Contemporary Management of Temporomandibular Disorders

Non-Surgical Treatment

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Gianluca Martino Tartaglia
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Editors

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Foreword

All human beings have mechanisms of adaptation that allow them to react to the changes that occur both in their external environment and in their internal environment. When these mechanisms are overcome, people develop what we know as disease.

Temporomandibular disorders (TMD) are a musculoskeletal disease that can partially or globally affect the different structures that constitute the cranio-cervicomandibular unit. Therefore, one of the most important challenges in clinical practice is to carry out a careful anamnesis, the clinical examination, and when necessary, carry out complementary exams that allow one to achieve a correct diagnosis regarding the causes and mechanisms determining that the individual's capacity for adaptation has been exceeded, generating the disease that we know by the name of TMD. Therefore, it is clear that the most important phase in the management of TMD is **diagnosis**.

For many years, teaching in different dental schools was based on the fact that muscles, joints, and occlusal problems were the main causes of TMD (Axis I). However, in the early 1990s, several authors emphasized the importance of psychosocial factors (Axis II) in the genesis of TMD. Actually, there is currently a consensus that the cause of TMD is multifactorial, and that is the reason why in Volume II the relationship between bruxism during sleep and TMD (Chap. 1) and the relationship between trauma and whiplash injury in TMD (Chap. 2) are analyzed.

Based on the importance of considering the state of Axis I and Axis II in the diagnosis of the patient with TMD, Part III of this volume discusses management with respect to the dental treatment of TMD, and Parts II and IV discusses treatment with respect to the medical and adjunctive therapies for the management of TMD.

I invite readers to enjoy the careful material prepared by the authors of this Volume II, which is based on solid scientific evidence and fruitful clinical experience achieved through many years of professional practice.

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Rodolfo Miralles

Preface

The editors and contributors to this text find the study of temporomandibular joint disorders to be strikingly interesting and interlinked with many other fields, such as rheumatology, imaging, pain management, mental health, neuroscience, rehabilitation, dentistry, and surgery. We are all innovators in our own way, striving to bring to the field morsels of information arising from clinical and laboratory experience and research. No doubt, however, that many clinicians are convinced that TMD is a black hole, a field that is shrouded in mystery from which some patients emerge and others are lost in an endless loop of pain medication and soft diets. Volume II will hopefully inspire creativity in the diagnosis and care of TMD patients and will inform many types of healthcare providers.

The three editors of this book met in Barcelona, Spain, in 2016 to attend the TMJ Bioengineering Conference. In this beautiful and historic city, we heard incredible presentations and met an international group of surgeons, clinicians, and scientists. Many papers were presented by eminent speakers, and it was in this amazing European venue that the idea of collecting much of this incredible knowledge into a new contemporary text began to take shape. It is our desire that the reader learns and appreciates the temporomandibular joint for its complex beauty and biomechanics and gains an appreciation of the different modes of treatment. Our patient outcomes highlight the need for furthering our understanding of the mysteries of the temporomandibular joint.

Volume II focuses on the nonsurgical treatment of temporomandibular disorders. Part I informs us about the important role of bruxism and trauma in the development or perpetuation of TMD. Part II is concerned with medical treatment. Here we look at the role of various classic and specialty drugs that can be utilized in treatment. Part III reviews occlusion and its impact on temporomandibular disorders from both an orthodontic and prosthodontic perspective. We also present the orofacial evaluation of the TMD patient, which includes information on complementary exams, exercises, and function training. In Part IV, adjunctive therapies are discussed, which can significantly improve treatment outcomes. These topics include physical therapy, dry needling, laser, ultrasound, TENS, and chiropractic. We end this volume with the topic of psychiatric considerations for TMD and chronic pain patients. All TMD clinicians, surgeons and nonsurgeons, will recognize the impact that pain and dysfunction has upon a patient's mental health and his or her recovery trajectory.

The editors and the authors of the chapters herein hope that the valuable information presented will help you grow your knowledge base and improve your ability to successfully treat a wide variety of TMD disorders.

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Milano, Italy — Gianluca Martino Tartaglia
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Contents

Part I

Special Considerations: Bruxism and Trauma

Sleep Bruxism and Temporomandibular Disorders

1

Marc Schmitter

Abstract

TMDs have a multifactorial etiology: besides others, psychosocial and genetic aspects, habits, trauma, and bruxism have been proposed to cause and/or perpetuate TMD. This chapter will explore how investigators have attempted to diagnose and quantify bruxism and determine its relationship to temporomandibular disorders.

Temporomandibular disorder (TMD) is a collective term for a heterogeneous group of disorders of the temporomandibular joint (TMJ) and related muscles [1]. In the orofacial region, TMD is the most common cause of non-dental and noninfectious pain [2]. The most common complaint of patients with TMD is myofascial pain (MP) of the masticatory muscles [3]. MP in the orofacial area often occurs in conjunction with widespread pain throughout the body [4]. TMDs have a multifactorial etiology: psychosocial and genetic predispositions, habits, trauma and bruxism, and others have been proposed to cause and/or perpetuate TMD. This chapter will explore how investigators have attempted to diagnose and quantify bruxism and determine its relationship to temporomandibular joint disorders.

Bruxism is defined as "a repetitive jaw muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible" [5]. Bruxism has two circadian manifestations: it can occur during sleep (sleep bruxism) or during wakefulness (awake bruxism). According to a recent consensus paper, bruxism can be classified as "possible" (self-report), "probable" (self-report plus clinical examination), or "definite" (self-report plus clinical examination, plus polysomnographic recording) [5].

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The relation between TMD and bruxism can be assessed using either clinical studies or experimental studies (including finite element analysis, FEA) as described below.

However, given the current evidence, the relationship between bruxism and TMD seems to be still controversial at first glance. There are two major reasons for this dilemma: first, the complexity of the etiology of both bruxism and TMD and second the diagnostic uncertainty of both disorders [6].

1.1 TMD Diagnostics

When diagnosing TMD, it is mandatory to distinguish between myogenic and arthrogenic findings: myogenic and arthrogenic TMD might be caused and/or perpetuated by different causes. Thus, without this differentiation, it might be difficult to identify risk factors, including bruxism. However, most TMD patients suffer from myogenic pain—arthrogenic pain is much less common.

The clinical examination is the first step when assessing TMD-related problems. Muscle-related TMD can be diagnosed reliably when using standardized clinical examination protocols, e.g., the RDC/TMD (Research Diagnostic Criteria for Temporomandibular Disorders) or the DC/TMD (Diagnostic Criteria for Temporomandibular Disorders). Using non-standardized clinical examination protocols often results in unreliable and invalid results. However, it has been shown that the diagnosis of arthrogenic TMD is much more challenging as the clinical examination is not sufficiently reliable and valid, independent of the examination protocol used. Thus, the clinical examination should be complemented with imaging modalities. Imaging techniques offer distinct advantages over clinical evaluation as they can give definite evidence of pathological changes. A number of different techniques for imaging the temporomandibular joint (TMJ) have been described in the literature. Some imaging modalities, however, are unsuited to imaging soft tissue and cartilage structures (e.g., computed tomography). Other modalities provide useful images of these structures but have the disadvantage of being invasive (arthrography2) or involve X-ray. When a suitable surface coil is used, magnetic resonance imaging (MRI) provides good-quality images and gives definition of soft and hard tissue structures without radiation or invasiveness [7–9].

However, in many studies assessing the relationship between TMD and bruxism, neither standardized clinical examination protocols nor imaging modalities have been used. Moreover, some studies do not distinguish between arthrogenic and myogenic TMD. This results in a severe bias with respect to the valid identification of TMD patients. Thus, these types of reports make it difficult to draw any solid conclusions or add to the existing knowledge base.

1.2 Diagnosis of Bruxism

The diagnosis of bruxism is challenging also: it has to be distinguished between sleep and awake bruxism, it may fluctuate over time, it can be caused by drugs, it could be mild to moderate or severe, and it might be influenced by hormonal aspects, psychosocial aspects, etc.

Unfortunately, for awake bruxism there is relative paucity of studies. Cioffi et al. [10] concluded in their study that "individuals with masticatory muscle pain have an increased frequency of both high- and low-intense daytime clenching episodes." Other studies [11, 12] found that daytime clenching has to be considered to be a risk for TMD. However, the diagnosis of daytime bruxism is mainly based on questionnaires, which is associated with some limitations as objective data are missing relying on that methodology alone.

The gold standard for the diagnosis of sleep bruxism (SB), which is the focus of the present chapter, is polysomnography (PSG) [13]. As this technique is both time-consuming and costly, few studies used this technique when assessing the relation between sleep bruxism and TMD. Furthermore, these studies use PSG for one night only. This may miss SB as it might fluctuate over the course of a few nights, especially for infrequent grinders [14, 15]. However, unfortunately, both anamnesis (self-report/questionnaire) and clinical examination are unreliable [16], and the results should be interpreted with care. This is not surprising as clenching is often not detected by sleeping partners (because it is without noise), and wear can be caused by different sources and might, therefore, occur independent of bruxism. Thus, it seems to be hard to diagnose sleep bruxism in an adequate manner.

In 2012, an international consensus group [5] proposed the following approach to deal with these problems: sleep bruxism should be distinguished between possible (self-report), probable (self-report *plus* clinical examination), and definite (self-report *plus* clinical examination *plus* polysomnographic records). However, this proposal cannot help to overcome the aforementioned problems with respect to the validity of the diagnoses, as polysomnographic recordings are cost-intensive, time-consuming, and not available everywhere.

In recent years, portable EMG devices for domestic use and special occlusal splints have been introduced to diagnose sleep bruxism. Splints, however, have a significant disadvantage: they can interfere with occlusion that might bias the results of the measurement [17]. In fact, splints are often used as a biofeedback device to decrease activity. Thus, sleep bruxism cannot be distinguished from iatrogenic muscle activity caused or inhibited by the splint and "real" parafunctional activity. In contrast, portable EMG devices have several advantages: the devices do not interfere with occlusion, and they are easy-to-use, have reasonable accuracy and precision [18], and have high subject adherence and can be used for several nights at home. For some devices, a high sensitivity and specificity (compared to PSG) have been shown. Consequently, these devices might seem to support the valid diagnosis of sleep bruxism.

1.3 Sleep Bruxism and TMD

1.3.1 Studies Using Clinical Examination and/or Anamnesis

There are several studies quantifying the presence or absence of sleep bruxism using questionnaires and/or a clinical examination. This is not astonishing: this kind of study can be done very easily and rapidly, including a large number of subjects.

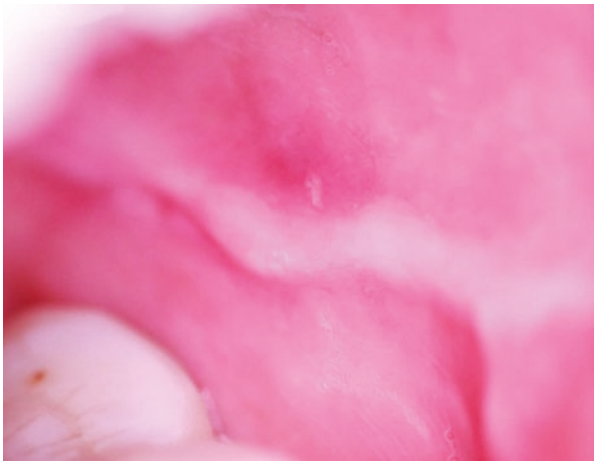

Fig. 1.1 Impressions in the cheek in a patient suffering from sleep bruxism

However, as previously described, these studies have a high risk of bias: it is very hard for a person to know about his/her own sleep bruxism. The bed partner may notice grinding, whereas clenching remains undetected. Raphael et al. [19] concluded that "self-reported SB failed to significantly predict the presence or absence of either moderate or severe SB as assessed by PSG." Thus, studies using questionnaires, only, should not be used to answer the question if TMD is related with bruxism. But what about the clinical examination? Wear facets, impressions in the cheek (Fig. 1.1), impressions in the tongue, muscular fatigue in the morning, etc. have been recommended to diagnose sleep bruxism [20]. However, Castroflorio et al. [16] found that the "false-positive and false-negative rates were unacceptable for all clinical signs/symptoms." In addition, the investigators concluded that clinical findings of bruxism did not correlate well with sleep bruxism diagnosed by portable EMG/ECG recorder. In other words, clinical findings commonly associated with bruxism may be seen in patients who do not show evidence of bruxism on portable EMG/ECG, and those who show bruxism on portable EMG/ECG may not show physical evidence of bruxism on clinical exam. Consequently, studies using clinical examinations have a high risk of being unreliable as well. In conglomerate, when addressing the relationship between TMD and bruxism, studies based on questionnaires and clinical examinations should be interpreted with care because the diagnosis of bruxism itself is problematic.

However, it is worth mentioning that most of the studies based on clinical examination protocols found a positive association between bruxism and TMD.

1.3.2 Studies Using Electromyography in a Home Setting

Several studies have shown that portable EMG devices (Fig. 1.2: two examples for portable EMG devices) are helpful to reliably diagnose sleep bruxism [16, 21]. There are several products, including disposable, single-use EMGs and EMGs in combination with heart frequency recordings in order to improve the validity of the

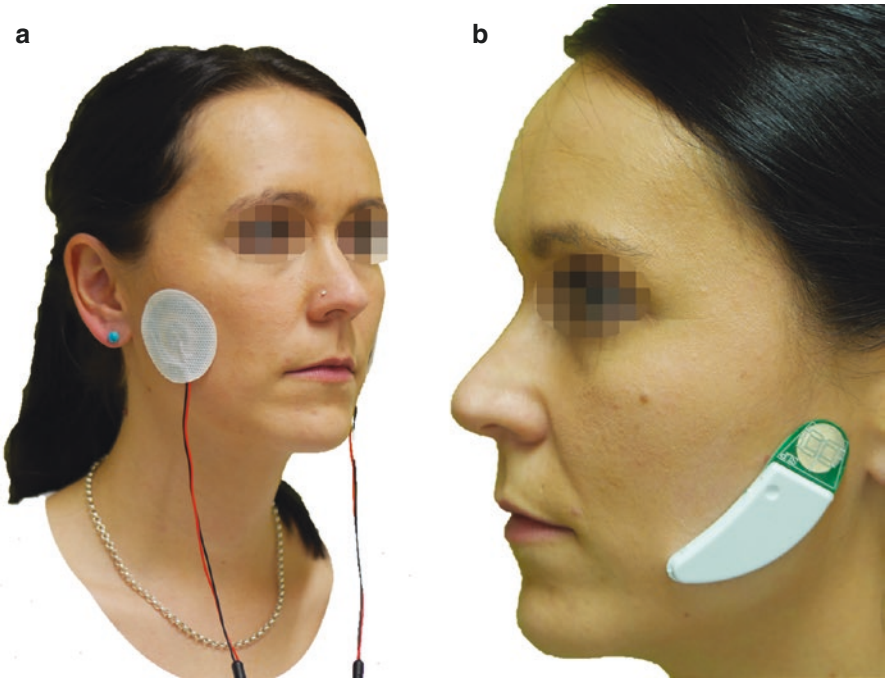

Fig. 1.2 (**a**) (Left side): BruxOff, OTBioenettronica, Italy. Records masseter muscle activity on both sides and heart frequency in order to improve validity of the measurement. Can be used for multiple nights. (**b**) (Right side): BiteStrip, SLP – Scientific Laboratory Products, Ltd, Israel. Records masseter muscle activity on one side. Disposable device can be used for one night only

measurement and exclude false-positive events. However, few studies used both portable EMG devices and reliable clinical TMD examinations in order to assess the association between TMD and sleep bruxism. Fortunately, a new generation of portable EMG devices has been introduced, which are easier to use, less expensive, and produce validated results. Schmitter et al. [22] found in their EMG study of 44 subjects that in patients suffering from myofascial pain, 71.4% were sleep bruxers. In contrast, in healthy subjects only 13.6% were sleep bruxers.

This literature provides increasing evidence that there is a positive association between myofascial pain and sleep bruxism, when a validated portable EMG confirms diagnosis of sleep bruxism.

1.3.3 Studies Using Polysomnography (PSG)

Few studies used PSG to assess the relation between TMD and bruxism. As previously described, PSG has some limitations also, although it is the gold standard in diagnosing sleep bruxism. Jimanez-Silva et al. [23] identified seven studies in their systematic review on this topic using PSG for the diagnosis of sleep bruxism. Four

of these studies used the RDC/TMD to diagnose TMD, which is a reliable clinical examination protocol.

Three of these seven studies could not find an association between bruxism and TMD; the other four studies found a positive relation. Further, both myofascial pain [24] and TMJ noises [25] were found to have an association with bruxism. Given that PSG is the benchmark for diagnosing sleep bruxism, these results have a high impact. However, as about 43% of the studies could not find an association between TMD and sleep bruxism, no decisive conclusion can be drawn. Furthermore, the quality of evidence of these studies was rated to be heterogeneous, and none of these studies obtained the highest score using the Newcastle-Ottawa Scale [23].

Nonetheless, it can be concluded that there is a clear trend indicating an association between TMD and sleep bruxism using PSG.

Although every endeavor has been made performing numerous studies, including questionnaires, clinical assessment, EMG, and PSG, the question of whether TMD is associated with bruxism cannot be conclusively answered. Thus, there is insufficient evidence to proof that there is an association between bruxism and TMD.

However, there are experimental and finite element studies available, which might help to answer this question.

1.3.4 Experimental Studies Using Bruxism Simulation and Studies Using Finite Element Analysis (FEA)

Taking into account biomechanics and the proposed pathophysiology of degenerative joint disease as laid out in Chap. 7, it seems obvious that clenching and/or grinding might result in higher muscle and joint strain. Higher strain, in turn, might cause pain and discomfort and signs of breakdown. However, it is necessary to validate these assumptions using both experimental studies and computer-based simulations.

1.3.4.1 Experimental Clinical Studies on Bruxism

In most experimental studies, clenching/grinding was simulated in healthy volunteers and/or in patients suffering from TMD [26–29]. Thus, daytime bruxism was simulated rather than sleep bruxism.

The duration of induced clenching, its intensity, and location (centric versus eccentric) varied from study to study. Some authors preferred 15 min at about 20–25% of the maximal voluntary clenching (MVC) force; others used up to 60 min or even some hours at different intensity levels (50% MVC, 10% MVC) [26–29]. This variation highlights the heterogeneity of the experimental studies and may be contributing to inconsistent findings. However, despite these problems, *most authors found that clenching results in low to high pain levels in subjects* [23], *affirming the aforementioned biomechanical considerations and the association between bruxism and TMD.*

1.3.4.2 Finite Element Analysis

FEA is a sophisticated, computer-based way to analyze stress in the TMJ. Based on this computer simulation, stress distribution and stress changes can be calculated and visualized using a specific software (Fig. 1.3). However, prior to the simulation, detailed data has to be acquired: material parameters of the tissue, muscle forces, etc. Without reliable and valid data, no reliable and valid simulation can be expected. This is the major limitation of this approach—particularly as it is often difficult to assess the validity of the selected parameters (e. g., material properties of the tissue).

Several studies used FEA in order to simulate the effects of bruxism on the TMJ [30–33]. Different FEA models have been used, based on different assumptions and material parameters. Despite these differences, most FEA studies found an increased stress in the TMJ due to clenching/grinding. *Consequently, based on the results of the FEA studies, an impairment of the temporomandibular joint disc and surrounding tissue with bruxism seems to be plausible, resulting in pain and discomfort.*

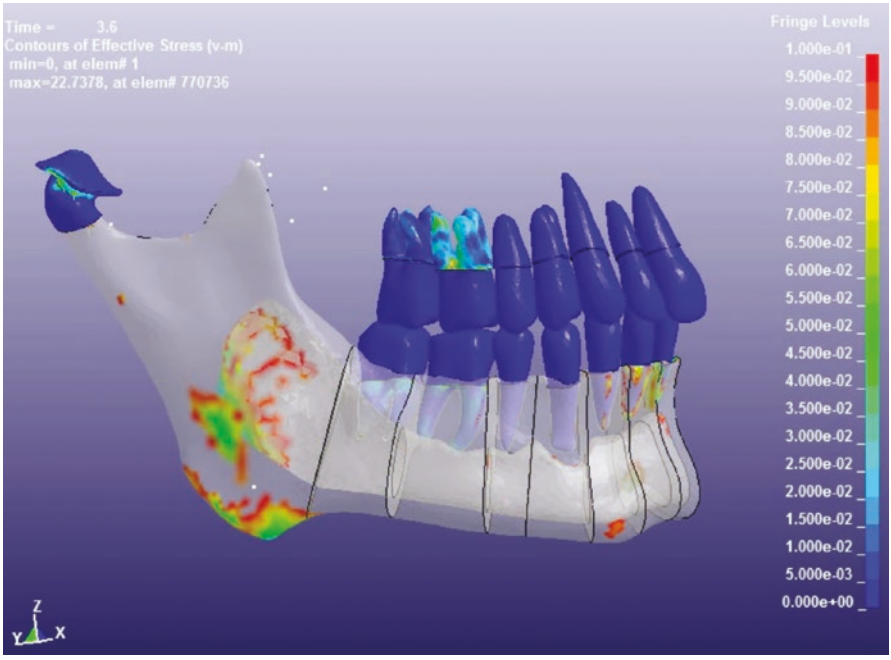

Fig. 1.3 Strain in the mandible, the temporomandibular joint, and the periodontal gap visualized using finite element analysis. FEA was performed within a DFG grant by the Karlsruhe Institute of Technology in cooperation with the working group for experimental biomechanics and oral physiology in the department of prosthodontics, University of Würzburg, Germany

1.4 Summary

The results of the different studies with varying study designs, assessment tools, and predictor variables indicate that there is a positive association between sleep bruxism and TMD, although a definitive conclusion remains elusive. The reasons for this uncertainty are manifold: non-standardized examination protocols, limited number of subjects, insufficient reliability of a bruxism diagnosis, and duration of the assessment of bruxism. PSG continues to be the best available method to diagnose bruxism.

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The Role of Trauma and Whiplash Injury in TMD

2

Sonia Sharma, Richard Ohrbach,
and Birgitta Häggman-Henrikson

Abstract

The effects of direct or indirect trauma to the jaw and temporomandibular joint (TMJ) can have both short- and long-term consequences. This chapter will explore direct and indirect trauma in relation to temporomandibular disorders (TMDs).

- Definition of trauma and role of injury
- Micro- vs. macrotrauma
- Epidemiology of trauma
- Direct trauma and TMDs
- TMD and whiplash injury
- Consequences of trauma: pain and functional disturbances
- Considerations for treatment in relation to TMD and trauma

2.1 Definition of Trauma and Role of Injury

In 2004, an intriguing if not unsettling commentary by Langely et al. identified that there is a substantial problem with defining injury [1]. Most research has relied on the "energy definition" for defining injury, described as "damage to the body

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produced by energy exchanges that have relatively sudden discernable effects." However, the authors noted that what is meant by "damage to the body" is not readily interpretable. Often, injury has been defined as external causes of injury, which is circular. One may assume, however, that researchers using such type of circular definition have implicitly considered some form of tissue damage as a necessary consequence of specific kinds of events. Clearly, there are significant problems regarding the nuanced understanding of "injury," problems that impact profoundly on its scientific investigation.

Many methodological problems exist within injury epidemiology [2]. It is a relatively young field, and consequently data about injury as an epidemiological exposure are sparse. Firstly, as described above, injury has been defined in a variety of ways.

Secondly, approaches used to monitor injuries are diverse, ranging from reports on death certificates for fatal injuries to specific injury surveillance systems (e.g., traumatic brain injury systems) and to registries that attempt to record all injury events. Furthermore, because the spectrum of injuries is quite broad, the sources used to monitor injuries are largely defined by the type of injury, the level of severity, and the legal implication of the injury. Most injuries are relatively minor and can be managed without any professional medical attention and therefore are self-reported, whereas more severe injuries will require medical attention by a professional if not hospitalization, and the most severe injuries can be fatal. These factors affect the available information about injury and its consequences [2].

Thirdly, injuries can be classified in a variety of ways, such as by the anatomy, event, or etiological mechanism, each having its own advantages in ways that public health can be applied to prevent further injuries [3]. However, the most common classification system used by insurance companies and medical experts in identifying injury are the ICD codes, which focus only on the event and body region but lack criteria for injury. Lastly are the varying calculations used for estimating epidemiological estimates of incidence. For example, some studies have adopted a term called "clinical incidence" calculated as the number of injury events divided by the number of athletes at risk (rather than cumulative incidence, calculated as the number of injured athletes divided by the number of athletes at risk), making comparisons within specific injurious event types and rates across studies difficult to compare [4].

The following sections will provide an overview of the epidemiology of injury and elaborate on specific types of nonfatal injuries to the jaw. Studies show that the common sources for jaw injuries include falls, sport injuries, motor vehicle accidents and whiplash trauma, and head and neck injuries, as well as injury from medical and dental procedures.

2.2 Micro- and Macrotrauma

Micro-trauma is often considered as an initiating factor for TMD. Musculoskeletal micro-trauma represents low magnitude forces which can lead to physical damage over time, depending on the intensity and duration in relation to the resistance of

the structures and capacity of the individual [5, 6]. The most common cause of micro-trauma is overuse behaviors; in the orofacial region, para-functional behaviors such as tooth clenching and grinding are considered risk factors for the development of TMD.

It can be difficult to determine whether physical damage is related to micro-trauma. Although the presence of tight bands and myofascial trigger points in painful muscles is widely regarded as an indication of a myofascial pain disorder, studies indicate poor inter-examiner reliability [7], and the presence of this phenomenon has been debated [8, 9]. Given, however, the role that overuse behaviors have as a stated etiology [10] and empirically have an onset and perpetuation of myofascial pain [11–13], perhaps myofascial pain disorders should be regarded more as a construct, comprised of multiple indicators, rather than as a simple physical diagnosis. The role of micro-trauma as a factor in the development of TMD requires more research.

Physical damage might wrongly be assumed to be related to parafunctions, so that what might be a behavioral problem [11, 14] is perceived by the clinician as a physical disorder with associated tissue damage as the source of the pain. This pattern of thinking—the belief that physical damage has occurred and therefore must be the focus of physical intervention—has dominated the TMD field for decades, but it does not consider levels of diagnosis [15]. Micro-trauma remains an important possibility for the TMD pain that has onset without known cause [16]. Thus, micro-trauma has been regarded as an initial cause of TMDs, particularly for disorders affecting the TMJ, but this appears to be largely based on speculation. There is emerging evidence however on the importance of micro-trauma [17, 18] and that women in particular [19] may be more susceptible to the repeated forces of sleep bruxism. These findings may have implications for the understanding of the association between overuse behaviors and TMDs.

2.3 Epidemiology of Trauma

The four leading external causes of medically consulted injury episodes according to the NHIS 2014 data, as events per 1000 population per year, were falls [47], being struck by a person or an object [14], transportation [11], and overexertion [12, 20]. An integrated database of the NHIS from 2004 to 2014 had similarly reported falls as the leading cause of nonfatal injury for all age groups followed by transportation-related injury, being struck by object or person, and “other injures” [21]. Although falls are among many types of injuries that may result in a jaw injury, fall injuries are also unique due to the discreet form of the event and the resulting pathology. A fall is usually reported when it has an impact on function, whereas, for example, a motor vehicle accident (MVA) is usually reported irrespective of a resulting pathology. This form of distinction of the event and the resulting pathology helps with better understanding of the general use of the term “injury.” Across the world fall injuries accounted for 250.0 million prevalent cases of injury and were the 12th leading cause of disability. As mentioned above, the prevalence for fall injuries for

2014 was 47 per 1000 population per year, and as expected the prevalence was highest for individuals over 75 years old at 131 per 1000 population per year [20].

2.3.1 Motor Vehicle and Transport Injuries

Around the world MVA exclusively, not including motorcyclist, cyclist, and pedestrian injuries or other road and transport injuries, constituted 60.7 million prevalent cases in 2013 [22]. Whiplash trauma, often associated with MVA, has a reported incidence in the region of 1 per 1000 inhabitants [23].

2.3.2 Sports Injuries

For the year 2014, the prevalence for self-reported medically consulted sports injury and poisoning episodes in the United States was about 22 per 1000 population. Unfortunately, sports injuries and poisoning episodes as reported by the NHIS cannot be parsed separately. Annualized age-adjusted prevalence for both sports and poisoning episodes was the highest for ages between 12 and 17 years at 83 episodes per 1000 population followed by ages 18–44 years at 22 per 1000 population, while the lowest occurred in those above 45 years at 8 episodes per 1000 population per year. Injuries reported by males at 27 per 1000 population per year were nearly twice than those reported by females at 15 per 1000 population per year. Estimates by education were higher for those with a bachelor's degree or higher at 20 episodes per 1000 population per year and lowest for some college education at 18 per 1000 population [24].

2.3.3 Head and Neck Injuries

According to the Nationwide Emergency Department Sample (NEDS), the authors found that of the more than 131 million emergency department (ED) visits reported in 2011, 4% were attributed to head and neck injuries. The most common mechanisms of head and neck injury were falls (39%) followed by blunt trauma (26%) and motor vehicle traffic (7%). Among the top ten diagnoses based on the ICD-9-CM codes were unspecified head injury (21%); contusion of the face, scalp, or neck (18%); open wound of the forehead (12%) and scalp (10%); open wound of the lips (5%); abrasion of the head (4%); open wound of the jaw (3%); closed nasal bone fracture (2%); unspecified open wound of the face (2%); and foreign body in the ear (2%) [25].

2.3.4 Jaw Injuries

Injuries to the jaw can range from minor laceration of the soft tissue structures to more severe damage such as fractures of the hard tissues. Moreover, jaw injuries can be brought about by a number of different traumatic events. As there is no one single

source that reports on the epidemiology of jaw injuries, this section will describe reports in various publications and in different populations. The Nationwide Emergency Department Sample (NEDS), based on 970 hospitals in 27 participating states, reported in 2007 a total of 407,167 ED visits for facial fractures, with a higher frequency in men (68%). Among the various traumatic events associated with facial fractures, assaults were the highest (37%), followed by falls (25%), motor vehicle accidents (12%), and transport and cyclist accidents (2%) [26]. Besides assaults, falls, and accidents, other forms of injuries that can affect the jaw include head and neck injuries as described in the prior section and traumatic brain injuries. Cassidy et al. found out that among adults who suffered mild traumatic brain injuries after a motor vehicle collision, 26% reported soreness in the jaw, and 40% reported face pain over a period of 2 years after the injury [27].

Iatrogenic forms of injuries such as oral intubation, laryngoscopies, and dental treatments have also been reported to be sources of dental injuries. However, because dental injuries encompass injuries to structures inclusive of the lips, teeth, tongue, etc., it is difficult to parse out from the literature how many injuries affect the jaw bones or muscles specifically. For example, in a pilot study on adults who underwent elective surgery under general anesthesia, more than 80% experienced oral injuries inclusive of injuries to the teeth, lips, tongue, and oral cavity after endotracheal oral intubation [28].

2.4 Direct Trauma and TMDs

Direct trauma (i.e., injury from a direct force to the body) may cause a variety of injuries such as to soft tissues (the TMJ disc, ligaments, muscles, and nerves) as well as bony structures. The most common mandibular fracture is condylar neck fracture which, in addition to pain, may affect both function and growth. Most injuries caused by direct trauma will only cause short-term pain, as part of the normal healing process. The relationship between TMD and other types of direct trauma such as dental and medical procedures, yawning, and sustained jaw opening is described in more detail below.

2.4.1 Dental and Medical Procedures

Studies suggest a positive association between dental treatment and TMD. Akhter et al. studied the association of jaw injury, third molar removal, and orthodontic treatment with TMD in 2374 Japanese university students through self-administered questionnaires. TMJ pain, difficulty in mouth opening, and other more general jaw symptoms were significantly associated with jaw injury. TMJ clicking was associated with extraction of third molars, but no associations were found between TMD symptoms and orthodontic treatments [29]. In a case-control study in 469 adults in Seattle, the odds of facial trauma were twofold among TMD cases compared to those with no TMD [30]. Facial trauma was assessed using a self-report

questionnaire and was defined as been hit, having had a car accident, sports injury, or other accident where one had received a hard blow or bang to the jaw or face. In the same study, an even stronger positive association with TMD was found for third molar removal. For both facial trauma and tooth extraction, their associations with TMD were not altered by further adjustment for race, marital status, income, or education [30]. However, a possible limitation of both these studies is the potential for recall bias, where individuals may have attributed their current pain to a prior injury. Also using a retrospective prospective design, Huang et al. found an association between third molar extraction and first onset of TMD using insurance records. This was a representative sample of individuals living in Washington State with dental insurance, and findings are generalizable only to this select population [31].

In a prospective study that enrolled 2217 individuals in southwest Washington and northwest Oregon, individuals aged 15–20 years who had one or more third molars removed during the 5-year study period had a nonsignificant increased risk of TMD. The authors of this study concluded that though most third molar extractions occur during the teenage years and are complicated due to impaction, the risk of TMD in this age group is only around 1% of the overall population, whereas the attributable risk of TMD is 23%. The results indicated that almost a quarter of all TMD cases in this age group might be related to third molar extraction [32]. However, the investigators for the above study used the International Classification of Disease, 9th edition (ICD-9), and diagnostic codes 524.6 through 524.69 to identify TMD. In a subsequent study that used pain on wide opening or pain in the temple, jaw joint, or jaw muscles as the criterion measures for TMD, Huang et al. conducted a prospective analysis on patients of dentists enrolled in a dental practice-based research network in the Pacific Northwest, from May 2009 through September 2010. Of the 517 participants aged 16–22 years with follow-up data, 201 had third molars removed, and of these 38 developed pain on wide opening and 19 had pain in the temples, jaw joint, or jaw muscles. Compared to those who did not undergo third molar extraction, those who did had a significant 5.2 and 2.2 times higher risks for pain on wide opening and pain in the temples, jaw joint, or jaw muscles, respectively, after adjustment for age and gender [33].

2.4.2 Yawning and Sustained Jaw Opening

Studies on jaw injury due to yawning or prolonged mouth opening are scarce. The OPPERA baseline case-control study assessed jaw injury in 3263 controls and 185 TMD cases using a self-report questionnaire and examined TMD clinically using the RDC/TMD. The above study found that any injury (OR, 4.2) and specifically yawning (OR, 7.3) and prolonged mouth opening (OR, 8.3) were strongly associated with TMD [13] and indicate that the association between injury and TMD can range between OR, 4.2, and OR, 8.3. However, because the above study used a cross-sectional design, TMD cases could have attributed their pain to an injury and may overreport on the questionnaire.

In summary major limitations of these studies were either absence of comparison group (e.g., a group without TMD or a group without injury) [34–40] or the

comparison groups were not clearly defined potentially leading to misclassification and biased measures of association [41, 42]; the extent of the threats to validity is difficult to predict in the absence of validation studies. Some studies used non-validated protocols or a questionnaire to diagnose subsequent onset of TMD which introduces the potential for misclassification [30, 39, 40, 42–46]. Some studies either did not adjust for confounders resulting in distorted associations or did not adequately adjust for confounders resulting in residual confounding.

2.5 TMD and Whiplash injury

In 1928 Harold Crowe first coined the term "whiplash" [47]. Whiplash trauma is described as an acceleration-deceleration mechanism of energy transfer to the neck, which manifests overwhelmingly as soft tissue injuries. Although most individuals will recover, about one in three will develop long-term symptoms [48]. "Whiplash-associated disorder" (WAD), a term given by the Québec Task Force, refers to clinical manifestations of whiplash [49] within a classification system: grades I to III refer to neck pain with no physical signs, with musculoskeletal signs, and with neurological signs, respectively, and grade IV refers to fractures. Though the soft tissue injury of a WAD is typically referred to as cervical sprain or strain, the exact physiologic mechanism is not known, and there may or may not be damage to soft tissue, joints, ligaments, and muscles in the neck, posterior shoulder, and upper thoracic regions. Although MVA is the most common source of WAD, the disorder can also occur as a result of falls or other mishaps [50]. Common symptoms following whiplash trauma include neck pain, impaired cervical mobility, and headache, but symptoms are heterogeneous and relate to the mechanical injury to the neck, to psychological and behavioral factors, and to pain sensitization. For example, when individuals are observed soon after a reported whiplash injury, significantly higher depression symptoms and both pain and non-pain whole body symptoms are also reported, underlining the importance of psychosocial factors following trauma and complicating the purported relationship between the trauma and the primary subsequent symptom of pain; see Fig. 2.1.

A functional connection between the jaw and the neck regions is suggested by their direct anatomical and biomechanical relationships. The location of the trigeminal nucleus, which penetrates into the upper cervical segments, reflects a linkage in sensorimotor input between the jaw and neck regions. A wide range of head-neck movements are influenced or initiated by input from orofacial structures, indicating that movement of the head is an integral part of normal oral function. It has been suggested that a local injury to the TMJ due to "mandibular whiplash" might occur concurrent with cervical trauma [51], but this proposal has later been refuted by prospective studies showing that the high prevalence of TMD pain after whiplash trauma was not associated with any structural changes in the TMJ [52, 53]. Instead, these findings provide support for a neurobiological explanation suggesting that a whiplash trauma can indirectly affect the jaw system and contribute to TMD; purported mechanisms include functional integration, spread of pain between the neck and jaw regions, and widespread pain caused by sensitization mechanisms.

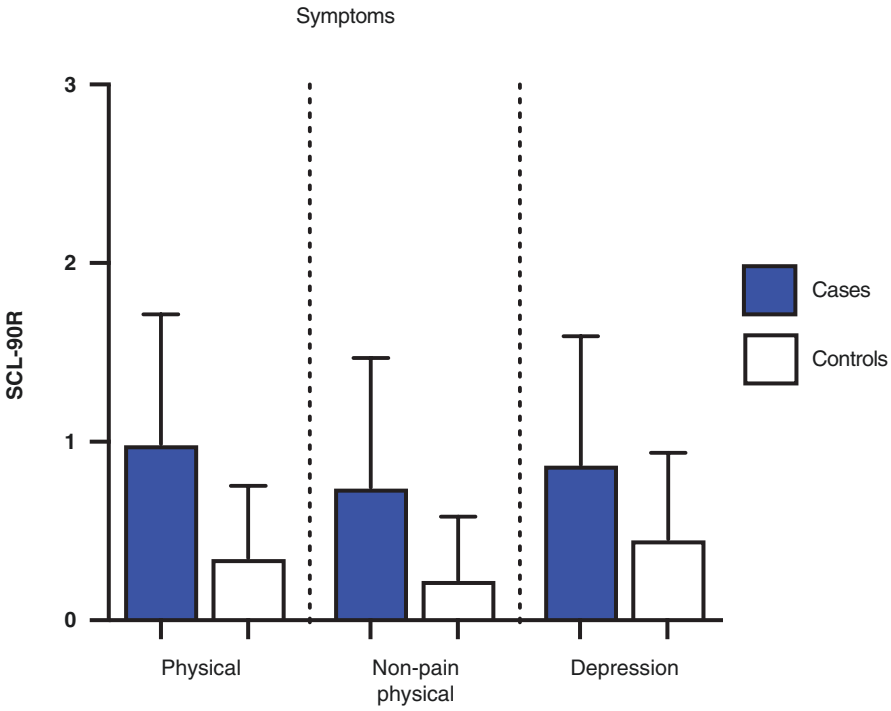

Fig. 2.1 Differences between scores on the subscales physical symptoms, non-pain physical symptoms, and depression as measured by the Symptoms Checklist-90R (SCL-90R). Data were collected from individuals with a recent (i.e., within the last month) whiplash trauma (cases) and from individuals without a history of whiplash trauma (controls). The control group was matched for age and gender and was recruited by advertising, with the only exclusion criteria being history of head-neck trauma. Post hoc testing indicates that all group comparisons are significant, $p < 0.0001$. The results were presented at the 15th World Congress of Pain, International Association for the Study of Pain (IASP); https://event.crowdcompass.com/wcp2016/activity/tmp42LWT6l

2.5.1 Development of TMD After Whiplash Trauma

Although it has been reported that the incidence of jaw-face pain and dysfunction following acute whiplash injury is low [38, 54], chronic WAD patients report pain and dysfunction also in the jaw-face regions [55]. Most epidemiologic studies that have investigated the association between injury and TMD have focused on injuries attributed to MVA and whiplash, and the majority of these studies were of cross-sectional design on patients with chronic WAD [41, 51, 56–58]. In a cross-sectional study comparing individuals in the acute stage within 1 month after whiplash trauma with controls without a history of trauma, individuals with whiplash trauma reported more jaw pain and dysfunction [59] and reduced chewing capacity [60]. Furthermore, the reported correlation between the intensity of jaw and neck pain may imply that jaw pain in the acute stage after whiplash trauma is related to referral or spread of pain from the neck rather than originating locally in the TMJ (Fig. 2.2).

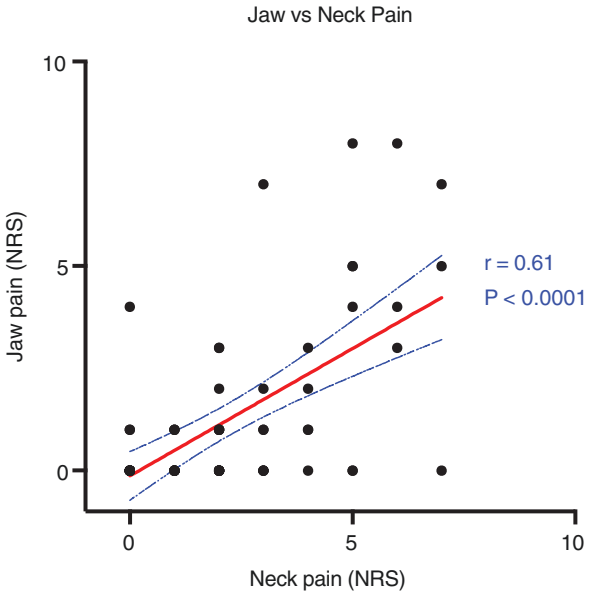

Fig. 2.2 Scatterplot between jaw and neck pain intensity (numerical rating scale [NRS]) for individuals with a recent neck trauma ($n = 70$), showing a moderate positive correlation between jaw and neck pain but with large inter-individual variability in pain ratings. Adapted from Häggman-Henrikson et al. (2016) [59]

Retrospective studies have assessed the association and temporality between WAD after a collision and TMD. Among 7763 Canadian adults who filed collision-related personal injury claims, and were followed up prospectively for pain symptoms at 4, 8, and 12 months, 636 were identified with WAD. The incidence of reduced and painful jaw movement was higher (RR, 3.36, 95% CI, 2.36–4.78) in subjects with WADs (15.8%) than in those without WADs (4.7%); and the onset of reduced and painful jaw movement was associated with female sex and age below 50 years. Major limitations of this study include not having adjusted for potential confounders and the use of self-reported questionnaire to assess TMD [61].

There are few prospective studies where temporality may be more directly assessed and incidence of TMD calculated [38, 42, 52]. In general, these studies have also found a positive association between WAD injury and TMD risk. In a prospective study that enrolled 60 patients from a hospital emergency department that were involved in rear-end car collisions with WAD grades I to III found approximately sevenfold increase risk of TMD after 1 year when compared to age- and sex-matched nonexposed individuals from the same geographic region [52].

2.6 Consequences of Trauma: Pain and Functional Disturbances

The consequences of trauma can be related to many different factors, including the type of injury, the structures involved, the context of the trauma, as well as psychosocial factors that may affect the healing process. In line with this, the consequences can range from functional disturbances related to damage to the

TMJ or other hard or soft tissue, to acute pain as part of the healing process, or to the development of chronic pain conditions.

2.6.1 The Role of Pain as a Primary Symptom of Injury

One of the most immediate and vital physiological responses of the body to tissue damage or potential tissue damage is pain. The International Association for the Study of Pain (IASP) has defined pain as "An unpleasant sensory and emotional experience *associated with actual or potential tissue damage, or described in terms of such damage" (emphasis added).* Injury is not only sensory, however, but it can also elicit an emotional response by disrupting the brain's homeostatic regulatory system through activation of the neural, hormonal, and behavioral systems, thereby producing stress. Furthermore, the extent and severity of injury will govern which mechanisms are activated through a genetically predetermined system [62].

2.6.2 Nociception and Pain

Nociception, defined as "the neural process of encoding noxious stimuli," is the response of peripheral receptors to stimuli that are harmful or potentially harmful to the body. Nociception encompasses the peripheral nerve from receptor to first synapse in the spinal cord. In contrast, pain is a process that occurs from the first synapse in the spinal cord up into the brain [63]. Whether nociception is perceived as pain, which is a conscious experience, depends on many factors [64], which is further explained subsequently. In general nociception is an evolutionary, protective mechanism that warns of a harmful or potentially harmful stimulus regardless of the particular response it engenders. If ignored, nociceptive responses to harmful stimuli can set up a cascade of more complex neurobiological pain mechanisms that may project into a pain disorder. Based on the underlying neurobiological mechanisms that are generated by diverse etiologic factors, pain can be divided into four primary types: nociceptive, inflammatory, neuropathic, and functional [65].

Nociceptive pain is the most basic and vital physiologic response of the body that warns of a potentially damaging threat to the body through the activation of nociceptors [63]. Nociceptors are mostly comprised of high-threshold sensory receptors located within the peripheral somatosensory nervous system which are capable of transducing a noxious stimulus such as mechanical or chemical [65]. Inflammatory pain, in contrast, is part of a healing response of the body that occurs once tissue damage has occurred. Inflammatory pain marks a shift in the body's response from protection, when a noxious, potentially damaging stimulus results in nociception, to a restorative response, when damage has occurred, and the body's response is to promote healing. Both nociceptive pain and inflammatory pain are adaptive responses of the body, whereby they contribute to survival by protecting and healing [65].

Two other types of pain represent abnormal functioning of the pain system. Neuropathic pain is defined as "pain caused by a lesion or disease of the

somatosensory nervous system." Based on whether the lesion or disease lies in the periphery as in after a dental procedure or in the central somatosensory nervous system as in multiple sclerosis or stroke, neuropathic pain can be divided into peripheral or central. Some characteristics of this type of pain are allodynia, hyperalgesia, or hyperpathia [63, 66]. Functional pain is a form of pain where no physical evidence of abnormality can be detected simultaneous with abnormal responsiveness or function of the nervous system resulting in increased sensitivity of the pain system, such that symptoms are amplified. Common conditions which include fibromyalgia, irritable bowel syndrome [65], and also TMD have been suggested to be a functional pain disorder [67, 68].

Although the different pain types can be separated based on their underlying mechanisms, they share some common characteristics such as they can also be spontaneous, that is, due to the increased sensitivity of the nervous system during inflammation or neuropathic or functional pain, they could arise in the absence of any identified peripheral stimulus, or they could arise from non-noxious stimuli (allodynia) or may have exaggerated or prolonged responses from noxious stimuli (hyperalgesia) [65].

2.6.3 Mechanisms Linking Injury to a Chronic Pain Condition

Inflammation is a complex, protective biological response to harmful stimuli, such as injury, which involves the blood vessels, mediators such as amino acids, and immune cells such as neutrophils that eliminate the pathogens and tissue damage that is associated with the injury. A number of biological pathways from injury leading to TMD pain have been identified in the literature. One such pathway is through inflammation, which includes the synthesis and release of inflammatory mediators as part of the innate immune response to tissue damage. Furthermore, some of the mediators involved in the inflammatory process are also involved in sensitizing the nociceptive terminals. This sensitization, termed peripheral sensitization, includes an increase in responsiveness of nociception and decrease in the threshold of the high threshold and salient nociceptors in the periphery [65]. In addition, injury also induces a local electrophysiological response. This leads to the recruitment of new nociceptors at the injury site, which enhances pain perception. The resulting increase in pain levels, in turn, helps protect the injured tissues from repeated injury [69].

Central sensitization is a condition of the nervous system that is associated with the development and maintenance of chronic pain. When central sensitization occurs, the nervous system exhibits a process called "windup," which refers to decreasing threshold in response to a repeating stimulus. The net effect is a form of pain amplification associated with upregulation of the central nervous system in response to recurrent stimulation. This persistent, or upregulated, state of reactivity subsequently maintains pain even after the initial injury might have healed. Central sensitization involves a heightened sensitivity to the sensation of touch or nociception, manifesting as allodynia and hyperalgesia,

respectively. Allodynia occurs when pain is evoked from non-harmful stimuli (e.g., touch), and hyperalgesia occurs when a harmful stimulus evokes a painful response that is greater than it normally should be [65].

Psychosocial factors, such as the stress-response to life events, also play a role in the development of central sensitization and chronic pain conditions. Studies have shown a relationship between stress and lowering of pain thresholds [70] and between pre-existing anxiety conditions, catastrophizing, coping, self-efficacy, rumination about pain, and higher pain sensitivities [71, 72]. In addition, previous trauma and existing pain conditions are general risk factors for the development of chronic pain. Taken together, these studies indicate that the pre-existing state of the nervous system and psychosocial factors are important determinants for the development of chronic pain following the onset of acute pain.

2.7 Models of Injury Underlying Onset of Pain Disorders

Wall's injury to pain model proposed three stages of the body's response to injury: immediate, acute, and chronic. Each stage can last as short as seconds to as long as days and represent a disruption in the brain's homeostatic regulatory systems by activating the sensory, neural, and hormonal systems as well as behavioral activity whose goal is to reinstate homeostasis [73]. The first immediate stage after injury can actually be painless, which typically occurs in situations such as fighting, escaping, and obtaining aid which are of the highest importance for survival, for example, in soldiers during war or in individuals after a car accident.

Once there is safety from the source of injury, the next acute stage is characterized by recognition of tissue damage, anxiety, and pain directed at assuring self-protection and initiating treatment to minimize future consequences of the damage. Once safety and initial treatment are achieved, a quiet phase of inactivity is sought for optimum healing and recovery from the injury. However, if this third stage characterized by pain and a depressive state persists beyond the period for recovery, the process of post-injury tissue healing is transformed into a disorder. This is classifiable as an acute pain disorder characterized by functional and behavioral changes which may further progress into a chronic condition [73].

2.7.1 The Homeostasis Model

This model is based on the fact that injury does not merely produce pain [74]. Stress, which is the brain's homeostatic regulator system response to reinstate recovery in the event of a stressor such as injury, is a vital part in the process of chronic pain [62]. This perception of threat, such as to an injury, activates the sympathetic nervous system, which allows the organism to "fight" or "flee" from a threat, accompanied by the release of cortisol in order to mobilize glucose reserves that will replenish the energy expended in the initial response. However, prolonged activation of the stress

regulation system will generate breakdowns of the muscle, bone, and neural tissue and can produce a vicious cycle of pain-stress-reactivity [75].

Both Craig's homeostasis model and Wall's model on injury propose that stress may enhance immediate or delayed pain after injury supporting the link between neurobiological stress systems and variation in pain outcomes after trauma.

2.7.2 The Fear-Avoidance Model

This model describes a common behavioral consequence of pain where the manner in which the pain is interpreted can lead to two contrasting behaviors. Fear and anxiety of pain, which is helpful in learning and preventing one from doing potentially harmful activities, in excess of the actual risk of harm or injury can become dysfunctional. If the pain is interpreted as non-threatening, there is continuation of healing-stage appropriate daily activities leading to recovery [76]. If, in contrast, the pain is interpreted as threatening, then dysfunctional pain-related fear emerges which is associated with safety-seeking behaviors such as avoidance/escape and hypervigilance. The long-term consequences, such as disability and disuse, in turn may lower the threshold at which subsequent pain will be experienced [76]. The experimental and observational support for the fear-avoidance model is extensive, even though there is an ongoing debate regarding the precise meaning of the term fear which can often be interchangeably used with anxiety [77].

Collectively, the proceeding information leads to the following expansion from Wall's original model [73]. As stated above, when the phase of recovery of the damaged tissue, characterized by depression and pain symptoms, persists beyond the necessary period for recovery, the condition may develop into a pain disorder and may with time become chronic (Fig. 2.3).

Pain persisting for some time elicits distress. The work by Vlaeyen suggests that initial distress, expected to occur in the presence of persistent pain, is itself facilitated by pre-established cognitive structures that include somatic vigilance, symptom amplification, and catastrophizing. Fundamental beliefs about the meaning of pain transform the brain's capacity to modulate ongoing nociception and that as

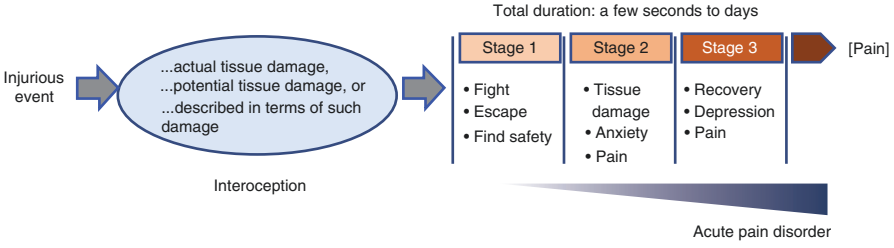

Fig. 2.3 On the nature of injury. The three stages in development of acute pain disorder, Adapted from Wall PD, On the relation of injury to pain. Pain, 1979

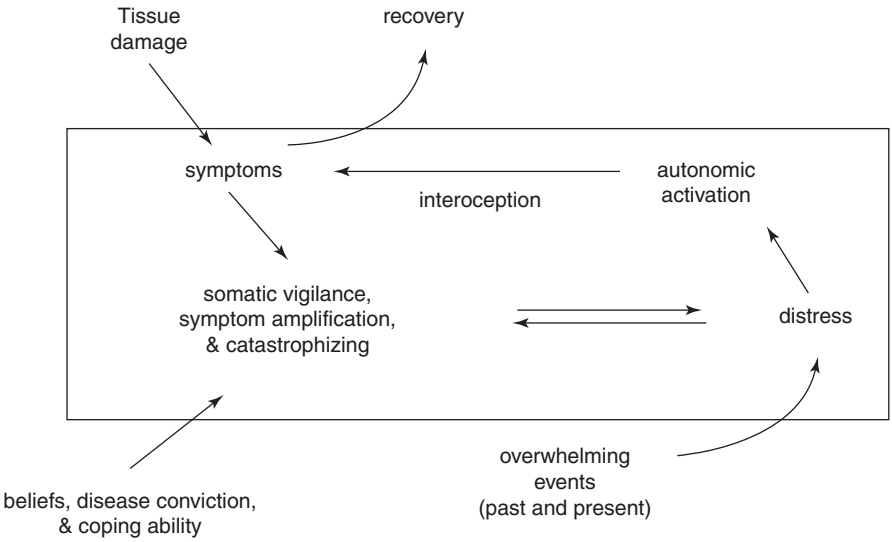

Fig. 2.4 Physical and psychological processes directly associated with functional disorders are shown inside the box. Outside the box are exogenous processes that represent life events, tissue damage, and trait characteristics in the form of beliefs and coping style which is illustrated as existing separate from the functional disorder. Modified from Sullivan and Katon (1993) [78]

pain continues, both physical and, later, mental deconditioning occur, ultimately leading to illness in all of its aspects [76]. Figure 2.4 provides an overview of the currently understood processes underlying the progression into chronic pain.

