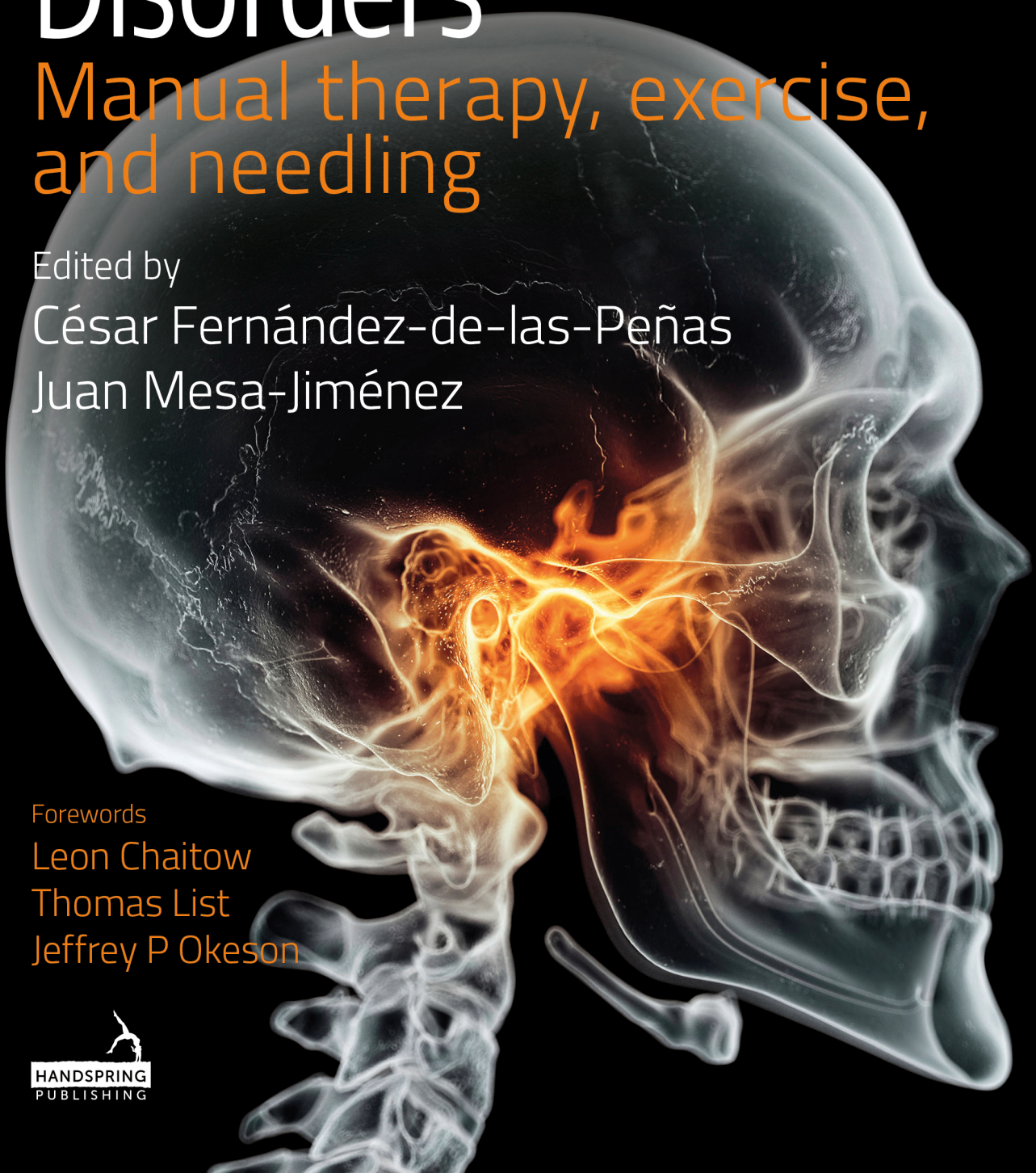
Temporomandibular Disorders

Manual therapy, exercise, and needling

Edited by
César Fernández-de-las-Peñas
Juan Mesa-Jiménez



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Leon Chaitow
Thomas List
Jeffrey P Okeson

HANDSPRING PUBLISHING

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FOREWORD by Leon Chaitow

What emerges from the pages of this absorbing book is a revelation. There is a proverb, attributed to various cultures, that states 'It takes a village to raise a child' and, as this textbook illustrates, it takes a large team to comprehensively describe temporomandibular disorders (TMDs) and to accurately and safely examine, identify, understand, treat, and manage these extremely widespread musculoskeletal pain conditions, that are (depending on the diagnostic criteria) second only to nonspecific low back pain in prevalence worldwide, affecting approximately 10 per cent of the adult population.

The opening chapters set the scene by clearly outlining and detailing the examination, classification, including trigeminal nociceptive processing, pathophysiology, sensory testing, and referred pain associated with TMDs (and orofacial pain). Now, it might be thought that defining TMDs would be straightforward. However, as is forensically explained in the first chapter – Definition, epidemiology and etiology of painful temporomandibular disorders – in order to manage these conditions effectively, it is vital that clinicians understand and appreciate the multiple factors that can influence the evolution and maintenance of the dysfunction and pain associated with TMDs.

Before the chapters that detail the effective examination and manual therapy treatment and management of TMDs, it becomes apparent that, critical to optimal management, there must be awareness that a TMD is rarely an isolated disorder with a single 'cause', but is usually the result of a wide range of interacting adaptations, factors, and influences. Some of these etiological features may be preventable, and/or reversible, while some are historical (injury for example) or inherent. For example, there are unexpected ethnic and racial differences, with a clear discrepancy between the incidence of TMDs amongst, e.g., African Americans (3.8 per cent), mixed-race White/Native Americans (12.7 per cent), and Asians (2 per cent). Other potentially significant influences range from educational, occupational and socioeconomic features, to body weight, physical activities, coexisting conditions, habits such as smoking, as well as biomechanical and psychological factors. Unsurprisingly, these same features are also common risk factors for nonspecific low back pain and chronic neck pain.

The chapters on examining for TMD and orofacial pain clearly describe the need for a comprehensive clinical history, together with a detailed evaluation of the temporomandibular joint itself, the masticatory muscles, and the vital structural and functional connections to the cervicothoracic spine, including possible influences such as posture, neurology, ligamentous stability, arterial dysfunction, segmental ranges of motion and mobility, and the functionality of the deep neck flexor muscles. All, or any, of these topics can potentially be major features in the evolution of TMDs, making their assessment essential for an understanding of the particular influences in any given case.

The chapters covering manual therapy interventions provide evidence-informed details regarding therapeutic exercise, joint manipulation and mobilization, management of referred pain (trigger points), as well as a range of soft tissue methods, postural re-education and training. Among these insightful manual therapy chapters there is also a clear and detailed exposition of the role of fascial anatomy in relation to the cranio-cervico-mandibular region. This chapter includes an extraordinarily detailed and complex outline of fascia in relation to the act of chewing, as a part of the survey of multiple dynamic fascial links, connections and functions in the mouth, throat, head, and neck. Also receiving appropriately detailed coverage in relation to TMD management are dry needling, and – usefully in a separate chapter – acupuncture, as well as current

perspectives on pain psychology and treatment of the brain. One of the final chapters – Treating the brain in temporomandibular disorders – includes a fascinating exploration of pain neuroscience education and brain exercises.

As a clinician, this reader now has a far clearer understanding as to the host of influences governing the complex issues around TMDs. This fine textbook has been brilliantly conceived and thoughtfully realized, and all concerned deserve congratulations.

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FOREWORD by Thomas List

I am very pleased, and honored, to be writing a foreword to this book. The gap between our theoretical knowledge of mechanisms, treatment efficacy, and effectiveness outcomes, and our practical clinical experience in how to apply physical therapy in temporomandibular disorder (TMD) pain patients has long needed such a discussion as this book provides. The wide array of treatment modalities in the domain of physical therapy can be confusing when considering which of the available therapies is best suited to a particular situation. This book focuses on best uses of manual therapy, therapeutic exercises, postural training, dry needling, and acupuncture in the treatment of chronic TMD pain patients.

A large epidemiological study of 46,394 participants in Europe reported that 19 per cent of the population had moderate to severe chronic pain. Two-thirds used non-medication treatments, for example massage (30 per cent), physical therapy (21 per cent) and acupuncture (13 per cent), and 38 per cent reported that it had been extremely helpful. Interestingly, the type of physical therapy and the prevalence of its use ranged widely between countries, indicating large cultural differences. Most physical therapies are designed to treat various musculoskeletal pain disorders in the body. Although many features of the masticatory system are admittedly unique, we have learned that the mechanisms by which nociceptive impulses are initiated, transmitted, and perceived are not, as pain is more or less common throughout the body. This indicates that interventions which have been found to be useful at other sites in the body may also be useful in TMDs. Although evidence is limited, some modalities of physical therapy, such as jaw exercises, have been recommended in Swedish national guidelines in health care and as an integrated part of self-care in several publications. Based upon moderate evidence, the Swedish national guidelines for the treatment of orofacial pain currently recommend jaw exercises for TMDs, particularly from a health-economic perspective.

