CHAPTER 8

Pain and dysfunction of the temporomandibular joint

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1. Introduction

Temporomandibular disorders (TMD) refer to various conditions affecting the temporomandibular joint (TMJ), masticatory muscles and contiguous tissue components. Two common types of painful TMD are encountered: myogenous or muscle-generated pain (see Chapter 7), and arthrogenous or joint-generated pain. As discussed in Chapter 7 and below, the use of the term TMD is problematic; it forcefully integrates what are probably distinct biological entities into one diagnostic family. The pooling of data from epidemiologic, clinical and therapeutic studies of 'TMD' is therefore misleading. An overview of the overall epidemiology and general characteristics of TMDs was presented in Chapter 7.

This chapter focuses on pain and dysfunction originating in the TMJ proper, particularly as a result of disc derangements or osteoarthritis. Other relatively rare joint-related entities that may present as TMJ pain are extremely important in differential diagnosis but are beyond the scope of this chapter; some have been partly covered in other chapters (Table 8.1).

When discussing TMJ disorders the term 'internal derangement' is often used as a diagnosis. However, internal derangement is a classification, not a diagnosis, and includes disc displacements (joint derangements) and degeneration of the TMJ (Stegenga 2001). Joint *derangement* refers to a disarranged condition, focusing on a disturbance in mechanical operation. For accurate diagnosis the structures and the mechanical problem must be defined; for example disc displacement with reduction. On the other hand, joint *degeneration* is associated with disintegration or tissue damage to the joint constituents, as is the case in TMJ osteoarthritis.

Many patients report the onset of TMJ symptoms subsequent to trauma; others describe an insidious onset with no apparent cause. However, accumulating data suggest that many TMJ disorders may primarily be due to overuse or overloading of the system. We review current thinking on pathogenesis, diagnosis and treatment and discuss pain and dysfunction of the TMJ based upon the biomechanical and biochemical events underlying pain and joint dysfunction. Although knowledge of the aetiology of TMJ disorders is limited, the prognosis of most TMJ problems is good and many patients improve spontaneously in terms of both signs and symptoms. Our treatment approach should therefore be in line with this natural process. Adopting a conservative treatment with an aim to reduce the inflammation, encourage healing and repair and restore function consistently results in successful outcomes for most of our patients.

2. Classification

We employ the diagnostic criteria published by the American Academy of Orofacial Pain (AAOP) (Okeson 1996) and by Dworkin and LeResche (1992) (Research Diagnostic Criteria for Temporomandibular Disorders, RDC-TMD). The systems are very similar in approach and content and their merits are described in Chapter 7; the AAOP's criteria are intuitive and highly applicable in the clinical setting whilst the RDC-TMD are extremely detailed (in both the examination technique to be used

Table 8.1 Differential Diagnosis: Entities Not Covered in This Chapter That May Present With TMJ Pain

Mechanism		Structure/Entity	Comments	Chapter
Referred	*Regional*	• Dental	Referral area related to pain intensity	5
		• Ear	Very common	6
		• Muscular	Very common; pterygoids, masseter	7
		• Neurovascular	Hemicrania continua	9, 10
		• Neuropathic	Glossopharyngeal, nervus intermedius neuralgias	11
	Distant	• Cardiac	Usually left-sided, associated with exertion	13
Systemic	*Autoimmune*	• Rheumatoid • Psoriasis • SLE • MCTD • Ankylosing spondylitis • Systemic sclerosis	Patient's primary disorder has usually been diagnosed and requires specific TMJ-related management	14
Infectious	*Primary*	• Bacterial	Usually staphylococcal	14
	Reactive	• Reiter's syndrome		
Tumour	*Benign*	• Usually bone or cartilage	Uncommon: new onset malocclusion, painless swelling	14
	Malignant	• Primary	Uncommon, usually chondrosarcoma. Painful mass.	
		• Metastatic	More common than primary; breast, lung, prostate	
		• Referred pain	Nasopharyngeal carcinoma	
		• Post-therapy	Surgery, radiotherapy	

TMJ, temporomandibular joint; SLE, systemic lupus erythematosus; MCTD, multiple connective tissue disease.

and the diagnostic criteria) and should be employed for TMD research. However, the RDC-TMD criteria do not include polyarthritides or tumours that affect the TMJ and do not deal at all with orofacial pain; this limits its application for routine use in specialized or oral and maxillofacial surgery clinics. Additionally the AAOP system is built upon and integrated with the International Headache Society's (IHS) classification of head and face pain (Olesen *et al* 2004); see Chapter 1. The combined systems thus allow for extensive diagnostic options.

3. Patient Assessment

3.1. Interview and Clinical Examination

Patient interview and evaluation, leading to accurate diagnosis, are the keys to appropriate treatment; but bear in mind that each piece of information may be misleading. It is therefore crucial to evaluate each patient as if assembling a new puzzle. A conclusion can be drawn only when all pieces are collected and properly placed; missing pieces should be looked for and accordingly misleading pieces should be recognized and discarded (see Chapter 1, The Diagnostic Process).

Accurate history taking and thorough physical examination techniques are beyond the scope of this chapter. Examination of the TMJ is rarely performed without a thorough head and neck examination including masticatory, neck and pericranial muscles. The complete examination of the head and neck musculature is essential to diagnose comorbid muscle problems (Chapter 7).

The basic examination techniques and anamnestic approach to orofacial pain patients have been reviewed in Chapters 1 and 7. We therefore present only aspects of the diagnostic process relevant to patients with TMJ problems. In our practice, patient evaluation includes a self-filled questionnaire detailing demographic information and a comprehensive history of the main complaints in the patient's own words. This includes initial symptoms, their characteristics, onset and duration, triggering, modifying or aggravating factors and the presence of oral habits (e.g. clenching). Pure TMJ pain (but not referred pain) is usually accompanied by complaints of dysfunction such as reduced chewing ability. Location and referral patterns should be described in words and marked by the patient on a diagram of the head and neck region. Patients with pain of intra-articular origin usually locate pain around the joint and ear that may radiate to adjacent structures, especially the ear. Pain is increased upon forced opening, on biting and/or chewing on the contralateral side and on loading. Severity of pain at rest and during function as well as the extent of any dysfunction should be assessed by using visual analogue scales (VAS); see Chapter 3. In

addition to quantifying an admittedly subjective experience VAS records enable the clinician to assess changes over time and treatment response in a more objective manner (see also use of pain diaries; Chapter 1). Admittedly there is some disagreement as to the use of questionnaires but we find them time-saving and extremely useful.

A dental history and examination (see Chapter 5) are important to exclude primary dental pathology or secondary occlusal problems such as deviation of the dental midline, missing teeth, collapsed or open bites. The patient's previous treatments such as drug dosages and duration, physical exercises and the result these obtained should be recorded. The patient's adherence to these treatment protocols are important—often treatment is prescribed but not carefully followed and may lead to 'treatment failure'. General health problems and current or past medications are highly relevant to diagnosis and treatment planning (see drug interactions and contraindications in Chapters 15 and 16). Psychosocial factors are important modifiers of disease progression and treatment response (see Chapters 4 and 7)and a basic psychosocial history, with or without the aid of pre-prepared forms, may be useful. The RDC-TMD clinical examination protocol and techniques are highly recommended. They include careful observation, determination and recording of clinical signs and symptoms.

The clinical examination of the TMJ should begin by allowing the clinician to familiarize himself with the patient (behaviour, relative sensitivity) and their articular problem. Examination and interview should be performed facing the patient so that responses can be adequately assessed. Following the initial interview, we begin by palpating the lateral pole of the joint in the preauricular area and asking the patient to perform basic jaw movements; this also familiarizes the patient with the examination procedures to follow. The joint should be carefully palpated with uniform pressure from its lateral aspect and posteriorly via the external auditory meatus both in the open and closed positions. The degree of resultant pain and sensitivity may be recorded on a simple ordinal scale (0=no pain, 1=mild pain, 2=moderate pain and 3=very painful) or employing a 10-cm VAS scale.

The presence and characteristics of joint sounds (Box 8.1) should be recorded. This may be performed digitally or via stethoscopic auscultation; often sounds are present continuously during movement or at particular positions and these should be recorded. Maximal unassisted and assisted interincisal mouth opening should be accurately measured with a millimetre ruler; the presence and pattern of deviation if it exists should be recorded. For example, persistent deviation to one side is characteristic of ipsilateral osteoarthritis or disc derangement without reduction. Deviation during opening that corrects itself following a joint sound (the classical 'S'-shaped mouth opening) is characteristic of ipsilateral disc displacement with reduction. Pain produced on assisted opening, its severity and very importantly its location should be carefully noted (Figs. 8.1a and 8.1b). Other characteristics associated with limitation in jaw movement should be recorded when present; anchored disc phenomenon is associated with a strictly limited mouth opening whilst other disorders may allow increased opening with manual assistance. Other jaw movements such as protrusion and lateral excursions should be accurately assessed and measured on a millimetre (mm) scale, and the exact location of resultant pain recorded. These measurements are reliable in differentiating TMJ patients versus controls (Dworkin and LeResche 1992; Dworkin *et al* 2002; Celic *et al* 2003). A joint-loading test performed by biting on a wooden stick on the canines and molars on both sides followed by asking the patient to point to the pain location and define its intensity is useful (Fig. 8.2a). Intra-articular inflammatory processes are characterized by pain or sensitivity when the patient bites contralaterally to the affected TMJ whilst muscle disorders usually result in pain ipsilateral to the loading (Fig. 8.2b).

Box 8.1 Defining Joint Sounds

Click

- A brief and distinct sound of limited duration occurring during mandibular movement.
- The sound is usually of a 'sharp' or 'popping' nature.
- Clicking noises may occur during opening or closing jaw movements; when they occur on both the click is termed 'reciprocal'.
- Reproducible clicks refer to sounds consistently present on clinical examination and not only as a patient complaint.

Crepitus

- A sound that is present continuously during jaw movement and is therefore not brief, like a click.
- Crepitus reflects the noise of bone grinding against bone or cartilage on cartilage, and sounds or feels like the grinding of stones or walking on snow or sand.
- RDC-TMD further subdivides crepitus into coarse or fine crepitus.
- Coarse crepitus is not muffled and quite prominent whilst fine crepitus is a more subtle grating sound often over a longer period of jaw.
- Fine crepitus may be often described as a rubbing or crackling sound on a rough surface.

RDC-TMD, Research Diagnostic Criteria Temporomandibular Disorders.

3.2. Imaging

Imaging of the TMJ is a complementary modality to clinical examination and is used to confirm diagnosis, aid in treatment planning and assess disease progress. Certain modalities such as arthrography (radiography with contrast medium) have greatly contributed to our understanding of TMJ disorders and the high correlation between specific clinical signs and anatomic disc

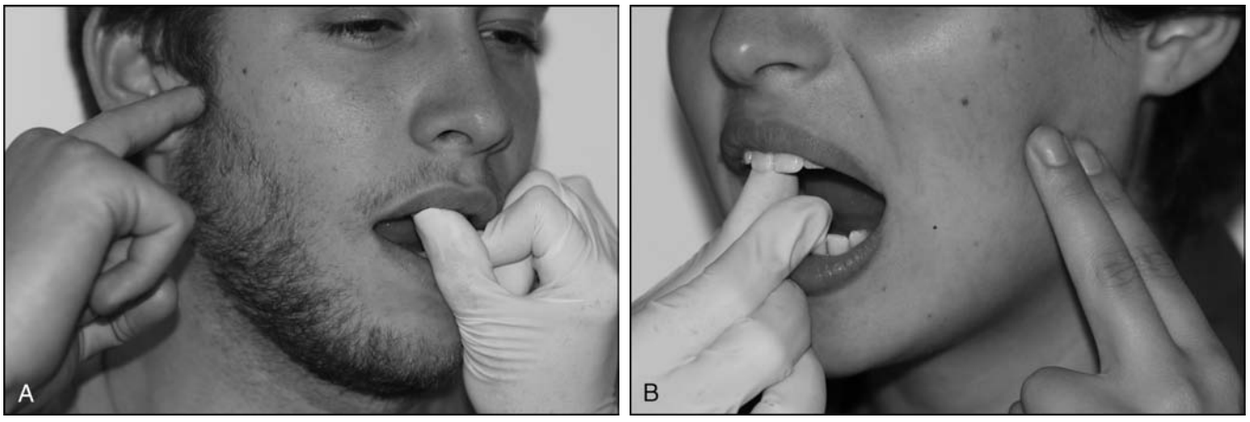

Fig. 8.1 • Loading test. In patients with painful intra-articular disorders or degeneration of the temporomandibular joints loading by clenching the teeth on a wooden stick or spatula induces pain located to the contralateral joint (a). In muscle disorders clenching usually induces ipsilateral pain over the masseter muscle (b).

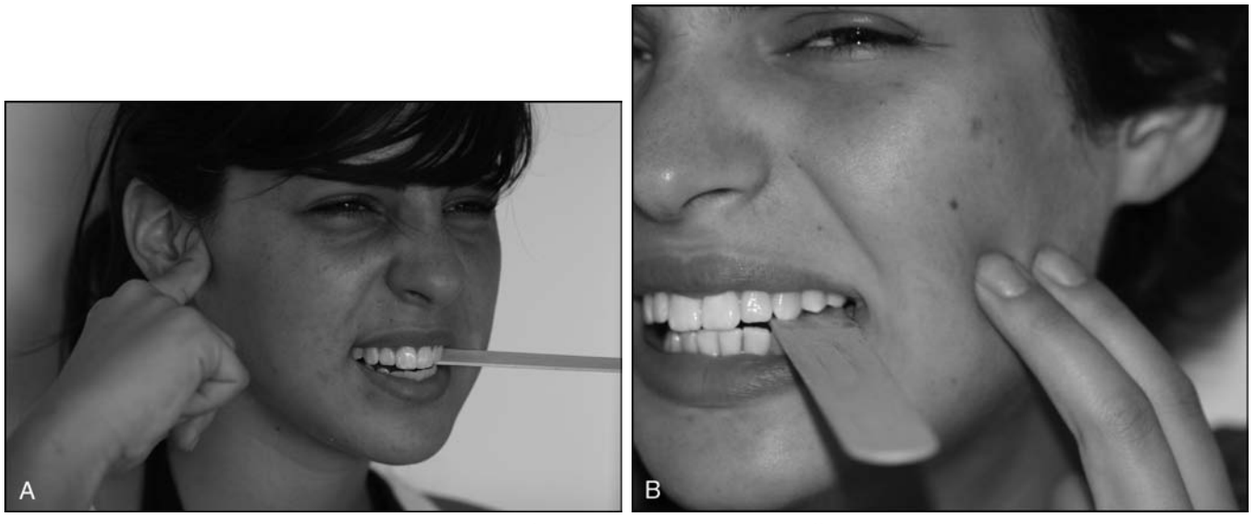

Fig. 8.2 • Forced or assisted opening. Assisted opening will often induce strong pain in the affected temporomandibular joint (a). In masticatory myofascial pain this is often localized over the masseter muscle (b). Additionally this examination demonstrates the amount of opening that may be additionally gained by mild force; in muscle this may be substantial (5mm or more), in disc displacement without reduction moderate (less than 4mm) whilst in anchored disc phenomenon the limitation is 'hard' with no possibility of increase.

derangements. The correlation was so high that arthrography is not needed today (Nitzan *et al* 1991a) and in any event has been replaced by magnetic resonance imaging (MRI). Indeed, clinical examination and the co-occurrence of certain signs may accurately predict the presence of some imaging abnormalities on MRI, such as effusion (Manfredini *et al* 2003). However, some clinical diagnoses do not always accurately predict the precise intra-articular disorder. Thus, the predictability of the clinical examination in disc displacement and TMJ osteoarthritis when compared to MRI or arthroscopy was 43–71% (Roberts *et al* 1991; Paesani *et al* 1992; Israel *et al* 1998). Following clinical assessment, imaging studies may be needed to complete the diagnostic process but careful patient selection is important.

For routine screening or evaluation, panoramic radiographs are adequate. For the basic evaluation of the TMJ, transpharyngeal and transcranial radiographs in the closed and open-mouth positions provide information on the hard tissue structures and range of movement (Brooks *et al* 1997). The decision to obtain other imaging modalities of the TMJ, such as computerized tomography (CT), MRI or bone scan should be deferred until the diagnostic process indicates a need for these. CT scanning provides the most complete three-dimensional reproduction of TMJ bone anatomy, but is not essential for diagnosis (see Fig. 8.3). CT is valuable in assessing the degree of joint degeneration or the possible presence of ankylosis, and essential mostly prior to open surgical intervention (Fig. 8.3). However, accurate information of all the soft tissue elements of the TMJ can only be obtained employing MRI. Indeed MRI may depict joint abnormalities not seen with any other imaging method and thus is the best method for making a thorough imaging assessment of the

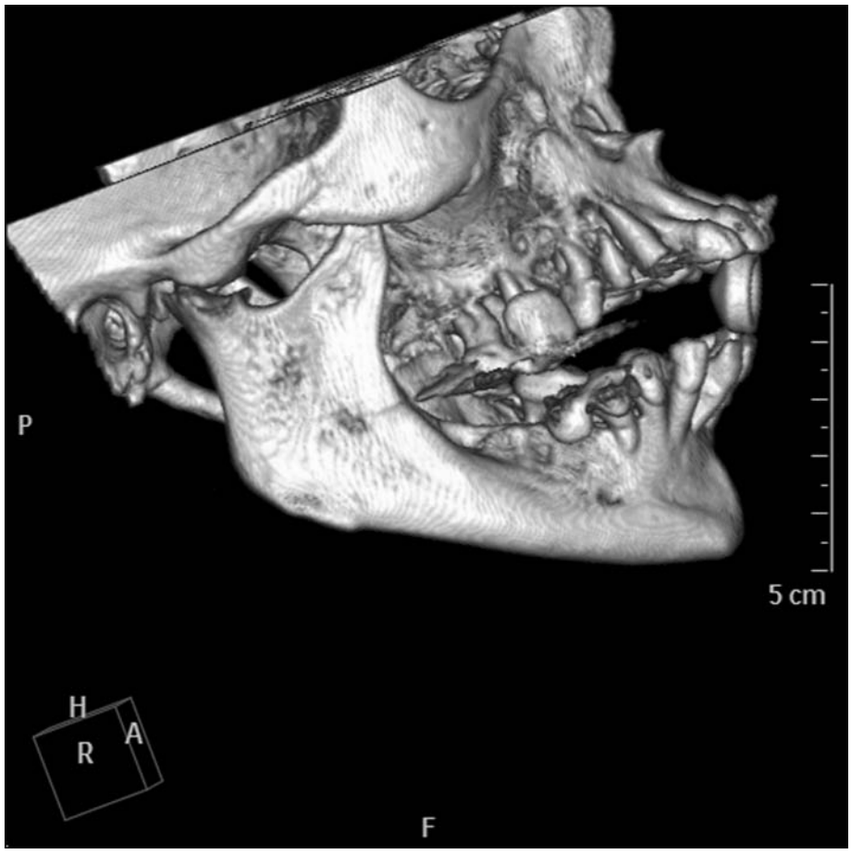

Fig. 8.3 • Three-dimensional (3D) computerized tomography. Reconstruction of images provides a 3D image that is especially useful for assessment of TMJ anatomy. In this case the right temporomandibular joint shows degenerative changes consistent with osteoarthritis and the presence of ankylosis.

TMJ, when this is indicated (Larheim 2005). When MRI is used, some disc displacements may be detected in asymptomatic individuals. Consecutive MRI images may be performed to create a dynamic representation of joint movement and this clearly shows the causes for limitation, such as anchored disc or disc displacements without reduction. Additionally, MRI is able to detect joint effusion and mandibular condyle marrow abnormalities (Takahashi *et al* 1999; Guler *et al* 2005). It is important to realize, however, that MRI as a sole modality is not sufficient for the diagnosis of TMJ pathologies and must be integrated with clinical and anamnestic findings (Emshoff *et al* 2003a; Widmalm *et al* 2006). Bone scanning (scintigraphy or radionuclide studies) offers information on the metabolic activity of bone, and can show increased activity in osteoarthritic joints, even in the absence of radiographic changes (Epstein *et al* 2002). Severe osteoarthritic joint degeneration associated with a negative bone scan indicates inactive disease, making scintigraphy an invaluable tool.

3.3. Other Special Tests

3.3.1. Joint Sounds

Diagnosis of many intra-articular disorders relies on the clinical detection and characterization of joint sounds; different examiners may not agree on the presence and characteristics of these sounds (Dworkin *et al* 1990). Patient confirmation of digitally detected sounds improves the agreement between two independent examiners to about 90% (Goulet and Clark 1990). Techniques using vibration or sound sensors to characterize specific TMJ dysfunction conditions have been tested (Toolson and Sadowsky 1991; Ishigaki *et al* 1993). Unfortunately, these studies suffer from research design flaws that make their conclusions questionable (Baba *et al* 2001). Poor reproducibility of joint sounds can be best explained by the fact that joint sounds vary over time (Kononen *et al* 1996).

