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Current Concepts on Temporomandibular Disorders

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Chapter 2

TMD Classification and Epidemiology

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The term temporomandibular disorders (TMD) embraces a number of conditions characterized by signs and symptoms involving the temporomandibular joint (TMJ), masticatory muscles, or both.[1] Its introduction is a recent phenomenon (last decade); before that, many terms were used including: myofascial pain syndrome, TMJ syndrome, TMJ dysfunctional syndrome, and Costen's syndrome.[2,3]

TMD signs are objective findings such as joint noises and limitations or abnormalities in jaw function; symptoms are subjective findings either reported by the patient, such as the anamnestic report of pain in the TMJ area or within the masticatory muscles, or elicited by the operator during the clinical assessment, such as pain in response to TMJ or masticatory muscle palpation. Despite such signs and symptoms being well described in the literature, both in terms of their epidemiologic relevance and the associated diagnostic pathways, uncertainties still remain about the pathophysiology of several TMD-related symptoms at the individual level.

Prevalence of TMD Signs and Symptoms in the General Population

TMD are considered the most common orofacial pain conditions of non-dental origin, even though the reported prevalence of TMD differs between investigations.[4] The classically described triad of clinical signs for TMD is: muscle and/or TMJ pain; TMJ sounds; and restriction, deviation, or deflection of the mouth opening path.[5] Nonetheless, a multitude of signs and symptoms such as earache, headache, neuralgia, and tooth pain may also be present as TMD-related or unrelated ancillary findings that need to be considered in the differential diagnostic process.

The actual prevalence of TMD at population level is a matter of debate, due to the lack of homogeneity in the diagnostic criteria adopted by various research groups, and there is evidence that the prevalence of TMD signs and symptoms may also be high in non-patient populations.[6] In particular, early reviews suggested that the prevalence of TMD in the general population ranges from 1% to 75% for objective signs and from 5% to 33% for subjective symptoms.[7] TMD symptoms are considered to have a gaussian distribution in the general population, with a peak in the age range

between 20 and 40 years for the most common forms and a lower prevalence in younger and older people. Females are predominantly affected by these disorders but, even though the reported numbers of females are relatively high in patient populations (see later in this chapter), it seems that, with regard to the prevalence of TMD signs at general population level, no significant gender differences exist.

Despite the existence of wide ranges of prevalence of TMD signs and symptoms reported in the different studies, the literature seems to be more consistent if one considers only the prevalence of the main TMD signs and symptoms (Table 2-1).

Two main factors seem to responsible for the differences in the findings among the studies, namely the TMD assessment (clinical studies vs. interview/questionnaire investigations) and the demographic characteristics of the study population (female:male ratio, mean age, age range). Interview studies conducted on Caucasian and Asian populations[10,16,18] reported a 6% to 30% prevalence of self-reported joint sounds, 5% to 33% for jaw pain, and 4% to 16% for any abnormality or limitation in mouth opening. Differences between studies are minimal if one considers the percentage of subjects with the same level of impairment and frequency of symptoms. For instance, self-reported severe mouth limitation, frequent joint sounds, and severe jaw pain affect approximately 4% to 6% of the general population.

Interesting data have also emerged from studies in which TMD signs and symptoms were assessed by means of clinical examination, which provide consistent findings for the percentage of subjects with clicking (8% to 20%), crepitus (4% to 15%), TMJ pain (4% to 10%), muscle pain (3% to 17.5%), and mouth opening limitation (4% to 9%). Differences in the demographic features of the samples and in the clinical protocols used to diagnose TMD may be responsible for some "out of range" findings that have characterized some studies, but useful information can be drawn from the investigations based on the Research Diagnostic Criteria for TMD (RDC/TMD, see later in this chapter).

Rantala and coworkers[14] found that joint sounds and muscle pain on palpation were the two most common TMD findings, involving 10% to 15% of the subjects, while other TMD-related signs and symptoms appeared to be rare in the non-patient population under investigation. Female gender was significantly associated with pain symptoms, in accordance with other painful musculoskeletal disorders,[19] while joint sounds and other objective signs seemed to be less gender-related.

Casanova-Rosado and coworkers[9] found a 46.1% prevalence of at least one TMD sign or symptom in a population of young adults and adolescents, with a mean age of about 17 years. Despite the authors' conclusion that their data could confirm that the disease appears even at a young age, it should be kept in mind that most clinical signs in this study were of moderate severity. More specifically, the prevalence of joint clicking was about 15%, in line with that described also by Rantala and coworkers,[14] and crepitus, which may indicate an ongoing inflammatory-degenerative process within the TMJ, was not found in any of the study subjects. Muscle pain on palpation in at least three facial sites was also relatively high (10.9%), and TMD prevalence was significantly higher among women than men (52.9% vs. 37.9%).