2.7.3 History of Trauma in Patients with TMD

Prior epidemiological studies have suggested an association between injury and TMD pain. For example, data from the North Finland Birth Cohort study indicated a positive association between facial trauma and facial pain; the sample was a group of 5696 individuals born in the year 1966 in Finland who were recruited by mail and queried using a questionnaire on facial pain, other symptoms, and traumas to the face and body [79]. The unadjusted odds ratios for the association between facial pain and facial trauma were 2.1 for men and 2.3 for women and between facial pain and TMJ trauma were 3.2 for men and 4.4 for women [79]. A systematic review found that one in three patients with chronic TMD have a history of whiplash trauma to the head-neck [80]; these studies, however, were case-series or case-control studies in various types of clinical settings. Individuals with chronic TMD pain and with a prior history of regional trauma report more severe and frequent symptoms and signs such as headaches, facial pain, severe jaw dysfunction, and sleep disturbances [34, 44, 56, 81, 82].These patients also respond less well in reaction time tests and

overall tire more easily [81]. Since many of these symptoms are associated also with closed head injuries [83], centrally mediated changes may account for the uniqueness of jaw-face pain and dysfunction in patients with a history of head-neck trauma. It has also been suggested that the prognosis for recovery is lower for this patient group [44, 82, 84].

2.8 Considerations for Treatment in Relation to TMD and Trauma

TMD is the most common reason for chronic pain in the orofacial area. As the focus of this chapter is the role of trauma in TMD, it does not cover acute management of soft tissue injury and fractures and the associated healing in the acute stage. As described in previous sections, the transition from injury into an acute or chronic pain condition is complex and related to many different factors.

The treatment principles for TMD in relation to trauma should follow the current general guidelines for TMD treatment, taking into account the contributing factors for the individual patient and favoring basic self-management skills as the first part of treatment:

- Disease information with the goal to increase understanding of pain mechanisms so that the patient has realistic expectations regarding what can be achieved with regard to treatment of TMD related to trauma.
- Self-management consisting of jaw relaxation and jaw exercises to reduce muscle tension, increase blood flow, and reduce pain.
- Stabilization splint for use during sleep when sleep bruxism is present, with the goal to reduce the associated muscle pain.
- These treatments should be combined with psychological assessment, cognitive behavioral therapy, and physiotherapy as needed.

2.9 Conclusions

As described in this chapter, the literature on injury is fraught with challenges and complexities associated with case definitions and ascertainment of injuries, both of which substantially impact on reliable interpretations of estimates from currently available studies. Overall, the literature relies almost exclusively on the self-report from the people surveyed about prior injury. While these reports are not accompanied by any objective evidence of tissue damage, the role of self-reported injury ranging from presumably objectively observable injury to non-observable injury should be considered of equal importance. However, attribution bias must be simultaneously considered when pain has become chronic. In contrast, literature relying on reports from emergency departments is usually based on ICD codes requiring diagnosis from medical professionals; yet, such codes do not assure the presence of observable injury. Therefore, analysis in the injury literature has largely been based

on self-report, but this should not be construed in a negative light; rather, self-report can be very reliable, and, in the case of pain, self-report is all that exists. Further research should examine the relationship of such self-report to interoceptive cues and measures of tissue damage, both overt and non-observable.

As described above, jaw injuries can be caused by a wide variety of events that impact in different ways. Because there is no single literature source that reports on the different types of injuries, injury of different types has been reported by different literatures with injury operationalized in different ways, which then leads to no direct comparisons of epidemiological estimates among the different types of injuries. The relative significance of one form of injury compared to another, for purposes here of causing damage to the jaw, remains unknown.

The above-stated complexities lead to further issues when describing associations of injuries with pain conditions. The challenge lies in how to model injury; for example, should association studies consider the event as the exposure or overt tissue damage as the exposure, or should tissue damage be considered implicit and be considered as the pathway from event to, for example, a pain disorder? In summary, these complexities beg for more reliable and valid definitions of the spectrum of injury.

Finally, with regard to the role of trauma in TMDs, the clinician should aim for facilitating the normal healing process in the acute stage, as this will be sufficient for most patients. For those patients whose acute pain following a trauma transitions into a chronic pain condition, the multiple risk determinants associated with TMD onset and transition to chronic should be considered and the treatment tailored accordingly rather than assuming that only the history of trauma is relevant to the chronic condition.

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Part II

Medical Treatment

Medical Management of TMD

3

Rebeka G. Silva, Valeria Gerloni, and S. Thaddeus Connelly

Abstract

Understanding how to use medical management to address temporomandibular disorders pain is an essential component of the provider's armamentarium. Patients who benefit from medical management include those with a wide variety of acute and chronic diagnoses, encompassing intra-articular and extra-articular disorders, as well as the postsurgical patient. This chapter covers selected medication categories and minimally invasive interventions that go beyond the first-line advice of soft diet and warm compresses.

3.1 Introduction

The TMJ surgeon often wishes that he or she can select the proper operation for any given patient, and that the dysfunction or condition will be cured, because that is how we think and it is what we do best. All surgeons should be comfortable treating immediate postoperative pain. But what about the patient for whom the diagnosis is unsure, or for whom surgery is not indicated or not yet decided upon by either patient or surgeon? What about the individual who requires a lengthy postoperative follow-up that may span months or years? Medical management has several objectives: one is to control pain and to improve function. The second objective is to

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affect the pathophysiologic process, by either halting it or reversing it. In some cases, medical management might be used as a diagnostic tool to rule in or rule out possible diagnoses. This chapter section will serve as a primer for the surgeon and other dental or medical providers on medical management and nonsurgical therapeutic interventions that may be considered for patients with TMD, in concert with a possible referral to a physical therapist or chronic pain specialist. These in-office treatment modalities are efficacious for a wide variety of diagnoses and can have a large impact on the patient's quality of life.

As often occurs, new patients may present to the clinician complaining of "TMJ" and it is up to the clinician to sort out where the primary problem lies. One way to begin is to understand that many patients fall neatly into one of three groups. The predominant clinical picture will either be:

- Internal derangement
- Degenerative joint disease
- Myalgia/myospasm

The clinician must also realize that many patients, perhaps most, have a combination syndrome on initial presentation and the challenge will be to figure out whether joint or muscle is the largest contributor to the pain and dysfunction scenario. Our advice is to determine which etiology predominates and begin with a treatment algorithm geared toward that causation.

The classic elements of conservative nonsurgical treatment for TMD taught in dental school are soft diet, moist heat to the jaw muscles, and NSAIDs, with the possible addition of muscle relaxants and behavior modification (i.e., stress reduction, improvement of sleep, exercise). Inadequate relief or outright treatment failure will often lead to a medication prescription in one or more drug categories or to a minimally invasive intervention. The treatment elements covered in this chapter section include:

- NSAIDs
- Opioids
- Tumor necrosis factor-α inhibitors
- Muscle relaxants
- Tricyclic antidepressants
- Topical medications
- Dietary supplements
- Trigger point injections
- Intra-articular injections
- Botulinum toxin

3.2 Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

NSAID therapy is the backbone of TMD conservative management and is a first-line drug class for many clinicians. The use of NSAIDs for pain management compares very favorably with other traditional TMD nonsurgical interventions, such as

splint therapy. For example, 1 week of diclofenac can yield significant improvements in TMD symptoms in patients with TMJ osteoarthritis, compared to 1 month of splint therapy [1]. As a result, for patients who have no contraindication and who can tolerate a daily NSAID regimen, the clinician is well advised to incorporate NSAIDs in the treatment algorithm for TMD patients.

3.2.1 Method of Action and Adverse Effects

A joint that is exposed to excessive mechanical stress shows upregulation of proinflammatory cytokines and matrix metalloproteinases (MMPs) in chondrocytes, which can lead to osteoarthritis. NSAIDs inhibit cyclooxygenase (COX) enzymes involved in the synthesis of prostaglandins and other mediators of inflammation. There are two different types of COX enzymes that are targeted for inhibition by different classes of NSAIDs. The COX-1 enzyme, which is expressed in the gastrointestinal (GI) tract, kidney, platelets, and blood vessels, is inhibited by COX-1 inhibitors, the classic and most familiar of which is ibuprofen. The COX-2 enzyme, which is induced under inflammatory conditions and is found in macrophages, leukocytes, and fibroblasts, is inhibited by both the COX-1 and selective COX-2 inhibitors. Thus, COX-1 inhibitors are considered nonselective and have a greater risk for GI adverse effects than COX-2 inhibitors, because prostaglandins play a protective role in maintaining gastric mucosal integrity. Adverse events include bleeding and ulceration, and this drug class should be used with caution in elderly patients and those with a prior history of peptic ulcer disease and/or GI bleeding. NSAID use may also result in an increased risk of serious cardiovascular (CV) thrombotic events, myocardial infarction, and stroke. The risk escalates with length of use, and patients with pre-existing CV disease or risk factors for CV disease may be at greater risk. NSAID use can be associated with dose-dependent hypertension in many normotensive and hypertensive individuals because of decreased renal function due to the inhibition of COX-2 in the kidneys, which causes water and sodium retention [2–4].

Nonselective NSAIDs, which inhibit both COX-1 and COX-2 enzymes, are proven to attenuate inflammation and reduce hyperalgesia in the TMJ [5], and selective COX-2 inhibitors are also beneficial in cartilage injury induced by cytokines and high-magnitude cyclic mechanical strain [6]. Kawashima showed that COX-1 and COX-2 NSAIDs reduce the expression of inflammatory factors in fibroblast-like synoviocytes derived from the TMJ [7]. However, Ta showed that naproxen, a nonselective COX inhibitor, was significantly more efficacious in treating TMD pain from internal derangement than celecoxib, a selective COX-2 inhibitor. Unfortunately, the benefit came at the price of 40% higher incidence of GI symptoms in the naproxen group compared to the celecoxib or placebo groups [8].

On the other hand, one must consider that NSAIDs have been associated with increased cardiovascular risk, and in the scientific literature, naproxen still has the best cardiovascular safety profile. A meta-analysis by Trelle et al. revealed that rofecoxib had the greatest risk of cardiac infarction, while naproxen showed the lowest cardiovascular risk [9]. A more recent review of the literature by Olmo et al. confirmed that diclofenac and cyclooxygenase-2 inhibitors (especially rofecoxib) are associated with an increased cardiovascular risk, and naproxen has the smallest risk [10].

3.2.2 Effectiveness

For any given population, the efficacy of the different NSAIDs at equipotent doses may be quite similar; however, individual patients often differ in their individual response to the various NSAIDs on the market. The reasons for this are incompletely understood and may include various factors such as expression of adverse reactions, relative COX-1 versus COX-2 inhibition, presence or absence of comorbid conditions, and genetic variations in absorption, distribution, and metabolism [11–14]. It is, therefore, difficult to identify the safest or best NSAID for an individual patient. Ibuprofen is considered by many to be quite safe, but increasing the dose of ibuprofen, or any other NSAID, is associated with an increased risk of an adverse event.

3.2.3 Dosing Guidelines

For patients in the early phase of treatment, NSAIDs are frequently recommended for an around-the-clock dosing regimen because this improves the anti-inflammatory effect. NSAID dose and frequency should be adjusted to the lowest effective dose for the shortest duration possible to suit the individual patient's treatment goals. At higher doses, the clinician should ascertain that an increased clinical benefit is observed to outweigh the potential for increased risk of adverse events. Studies looking at patients taking blood pressure medications show that adherence to the drug regimen is enhanced when medications are dosed less frequently [15]. However, patients being treated for pain may be more likely to comply with nearly any dose schedule that results in pain relief. Nevertheless, the clinician may wish to take dosing frequency into account when prescribing an NSAID regimen. Patients with an inflammatory condition who have a poor response to one NSAID should be assessed to determine whether an adequate anti-inflammatory dose was used. It is not unreasonable to switch patients to a different NSAID if adequate pain relief is not achieved; however, this has not been well studied in a prospective randomized clinical trial. One rule of thumb is to allow for at least 2 weeks of treatment before switching drugs. Bingham found that the response after 2 weeks was predictive of the more long-term response seen at 12 weeks [16].

Ibuprofen: nonselective COX inhibitor
Osteoarthritis and rheumatoid arthritis 300–800 mg PO tid-qid Maximum dose: 3200 mg/day
Mild-moderate pain 400 mg PO q4–6 h prn Maximum dose: 2400 mg/day
Anti-inflammatory use 600 mg PO qid for 7–14 days, or 800 mg PO tid for 7–14 days Maximum dose: 2400 mg/day

Naprosyn: nonselective COX inhibitor
Osteoarthritis and rheumatoid arthritis 250–500 mg PO q12h Maximum dose: 1500 mg/day
Mild-moderate pain and anti-inflammatory use 250–500 mg PO q12h Maximum dose for acute pain: 1250 mg/day Maximum dose for maintenance: 1000 mg/day
Note: 500 mg naproxen is equivalent to 550 mg naproxen sodium
Indomethacin: nonselective COX inhibitor
Osteoarthritis and rheumatoid arthritis Indomethacin: 25 mg PO bid-tid Increase daily dose by 25–50 mg q7 days, especially during acute flares. For persistent night pain or morning stiffness, take a larger portion of total daily dose (up to 100 mg) at bedtime Maximum dose: 200 mg/day Indomethacin extended-release: 75 mg extended-release PO qd Maximum dose: 150 mg/day
Mild-moderate pain Indomethacin: 25–50 mg PO tid prn Maximum dose: 200 mg/day
Diclofenac: nonselective COX inhibitor
Osteoarthritis Diclofenac capsule: 35 mg PO tid Maximum dose: 100 mg/day Diclofenac potassium tablet: 50 mg PO bid-tid, or 75 mg PO bid Maximum dose: 150 mg/day Diclofenac sodium delayed-release: 50 mg PO bid-tid, or 75 mg PO bid Maximum dose: 150 mg/day Diclofenac sodium extended-release: 100 mg PO qd Maximum dose: 100 mg/day
Rheumatoid arthritis Diclofenac potassium tablet: 50 mg PO tid-qid or 75 mg PO bid Maximum dose: 200 mg/day Diclofenac sodium delayed-release: 50 mg PO tid-qid or 75 mg PO bid Maximum dose: 200 mg/day Diclofenac sodium extended-release: 100 mg PO qd-bid Maximum dose: 200 mg/day
Mild-moderate pain Diclofenac capsule: 18–35 mg PO tid Maximum dose: 100 mg/day Diclofenac potassium tablet: 25–50 mg PO tid Maximum dose: 200 mg on first day, then 150 mg/day
Celecoxib: selective COX-2 inhibitor
Osteoarthritis 200 mg PO qd, or 100 mg PO bid
Rheumatoid arthritis 100–200 mg PO bid
Acute pain 200 mg PO bid May start at 400 mg PO × 1 and give additional 200 mg on day 1 if needed
Note: Start at 50% dose if patient is a poor CYP2C9 metabolizer

3.2.4 Aspirin: A Nonselective COX Inhibitor

Unlike NSAIDs, which reversibly curbs platelet aggregation through inhibition of the COX-1 enzyme within the platelets, aspirin irreversibly inhibits platelet COX-1. As a result, aspirin is invaluable in lowering the risk of thrombotic cardiovascular events. For patients who need a typical NSAID, this attribute may be important enough to warrant its concurrent use on an occasional short-term basis. If concurrent use is necessary, aspirin should be taken at least 2 h before the NSAID. It should also be noted that while COX-2 inhibitors are known to spare the GI mucosal generation of prostaglandins, this effect can be offset by the use of low-dose aspirin if it is taken together. Strategies to prevent aspirin and non-aspirin NSAID gastropathy include concomitant administration of a prostaglandin analogue such as misoprostol, reduction of gastric acid production through the use of histamine H2-receptor antagonists or proton-pump inhibitors (PPI), and *H. pylori* eradication.

Aspirin: nonselective COX inhibitor
Osteoarthritis and rheumatoid arthritis
Start with 3 mg/day PO divided q4–6 h and adjust down
Mild-moderate pain
325–650 mg PO q4–6 h prn
Maximum dose: 4 g/day

3.3 Opioids

It is often tempting to prescribe opioids for acute TMJ pain. Opioids are non-ulcerogenic and may be added to NSAIDs and acetaminophen for increased benefit.

Opioids induce analgesia by activating opioid receptors on nociceptive primary afferent neurons, the nerves involved in the initial perception of pain. In the head, throat, and upper neck, when a pain stimulus (either thermal, chemical, or mechanical) crosses the pain threshold of these specialized nerves, depolarization triggers an action potential. Pain impulses then propagate along the nerve axon, following trigeminal (V) nerve branches, and into the medulla where the nociceptive primary afferent nerve synapses with a second-order neuron. The pain signal travels along the second-order neuron to the thalamus, where the signal is distributed to different parts of the brain. Peripheral nociceptive nerve endings are activated by many different chemical mediators that are released by macrophages, mast cells, and injured cells. The peripheral nociceptive nerve endings also possess inhibitory receptors, and it is here that opioids exert their positive effect on pain [17].

All the commonly prescribed narcotics have been used to mitigate TMD pain. Frequent side effects that the provider will be called upon to manage include dry mouth and constipation. However, patients with acute pain do

benefit from short-term narcotics for break through pain, or if there are contraindications to prescribing NSAIDs. Patients should be selected carefully, and the provider is advised to limit the initial prescription to a 7–14-day course. When several weeks of narcotics are needed in combination with active TMD therapy, the dose should be frequently assessed to see if it could be titrated down. Narcotics to control long-term TMD pain should be avoided where possible due to the obvious risk of escalating tolerance, abuse, addiction, and diversion. However, if chronic pain management is required, the provider should seek assistance from a pain management specialist. Frequently, opioid contracts are established with the patient at this point, to create a good understanding of everyone's expectations and responsibilities. Switching immediate-release opioids to a long-acting or sustained-release formulation of morphine or oxycodone can mitigate severe breakthrough pain and withdrawal symptoms. Establishing a chronic narcotic regimen as part of TMD treatment assumes that the TMD workup is as complete as possible and that the diagnosis is firmly established. Tumors, severe rheumatoid arthritis with effusion, and other arthritidies are associated with significant pain and can be missed without the proper diagnostic testing.

For selected patients with intracapsular pain, opioids may be injected intra-articularly with good pain relief. Sipahi et al. showed that arthrocentesis followed by either morphine or tramadol injection into the joint space resulted in sustained and significant reduction in pain in a double-blind, placebo-controlled study of patients with internal derangement that had poor response to conservative treatment [18]. These results have also been borne out by other investigators [19–21]. The use of opioids intra-articularly avoids the systemic side effects caused by oral or parenteral administration, and intuitively, maximum benefit is achieved because the drug is administered directly on the mu opioid receptors present in the inflamed intra-articular tissues. Both morphine and tramadol have excellent analgesic efficacy. Morphine works in the classic way, by binding to the opioid receptors of the primary afferent neurons as previously described, which inhibit the activation of the pain signal to the CNS. Tramadol combines opioid receptor activity with reuptake inhibition of serotonin and norepinephrine, thus raising the brain levels of these compounds and contributing to an antidepressant efficacy that is not found in traditional opioids [22]. Tramadol is also successful as an oral agent for patients with osteoarthritis and postoral surgery pain, especially when combined with acetaminophen (Ultracet) [23, 24].

Tramadol (37.5 mg) and acetaminophen (325 mg)
Moderate to severe pain Two tablets PO q4–6 h prn Maximum dose: Eight tablets/day (300 mg tramadol and 2600 mg acetaminophen)
Note: Caution advised with concomitant use of drugs affecting CYP450. Has drug interactions with quinidine, tricyclic antidepressants, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors, and others
Note: For short-term use (up to 5 days) for acute pain and for whom alternative treatments are inadequate

3.4 Tumor Necrosis Factor-α Inhibitors and Interleukin-1 Receptor Antagonists

3.4.1 Background

The overproduction of the inflammatory cytokine tumor necrosis factor-alpha (TNF-α) plays a key role in the maintenance of many chronic inflammatory rheumatic diseases, particularly those that depend on the relationship between T cells and macrophages [25]. TNF-α has been shown to induce bone reabsorption, to inhibit proteoglycan [26, 27] and collagen synthesis [28, 29], to induce prostaglandin E_2 and collagenase release from synovial cells, and to stimulate fibroblast growth [30]. TNF-α also plays a pivotal role in the enhancement of inflammatory cell trafficking into synovium, by regulating the expression of adhesion molecules on the endothelial cells [31, 32]. It also upregulates the production of other pro-inflammatory cytokines, such as interleukin-1 (IL-1) and interleukin-6 (IL-6), and the expression of their respective receptors [33]. TNFα blockade has been therapeutically achieved through the administration of monoclonal antibodies against TNFα (mAb) or soluble TNFα receptors (sTNFR).

Five anti-TNFα agents are currently approved in Europe and the United States for use in adult-onset rheumatoid arthritis (RA), spondyloarthritis (SpA), and psoriatic arthritis (PsA): the fusion protein combining two p75-TNF-α receptors with a Fc fragment of human IgG1 (etanercept [Enbrel®]); the chimeric, human and murine mAb against TNF-α (infliximab [Remicade®]); the fully human TNF-α mAbs (adalimumab [Humira®] and golimumab [Simponi®]); and the PEGylated Fab' fragment of a humanized mAb against TNF-α (certolizumab [Cimzia®]). Etanercept and adalimumab are also approved in Europe and United States for use in polyarticular Juvenile Idiopathic Arthritis (JIA). Infliximab is approved only for JIA-related uveitis.

Monoclonal antibodies, in particular the chimeric antibody infliximab, specifically and potently bind and neutralize not only the soluble TNF-α but also its membrane-bound precursor inducing cell lysis by apoptosis; their action mechanism is different and more selective than that of the recombinant p75-TNF-α receptor Fc fusion protein (etanercept), which does not cause cell lysis and inhibits not only TNF-α but also lymphotoxin (LT, formerly called TNF-β); however, the role of lymphotoxin in the pathogenesis of rheumatic diseases is not well understood [34].

Controlled trials have shown that the TNF-α inhibitors significantly reduce symptoms and signs, improve function and quality of life, and reduce radiological damage in RA and related diseases. Concurrent treatment with methotrexate (MTX) appears to enhance the therapeutic response.

The TMJ can be affected by a variety of immune-mediated disorders including RA, SpA, PsA, and primarily JIA, so biologics such as TNF-α inhibitors may be beneficial in the treatment of this patient cohort. Inflamed human synovial fluid and synovial membrane have been found to have elevated TNF levels, which signal synovial cells to produce metalloproteinases and collagenase [35]. Furthermore, TNF-α has been identified in the TMJ synovial fluid in patients with chronic

inflammatory TMJ disorders, which is the rationale for the use of a TNF-α inhibitor in the treatment of immune-mediated TMJ disease [36]. Etanercept was shown to be an effective nonsurgical treatment in a patient with severe psoriatic arthritis affecting the TMJ, allowing the patient to achieve full remission of the TMJ symptomatology as well as the skin lesions [37].

Biologic drugs, such as TNF inhibitors, refer to genetically engineered drugs that have been designed to modulate a specific aspect of the underlying immune process, thus avoiding generalized immunosuppression, with the hope that such specificity will result in higher efficacy and fewer adverse events (AEs) than traditional cytotoxic disease-modifying antirheumatic drugs (DMARDs). However, as use of these agents has increased worldwide during the post-marketing period, infrequent AEs that were not apparent in the randomized controlled trials (RCTs) required for registration have emerged [38–40]. The neutralization of TNF is associated with an increased risk of certain serious but uncommon AEs, including serious bacterial infections such as tuberculosis (TB) and certain opportunistic infections, malignancy/lymphoma, congestive heart failure, demyelinating and neurological disorders, injection-site reactions or infusion-related systemic reactions, newly induced autoantibodies, and lupus-like disease. However, several of these risks (e.g., lymphoma and serious infections) are associated with either the disease per se or the concomitant and previous immunosuppressive treatments [41]. These AEs may be related to blockade of TNF and may therefore represent class effects of these agents. Overall, tolerability of anti-TNF therapy in JIA is good. However, the severity and degree of the risk may not be the same with all agents. In general, infliximab was discontinued more often than etanercept or adalimumab because of AEs, mainly infusion reactions [42, 43]. Therefore, anti-TNF agents should be prescribed with caution due to their potential risks (Atzeni 2015). Thus, in addition to evaluating the indication of this biologic therapy, a careful diagnostic workup before starting the treatment is needed; in particular, patients must be screened for tuberculosis (TB), hepatitis B virus (HBV) infection, and hepatitis C virus (HCV) infection. Of course, the treatment should not be initiated in patients with active infections, until those are controlled. Moreover, specific attention should be paid to patients who have travelled in areas of high risk of tuberculosis or endemic mycoses, such as histoplasmosis, coccidioidomycosis, or blastomycosis. Finally, a close monitoring of patients during the therapy should be warranted, in order to timely evidence infectious reactivation by the aforementioned agents and serious and/or opportunistic infections [44].

The use of TNF-α inhibitors in the contemporary treatment of JIA is an exciting field of study. Clinically detectable TMJ involvement in patients with JIA has been reported to vary, affecting up to 75% of patients [45–48]. Anti-TNF agents, often administered with methotrexate (MTX), a disease-modifying antirheumatic drug (DMARD), have been used to manage pediatric and adult patients who have autoimmune or immune-modulated inflammatory arthritidies of the TMJ [49–51].

3.4.2 Etanercept (Enbrel®)

Etanercept (ETN) is a soluble TNF-binding dimer molecule, consisting of the extracellular ligand-binding portion of the human 75 kDa TNFR2 linked to the Fc portion of the human Immunoglobulin G1 (IgG1). The recommended dose in adult is 50 mg given once weekly and in children is 0.8 mg/kg (up to a maximum of 50 mg per dose). The administration of ETN must be discontinued if patients develop serious infection. ETN is a well-tolerated drug with generally mild injection site reactions being the main AEs in placebo-controlled studies. More serious AEs such as skin vasculitis or drug-induced lupus have been rarely described, as well as extracutaneous manifestations (e.g., pancytopenia, aplastic anemia, changes in mood, weight gain, autoimmune hepatitis, cholecystitis, macrophage activation syndrome, etc.) [52].

In clinical trials, no interactions have been observed when ETN was administered with glucocorticoids, salicylates (except sulfasalazine), and nonsteroidal anti-inflammatory drugs (NSAIDs). Adult patients receiving combination therapy with ETN and sulfasalazine experienced a statistically significant decrease in mean white blood cell counts compared to the single therapies [53]. Unfortunately, not all patients have clear benefits from therapy with ETN; observational registries suggested that only 40–50% patients have an optimal response. According to EMA recommendations, the discontinuation of treatment should be considered in patients who show no response after 4 months.

The biological mechanisms underlying drug failure of ETN are largely unknown. A few studies provided some highlights on mechanisms or biomarkers affecting the therapeutic response to ETN. Some of those might be extended also to the other TNF antagonists [54]. However, while waiting for the availability of prognostic biomarkers, an earlier use of ETN might improve the natural history of RA and JIA. Indeed, recent studies suggested that earlier use of TNF inhibitors could improve patient remission rates and functional status as well as slow disease progression [55].

3.4.3 Infliximab (Remicade®)

Infliximab (IFX) is a chimeric mAb directed against TNF-α. It is named "chimeric" because it refers to the use of both murine and human components to make this molecule. The variable domains of the heavy and light chains of the antigen-binding portion of IFX are murine (and act as binding sites for TNF-α), while the constant Fc domain is 75% human IgG1 isotype. IFX is produced in murine hybridoma cells by recombinant DNA technology [56].

Unlike ETN, IFX can bind not only to soluble TNF-α molecules but also to membrane-bound TNF molecules, leading to both antibody-dependent cell-mediated cytotoxicity (ADCC) and complement-dependent cytotoxicity (CDC), and it does not interact with lymphotoxin (TNF-ß). The binding between IFX and TNF-α rapidly forms stable complexes, resulting in the loss of TNF-α bioactivity [57].

Currently, EMA recommendations for prescribing IFX in rheumatic diseases do not include JIA; IFX is allowed only in adults, in the setting of RA, SpA, and PsA. IFX is administered by doses of 3 mg/kg in adults and 5–6 mg/kg in children, given as an intravenous infusion followed by additional infusions at 2 and 6 weeks after the first infusion and every 8 weeks thereafter. Available data do not support further IFX treatment in patients not responding within the first 10 weeks of treatment. Intervals between consecutive administrations can be adjusted, according to the individual clinical response, as some patients may require a shorter dosing interval to maintain clinical benefit, while for others a longer dosing interval may be sufficient. However, shortening the dose intervals may put one at greater risk for adverse reactions [58]. The administration of IFX in combination with MTX is recommended in rheumatic diseases: in addition to the clinical benefit, the concomitant use prevents the development of anti-IFX antibodies, which seem to correlate with infusion reactions, accelerated clearance, and loss of clinical benefit [59].

Infections remain the major concern with the use of IFX. Many more cases of tuberculosis have been reported in patients treated with IFX than in those treated with ETN, probably because of the destabilization of previously formed granulomata. Other infections that have occurred with greater than expected frequency include histoplasmosis, coccidioidomycosis, and listeriosis.

In addition to the AEs noted with ETN, great attention should be paid to the risk of infusion reactions, ranging from mild allergic reactions to anaphylactic reactions, which may occur, more commonly on the second or third infusions. Therefore, IFX must be administered under close observation. Patients may be pre-treated with an antihistamine and/or hydrocortisone, because of previous reactions: in these cases, the infusion rate may be slowed in order to decrease the risk of recurrence.

3.4.4 Adalimumab (Humira®)

Adalimumab (ADM) is a recombinant human IgG1 mAb against TNF. It is produced by recombinant DNA technology using a mammalian cell expression system. ADM acts in a similar fashion to IFX, as it can bind TNF-α both in the circulation and on the cell surface.

In adult patients, ADM is usually administered subcutaneously at a dose of 40 mg every 2 weeks. The initial recommended dosing in children is 24 mg/m^2 body surface area. In North America, the tendency is to administer 20 mg for patients weighing less than 30 kg and 40 mg for patients weighing more than 30 kg. According to EMA recommendations, the dose of ADM for patients with JIA, aged 2–12 years, is 24 mg/m^2, up to a maximum single dose of 20 mg (for patients aged 2–4 years) and up to a maximum single dose of 40 mg (for patients aged 4–12), administered every other week [60].

Like other TNF antagonists, serious infections due to bacterial, mycobacterial, fungal, parasitic, viral, or other opportunistic infections (such as listeriosis, legionellosis and pneumocystis) have been described during the therapy with ADM. In the available clinical trials, sepsis, pneumonia, pyelonephritis, and septic arthritis

have been reported. Despite prophylactic therapy, cases of reactivated tuberculosis have occurred in patients treated with ADM. Moreover, reactivation of hepatitis B in patients who are chronic carriers of this virus (i.e., surface antigen positive) can occur when receiving a TNF antagonist including ADM. Serious systemic allergic reactions associated to ADM have been rarely described in the clinical trials. As in the case of IFX, antibody production against the biologic drug was lower when ADM was given with MTX. Thus, the administration of ADM without MTX resulted in increased formation of antibodies, increased clearance, and reduced efficacy of the therapy. Actually, there is no evident correlation between the presence of anti-ADM antibodies and the occurrence of AEs [60].

3.4.5 Golimumab (Simponi®)

Golimumab (GLM) is a recombinant human IgG1k mAb against TNF, produced by using a murine hybridoma cell line, through recombinant DNA technology. The amino acid sequence of constant regions of the heavy and light chains of this mAb is identical to the corresponding constant regions of IFX. However, in contrast to IFX, the heavy and light variable regions are of human sequence [61]. GLM is able to bind and inhibit both soluble and transmembrane human TNF. It forms high affinity and stable complexes preventing the binding of TNF-α to its receptors [62].

The FDA and EMA approved GLM for the treatment of moderately to severely active adult RA, PsA, and SpA. Currently, EMA approved the indication of GLM for the treatment of JIA in children with a body weight of at least 40 kg, who have responded inadequately to previous therapy with MTX and in combination with it. The recommended dosage is 50 mg subcutaneously once a month, in combination with MTX. The most frequent AEs were infections and injection site reaction. The most common reported infections with GLM are upper respiratory tract infections, while the most serious include sepsis, pneumonia, TB, invasive fungal and opportunistic infections, and HBV reactivation [61]. The formation of antibodies to GLM did not appear to have a substantial impact on clinical efficacy or to be associated with injection site reactions. The follow-up during the treatment with GLM is similar to that described for other anti-TNF agents. No interaction studies have been performed. Although the concomitant use of MTX resulted in higher steady-state concentrations of GLM, no dose adjustment of either GLM or MTX is needed so far [61]. Subcutaneous administration, permitting home self-injection, and less frequent dosing regimen (GLM is administered once/month, as compared with once/week for ETN, once/2 weeks for ADM, and once/2 or 4 weeks for certolizumab pegol) might promote adherence.

3.4.6 Certolizumab Pegol (Cimzia®)

Certolizumab pegol (CTZ) is a pegylated (conjugated with polyethylene glycol, PEG) humanized Fab′ fragment of a mAb that binds TNF. As opposed to IFX, ADM,

and GLM, which are full-length bivalent IgG1 mAb, CTZp is a monovalent Fab antibody fragment. CTZ is expressed in *Escherichia coli* system by DNA recombinant technology and, then, is conjugated to PEG [63]. CTZp has a high affinity for human TNF-α that is selectively neutralized, but it does not bind lymphotoxin α (TNF-β). CTZp was shown to link membrane-associated TNF in a dose-dependent manner, too. As CTZp does not contain the Fc region, which is normally present in a complete antibody, it cannot fix complement, induce CDC, or cause ADCC [57, 62].

The PEGylation substantially delays the elimination of CTZp from the circulation by a variety of mechanisms, including decreased renal clearance, decreased proteolysis, and decreased immunogenicity. The half-life was estimated to be approximately 14 days, regardless of the administered dose. FDA approved the use of CTZp for the treatment of RA and Crohn's disease in 2008 [64].

3.4.7 Anakinra (Kineret®)

Because interleukin-1 (IL-1), a powerful pro-inflammatory cytokine, in addition to TNF-α, is also an important mediator of TMJ intracapsular pathology [65], drugs that block IL-1 have shown promise in the treatment of a wide variety of autoinflammatory syndromes [66–68]. Anakinra (Kineret®), the first direct and selective interleukin-1 receptor antagonist, was approved for treatment of moderately to severely active rheumatoid arthritis in adults who have failed one or more DMARDs. Other IL-1 inhibitors, currently approved for the treatment of specific rare autoinflammatory orphan diseases, include rilonacept and canakinumab [69]. The exploration of the IL-1 pathway and its role in human disease is driving research into targeted therapies for diseases that produce organ-specific as well as systemic inflammation.

3.5 Muscle Relaxants

Muscular pain is a common symptom described by those who splint their muscles to prevent painful jaw movement. Muscle splinting is a condition of hypertonicity that accompanies injury; the contracture locks the painful joint to reduce the jaw's range of motion. Muscle relaxants can provide welcome relief from myalgia and myospasm when prescribed early and on a short-term basis, as they are not as effective in chronic musculoskeletal pain [70]. When taken before bedtime, improved sleep quality is promoted and the TMJ and associated muscles can rest, especially when combined with a well-adjusted occlusal splint. In bruxers and clenchers with secondary joint pain in addition to pain in the muscles of mastication, muscle relaxants can help unload the joint and allow for reduction of compressive, and potentially destructive, condylar forces. In contrast, no change in disc position is to be expected for those patients with internal derangement. Interestingly, in a double-blind placebo-controlled study by Alencar et al. that randomly assigned patients with morning jaw pain to groups that received placebo, cyclobenzaprine, or tizanidine, in addition to a

program of patient education, muscle relaxants were not found to add to jaw comfort. The patient education program included an explanation of myofascial pain and possible etiologies, instructions in how to achieve mandibular rest position, diet modification, heat therapy to the muscles, and the use of ibuprofen as a rescue medication. The data revealed that patients in all three groups improved but that no statistically significant difference among the groups could be observed [71].

Myorelaxant drugs have been observed to have efficacy in pain relief in patients with back pain when combined with antispastic drugs, such as tizanidine, benzodiazepines, and baclofen [72]. However, providers should exercise caution when using drug combinations because the systemic effects are many; the adjunctive use of physical therapy and/or other non-drug modalities may be considered to be drug-sparing for patients suffering from acute and chronic TMD pain, or other musculoskeletal conditions.

Myorelaxants and anti spastic drugs
Cyclobenzaprine Immediate-release: Initial dose: 5 mg PO up to tid; may increase up to 10 mg PO tid Extended-release: Initial dose: 15 mg PO qd, may increase to 30 mg PO qd Duration of therapy: limit to 2–3 weeks Do not prescribe if taking MAO inhibitors within past 14 days
Carisoprodol 250–350 mg up to three times daily Duration of therapy: limit to 2–3 weeks Habit-forming, potentiates opioids
Methocarbamol Initial dose: 1500 mg qid for 2–3 days, up to maximum of 8 g/day Maintenance dose: 4000–4500 mg/day in divided doses
Baclofen Initial dose: 5 mg tid × 3 days, then 10 mg PO tid × 3 days, then 15 mg PO tid × 3 days, then 20 mg PO tid × 3 days Maintenance dose: Should be individualized Maximum dose: 80 mg/day
Tizanidine Initial dose: 2 mg PO q6–8 h prn; may increase dose by 2–4 mg every 1–4 days until satisfactory reduction of muscle tone Maximum single dose: 16 mg Maximum daily dose: 36 mg in divided doses Not known to be habit-forming
Diazepam 2–10 mg tid-qid prn

3.6 Tricyclic Antidepressants

As with many other types of pain disorders, TMD is often accompanied by depression and anxiety that benefits from concurrent treatment. Some drug categories used to treat depression, such as selective serotonin reuptake inhibitors (SSRIs),

can actually induce bruxism [73–77]. Tricyclic antidepressants (TCAs) may be a non-habit-forming alternative to treat depression where SSRI bruxism is a problem.

The research has been mixed regarding whether low doses of TCAs are effective in treating the chronic pain associated with TMD [78–83]. For example, Haviv et al. showed that 54.7% of patients with persistent facial pain and tenderness of regional muscles reported improvement on low-dose amitriptyline or its metabolite nortriptyline [78]. Bendtsen and Jensen studied amitriptyline versus citalopram versus placebo in a double-blind three-way crossover study of non-depressed patients with chronic headache. They concluded that amitriptyline at a daily dose of 75 mg reduced tenderness and headache significantly more than placebo, while citalopram, an SSRI drug, had no significant effect [81]. Some investigators have found that TCAs reduce the number of sleep disturbances and therefore postulated that this drug class could be effective in the treatment of nocturnal bruxism; however, no significant differences were found between amitriptyline and placebo for masseteric EMG activity or in sleep duration in two double-blind clinical trials [82, 84]. Nevertheless, patients with sleep bruxism, which is associated with sleep disturbances, abnormal tooth wear, jaw muscle fatigue upon awakening, and dental sensitivity from excessive forces, may be candidates for a trial of TCAs. When in doubt, full-night polysomnography (PSG) with electromyography of the masseter and temporalis muscles is the gold standard and can identify other sleep-related problems such as obstructive sleep apnea and periodic limb movement disorder.

In those taking other anticholinergic drugs, or taking antihypertensive medications, the TCA regimen should begin with the lowest dose. In all patients, the dose may be gently increased every 1–2 weeks until benefit is achieved; titrate every 3–4 weeks for the elderly. The main side effects for amitriptyline and nortriptyline are dry mouth, dizziness, constipation, blurred vision, and daytime sedation. Patients occasionally report agitation and difficulty sleeping with nortriptyline when taken at night. If this happens, switching the dose to the morning or in two divided doses may help.

Tricyclic antidepressants
Amitriptyline 10–25 mg PO prior to bed, gradually taper up to effect Tablet strengths: 10, 25, 50, 75, 100, and 150 mg Most responders achieve benefit with dose 25–30 mg per day Maximum dose is 150 mg per day
Nortriptyline 10–25 mg PO 1 h before bed, gradually taper up to effect Tablet strengths: 10, 25, 50, and 75 mg Most responders achieve benefit with dose of 25–75 mg per day Maximum dose is 150 mg per day
Risk of serotonin syndrome if taken in combination with serotonin reuptake inhibitors (SSRIs) or monoamine oxidase inhibitors (MAOIs)

3.7 Topical Medications

Medicated patches, well tolerated by many patients, are a convenient delivery system to bring localized pain relief to the head and neck. For patches containing NSAIDs, the gastrointestinal tract is bypassed, avoiding the usual adverse GI effects that many patients experience. In addition, topical medications act peripherally because they are absorbed right at the site of application, thus affecting a change on the peripheral receptors only, resulting in insignificant serum drug concentrations, little to no systemic side effects, and improved patient compliance. Drug interactions are avoided and titration of dose is not necessary. If patches are not available or too expensive, one may try substituting a layer of medicated ointment/gel directly to the skin at the site of pain, and cover with a dressing. In contrast, transdermal medications, such as transdermal nicotine or fentanyl, do require a minimum serum concentration in order to be effective, which means that systemic side effects and drug interactions are possible, and titration is necessary [85].

There is a paucity of data on the use of topical NSAIDs for TMJ degenerative joint disease, but topical NSAIDs have been shown to provide good pain relief in patients with chronic musculoskeletal pain of the knee and hand, with reduced incidence of gastrointestinal adverse effects compared with oral NSAIDs [86]. The best data is for topical diclofenac, with differences in formulation being the single largest factor in determining efficacy of the product. A meta-analysis by Lin et al. found improvement in pain and function in patients with osteoarthritis of the knee, hand, or hip when treated with topical NSAID during the first 2 weeks, although the effect dropped off in weeks 3 and 4 [87]. Consistent with this, a meta-analysis by Bjordal's group showed that topical NSAIDs were best at reducing knee pain from osteoarthritis within 1.6 weeks, with a lesser effect after 4 weeks [88]. A retrospective study examining patients with orofacial neuropathic pain found that topical medication (a gel containing carbamazepine 4%, lidocaine 1%, ketoprofen 4%, ketamine 4%, and gabapentin 4%) was found to provide rapid pain relief alone or when used in combination with systemic medication [89]. In contrast, topical NSAID application failed to demonstrate efficacy in patients with TMJ degenerative joint disease, as concluded by Senye et al. through a systematic review [90]. One reason for this may be the relatively small surface area at the TMJ for application of sufficient topical medication to effect suitable absorption of the medication. At this time, topical formulations of NSAIDs, including patches, should be considered to be adjunctive alternative treatment modalities for TMJ pain, especially for patients who prefer not to take oral medications or are at high risk for GI complications from this drug class.

Lidocaine patches and gels have not been well studied in TMJ patients. When used for the treatment of head and neck postherpetic neuralgia, however, topical lidocaine is been effective and well tolerated [91, 92]. The pain threshold of the facial skin at the cheek overlying the masseter muscle is susceptible to topical local anesthetic gel, and these results should be further studied to determine the efficacy of this modality for myofascial pain associated with TMD [93]. One is encouraged by studies showing that the lidocaine 5% patch results in pain relief for patients with moderate-severe myofascial pain of the lower back and for myofascial pain of the

trapezius [94, 95]. In summary, it appears that topical lidocaine, including the 5% patch, is a reasonable adjunctive treatment for selected patients with myofascial pain of the head and neck.

Patches
Diclofenac 1.3% patch (Flector)
One patch placed over painful joint up to twice daily
The patch may be trimmed with scissor to fit affected area
Lidocaine 5% patch (Lidoderm)
700 mg dose
One patch placed over painful area for 12 h, then off for 12 h
The patch may be trimmed with scissor to fit affected area

3.8 Dietary Supplements

Over-the-counter dietary supplements such as glucosamine sulfate and chondroitin sulfate are popular among those seeking adjunctive remedies for joint pain and dysfunction. Glucosamine is a monosaccharide and is primarily produced by processing chitin from the shells of shellfish. It is commonly commercially available as a supplement that is combined with chondroitin sulfate. Chondroitin is a glycosaminoglycan (GAG) that is a long-chain repeating disaccharide consisting of alternating sugar molecules. It is naturally found in articular cartilage and has been characterized as supporting cartilage compression resistance as well as joint mobility and flexibility [96].

Both glucosamine and chondroitin, separately or in combination, have not been definitively shown to benefit patients with osteoarthritic joints. The data is quite mixed, leading many groups to reject any recommendation for these supplements. The American College of Rheumatology (ACR) concluded there was no evidence of benefit for osteoarthritis [97]. The American Academy of Orthopedic Surgeons (AAOS) in its 2017 Clinical Practice Guideline on the Management of Osteoarthritis of the Hip includes a comment that moderate strength evidence did not show that glucosamine sulfate was any better than placebo [98]. The National Center for Complementary and Integrative Health notes that experts disagree on the benefit of glucosamine and chondroitin in knee and hip osteoarthritis, although the group observed that several European studies demonstrated benefit for knee pain when participants took a large, once-daily dose of glucosamine sulfate [99]. This was in contrast to the findings of a large NIH trial that failed to show statistical improvement with glucosamine hydrochloride and chondroitin sulfate together or alone, for patients with mild knee pain. For a small subset of study subjects with moderate to severe pain, there was a statistically significant reduction in pain with the combination of glucosamine and chondroitin [100]. Regarding a proposed benefit between glucosamine and chondroitin and a reduction in the risk of osteoarthritis, the FDA has tentatively concluded that sufficient evidence does not exist. However, it is generally accepted that these dietary supplements are quite safe at doses of glucosamine sulfate 1500 mg and chondroitin sulfate 1200 mg daily.

Okazaki et al. in 1997 quantified the different disaccharide isomers of chondroitin in diseased versus healthy TMJ synovial fluid and concluded that the analysis was a good indicator of the degree of internal derangement [101]. In a sheep TMJ model, Ishimaru et al. demonstrated that synovial fluid chondroitin sulfate showed a positive correlation with the severity of surgically created TMJ osteoarthritis. This suggested that the increase in chondroitin in synovial fluid represented either release of the molecule from ongoing articular degradation or from hypersynthesis resulting from the joint's attempt to initiate repair [102]. In either scenario, the biochemical alteration of the synovial fluid in diseased joints was confirmed.

Shankland in 1998 reported that 80% of his study subjects experienced a decrease in TMJ noise with an associated decrease in joint pain and swelling with a combination of glucosamine, chondroitin, and vitamin C [103]. Other investigators have also demonstrated the benefit of glucosamine sulfate (1500 mg per day) compared to ibuprofen. Study participants in the glucosamine sulfate intervention groups showed superior therapeutic value with respect to pain relief and jaw opening, as well as having a lower rate of adverse effects [104, 105]. Nguyen et al. studied the combination of glucosamine and chondroitin versus placebo in TMD patients and concluded that the dietary supplements benefited patients, who were also able to reduce the number of daily nonprescription medications needed [106]. The effect of glucosamine-chondroitin combination was also shown to be as effective as tramadol, a narcotic analgesic, in a group of patients with painful internal derangement [107]. Unfortunately, glucosamine was not superior to placebo in a well-designed double-blind, randomized controlled trial over 6 weeks, for patients with painful TMJ osteoarthritis [108].

It is clear that the use of glucosamine and chondroitin is controversial. There is insufficient evidence for providers to turn to in order to make a recommendation for or against. However, the supplements are safe and well tolerated, and there are possibly subsets of patients who may benefit, thus avoiding the adverse effects of prescription drugs.

3.9 Trigger Point Injections

Trigger point injections have been well established to be of highly effective to relieve pain and improve associated symptoms of depression for individuals with myofascial pain related to the masticatory muscles and other muscles of the head and neck [109]. It is a technique that is relatively easy to perform in an office or outpatient clinic setting, and the injections themselves are well tolerated by patients. Experience in the technique has a relatively short learning curve. The goal of the trigger point injection is to eliminate the trigger point and to soften and relax the tender, hyperirritable, and firm areas in the target skeletal muscle. How trigger points develop is not well understood, but the hypothesis is that trauma, microtrauma, or repetitive stress is responsible [110].

Trigger points may be active or latent [111]. Active trigger points are tender when palpated, and if sufficient pressure is exerted (2–4 kg/cm^2 of pressure for 10–20 s), the patient will experience referred pain to distant sites. The referred pain

does not necessarily adhere to a dermatome or follow muscular anatomy, but it is reproducible. Latent trigger points are painful to palpation but the pain does not radiate. When a muscle with trigger points is stretched or contracted, there is significant pain leading to a reduction in jaw or neck range of motion. Trigger points associated with myofascial pain may be palpated as taut muscle bands or firm nodules within the tissue deep to the skin and subcutaneous tissue. Classically, when one flicks a finger over the taut band of muscle, like snapping a guitar string, a muscle contraction is noted, also known as a "twitch response" [109]. The twitch response is also often seen when the needle is inserted into the trigger point. Precise localization of the trigger points, a very hands-on maneuver accomplished with the patient's help, is the key to excellent pain relief, which the patient often experiences immediately after the injections.

Interestingly, dry needling or acupuncture needling has been found to be as effective as injection of local anesthesia for the treatment of trigger points [112–114]. It seems that disruption of the trigger point is not dependent on the numbing produced by the local anesthesia, although perhaps post-needling pain is reduced with local anesthesia.

Trigger point injections are often combined with other modalities such as splint therapy, muscle relaxants, and/or antidepressant medication, which may add to the efficacy of treatment of those diagnosed with myofascial TMD pain [115, 116].

3.9.1 Author's Technique

Trigger point injection
Clean skin with alcohol pads. Palpate masticatory and neck muscles to identify trigger points. Be sure to exert sufficient pressure, and determine if the trigger point palpation results in radiating pain and a possible twitch response (active trigger point) or localized pain (latent trigger point). Mark trigger points with a skin marker
Needle and syringe selection: 1.5-in. 27 gauge dental needle on a dental aspirating syringe or a 1-in. 27 gauge needle on a 3 cm^3 disposable syringe. Thirty gauge needles are too soft and harder to use because it is more difficult to get feedback on needle position within the muscle
Injection solution selection: Commonly available dental local anesthetics work very well (a) Bupivacaine 0.5% with 1:200,000 epinephrine. (b) Lidocaine 2% with 1:100,000 epinephrine. (c) Articaine 4% with 1:100,000 or 1:200,000 epinephrine. (d) Mepivacaine 3% plain, lidocaine 1% plain, or lidocaine 2% plain: For cases where there is a contraindication to epinephrine, or if a preservative-free solution is desired. Local anesthetic solutions without epinephrine are more likely to result in localize bleeding and possible skin ecchymosis.
Press alcohol pad once again over the injection site, pinch or stabilize muscle, introduce needle into the skin, and advance into the skeletal muscle's trigger point. Aspirate and inject approximately 0.5 mL of local anesthesia, less in smaller, thinner muscles and more in thicker, deeper muscles. Some operators withdraw the needle and reenter the trigger point from various angles, injecting small amounts each time
Apply some pressure to the site of each trigger point injection for hemostasis. Use ice pack if bleeding occurs or hematoma begins to form
After injection(s), take the patient through the range of motion for the jaw or neck several times, allowing the injected muscle to contract and stretch

Precautions

- Avoid vasovagal response by reclining the patient prior to injection.
- Do not inject into any area where there is frank inflammation or infection.
- Prior to injection, determine if the patient's medical history includes drugs or medical conditions that may predispose to increased bleeding.

In conclusion, trigger point injections are very efficacious for relief of pain that is myofascial in origin. Trigger point injections are repeatable and can be combined with other therapies, including splint therapy, physical therapy, and medications. It should be noted that individuals with long-standing myofascial pain are often in psychological distress; therefore providers should identify those patients early and refer for psychological evaluation for possible concurrent treatment with antidepressants.

3.10 Intra-articular Joint Injections

The delivery of medication directly to the joint space is safe and avoids many of the untoward effects of systemic medications that are taken orally or parenterally.

3.10.1 Steroids

Intra-articular steroid injections have a proven pain relief effect for patients with inflammatory temporomandibular joint disease, although the optimal dose and re-dose interval is still debated. Corticosteroids delivered to the TMJ superior joint space pass through cell membranes and bind to glucocorticoid receptors within the cytoplasm, which reduces synovial inflammation by blocking production of prostaglandins and leukotrienes and increasing the production of anti-inflammatory proteins such as interleukin-10 and interleukin-1 receptor antagonist. In addition, glucocorticoid injection reduces the level of serotonin and tumor necrosis factor-alpha (TNF-α) within TMJ synovial fluid. For example, high pre-treatment levels of TNF-α decrease following injection, and this is predictive of pain elimination [36, 117]. The molecular activity described has the effect of decreasing pain, resolving joint effusion, and promoting early return to function. The addition of physical therapy or splint therapy after intra-articular injection may further improve the level of benefit of the potent anti-inflammatory effect. The onset of action is quite rapid, often within 24 h, and the author's experience is that the clinical effect lasts 4–8 weeks and often much more.

Several different corticosteroid injection regimens have been tested, including triamcinolone acetonide 40 mg/mL concentration (Kenalog-40), triamcinolone hexacetonide (Aristospan 20), and methylprednisolone acetate 40 mg/mL (Depo-Medrol). Triamcinolone hexacetonide has lower solubility and longer duration of action than triamcinolone acetonide. The corticosteroid of choice is often combined with a long-lasting local anesthetic, such as lidocaine 2% or bupivacaine 0.5%, for

added comfort. Arabshahi et al. found that most children with juvenile idiopathic arthritis had pain resolution and improved maximum opening following either 1 cm^3 (40 mg) of triamcinolone acetonide or 1 cm^3 (20 mg) of triamcinolone hexacetonide [118]. Samiee et al. analyzed a series of TMD patients with painful disc displacement without reduction, all of whom received an intra-articular injection of 1 mL of 2% lidocaine without epinephrine mixed with and 0.5 mL (20 mg) of triamcinolone acetonide as a single, first-line treatment. A statistically significant improvement in mouth opening was noted, with a trend toward improved comfort [119]. Common indications for intra-articular steroid injections are in the table below.

Common indications for TMJ intra-articular steroid injections
Adult and juvenile rheumatoid arthritis
Crystal-induced arthritis (gout, pseudogout)
Spondyloarthropathy (psoriatic arthritis)
Osteoarthritis
Capsulitis
Synovitis

Complications from TMJ intra-articular steroid injections are rare. Some caution against the use of intra-articular steroid injections because high doses and multiple injections have been reported to be associated with articular cartilage destruction, infection, and disease progression [120, 121]. Patients with diabetes may find that blood glucose levels rise following an intra-articular injection, but careful technique and low injection volume make this an uncommon problem. Caution is advised when treating children with juvenile idiopathic arthritis due to the impact of significant mandibular growth suppression by corticosteroids repeatedly introduced close to the mandibular growth center [122]. This has also been demonstrated in animals, where multiple corticosteroid injections in the TMJ negatively affected dentofacial development [123].

3.10.1.1 Author's Technique

The injection technique is easily done in clinic on an awake, cooperative patient. Stoll et al. demonstrated that an experienced oral and maxillofacial surgeon can safely and accurately perform intra-articular steroid injections for children with juvenile idiopathic arthritis without CT guidance [124]. The author has a preference for corticosteroids and local anesthetics used together, in order to give patients rapid relief of pain due to the initial action of the local anesthetic. After the anesthesia wears off, many patients experience an increase in pain from the injection until the corticosteroid effect begins, usually a day later.