3.3.2. Laboratory Findings

Isolated TMJ disease will rarely be accompanied by changes in haematological, biochemical or autoimmune profiles. TMJ involvement in polyarthritides is, however, accompanied by a number of diagnostic and prognostic markers. Direct markers provide a measure of cellular response or changes in the affected tissues and are largely metabolic markers. One example is antigenic keratin sulphate (AgKS) found almost exclusively in cartilage aggrecan. Tissue destruction releases these into body fluids, allowing them to be measured; in most patients with polyarticular osteoarthritis serum AgKS is high (Thonar *et al*

1985). Indirect markers have the ability to influence the metabolism of cells in the affected tissues and include proteolytic enzymes, growth factors and proinflammatory agents. Erythrocyte sedimentation rate and C-reactive protein are indirect systemic markers of inflammation. Inflammatory markers may also be assessed from synovial fluid as described in the pathophysiology section. Synovial fluid analysis is probably the most promising test in intra-articular disorders but is relatively more invasive than venipuncture.

Ultimately, the final diagnosis is based on the skilful integration and interpretation of the patient's complaint (pain, dysfunction), the history, the clinical examination and the radiographic and laboratory findings.

4. Diagnosis of TMJ Disorders

4.1. Historical Perspective

Till the late 1980s disorders of the TMJ and the muscles of mastication were pooled together under a variety of diagnostic terms such as temporomandibular dysfunction syndrome (TMPDS). However, it became clearly apparent in the 1980s that TMPDS included both muscular and joint-based problems that needed to be categorized separately (Eversole and Machado 1985). Concomitantly disc displacement was the postulated cause of joint pain, limited mandibular movement and joint sounds (Westesson and Rohlin 1984; Wilkes 1989; Milam and Schmitz 1995). Naturally at this point a variety of surgical interventions were developed to restore normal TMJ anatomy (disc displacements) and function that led to apparently successful outcomes. However, these procedures were based on limited awareness of the differential diagnosis and pathogenesis of TMJ pain and dysfunction. For example, patients with painless clicking joints but severe muscle pain underwent unwarranted surgical intervention. The result was severe joint and muscle pain accompanied by an inability to exercise the jaw, leading to further complications. Subsequently, many cases complained of recurring and severe signs and symptoms, leading to further, multiple surgical interventions. These repeated interventions sometimes resulted in such severe complications that a need and demand for joint replacements was created (Kent *et al* 1993; Dolwick and Dimitroulis 1994; Mercuri *et al* 1995). Thus, although TMJ pain and dysfunction as a clinical entity has been given much attention, efforts to explain the factor(s) underlying this phenomenon appeared only at a later stage (Haskin *et al* 1995; Zardeneta *et al* 1998).

4.2. Current Thinking

The reported results of therapeutic interventions aimed at disc displacements and data from recent research have stimulated a rethinking of the role played by disc displacement in TMJ complaints (Hall 1995). Disc displacement may be a physiological change (Scapino 1983), often diagnosed in normal individuals and not associated with joint pain (Kircos *et al* 1987a). Over one-third of joints in asymptomatic volunteers were found to have moderately or severely displaced discs (Katzberg *et al* 1996; Morrow *et al* 1996; Tallents *et al* 1996). Contralateral discs in asymptomatic joints of patients with unilateral TMJ problems were found to be displaced as often as the disc on the symptomatic side (Davant *et al* 1993). Conversely normal disc position is observed on imaging in about a quarter of clicking joints (Davant *et al* 1993). In addition, lavage of the upper joint compartment using arthroscopy (Sanders 1986; Nitzan *et al* 1990) or arthrocentesis (Nitzan *et al* 1997), neither of which change the disc position (Montgomery *et al* 1989; Moses and Poker 1989), were found to markedly improve function and alleviate pain. It has become clear that disc displacement is not always the underlying cause of clicking joints; see below. Thus, accurate diagnosis of the origin of pain and/or dysfunction is crucial prior to any treatment recommendations. Gradually, studies on the 'position of the disc' have shifted to the search for the intra-articular, bio-mechanical and biochemical events underlying joint pain and dysfunction (Nitzan and Dolwick 1991; Dolwick 1995).

5. The Temporomandibular Joint

Understanding the functional anatomy of the TMJ is essential for a thorough clinical examination, interpretation of findings and understanding the intricacies of the disorders that afflict this joint. The maintenance of a healthy and functional joint involves the interaction between its constituent tissues; bone cartilage, synovium, capsule, disc, blood vessels and innervation. Since much of the articular cartilage is avascular, it is dependent on the synovial fluid for its nutrients, lubricating agents and metabolic homeostasis.

5.1. Anatomy and Function

The temporomandibular joint is a ginglymoarthrodial synovial joint, that is, a joint capable of hinge and sliding movements. It is encapsulated, bathed in synovial fluid, stress-bearing and capable of allowing opening, lateral and protrusive movement of the mandibular body. Condylar movements are protected from direct contact with the bony architecture of the fossa through an intricate system of fibrocartilage and synovial structures. The TMJ is unique, compared with other load-bearing joints, in anatomical functional and genetic regulation (Luyten 1997).

There are two joint compartments separated by an interarticular disc, and thus four articular surfaces. Joint rotation (hinge) occurs largely in the lower joint space whilst sliding (translation) occurs within the upper joint space. The articular surfaces, including the disc, are all fibrocartilage rather than chondrocartilage as in other joints.

Fibrocartilage is considered more resistant to tensile or shear forces associated with full-range mandibular movements. Articular cartilage is made up of collagen, proteoglycan and chondrocytes. A dense network of aggrecan (aggregating chondroitin sulphate proteoglycan) and collagen fibres provides the necessary biomechanical properties to cartilage (see below on loading). The TMJ is the only joint in the body with vascularized tissue within the capsular ligament. Jaw movements require active participation and perfect coordination between the left and right TMJs.

The articular disc is made up of dense fibrous connective tissue and divides the joint cavity into upper and lower joint spaces. The disc is shaped to match the condyle and fossa: concave inferiorly and convex superiorly. If sectioned anteroposteriorly the anterior portion (or anterior band) is thicker than its central portion (or intermediate zone). Posteriorly the articular disc is thickest (termed the posterior band). Both the posterior band and the intermediate zone thin laterally. Laterally and medially collateral ligaments attach the disc to the condylar head. It has been suggested that the disc imparts the TMJ with abilities to withstand impressive and prolonged compression, relative to joints without a disc (Tanaka *et al* 2004). It is significant, however, that patients who underwent meniscectomy presented excellent function 30 years later (Silver 1984; Eriksson and Westesson 1985; Tolvanen *et al* 1988; Takaku and Toyoda 1994).

During function the lateral and medial discal collateral ligaments, which attach the disc to the condyle, allow for rotational movement of the condyle on the inferior surface of the disc. The superior surface of the disc translates or slides along the posterior aspect of the articular eminence during full mouth opening. Limited lateral movements are also possible. During all movements of a normal TMJ the interarticular disc is always positioned between the fossa/eminence and condyle by the action of the superior lateral pterygoid muscle and the uppermost elastic properties of the posterior attachment known as the posterior, superior retrodiscal lamina of the retrodiscal tissue. Translation of the condyle occurs as a result of the action of the inferior lateral pterygoid muscle which protrudes the mandible, acting in concert with other mandibular depressors and the infra- and suprahyoid musculature. Movement of the disc is controlled during opening by the superior retrodiscal lamina which passively pulls the disc posteriorly as the condyle translates anteriorly. During closing, the superior lateral pterygoid muscle contracts eccentrically, stabilizing the disc against the distal slope of the articular eminence.

5.2. Load Distribution and Lubrication

The arrangement of the teeth, muscles and TMJ is similar to the arrangement of a class 3 lever and predicts that during clenching the TMJ is loaded by muscle activity. Experimental evidence demonstrates that forces acting on the TMJ are both compressive and tensile (Herring and Liu 2001; Sindelar and Herring 2005). The bone architecture of the condyle includes fine, vertically oriented bony trabeculae ideally suited for compressive loading. The articular eminence has thick cortices with the trabeculae oriented approximately transversely and suited for tensile and shearing forces (Herring and Liu 2001). The articular disc is also subjected to both compression and shearing (Sindelar and Herring 2005).

The normal articular surfaces are smooth and possess a high surface energy (Ghadially *et al* 1982; Bloebaum and Radley 1995; Hills 1996), thus requiring an efficient lubrication system. Indeed the smooth movements of the TMJ are possible as a result of sophisticated lubricating and shock-absorbing mechanisms. The synovial membrane, which lines the two layers of the joint capsule and the disc (except for the articulating surface), produces synovial fluid and supplies the nutritional needs of the joint (Dijkgraaf *et al* 1996). The lubricating abilities depend upon the synovial membrane and fluid, the disc and the articular cartilage. The latter are microporous, allowing permeability of the synovial fluid. The permeability and mechanical response of the joint are mutually dependent (de Bont *et al* 1985). It is important to understand that TMJ movements are responsible for the efficient generation of lubrication, blood supply, load absorption mechanism and normal mandibular growth.

In situ the joint space is filled by the highly viscous synovial fluid (SF), containing hyaluronic acid (HA) and the glycoprotein lubricin (Swann *et al* 1981). HA is a polymer of D-glucuronic acid and D-*N*-acetylglucosamine, which is highly unstable and degrades under inflammatory conditions (Nitzan *et al* 2001). Lubricin is composed of ~44% protein, ~45% carbohydrates and ~11% surface-active phospholipids (Swann *et al* 1981), and is suggested to facilitate joint lubrication (Hills and Butler 1984; Schwarz and Hills 1998). The surface-active phospholipids protect the articular surfaces, and are highly effective as major boundary lubricants (Schwarz and Hills 1998; Hills 2000). Lubricin and proteolipid, which have been isolated from synovial fluid, seem to facilitate surface-active phospholipid deposition at articular surfaces (Schwarz and Hills 1998).

An electron-dense layer has been identified in the TMJ that maintains proper joint function and prevents adherence of the articular surfaces (Marchetti *et al* 1997). Osmiophilic layers with embedded vesicular structures have been demonstrated in the TMJ (Clark *et al* 1999). The dominant presence of phosphatidylcholine, a surface-active phospholipid, in rat TMJ was demonstrated in connection with hyaluronic acid and fibronectin (Zea-Aragon *et al* 2005). Upon exposure to phospholipase A2 (PLA2) the osmiophilic droplet cluster in centrifuged SF degraded and the immunolabelling for phosphatidylcholine was clearly decreased (Zea-Aragon *et al* 2005). PLA2, part of the inflammatory process, is naturally secreted into the synovial fluid by the synoviocytes, chondrocytes and osteoblasts, and probably acts specifically on PLs. Addition

of PLA2 to the SF, *in vitro*, significantly increases the measured friction (Hills and Monds 1998).

HA, although not itself a lubricant, forms a 'full fluid film' that keeps the articular surfaces apart and acts as a cushion, preventing generation of friction and thus has an indirect role in joint lubrication (Nitzan *et al* 2001). An *in vitro* study revealed that HA protects PL membranes (liposomes) from lysis by PLA2 by their mutual adherence (Nitzan *et al* 2001; Zea-Aragon *et al* 2005). The lubricating mechanism is therefore intricate and may be disturbed by disruption of any one of its elements; therapy by the specific injection of HA is thus of questionable value.

The normal subchondral bone contains (fatty) bone marrow and trabecular bone with many arterial terminal branches. Subchondral bone marrow accounts for more than 50% of the glucose, oxygen and water requirements of cartilage and is, therefore, important for cartilage metabolism (Imhof *et al* 2000). Additionally, shock absorption in synovial joints is shared by the articular cartilage and the subchondral bone; 1–3% of load forces are attenuated by cartilage while normal subchondral bone is able to attenuate about 30% of the loads (Imhof *et al* 2000). Thus, the subchondral bone has a role protecting articular cartilage from damage caused by excessive loading.

Various methods have been used to assess the amount of load generated in the TMJ. Forces of up to 17.7 kg have been recorded in Macaca monkeys and contact stress in human TMJs is similar to that in the hip and knee joints (Boyd *et al* 1990; Chen and Xu 1994). Intra-articular pressure indirectly measures load; it is negative under most conditions, and reaches high positive values in synovial joints only at the extremes of movement (Ward *et al* 1990). In weanling pigs, intra-articular pressure in the superior TMJ compartment was as high as 20 mmHg during masticatory movement (Ward *et al* 1990). In awake humans, intra-articular pressure in open mouth position is negative and becomes positive in clenched mouth position (Nitzan 1994). These fluctuating intra-articular pressures play a major role in governing joint nutrition, waste removal and condylar growth (Nitzan 1994).

5.3. Innervation

Studies have shown that the TMJ receives small diameter afferents (nociceptors), proprioceptors (including Ruffini, Pacinian), sympathetic and parasympathetic efferents (Dreessen *et al* 1990; Kido *et al* 1993, 1995, 2001; Uddman *et al* 1998; Haeuchi *et al* 1999). Surprisingly the articular proprioceptors become active only at extremes of jaw movement and it is postulated that muscle proprioceptors control routine jaw movements. Sensory innervation is largely from the mandibular branch of the trigeminal nerve via its auriculotemporal branch, although the masseteric and deep temporal branches also participate (Uddman *et al* 1998; Davidson *et al* 2003). Additionally there are fibres originating from the upper cervical dorsal root ganglia (Uddman *et al* 1998); these may be important in patterns of pain referral. The intra-articular distribution pattern of these fibres is anatomically peripheral (i.e. present in the capsule and synovium) with the central parts of the articular disc, condylar head and fossa largely non-innervated. The periphery of the human articular disc is, however, sparsely innervated (Haeuchi *et al* 1999).

The sensation of pain requires the presence and activity of nociceptors but in normal circumstances most of the articular disc is avascular and largely non-innervated. Additionally the articular surfaces of the fossa, eminence and condyle are not innervated. Thus pain from within the joint is usually due to inflammation or injury of the capsule, the highly vascularized and innervated retrodiscal tissues or inflammation of the synovial tissues. The autonomic nervous system, particularly the sympathetic nervous system, is involved in the modulation of pain (see Chapters 2, 11).

5.3.1. Effects of Inflammation

Following inflammation clinical and experimental evidence suggests significant changes in the innervation pattern and neuronal characteristics of the TMJ. Following experimental TMJ arthritis in the rat nerve sprouting into the central part of the articular disc has been shown to occur (Shinoda *et al* 2003). This was associated with behavioural changes, such as reduced food and water intake suggestive of pain. TMJ inflammation significantly increased numbers of heat-sensitive units and induced a lowered heat threshold (Takeuchi *et al* 2004). Mechanical thresholds also tended to be lower, suggesting that inflammation may sensitize nociceptors in the TMJ, and cause hyperalgesia and allodynia (Takeuchi *et al* 2004). There is also evidence for the establishment of central sensitization following TMJ inflammation. Expanded receptive fields, thresholds reduced to mechano stimulation and prolonged neuronal discharges have been documented following TMJ inflammation (Broton *et al* 1988; Sessle and Hu 1991; Iwata *et al* 1999; Lam *et al* 2005; Takeda *et al* 2005). The sympathetic nervous system has a significant pain-modulating capacity and this has been demonstrated in experimental arthritis of the TMJ (Rodrigues *et al* 2006).

Clinical correlates of these changes are difficult but have been documented. Inflammation of the TMJ is clinically characterized by a synovitis with increased vascularity and synovial hyperplasia usually accompanied by neuronal structures (Murakami *et al* 1991; Gynther *et al* 1994, 1998). Sensory thresholds of the skin overlying inflamed TMJs were significantly lower than in non-inflamed TMJs, indicating peripheral sensitization (Eliav *et al* 2003). These changes, and patient's pain ratings, were reversed by arthrocentesis, suggesting that inflammatory cytokines may have been responsible. Immunohistochemical analysis of articular discs from humans with disc

displacements revealed more intense substance P (SP)-like reactivity than in control subjects (Yoshida *et al* 1999).

Taken together the evidence points to a dynamic response of the TMJ constituent tissues to inflammation and includes neuronal plasticity and neuroanatomical changes. Clinically this should be interpreted as indications for early, effective (but conservative) interventions in painful TMJ conditions. This may be valuable in the prevention of nerve sprouting and neuronal plasticity important in the establishment of chronic TMJ pain.

6. Pathophysiology of TMJ Disorders: General Factors

In the following section we examine the pathophysiological events associated with TMJ disorders under two major headings: intra-articular and extra-articular factors. These may be involved in various degrees in different pathologies; factors known to be specifically associated with particular diagnoses are further elaborated in individual sections.

6.1. Intra-articular Events

It is generally accepted that joint derangements involves a complex interaction between the tissues that make up the joint; these components may individually initiate disease but also interact to modify disease progression (Stegenga 2001; Martel-Pelletier *et al* 2006). There is debate regarding the initiating factor or event in joint derangements. Joint function remains normal as long as its adaptive capacity is not compromised (Boyd *et al* 1990; Stegenga *et al* 1991; Nitzan 1994; Milam and Schmitz 1995). Overloading, immobilization and trauma are the major factors associated with joint derangement and disruption of its integrity (Stegenga *et al* 1991; Alexander 2004). Parafunction, such as clenching, is a good example of repetitive jaw motion associated with possibly high and unevenly distributed impact loading that may elicit marked damage to synovial joints such as the TMJ (Nitzan 1994). Overloading is capable of inducing direct and indirect cellular events, neuronal activation and the triggering of a cascade of molecular events that lead to the degradation of the joint constituents by a number of mechanisms. These events include the release of free radicals, neuropeptides, cytokines, proinflammatory agents, enzymes and growth factors (reviewed below). This leads to the establishment of conditions for joint derangement, degeneration and chronic pain. Excessive force, as in macrotrauma, leads to direct cellular and tissue damage with an additional massive release of intracellular contents. This type of injury with microbleeding may also be the source of redox active iron (Zardeneta *et al* 2000), which acts much in the same way as free radicals (see below).

6.1.1. Production of Free Radicals

Free radicals are highly unstable and reactive and will rapidly interact with surrounding molecules to initiate chemical reactions and/or induce tissue injury. Cells, such as synoviocytes, generate free radicals in response to excessive loading (Fukuoka *et al* 1993) and inflammatory cytokines (Kawai *et al* 2000). Free radicals may also be generated as a result of hypoxic-reperfusion cycles associated with overloading (Blake *et al* 1989; Merry *et al* 1991). Temporary hypoxia is a natural outcome of TMJ capillary bed compression occurring during loading (e.g. tooth clenching); re-oxygenation upon cessation of overloading may initiate a hypoxic-reperfusion cycle evoking non-enzymatic release of reactive oxygen species or ROS (i.e., superoxide and hydroxyl anions) (Blake *et al* 1989; Merry *et al* 1991). The highly reactive ROS may enter into rapid chemical reactions in various tissues or with important molecules in the synovial joint (Sheets *et al* 2006). ROS have a number of detrimental effects that may lead to joint disease:

- Induction of neuropeptide release by sensory afferents (see below);
- Initiation of the formation of adhesions (Dijkgraaf *et al* 2003; Sheets *et al* 2006);
- Induction of inflammatory cytokines and activation of transcription of genes involved in the pathogenesis of joint disease (Fukuoka *et al* 1993);
- Inhibition of HA biosynthesis and initiation of its degradation (Zardeneta *et al* 1998), thus decreasing SF's viscosity (Merry *et al* 1991). ROS degradation of HA removes an essential protection mechanism. An *in vitro* study has shown that in the degraded form HA fails to protect continuity of the surface-active phospholipid layer (Dan *et al* 1996; Nitzan *et al* 2001). Thus, phospholipase-A2 secreted into the SF following any inflammatory event is able to extensively lyse surface-active phospholipids (Dan *et al* 1996; Nitzan *et al* 2001). This will reduce or eliminate the continuity of the boundary surfactant layer essential to the integrity of articular surfaces. In the TMJ, uncovering of the articular surface is speculated to be an initiating factor in joint derangement such as disc displacement. PLA2 is also a key element in the production of fatty acid derivatives such as prostaglandins and leukotrienes; and
- Inhibition of the activity of proteolytic enzyme inhibitors by oxidation; e.g. tissue inhibitor of metalloproteases (TIMP) (Zardeneta *et al* 1998; Kanyama *et al* 2000; Shinoda and Takaku, 2000), thus removing an important homeostatic mechanism.

6.1.2. Neuropeptides

Proinflammatory and nociceptive neuropeptides are released in the TMJ by nociceptive trigeminal nerve terminals found in the retrodiscal tissue and capsular

ligaments. These include SP, calcitonin gene-related peptide (CGRP), neuropeptide Y (NPY) and vasoactive intestinal peptide (VIP) (Holmlund *et al* 1991; Appelgren *et al* 1995, 1998; Kopp, 2001). SP and CGRP are released by sensory fibres, NPY by sympathetic fibres and VIP by parasympathetic fibres. Analysis of the synovial fluid of patients with TMJ disorders has shown elevated levels of CGRP (Sato *et al* 2004), SP (Henry and Wolford 2001), NPY and VIP (Alstergren *et al* 1995), providing further evidence that the human TMJ is innervated as described in rodent experiments (Haeuchi *et al* 1999). Neuropeptide release can be initiated by intra-articular mechanical and nociceptive stimuli as in overload; this effect is reversible by opioids. The primary effects (nociceptor activity, neurogenic inflammation) are followed by the appearance of various enzymes and cytokines that were linked by *in vitro* and *in vivo* studies to the biological activities leading to the typical degenerative alterations (van der Kraan and van den Berg 2000; Pufe *et al* 2004a). It is important to appreciate that neuropeptides released by nerve terminals and the resultant neurogenic inflammation are normally essential elements of healing and repair and a disruption in this balance occurs in arthritis (Levine *et al* 2006).

