ELSEVIER
SAUNDERS

Oral Maxillofacial Surg Clin N Am 20 (2008) 159–168

ORAL AND
MAXILLOFACIAL
SURGERY CLINICS
of North America

Internal Derangements of the Temporomandibular Joint

Reny de Leeuw, DDS, PhD

Orofacial Pain Center, University of Kentucky, College of Dentistry, 800 Rose Street, Lexington, KY 40536-0297, USA

Definition of various internal derangements

Internal derangement is defined as any interference with smooth joint movement. Although the term therefore includes all types of intracapsular interferences that impede smooth functional joint movements, with regard to the temporomandibular joint (TMJ) the term is typically used interchangeably with disc displacement. In this article, several other types of internal derangements are discussed. Besides the common derangements, disc adherences, disc adhesions, subluxations, and dislocations of the disc–condyle complex are also discussed (Table 1).

Stegenga and de Bont [1] most recently made a case for a name change of the phenomenon of disc displacement. They argued that adhering to the so-called ''12 o'clock position'' as the normal position of the posterior band of the disc relative to the condyle might lead to overdiagnosis of this particular disorder, because a displaced disc does not always lead to clinical symptoms. They therefore proposed to replace the word ''displacement'' with ''derangement,'' which would indicate that the displaced disc actually interferes with smooth joint movement and causes some type of dysfunction to the individual. Imaging studies have found disc displacements in 23% to 33% of asymptomatic volunteers [2–4].

A disc derangement is defined as a malpositioning of the articular disc relative to the condyle and eminence. Theoretically, a disc may be displaced to varying degrees and in any direction (ie, anterior, posterior, lateral, or medial). Rarely is a disc displaced purely in one direction, with the possible exception of anterior displacement. Posterior displacements have been described but are infrequent [4–7]. Pure sideways displacements seem to be rare also [2,8] and may be related to more advanced stages of derangement [9]. The most common type of disc derangement is an anterior displacement [4]. Controversy exists about which type of displacement is next most common. Some studies report more often anteromedial derangements [10,11], some found more often anterolateral derangements [2,8], and others report an even distribution of anteromedial and anterolateral derangements in patients and healthy volunteers [4].

With regard to clinical diagnosis and treatment, two predominant stages of disc derangements are distinguished. The respective conditions are called disc derangement with reduction and disc derangement without reduction. In a normal TMJ, the disc is positioned over the condylar head with the posterior band situated in the 12 o'clock (superior to the condyle) position and the intermediate zone situated in the 1 o'clock (superior-anterior to the condyle) position. On opening the disc–condyle complex translates in a forward direction. Although the condyle also rotates forward, the disc relatively rotates in a posterior direction over the condyle. Disc derangement with reduction is typically defined as a condition in which the articular disc of the TMJ is (most often anteriorly) displaced while the mouth is closed and the teeth are together in maximal occlusion. On opening, the condyle pushes against the posterior band of the disc until the condyle is able to slide or snap under the posterior band of the disc, and the disc reduces to its position on top

E-mail address: rdele0@email.uky.edu

doi:10.1016/j.coms.2007.12.004

Table 1
Definitions of internal derangements of the temporomandibular joint

Disc derangement	A malpositioning of the articular disc relative to the condyle and eminence
With reduction	The articular disc resumes its normal position on top of the condyle on opening
Without reduction	The articular disc remains malpositioned on opening attempts, resulting in restricted mouth opening in acute cases
Disc adherence	A temporary sticking of the disc either to the fossa or to the condyle
Disc adhesion	A fibrotic connection between the disc and the condyle or the disc and the fossa
Subluxation (hypermobility)	An overextension of the disc–condyle complex on opening beyond the eminence
Joint dislocation	A dislocation of the entire disc–condyle complex beyond the eminence combined with the inability to return passively into the fossa

of the condyle. Overcoming the thick posterior band of the disc is believed to be responsible for the clicking or popping sound. On closing the mouth, the disc stays behind and slips off the condyle, which may be accompanied by a clicking sound. Typically, the opening click occurs later during the opening movement, whereas the closing click often occurs close to maximal occlusion.

Disc derangement without reduction is defined as a condition in which the condyle is unable to slide or snap back underneath the disc. The (anteriorly) displaced disc thus does not reduce to its position on top of the condyle during the opening movement. The disc is obstructing further translation of the condyle and consequently the opening and contralateral movements are impaired.

Disc adherence is defined as a temporary sticking of the disc either to the fossa or to the condyle. This adherence can be caused by prolonged static loading or lack of lubrication or a combination thereof. Oftentimes patients report difficulties with jaw opening on awakening. On attempts to move the jaw, generally the adherence can be overcome; this is often accompanied by a loud single pop or click. The condition should not be confused with disc derangement with reduction or subluxation. Another condition that has similar characteristics has been termed anchored disc phenomenon [12]. It is believed that the disc is stuck to the fossa because of compromised lubrication. A strong adhesive force prevents the disc from being separated from the fossa or condyle by simply moving the jaw. This phenomenon may resemble a disc derangement without reduction, although a history of clicking is frequently absent and the limitation of mouth opening is said to be more severe [12]. Disc adhesion is defined as a fibrotic connection between the disc and the condyle, or the disc and the fossa. This condition is characterized by limited jaw movements. In contrast with an adherence, an adhesion cannot be overcome by simple jaw movements. This condition should be distinguished from disc derangement without reduction or fibrous ankylosis. For disc adherence or adhesion to occur the disc does not have to be deranged.