TMJ intra-articular steroid injection Author's technique
Clean preauricular skin with alcohol wipes.
Palpate for condylar head by asking patient to open and close. Measure landmarks for condylar head/fossa, and mark skin with marker

Position the patient in a semi-supine manner. Some exhibit vasovagal reactions with injections.
Local anesthesia given to skin with 27 gauge needle. Enter the skin using an up-angle of 30° and anterior angle of 30°, to avoid entering the ear canal. Determine the location of the superior joint space by contacting the needle tip against the bone at the root of the zygomatic arch, lateral to the articular fossa. If uncertain whether needle tip is on the condylar head or the zygomatic arch, ask patient to open and close to determine if needle tip deflects with the movement. If so, angle needle more superiorly or reenter skin slightly more superiorly. – Use ~1 cm^3 of local anesthesia of choice.
Mix maximum of 0.5 cm^3 Kenalog-40 (total triamcinolone 20 mg) with 0.5 cm^3 of local anesthesia. – Author mixes triamcinolone with 0.5 cm^3 of bupivacaine 0.5% 1:100,000 epinephrine, for total injection volume of 1 cm^3 per joint – Use 3 cm^3 syringe with 1″ 27 gauge needle; 30 gauge needles are too flexible
Enter skin in a similar manner as for the local anesthesia, and advance needle to enter the superior joint space. – If overlying tissues are thick, advance needle as much as 20 mm, or nearly the length of the 1″ needle – The patient should relax jaw and open slightly, facilitating entry of needle into the joint space
Inject slowly – If significant pressure during injection is encountered, reposition needle slightly – The patient may complain of pressure sensation or hear noise
Wait a few seconds for solution to settle into the joint space, then withdraw needle.

Postinjection Findings: Due to the local anesthesia, the patient may have temporary palsy of the temporal and zygomatic branches of the facial nerve. Pain relief may be instantaneous, also due to the local anesthesia. Because of the volume injected at the level of the superior joint space, it is common to see a small posterior open bite on the ipsilateral side immediately after the injection. Once the steroid solution is fully absorbed, usually a matter of several hours to 1 day, the occlusion will return to preinjection state.

Postinjection Directions: Soft, non-chewy diet is advised for 24 h after the injection.

Recommended Re-dose Interval: For rheumatoid arthritis, injections may be given every 3 months. For osteoarthritis, longer intervals are recommended, such as every 4–6 months if significant discomfort returns. For reasons that are not well understood, the author has frequently noted that the best result is usually achieved with the first intra-articular steroid injection and a somewhat less effective result with subsequent injections.

3.10.2 Hyaluronic Acid

Viscosupplementation is the term used for the injection of a lubricating fluid that is injected into a joint. The terms hyaluronic acid, “HA,” sodium hyaluronate, and hyaluronan are often used interchangeably, and all refer to a natural complex carbohydrate of the glycosaminoglycan group that is a heavy, long-chain polysaccharide

polymer. It is found in high concentrations in synovial fluid, the skin, and the eye. In joints, HA contributes to the viscosity of the synovial fluid and is felt to act as a shock absorber for the joint because large hyaluronan aggregates trap water molecules and thus improve the ability of cartilage to resist compressive forces. Even in the absence of osteoarthritis, the concentration of HA within knee synovial fluid decreases substantially with age across all different HA molecular weight groups, suggesting that age-related changes in HA are related to age-related changes in knee cartilage deterioration [125]. The goal of viscosupplementation, therefore, is to replenish the deficit.

The original polysaccharide was isolated from bovine vitreous humor, and now, HA that is used for medical purposes is derived from rooster comb, umbilical cord, or bacterial cultures. HA has been most extensively studied in the knee osteoarthritis model, and depending on the preparation, the solution is commonly injected weekly for 3–5 weeks for knee osteoarthritis. Although HA is cleared from the joint within a day, some commercially available HA has been modified to cross-link the molecules, which increases the molecular weight and lengthens the half-life [126]. In 2013, the American Academy of Orthopedic Surgeons (AAOS) revised the clinical practice guideline for knee osteoarthritis. Based on a meta-analysis of 14 studies, the AAOS no longer recommends HA intra-articular injection as a method of treatment for symptomatic osteoarthritis of the knee, even though a few studies did report statistically significant benefit [127].

HA is present in TMJ synovial fluid, and it is part of the lubrication system that decreases friction between the disc and the fossa during jaw movement [128]. HA appears to protect surface-active phospholipids, the main boundary lubricant, from degradation [129]. HA used in the TMJ, either as a stand-alone intra-articular injection or following arthrocentesis, has been studied by several investigators. Bertolami reported that a single HA injection resulted in statistically significant improvement in pain, joint movement, and joint noise in a group of patients with reducing displaced discs over 6 months [130]. Alpaslan studied outcomes following TMJ arthrocentesis with and without HA and showed that both techniques resulted in increased function and improvement in pain, although there was a trend toward greater improvement with HA [131]. Goiato's systematic review of TMJ intra-articular injections found that HA as well as corticosteroid and nonsteroidal anti-inflammatory drug injections can all be used with satisfactory results, citing that further study is needed to identify the best protocol, as well as to determine the best molecular weight of HA to use [132]. In contrast, Gencer et al. reported that their single-injection HA protocol did provide better pain relief scores when compared to a single intra-articular injection of betamethasone, tenoxicam, or saline at 6 weeks [133].

Guarda-Nardini and Manfredini's group has extensively reported on benefits of multiple cycles of arthrocentesis combined with hyaluronic acid injection, although many studies are case series or do not include a control group [134–138]. They also attempted to find predictive factors for early HA effectiveness in a group of patients with TMJ degeneration who underwent arthrocentesis combined with HA injection.

Improvement was only found in cases of high baseline pain levels and if the joint degeneration was unilateral, instead of bilateral [139]. A comparison between single- and multiple-session viscosupplementation protocols for TMJ disorders found that the five-session protocol of TMJ lavage plus HA injection was superior to any single-session protocol [140]. It should be noted that this group's studies frequently involve arthrocentesis lavage and not simply intra-articular injection without lavage. Therefore, the question of whether HA is effective in TMJ intracapsular disease in a single-injection or multiple-injection protocol, *without lavage*, has not been answered.

In conclusion, two chapter authors (RS, STC) have not noted benefit from a three-injection HA protocol *without lavage* and had many patients complaining of injection pain and joint tenderness following HA injection. Several patients declined to complete the three-injection protocol due to injection pain.

3.10.3 Opioids

As established above, intra-articular steroid injection works very well to reduce the effect of inflammatory mediators in patients with painful joints. Intra-articular injection has the advantage of decreasing systemic side effects while delivering benefit directly to the affected tissues. The analgesic efficacy of morphine within the knee joint has been shown to be superior to intramuscular morphine following arthroscopy, even though plasma concentrations are similar, adding to the evidence about morphine's peripheral benefit [141]. Increasingly, opioids given to the TMJ intra-articular space have been studied and found to produce excellent analgesic effects through their interaction with opioid receptors within the joint [142].

Sipahi's double-blind, placebo-controlled study of patients with internal derangement found that morphine 1 mg or tramadol 50 mg given intra-articularly following standard arthrocentesis gave significant pain relief, with morphine showing a longer-lived benefit [18]. Kunjur et al. reviewed 405 cases of TMJ arthrocentesis with intra-articular morphine infusion, showing success of 90% in the reduction in pain scores after 1 year [143]. The differences in benefit between morphine and other opioids may have something to do with the lipid solubility of the molecule, with lower lipid solubility associated with improved pain relief within the TMJ. Lower lipid solubility and the relative low exchange rate within the closed joint environment may also contribute to the surprisingly long-lasting benefit of intra-articular morphine.

In contrast to the benefit of morphine following arthrocentesis, the use of morphine as a single-dose intra-articular injection *without* arthrocentesis has been studied as well. List et al. found that as little as 0.1 mg of morphine yielded a reduced pain score at maximal opening [144]. Ziegler et al. demonstrated the best and longest lasting analgesic effect with 10 mg of morphine injected into the TMJ intra-articular space three times at an interval of 48 h between injections. Interestingly, even plain saline injection resulted in an increase in maximum incisal opening, but

this was attributed to the pumping effect of the intra-articular injection that widens the superior joint space and reverses negative intra-articular pressure [19].

Opioid receptors have anti-inflammatory effects, including mu and kappa receptors. Morphine acts primarily via activation of the mu opioid receptor. Mu receptor agonists have well-known side effects when they affect the central nervous system, including nausea, vomiting, sedation, respiratory depression, constipation, etc. New drugs are being developed that target peripherally acting kappa opioid agonists, which may have an improved side effect profile. Chicre-Alcantara's group demonstrated that local activation of kappa opioid receptors in the TMJ is associated with a decrease in plasma extravasation and neutrophil migration, which constitutes an anti-inflammatory effect. In addition, kappa receptor activation yields potent pain relief. As a result, research efforts to develop new drugs that preferentially or exclusively activate peripheral kappa opioid receptors is underway, holding the promise for the treatment of inflammatory TMJ pain syndromes [145].

In conclusion, the scientific evidence and authors' experience support the use of a minimum of 1 mg and probably as much as 10 mg of intra-articular morphine injection *without arthrocentesis* for long-lasting pain relief in patients with inflamed TMJ intra-articular conditions.

3.10.4 Amniotic Fluid

Amniotic fluid collected from normal, full-term pregnancies scheduled for C-section deliveries has been touted for its beneficial properties for clinical applications. Amniotic fluid, which protects and lubricates the contents (fetus) of a closed environment, has biologic properties that include anti-inflammatory and antimicrobial characteristics [146]. Fluid characteristics and chemistries are similar between donors, but differences are noted in hyaluronic acid levels and types and quantities of cytokine proteins [147]. Amniotic fluid can contain a small number of stem cells as well, which theoretically give the fluid regenerative properties [148, 149].

It is hypothesized that the cushioning action that amniotic fluid provides to the fetus, similar to the cushioning action of synovial fluid, can be applied to joints. Cryopreserved amniotic fluid in intra-articular joint injections has been studied in osteoarthritic pain in the knee, as an alternative to hyaluronic acid injection. Amniotic fluid-treated patients had impressive improvements in pain at 30 and 90 days in a single-arm, prospective study [150]. Amniotic suspension allograft, which has cryopreserved particulated human amnion and amniotic fluid-derived cells, was studied in a small cohort of patients with symptomatic knee osteoarthritis and was shown to produce improvements in pain, symptoms, and quality of life, among other parameters [151].

In light of the promising results achieved with amniotic fluid, a group of 15 patients with painful TMJ degenerative joint disease or internal derangement were given a one-time intra-articular injection of 1.0 cm^3 amniotic fluid in a prospective, open-label pilot study. Of the 15 participants, 14 reported reduction in pain and demonstrated an increase in maximum opening [152]. The results of the study

support the hypothesis that amniotic fluid is an effective treatment alternative for painful TMD, with the potential to decrease opioid medication use and improve quality of life, and should be studied in a formal clinical trial.

3.11 Botulinum Toxin (BTX-A)

3.11.1 Clinical History

The history of BTX-A dates back to the discovery and description of the food-borne illness, botulism, a potentially fatal neuroparalytic disease. Botulism was initially described by Justinus Kerner, a German physician, when the consumption of sausages (in Latin, botulus means "sausage") resulted in many deaths in South Western Germany between 1817 and 1822 [153]. During this time, it was postulated that the deaths were due to a toxin within the contaminated ham. At the end of the nineteenth century, microbiologist, Emile Pierre van Ermengem, identified what was responsible for producing the deadly toxin, *Clostridium botulinum*, an anaerobic bacteria [154]. Snipe and Sommer later identified and purified the toxin, BTX-A, in 1928 [155]. Since its discovery, additional research has led to the identification of seven types of botulinum toxin (A–G) with types A, B, E, and F causing human botulism [156].

Approximately 70–100 new cases of botulism are reported in the United States each year, of which 65% are infant botulism [157]. Following infection with BTX-A, most commonly through consumption of infected foods, symptoms usually present within 18–36 h and can occur in as few as 6 h of infection [158]. Infection causes muscle paralysis affecting the ability to speak, swallow, and move one's eyes. If not treated promptly, botulism can affect the muscles of respiration and result in respiratory arrest and death.

The mechanism of how BTX-A inhibits muscle contraction at the neuromuscular junction was first reported in 1949, by Burgen [159]. Since the elucidation of its mechanism of action, there has been tremendous improvement in the treatment of botulism. In addition, much research has been done with regard to using BTX-A as a therapeutic agent. In 1822, Kerner proposed that smaller doses of BTX-A can be used to treat diseases that result from hyperactivity of the autonomic nervous system [160]. Yet it was not until 1980 that Alan B. Scott, a San Francisco ophthalmologist, first reported a therapeutic usage of BTX-A for the treatment of strabismus. In his report, BTX-A was injected into the extraocular muscles of 67 monkeys with strabismus. He observed complete paralysis following injection of BTX, and correction of strabismus lasted 4–5 days and gradually diminished depending on the dosage used [161]. Subsequently, in 1989, the FDA approved Allergan's Biological License Application for two BTX-A clinical products for treating dystonia complications: blepharospasm, strabismus, and seventh cranial nerve disorders [162].

The first cosmetic application of BTX-A was reported in 1992, by Binder, who treated patients with headache disorders with concomitant hyperfunctional facial lines or other dystonias and observed a significant reduction in headache symptoms

[163]. Since then, the FDA has approved BTX-A for the treatment of cervical dystonia, chronic migraines, detrusor overactivity, upper limb spasticity, and primary axillary hyperhidrosis. Other applications of BTX-A include treatment of nystagmus, hemifacial spasms, dystonias, and chronic pain.

3.11.2 Structure of Botulinum Toxin

Botulinum toxins A–G share a similar core structure but are diverse in their serology and antigenicity [164]. For example, the structure of BTX-A is 900 kDa, of which 750 kDa are accessory proteins and the remaining 150 kDa encompasses the toxic moiety (Fig. 3.1) [165]. The accessory proteins may include dimers of L-complex components and help to protect the core. The toxic moiety is composed of two polypeptide chains that are inactive when bound together, one heavy chain (100 kDa) and one light chain (50 kDa) that are covalently bound via a disulfide bond. Once the toxin enters the cytosol of a neuron, the reducing capacity of the cytosolic environment cleaves the disulfide bond and produces the active individual chains [166].

The heavy chain consists of two domains that include a C-terminal domain (–COOH) and an N-terminal domain ($–NH_2$). The C-terminal domain is the membrane acceptor-binding domain (Hc) and is responsible for binding the toxin to the cell surface. When bound to the cell surface, the N-terminus helps translocate the light chain across the endosomal membrane. The zinc-dependent light chain is the main toxic agent and has enzymatic properties. When bound to zinc, the light chain-zinc complex cleaves peptide bonds that make up the soluble

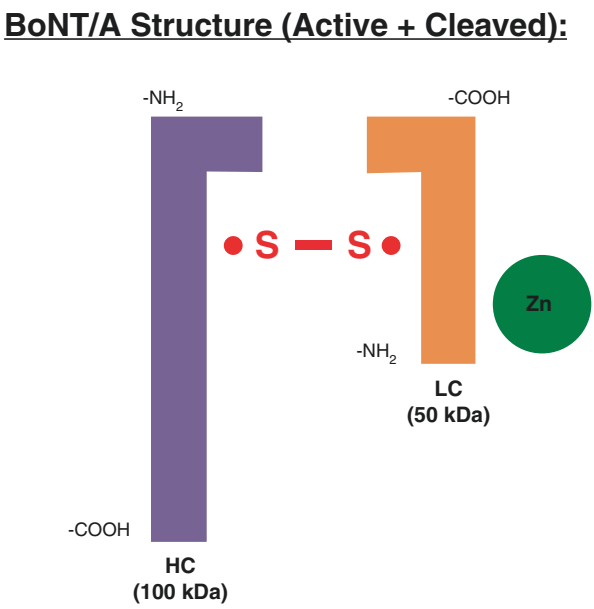

Fig. 3.1 Structure of BTX-A

N-ethylmaleimide-sensitive fusion attachment protein receptor (SNARE) [167]. Botulinum toxins A–G each cleave a specific peptide bond within SNARE [168].

3.11.3 Peripheral Mechanisms

The peripheral mechanism of action of BTX-A is generally well understood. The overall process begins with entry of botulinum toxin into the neuromuscular junction (NMJ) and inhibition of the release of peripheral neurotransmitters. In a normally functioning cholinergic nerve ending, the presynaptic cell contains vesicles of acetylcholine (Ach). Neuronal stimulation leads to a cascade of events that ultimately result in the fusion of Ach-containing vesicles with the nerve membrane. Fusion is made possible by the SNARE group of proteins described above. Following fusion, Ach is released into the synaptic cleft of the NMJ. Binding of Ach to Ach-receptors activates a cascade event that results in muscle contraction.

The effect of BTX-A is initiated by the dissociation of the core complex from its surrounding accessory proteins and movement of the core complex toward the nerve ending within the extracellular space. When in close proximity, the C-terminal domain of BTX-A heavy chain (HC) binds with high affinity to a receptor on the presynaptic cholinergic end allowing receptor-mediated endocytosis of BTX-A (Fig. 3.2) [169]. The specificity of binding to the presynaptic cholinergic receptor is

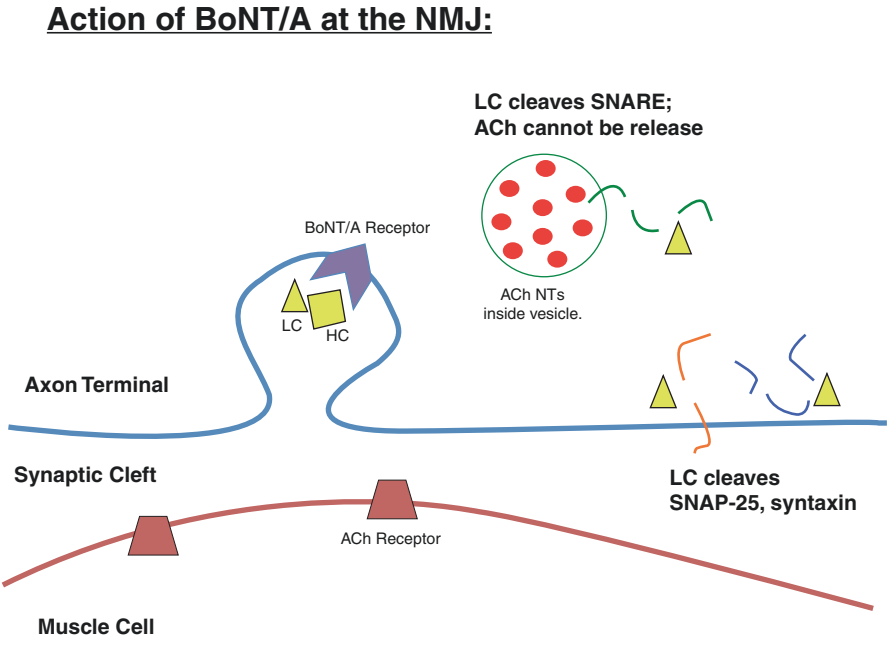

Fig. 3.2 Receptor-mediated endocytosis of BTX-A

in part due to the presence of gangliosides on the presynaptic receptor. After binding, BTX-A travels within vesicles down the presynaptic cell where the light chain (LC) is released into the cytoplasm. Once released, the light chain refolds into a soluble zinc-dependent metalloprotease [170].

When bound to zinc, the activated light chain cleaves the t-SNARE protein, SNAP-25 (synaptosomal-associated protein), which inhibits the release of Ach into the synaptic cleft preventing muscle contraction. Since the muscle does not contract, it instead relaxes leading to chemical denervation and recovery of new dendrites. Interestingly, it has been found that BTX-A cleaves less than 10% of the SNAP-25 protein despite the essentially full paralysis of the affected muscle. Other studies have proposed that the SNARE complex is completely destroyed in the presence of BTX-A [171]. In adult mice studies, the light chain of BTX-A was shown to remain stable between 120 and 180 days in vivo and approximately 80 days in vitro [172]. This stability is responsible for the long duration of action for BTX-A. Of the seven types of botulinum toxin, BTX-A was shown to be most resistant to degradation [173].

It has been postulated that the antinociceptive properties of BTX-A are due to the modulation of calcitonin gene-related peptide (CGRP) and substance P. In sensory neurons, the release of these neuropeptide neurotransmitters are inhibited by BTX-A, which inhibits nociception and causes a change in the blood supply or vascular function [174]. This process also occurs at SNAP-25 and inhibits the sensitization of pain. Welch demonstrated that BTX-A inhibited the release of substance P in a primary culture of embryonic dorsal root ganglia (DRG) in an animal model [175]. BTX-A has also been shown to inhibit the release of norepinephrine and inhibit its downstream effects. Nerve conduction velocity has also been shown to be affected. Moreno-Lopéz studied the effects on cranial nerves within the peripheral nervous system in a feline model, and it was observed that after injection of high doses of BTX-A, nerve conduction velocity was reduced by 21% within the abducens motor neurons [176].

3.11.4 Author's Technique

Onabotulinumtoxin A ("Botox") or incobotulinumtoxin A ("Xeomin") is utilized in this technique. The distinct formulations are reported to be dose-equivalent using standard unit dosing. The author's unpublished experience is that while incobotulinum toxin A appears to have a faster onset, the therapeutic effect may be several weeks shorter in duration compared to onabotulinumtoxin A for myofascial pain of the muscles of mastication. Three locations in the masseter muscle and two locations of the anterior ½ of the temporalis muscle are identified for injection. Each injected site receives 10 units of onabotulinumtoxin A ("Botox"). Thus, each masseter muscle receives 30 units and each temporalis muscle receives 20 units of Botox. Patients are given aftercare instructions including remaining upright for 3 h, avoidance of rubbing the injection sites, and adherence to a non-chewy diet for 1 day (Fig. 3.3).

Botox injection to Muscles of Mastication Author's technique
Mark and prep skin
• Alcohol wipe prep
Dilute Botox to 25 units/cm^3 with normal saline
• Gentle handling of vial; **do not** shake the vial to prevent breakage of the BTX-A chains
Bilateral injection of muscles of mastication
• 27 gauge needle
• 2–3 sites per muscle as clinically indicated, without use of local anesthesia. Separate injection sites by ~2 cm.
• 30 units per masseter muscle
• 20 units per temporalis muscle
Post-injection instructions
• No rubbing of injected sites for at least 24 h
• Non-chewy diet
• Remain upright for 3 h

The well-known myorelaxant effect of BTX-A is thought to be primarily responsible for the pain reductions achieved in TMD patients following injection [177, 178]. The temporary partial paralysis acts to relieve the chronic muscle overload in the muscles of mastication leading to decreased feedback from persistent fiber contraction, improved blood flow, and decreased release of proinflammatory mediators with less sensitization and activation of nociceptive neurons [179–181]. However, it has been our clinical observation that patients can complain of pain returning in as few as 3–4 months post-Botox injection, a time point at which the muscles, based on clinical exam, have not yet

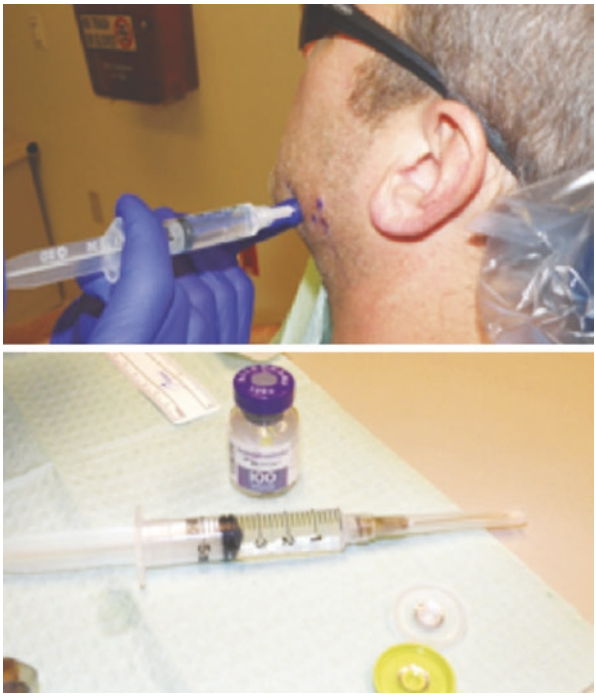

Fig. 3.3 Botox is diluted to 100 units/4 cm^3 saline and is injected into the masseter muscle (left). A 5 cm^3 syringe and 27 or 30 gauge needle are used (right)

fully recovered. This partial muscle recovery has been noted in the literature and supports our clinical observations. It has been shown that muscles do not fully recover their original volume and contractile ability when measured at 6 or 12 months postinjection as determined both physiologically and histologically in animals [182] and in humans via magnetic resonance imaging [183], respectively. Interestingly, when the patients with early recurrent pain return to clinic and are administered a repeat injection of BTX-A, it is again effective in reducing pain despite the incomplete muscle recovery. One explanation for this is that BTX-A is simply affecting the new neuronal sprouts that form at the neuromuscular junction prior to the regeneration of the original atrophied components [184]. Another explanation is that there are alternate pathways for BTX-A to exert its antinociceptive effects separate from its action on the muscle at the NMJ [185]. Preclinical studies have shown that BTX-A can be delivered to the trigeminal ganglion by retrograde axonal transport via sensory neurons [186], where it may act to inhibit the activation of pain-mediating second-order neurons and to block the stimulated release of calcitonin gene-related peptide (CGRP), glutamate, and substance P [185]. Interestingly, these are some of the same mediators that chronic pain and PTSD have in common as a possible shared pathophysiologic pathway.

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Part III

Dental Treatment of Temporomandibular Disorder

Occlusal Diagnosis and Treatment of TMD

4

Kazumi Ikeda

Abstract

Careful evaluation of the occlusal scheme with an accurate diagnosis and treatment plan before orthodontically modifying the occlusion is a critical step in solving patients' TMD problems; however, it is underappreciated and underutilized. To begin, the goals of occlusal treatment must be clearly defined before starting treatment. They should not be ambiguous, and clear goals are needed for each area affected with treatment. Changing the occlusion will influence the following areas:

- Functional occlusion
- TMJ
- Three-dimensional position of the mandible
- Periodontal tissues
- Dental and facial esthetics

4.1 Defining Treatment Goals

Goals of occlusal treatment must be clearly defined before starting treatment. They should not be ambiguous, and clear goals are needed for each area affected with treatment. Changing the occlusion will influence the following areas:

4.1.1 Functional Occlusion

It is important that the occlusion is in harmony with the TMJ to reduce any tension of the masticatory muscles [1–3]. This can be achieved by providing even bilateral

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contacts of the posterior dentition at mouth closure, establishing proper canine guidance in lateral movement and anterior guidance in protrusive movement [4, 5], and eliminating posterior occlusal interferences during mandibular movements [6].

4.1.2 TMJ

Efforts should be made to prevent disc displacement (DD) from occurring. If DD is already present, attempt to arrest its progression. It is also important to prevent any loosening of the collateral and lateral ligaments attaching the disc to the condyle and pay attention to the masticatory muscles and neuromuscular system that are closely related with the TMJ [7].

4.1.3 Three-Dimensional Position of the Mandible

Changing the occlusion will alter the position of the mandible. Conversely, mandibular position may be improved by changing the occlusion. Chin position should be harmonized with the occlusion while creating a balance between the nose, lips, and chin for facial esthetics, in order to avoid excessive lip and perioral muscle strain during mandibular closure [8, 9].

4.1.4 Periodontal Tissues

Close attention must be paid to the position of the connective tissue attachment and the thickness of the gingiva [10], as well as to the thickness and form of the alveolar bone.

4.1.5 Dental and Facial Esthetics

Andrews describes the characteristics of the ideal alignment of the dentition in his six keys to normal occlusion [11]. The teeth that are optimally aligned should also be in harmony with the TMJ [12]. Furthermore, it must be kept in mind that the act of restoring occlusal function also affects facial esthetics.

4.2 TMJ as a Treatment Goal

4.2.1 So-Called CR

1. The topic of centric relation (CR) comes up whenever the subject of mandibular position is discussed in dentistry. By definition, CR is the most anterior-superior position of the condyle in the glenoid fossa, where the condyle is in close contact

with the articular eminence with the disc interposed in between. The posterior band is at the 12 o'clock position when the disc is "on" (Figs. 4.1 and 4.2) [13].

2. The "disc-on" state is advantageous for TMJ function because the disc is in a position to help the condyle to move up and down the eminence smoothly. Sicher stated in his oral anatomy textbook that the condyles, discs, and eminentia are in close contact in all movements and at rest [14]. Nowadays this can be visualized on cine-mode MRI images (Fig. 4.3) [15]. With the availability of MRI for TMJ imaging in daily practice, it is possible to objectively assess the disc position and the condyle-eminence relationship.

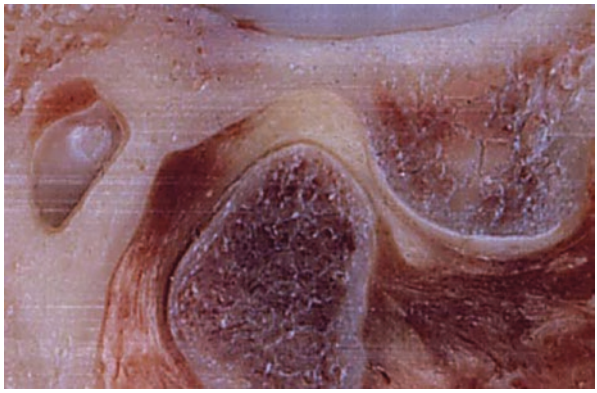

Fig. 4.1 Cryosection of TMJ. The articular disc is properly interposed between the condyle and the articular eminence. The optimum condylar position is most anterosuperior in the fossa (Courtesy, Dr. Isberg, Sweden)

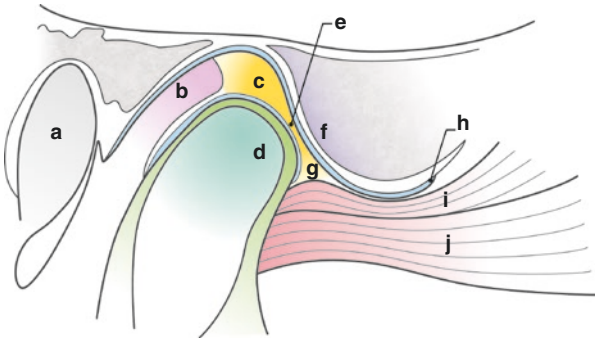

Fig. 4.2 The normal TMJ: (*a*) auditory canal, (*b*) posterior disc attachment, (*c*) posterior band, (*d*) condylar head of mandible, (*e*) intermediate zone, (*f*) articular eminence, (*g*) anterior band, (*h*) anterior limit of capsular insertion, (*i*) superior head of lateral pterygoid muscle, (*j*) inferior head of lateral pterygoid muscle

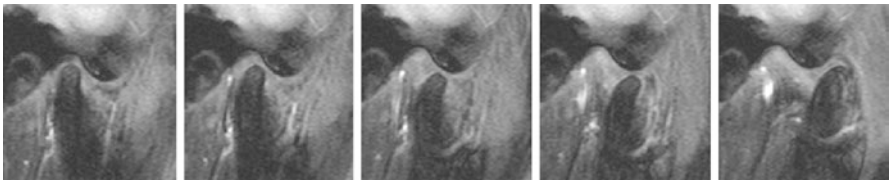

Fig. 4.3 Cine-mode MRI of a normal TMJ shows the disc and condylar movements during mouth opening. As the condyle smoothly rotates and translates, the functional surfaces of the condyle and articular eminence maintain an intimate relationship with the intermediate zone of the disc interposed between them

3. The disc is known to have the architecture suitable for its function [16, 17]. When the occlusion is in harmony with the TMJ, the masticatory muscles can function without excessive strain and serve to maintain the form and elasticity of the disc.
4. With the introduction of MRI in clinical practice, we are now beginning to understand that patients having this well-designed "disc-on" state in both the right and left joints are a small minority, rather than a majority [18–20], among children and adolescents, as well as adults [21, 22].

4.2.2 Is Disc Displacement (DD) a Normal Variation?

DD, which does not fit the definition of CR, is so common that some people regard it as a normal variation. However, it is not a normal variation. Even incipient DD is associated with an inflammatory condition called joint effusion, which can be visualized on T2-weighted MRI images [23]. Incipient DD can be detected on cone beam computed tomography (CBCT) as well because it is accompanied with condylar displacement in the fossa [24]. Naturally, DD affects mandibular function (Fig. 4.4). DD in growing children can have major impact on their mandibular growth as demonstrated in animal experiments and studies using cephalograms of growing patients with DD [25–30]. Thus, DD is not a normal variation as suggested by some academicians.

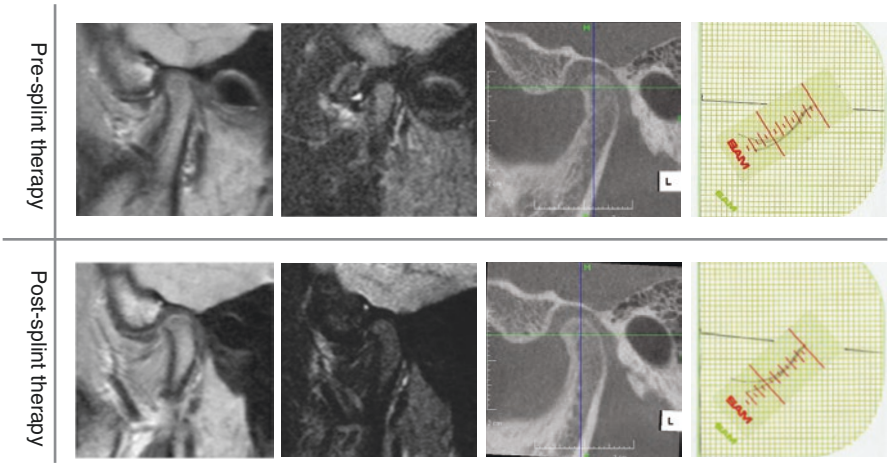

Fig. 4.4 The pre- and post-splint therapy images from MRI (proton-density-weighted and T2), CBCT, and Axiography recording the jaw movements of a patient's left TMJ. Stabilization-type splint was used approximately 15 h daily for 8 months. MRI shows an improved disc position after splint therapy, and complete reduction of joint effusion is evident from the T2 images. CBCT images illustrate a slightly more forward position of condyle post-splint therapy. With improved disc position, joint movements also improved. The reverse curve at the onset of mouth opening on Axiography that signifies anterior disc displacement is reduced after splint therapy

4.3 Normal TMJ

4.3.1 Normal Disc Position

The articular disc is comprised of very dense collagen fibers. Its thinner central part called the intermediate zone primarily supports mandibular function [31]. It is interposed between the anterosuperior articular surface of the condyle and the articular eminence. The thickest part of the disc called the posterior band is at the 12 o'clock position of the fossa on a sagittal image of a normal TMJ. The disc is firmly attached to the medial and lateral poles of the condyle via the collateral ligaments without mediolateral displacement on a coronal image of a normal TMJ (Fig. 4.5) [32]. According to Hansson et al. [16], in the coronal view, the disc is slightly thickened on the medial aspect, and slightly thinner laterally. Laterally, the disc attaches directly below the lateral pole, which in adults are slightly lower in position to the medial attachment.

4.3.2 Normal Condylar Position Depicted by Imaging

The condyle tends to assume a particular position when the disc is "on." It is therefore necessary to know the normal position of the condyle for diagnostic purposes.

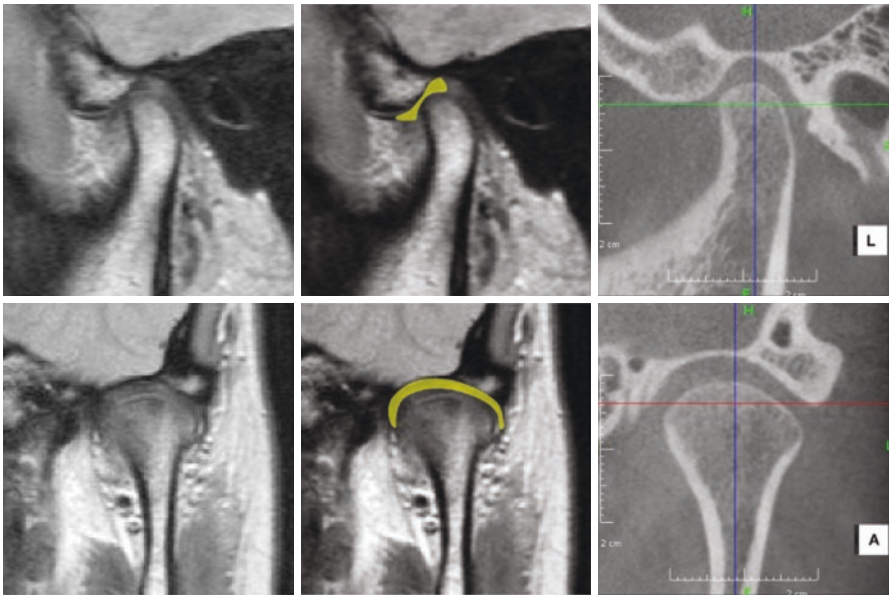

Fig. 4.5 MRI and CBCT of a normal TMJ in sagittal and coronal planes

Weinberg postulated in the 1970s that normal condylar position is concentric and that the condyle is displaced posteriorly in patients with joint problems [33]. He used transcranial radiographs. Pullinger reported in the 1980s that normal condylar position was variable (anterior 29.7%, central 43.2%, posterior 27.1%) in an asymptomatic population [34]. Tomography was employed as a technique for displaying a cross section through an object. Why was normal condylar position variable in his study? One reason is the way he acquired the images. He used a fixed angle of projection, instead of a patient-specific long axis of the condyle. With a projection of the joint at a fixed angle, the position of the condyle in the fossa appears different depending on the inclination of its long axis, which varies from patient to patient. Better would have been cross-sectional images of the condyle obtained perpendicular to its long axis using submentovertex (SMV) radiographs [35]. Another reason has to do with sample selection. Without the use of MRI, it might have been difficult to exclude asymptomatic DD from the study sample. Condylar position changes even with mild asymptomatic DD [24]. The sample for statistical analysis might have included joints that were not normal with incipient DD. Another limitation of his study was the use of central images only. It is important to examine the lateral and medial imaging planes as well. Condylar position in the fossa changes in three planes of space as disc position changes three-dimensionally. This necessitates the evaluation of condylar position in both sagittal and coronal planes.

With the advent of CBCT, condylar position can be assessed at a relatively low radiation dose and a high precision with a pixel size of 0.1–0.2 mm [36, 37]. Ikeda et al. acquired three-dimensional measurements of normal condylar position in a carefully selected "disc-on" sample based on MRI findings [38, 39]. Beyond the absence of symptoms, additional strict inclusion criteria were used: "disc-on" status confirmed on MRI, absence of joint effusion on T2-weighted MRI, no contributory history, no abnormal finding in chairside examination, and limited ligament looseness with $\leqq$1 mm of immediate side shift on Axiography recording. The three-dimensional positional data for normal condylar position relative to the fossa is shown in Fig. 4.6.

4.3.3 Abnormal Disc Position

As noted above, only a few adults or children have TMJs with normal disc position. It must be kept in mind that DD occurs in three planes of space. When disc position is evaluated on MRI, medial, central and lateral images in the sagittal plane are utilized to identify the extent of displacement. In addition, a coronal image is evaluated to detect any medial or lateral shift. While complex displacement patterns are less frequent in children than adults, advanced DD may be present in adolescents [22]. The same assessment procedure should be used for both children and adults (Fig. 4.7).

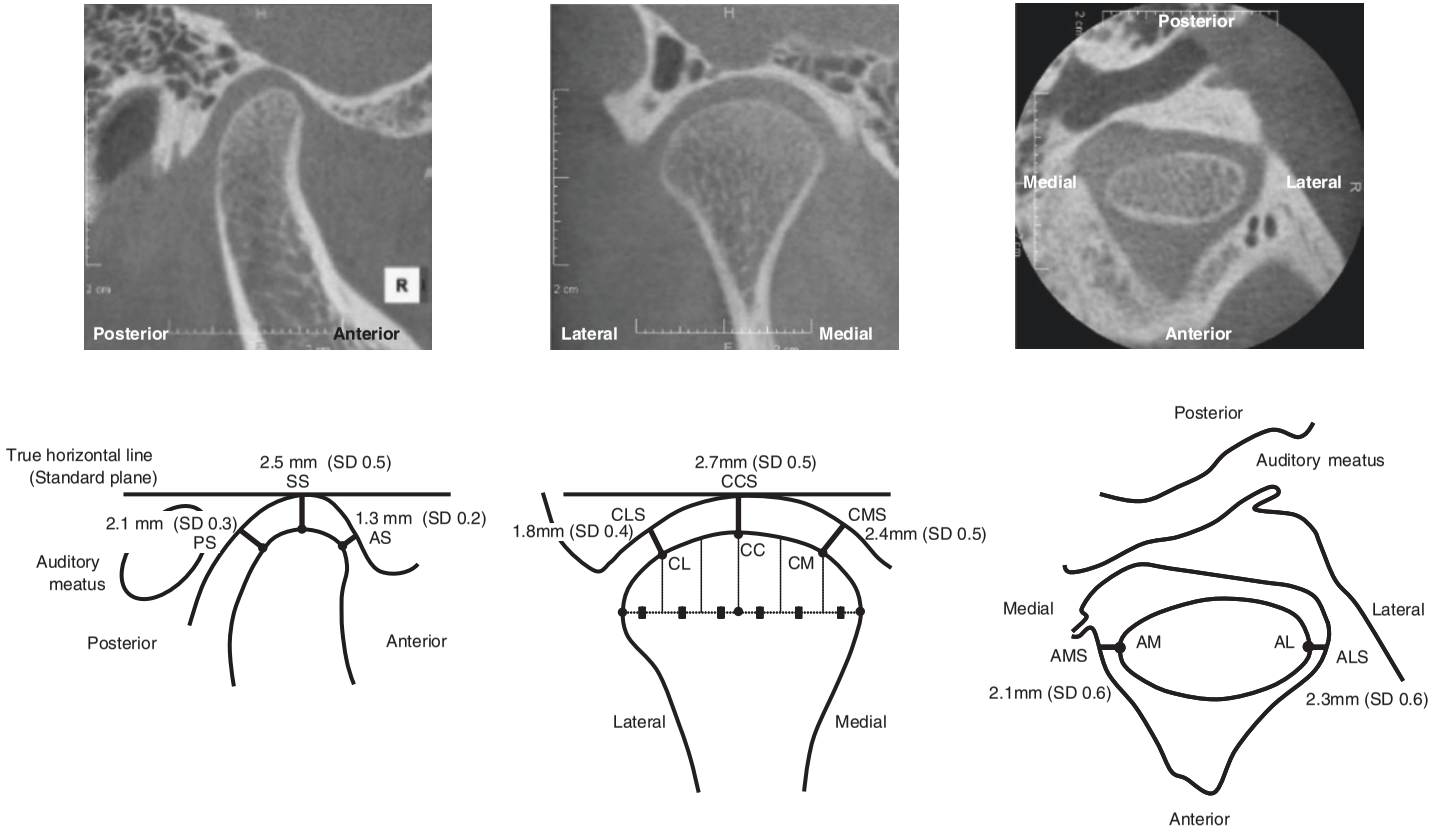

Fig. 4.6 Optimum condylar position in sagittal, coronal, and horizontal and space data of each section with illustrations. *AC-AS* anterior space, *SC-SS* superior space, *PC-PS* posterior space, *CM-MS* medial space, *CC-CS* central space, *CL-LS* lateral space, *AL-LS* lateral space, *AM-MS* medial space

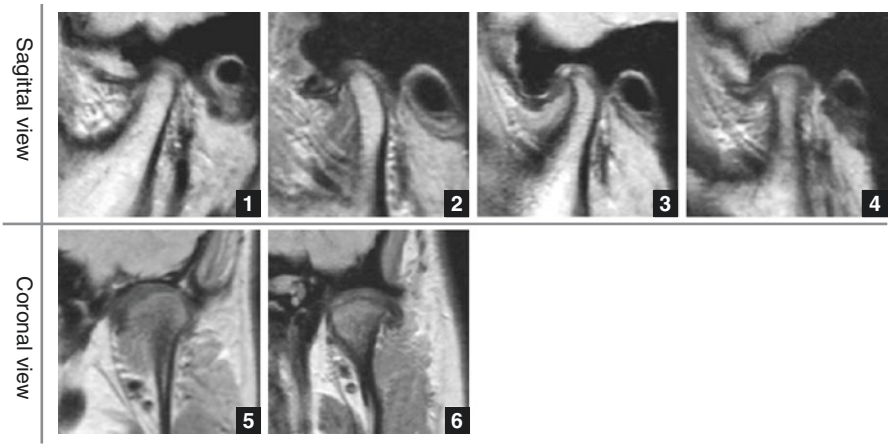

Fig. 4.7 MRI of various disc displacements. *1–4* show displacement in the sagittal plane and *5–6* in the coronal plane. *1* is an incipient displacement; *2* is slightly more progressed partial displacement; *3* is complete displacement; *4* is a chronic complete displacement with altered disc morphology, making it unlikely to reduce; *5* is a left TMJ showing a medial displacement; and *6* is a lateral displacement

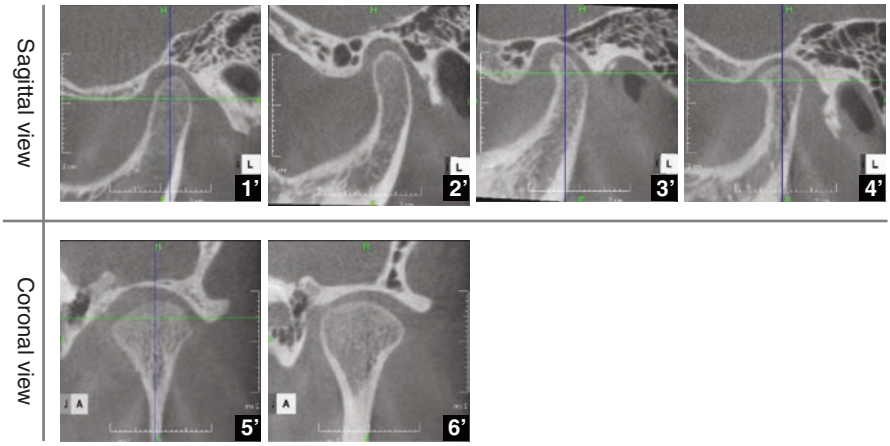

Fig. 4.8 CBCT images of the same joints in Fig. 4.7. Even with a slight anterior displacement, the condyle moves posteriorly, and as the DD worsens, the condyle moves up and posteriorly. With advanced DDs, there are morphological changes to the condyle and the eminence. *1'* is an incipient displacement; *2'* is slightly more progressed partial displacement; *3'* is complete displacement; *4'* is a chronic complete displacement with altered disc morphology, making it unlikely to reduce; *5'* is a left TMJ showing a medial displacement; and *6'* is a lateral displacement

4.3.4 Effects of DD on Condylar Position in the Fossa

Condylar position in the fossa is extremely sensitive to DD. Minimal DD cannot be disregarded as a normal variation. The eminence and condyle also undergo morphological changes as DD progresses (Fig. 4.8) [40].

4.3.5 Research on Changes in Mandibular Position Caused by DD

Experience with MRI and CBCT images reveals that there are patterns of change in condylar position in the fossa specific for each type of DD. Ikeda and Kawamura conducted a study to determine if the direction and extent of DD can be estimated from specific patterns of change in condylar position on CBCT images [24].

The line connecting the 12 o'clock position of the fossa and the most inferior point of the eminence was divided into three equal parts. Changes in joint spaces were measured in a group with the posterior band of the disc located within the superior third of the eminence (partial DD) and a group with the posterior band of the disc displaced down to the inferior third of the eminence (total DD with reduction) as shown in Fig. 4.9 (see the article for details) [24]. Mediolateral displacement of the disc and the condyle was measured in the coronal plane (Fig. 4.10).

4.4 Establishing the Stages of Disc Displacement

Staging helps diagnose and plan the treatment of DD. Rasmussen divided temporomandibular arthropathies into six phases in 1980 [41]. Hatcher graphically illustrated the progression of degenerative joint disease (DJD) based on CT images [42]. Today's advanced imaging technology can provide objective information on the absence or presence of DD, extent of DD progression, and its effects on clinical symptoms and bone morphology. Clinicians need a staging system developed based on such information as the basis for diagnosis and treatment planning. Without information on stages of DD progression and approaches to arresting its progression, they would not know how to treat the problem or assess treatment results. Staging also facilitates communication among clinicians. A staging system that provides a simple and clear image of each stage without too much detail is needed.

4.4.1 DD Is Three-Dimensional

Because DD occurs in three planes of space, anterior displacement should be assessed on medial, central, and lateral areas of the condyle. The disc may shift sideways as well. A coronal image in the center of the condyle should also be used in conjunction with a couple of images anterior and posterior to the central image to check sideways disc displacement medially or laterally before determining the stage of DD (Fig. 4.11).

4.4.2 Where Is the 12 o'Clock Position on MRI?

Evaluation of the extent of DD requires the identification of the 12 o'clock position on MRI images. Without this reference, there is no way of differentiating incipient DD from normal disc position. Where is this reference position on

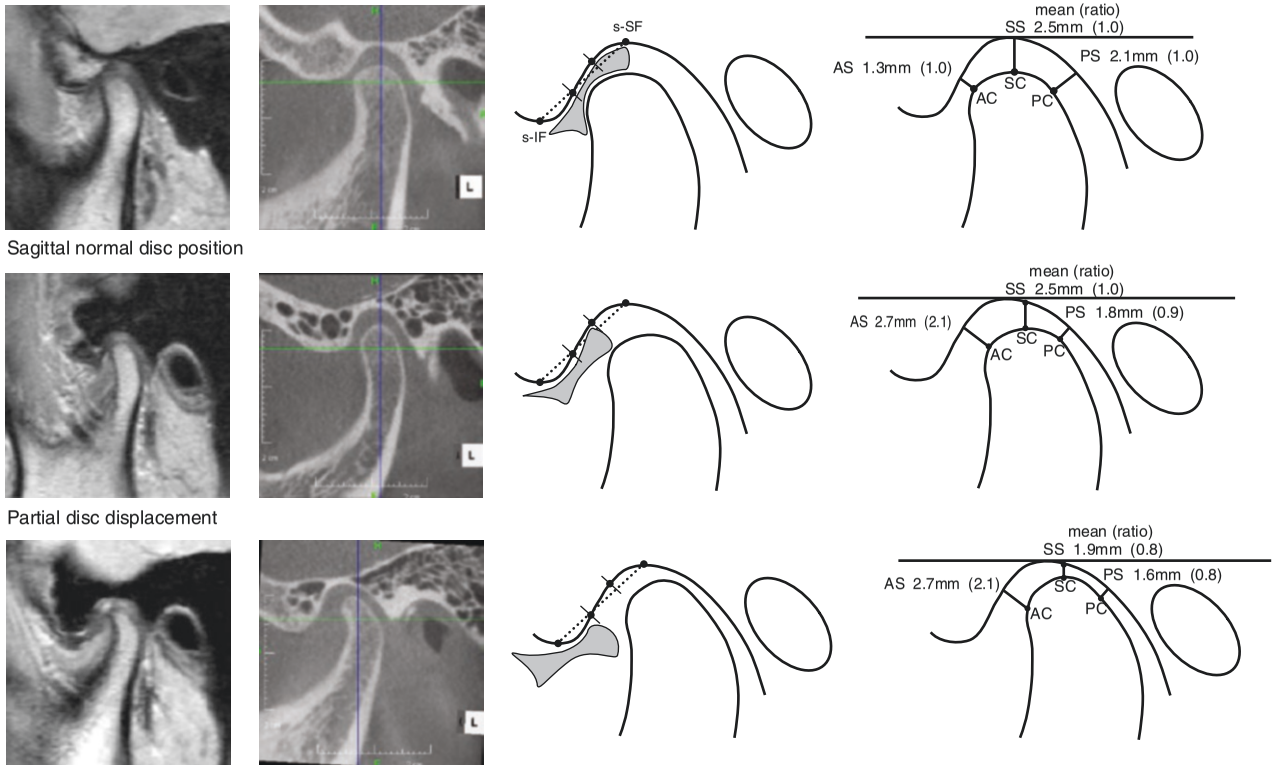

Fig. 4.9 Condylar position in the fossa changes with various degrees of disc displacement, making it possible to estimate the presence and severity of DD based on the spatial relationship of the condyle and the fossa. In the sagittal plane, partial DD is accompanied by a posterior, rather than a vertical, condylar position change. With total DD, the condyle shifts slightly posteriorly and dramatically more superiorly, and the superior joint space is significantly reduced

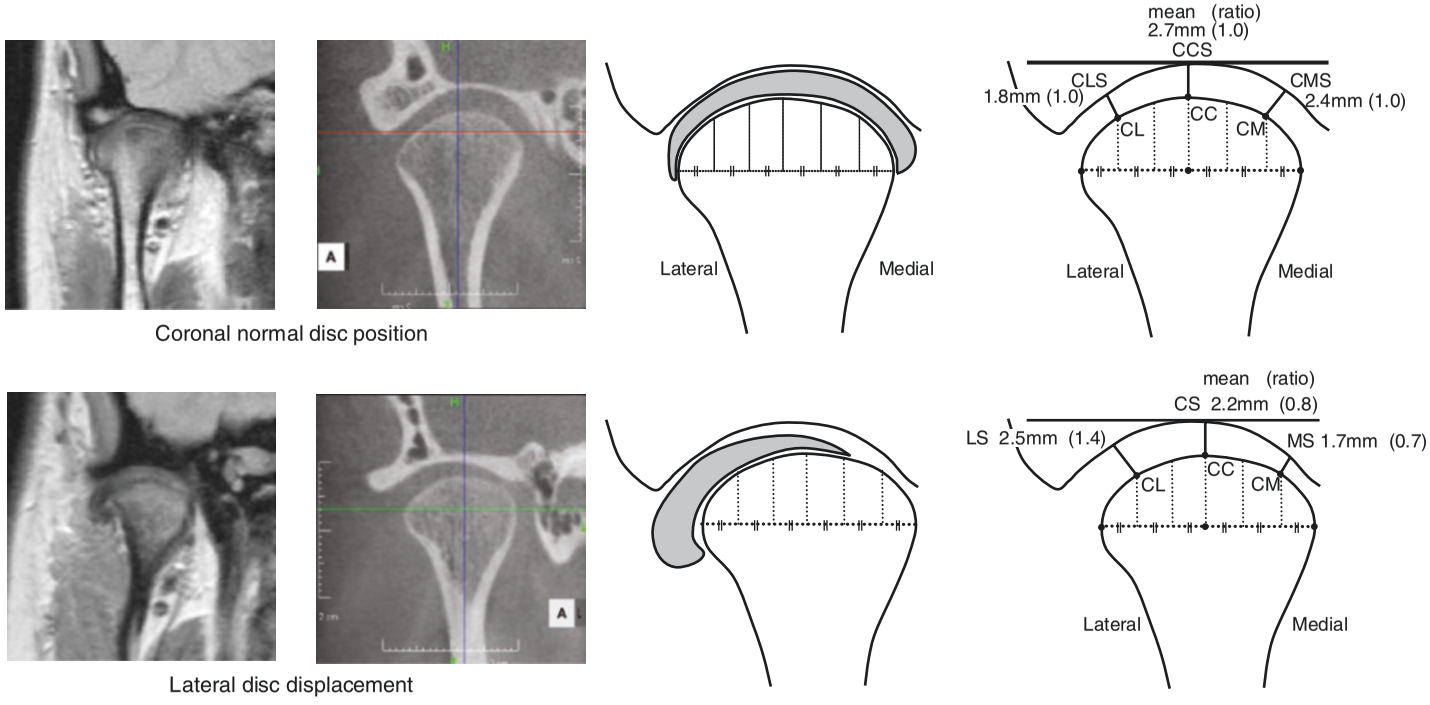

Fig. 4.10 In the coronal plane, the medial and lateral DD can cause the condyle to shift in the reverse direction. From the medial/lateral change in the condylar position, the disc position can be estimated. In a normal joint, the disc is slightly thicker medially, and consequently the joint space is the largest at the medial aspect, while the lateral aspect is the smallest. As the disc shifts laterally, the lateral joint space visibly increases, and the medial space decreases

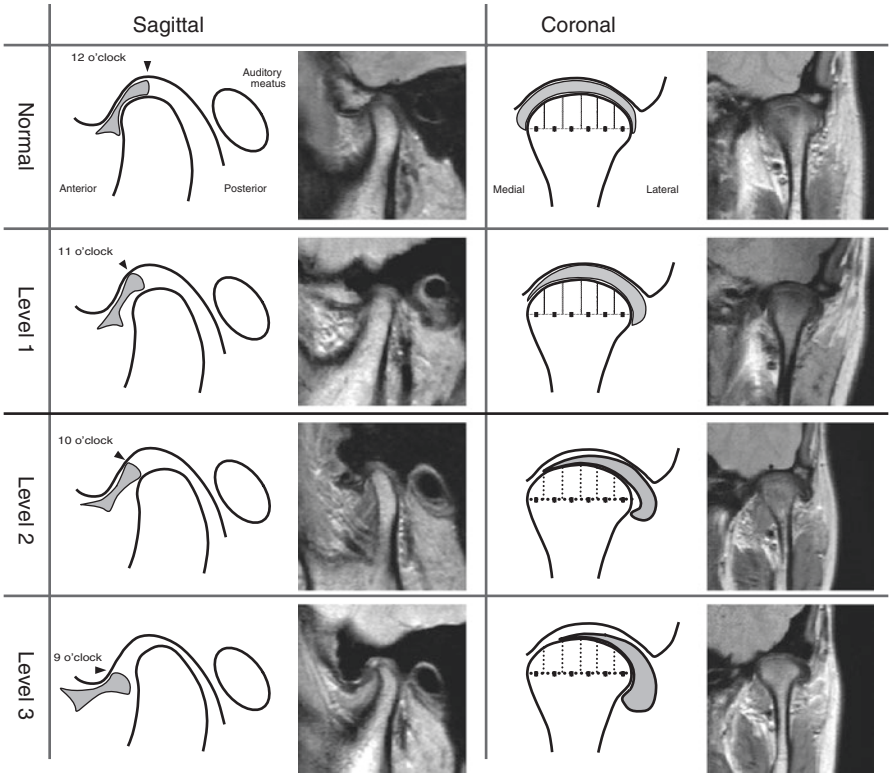

Fig. 4.11 In the sagittal cut, the position of posterior band is assessed: the 12 o'clock position as normal; the 11 o'clock position as Level 1; the 10 o'clock position as Level 2; the 9 o'clock position, which is inferior to the functional area of the condyle, as Level 3. In the coronal cut, sideways displacement of the disc is used. Level 1, Level 2, and Level 3 represent 1/3, 1/2, and 2/3 or greater sideways displacement, respectively

MRI? Ikeda conducted a study to locate the 12 o'clock position on MRI images using CBCT images acquired in the same period of time (Fig. 4.12) [43]. A true horizontal line (THL) can be drawn on CBCT images, which are acquired in the natural head position. MRI and CBCT images were superimposed by matching their magnification. In a search for an anatomical reference line approximating a line parallel to THL, it was shown that a line connecting the center of the auditory canal and the most inferior point of the articular eminence was most appropriate [43].