Several physical therapy modes are reportedly beneficial because they often activate the endogenous pain inhibitory modulation system; have few side effects; activate the patient by increasing body awareness and providing new pain-relief tools for home use; and facilitate communication with care providers. An additional benefit is that multimodal treatment with other therapies becomes easier and potentially more effective.

Chronic pain is often complex with comorbid pain conditions. An optimum treatment outcome almost always requires multidisciplinary collaboration with other medical disciplines. Although this book provides information on physical therapies useful for TMD patients and targets TMD professionals, such treatment may be delivered by other health professionals who may find the information contained between the covers of this book useful.

The book is divided into four parts, each containing several chapters. The first part deals with the epidemiology and classification of TMDs, nociceptive processing, and the pathophysiology of the masticatory system. These chapters provide the clinician with a deep understanding of the basic science of chronic pain. Part 2 focuses on the clinical case history and the clinical examination of the masticatory system and upper cervical region. The chapters in Part 2 detail the currently tested and accepted methods for assessing and examining the patient. Part 3 reviews various manual therapies for TMDs and neck disorders. This section highlights the available evidence-based literature and provides readers with scientifically sound and effective support for the use of these therapies. Part 4 discusses other interventions, such as acupuncture, and in addition,

the final chapter uses a biopsychosocial perspective to set up a framework for integrating physical therapies with other therapies in the management of chronic TMD pain patients.

Although the field of TMDs and orofacial pain has made great strides in the last few decades, clinical situations continue to remind us of the limits to our knowledge. As clinicians, we meet patients seeking help with pain and suffering on a daily basis. We must determine, to the best of our ability – based on the best scientific evidence available, our own clinical experience, and potential value to the patient – the treatment that will best provide an optimal outcome and quality of life for just this patient sitting in our chair. The present book was conceived and written for this purpose.

Treatment needs to be tailored for the individual chronic pain patient; this often implies different approaches or combinations of treatment modes such as behavioral therapies, pharmacologic treatment, occlusal therapy (splints), and physical therapy. One guideline overshadows all else: Select – always – the most conservative approach, and above all, do no harm. Patients need to feel believed, to know that all attempts to arrive at a correct diagnosis have been made, and to understand that appropriate treatment or referral to other specialists and therapists has been done when necessary.

I congratulate the contributing editors and authors, many of whom are recognized, leading experts in their fields and have contributed significantly to our current knowledge through their research and scientific publications. This book is a gem in its field, providing effective, trustworthy information to clinicians that will help alleviate orofacial pain and the suffering of their patients, and thus to some measure, or substantially, improve the daily experience of chronic pain patients.

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November 2017

FOREWORD by Jeffrey P. Okeson

During my professional career, I have had the opportunity to witness some very positive changes for our patients. One of these has been the blending of professional efforts in the area of orofacial pain. As we attempted to better understand pain, we began to appreciate the complexity of this field. Pain is one of the most powerful negative emotions we humans experience, yet we often struggle to help our suffering patients. We have come to learn that pain is far more than a sensation. Instead, pain is actually an experience, far more complex than a simple sensation. We have also learned that common sources of peripheral injury, thought to be the source of most pains, are not the problems we clinicians face. We now understand that when nociception enters the central nervous system it is greatly influenced by excitatory and inhibitory mechanisms. As a result, we have come to appreciate that pain is not exclusive to one medical discipline. Instead, our patients deserve the best that every discipline can offer to reduce their suffering.

This textbook is an example of this progressive thinking as it combines input from three different professions with the idea of providing the best care for our patients. In acute injuries, physical therapy can provide the necessary management that assists in recovery. It is important to recognize that when pain becomes chronic, central factors become a predominant component of maintaining the pain. With these patients, a multiprofessional team adds an important dimension to patient recovery. This textbook offers information from well-known authorities in physical therapy, orofacial pain and clinical psychology, which will help the clinician better understand what each discipline can offer. This multiprofessional effort offers the best possible success for patient management. A text like this is rare and the authors should be commended for their combined work. This professional endeavor is a reflection of the evidence-based science and the state-of-the-art efforts our patients deserve. The information found in this text will help all clinicians better evaluate and manage their patients.

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PREFACE

The term temporomandibular disorder (TMD) encompasses pain in the head and face, a condition which can be highly distressing and disabling for the patient. As clinicians, we should focus our attention on the therapeutic approaches than can help those patients. It is increasingly clear that the value of manual therapy, exercise, and needling therapies can be understood through the emerging concepts of pain neuroscience, and that all these interventions come together in a biopsychosocial model. In fact, manual therapy and exercise is probably the therapeutic combination most commonly used by many health care professionals for treating patients with chronic pain. Today, it is universally accepted that the central nervous system plays a critical role in the personal experience and clinical presentation of pain, and that manual therapy, exercise or needling therapies trigger peripheral and central nervous system responses. It was against this background of a growth in understanding of mechanisms that we were inspired to bring together a wide range of contributors from all over the world to provide a comprehensive and practical account of the diverse approaches to assessing and treating TMDs.

In conceiving and editing this book we have adopted the evidence and clinically informed paradigm. We believe that a combination of evidence and clinical experience should guide all clinicians in the management of individuals with chronic pain. The main feature of the evidence-based paradigm is that diagnosis and management should be guided mainly by the best available scientific evidence; however, the relevance of this doctrine can be limited since there is no good evidence for all intervention or diagnostic procedures that therapists use in daily practice. Although evidence-based practice is in continuous evolution, the evidence-*informed* paradigm is considered more appropriate since the clinician takes the best available scientific evidence and combines it with clinical experience while bearing in mind the patient's expectations and beliefs.

Throughout this textbook, chapter authors have integrated clinical experience and reasoning based on a neurophysiologic rationale with the most up-to-date evidence, thereby in effect combining the best of evidence-based and clinically based paradigms, mimicking what clinicians do in everyday clinical practice. We believe that this approach has created a textbook that truly provides practicing clinicians with what they need to know for real-life screening, diagnosis, and management of patients with TMD pain. This should be especially valuable since the multifactorial etiology and presentation that patients with TMD may exhibit can create a real challenge to the clinician.

The textbook is divided into four parts. In Part 1, several authors review the epidemiology and classification of TMD pain syndromes and the neurophysiological mechanisms underlying craniofacial pain. In Part 2, authors set out the steps for taking a comprehensive history in patients affected by TMD and the basic principles for the physical examination. In this section, authors clearly demonstrate the relevance of regional interdependence by showing why the thoracic and cervical spine should be also assessed in individuals suffering from TMD. The remaining parts cover therapeutic interventions for TMDs. Part 3 describes several manual therapy interventions, including joint, muscle, fascia, and neural interventions, and also therapeutic exercises. Finally, Part 4 covers other therapeutic options, including different needling therapies, by placing the field of these interventions within the context of contemporary pain neurosciences and neuroscience education.

We anticipate that this textbook will become the standard for manual management of individuals with TMDs and we hope that it will bridge apparent differences in opinion. We aim to unite different health care disciplines using manual therapy, exercise, and needling therapies as their therapeutic approach. We hope that the current textbook will ultimately benefit patients worldwide.