Subluxation (sometimes referred to as hypermobility) is defined as an overextension of the disc–condyle complex on opening. On opening, the disc–condyle complex passes beyond the eminence. Typically, this is accompanied by a dull thud-like sound. The sound may be reciprocal (ie, may occur on opening and on closing). The sound typically occurs, unlike in the case of disc derangement, late in the opening phase (almost at maximum opening) and early in the closing phase. The subluxation may be habitual, meaning that the disc–condyle complex passes the eminence back and forth without causing pain, discomfort, or dysfunction during routine opening. Several cross-sectional studies have attempted to relate generalized joint hypermobility to TMJ hypermobility, yet the results of a systemic review revealed that controversies about this continue [13].

A joint dislocation is similar to a subluxation in that the condyle–disc complex passes beyond the eminence. In this case, however, the patient is unable to close the mouth because the disc–condyle complex is trapped in front of the eminence.

Etiology/pathophysiology

The most common etiologic factor in the development of internal derangements is trauma. Macrotrauma, such as a hit or blow to the face, may result in direct tissue injury and immediate derangements of the TMJ components. Oral intubation and dental/surgical procedures that involve prolonged mouth opening or excessive forces, such as difficult extractions, also have the

propensity to cause direct tissue injury. In addition, such trauma could result in elongation of ligaments, creating internal joint laxity, which may set the TMJ up for a slower development of derangement. Under normal physiologic conditions, a balance exists in synovial joints between tissue breakdown and repair. When the balance is disturbed by a mechanical, biomechanical, or inflammatory insult the internal cartilaginous remodeling system may fail, resulting in accelerated tissue breakdown [14]. The intrinsic changes in the joint components may induce a disc derangement.

Microtrauma may be another etiologic factor in the development of internal derangements. Microtrauma is defined as application of prolonged repetitive forces, such as in clenching or grinding. The repetitive forces may result in tissue failure in several ways. When the force is within physiologic limits, but is applied to articular cartilage that has a reduced adaptive capability, or when the force exceeds the adaptive capability of normal cartilage, tissue degeneration may ensue [15].

Under normal physiologic conditions, a balance also exist between formation of free radicals and neutralizing mechanisms [16,17]. Mechanical loading of the joint may result in local hypoxia. Reperfusion of hypoxic cells can lead to an explosive increase in free radicals [16]. These free radicals may lead to degradation of hyaluronic acid, which is an important component of synovial fluid. Degradation of hyaluronic acid in turn may impair the lubrication of the TMJ [18]. The impaired lubrication may increase friction between the surfaces of the different joint components. Not only could this lead to adherences or an anchored disc but also it may precipitate a disc derangement [19]. In addition, it has been proposed that adhesions may be formed by free radical–mediated crosslinking of fibrinogen and fibronectin [20].

Indirect trauma, such as that related to acceleration-deceleration (whiplash) injuries in the absence of a direct trauma to the face, has been related to symptoms of temporomandibular disorder (TMD). The most recent prospective controlled study indicated that 1 out of 3 patients who had whiplash accidents without direct trauma to the head developed TMJ pain or associated symptoms within 1 year of the accident compared with 1 out of 16 of the control group [21]. In contrast, a previous prospective study showed that there were no differences between whiplash patients and controls at 6 months regarding TMJ pain and clicking [22].

Clinical diagnosis, including imaging and differential diagnosis

The most salient sign of a disc derangement with reduction is a repeatable, audible click on opening. Often there is also a click on closing, but this may be less noticeable. When there is a closing click it is called reciprocal clicking. On occasion the click is not audible but may be heard by auscultation. In addition, the shift in disc position may be felt by palpation. The click may coincide with a momentary deviation from the midline during the opening movement. Other signs of disc derangement with reduction may include a click on protrusion or lateral movements. Typically, the mouth opening is not restricted and this condition usually is not painful by itself [23].

Conventional radiographs may be used to rule out degenerative joint diseases. Generally, a disc derangement with reduction presents with no to mild radiographically visible degenerative changes. These changes are mostly limited to flattening and sclerosis [24]. The clinical diagnosis of disc derangement can be confirmed by soft tissue imaging, although the mild nature of this condition does not necessitate routine soft tissue imaging. Differential diagnosis may include adhesions, deviation in shape, or arthritis.

The most salient sign of an acute disc derangement without reduction is sudden limited mouth opening. The patient's history in the case of disc derangement without reduction is essential and usually includes a sudden cessation of clicking accompanied by a limitation of mouth opening. The clinical signs are related to the obstruction of translation of the condyle by the disc. They include a limited mouth opening, limited contralateral movement, deflection on protrusion, and opening to the ipsilateral side [23,25]. This condition is often accompanied by pain. Palpation of the affected joint may be painful and reveals a limited translation of the obstructed condyle.

Generally, an acute disc derangement without reduction presents with no to mild radiographically visible degenerative changes. If the derangement persists and becomes chronic, usually pain subsides and joint mobility increases, even without treatment [26,27]. Mobility-related clinical signs and symptoms may gradually fade, although crepitus may emerge [28]. At the same time, degenerative changes may become radiographically visible [24]. These changes are generally moderate, consist predominantly of flattening and sclerosis of the load-bearing areas of the joint, and most

often stabilize over a few years [24,29,30]. In cases with severe degenerative changes, occlusal disturbances, such as anterior open bite or a unilateral open bite, may develop. In general, the visible osteoarthritic changes should be seen as articular remodeling in an attempt for the joint to establish a new balance between form and function [30]. Conventional radiographs may be used to identify degenerative changes. The position and shape of the disc can be observed with soft tissue imaging, but unless surgery is planned, routine imaging is not warranted. Differential diagnosis may include arthritis, disc adherences or adhesions, fibrotic ankylosis, myospasm, and neoplasia.