4.4.3 DD Findings from the Four Images Are Put Together for Staging with the Following Criteria

1. Stage 1: The disc is slightly out of normal position toward the 11 o'clock position in the sagittal plane and slightly displaced sideways and/or thickened in the

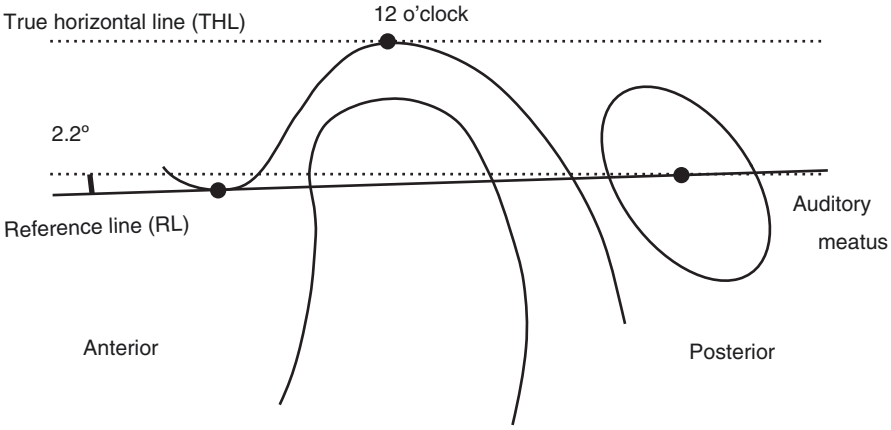

Fig. 4.12 Establishing the 12 o'clock position on MRI. A study revealed that the true horizontal line on CBCT is approximately in line with an anatomical reference line on the MRI that passes through the center of the auditory meatus and the most inferior point of the eminence. THL can be established directly on an MRI by rotating the reference line 2.2° clockwise. The 12 o'clock position can be located by moving THL up parallel to itself until it touches the deepest point on the glenoid fossa

coronal plane. The patient is typically asymptomatic. DD is so incipient that the signs may be easily missed without careful chairside examination. None of the four MRI images (three sagittal, one coronal) show Level 2 DD. Only Level 1 DD is seen in this stage.

2. Stage 2: Stage 2 DD is found in children as well. DD in this stage is prevalent in adolescents, an age group suitable for orthodontic treatment. A part of the disc remains between the articular eminence and the functional surface of the condyle. This stage encompasses such a wide spectrum of conditions between the incipient Stage 1 and the more advanced Stage 3 that it is divided into three substages for clinical purposes.
   (a) Stage 2A: Of the three sagittal images, two still show Level 1 DD. Sideways displacement is Level 2 or lower in the coronal plane.
   (b) Stage 2B: Of the three sagittal images, only one shows Level 1 DD. Sideways displacement is Level 2 or lower in the coronal plane.
   (c) Stage 2C: All three sagittal images show at least Level 2 DD; none show Level 1. Sideways displacement in the coronal plane is Level 2 or lower.
3. Stage 3: While Stage 1 and 2 represent early to moderate DDs, more advanced and established DDs are classified as Stage 3. The posterior band of the disc is at the 9 o'clock position or inferior to the functional surface of the condyle in all three sagittal cuts. Sideways displacement of Levels 1–3 may be present in the coronal plane. The disc is still reducible on opening. Great care must be taken when performing a modality of treatment that alters an individual's occlusion as it may cause the state of jaw locking mentioned below. It is therefore necessary

to stabilize mandibular position before proceeding to occlusal therapy. DD in very early Stage 3 may be reversed to Stage 2 with joint stabilization using a splint.

4. Stage 4: In this most advanced stage of DD, the disc is no longer interposed between the functional surfaces of the eminence and the condyle, getting in the way of opening and lateral movements of the jaw. Jaw locking is a common finding in this stage. Attempts to force the jaw open cause joint pain in the acute phase. The amount of mouth opening increases with time as the condition becomes chronic. Though rare, there are pre-orthodontic teenage patients already in Stage 4 who have never experienced any difficulty or recognized any problem in the joints. When problems are suspected in totally asymptomatic patients, a detailed history including trauma should be taken and studied carefully in conjunction with findings from chairside examination and TMJ imaging, so that a precise diagnosis can be made.

4.5 Progression of DD

4.5.1 Continuum of Change in DD

David Hatcher, an American maxillofacial radiologist, has proposed a continuum of degenerative changes on CT images in relation to clicking, limitation of opening, and pain summarized in a schematic diagram [42]. He maintains that TMJ disorders with different symptoms are not separate entities, but represent a continuum of hard and soft tissue changes. Figure 4.13 illustrates the course of DD progression with corresponding CBCT images of the joints of the same patient and typical symptoms associated with these changes. It is reasonable to accept the concept of a continuum of DD progression, bony changes on CBCT, and symptoms attributable to those changes. The four CBCT images representing Stage 4 DD vary in the time of DD onset and the pattern of bony changes from simple morphological changes to more complex ones.

4.5.2 Relationship of DD with Signs and Symptoms

- Stage 1 and early Stage 2 DDs are asymptomatic or minimally symptomatic. Patients rarely complain of symptoms. Only careful chairside examination including patient history and palpation and imaging such as MRI will reveal these conditions.
- Distinct clicking sounds occur in late Stage 2 to Stage 3. A loud clicking sound is heard when the condyle moves onto the posterior band of the displaced disc. There is only a softer sound when the disc slides off the condyle at the end of closing. The patient may complain of symptoms without the clinician's inquiry. When the disc is displaced sideways medially, palpation may not detect the displacement. More advanced Stage 3 DD may be associated with thickening of the

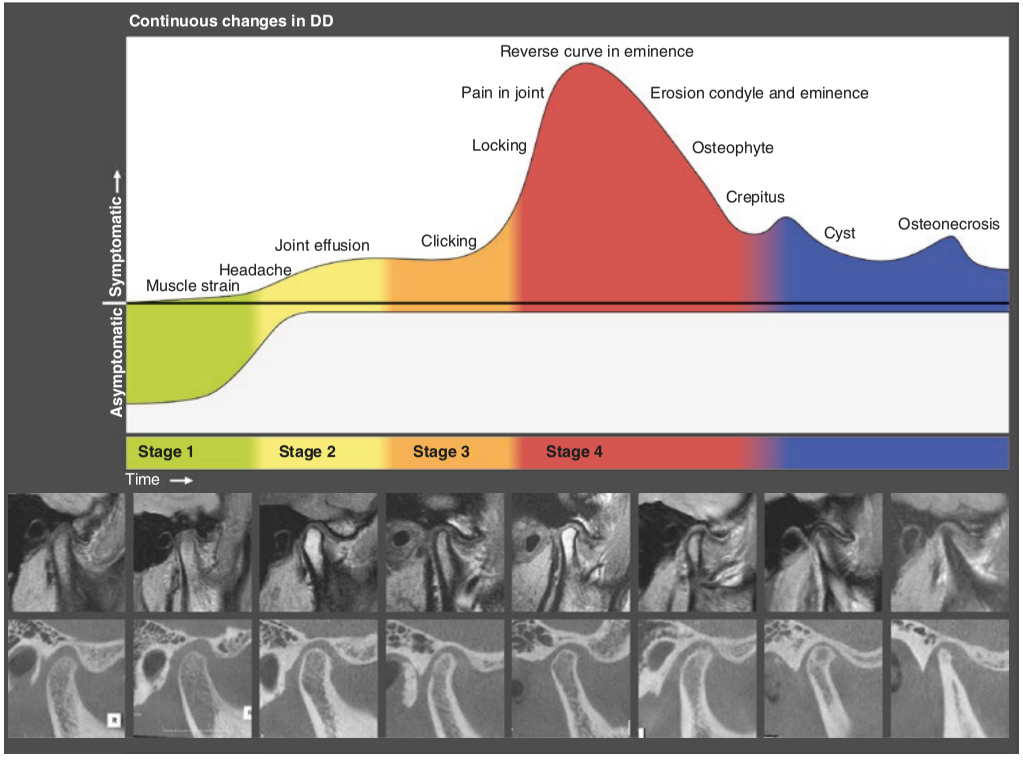

Fig. 4.13 There are minimal clinical symptoms associated with Stage 1 DD. Only when compared with normal joint images, the incipient displacement can be discerned. The change in condylar position is also noted, demonstrating that slight displacement of the disc can cause a clear condylar position change. As the DD progresses, the condylar position continues to shift, and morphological changes in the hard tissues are observed. Furthermore erosion, sclerosis, osteophyte, cyst formation, and necrosis can be seen in the advanced stage

posterior band of the disc. The patient may experience tentative lock or inability to open the mouth particularly upon awakening in the morning or after many hours of work. Although he manages to open the mouth by shifting the jaw slightly sideways, he often feels discomfort in the joint area.

- The acute phase of Stage 4 DD is often accompanied by the sudden limitation of opening. The patient experiences "locking" of the jaw and feels pain when attempting to open the mouth beyond a limited comfortable opening. The clinician can easily diagnose the condition by checking the amount of opening at chairside. The amount of mouth opening may also be reduced to 35–40 mm in the presence of masticatory muscle strain due to parafunction. When the jaw locks, the maximum mouth opening ranges from 10 to 20-some mm.
- In the chronic phase of Stage 4 DD, trismus is often resolved, and the patient may have forgotten about the experience. However, he may report his previous experience with symptoms such as clicking, pain, and tentative or complete jaw locking if the clinician asks about them during the initial history taking. Crepitus or grating sounds may be detected by chairside palpation. As pseudo-disc formation progresses in the posterior disc attachment area after a long period of the chronic phase, crepitus is often indiscernible and pain is decreased. The maximum mouth opening may be near normal. Some patients show almost normal sagittal movement tracings when their mandibular movements are recorded. However, their immediate side shift is close to or even greater than 2 mm, indicating that the ligaments are loose.

4.5.3 Trial of DD Mapping on CT Images

MRI is not always available, while CBCT is widely used in clinical practice. Because of the importance of knowing disc status for diagnosis and treatment planning, methods to visualize DD using CBCT image data have been sought (Fig. 4.14). The space between the condyle and fossa varies with changes in condylar position due to altered disc position. Three-dimensional changes in joint space can be displayed in different colors on the surfaces of the condyle and fossa to indicate the position of the posterior band and thereby represent disc position. Dr. Tadanori Furuya (private practice in Hokkaido, Japan) has shown the techniques of reflecting DD on CBCT images,a demonstration of color mapping. It has the following advantages:

- DD can be recognized three-dimensionally on CT images.
- The relationship between changes in bone morphology and DD can be visualized on CT images.
- Disc position can be estimated using the color mapping technique on CBCT when MRI is not available or feasible.

Efforts should be made to prevent DD from progressing by getting a good grasp of the disc status before DD reaches an advanced stage in young patients. It is important to know the extent of its progression prior to the start of any treatment that alters the existing occlusion, so that measures can be taken to improve the condition

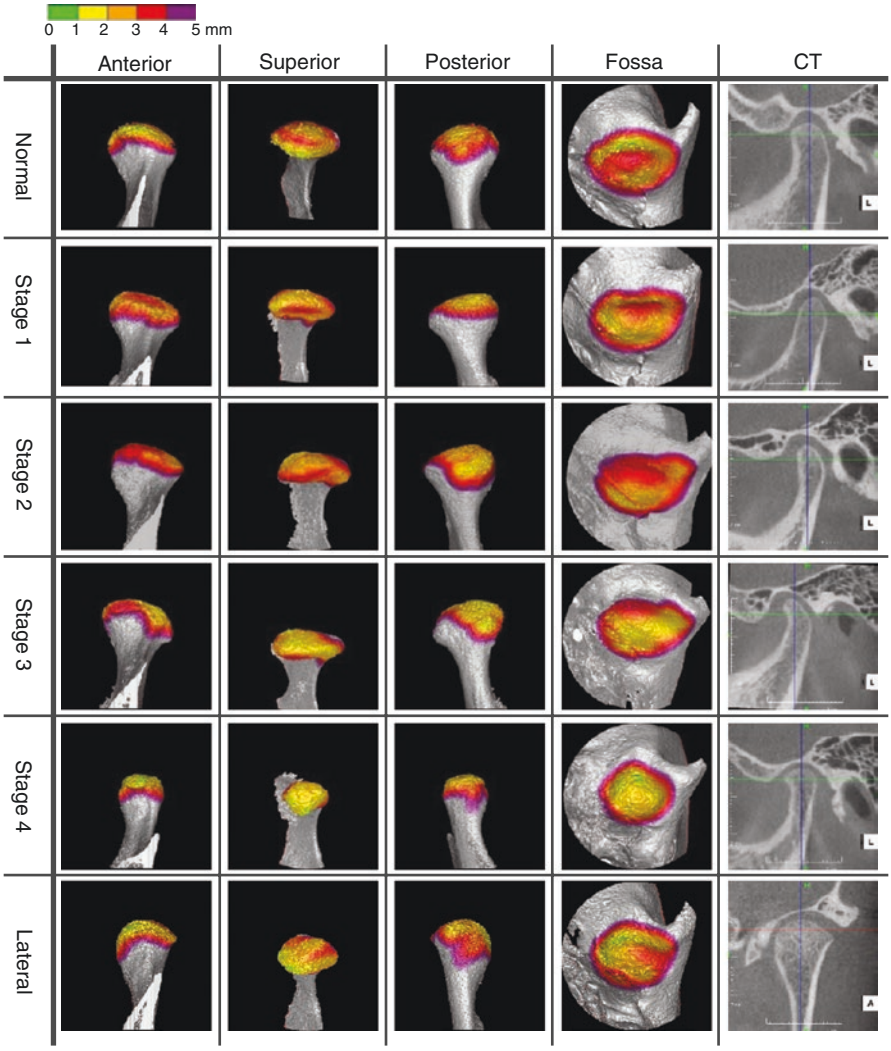

Fig. 4.14 Color-mapped joint spaces of adolescent patients with various DDs. The superior view of a normal condyle has a red band, which corresponds to the posterior band of the disc. The anterior green zone is the intermediate zone of the disc. The red band or posterior band moves anteriorly to a lower position as DD progresses from Stage 1 to Stage 2. The red band drops out of the functional surface of the condyle in Stages 3 and 4. Disc status can thus be estimated using this color mapping technique

and reduce the severity if possible. Although an MRI scan is required for a definitive diagnosis, color mapping of DD enables three-dimensional visualization of changes in the position of the posterior band with the progression of DD including sideways displacement. Interpretation of Stage 1 DD is relatively difficult even with MRI. The color mapping technique can depict subtle changes in the incipient stage of DD and

is therefore useful in considering ways to arrest or prevent its progression. For more advanced DD, the extent of DD progression can be estimated based on changes in condylar position and bone morphology observed on CBCT images in conjunction with TMJ history and chairside examination.

4.6 CR as a Goal

In patients seeking occlusal therapy, it is difficult to reconstruct the occlusion in the CR position defined in healthy joints with the "disc-on" status. DD is not just an adults' problem. Incipient DD is present in children. DD has already progressed to Stage 4 in some adolescents. Many adults seeking orthodontic treatment have advanced DD. Unfortunately the CR defined for normal "disc-on" joints cannot be used as a basis for the treatment of many patients. Because a great majority of patients undergoing occlusal therapy have DD, one must find a way to establish a stable foundation on which to build treatment for these patients. One needs to confirm how reliable a mandibular position we can obtain prior to definitive treatment.

4.7 Recognizing Changes in the TMJ in the Presence of DD

Studies using cryosections of the joints revealed that disc shape and its position can be changed in many individuals [32]. Arthrography has also played an important role in discovery of DD. Farrar noted the presence of DD in his clinical practice and published a way to treat the condition, spreading the notion of DD in dentistry [44]. However, DD has been recognized as a distinct clinical entity since the advent of MRI [45–47]. Researchers began to use MRI in the mid-1980s, and internal derangement in the TMJ has been visualized objectively in patients since then. The clinical application of imaging for visualization and diagnosis of DD therefore has a relatively short history. What are the changes observed on MRI images when DD is present?

1. The disc becomes deformed (Fig. 4.15).
2. Fibrosis occurs in the posterior attachment (Fig. 4.16).

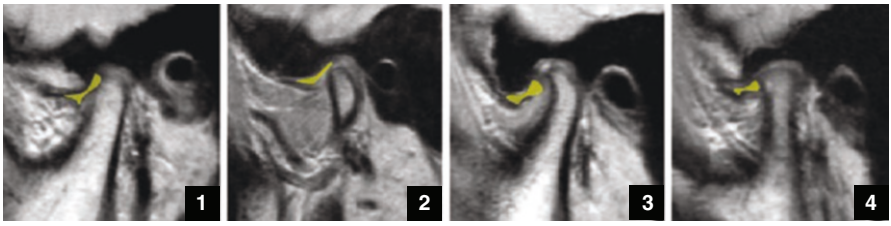

Fig. 4.15 (1) Elongation of the intermediate zone, (2) flattening of the posterior band, (3) hypertrophy of the posterior band, (4) reduction of the intermediate zone causing the anterior and posterior bands to come closer together

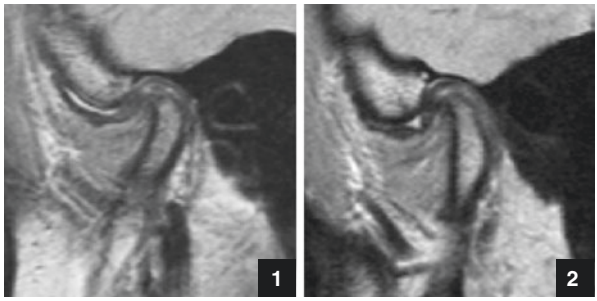

Fig. 4.16 (1) Fibrosis in posterior disc attachment, (2) pseudo-disc formation

3. Joint effusion is observed even in the presence of incipient DD (Fig. 4.17).
   Although synovial fluid is present in the upper and lower joint spaces, a small quantity of this fluid suffices for normal function as long as the articular surfaces are kept lubricated. When DD, even an incipient one, is present, T2-weighted MRI images show an increased amount of synovial fluid. This increased intra-articular fluid called joint effusion contains proinflammatory cytokines such as interleulin-1β (IL-1β), IL-6, IL-8, etc. DD is thus detrimental to the function and internal milieu of the TMJ [48]. Berteretche demonstrated that partial DD adversely affected the secondary cartilage in an animal experiment [26], suggesting that DD in growing children may negatively influence the secondary cartilage and condylar growth.
4. Bone hypertrophy and deformation occur in the condyle, eminence, and fossa (Fig. 4.18).
5. Changes and edema occur in the muscles attached to the disc and condyle (Fig. 4.19).
6. Ligament laxity can be identified with immediate side shift measurement. Measurements are made, while the clinician applies pressure to the gonial angle toward the contralateral posterior temporal area (Fig. 4.20). The amount of side shift during lateral jaw movement is generally increased with the pressure application than when the patient performs the movement on his own. Immediate side shift is measured when the jaw moves 4 mm laterally (Fig. 4.21). The amount of immediate side shift increases as ligament laxity increases. It is often greater than 2 mm in the chronic phase of Stage 4 DD (Fig. 4.22).
7. Changes in condylar movement paths (Fig. 4.23).
   Three movements in the sagittal plane (protrusive, lateral, and opening) are scribed as movements of the axis of rotation of the condyle [49].

4.8 Occlusion Observed in the Mouth

4.8.1 So-Called CO

Care must be taken when evaluating the patient's occlusion during the initial examination. Patients have a strong tendency to bring the teeth together by shifting the jaw to a position where the maxillary and mandibular teeth fit together best (Fig. 4.24). This is controlled on a subconscious level by the neuromuscular mechanism [7].

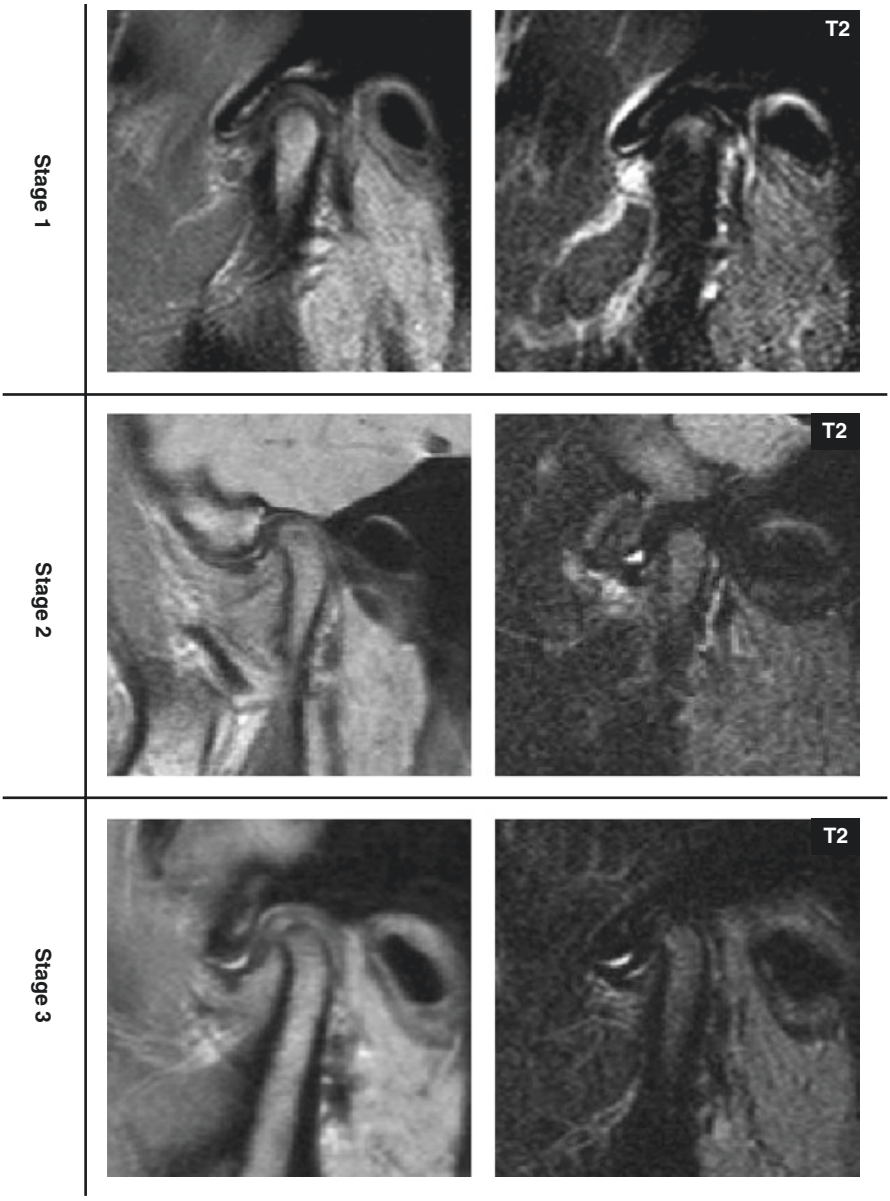

Fig. 4.17 Proton-density-weighted and T2-weighted MRI of DD in various stages. T2 MRI of Stages 1–3 DD illustrating the presence of joint effusion. Joint effusion can be seen even in the incipient stage of displacement

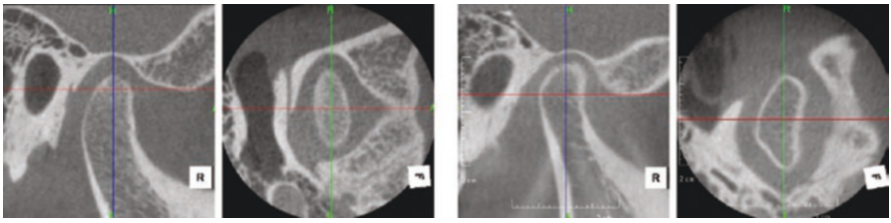

Fig. 4.18 Comparison between a normal TMJ and advanced DD. The smooth outline of the hard tissue surfaces has been altered due to the joint dysfunction caused by advanced DD. Localized sclerosis is also evident

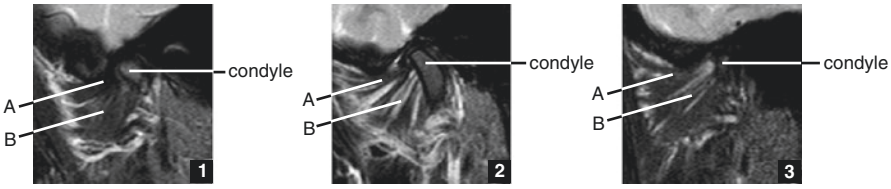

Fig. 4.19 T2 MRI showing (*A*) superior head of lateral pterygoid muscle and (*B*) inferior head of lateral pterygoid muscle. 1 is normal, 2 shows edema in the lateral pterygoid muscle, and 3 shows edema and disarrangement of muscle fibers

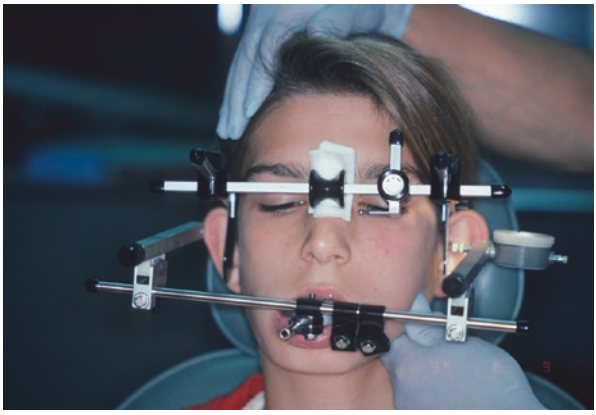

Fig. 4.20 Recording of an immediate side shift induced with the application of pressure to the left gonial angle toward the right temporalis

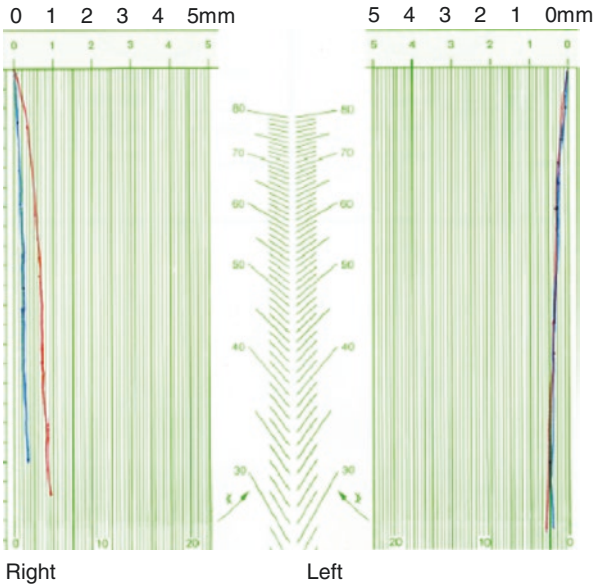

Fig. 4.21 Immediate side shifts in the coronal plane were measured for the right and left joints. Border movement is shown in red and unguided movement in blue. The size of immediate side shift is less than 0.5 mm, indicating minimal joint laxity

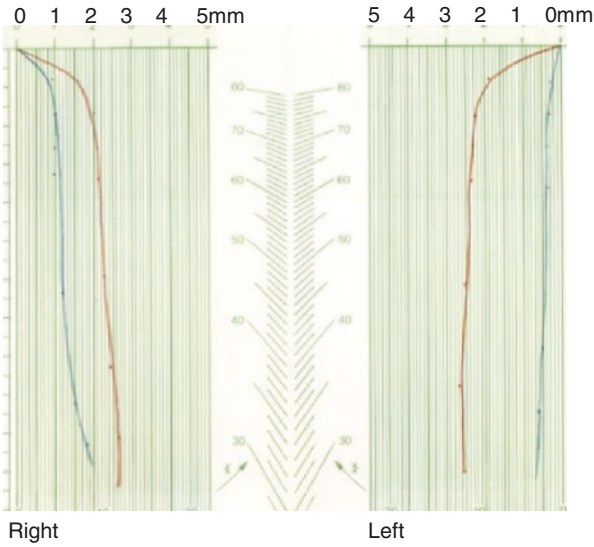

Fig. 4.22 This patient has loose joints with more than 2 mm of immediate side shift on both sides, indicative of Stage 4 DD

4.8.2 How to Find Occlusal Discrepancy During the Initial Examination

- Have the patient close on to a piece of wax and record his habitual occlusal position (so-called CO) (Fig. 4.25).
- Next, take an anterior CR wax record by guiding the jaw so that the patient can maintain 3 mm of space between the maxillary and mandibular posterior teeth without protruding the jaw. Chill the wax to harden it. Soften another piece of

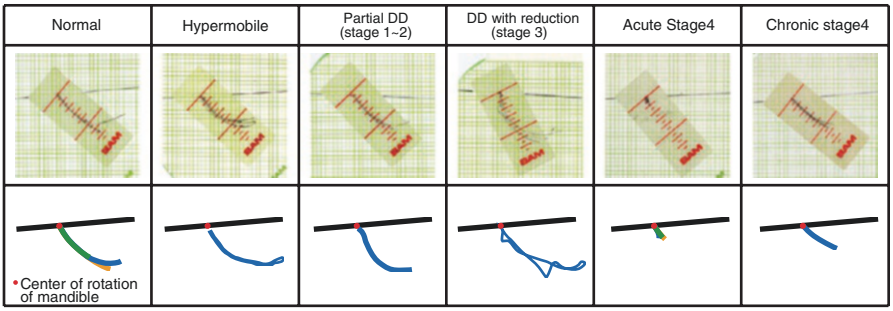

Fig. 4.23 The terminal hinge axis (red dot) is the starting point of its protrusive (green), opening (blue), and mediotrusive (orange) movement paths recorded in the sagittal plane. When the three lines don't superimpose for the first 8 mm, it's indicative of an unstable joint with DD and joint laxity. The reverse curve at the beginning of movement is indicative of incipient DD and may aid in confirmation of DD when combined with other records. The characteristic reverse curve associated with mandibular movement in early stages of DD is a sensitive indicator of DD. The reverse curve is formed when the condyle climbs over the posterior band of the displaced disc at the beginning of opening or at the end of closing

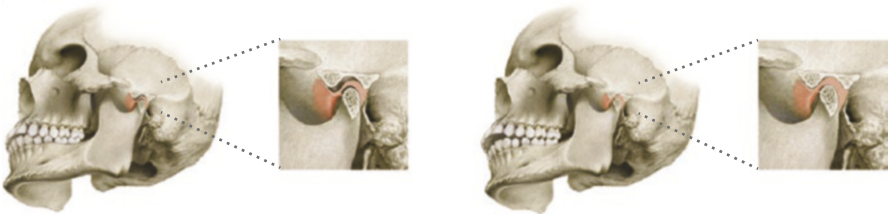

Fig. 4.24 The left illustrations show the teeth in maximum cuspation, but the condyles are not seated in the fossae. This is observed in many patients seeking orthodontic treatment. The right illustrations show the joints that have been stabilized, but the teeth no longer fit. This is a situation often encountered after splint therapy

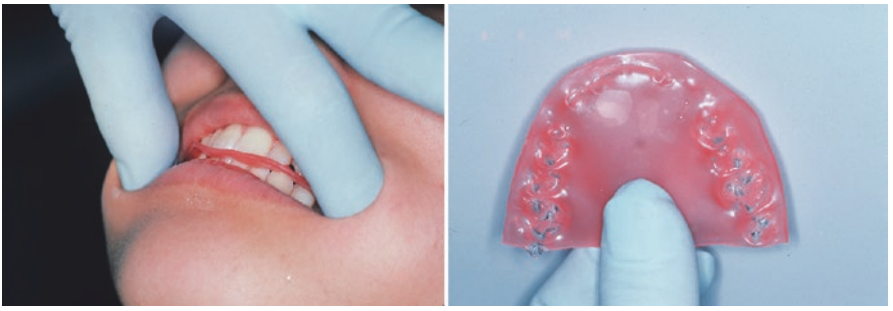

Fig. 4.25 CO occlusal registration. A thin but dimensionally stable piece of wax is used to record the patient's CO or habitual occlusal position. This is an important record used to measure condylar displacement with the condylar position indicator (CPI). The CO occlusal record is not used to mount models on an articulator

wax for the posterior teeth, and place it in the mouth together with the hardened anterior section. Take the posterior section of the "power centric" interocclusal registration by asking the patient to close into the soft posterior wax with the help of his masticatory muscle power (Fig. 4.26) [50].
- Mount models using the CR bite record.
- Measure the difference between CO and CR as discrepancies at the TMJ level using an instrument for tracking condylar positions such as CPI and MPI. These measurements show how far the condyle is displaced in the fossa in three planes of space from the initially captured position of the mandible (CR) to the habitual occlusal position of the maxillary and mandibular teeth (CO) (Figs. 4.27 and 4.28) [51].

4.8.3 Mounting the Initial Models in CR on an Articulator Uncovers the Patient's Premature Contacts That Cannot Be Detected in the Mouth

The above Sects. 4.8.1 and 4.8.2 are illustrated in an actual patient. Her molar relationship observed in the mouth is Angle Class I. However, her initial models mounted in CR reveals a full Class II molar relationship with a large overjet. Her CO-CR discrepancy measurements indicate that both of her condyles are displaced down and forward in the fossa when she brings her teeth together. CBCT images also show clearly that both condyles shift down and forward in the fossae (Figs. 4.29, 4.30, 4.31, 4.32, and 4.33).

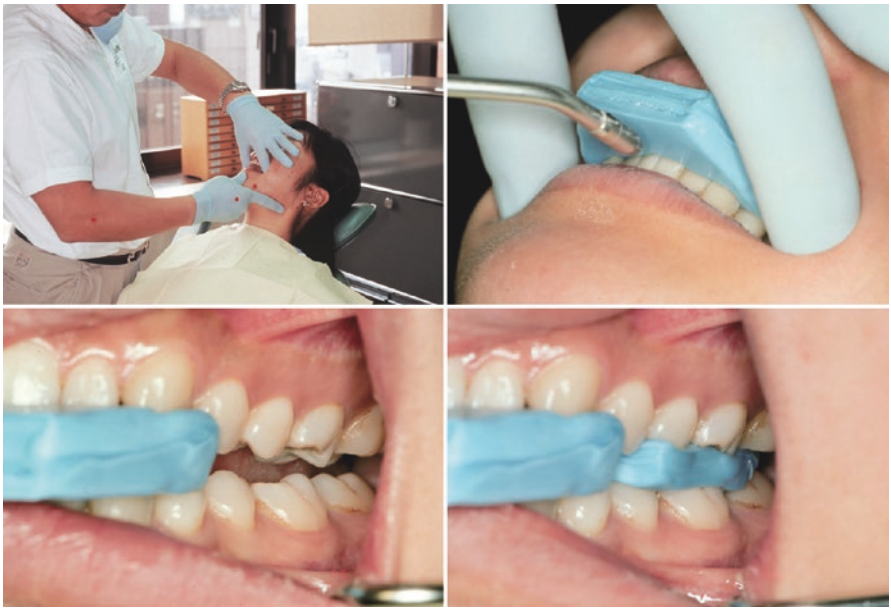

Fig. 4.26 CR occlusal registration. This technique is used to record the patient's initial CR. The initial CR should not be mistaken for the true CR, which is extremely difficult to capture at chair side during the initial examination. The purpose here is to obtain the best possible seating of the condyles in the fossae on that day. The patient's true CR can be registered only after the joints have been stabilized

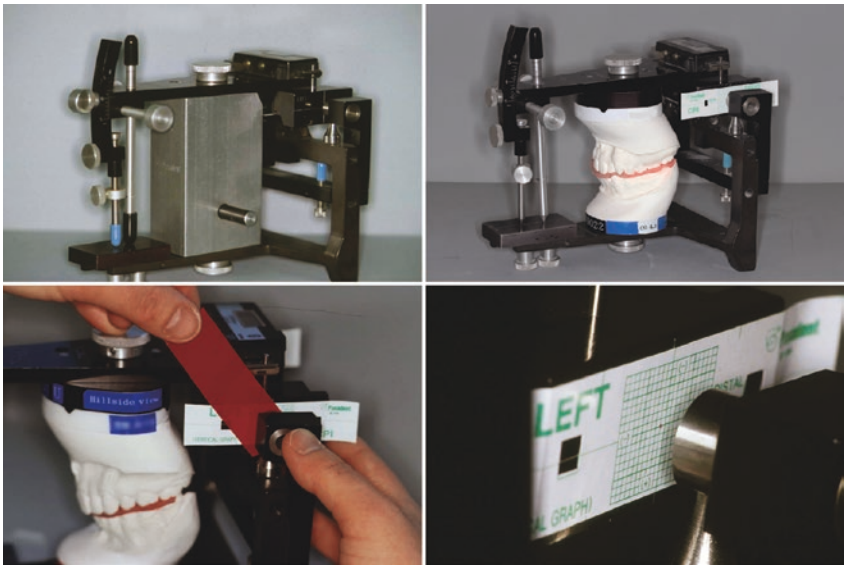

Fig. 4.27 CPI (condylar position indicator). CPI measures three-dimensional displacements of the condyles with the CO wax bite. This procedure is affected by the accuracy of the CR and CO wax bites, models, and their mounting. Thus, care must be taken in performing each of the preceding steps that requires a high level of accuracy, as well as in handling the CPI

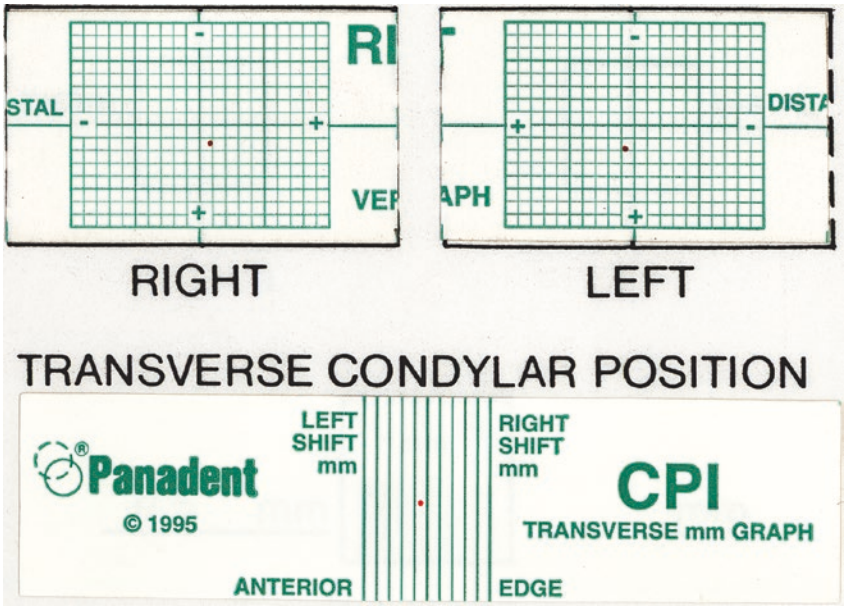

Fig. 4.28 CPI data. CPI data consists of three graphs, right and left sagittal graphs and transverse graph. The top two graphs are used to measure condylar displacements in the sagittal plane, which are shown in red. The origin of the coordinate represents the initial CR position. The red dot shows the CO position. The bottom record indicates condylar displacement in the frontal plane. A transverse discrepancy of greater than 0.5 mm raises a concern

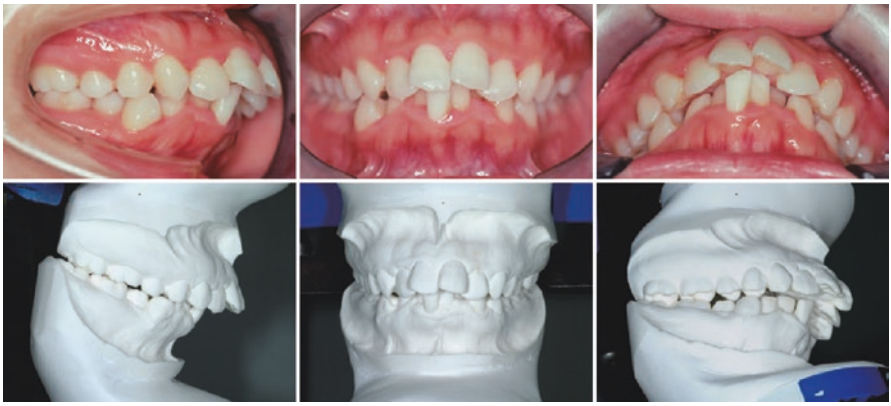

Fig. 4.29 21-year-old female, initial intraoral photos and mounted models based on the initial CR records. Patient's habit of posturing the mandible forward is evident

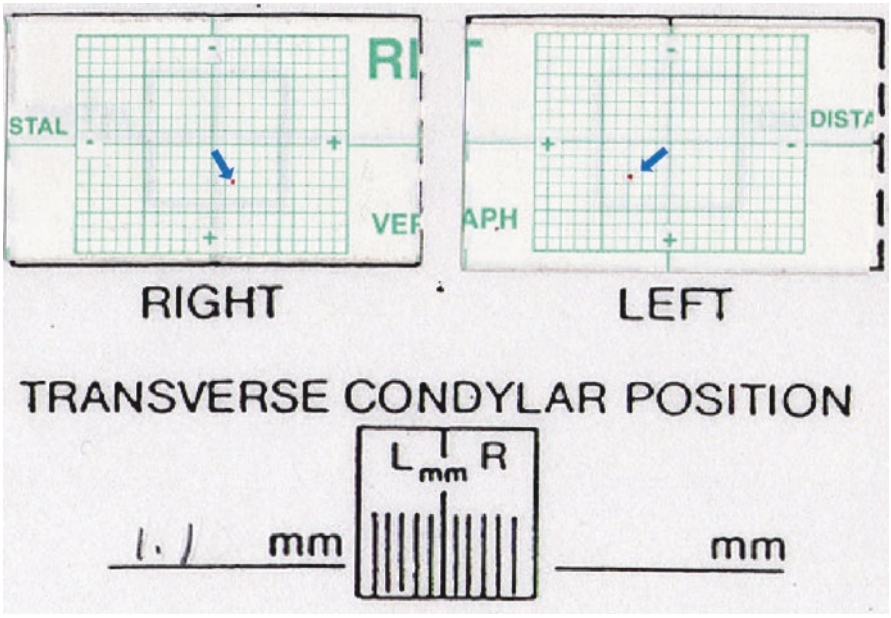

Fig. 4.30 CO-CR discrepancy at the right and left joint level measured using the CPI data. The red points are CO, and the central crossing is the initial CR position. The CPI data three-dimensionally measures the joint movement from CR to CO

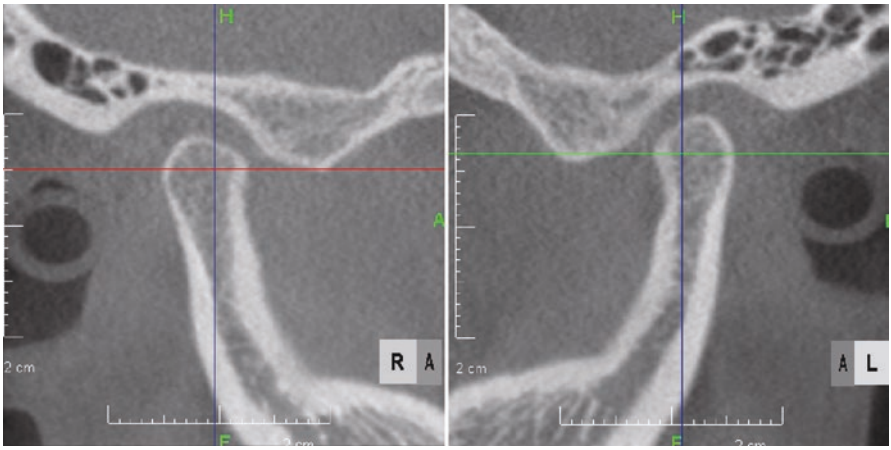

Fig. 4.31 Sagittal CBCT with the patient in CO. Both condyles are shifted anteriorly. The condylar head and the eminence have flattened, and they appear to function primarily by anteroposterior movements rather than rotation

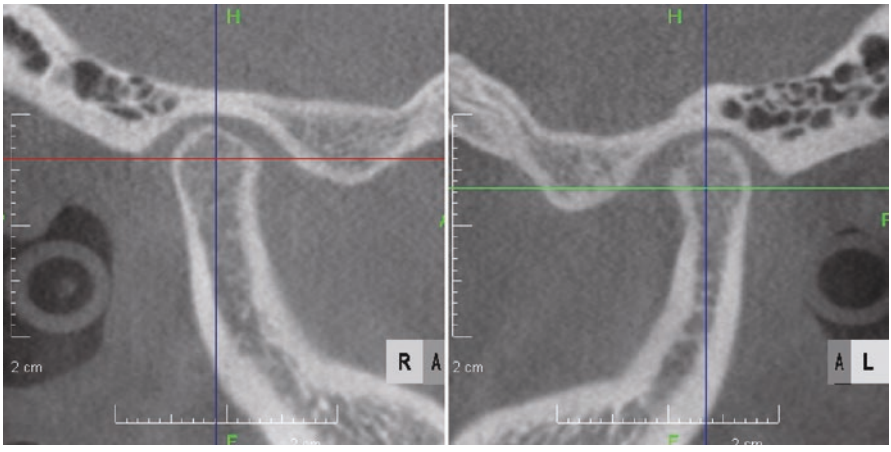

Fig. 4.32 Sagittal CBCT after splint therapy. Both condyles have seated upward with a very small superior joint space. The MRI confirmed that both joints are Stage 4 DD

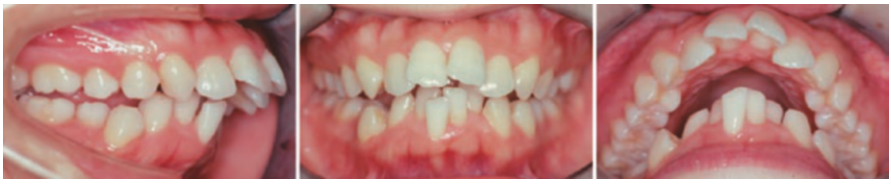

Fig. 4.33 Intraoral photos after splint therapy. Due to the positional changes of both condyles, there is obvious change at the occlusal level

4.9 Stabilization of Mandibular Position

The patient's true mandibular position may be difficult to capture during the initial examination due to tight muscles. Patient TMJ history and chairside examination often provide clues to problems in the TMJ such as DD. When problems are suspected, it is necessary to check the patient's mandibular position to make an accurate diagnosis and determine if the existing mandibular position is stable enough to serve as a foundation for treatment.

The following are examples of DD conditions that would make the stabilization of mandibular position difficult:

- Advanced Stage 3 DD is unstable because it may progress to Stage 4. Stable mandibular position is difficult to obtain during this transition period.
- In the acute phase of Stage 4 DD, mandibular position may change significantly, necessitating cautious monitoring until the condition becomes chronic to allow treatment planning after stabilization of the joint. It is important to bear in mind that stable mandibular position in this situation is different from the one in the "disc-on" setting.
- Stage 4 DD is often associated with degenerative bony changes such as erosion and subchondral cysts (Fig. 4.34). This may continue to progress with further bone resorption. Progression of bone degeneration or resorption would naturally cause changes in the occlusion.

4.10 Process of Stabilizing Mandibular Position

(a) Stabilization-type splints are used to stabilize the mandibular position. A CR wax bite record taken during the initial examination is used as the patient's initial mandibular position for splint fabrication. For Stage 3 DD patients without the disc in place or patients with TMJ pain, the operator must watch for any sign of discomfort or pain during bite registration and instruct the patient to stop squeezing into the wax just before feeling any discomfort.

(b) What is a stabilization-type splint (Figs. 4.35, 4.36, and 4.37)?

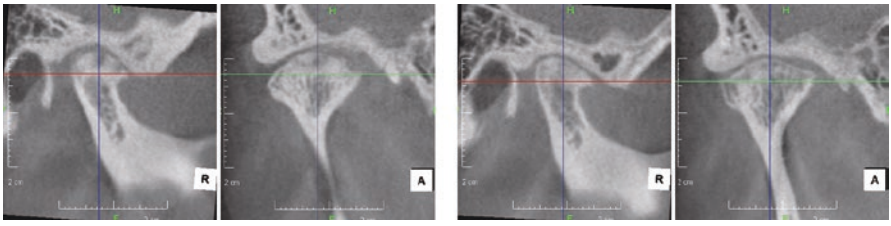

Fig. 4.34 Mandibular position is altered by resorption and remodeling of the condyle, and the eminence and the condylar position change within the fossa. In advanced DD, more time is needed to achieve a stable joint

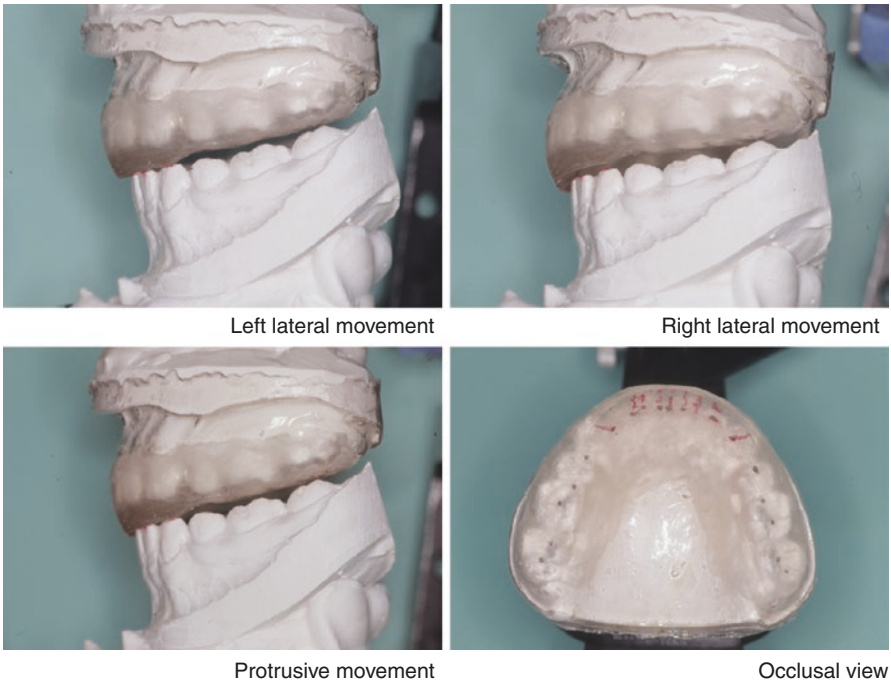

Fig. 4.35 Stabilization-type splint. An upper full coverage stabilization splint with a mutually protected occlusion is used to achieve a stable mandibular position. The splint is constructed on an articulator. In closure there are simultaneous centric contacts of lower buccal (centric) cusps with no actual contact of the anterior teeth (0.0005″ clearance). The splint is adjusted to eliminate occlusal interferences in protrusive and lateral movements of the mandible

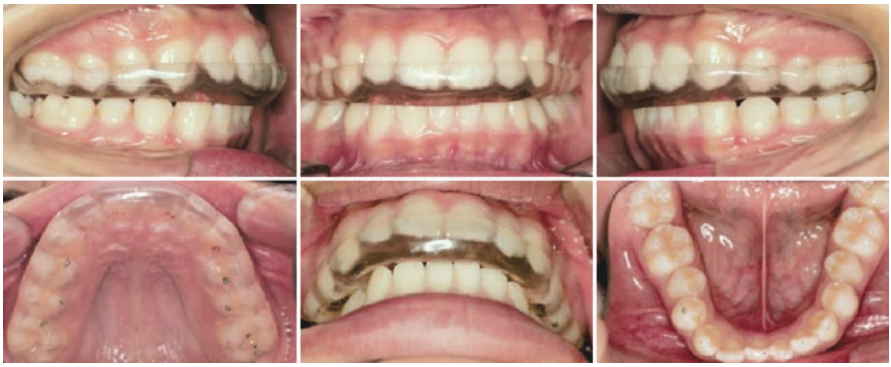

Fig. 4.36 Stabilization-type splint in the mouth. These intraoral photographs show the stabilization-type splint placed in the mouth. Note the smooth surface and neat shape of the splint for minimal patient discomfort

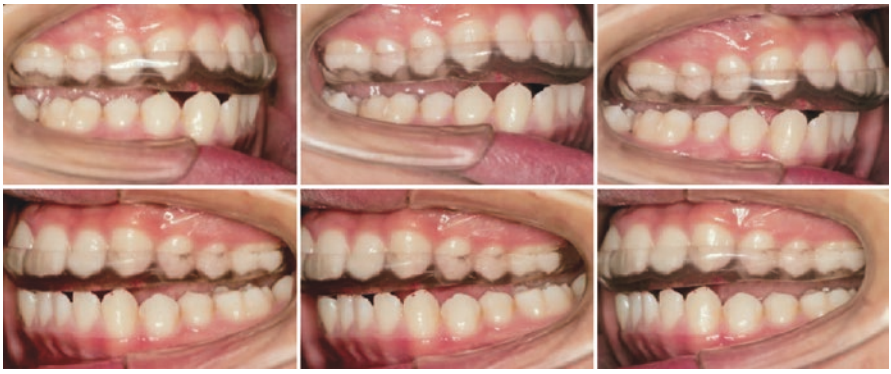

Fig. 4.37 Function and form of the splint. The function of the splint needs to be checked for adequate disclusion of the posterior teeth in protrusive and lateral movements of the mandible

Dyer and others used stabilization-type splints for treatment in the early 1970s [52]. Michigan-type splints including the one developed by Ramfjord are based on similar concepts with the only difference in the incisal guidance [53]. Clark commented about the stabilization-type splint in the treatment of TMD patients: "Stabilization appliances are preferable over repositioning appliances for most patients since they produce a positive effect on jaw function, muscle activity, and muscle pain without the need for the subsequent dental stabilization" [54]. However, this type of splint requires clinician's experience and skills for bite registration, precise fabrication of the appliance, close monitoring, and regular adjustment, and it must be used while paying attention to what stage of DD the patient is in.