Interestingly, Schmitter and coworkers[12] applied the standardized RDC/TMD protocol to investigate the prevalence of TMD in a non-patient population of older subjects. In comparison with a group of young subjects, the so-called "geriatric" group, averaging 83 years of age, showed a markedly higher prevalence of objective clinical signs, such as joint clicking (38% vs. 7%) and crepitus (21% vs. 0%), presumably related to age-related joint degeneration. By contrast, subjective clinical symptoms, such as TMJ and muscle pain, were more prevalent in the younger subjects (16% vs. 0% and 22.7% vs. 10.3%, respectively). No gender-related differences were detected.

Taken together, these findings suggest that the common belief that TMD affects mainly

Table 2-1 Prevalence of most common TMD signs and symptoms in the general population.

Study's first author and year	Study sample	Examination method	Prevalence (%)					
			Clicking	Crepitus	TMJ pain	Muscle pain	Jaw pain	Mouth opening limitation
Rutkiewicz, 2006[8]	6335 (M=2869; F=3466; age 30–80)	Clinical	15	8	4	14	NI	9
Casanova-Rosado, 2006[9]	506 (46% males; age 14–25)	Clinical (RDC/TMD)	15.6	0	0	10.9	NI	5.9
McMillan, 2006[10]	1222 (M=505; F=717; age >18)	Interview	NI	NI	6.9	3.2–6.1	5	NI
Johansson, 2006[11]	12,468 (M=46%; two cohorts aged 50 and 60 years)	Interview	NI	NI	NI	NI	12.1	11.1
Schmitter, 2005[12]	58 geriatric (M=11; F=47; age range 68–96); 44 young (M=14; F=30; age range 18–45)	Clinical (RDC/TMD)	G 38; Y 7	G 21; Y 0	G 0; Y 16	G 10.3; Y 22.7	NI	NA*
Gesch, 2004[13]	4289 (M=2109; F=2180; age range 20–79)	Clinical	20	7.2	2–6.9	3.5–17.5	NI	9.1
Rantala, 2003[14]	241 (48% males; mean age 48 ± 7)	Clinical (RDC/TMD)	10–13.7	7–15	2.1–3.7	12.9	14.9	0.4
Nassif, 2003[15]	523 males (age 18–25)	Clinical	17–24.7	4.2	9.9	3–13.7	13	27.7
Pow, 2001[16]	1526 (M=769; F=757; age >18 years)	Interview	6–29.9	NI	NI	NI	33	4–8.2
Otuyemi, 2000[17]	308 (M=207; F=101; age range 17–32)	Clinical (Helkimo Index)	8.1	NI	2.6	3.2	NI	4.5
Goulet, 1995[18]	897 (M=400; F=497; age range >18)	Interview	9–30	NI	NI	NI	7–30	4–16

M – male; F – female; NI – not investigated; RDC/TMD – Research Diagnostic Criteria for temporomandibular joint disorders

people within the 20–40 years age range may be open to question. TMD signs and symptoms are almost equally prevalent in the different age groups, and the classic gender- and age-related features of TMD patients actually refer to TMD treatment-seeking subjects, who form a minority of those having TMD-related signs or symptoms.[20] Females complaining of painful TMD symptoms are predominant among patient populations (see later in this chapter), but objective TMD signs are almost equally distributed among the sexes and ages. This point confirms that pain is the cardinal symptom of TMD, on the assessment of which the diagnosis should be based, and on the management of which treatment should be targeted. Thus, over the years, the pathologic importance of some of the clinical signs, such as the presence of clicking, has diminished in contrast with past beliefs.

General Concepts of TMD Etiology

Many of the uncertainties that still characterize the TMD field are due to the complex etiopathogenesis of such disorders. At present, there is evidence that there is no place for a single etiologic factor or a sole etiopathogenetic theory that might be responsible for TMD onset as a whole.[21] Indeed, a multifactorial etiology has been repeatedly described, and different factors are likely to have a different role in the etiopathogenesis of different TMD symptoms.[22]

In dedicated chapters, Murray and Peck, Paesani, and Nitzan and Roisentul have provided their important contributions to summarize up-to-date knowledge of the etiopathogenesis of muscle disorders, disc displacement, and osteoarthritis, respectively. To enable readers to comprehend such scientifically sound observations as those expressed later in the book, some fundamentals of TMD etiopathogenesis are introduced in this chapter.

