Nomenclature and classification of temporomandibular joint disorders

B. STEGENGA  Departments of Oral and Maxillofacial Surgery and Dentistry and Oral Hygiene, University Medical Center Groningen, University of Groningen, The Netherlands

SUMMARY Currently, there are basically two approaches to classification, one based on structural and one on positional changes occurring within the joint. Despite the increase in knowledge of pathologic changes occurring within the temporomandibular joint (TMJ), the disc still seems to be a central issue in nomenclature and classifications of TMJ disorders. Basic pathologies of the TMJ involve inflammation and degeneration in arthritic disorders (irrespective of the presence or position of the disc) and structural aberrations in growth disorders. Some internal derangements may occur independent of underlying pathology, e.g. because of a traumatic event. In this position paper, a classification of TMJ disorders is proposed based on basic structural changes occurring in the joint.

KEYWORDS: temporomandibular joint disorders, classification

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Introduction

Diagnosing temporomandibular disorders (TMDs) basically consists of three consecutive phases (1). It starts with classification of the findings from the history and clinical examination into a broad diagnostic category, followed by specification of the physical disorder within this category (yielding a specific axis I diagnosis). The third phase involves individualisation by indicating the degree of function impairment (both physically and psychosocially) imposed by disorder (yielding the axis II diagnosis).

Currently it is commonly agreed to separate joint disorders from muscle disorders, although joint and muscle symptoms frequently occur simultaneously. This is also reflected in the current RDC/TMD groups of disorders (2).

This article focuses on the basis for the nomenclature for disorders affecting the temporomandibular joint (TMJ).

Current approaches to nomenclature

By definition, the TMJ is basically a complex synovial joint attributed to the presence of the articular disc. Anatomically, the disc plays a central role, being part of the articulating surfaces within both the lower and the upper joint compartment. Nevertheless, the disc became the central issue in diagnostic classifications no earlier than the last two decennia of the past century (3). Disc displacements appeared to explain many of the clinical signs and symptoms displayed by patients and since then played a central role in the diagnosis of TMDs (4). This way of thinking has led to an approach to TMD terminology focusing on intra-articular positional changes of the disc (internal derangements). These internal derangements explain most of the mechanical manifestations occurring in the joint, including:

1 Disc derangements (reducing, permanent), explaining clicking sounds and movement restriction because of the obstruction of condylar movement by the disc
2 Subluxation and luxation of the disc-condyle complex, representing the TMJ hypermobility disorders
Adherence, adhesions, and ankylosis of joint surfaces resulting in TMD characterised by hypomobility

These positional disorders are frequently classified separate from pathologies of the joint (5) (Table 1). With the increase in knowledge of pathologic changes occurring within the joint (6), it is striking that the disc still seems to be a central issue in many schools of thought today, including the RDC/TMD (2).

Structurally, the TMJ is a system of interdependent connective tissues with two major specific functions, i.e. the control of mandibular positions and movements during jaw functions, and contribution of the growth of the mandible (7). It is therefore not surprising that another approach to TMD terminology, focusing on pathologic changes of connective tissues, results in two major categories of structural disorders:

1 Arthritic disorders (i.e. inflammatory disorders affecting the joint), which are mainly characterised by pain and function impairment
2 Growth disorders, which are mainly characterised by facial asymmetry

The nomenclature of disorders and systems to classify them are ideally based on aetiology. However, the aetiology of most TMDs is unknown. As a result, many aetiologic theories have been suggested, most of which are lacking sound scientific evidence. Currently, there seems to be consensus that TMDs have a multifactorial aetiology, which in fact is somewhat misleading, because evidence for more than one simultaneous aetiologic moment is also lacking.

### Joint loading and adaption

Whatever the aetiologic event may be, most joint disorders display a disturbance of the balance that exists between joint loading on the one hand, and the adaptive capacity of the connective tissues on which loads are imposed on the other (Fig. 1). Basically there are two ways in which this disturbance may occur (7, 8):

1 Absolute overloading (Fig. 1a): excessive physical stress imposed on tissues with normal adaptive capacity (9, 10), for example because of traumatic injury, or possibly because of functional loading (11) or parafunctional loads (12)
2 Relative overloading (Fig. 1b): normal physical stress imposed on connective tissues with reduced adaptive capacity, for example because of ageing (13), systemic disease (e.g. autoimmune diseases, metabolic disorders), hormonal changes (14), and genetic factors.

What is actually happening when a joint is loaded? The loading is imposed on connective tissues, each of which has a specific capacity to adapt (8). When loading occurs within the limits of adaptive capacity, synthesis of new tissue will remain in balance with tissue breakdown, in this way maintaining tissue integrity. So joint tissues are changing continuously, but tissue integrity is maintained. The mechanisms that are involved include modelling and remodelling (15). Sometimes compensatory changes occur. These responses may be referred to as ‘adaptive’.