(c) A great deal of information can be gathered from a patient's response during the stabilization process. Splint therapy is a reversible treatment that temporarily improves the occlusion using a splint to determine if the patient's problem in the TMJ area can be resolved by occlusal therapy. It also allows three-dimensional measurement of each case's deviation from normal once the joints have been stabilized. Predictability of treatment results can be improved with proper assessment of the present status of the joints.

4.11 Summary of Stabilization Process of the Mandibular Position

1. Stabilization of mandibular position enables clinicians to:
   (a) Determine if there is a relationship between patients' TMJ complaints and their occlusion prior to the start of definitive occlusal treatment.
   (b) Identify true discrepancies in the mandibular position three-dimensionally.
   (c) Confirm whether or not stable mandibular position can be obtained. If there is uncertainty, the prognosis of any procedure requiring major occlusal

change will be unpredictable. Prudence and caution are warranted when performing irreversible procedures.

2. What are the characteristics and criteria of stable mandibular position?
   (a) Significant improvement in the patient's initial complaint.
   (b) The improvement is sustainable.
   (c) There are objective data indicating stability (e.g., MRI, CBCT, mandibular movement, CPI data).
   (d) The stable mandibular position can be confirmed clinically and utilized for treatment planning.
3. Indicators of readiness for treatment planning following stabilization:
   (a) The patient's initial complaint in the TMJ area has been reduced or resolved with splint therapy, demonstrating that the complaint is related to the occlusion.
   (b) The clinician has identified the extent of the patient's problem, allowing development of a comprehensive treatment plan covering all problem areas including skeletal discrepancy, facial profile, intraoral occlusal discrepancy, and stress on the periodontium.
   (c) Preferably the patient is well aware of the relief of TMJ-related symptoms as a result of occlusal improvement with splint therapy. This will increase patient compliance with modalities of treatment that may significantly alter the existing occlusion, such as prosthodontics, implant therapy, orthodontics, and orthognathic surgery.

4.12 Case Example

Trying to manage a case like the following without the process of stabilizing mandibular position described above is a long, rough road, and end results will never be stable. This patient had started orthodontic treatment elsewhere but soon discontinued it. The process of stabilizing his mandibular position prior to orthodontic treatment will be illustrated below.

4.12.1 Gathering Information by History and Record Taking

1. Medical/dental history:
   - Medical:
     General medical health and any significant medical condition, such as cardiac, renal, liver, or neurological diseases
   - Dental:
     Tooth sensitivity, severe tooth wear, and TMJ-related symptoms including joint sounds and limited mouth opening. The patient was aware of parafunctional habits. He went to an orthodontist 8 years ago but discontinued the treatment after 3 months.
2. Initial findings:
   - Face: Retruded chin, short lower face height, slight mandibular shift to the right, and everted lower lip (Fig. 4.38).

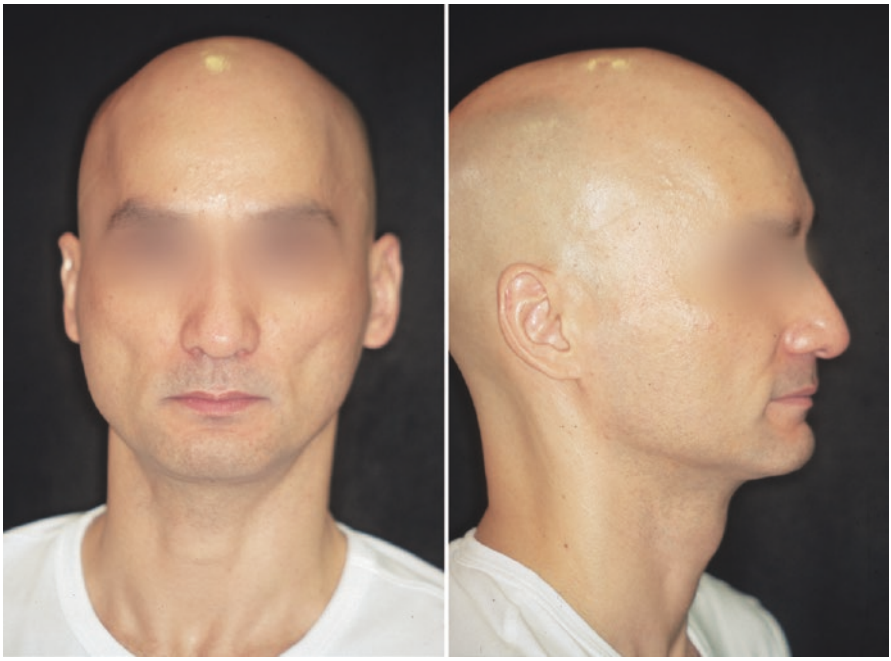

Fig. 4.38 A 48-year-old Caucasian male initial frontal and profile facial photos. Chief complaint is teeth misalignment

- TMJ: Tight masticatory muscles on palpation, tinnitus, joint sounds during jaw function, and bruxism. Imaging revealed advanced DD on the right side with joint effusion on T2-weighted MRI images (Fig. 4.39) and DD in transition from moderate to advanced stage on the left side with joint effusion (Fig. 4.40). The overall disc status was therefore rated as advanced DD. He complained of tinnitus and joint noise. He was also a bruxer. His muscles were tight when palpated.
- Recording of condylar movement with Axiograph: His left joint was considered loose because of a large immediate side shift of 1.8 mm. Condylar movement in the right joint was limited (Figs. 4.41 and 4.42).
- CBCT images: Both condyles were displaced posteriorly due to DD, accompanied by flattening and bone sclerosis of the eminence (Fig. 4.43).

3. Mounted models (in initial CR):
   - Initial CO-CR discrepancies were measured at the joint level using CPI. Because his muscles were tight, these were his initial CO-CR discrepancies that could be captured on the first attempt (Fig. 4.44).
4. Intraoral photographs:
   - He had generalized attrition of teeth, as well as gingival recession and tori, indicating parafunction. He showed a collapsed bite with many endodontically treated teeth and a couple of missing molars (Fig. 4.45). There was a major difference between his bite observed in the mouth and that on mounted models, which was indicative of occlusal instability.
5. Cephalograms:
   - P-A: Asymmetry of the rami was observed, a clear sign of mandibular asymmetry.

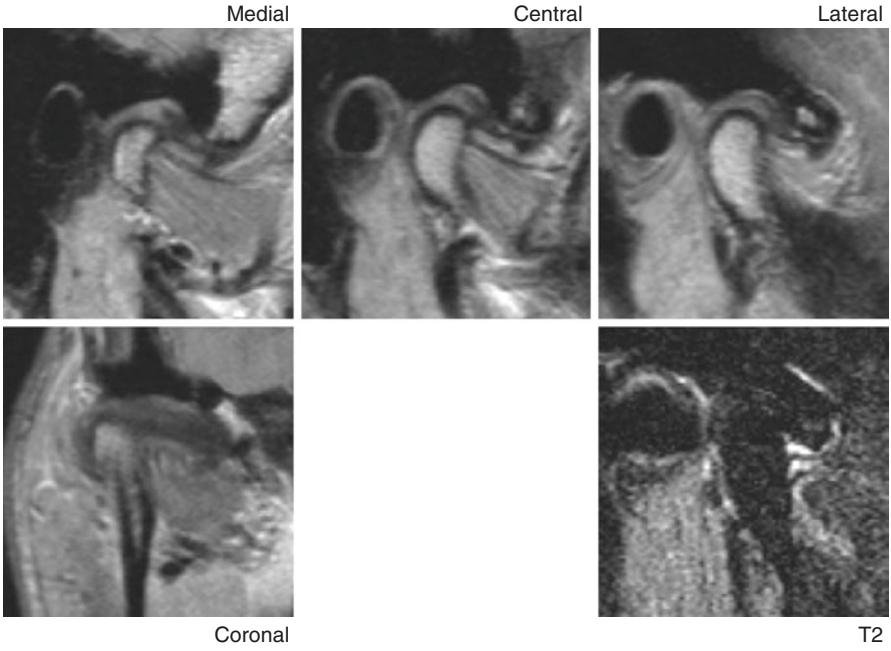

Fig. 4.39 MRI of the right TMJ. Advanced DD and significant joint effusion are noted. DD is in Stage 3 as the disc reduces upon mouth opening

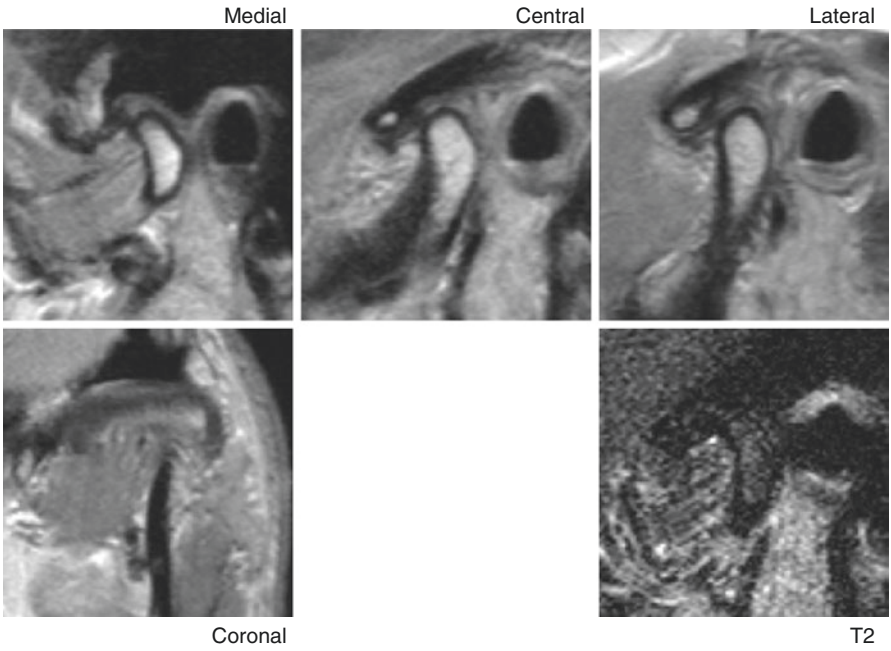

Fig. 4.40 MRI of the left TMJ. The posterior band of the disc lies near the 9 o'clock position, making it unstable with a chance of advancing toward a complete displacement. Joint effusion is present although less compared to the right side

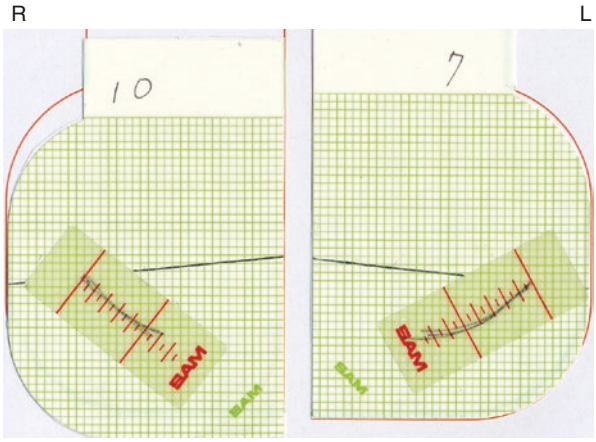

Fig. 4.41 Sagittal jaw movement recording. The red dot is the terminal hinge axis at initial, and the black line is the axis orbital line. Sagittally, the right side has limited range of movement

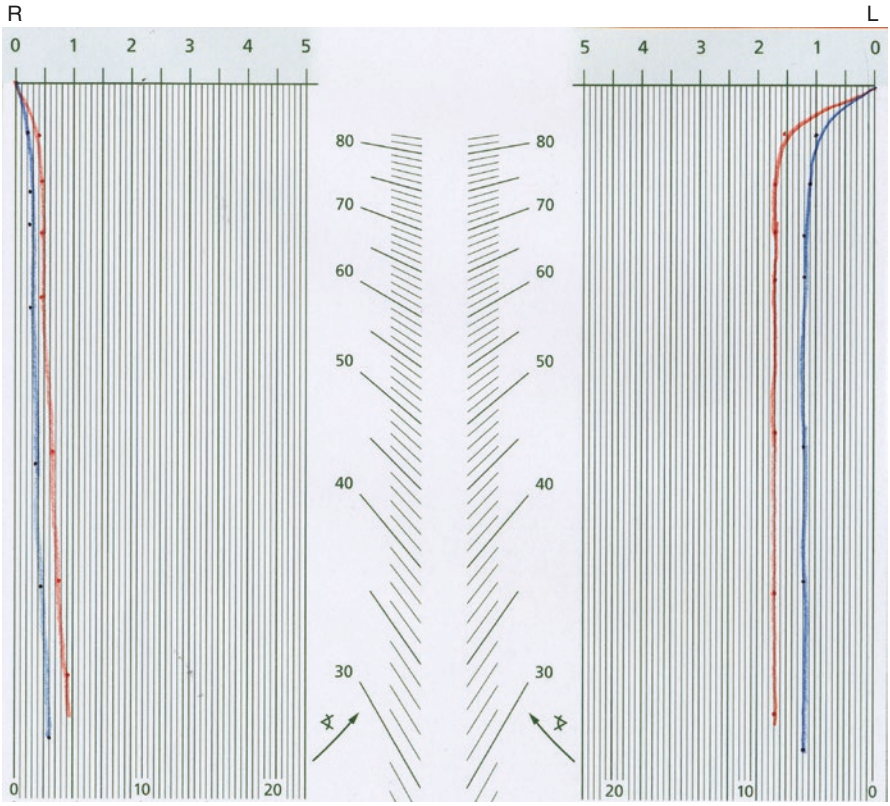

Fig. 4.42 Immediate side shift recording. The blue line is marked with the patient moving the jaw by himself, and the red line is drawn as pressure is applied during jaw movement. Sideshift is large, especially on the left side, suggestive of joint laxity

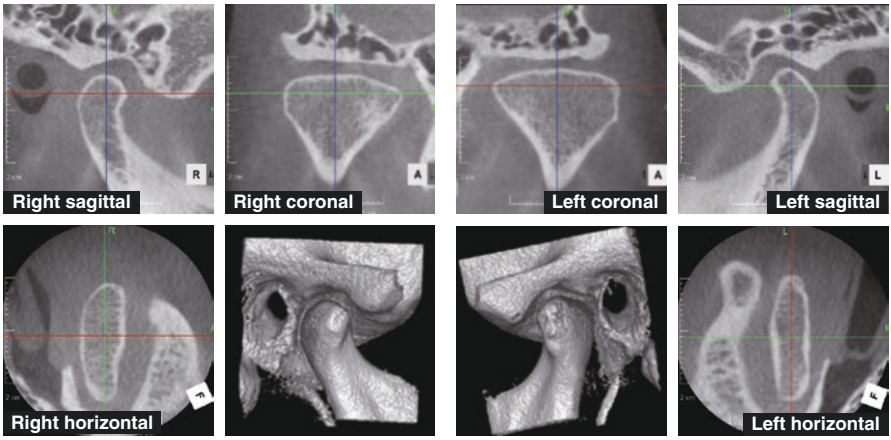

Fig. 4.43 Initial CBCT images of the right and left TMJ. The functional surfaces that are supposed to be rounded show some flattening

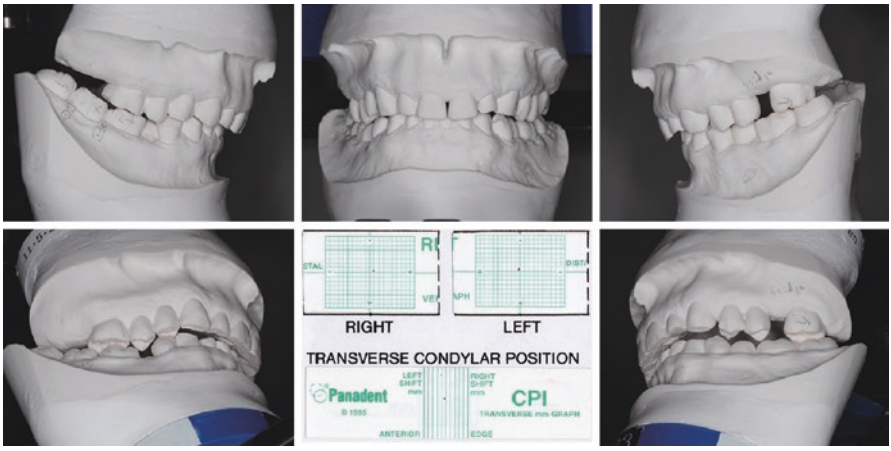

Fig. 4.44 Initial mounted models in CR and CPI data. The bite is different from what was seen intraorally

- Lateral: The chin was slightly retruded. The lower face height to upper face height ratio was unfavorable due to a short lower face (Fig. 4.46).

These initial records provide only a rough estimate and do not form a basis for diagnosis and treatment planning. His initial CR may change because he has unstable disc position and joint effusion. The process of stabilizing his mandibular position is thus needed. A splint is used to create an ideal occlusion without changing tooth position, observe the effects of occlusal changes with the splint on his chief complaint and TMJ symptoms, and determine if there is a relationship between them.

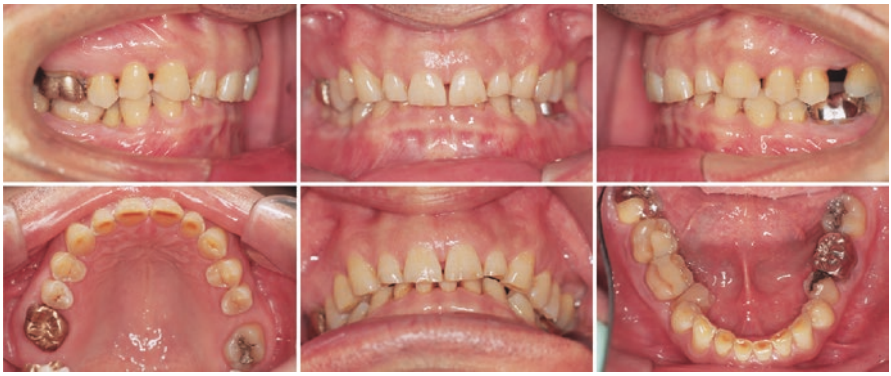

Fig. 4.45 Initial intraoral photographs. The bite is unstable and severe attrition and overall damage to the dentition are evident

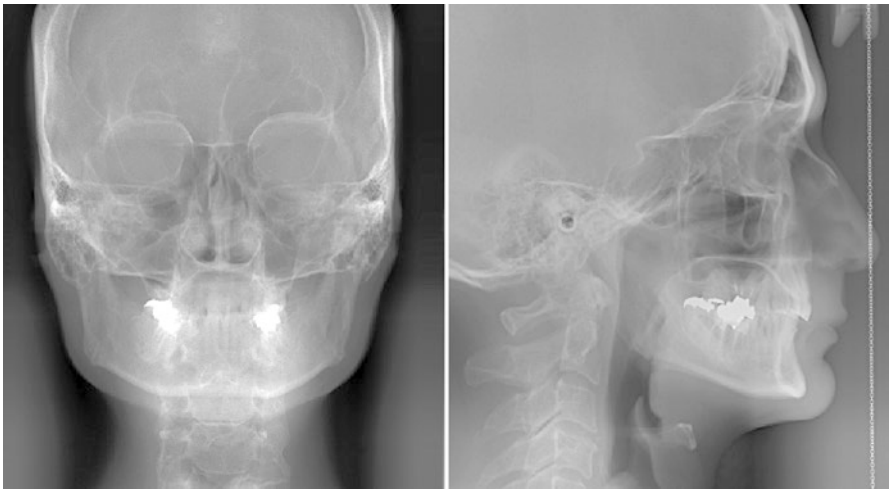

Fig. 4.46 Initial frontal and lateral cephalograms. The lower facial height is reduced and mandibular asymmetry to the right is noted

4.12.2 Records Taken After Splint Therapy Using a Stabilization-Type Splint

1. Changes in the TMJ on MRI:
   Post-splint MRI images show improvements in disc position and morphology with the formation of pseudo-discs in both joints. This is associated with a marked reduction in joint effusion on the right side (Figs. 4.47 and 4.48).
2. Facial changes:
   - Post-splint facial photographs still show a retruded chin, a short lower face height, and asymmetry to the right.

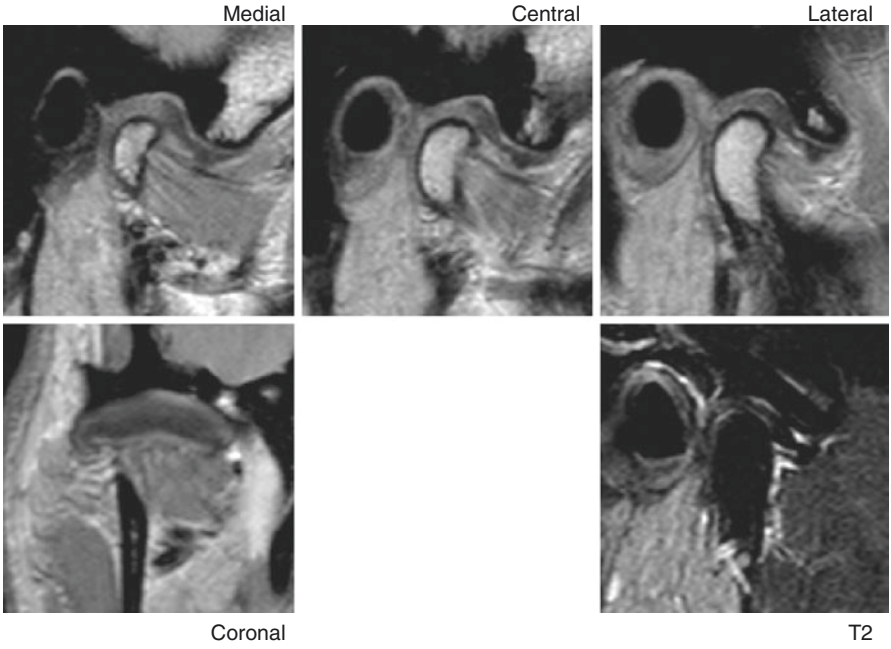

Fig. 4.47 Post-stabilization MRI of the right joint. Disc status is improved with a pseudo-disc formation, and joint effusion is dramatically reduced

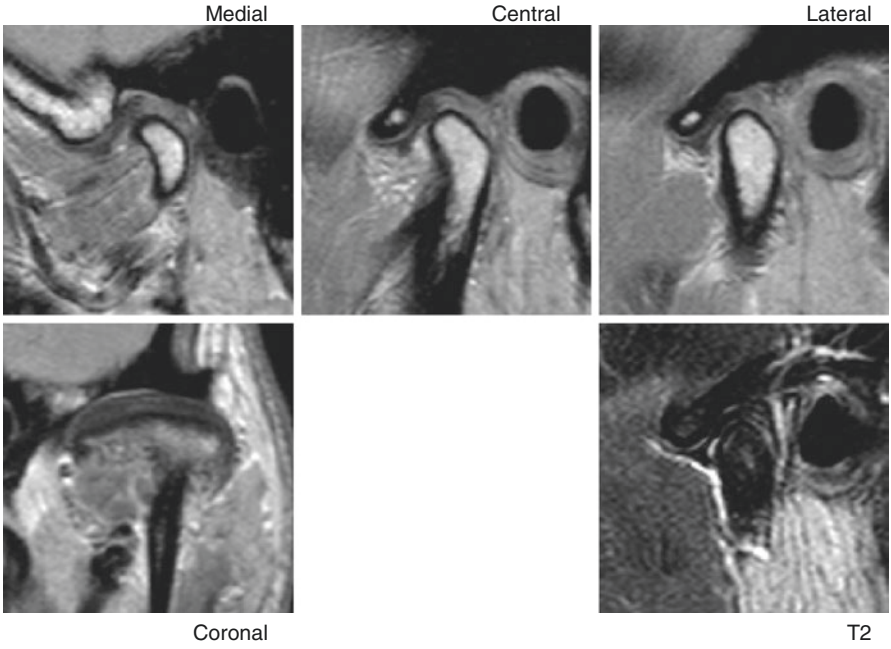

Fig. 4.48 Post-stabilization MRI of the left joint. Disc position is significantly improved

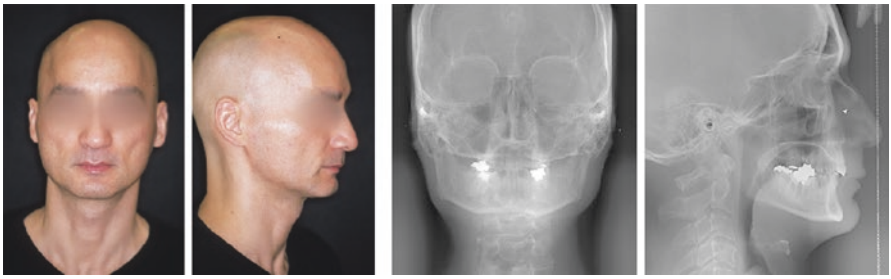

Fig. 4.49 Post-stabilization facial photographs and cephalograms. No major changes are noted, but the mandible has receded with a slight clockwise rotation

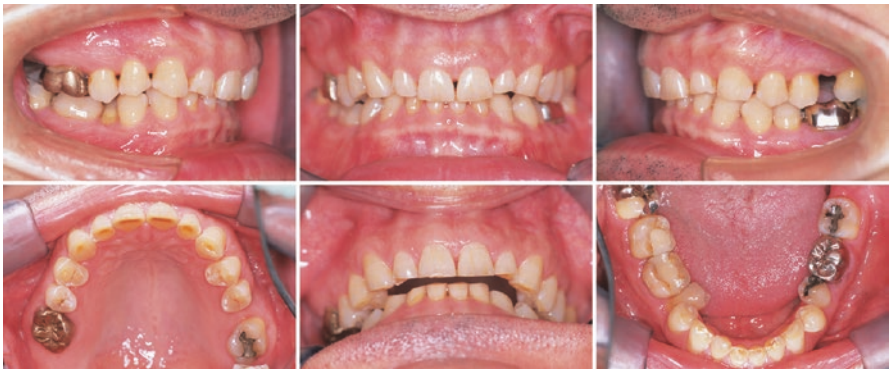

Fig. 4.50 Post-stabilization intraoral photographs. It is evident that the patient had been posturing

- Cephalograms exhibit asymmetry of the rami and mandibular retrusion (Fig. 4.49).

3. Intraoral changes:
   Occlusal discrepancy is evident in the mouth because of changes in the position of both condyles in the fossae through the process of TMJ stabilization (Fig. 4.50).
4. Changes in CPI data to monitor changes in condylar position in the fossa:
   CPI data is used for tracking changes in condylar position in the fossa. CPI data remaining constant for 3 months or three consecutive recordings is an indicator of joint stability. This patient's CPI records show a more complicated path to stability in the right joint with more advanced DD than in the left joint (Fig. 4.51).
5. Patient's subjective judgment:
   Patient self-assessments are recorded at every appointment during splint therapy. This patient reported that tinnitus was completely resolved and clicking was reduced. He experienced no headache during the therapy. He wore the splint 24 h a day for 10 months.

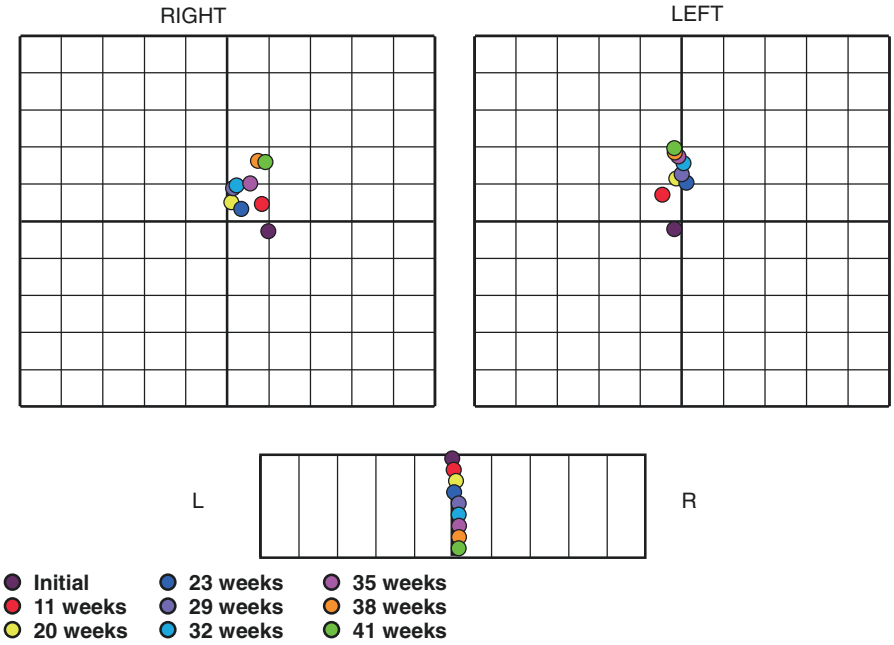

Fig. 4.51 Changes in CPI data during splint therapy. The right side with advanced DD shows a more complex change in condylar position

6. Mounted models:
   He shows an even larger CO-CR discrepancy on the post-splint mounted models than on the pre-treatment ones using the initial CR record (Fig. 4.52).
7. CBCT images of the TMJ:
   His condyles remain posteriorly displaced with no significant morphological change (Fig. 4.53).
8. Changes in Axiographic tracings of mandibular movements:
   The range of movement was increased on the right side. The size of immediate side shift, an indicator of joint laxity, was decreased (Fig. 4.54).

4.12.3 Comparison Before and After Stabilization of Mandibular Position

- Changes in the TMJ:
  Changes in disc position and morphology are seen along with reductions in joint effusion (Fig. 4.55). In this patient, changes occurred in both joints with the splint, which was adjusted at every appointment to provide anterior guidance and equal, simultaneous bilateral contact of the posterior teeth.
- Changes on cephalograms:

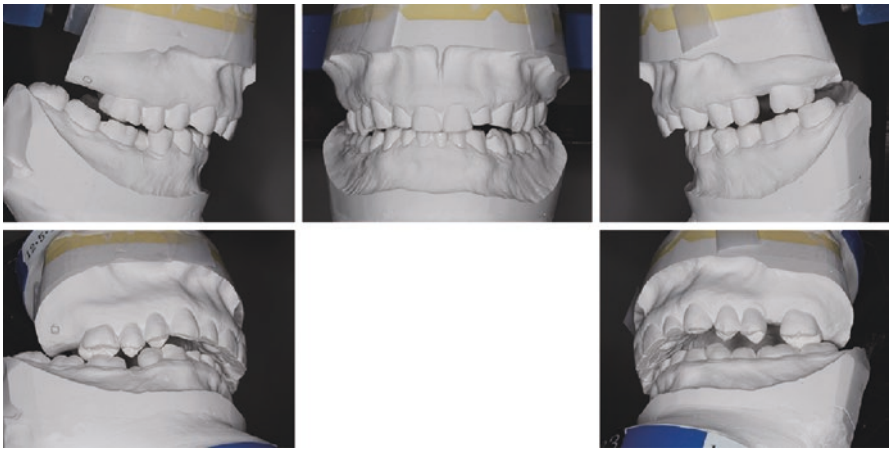

Fig. 4.52 Post-stabilization hinge-axis mounting

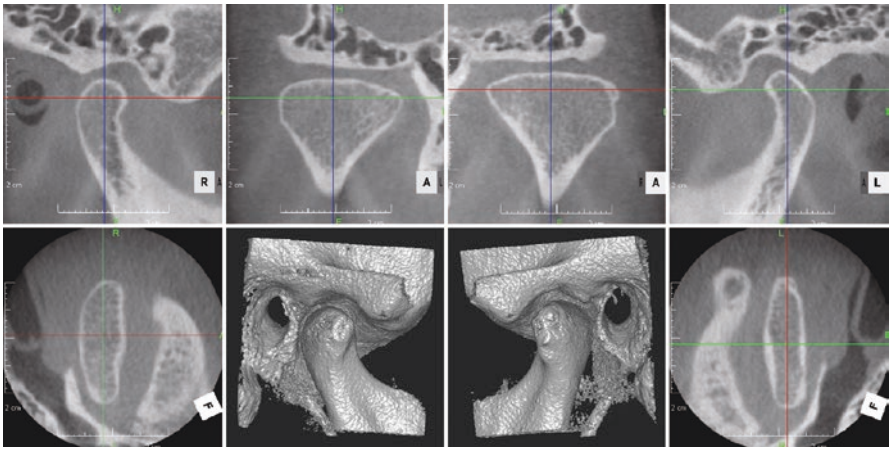

Fig. 4.53 Post-stabilization CBCT

The mandible rotated slightly clockwise with changes in condylar position, resulting in a small increase in lower face height (Fig. 4.56).

- Changes in bite:
  His occlusal discrepancy could not be detected in the mouth due to tense muscles during the initial examination, but partially uncovered on mounted models (Fig. 4.57). When his mandibular position was stabilized with splint therapy, nearly the same CO-CR discrepancy was observed in the mouth and on the mounted models as shown in the lower panel of Fig. 4.58.
- Changes in the muscles on palpation:
  The masseter muscles were tense initially but are relaxed after splint therapy. Headache has disappeared.

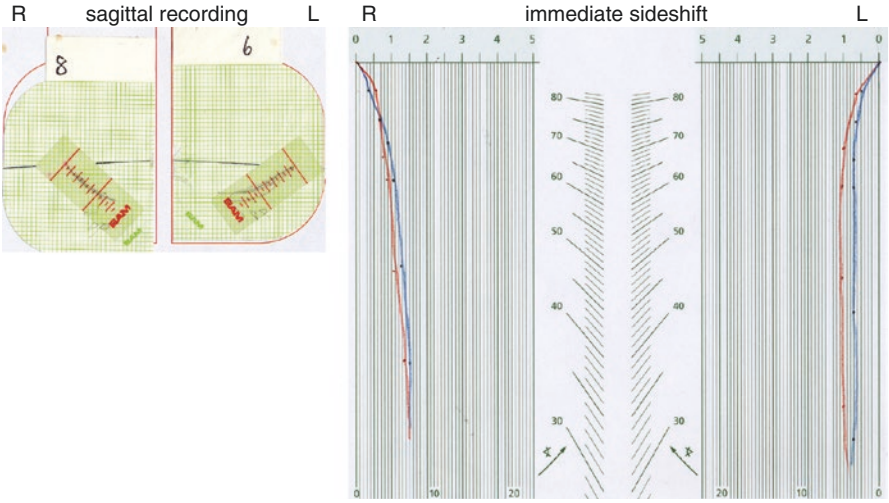

Fig. 4.54 Post-stabilization Axiograph recording

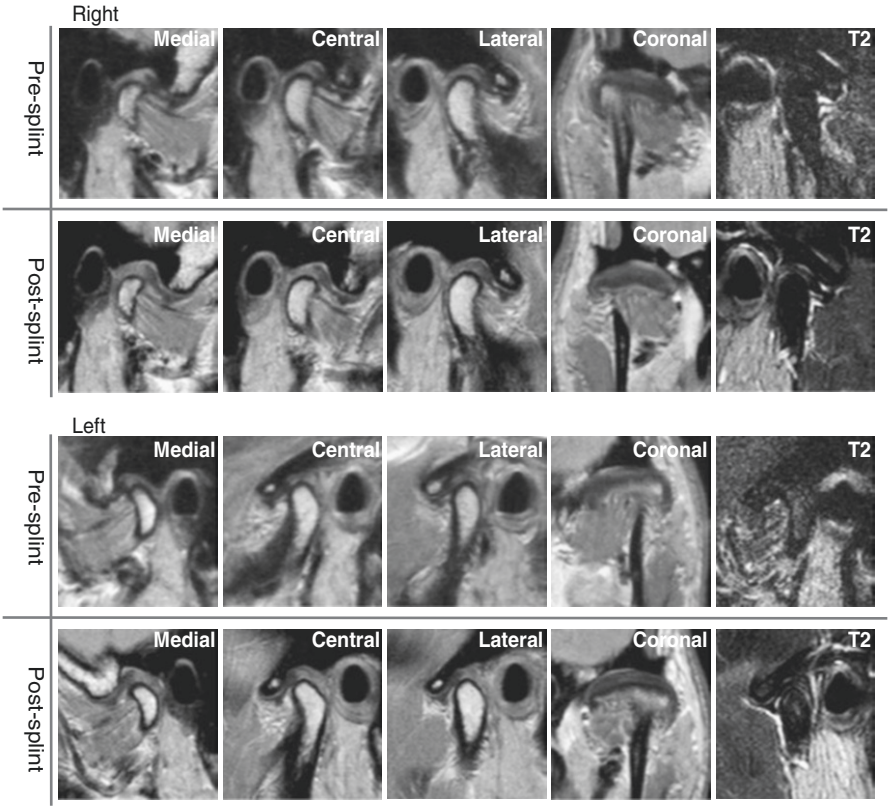

Fig. 4.55 Pre- and post-splint therapy MRI comparison. Note the improvement in disc status after splint therapy

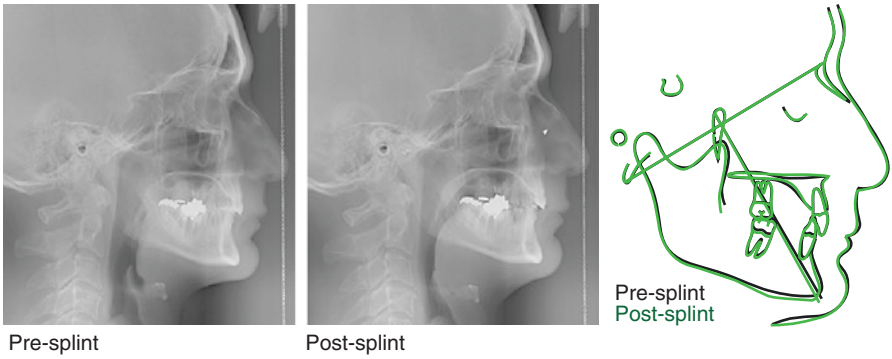

Fig. 4.56 Pre- and post-splint therapy cephalograms and superimposition. There is slight clockwise rotation of the mandible

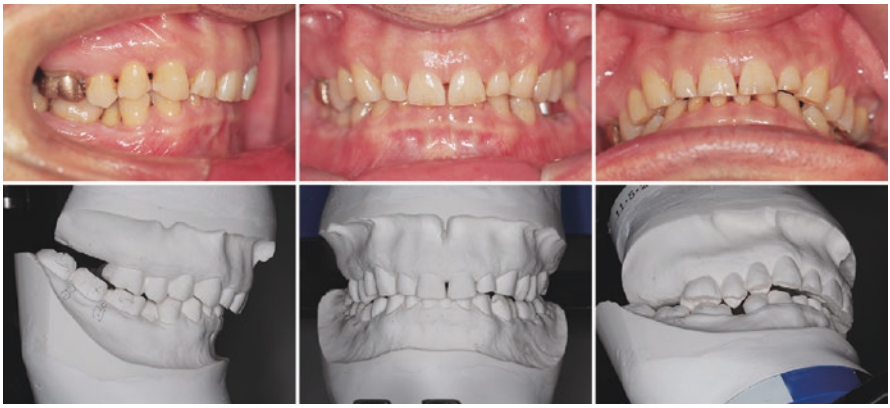

Fig. 4.57 Initial intraoral photographs compared to initial CR mounting. The initial mounting reveals a bite discrepancy not seen in the mouth

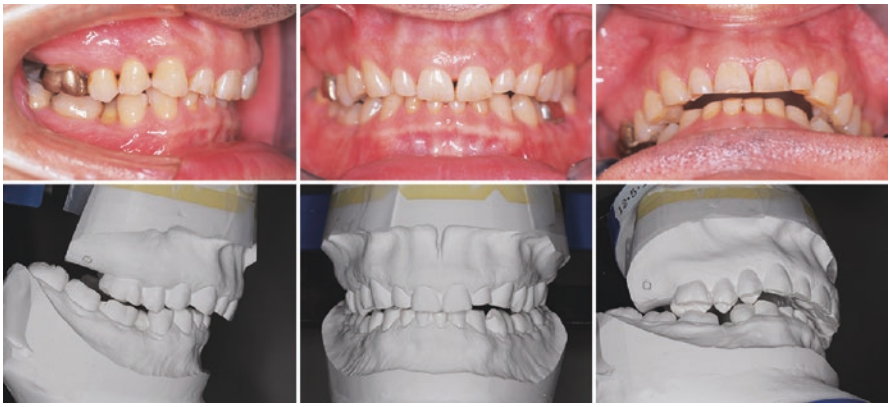

Fig. 4.58 Post-splint therapy intraoral photographs compared to post-splint therapy hinge-axis mounting. The occlusion is the same between the mouth and the models

- Changes in Axiographic mandibular tracings:
  His post-splint immediate side shift is within a normal range of 1 mm on both sides. TMJ laxity was reduced (Fig. 4.59). Splint therapy confirmed that mandibular position could be stabilized. His chief complaint was resolved. Once the joints are stable, the patient's true problems can be identified three-dimensionally, establishing a guide for treatment planning.

4.12.4 Formulation of a Problem List

- Face: Short lower face height, slightly retruded chin, and everted lower lip in profile view. Deviation of the chin and the whole lower face to the right in frontal view
- TMJ: Presence of DD, which increases susceptibility to joint instability compared with "disc-on" cases
- Skeletal: Mandibular deviation to the right due to a shorter right ramus, mandibular retrusion, and decreased lower face height
- Occlusion: Deep bite, midline shift, Class II canine relationship, severe tooth wear, and missing molars

4.12.5 Treatment Plan

How to solve the problems on the list is summarized below. STO is created, and predictions are made with routinely used techniques to see if good results can be obtained (Fig. 4.60).

- Vertical control:
  - Re-establish the lower face height.
  - Intrude the upper and lower incisors.

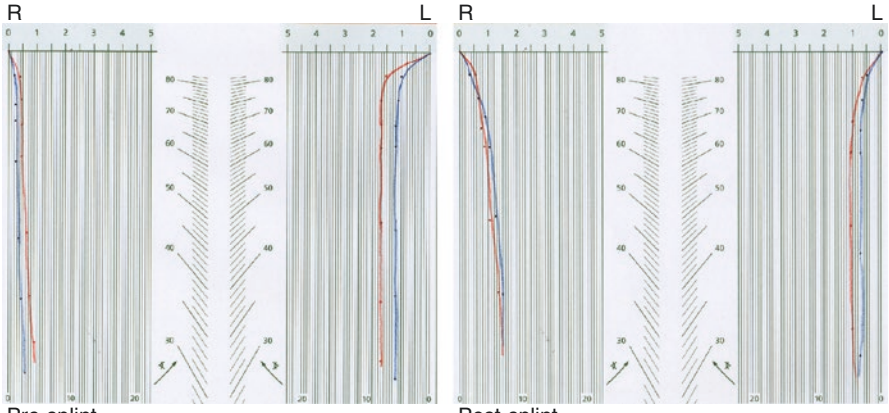

Fig. 4.59 Pre- and post-splint therapy immediate sideshift comparison. There is reduction in the joint looseness

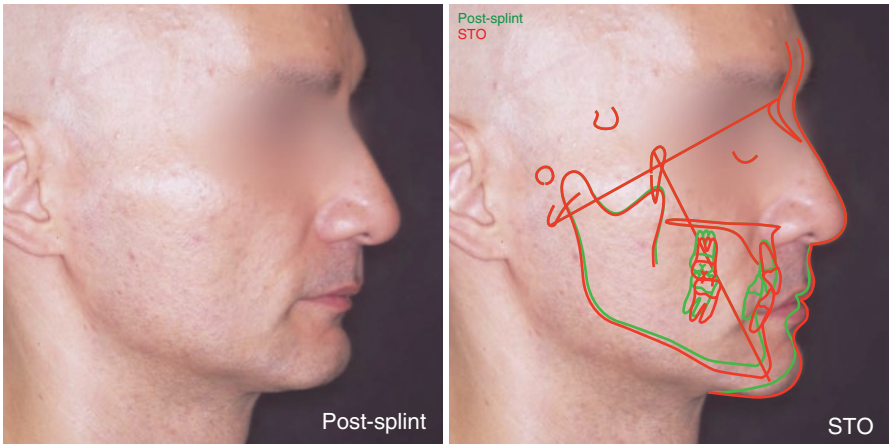

Fig. 4.60 Treatment planning with the surgical movement using surgical treatment objective (STO)

- A-P control:
  - Advance the mandibular position.
  - Correct the mandibular incisors position for the advancement of the mandible.
- Transverse control:
  - Change the mandibular position toward the left.
- Tooth alignment:
  - Reduce the overbite by intruding upper and lower incisors.
  - Correction of lower crowding by retracting lower molars.

4.12.6 Posttreatment Records

- Facial photos and cephalograms: The lower face height was increased. Midline shift was corrected. Lower lip shape was improved with the chin moved forward (Fig. 4.61).
- Intraoral photos: The dental midlines are coincident. Both canine and molar relationships are Class I. The maxillary and mandibular dentitions are harmonized with improved incisor inclinations. Severely worn teeth were restored with composite resin, and the overjet and overbite were improved (Fig. 4.62).

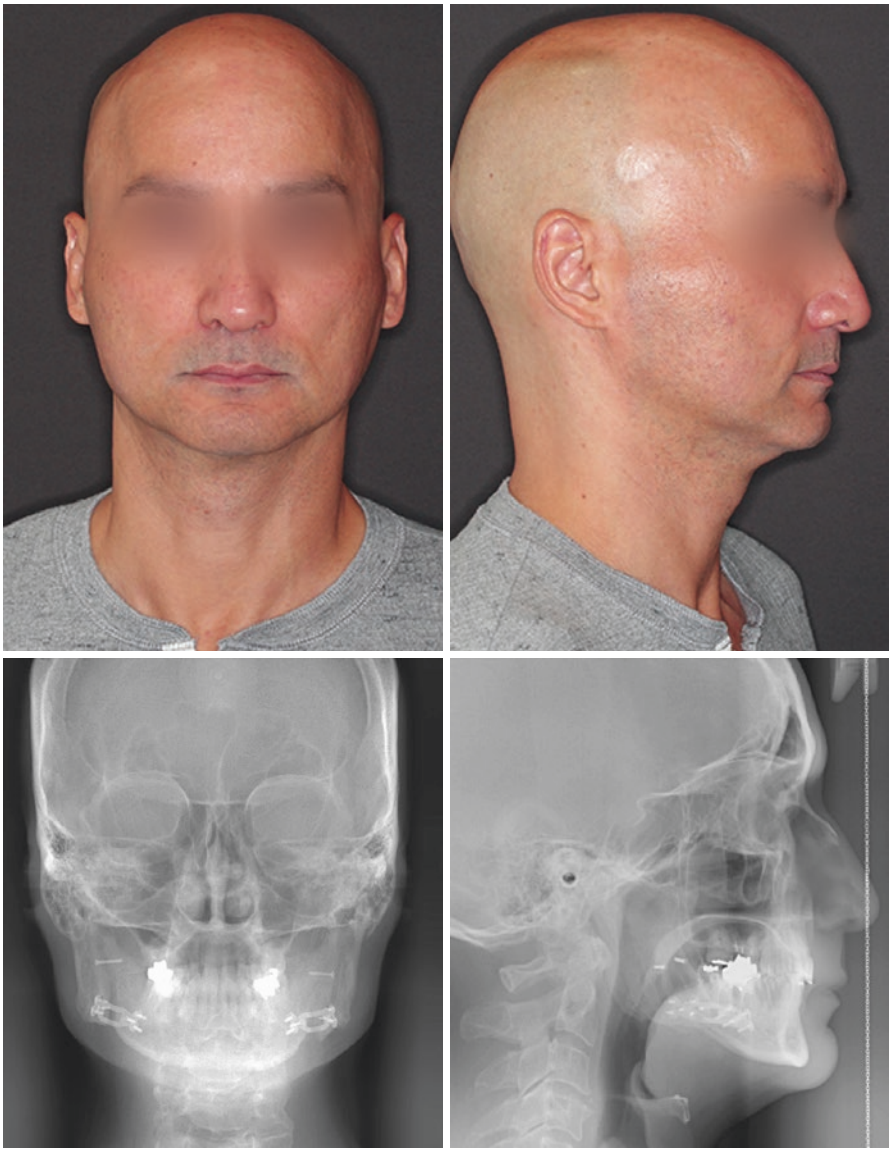

Fig. 4.61 Posttreatment facial photos and cephalograms

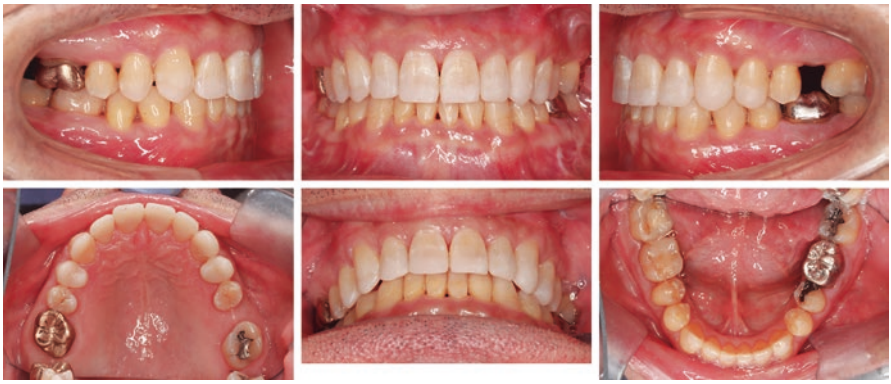

Fig. 4.62 Posttreatment intraoral photographs

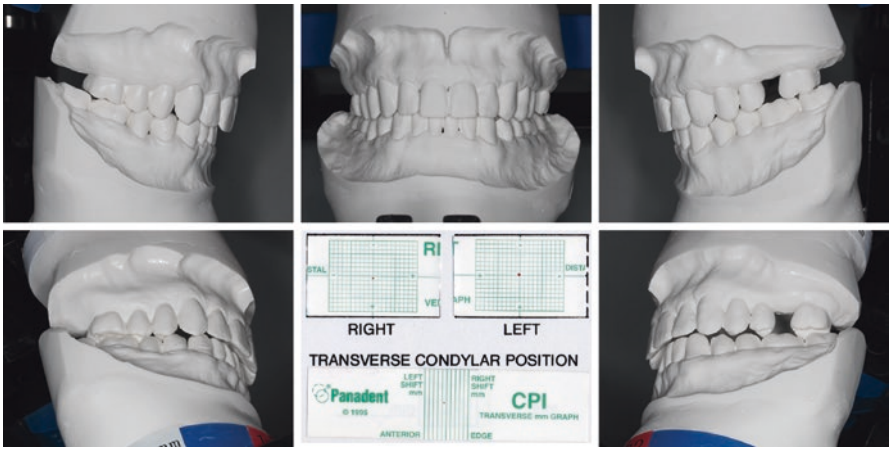

Fig. 4.63 Posttreatment mounted models and CPI data. The amount of CO-CR discrepancy is minimal

- Mounted models: There is only minimal CO-CR discrepancy. The joints will be restabilized with a splint followed by occlusal equilibration prior to prosthodontic treatment. The patient will be followed for more than 6 months before starting prosthodontics treatment (Fig. 4.63).
- CBCT images: The patient was treated by surgical-orthodontic approach with sagittal split ramus osteotomy (SSRO), which may have stressed the joints. However, no significant changes are observed in condylar position or condylar, fossa, or eminence morphology (Fig. 4.64).

