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Chapter 1

General Introduction



Clinicians are often faced by patients exhibiting signs and symptoms of temporomandibular disorders (TMDs). TMD is a term embracing a number of clinical problems involving the temporomandibular joint (TMJ), muscles of mastication, and associated structures (Okeson, 1996). Signs and symptoms of TMDs include muscle and/or joint pain, and functional disturbances, such as TMJ sounds on movement, limited mandibular mobility, and deviations in jaw movement (McNeill, 1993). Functional disturbances within the TMJ are, after muscle pain, the second most common form of TMD (De Leeuw, 2008). These disturbances are characteristic for so-called TMJ internal derangements (IDs), which are the topic of this thesis. In this chapter, first, the normal anatomy and function of the TMJ is described. Secondly, the term internal derangement is defined, and the two most common types of IDs are introduced- TMJ hypermobility and anterior disc displacement with reduction. Thereafter, aspects of the diagnosis, mechanisms and risk factors, and prognosis of these derangements are discussed. This chapter ends with a synopsis of the general aims of the thesis.

The temporomandibular joint

The temporomandibular joint is one of the most complex articulations in the human body. It is a synovial, paired, load-bearing joint, which underneath its capsule consists of the following structures (fig. 1): *condylus mandibularis*, *fossa mandibularis*, *discus articularis*, and *eminentia articularis* (Gray, 1918). The mandibular condyle and fossa are covered by a layer of dense fibrocartilage. The articular disc, also consisting of dense fibrous connective tissue, is placed at the region of high load between the condyle and the fossa. Within the disc, three zones are recognized: (1) an anterior disc band, in which, in some joints, attachments of the superior head of the lateral pterygoid muscle merge (Meyenberg *et al.*, 1986), and which fuses with the anterior part of the articular capsule (Obrez and Gallo, 2006); (2) an intermediate zone, which is the thinnest and most loaded part of the disc; and (3) a posterior disc band that is normally the thickest discal portion (Hansson *et al.*, 1977). Posteriorly, the articular disc continues as the bilaminar zone (Rees, 1954). Its inferior layer attaches the disc to the posterior part of the collum mandibulae through a fibrous tissue, rich with collagen fibres. The superior layer of the bilaminar zone connects the disc with the posterior wall of the mandibular fossa and contains elastine fibres that unfold and stretch during forward movement of the disc-condyle complex at mouth opening. The articular disc separates the intra-articular space into two chambers filled with synovial fluid: an upper discotemporal compartment, and a lower discomandibular compartment.

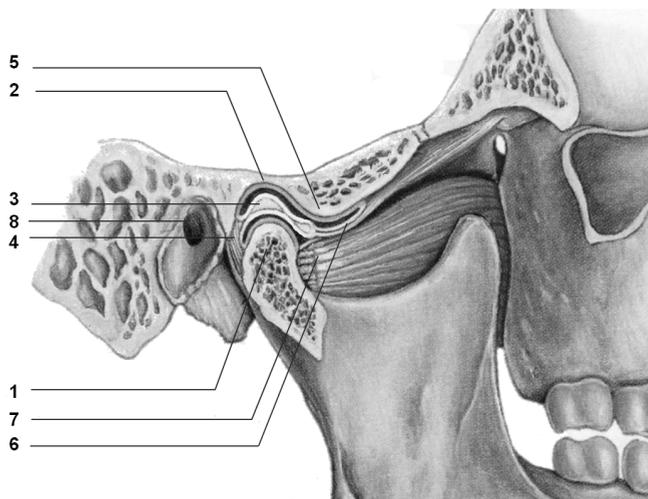


Figure 1. Normal anatomy of the human temporomandibular joint and its surrounding structures. 1- condylus mandibularis; 2- fossa mandibularis; 3- discus articularis; 4- bilaminar zone; 5- eminentia articularis; 6- anterior part of the joint capsule; 7- m. pterygoideus lateralis; 8- meatus acusticus externus.

