

Temporomandibular Disorders as a source of orofacial pain

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Abstract

Since decades, the dental profession has been challenged by pain and dysfunction of the masticatory system. In recent years, a better understanding of the pathophysiological mechanisms of masticatory pain paralleled the increase of basic and clinical research focusing on pain in general. Consequently, so-called diagnostic techniques and treatment procedures, based upon hypothetical, sometimes dogmatic, etiological mechanisms, are increasingly questioned, and the ill-supported thoughts are gradually replaced by research-based insights. In addition, a better communication between basic scientists, researchers, and medical or dental practitioners focusing on musculoskeletal pain, has led to an improved quality of research on pain and dysfunction of the masticatory muscles and the temporomandibular joint (TMJ). This paper will try to review this progress, comment on the clinical implications, and give some suggestions for future research.

Key words : Orofacial pain ; temporomandibular joint ; myofascial pain ; masticatory system.

Introduction

Epidemiological studies, especially in Scandinavia, have illustrated the high prevalence and incidence of signs and symptoms grouped under the heading Temporomandibular (or Cranio-mandibular) Disorders (for review see Leresche, 1997). Isolated complaints of clinical pain in the joints and muscles and minor signs of dysfunction were reported in the majority of the population. The real treatment need, however, is estimated at 2 to 5% (Carlsson, 1999). The comparison of clinical studies and the interpretation of treatment procedures and results has been hampered by the lack of standardized diagnostic criteria for (the different subgroups) of temporomandibular disorders (TMD).

CLASSIFICATION OF TEMPOROMANDIBULAR DISORDERS

Several classifications have been suggested : the ad hoc committee of the International Headache Society (1988) mentioned temporomandibular disorders and related pain in sections 2.2 and 11.7,

while also other proposals were published, based upon the orthopedic literature, biopsychosocial models or rheumatological classifications. Thorough review of the existing literature by expert committees (Dworkin and LeResche, 1992) criticized the existing taxonomy because of the descriptive nature, the lack of validation, poor specificity, and impossibility of having multiple diagnoses. Consequently, a set of Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) were formulated, based on operational definition of the terms used, epidemiological data, specification of the examination methods, and established reliability of the measurements. As in most classifications of pain syndromes, a dual axis system was applied, allowing a physical diagnosis to be coordinated with operationalized assessment of psychological distress and psychosocial dysfunction. The validation and clinical testing of these RDC/TMD is currently in progress in several centers (Huggins *et al.* 1996). According to Axis I of the RDC/TMD, three subgroups are defined : muscle disorders, disc displacements, and a group covering arthralgia, arthritis, and arthrosis (Table 1). Muscle spasm, myositis, and contracture of the masticatory muscles, as well as the polyarthritides involving the TMJ and acute traumatic injury, are rare and sometimes ill-defined. Consequently, these diagnoses were deliberately omitted from the classification.

Table 1

Research diagnostic criteria , axis I : clinical conditions
(Dworkin and LeResche, 1992)

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|---|
| Group I : Muscle Disorders |
| – Myofascial pain |
| – Myofascial pain with limited opening |
| Group II : Disc displacements |
| – Disc displacement with reduction |
| – Disc displacement without reduction with limited opening |
| – Disc displacement without reduction without limited opening |
| Group III : Arthralgia, arthritis, arthrosis |
| – Arthralgia |
| – Osteoarthritis of the TMJ |
| – Osteoarthrosis of the TMJ |

The development of such a classification is a continuous process and will make progress in line with a better understanding of the cause(s) and natural progression of these disorders.

Masticatory muscle pain

Muscle pain (myalgia) is considered the most common source of pain in patients presenting with chronic pain (Simons, 1988) as well as in an asymptomatic population (Carlsson, 1999). The use of the terms "myofascial pain syndrome (MPS)", "trigger points" (Travell and Simons, 1983) and some suggested diagnostic criteria (e.g., twitch response, taut band) are still debated. Recent research (Drewes *et al.*, 1993) could not confirm the presence of histological changes at the site of the trigger points. In addition, the spread of the pain sensation after local injection of hypertonic saline in muscles only partially parallels the described referred pain patterns (Stohler and Lund, 1995; Graven-Nielsen *et al.*, 1997). The vague term MPS tends to be replaced by the diagnosis "fibromyalgia" which is defined as widespread pain with tenderness at 11 out of 18 specific points located all over the body (Wolfe *et al.*, 1990). Unfortunately, none of these points is situated in the orofacial region. The overlap between fibromyalgia and TMD is poorly documented: 18 to 35.5% of fibromyalgia patients reported jaw pain (Leavitt *et al.*, 1997); more widespread body pain and other symptoms of fibromyalgia are infrequent in TMD populations (Dao *et al.*, 1997; Cimino *et al.*, 1998). More systematic studies are needed to understand the nature of both conditions.