From a historical viewpoint, concepts about TMD etiopathogenesis went through the same shift from a dentally based to a medically based model, which also embodies the recent changes in the diagnostic and treatment approach to TMD patients.[23] Since the time when Costen, in 1934,[24] described his oto-mandibular syndrome, supposedly related to the loss of occlusal support, occlusal factors have been considered a major, and by many authors the sole, causal agent for TMD. Over the years, several etiopathogenetic theories have been postulated that assigned a central role to occlusal abnormalities, and several occlusal disharmonies have been correlated with TMD.[25] However, multivariate analysis investigations involving logistic regression models suggest that occlusal variables account for up to 27% of the total amount of variance for TMJ disorders[26] and up to 10% for masticatory muscle disorders.[27] This means that the influence of dental occlusion on the onset of TMD is much smaller than was believed in the past and leaves the door open for the search for other potential risk factors for TMD.

At present, the role of psychosocial factors, the involvement of which in the etiopathogenesis of TMD was already emphasized by Lupton and Schwartz early in the 1950s (see, for example, the review by Molin[28]), is gaining attention, and the number of papers documenting an association of TMD with stress, anxiety, depression, and somatization is rapidly increasing.[22,29] A classification system that provides an assessment of the psychosocial impairment along with the physical impairment, the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD),[30] which is being updated at the time of writing of this book,[31,32] has been approved by the scientific community and will be described in detail later in this chapter and in other parts of the book. Nonetheless, the actual causal link between TMD and psychosocial factors is yet to be clarified, and psychosocial impairment has been assumed to be a consequence rather than a cause of TMD in the majority of patients.[33] Among the psychosocial factors, the role of stress sensitivity

deserves attention due to its link with teeth clenching (see the review by Manfredini and Lobbezoo[34]), which is another major risk factor for TMD that will be described in detail later in the book.

An intriguing clinical hypothesis is that teeth clenching may be most dangerous in patients with particular skeletal characteristics associated with certain muscular force vectors exerted on the TMJ (see Chapter 8). The issue of the prevalence of TMD in patients with different musculoskeletal profiles has not been addressed exhaustively in the literature so far, and future studies should take into account the assessment of TMJ (over)load provoked by muscle forces exerted during functional and non-functional jaw movements in patients with different facial morphology.

Moreover, there is currently a compelling need to provide biologically plausible explanations for the higher prevalence of painful TMD in females.[35] Several studies have been conducted to assess the etiopathogenetic role of gender differences in the levels of some hormones.[36,37] Efforts are currently ongoing to identify potential neuroendocrine pathways leading to TMD predisposition in the female sex[38] as well as to identify early predictors of onset of TMD and facial pain.[39] Also, as shown in Chapters 1 and 6, progress has been made in the knowledge of histologic and tissue characteristics of the TMJ and in the attempt to relate the different risk factors with a specific pathogenetic pathway. Recent advances have pointed out that genetic factors may also have some importance in the onset of TMD symptoms.[40] Last, but not least, there is a need to go deeper into the phenomenon of neuroplasticity, which seems to play a major part in the explanation of persistence of TMD pain.[41] Persistent TMD pain is likely to be a manifestation of chronic neuropathic pain, which bears little relation to the original source of pain and is maintained by the phenomenon of central sensitization, even after the removal of the noxious stimulus.

Taken together, the literature on the etiopathogenesis of TMD suggests that they are not dental-related, but, rather, they are part of the wider family of orofacial pain disorders, which accounts for the need to consider neurologic, endocrine, and psychosocial factors during the diagnostic process.

These current concepts, which impact on etiopathogenesis as well as diagnosis and management of TMD, will be the main focus of this book and will be discussed with particular attention to their implications for the clinical practitioner.

TMD Classification Schemes

In the case of TMD, the search for a causal factor is often frustrating and unproductive, to the point that past and current classification schemes are not based on the etiology of symptoms. This may explain why the history of TMD literature is rich in taxonomic and classification proposals that have failed to achieve international consensus and have prevented gathering comparable data from different studies (for a review see Suvinen and coworkers,[22] and Okeson[2]). At present, two classification systems are widely adopted in the TMD literature, the American Academy of Orofacial Pain (AAOP) classification[42] and the RDC/TMD.[30]

AAOP Classification (Box 2-1)[42]

This classification divides TMD in articular and masticatory muscle disorders, for each of which a detailed description of symptoms and some pathogenetic information is provided.