### Table 1. Approaches to classification of articular temporomandibular disorders

<table>
<thead>
<tr>
<th>Type</th>
<th>Aetiology</th>
<th>Pathology</th>
<th>Clinical manifestations</th>
<th>Terminology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structural</td>
<td>Overloading</td>
<td>Inflammation</td>
<td>Arthralgia (inflammation)</td>
<td>Arthritic disorders</td>
</tr>
<tr>
<td></td>
<td>Traumatic</td>
<td>Low-grade</td>
<td>Function impairment</td>
<td>Low-grade</td>
</tr>
<tr>
<td></td>
<td>Functional</td>
<td>High-grade</td>
<td>(because of pain and internal derangements)</td>
<td>Osteoarthritis</td>
</tr>
<tr>
<td></td>
<td>Systemic disease</td>
<td>Degeneration</td>
<td>Facial deformity</td>
<td>Traumatic arthritis</td>
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<tr>
<td></td>
<td>Autoimmune</td>
<td>Deformation</td>
<td></td>
<td>High-grade</td>
</tr>
<tr>
<td></td>
<td>Metabolic</td>
<td></td>
<td></td>
<td>Infectious arthr</td>
</tr>
<tr>
<td></td>
<td>Structural abberation</td>
<td></td>
<td></td>
<td>RA</td>
</tr>
<tr>
<td>Positional</td>
<td>Tissue deformation</td>
<td>Deformation</td>
<td>Arthralgia (strain)</td>
<td>Metabolic arthr</td>
</tr>
<tr>
<td></td>
<td>Trauma</td>
<td></td>
<td>Clicking</td>
<td>Growth disorders</td>
</tr>
<tr>
<td></td>
<td>Adherence of surfaces</td>
<td></td>
<td>Closed lock</td>
<td>Internal derangements</td>
</tr>
<tr>
<td></td>
<td>Structural disease</td>
<td></td>
<td>Hypermobility</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Open lock</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hypomobility/adhesion – ankylosis</td>
<td></td>
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</table>
On the other hand, when the joint is loaded to an extent beyond the limits of a tissue’s adaptive capacity, synthesis cannot keep pace with increased breakdown, which will result in tissue of inferior quality (by definition: degeneration) or to (permanent) deformation of tissues. Compensatory mechanisms cannot avoid these changes to occur. In other words, the response to loading is now ‘maladaptive’, resulting in pathologic changes.

Pathologic changes of the TMJ

During the past years, the evidence for several mechanisms occurring in synovial joints in general and the TMJ in particular is rapidly accumulating (6, 16). The major events occurring in joints when tissues respond to loading with maladaptive changes include the following:

1. Imbalance of matrix-metalloproteinases and their tissue inhibitors (17, 18) which are regulated by vascular endothelial growth factor; this factor has been shown to be expressed in the pathologic TMJ osteoarthritis (19, 20). This results in a relative surplus of enzymes breaking down the tissue matrices (i.e. collagenases, proteolytic enzymes).

2. Production of several inflammatory mediators, such as interleukins (21–23).

3. Release of several neuropeptides, such as substance P and calcitonin-related peptides (24).

There is an intimate relationship between changes occurring within the articular cartilage and inflammatory changes within the synovial membrane.

So, by several interacting molecular mechanisms, a maladaptive response to loading may result in pathologic changes of the cartilage matrix, the subchondral bone, and the soft tissues associated with the joint. The major pathologic changes include inflammation, degeneration and deformation (25).

Clinical manifestations

These pathologic changes may eventually result in clinical manifestations, of which pain, mechanical derangements (which are especially obvious in joints with a disc or meniscus such as the TMJ and the knee joint), and facial deformity are the most prominent.

Pain of the joint is represented in the RDC/TMD classification group IIIa by the general (non-diagnostic) term ‘arthralgia’. Most commonly, joint pain occurs as a consequence of inflammatory changes, and the corresponding diagnostic term should be ‘arthritis’. These inflammatory changes may be related to degenerative changes, but also attributed to trauma, systemic disease, and irritation of soft tissues associated with the joint. Other, less common, forms of joint pain include pain because of strain of ligaments, neuropathic pain, and pain referred from a distant source (e.g. muscle pain referred to the joint area).

Mechanical derangements represent a group of clinical manifestations that are covered in the RDC/TMD group II. These conditions may include derangements of the disc (i.e. disarranged conditions focusing on a mechanical disturbance because of the disc displacement), accounting for common clinical signs as clicking and locking.

Facial deformity may range from very subtle forms (for example in TMJ osteoarthritis, which is a gradual process in which dimensional differences between the affected and non-affected side may eventually develop), to severe and readily visible deformations.
changes mainly result from loss of posterior vertical dimension as a consequence of condylar resorption or degeneration. Usually this occurs gradually and the dentition is able to adapt, resulting in tilting of the occlusal plane. When these changes occur rapidly, the major sign is an anterior open bite. However, usually deformations occur as a result from growth disorders of the joint (e.g. condylar hyperplasia).

Implications for nomenclature and classification

In the literature, two broad groups of disorders are distinguished, focusing on structural and positional changes, respectively. Structural changes result from either absolute (traumatic or functional) or relative overloading where the adaptive capacity of the affected tissues is impaired, e.g. because of systemic disease. Positional changes (becoming manifest as mechanical derangements) mainly result from tissue deformation because of trauma or, more gradually, resulting from structural disease. These structural conditions display pathologic changes, of which inflammation of various grades is the most common. Structural conditions may become clinically manifest as pain, function impairment because of mechanical derangements, and facial deformity.