Adherences are difficult to diagnose clinically. The most important information, the transient inability to open the mouth wide after a period of static loading, resolved by moving or manipulating the jaw, and the accompanying single pop, may be derived from the history. Rarely do these types of adherences occur while the patient is in the clinic. The disc may be anchored for a more prolonged time when the patient is unable to overcome the adhesive force between the surfaces of the disc and the fossa [12]. In such cases, the patient presents with limited mouth opening, deflection to the ipsilateral side on opening and protrusion, and restricted contralateral movements. The restriction of the mouth opening may be more severe than in the case of disc derangement without reduction because the adhesive force prevents any translation in the joint and mobility is strictly dictated by rotation of the condyle. Technically speaking, adherences can occur in the lower joint space also, in which case rotation of the condyle is impaired. The clinical presentation of a patient who has an adherence in the lower joint space is similar to that of a patient who has an upper joint space adherence. Because movement of the condyle is now dictated by translation, the movement may be irregular with a sensation of stiffness.

Conventional radiography is not indicated to detect adherences but may be used to rule out degenerative changes. Magnetic resonance imaging may be used to depict a static (ie, nonmoving) disc. Differential diagnosis may include disc derangements.

Persistence of adherences and articular tissue changes related to osteoarthritis may result in adhesions [20]. Adhesions are common in painful TMJs with limited mobility that are refractory to conservative therapies [31]. Adhesions are difficult to diagnose clinically, and the history and clinical findings obtained from the patient who has adhesions may not differ from those of a patient who has disc derangement without reduction or anchored disc.

Adhesions can be detected by arthroscopy, magnetic resonance arthrography, and plain film arthrography, although medial adhesions seem to be more difficult to discern with the latter technique [32]. Differential diagnosis may include disc derangement without reduction, prolonged adherence, and fibrous ankylosis.

A joint dislocation is commonly known as an ''open lock.'' The patient is unable to close the mouth or fully occlude. There may be considerable variability in the extent that the mouth remains open. The patient may present with a prognathic profile and class III malocclusion attributable to the protrusion of the mandible.

A conventional radiograph, such as a transpharyngeal or transcranial projection with ''closed'' mouth, reveals that the condyle is dislocated in front of the eminence.

Treatment and prognosis

If a disc derangement with reduction is asymptomatic (ie, the patient is not bothered by it) this condition does not warrant treatment [33]. Clicking and disc derangement may persist for several years without any progression and without development of radiographically visible degenerative changes [24,28]. In addition, signs and symptoms of temporomandibular disorders in the general population fluctuate considerably over time and rarely result in significant dysfunction [34,35]. Taking into account the possibility of spontaneous resolution, the initial treatment should be limited to nonaggressive measures, such as counseling (reassurance and education, rest, instructions to avoid loading, control of contributing factors, such as parafunctional habits) and mild analgesic or anti-inflammatory medicines [36].

Several authors report the use of anterior repositioning appliances for the treatment of disc derangement with reduction with variable rates of success [37–41]. An anterior repositioning appliance is indicated in a patient who has painful clicking or frequent locking, if the clicking can be eliminated by having the patient close down in a protrusive position [42]. The patient is instructed to open the mouth; on doing so the deranged disc reduces to its position on top of the condyle. The patient is then allowed to close the mouth while keeping the mandible protruded, which prevents the disc from deranging again.

The ultimate treatment position is defined by the least protrusive position in which the disc remains in place. The correctness can be checked by having the patient open and close in the chosen position. The click should not reoccur when the position is chosen correctly. The anterior repositioning appliance should be worn at nighttime only. The goal of this treatment is to allow the retrodiscal tissues to recover and adapt and to bring the patient back to a pain-free clicking state. This procedure is not intended to recapture the disc or eliminate clicking permanently, because this has been shown to be unattainable [38,39,43–45].

In the case of an acute disc derangement without reduction, an attempt should be made to unlock the patient. This can be done by gently manipulating the affected TMJ in a downward and then medial direction. During this manipulation the disc may snap back onto the condylar head. Sometimes this procedure is sufficient and the patient may not lock for some time. If the patient locks again immediately on closing, however, an anterior repositioning appliance may be indicated. In such a case, after the patient has been unlocked, the appliance should be made immediately and worn full time for about 5 days, after which the patient should gradually reduce the use of the appliance to avoid malocclusion. Several surgical options are available for disc derangement with reduction; however, they are rarely indicated. These procedures are described below because they may apply to disc derangement with and without reduction.