Congenital or developmental disorders include aplasia, hypoplasia, hyperplasia, and neoplasms. The most common form of aplasia is hemifacial microsomia, which is characterized by a unilateral lack of growth of the condyle. Hypoplasia is less severe than aplasia and may affect the cranial bones or the mandible. Conversely, hyperplasia is the overdevelopment of the cranial bones or the mandible. Dysplasia is a slowly progressing hyperplasia characterized

Box 2-1 AAOP classification of temporomandibular disorders (De Leeuw[42])

TMJ articular disorders
Congenital or developmental disorders
- Aplasia
- Hypoplasia
- Hyperplasia
- Dysplasia
- Neoplasia

Disc derangement disorders
- Disc displacement with reduction
- Disc displacement without reduction

TMJ dislocation
Inflammatory disorders
- Synovitis and capsulitis
- Polyarthritides

Non-inflammatory disorders
- Primary osteoarthritis
- Secondary osteoarthritis

Ankylosis
Fracture
Masticatory muscle disorders
Local myalgia
Myofascial pain
Centrally mediated myalgia
Myospasm
Myositis
Myofibrotic contracture
Masticatory muscle neoplasia

by the presence of fibrous connective tissue, which usually occurs in childhood and adolescence and becomes inactive following skeletal maturity. Neoplasms can be benign or malignant, though they are a rare cause of TMD; squamous cell carcinomas of the maxillofacial region and primary nasopharyngeal tumors are the two most common malignant conditions of the maxillofacial area. All congenital or developmental disorders are characterized by abnormal facial morphology of varying degree, and they are treated surgically by a number of approaches.

Disc derangement disorders are represented by different conditions of articular disc displacement, which is an abnormal relationship between the disc and the condyle. Disc displacement with respect to the condyle usually happens in an anteromedial direction and may be with or without reduction. The former is characterized by a misalignment of the disc–condyle complex in the closed mouth position that improves during mouth opening, therefore, reduction. The latter is maintained during condylar translation and may be associated with limitation in mandibular range of motion. Such conditions, as well as their clinical signs (clicking during reduction, deviation of the jaw opening pattern toward the affected side, inability to open the mouth in the acute stages of displacement without reduction) have been well described in the literature. Importantly, the pathologic significance of disc displacement has reduced in recent years, and there is now consensus that asymptomatic disc displacements should be left untreated.

TMJ dislocation is a condition in which the condyle goes beyond the articular tubercle during translation and is unable to regain its normal position within the glenoid fossa. Clinically, the patient cannot close the mouth. Dislocation may be momentary or prolonged and may be accompanied by pain at the moment of dislocation. Behavioral and physiotherapeutic approaches are often enough to avoid recurrent episodes of dislocation, but surgery may be needed in a minority of patients to normalize joint tubercle morphology.

Inflammatory disorders of the TMJ include capsulitis, synovitis, and the polyarthritides. Synovitis, described as the inflammation of the synovial fluid of the TMJ, and capsulitis, which is inflammation of the joint capsule, may be related to infections or trauma, and they are impossible to differentiate clinically. The polyarthritides are characterized by TMJ inflammation and structural changes as part of systemic, generalized polyarthritic diseases, such as a number of rheumatologic conditions (among others, rheumatoid arthritis, psoriatic arthritis, infectious arthritis, autoimmune disorders). Pain and joint degeneration are the cardinal manifestations of these disorders, and the

management of such symptoms is the target of treatment.

Non-inflammatory disorders are represented by primary and secondary osteoarthritis. Osteoarthritis, also called osteoarthrosis or degenerative joint disease, is characterized by joint degeneration, bone surfaces remodeling, and articular cartilage loss, and may be *primary*, ie no identifiable etiological factor, or *secondary*, ie due to an identifiable etiologic factor or event. In the latter, which is mainly due to micro- or macrotrauma, or to systemic diseases, treatment should be directed toward the resolution of the primary causal factor.

Ankylosis is a fibrous or, less often, osseous adhesion of the articular surfaces, which prevents the patient from opening the mouth wide. Joint ankylosis is often the long-term consequence of a traumatic event, but it may also be a consequence of repeated infections in the ear or TMJ area, or even a post-surgical complication after interventions in the TMJ area. Treatment is surgical, and in recent years long-term positive findings have been described with total TMJ prosthesis.

Fracture may be due to direct TMJ trauma or to trauma to the jaw. Consequences may range from mild TMD symptoms to severe deformations of the joint bone components, demanding surgery.

Local myalgia is muscle soreness with pain in the masticatory muscles during function. It is usually bilateral and described as a cramp-like feeling. It has been thought to be associated with prolonged non-functional jaw activities, which lead to delayed-onset muscle soreness, eg after prolonged activation of the masticatory muscles as that exerted by masseter and temporal muscles during jaw-clenching activities. It is difficult ot distinguish from other differential diagnoses of muscle pain.