Cesar Fernández-de-las-Peñas, Juan Mesa-Jiménez
Madrid, Spain
January 2018

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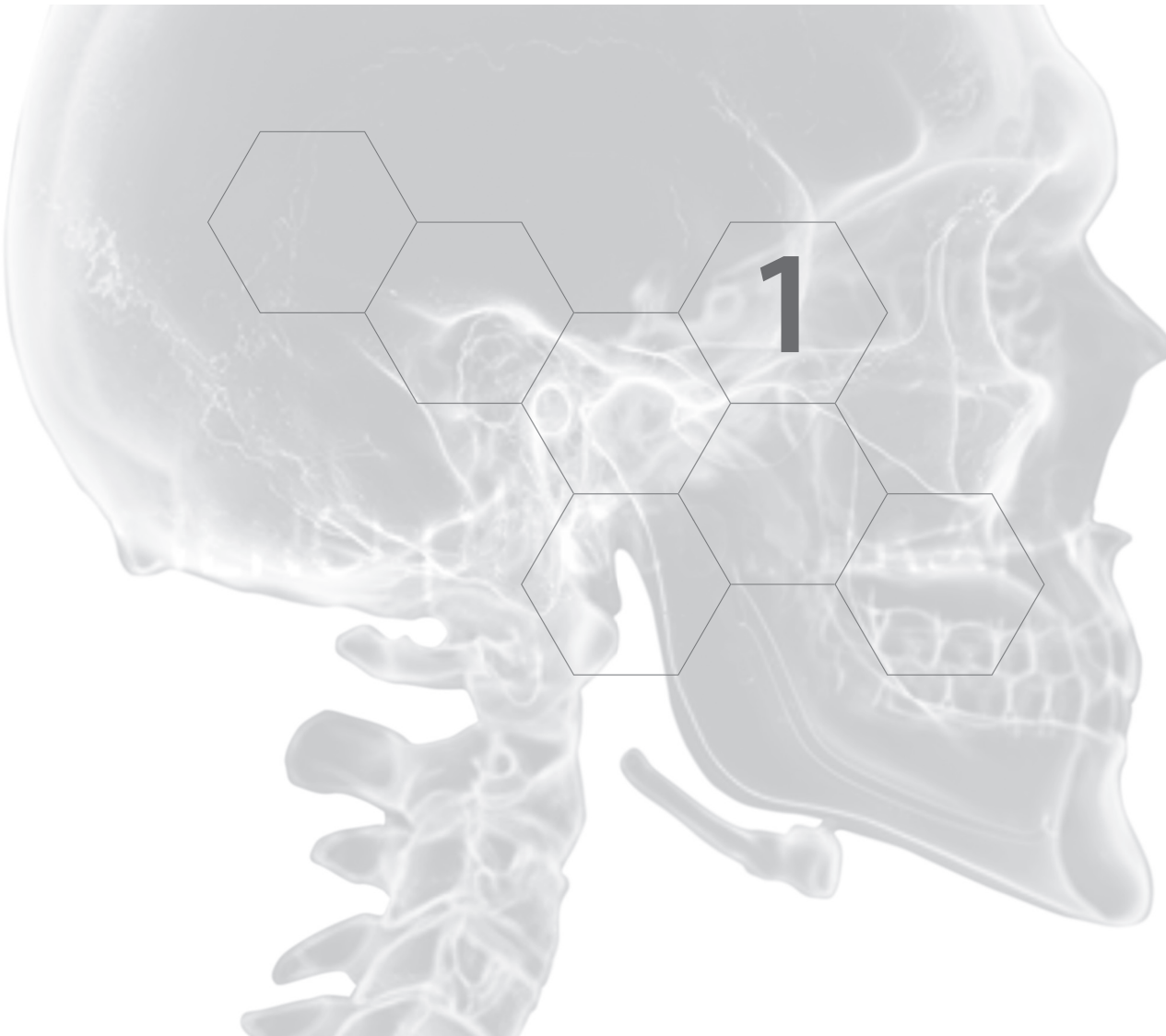
After many years working with patients with orofacial pain, and in consultation with Handspring Publishing, we agreed that the time had come to put together a textbook about TMDs. We did not really grasp how many people would eventually become involved in this project, but the result is the current textbook – made possible through the efforts of many individuals.

First, we would like to thank our coauthors, who prepared the chapters, and created a worldwide collaboration. In addition, the authors come from different health care professions, making this book a true multidisciplinary collaboration. Secondly, we would also like to acknowledge our patients who have taught us so much about what it is like to live with acute and chronic persistent TMD pain. Thirdly, we would like to acknowledge Handspring Publishing for entrusting us with the preparation of this textbook. We have been very impressed with the professionalism and enthusiasm of the folks at Handspring, and much appreciate their guidance and attention to detail.

Lastly, we are grateful to our families, friends, and colleagues. We realize that our professional activities, such as writing book chapters and teaching courses, do take us away from our families and friends. We appreciate their understanding and support for our endeavors.

Cesar Fernández-de-las-Peñas, Juan Mesa-Jiménez
Madrid, Spain
January 2018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PART 1

Introduction to temporomandibular disorders

Chapter 1

Definition, epidemiology and etiology of painful temporomandibular disorders

Sonia Sharma, Richard Ohrbach

Definition of temporomandibular disorders

Temporomandibular disorders (TMDs) are a group of pain conditions that affect the hard and soft structures of the orofacial region and are characterized principally by pain, limitation in jaw opening, and temporomandibular joint (TMJ) noises (de Leeuw & Klasser, 2013). Based on these principal characteristics, approaches have differed considerably as to how to conceptualize the disorders beyond their basic musculoskeletal character, which has in turn lead to differing diagnostic criteria. In fact, taxonomies and criteria are addressed in Chapter 2 of this textbook; here, we focus on definitions, and how definitions have influenced our understanding of the epidemiology and etiology of TMDs. Furthermore, definitions directly influence taxonomies, criteria, clinical practice, and research, including both the application of clinical research and research focused on classification. In this chapter, we apply the following definitions: 'TMDs' refer to the rubric as a whole, while 'a TMD' is a specific disorder.

According to the most widely accepted diagnostic methods and criteria, TMDs are the second most commonly occurring musculoskeletal condition in the USA after chronic low back pain (Lipton et al., 1993; NIDCR, 2014). In fact, worldwide estimates of their prevalence – approximately 10 per cent of the adult population are affected– appear to mirror those in the USA (Lipton et al., 1993; NIDCR, 2014). How TMDs broadly are defined will substantially influence their prevalence, as discussed in detail in the section on epidemiology later in this chapter; briefly, estimates can be as low as a few per cent if criteria are very restrictive, and as high as 60 per cent if criteria are relaxed. Very restrictive criteria might include requirement of multiple findings of masticatory musculature pain during mobility testing and palpation, and a high number of days, e.g., 20 in the previous month, with masticatory pain. Very relaxed criteria might be the presence of a single TMD-type symptom, say any TMJ clicking, in the previous six months. Therefore, to address this wide range of possibilities we should consider first the case definition.

Case definition

The purpose of a case definition is to provide an instantiated clarity, even if only temporarily, into a disorder; the clarity of that definition permits separation from other, perhaps more established (though not necessarily valid), diagnostic concepts. The particular case definition is constructed to fit within the context of intended usage in order to maximize reliability (in contrast to clinical diagnosis which is constructed to capture disease as it appears to occur in the natural world), which then permits the meaningful depiction of incidence, prevalence, risk factors, natural history, and clinical course of the disorder, without the burden of restrictions that might be imposed by a conventional diagnosis. An informed case definition is imperative when developing taxonomies and also when revising taxonomies; the instantiated clarity helps avoid circularity when bootstrapping diagnostic concepts from one phase of development to the next phase. In many instances, a given phase of development of a disease taxonomy may represent the current (and best) reference standard for the field; the improvement of a reference standard from one version to the next requires the inclusion of a third perspective.

Prior to the development of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) there was no single agreed-upon definition for TMDs as a global term encompassing a variety of subtypes. Several research groups focused on pain in the structures of the temporomandibular region

as the defining characteristic of a TMD, and hence the prevalence reported for TMDs has varied widely across studies. Based on the rationale that pain rather than symptoms such as joint sounds or jaw locking had a greater impact on individual suffering, interference with usual activities, increased economic burden due to lost productivity, and seeking health care, Dworkin & LeResche introduced the RDC/TMD (Dworkin et al., 1990; Dworkin & LeResche, 1992). This set of diagnostic criteria revolves around a common case definition for a given type of disorder, standardized examination methods, and standardized methods of gathering self-report information (Dworkin & LeResche, 1992). For further information on this, see the sections below on diagnosing TMDs using the RDC/TMD.

Given the wide differences in the prevalence of TMDs reported, ranging from just a few per cent to up to 60 per cent depending on how the disorders have been variously defined, the determination of the criteria that will correctly identify a 'real' disorder, that is one existing in nature and not just in the mind of the clinician, benefits from the inclusion of a case definition. For research purposes, choosing between a restrictive versus a relaxed approach is generally determined by the research goal for many types of studies. Studies purporting to have clinical generalizability will adhere to standards more suitable for the clinic: tests readily available in the clinic will be given priority. A clinician who believes that even a single episode of a TMJ click is a potential indicator of a subsequent clinical disorder will have a very low threshold for what qualifies as a disorder; a clinician concerned about, for example, the consequences of over-treatment for TMJ clicking that most of the time has very low morbidity will adopt, in contrast, a much higher threshold for severity of symptoms or findings in determining what qualifies as a disorder. Because TMDs, like most chronic pain disorders, have few, if any, pathognomonic markers, there is no clear objective marker of disease to serve as a reference standard by which to determine pathology versus nonpathology. This is particularly true for the disorders primarily characterized by pain, but even for joint disorders the existing reference standard of imaging only discloses the physical status of the joint structures, and the relationship between imaging-based findings and a disorder with clinical relevance has low diagnostic specificity, with the clinical disorder better explained by behavioral and psychological variables (Dionne et al., 2008; Johansson et al., 2008; Leeuw et al., 2007; Türp et al., 2016; Verkerk et al., 2015; Wasan et al., 2005). Consequently, reasoned decisions for how to construct a case definition, particularly for clinical purposes, revolve around multiple considerations.