THE GENERATION AND PROCESSING OF MUSCLE PAIN

Knowledge on how noxious information is registered and transmitted from the muscles to the central nervous system (CNS) has been obtained in animal studies (Mense, 1993). In contrast with this body of knowledge on the spinal afferents involved in muscle and joint nociception in limbs, data on the physiological properties of nociceptors in the jaw muscles and the TMJ is scarce (Hannam and Sessle, 1994). From the animal studies, however, one could deduct that also in the trigeminal system, heavy mechanical or chemical agents predominantly excite free endings, served by small-diameter afferents (Group III and IV), which, similar to limb muscles, are subject to peripheral sensitization and are involved in neurogenic inflammation (Sessle, 1999). These afferents project to various sites of the trigeminal (V) sensory complex, but especially the subnucleus caudalis (medullary dorsal horn) and the ventrobasal and posterior complex of the thalamus. Virtually no information exists concerning the projection from thalamus to cortex as regards the orofacial area.

ETIOLOGY OF MUSCLE PAIN

Several etiological hypotheses from the past have been challenged and refuted:

- the importance of malocclusion in the etiology of muscular pain (and other symptoms of TMD) has been calculated as "minor" (McNamara *et al.*, 1995), since the correct interpretation that simple correlations do not implicate cause-effect relationships (De Laat *et al.*, 1986),
- the theory of the "vicious cycle", which suggested that "pain causes muscle hyperactivity which in turn causes more pain" (Laskin, 1969), has been challenged: critical evaluation of the literature (Lund *et al.*, 1989) and recent experimental data indicate that a) parafunctional habits are a very common event, usually not resulting in TMD symptoms (Lobbezoo and Lavigne, 1997), b) that bruxers with pain exhibit less episodes of bruxism per hour than bruxers without pain (Lavigne *et al.*, 1997), and c) that heavy exercise results in short-term pain and does not trigger the "vicious cycle", but merely produces a training effect (Svensson and Arendt-Nielsen, 1996). In addition, Lund *et al.* (1989) showed that TMD patients do not show increased postural electromyographic (EMG) activity of jaw muscles or signs of central motoneuronal hyperexcitability (Cruccu *et al.*, 1997). Muscle pain seems to result in a decreased maximal voluntary contraction and bite force (Lund *et al.*, 1989) in humans, as well as smaller and slower neuronal discharges in rabbits (Westberg *et al.*, 1997).

Instead of a "vicious cycle", these findings all fit in a pain-adaptation model including a diminished capacity to perform work against load and a reduction of speed and range of motion (Lund *et al.*, 1989; Westberg *et al.*, 1997).

As a consequence of these developments, the attention has been turned away from the local occlusal and dental factors mentioned above, and focused more on systemic factors. Muscle tenderness might be related to prolonged central sensory hyperexcitability and changes in central processing resulting from a peripheral injury (Reid *et al.*, 1994; Ren and Dubner 2000). Other studies illustrated the association between temporomandibular problems, segmental limitations of the cervical spine (especially in the C0-C3 region) and cervical muscle myalgia (De Laat *et al.*, 1998). In this study, the best discriminator between patients and controls appeared a skin folding test, reported as "painful" by most of the patients and none of the controls. This might reflect an overall increase in sensitivity towards an otherwise non-painful stimulus. Recent years have been characterized by studies focusing on the influence or possible etiologic role of female hormones (Leresche *et al.*, 1997; Dao and Leresche, 2000; Cimino *et al.* 2000;

Isselee *et al.* 2001) in an attempt to elucidate why females outnumber males up to 10 times in patient populations. Slight fluctuations over the menstrual phases, which appear to be of limited clinical significance have been reported in symptom-free subjects and patients with myofascial pain.

