University of Groningen

Nomenclature and classification of temporomandibular joint disorders

Stegenga, B.

Published in:
Journal of Oral Rehabilitation

DOI:
[10.1111/j.1365-2842.2010.02146.x](https://doi.org/10.1111/j.1365-2842.2010.02146.x)

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2010

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Stegenga, B. (2010). Nomenclature and classification of temporomandibular joint disorders. *Journal of Oral Rehabilitation*, 37(10), 760-765. <https://doi.org/10.1111/j.1365-2842.2010.02146.x>

Copyright

Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: <https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment>.

Take-down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): <http://www.rug.nl/research/portal>. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

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

Accepted for publication 10 July 2010

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

Position paper. Presented at the International RDC/TMD consortium – IADR – Toronto 2008.

3 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 aetiological 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 aetiological moment is also lacking.

Joint loading and adaption

Whatever the aetiological 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

Type	Aetiology	Pathology	Clinical manifestations	Terminology
Structural	Overloading	Inflammation	Arthralgia (inflammation)	Arthritic disorders
	Traumatic	Low-grade	Function impairment	Low-grade
	Functional	High-grade	(because of pain and internal	Osteoarthritis
	Systemic disease	Degeneration	derangements)	Traumatic arthritis
	Autoimmune	Deformation	Facial deformity	High-grade
	Metabolic			Infectious arthr
	Structural aberration			RA
Positional				Metabolic arthr
				Growth disorders
	Tissue deformation	Deformation	Arthralgia (strain)	Internal derangements
	Trauma	Degeneration?	Clicking	
	Adherence of surfaces		Closed lock	
	Structural disease		Hypermobility	
			Open lock	
			Hypomobility/adhesion – ankylosis	

Fig. 1. Balance between joint loading and adaptive capacity (AC). (a) Absolute overloading: increased load, normal AC. (b) Relative overloading: normal load, decreased AC.

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. These

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 'arthralgia' (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 overloading), 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.

Table 2. Classification of articular temporomandibular disorders

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

- 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

1. Stegenga B. Temporomandibular joint degenerative diseases: clinical diagnosis. In: Stegenga B, de Bont LGM, eds. *Management of temporomandibular joint degenerative diseases: biologic basis and treatment outcome*. Basel: Birkhauser; 1996:13.
2. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders. Review, criteria, examination and specifications. *J Craniomandib Disord*. 1992;6:301–355.
3. Farrar WB, McCarty WJ. *A clinical outline of temporomandibular joint diagnosis and treatment*. Montgomery AL: Normandie; 1982.
4. Osborn JW. The disc of the temporomandibular joint: design, function and failure. *J Oral Rehabil*. 1985;43:889–898.
5. De Leeuw R (ed). *Orofacial pain: guidelines for classification, assessment, and management*. 4th ed. Chicago: Quintessence; 2008.
6. Milam SB. TMJ Osteoarthritis. In: Laskin DM, Greene CS, Hylander WL, eds. *TMDs: an evidence-based approach to diagnosis and treatment*. Chicago: Quintessence; 2006:105–124.
7. Stegenga B. Osteoarthritis of the temporomandibular joint organ and its relationship to disc displacement. *J Orofac Pain*. 2001;15:193–205.
8. Stegenga B, de Bont LGM, Boering G, van Willigen JD. Tissue responses to degenerative changes in the temporomandibular joint. *J Oral Maxillofac Surg*. 1991;49:1079–1088.
9. Stegenga B, de Bont LGM, Boering G. Osteoarthrosis as the cause of craniomandibular pain and dysfunction: a unifying concept. *J Oral Maxillofac Surg*. 1989;47:249–256.
10. Nitzan DW. The process of lubrication impairment and its involvement in temporomandibular joint disc displacement: a theoretical concept. *J Oral Maxillofac Surg*. 2001;59:36–45.
11. Milam SB, Schmitz JP. Molecular biology of temporomandibular joint disorders: proposed mechanisms of disease. *J Oral Maxillofac Surg*. 1995;53:1448–1454.
12. Gallo LM, Chiaravallotti G, Iwasaki LR, Nickel JC, Palla S. Mechanical work during stress-field translation in the human TMJ. *J Dent Res*. 2006;85:1006–1010.
13. Tanaka E, Sasaki A, Tahmina K, Yamaguchi K, Mori Y, Tanne K. Mechanical properties of human articular disk and its influence on TMJ loading studied with finite element method. *J Oral Rehabil*. 2001;28:273–279.
14. LeResche L. Epidemiology of temporomandibular disorders: implications for the investigation of etiologic factors. *Crit Rev Oral Biol Med*. 1997;8:291–305.
15. Stegenga B, de Bont LGM. TMJ Growth, adaptive modeling and remodeling, and compensatory mechanisms. In: Laskin DM, Hylander WL, Greene C, eds. *TMDs. An evidence-based approach to diagnosis and treatment*. Chicago: Quintessence; 2006:53–67.
16. Haskin CL, Milam SB, Cameron IL. Pathogenesis of degenerative joint disease in the human temporomandibular joint. *Crit Rev Oral Biol Med*. 1995;6:248–277.
17. Zardeneta G, Milam SB, Lee T, Schmitz JP. Detection and preliminary characterization of matrix metalloproteinase activity in temporomandibular joint lavage fluid. *Int J Oral Maxillofac Surg*. 1998;27:397–403.
18. Pufe T, Harde V, Peterson W, Goldring MB, Mentlein R. Vascular endothelial growth factor (VEGF) induces matrix metalloproteinase expression in immortalized chondrocytes. *J Pathol*. 2004;202:367–374.

