evaluate the appearance of muscle pain with overfunction. Karibell et al. (22), after inducing the chewing of gum for 6 minutes, found pain to increase in both males and females in the patient group, though unexpectedly it also increased among the women in the control group – thus supporting the hypothesis of increased female susceptibility.

Miyake et al. (23), in a group of 3557 university students, found that chewing gum on one side of the mouth only, and tooth clenching, increased the risk of TMD – though the corresponding odds ratio (OR) only reached 2 for limitation in oral aperture among the subjects that chewed gum on one side only.

In a study published by Winocur et al. (24) in Tel Aviv (Israel) among 323 females aged 15-16, it was seen that those individuals with an intense habit of chewing gum (more than 4 hours a day) associated pain in the ear region at rest and during movement, as well as a greater prevalence of joint sounds. What the authors referred to as "jaw play" (the habit of forced mandibular lateralization or protrusion movements without occlusal contact) appeared less often

than chewing gum (14.2% versus 62.4%), though it was significantly associated with joint pain at rest and during movement, a sensation of tiredness during mastication, and joint sounds and blocks. The authors concluded that "jaw play" is the parafunctional habit with the greatest deleterious potential, and that chewing gum contributes to joint sounds and pain.

Bruxism (Figure 2a, 2b)

The prevalence of bruxism in the adult population is around 20%, and is similar to that recorded in children. In a recent study conducted in Boston by Cheifetz et al., parent interviewing revealed that 38% of the children (in a group of 854 with a mean age of 8.1 years) presented bruxism. However, only 5% of the parents reported subjective symptoms of TMD in their offspring (25).

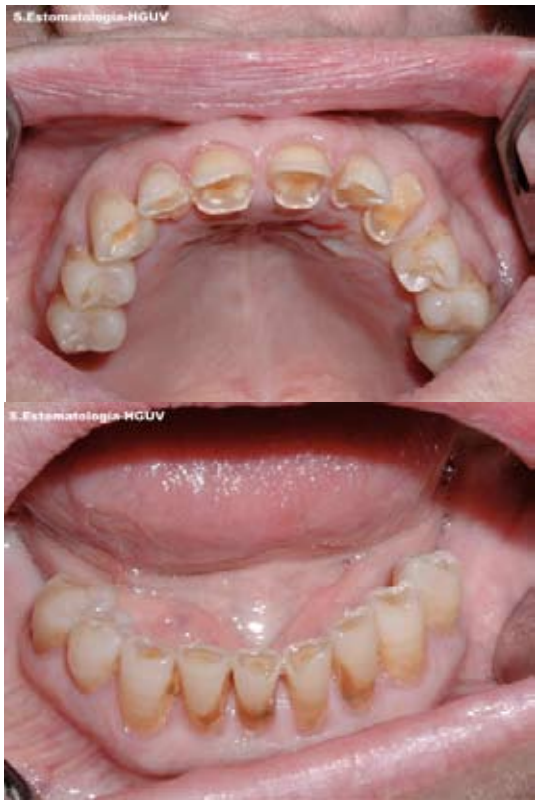

Fig. 2. a y B. Bruxism.

The greatest incidence of bruxism is between 20 and 50 years of age, after which the habit progressively decreases.

Regarding the etiology of bruxism, the intervention of occlusal interferences was initially postulated, though at present emotional stress is considered to be the principal triggering factor. Other factors that have been related to the origin of bruxism are certain drugs, central nervous system disorders, and a certain genetic and/or familial predisposition.

Magnusson et al. (26), in a longitudinal study of 420 individuals followed-up on for 20 years, reported a significant correlation between bruxism and TMD. Dental crowding at the start of the study was seen to be a predictor of TMD.

Huang et al. (27), in a study of 274 patients diagnosed with myofascial pain (n=97), arthralgia (n=20), and myofascial pain plus arthralgia (n=157), found the diagnosis of myofascial pain to be significantly associated to tooth clenching (OR=4.8). In the group of patients with myofascial pain plus arthralgia, the odds ratio was 3.3 versus the control group.

Stress, anxiety and other psychological factors

In 1955, Laszlo Schwartz et al. reported that a group of patients within the population classified as presenting "TMJ syndrome" could be characterized by painful limitation of mandibular movement caused by masticatory muscle spasm, and that this syndrome (known as mandibular pain-dysfunction) was probably of myofascial origin. Emphasis was placed on psychological stress rather than on occlusal disharmony, as primary cause of the problem.

In 1969, Daniel Laskin proposed the psychophysiological theory of myofascial pain, where stress is defined as a major causal factor. According to this theory, stress induces muscle hyperactivity. Fatigue resulting from such hyperactivity in turn would cause muscle spasms, with the following consequences: contracture, occlusal disharmony, internal derangement and degenerative arthritis. These factors would be able to alter the occlusion pattern during mastication, and this alteration therefore would be the effect rather than the cause of the pain-dysfunction syndrome.

Different studies (28) have confirmed that patients with myofascial pain and with myofascial pain associated to arthralgia, arthritis or arthrosis suffer increased levels of depression and somatization than those diagnosed only with disc displacement.

Orthodontic treatment

The possibility that orthodontic treatment could cause TMJ pathology has been extensively dealt with in the scientific literature. Despite the diverse methodological approaches involved, the great majority of studies conclude that orthodontic treatment neither improves nor worsens TMD.

Kim (29) reviewed 31 publications on orthodontics and TMD. He drew attention to the heterogeneity of the methodologies involved in these studies, and pointed out that only one of the reviewed articles found tooth extraction during orthodontic treatment to change the prevalence of TMD. The author concluded that orthodontic treatment does not increase the prevalence of TMD. Mohlin et al. (30) are of the same opinion. In a study conducted in Gothenburg (Sweden) involving 337 patients followed-up on between 11 and 30 years of age, they found that orthodontic treatment neither prevents nor improves dysfunction of the TMJ.

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