DIAGNOSIS OF MUSCLE PAIN

Since no metabolic or immunologic markers are available, and in correspondence with the data on muscular activity (Lund *et al.*, 1989), which make the use of EMG recordings useless at the present time, the clinical diagnosis of masticatory myalgia is made by algometry. Manual palpation (Dworkin *et al.*, 1990) and several kinds of algometers allow a consistent and significant difference between groups with and without muscle pain. It should be noted, however, that higher counts of tender points are seen after pressure algometry when compared to digital palpation (Cott *et al.*, 1992). This might result in confusion for diagnoses in which the number of tender points is conclusive (Wolfe *et al.*, 1990, Dworkin and Leraesche, 1992). Pressure algometry has been proven reliable tool (Jensen *et al.*, 1992) and reproducible but highly variable between subjects (Isselee *et al.*, 1997).

TMJ disk displacements and related pain

A renewed interest on the association between TMJ disk displacements and signs and symptoms of TMD (Farrar, 1971) led to an increased focus on disc position and, consequently, treatments aimed at reestablishing "normal" relationships between disc and condyle. A clinical distinction has been made between a (usually anteromedial) displacement of the disc *with* reduction (clicking joint) and *without* reduction (closed lock). The latter situation could result in the presence or absence of limitation of mouth opening (Dworkin and Leresche, 1992). The motivation for early diagnosis and treatment of disc displacements was based on the assumption that the anterior position of the disc was directly related to the occurrence of pain, limitation of mandibular movement (in case of non-reducing disc displacements), and the development of osteoarthritis. Recently, however, the importance of disc position and the relationship between disc displacement and degenerative joint disease have been revisited, which changed the interpretation of as well as the need for elaborated diagnostic techniques and treatment.

EPIDEMIOLOGICAL FINDINGS AND NATURAL COURSE

Even if comparison between studies is hampered by the lack of standardization regarding definition and diagnosis of joint sounds, it is clear that the symptom is very common in the general population

(30-50%). In addition, recent longitudinal data obtained in adolescents (Könönen *et al.*, 1996, Magnusson *et al.* 2000) and patients (Greene and Laskin, 1988), indicate that TMJ clicking only rarely develops into joint locking and that the symptom might be a bad predictor of such locking. Clicking of the TMJ appears to be a cyclic and poorly predictable symptom, which does not warrant treatment as such (Okeson, 1996).

TMJ DISC DISPLACEMENT AND PAIN

Movements of the jaw in patients with disc displacement lead to increased pain, which suggests that traction or pressure on the ligaments and retrodiscal tissues are the main cause of the pain (Tenenbaum *et al.*, 1999). Clicking and even locking, however, is very often not accompanied by pain, which questions this direct relationship. A possible explanation might be offered when micro- or macrotrauma is considered as the main etiology for disc displacements: if the internal derangement develops *slowly*, the neighbouring tissues will gradually adapt to the altered biomechanics without pain, while in case of *sudden* or massive trauma pain will occur. Long-term longitudinal studies have indicated that non-invasive procedures yield long-lasting results in most patients, regardless of disk position (de Leeuw *et al.*, 1994; Kai *et al.*, 1998).

TMJ DISK DISPLACEMENT AND OSTEOARTHRITIS

As mentioned before, the interaction between internal derangement and the development of degenerative changes is still unclear (Stegenga, 2001). Both in primary and secondary osteoarthritis, a mechanical, biochemical, inflammatory or immunologic insult disturbs the equilibrium between form and function maintained by continuous remodeling, and as a result cartilage breakdown occurs (de Bont, 1996). In this view, the displacement of the disc might be considered an etiologic (co-) factor because of the possible overload of condylar cartilage, but also a sign of osteoarthritis, where the altered sliding properties of the cartilage or deterioration of the synovial fluid give rise to friction, wear, and possibly disk displacement.