19. Tanaka E, Aoyama J, Miyauchi M, Takata T, Hanaoka K, Iwabe T. Vascular endothelial growth factor plays an important autocrine/paracrine role in the progression of osteoarthritis. *Histochem Cell Biol.* 2005;123:275–281.
20. Sato J, Segami N, Kaneyama K, Mashiyama Y, Fujimura K, Yoshitake Y. Vascular endothelial growth factor concentrations in synovial fluids of patients with symptomatic internal derangement of the temporomandibular joint. *J Oral Pathol Med.* 2005;34:170–177.
21. Kubota E, Kubota T, Matsumoto J, Shibata T, Murakami KI. Synovial fluid cytokines and proteinases as markers of temporomandibular joint disease. *J Oral Maxillofac Surg.* 1998;56:192–198.
22. Sato J, Segami N, Nishimura M. Expression of interleukin 6 in synovial tissues in patients with internal derangement of the temporomandibular joint. *Br J Oral Maxillofac Surg.* 2003;41:95–101.
23. Hamada Y, Kondoh T, Holmlund AB, Yamamoto M, Horie A, Saito T. Inflammatory cytokines correlated with clinical outcome of temporomandibular joint irrigation in patients with chronic closed lock. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2006;102:596–601.
24. Alstergren P, Appलगren A, Appलगren B, Kopp S, Lundberg T, Theodorsson E. Co-variation of neuropeptide Y, calcitonin gene-related peptide, substance P and neurokinin A in joint fluid from patients with temporomandibular joint arthritis. *Arch Oral Biol.* 1995;40:127–135.
25. De Bont LGM, Dijkgraaf LC, Stegenga B. Epidemiology and natural progression of articular temporomandibular disorders. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1997;83:72–76.
26. Mercuri LG. Surgical management of TMJ arthritis. In: Laskin DM, Greene CS, Hylander WL, eds. *TMDs: an evidence-based approach to diagnosis and treatment.* Chicago: Quintessence; 2006:455–468.
27. Tanaka E, Detamore MS, Mercuri LG. Degenerative disorders of the temporomandibular joint: etiology, diagnosis and treatment. *J Dent Res.* 2008;87:296–307.

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