The proper terms that should be used for structural TMJ changes are ‘arthritic disorders’ (including osteoarthritis, traumatic arthritis, infectious arthritis, rheumatoid arthritis, metabolic arthritis, depending on the underlying cause or mechanism), and growth disorders (including condylar hyperplasia, and several tumours which fortunately are relatively rare). Positional changes present as internal derangements, such as clicking, locking, hypermobility, and hypomobility, mainly owing to obstruction by the disc or disc-condyle complex. These disorders may occur in isolation (e.g., because of trauma or related to a connective tissue anomaly), but are usually the result (and manifestation) of an underlying pathologic process.

To decide on the terminology that we should use to designate articular temporomandibular disorders, we should address several issues.

First, we should make the choice to base our diagnostic terminology on structural (pathology-based) changes or on their clinical manifestations. The RDC/TMD are now a mixture of pathological and clinical entities. To distinguish specific diagnostic groups, terms such as ‘arthalgia’ (RDC group IIIa) and ‘disc derangements’ (RDC group II) are not suitable, because they represent non-specific clinical manifestations of underlying disease processes.

Second, and more importantly, the aim of a diagnosis is to provide a basis for effective treatment. The objectives for managing structural conditions are to re-establish the balance between synthesis and breakdown, and to optimise the circumstances for repair and healing. These objectives can be achieved by controlling risk factors (mainly related to over-loading), reducing pain (i.e., anti-inflammatory medication and arthrocentesis), and improving function (educating exercises, physical therapy). On the other hand, current treatment modalities do not affect the position of the disc. So, despite the general focus on diagnosing the position of the disc, treatment is mainly focused on structural changes, although it would seem rational to direct treatment of positional changes at restoring the altered position of the disc. This poses the question as to why it is necessary to distinguish between several diagnostic categories of disc displacement (such as in RDC/TMD group II disorder) in the first place!

Conclusion

Basic pathologies of the TMJ involve inflammation and degeneration in arthritic disorders (irrespective of the presence or position of the disc!) and structural aberrations in growth disorders. These pathologic changes may become clinically manifest as pain and internal derangements. Some internal derangements may occur separate from an underlying pathologic process, e.g. because of a traumatic event or related to congenital or developmental ligamentous laxity. Our current treatment approaches mainly focus on influencing the pathologic changes and do not normally alter positional changes of the disc.

Therefore, it appears that the most rational approach is to classify TMJ disorders in the following main categories (Table 2):

1. Arthritic disorders, consisting of low-grade and high-grade conditions (26, 27). The arthritic disorders are characterised mainly by pain, and later in the disease course possibly by internal derangements and in some cases facial deformity. Treatment is aimed at controlling risk factors and the inflammatory response.

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2 Growth disorders, which are mainly characterised by facial deformity. Treatment is aimed at removing the tumour and correcting of the deformity. Therefore, usually surgery is indicated.

3 Non-arthritic disorders in which the mechanical derangement is the main characteristic. Examples include luxation (possibly related to structural joint laxity) and acute (traumatic) disc dislocation. Treatment is specifically aimed at reducing the mechanical obstruction.

References


Table 2. Classification of articular temporomandibular disorders

<table>
<thead>
<tr>
<th>Category</th>
<th>Clinical characteristics</th>
<th>Diagnosis based on...</th>
</tr>
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<tbody>
<tr>
<td>Low-grade arthritic disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>Mild–moderate pain</td>
<td>Risk factors (overloading, trauma)</td>
</tr>
<tr>
<td>Traumatic arthritis</td>
<td>Possible internal derangement</td>
<td>Clinical characteristics</td>
</tr>
<tr>
<td></td>
<td>Possible mild facial deformity</td>
<td>Imaging (local degeneration) Synovial fluid analysis (?)</td>
</tr>
<tr>
<td>High-grade arthritic disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>Moderate–severe pain</td>
<td>Risk factors (e.g. infection, systemic disease)</td>
</tr>
<tr>
<td>Infectious arthritis</td>
<td>Possible internal derangement</td>
<td>Clinical characteristics</td>
</tr>
<tr>
<td>Metabolic arthritic disorders</td>
<td>Possible facial deformity</td>
<td>Imaging (diffuse degeneration) Synovial fluid/blood analysis</td>
</tr>
<tr>
<td>Growth disorders incl. Neoplasms</td>
<td>Swelling</td>
<td>Risk factors (e.g. ligamentous laxity)</td>
</tr>
<tr>
<td>Non-arthritic mechanical disorders</td>
<td>Internal derangement</td>
<td>Clinical characteristics</td>
</tr>
<tr>
<td></td>
<td>(hypermobility, luxation, hypomobility, acute disc dislocation)</td>
<td></td>
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</tbody>
</table>


Correspondence: Boudewijn Stegenga, University Medical Center, Department of Oral & Maxillofacial Surgery, PO Box 30-001, 9700 RB Groningen, The Netherlands. E-mail: b.stegenga@khir.umcg.nl