DIAGNOSIS OF TMJ DISK DISPLACEMENT

Disc displacement with and without reduction are *clinical* diagnoses. Clicking of the TMJ can be traced using manual palpation or stethoscopy with fair to good intra- and interobserver reliability (Dworkin *et al.*, 1990). Electronic devices have proven higher reliability, but are not necessary in clinical settings. For non-reducing discs, the diagnosis may be complicated in patients with joint hypermobility, where the limitation of mouth

opening or the asymmetric lateral movements appear less marked. In these patients, MRI offers a reliable non-invasive tool without the hazard of radiation to study disc position (De Laat *et al.*, 1993). The use of this expensive technique should, however, be limited to these doubtful cases.

Degenerative/inflammatory disorders

The complex changes that take place in the articular fibrocartilage are illustrated in animal models and developments in molecular biological research start to elucidate the complex changes which occur at the level of the articular fibrocartilage, and the role of loading, matrix components, cytokines, and neuropeptides (Stegenga, 2001). Factors like female hormones, age, sympathetically mediated effects related to pain or psychological stress, trauma, systemic illness, diet and smoking are all mentioned to change the adaptive capacities of the TMJ. Local inflammatory processes lead to cartilage breakdown and damage of the joint, both in the inflammatory (e.g. rheumatoid arthritis) and degenerative disorders. Proteases, cytokines, growth factors and arachidonic acid appear to play an important role in maintaining normal tissue turnover (Mankin and Brandt, 1992 ; Kopp, 2000). The local peripheral nervous system appears not only to signal nociception, but also takes active part in the inflammatory process (neurogenic inflammation) (Sessle, 1999). The sympathetic fibers release substance P, calcitonin gene-related peptide (CGRP), neurokinin A and neuropeptide Y. These substances were found in the synovial fluid of arthritic patients (Appelgren *et al.*, 1991) and are thought to mediate and modulate the inflammatory disease and the concomitant pain (Kopp, 2000). Care should be taken, however, not to confuse between real markers of disease and products possibly resulting from the inflammatory process itself.

Management of masticatory disorders

A lack of prospective studies, randomized clinical trials, clear inclusion/exclusion criteria for subjects and standards for treatment outcome, have characterized research on the treatment of pain and dysfunction of the masticatory system until recently. Some principles with regard to management can be put forward :

- treatment goals are a decrease of pain, decreased loading of the masticatory system, and restored mandibular movements and oral function. In this respect, early treatment of significant signs and symptoms is advocated, to prevent chronicity. Chronic pain and dysfunction may lead to more psychosocial factors and altered care-seeking behavior (Fricton, 1991).

- since both physical and psychological contributing factors need to be considered, a multidisciplinary approach is advocated for both assessment and management of the disorders.
- as mentioned earlier, there is increasing evidence that signs and symptoms of TMD are self-limiting and resolve without apparent side-effects. Consequently, the use of non-invasive, reversible treatment procedures should be promoted over surgical interventions. Conservative treatment has proven efficacy in relieving pain and dysfunction in 50 to over 90% of patient populations examined (Okeson and Hayes, 1986 ; Kai *et al.*, 1998), also over longer periods of time (de Leeuw *et al.*, 1994)
- the position of the temporomandibular disc position seems less critical with regard to the development of pain (Tenenbaum *et al.*, 1999) and accumulating evidence suggests TMJ-clicking is not a determinant factor in the development of closed lock of the TMJ (Könönen *et al.*, 1996 ; Magnusson *et al.*, 2000). Intra-oral appliances or surgical techniques aimed at repositioning the disc into its “best” position, therefore, should reconsider their goal, and moreover have been reported to be only moderately successful in stabilizing the disc position or avoid TMJ-clicking on the long term (for review, see Dao and Lavigne, 1998).
- it is striking that comparable results are obtained with whatever treatment executed, which questions the value of intensive treatment in comparison with time- or placebo-effects. Only very few randomized clinical trials have been performed which indicate that biofeedback, antidepressant (amitriptyline) or relaxing (clonazepam) medication, and also acupuncture are more effective than placebo (see Dionne, 1997, for review). Conversely, no such studies could validate the reported clinical success of interocclusal appliances, NSAID's, muscle relaxant medication and various physical treatment procedures (Clark *et al.*, 1995). The real effect of arthroscopic lysis and lavage in comparison with non-invasive treatment is still under discussion (Stegenga *et al.*, 1993).

In conclusion, and based on the (limited) data available, a conservative, non-invasive, reversible approach appears effective in most patients.

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