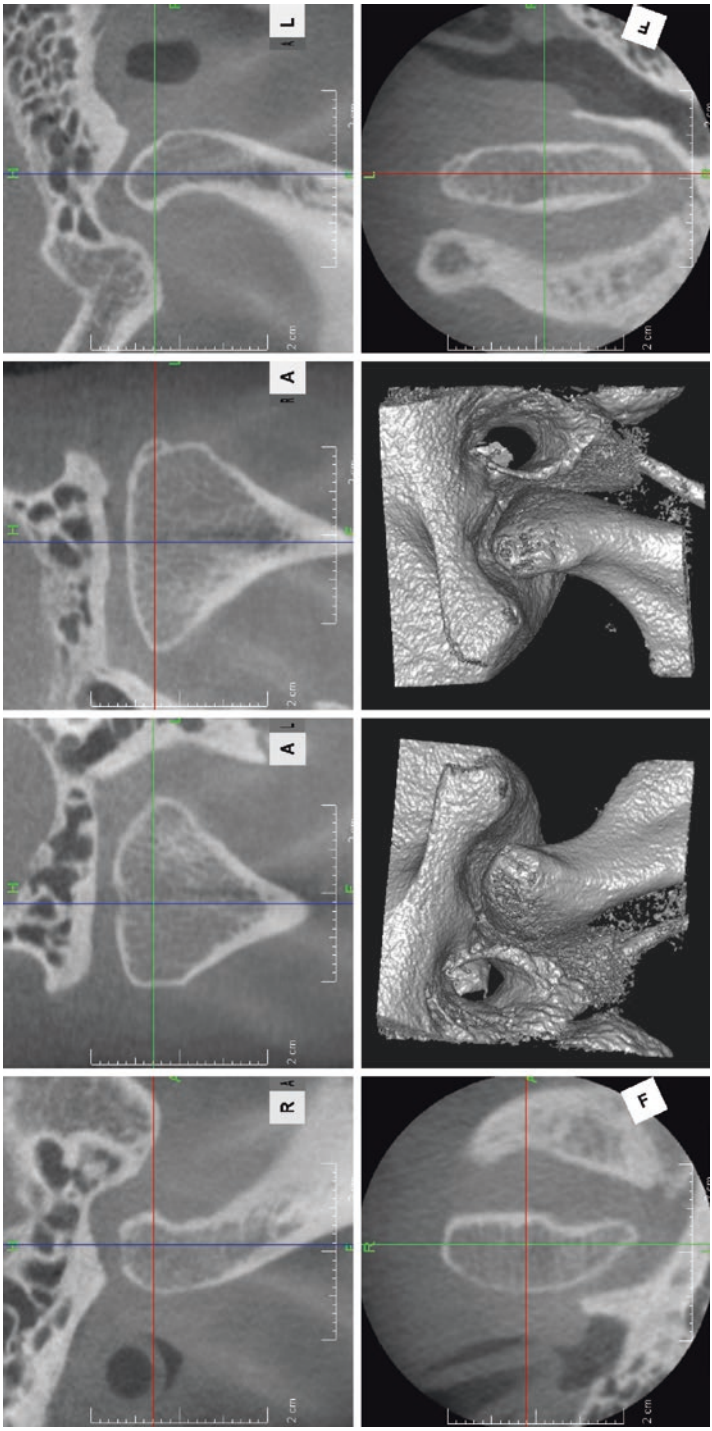

Fig. 4.64 Posttreatment CBCT of the TMJ

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TMD and Occlusion

5

Michael Jacobs

Abstract

Historically, the role occlusion plays in TMD has proven controversial and inconclusive. Some studies demonstrate an association between TMD and occlusion (Pullinger et al., J Prosthet Dent 59:363, 1988), and others find no connection (Dworkin et al., J Am Dent Assoc 120:273, 1990). Given this lack of consensus and clear causal relationship between occlusion and TMD, the idea of treating TMD by making modifications to the occlusion seems unmerited. On the other hand, as a major component of the stomatognathic complex, the occlusion is of course an important consideration when formulating a diagnosis and treatment plan for the TMD patient. Specifically, it is critical to develop a stable and reproducible occlusion to achieve a stable result of various surgical and nonsurgical manipulations aimed at directly treating the TMD. Additionally, as discussed below, the goals of obtaining a mutually protected occlusion and minimizing the centric relation/centric occlusal variance may very well, either directly or indirectly, contribute to a decrease in TMD symptoms and improvement in function. Regardless, the reality is that the restorative dentist will be called to provide dental care for the TMD patient and this necessitates that the clinician possesses an understanding of TMD and the dental considerations involved. Thus, the purpose of this chapter is to demonstrate a conservative diagnostic and treatment protocol that can be used as a guideline to provide appropriate dental care to the TMD patient.

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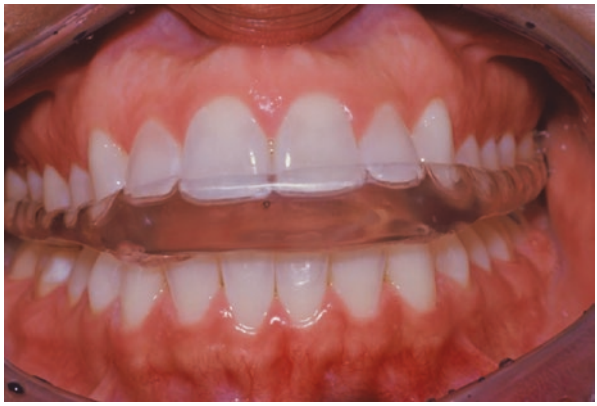
5.1 Overview

Temporomandibular disorder (TMD) is a "catchall" term that describes numerous functional disorders of the masticatory system. Historically, the role occlusion plays in TMD has proven controversial and inconclusive. Some studies demonstrate an association between TMD and occlusion [1], and others find no connection [2]. Given this lack of consensus and clear causal relationship between occlusion and TMD, the idea of treating TMD by making modifications to the occlusion seems unmerited. On the other hand, as a major component of the stomatognathic complex, the occlusion is of course an important consideration when formulating a diagnosis and treatment plan for the TMD patient. Specifically, it is critical to develop a stable and reproducible occlusion to achieve a stable result of various surgical and nonsurgical manipulations aimed at directly treating the TMD. Additionally, as discussed below, the goals of obtaining a mutually protected occlusion and minimizing the centric relation/centric occlusal variance may very well, either directly or indirectly, contribute to a decrease in TMD symptoms and improvement in function. Regardless, the reality is that the restorative dentist will be called to provide dental care for the TMD patient and this necessitates that the clinician possesses an understanding of TMD and the dental considerations involved. Thus, the purpose of this chapter is to demonstrate a conservative diagnostic and treatment protocol that can be used as a guideline to provide appropriate dental care to the TMD patient.

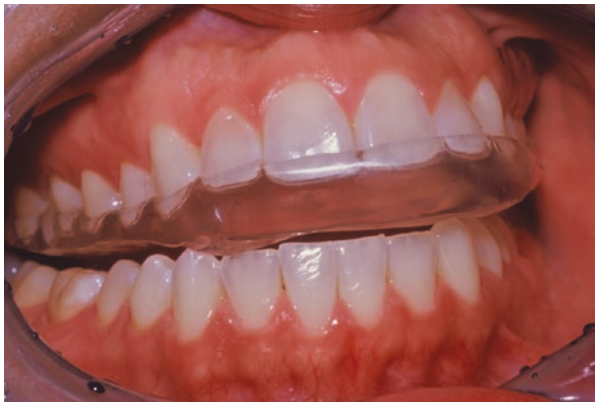
5.2 Diagnosis

As in all of dentistry, an appropriate diagnosis is critical to allow for the development of an effective treatment plan. At the initial consultation, the clinician should take a detailed approach with the TMD patient. This is especially true if extensive restorative work is anticipated, possibly including alteration in the occlusal vertical dimension (OVD). It is common for the TMD patient's chief complaint to involve muscle or joint pain. Careful attention should be paid to this report, because Okenson [3] has stated, "Problems with bringing the teeth together in intercuspal position are answered in the muscles. Once the teeth are in occlusion, problems with loading the masticatory structures are answered in the joints." If affirmation is given to one of these circumstances as derived from the patient report and clinical exam, then dentistry can play a role in solving his/her TMD problem. This is particularly applicable when the plan includes the development of a stabilizing occlusion. Further, in considering the treatment timeline, as a general guideline, no extensive definitive dental treatment should be undertaken until the patient is comfortable from a joint/muscle standpoint. If patient comfort cannot be obtained, no large-scale treatment should be initiated. Resolving the symptoms of the patient before definitive dentistry is undertaken is a basic treatment tenet [4]. Otherwise the restoration may not provide a stable result.

Osteoarthritis, osteoarthrosis, traumatic incidents, rheumatoid arthritis, and disk interferences are some of the major causal agents of TMD [5]. A preliminary diagnosis can be obtained via a medical history, careful clinical exam, and imaging of the



Picture 5.1 Nightguard—bilateral simultaneous posterior contacts and anterior disclusion



Picture 5.2 Nightguard—right laterotrusive anterior disclusion

temporomandibular joint as per the DC/TMD criteria (Schiffman/Ohrbach). If the problems are relatively mild, simple reversible treatment (occlusal splints) may be a good starting point to help resolve the patient's discomfort [6]. The splint or nightguard develops an occlusion where the posterior contacts are small and distinct and there are no laterotrusive or mediotrusive posterior contacts on the nightguard in these respective movements (Picture 5.1). The posterior contacts should hold 8-μm-thick shim stock, and the anterior teeth contacts on the nightguard should drag the shim stock. The anterior portion of the nightguard develops anterior disclusion (Picture 5.2). If no relief of pain or symptoms is obtained from the nightguard and, on further analysis, severe joint destruction and disk displacement are noted, then a consult to specialist should be obtained.

5.3 Temporomandibular Joint and Occlusion Determinants

The temporomandibular joint (TMJ) along with the muscles and ligaments supporting and moving these connected joints profoundly influences the dentist's ability to provide competent, durable restorative dentistry. This complicated

joint both rotates (inferior compartment) and glides (superior compartment). A practical working knowledge of the temporomandibular joint (TMJ) and mandibular movements is essential in planning appropriate treatment. When planning larger restorative treatment that involves occlusal vertical dimension (OVD) changes, the roles of mandibular movement and the TMJ are especially important.

The temporomandibular joints are considered the posterior determinants of occlusion, and the teeth are considered the anterior determinants of occlusion [7]. Historically, the mandibular movements are evaluated and recorded as part of the diagnostic work-up. The goal of these recordings is to gain insight into how the TMJ, muscles, and ligaments influence the mandibular movement and subsequently how these movements might influence restorative dentistry. It was hoped that the relationship between the TMJ, the mandibular movements, and the teeth would be revealed. This information is then evaluated and provides some insight into what type of occlusal form would yield the "best and most predictable" therapeutic occlusion. The term "mutually protected" occlusion was coined to describe this type of occlusion [8]. This was described as an occlusion where the posterior teeth protected the anterior teeth and the anterior teeth protected the posterior teeth. In relation to this, an early evaluation of what healthy "good" dentitions had in common was also undertaken. These early appraisals while empirical were a starting point in developing a therapeutic occlusal philosophy. Long before Williamson's frequently cited study regarding muscle activity and occlusal type [9], gnathologist Charles Stuart was advocating a "mutually protected" occlusion [10]. This evaluation of the normal functional masticatory system was used to describe a "gold standard" that could be used to re-create and reorganize dentitions that were worn and in poor condition. This was especially useful in Angle Class I occlusions. Subsequently, the development of highly adjustable articulators that allowed a more precise study of mandibular movements and allowed the quantification and recreation of Bennett movement and immediate side shift was a major step forward in assisting the clinician in developing a therapeutic occlusion. This knowledge was helpful in trying to recreate mandibular movements on the articulator that guided the construction of a restoration that required less adjustment and was more in harmony with the masticatory apparatus.

The shape of the glenoid fossa and the way the disk and condyle move in that fossa will influence the restoration cusp inclination, cusp shape, and cusp paths. For example, a steep condylar guidance will allow more posterior cusp height. However, if the condylar guidance is shallow, this will influence the posterior restorations to have shorter cusps. Another example of condylar influence is if there is a pronounced immediate lateral translation with minimum anterior guidance, this situation would dictate the use of shorter posterior cusps. When the compensating curve, plane of occlusion, incisal guidance, cusp inclination, and condylar guidance are taken into consideration, we have Hanau's Quint [11]/Theilman's formula which relates all five aspects of Hanau's Quint to allow the restorative dentist a method of evaluating how these five factors interrelate and can affect occlusion. While Theilman's formula is primarily a method of considering balanced

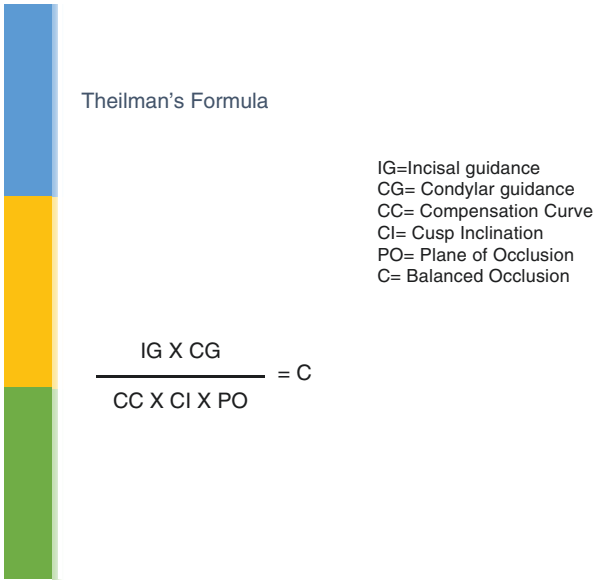


Fig. 5.1 Theilman's formula

occlusion for complete dentures, this formula can also be used to evaluate the dentate patient's occlusion. For example, in the dentate patient, if the incisal guidance is increased, the cusp height and inclination of the posterior restoration can be increased. However, if the incisal guidance is decreased, the cusp height and inclination of the posterior restoration must be decreased to prevent occlusal interferences (Fig. 5.1).

5.4 Occlusion's Role in TMD

As pointed out above, if the patient has symptomatic joints or muscles, care should be taken in delineating the cause of that pain and soreness before treatment is started. Unfortunately, the source of the pain and dysfunction is usually multifactorial and typically requires multiple interventions to control. Historically, masticatory muscle dysfunction was thought of as being primarily related to occlusal disharmony. In fact, dentistry went through a phase where many occlusal equilibrations were accomplished to help alleviate TMD. The advisability of that approach has been since questioned [12]. Currently, occlusion has taken on a much smaller contributing role in TMD. In fact, some believe occlusion plays no role in the development of TMD [13]. A more biologic and less mechanical approach is the current treatment philosophy with less emphasis on occlusion. Pullinger, Seligman, and Gornbein [14] evaluated the literature regarding the movement of the mandible and functional occlusion as it related to TMD. They evaluated eccentric occlusal contacts, clenching, bruxism, and tooth wear patterns. Their conclusions were that none of these factors could be determined to

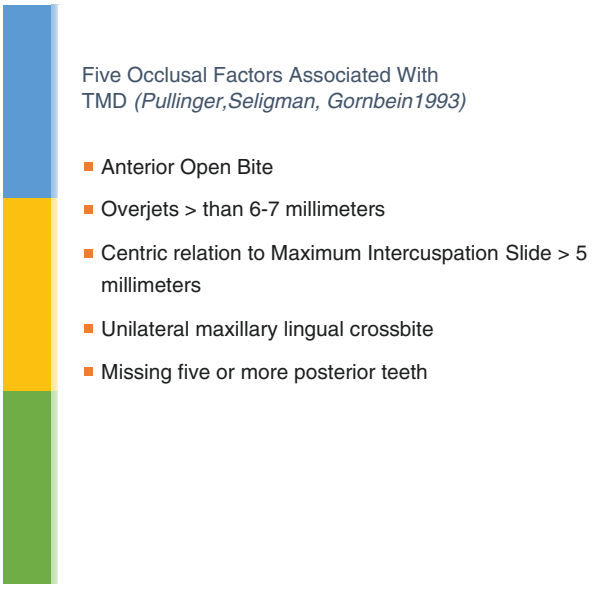


Fig. 5.2 Five occlusal factors associated with TMD [17]

cause TMD. However, the authors noted that there were five occlusal factors that could possibly be associated with TMD. They were an anterior open bite, overjets greater than 6–7 mm, a centric relation (CR) to maximum intercuspation (MI) slide of greater than 5 mm, unilateral maxillary lingual crossbite, and missing five or more posterior teeth (see Fig. 5.2).

These five occlusal factors are only possibly associated with TMD, and there is still no compelling evidence for TMD being caused primarily by occlusal disharmonies. Further, Seligman [15] states that only 10–20% of TMD patients may be delineated by occlusal risk factors. This leads us to the conclusion that today's clinician should consider the occlusion and note the status of the above occlusal factors, but it is necessary for him/her to consider other potential causes of TMD in formulating a correct diagnosis and appropriate therapeutic strategy.

To continue, the most pertinent clinical question is how do we comprehensively treat the TMD patient who requires care. After the systematic evaluation of the muscles and joints is completed and it is delineated whether the primary problem is muscle related (patient touches side of face) or joint related (patient touching the joint itself) when queried, it may be determined that the primary source of pain is both muscle and joint dysfunctions. Then, in either instance the source of the pain should be addressed. Every attempt should be made to make the patient more comfortable before dental treatment is started. If pain is not resolved, then dental treatment should be delayed. As previously noted, Okenson states that problems in bringing the teeth together in occlusion is muscle related and problems associated the loading the joints once the teeth are in occlusion is joint related. This straightforward statement is a significant help in determining whether dental treatment is indicated and should be initiated.

5.5 Occlusal Evaluation and Treatment Planning

An accurate centrically related diagnostic mounting is a good starting point in assessing the clinical situation and developing a stable restorative plan. This will further allow for the evaluation of centric relation (CR) and maximum intercuspation (MI). Most patients have a discrepancy between CR and MI. If this discrepancy is 1 mm or less, it is considered "normal," and the patient can adapt to this discrepancy. Approximately 90% of patients have some degree of CR to MI slide [16]. A CR to MI slide of greater than 5 mm has been associated with TMD [17].

Accuracy and precision are essential when a diagnostic mounting is accomplished. The impressions and subsequent cast must be meticulously made. An arbitrary facebow along with a centric jaw relation record is used for the initial evaluation. The relationship between the maxillary cast and mandible cast can be more carefully evaluated and studied on an articulator than trying to evaluate this relationship clinically. Moreover, the first tooth contact in centric relation can be more easily seen and evaluated. This first contact is important because if the first contact on mounted casts is the same as what is found intraorally, it is a verification of the accuracy of the diagnostic mounting. Moreover, the mounted casts can be moved into MI and the incisal pin set against the incisal table. This established the patient's current occlusal vertical dimension. The diagnostic casts are then allowed to open in the articulator and move back into CR position. At this time with the cast mounted in CR, the casts are allowed to close and touch at the first CR contact position. The number of millimeters the incisal pin is off the incisal table at first CR contact as compared to the MI incisal edge position gives some indication of how much vertical and protrusive CR to MI slide is present. Typically this difference is 0.5–1.5 mm. This small difference in most cases is considered not clinically significant. However, if the CR-MI slide is very large (>4–5 mm), this can be clinically relevant. This information is critical in determining if an occlusal adjustment will be necessary or prudent to make CR and MI coincident. CR is a good starting position for a larger restorative case because it is repeatable and the condyles are in their most orthopedically stable position. The centric jaw relation record can be made using various techniques. The leaf gauge, bimanual manipulation, chin point guidance, and Lucia gig are all common methods of obtaining a centric relation record. Personal preference will dictate which one of these techniques is used. Three interocclusal records are made. The casts are mounted using one of the records, and the other records are used to verify the diagnostic mounting. The goal is to position the condyles in what has been traditionally called centric relation. Okeson offers the profession a more descriptive and in this author's opinion a better term. He describes this position as the ***optimum functional position*** [18]. This position is defined as when the condyles are in the most superior anterior position in the mandibular fossa, resting against the articular eminence with the articular disk properly interposed between the condyles and the mandibular fossa. Okeson describes this position as the most musculoskeletally stable position of the mandible.

After the casts are diagnostically mounted in centric relation, the relationship of the maxilla and the mandible has been captured. The relationship is verified using the additional interocclusal records. If the additional records do not verify the mounting, the jaw relation record is repeated until verification is accomplished. This is a critical

record and must be as accurate as possible. At this point the diagnostic mounting can be used to find and evaluate the first tooth contact. The maxillary and mandibular casts are carefully closed in CR until the first tooth contact is made. This is verified with shim stock (8 μm) and marked with very thin AccuFilm 2 articulating paper (21 μm). This initial contact usually occurs in the second molar areas. More specifically, the initial contact usually occurs on the distal inclines of the posterior mandibular teeth and the mesial inclines of the posterior maxillary teeth. These contacts, the distal inclines of the mandibular teeth and the mesial inclines of the maxillary teeth, have been described by gnathologists as "equalizers" contacts. The mesial inclines of the mandibular teeth and the distal inclines of the maxillary teeth have been described as "closure stoppers." Typically the "equalizer" contacts hit and slide until MI is obtained. This hit and slide is clearly delineated in the sagittal view of Posselt envelope of motion [16] (Fig. 5.3). If the first contact occurs in the same area as was clinically noted, this is another verification of the accuracy of the diagnostic mounting.

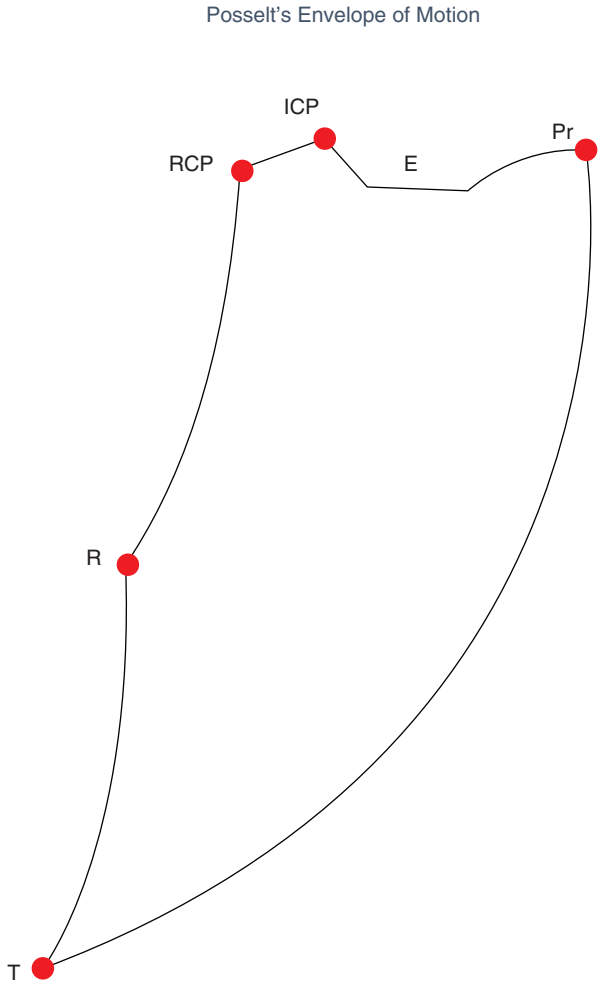
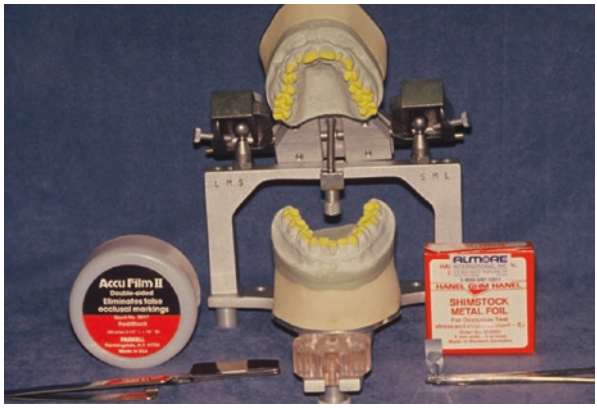


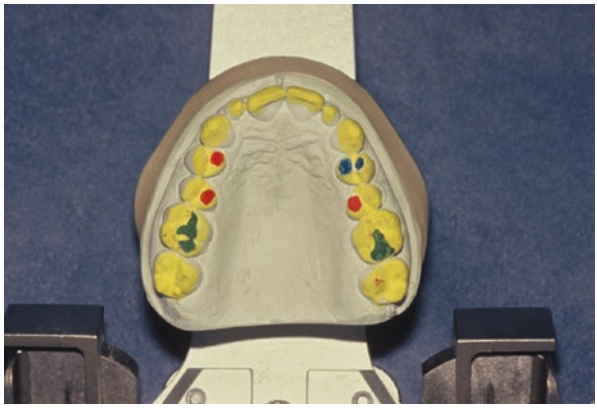
Fig. 5.3 Posselt envelope of motion

5.6 Initial TMD Patient Preparation for Comprehensive Treatment

This initial diagnostic mounting can give insight where the first point of contact is located and how much of an occlusal discrepancy is noted between CR and MI. This mounting can also be used to make the first treatment splint or nightguard. The nightguard is used to develop an ideal occlusal pattern and help relax the muscles, decrease joint inflammation, and more evenly distribute occlusal force. This occlusal pattern is usually bilateral simultaneous posterior occlusal stops. The posterior contacts hold shim stock. Shim stock is a feeler gauge that is 8 μm in diameter. The nightguard's anterior stops drag shim stock, and an anterior guidance ramp is developed so that in any lateral and anterior movement, the mandibular anterior teeth engage the ramp and immediately separate the posterior teeth effectively creating anterior guidance and a Class III lever system. Another name for this type of splint is the mandibular stabilization prosthesis. The goal of this device is to decrease muscle fatigue, spasm, tenderness, joint edema, and inflammation [19]. The TMD patient is asked to wear the nightguard all the time except while eating. After 1 month of continuous nightguard wear, the patient is re-evaluated. If the patient's muscles and joints are more comfortable, then a new diagnostic mounting is made and analyzed. A hinge axis location can be accomplished to improve the accuracy of the second mounting. After the patient has worn the splint and is comfortable, an occlusal adjustment can be considered. It is important to point out this occlusal adjustment is ***not used to treat TMD*** but it is accomplished to aid in the development of the therapeutic occlusion. A stable therapeutic occlusion will be a positive development for patients with TMD. Plus it is critical for the dependability and longevity of the patient's dentition and subsequent restoration. A critical review of the literature by Tsukiyama concluded that the current evidence does not favor the use of occlusal equilibration to treat TMD [20]. The purpose of this occlusal adjustment is to improve jaw relationships and to make CR and MI more coincident. Another goal is to improve and smooth anterior guidance and modify traumatic occlusion. Typically, another carefully made and verified diagnostic mounting is done, and the occlusal adjustment is accomplished on the mounted casts. This gives the clinician a preview of what the clinical occlusal adjustment might entail. This cast adjustment serves as a guideline for the future clinical occlusal adjustment. This clinical occlusal adjustment is accomplished after the "practice" cast adjustment and only when the patient is comfortable (Pictures 5.3 and 5.4). It is possible that once the adjustment is accomplished and there is no longer a hit and slide, the anterior teeth may "uncouple." This "uncoupling phenomenon" can be evaluated during the cast occlusal adjustment. A plan on how to recouple the anterior teeth should be in place to allow for the development of anterior guidance. An incisal guide angle that is 5° greater than the condylar guidance is considered ideal; it is also recommended that the incisal guide angle not be greater than 10° larger than the condylar guidance [21]. In many treatment plans, this type of extensive diagnostic work-up is used primarily for a larger reconstruction. If that is the case, the uncoupling of the anterior teeth is not such an



Picture 5.3 "Practice" occlusal cast adjustment on diagnostically mounted casts



Picture 5.4 "Practice" occlusal adjustment prepares clinician for clinical occlusal adjustment

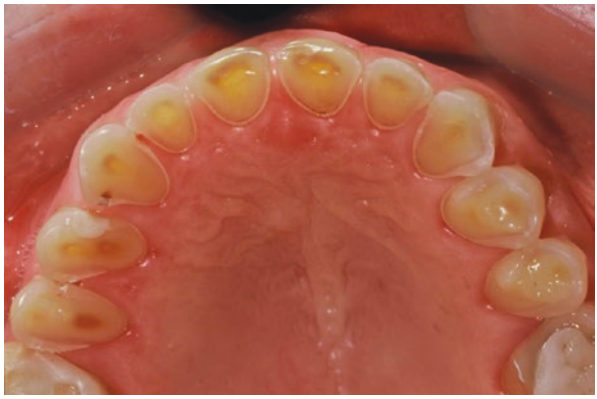
issue since the anterior teeth can be re-engaged via the planned anterior restorations. Moreover, this additional anterior restorative space created via the occlusal adjustment can be advantageous when anterior restorations are required and planned. After the clinical occlusal adjustment is accomplished, a new mounting is made. The mounting and casts are evaluated, and in many instances, an increase in vertical dimension is required. This is especially the case in GERD patients, bruxing patients, and the patient with a highly abraded dentition. Moreover, this additional restorative space is needed to address the patient's esthetic desires. After the need for a new OVD is determined, a full-mouth diagnostic wax-up is accomplished to provide a restorative guide. This should only be accomplished if the patient was comfortable with the treatment nightguard at the proposed increase in OVD.

When the goal of TMD patient comfort has been obtained and CR equals MI via occlusal adjustment, a more predictable therapeutic occlusion can be developed. At this stage the occlusal treatment plan for a TMD patient is similar to that for the non-TMD patient. Anterior disclusion, mutual protection, and an appropriate OVD are the treatment goals.

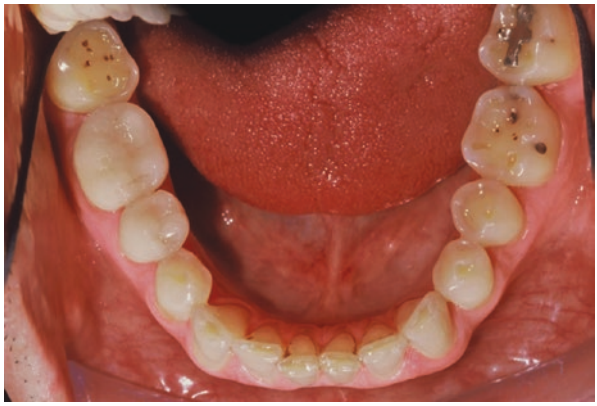
5.7 Clinical Example of Treatment Goals

The following TMD patient who also presented with gastroesophageal reflux disease (GERD) will be used to illustrate the diagnostic process, treatment planning, and clinical treatment.

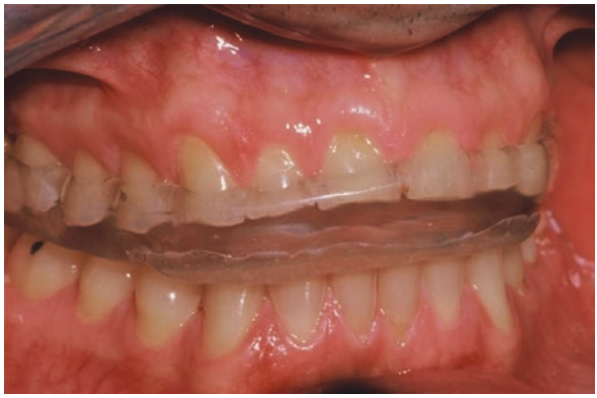
The patient was a 45-year-old male with history of GERD and TMD. The GERD was successfully treated by his gastroenterologist, and the patient was interested in restoring lost tooth structure (Pictures 5.5 and 5.6). He was also concerned with his chronic jaw and muscle pain. An initial diagnostic mounting was done in centric relation. It was noted that there was a significant discrepancy from CR to MI. This is one of the indicators that dental intervention may help a TMD patient. Because of the large CR-MI discrepancy, occlusal adjustment was considered to help place the condyles in the optimal functional position in the glenoid fossa. A maxillary splint (Picture 5.7) was made, and the patient was asked to wear the splint except while eating. The maxillary splint has small simultaneous posterior contacts, and anterior disclusion was developed with the splint. After 1 month, the patient was re-evaluated. An increase in patient comfort was noted.



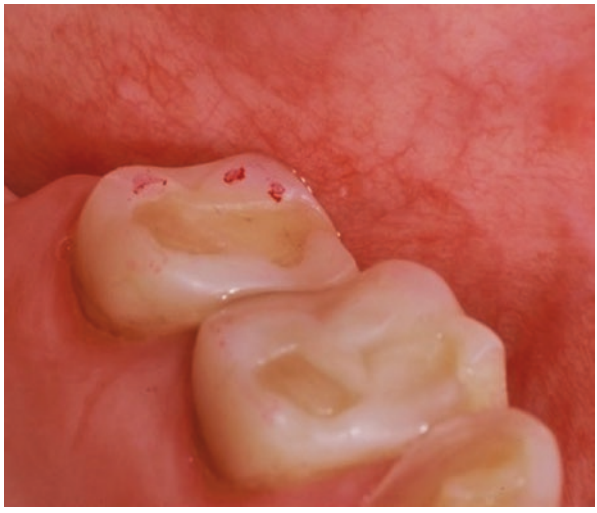
Picture 5.5 Eroded and abraded maxillary dentition



Picture 5.6 Eroded and abraded mandibular dentition

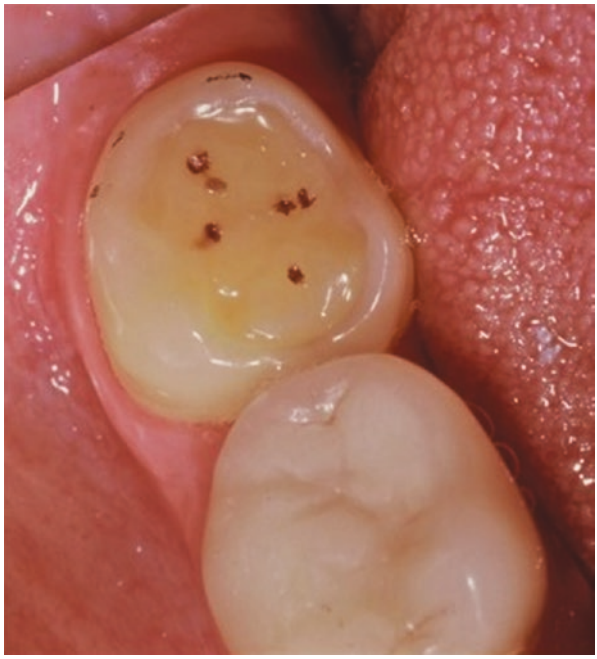


Picture 5.7 Maxillary nightguard—trial increase in occlusal vertical dimension (OVD)

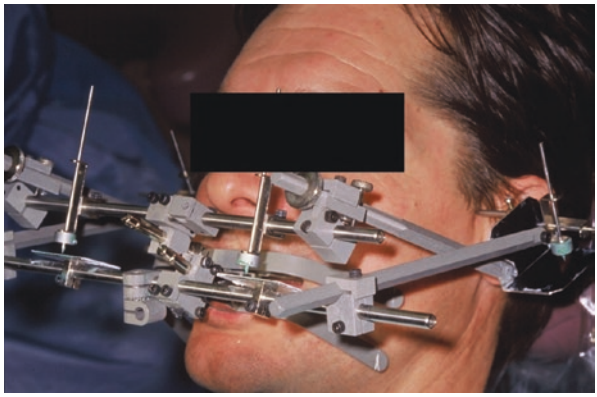


Picture 5.8 First tooth contact in centric relation—mesial incline #2 (equalizer)

A new diagnostic mounting was made in centric relation, and a "practice" occlusal adjustment was made on the casts of this mounting to evaluate how much occlusal change would be necessary to make MI and CR coincide. After the "practice" diagnostic mounted casts were adjusted, it was determined the same type of adjustment could be accomplished clinically. The leaf gauge was used to help with the clinical adjustment. The first tooth contact in centric relation was on the mesial incline of #2 and the distal incline of #31 (Pictures 5.8 and 5.9). This first contact matches the diagnostic mounting first tooth contact. The clinical adjustment's goal was to make CR and MI coincide. The clinical adjustment was done, and this adjustment sequence followed a similar pattern as practice cast adjustment. The patient's splint was also modified. Since the patient was comfortable with the splint at the new vertical dimension, it was determined a recording of mandibular movements would have some value. A stereographic recording was made to primarily evaluate the amount of immediate lateral translation that might



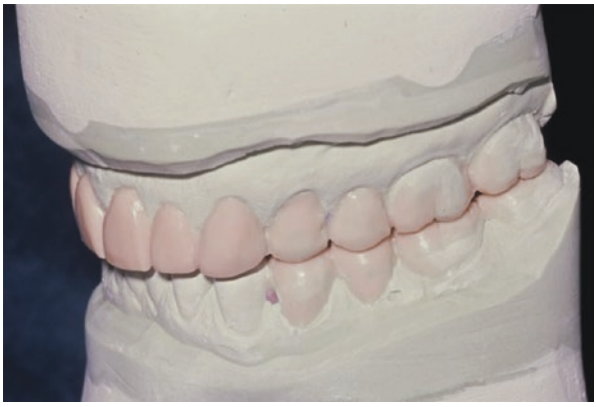
Picture 5.9 First tooth contact in centric relation—distal incline #31 (equalizer)



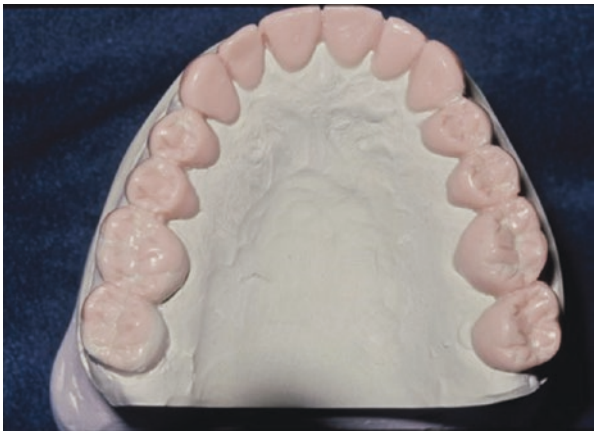
Picture 5.10 Stereographic recording using Stuart recorder

be present (Picture 5.10). The stereographic recording demonstrates a minimum amount of immediate lateral translation on both the right and left horizontal recording plates (Picture 5.11). Because of this limited amount of immediate translation, it was determined a semi-adjustable articulator (Whip Mix 2240) would be an adequate instrument to restore the patient's dentition. If a significant amount of immediate lateral translation was noted, a more fully adjustable articulator could be used (C.E. Stuart Gnathological Computer). After the adjustment, a new diagnostic mounting was made. A diagnostic wax-up was then made at the increased vertical dimension that was based on the splint therapy (Pictures 5.12,

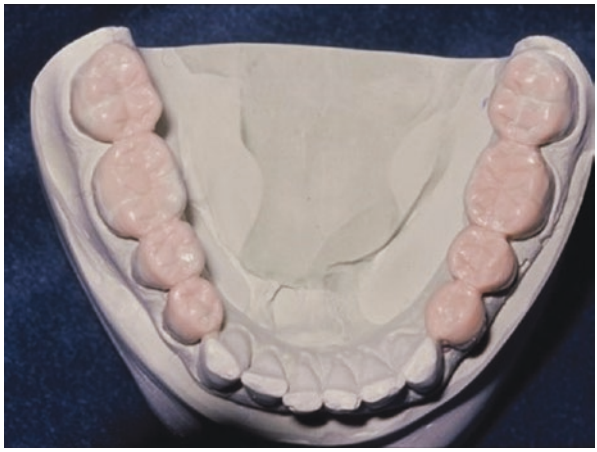
Picture 5.11 Both right and left posterior horizontal recording plates demonstrated minimal immediate lateral translation



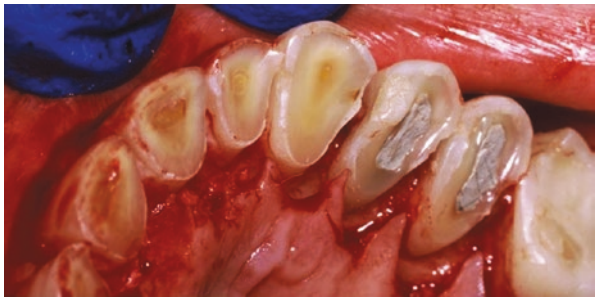
Picture 5.12 Diagnostic wax-up at planned OVD increase



Picture 5.13 Diagnostic maxillary wax-up

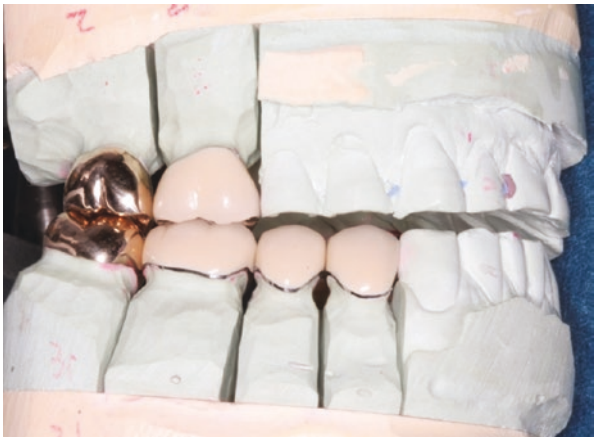


Picture 5.14 Diagnostic mandibular wax-up

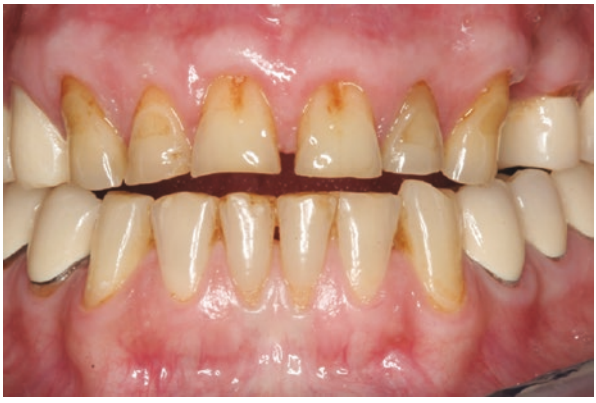


Picture 5.15 Crown lengthening 4–13

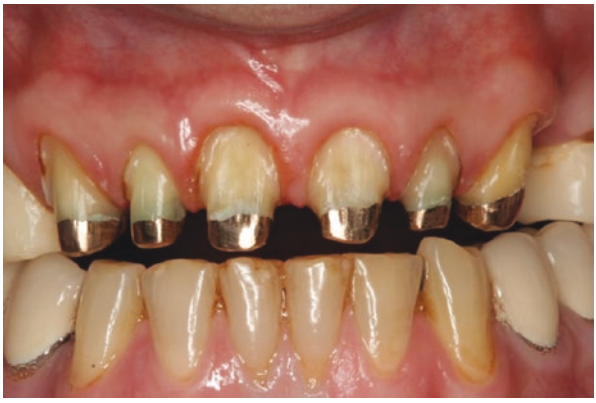
5.13, and 5.14). The splint had served as a predictive preview if the patient could tolerate and be comfortable at the new occlusal vertical dimension. Because of the erosion and abrasion, it was determined crown lengthening, endodontics, and cast gold post and cores would be necessary to allow the restoration of 4–13 (Picture 5.15). Three months after the crown lengthening surgery, the occlusal reorganization and restoration of the teeth proceeded. The posterior molar teeth were prepared to accept provisional restorations at the new vertical dimension. The final impressions were made, and the molar provisional restorations were based on the contours of the diagnostic wax-up. The molar restorations would increase the OVD and allow restoration of the remaining abraded and eroded teeth (Picture 5.16). The posterior molar restorations were made and delivered (Picture 5.17); this increase in OVD allowed the restoration of 4–13. The cast gold post and cores were made and delivered (Picture 5.18). The provisional restorations that were then made for the cast post and cores closely reflected the contours of the diagnostic wax-up (Picture 5.19). After a month of provisional restoration wear, the patient noted he was comfortable and was pleased with the provisional restoration's esthetics. Moreover, the therapeutic occlusion was stable. Anterior



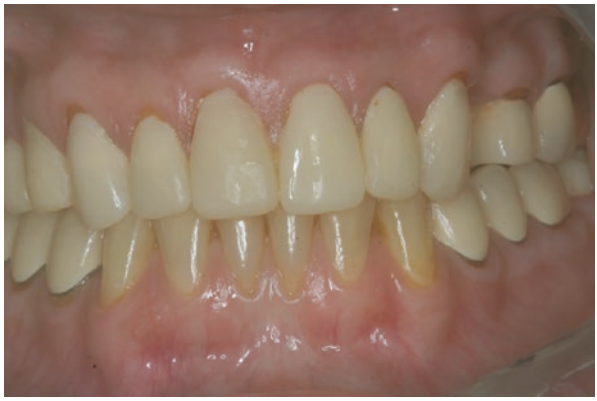
Picture 5.16 Posterior molar restorations on working casts (note increase in OVD)



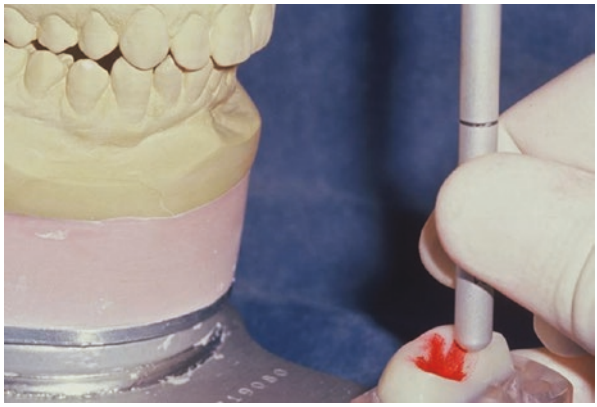
Picture 5.17 Clinical presentation of increase in OVD



Picture 5.18 Cast gold post and cores were delivered to allow restoration of 4–13

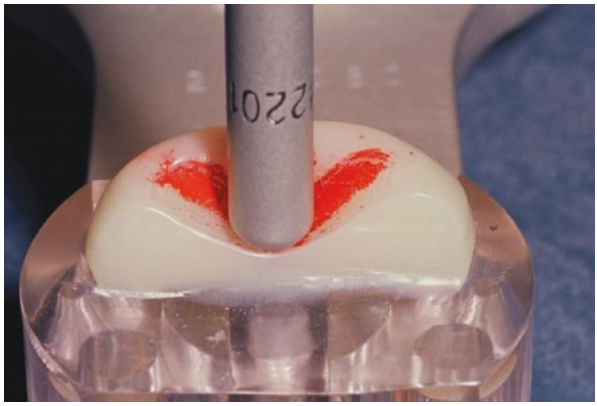


Picture 5.19 Provisional restoration made incorporating the same contours as diagnostic wax-up

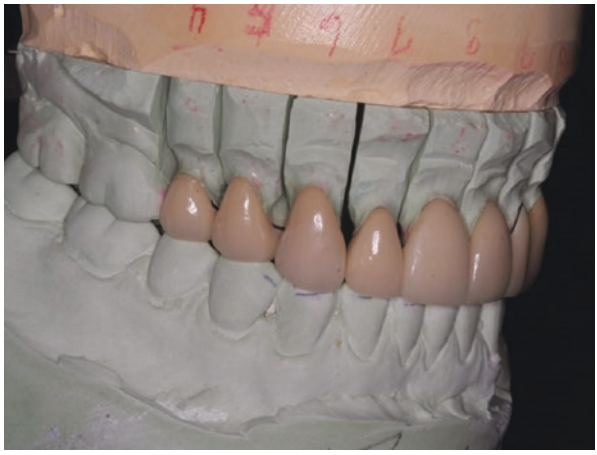


Picture 5.20 Making custom incisal guide table

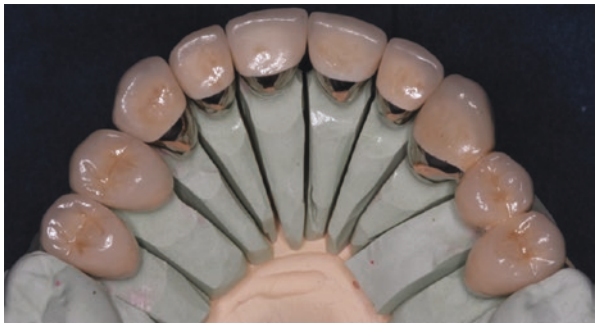
disclusion was developed with the provisional anterior restorations. A new diagnostic mounting was made with the provisional restorations in place. A custom incisal guide table was then fabricated from these diagnostically mounted casts (Pictures 5.20 and 5.21). This custom incisal guide table would be used to develop the anterior guidance for the final restorations. Since the patient was comfortable with the anterior provisional restorations, a final impression, jaw relation record, and stick bite was made. The maxillary final cast was cross-mounted against the previously diagnostically mounted mandibular cast. The custom incisal table was used to allow the laboratory technician to "mimic" the accepted provisional restoration contours. The planned final restorations were made (Picture 5.22). The custom incisal guide table was critical in the development of the permanent anterior restoration's lingual contour. This lingual contour is critical in developing acceptable anterior guidance (Picture 5.23). The final anterior restorations were delivered (Picture 5.24). The patient's permanent restoration was at an increased re-established vertical dimension and with appropriate anterior guidance (Picture 5.25). The occlusal view of both the maxillary and mandibular restored dentitions



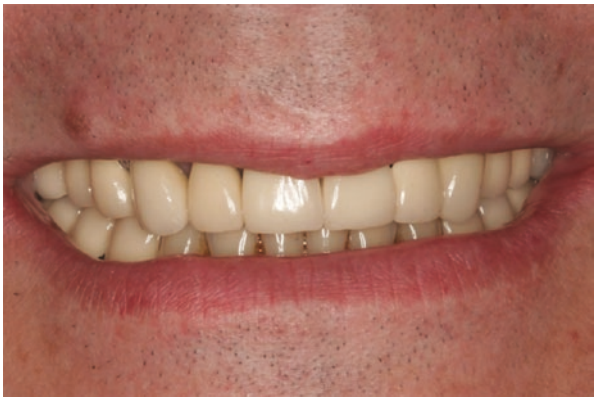
Picture 5.21 Custom guide table allows fabrication of final restorations that mimic the contours of the proven provisional restorations



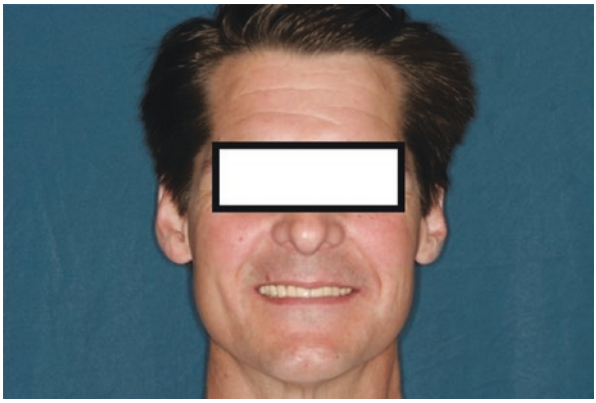
Picture 5.22 Final restorations 4–11 that have similar contours as clinically proven provisional restorations



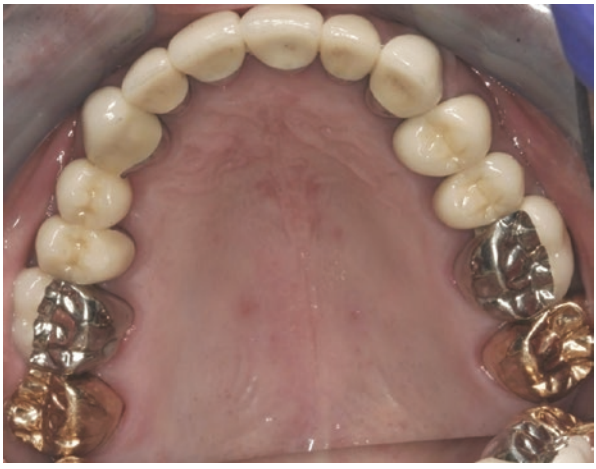
Picture 5.23 Occlusal view of final restorations. Lingual concavity development is critical for anterior guidance



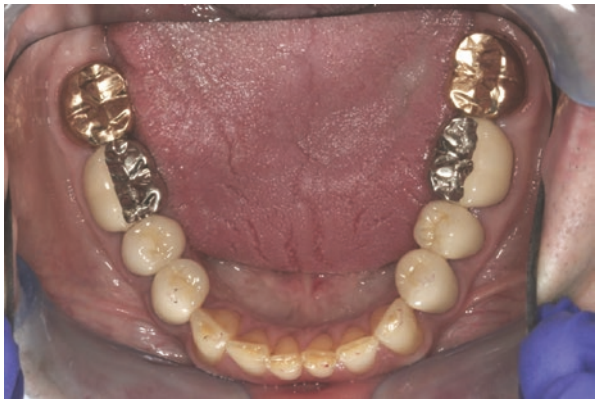
Picture 5.24 Clinical view of final restorations



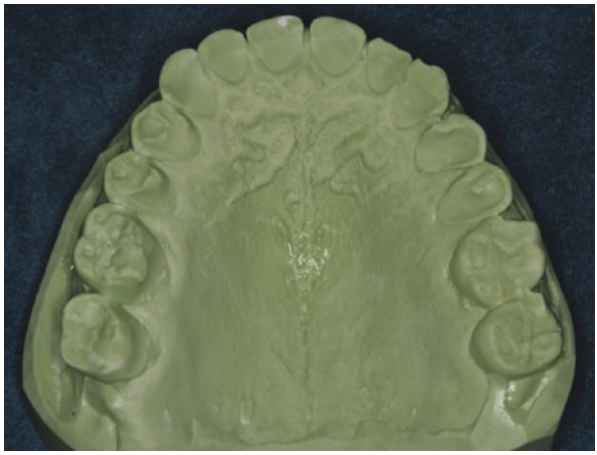
Picture 5.25 Full face view



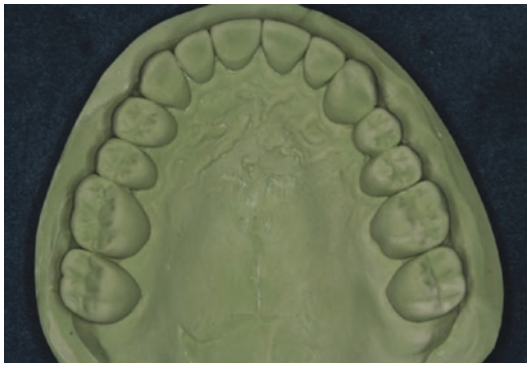
Picture 5.26 Occlusal view of maxillary restorations



Picture 5.27 Occlusal view of mandibular restorations

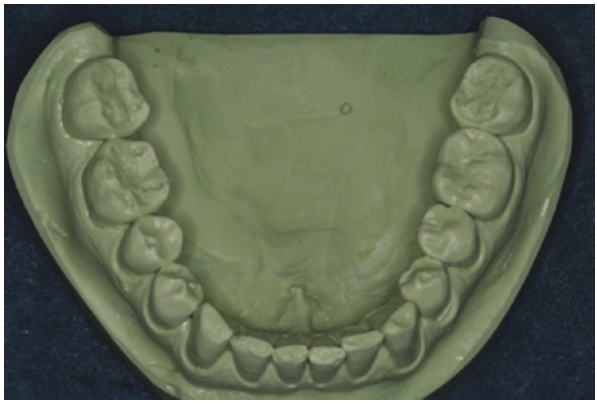


Picture 5.28 Before treatment maxillary diagnostic cast

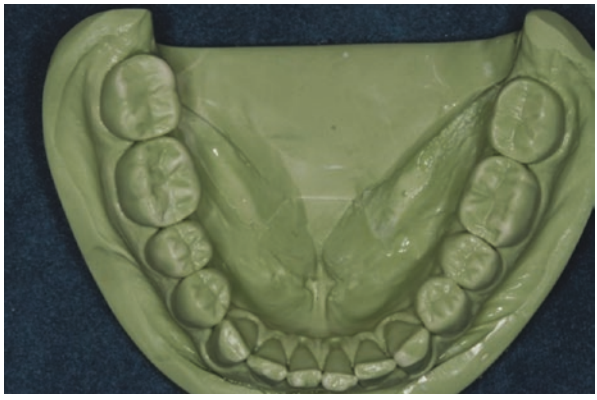


Picture 5.29 After treatment maxillary cast

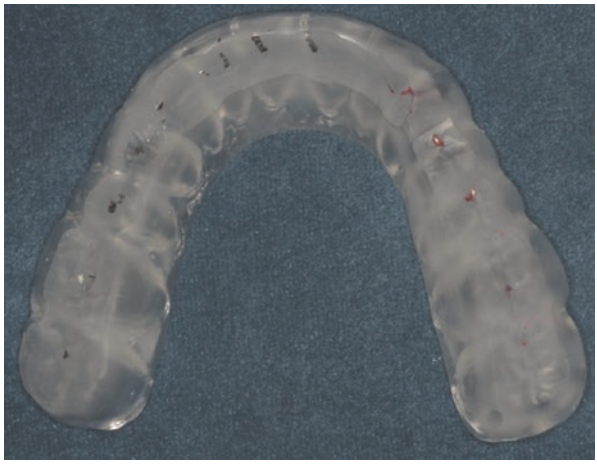
is presented (Pictures 5.26 and 5.27). The before treatment and after treatment cast photographs demonstrate a significant amount of occlusal reorganization (Pictures 5.28, 5.29, 5.30, and 5.31). Moreover, the patient's TMD complaints significantly improved. In summary, the TMD patient that requires extensive restoration of their dentition should be carefully approached. Resolution of the



Picture 5.30 Before treatment mandibular diagnostic cast



Picture 5.31 After treatment mandibular cast



Picture 5.32 Nightguard used to protect restoration

patient's TMD discomfort before reconstruction seems to be a logical, necessary, and practical approach. Moreover, after a larger reconstruction is delivered, frequent patient monitoring and follow-up are suggested. A post-reconstruction splint is also considered a must to protect the patient's investment (Picture 5.32).