6.1.3. Cytokines

Cytokines are small proteins released by cells that have specific effects on cell–cell communication or interaction, and cell behaviour. The cytokines includes the interleukins (IL), lymphokines and cell signal molecules, such as tumour necrosis factor (TNF) and the interferons, which trigger inflammation. Among the cytokines reported in osteoarthritic joints are interleukin 1 (IL-1), interleukin 6 (IL-6) and TNF-α, which are associated with cartilage degradation (Rossomando *et al* 1992; Kopp 2001; Ogura *et al* 2002, 2005; Pufe *et al* 2004a). Some of these molecules are correlated with disease severity and may be used to predict therapeutic outcomes (Hamada *et al* 2006; Kaneyama *et al* 2007). Cytokines exert their effects via a number of mechanisms:

- Potent proinflammatory effects; accumulation of prostaglandins and other molecules; see below;
- * IL-1 and TNF-α, which are known inducers of the synthesis and activation of metalloproteases by chondrocytes, possibly leading to increased tissue destruction;
- Stimulation of sensory nerve endings, inducing pain and the release of proinflammatory neuropeptides; and
- Generation of free radicals; see above.

6.1.4. Proinflammatory Agents

A number of molecules active in the inflammatory process (see Chapter 15) have been identified in disorders of the TMJ; some are closely related to clinical symptoms and therapeutic response (Murakami *et al* 1998a; Alstergren and Kopp 2000; Kaneyama *et al* 2007). Significant correlations include levels of prostaglandin (PGE2) and pain on movement (Alstergren and Kopp 2000). PGE2 is involved in the pathogenesis of osteoarthritis and induces the production of cytokines, proteases and ROS.

6.1.5. Enzymes

These are released within the osteoarthritic joint by chondrocytes of the articular cartilage, by the lining cells of the synovial membrane and by the osteoblasts in subchondral bone. The enzymes are mainly metalloproteases, serine proteases, thiolproteases and aggrecanases; all are known for their collagen and proteoglycan lysis activity and have been detected in human TMJ disorders (Kanyama *et al* 2000; Yoshida *et al* 2006).

6.1.6. Bone Morphogenetic Proteins (BMP) and Growth Factors

Development of chondrocytes in articular cartilage is arrested prior to final maturation by growth factors, circumventing mineralization and apoptosis. The exact role these factors play in joint disease is unclear; they may be involved in attempts at repair (decompensation). On the other hand some growth factors have been shown to induce catabolic processes and activate proteases (Martel-Pelletier *et al* 2006). In osteoarthritic joints cartilage and osteophytes express growth factor genes, such as BMP-2 and –4, probably involved in allowing the cells to complete the maturation cycle (Martel-Pelletier *et al* 2006). Insulin growth factor (IGF-I), BMP-2 and transforming growth factor-β (TGF_{β}), which are related to matrix synthesis, have been identified in the synovial fluid of TMJ disorders (Gotz *et al* 2005; Matsumoto *et al* 2006). Once the exact mechanisms underlying the effect of the various factors in osteoarthritis are elucidated, some of these may be beneficial in the treatment of TMJ disorders (Detamore and Athanasiou 2005; Almarza and Athanasiou 2006).

6.1.7. Interactions and Progressive Damage

The continued presence of proinflammatory agents and neuropeptides will themselves activate trigeminal fibres and further drive the production of neuropeptides—these interactions are schematically represented in Fig. 8.4. As a result of the accumulation of free radicals, neuropeptides, cytokines and proteases there is damage to the lubrication system and the collagen structure of the cartilage, and an increased volume of proteoglycans. Damage to the lubrication system may lead to stickiness of the disc and derangements (see below).

More rarely severe damage to the joint ensues. Cartilage swelling and softening occurs (chondromalacia) followed by the breakdown of cartilage by proteases (de Bont *et al* 1985) and release of proteoglycans into the synovial fluid (Israel *et al* 1997; Ratcliffe *et al* 1998). This

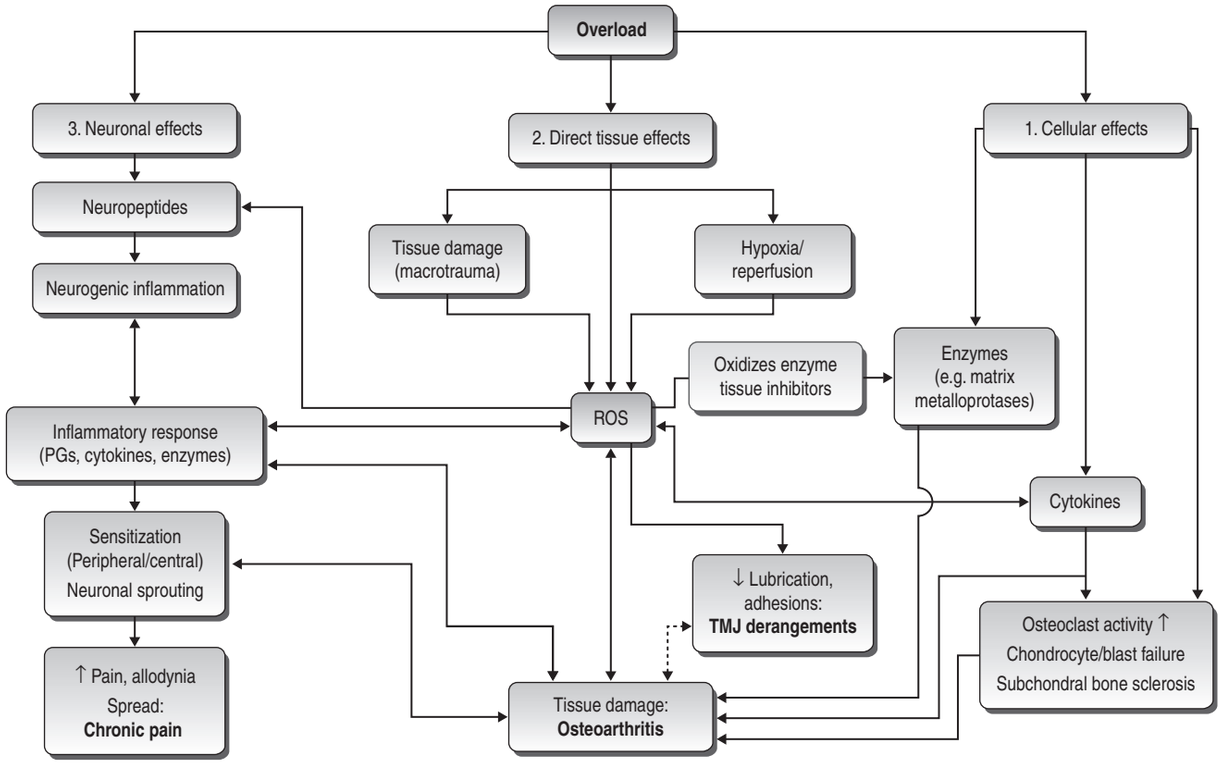

Fig. 8.4 • Schematic summary of events leading to pain and TMJ disorders (for details see text). Overload is considered to be a major factor in the initiation of articular tissue damage. It is important to appreciate that extra-articular factors, such as nutrition, atheromatous disease (hypoxia) or obesity, may predispose to joint disease; the overload may therefore be relative. Female gender is also a significant predisposing factor. Overload exerts its damaging effects via a number of mechanisms, including direct tissue, cellular and neuronal effects.

1. Cellular effects include the induction of cytokine and enzyme release from resident tissue cells. Released cytokines are proinflammatory and algesic molecules also known to induce the release of reactive oxygen species (ROS; see below). Cellular enzymes released are largely proteases that induce tissue damage. Mechanical overload also leads to increased activity of osteoclasts and chondrocyte/chondroblast failure, disturbing the TMJ's repair abilities and increasing tissue destruction.
2. Direct tissue effects are the result of damage (macrotrauma with massive tissue injury) and hypoxia reperfusion injury induced by repetitive capillary bed
compression and release (e.g. clenching). Hypoxia reperfusion cycles result in the production of ROS that have multiple deleterious effects on joint function. ROS are able to induce direct tissue damage, they also induce the release of neuropeptides from afferent fibres, cytokines and enzymes from cells and the oxidation of tissue inhibitors of enzymatic proteases, thus increasing tissue destruction. The lubrication system is severely affected by ROS, leading to increased friction and the formation of adhesions. This may underlie the initiation of some TMJ derangements.
3. Neuronal effects are caused by mechanical activation of trigeminal afferents inducing peripheral release of neuropeptides and subsequent neurogenic
inflammation. If unchecked, neurogenic inflammation induces an inflammatory response with classical proinflammatory agents (e.g. prostaglandins, PG) and induces the release of cytokines and tissue-damaging enzymes. Prolonged inflammation will result in neuronal changes (plasticity) including peripheral and central sensitization and neuronal sprouting into the central portion of the articular disc. These events are clinically represented as ongoing (spontaneous) pain, allodynia (pain on movement, touch) and spread of pain to adjacent structures (ear, temple, mandible).

results in weak cartilage that is unable to withstand loads and thus deforms. Signs of fibrocartilage disintegration include the appearance of vertical and horizontal splits and cartilage thinning (de Bont *et al* 1985; de Bont *et al* 1986). Moreover tissue loading results in chondroblast and chondrocyte failure and osteoclastic bone destruction, leading to impaired repair capacities and structural damage (Stegenga 2001).

Painful TMJs usually prevent the patient from performing mandibular movements. The above data stress the importance of maintaining joint movement (with minimal loading) so as to encourage elimination of damaging products and promote healing.

6.2. Extra-articular Factors

Recent evidence suggests that a number of extra-articular factors may significantly affect the initiation and progression of joint disease. These include nutrition, bone physiology, genetics and gender.

6.2.1. Nutrition

In general suppressed or abnormal synthesis related to insufficient or disturbed nutrition may manifest as degenerative disease. A direct connection between deficient nutrition and joint disorders in general (TMJ disorders

included) is rare and has not been clearly established (Cimmino and Parodi 2005). However, recent research demonstrates that certain foods or nutritional supplements may offer protection from, or alleviate joint disorders (Ameye and Chee 2006). The anti-inflammatory effects of omega-3 fatty acids (see Chapter 17) are well established, and these supplements have been successfully applied in patients with rheumatoid arthritis (Goldberg and Katz 2007). There is also preliminary evidence for the beneficial effects of vitamins C and D in arthritic conditions (McAlindon 2006). Glucosamine with or without chondroitin sulphate is beneficial in arthritic conditions, but is of slow onset (Sarzi-Puttini *et al* 2005; Clegg *et al* 2006). Preliminary evidence suggests that these may be useful in TMJ disorders (Shankland 1998; Nguyen *et al* 2001; Thie *et al* 2001). Based on the above it is reasonable to predict that data demonstrating efficacy of these agents in TMJ disorders will continue to accumulate. Consequently these supplements will gradually be incorporated into the management of TMJ disorders.

6.2.2. Genetics

Genetic variations in the initiation and maintenance of chronic pain syndromes have been extensively studied (see Chapters 7, 9–11. The onset of masticatory muscle pain has been linked to variants (haplotype) of the gene encoding catecholamine-*O*-methyltransferase (COMT) (Diatchenko *et al* 2005). Mutations in genes encoding collagen have been associated with degenerative joint disease (Cimmino and Parodi 2005); however, no similar data are currently available for TMJ disorders but it seems reasonable that a genetic link will be established by future research.

6.2.3. Gender

Pain, dysfunction and clinical signs of TMJ osteoarthritis are more common in females than in males (Agerberg and Inkapool 1990; De Kanter *et al* 1993; Yap *et al* 2003). Although the reasons for this are unclear they are probably linked to the following findings:

- Recent evidence indicates that female hormones modulate the release of neuropeptides from trigeminal ganglion cells (Puri *et al* 2005).
- TMJ afferent activity following intra-articular glutamate injection (excitatory neurotransmitter) is greater in female than in male rats (Cairns *et al* 2001). Oestrogen increases the excitability of rat TMJ afferents and amplifies sensitization secondary to inflammation (Flake *et al* 2005, 2006). Experimental data suggest that testosterone may reduce TMJ damage by modifying the inflammatory response (Flake *et al* 2006).
- The central integration of pain signals originating from the TMJ region differs between male and female rats (Bereiter *et al* 2002) and varies over the oestrus cycle (Okamoto *et al* 2003). Pro-oestrus females rats showed a higher level of central neuronal activation following TMJ inflammation than male rats (Bereiter *et al* 2002). Additionally morphine caused a greater dose-related reduction in nociceptive markers in males than in females.
- Various cell types within TMJs, including synoviocytes and neurons, express oestrogen receptors and their activation is thought to contribute to joint hypermobility, increased matrix metalloprotease activity and a decreased content of collagen and protein in articular disc (Abubaker *et al* 1993, 1996).

Taken together the data suggest that females are more prone to tissue damage in the TMJ and that this damage expresses itself more severely. It also seems that females may be more resistant to pharmacologic treatment.

7. The Painful TMJ

Arthralgia is a term used in the RDC-TMD classification and is defined by the presence of sensitivity to pressure on the TMJ and joint pain on mandibular movements (Table 8.2): these signs usually indicate capsulitis. Many of the disorders and degenerative diseases of the TMJ are often painless. When pain is present it usually indicates an active inflammatory and/or a neuropathic process (see the section above on innervation). Because it is often difficult to accurately identify the underlying process we usually qualify our diagnoses as with or without pain. Similarly since many TMJ disorders present with or without dysfunction diagnoses should be similarly qualified. The following sections deal with individual clinical conditions: joint inflammation, derangements and joint degeneration. Within each section the clinical features are described, specific pathophysiological events highlighted and general treatment options outlined. Full descriptions of the individual treatment options can be found at the end of the chapter.

7.1. Capsulitis and Synovitis

The AAOP define capsulitis and synovitis together (Table 8.2). We will discuss these entities separately in an attempt to differentiate between the two conditions.

7.1.1. Capsulitis

Although there is little data concerning the clinical presentation of capsulitis, it is easily diagnosable. The patient complains of pain around the affected TMJ, particularly during function, and the joint is tender to pressure. In pure capsulitis there are no joint sounds and no findings on plain radiography. However, capsulitis may accompany disc derangements.

Table 8.2 Diagnostic Criteria and Symptomatology of Capsulitis, Synovitis or Arthralgia

Parameters	AAOP[a]: Capsulitis, Synovitis[b]	RDC-TMD[c]: Arthralgia
Diagnostic signs	• Localized TMJ pain • Pain exacerbated by: – Function – Joint loading – Palpation.	• Pain on palpation of the joint on the lateral pole and/or via external auditory meatus • Complaint of: – Pain over the joint; – Pain on function; or – Pain on assisted or unassisted mandibular movements • No coarse crepitus (see Box 8.1).
Findings or comments	• Pain may be present at rest. • Pain may cause limited range of movement. • Fluctuant swelling over affected TMJ may be found. • Ear pain may occur.	
Imaging	• A bright MRI (T2-weighted) signal may be detected if an effusion is present • No extensive osteoarthritic changes are detected.	• Not included
Authors' comments	• In our opinion synovitis is usually distinguishable from capsulitis by the presence of pain on joint loading with contralaterally applied occlusal force (load test); see text.	• Arthralgia is in our view a nonspecific term with specific criteria reminiscent of capsulitis; it has proven invaluable for research purposes. We recommend clinicians attempt to reach a clinical diagnosis that may have features of capsulitis, synovitis, etc. See text for further comments.

AAOP, American Academy of Orofacial Pain; RDC-TMD, Research Diagnostic Criteria Temporomandibular Disorders.
[a] Adapted from Okeson (1996).
[b] Clinically indistinguishable.
[c] Adapted from Dworkin and LeResche (1992).

Treatment should initially include analgesics or non-steroidal anti-inflammatory agents (NSAIDs; see Chapter 15), and physiotherapy to maintain joint mobility. Alternatively periarticular steroids have proven beneficial in our experience.

7.1.2. Synovitis

Characteristically TMJ synovitis causes spontaneous pain, local tenderness to palpation and evoked pain on mandibular movement. It is our opinion that pain on joint loading is a particular feature of synovitis not present in capsulitis. Rarely, fullness over the joint is detectable due to joint effusion, and may induce a sense of acute ipsilateral malocclusion. Joint effusion may be detectable with MRI. Synovitis may occur following external trauma to the joint (falls, blows, traffic accidents) or from joint overload such as repetitive and prolonged clenching. Treatment includes analgesics or NSAIDs, physiotherapy (with no loading) to maintain joint mobility and reduction of joint loading with an intraoral appliance (IOA; see below). In resistant cases arthrocentesis and/or intra-articular steroids have proven beneficial in our experience.

8. Derangements of the TMJ

In its broadest sense the term internal derangements of the TMJ includes all of the intra-articular disorders characterized by dysfunction based on localized anatomical faults (Stegenga 2001). Below we discuss derangements that are accompanied by significant dysfunction, including clicking joints, limited mouth opening and open lock.

8.1. The Clicking Joint

Clicking sounds from TMJs are very common and are reported in patients not seeking treatment: 8.9% in children (Keeling *et al* 1994) and 6–48% in larger population studies (Locker and Slade 1988; De Kanter *et al* 1993). Most often clicking joints are detected by the clinician in patients previously unaware they had joint sounds (Hardison and Okeson 1990), suggesting that clicking is not viewed by patients as a significant treatment-seeking symptom.

Although clicking is commonly considered the first sign of a TMJ derangement the clinical value of a clicking

joint as a diagnosis is doubtful. Clicking sounds per se are of no prognostic value and are not an absolute indication for treatment; only 7% of patients with clicking joints progressed over 1–7.5 years to a bothersome problem (Randolph *et al* 1990). In a large study that followed up patients over a 20-year period, joint sounds rarely progressed to clinically significant problems (Magnusson *et al* 2000).

Clicking may not be a permanent feature of jaw movements and is referred to as intermittent clicking, a condition that is largely asymptomatic. Persistent clicking is a condition characterized by joint sounds consistently occurring during function; usually at variable points during mandibular movement.

8.2. Persistent Click

The patient may present with clicking sounds upon mouth opening, closing or both. There is no limitation in mouth opening but when unilateral the mandible may deviate to the affected side on mouth opening. Clicking may be associated with mild to severe pain and sensitivity to palpation or on loading. These symptoms may cause limitation in function. Some patients may experience intermittent locking, where they are unable to open the jaw to their normal range, but this may be eliminated by active manipulation of the jaw. Joint noise may often be associated with, but is not caused by, muscular pain.

Most often, persistent clicking is produced when an anteriorly displaced disc is reduced to its proper relationship with the condyle during mouth opening (Fig. 8.5). After reduction, the disc-condyle complex moves together as they slide down the posterior slope of the articular eminence, resulting in most cases in normal mouth opening. Upon closure of the mouth, the disc slips to its anteriorly displaced position and this is associated with a clicking sound during closure (Fig. 8.5). This pattern of joint clicking on opening and closing is referred to as a 'reciprocal click' and is pathognomonic of disc displacement with reduction; see below.

8.2.1. Disc Displacements

Disc displacement is defined as an abnormal anatomical relation between the condylar head and the articular disc when the teeth are in normal occlusion: in most cases the disk is anteriorly displaced. Disc displacements may be divided into two major categories: disc displacements with reduction (DDwR) and disc displacement without reduction (DDwoR); see Fig. 8.5. Patients with DDwR or DDwoR account for about 9% of clinic patients, and may also be seen in conjunction with muscular disorders (8%) or together with osteoarthritis, joint pain and muscle disorders (12%) (Lobbezoo *et al* 2004). Disc displacements as diagnosed with the RDC-TMD criteria are relatively rare in the community—about 3%—and are more commonly seen with muscle disorders (8%) (Lobbezoo *et al* 2004).