Myofascial pain is characterized by a regional, dull, aching pain within the muscle and is associated with the presence of trigger points. With respect to local myalgia, myofascial pain is characterized by pain at rest and pain aggravation and referral with provocation of trigger points.

Centrally mediated myalgia is a chronic, continuous muscle pain disorder. It may present as a myositis-like pain without signs of inflammation or it may be accompanied by signs of neurologic inflammation, likely due to prolonged nociceptive input to the central nervous system. The presence of persistent pain is more important than its duration and intensity for the onset of the centrally mediated mechanisms that are at the basis of the development of pain chronicity. Treatment should be started as early as possible and directed toward achieving a desensitization of central mechanisms.

Myospasm is an acute condition characterized by a sudden, involuntary tonic muscle contraction, such as a cramp or trismus. Mouth opening is markedly reduced due to the continuous muscle contraction. Myospasm is a rare condition in facial muscles.

Myositis is an inflammatory condition presenting with the classic clinical signs of tissue inflammation, such as swelling, redness, and increased temperature. Trauma is considered a potential source of muscle inflammation as well as infections spreading from nearby tissues. Inflammation can occur also in the tendinous attachments of the muscle, viz., tendinitis.

Myofibrotic contracture refers to the chronic, painless shortening of a muscle due to fibrosis of tendons, ligaments, or, more rarely, muscle fibers. Such a rare condition is sometimes observed after periods of prolonged muscle immobilization, as in the case of postoperative intermaxillary rigid fixation.

Masticatory muscle neoplasia can be benign or malignant and needs to be confirmed by imaging and biopsy.

Research Diagnostic Criteria for Temporomandibular Disorders (Box 2-2)[30]

The RDC/TMD guidelines provide standardized criteria for a two-axis diagnosis. This means that, along with a physical diagnosis (axis I), the patient receives a psychosocial diagnosis as well (axis II).

Box 2-2 Research diagnostic criteria for temporomandibular disorders (Dworkin and LeResche[30])

Axis I diagnoses
Group I
a. Myofascial pain
b. Myofascial pain with limited opening
Group II
a. Disc displacement with reduction
b. Disc displacement without reduction with limited opening
c. Disc displacement without reduction without limited opening
Group III
a. Arthralgia
b. Osteoarthritis
c. Osteoarthrosis
Axis II diagnoses
Depression
- No depression
- Moderate depression
- Severe depression

Non-specific physical symptoms
- No somatization
- Moderate somatization
- Severe somatization

Chronic pain severity
Grade 0: low disability
Grade I: low disability, low intensity
Grade II: low disability, high intensity
Grade III: high disability, moderately limiting
Grade IV: high disability, severely limiting
Jaw limitation scores

Axis I of the RDC/TMD classification system is a clinically based assessment taking into account both anamnestic and clinical parameters of evaluation. It provides criteria for the diagnosis of three main groups of disorders: muscle disorders (group I), disc displacements (group II), and other joint disorders, such as arthralgia, osteoarthritis, and osteoarthrosis (group III). A detailed description of the RDC/TMD is beyond the scope of this chapter, and readers are referred to chapters on the clinical and psychosocial diagnosis for more detailed description of such criteria, but some reference to the criteria needed for diagnoses will be helpful here for the comprehension of the concepts leading to their formulation.

Muscle disorders (group I) are diagnosed on the basis of anamnestic reports of pain in the muscles of mastication, and clinical assessments of pain at palpation of at least three out 20 muscular sites in the facial area (10 for each side). The only distinction among muscle disorders is made when mouth opening is less than 40 mm. When criteria for group I diagnosis are satisfied, a diagnosis of myofascial pain has to be made, with or without restricted mouth opening, on the basis of the range of jaw motion.

The diagnostic group of disc displacements (group II) aims to detect conditions in which the TMJ disc is anteriorized with respect to the mandibular condyle. Three diagnostic subgroups are identified: displacements with reduction, and displacements without reduction with or without restricted mouth opening. The main criterion for diagnosis of disc displacement with reduction is the presence of reciprocal clicking during jaw movements (audible during both jaw opening and jaw closing movements) that is not fixed (audible at different stages of motion during the jaw opening and jaw closing movements). A disc displacement without reduction is diagnosed when a history of previous clicking is accompanied by its absence at clinical assessment and by a deflection during jaw opening. When the mouth opening is less than 35 mm, a diagnosis of displacement without reduction with restricted mouth opening can be made, while a mouth opening of more than the cut-off value points toward the diagnosis of disc displacement without reduction and without restricted mouth opening.

The third group of diagnoses – arthralgia, osteoarthritis, and osteoarthrosis (group III) – is based on findings on joint palpation, ie the presence of pain on palpation and crepitus, alone or combined.