If in the case of an acute disc derangement without reduction manipulation is unsuccessful to unlock the jaw, there are many nonsurgical and surgical options to choose from. Studies show that if minimal treatment is provided [28,29,46], or even if nothing is done [26,27,47], the signs and symptoms gradually improve. Treatment speeds up the natural progression process, however, and is primarily aimed at restoring the balance between form and function, also called adaptation [30]. Similar to disc derangement with reduction, nonaggressive measures, such as reassurance and education, rest, instructions to avoid loading, control of contributing factors (such as parafunctional habits), and mild analgesic or antiinflammatory medicines, should be provided as first-line treatment [36]. An interocclusal appliance (ie, a stabilization appliance) may also be helpful if the patient complains of pain that is worse on awakening. Physical therapy aimed at reduction of pain and dysfunction may be indicated also [48]. Once the patient is pain free, additional treatments aimed at reducing the limited mouth opening should be implemented. These may consist of simple stretching exercises within a pain-free range performed by the patient, or manipulation techniques performed by a physical therapist. In rare cases the restriction of mouth opening is persistent and more aggressive therapies may be needed. Several studies show that a disc derangement without reduction responds well to nonsurgical treatment [28,30,49,50]. If these nonsurgical treatments fail, however, an evaluation for the appropriateness of surgical intervention is indicated.

As outlined in the Clinical Practice Guidelines for TMJ surgery of the American Association of Oral and Maxillofacial Surgeons, TMJ surgery is only indicated when nonsurgical therapy has been ineffective. TMJ surgery is not indicated for asymptomatic or minimally symptomatic cases and should not be performed in an attempt to prevent TMD [51]. Indications for surgery include moderate to severe pain or dysfunction that is disabling [51]. Most often surgery is indicated for persistent painful intracapsular conditions. When clicking is interfering significantly with the patient's quality of life, however, and nonsurgical procedures have been ineffective to improve the patient's quality of life, surgical procedures may also be indicated.

Although the guidelines state that surgical procedures are indicated only after reasonable efforts with nonsurgical modalities have failed, there may be one exception. When it is debatable whether the diagnosis is disc derangement without reduction or disc adherence, an arthrocentesis may be performed. An arthrocentesis is a minimally invasive procedure typically associated with good outcomes for patients who have a closed lock [52]. A disc adherence may be instantly released by such a procedure and recurrence of the adherence seems to be infrequent [53]. A locked jaw because of a deranged disc may also be resolved, although most frequently the improvement of dysfunction and mouth opening are not related to an improved disc–condyle relationship [54–58]. Although there are studies promoting the use of sodium hyaluronate during arthrocentesis, there are no controlled trials indicating that the use of sodium hyaluronate results in significantly better treatment outcomes than arthrocentesis alone [59].

Most surgical procedures for disc derangements are aimed at improving the disc–condyle

relationship. Such procedures include modified condylotomy, discoplasty, and disc repositioning. Another procedure includes discectomy with or without replacement of the removed disc.

Discectomy has long been advocated to reduce the mechanical problems related to disc derangements. Discectomies have resulted in elimination of pain and improvement of function. Success rates vary from 43% to 93% for this procedure [60–66]. Significant radiographically visible degenerative changes, which resemble those associated with degenerative joint disease, may occur with this procedure [61,66]. There seems to be no advantage to replacing the disc with temporary or permanent implants or grafts [61,67–70]. Evidence of the formation of a pseudodisc after discectomy has been reported [65,71].

The modified condylotomy consists of an intraoral vertical ramus osteotomy, in which the condylar segment is allowed to move slightly inferiorly [72]. This supposedly creates space to allow for a better disc–condyle relationship. The advantage of this procedure is that it avoids intracapsular surgery. The disadvantage of the procedure is that it is hard to predict what the position of the condyle will be and whether the condyle–disc relationship will improve. This procedure is generally only recommended in early stages of disc derangements with reduction [73], when it is more likely that the disc still has its original saddlelike shape. Disc derangements in the absence of pain generally do not warrant treatment, and several more conservative treatment options have proven successful in the treatment of early disc derangements. The indications and use of this particular procedure are therefore limited.

Discoplasty is a surgical open-joint procedure in which the disc is repaired or its shape improved. Oftentimes this procedure is used concomitant with disc repositioning techniques. Although reports on pain and dysfunction are favorable, relapse of disc position is frequent [74,75]. Disc repair and disc repositioning can also be achieved with arthroscopy [76]. Similar positive treatment outcomes were obtained in a randomized study wherein one group of patients received open-joint surgery with disc repositioning and the other group was treated arthroscopically with lysis, lavage, and capsular stretch [75]. Note that in the latter case the arthroscopic procedure did not aim to improve the disc–condyle relationship.

Arthroscopy is a minimally invasive closed-joint procedure. It has proven successful for treatment of different stages of derangements, even if the main goal was not aimed at improving the disc–condyle relationship [77–82]. Mobilization seems of more importance for reduction of signs and symptoms of derangements. With arthroscopy, the deranged disc can be repositioned or reshaped [83].

Adherences may be treated with reassurance and education, instructions to avoid static loading of the TMJs, and control of parafunctional habits. If the patient reports that the jaw is also locked on awakening, an interocclusal appliance (ie, a stabilization appliance) may be indicated. If the patient and the health care provider are unable to unlock the jaw, arthrocentesis is indicated. Adherences can also be alleviated with arthroscopy [83], although this procedure may rarely be indicated in lieu of arthrocentesis.

Adhesions are difficult to manage with nonsurgical measures. To break adhesions lysis, lavage, and hydraulic distention may be sufficient. More likely, arthroscopy will be necessary to release or ablate adhesions [83].