The structure of the temporomandibular joint allows the performance of complex movements with a high degree of freedom. Mandibular motion results from combined condylar rotatory and translatory movements that occur respectively, in the lower and the upper TMJ compartment (Hylander, 1992). Mandibular movements are driven by the muscles of mastication, and are limited by the joint capsule and some of the TMJ ligaments (viz., the temporomandibular and stylomandibular ligaments) (Hylander, 2006), as well as by the stretched jaw-closing muscles at maximum mouth opening (Koolstra *et al.*, 2001). Smooth mandibular motion, however, can be hampered by the presence of so-called TMJ internal derangements.

Internal derangements of the TMJ

Internal derangements (IDs) of the TMJ are, from an anatomical point of view, deviations in position or form of the intra-articular tissues (The glossary of prosthodontic terms, 2005). Clinically, IDs are only manifested when they interfere with normal, smooth mandibular movements (McNeil, 1993). The two most prevalent types of IDs, encountered in nearly 40% of the adult population, are TMJ hypermobility and anterior disc displacement with reduction (ADDR), with the latter being the most prevalent TMJ derangement (Huddleston Slater *et al.*, 2007).

TMJ hypermobility

From an anatomical view point, TMJ hypermobility has been defined as an excessive mobility of the joint (The glossary of prosthodontic terms, 2005), with the mandibular condyle traveling beyond the articular eminence. According to another, clinical definition, TMJ hypermobility is noted as such only when functional interferences on movement are present (i.e., clicking joint sounds in the last part of mouth opening and first part of closing, and jerky mandibular movements when the condyle snaps along the articular eminence). In the latter definition, because of the presence of clinical signs, this internal derangement is referred to as symptomatic TMJ hypermobility (Huddleston Slater *et al.*, 2004, 2007; Kalaykova *et al.*, 2006). Hypermobility is usually considered a harmless TMJ internal derangement, except for cases, when the condyle stays locked in front of the eminence, and cannot be repositioned by the patient him/herself (an open lock, TMJ dislocation, or luxation) (for a review, see Shorey & Campbell, 2000).

Anterior disc displacement with reduction

In a TMJ with an ADDR, in the closed mouth position, the articular disc is located anteriorly to the condyle, instead of being on top of it. During mouth opening, the disc reduces by slipping back on top of the condyle. At the end of closing, the disc gets again anteriorly displaced (Farrar and McCarty, 1982). Clinically, an ADDR is accompanied merely by reciprocal clicking joint sounds at the moments of disc reduction and dislocation, and is usually a non-problematic condition (Okeson, 1996). However, an ADDR may cause serious complaints if the disc reduction gets hampered (an anterior disc displacement without reduction, ADDWoR). The latter may result in a severe and often painful mouth opening limitation (a closed lock) (Farrar and McCarthy, 1982). The loss of disc reduction can be permanent, but also temporary, because subjects with an ADDR sometimes report periods of sudden, transient, and recurrent mouth opening limitations (intermittent locking) (Westesson and Lundh, 1989).

Diagnosis

In order to gain insight into the risk factors and prognosis of IDs, and the conditions, under which those become problematic to ID patients, it is important to be able to distinguish between the types of TMJ derangements. TMJ IDs could be recognized by visualization of the intra-articular relations, for example, by using magnetic resonance imaging (MRI) (Larheim, 2005), or by detection of ID clinical signs, for example, on a clinical TMJ

examination or on mandibular movement recordings (Mauderli *et al.*, 1988; Huddleston Slater *et al.*, 2004). MRI is considered the gold standard for TMJ ID diagnostics (Larheim, 2005). It is, however, an expensive and elaborate technique with a low availability, on which the intra-articular relationships are sometimes difficult to interpret (Palla, 2009). Therefore, clinical examination is the diagnostic method that is most widely used in daily practice, and in research with large study samples (Huddleston Slater *et al.*, 2007).