For the psychosocial diagnosis (axis II), the patient is given a rating for jaw disability,

chronic pain, and depression by using validated questionnaires, which allows assessment of the psychosocial aspects that have to be addressed at the therapeutic level.

Although the RDC/TMD guidelines do not allow diagnosis of less frequent conditions, or pathologies that do not show a clear origin and natural progression (such as traumatic injuries, neoplasm of condyle, acute traumatic injuries, polyarthritis, atypical facial pain, and headaches), they actually represent the standard of reference for TMD diagnosis and classification in the research setting, and allow cross-cultural and multicenter comparisons, both in the patient and non-patient populations.

Comments on the AAOP and RDC/TMD Guidelines

The two classifications are intended for different purposes.

The AAOP classification is a clinically oriented taxonomic proposal that contains some referrals to the plausible pathogenesis of the different disorders. Unfortunately, the discriminatory power of the proposed criteria to differentiate between the diagnostic categories has never been tested. Moreover, the actual existence of some masticatory muscle disorders, or at least the possibility to specifically identify them in the clinical setting, is a matter of major debate, and more studies are needed to fine-tune the process of differential diagnosis. Distinguishing between, for example, local myalgia and myofascial pain is not simple, if even possible, and strong efforts should be directed toward the identification of a specific pathophysiology for each disorder. Nonetheless, it should be kept in mind that the AAOP guidelines provide a quantity of information that is potentially useful in the clinical setting.

By contrast, the RDC/TMD was developed as an instrument designed for research purposes, to implement standardization of diagnoses and to allow comparison of findings between different studies. Since the time of its introduction in the TMD literature, the RDC/TMD has rapidly gained popularity among researchers and its use has contributed greatly to improved knowledge about TMD epidemiology. Validation of many of the physical and psychosocial diagnostic protocols included in the RDC/TMD has been achieved, and the efforts made by the dedicated consortium to translate the guidelines in as many languages as possible, to ease its dissemination, over the years has been laudable. Notwithstanding, there is consensus within the scientific community that information gained with the use of the RDC/TMD should be used to modify the diagnostic criteria in accordance with the currently available scientific knowledge.

In summary, the strengths of the RDC/TMD classification (standardization of criteria, simple taxonomic groups), which have led to its widespread use among epidemiologists and researchers, are not so helpful in the clinical setting, where the use of a wider classification system providing etiopathogenetic information as well is more appropriate. This is the reason for the widespread adoption of the AAOP classification system for TMD assessment in the clinical setting. The quantity of potentially clinically useful information provided by the AAOP guidelines is much superior to that of the RDC/TMD, but most of it is empirically based and, consequently, not suitable for research purposes. For these reasons, the two classification schemes can coexist, and may be assumed to be the current standards of reference in their respective settings.

Prevalence of TMD in Patient Populations

A limitation of current knowledge on TMD is that most data have come from studies performed at general population level, while a detailed description of populations of patients attending TMD clinics all over the world, which see the most severe and treatment-seeking cases, is much more useful in providing a snapshot

of the real clinical impact of such conditions. Populations of patients at tertiary clinics have been described in several studies, but even here, generalization of data is limited by the frequent use of non-standardized diagnostic and classifying procedures. Thus, comparison of clinical findings is possible between studies using the RDC/TMD (Table 2-2). (Note: psychosocial findings will be discussed in Chapters 3, 11, and 21).

All the investigations reported a higher prevalence of at least an RDC/TMD diagnosis in females, in a ratio ranging from 2.6:1 to 7.3:1. Mean age of patients was quite similar in all studies. With regard to RDC/TMD axis I diagnoses, the prevalence of group I disorders (muscle disorders) ranges between 31%, as reported in Asian patients,[48] and 76%, as reported in the first cross-cultural study conducted more than a decade ago on populations of Swedes and Americans.[49] Apart from this large variability, another notable difference concerns the prevalence of myofascial pain with limited opening, which was less than 2% in a study on Italian patients and up to 30% in other investigations. Considering that all the studies were performed by trained and calibrated investigators, such differences are quite unexpected and hard to explain on the basis of racial differences alone. Gender differences in the prevalence of myofascial pain were not investigated in all studies. A female:male ratio of 4.3:1 was reported in one study,[44] but, in contrast, no significant sex differences were found in a study on Asians,[48] even though women of childbearing age constituted the majority of the group I patients (3.3:1) in this study. Significant gender differences in RDC/TMD diagnosis of myofascial pain were reported in some studies on non-patient populations, making it possible to hypothesize that myofascial face pain shares epidemiologic characteristics with other musculoskeletal pain conditions,[50] so that biologic mechanisms underlying gender differences in pain should be applied to TMD patients as well.[19]