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Oral Motor Treatment of TMD

6

Cláudia Maria de Felício

Abstract

Patients with temporomandibular disorder (TMD) exhibit impairment of orofacial muscles, mastication, and swallowing. The objective of oral motor treatment is to rehabilitate the stomatognathic (or orofacial) functions and to resolve the signs and symptoms of TMD. The basis of this treatment, the way it is performed, and the possibility of combining it with other modalities and future perspectives are discussed in this chapter.

6.1 Introduction

The present chapter focuses on the application of orofacial motor treatment or therapy (OMT) for temporomandibular disorder (TMD). A brief argument is presented in order to justify OMT, followed by a general view of orofacial functions and the changes related to TMD, and finally by a description of the methods for evaluation and the therapeutic conducts.

The main characteristic of TMD is orofacial pain, with an impact on the functions of the stomatognathic system such as a sensation of limitation of mastication [1–3], and, to a lesser extent, of speech. In addition, real impairment of orofacial (or stomatognathic) functions has been detected in persons with TMD [4–9].

Conservative methods focusing on pain are recommended for the treatment of TMDs, although, thus far, no modality has been considered to be definitive and not

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all patients respond in a favorable manner. However, a question that has not been properly targeted in most treatment plans is the need to rehabilitate the orofacial motor functions, which are negatively affected in many of the individuals who seek treatment.

As can be easily observed in many investigations, the measurement of the results of functional recovery is limited to an increase of the limits of mandibular movements, associated or not with subjective scales regarding the functional limitations or difficulties. However, this does not necessarily mean that the orofacial functions have been rehabilitated [4, 9]. The relief of pain after antalgic treatment such as low-intensity laser therapy, for example, permits a reduced perception of the difficulty in masticating [10] and an increase of maximal mouth opening [2, 3, 11] and of masticatory efficiency [12], i.e., better food grinding, but does not solve the orofacial myofunctional disorders (OMD) [6, 9].

OMD include changes of orofacial muscles and stomatognathic functions such as mastication, swallowing, and speech that can have a negative impact on oral and general health [13]. An altered motor performance may precede TMD and muscle pain and may also act as a factor in the maintenance of pain [14] or of impairment [15]. Thus, in order to restore the orofacial functions, it is necessary to promote appropriate levels of mobility and coordination of the tongue, lips, cheeks, and jaw muscles, as well as functional recovery in a manner compatible with the occlusion and the temporomandibular joints (TMJs). These are the objectives of orofacial motor therapy, also called orofacial myofunctional therapy (OMT), so that an individual will be able to chew, swallow, and speak without feeling pain and without aggravating the problem [16, 17].

6.2 Orofacial Functions

The orofacial functions involve many other muscles in addition to those of the mandible, which have not been considered in most of the proposed treatments of TMD [9] or even in investigations of the characteristics of affected patients. Thus, these questions should be explored in depth since mastication and swallowing are frequently impaired in patients with TMD.

The ingestion of solid food requires the coordination of various muscles of the orofacial region in terms of amplitude and timing, in order to guarantee appropriate food grinding, the transfer of the bolus, and safe swallowing [18]. During food processing, jaw movements are coordinated with the movements of the tongue, cheek, soft palate, and hyoid bone so that the particles are reduced in size and mixed with saliva [19, 20].

The sensory inputs derived of the orofacial region are used by the central nervous system to define and guide the motor commands so that the food will be processed until it forms a homogeneous bolus [19, 21] and the shape and propulsive force of the tongue will be prepared to transport it efficiently through the pharynx [22]. At the end of the preparation of a food portion, there is a predominance of vertical movements of the mandible and tongue. The bolus is placed on the tongue surface,

the anterior tongue surface first contacts the hard palate just behind the upper incisors, and the area of tongue–palate contact gradually expands backward, squeezing the food back along the palate to the oropharynx [20]. Airway-protective reflexes occur simultaneously in order to prevent aspiration. The swallowing of an unprepared bolus cause risk of aspiration, choking, or an increasing load on the digestive system [23].

Thus, the orofacial functions involve a sophisticated sensorimotor network that combines the activity of various muscles in different ways in order to perform semiautomatic tasks, such as mastication and swallowing, or voluntary tasks such as speech [21]. The orofacial region receives its motor and sensory nerve supplies from the brainstem. The main cranial motor nuclei that control orofacial behaviors are the trigeminal (V), facial (VII), ambiguus [NA, which gives rise to glossopharyngeal (IX) and vagus (X) cranial nerves], and hypoglossal (XII) nerves [18].

The orofacial sensory innervation consists of exteroceptive, proprioceptive, and nociceptive afferent fibers. The main afferent pathway is the trigeminus nerve (V), with sensory fibers from the maxillary and mandibular divisions that have their primary afferent cell bodies in the trigeminal ganglion (CNV ganglion) and send input into the trigeminal sensory nuclei [22, 24]. Visceral afferents such as those supplying lingual, laryngeal, and pharyngeal taste buds and mechanoreceptors project to the solitary tract nucleus in the caudal brainstem [21, 24]. Both the trigeminal brainstem sensory nuclear complex and the solitary tract nucleus relay orofacial sensory information from the brainstem via the thalamus to higher brain areas that include face primary somatosensory cortex (SI) and primary motor area (MI), which in turn project to the brainstem and can thereby exert modulatory influences on orofacial motor activities [21].

The sensory impulses derived from orofacial tissues are relevant to the orofacial motor control, with the integrated participation of the MI in the cerebral cortex, the subcortical regions, and the generators of central patterns in the brain stem in order to satisfy the specificities of each function, the variations in their course, and the coordination, for example, between mastication, breathing, and swallowing [18, 21, 22, 24, 25].

Several variables influence the performance of orofacial functions, such as dental occlusion; the activity of jaw-closing, suprahyoid, facial, and tongue muscles; the TMJs, pain; and age [5, 21–23]. For this reason, a poor performance in patients with TMD should not be attributed only to pain, but should be analyzed in a more encompassing manner. For instance, if the tongue is unable to transport, select, and position the food, the masticatory efficiency may be impaired [20, 23].

Negative conditions may lead to the adaptation of orofacial motor behaviors such as changes in movement, posture, and activity. Some motor adaptations may be beneficial (protection of tissues and structures), whereas others lead to an abnormal tissue overload and injury that stimulate the nociceptors, resulting in susceptibility to the development of a variety of chronic sensorimotor dysfunctions [15]. In turn, nociceptive stimuli such as those occurring in orofacial pain tend to modify the orofacial motor behaviors and to produce cortical neuroplasticity (reorganization of the representation of orofacial structures and changes in excitability) [21], some of

which persist after resolution of pain due to previous experiences that influence the sensory processing and the programming of the efferent motor impulse [25].

Knowledge about the participation of superior cerebral centers in the generation and control of semiautomatic orofacial functions and about the fact that neuroplasticity may underlie various pathological conditions [21] has implications about how alterations in orofacial muscles and function are interpreted. In addition, studies demonstrating that the primary motor cortex (face-M1) can also submit to neuroplasticity according to the learning processes based on novel motor-skill training [21, 24] such as tongue protrusion [26], with advantageous results regarding an increased mechanical performance [14] or precision, can potentially contribute to a rational choice of strategies for orofacial motor treatment.

6.2.1 TMD and Orofacial Myofunctional Status

Patients with TMD show worse orofacial myofunctional conditions, involving the appearance/posture and mobility of lips, mandible, and cheeks, as well as swallowing and mastication, compared to healthy individuals [4, 5, 8, 9, 27].

Facial asymmetry is a common finding in patients with TMD. The lips show changes in appearance and position ranging from non-occlusion (less frequent) to tense occlusion with loss of natural volume that may be due to malocclusion (e.g., overjet) or to dental clenching, thus being associated with a change in mandibular rest.

The tongue is usually positioned on the mouth floor or is interposed between the dental arches due to reduced neuromuscular control or in order to stabilize the mandible at rest and during swallowing, or in order to relieve pain, acting like an occlusal splint. Marks on the tongue as well as the cheeks can be easily observed, with characteristics ranging from hypofunction to hyperfunction [4, 28].

Impaired mobility may be due either to the lack of appropriate use during the functional activities or to the restrictions imposed by the TMJs, or both, and may adversely affect mastication, swallowing, and speech [16]. The reduced precision of the execution of simple movements occurs, in decreasing order of frequency, in the mandible, tongue, lips, and cheeks. The mandible mainly shows the inability to perform ample movements without deviations, and the tongue is unable to contract or to maintain contraction.

The most frequent functional manifestations observed are contraction of perioral muscles, tongue interposition and associated movements suggestive of changes during swallowing [28], and predominance of the unilateral masticatory type [9, 29, 30]. The prevalence of unilateral mastication has been observed in patients with chronic TMD of the disk displacement reduction type (TMD-DDR) of severe degree (80% of cases) or moderate degree (74%), in contrast to the normal alternate bilateral pattern (87%) in control subjects [31].

The main characteristics of the speech of these patients are the reduced movements of the articulators. In addition, a lateral deviation in the opening and closing trajectory of the mandible is not uncommon in patients with disk displacement with

reduction [27], and it may appear in their speech. Another possibility is the occurrence of some premature dental contact causing an adaptation (deviation) in the movement of mandible closure in order to avoid it. Thus, the same occurs in the production of the /s/ and /z/ phonemes when the mandible is closer to the maxilla compared to the production of other phonemes [28].

All of these manifestations can be observed clinically without the need for any type of equipment. However, several resources have been of help for more in-depth research about the relationship between the TMD and the oral motor functions.

According to previous studies, experimental jaw muscle pain rather than stereotyped changes as increased or decreased muscle activity may involve differential effects with redistribution of the activity in the painful muscle itself and among contralateral or agonist muscles according to the task [32]. In addition, despite the reduced bite force, mastication can be maintained with minor compensatory changes, at least if mastication does not exacerbate pain [33].

In other studies conducted on patients with TMD, poor coordination among the masticatory muscles has been observed by surface electromyography during maximum voluntary contraction (MVC) [34] and during mastication [9]. The reorganization of muscle activity for mastication in patients with TMD-DDR was characterized by a reduced ability to differential muscle recruitment and an increased participation of the balance (non-working) side, which worsens with increasing severity of symptoms [31].

Impaired masticatory function has also been detected by three-dimensional kinematic analysis. A recent study reported that patients with chronic TMD-DDR were able to achieve maximum excursions of jaw movements comparable to healthy subjects' performances. However, they showed deviation of the trajectory of mouth opening, asymmetry of mandible laterotrusion, asynchronous condylar translation in jaw-closing and jaw-opening movements, and reduced jaw-closing velocity [27].

These changes in muscle coordination and jaw movements in patients with DDR reflect TMJs impairment, symptomatology severity [31], and the adapted oral motor behaviors [27]. Therefore, regardless of the sequence of factors and events that led to the changes described, when a patient seeks treatment, it is because his stomatognathic system can no longer bear the functional loads without the occurrence of discomfort, pain, or compensations. Thus, OMT is indicated for patients whose orofacial motor behaviors are altered, whether they arose before or due to TMD aiming to reestablish motor strategies.

6.3 Interview and Exams

To plan OMT it is essential to obtain information about the history of patients with TMD and their signs and symptoms, as well as to explain to them the nature of TMD, the treatment modalities, and the results one intends to achieve. Thus, the interview is held in the form of a dialogue rather than simply in the form of questions and answers.

Next, data are obtained using validated protocols such as the protocol for multiprofessional centers for the determination of signs and symptoms of temporomandibular disorders (ProTMDmulti) [35] and the protocol of orofacial myofunctional evaluation with scores (OMES protocol) [5].

The ProTMDmulti is a self-report questionnaire about TMD signs and symptoms, such as muscular pain, joint pain, neck pain, otalgia, tinnitus, ear fullness, tooth sensitivity, joint noise, and difficulty swallowing and speaking. The subject is asked to rate according to their perception of the severity of the signs/symptoms on a scale in which zero indicates absence and ten the highest possible severity. The questions are presented four times so that the subject will answer according to what he feels when he wakes up, when he chews, when he speaks, and when he rests. The sum of the scores attributed to each sign/symptom in the four situations evoked provides information about severity. The total sum score indicates the severity of TMD, which ranges from 0 (absence) to 400 (the highest possible severity). For detailed information see De Felício et al. [35].

The OMES protocol permits the evaluation and determination of orofacial myofunctional status. The assessment is performed by visual inspection and consists of the examination of appearance/posture and mobility of lips, tongue, cheeks, mandible, the configuration of the hard palate, and the breathing, swallowing, and chewing functions [5]. The OMES protocol has predetermined scores, with the highest value indicating the best orofacial myofunctional condition and the lowest value the worst myofunctional degree. The OMES has proved to be useful for OMD diagnosis in clinical practice and research on patients with TMD [4, 8, 27], as well as to determine if there is an effect of treatment on orofacial muscles and functions [6, 9, 36]. For speech evaluation, other protocols focusing on articulatory production are used and should be chosen according to the local language.

The presence of parafunctions or deleterious orofacial habits such as chewing gum; nail biting; object or lip, cheek, and tongue biting; and daytime or nighttime bruxism impairs the equilibrium of the stomatognathic system. Thus, they should be investigated and the patient should be counseled. If necessary, the patient should be referred to a psychologist.

The patients are also asked about their difficulty to chew foods commonly belonging to the Brazilian diet, such as rice and beans, pasta, boiled potatoes, chicken in sauce, bread, steak, apples, peanuts, and barbecued meats. They rate using a 10-point scale in which one indicates the lowest difficulty in chewing and ten the highest. The score of difficulty to chew is obtained by the sum of the points attributed [16]. Patients with TMD discriminate in a very precise manner the degree of difficulty to chew, and the scores they attribute seem to be related to the texture of foods but also to the symptoms and structural and functional conditions of the stomatognathic system. Whereas the scores attributed by healthy individuals to foods such as boiled potatoes and pasta differ little from those attributed to peanuts and barbecued meats, these differences are considerable in the replies of patients.

In addition to the above data, the diagnosis of TMD is fundamental for the planning of OMT. In our group, clinical data were obtained using the research

diagnostic criteria for TMD, axis I, and currently the diagnosis is performed in accordance with the "diagnostic criteria for the most common pain-related temporomandibular disorders" (DC/TMD) [37].

Also, the results of analysis of functional occlusion are necessary in order to understand some functional impairments such as unilateral chewing preference due to occlusal interference and to plan the rehabilitation of functions without causing additional traumas.

6.3.1 Complementary Exams

In clinical practice, noninvasive complementary exams are performed for a better understanding of muscle function or dysfunction whenever necessary, and those most frequently used in our laboratory are listed below:

6.3.1.1 Surface Electromyography (sEMG)

sEMG is useful to elucidate the masticatory muscle function and adaptation in patients with TMD [31]. The analysis of the anterior temporal and masseter muscles in MVC is performed using the sEMG standardization protocol described by Ferrario et al. [38] which permits the determination of the indices of symmetry between the left and right temporalis anterior (POC_T) and masseter muscles (POC_M), potential lateral displacing of components given by unbalanced contractile activities of contralateral masseter and temporalis muscles (POC_{TORS}), and the total standardized muscle activities (IMPACT). Another index is used to assess the degree of cooperation between bilateral temporal and masseter muscles' activities (POC_{TM}) [31].

In the sEMG analysis of mastication, the activity of the four muscles (left and right temporalis anterior and masseter) is recorded during the task of 15-s unilateral, left, and right chewing of a pre-softened sugarless gum. The quantitative bivariate analysis of the differential EMG signals of the paired masseter and temporal muscles is performed using the Lissajous plot, a method proposed by Kumai [39] to graphically show the cooperative work of masticatory muscles, and later developed including its standardization [40].

6.3.1.2 Mandibular Kinematics

Three-dimensional (3D) kinematic analysis is used as a supporting method to deepen the comprehension of oral motor control and TMJ function [27]. Mandibular and condylar kinematics are analyzed during free maximum jaw movements—opening-closing, protrusion, bilateral laterotrusions, and unilateral chewing.

6.3.1.3 Tongue and Lip Strength

Because of the relevance of the tongue and lips for the execution of orofacial functions, the tongue pressure that an individual can produce by pressing a standardized air-filled bulb is measured, during anterior tongue elevation, tongue protrusion, and

swallowing, in kilopascals (kPa). Lip pressure is measured with the individual pressing lips together with maximum effort, according to a previously published method [41].

Once the assessment is completed, the results must be organized, related, and interpreted in order to explain them to the patient and to elaborate a treatment plan.

6.4 Treatment

Exercised-based treatment has been long considered relevant for the rehabilitation of patients with musculoskeletal disorders and a wide variety of painful conditions [42] because it reduces the sensitivity to noxious stimuli or pain perception and it improves the main symptoms of TMD, the self-perception of functional impairment, and the limits of mandibular movements [43, 44].

However, since most patients with TMD, especially those with the chronic form of the condition, have deficits in orofacial motor skills and maladapted functional processes, it is also necessary to promote an appropriate motor control and to reverse maladapted behaviors for establishing a protective adaptation.

For these reasons, OMT is an exercise-based modality which includes strategies for lips, tongue, cheeks, and mandible aiming at the recovery of voluntary contraction and the mobility and coordination of each component, as well the training of mastication, swallowing, breathing, and speech when appropriate. Therefore, it differs but is not conflicting with the physiotherapy. In Brazil, OMT is the responsibility of speech pathologists. The degree of muscle and orofacial function impairment is the primary point considered for the determination, or not, of OMT.

The OMT program previously proposed for TMD [16] involves the following objectives:

- To guide the patients and to instruct them about TMD and treatment
- To minimize or eliminate the symptoms
- To habilitate the muscles to perform precise and coordinated movements
- To rehabilitate the orofacial functions

The OMT program lasts 120 days. The sessions with the therapist are held once a week during the first month and every 15 days thereafter. The patient should practice his training at home on a daily basis. Improvement of orofacial myofunctional status, reduction of pain upon palpation, reduction of symptoms, and increased limits of mandibular movements have been detected when the program is applied as a single treatment [4, 9] or in combination with laser therapy [9, 36]. OMT may also be combined with dental, psychological, or physiotherapeutic treatment.

Basic directions are given below, with emphasis on the fact that the first two objectives are appropriate for all types of TMD diagnosis and, in general, are included in various proposed treatments in which the strategies may vary.

6.4.1 Explaining TMD and Treatment to the Patients and Providing Guidance

It is necessary to explain the set of signs and symptoms of TMD to the patients so that they will understand, among other things, that they are part of a single condition rather than representing multiple problems, as the patients often feel in our clinical practice.

Guidelines about the possible adverse effects of parafunctional habits are basic to interrupt it. Also, the explanations about the structure and function of the stomatognathic system, the damage detected during the assessment, the way the treatment will be applied, and the general and individual objectives of each therapeutic strategy permit better patient adherence to and engagement in treatment. These orientations are essential in exercise-based therapies, in which the patient must participate in an active manner on a daily basis. Starting during the phase of interview and assessment, guidelines will continue to be offered in various situations in the course of OMT.

6.4.2 How to Minimize or Eliminate the Symptoms

The orofacial pain is the first symptom to be minimized and, if possible, eliminated because it represents the main expectation of the patients regarding treatment. Also, the presence of pain causes difficulty to orofacial exercising although it does not prevent their execution if performed with caution. The resources presented below are employed together in order to favor blood flow, muscle tissue oxygenation, and the elimination of metabolic residues. Besides, they contribute to the reestablishment of the resting posture of the mandible when patients kept the teeth in occlusion instead of maintaining free-way space and the vertical dimension at rest (VDR).

Warm compresses are applied to the craniofacial muscles and possibly extended to the cervical region and the shoulders. The recommended duration of application is 20 min, and the temperature should be tested by hand before applying the compress to the face. The compresses could be replaced with commercially available hot packs.

Massage the masseter, and temporal muscles are massaged with moderate digital pressure and with circular movements for 10 min after heat therapy. Massage is contraindicated in the region of the temporal artery and in cases in which trigger points are present, so that headache or myofascial pain will not be induced [16, 17].

Relaxation of the neck and shoulders involves a sequence of slowly executed rotation movements of the shoulders and then of the neck, which are repeated 5–10 times each [16, 30].

Relaxation of jaw muscles. To promote relaxation of mandible elevator muscles, the patient is instructed to occlude his lips gently and disocclude his teeth maintaining free-way space and VDR. Then the patient puts his anterior surface of the tongue in contact with the palatal rugae and slowly slides forward and backward without exerting pressure or elevating the tongue apex [28].

During this exercise, there should be no mandibular movements or contraction of the submental muscles, except when the patient swallows. When the patient starts it, the lips can be kept disoccluded to permit the observation of tongue positioning and movement. The exercise lasts 5 min, and this time can be increased to 15 min, with brief intervals, with at least three repetitions per day [16].

As mentioned earlier, these therapeutic resources are of help for the reestablishment of the resting posture of the mandible. Since many persons do not even know whether they keep their teeth occluded and should not do so, it is also relevant to include in the therapy sessions some time for self-perception of the level of muscle contraction and the mandibular and body posture induced by the therapist's verbal instructions.

The sessions start with the strategies described above. As the patient masters the techniques and practices them at home, and pain, as well as other symptoms, decrease, these take up less time during the session, and the duration of the orofacial motor exercises increases.

6.4.3 Orofacial Motor Exercises

6.4.3.1 Preparation for Precise Movements and Orofacial Functions

This stage mainly consists of training in order to achieve precise and symmetrical movements with appropriate levels of contraction, followed by coordination of the lips, tongue, cheeks, and mandible. When a subject is unable to follow the model needed for a mobility task due to deficient proprioception or to the inability to perform the necessary levels of muscle contraction, the exercises could be preceded by tactile stimulation. The stimulus is applied according to the inability detected, with light pressure and rapid movements. Two examples are given below:

Inability to maintain the lips occluded

- The subject should be asked to occlude his lips and then the bristles of a plastic toothbrush should be passed from his mentolabial sulcus to the vermilion line of the lower lip. Counter-resistance is applied to the mentalis muscle to prevent it from aiding the elevation of the lower lip [28]. This maneuver is employed after the determination of absence of nasal obstruction, which requires compensating inspiration through the mouth.

Inability to sharpen and protrude the tongue

- Ask the subject to open his mouth and place the tongue apex on the right labial commissure. Pass the bristles of a toothbrush from the posterior region of the tongue to its apex. The stimulation is repeated with the tongue apex on the left labial comissure.
- Ask the subject to open his mouth and to try to protrude his tongue. Using two toothbrushes, stimulate the margins of the tongue from the posterior region to the apex.

6.4.3.2 Mobility Exercises

Instructions: the movements to be trained are those, which the patient was unable to perform with precision during his evaluation. According to the specificity principle, during the first stage, each structure is trained separately. All movements should be carried out slowly, and, when the target is reached, the position should be maintained for 3 s. In general, contraction is followed by an equal time of rest and then by another contraction. The number of repetitions is stipulated by avoiding to reach the fatigue limit, which can be observed based on the difficulty to continue the task with precision or in the presence of tremor. During the exercises for the lips, tongue, and cheeks, a thumb should be kept under the chin to support the mandible and to prevent undesirable movements. Also, to prevent a compensatory participation of the lips during the tongue exercises, the chin should be slightly pulled downward with the index finger. The therapist instructs and presents a model so that the patient will be able to perform the exercises. Training is conducted with the subject sitting on a chair with a backrest, with his back straight, and without deviation of the head. Support with a mirror is essential, especially at the beginning of each new task. Home training should be performed at least three times a day, and therefore the number of weekly exercises should not be excessive. The execution of the exercises should not produce noise in the TMJs or pain during practice or after it. Monitoring by the therapist should be frequent.

Lips

- Protruding the lips joined
- Lateralizing the joined lips to the right
- Lateralizing the joined lips to the left

Tongue

In the following exercises, the tongue should perform movements without any aid of the lips.

- Sharpening and protruding the tongue
- Placing the tongue apex on the right labial commissure and then on the left, always with the apex orientate upwards, with no resting interval until 20 repetitions are performed
- Raising the tongue until the apex touches the midpoint of the vermilion line of the upper lip
- Lowering the tongue until the apex touches the midpoint of the vermilion line of the lower lip

Cheeks

- With the lips occluded and the mandible at rest, air should be held in the mouth and transferred to the cheek from the right to the left side, with alternate cheek inflation and without a resting interval until achieving 20 repetitions.

Mandible

Performing repeated mandibular movement without deviations:

- Opening the mouth about 20 mm and closing it.
- Opening the mouth to a comfortable limit and closing it, keeping the tongue apex in contact with the incisive papilla. If there is deviation of the movements, the tongue apex should be slightly shifted in the opposite direction [45]. The best positioning in order to achieve symmetrical movements should be established with the therapist. A prerequisite for the execution of this exercise is the ability to maintain tongue elevation [16].
- Lateralizing the mandible starting from the midline until reaching the canine guide (when present). Repeating up to ten times for the right side and then for the left. In order to avoid occlusal interference during this movement, the use of an occlusion splint or the placement of a 9 mm-thick latex tube between the upper and lower incisors as if it were a soda straw is recommended. For the rightward and leftward movements, when there is asymmetry between sides, the extension of the movement, the side, as well as the number of repetitions can be adapted to the needs of the patient.

6.4.3.3 Force and Resistance Exercises

To strengthen the orofacial musculature training against resistance can be applied [46, 47]. Sustained contraction also has the potential to improve muscular endurance. However, the mobility must be adequate, with precise movements to prevent compensations.

Tongue

- Sharpening and protruding the tongue, maintaining protrusion for 3 s. Time should be gradually increased according to the ability of the patient to maintain voluntary contraction [16, 17].
- Coupling the tongue to the palate, lowering the mandible, and elevating it until the upper and lower lips touch, without occluding the teeth. The number of repetitions depends on the ability of the subject to maintain coupling and should be increased up to 20 times. This training is indicated to achieve correct positioning of the tongue at rest and during swallowing and should start with sequential movements opening and closing the mouth without intervals. Posteriorly, jaw opening is maintained for 10 s.

Cheeks

- Resisting with the cheek, a non-intense force applied from inside out using a tongue depressor. Excessive mouth opening should be avoided.

Jaw Muscles

- Biting a latex tube, keeping it compressed for 3 s. For better muscle engagement, the thickness of the tube can be increased from 5 to 9 mm. In cases of muscle asymmetry, training can be performed only on the side with deficient muscle function.

6.4.3.4 Coordination Exercises

- Performing coordinated movements of the tongue, cheeks, and lips in order to transfer solid food, such as a small piece of candy from the right to the left oral vestibule and vice versa
- Speech training with monitoring of movement amplitude and articulatory precision

6.4.4 Function Training

During the OMT process, there is a hierarchy, but in some phases, the goals intertwine, e.g., breathing training can be performed together with the strategies for the relief of symptoms. Also, it is not possible to follow all the previous steps and only later instruct the patients about mastication.

6.4.4.1 Respiration

The patient should breathe in and out through the nose and simultaneously performs diaphragmatic breathing. During the initial phase, ideally the patient should lie down or be in a reclining position.

6.4.4.2 Mastication

Instructions about mastication should be provided since the initial phase of treatment because people eat several times a day and should do so in the least possible traumatic manner. According to our experience, because of pain and a sensation of difficulty to chew, persons with TMD tend to choose foods that do not require masticatory force, so that it is seldom necessary to instruct them about consuming a soft diet. Thus, work with these patients is directed at masticatory movements and types. It should be kept in mind that unilateral mastication predominates among patients with TMD [8, 9, 29], whether as a preference or as a chronic pattern [31], whereas the ideal type is alternate bilateral mastication.

However, it is not always possible to reach the ideal pattern. Thus, the goals of rehabilitation of mastication and swallowing should take into account the dynamics of the relationship between occlusions and muscles. From the viewpoint of occlusion, there should be bilateral occlusal guides and no occlusal interference, mainly on the balance side [16]. From the viewpoint of the muscles, there should be muscle equilibrium and coordination in order to transfer food from one side to the other of the oral cavity, to grind it, and to transport it to the oropharynx [18, 20]. Also, during or after mastication, the patients should have either joint noise or pain [16].

Failure to satisfy these requirements may represent an aggravating factor for TMD. For this reason, eventually, during the initial phase, the patient should be helped to change from the unilateral masticatory pattern to the simultaneous bilateral with a predominance of vertical mandibular movements over lateral ones. At the course of TMO, it is possible to overcome muscular limitations, pain, and often the presence of noise during mastication. Thus, if no occlusal impediments are

present, training in bilateral and alternate chewing will be applied. Training is performed with foods of different textures so that mastication will be appropriate for the most diverse situations.

6.4.4.3 Swallowing

Mobility and resistance training and the establishment of resting posture are preparatory for swallowing. Moreover, the pain relief and a better bolus preparation during mastication favor this function. For specific training of swallowing, when a subject masters the exercise of the tongue coupling to the palate, he is instructed to occlude his lips and teeth and to swallow saliva when he feels the need for it, maintaining the positioning of the tongue. However, before establishing this type of training, it is necessary to determine if there is no premature occlusion or reduced vertical dimension, which can potentially cause muscle adaptations.

6.4.4.4 Speech

Mobility exercises potentiate speech articulation. Additionally, speech training with precise movements is included in some sessions, without an exaggerated amplitude.

Upon completion of the OMT program, all questionnaires, evaluations, and exams are applied again in order to verify the effects of treatment. Thus, the therapeutic exercises will be discontinued, because the acquired news orofacial motor patterns will function as protectors of the stomatognathic system. According to the previous study, the positive results were maintained in the 3-month follow-up [9].

Finally, further studies on orofacial motor rehabilitation are required. On the one hand, it is necessary to verify if the OMT program here presented promotes neuroplasticity to the primary motor area (MI) and the primary somatosensory area (SI) that are involved in sensorimotor integration and control of orofacial motor functions [21]. On the other hand, the paradigm based on novel motor-skill training which successfully induces neuroplasticity in corticomotor control [10, 21, 24, 26] needs to be tested on patients who have orofacial motor disorders and analyzed the long-term effect.

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Part IV

Psychiatric Considerations and Adjunctive Therapies

Adjunctive Therapies for Temporomandibular Disorders

7

César Fernández-de-las-Peñas and Kimberly Bensen

Abstract

Proper management of patients with temporomandibular pain disorders (TMD) needs a multidisciplinary approach including several professionals such as medical doctors, dentists, physical therapists, osteopaths, chiropractors, acupuncturists, orthodontists, surgeons, and psychologists [1]. Each professional will conduct an exhaustive clinical examination and will plan a multimodal program for these patients. In fact, treatment should be personalized based on the patient's experience and including different therapeutic strategies such as passive and active techniques, active listening, empathy, and management of psychosocial issues, i.e., depression, anxiety, and catastrophizing, always based on clinical findings during the history and examination. For instance, dentists could recommend hard stabilization appliances [2] or splint therapy [3] for reducing pain in patients with TMD, whereas physical therapists can apply manual therapies or exercises for the same objective. Clinical and scientific evidence suggest that an interdisciplinary work between dentistry and physical therapy increases the effects of each isolated intervention for patients with TMD [4]. The current chapter summarizes different adjunctive therapies including physical therapy and chiropractic therapy for TMD.

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7.1 Introduction

Proper management of patients with temporomandibular pain disorders (TMD) needs a multidisciplinary approach including several professionals such as medical doctors, dentists, physical therapists, osteopaths, chiropractors, acupuncturists, orthodontists, surgeons, and psychologists [1]. Each professional will conduct an exhaustive clinical examination and will plan a multimodal program for these patients. In fact, treatment should be personalized based on the patient's experience and including different therapeutic strategies such as passive and active techniques, active listening, empathy, and management of psychosocial issues, i.e., depression, anxiety, and catastrophizing, always based on clinical findings during the history and examination. For instance, dentists could recommend hard stabilization appliances [2] or splint therapy [3] for reducing pain in patients with TMD, whereas physical therapists can apply manual therapies or exercises for the same objective. Clinical and scientific evidence suggest that an interdisciplinary work between dentistry and physical therapy increases the effects of each isolated intervention for patients with TMD [4]. The current chapter summarizes different adjunctive therapies including physical therapy and chiropractic therapy for TMD.

7.2 Physical Therapy and Temporomandibular Disorders

Physical therapy include different physical agents for the management of TMD, e.g., manual therapy, therapeutic exercise, dry needling, electro-physical modalities (e.g., low-level laser, therapeutic ultrasound), or electro-analgesic modalities such as transcutaneous electrical nerve stimulation (TENS). Since TMD can affect different structures or tissues such as the temporomandibular joint (TMJ), the masticatory musculature, or their associated tissues (e.g., ligaments, connective tissues), the appropriate technique would depend on the affected tissue responsible of nociception. Physical therapy has evolved from a biomechanical point of view to a neurophysiological conceptualization. In this updated scenario, clinicians should consider potential neurophysiologic and tissue mechanisms underlying the effects of any intervention that they will apply on each patient. In fact, the challenge for clinicians is how to select proper treatment for each patient who is likely to be different with an individual clinical presentation. An important topic to consider for determining the proper therapeutic program is to determine if the clinical pattern of a patient exhibits a more peripheral or more central input dominance. This clinical reasoning is based on better understanding of nociceptive mechanisms in patients with chronic pain. Pfau et al. described two subgroups of patients with TMD grouped by their sensitization mechanism: one group (sensitive) exhibiting more central sensitization and other (nonsensitive) group showing more peripheral sensitization [5]. This classification agrees with literature supporting that identification of nociceptive processing is highly

important since the presence of central sensitization can constitute a worse prognosis factor for physical therapy [6] and also may determine the dose of the interventions, e.g., intensity, amplitude, and frequency of the sessions [7]. Therefore, it seems that identification of sensitization mechanisms in patients with TMD implies a complex clinical reasoning [8].

There is evidence suggesting that individuals with TMD exhibit widespread pressure pain hypersensitivity over different tissues, e.g., muscle, joint, and nerve [9], as a manifestation of central sensitization; nevertheless, the magnitude of this sensitization is higher within the trigeminal area since widespread pressure hypersensitivity is associated with the intensity and duration of the symptoms in the orofacial area [5]. Therefore, if a particular patient with TMD seems to be more mediated by peripheral nociception, specific treatment of the affected tissue and application of exercises and functional activities should be encouraged. In this patient, localized treatments targeting muscles or joints of the TMJ responsible of nociception would be the first therapeutic step for decreasing central sensitization. If a patient with TMD seems to be more centrally mediated, a multimodal approach including pharmacological, physical, and cognitive interventions is encouraged. In these patients, it may be not possible to clearly identify a particular tissue responsible for nociception; therefore, patients should be also educated on optimizing normal functional movements and on undertaking active and specific exercises, in combination with proper passive manual therapies and educational programs.

7.3 Scientific Evidence for Physical Therapy Modalities

A national survey conducted in the United Kingdom showed that, despite limited evidence, physical therapy is usually considered to be an effective treatment option for TMD, with jaw exercises (79%), therapeutic ultrasound (52%), manual therapy (48%), acupuncture (41%), and laser therapy (15%) being considered the most effective modalities for managing TMD [10]. This section chronologically reviews scientific evidence for several physical therapy therapeutic options for the management of patients with TMD.

An old review (1999) found little evidence supporting the use of ultrasound therapy in the treatment of musculoskeletal disorders [11]. This review analyzed 13 randomized placebo clinical trials, of which 4 included individuals with TMD. Most studies did not clinically or statistically support the use of ultrasound therapy for TMD [7].

In 2006, McNeely et al. found few studies investigating the effectiveness of manual therapies for the management of TMD and concluded that the methodological quality of these studies was poor [12]. This review concluded that the use of manual therapies combined with active exercises may be effective for reducing pain and improving function in TMD, although more high-quality studies are clearly needed [8]. Another systematic review also published in 2006 concluded that active

exercises combined with manual mobilizations may be effective for TMD and that postural training may be used in combination with other interventions [13]. Again, authors also pointed out for the low methodological quality of the available trials since most papers included were case series [9].

List and Axelsson, in a critical review of systematic reviews, analyzed the evidence of 23 qualitative systematic reviews and seven meta-analyses regarding the management of TMD [14]. These authors investigated the results of reviews on occlusal appliances, occlusal adjustments, bruxism, physical therapy, drug treatment, surgery, and behavioral therapy and concluded that there is limited evidence supporting the application of occlusal appliances, acupuncture, behavioral therapy, exercise, and pharmacological drug treatment for TMD [10]. An important conclusion of this review was that occlusal adjustments seem to have no effect and should be avoided [10].

Brantingham et al. conducted, in 2013, a systematic review focusing on manual and manipulative therapy conducted by chiropractors and concluded that there is limited evidence that manual and manipulative therapy may be helpful at short-term (≤3–6 months) for TMD [15].

The review by Calixtre et al. published in 2015 aimed to determine the effectiveness of manual therapies in isolation, not the application of multimodal treatments [16]. This review found low to moderate evidence supporting that myofascial release and massage applied on the masticatory muscles and spinal manipulative therapy applied to the upper cervical spine are more effective than control for the management of TMD pain [12].

The most recent meta-analysis analyzed the effects of manual therapy and exercises for the management of TMD symptoms. Armijo et al. concluded that manual therapy alone or combined with exercises at the jaw or cervical spine showed promising benefits for treatment of TMD, although effect sizes were low to moderate and related to the kind of TMD [17]. This meta-analysis found that:

1. Manual therapy significantly reduced pain (mean difference, 1.35 cm; 95% CI 0.91–1.78) at short-term compared with botulinum toxin or waiting list (mean difference, 1.31 cm; 95% CI 0.86–1.76) in myofascial TMD.
2. Head and neck exercises were significantly effective for increasing pain-free maximum mouth opening (mean difference, 5.94 mm; 95% CI 1.0–12.9) and decreasing pain (standardized pooled mean difference, 0.43; 95% CI 0.02–0.87) compared with a control group in myofascial TMD.
3. A trend, but not significant, effect of exercise (standardized pooled mean difference, 0.68; 95% CI −0.04, 1.40) against a control group was observed for arthrogenous TMD, including disc displacement with or without reduction.
4. Posture correction exercises were significantly more effective for increasing maximum pain-free mouth opening (mean difference, 5.54 mm; 95% CI 2.93–8.15) and for decreasing symptoms and disturbances with daily life activities (standardized pooled mean difference, 1.13; 95% CI 0.48–1.78) than a control group for myofascial TMD [13].

No significant adverse event was observed with any manual therapy procedure [13].

The meta-analysis conducted by Martin et al. also showed significant differences and large clinical effects on active, but not passive, mouth opening (standardized pooled mean difference, 0.83; 95% CI 0.42–1.25) and on pain during active mouth opening (standardized pooled mean difference, 1.7; 95% CI 1.1–2.3) favoring musculoskeletal manual techniques compared to other conservative treatments for TMD [18]. Although current evidence clearly supports that manual therapies and exercise can be effective for the management of TMD, it is not possible to draw any firm conclusion about the most effective techniques to improve pain and range of motion. This is related to the fact that most studies, as it is commonly done in clinical practice, have combined articular and soft tissue techniques [8–14].

A recent systematic review [19] and a meta-analysis [20] observed a moderate clinical effect (standardized pooled mean difference, −0.6; 95% CI −0.73, −0.47) for the application of low-level laser (dosages and treatments with wavelengths of 780 and 830 nm) on the masticatory muscles or joint capsule for TMD, although the optimal parameters are still controversial.

7.4 Manual Therapies

The American Academy of Orthopedic Manual Physical Therapists (AAOMPT) defines manual therapy/orthopedic manual physical therapy as "any hands-on treatment provided by the physical therapist." Manual therapy may include moving joints in specific directions, at a different speed and amplitude (joint mobilization and manipulation), soft tissue interventions, stretching interventions, passive movements of the affected body part, or having the patient move the body part against the therapist's resistance to improve muscle activation and timing (exercises) [21]. Manual therapies have been traditionally used for increasing restricted range of motion, reduce local ischemia, stimulate proprioception, break fibrous adhesions, stimulate synovial fluid production, and reduce pain symptoms. Nevertheless, recent theories suggest that manual therapy-induced effects are caused by neurophysiological mechanisms including the activation of periaqueductal gray substance; lessening of temporal summation; reduction of nociceptive substances, e.g., cytokines and substance P; and changes in muscle activity and motor-neuron pool activity [22].

Several manual therapies are clinically proposed for the management of TMD pain. In fact, patients with TMD can be treated with manual therapies targeting the TMJ (Fig. 7.1), the musculature (Fig. 7.2), or the neural tissues (Fig. 7.3), depending on the structure responsible of nociception. Additionally, preliminary evidence suggests the effectiveness of the inclusion of manual therapies targeting the cervical spine (Fig. 7.4) for the management of individuals with TMD [23, 24]. Since the cervical spine is biomechanically and neurophysiologically associated with the thoracic spine, some authors proposed the inclusion of thoracic spine manipulation (Fig. 7.5), although the isolated application of upper thoracic spine thrust manipulation did not lead to a reduction in pain in TMD [25].

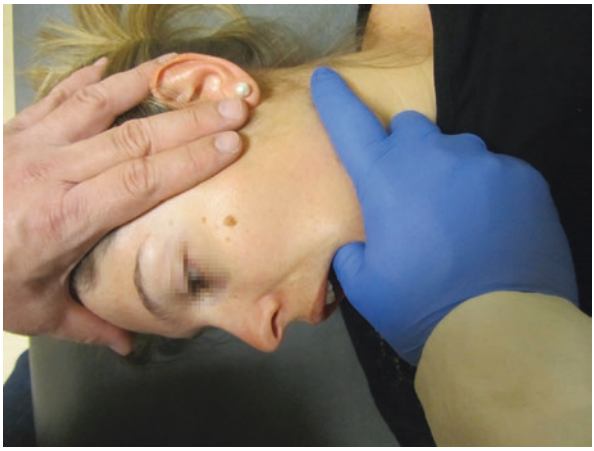

Fig. 7.1 Mandibular distraction mobilization technique

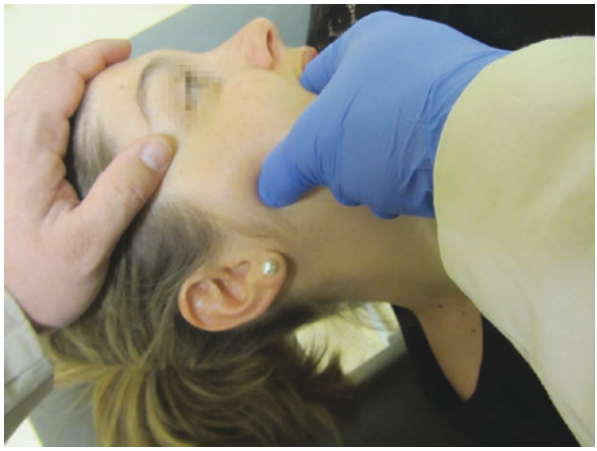

Fig. 7.2 Intraoral massage of the masseter muscle

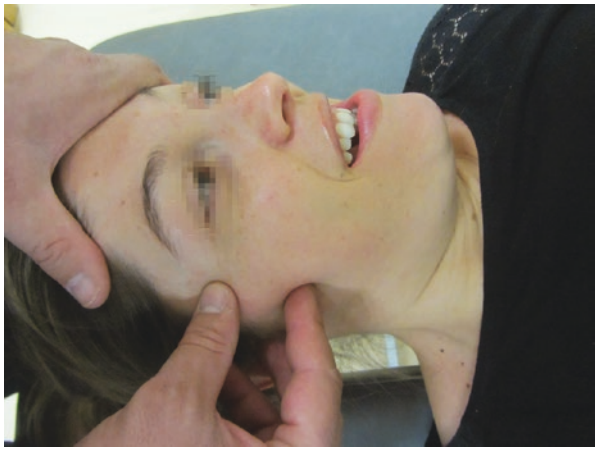

Fig. 7.3 Neural mobilization of the right trigeminal nerve

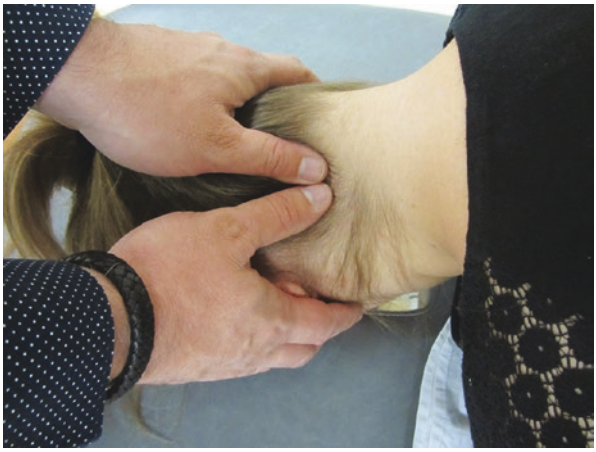

Fig. 7.4 Posterior-anterior joint mobilization of the upper cervical spine

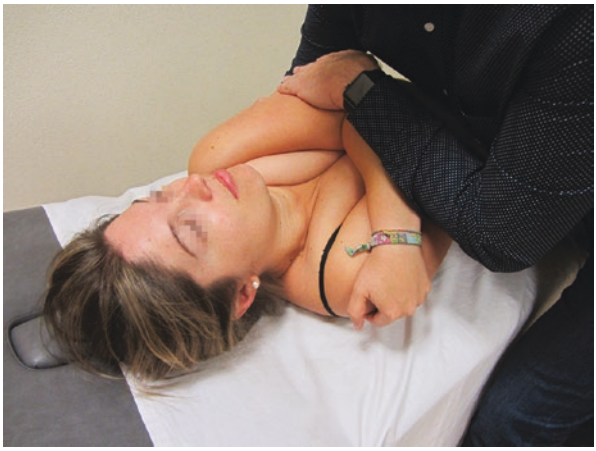

Fig. 7.5 Upper thoracic spine thrust manipulation

It is important to consider that common clinical practice use different manual therapies with other interventions such as exercise and educational program. This multimodal approach is supported by current scientific evidence where combination of manual therapy, education, and exercise has demonstrated to be effective for myofascial TMD [26–28], TMJ arthralgia [29], or anterior disc displacement without reduction (closed lock) [30].

7.5 Exercise Programs

Therapeutic exercise interventions are prescribed to address specific TMJ impairments and to improve the function of the cranio-cervico-mandibular system. Exercise programs should be designed to improve muscle coordination, relax

hypertonic muscles, increase range of motion, and increase muscle proprioception. Recent evidence supports the use of exercises to correct head and neck posture (Fig. 7.6) and active oral exercises (Fig. 7.7) for reducing TMD pain and improving orofacial motor function [13]; however, no information regarding dosage, frequency, or adherence to exercise programs are currently available. Further, there is no trial investigating the effectiveness of the isolated application of therapeutic exercises for TMD pain; so we do not currently know the effects of this intervention on isolation.

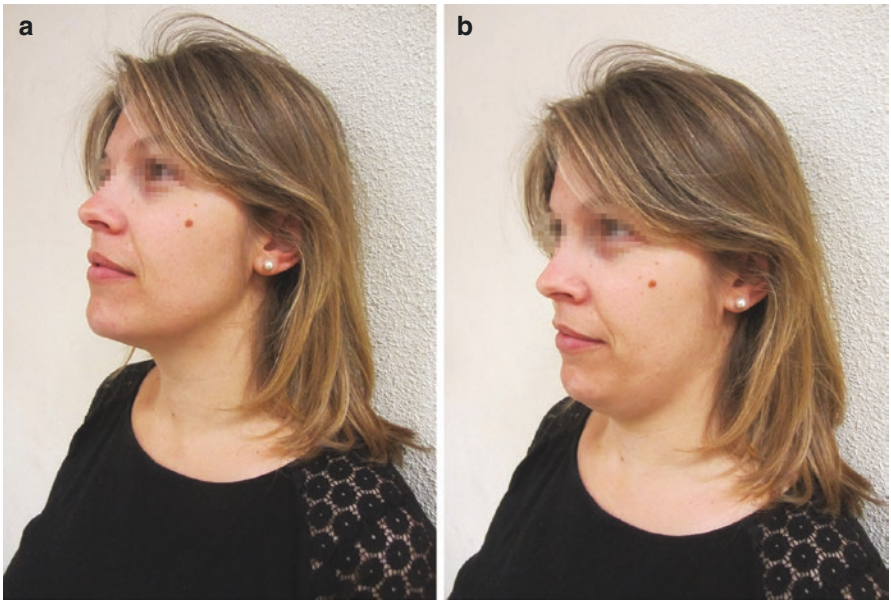

Fig. 7.6 Corrective exercise for forward head posture against the wall

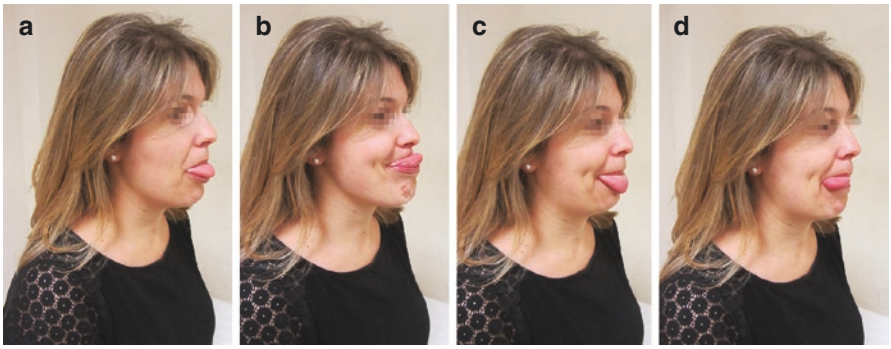

Fig. 7.7 Active oral exercise with the tongue

In addition to specific therapeutic exercises, general aerobic exercise has been shown to improve strength, flexibility, and functional capacity and also induce analgesia [31]. Nevertheless, it is important to note that patients with central sensitization, such as TMD, can exhibit an abnormal pain threshold response to exercise since aerobic exercise usually exerts exercise-related hypoalgesia by activating the descending inhibitory pain mechanisms. In individuals with TMD pain exhibiting central sensitization, this situation is the opposite; exercise induces hyperalgesia [32]. Therefore, clinicians need to assess these exercise-induced mechanisms, since activation of descending inhibitory pathways will be extremely helpful during the treatment process of patients with myofascial TMD.

7.6 Trigger Point Dry Needling

Different needling therapies can be applied in individuals with TMD depending on the health-care profession: acupuncture (acupuncturists), dry needling (physical therapists), and botulinum toxin type A (medical doctors or dentists) [33].

The American Physical Therapy Association (APTA) defines dry needling as "skilled intervention using a thin filiform needle to penetrate the skin that stimulates TrPs, muscle, and connective tissue for the management of musculoskeletal pain disorders" [34]. Clinical rational for application of dry needling is that the referred pain elicited by active trigger points in the head and neck muscles reproduces the pain symptoms in patients with TMD [35]. Recent meta-analyses support the use of trigger point dry needling for reducing pain in upper quadrant syndromes [36] and multiple body areas [37], including myofascial TMD pain.

Several studies have demonstrated that trigger point dry needling of different muscles, such as the lateral pterygoid or masseter (Fig. 7.8), is effective for reducing pain and increasing active mouth opening in individuals with myofascial TMD [38–40] or sleep bruxism [41]. A small study found that trigger point dry needling was

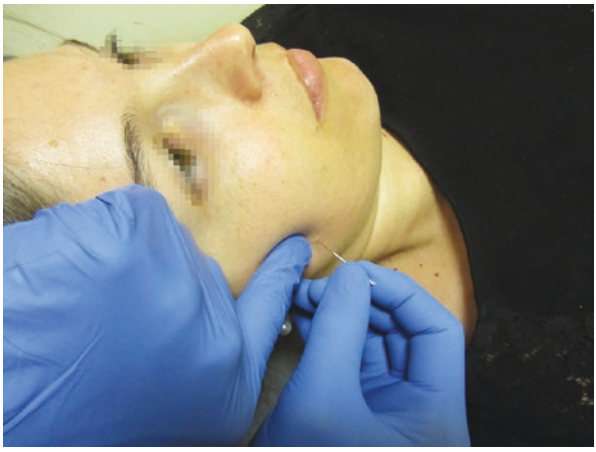

Fig. 7.8 Dry needling of trigger points in the right masseter muscle

also effective for improving TMD-related symptoms such as tinnitus [42]. Interestingly, trigger point dry needling has shown better effects for reducing pain and improving jaw movements compared with methocarbamol/paracetamol drug treatment [43, 44].

An important topic to discuss is that the effect of dry needling arises in the own needle and not in any substance associated with it. This is supported by several studies demonstrating similar outcomes after application of trigger point dry needling and injections of lidocaine or botulinum toxin A in patients with TMD and headaches [45–47]. In fact, a meta-analysis found no significant differences in pain between dry needling and lidocaine injections immediately (standardized pooled mean difference, 0.41; 95% CI −0.15, 0.97), at 1 month (−1.46, 95% CI −2.04, 4.96) and 3–6 months (−0.28, 95% CI −0.63, 0.07) after the treatment [48].

Nevertheless, as manual therapies, trigger point dry needling should be integrated into a multimodal management [49].

7.7 Low-Level Laser Therapy

Several authors have proposed the application of low-level laser (i.e., application of light amplification by stimulated emission of radiation) for the management of patients with TMD. The basic therapeutic effects of low-level laser include biostimulation, regeneration, antinociception, and anti-inflammatory of the affected tissues [50]. Others have reported that low-level laser could affect the synthesis of prostaglandin, causing arachidonic acid to enter endothelial tissues allowing them to generate vasodilatation and anti-inflammation [51].

Evidence supports the use of low-level laser therapy over the masseter, temporalis (Fig. 7.9), and pterygoid muscles for the management of TMD [15, 16]. Additionally, low-level laser seems to be more effective than other modalities such as transcutaneous electrical neural stimulation (TENS) [52]. However, the most controversial topics are the number of session, the type of laser, time of application,

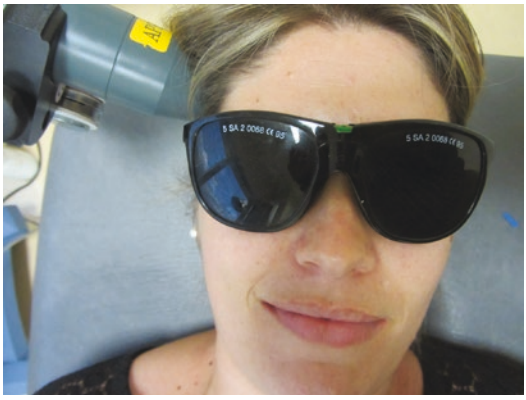

Fig. 7.9 Application of low-level laser over the right lateral pterygoid muscle

and proper dose since various doses, methods, and modes of low-level laser could also result in different treatment effects. In fact, the radiation dosage is determined by the irradiation time and treatment course. The key for an effective treatment is the adequacy of the dosage delivered to the tissue.