Threshold considerations

Based on a simple but widely accepted TMD case definition for epidemiologic purposes, TMDs are associated with pain and disability and affect approximately 5–12 per cent of the population of the USA, and the estimated annual cost is US$4 billion (Lipton et al., 1993; NIDCR, 2014). Here, cost for treatment is determined by the threshold for what constitutes a disorder and, thereby, should be treated. For example, a low threshold for qualifying as a disorder may lead to multiple treatments as trial and error, and thereby increase costs for treatment and consequently lead to over-treating the disorder. A high threshold, in contrast, has the potential to identify the disorder beyond its representation by only simple symptoms and can therefore direct more targeted treatments, thereby reducing costs. Thresholds can be considered from within the disorder or based on the consequences of the disorder.

In order to consider the threshold by which a case definition should identify a clinical disorder, some critical terms require definition. Biomedical vocabulary standards for disease concepts vary widely and consequently for the present purposes we will use a set of definitions developed for ontology (Scheuermann et al., 2009). The selected definitions

of primary importance for this chapter are taken verbatim from this source:

- ***Disorder.*** A causally relatively isolated combination of physical components that is clinically abnormal and not readily reducible to some other entity.
- ***Pathological process.*** A bodily process that is a manifestation of a disorder.
- ***Diseases.*** A disposition to undergo a pathological process due to one or more disorders.
- ***Sign.*** A bodily feature of a patient that is observed in a physical examination and is deemed by the clinician to be of clinical significance.
- ***Symptom.*** A bodily feature of a patient that is observed by the patient and is hypothesized by the patient to be a realization of a disease.
- ***Normal value of a test (or finding).*** A value that is based on a statistical treatment of values from a reference population.
- ***Preclinical manifestation of a disease.*** A disease manifestation that exists prior to its becoming detectable in a clinical history taking or physical examination.
- ***Diagnosis.*** A conclusion of an interpretative process that has as input a clinical picture of a given patient and as output an assertion to the effect that the patient has a disease of a certain a type.

A case definition needs to be specific to the purpose of what needs to be identified, and within that it can be based on only primary characteristics (for example signs, symptoms or biomarkers), on a combination of primary characteristics, or on the inclusion of secondary characteristics. Table 1.1 gives a summary of pain terminology, representing domains that are widely regarded as primary domains of experienced pain and upon which case definitions are typically based.

One aspect of determining a threshold within the selected characteristics for the purposes of constructing a case definition resides on the normal value of the test or finding, and that in turn depends on a population of values in order to identify normal variability and thereby discriminate abnormality as a possible marker of a pathological process. One example within the diagnosis of masticatory muscle myalgia is whether the evaluation need only disclose one painful masticatory muscle from provocation testing (Diagnostic Criteria for Temporomandibular Disorders [DC/TMD]) or three such muscle sites (RDC/TMD) as a threshold for myalgia within the respective diagnostic systems. This may depend on the presence of a cofactor (for example the provoked pain must replicate the pain of clinical complaint, as in the DC/TMD) in order to protect against false positive diagnoses, given that hyperalgesia is expected in clinically normal individuals due to variations in pain perception, which leads to the ubiquity of simple tenderness from palpation unrelated to any clinical disorder (Dworkin & LeResche, 1992). In this example, the threshold for a disorder is based on the normal value of a test: in the DC/TMD, individuals without pain may have painful muscles due to simply being pain sensitive, but only individuals with a disorder will report familiarity of the provoked pain to some other recent experience (pain recognition); whereas within the RDC/TMD, the threshold of three painful muscle sites was based on the fact that non-cases may report pain from provocation, but statistically no more than two such sites are reported by individuals who did not report a recent history of pain. Normative values help to define an expected prevalence of a disorder versus the prevalence of a finding; for example, if positive findings for anterior disc displacement of the TMJ occur in one-third of the population, then this finding could result in one-third of the population having a disorder if the criteria are based solely on the imaging finding.

The threshold for identifying a disorder can also depend on its consequences, and TMD has three major types of consequences: limitation in function, disability, and chronic pain. Each of these major consequences critically questions what we mean by disorder. As defined above, a disorder is a combination

Table 1.1
Summary of pain terminology (adapted from Blau, 1982).

Pain attributes	Definition	Examples of common terms
Location	Site and spread of pain	Localized Radiating, referred, shooting
Intensity	Magnitude (amount) of pain in a single episode; includes temporal aspects of magnitude	6 out of 10 (on a scale of 0–10) Fluctuating, steady
Duration	Period of time of a single episode, that is, between two pain-free periods	Hours, days, weeks, months
Frequency	Pattern of pain over time	Every morning or evening Continuous (a pain 'episode' that persists for a month or more) Intermittent (two episodes a week)
Quality	Sensory and emotional aspects of an experience	Aching, burning, stabbing Punishing, intolerable
Modifying factors	Factors that initiate, increase, or decrease pain associated with an episode	Touching, washing, coughing, talking, medication, heat, cold
Timecourse	Course of pain over a long period of time	Acute, subacute, chronic, recurrent

of characteristics that are clinically abnormal, but if clinically abnormal characteristics do not disrupt the state of a subject's being, then the characteristics are merely findings. In short, can a constellation of features (symptoms, signs, or both; biomarker-based findings) be a disorder if there is no consequence (Wakefield, 1992)?

TMDs, as musculoskeletal disorders, are assumed to result in functional limitation of the masticatory system, with the extent of functional limitation only somewhat proportional to the severity of the TMD (Ohrbach, 2001; Ohrbach et al., 2008a). Functional limitation includes domains of mastication, jaw mobility, and verbal and emotional expression. If functional limitation is used as a measure of overall severity, individuals with a single positive finding (for example sporadic TMJ clicking) may well report no functional limitation and would correspondingly not be considered to have a painful disorder. An obvious exception would be when isolated clicking has prognostic value for subsequent development of a severe disorder; available data do not support a likely progression from simple clicking to a later disorder that, had the click been 'treated' as a preventive step, the disorder would have been more likely either prevented or minimized. Consequently, both pain and functional limitation represent sensible measures by which a threshold for a disorder may be determined based on available signs, whereas signs alone are poor indicators of a disorder as distinct from either a condition or the range of normal.

Disability is a person-level construct rather than a system-level construct of functional limitation. Disability associated with activities of daily living is a consequence of a TMD. While disability is not likely to occur in the complete absence of any functional limitation, disablement models clearly indicate that disability and functional limitation are not necessarily hierarchically organized (Ohrbach, 2001; Osterweis et al., 1987). The biopsychosocial model of pain indicates that disability due to a disorder is the consequence of not only any biological changes but also necessarily due to contributions from both psychological and social factors (Dworkin, 1991).

A final and relatively common consequence of a TMD is the development of chronic pain associated with that disorder. Three definitions of chronic pain are pertinent: pain that persists beyond the time of usual tissue healing, pain that has not responded to usual treatment for the identified disorder, or, the most commonly used, pain that persists beyond three months or beyond six months (depending on which standard one uses) (Turk & Rudy, 1987). All three of these definitions are useful at different times within both research and clinical practice; all three of these definitions are also more complex than usually regarded. The first definition assumes, based on the IASP pain definition (see below), that some sort of tissue damage (broadly defined) is present prior to the initial perception of pain, and that normal biological factors will result in healing of that tissue damage. Wall's model of injury (Wall, 1979) indicates that if pain persists beyond the normal healing time, then there is the increased probability of developing an acute pain disorder; moreover, Wall also indicates the increased probability of transition from injury to a chronic pain disorder. The possibility during the normal healing stages of transition to either acute or chronic disorder highlights the presence of risk factors occurring early in the normal healing process as well as later in the healing process. The implication of the IASP definition of pain as well as Wall's subsequent insights is that while obvious tissue damage does heal (and that pain may persist beyond that final healing stage, with the implicit conclusion that with no tissue damage, there should be no remaining source of peripheral nociception), pain may persist, and that pain may be causally related to the initial injury and associated nociception. There are, as Wall's insights suggest, several complications with a simple direct causal pathway. One is that primary nociception (primary and inflammatory; [Woolf, 2004]) may give way to neuropathic pain over time but with the interpretation that the primary tissue injury has healed and a new condition, functional alteration of the nociception-related parts of the nervous system, may emerge. Yet, primary nociception may still remain even after initial tissue healing, and in relation to more complex dysfunctional aspects that appear to now be more clearly defined in the pain research literature. Instead of chronic pain being a disorder of the central nervous system (CNS) in the absence of nociception, given the apparent absence of tissue damage, chronic pain, as a central disorder, may be more likely to co-exist with peripheral disorders that began as some sort of tissue damage (again, broadly defined intentionally here in order to be inclusive) but then progresses to dysfunction of a different sort, associated with nociception, but poorly understood (Moseley, 2003).