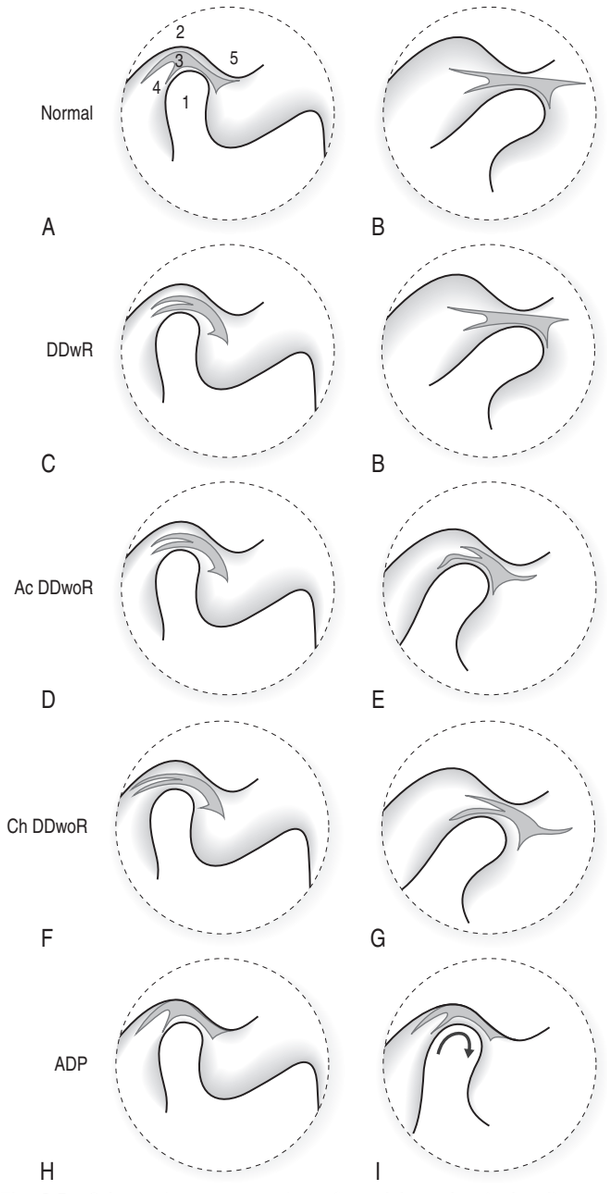

Fig. 8.5 • Schematic representation of condyle and articular disc movements in various disorders. In normal circumstances (A) the condyle (1) is located in the fossa (2) with the articular disc (3) located above and slightly anteriorly. The highly vascular and innervated retrodiscal tissues (4) do not articulate. (5) is articular eminence. During opening (B) and closing, the disc and condyle are coordinated and move smoothly together. (C) In disc displacement with reduction (DDwR) the disc is anteriorly displaced. During opening the condyle meets the displaced disc, which causes a temporary obstacle to movement and thus deviation to the affected side. The condyle is able to reduce under the disc (opening click) and subsequently opening is undeviated and unlimited (B). On closing the condyle disc relation is again disturbed (closing click), leaving the disc displaced anteriorly. Disc displacement without reduction (DDwoR) may be (D) acute (Ac) or (F) chronic (Ch). (E) In Ac DDwoR the disc remains anteriorly displaced during opening movements, with limited mouth opening (35 mm or less). The repetitive forces during mouth opening eventually deform the disc structure and stretch the posterior attachment that has undergone adaptive changes to act as an articulating disc. Consequently mouth opening is far less limited (more than 35 mm) and may even approach normal values (G). In anchored disc phenomenon (ADP) the disc adheres to the fossa (H) and allows for rotatory movements only (I); thus mouth opening is severely limited (less than 30 mm). See text for further details.

8.2.2. Disc Displacement with Reduction (Table 8.3, Fig. 8.5)

Displacement of the articular disc is usually anteriorly or anteromedially (Prinz 1998b) but other rare displacements have been described (Westesson *et al* 1989; Huddleston Slater *et al* 2005). DDwR is characterized by a reproducible joint noise occurring during opening and closing mandibular movements (Okeson 1996). The click during opening occurs at about 20–25 mm interincisal opening; the click on closing invariably occurs at a smaller interincisal opening (15–20 mm) (Dworkin and LeResche 1992). Jaw movements on opening have a classical 'S' shape: initial deviation to the affected side and following a click returns to undeviated and unrestricted maximal opening.

Patients with DDwR are usually asymptomatic and may not even be aware of their joint sounds. TMJ sensitivity and pain may occur spontaneously or secondary to joint loading or other function. When present, pain is usually moderate but may occasionally be more severe (VAS scores of up to 7 on a 10-cm scale) (Conti *et al* 2006). When asked to protrude the jaw and then open, the click is usually eliminated (Dworkin and LeResche 1992).

8.2.2.1. Pathogenesis of Disc Displacement

Many aetiologic factors to explain the occurrence of disc displacement have been proposed. The suggestion that spasm of the superior head of the lateral pterygoid muscle is responsible for the displacement of the disc was rejected (Eriksson *et al* 1981; Mao *et al* 1992). Joint laxity might be a contributing factor but is not prevalent enough relative to the prevalence of disc displacement (Westling 1992). Trauma was thought to cause disc displacement; however, several studies have failed to confirm significant relationships between indirect trauma and disc displacement (Isacsson *et al* 1989; Katzberg *et al* 1996). For example, the prevalence of disc displacement in patients with and without history of whiplash is similar (Tasaki *et al* 1996).

Displacement of the disc seems to be caused by impairment of free articular movements caused by disruption of the lubrication system (Ogus 1981, 1987; Stegenga *et al* 1991; Nitzan 2001). It has been suggested that the lubrication system in the TMJ is relatively stable even with prolonged compression compared to other joints without disc (Tanaka *et al* 2004). Still, the adaptive capacity of the joint structures is often exceeded by prolonged overloading and the viscoelastic properties are affected, leading to increased shear stress in the disc which in turn leads to fatigue and damage. This is associated with generation of free radicals (Cai *et al* 2006) detrimental to the lubrication system; see Fig. 8.4 (Takahashi *et al* 1996; Nitzan *et al* 2002). It seems that as a result of repetitive disc hesitation the ligaments are gradually stretched (Ogus 1981). The disc adheres to the fossa, increasing disc mobility and finally inducing its displacement (Stegenga *et al* 1991; Nitzan 2001; Stegenga 2001) (Fig. 8.6). In support, TMJ arthrography in disc displacement with reduction demonstrates disfiguration of the lower compartment caused by the condyle sliding under the 'hesitating' disc and stretching the anterior wall of the capsule.

Accumulating data is driving research in the direction of the biological mechanisms of joint lubrication; thus disc mobility may be much more important than disc position (Benito *et al* 1998; Takatsuka *et al* 2005; Ohnuki *et al* 2006). Additional mechanisms potentially involved in disruption of disc mobility have been discussed earlier (see General Factors).

Table 8.3 Diagnostic Criteria and Symptomatology of Disc Displacement with Reduction (DDwR)

Parameters	AAOP[a]	RDC-TMD[b]
Diagnostic signs	• Reproducible joint sounds on opening and closing movements of the mandible • Pain may be present • Precipitated by joint movement • Deviation of mouth opening • Coincides with opening click • Unrestricted mouth opening	• Reciprocal click on opening and closing[c] – Click occurs on opening at an interincisal opening $\geq$5 mm than that on closing • May be accompanied by click on protrusion or lateral excursion[c]
Findings or comments	• Patients may report episodic and momentary impairment of jaw movement that may be resolved by mandibular movement or manipulation.	• The RDC-TMD criteria require that when DDwR is accompanied by pain a second diagnosis of joint pain or arthralgia (see Table 8.2) is made
Imaging	• Displaced disc that reduces on mouth opening • No extensive bone changes	• Not included

AAOP, American Academy of Orofacial Pain; RDC-TMD, Research Diagnostic Criteria Temporomandibular Disorders.
[a] Adapted from Okeson (1996).
[b] Adapted from Dworkin and LeResche (1992).
[c] Reproducible on two of three consecutive trials.

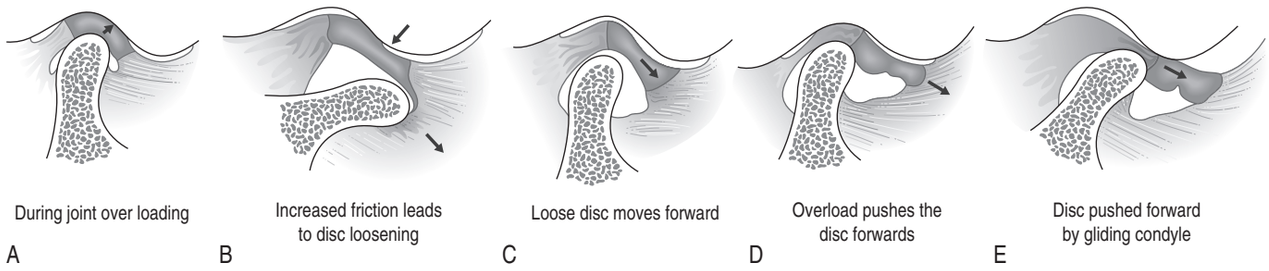

Fig. 8.6 • Pathophysiology of disc displacements. Displacement of the disc is thought to begin by the disruption of the lubrication system leading to increased friction during articular movements and subsequent tissue disruption.
In prolonged overloading the viscoelastic properties of the disc are damaged (A). Concomitant generation of free radicals disrupts the lubrication system, resulting in repetitive disc hesitation and gradual stretching of the ligaments. The disc adheres to the fossa (B), increasing disc mobility and finally inducing its displacement (C). The displaced disc is now repetitively pushed forwards, causing further displacement on clenching (E) with further displacement on opening and overloading of the posterior attachment (D); see text.

Data from Ogus (1981, 1987), Stegenga *et al* (1991), Takahashi *et al* (1996), Nitzan (2001) and Nitzan *et al* (2002).

8.2.3. Intermittent Clicking

Not all clicking joints have verifiable disc displacement and the question arises as to what causes clicking in these cases. Intermittent clicking may also be caused by disc hesitation or lagging as a result of 'stickiness' in the upper compartment following transient overloading or clenching (Prinz 1998a). The clicking noise is assumed to occur upon release of the disc or while the condyle moves against the lagging disc. Other possible mechanisms that may induce intermittent or persistent clicking include joint hypermobility (Johansson and Isberg 1991), enlargement of the lateral pole of the condyle (Griffin 1977), irregularities of the articular eminence (Pereira *et al* 1994b) and adhesions or intra-articular bodies (Bewyer 1989; Montgomery *et al* 1989).

8.2.4. Clicking Joint: Treatment Guidelines

The treatment of a clicking joint, including DDwR, is dictated primarily by the presence of pain or dysfunction. Reciprocal or intermittent click with no significant symptoms should not be treated. The majority of patients with DDwR do not progress to DDwoR (Sato *et al* 2003). Unfortunately, we currently have no means by which to predict which patients will deteriorate; se also discussion below on prognosis of disc displacements. In the presence of pain or dysfunction, therapy should begin with conservative options such as joint unloading (appliances, behavioural modification), physiotherapy and medication. This will improve pain but leaves clicking largely unaffected. When clicking is associated with severe joint pain with no response to non-surgical treatment arthrocentesis may improve symptoms but clicking usually remains or recurs. Surgical arthroscopy, disc repositioning, condylotomy, diskectomy or disc anchorage may be considered when an intolerable loud sound and/or pain resistant to conservative therapy persists. Each procedure should be enhanced by reducing joint loading and by physical therapy. Recurrence of clicking may and often occurs but is usually less annoying to the patient.

In cases were clicking is secondary to adhesions lavage of the upper compartment releases the disc and enables both the condyle and the disc to move simultaneously, preventing the clicking noise. Alternatively if clicking is caused by irregularities, anatomical changes or loose bodies an alternative approach such as arthroscopy or surgery is recommended (Hall *et al* 2005; Smolka and Iizuka 2005; Gonzalez-Garcia *et al* 2006; Undt *et al* 2006; Dolwick 2007). Alleviation of symptoms is obtained regardless of the post-treatment disc position, which usually remains anteriorly displaced (Takatsuka *et al* 2005; Ohnuki *et al* 2006). It has therefore become evident that disc position may be relatively unimportant in TMJ disorders (Dolwick 1995).

8.3. Limited Mouth Opening

In general, the TMJ itself accounts for only a small proportion of disorders causing limitation. Most TMD disorders are muscular in origin (Lobbezoo *et al* 2004) and these account for most cases of limitation of mouth opening. TMJ disorders underlying limited mouth opening include disc displacement without reduction (Han *et al* 1999; Carvajal and Laskin 2000; Guler *et al* 2005) or closed lock (Emshoff *et al* 2000a,b; Reston and Turkelson 2003; Hamada *et al* 2006), anchored disc phenomenon (Nitzan 2003), fibrous ankylosis (McCain *et al* 1992) and osteoarthritis (Emshoff *et al* 2003b). Arthrocentesis seems efficient for these entities, other than for fibrous ankylosis (Montgomery *et al* 1989; Goudot *et al* 2000; Alpaslan and Alpaslan 2001; Guler *et al* 2005). However, it is important to realize that each of these is an independent disorder that requires an individually tailored approach based upon clear diagnostic criteria.

8.3.1. Disc Displacement without Reduction (Table 8.4, Fig. 8.5)

The basic alteration of disc position is the same as in DDwR only that in DDwoR the condyle is unable to

Table 8.4 Diagnostic Criteria and Symptomatology of Disc Displacement without Reduction (DDwoR)

Parameters	AAOP[a]: Acute	RDC-TMD[b]: With Limited Opening
Diagnostic signs	• Persistent marked limitation of mouth opening ≤35 mm • Deviation to the affected side on mouth opening • Markedly limited contralaterotrusion	• History of limited mouth opening – Unassisted opening ≤35 mm – Passive stretch improves opening by ≤4 mm • Uncorrected deviation to the affected side on opening or contralateral excursion <7 mm • Absence of reciprocal click—other joint sounds allowable
Findings or comments	• Pain on forced mouth opening • History of clicking • Ceased when DDwoR began • Affected TMJ tender to palpation	• The RDC TMD criteria require that when DDwoR is accompanied by pain a second diagnosis of arthralgia (joint pain, see table 8–2) is made
Imaging	• Disc displaced that does not reduce on opening • No extensive osteoarthritic changes—mild to moderate changes allowable	• Not included.
Authors' comments	• The current definition of acute DDwoR includes a subgroup of limited mouth opening caused by anchored disc phenomenon (ADP). The major factor that may finally differentiate is clinical response (mouth opening, pain) to arthrocentesis, which is dramatic in ADP and not so significant in DDwoR.	
Parameters	**AAOP[a]: Chronic**	**RDC-TMD[b]: Without Limited Opening**
Diagnostic signs	• History of sudden onset limited mouth opening that began >4 months ago	• History of limited mouth opening – Unassisted opening >35 mm – Passive stretch improves opening by ≥5 mm • Uncorrected deviation to the affected side on opening or contralateral excursion ≥7 mm • Absence of reciprocal click—other joint sounds allowable
Findings or comments	• If pain present, markedly less than in acute stage • History of clicking • Ceased when DDwoR began • Gradual resolution of mouth opening	
Imaging	• Disc displaced that does not reduce on opening • Moderate osteoarthritic changes allowable	• Evidence of non-reducing disk displacement on arthrography or MRI
Authors' comments	• The definition of limited mouth opening in our view includes an opening significantly less than the patient's original normal value	

AAOP, American Academy of Orofacial Pain; RDC-TMD, Research Diagnostic Criteria Temporomandibular Disorders; MRI, magnetic resonance imaging; TMJ, temporomandibular joint.
[a] Adapted from Okeson (1996).
[b] Adapted from Dworkin and LeResche (1992).

reduce onto the disc during opening. Accumulated data have shown that DDwoR may occur with or without clinically significant limited mouth opening (Dworkin and LeResche 1992); the AAOP refer to these as acute or chronic DDwoR, respectively, in the belief that adaptive changes that restore normal range of mouth opening in DDwoR occur over time (Okeson 1996). We define clinically significant limited mouth opening as less than 40 mm interincisal or when the patient subjectively reports discomfort/dysfunction associated with an opening that is less than their usual maximal opening.

Early on, disc displacement without reduction is characterized by limited mouth opening (usually 25–35 mm) that usually develops gradually. A history of clicking is obligatory to make this diagnosis (Okeson 1996). Mouth opening is associated with deviation to the affected side and lateral excursion to the contralateral side is markedly limited. Assisted mouth opening is painful but

usually results in an increase of about 4mm or less. The affected joint may be symptomatic, with localized pain occurring spontaneously or on jaw movements. Pain over the affected TMJ is usually moderate with VAS scores of about 6, but may be more severe particularly in patients seeking treatment. Patients often complain of chewing problems and reduced masticatory efficiency (Peroz and Tai 2002). There is usually pain on palpation of the affected joint, and on loading (see Fig. 8.2). Pain is due to stretching and overloading of the yet unadapted and highly innervated retrodiscal tissue. In the absence of problematic limitation of opening or pain, this disorder might remain unnoticed by the affected individual. Plain open-mouth or CT radiographs of the TMJ invariably demonstrate some condylar sliding, however limited. In arthrography and MRI, the disc is located in front of the condyle in both closed- and open-mouth positions.

Over time pain may be markedly reduced (VAS scores 1–2) and maximal opening may approach normal values (Choi *et al* 1994; Sato *et al* 1997, 1999). This stage is termed 'chronic DDwoR' by the AAOP (Okeson 1996) and 'DDwoR without limited opening' by the RDC-TMD (Dworkin and LeResche 1992).

8.3.1.1. Pathogenesis of Disc Displacement Without Reduction

The pathogenesis of DDwoR is thought to involve processes similar to that discussed earlier; see general pathophysiological processes (Fig. 8.4) and particular processes associated with DDwR (Fig. 8.6).

8.3.2. Prognosis of Disc Displacements

Some studies have suggested that disc displacements will naturally progress to osteoarthritis (Rasmussen 1981; de Leeuw *et al* 1994). However, osteoarthritis can afflict TMJs with or without discs (Eriksson and Westesson 1985; Takaku and Toyoda 1994; Tolvanen *et al* 1988), and may appear prior to signs of disc displacement (de Bont *et al* 1986; Pereira *et al* 1994a), suggesting that these disorders may be independent. Degenerative changes in the condyle are related to female gender, joint immobilization, increased load, atheromatous disease, increased age and a reduced dental arch length and less-to-disc position (Luder *et al* 1993; Luder 2002; Alexander 2004). Most prospective studies have shown that the vast majority of patients with symptomatic DDwoR either remain static or improve spontaneously (Lundh *et al* 1992; Sato *et al* 1997; Kurita *et al* 1998). The reduction in symptoms and restoration of function in DDwoR probably reflects adaptive intracapsular changes that include an increase in dense connective tissue, decreased vascularity and decreased innervation (Isberg and Isacsson 1986; Kurita *et al* 1989). These changes are also present in elderly individuals, irrespective of disc position, so they may be both adaptive and age-related events (Scapino 1991; Luder *et al* 1993; Pereira *et al* 1996). The end result is that the retrodiscal tissues are modified and able to act as a functional disc.

More recently disc mobility and not disc position has emerged as highly important in the prognosis of TMJ disorders (Benito *et al* 1998). This is interesting in that it links the lack of adequate joint lubrication to the development of advanced TMJ disorders.

8.3.3. Treatment of Disc Displacement Without Reduction

In early (acute) DDwoR patients with pain, conservative management may improve symptoms but mouth opening usually remains limited. Arthrocentesis will improve symptoms and marginally increase mouth opening; since arthrocentesis does not alter disc position this is to be expected. Arthrocentesis should be accompanied by conservative options such as joint unloading, appliances, behavioural modification, physiotherapy and analgesic or anti-inflammatory medication. This supportive mode of treatment encourages adaptation of the posterior attachment to act as a disc (Scapino 1991).

The decision to treat chronic DDwoR (or with no limited mouth opening) depends largely on the assessment of functional capabilities as described by the patient and the presence of pain. If treatment is offered it should include conservative options with or without arthrocentesis. Surgical options are considered for patients who remain with pain and significant dysfunction despite conservative management.

8.4. Anchored Disc Phenomenon (ADP) (Fig. 8.5, Case 8.1)

Anchored disc phenomenon is characterized by sudden severe and persistent limited mouth opening, ranging from 10 to 30mm (considerably lower than in disc displacement without reduction), with deviation towards the affected side of the mandibular midline on opening. The movement towards the contralateral side is limited and often painful. On protrusion, the mandible deviates towards the ipsilateral side. History of clicking is *not* obligatory (see DDwoR), but may be present in up to 70% of cases. There is usually no pain in the TMJ upon loading. Forced mouth opening evokes pain in the affected joint and is characterized by an inability to increase maximal opening; the limitation in ADP feels 'hard' relative to that in DDwoR. In long-lasting ADP, the clinical characteristics are less pronounced. In plain open-mouth radiographs and CT scans, the TMJ shows evidence of a non-sliding, but normally structured condyle—rotatory movements are, however, present (Nitzan *et al* 1997; Nitzan 2003; Sanroman 2004). In MRI, the disc appears stuck to the articular eminence and the condyle slides underneath it (Rao *et al* 1990; Sanroman 2004).

Case 8.1 Anchored Disc Phenomenon, 30-year-old Female Patient

Present complaint

Limited mouth opening. The patient reported that she cannot even bite a sandwich and must cut the food into small pieces. She also cannot chew on the left side due to pain.

History of present complaint

Three months prior to her referral she woke in the morning and could not open her mouth. Her dentist referred her to physiotherapy with no improvement. The patient claimed she had no joint clicking in the past.