An acute joint dislocation is treated by manipulation of the jaw in a slightly forward, then downward, direction. Once sufficiently cleared from the eminence, the condyle-disc complex snaps back into the fossa. Sometimes it is sufficient to ask the patient simply to yawn, on which the joint may overcome the eminence and return to the fossa. The manipulation technique should be taught to the patient in addition to instructions to avoid opening wide. In case of a chronic dislocation, the patient may need to be put under general anesthesia to unlock the jaw. In addition, intermaxillary fixation may be needed to avoid relapse. When a patient suffers from uncontrollable repeated dislocations, treatment depends on the cause of these dislocations. Eminectomy, by way of arthroscope or the traditional open-joint surgery, has been a widely used method to treat recurrent or habitual dislocations [84–86]. Although this method was originally described in 1951 by Myrhaug [87], estimates of its efficacy are mostly based on small sample case series or case reports. Other methods used to reduce the recurrent or habitual dislocations include techniques aimed at creating a barrier to limit condylar translation [88–91] and injection of sclerosing agents [92]. Downfracturing of the eminence, placement of miniplates or screws, or cranial bone or other autografts have been used to create barriers. No randomized controlled trials or comparative trials were found in the English

literature regarding the efficacy and safety of these procedures. If dystonia or spasm of the lateral pterygoid is responsible for the dislocations, injections with botulinum toxin may be useful [93,94].

Summary with an emphasis/impact on oral surgeons

TMJ internal disc derangements most often respond well to nonsurgical methods. An asymptomatic click does not warrant treatment. In line with the Clinical Practice Guidelines for TMJ Surgery, surgical options should only be used in cases of moderate to severe persistent pain or dysfunction, after reasonable conservative treatment has proven ineffective. Possible exceptions are acute disc adherences, and adhesions, in which surgical methods, such as arthrocentesis and arthroscopy, may be the first treatment of choice. For disc derangements, a multitude of surgical procedures are available, but recent advances in technology and philosophy direct the surgical procedures of choice toward the minimally invasive technique, arthrocentesis, with arthroscopy as the next alternative.

References

[1] Stegenga B, de Bont LGM. TMJ disk derangements. In: Laskin DM, Greene CS, Hylander WL, editors. Temporomandibular disorders. An evidence-based approach to diagnosis and treatment. Chicago: Quintessence; 2006. p. 125–36.

[2] Tasaki MM, Westesson PL, Isberg AM, et al. Classification and prevalence of temporomandibular joint disk displacement in patients and symptom-free volunteers. Am J Orthod Dentofacial Orthop 1996;109(3):249–62.

[3] Katzberg RW, Westesson PL, Tallents RH, et al. Anatomic disorders of the temporomandibular joint disc in asymptomatic subjects. J Oral Maxillofac Surg 1996;54(2):147–53 [discussion: 153–5].

[4] Westesson PL, Larheim TA, Tanaka H. Posterior disc displacement in the temporomandibular joint. J Oral Maxillofac Surg 1998;56(11):1266–73 [discussion: 1273–4].

[5] Blankestijn J, Boering G. Posterior dislocation of the temporomandibular disc. Int J Oral Surg 1985;14(5): 437–43.

[6] Huddleston Slater JJ, Lobbezoo F, Hofman N, et al. Case report of a posterior disc displacement without and with reduction. J Orofac Pain 2005;19(4): 337–42.

[7] Chiba M, Watanabe N, Echigo S. Longitudinal MRI follow-up of non-reducible posterior disc displacement accompanied by bone marrow oedema in the mandibular condyle. Dentomaxillofac Radiol 2007;36(5):304–7.

[8] Whyte AM, McNamara D, Rosenberg I, et al. Magnetic resonance imaging in the evaluation of temporomandibular joint disc displacement—a review of 144 cases. Int J Oral Maxillofac Surg 2006;35(8): 696–703.

[9] Sener S, Akganlu F. MRI characteristics of anterior disc displacement with and without reduction. Dentomaxillofac Radiol 2004;33(4):245–52.

[10] Larheim TA, Westesson P, Sano T. Temporomandibular joint disk displacement: comparison in asymptomatic volunteers and patients. Radiology 2001;218(2):428–32.

[11] Nebbe B, Major PW. Prevalence of TMJ disc displacement in a pre-orthodontic adolescent sample. Angle Orthod 2000;70(6):454–63.

[12] Nitzan DW, Etsion I. Adhesive force: the underlying cause of the disc anchorage to the fossa and/or eminence in the temporomandibular joint—a new concept. Int J Oral Maxillofac Surg 2002;31(1):94–9.

[13] Dijkstra PU, Kropmans TJ, Stegenga B. The association between generalized joint hypermobility and temporomandibular joint disorders: a systematic review. J Dent Res 2002;81(3):158–63.

[14] Dijkgraaf LC, de Bont LG, Boering G, et al. The structure, biochemistry, and metabolism of osteoarthritic cartilage: a review of the literature. J Oral Maxillofac Surg 1995;53(10):1182–92.

[15] Stegenga B, de Bont LG, Boering G. Osteoarthrosis as the cause of craniomandibular pain and dysfunction: a unifying concept. J Oral Maxillofac Surg 1989;47(3):249–56.

[16] Milam SB. Pathogenesis of degenerative temporomandibular joint arthritides. Odontology 2005; 93(1):7–15.

[17] Milam SB, Zardeneta G, Schmitz JP. Oxidative stress and degenerative temporomandibular joint disease: a proposed hypothesis. J Oral Maxillofac Surg 1998;56(2):214–23.