The second definition of chronic pain – pain that has not responded to usual treatment – is useful in the clinic for assessing patient factors (for example adherence) and tissue systems treated to date (for example acupuncture but not physical therapy, for a presumed musculoskeletal pain). However, there is a potential but inherent circularity in this definition of chronic pain, as this book points to repeatedly in the various chapters on treatment modalities. That is, this definition assumes that all forms of 'usual treatment' for this patient have already been sufficiently identified and that the usual treatment was provided in a manner consistent with the goal of so-called 'good clinical practices.' Yet, as Moseley indicates, physical forms of treatment to the body can vary enormously in how

they focus on the specific tissue system (or part of the tissue system) deemed responsible for presumed ongoing nociception, and a sufficient schema at the treatment level is essential for incorporating all factors (for example psychological, social, or behavioral) into the treatment plan so that a sufficient critical mass of therapies capable of making the kinds of changes necessary to effect change in the individual's chronic pain is included (Moseley, 2003).

The third definition of chronic pain, based on a period of either three or or six months' duration (IASP, 2011), is clearly arbitrary, but in the clinic (or for research purposes) when one has no other milepost to anchor a potential classification of chronic pain for that subject, the time base is at least pragmatic. See Table 1.2 and Figure 1.1 for a summary of pain types according to duration. The classic intervals of three months and six months have emerged largely as a fail-safe approach: the body and the person have been given the benefit of the doubt for recovery from the initial event that caused the pain, and three months or six months later any persisting pain should therefore be considered evidence of a dysfunctional central pain processing network; treatment should therefore escalate from simply (and often only) focusing on the disorder per se at the bodily level and now incorporate consideration of the many psychological, social, and behavioral factors that can contribute to ongoing pain as well as interfere with response to usual treatments. The primary problem with this approach to defining chronic pain lies in the choice of temporal threshold: emerging data indicate that six months is too long and that three months is probably too long in terms of the factors that can emerge following initial onset, say via injury and which are already active in affecting behavior, interfering with typical treatment response, and perpetuating chronicity. Earlier treatment is superior (Epker et al., 1999).

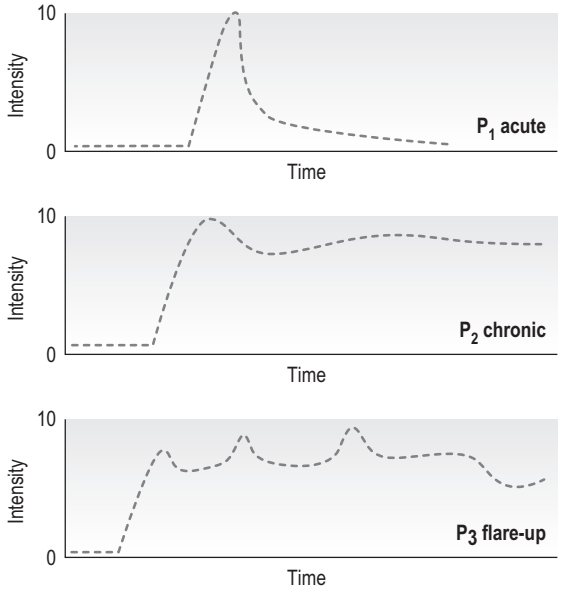

Figure 1.1
Time frame for types of pain.

In summary, the threshold for a clinically oriented case definition of a TMD appears to be best based on normative data from the population coupled with functional consequences specific to the identified organ system. The normative data, at this time, should be based on primary symptoms rather than signs, given the absence of prognostic validity for a case definition based solely on signs. Returning to the earlier example, if one-third of the population have anterior disc displacement of the TMJ as disclosed by imaging, but less than 10 per cent of those individuals report symptoms or functional consequences of the disc displacement, then this imaging finding would not constitute a disorder. If, in contrast, the imaging finding has prognostic value for increased risk in the future for developing TMD or for being a factor that contributes to worsening of the TMD after onset, then the imaging finding would constitute a disorder (as per the ontological definition above). Disability associated with a constellation of features is not a useful marker for setting the threshold of whether that constellation of features represents a disorder or

Table 1.2
Summary of pain types according to duration. Clinical assessment and characterization of an individual's reported pain result in classification within one of these categories. Case definitions also build on these time constructs (adapted from Von Korff, 1994).

Pain type	Definition
Acute	Pain of recent onset that is not recurrent or chronic and that has persisted for less than three months
Subacute	Less recent pain condition, sometimes incorrectly used to depict less severe pain which is in contrast to a severe acute pain
Chronic	Pain that persists for three months or more, or six months or more Pain that persists beyond the time of usual healing Pain that is nonresponsive to usual treatments
Recurrent	New episodes of pain bouts that repeatedly recur after pain-free periods over a longer timescale, for example, menstrual headache Also when pain is present for less than half the days in a specified time period (12 months), occurring in multiple episodes over the year
First-onset	Episode of pain that is the first occurrence of a particular pain disorder in a person's lifetime
Transient	A pain episode of not more than 90 consecutive days that does not recur over a 12-month observation period

not; this is largely due to the inclusion of other factors outside the target disorder contributing to the development of disability. The net effect would probably be a too extreme form of clumping. Similarly, the potential for the emergence of chronic pain may not be a reliable or even valid consideration for how to set the threshold of a disorder for diagnostic purposes.

Disability and chronicity are not integral to the disorder; recognizing this distinction is an important concept regarding the boundary of a disorder, which has, as one of its two core criteria, nonreducibility to another disorder. For example, should a TMD be defined to necessarily also include cervical pain or cervical dysfunction as part of the diagnostic criteria? The DC/TMD has been criticised for not being sufficiently inclusive because it excludes cervical findings. Certainly, a basis for that criticism could include the well-established neurological and mechanical linkage of symptoms and motor function, respectively, of the masticatory and cervical systems. But because either cervical or masticatory system problems could exist separately from the other system, a case definition based on a combined system would not represent the smallest nonreducible unit of a disorder.

Pain definition

While pain can be informally defined as physical suffering or discomfort caused by illness or injury, the scientific (and far more clinically useful) definition is 'An unpleasant sensory and emotional experience

associated with actual or potential tissue damage, or described in terms of such damage' (IASP, 2011). The implications of this definition are that three subtypes of pain exist: pain accompanying actual tissue damage, pain accompanying what is called 'potential tissue damage,' and pain accompanying a description (in some patient reports) involving reference to tissue damage (Smith et al., 2011). In one sense, these three aspects of tissue damage represent the degree of certainty on the part of the patient or provider for whether the initial pain onset was linked to evident nociception from known tissue damage. Note that pain accompanying the least certain form of evident nociception with actual tissue damage, pain described 'in terms of such damage,' has been included in a clinical category termed functional pain (Woolf, 2004) and it is well-known to be the source of much clinical and research speculation as well as confusion. That category probably accounts for the majority of individuals with chronic pain in general and certainly the largest category of individuals with chronic TMD pain. Moreover, it is likely to be the most common type of pain associated with the various disorders identified in this book for targeting via new treatments.

A proposed revised definition of pain is stated as 'pain is a distressing experience associated with actual or potential tissue damage with sensory, emotional, cognitive, and social components' (Williams & Craig, 2016). This definition notably eliminates the complex aspect of the current pain definition, phrased as '…or described in terms of such [tissue] damage'; Williams and Craig acknowledge that this phrase was a part of the current definition to intentionally include individuals who complained of pain in the clear absence of any detectable evidence of tissue damage, suggesting that this was more for political expediency than out of scientific necessity. Nevertheless, the authors further acknowledge that strong evidence of neuroplastic CNS changes in relation to the course of pain supports the wisdom of the decision to previously include that phrase, while further indicating that knowledge advances suggest that such individuals should now perhaps be classified within a different diagnostic system, reserving 'pain' for only those with actual or potential tissue damage. Yet, clinically more often than not, pain histories associated with, for example a TMD diagnosis, do not identify any particular prior 'actual or potential tissue damage' event strongly associated with known nociception. If such patients are compared to those with a clear history of prior injury, symptom descriptions are typically indistinguishable (aside from specifics related to an injury, if present), and the findings from both types of patients are equivalent in terms of resultant diagnoses (for example, myofascial pain). While the presence or absence of prior tissue damage may suggest differing mechanisms at the time of onset, the mechanisms associated with subsequent stages of transition to an acute pain disorder or further to chronicity may not differ between these types of subjects and consequently, the current definition qualifier of '…or described in terms of such [tissue] damage' appears to remain critically important for an appropriately inclusive domain of pain. Critical analysis of the current IASP definition readily encompasses this broad inclusive framework regarding the marked extremes in examiner certainty about prior injury and associated tissue damage, in that the absence of observable tissue damage does not exclude more subtle levels of tissue damage that we are unable with current methods to assess (Smith et al., 2011). In addition, the relation of a reported injury to nociception known to be determined by tissue damage is not understood; moreover, the range of stimulus, vis-à-vis injury, varies from doubtful tissue injury to certain tissue injury. Consequently, the current pain definition appears to be superior for capturing the relevant clinical phenomena for both diagnosis and treatment, especially in relation to new approaches to treatment using a case definition that builds upon 'pain' as currently defined.