In all studies, the most frequent group II diagnosis was disc displacement with reduction, with a prevalence ranging from about 10% to 35%, while disc displacement without reduction with or without limited opening had a lower frequency (0% to 12%). Disc displacement with reduction was also the most common group II diagnosis in non-patient populations, as reported in a study on Finnish non-patients, using the RDC/TMD and reporting comparable prevalence (15.8%).[14] Observations that disc displacement is common among non-patients as well as the high variability of disc position in asymptomatic subjects,[51] lend support to the hypothesis that disc displacement can be, in many cases, considered a non-pathologic or, at least, a non-treatment-requesting condition.

The prevalence of group III disorders was similar in almost all studies (about 50%), with some notable exceptions in the investigations in Israeli[43] and Asian[48] patients, which reported lower prevalence data. Some differences in the pattern of subgroup distribution are also quite evident; for example, in the investigations in Italian patients,[44,47] the prevalence of arthritis and arthrosis was higher than in other studies. This may be attributed to the fact that many forms of articular remodeling are clinically silent and can be diagnosed only by means of radiologic exams, as suggested by RDC/TMD guidelines, which were used on almost all patients of such studies and not in other investigations.

As for multiple diagnoses, they were not reported in all studies. In general, it is a common observation that a high percentage of patients presented more than one RDC/TMD diagnosis as a confirmation of the complexity of clinical symptomatology in TMD patients. Data about the relative frequency of single and multiple diagnoses are mostly important in terms of their prognostic impact; but, unfortunately, little attention has been given to such issues so far. In the patient populations for which data were reported, the prevalence of multiple diagnoses was near to 50%, suggesting that a combination of muscular and articular disorders is a frequent clinical reality and that an improvement in knowledge about how and when such disorders relate to each other is a compelling need for the near future.

Table 2-2 Studies on RDC/TMD diagnoses distribution in patient populations

First author and year of study	Study sample	RDC/TMD diagnosis (%)													
		Ia	Ib	IIa (right)	IIa (left)	IIb (right)	IIb (left)	IIc (right)	IIc (left)	IIIa (right)	IIIa (left)	IIIb (right)	IIIb (left)	IIIc (right)	IIIc (left)
Winocur, 2009[43]	298 (M=65; F=233)	47	18	36.2 (Joints)		12.8 (Joints)		8.1 (Joints)		14.1 (Joints)		6.4 (Joints)		2.9 (Joints)	
		M 14; F 86		38 (Patients); M 25; F 75						11 (Patients)		5 (Patients)		2 (Patients)	
Manfredini, 2006[44]	377 (M=101; F=276)	36.6	1.9	32.1	31.6	2.9	1.9	1.8	2.9	17.5	17.2	12.7	10.8	11.6	12.4
		M 25.7; F 40.2	M 0.9; F 2.1	M 33.6; F 31.5	M 23.7; F 33.6	M 0.9; F 3.6	M 0; F 2.8	M 0.9; F 2.1	M 1.8; F 3.2	M 14.8; F 18.4	M 9.9; F 18.4	M 13.8; F 12.3	M 5.0; F 12.6	M 7.9; F 13	M 7.9; F 14.1
Reiter, 2006[45]	115 (65 Israeli Jews, M:F= 1:2.4; 50 Israeli Arab, M:F=1:7.3)	50	32	28		2		0		40		6		4	
Plesh, 2005[46]	61 females	F 47.5	F 32.8	F 32.7 (Patients)		F 0 (Patients)		F 0 (Patients)		F 41 (Patients)		F 6.5 (Patients)		F 0 (Patients)	
Manfredini, 2004[47]	285 (M=63; F=222)	50.2		38.6; 22.8 Right; 19.3 Left						50.2; 27.4 Right; 30.2 Left					
Yap, 2003[48]	191 (M= 53; F=138)	13	18	12.5 (Patients)	9.9 (Patients)	2.6 (Patients)	4.1 (Patients)	0.5 (Patients)	1 (Patients)	11.5 (Patients)	10.9 (Patients)	0.5 (Patients)	0 (Patients)	1.0 (Patients)	1.5 (Patients)
		M 16.9; F 9.4	M 11.6; F 21.7	M 9.4; F 13.7	M 11.3; F 9.4	M 3.7; F 2.1	M 1.8; F 5	M 0; F 0.7	M 0; F 1.4	M 5.6; F 13.7	M 3.7; F 13.7	M 0; F 0.7	M 0; F 0	M 0; F 1.4	M 0; F 2.1
List, 1996[49]	82 Swedish	50	26	28	34	0–4				16	23	0–6			
	210 US	46	30	18	24					43	38				
	Overall			32 Right; 39 Left						25 Right; 31 Left					

M – male; F – female.
Unless otherwise specified, data for the prevalence of joint disorders (group II and III) are expressed per percentage of affected joints.