[18] Nitzan DW. The process of lubrication impairment and its involvement in temporomandibular joint disc displacement: a theoretical concept. J Oral Maxillofac Surg 2001;59(1):36–45.

[19] Nitzan DW. ''Friction and adhesive forces''—possible underlying causes for temporomandibular joint internal derangement. Cells Tissues Organs 2003; 174(1–2):6–16.

[20] Dijkgraaf LC, Zardeneta G, Cordewener FW, et al. Crosslinking of fibrinogen and fibronectin by free radicals: a possible initial step in adhesion formation in osteoarthritis of the temporomandibular joint. J Oral Maxillofac Surg 2003;61(1):101–11.

[21] Sale H, Isberg A. Delayed temporomandibular joint pain and dysfunction induced by whiplash trauma: a controlled prospective study. J Am Dent Assoc 2007;138(8):1084–91.

[22] Kasch H, Hjorth T, Svensson P, et al. Temporomandibular disorders after whiplash injury: a controlled, prospective study. J Orofac Pain 2002; 16(2):118–28.

[23] Okeson JP. Orofacial pain, guidelines for assessment, diagnosis, and management. Chicago: Quintessence Pub; 1996.
[24] de Leeuw R, Boering G, Stegenga B, et al. Radiographic signs of temporomandibular joint osteoarthrosis and internal derangement 30 years after nonsurgical treatment. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79(3):382–92.
[25] Stegenga B, de Bont LG, Boering G. Classification of temporomandibular joint osteoarthrosis and internal derangement. 2. Specific diagnostic criteria. Cranio 1992;10(2):107–16 [discussion: 116–7].
[26] Sato S, Nasu F, Motegi K. Natural course of nonreducing disc displacement of the temporomandibular joint: changes in chewing movement and masticatory efficiency. J Oral Maxillofac Surg 2002;60(8): 867–72.
[27] Sato S, Takahashi K, Kawamura H, et al. The natural course of nonreducing disk displacement of the temporomandibular joint: changes in condylar mobility and radiographic alterations at one-year follow up. Int J Oral Maxillofac Surg 1998;27(3):173–7.
[28] de Leeuw R, Boering G, Stegenga B, et al. Clinical signs of TMJ osteoarthrosis and internal derangement 30 years after nonsurgical treatment. J Orofac Pain 1994;8(1):18–24.
[29] Boering G. Temporomandibular joint osteoarthrosis. Groningen (Netherlands): Van Denderen; 1994.
[30] Kai S, Kai H, Tabata O, et al. Long-term outcomes of nonsurgical treatment in nonreducing anteriorly displaced disk of the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85(3):258–67.
[31] Israel HA, Langevin CJ, Singer MD, et al. The relationship between temporomandibular joint synovitis and adhesions: pathogenic mechanisms and clinical implications for surgical management. J Oral Maxillofac Surg 2006;64(7):1066–74.
[32] Zhang S, Yang C, Zheng J, et al. Plain film arthrography applied to the diagnosis of intra-articular adhesions of the temporomandibular joint. J Oral Maxillofac Surg 2007;65(2):212–7.
[33] Greene CS, Laskin DM. Long-term status of TMJ clicking in patients with myofascial pain and dysfunction. J Am Dent Assoc 1988;117(3):461–5.
[34] Kononen M, Waltimo A, Nystrom M. Does clicking in adolescence lead to painful temporomandibular joint locking? Lancet 1996;347(9008):1080–1.
[35] Magnusson T, Egermarki I, Carlsson GE. A prospective investigation over two decades on signs and symptoms of temporomandibular disorders and associated variables. A final summary. Acta Odontol Scand 2005;63(2):99–109.
[36] de Bont LG, Dijkgraaf LC, Stegenga B. Epidemiology and natural progression of articular temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83(1):72–6.
[37] Lundh H, Westesson PL, Kopp S, et al. Anterior repositioning splint in the treatment of temporomandibular joints with reciprocal clicking: comparison with a flat occlusal splint and an untreated control group. Oral Surg Oral Med Oral Pathol 1985;60(2):131–6.
[38] Moloney F, Howard JA. Internal derangements of the temporomandibular joint. III. Anterior repositioning splint therapy. Aust Dent J 1986;31(1): 30–9.
[39] Tallents RH, Katzberg RW, Macher DJ, et al. Use of protrusive splint therapy in anterior disk displacement of the temporomandibular joint: a 1- to 3-year follow-up. J Prosthet Dent 1990;63(3):336–41.
[40] Williamson EH. Temporomandibular dysfunction and repositioning splint therapy. Prog Orthod 2005;6(2):206–13.
[41] Tecco S, Caputi S, Tete S, et al. Intra-articular and muscle symptoms and subjective relief during TMJ internal derangement treatment with maxillary anterior repositioning splint or SVED and MORA splints: a comparison with untreated control subjects. Cranio 2006;24(2):119–29.
[42] Okeson JP. Management of temporomandibular disorders and occlusion. 6th edition. St. Louis (MO): Mosby; 2006.
[43] Lundh H, Westesson PL, Kopp S. A three-year follow-up of patients with reciprocal temporomandibular joint clicking. Oral Surg Oral Med Oral Pathol 1987;63(5):530–3.
[44] Okeson JP. Long-term treatment of disk-interference disorders of the temporomandibular joint with anterior repositioning occlusal splints. J Prosthet Dent 1988;60(5):611–6.
[45] Eberhard D, Bantleon HP, Steger W. The efficacy of anterior repositioning splint therapy studied by magnetic resonance imaging. Eur J Orthod 2002; 24(4):343–52.
[46] Rasmussen OC. Description of population and progress of symptoms in a longitudinal study of temporomandibular arthropathy. Scand J Dent Res 1981;89(2):196–203.
[47] Sato S, Kawamura H, Nagasaka H, et al. The natural course of anterior disc displacement without reduction in the temporomandibular joint: follow-up at 6, 12, and 18 months. J Oral Maxillofac Surg 1997;55(3):234–8 [discussion: 238–9].
[48] Suvinen TI, Hanes KR, Reade PC. Outcome of therapy in the conservative management of temporomandibular pain dysfunction disorder. J Oral Rehabil 1997;24(10):718–24.
[49] Minakuchi H, Kuboki T, Matsuka Y, et al. Randomized controlled evaluation of non-surgical treatments for temporomandibular joint anterior disk displacement without reduction. J Dent Res 2001;80(3):924–8.
[50] Murakami K, Kaneshita S, Kanoh C, et al. Ten-year outcome of nonsurgical treatment for the internal derangement of the temporomandibular joint with closed lock. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94(5):572–5.