Application of a case definition to development of a diagnostic system

The RDC/TMD system developed in 1992 consists of reliable and validated criteria used to

examine, diagnose, and classify most common forms of muscle- and joint-related TMD musculoskeletal conditions. Based on the biopsychosocial model of pain the RDC/TMD consists of a dual axis approach: Axis I (physical findings) and Axis II (pain-related disability and psychosocial status). The Axis I measures are used to obtain physical diagnosis through a clinical examination which assesses regional pain in the past 30 days as well as current pain from provocation of masticatory musculature and the TMJ via jaw mobility and palpation. The Axis II measures of the RDC/TMD are intended to determine the extent to which cognitive, emotional, or behavioral impairment contribute to the development and maintenance of TMDs. More specifically, these measures assess jaw disability during function, psychological status, and psychosocial level of functioning, and are obtained through reliable and validated behavioral and psychological tests. Based on the RDC/TMD clinical examination protocol, TMDs can include any of the three groups of diagnoses; Group I (muscle disorders), Group II (disc displacements) and Group III (joint disorders) or a combination of any of the subgroups.

In 2010, the RDC/TMD Validation Project was carried out to determine the sensitivity and specificity of the original RDC/TMD. Using a simple case definition of regional pain in the prior 30 days, revised draft diagnostic criteria were developed and two calibrated but independent examiners provided, by consensus, reference standard diagnoses for the pain disorders. According to the Validation Project the following sensitivity and specificity for the Axis I diagnostic algorithms were found for the two main muscle group diagnoses: Group Ia myofascial pain (sensitivity 0.65, specificity 0.92) and Group Ib myofascial pain with limited opening (sensitivity 0.79, specificity 0.92) (Truelove et al., 2010). Realizing that none of the individual diagnostic groups met the target sensitivity of ≥ 0.70 and specificity of ≥ 0.95, and that the targeted sensitivity and specificity were observed only when both Group I diagnoses were combined into any myofascial pain (0.87 and 0.98, respectively), the Validation Project's results strongly suggested a need for developing a revised RDC/TMD (Truelove et al., 2010).

Revising the eight Axis I RDC/TMD diagnostic algorithms subsequently demonstrated the evaluation method to be valid for the most common pain-related TMDs. The criterion measure for this study included a comprehensive history and clinical measures, panoramic radiographs, bilateral TMJ MRIs, and bilateral TMJ computed tomography. A calibrated board-certified radiologist interpreted all images and diagnoses were made by consensus of two TMD experts who independently assessed all participants using the criterion protocol. In case of disagreement final diagnoses were made using the radiologist-interpreted images. The sensitivity and specificity of the revised algorithm for myofascial pain (0.82, 0.99, respectively) and myofascial pain with limited opening (0.93, 0.97, respectively) exceeded the target levels of sensitivity and specificity. On combining diagnoses for any myofascial pain, both sensitivity (0.91) and specificity (1.0) were further increased (Schiffman et al., 2010). Further, the kappa coefficients increased from 0.60 and 0.70 in the original criteria to 0.73 and 0.92 in revised criteria for myofascial pain and myofascial pain with limited opening respectively (Schiffman et al., 2010).

In summary, a broader case definition was more useful than a constricted one for a heterogeneous pain condition such as myofascial pain. For example, the Validation Project recruited individuals with at least one of the three cardinal signs or symptoms of TMD (jaw pain, limited mouth opening, or TMJ noise) as potential cases of TMD; those without TMD signs and symptoms by history and on clinical examination were included as potential controls. This broad inclusion not only helps discriminate between patients with and without TMD pain, but also helps discriminate between patients with TMD pain and patients with orofacial pain complaints of non-TMD origin.

Epidemiology of temporomandibular disorders

Incidence and prevalence

Epidemiological studies that have used USA nationwide data such as that from the National Health Interview Survey (NHIS) and the National Health and Nutrition Examination Survey (NHANES) have mostly relied on the self-report of face or jaw pain. In addition to dental oral examinations performed, range of motion and muscle tenderness were also measured in NHANES in the interest of obtaining objective data on TMDs. However, these measures were insufficient to meet the requirements of valid diagnostic criteria of any type of TMD. Due to the use of different case definitions, and due to ambiguity in the use of terminology (such as point prevalence versus period prevalence as boundaries for time) the global prevalence of TMD pain varies considerably across studies. For example, using self-reported information, the prevalence estimates for facial pain have been found to range from 3.7% (Agerberg & Bergenholtz, 1989) and 4.6% (Plesh et al., 2011b) to 12% (Von Korff et al., 1988). Facial pain is often considered to represent TMD pain in large part because TMD pain represents a higher prevalence among orofacial pain compared to other nondental pains.

Attempting to subtype TMD pain based on self-report methods may not be accurate, as the method of capturing potential cases matters greatly in terms of confidence in case ascertainment, and consequently the prevalence of self-reported TMD pain subgroups such as myalgia and arthralgia varies across studies. See Table 1.3 regarding prevalence in adults with ambient TMD pain or functional TMD pain. Using NHIS data, which is based only on self-report, Lipton et al. (1993) reported an overall prevalence of 5%–12% based on subtypes of facial location and pain quality. More specifically with regard to TMD subtypes Lipton et al. (1993) reported a 6.5% prevalence for jaw joint pain and 1.5% prevalence for muscle pain. In contrast, when a validated clinical examination protocol (for example RDC/TMD) was used, the prevalence of individual diagnostic subgroups of pain-related TMD was found to be higher for a diagnosis of only myalgia at 25% than for TMJ conditions at 4.2%, which included both painful and nonpainful joint diagnoses (Drangsholt & LeResche, 1999). The most likely explanation for this set of contrasting findings is that respondents are unable to reliably distinguish muscle and joint structures when the mode of evaluation is only by self-report. Consequently, examinations are essential for prevalence estimates of subgroups to be accurate, but they may not be feasible in all settings. More general estimates that do not distinguish subtypes are likely reliable via self-report. Furthermore, methodological weakness and limitations of epidemiological studies on TMD pain prevalence do not only lack reporting prevalence estimates by subtypes, but also lack other attributes; see Table 1.4 for further details.

There are a few prospective studies that report TMD incidence. The two studies on adolescents that report incidence of TMD region pain or jaw pain show that the cumulative incidence varies from 1.8% (Heikinheimo et al., 1990) to 2.8% (Kitai et al., 1997) per year for a 3–5 year follow-up interval. The studies on adults aged 18–65 years reported a cumulative incidence of 2.2% (Drangsholt & LeResche, 1999; Von Korff et al., 1993). The above studies highlight that based on self-reported information, estimates of TMD incidence can vary, and again emphasize the importance of valid clinical measure of TMD. More recently the OPPERA study found the incidence rate of lifetime first-onset TMD to be 3.9% per year, based on a median follow-up of 2.8 years following enrollment in participants aged 18–40 years old (Slade et al., 2013). Prospective studies on persistent TMD are scarce, probably because of methodological challenges. One study that assessed for persistent TMD, defined as pain present for ≥ 180 days of the prior 360 days in a group of 1061 Health Maintenance Organization (HMO) enrollees aged 18–65 years, found an incidence rate of 1.2 per 1,000 person-years

Table 1.3
Pain prevalence by type of temporomandibular disorder (TMD).

TMD type	Reference	Disorder definition	Source of sample	Sample size	Age range (years)	Prevalence (%)
Prevalence of ambient TMD pain in adults	Helkimo, 1974	Facial and jaw pain	Finnish Lapps	600	15–65	12
	Molin et al., 1976	Frequent pain in front of ears	Swedish males in the military	253	18–25	5
	Szentpetery et al., 1986	Recent pain in face, neck, or around ears	Hungarians	600	12–85	5.8
Prevalence of functional TMD pain in adults	Agerberg & Carlsson, 1972	Face hurts when yawning	Swedes	1,106	15–74	12
	Osterberg & Carlsson, 1979	Pain when opening the mouth wide to take a large bite	Older Swedes	348	70	3
	Alanen & Kirveskari, 1982	Pain in jaw joint on chewing	Finns	853	18–57	5.2

(Von Korff et al., 1993). Further findings on persistent TMD from the OPPERA study are currently under evaluation and have not yet been published.

Risk factors

In contrast to etiology, risk factors represent a broader domain of factors contributing to a disease, including those that clearly precede disease onset as well as factors that may co-occur at onset or factors that are a consequence of the disease at some stage and which serve to perpetuate the disease. Given the difficulty of research designs in partitioning these various stage-specific roles, we make no distinction here in the timing of the contribution of a particular factor but instead present them as generally identified via cross-sectional designs.