General health

Healthy.

Patient evaluation

On the visual analogue scale (VAS, 10 cm) her pain level was 7.8 when opening was forced. Pain was specifically located at her right TMJ. Dysfunction was graded 8 out of 10.

Clinical examination

Maximal mouth opening (MMO) was about 24 mm with deviation to the right (see Fig. 8.7a). Upon forced passive opening pain was located in the right TMJ, but mouth opening remained unchanged. Lateral movements to the right were unrestricted but the patient felt she was unable to freely move her jaw to the left (see Fig. 8.7b). Protrusion was also restricted, with deviation to right. Upon palpation there was no TMJ pain, no masticatory muscles tenderness except for slight tenderness in her right external pterygoid. There were no clicking and pain was not generated upon right and left loading.

Imaging

Plain radiographs revealed normal anatomy. The condyle, however, demonstrated no sliding down the slope of the eminence. MRI demonstrated that at maximal opening the disc was stuck to the temporal bone and the condyle was only able to rotate under the stuck disc.

Diagnosis

The absence of a history of clicking, the sudden onset, the extremely limited mouth opening and the absolute resistance of the mandible to increase interincisal opening during active assistance suggested anchored disc phenomenon.

Treatment

Joint unloading by an interocclusal appliance followed by arthrocentesis with immediate rehabilitation of all joint movements. We now have four-year follow-up with no recurrence.

Discussion

It is important to begin by conservative therapy, such as unloading with an intraoral appliance, which may release the disc in 10% of patients. This is followed by arthrocentesis.

8.4.1. Pathogenesis of Anchored Disc Phenomenon

It has been suggested that the cause for limitation originates from a suction-cup effect, whereby the disc that clings to the articular eminence is responsible for the limitation of movement (Sanders 1986; Xu *et al* 2005). However, since the introduction of one needle into the upper joint space, which abolishes the vacuum, does not cure this limitation other adhesive forces between the disc and fossa have been suggested (Nitzan and Etsion 2002; Nitzan 2003; Sanroman 2004). Overloading of the joint is assumed to damage the normal lubrication of the joint (Fig. 8.4). Apparently, in the presence of suboptimal lubrication, adhesive forces can be generated between the pressed, denuded, smooth, elastic disc and the eminence (Nitzan and Etsion 2002; Nitzan 2003; Sanroman 2004). The disc might be in a normal or displaced position. Even a limited area of adhesion between the two opposing surfaces is capable of suddenly holding the disc from sliding down the slope of the eminence. Forced opening is not recommended, as the condyle is pulled away from the adhered disc. Such stretching of the joint's ligaments may traumatically disrupt the anatomical relationship between the condyle and the disc.

8.4.2. Treatment of ADP

Case 8.1 is a young woman with ADP that responded very well to arthrocentesis and joint unloading with good long-term stability (Fig. 8.7). Arthrocentesis neutralizes these adhesive forces, separates the flexible disc from the rigid surface of the eminence and enables smooth normal opening (Nitzan *et al* 1997; Sanroman 2004). Arthrocentesis should be enhanced by reducing joint loading and by physical therapy. The latter, which is not indicated as long as the disc is stuck, should be intensively used following disc release. In these circumstances, recurrence is rare, probably due to the awareness of the patient as well as the low likelihood that the two opposing articular surfaces will again become uncovered and adhere (Nitzan *et al* 1997).

8.5. TMJ Disorders Characterized by Inability to Close the Mouth

8.5.1. Open Lock versus TMJ Condylar Dislocation (Table 8.5)

Open lock is characterized by a sudden inability to close the mouth, and is usually released by the patient's manipulation. Mouth opening during open lock is usually less extreme than in condylar dislocation, and may range from 25 to 30 mm (Nitzan 2002). In plain radiographs and CT scans, the condyle in 'open lock' is located under the eminence and not in front of it, as would be expected in condylar dislocation. MRIs show the condyle to be located in front of the lagging disc (Nitzan 2002). The cause for open lock probably involves diminished lubrication with increased friction between the disc and the eminence. The disc, which normally moves together with the condyle, lags behind it, and consequently the condyle slides under and in front of the disc and cannot return to its former position in the fossa; hence, the mouth remains open.

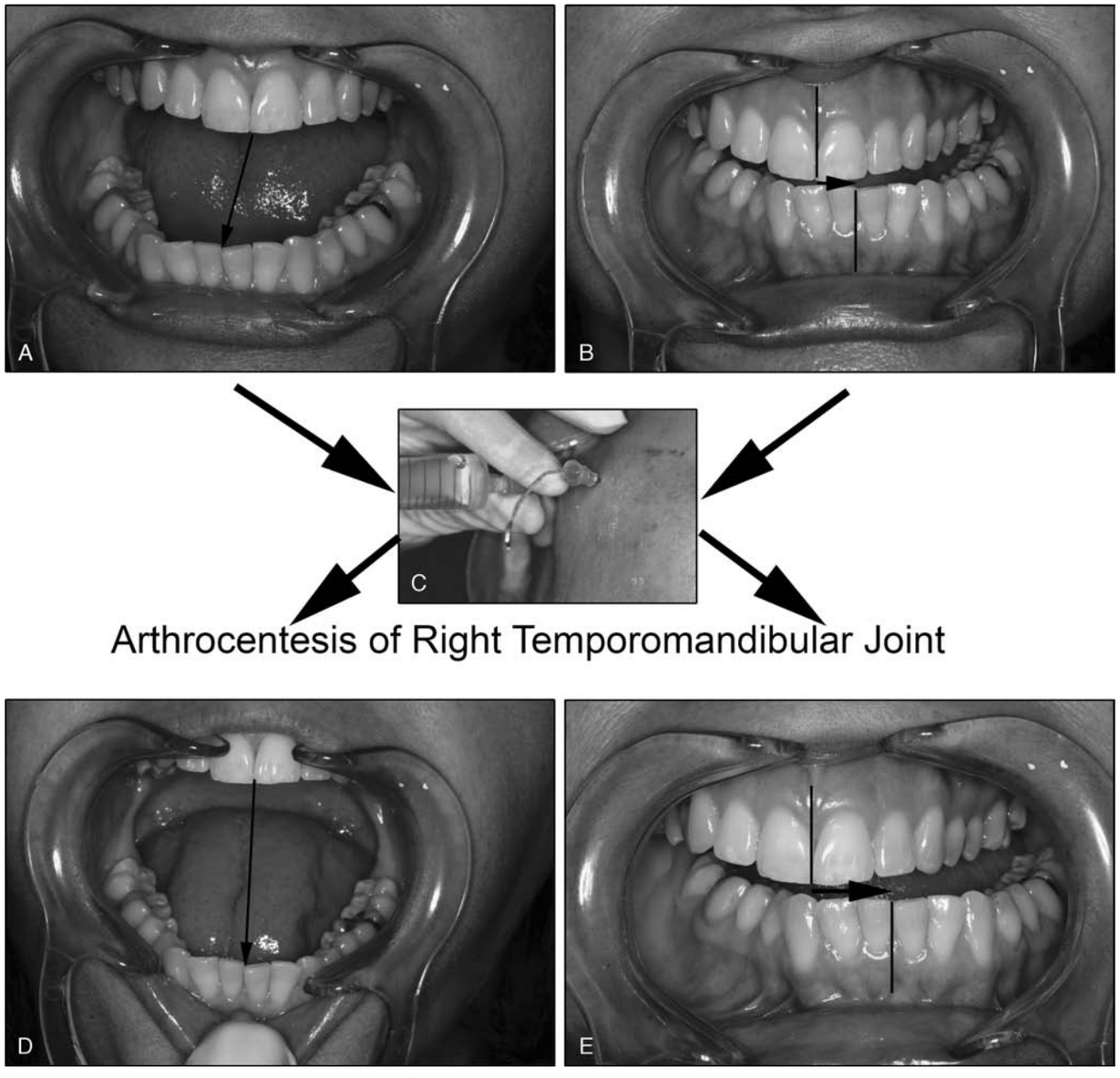

Fig. 8.7 • Patient with anchored disc phenomenon of the right temporomandibular joint. Prior to arthrocentesis mouth opening was limited and deviated to the affected side (a) and lateral excursion to the contralateral side was limited (b). Following superior joint space lavage (c) mouth opening improved markedly without deviation (d) and normal range of lateral excursion was regained (e). See Case 8.1.

Dislocation of the TMJ occurs when the mandibular condyle is displaced anteriorly beyond the articular eminence and the patient is unable to self-reduce the condyle. This is in contrast to subluxation, which is generally defined as a displacement of the condyle out of the glenoid fossa and anterosuperior to the articular eminence, which can be self-reduced by the patient (Shorey and Campbell 2000). There are multiple causes for dislocation that may be related to endogenous factors such as a lack of integrity of the joint ligaments or problems with the bony architecture of the joint surfaces (Shorey and Campbell 2000). Exogenous factors that may induce dislocation include trauma and imbalanced activity of the musculature acting on the joint, sometimes habitual or secondary to medications. Final diagnosis of either open lock or dislocation must be supported by imaging at the time of occurrence.

8.5.2. Treatment of Open Lock/Dislocation

For open lock non-surgical means are usually effective and if not, lavage of the upper compartment can restore sliding of the disc, allowing it and the condyle to move simultaneously. Preventing the condyle from moving in front of the disc provides relief, and with rare long-term recurrence.

For chronic dislocation injection of sclerosing solution (sodium morrhuate or sodium tetradecyl sulphate) has been suggested and induces scarring of the capsule and may prevent recurrence in some cases (Shorey and Campbell 2000). Similarly, injection of botulinum toxin to the lateral pterygoid muscle is claimed to be effective, but is by definition temporary. Surgical treatments for recurrent condylar dislocation include eminectomy (Guven 2005), capsule tightening, introduction of an obstacle

Table 8.5 Comparison between the Characteristics of Open Lock and Condylar Dislocation

Characteristics	Open Lock	Condylar Dislocation
Age	Younger	Older
Occurrence	Spontaneous in joints with internal derangement	Maximal opening (yawning, shouting, neurogenic, neuroleptic drugs, joint laxity)
Maximal mouth opening during the event	Maximal opening with protrusion	>Maximal opening
Reduction	Difficult but self-corrected	Usually professional
Condyle location on radiographs	In front and inferior to eminence	In front and superior to eminence
Magnetic resonance imaging (open-mouth position)	Trapped condyle. Located in front of the lagging disc	Condyle located in front of the eminence
Treatment	Arthrocentesis and unloading	Surgery if recurrent

(e.g. bone) to prevent dislocation and surgical stripping of the lateral pterygoid muscle (Shorey and Campbell 2000).

9. TMJ Degeneration: Osteoarthritis (Tables 8.6, 8.7)

As in other synovial load-bearing joints the TMJ is subject to pathological overload or systemic disease that may lead to tissue breakdown, pain and dysfunction. There is some disagreement between the AAOP's and the RDC-TMD's classification of degenerative joint disease. The RDC-TMD clearly defines 'osteoarthrosis' as a noninflammatory asymptomatic disorder that causes degeneration of the TMJ structures (Table 8.7). The AAOP terms this entity 'primary osteoarthritis', probably in view of the fact that in most cases secondary synovitis develops rapidly (Israel *et al* 1998; Stegenga 2001). There are patients with clinical and radiographic signs of degenerative joint disease with minimal if any symptoms or signs; they probably represent a subacute or chronic form of osteoarthritis. The appearance of symptoms is associated with the identification of intra-articular inflammatory molecules (Holmlund *et al* 1991; Israel *et al* 1991). Accordingly we adopt the thinking that osteoarthritis is the most appropriate terminology (Stegenga 2001) and suggest that this simply be further characterized as with or without pain and/or dysfunction.

9.1. Epidemiology and Clinical Features, Case 8.2

Osteoarthritis (OA) increases with age and may be related to mechanical factors such as loss of molar support with shortening of the dental arch (Luder 2002). It is more common in women than in men, particularly after the age of 40–50. In one study about 10% of women and 5% of men aged 65 were found to have TMJ crepitation, compared to about 5 and 4%, respectively, in 35 year olds (Agerberg and Bergenholtz 1989). Radiographic evidence of OA is very common (14–50%) in asymptomatic individuals, mostly unilaterally, but only 8–16% will have clinically detectable disease (Sato *et al* 1996; Toure *et al* 2005). Both clinical and radiographic findings are found in about 5% of cases (Sato *et al* 1996; Lobbezoo *et al* 2004; Toure *et al* 2005). These figures are comparable to that found in other joints of the body (Cimmino *et al* 2005). Patients with OA or pain of the TMJ make up about 6% of clinic samples; most often these entities are seen together with muscle pain with (18%) or without (35%) disc displacements (Lobbezoo *et al* 2004).

In the acute painful phase of TMJ osteoarthritis, patients typically complain of early morning joint stiffness that lasts more than 30 minutes. This is accompanied by severe joint pain at rest, during jaw movement, difficulty in yawning, biting and chewing. Sometimes the symptoms are accompanied by a sensation of swelling in the TMJ area (Kopp 1985; Zarb and Carlsson 1999). On physical examination, there is painful limited mouth opening, lateral excursions in both directions induce pain in the affected joint (even if only one joint is affected) and attempts to protrude the jaw beyond the limits imposed by the disorder elicit considerable pain in the affected joint; see Case 8.2. Crepitation in the arthritic joint, with or without clicking, may occur during jaw movement. Palpation of the affected joint can evoke mild to severe pain. History of clicking is variable. In general, these patients do not present any distinctive description, complaining of all or some of the symptoms, with severity varying considerably from mild to severe (Zarb and Carlsson 1999).

Imaging of an osteoarthritic joint may show only mild changes; however, advanced stages typically show erosion of the cortical outline, loss of intra-articular space, osteophytes, marginal spurs, subcortical cysts, subchondral bone sclerosis, reduced joint space and a perforated disc, among other features; see Fig. 8.8 (Dolwick and Aufdemorte 1985; Israel *et al* 1997; Imhof *et al* 2000). Inconsistency may exist between the clinical symptoms and imaging: mild clinical disease might be associated with severe imaging appearance and vice versa. Indeed early osteoarthritis may not be detectable by radiographic imaging (Holmlund and Hellsing 1988; Brooks *et al* 1992; Brooks *et al* 1997; Zarb and Carlsson 1999) and conversely radiographic changes are present in many asymptomatic individuals (Ericson and Lundberg 1968; Holmlund and Hellsing 1988). MRI may show the presence of a joint effusion, particularly in painful joints (Takahashi *et al* 1999). Scintigraphy is useful to assess the degree of disease activity.

Table 8.6 Diagnostic Criteria and Symptomatology of Degenerative Joint Disease

Parameters	AAOP[a]: Primary Osteoarthritis[c]	RDC-TMD[b]: Osteoarthritis
Diagnostic signs	• TMJ pain on function • TMJ tenderness to palpation • No identifiable aetiology • Positive imaging findings	• Signs and symptoms of joint pain (arthralgia; see Table 8.2) • Coarse crepitus from affected TMJ (see Box 8.1), or • Positive imaging findings
Findings or comments	• Limited range of movement: – Deviation to affected side • Crepitus or multiple joint sounds	
Imaging	• Evidence of structural bony change: – Subchondral sclerosis – Osteophytes – Erosion – Joint space narrowing	• Tomograms show: – Cortical erosion, or – Sclerosis, or – Flattening, or – Osteophytes
Authors' comments	• Osteoarthritis has an extremely variable clinical expression. It may present with no pain, minimal or no dysfunction and extensive radiographic degeneration only. Conversely there may be extreme pain and dysfunction in patients with minimal radiographic findings. These are difficult to classify with current criteria.	
Parameters	**AAOP[a]: Secondary Osteoarthritis**	**RDC-TMD[b]: Secondary Osteoarthritis**
Diagnostic signs	• As in primary • Documented disease or event associated with onset	• No clear criteria given. Dependent on rheumatologist's or other medical professional's diagnosis of polyarthritic condition.
Findings or comments	• As in primary	
Imaging	• As in primary	
Authors' comments	• See above. Secondary osteoarthritis may also occur after trauma, which is not discussed in this chapter.	

AAOP, American Academy of Orofacial Pain; RDC-TMD, Research Diagnostic Criteria Temporomandibular Disorders; TMJ, temporomandibular joint.
[a] Adapted from Okeson (1996).
[b] Adapted from Dworkin and LeResche (1992).
[c] The AAOP relates osteoarthritis as a noninflammatory condition although this is qualified by a statement that secondary synovitis is common. In contrast the RDC-TMD clearly define a noninflammatory degenerative disorder of the TMJ: osteoarthrosis; see Table 8.7.

Table 8.8 summarizes the differences between the clinical signs and symptoms, and imaging data of anchored disc phenomenon, disc displacement without reduction and osteoarthritis.

9.2. Specific Comments on the Pathogenesis of Osteoarthritis

Osteoarthritis involves the concomitant actions of inflammation, degeneration and attempts at repair. Degeneration is in essence a maladaptive response; a failure of the tissues to respond to the demands made of it. In osteoarthritis the usually well-kept balance between tissue synthesis (repair mechanisms) and breakdown (damage) is disturbed. Recent research has shown that osteoarthritis is a much more complicated than previously thought and the variable presentation is probably a result of the variety of factors linked with the disease. Awareness of these factors is essential for improving insight into the origin of the signs and symptoms and thereby the treatment approach.

As discussed earlier in the section on general pathophysiology, a number of processes may result from joint overloading; see Fig. 8.4. When overloading exceeds the joint's repair capacity a cascade of deterioration may be initiated. This may lead to disruption of the lubrication system, and wear of the articular cartilage that gradually penetrates the underlying bone (Mow and Ateshian 1997; Zarb and Carlsson 1999; Malemud *et al* 2003). Concomitantly, overloading is associated with a variety of mutilations to the subchondral bone leading to microfractures that induce subchondral bone sclerosis. When sclerosed,

Table 8.7 RDC-TMD Criteria for Osteoarthrosis of TMJ

Parameters	RDC-TMD: Osteoarthrosis
Diagnostic signs	• No signs and symptoms of joint pain (arthralgia; see Table 8.2) • Coarse crepitus (see Box 8.2) from affected TMJ, or • Positive imaging findings
Findings or comments	• There is no pain on function, loading or palpation
Imaging	• Tomograms show: – Cortical erosion, or – Sclerosis, or – Flattening, or – Osteophytes
Authors' comments	• As discussed in the text we opt not to use osteoarthrosis seeing as patients with osteoarthritis (OA) may be labile and frequently fluctuate between symptomatic and asymptomatic states. This probably reflects levels of inflammatory mediators within the joint and not separate diagnoses. We thus recommend the use of OA as a diagnosis with additional qualification as to the presence of pain or dysfunction as relevant; see text

RDC-TMD, Research Diagnostic Criteria Temporomandibular Disorders; TMJ, temporomandibular joint.
Adapted from Dworkin and LeResche (1992).

the subchondral bone does not provide the nutritional needs to the cartilage and does not function as an efficient shock absorber, both crucial for the integrity of the articular cartilage. It has been recently suggested that subsequent to the bony changes the cartilage is degraded and separates from the underlying bone. Other factors such as obesity and atheromatous disease may induce subchondral bone sclerosis and predispose the joint to damage.

9.2.1. Imbalance in Bone Physiology

The main radiologic features of OA are joint space narrowing and extensive remodeling of subchondral bone which is generally sclerotic. Bone changes may precede cartilage destruction and prominent alterations in subchondral bone suggest that this tissue plays a key role in the initiation of TMJ disease. Cartilage loss in knee osteoarthritis can often be predicted by the enhanced uptake of radioactive markers (scintigraphy) specifically by subchondral bone (Dieppe *et al* 1993). Moreover scintigraphic activity precedes episodes of radiographic degeneration. Bone cells, contrary to cartilage cells, are well supported by a capillary plexus. This, however, also exposes them to secondary effects of local or systemic disease that may affect local blood flow, see atheromatous disease below.

9.2.1.1. Bone Mass

It has been found that patients with OA have a significantly increased bone mass (Sinigaglia *et al* 2005). A high bone mass has been shown to play a role in the development of erosive changes in the TMJ (Flygare *et al* 1997). OA is characterized by an increase in material density in subchondral bone, constituting increased collagen matrix and abnormal mineralization ((Martel-Pelletier *et al* 2006). Osteoarthritic bone of the femoral neck is significantly stiffer than that of controls (Li and Aspden 1997). On the one hand this results in relative protection from fractures but also in uncompliant subchondral bone structure that is inadequate support for the articular cartilage; shear forces are generated and cartilage damage with cleft formation and separation from bone occurs. Although the debate is ongoing (Cimmino and Parodi 2005; McDonald Blumer 2005), OA seems less common in patients with osteoporosis; possibly because osteoporosis renders the bone more flexible (Sinigaglia *et al* 2005). Cartilage separation results in the ability of cytokines, growth factors and prostaglandins produced by the subchondral bone tissue to cross through the bone–cartilage interface and damage the cartilage (Imhof *et al* 2000; Lajeunesse and Reboul 2003). In support, osteoarthritic subchondral bone has significantly increased levels of cytokines, inflammatory mediators and matrix metalloprotease activity (Mansell and Bailey 1998; Martel-Pelletier and Pelletier 2005). Recent data have shown that bone resorption pits in subchondral bone may release matrix metalloproteases derived from cells in bone marrow into the articular cartilage (Martel-Pelletier *et al* 2006).