[51] Parameters of care for oral and maxillofacial surgery. A guide for practice, monitoring and evaluation (AAOMS Parameters of Care-92). American Association of Oral and Maxillofacial Surgeons. J Oral Maxillofac Surg 1992;50(7 Suppl 2):i–xvi 1–174.

[52] Al-Belasy FA, Dolwick MF. Arthrocentesis for the treatment of temporomandibular joint closed lock: a review article. Int J Oral Maxillofac Surg 2007; 39(9):773–82.

[53] Nitzan DW, Samson B, Better H. Long-term outcome of arthrocentesis for sudden-onset, persistent, severe closed lock of the temporomandibular joint. J Oral Maxillofac Surg 1997;55(2):151–7 [discussion: 157–8].

[54] Alpaslan C, Dolwick MF, Heft MW. Five-year retrospective evaluation of temporomandibular joint arthrocentesis. Int J Oral Maxillofac Surg 2003; 32(3):263–7.

[55] Alpaslan GH, Alpaslan C. Efficacy of temporomandibular joint arthrocentesis with and without injection of sodium hyaluronate in treatment of internal derangements. J Oral Maxillofac Surg 2001;59(6): 613–8 [discussion: 618–9].

[56] Emshoff R, Rudisch A, Bosch R, et al. Effect of arthrocentesis and hydraulic distension on the temporomandibular joint disk position. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89(3): 271–7.

[57] Hosaka H, Murakami K, Goto K, et al. Outcome of arthrocentesis for temporomandibular joint with closed lock at 3 years follow-up. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82(5): 501–4.

[58] Carvajal WA, Laskin DM. Long-term evaluation of arthrocentesis for the treatment of internal derangements of the temporomandibular joint. J Oral Maxillofac Surg 2000;58(8):852–5 [discussion: 856–7].

[59] Shi Z, Guo C, Awad M. Hyaluronate for temporomandibular joint disorders. Cochrane Database Syst Rev 2003;(1) CD002970.

[60] Bjornland T, Larheim TA. Discectomy of the temporomandibular joint: 3-year follow-up as a predictor of the 10-year outcome. J Oral Maxillofac Surg 2003;61(1):55–60.

[61] Dimitroulis G. The use of dermis grafts after discectomy for internal derangement of the temporomandibular joint. J Oral Maxillofac Surg 2005;63(2): 173–8.

[62] Eriksson L, Westesson PL. Discectomy as an effective treatment for painful temporomandibular joint internal derangement: a 5-year clinical and radiographic follow-up. J Oral Maxillofac Surg 2001; 59(7):750–8 [discussion: 758–9].

[63] Mazzonetto R, Spagnoli DB. Long-term evaluation of arthroscopic discectomy of the temporomandibular joint using the Holmium YAG laser. J Oral Maxillofac Surg 2001;59(9):1018–23 [discussion: 1024].

[64] Nyberg J, Adell R, Svensson B. Temporomandibular joint discectomy for treatment of unilateral internal derangements—a 5 year follow-up evaluation. Int J Oral Maxillofac Surg 2004;33(1):8–12.

[65] Takaku S, Sano T, Yoshida M. Long-term magnetic resonance imaging after temporomandibular joint discectomy without replacement. J Oral Maxillofac Surg 2000;58(7):739–45.

[66] Widmark G, Dahlstrom L, Kahnberg KE, et al. Diskectomy in temporomandibular joints with internal derangement: a follow-up study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83(3): 314–20.

[67] Eriksson L, Westesson PL. Temporomandibular joint diskectomy. No positive effect of temporary silicone implant in a 5-year follow-up. Oral Surg Oral Med Oral Pathol 1992;74(3):259–72.

[68] Fricton JR, Look JO, Schiffman E, et al. Long-term study of temporomandibular joint surgery with alloplastic implants compared with nonimplant surgery and nonsurgical rehabilitation for painful temporomandibular joint disc displacement. J Oral Maxillofac Surg 2002;60(12):1400–11 [discussion: 1411–2].

[69] Trumpy IG, Lyberg T. Surgical treatment of internal derangement of the temporomandibular joint: long-term evaluation of three techniques. J Oral Maxillofac Surg 1995;53(7):740–6 [discussion: 746–7].