Age and gender

Based on an inclusive definition of TMDs that queries for face or jaw pain in the past three months from the date of interview, the age-specific prevalence patterns for TMDs have been stable over the years. Using the NHIS data, Lipton et al. (1993) reported the following age-specific prevalence for face or jaw pain: 6.5% in those aged 18–34 years, 5.0% in 35–54 year-olds, 4.0% in 55–74 year-olds, and 3.9% among individuals ≥ 75 years old, indicating a slight decrease in TMDs as age increases. According to the

Table 1.4
Methodological weakness and limitations of epidemiological studies on TMD pain prevalence (extracted from Drangsholt & LeResche, 1999).

Methodological weakness or limitation	Studies (n=133) with problem
Inadequate sample size	> 80%
Study is not performed on representative sample of a defined population	> 50%
Case definition does not include pain or depends solely on physical assessment	> 50%
Case definition of pain is not explicit: it does not include severity or duration	> 95%
Age- and gender-specific proportions are not given	> 75%
No mention of spread or dispersion of data; that is, no confidence intervals	> 95%

most recent NHIS, the prevalence of pain in the face or jaw was 4.6% among all persons aged 18 years and older. The age-specific estimates were 5.0% in those aged 18–44 years, 4.6% in 45–64 year-olds, 4.2% in 65–74 year-olds and 2.6% in those ≥ 75 years old, again suggesting a monotonic decrease in prevalent jaw or face pain across the adult life-span (NCHS, 2014). However, it is important to be careful in interpreting estimates from national health surveys of face and jaw pain as necessarily representing TMD pain in that the estimates are based solely on self-report questions focusing on pain location. The only suitable epidemiologic tool that has been developed is a brief set of self-report questions with high sensitivity and specificity for TMDs (Gonzalez et al., 2011).

In contrast, some prospective studies show an increase in incidence of TMD with increase in age. More recently, the OPPERA study enrolled individuals between the ages of 18 and 44 years old who were confirmed to have never had diagnosable TMD, and based on subsequent development of clinically diagnosed TMD, the following age-specific incidence rate of TMD per year emerges: 2.5% for individuals aged 18–24, 3.7% in those aged 25–34, and 4.5% in those aged 35–44. Furthermore, using the age group of 18–24 years as the reference group, the association (hazard ratio [HR] statistic) between age and TMD showed a 40% increased risk for TMD among individuals aged 25–34 (HR: 1.4, 95% CI 1.0, 1.9) and a 50% increased risk among individuals aged between 35 and 44 years (HR: 1.5, 95% CI 1.0, 2.0) (Slade et al., 2013).

Cross-sectional studies show that prevalence estimates for men (0%–10%) and for women (2%–18%) vary and show that women are 1.5–2 times more likely to be afflicted with TMDs than men (Helkimo, 1974; Plesh et al., 2011a; Von Korff et al., 1988). Recent NHIS data have also shown higher prevalence of face or jaw pain in females (5.8%) than in males (3.4%) (NCHS, 2014). Furthermore, using pooled data from NHIS from the years 2000–2005, Plesh et al. (2011b) reported that females had a significantly higher frequency (odds ratio [OR]: 1.41, $P < 0.001$) of two or more comorbid pain conditions in comparison to males (Plesh et al., 2011b), indicating that the higher rate of TMD pain in females extends to additional pain disorders. Similar results have been reported in longitudinal studies. For example, Von Korff et al. (1993) also found a higher cumulative incidence of TMDs in women (2.6% per year) compared

to men (1.6% per year) in a three-year follow-up. More recently the OPPERA study reported a 3.6 cases of TMD per 100 person-years at-risk incident rate in females compared to males at 2.8 cases of TMD per 100 person-years at risk in a five-year follow-up, but this difference was nonsignificant (HR: 1.3 95% CI0.9, 1.7) (Slade et al., 2013).

Race and ethnicity

As seen with low back pain and neck pain, the prevalence of face or jaw pain is higher in individuals of mixed race. Recent prevalence estimates for jaw or face pain were 4.9% for single-race White adults and 4.0% for American Indian or Alaska Native adults. In contrast, a higher percentage (12.7%) was reported in both mixed-White and American Indian or Alaska Native adults (NCHS, 2014). But among single-race individuals, prevalence rates for Whites were still slightly higher (4.9%) compared to African Americans (3.8%) and Asians (2.1%) (NCHS, 2014). This pattern is the same or nearly the same as observed for back pain. Similarly, the OPPERA study has also shown that the incidence of clinically diagnosed TMD was only slightly higher in African Americans (4.6%) compared to Whites (3.0%). Furthermore, the association between race and TMD was higher for African Americans (HR: 1.4, 95% CI 1.0, 1.9) than for Hispanics (HR: 1.2, 95% CI 0.6, 2.1) (Slade et al., 2013). The prevalence for Whites and African Americans varies across the different studies but stays in the same range and it seems safe to conclude that they are approximately the same. Data indicate that the prevalence in mixed-race people is more limited; this is consistent with their low overall prevalence in the population.

Education and socioeconomic status

Studies on TMD and education or socioeconomic status are scarce. Using a population of health maintenance organization enrollees, education was not associated with any of the five major pain conditions including pain in the face (Von Korff et al., 1988). More recently, the NHIS data regarding education and jaw pain show that the differences in jaw pain among the different educational groups are minimal. For example, the 2014 NHIS found the following prevalence estimates for face or jaw pain by education level: 5.1% for less than high school diploma, 4.4% for high school diploma, 5.0% for some college education, and 4.4% for bachelor's degree or higher (NCHS, 2014). Furthermore, specifically for TMDs, the OPPERA study did not find significant associations between education and incidence of TMDs. In contrast, association with self-reported rating of satisfaction with material standards of life (a possible surrogate measure of socioeconomic status), showed a decreased association with incidence of TMDs (HR: 0.87, 95% CI 0.76, 0 .98). Similar results were reported when categorizing the variable: greater TMD incidence (HR: 1.71, 95% CI 1.16, 2.51) was found in the individuals with the lowest ratings (0–5) compared to the individuals with highest ratings (9–10) (Slade et al., 2013).

Body weight and physical activity

Literature on body weight and TMDs indicates that body mass index (BMI) is unlikely to be a putative risk factor for TMD pain. A study using participants from the University of Washington Twin Registry assessed the association of excessive weight and obesity with five distinct pain conditions and three pain symptoms, and further examined whether familial influences explained these relationships. After adjustment for age, gender, and depression, overweight twins were more likely to report TMD pain than normal-weight twins (OR: 1.49, 95% CI 1.03, 2.17); however, after further adjustment for familial influences or genetic factors, these associations hardly changed (OR: 1.44, 95% CI 0.99, 2.09) (Wright et al., 2010). Similarly, the OPPERA study found BMI to be a putative risk factor for first-onset TMD in analysis that adjusted for study site and demographic characteristics (HR: 1.13, 95% CI 1.00, 1.26), but its effect was attenuated to statistical nonsignificance after imputation for loss to follow-up (HR: 1.09, 95% CI 0.97, 1.23) (Sanders et al., 2013).

Physical activity has no documented studies regarding its association with TMD, despite emerging models suggesting that increased activity should increase pain resilience (Ambrose & Golightly, 2015; Ahn, 2013).

Comorbidity of pain at other sites

Using pooled data from NHIS from the years 2000–2005, Plesh et al. (2011b) have reported a prevalence of 4.6% for TMD-type pain, and of those who reported TMD-type pain approximately 59% had two or more additional complaints of pain either in the neck, low back, or another joint. Furthermore, with regard to involvement of other body sites across the different races, Plesh et al. (2011b) have also reported that in comparison to Whites, Hispanics (OR: 1.56, $P < 0.001$) and Blacks (OR: 1.38, $P < 0.01$) reported significantly higher frequencies of two or more pain complaints in the neck, low back or another joint. Similarly, the OPPERA study also found that incidence of TMD was higher for ≥ 2 comorbidities (HR: 2.70, 95% CI 2.02, 3.59) than that for a single comorbidity (HR: 1.42, 95% CI 1.00, 2.01) compared to no comorbidity. In addition, and compared to no back pain, ≥ 5 back pain episodes strongly predicted TMD risk (HR: 2.20, 95% CI 1.54, 3.14) (Sanders et al., 2013).