9.2.2. Atheromatous Diseases

Atheromatous diseases are highly correlated with OA (Conaghan *et al* 2005), and compromised blood supply to the subchondral bone is a possible inducer of osteoarthritis. This provides an explanation for the occurrence of osteoarthritis in older patients with atheromatous diseases even in unloaded joints (Pufe *et al* 2004a). Hypoxia triggers the secretion of vasculo-endothelial growth factor (VEGF), which is suggested to initiate joint degradation, via subchondral bone sclerosis (Pufe *et al* 2004b; Tanaka *et al* 2005; Murata *et al* 2006). Correspondingly, when we injected VEGF intra-articularly in an animal model OA was induced (unpublished data).

9.2.3. Obesity

Obesity is a well-known risk factor for OA, thought to act via increased joint loading. However, increased OA of the hand in obese patients suggests that other, probably metabolic factors are at play (Cimmino and Parodi 2005). The hormone leptin, derived from adipocytes, may be key to this relationship. Leptin plays a major role in preventing obesity by effects at the hypothalamic level and leptin resistance is associated with obesity. Levels of leptin are increased in osteoblasts within

Case 8.2 TMJ Osteoarthritis, 45-year-old Man

Present complaint

Sudden severe pain in his right joint associated with severely limited mouth opening. He was unable to talk without suffering pain and eating had progressively become nearly impossible.

History of present complaint

The patient, a lawyer in a specially demanding period, ran a normal but very intensive life until a month ago when sharp pain on the right side of the face had woken him up at night. Since then he could not bite or chew on the left side. Various medications such as analgesic nonsteroidal ant inflammatories had been tried with no improvement. There was no history of clicking or trauma in the past. The patient reported no generalized joint pain and felt well. Recent laboratory tests (routine) revealed a normal differential blood count, liver function and electrolytes.

Medical history

Hypercholesterolaemia controlled with 20 mg simvastatin daily.

Clinical evaluation

The patient evaluated his pain and dysfunction as 9 and 7.8, respectively, on a 10-cm VAS scale.

Clinical examination

Maximal mouth opening was 22 mm with slight deviation to the right. Upon slight forced opening excruciating pain was generated in the right TMJ. Lateral movements to the right were unrestricted but were painful and limited to the left, both associated with severe pain in the right TMJ. Protrusion was limited with deviation to the right and severe pain on the right side. The patient had a deep overbite, a deviated dental midline (to the left) but a well-maintained dentition. Upon contralateral loading severe pain was generated in the right joint.

Imaging

A CT scan demonstrated typical degenerative changes on the right TMJ, limited movement and no widening of the intra-articular space (Fig. 8.8).

Diagnosis

The signs and symptoms are typical of osteoarthritis.

Treatment

Joint unloading and right TMJ arthrocentesis were followed by immediate improvement in mandibular movements (Fig. 8.9). Intensive physiotherapy was supported by analgesic medication.

Discussion

The purpose of the treatment is to aid the patient and provide optimal conditions for healing and restored function. This is performed by providing an IOA with anti-inflammatory medication: reduced internal and external load. These were able to control pain but only marginally improved maximal mouth opening. Arthrocentesis eliminates inflammatory products followed by immediate rehabilitation of movements. It is interesting that local anaesthesia applied prior to arthrocentesis eliminated pain but did not improve mouth opening. Physiotherapy is an essential component and maintains adequate function.
The severe degenerative changes demonstrated by the CT are not to be misinterpreted as contraindicating arthrocentesis: it is our experience that there is no correlation between the severity of the degeneration (by CT scan) and the results of arthrocentesis.

osteoarthritic subchondral bone (Martel-Pelletier *et al* 2006). Leptin exhibits, in synergy with other proinflammatory cytokines, a detrimental effect on articular cartilage cells by promoting nitric oxide synthesis (Otero *et al* 2006). A connection between obesity and TMJ has not been shown; this may be due to differential metabolic control of the TMJ relative to other joints (Luyten 1997).

9.2.4. Jaw Movement

Immobilization is currently considered among the principal causes for joint deterioration mainly due to the absence of natural elimination of the virulent inflammatory core. Movements of the joint are necessary to induce fluctuating intra-articular pressures that function like a pump crucial for joint homeostasis.

Although most damage is associated with movement, immobilization of the joint is a dominant factor in joint deterioration. Interestingly, prolonged opening has led to osteoarthritis in mice (Fujisawa *et al* 2003), and immobilizing the joint in primates leads to marked thinning of the articular cartilage (Glineburg *et al* 1982).

Early and correct diagnosis associated with the appropriate treatment aiming to bring the joint back to normal movements is crucial. Adequate mobilization also avoids further complications such as ankylosis; see Fig. 8.3.

9.3. Treatment of Osteoarthritis

The physician's role is supportive, establishing ideal conditions for healing by unloading and enabling movement. As synovial joints are believed to be an adaptable organ our goal is to bring the symptomatic joint from an unadaptable state to an adaptable one, bearing in mind that we treat the patient and not the radiographic image. Especially in this

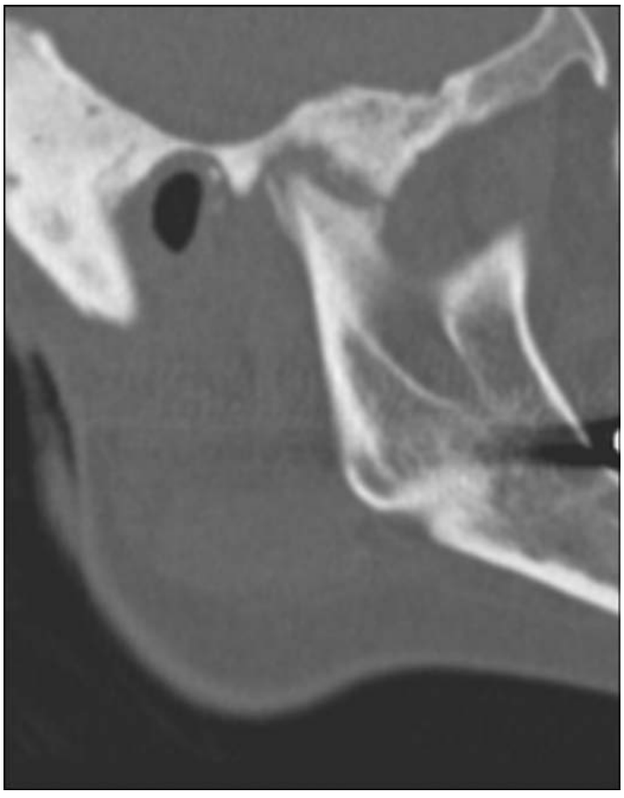

Fig. 8.8 • Section from computerized tomography showing osteoarthritis. The sagittal view shows the right temporomandibular joint. The head of the condyle is severely degenerated with flattening and osteophyte formation; see Case 8.2.

Table 8.8 Comparison between the Common Characteristics of the Anchored Disc Phenomenon, Disc Displacement without Reduction and Osteoarthritis

Characteristics	Anchored Disc Phenomenon	Disc Displacement Without Reduction	Osteoarthritis
Occurrence	Sudden	Gradual	Sudden/ gradual
Past clicks	No (30%)	Yes	No/Yes
Maximal mouth opening (mm)	15–25	30–45	10–30
Contralateral movement	Limited	Limited	Limited
Ipsilateral movement	Normal	Normal	Limited
Pain (self-assessment)	–	+	Severe to none
Dysfunction (self-assessment)	+	–	– to +++
Magnetic resonance imaging (open mouth position)	Disc stuck, located above and behind the condyle	Disc displaced-deformed, located in front of the condyle	Effusion +/– Adhesion +/– Disc displaced +/– Deformed disc +/– Perforated disc +/–
Effect of arthrocentesis	Excellent	Moderate	Very good (70%)

disorder, surgery should not be recommended unless non-surgical means have failed (Kopp 1985; Milam and Schmitz 1995; Nitzan 2003; Kurita *et al* 2004, 2006; Martinez Blanco *et al* 2004; Dimitroulis 2005b; Tanaka *et al* 2005). The prognosis of osteoarthritis following conservative management has been shown to be good and stable; although radiographic bone changes may show deterioration clinical signs and symptoms tend to improve (de Leeuw *et al* 1994, 1995a,b, 1996). Treatment should aim towards joint unloading, joint mobilization and pain control. Joint unloading is crucial for restoring lubrication and allowing healing of the cartilage. External loads may be reduced by modalities such as intraoral appliances, soft diet, and behavioural treatments. Reduction of 'internal' factors such as the inflammatory exudate may be attained by medication provided orally, intramuscularly or intra-articularly. Arthrocentesis is also able to mechanically rinse the joint and remove proinflammatory agents; this unloads, provides analgesia and releases adhesions, thus promoting joint mobility. Results are often dramatic; see Case 8.2, Figs. 8.8, 8.9.

To attain joint mobilization maximal pain control must be attained. Pain relief is rapidly achieved by pharmacological means (NSAIDs, analgesics; see below) or by joint unloading with IOA. Physiotherapy is a critical component essential for overcoming the acute stage of the disorder. Better functional performance allows for nutrition, efficient removal of waste and increased joint lubrication, thus establishing conditions that will allow healing of the TMJ constituents.

If pain and dysfunction are not eliminated surgical intervention should be planned. In our experience (Nitzan and Price 2001), arthrocentesis obviates the need for corrective surgery in 68.4% of patients that did not respond to other non-surgical treatment and were candidates for surgery. Following the arthrocentesis, maximal mouth opening increased from about 24 to 43 mm. Over a mean follow-up period of about 20 months (range 6–62 months), pain levels decreased substantially from VAS scores of about 7 to 2 and dysfunction levels were significantly improved. These outcomes are not perfect, but certainly suffice to obviate corrective surgery. However, in the remaining 31.6% of the cases similar symptoms were caused by joint pathologies such as bone spicules or fibrous ankylosis, which are not amenable to lavage (see Fig. 8.3). A similar rationale explains the effect of arthrocentesis in the treatment of TMJ rheumatoid arthritis and in other polyarthritides.

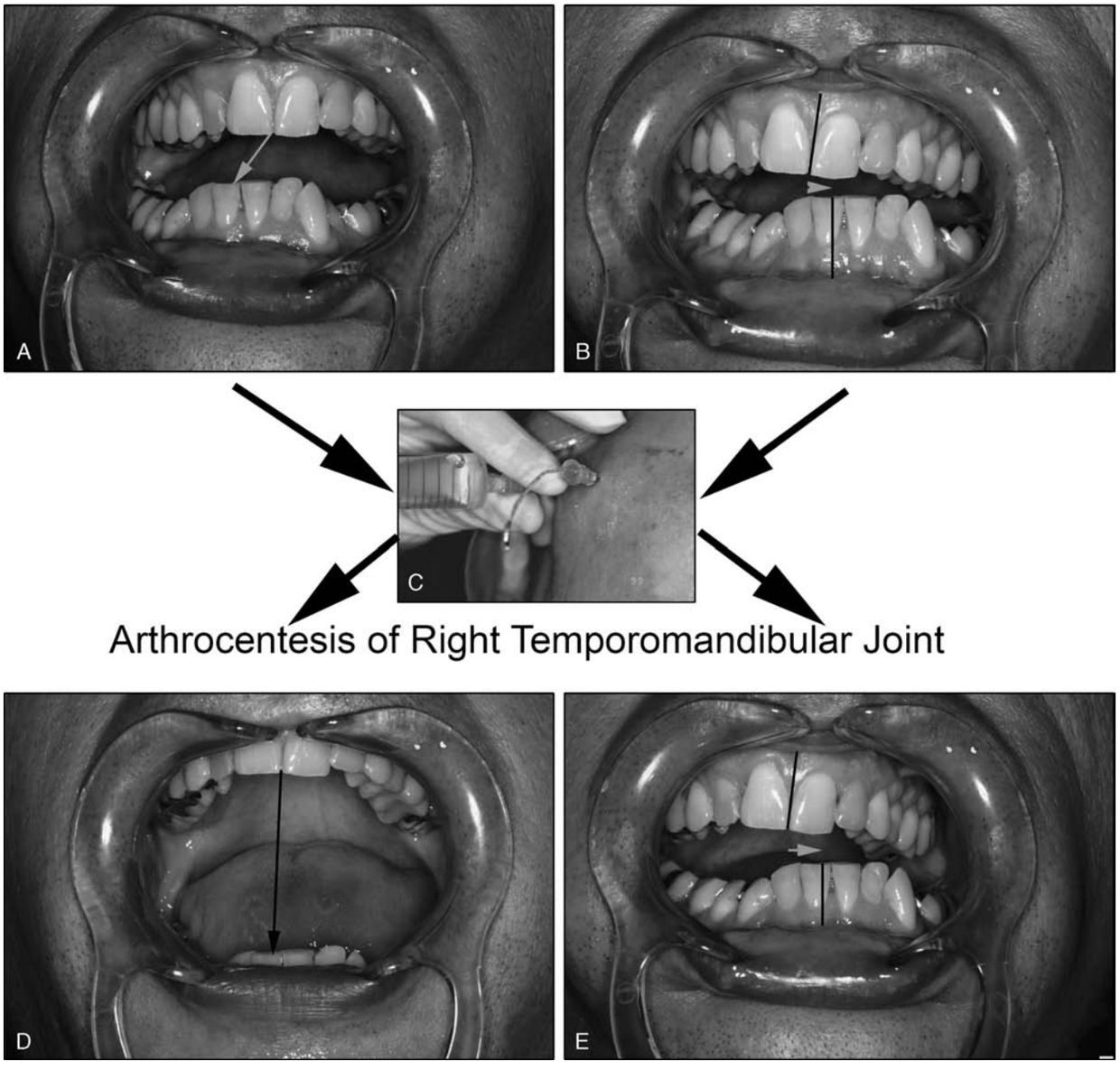

Fig. 8.9 • Patient with osteoarthritis of the right temporomandibular joint. Prior to arthrocentesis mouth opening was limited and deviated to the affected side (a) lateral excursion to the contralateral side was limited (b) and both caused severe pain. Following superior joint space lavage (c) mouth opening improved markedly without deviation (d) and normal range of lateral excursion was regained (e). See Case 8.2.

When arthrocentesis fails, surgical intervention is recommended; surgical arthroscopy, disc repair and repositioning, diskectomy and in very severe cases with marked loss of vertical dimension and malocclusion joint replacement are required. Replacement is accomplished by autogenic bone or an artificial joint (see below).

9.4. Secondary Osteoarthritis

A number of systemic disorders may induce a degenerative process within the TMJ that is often clinically and radiologically indistinguishable from primary osteoarthritis. However, these entities are usually accompanied by symptoms associated with the systemic disease, such as fatigue, pyrexia, anaemia and serology. In Table 8.9 we summarize salient features of systemic conditions that may induce TMJ pain and degeneration (Tegelberg and Kopp 1987, 1996; Celiker *et al* 1995; Yoshida *et al* 1998; Voog *et al* 2003; Helenius *et al* 2005, 2006).

9.5. Idiopathic Condylar Resorption (Condylysis)

Degenerative conditions of the condyle are usually primary and associated with increasing age and articular loading. Secondary osteoarthritis is related to a number of factors such as trauma or infection or due to systemic conditions that may involve the TMJ. Some cases have been reported following orthognathic surgery (Kerstens

Table 8.9 Laboratory Findings of Osteoarthritis That May Involve the TMJ

Diagnosis	Laboratory Findings	Clinical
Rheumatoid		
Prevalence 2–2.5% Peak onset 40–60 years	Rheumatoid factor (70–80%) Antinuclear antibody (15%) Elevated ESR (90%) Mild anemia (25%) HLA Dw5, HLA-DrW (50%)	Fatigue, weight loss, pyrexia. Bilateral joint pain (arthralgia) with crepitation. Open bite. Radiographic evidence of OA. TMJ involvement occurs in 50–75% of patients and signifies severe disease and usually not presenting symptom.
Psoriatic		
Prevalence 0.07% Peak onset 35–45 years	Seronegative. HLA-B27	Fatigue, weight loss, pyrexia, myalgia. Often unilateral pain of the TMJ. Psoriasis itself affects 1–2% of the population but arthritis is a rare complication and TMJ involvement even rarer.
Ankylosing spondylitis		
Prevalence 0.4–1.6% Peak onset 20–30 years	Seronegative. Elevated ESR (70%) HLA-B27	Joint pain, stiffness of TMJ that may result in ankylosis. TMJ involvement occurs in patients late in the disease
Reiter's syndrome		
Reactive to infection Peak onset 20–30 years	Seronegative	Fatigue, weight loss, pyrexia, lymphadenopathy. Acute unilateral joint pain.

TMJ, temporomandibular joint; OA, osteoarthritis; ESR, erythrocyte sedimentation rate.

et al 1990; Bouwman *et al* 1994) and prolonged steroid use has been associated with destructive joint disease.

However, there are cases, usually females aged 15–35 years, who present with condylar resorption with no apparent cause (Arnett *et al* 1996a,b). These have been termed idiopathic condylar resorption or condylysis (Okeson 1996); we adopt the former term. The patients complain of an anterior open bite and a variable degree of dysfunction. The presence of pain and sensitivity to pressure over the joints is inconsistent. Clinical findings also include a number of skeletal and occlusal features suggested to be involved in its pathogenesis (Wolford and Cardenas 1999). Radiography usually reveals bilateral condylar damage similar to that observed in OA, and scintigraphy is useful in assessing the current activity of bone destruction. By definition idiopathic condylar resorption must be seronegative with no biochemical or haematological abnormalities.

The pathophysiology is poorly understood but the high predilection for females suggests that hormonal influences are important. Idiopathic condylar resorption may be a severe form of OA or be related to disturbed blood supply to subchondral bone, as observed in hip joints.

Treatment of idiopathic condylar resorption is largely empirical; it is a rare condition and there are no prospective studies. We suggest joint unloading with an IOA and the judicious use of analgesics or NSAIDs during episodes of pain. Severe facial deformity or an occlusion with significant dysfunction to the patient may indicate a need for surgery. Surgery may consist of orthognathic restoration or TMJ replacement, depending on the case. Orthognathic surgery is not associated with consistent results and usually leads to exacerbation or reinitiation of resorption (Merkx and Van Damme 1994; Huang *et al* 1997). However, good results for surgery have been described when patient selection includes evidence that disease progression is arrested, and there is adequate perioperative joint unloading (Arnett and Tamborello 1990). It is essential that candidate patients fully understand the risks.

10. Pain in the TMJ: Differential Diagnosis

Common pathologies within the joint that present with pain and/or dysfunction have been reviewed. However, the location of the TMJ in the midst of the skull with resultant referral of pain, often makes patient evaluation complex; see Chapter 1 Importantly, the differential diagnosis of pain *felt* in the TMJ, but not originating from the joint, includes a number of causes. Masticatory myofascial pain, a far more common condition, affecting the lateral and/or medial pterygoid and/or the masseter will often refer pain specifically to the TMJ (Chapter 7). Many other adjacent structures may refer pain to the TMJ area including intraoral pathology (Chapter 5), the ear (Chapter 6) and neurovascular (Chapters 9 and 10) or neuropathic syndromes (Chapter 11). In addition a variety of extra-articular systemic disorders may directly affect joint growth, anatomy and function; see Tables 8.1 and 8.9. Although rare, primary tumours (benign/malignant) may originate from TMJ structures and distant tumours may metastasize to the TMJ, presenting with joint pain or sensitivity (see also Chapter 14) (Nwoku and Koch 1974; Allias-Montmayeur *et al* 1997). Most tumours arising in the TMJ will be of bone or cartilage origin (Bavitz and Chewning 1990; Warner *et al* 2000). In any clinical evaluation careful data collection and examination are crucial for avoiding misdiagnosis; imaging is a complementary tool. Figure 8.10 is a simplified flow chart to aid clinicians in the diagnosis of painful TMJ disorders that may be accompanied by limited mouth opening and/or joint sounds.