In summary, there is some evidence suggesting that the RDC/TMD classification system, in line with one of its proposed objectives, has allowed identification of some differences between the constitution of patient populations in tertiary centers all over the world, so that further investigations are needed to define possible cross-racial and cross-cultural patterns in the relative frequency of TMD conditions.

Natural Course of TMD

TMD are considered benign disorders with favorable prognosis, and the literature supports the view that symptoms are self-limiting in many cases, thus encouraging the adoption of conservative and reversible forms of treatment.[52] Fluctuation and self-remission of symptoms are a major source of bias for studies on the effectiveness of TMD treatments as well as one of the reasons why high success rates have been described for several treatment approaches. Nonetheless, some patients progress toward pain chronicity, mainly due to biopsychosocial reasons or due to sensitization phenomena that will be described later in the book. Notwithstanding, taking a look at the natural course of the disease might be useful to get a better insight into some of the etiopathogenetic, diagnostic, and therapeutic concepts that will be introduced later in the book.

Clinical signs have a different impact on the patient's jaw function and, more generally, quality of life, with respect to symptoms, to the point that many of the former have been stripped of the pathologic significance that was attributed to them in the past. Clicking within the TMJ, "abnormal" jaw-opening patterns (ie non-linear direction with respect to the sagittal plane, non-repeatable pattern of mouth opening and closing, slow movements) and suggested occlusion-related risk factors are examples of clinical signs that have also been found in asymptomatic TMD subjects and that can be no longer viewed as treatment-seeking findings. There is now much evidence showing that only a minority of clicking joints, ie joints with disc displacement with reduction, progress toward a diagnosis of disc displacement without reduction,[53] and that the presence of clicking in adolescence is not an indicator for the onset of more severe or painful TMJ disorders in adulthood.[54] Thus, past beliefs that disc displacements go through staged phases of anteriorization that inevitably end in a non-reducing displacement have been shown to be wrong. Besides, the actual relationship between disc displacement and degeneration of joint surfaces is far from being fully understood, and there is no evidence base for the hypothesis that damage to the articular cartilaginous and bone structures can be stopped by any treatment aiming at disc repositioning.[55] Moreover, the pathological significance and severity of some signs of joint degeneration, which are more common in older subjects, such as joint crepitus, should be assessed in terms of age-related structural changes.[56] In consideration of that, the need for treatment in the presence of joint sounds has to be determined at the individual level. According to current views, the only treatment-seeking clinical sign is the presence of severe limitation in mouth opening, a manifestation of several joint and muscular disorders.

As for clinical symptoms, ie muscle and TMJ pain, it is a common opinion that they are fluctuating in nature, and longitudinal observational studies on populations of TMD patients provide support for such observations.[57] Also, progression toward severe or persistent pain is rare.[58] Notwithstanding, in some cases pain becomes persistent, probably because of the phenomenon of central sensitization in response to prolonged peripheral hyperexcitability, and this is the reason why pain assessment in TMD patients deserves special attention by clinicians as early as possible in the diagnostic process. At present, suggested predictors of pain chronicity have been intensity and frequency of baseline pain as well as the concurrent presence of widespread pain in other body areas, but there is no doubt that greater knowledge of the pathophysiology of the different causes for

TMD pain is much needed. Once this has been accomplished, the natural course of clinical symptoms can be more fully comprehended.

Conclusions

TMD are a major cause of pain in the orofacial area. For years, the analysis of epidemiologic data has been made difficult by the absence of a standardized taxonomy for the diseases grouped under the term TMD and by the poor knowledge about their etiology. At present, the RDC/TMD classification system, which is undergoing a process of updating, seems to be the most suitable tool to help researchers to compare findings from different studies and to acquire greater knowledge of the distribution of TMD signs and symptoms in the general population as well as in patient samples.

The prevalence of mild TMD signs has been reported to be high in the general population, but the pathologic connotation of many of them has been diminished by longitudinal studies that have shown that they rarely progress toward severe disorders. The presence of combined muscle and joint disorders seems to be the most frequent condition among patients attending tertiary TMD clinics, even if very few studies have reported on this specific issue. Future studies need to be directed toward the description of the pathophysiology of the different TMD symptoms, in order to gather more information on the natural course of the disorders and better identify all the risk factors for pain chronicity and persistence.

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