In 1992, standardized clinical criteria for ADDR were introduced as part of the so-called Research Diagnostic Criteria for TMD (RDC/TMD; Dworkin and Le Resche, 1992). No scientific rationale was, however, presented to form the basis for these criteria, nor have these criteria been validated so far. Moreover, TMJ hypermobility was not included into the RDC/TMD system, thereby disabling the differential diagnostics between these two most common IDs that present with similar signs (*viz.*, reciprocal clicking). As recently the need for new, thoroughly revised diagnostic criteria for TMD was recognized (Steenks and De Weijer, 2009), in **Chapter 2**, the current RDC/TMD for ADDR are evaluated and modified, based upon evidence accumulated while performing numerous mandibular movement recordings in patients and controls with or without ADDRs.

Mechanisms and risk factors

Symptomatic TMJ hypermobility has been related to the ability of a condyle to move in front of, and superior to, the crest of the articular eminence, where snapping of the condyle over the eminence crest would be the mechanism responsible for the interferences with smooth mandibular movements (McNeill, 1993; Huddleston Slater *et al.*, 2004). The role of this mechanism in the symptoms of TMJ hypermobility is studied in **Chapter 3**. In this chapter, it is tested whether the condyles of symptomatically hypermobile subjects are translated further beyond the crest of the articular eminence than those of non-hypermobile subjects.

In the etiology of ADDR, several risk factors have been suggested, among which TMJ loading and microtraumatic events resulting from oral parafunctions (Stegenga and De Bont, 2006), hypermobility (Johansson and Isberg, 1991; Hirsch *et al.*, 2008), and changes in intra-articular relations during bodily growth in adolescence (Huddleston Slater *et al.*, 2007). TMJ loading could also be of influence on the reducing capacity of the TMJ once an ADDR has developed, because the loss of molar support and tooth-wear have been found associated with a loss of disc reduction (Lundh *et al.*, 1987). In **Chapter 4**, the role of oral parafunctions and symptomatic TMJ hypermobility as possible risk factors for ADDR and intermittent locking is assessed in an adolescent population sample. Moreover, in **Chapter**

5, in an experimental study, the effect of TMJ loading by means of an intensive chewing exercise upon the reduction of the anteriorly displaced disc is tested.

Prognosis

A large number of longitudinal studies on the course of TMD signs and symptoms have concluded that joint clicking is a fluctuating phenomenon that usually does not lead to locking (Wänman and Agerberg, 1990; Könönen *et al.*, 1996; Kitai *et al.*, 1997; Henrikson and Nilner, 2003; Magnusson *et al.*, 2005). In those studies, however, no differential diagnosis was made between the types of IDs causing the joint clicking, and therefore, no inferences are possible regarding the prognosis of ADDR or TMJ hypermobility. Other authors have found that in patients with a history of intermittent locking, the ADDR clicking may disappear over time (Lundh *et al.*, 1987; Westesson and Lundh, 1989; Sato *et al.*, 2003). While in some patients, ADDR clicking was replaced by a long-lasting locking, in others, clicking had disappeared with the preservation of normal joint mobility (Lundh *et al.*, 1987; Sato *et al.*, 2003). Because no TMJ imaging was performed at the disappearance of clicking, it was unknown which functional and/or morphological alterations within the TMJ were responsible for this disappearance. Had the ADDR been converted to an ADDWoR without the symptoms of locking, or conversely, had the disc position within the TMJ got normalized? In **Chapter 6**, the two-year natural course of ADDR was studied. The hypothesis was tested that the disappearance of clicking is related to a gradual loss of reducing capacity of the disc.

Synopsis

The topic of this thesis is internal derangements of the temporomandibular joint. In several chapters, aspects of the diagnosis, mechanisms and risk factors, and natural course of these TMJ derangements will be studied. The aim of the thesis is three-fold: (1) to evaluate the current clinical criteria for the recognition of ADDR (**Chapters 2**); (2) to study the mechanisms and risk factors for symptomatic TMJ hypermobility, ADDR, and the loss of disc reduction (**Chapters 3, 4 and 5**); and (3) to study the natural course of ADDR (**Chapter 6**).

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