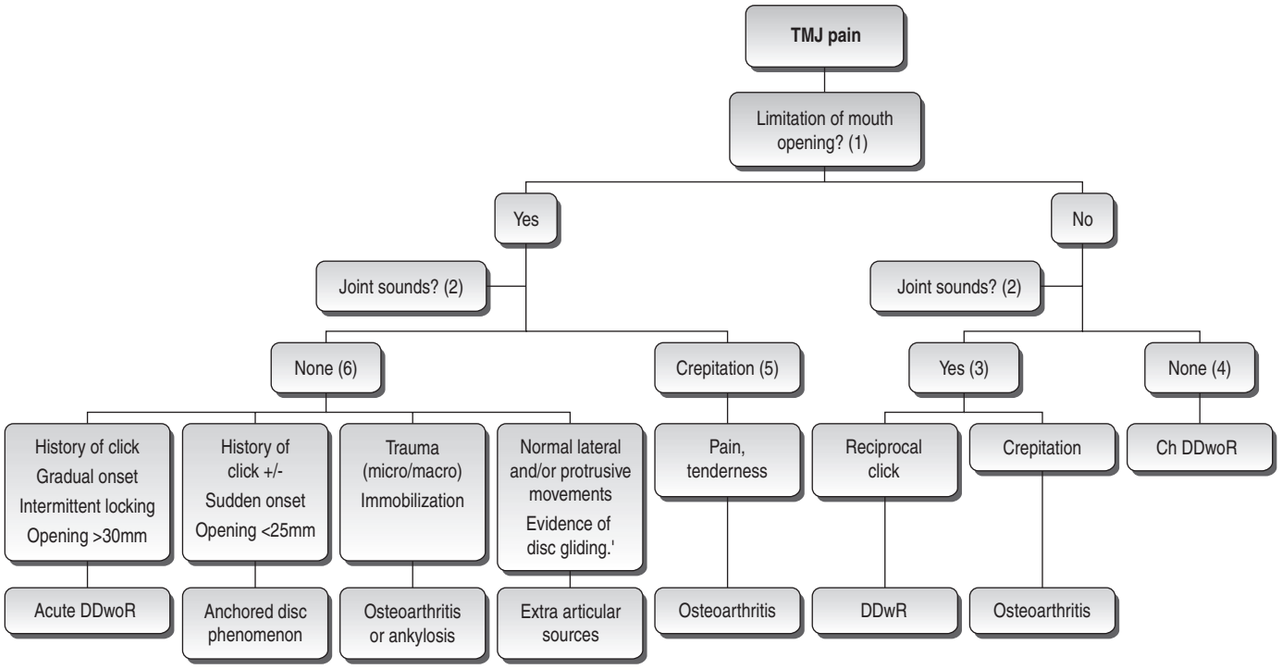

Fig. 8.10 • Flow diagram to aid in the diagnosis of painful temporomandibular joint disorders. A possible approach, as shown here (1), is to initially divide between patients with and without limitation of mouth opening (see text for definition). The presence of joint sounds (2) further subdivides these groups. In patients with no limitation in mouth opening and joint sounds (3) the quality may rapidly aid in diagnosis; crepitation is classical of osteoarthritis and reciprocal clicking of disc displacement with reduction (DDwR). No limitation of mouth opening in the absence of joint sounds (4) may be due to long-standing or chronic disc displacement without reduction (ChDDwoR). Limited mouth opening with crepitation is often present in active osteoarthritis (5); see also Case 8.2. The absence of joint sounds accompanied by limited mouth opening and a symptomatic TMJ is often difficult to diagnose (6). A history of clicking joints with a gradual onset of 'locking' or limitation of opening that is around 30mm suggest acute disc displacement without reduction (AcDDwoR). In contrast an acute onset of limited mouth opening of less than 25mm in a young patient with no history of clicks suggests anchored disc phenomenon (ADP). A history of clicks in the past does not exclude ADP. Macrotrauma, TMJ surgery or long-standing osteoarthritis that has been inadequately managed suggests that ankylosis is present (see Fig. 8.3). Extra-articular

11. Treatment of TMJ Disorders

Since the aetiology of TMJ disorders is considered multifactorial, numerous forms of ostensibly targeted therapies have been designed. Modalities include mechanistic approaches such as orthotic or occlusal therapies, and biopsychosocial approaches that integrate behavioural therapies into the standard medical model of disease. Interestingly many of these interventions, alone or in combination, have an equally positive impact in reducing or eliminating patient's symptoms. Studies clearly show no superiority of any one treatment for patients with TMJ disorders; successful treatment outcomes typically follow a multimodal and often multidisciplinary approach.

Treatment of TMJ problems may be compared to the treatment of any other skeletal disorder. The ultimate goals are to decrease pain and increase function. Indeed, TMJ pain and/or dysfunction are the clearest indicators that treatment should be offered. Dysfunction includes limited mandibular range of motion and reduced functionality. However, it is important to clearly state the treatment objectives to the patient; elimination or alleviation of pain and dysfunction may not always be accompanied by eradication of joint sounds. It is important to stress to the patient that long-term follow-up of clicking joints show no significant deterioration in the vast majority of patients (Randolph *et al* 1990) and no advantage of surgical over non-surgical techniques in controlling deterioration (Greene and Laskin 1988). A conservative approach is therefore successful for most patients.

On the rare occasions when a patient complains of a disturbing joint sound even in the absence of pain or dysfunction surgical treatment may be offered; see below.

11.1. Non-surgical Options

Non-surgical treatment of painful, dysfunctional TMJs commonly relies on the use of patient education and awareness techniques, joint unloading by functional modification or appliances, physical therapy or manipulation (Jagger 1991; Nicolakis *et al* 2001), bite appliances (Ekberg 1998; Ekberg *et al* 1998; Ekberg and Nilner 2002), medications (Ekberg 1998) and cognitive behavioural therapy. Other less common modalities include warming (Dahlstrom 1992), soft laser (Fikackova *et al* 2006) and complementary

or alternative medicine (DeBar *et al* 2003; Ernst and White 1999; see Chapter 17), but their efficacy is unclear. Because most patients with painful TMJs have a good prognosis the aim of treatment is palliative and supportive. Even in patients with DDwoR the use of a conservative approach is effective (Murakami *et al* 2002) and is in no way inferior to surgical intervention in controlling signs and symptom (Schiffman *et al* 2007). However, patients with a history of a persistently limited mouth opening (<30mm) have been shown to be relatively resistant to non-surgical intervention (Iwase *et al* 2005).

11.1.1. Joint Unloading: Functional Behavioural Modification

In some patients clenching or other parafunctional habits (protrusion) may be associated with TMJ pain; behavioural modification to stop these will alleviate symptoms. In most cases of pain an initial period of rest and a soft diet is also useful. Rest, which is beneficial for any symptomatic synovial joint, is not totally relevant for the TMJ since it is involved in essentially daily functions such as eating, swallowing and speech. Moreover sleep parafunction is a major source of TMJ overloading and is not controllable by the patient, so appliances are indicated. The use of appliances is described below and more extensively in Chapter 7.

Enrolling the patient to actively participate in the treatment with the aim to identify and reduce potentially damaging parafunction is essential. This approach may be enhanced with the use of biofeedback and stress management (see Chapters 4 and 7).

11.1.2. Physical Therapy

Joint mobilization is essential to maintain its long-term function and mobility. Exercises can be taught by the treating physician but in difficult cases or when cooperation is not achieved it is essential to refer the patient to a professional physiotherapist. Exercises that we prescribe include passive and active symmetric movements in all directions (Nicolakis *et al* 2001); upon maximal movement further stretching exercises are recommended to increase range of motion. Exercises combined with anti-inflammatory drugs are an effective strategy (Yuasa and Kurita 2001). Careful follow-up and meticulous recording of the extent of motion is essential. In addition the patient should be actively involved in goal setting and follow-up.

11.1.3. Bite Appliances (see also Chapter 7)

In the short term parafunctional habits may be controlled with appliances or drugs and thus reduce joint loading, but habits tend to recur when appliances are removed or following long-term use (Solberg *et al* 1975; Sheikholeslam *et al* 1986; Rugh and Harlan 1988).

There is evidence to suggest that the use of stabilization is beneficial for reducing pain severity in patients with painful joint disorders, at rest and on palpation, when compared to no treatment (Ekberg 1998; Ekberg *et al* 2002; Forssell and Kalso 2004). Repositioning appliances have been advocated to treat internal derangements and to 'recapture' the disc. However, in the long term, clicks or abnormal disc positions tend to recur (Lundh *et al* 1985; Tallents *et al* 1990) and these appliances fail to recapture the disc in ID without reduction (Eberhard *et al* 2002). Indeed repositioning appliances have no significant benefit over stabilization appliances in the treatment of TMJ sounds (Tecco *et al* 2004) and may induce irreversible occlusal changes. Recent data suggest that flat centric splints will relieve clinical symptoms more efficiently than repositioning splints in patients with DDwoR (Schmitter *et al* 2005). In a randomized clinical trial, a group of patients with painful DDwR were treated with three types of bite plates; flat occlussal, canine-guided and non-occluding (Conti *et al* 2006). After six months all patients with occluding splints had significant improvement in pain with no difference between the type of guidance (Conti *et al* 2006). Interestingly the frequency of joint noises decreased over time, with no significant differences among the three groups. This effect has also been observed in non-treatment groups included in a similar study, suggesting natural fluctuation in the expression of clicks (Conti *et al* 2005).

Reduction of TMJ loading may be obtained by constructing an appliance with occlusal contacts in the posterior region only (Nitzan 1994). This type of interocclusal appliance reduces the pressure generated in the joint during active clenching by 81.2% (Nitzan 1994). The use of techniques to off-load the TMJ is not unlike the effect of different bandaging techniques as an adjunct to orthopaedic management.

It is imperative that patients are informed that intraoral appliances are a temporary means of management; long-term use is associated with uncontrolled changes in occlusion.

11.1.4. Medications

Most cases with TMJ pain will improve with the use of mild analgesics and NSAIDs. NSAIDs, both topically and systemically, have been shown to be effective in TMJ disorders (Ekberg 1998; Thie *et al* 2001; Di Rienzo Businco *et al* 2004). There are data suggesting that the nonspecific NSAIDs, such as naproxen, may be more effective than specific cyclooxygenase (COX)-2 inhibitors in the management of TMJ pain (Ta and Dionne 2004). Since naproxen has a better cardiovascular safety profile than COX-2 inhibitors, this would suggest their preferential use; see Chapter 15. In severe cases, the combination of initial steroid therapy followed by an NSAID was as effective as surgical or other management of DDwoR (Schiffman *et al* 2007).

In the absence of extensive and specific research on drug use in inflammatory TMJ disorders (List *et al* 2003) results of experimental models or of clinical trials of other

joints affected by osteoarthritis may be extrapolated. For example, both acetaminophen and COX-2 inhibitors were able to reverse the behavioural effects of experimental TMJ inflammation in rats (Ahn *et al* 2005). Celecoxib (200–400 mg/d) was compared to acetaminophen (slow release formulation 3990 mg/d) in patients with osteoarthritis in various sites (Yelland *et al* 2007). Paracetamol was found to be as effective as celecoxib by most patients (Yelland *et al* 2007). A systematic review on the treatment of osteoarthritis of the knee or hip concluded that acetaminophen in doses of about 4 g daily is clinically effective and superior to placebo (Towheed *et al* 2006). However, NSAIDs (e.g. ibuprofen 1200 mg/d, diclofenac 75–150 mg/d, celecoxib 200 mg/d) improve pain, function and stiffness more than acetaminophen, especially in moderate to severe cases. Patients taking traditional NSAIDs were more likely to have adverse gastrointestinal effects but otherwise there was no major difference in side effects relative to acetaminophen. This would suggest that NSAIDs should also be employed in TMJ osteoarthritis; however, most of the studies were only about 6 weeks long (Towheed *et al* 2006). For long-term management the efficacy of NSAIDs must be countered with severe side effects on the gastrointestinal, renal and cardiovascular systems—factors that may favour the use of acetaminophen. Renal and cardiovascular risks are particularly relevant for the specific COX-2 inhibitors that also involve increased cost. Systemic side effects may be minimized by the use of topical application of diclofenac (see Chapter 16).

Taken together the data suggest that for short-term management of TMJ pain and osteoarthritis NSAIDs (naproxen, ibuprofen) are most likely to be effective, with minimal side effects. It is important to emphasize that renal and cardiovascular risks are increased within one month of NSAID use (see Chapter 15), so we suggest titrating clinical response and transferring patients at the earliest opportunity to other analgesics. Particularly in patients with medical comorbidity (cardiovascular, renal) acetaminophen is the drug of choice, although in patients with gastrointestinal problems NSAIDs may be used in conjunction with a proton pump inhibitor (consult the managing physician). For long-term management, the use of mild analgesics, such as acetaminophen, is effective for patients with TMJ pain and certainly safer. The advantages of mild analgesics over NSAIDs are discussed in Chapter 15. Rarely stronger analgesics such as mild opioids may be needed for short-term therapy in severe cases, or postoperatively. All the above medications, drug dosages, schedules and side effect profiles are extensively reviewed in Chapter 15. More recent data suggest that drugs specifically acting on cytokines may be clinically useful (Kopp *et al* 2005). Infliximab, an anti-TNF-α agent, was given systemically to patients with TMJ pain who reported significantly reduced pain levels (Kopp *et al* 2005).

When comorbid muscle pain is present the use of adjuvant analgesics is beneficial. These drugs include muscle relaxants (cyclobenzaprine), antidepressants (e.g. amitriptyline) and anticonvulsants (gabapentin, clonazepam) and are reviewed in Chapter 16. The analgesic effects of antidepressants may account for their use in patients with articular disorders (Plesh *et al* 2000; Rizzatti-Barbosa *et al* 2003; Johansson Cahlin *et al* 2006). Based on two pilot studies, chondroitin sulfate or glucosamine hydrochloride are effective in TMJ pain (Nguyen *et al* 2001; Thie *et al* 2001); see also Chapter 17.

11.2. TMJ Surgery

Surgery of the temporomandibular joint plays a small but important role in the management of patients with TMJ disorders. The literature has shown that about 5% of patients with TMJ disorders require surgical intervention and a spectrum of invasive procedures, from simple arthrocentesis to more complex open joint surgical procedures, is available. Each surgical procedure should have strict criteria for which cases are most appropriate. However, each procedure has its enthusiastic supporters, and specific criteria for the most appropriate intervention in each diagnosis are lacking. Thus the literature is based more on observation than scientifically controlled studies. Recognizing that scientifically proven criteria are lacking we will discuss the criteria for each procedure, ranging from arthrocentesis to complex open joint surgery. The discussion will include indications, brief descriptions of techniques, outcomes and complications for each procedure.

11.2.1. Indications

Indications for surgery of the temporomandibular joint may be divided into relative and absolute (Dimitroulis 2005a,b). Absolute indications are reserved for those where surgery has an undisputed role such as tumours, growth anomalies and ankylosis of the TMJ.

Surgical intervention of the dysfunctional or painful joint must be based upon clearly defined criteria, such as:

1. Pain and/or dysfunction of such a magnitude as to constitute a disability to the patient;
2. Failure of non-surgical therapy to resolve the problem;
3. Documentation of TMJ intracapsular pathologic condition or anatomic derangement that is a major source of the patient's pain and/or dysfunction; and
4. Amenability of the condition to surgical intervention.

While the indications for surgery may appear clear, they are in fact, nonspecific. The first criteria, significant TMJ pain and dysfunction, may be the most important. What distinguishes the surgical candidate from the group of non-surgical patients is localization of the pain and dysfunction to the TMJ. The more localized the pain and dysfunction are to the TMJ, the better the prognosis for a successful surgical outcome. Conversely, the more diffuse the pain and dysfunction, the less likely surgical

intervention will succeed. The decision for surgical intervention should be made based on clinical findings in conjunction with the impact of the pain and dysfunction on the well being of the patient, balanced against the prognosis if surgery is not performed. Surgical candidates should have localized, continuous TMJ pain that is moderate to severe, and becomes worse during mandibular function, i.e. talking or chewing. They may present limited movements with or without pain or other signs of dysfunction such as painful clicking, crepitation or locking of the TMJ.

The second criterion, refractory to non-surgical treatment, is also nonspecific. Temporomandibular disorders encompass a wide variety of aetiologies and complaints, and therefore, there is no clear consensus on a protocol for conservative or non-surgical treatment. None the less, most clinicians understand what non-surgical or conservative therapy involves. It typically includes a combination of patient education, medication, physical therapy, an occlusal appliance and possibly counselling or behavioural therapy. Most patients will respond successfully to this treatment, while others may see a reduction in symptoms over time, even in the absence of treatment. Therefore a surgical consideration should be reserved only for patients who fail to respond successfully over a reasonable period of time. It must also be stressed that not all patients who fail non-surgical treatment are surgical candidates. Surgical treatment must be limited to those who respond to the first criteria of pain and dysfunction arising from within the TMJ. Patients who have pain and dysfunction arising from the masticatory muscles or other non-TMJ sources are not surgical candidates and may be made worse by surgical intervention.

The third criterion, imaging evidence of disease, appears to be the most objective; however, imaging should not be interpreted in isolation. The correlation of disc derangement, dysfunction, and osteoarthrosis found on imaging with pain is poor (Kircos *et al* 1987b; Kozeniauskas and Ralph 1988). Therefore imaging should be used only to confirm and support the clinical findings. Surgery cannot be performed on the basis of imaging alone.

Surgical interventions include arthrocentesis, arthroscopy, condylotomy and open joint procedures such as disc repositioning and discectomy. Randomized clinical trials comparing these procedures do not exist, so the surgical procedure selected is based mostly on the surgeon's experience. Each procedure does have specific benefits as well as risks. Therefore, the procedure which has the highest potential for success with the lowest risks and most cost effectiveness should be chosen for the patient's specific problem.

Based on our experience arthrocentesis and arthroscopic lavage and lysis should be used for TMJ pain during function, intermittent clicking or limited opening (Kendell and Frost 1996). Condylotomy for TMJ pain with little or no restriction of opening and open TMJ surgery should be reserved for advanced cases of internal derangement and osteoarthrosis.

11.2.2. TMJ Arthrocentesis

In our view, arthrocentesis is probably the first surgical procedure that should be employed in patients with TMJ pain and dysfunction that have not responded to conservative therapy.

Studies on the effect of arthrocentesis on DDwoR have shown consistently improved mouth opening and decreased pain (Han *et al* 1999; Carvajal and Laskin 2000; Emshoff *et al* 2003b; Yura and Totsuka 2005). Our clinical experience suggests that range of jaw movements in DDwoR only improve marginally following arthrocentesis: interincisal opening may increase from about 32 to 36 mm, and lateral excursions from 8 to 8.5 mm. Pain and dysfunction levels are improved but response is unpredictable. These findings are expected as arthrocentesis and arthroscopic lavage are incapable of changing the disc's shape or position (Montgomery *et al* 1989; Moses and Poker 1989). The marked improvement described in the literature is probably in a group of patients that were not strictly defined and may include other disorders.

In a group of patients with disc displacements resistant to conservative therapy arthrocentesis was not significantly different to arthroscopy for decreasing patient reports of pain and increasing functional mobility of the mandible (Fridrich *et al* 1996). Since arthrocentesis is a simple outpatient procedure performed under local anaesthetic and is relatively safer than arthroscopy, it should be tried first.

11.2.2.1. Technique

Murakami et al (1987) were the first to offer a systematic description of TMJ arthrocentesis, which they termed 'manipulation technique followed by pumping and hydraulic pressure'. Arthrocentesis of the TMJ, as we present here, is a modification of the traditional method, and in which two needles instead of one are introduced into the upper joint space. This adaptation permits massive lavage of the joint, in addition to aspiration and injection (Nitzan et al 1991b).

The patient is seated inclined at a 45° angle, with the head turned to the unaffected side to provide an easy approach to the affected joint. After proper preparation of the target site, the external auditory meatus is blocked with cotton soaked in mineral oil. The points of needle insertion are marked on the skin, as follows. A line is drawn from the middle of the tragus to the outer canthus. The posterior entrance point is located along the canthotragal line, 10 mm from the middle of the tragus and 2 mm below the line (Fig. 8.11). The anterior point of entry is placed 10 mm farther along the line and 8–10 mm below it. These markings over the skin indicate the location of the articular fossa and the eminence of the TMJ. It is important that these points are used as guides; the precise location needs to be confirmed by careful examination of the patient's anatomy.

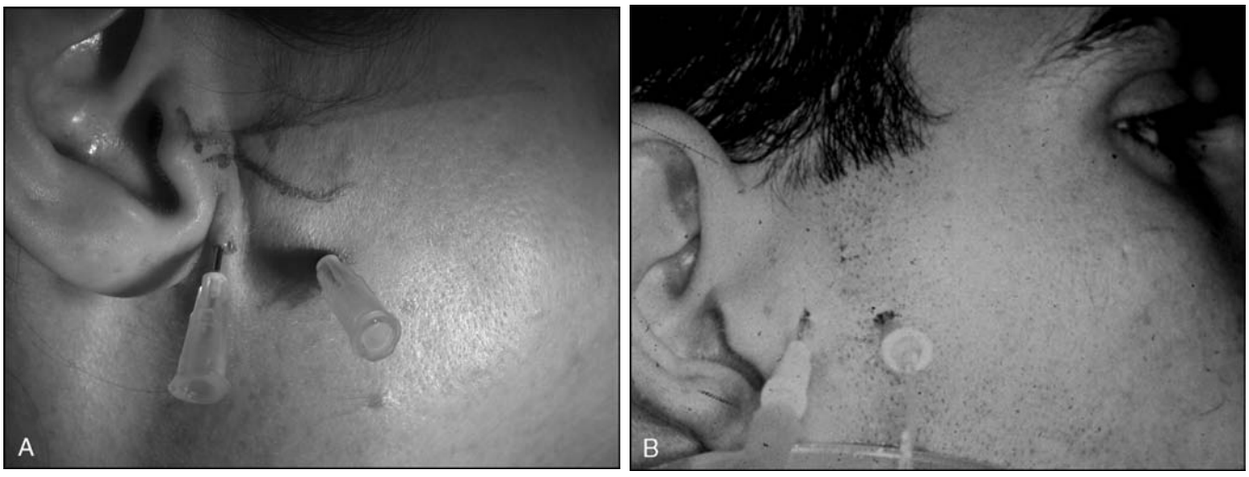

Fig. 8.11 • Arthrocentesis. Two needles are placed in the anterior and posterior recesses of the upper joint space (a). Irrigation is then performed (b) as described in the text.