The light used with low-level laser is typically of narrow spectral width in the red or near-infrared spectrum (600–1,000 nm), with a power density (irradiance) between 1 mW and 5 W/cm^2. Previous studies investigating the effects of low-level laser in TMD patients have used different doses: (1) total dose, 4 J/cm^2; wavelength, 904 nm; intensity, 0.6 W; time, 60 s [53], or (2) total dose, 5 J/cm^2; wavelength, 780 nm; intensity, 20 mW; time, 10 s [54]. The heterogeneity of laser application is manifested in the review conducted by Maia et al. who observed that the energy density used in published studies ranged from 0.9 to 105 J/cm^2, while the power density ranged from 9.8 to 500 mW [55]. In addition, the number of sessions varied from 1 to 20, whereas the frequency of applications ranged from daily for 10 days to 1 time per week for 4 weeks [51].

As most physical therapy interventions, low-level laser combined with exercises was more effective for TMD rehabilitation than low-level laser alone [56].

7.8 Therapeutic Ultrasound

It has been proposed that therapeutic ultrasound can reduce inflammation, promote muscular relaxation, increase blood flow, and induce analgesia by increasing the temperature in the targeted tissue. Based on these effects, therapeutic ultrasound is clinically applied over the masticatory musculature. Nevertheless, current evidence for therapeutic ultrasound in individuals with TMD is lacking and controversial. It seems that the isolated application of therapeutic ultrasound is not effective for TMD pain [7]; however, the combination of home exercise with therapeutic ultrasound was more effective for decreasing pain and increasing mouth opening than home exercises alone for patients with TMD [57]. Again, the topic of dose and time of application remains unknown.

7.9 Transcutaneous Electrical Neural Stimulation (TENS)

The application of TENS consists of a controlled exposure of electrical current to the surface of the skin, causing muscle relaxation and decrease in pain. It is usually applied with an electronic device producing pulsed biphasic electrical waves through the electrodes placed on the skin surface [58]. The effectiveness of TENS in patients with TMD [59] and bruxism [60] has been documented in some studies. In fact, one study observed that TENS reduced both pain and muscle activity of the anterior portion of the temporal muscle but increased the activity of the masseter muscles suggesting a complex mechanism of action of this intervention [61].

7.10 Neuroscience Education

The difficulty for proper long-term management of patients with TMD usually lies in the complex task of changing the attitudes, lifestyles, and social and physical environment of the individual. This hypothesis is based on the premise that pain is potentially influenced by inappropriate cognitions, emotions, and behaviors including catastrophizing, hyper-vigilance, avoidance behavior, and somatization. Several of these psychological disturbances should be treated by the appropriate professional, i.e., a psychologist; however, neuroscience education can be also applied by physical therapists. There is evidence suggesting that neurophysiology education aiming at conceptualizing pain is effective in patients with chronic pain [62] and should be included in the initial phase of treatment in individuals who have inappropriate beliefs about their pain symptoms and complaints. If not, a poor understanding of their pain may lead to the acquisition of maladaptive attitudes, cognitions, and behaviors and a consequent poor compliance to any active exercise program. In fact, Kalamir et al. found that the inclusion of short talks on the anatomy, physiology, and biomechanics of the jaw plus instruction and supervision of self-care exercises increased the effects of oral myofascial therapies in patients with TMD at short- and long-term follow-ups [63].

7.11 Chiropractic Introduction

Kimberly Bensen

Chiropractic is the third largest health-care profession [64] and is the largest, most regulated, and best recognized of the complementary and alternative medicine (CAM) professions [65].

Chiropractic is now a well-acknowledged health profession routinely used by the public, capable of broad diagnostic activity, conservative treatment, and health promotion. It has developed a respected scientific evidence base, and Doctors of Chiropractic (DCs) are embedded in a growing number of health delivery and reimbursement systems, including Workers' Compensation programs, Medicare, the Veteran's Health Administration, and the US Department of Defense. The practice of chiropractic is a licensed health-care profession in all 50 states, the District of Columbia, the US Virgin Islands, Puerto Rico, and other territories of the United States. All of these licensing jurisdictions accept or require graduation from a Council on Chiropractic Education (CCE)-accredited educational program, and all recognize the CCE Standards as the educational requirements for chiropractic licensure [66].

Chiropractic care is a systems-based, whole-person approach to health care. It incorporates the recognition that all aspects of the body are interrelated and interdependent and that the body has powerful self-healing mechanisms. Their primary focus is on disorders of the musculoskeletal system and the nervous system and the effects of these disorders on general health. Chiropractic services are used most

often to treat neuromusculoskeletal complaints [67]. They make adjustments using their hands or a small instrument to apply a controlled, sudden, specific, high-velocity thrust, with a low-level force to a joint that has abnormal movement patterns, and fail to function normally, hypermobility, hypomobility, or dyskinesia (also called a subluxation) [68]. The goal of chiropractic adjustment is to reduce the subluxation; correct structural alignment; improve musculoskeletal joint range of motion, nerve irritability, and pain [69]; decrease inflammation [70]; and stimulate the body's communication system to work more effectively to initiate, control, and coordinate the various functions of the cells, organs, and systems of the body [71]. They incorporate modalities and procedures, lifestyle counseling, nutritional advice and therapy, and other measures that lie within the professional and legally authorized scope of practice of Doctors of Chiropractic (DC) [66].

The DC's license also includes training and use of physiotherapies (as described in the previous physical therapy pages) such as: mobilization or soft tissue techniques, use of complementary measures, such as passive modalities, air, cold, diet, exercise, heat, light, massage, ultrasound, TENS, galvanic current, motor nerve stimulation, therapeutic ultrasound, electrical muscle stimulation, H-wave, galvanic current, FDA-approved laser, decompression, traction, ergonomics, posture training, nutritional counseling and supplementation, therapeutic and rehabilitative exercises and stretches, manual hands-on or tool muscle work including trigger point therapy, neuromuscular reeducation, active release type work, acupressure, deep tissue massage, as well as bracing, strapping, and orthoses. Their license limits the puncturing of the skin, surgery, or prescription of medications. Not all states recognize all aspects of training in their scope of practice licensure [72]. The licensed scope of practice is as broad as the individual doctor's practice philosophy, and not all DCs practice all modalities. Most DCs are not yet trained in comprehensive TMD-specific diagnosis and treatment (Figs. 7.10, 7.11, 7.12, 7.13, 7.14, and 7.15).

Chiropractic has earned recognition for its safe and effective treatment for a wide range of health conditions through the use of natural, nonsurgical, and drugless methods of treatments [64], enhancing patient quality of life and performance; promoting vitality, wellness, and patient empowerment; improving the quality of life without drugs or surgery; and offering readily accessible care [67].

7.12 Chiropractic and TMD

The most common conditions treated by chiropractors are back pain, neck pain, and headaches. The best available evidence supports manipulative therapy as a reasonable option for many of these complaints. Manipulative therapy also holds potential value for the treatment of a variety of extremity conditions, including the TMJs [73].

TMD is a multifactoral condition consisting of some or all of these components: joint subluxations, body positioning, muscle imbalance, neurologic interference, nutritional considerations, occlusion, and psychosocial/psychosomatic characteristics.

The subluxation of the temporomandibular joint is fairly frequent [74, 75], and TMD is the second most common musculoskeletal condition [76]. Chiropractic

Fig. 7.10 Cervical adjustment

Fig. 7.11 Cranial instrument-assisted adjustment

Fig. 7.12 "Infinity" manual mandibular mobilization

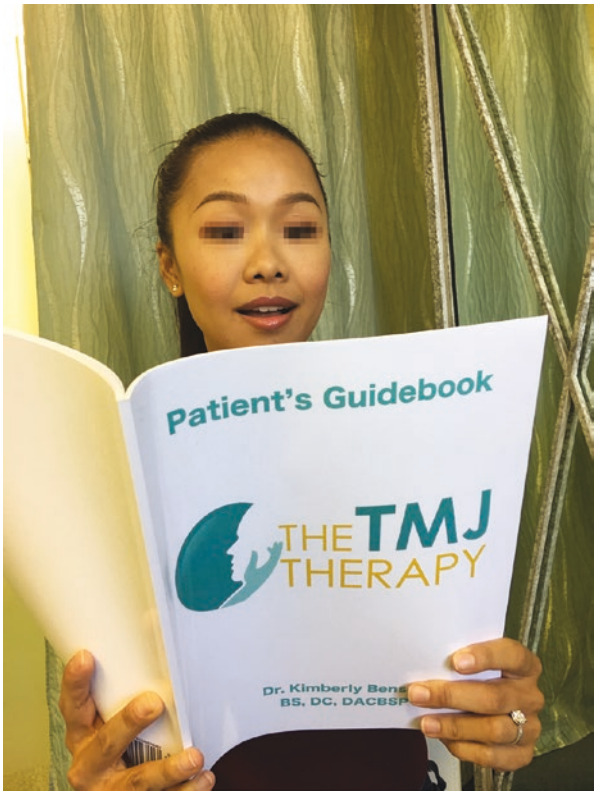

Fig. 7.13 Behavior modification lessons

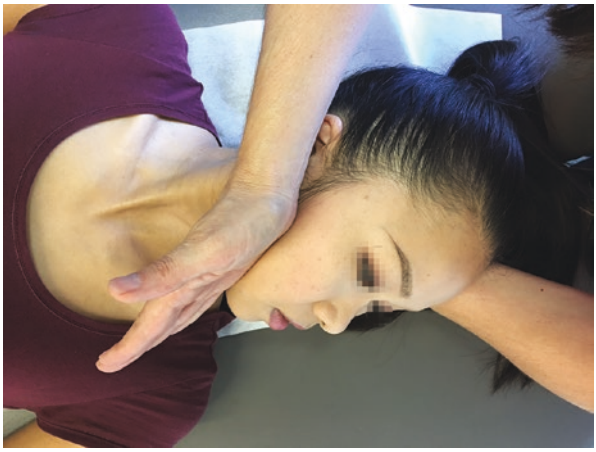

Fig. 7.14 Mandibular adjustment

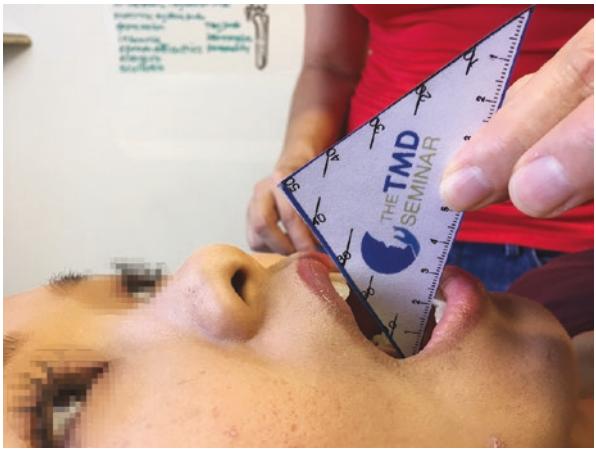

Fig. 7.15 Mandibular measurement

treatment, in a retrospective series, showed that all patients selected for that case series showed a reduction of temporomandibular dysfunction symptoms [77]. The main outcome measures change from baseline to follow-up of visual analog scale (VAS) for temporomandibular joint pain and maximum active mouth opening without pain. Their full spine and TMJs were assessed and adjusted in accordance with the advanced protocol of Activator Methods International. Participants were typically seen three times per week for 2 weeks and according to individual progress thereafter for 6 more weeks. Results of this prospective case series indicated that the TMD symptoms of these participants improved following a course of treatment using the Activator Methods International protocol. Consequently, further investigation of this type of chiropractic treatment for patients with the articular type of TMD is warranted [78].

Dysfunction is a process, not a static condition. Physiologic manifestations of the process of a dysfunctional joint are that the tissues undergo constant changes,

including hyperemia, congestion, edema, minute hemorrhages, fibrosis, local ischemia, atrophy, and tissue rigidity. Eventual rigidity and adhesions form not only in joint capsules but also in the ligaments, tendons, and muscles themselves [79].

Manual therapy (including soft tissue techniques, mobilization, exercise, and manipulation) is postulated to reduce local ischemia, stimulate proprioception, break fibrous adhesions, stimulate synovial fluid production, and reduce pain among other effects [80].

A joint can become subluxated and malfunctional as a kinesiopathogical joint lesion (usually hypomobility) that develops as a consequence of micro- and/or macrotrauma which cause tissue injury and associated inflammatory response, degenerative changes in muscular and connective tissues, decreased descending inhibitory pathway activity due to aberrant psychological states, dysafferentation (i.e., increased nociception), and decreased mechanoreception which can be caused by some or all of the above [81].

This single-subject case study was conducted to investigate the capability of chiropractic manipulation of the temporomandibular joint (TMJ) in treating unilateral anterior displacement of the articular disc with adhesion to the articular eminence. A specific joint manipulation was designed to reduce the anteriorly displaced and adherent TMJ disc. The findings of this study show this specific manipulation of the TMJ may be appropriate for the conservative treatment of adherent anteriorly dislocated disc [82].

Precise and complex balance of the head and neck muscles must exist to maintain proper head position and TMJ function. To understand the effects the muscles have on each other and their bony attachments, there are pairs of muscles that are agonistic or antagonistic, stabilizing or moving the head, neck, and TM joints. These muscle attachments on bones are craniomandibular, craniovertebral, mandibulohyoid, and hyoclavicular/thoracic. There is a highly coordinated, complex neuromuscular event that occurs to synchronize the muscular pulley system of the neck, cranium, and jaw in order to complete all aspects of jaw movement. Each of the major muscles acts like an elastic band. The tension provided must precisely contribute to the balance that maintains the desired head position. If one elastic band is stretched, is adhered, or breaks, the balance of the entire system is disrupted and the head position is altered. When one muscle is compromised, another can compensate, causing abnormal movements [83–85]. To fully assess the cause of the TMJ dysfunction, all associated structures must also be evaluated (Figs. 7.16, 7.17, 7.18, 7.19, and 7.20).

Neurologically, central sensitization and convergence are the primary mechanisms behind referred pain, which can enable tooth pulp pain to be perceived as masseter or facial muscle pain, and posterior neck muscles can generate referred pain to the forehead, periodical, vertex, temple, occipital, postauricular, and ear [86].

The correlation between primary temporomandibular joint disorders and the cervical spine dysfunction is well documented [87–94].

And therefore, we also must include the cervical spine in evaluation and treatment protocols.

Fig. 7.16 Occipital adjustment

Fig. 7.17 Postural exercise

As a case of the concept of integrated dental-orthopedic and craniochiropractic care for treating structural disorders of the jaw, neck, and spine demonstrated, the position of the jaw and head and neck is inextricably linked. Some dental treatment can cause the inability of the head and neck to adapt to maxillary and mandibular

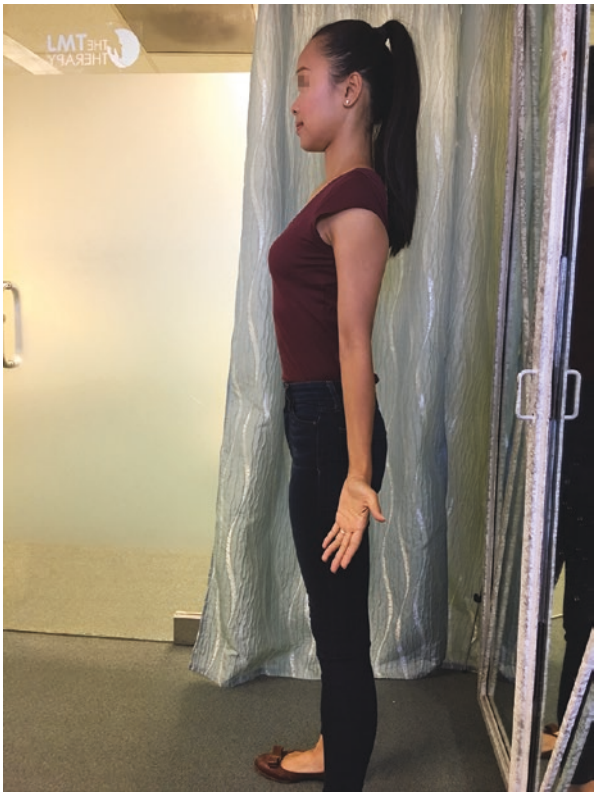

Fig. 7.18 Postural exercise 2

changes, and this study showed that mandibular position improved in response to chiropractic treatments [95].

As the issue of cranial and facial bone compliance and its affect on occlusion and TMJ functioning has gained greater acceptance in the dental and chiropractic fields, what has become a common theme between our professions is the relationship between the stomatognathic system and posture. While the pelvis and the TMJ might seem to be distant and unrelated aspects of our patients' presenting symptoms, research is suggesting otherwise. The rationale for greater relationships between chiropractors and dentists has been discussed in the literature, since in some cases, the only possible chance of a patient having any resolution of their TMD/CMD was with co-treatment [96].

Another study investigated the effect of a rehabilitation program based on cervical mobilization and exercise on clinical signs and mandibular function in subjects with temporomandibular disorder. In this study, 12 women (22.08 ± 2.23 years) with myofascial pain and mixed TMD, according to the Research Diagnostic Criteria for Temporomandibular Disorders, were evaluated three times: twice before (baseline phase) and once after intervention. Self-reported pain, jaw function [according to the Mandibular Functional Impairment Questionnaire (MFIQ)], pain-free maximum mouth opening (MMO), and pressure pain thresholds (PPTs) of both

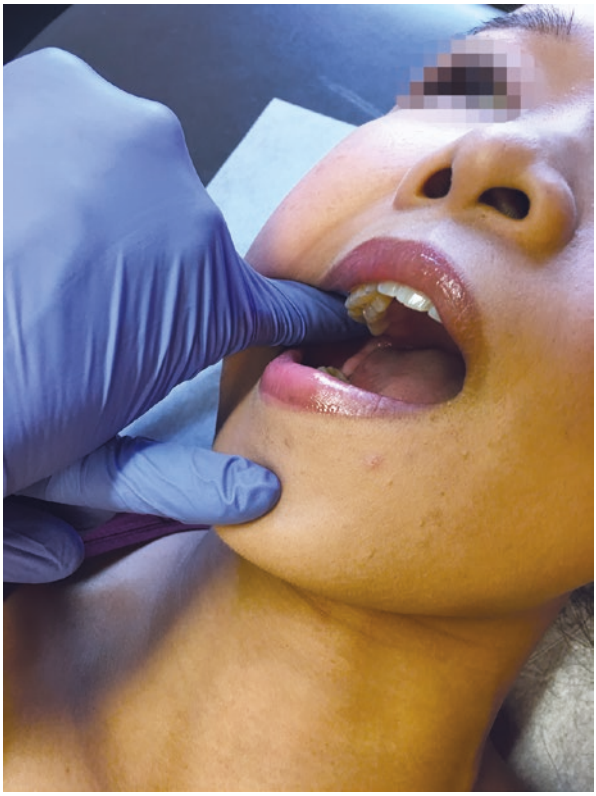

Fig. 7.19 Intraoral lateral pterygoid muscle therapy

Fig. 7.20 Cranial manual adjustment

masseter and temporalis muscles were obtained. Baseline and post-intervention differences were investigated, and effect size was estimated through Cohen's d coefficient. Despite the small sample size, the results showed jaw function improved 7 points on the scale after the intervention, and self-reported pain was significantly

reduced. Pain-free maximum mouth opening varied from 32.3 ± 8.8 mm to 38 ± 8.8 mm and showed significant improvement with moderate effect size when compared to the baseline phase. Pressure pain thresholds also increased with moderate effect size, and subjects had the baseline values changed from 1.23 ± 0.2 to 1.4 ± 0.2 kg/cm^2 in the left masseter ($P = 0.03$), from 1.31 ± 0.28 to 1.51 ± 0.2 kg/cm^2 in the right masseter ($P > 0.05$), from 1.32 ± 0.2 to 1.46 ± 0.2 kg/cm^2 in the left temporalis ($P = 0.047$), and from 1.4 ± 0.2 to 1.67 ± 0.3 kg/cm^2 in the right temporalis ($P = 0.06$). The protocol caused significant changes in pain-free maximum mouth opening, self-reported pain, and functionality of the stomatognathic system in subjects with myofascial TMD, regardless of joint involvement. Even though these differences are statistically significant, this study concludes that their clinical relevance is still questionable [97].

A recent case study presented a patient who had complaints associated with a disorder of the TMJ and cervical atlas subluxation. Vertebral subluxation of the cervical spine may result in a TMJ disorder and that removal of subluxations through adjustments may alleviate the extra vertebral complaint. The conclusion was to suggest that the treating chiropractor consider the possibility that some of these disorders may be effects of subluxations through referred pain or dysfunction in the craniovertebral kinematic chain [98].

The jaw is suspended from the cranium by a tonus of many muscles to hold the mandible in correct anatomical and functional position. The association, as found in the study done by Lee WY, Okeson JP, and Linderoth J., is that the head was positioned more forward in the group with temporomandibular disorders than in the control group [99].

Dr. Okeson's textbook entitled *Management of Temporomandibular Disorders and Occlusion* 5th Edition, states "the skull is supported in position by the cervical spine." It is not, however, centrally located or balanced over the cervical spine. In fact, if a dry skull were placed in its correct position on the cervical spine, it would be overbalanced to the anterior and quickly fall forward. Any balance becomes even more remote when the position of the mandible hanging below the anterior portion of the skull is considered; it is obvious that a balance of the skeletal components of the head and neck does not exist. If the head is to be maintained in an upright position so one can see forward, muscles that attach the posterior aspect of the skull to the cervical spine and shoulder region must contract.

Some of the muscles that serve this function are the trapezius, SCMs, splenius capitis, and longus capitis. It is possible, however, for these muscles to over contract and direct the line of vision too far upward; to counteract this action, an antagonistic group of muscles exists in the anterior region of the head: the masseter, suprahyoids, and infrahyoids. When these muscles contract, the head is lowered. Thus a balance of muscular forces exists that maintains the head in a desired position. These muscles, plus others, also maintain proper side-to-side position and rotation of the head, as supported by additional studies [99].

Other literature suggests a correlation between the orientation of craniofacial planes and temporomandibular disorder in young adults with normal occlusion.

In this instance, TMD might be mainly associated with head posture rather than with craniofacial morphology or specific pathophysiology [100]. Forward head posture is associated with cervical curve or lack of it, and the study done by D'Attilio, on a cross-sectional group of 50 females with mean age of 28.9, offered support of the existing literature demonstrating evidence of correlations between cervical alterations and cervical pain and the existence of cervical pain in subjects with temporomandibular joint internal derangement [101]. There is a significant association between TMD treatment and reduction of cervical spine pain, as far as improvement of cervical spine mobility, as discussed in BioMed Research International (2014). Cervical spine and TMJ positioning-proprioceptive positioning [102] and the biomechanical relationship between the craniocervical region and the dynamics of the TMJ, as well as trigeminal nociceptive processing, are different in different craniocervical postures.

Vladimir Janda documented the influence of the somato-sensorimotor system and the neuromuscular imbalances that can develop, potentially leading to an unfavorable chain reaction affecting the whole body, articular, muscular, and neurological system in his work "*The Assessment and Treatment of Muscle Imbalance*". He terms the head forward posture as the Upper Cross Syndrome (UCS) as well as a condition now known as proximal or shoulder girdle crossed syndrome. In UCS, tightness of the upper trapezius and levator scapula on the dorsal side crosses with tightness of the pectoralis major and minor. Weakness of the deep cervical flexors ventrally crosses with weakness of the middle and lower trapezius. This pattern of imbalance creates joint dysfunction, particularly at the atlanto-occipital joint, C4–C5 segment, cervicothoracic joint, glenohumeral joint, and T4–T5 segment. Janda noted that these focal areas of stress within the spine correspond to transitional zones in which neighboring vertebrae change in morphology. Specific postural changes are seen in UCS, including forward head posture, decreased cervical lordosis and thoracic kyphosis, elevated and protracted shoulders, and rotation or abduction and winging of the scapulae [103].

Further, TMD can be considered to be associated with an imbalance of the whole physical body. According to a literature search by Brantingham JW, Cassa TK, Bonnefin D, Pribicevic M, Robb A, Pollard H, Tong V, and Korporaal C., called Manipulative and multimodal therapy for upper extremity and temporomandibular disorders: a systematic review, there is a fair level of evidence for the implementation of manual manipulative therapy (MMT) to specific joints and the full kinetic chain combined with exercise and/or multimodal therapy for lateral epicondylopathy, carpal tunnel syndrome, and temporomandibular joint disorders. The authors' intent was that the study would help guide practitioners in the use of MMT, soft tissue techniques, exercise, and/or multimodal therapy for the treatment of a variety of upper extremity complaints in the context of the hierarchy of published and available evidence [103].

Given the correlation between global body posture, muscle imbalances caused by forward posture of the head in Upper Cross Syndrome, and temporomandibular joint internal derangement, a full assessment of the whole kinematic chain, (above and below the TMJs) and treatment plan should be incorporated and considered

with any patient expressing TMD symptomotology. Posture training and TMD self-management instructions are significantly more effective than TMD self-management instructions alone for patients with TMD who have a primary muscles disorder [104].

The studies are pointing to a multimodal approach for TMD patient management. Collaborative approach between chiropractic and dentistry [105], with a fair level of evidence for manipulative and multimodal therapy to specific joints and the full kinetic chain combined generally with exercise and/or multimodal therapy TMJ disorders [106], and some findings may suggest interconnectivity between the craniocervical junction and an individual's occlusal contacts and support the need for further integration between chiropractors and dentists seeking to co-manage temporomandibular joint disorders [107].

7.13 Conclusion

TMD is a multifactoral condition consisting of some or all of these components: joint subluxations, body positioning, muscle imbalance, neurologic interference, nutritional considerations, occlusion, and psychosocial/psychosomatic characteristics.

Chiropractors are ideal for initial, co-management, and adjunct care, as they are known for a wholistic approach to health and wellness. Seeing the body as a kinematic chain, they employ joint manipulation, physiotherapies as their practice philosophies allow, nutrition, posture/ergonomic, stress, and lifestyle advice. However, the DC's scope of practice varies from state to state, and their individual philosophies also vary, as well as their interest and/or advanced training in TMD and rehabilitation techniques.

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Treating the TMD/Chronic Pain Patient: Psychiatry and Psychology

8

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Abstract

From the outside, it has been very common for clinicians to dismiss TMJ patients as "crazy," "difficult," or "high maintenance." This sentiment conveyed both a lack of knowledge of TMJ pathology and mental illness on the part of the health professions. Fortunately, the true situation is gradually coming to light, and doctors are gaining an increased appreciation for how connected the mind and body really are. In fact, there is no true separation at all; it has always been something we created to make sense of a system we did not understand. Fortunately, our ideas of how the mind and body interact have evolved, and it is unmistakably critical to be able to assess both the mind and body together in order to ultimately make an ailing person whole again. This chapter is devoted to explaining how psychologists and psychiatrist evaluate, diagnose, and treat TMD/chronic pain patients.

8.1 Chronic Pain from a Psychiatric/Psychological Perspective

From a biopsychosocial perspective, the role of psychological assessment and intervention is integral to the treatment of all medical conditions, to the extent that the psychosocial factors are impacting treatment and outcomes for each particular

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person. Factors such as depression, anxiety, catastrophizing, and pain acceptance have all been linked to pain outcomes. As a result, it is crucial that when present, these factors be addressed in tandem or prior to medical interventions. At a minimum, psychological interventions can prepare patients for medical interventions and give patients realistic expectations and perspective on their situation.

For instance, when a patient has a phobia of needles, this must be addressed prior to Botox injections with the help of exposure therapy. A patient with depression may be reluctant to eat, but then only willing to do so in larger quantities at a time, causing more pain. This can be addressed by teaching pacing skills and using cognitive restructuring.

8.2 Characterization of the Problem

Psychological assessments typically include a combination of a clinical interview and self-administered assessment forms. The goal is then to develop a case conceptualization to identify potential sources of physical and emotional problems, dysfunctional behavioral patterns, and current level of distress and disruption due to these symptoms [1] and to identify mental health diagnoses according to Diagnostic and Statistical Manual (DSM) criteria [2]. This allows for a personalized treatment plan, as well as continuity of care across providers when using objective, checklist-based criteria as defined by the DSM.

Specific to conceptualizing the pain problem, thorough psychological assessments typically derive information from both the clinical interview and the specific pain psychology assessments such as those detailed below (e.g., PSC, PASS). Similar to how a psychological problem is described, the goal is to (a) identify how psychological symptoms may interact with pain conditions (e.g., anxiety and muscle tension; fear of movement and deconditioning) and (b) diagnose the presence of mental health diagnoses as it relates to pain conditions (somatic symptom disorder, illness anxiety disorder, conversion disorder). In addition to DSM diagnosis, patterns of dysfunctional behavior may exacerbate pain and identified for treatment targets such as fear avoidance, pain catastrophizing, poor sleep hygiene, poor coping strategies, patterns of pain flare and extended recovery, and treatment adherence to medical recommendations. Such patterns can maintain and exacerbate pain and reduce function and quality of life.

8.3 Pain Psychology Workflow from Initial Encounter to Diagnosis and Treatment

8.3.1 Chief Complaint

In the biopsychosocial model, it is first important to assess the subjective qualities of the pain, location, intensity, and affective qualities (tight, sharp burning, etc.) in order to understand the patients subject experience of the pain. Furthermore, identifying other comorbid pain conditions may give insight into appropriate treatment.

Alleviating and exacerbating factors are assessed both to understand the experience and also to hint at a patient's coping skills. Does he avoid eating anything solid, avoid speaking? Or rather, does he continue to do these things but take medicines or other substances to cope? Similarly, a patient's daily activities are assessed to understand what he is doing in spite of the pain. It is also important to identify if activities are being avoided due to pain, both to understand what valued activities he has given up and what represents avoidance behavior due to fear of pain.

Patients should be asked what control they feel they have over the pain; can they take a medicine to decrease it, can they avoid chewing in a particular way, and can they relax all the muscles in their face? A most distressing aspect of chronic pain can be the perception that one has no control. Ultimately, a main goal of psychological therapy for pain is for the patient to discover ways that he has control over his pain.

It can also be helpful to understand what role others play in the maintenance of the pain condition. First, how do others come to know about the patients' pain? Some patient's will verbally express their pain to others, and some will avoid mentioning it but instead will make facial grimaces, rubbing, or holding their face in a particular manner to alleviate the pain. Some have learned, either from current or past experience with pain, that it is best to restrict any expressions of pain. Others have high levels of pain verbalizations and behaviors. When family members are present, it can be useful to determine the patient's level of insight regarding their own pain behaviors. Further, the result of this behavior on the family members or friends is important to assess; are patients met with judgment, solicitous behaviors, or minimal responses?

From a psychological perspective, it is important to understand what the patient believes is causing his pain. What importance does he give to musculoskeletal factors, what role does he give to stress, and what role does he give to nerve pain? This assessment helps to tailor justification for psychological therapy in the future. For instance, if he understands that his pain is due to weak and tired muscles around the joint, he may be more interested in learning strategies to relax and strengthen those muscles using relaxation and physical therapy.

Finally, an assessment must include patients' expectations for the future. What do they believe will come of this pain? Do they hold catastrophic beliefs that this pain will ruin their life leaving them alone and destitute, or do they believe they can live with it and manage it? Do they expect it to get better, worse, or stay the same? Which interventions do they believe will be effective for this condition? This information allows the psychologist to evaluate patients' beliefs about pain and to tailor interventions in the future.

8.3.2 History of Present Illness

Patients often begin with a detailed history of their pain condition from beginning to present. However, they can unwittingly leave out important details. From a psychological perspective, the context of when the pain began for the first time can hold

information about traumatic experiences, experiential learning, and what types of stressors or changes may have been present in their life at that time.

Moreover, each patient's approach to treatment is assessed. Did they wait for several months or years to be evaluated? Did they seek only the top specialists? Did they have several surgeries for the pain and try many medications, or do they prefer naturopathic approaches? This information may be related to their expectations for what can be helpful to them and put in perspective their conceptualization of their own pain. Further, it is helpful to know if, in their view, their pain is improving, worsening, or not changing. Have they had any periods of remission or reductions of the pain? If so, what contextual factors were present?

All of these questions pertain to the patients' beliefs about causation, maintenance, and solutions for their pain problem. Several negative beliefs can be present in the history and can lead to negative expectations and negative outcomes. On the other hand, realistic beliefs and active coping strategies can optimize outcomes in both psychological and medical interventions.

8.3.3 Additional Assessment Information

To complete the evaluation, an assessment of daily habits (sleep, nutrition) and use of substances (caffeine, nicotine, alcohol, marijuana, and illicit drugs) and medication misuse or overuse is collected. Current symptoms of depression, mania, anxiety, panic, trauma, and psychosis are assessed.

A comprehensive psychosocial history is obtained, including adverse childhood events, family of origin relationships, current family and relationships, education, and employment. Furthermore, a personal psychological history and treatments are taken as well as a family history of mental illness and substance abuse.

This information can inform a psychiatric or substance use disorder diagnosis if needed. Typical psychiatric conditions, which overlap with chronic pain and TMD, specifically include adjustment disorders, major depressive disorder, anxiety disorders, somatic symptom disorders, and post-traumatic stress disorder.

8.3.4 Assessment Tools and Data Collection

Psychological assessments have utility in providing objective data to psychological evaluations. There are psychological screening tools to briefly capture various psychological dimensions pertaining to pain, and there are multidimensional psychological assessments. The list below is not meant to be an exhaustive list of psychological inventories, merely a selection from possible constructs that are important to consider. Measures that are reliable, valid, and freely available are favored. They are also available freely in multiple languages.

Brief screening tools for psychological dimensions include:

- *Pain:* The Brief Pain Inventory (BPI) short form is freely available and widely used to capture several dimensions of pain [3]. It looks at the severity, intensity, and the biopsychosocial impact of the pain on a person's life.
- *Depression:* The Patient Health Questionnaire 9 (PHQ9) is a self-administered tool based on DSM-IV criteria for diagnosing depressive disorder [4]. There are nine items based and it aims to predict the presence and severity of depression.
- *Anxiety:* The Pain Anxiety Symptom Scale (PASS) was specifically developed to measure anxiety related to pain [5]. The PASS is the only instrument designed specifically to assess four components of pain-related anxiety: (a) cognitive, (b) fear, (c) escape/avoidance, and (d) physiological. There is also a short form (20 items) that is freely available.
- *Catastrophizing:* The Pain Catastrophizing Scale (PCS) is a 13-item self-report scale [6]. It measures the construct of catastrophizing for patients with chronic pain. Catastrophizing is characterized as a pattern of magnified negative expectations for outcomes. This psychological construct has been shown to affect self-reported pain levels, mood, and how much dysfunction is caused by pain.
- *Disability and self-efficacy:* The Pain Self-Efficacy Questionnaire (PSEQ) measures self-reported confidence in one's ability to cope with their pain [7]. Self-efficacy is a measure of one's own perception of their ability to anything; in this case it is directed at pain coping. This construct has an impact on pain outcomes, specifically for function and disability.
- *Multidimensional psychological assessments:* The Battery for Health Improvement 2 (BHI2) is a standardized psychometric measure, a primary purpose of which is to perform evaluations of patients with pain and injury [8]. The BHI2 has medical scales, psychological scales, and social scales which allow it to be defined as a biopsychosocial instrument. It also has validity scales which can be useful in determining response style (random, self-disclosure, defensiveness). The measure has 217 multiple choice items and takes between 30 and 35 min to complete.

8.3.5 Typical Diagnostic Assessment

Typical psychiatric conditions, which overlap with chronic pain and TMD specifically, include adjustment disorders, major depressive disorder, anxiety disorders, somatic symptom disorders, and post-traumatic stress disorder. Even if no formal psychiatric diagnosis can be made, a diagnosis of "psychological and behavioral factors associated with disorders or diseases classified elsewhere" can be given, specifying which psychosocial factor is influencing which medical condition, i.e., high pain catastrophizing associated with TMD.

The diagnosis being treated primarily is listed first in the diagnosis list. For instance, if a patient is diagnosed with an adjustment disorder with anxious features and temporomandibular joint disorder, therapy would be aimed at both diagnoses,

but the adjustment disorder would be listed first because it is the primary disorder being treated. On the other hand, if the patient presents with a history of recurrent major depression, but is not in a current depressive episode, then TMD would be listed first.

8.4 Treatment Tool Box

8.4.1 Behavioral Approaches

1. *Relaxation training* is intended to both provide tense muscles the opportunity to relax and to provide the patient with some level of control over their experience. Relaxation can be self-initiated or the patient can listen to an audio recording and practice relaxation. With all relaxation training, a good deal of home practice is encouraged in order to learn the methods and be able to use them in a wide variety of settings.
2. *Diaphragmatic breathing*, a method of breathing, is taught to patients to increase control over their autonomic nervous system. It is a slow conscious breath, where the inhale is taken down to the bottom of the lungs and the abdomen expands. The exhale is done slowly from the bottom to the top of the lungs. The technique is demonstrated and then practiced by the patient. The patient is instructed to practice this at least twice a day for 5–10 min.
3. *Progressive muscle relaxation* is a technique that involves tension on various muscle groups for 10 s, then releasing and paying attention to the sensation of release and relaxation in the muscle group. This technique is particularly helpful for patients who have chronic muscle tension and who can learn what a muscle feels like when it is releasing.
4. *Autogenic training* involves repetitions of a set of visualizations that induce a state of relaxation and is based on passive concentration of bodily perceptions (e.g., warmth of arms, hands), which are facilitated by self-suggestions. For example, a patient may repeat to themselves, "my face is relaxed and warm, my jaw is warm and heavy, my mouth is relaxed and comfortable."
5. *Visualization/guided imagery* is typically led by a therapist. It can go beyond visualizing bodily states and invites the patients to imagine themselves in a relaxed and comfortable setting.
6. *Hypnosis* has long been used to treat TMD. It first relies on inducing a deeply relaxed state in the patient and then providing suggestions for relaxed muscles, reduced sensitivity to pain, and increased function. Typically it is taught in a session with a trained hypnotherapist and practiced at home over several sessions.

8.4.2 Operant Behavior Training

Operant behavior training focuses on pain behaviors as targets for treatment. Pain behaviors such as verbalizations, actions, or facial expressions that occur in response

to pain were potentially useful in response to acute pain when they elicited support via communication of danger or threat. However, in chronic pain they are thought to be dysfunction in the absence of danger and have found to predict disability. Operant behavior training is intended in provide learning opportunities to experience new pain behavior patterns.

1. *Graded activity* is where an activity or exercise is selected based on relevance to the functional limitations of the patient and/or their presenting pain complaint. This activity is performed to the point of pain or fatigue to establish a baseline and then is lowered by 10–25% and is prescribed, and the patient receives reinforcement for doing it. The activity is gradually increased, resulting in improved function. For instance, if a patient can talk for 5 min but then experiences pain, a graded activity may be to read aloud for 4 min, provide reinforcement, and gradually increase the amount of time.
2. *Activity pacing* involves breaking down an activity into smaller components and using breaks to reduce pain and increase function.
3. *Time contingent medication schedules* are implemented when a patient begins taking more medication than prescribed or in situations driven by mood. Taking medication on a time-based schedule as opposed to a pain-based schedule changes the contingencies of the medication-taking behavior.

8.4.3 Biofeedback

Biofeedback is a treatment that helps patients understand and control their physiological responses across multiple domains, including heart rate, breath, sweat, brainwaves, muscle tension, and peripheral temperature. It utilizes much of the skill development mentioned previously in relaxation training but allows the patient to gain concrete insight to the relationship between mind and body. This is often a transformative intervention for patients with chronic pain as it provides visual evidence of how they can consciously control their bodies' reactions (e.g., heart rate, muscle tension) by merely slowing their breath, completing exercises such as progressive muscle relaxation, or thinking of something calming. Biofeedback is often used in conjunction with cognitive behavioral therapy (CBT) to demonstrate the effects of thought change on the body (e.g., patient tells a story of a stressful experience and watches physiological stress response increase on the screen and then discusses coping strategies or alternate perspectives on the problem and watches a decrease on sympathetic nervous system measures such as decrease in heart rate and sweat). Typical course of treatment ranges from 2 to 10 sessions in most pain-related settings.

8.4.4 Cognitive Behavioral Therapy (CBT)

It is a well-established therapy for chronic pain. The goals of CBT are to help the patient reconceptualize their chronic pain condition as a chronic disease, that pain

can be malleable and potentially controllable, and to emphasize an active role in self-management. The focus of CBT is on skills acquisition of cognitive reframing and behavioral techniques as described above in relaxation skills and operant behavioral skills. CBT is typically provided in individual sessions ranging from 6 to 12 sessions and commonly studied in group format for 8–10 sessions. CBT can also be delivered in an online format where homework and lessons can be done at one's own pace. This can be helpful if a patient does not have access to CBT nearby.

8.4.5 Acceptance-Based Approaches

In Acceptance and Commitment Therapy (ACT), the goal is to facilitate a person's engagement with a range of valued activities in the presence of pain and to change the control over behavior by altering the context.

Mindfulness has a growing base of evidence for mood, anxiety, and pain management. Mindfulness is an awareness of mind and body and often learned through movement, meditation, and guided practice. In particular, the 8-week program Mindfulness-Based Stress Reduction (MBSR) has significant research to demonstrate reduction in depression, anxiety, and even pain symptoms in patients with chronic pain conditions, including TMD specifically. Importantly, this approach can be effective and useful for many populations, including healthy populations looking for wellness and stress management strategies. Thus, this recommendation is appropriate for most patients, ranging from those with diagnosed mental health disorders to those resistant or disinterested in other potentially stigmatizing treatments delivered through a mental health setting.

8.4.6 Self-Help

Both for patients with and without known psychopathology, clinicians may describe the relationship between "stress management," pain, and quality of life as a nonstigmatizing way to approach self-management of pain. There are a number of self-help books, websites, and mobile applications that can be helpful to patients with TMD. As these resources can quickly change over time, providers may self-manage an updated list or alternatively describe the importance of relaxation training and changing perspective on pain and stress and provide search terms for patients to follow up on such as MBSR, progressive muscle relaxation, and handouts on diaphragmatic breathing.

8.5 Outcomes and Evidence

In recent reviews comparing psychosocial interventions to usual care for patient with TMD, CBT has shown improved long-term self-report of pain and improvement in depression over usual care [9, 10]. Self-management interventions have

also shown positive results for patient with TMD [10]. However, in spite of these promising results, access to CBT delivered by a trained health psychologist remains difficult. A few studies have looked at the combination of CBT with interventions like biofeedback and hypnosis in comparison to usual care [11, 12]. Results have been encouraging in a few studies. In our clinical experience, the combination of usual care, including myofascial massage, and use of splints, along with pain medications, and cognitive behavioral therapy has good benefit for patient with TMD.

8.6 Ramifications of Comorbid Psychiatric Illness and Chronic Pain

8.6.1 Evidence Supports a Strong Clinical Relationship Between Psychiatric Illness and Chronic Pain

Psychiatric comorbidities complicate treatment of patients with chronic pain and opioid use disorder. For instance, in one study of patients with chronic pain and opioid disorder, common current anxiety diagnoses included post-traumatic stress disorder (21%), generalized anxiety disorder (16%), and panic disorder without agoraphobia (16%). Common current mood diagnoses included major depressive disorder (40%) and dysthymia (11%). A majority of patients (52%) had a personality disorder [13]. Conversely, the prevalence of pain symptoms in patients with depression has been reported to range from 15 to 100%, with the mean prevalence being 65% [14].

Common mental disorders, such as depression and anxiety, are known to be associated with higher pain intensity, more pain complaints, and higher pain interference with daily activities. The high rates of comorbidity among chronic pain and psychiatric disorders have been well documented. In the general population, the 12-month prevalence rate for major depression is 6.7%, yet having one or more chronic pain conditions increases the rates of depression to 10–30% [15]. Additionally, over 75% of depressed patients in primary care complain of painful physical symptoms such as headache, stomach pain, neck and back pain, as well as non-specific, generalized pain. The presence of such symptoms predicts a greater severity and a less favorable outcome of depression with a poorer health-related quality of life [16].

A close association has also been observed between pain and anxiety disorders. For example, 12-month prevalence rates of panic disorder are estimated to be 1–2.7% in the US general population, in comparison to 13–15% in patients with chronic headaches. The prevalence of PTSD has also been shown to be elevated in various pain populations. The 12-month prevalence rate of PTSD is 3.5% in the general population, in comparison to 30–50% in patients who developed pain as a result of motor vehicle accidents and 15% in patients seeking treatment for idiopathic facial pain [15].

The causal relationship between chronic pain and various psychiatric disorders has been a matter of interest. Prospective studies have suggested that chronic pain

can cause depression and that depression can also cause chronic pain. A bidirectional relationship likely underlies the strong association between pain and common mental disorders. This means that psychiatric disorders are not solely a reaction to chronic pain. They also predispose individuals to the development of chronic pain [15].

Regardless of whether the pain precedes the depression or vice versa, the implications of having both disorders are significant. People with both chronic pain and depression have worse pain outcomes than those with chronic pain alone and several studies suggest that depression adversely affects the effectiveness of treatments for pain [17]. For example, in terms of opioid treatment in those patients with chronic non-cancer pain, "negative affect" (cluster of related, concurrent negative emotions and thoughts, such as high levels of depression, anxiety, and pain catastrophizing), has been described to predict decreased effect to opioid analgesia [18].

Several studies show that depression significantly increases disability in chronic pain populations. Among people with chronic pain, depression has been associated with higher health-care costs, increased economic burden for patients and their employers, and increased risk of chronicity. In addition, depression has been associated with suicide attempts and suicide completion among people with chronic pain [17].

8.6.2 Evidence Supports Strong Neuroanatomical Overlap Between Pain and Mood

The interactive relationship between mood and pain is clearly evident from the experimental pain studies. Pain is a multidimensional subjective experience, which can be assessed along two dimensions, i.e., sensory pain intensity, which describes the discrimination of the stimulus intensity, and affective pain unpleasantness, which describes the emotional impact of the stimulus [19, 20]. During brief experimental noxious stimuli, these dimensions are highly correlated [21–23], yet even subtle alterations in the cognitive, emotional, and/or physiological state of the individual influence the pain experience [24–26]. Hence, in healthy individuals, induction of sad moods worsens the feelings of pain, especially when the emotional impact of pain is measured [27, 28]. Likewise, individuals with depression show increased emotional reactivity to experimental pain [29–32] and, in fact, demonstrate increased emotional response to stimuli that are not even painful, a phenomenon termed *emotional allodynia*, because stimuli that are normally *not* unpleasant produce an unpleasant feeling in the depressed subjects [33–35]. Clinical "allodynia" refers to an unpleasant light touch. Such affective feeling of unpleasantness is uncoupled from the sensory discrimination of intensity or pain, in stark contrast to healthy individuals, who begin to report unpleasantness at pain threshold [34, 36, 37]. Heightened emotional reactivity and emotional biasing of daily self-reports of subjective pain experience is consistently observed in chronic pain patients, especially in those with a history of depression and increased daily depressive moods [29, 38, 39] and in patients with comorbid chronic pain and depression [40].

On the neural level, segregated neural pain-processing networks of the affective and sensory pain dimensions are evident in healthy brains [41], in those with depression [36] and chronic pain and in those with comorbid chronic pain and depression [40]. Such segregated, yet integrated, neural processing of sensory and emotional aspects of pain experience can be parsimoniously described by the neuroanatomical substrate underlying interoception or the perception of the physiological condition of the entire body [42–44]. According to this neuroanatomical evidence, pain is homeostatically driven, i.e., pain begins as an affective bodily feeling that signals an urgent need, like itch or hunger [45]. The main forebrain projections of the interoceptive pathway are the insular cortex and anterior cingulate cortex, subserving sensory and motor aspects of pain, respectively [44]. Importantly, the insular and cingulate cortices interact strongly with each other and with other forebrain regions (e.g., hypothalamus, amygdala, ventral striatum) to create a so-called "healthy" experience of pain [44]. Such activation pattern is typically disrupted in those with chronic pain or psychopathology or both [46], where increased interoceptive focus [37] and/or decreased behavioral motivations [47] can amplify and maintain chronicity of chronic pain.

Mounting clinical and experimental evidence now shows tremendous benefit of targeting emotional symptoms in those with chronic pain. For example, changes in depressive symptoms severity were the only significant predictor of pain-related disability in highly disabled veterans with chronic, non-cancer pain [48], clearly supporting the tight link between depressive symptoms and increased levels of pain-related disability. On the neural level, reducing negative thoughts about chronic pain and/or depression with cognitive behavioral therapy showed dramatic improvement in brain structure and function [49], particularly related to motivational behavior within the cingulate and medial prefrontal cortices [50].

8.6.3 Antidepressants Target Separate Pathways in a Temporally Differential Manner to Affect Both Pain and Psychiatric Symptoms

The selective reuptake of norepinephrine is believed to be responsible for the analgesic effects of select antidepressants, including serotonin-norepinephrine reuptake inhibitors (SNRIs), and tricyclic antidepressants (TCAs). Proposed mechanisms of action include norepinephrine-mediated activation of descending inhibitory pathways projecting from the supraspinal centers and terminating in the dorsal horn of the spinal cord. Another possible mechanism includes improvement of depressive or anxiety symptoms. For example, secondary analyses of four randomized trials of duloxetine for fibromyalgia revealed that 69% of pain improvement was attributed to the direct analgesic effects of the drug, whereas 31% of pain improvement was attributed to reductions in depressive symptoms. In addition to fibromyalgia, neuropathic pain, and musculoskeletal pain, SNRIs are effective for the treatment of major depressive disorder and anxiety disorders including generalized anxiety disorder and panic disorder [51].

Similar to SNRIs, the proposed analgesic effects of TCAs are mediated by activation of descending inhibitory pathways, but other possible mechanisms include blockade of voltage-gated sodium channels, inhibition of *N*-methyl-D-aspartate receptors, and interaction with opioid receptors. The major adverse effects of TCAs are related to blockade of muscarinic, histamine 1 and α1-adrenergic receptors. Type 1 antiarrhythmic properties are due to sodium channel blockade, which could, in part, account for the increased rate of sudden cardiac death and myocardial infarction. Although neuropathic pain is the most widely recognized pain indication for TCA use, these medications, particularly nortriptyline and amitriptyline, also have proven efficacy for fibromyalgia and chronic low back pain [51].

A major issue in the literature has been whether the pain effects of these drugs are a direct analgesic effect through their antidepressant effect or through both effects. Recent statistical evidence utilizing correlation and path analysis indicates that the analgesic effect is direct and can be separated from the antidepressant effect. In addition, the analgesic effect appears to occur earlier than the antidepressant effect. Thus, antidepressants can also aid in the treatment of associated pain, while any effect on the depression could in turn aid pain treatment [14].

Interestingly, the antidepressant efficacy and low side effect profile of selective-serotonin reuptake inhibitors (SSRIs) have made them the most widely prescribed class of antidepressants. Pain patients whose depression responds to an SSRI may have less pain, a finding that is attributable to improvements in the affective components of their pain; there is little evidence to support the independent analgesic activity of SSRIs [52].

Serotonin-norepinephrine reuptake inhibitors (SNRIs) have been examined in several randomized, double-blind studies of the treatment of pain associated with depression. Three randomized, double-blind studies found that duloxetine 60 mg once/day significantly reduced painful symptoms compared with placebo in patients with major depressive disorder (MDD). Another randomized, double-blind study compared the effectiveness of duloxetine 40 mg/day, duloxetine 80 mg/day, and paroxetine (an SSRI) 20 mg/day versus placebo in 353 patients with MDD. Duloxetine 80 mg/day caused a significantly greater improvement in overall pain severity compared with placebo ($P < 0.001$); however, neither duloxetine 40 mg/day nor paroxetine was more effective than placebo for relieving pain [53]. The beneficial effect of venlafaxine on both depression and pain was documented in an observational, prospective study of patients with depressive symptoms and comorbid chronic pain and in an 8-week study of patients with first-episode depression with painful symptoms. However, the results of a recent 6-week investigation of the efficacy of 150 mg of venlafaxine in patients with comorbid depression and chronic low back pain showed that only 26.4% of patients responded in both conditions, suggesting a weak therapeutic effect on pain [54].

The important issue in clinical practice is whether SSRIs, considered as a first-line treatment of depression, are as effective as SNRIs in patients with pain symptoms. One multicenter, randomized, non-blinded, parallel-group 12-week trial compared the efficacy of duloxetine with generic SSRIs (citalopram, fluoxetine, paroxetine, or sertraline). Their data showed no significant differences in the depression remission rate. However, the effect of duloxetine on pain symptoms was

significantly better, in comparison to SSRIs. The aim of several 7- to 9-week head-to-head trials was to compare the efficacy of duloxetine (40–120 mg/day) and paroxetine (20 mg/day) in depressed patients with pain symptoms. Two pooled analyses of these studies found no significant difference between the two drugs in the reduction of painful symptoms. Thus, the current evidence from clinical trials is insufficient to speculate about the superiority of either agent over the other in the treatment of MDD with accompanying pain [54].

As for treatment implications, the Stepped Care for Affective Disorders and Musculoskeletal Pain (SCAMP) study was a randomized clinical trial consisting of 12 weeks of optimized antidepressant therapy (step 1) followed by six sessions of a pain self-management program delivered over 12 weeks (step 2) and finally a 6-month continuation phase in which symptoms were monitored and treatments reinforced. Optimized antidepressant therapy followed by a pain self-management program resulted in substantial improvement in depression as well as moderate reductions in pain severity and disability [55].

While limited by its retrospective study design and the use of antidepressants as a part of multimodal treatment of pain, 1506 chronic non-cancer pain charts at an outpatient multidisciplinary pain center recorded the prescription of at least one antidepressant. Most patients received a combination of medications and procedures. Tricyclic antidepressants (TCAs), which include secondary and tertiary amines, as well as SSRI/SNRIs and atypicals, all appear to show similar favorable pain response rate of ~75%. Patients with comorbid depression ranged from 25 to 30% in those taking TCAs, to 64%, 44%, 52%, 57%, 62%, and 44% in those prescribed SSRIs, SNRIs, bupropion, mirtazapine, trazodone, and nefazodone, respectively [55]. Curiously, the data suggested that in the context of multimodal treatment for chronic pain, antidepressant therapy at both low and therapeutic doses demonstrates similar response rates [56].

Interestingly, a recent evidence-based structured review has demonstrated consistent evidence that antidepressant treatment of depression may be negatively affected by the presence of pain for achieving depression response/remission in patients with depression and pain, and pretreatment pain levels will predict antidepressant depression remission [14].

Regardless, antidepressants can inhibit neuropathic pain even when the patient is not in a depressive state. In addition, the effects of antidepressants on depression characteristically take approximately 4 weeks to be observed from the time the drug is first taken, whereas the analgesic effects on chronic pain manifest in as little as few days to 1 week. Therefore, the analgesic effects of antidepressants on chronic pain likely involve a mechanism different from that mediating their antidepressive effects [57].

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