[70] Schliephake H, Schmelzeisen R, Maschek H, et al. Long-term results of the use of silicone sheets after diskectomy in the temporomandibular joint: clinical, radiographic and histopathologic findings. Int J Oral Maxillofac Surg 1999;28(5):323–9.

[71] Hansson LG, Eriksson L, Westesson PL. Magnetic resonance evaluation after temporomandibular joint diskectomy. Oral Surg Oral Med Oral Pathol 1992; 74(6):801–10.

[72] Hall HD. Modification of the modified condylotomy. J Oral Maxillofac Surg 1996;54(5):548–51 [discussion: 551–2].

[73] Hall HD, Navarro EZ, Gibbs SJ. Prospective study of modified condylotomy for treatment of nonreducing disk displacement. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89(2):147–58.

[74] Montgomery MT, Gordon SM, Van Sickels JE, et al. Changes in signs and symptoms following temporomandibular joint disc repositioning surgery. J Oral Maxillofac Surg 1992;50(4):320–8.

[75] Politi M, Sembronio S, Robiony M, et al. High condylectomy and disc repositioning compared to arthroscopic lysis, lavage, and capsular stretch for the treatment of chronic closed lock of the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103(1):27–33.

[76] McCain JP, Podrasky AE, Zabiegalski NA. Arthroscopic disc repositioning and suturing: a preliminary report. J Oral Maxillofac Surg 1992;50(6):568–79 [discussion: 579–80].

[77] Holmlund AB, Axelsson S, Gynther GW. A comparison of discectomy and arthroscopic lysis and lavage for the treatment of chronic closed lock of

the temporomandibular joint: a randomized outcome study. J Oral Maxillofac Surg 2001;59(9): 972–7 [discussion: 977–8].

[78] Kurita K, Goss AN, Ogi N, et al. Correlation between preoperative mouth opening and surgical outcome after arthroscopic lysis and lavage in patients with disc displacement without reduction. J Oral Maxillofac Surg 1998;56(12):1394–7 [discussion: 1397–8].

[79] Miyamoto H, Sakashita H, Miyata M, et al. Arthroscopic surgery of the temporomandibular joint: comparison of two successful techniques. Br J Oral Maxillofac Surg 1999;37(5):397–400.

[80] Murakami K, Segami N, Okamoto M, et al. Outcome of arthroscopic surgery for internal derangement of the temporomandibular joint: long-term results covering 10 years. J Craniomaxillofac Surg 2000;28(5):264–71.

[81] Murakami KI, Tsuboi Y, Bessho K, et al. Outcome of arthroscopic surgery to the temporomandibular joint correlates with stage of internal derangement: five-year follow-up study. Br J Oral Maxillofac Surg 1998;36(1):30–4.

[82] Smolka W, Iizuka T. Arthroscopic lysis and lavage in different stages of internal derangement of the temporomandibular joint: correlation of preoperative staging to arthroscopic findings and treatment outcome. J Oral Maxillofac Surg 2005;63(4):471–8.

[83] Koslin MG. Advanced arthroscopic surgery. Oral Maxillofac Surg Clin North Am 2006;18(3):329–43.

[84] Sensoz O, Ustuner ET, Celebioglu S, et al. Eminectomy for the treatment of chronic subluxation and recurrent dislocation of the temporomandibular joint and a new method of patient evaluation. Ann Plast Surg 1992;29(4):299–302.

[85] Holmlund AB, Gynther GW, Kardel R, et al. Surgical treatment of temporomandibular joint luxation. Swed Dent J 1999;23(4):127–32.

[86] Sato J, Segami N, Nishimura M, et al. Clinical evaluation of arthroscopic eminoplasty for habitual dislocation of the temporomandibular joint: comparative study with conventional open eminectomy. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95(4):390–5.

[87] Myrhaug H. A new method of operation for habitual dislocation of the mandible; review of former methods of treatment. Acta Odontol Scand 1951; 9(3–4):247–60.

[88] Bakardjiev A. Treatment of chronic mandibular dislocations by bone plates: two case reports. J Craniomaxillofac Surg 2004;32(2):90–2 [discussion: 93].

[89] Fernandez-Sanroman J. Surgical treatment of recurrent mandibular dislocation by augmentation of the articular eminence with cranial bone. J Oral Maxillofac Surg 1997;55(4):333–8 [discussion: 338–9].

[90] Shibata T, Yamashita T, Nakajima N, et al. Treatment of habitual temporomandibular joint dislocation with miniplate eminoplasty: a report of nine cases. J Oral Rehabil 2002;29(9):890–4.

[91] Guven O. Inappropriate treatments in temporomandibular joint chronic recurrent dislocation: a literature review presenting three particular cases. J Craniofac Surg 2005;16(3):449–52.

[92] Matsushita K, Abe T, Fujiwara T. OK-432 (Picibanil) sclerotherapy for recurrent dislocation of the temporomandibular joint in elderly edentulous patients: case reports. Br J Oral Maxillofac Surg 2007;45(6):511–3.

[93] Daelen B, Thorwirth V, Koch A. Treatment of recurrent dislocation of the temporomandibular joint with type A botulinum toxin. Int J Oral Maxillofac Surg 1997;26(6):458–60.

[94] Moore AP, Wood GD. Medical treatment of recurrent temporomandibular joint dislocation using botulinum toxin A. Br Dent J 1997;183(11–12): 415–7.