11.2.2.1.1. Stages in arthrocentesis.

1. A local anaesthetic is injected at the planned entrance points, avoiding penetration into the joint and injection into the synovial fluid.
2. If samples of synovial fluid are needed for research or diagnostic assessment the procedure is continued by injecting 1 mL of lactated Ringer's solution into the superior compartment at the posterior point, and immediately aspirated. This procedure is repeated three times to obtain a sufficient amount of fluid for diagnostic and research purposes.
3. If the procedure is purely for therapeutic purposes stage 2 is omitted. Surface anaesthesia from stage 1 is then followed by injection of 2 to 3 mL bupivacaine 0.5% to distend the upper joint space and anaesthetize the adjacent tissues through the posterior point using a syringe with a 19-gauge needle. A second 19-gauge needle is then inserted into the distended compartment in the area of the articular eminence (anterior point of entry) to enable free flow of Ringer's solution through the superior compartment (Fig. 8.11). Slight adjustment of the needle position may be necessary. In cases of sluggish outflow, additional needles may be inserted into the distended compartment to enhance the transport of the solution. Zardeneta *et al* (1997) recommend a free flow of 100 mL of Ringer's solution, because denatured haemoglobin and various proteinases were recovered in this fraction. Later studies suggested that 300–400 mL should be used for the washout of bradykinin, IL-6 and proteins (Kaneyama *et al* 2004). A simplified procedure is one in which the second needle is inserted next to the first one, into the posterior rather than the anterior recess, and saline is then flushed through the upper compartment. During the lavage, the mandible is moved through opening, excursive and protrusive movements to facilitate lysis of adhesions (Segami *et al* 1990).

11.2.2.2. Mode of Action

By forcing apart the flexible disc from the fossa and by washing away degraded particles and inflammatory components arthrocentesis re-establishes joint movements and reduces both load and pain, which is the hallmark of joint health. Upon termination of the procedure and following the removal of one needle, medication can be injected into the joint space. Hyaluronic acid is an example of such a supplement of arthrocentesis (Alpaslan *et al* 2000; Xinmin and Jian 2005), the effectiveness of which is still debated (Shi *et al* 2003). The potency of hyaluronic acid will be minimal if inflammatory products in the affected joint are allowed to degrade it, but with the removal of the inflammatory products by arthrocentesis the hyaluronic acid remains intact and is probably more effective.

Reports on the elimination of products from the diseased TMJ by arthrocentesis suggest that this may be a major mode of action; see the earlier section on pathophysiology. Arthrocentesis is also an important diagnostic tool. Adhesions or osteophytes are not always unambiguously diagnosed by available imaging techniques as the causes of limitation or pain. Failure of lavage implies that surgical means may be required and are legitimate in order to release the joint and restore movement. Indeed, arthrocentesis is a prerequisite for most TMJ surgical intervention.

11.2.2.3. Complications

One should be aware that temporary facial weakness or paralysis, as a result of the use of a local anaesthetic, and/or swelling of the neighbouring tissues caused by perfusion of Ringer's solution might occur during arthrocentesis. Both signs are transient and disappear within hours. Numerous other complications as a result of arthrocentesis, including extradural haematoma, have been described. Correct diagnosis, appropriate treatment approach and careful surgical technique should prevent complications.

11.2.3. TMJ Arthroscopy

TMJ arthroscopy is a minimally invasive procedure but is, however, usually performed under general anaesthesia in the operating room. It is very much an equipment-dependent procedure that requires considerable manual dexterity on the part of the surgeon. TMJ arthroscopy now plays a major role in the surgical management of TMJ internal derangement and osteoarthrosis.

11.2.3.1. Technique

TMJ arthroscopy involves the placement of an arthroscopic telescope (1.8–2.6mm in diameter) into the upper joint space (UJS) of the TMJ, and a camera is attached to the arthroscope in order to project the image onto a television monitor. The surgeon must conceptualize a three-dimensional space on a two-dimensional image. A second access instrument is placed approximately 10–15mm in front of the arthroscope. This access point provides an outflow portal for irrigation and access for instrumentation of the joint space. The examination is started posteriorly by identifying the posterior attachment tissue. The synovial lining is inspected for the presence of inflammation such as increased capillary hyperaemia. The junction of the posterior band of the disk and posterior attachment tissues can be identified. Movement of the joint allows for the identification of clicking or restriction in movement of the disk. As the arthroscope is moved through the UJS the articular cartilage is inspected for the presence of degenerative changes such as softness, fibrillation or tears. The joint space is also inspected for the presence of adhesions, loose bodies or other pathology. The integrity of the disc or perforations of the disc or posterior attachment tissues can be identified. Although the lower joint space (LJS) is not usually examined, the presence of a perforation in the disk or posterior attachment may allow limited examination of the LJS and condyle. Although sophisticated operative techniques ranging from ablation of adhesions with lasers to plication of the disk have been developed, most surgeons limit the use of arthroscopy to lysis of adhesions and lavage of the UJS. Lysis of adhesions is accomplished most often by sweeping either the arthroscope or the irrigation cannula through the adhesions and tearing them. After completion of the examination the joint space is thoroughly irrigated to remove debris and small blood clots. The patient is usually discharged the same day after recovering from the anaesthesia. Postoperative care includes a non-chew soft diet for a few days, range of motion exercises continued for several days, an intraoral appliance and analgesics as necessary for pain control.

Multiple studies report 80–90% success rate with arthroscopic lysis and lavage for the management of patients with painful limited mouth opening (McCain *et al* 1992; Murakami *et al* 1998b, 2000; Reston and Turkelson 2003; Hall *et al* 2005; Smolka and Iizuka 2005; Schiffman *et al* 2007). The majority of patients have decreased pain and improved mouth opening. Murakami *et al* (1998b, 2000) have shown in studies with 5- and 10-year follow-up that arthroscopic lysis and lavage is successful for all stages of internal derangement, and that results are comparable to those obtained with open surgery procedures. Data from surgical arthroscopic techniques such as disk repositioning are difficult to interpret and it is unclear whether the outcomes are better than those obtained with simple lysis and lavage.

11.2.3.2. Mode of Action

Basically arthroscopy is a sophisticated method for lavage and lysis of adhesions in the TMJ (see above section on arthrocentesis). It offers the additional benefits of allowing the use of rotatory instruments, lasers and other microsurgical implements to modify anatomy, cauterize tissues, repair the disc and remove loose bodies.

11.2.3.3. Complications

The advantages of TMJ arthroscopy are that it is minimally invasive and causes less surgical trauma to the joint. Surgical complications are rare and mostly limited to reversible effects, some similar to those observed in arthrocentesis (Gonzalez-Garcia *et al* 2006). Patient recovery is rapid and healing time is shorter than with open surgical procedures. The disadvantages include the surgical limitations, the necessity for sophisticated equipment and a high level of training.

11.2.4. Modified Condylotomy

The modified TMJ condylotomy is the only TMJ surgical procedure that does not invade the joint structures. It is a modification of the transoral vertical ramus osteotomy used in orthognathic surgery. Although some authors recommend modified condylotomy as the surgical treatment of choice for all stages of TMJ internal derangement it seems to be most successful when used to treat painful TMJ internal derangement without reduced mouth opening (Hall *et al* 1993).

11.2.4.1. Mode of Action

The objective of the procedure is to surgically increase the joint space between the condyle and the fossa, thus allowing repositioning of the condyle anteriorly and inferiorly beneath the displaced disc.

Hall *et al* (1993) reported good pain relief in about 90% of 400 patients treated over a 9-year period with modified condylotomies. In follow-up studies 94% success in patients with disk displacement with reduction has been reported. Interestingly 72% of these patients had a normal disk position when evaluated with follow-up MRIs (Hall *et al* 2000a). This would suggest that the procedure is able to provide optimal conditions for the restoration of normal disc–condyle relationship. However, in a group of patients with disk displacement without reduction the success rate for modified condylotomy was slightly less at 88% (Hall *et al* 2000a).

11.2.4.2. Technique

The modified condylotomy is performed under general anaesthesia usually as an outpatient procedure but may require overnight stay in the hospital. An incision is made intraorally along the anterior border of the mandibular ramus. After exposure of the lateral aspect of the mandibular ramus a vertical cut is made posteriorly to the lingula from the coronoid notch to the mandibular angle. After mobilization of the condylar segment the medial pterygoid muscle is stripped from the segment. The mandible is then immobilized using maxillomandibular fixation (MMF). Although the surgery is simple, there is a period of postoperative rehabilitation involving 2–3 weeks of MMF followed by training elastics so that the occlusion is maintained.

11.2.4.3. Complications

The most significant potential complication of the modified condylotomy is excessive condylar sag, resulting in malocclusion. In one series of cases there was only a 4% complication rate which consisted primarily of minor occlusal discrepancies (Hall *et al* 2000b). Despite the simplicity of the procedure and its high success rate the procedure has not become widely used. The reasons for this are unclear but are most likely related to the necessity for MMF and the fear of excessive condylar sag resulting in an unstable condylar position with malocclusion.

11.2.5. Open Joint Surgery (Arthrotomy)

Open joint surgery is indicated for those patients with TMJ internal derangement and osteoarthritis who have failed to respond to simpler surgical procedures or have failed previous open surgery. In the cases of previous surgery the surgeon must be very hesitant to perform repeated surgery because the success rate for repeat surgery is very low; in fact after two surgeries, it may approach zero. The surgeon must be certain that the source of the pain and/or dysfunction is arising from within the joint. Severe mechanical interference such as loud, hard clicking or intermittent locking associated with loud, hard clicking is an indication to perform open surgery without first performing simpler procedures because experience indicates that simpler procedures are rarely successful in these cases.

Although the use of open joint surgery has decreased significantly it still has a small but important role in the surgical management of TMJ disorders. While other surgical procedures provide a limited range of options open TMJ surgery provides the surgeon with an unlimited scope of procedures ranging from simple debridement of the joint to removal of the disk. Disk repositioning procedures are less commonly performed today compared to the 1980s and 1990s, because most patients with disks that can be preserved are successfully treated with simpler procedures. Advanced cases of internal derangement which have degenerative disks and severe arthritic changes may require partial diskectomy. Arthroplasty in the form of bone contouring of the articular eminence or condyle is sometimes necessary, particularly with disk repositioning procedures.

Open joint surgery is performed under general anaesthesia in the hospital, and usually requires a one- to two-day stay. The most common surgical approach is via a preauricular incision. Incorporating the tragus of the ear into the incision line is often used for cosmetic purposes. Exposure of the capsule is carefully performed in order to protect the temporal branches of the facial nerve. After exposure of the capsule the UJS is entered, and it is inspected for the presence of adhesions. The contour and integrity of the fossa and eminence are evaluated and lastly the disk is visualized. Evaluation of the disk includes its colour, position, mobility, shape and integrity.

11.2.5.1. Disk Repositioning

If the disk is intact and can be repositioned without tension then disk repositioning can be performed by removing excess tissue from the posterior attachment tissues, repositioning the disk and stabilizing it with sutures. Bone recontouring of the glenoid fossa and/or articular eminence is generally performed, especially in cases of gross mechanical interference or advanced degenerative joint disease. The goal of disk repositioning surgery is the elimination of mechanical interferences to smooth joint function. After completion of the intra-articular procedures, the UJS is irrigated and the soft tissues are closed.

Exercises to improve range of motion are started immediately after the surgery. Continuation of postoperative conservative treatment is very important in order to assure a successful outcome. A soft non-chew diet is recommended for six weeks after the surgery.

The literature indicates that disk repositioning surgery is successful in 80–95% of cases; however, experience indicates that this may be an overestimate (Marciani and Ziegler 1983; Hall 1984; Piper 1989; Dolwick and Nitzan 1990, 1994). It has been found that while disk repositioning surgery significantly reduced pain and dysfunction in 51 subjects evaluated up to six years postoperatively, improvement in disk position was not maintained over the follow-up period for most patients (Montgomery *et al* 1992). Despite these findings the preservation of a healthy, freely mobile disk is justified.

11.2.5.2. Diskectomy

A diseased or deformed disk that interferes with smooth function of the joint and cannot be repositioned should be removed. Only that portion of the disk which is diseased and deformed needs to be removed. The synovial tissues should be preserved as much as possible. After removal of the disk only minimal bone recontouring should be performed as exposure of bone marrow may result in heterotopic bone formation. Limitation caused by the heterotopic bone can be prevented by intensive physiotherapy. In order to minimize the risk of heterotopic bone formation the placement of an interpositional fat graft into the joint space is recommended. After completion of the

intra-articular procedures, the joint space is irrigated and the soft tissues are closed.

The postoperative findings are the same after diskectomy as described for disk repositioning. The postoperative recommendations are also the same except that a soft non-chew diet is recommended for six months.

Diskectomy of the TMJ has the longest follow-up studies of any procedure for management of TMJ internal derangement. There are four studies with at least 30 years follow-up that report excellent reduction in pain and improvement in function in most patients (Silver 1984; Eriksson and Westesson 1985; Tolvanen *et al* 1988; Takaku and Toyoda 1994). Postoperative imaging studies of diskectomy patients generally show significant changes in condylar morphology (Eriksson and Westesson 1985). These changes are thought to be adaptive changes and not degenerative. Despite the excellent long-term success associated with TMJ diskectomy surgeons seem reluctant to perform this procedure.

Immediately after the surgery the patient may experience swelling in front of the ear and a slight change in occlusion and limited mouth opening, which usually resolves in about two weeks. All patients experience some numbness in front of the ear, which resolves in about six weeks. Patients normally have moderate discomfort, which lasts one to two weeks. The most significant complication associated with open surgery is facial nerve injury. While total facial nerve paralysis is possible, it is rare. Inability to raise the eyebrow is the most commonly observed finding and occurs in about 5% of cases, but usually resolves within three months. It is permanent in less than 1% of cases. Other complications are limited opening and minor occlusal changes. The complications associated with diskectomy are similar to those associated with disk repositioning. The growth of heterotopic bone is more common after diskectomy than other TMJ surgical procedures. This can be a significant complication which can result in complete ankylosis of the joint. The frequency of occurrence of heterotopic bone formation is unclear.

11.2.5.3. TMJ Replacement

A complete discussion of total TMJ replacement is beyond the scope of this chapter. The discussion will be limited to alloplastic total joint replacement in adult patients who have advanced degenerative joint disease, ankylosis or complications of previously preformed open surgery. The use of alloplastic materials to reconstruct or replace the diseased tissues of the TMJ caused disastrous results in the 1980s and 1990s. The use of Proplast-Teflon and Silastic implants caused significant foreign body reactions with severe destruction of the TMJ structures (Dolwick and Aufdemorte 1985; Heffez *et al* 1987; Westesson *et al* 1987; Kaplan *et al* 1988). This experience has led some surgeons to reject the use of alloplastic TMJ prosthesis in favour of autologous tissues such as costochondral grafts for TMJ reconstruction (MacIntosh 2000). While there are advantages to using autologous tissues, recently developed alloplastic TMJ prostheses provide safe and predictably successful reconstruction of the TMJ (Mercuri 2000; Quinn 2000).

Two basic prosthetic joints will be discussed: a patient-fitted prosthesis and stock prostheses. A patient-fitted prosthesis is a custom-made implant which has been used for over 10 years (Mercuri 2000). The prosthesis consists of a glenoid fossa implant which has an articular surface made of high molecular weight polyethylene attached to a pure titanium mesh. The body of the condylar prosthesis is made of medical grade titanium alloy with a cobalt chromium molybdenum condylar head. The process for making the prosthesis requires that a head CT be obtained from which an acrylic model of the patient's skull can be made. The planned surgery is performed on the model. The prosthesis is designed on the model and sent to the surgeon for approval. After approval the patient's prosthesis is made using CAD CAM technology.

In contrast a stock prosthesis consists of a prefabricated implant available in various sizes: small, medium, and large for the fossa, and three lengths (45, 50 and 55mm) for the condyle. The prosthesis includes a glenoid fossa component made of high molecular weight polyethylene and a condylar component made of cobalt chromium molybdenum alloy. The articular surface is the same on all three implants and only the flange varies in size.

The surgical placement is essentially the same for both implants. The surgery requires preauricular and retromandibular incisions for access to the TMJ and mandibular ramus. A gap arthroplasty is performed by removing either the diseased condyle or ankylosed bone. Generally a coronoidectomy is also performed. After the teeth are placed into maxillomandibular fixation the implants are fitted and secured using titanium screws. Stock implants are more difficult to place than the patient-fitted implants because the bony structures must be reshaped to fit the implants. The MMF is released, the occlusion is verified and the range of motion determined. If these are acceptable the wounds are irrigated, a fat graft is placed around the condyle and the soft tissues are closed.

The criteria used to determine success in complex, chronic TMJ pain patients are somewhat relative and as such precise success rates are difficult to determine. Successful outcome generally means that the patient has reduced pain levels, increased range of motion, improved function and an absence of surgical complications. Using these criteria the success rates are high for both prostheses.

Patients who have had multiple TMJ surgical procedures and who suffer from chronic pain generally experience about 50% pain reduction and gain 10–15mm of mandibular opening. It should be emphasized that total TMJ replacement is not necessarily a solution to the management of chronic TMJ pain. The TMJ prosthesis can be used to predictably restore occlusion and increase range of motion but pain relief is variable. On the other hand, both TMJ prostheses predictably provide pain-free restoration of occlusion and range of motion for patients who have TMJ reconstruction for ankylosis, tumours or other

conditions where pain is not an original component of the condition. This may suggest that the presence of pre-existing pain is a negative prognostic factor and may be related to plastic changes of the nervous system in persistent pain states (see Chapters 2 and 11).

The most significant complication following TMJ reconstruction with alloplastic implants is facial nerve injury. While uncommon it does occur more frequently than following routine open joint surgery, especially in patients with previous, multiple TMJ surgeries. The formation of heterotopic bone is a common complication, occurring in as many as 20% of the cases. Other complications such as infection, foreign body and allergic reactions, malocclusion and implant failure can occur but are rare. Complications requiring implant removal are unusual.

Unquestionably the patient-fitted prosthesis provides the best TMJ reconstruction. The surgery is easier to perform and the implants fit more accurately than a stock prosthesis. However, there is a need for both types of implants. Patient-fitted implants require 1–3 months to manufacture so immediate TMJ reconstruction is not possible. They are also more expensive than stock prosthesis. In addition, there are several situations in which two surgeries are necessary in order to use a patient-fitted prosthesis:

1. Patients who require significant mandibular repositioning to correct large malocclusions;
2. Patients with extensive bony ankylosis requiring large amounts of bone removal;
3. Patients with foreign bodies such as previously placed alloplastic TMJ prostheses that must be removed before an accurate CT can be obtained; and
4. Combination of 1 and 2.

When two surgeries are required it can be problematic to maintain the occlusion and function after the first surgery during the time the prosthesis is being constructed. Additionally two surgeries are inconvenient for the patient, prolong healing time, expose the patient to greater risks of complications and are more expensive. Stock joints can provide adequate reconstruction with a single operation in these situations. Conversely there are situations where a stock prosthesis cannot be used. These occur in patients who have extensive bone loss at either the lateral aspect of the fossa and articular eminence or the mandibular ramus, resulting in inadequate bone for placement of a stock prosthesis. There is great flexibility in the design of patient-fitted prosthesis which allows them to be adapted to a variety of complex clinical situations. The surgeon must be familiar with both types of prostheses so as to successfully meet the needs of the variety of TMJ patient conditions requiring TMJ replacement.

12. Conclusions

Complex referral patterns in the head and neck makes accurate diagnosis of TMJ-related pain difficult. Additionally diagnostic criteria are often not adhered to, leading to misdiagnosis of muscle-related pain as a TMJ disorder.

Most painful disorders of the TMJ may be successfully treated by conservative means. Surgical techniques include minimally invasive options such as arthrocentesis and arthroscopy, which have been shown to be highly effective in many patients resistant to conservative management. Relative indications for open surgical intervention include failed conservative therapy, and structural abnormalities causing pain and dysfunction. Open surgery allows for the most flexible treatment options but carries the greatest morbidity. However, open surgery is absolutely indicated for some TMJ disorders, mainly tumours and ankylosis. In a minority of cases prosthetic replacement of the TMJ may be needed and custom or stock options are available. It is essential that practitioners be familiar with all surgical techniques so as to be able to discuss options with the oral and maxillofacial surgeon involved. This will result in more optimal management of orofacial pain patients.

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