Smoking

Studies that have examined the association of smoking with TMDs have found, compared to non-tobacco users, that tobacco users had a higher odds for TMD (OR: 4.56, 95% CI 1.46, 14.24) (Weingarten et al., 2009). Furthermore, a dose-response relationship was noted between smoking and the intensity of TMD pain using a 0–10 numeric rating scale: among light smokers (mean: 5.8 ± 1.8); among moderate smokers (mean: 6.3 ± 2.3) and among heavy smokers (mean: 8.1 ± 1.4) (Melis et al., 2010). Interestingly, among female Caucasians, a stronger association between smoking and TMD occurs in younger women < 30 years (OR: 4.1, 95% CI 1.6, 11.4) than in older women ≥ 30 years (OR: 1.2, 95% CI 0.6, 2.8). Furthermore, after adjusting for allergy-related conditions, cytokine mediators and psychological variables, the association was reduced by approximately 45% in both younger women (OR: 2.3, 95% CI 0.81, 6.43) and in older women (OR: 0.66, 95% CI 0.26, 1.68). The above findings indicate that the effect of one or more of the explanatory factors was higher among the younger than the older individuals (Sanders et al., 2012).

Occupational factors

The most common occupation potentially related to TMDs appears to be playing musical instruments, which has received widespread consideration albeit mostly based on poor-quality studies (van Selms et al., 2017). Studies on other occupations and their potential relation to TMDs are rare, and therefore for our purposes studies on musicians and TMDs will be further described. A case-control study, notable for its overall methodological quality, found greater prevalence of signs and symptoms of TMDs among a group of violinists than among control subjects who did not play musical instruments (Rodriguez-Lozano et al., 2010). Compared to controls the most commonly and significantly different detected clinical features in the violinists were parafunctional habits such as tongue thrusting, mouth breathing, and biting of nails (26.8%), TMJ sounds (51.2%), and pain on maximum mouth opening (24.4%). Overall, the evidence from association studies of musical instrument playing and TMDs is mixed; associations are more common in studies utilizing clinical examinations for case ascertainment (van Selms et al., 2017). Evidence from experimental trials, however, varies. For example, one study of wind instrument players found that the contractive load on jaw closing muscles measured using EMG activity of jaw muscles was small when playing both medium and high tones on a wind instrument, and playing an instrument for a long time did not induce fatigue of the jaw-closing musculature (Gotouda et al., 2007). An experimental study of 30 musicians that examined the effectiveness of oral splints for TMD found

that treatment with oral splints contributed to a significant decrease in dental or TMJ pain in 83% of the participants (Steinmetz et al., 2009). Note: most studies regarding musical instruments and TMDs have focused on TMD symptoms but not TMD diagnosis. A related experimental study found striking evidence among 14 musicians with upper limb pain for increases in same muscle activity in response to pain experience recall and for increases in the opposing trapezius in response to stress imagery (Moulton & Spence, 1992) suggesting that, as stated in our section on etiology, single causes for TMDs (or other musculoskeletal pain) are the exception, and instead the compounding effect of multiple-risk determinants is important.

Psychological factors

An abundance of evidence exists explaining the role that psychological factors play in TMD onset and chronicity. For TMDs specifically, mood disorders and personality disorders are significantly linked to muscle disorders, as opposed to disc or joint disorders (Kight et al., 1999). Studies also show that psychological distress is associated with greater severity and persistence of TMD-related clinical symptoms. More specifically, psychological stress and depression levels are found to be higher in individuals with chronic TMDs (Dworkin et al., 1990; Gatchel et al., 2007; Keefe et al., 2004). Moreover, the OPPERA study found an array of psychological factors that were associated with TMDs, among which the highest hazard ratio was found when using the Pennebaker Inventory of Limbic Languidness – a measure of somatic symptoms, for example aches, soreness, and tightness (HR: 1.44, 95% CI 1.29, 1.60). Among the four identified latent psychological constructs, stress and negative affectivity components (HR: 1.12, 95% CI 0.97, 1.30) and global psychological symptoms (HR: 1.33, 95% CI 1.18, 1.50) were the strongest risk factors, but showed only a modest effect, with a 12% and 33% increase in risk for first-onset TMD respectively (Fillingim et al., 2013). Furthermore, using repeated measures of stress, the OPPERA study found that stress measured during the same three-monthly period as TMD onset was associated with a 55% increased risk for TMD (HR: 1.55, 95% CI 1.34, 1.79) (Slade et al., 2015). Both of these studies showed that the estimated associations with stress that are measured at enrollment and during the three-monthly follow-up period are likely to be underestimated, because the values measured at both time points do not capture the accumulated effects of stress and global psychological symptoms that are intrinsic to everyday experiences. For example, the increased association with stress reported by Slade et al. (2015) using a measure that is more proximal in time to the outcome still demonstrates a one-time measure of a construct that is transitionally or temporally dynamic and is insufficient to capture its true association.

Etiology of painful temporomandibular disorders

The suspected etiology of TMDs has been as broad as the imagination of both clinician and researcher, but opinions have largely been based on case-series or cross-sectional studies, neither of which represents adequate approaches to the determination of etiology. Here, we address major domains related to the potential etiology of TMDs. Advances in identifying genetic and epigenetic etiological factors associated with pain and TMDs in particular are substantial (Belfer et al., 2013; Diatchenko et al., 2013; Smith et al., 2013); however, this complex area is beyond the scope of the present chapter.

The Bradford Hill criteria for causation and therefore for understanding etiology are appropriate and productive for bacterial diseases, but they are less useful for complex diseases, a category to which TMDs clearly belong (Ohrbach et al., 2015; Rothman & Greenland, 2005). Consequently, in considering the etiology for complex diseases such as TMD, the concept of risk determinants becomes more appropriate. This will perhaps become clearer by the end

of this section, as we present the evidence in support of a simple summary:

'TMD is less often a single isolated disorder and rather is more often the result of multiple risk determinants occurring together or in some sequence specific to an individual, such that no single risk determinant is sufficient, on its own, to "cause" TMD.'

— (Slade et al., 2016)

Injury

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. Among the various traumatic events that have been reported in the literature, assaults are the most frequent at 37% of all facial fractures in emergency department visits, followed by falls at 24.6%, motor vehicle accidents at 12.1%, transport accidents at 2%, and pedal cyclist accidents at 1.6% (Allareddy et al., 2011). In addition to assaults, falls and accidents as sources of injuries to the jaw, other forms of injuries that can affect the jaw include head and neck injuries (Cassidy et al., 2014).

Furthermore, 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 lips, teeth, tongue, etc. it is difficult to parse out from the literature how many injuries affect the jaw bones or muscles specifically. For further details on sources of jaw injuries see the article by Sharma (2017).

Microtrauma

Musculoskeletal microtrauma represents low magnitude forces insufficient to cause sudden disruption in the overt integrity of the involved tissue but which over time lead to physical damage to the body (Fernandez et al., 1995; Hauret et al., 2010). The most common cause of microtrauma appears to be overuse behaviors, with parafunctional habits being a dominant source for the orofacial region and with sports activities being the main source for the remaining body regions. Overuse, in contrast to normal use, is determined based on a combination of extent of load imposed by the behavior, frequency of the behavior, the duration of each particular behavior, the duration of all behaviors per bout, the extent of recovery periods, and the calendar time over which this occurs. In general, no specific measure of overuse behavior exists, and the threshold at which overuse, as opposed to normal use, is identified is generally unknown. Instead, the presence of overuse is often inferred when the threshold for tissue adaptation is exceeded, and either signs or symptoms putatively of microtrauma have appeared. Other potential causes of microtrauma include repeated strain (such as the biological response to repeated external forces on the body, for example from contact sports) and the effects of deconditioning resulting in decreased tissue resilience to load (Nørregaard et al.,1997).

Physical damage may be readily assessable with a disorder such as traumatic arthropathy or tendonitis, often stated to be the result of microtrauma; the clinician may be certain of the diagnosis, and inference leads to the putative etiology of microtrauma based on good evidence in the particular patient of microtrauma-inducing behaviors such as forced marching over long distances, over many days, without rest periods for tissue recovery. In such a context, microtrauma appears to be strongly supported by empirical data (Hauret et al., 2010).

Contrariwise, the presence of physical damage associated with microtrauma as the inferred etiology may not have the same certainty as tendonitis. For example, the clinical presence of findings such as tight bands and myofascial trigger points in the muscle associated with a pain complaint is widely regarded as diagnostic evidence for a myofascial pain disorder. While the clinician may be as certain of the diagnosis here as with, say, tendonitis, despite the softer findings compared to tendonitis, examiner

reliability studies indicate that these findings have moderate to poor interexaminer reliability (Gerwin et al., 1997) and the nature of the observed phenomenon is not fully accepted by some authors (Cohen & Quintner, 2008; Quintner et al., 2015). Given, however, the strong role that overuse behaviors have as a stated etiology (Travell & Simons, 1983) as well as the strong role they appear to have empirically in both onset and perpetuation of myofascial pain (Glaros et al., 2016; Ohrbach et al., 2013; Ohrbach et al., 2011), perhaps myofascial pain disorders should be regarded more as a construct comprised of multiple indicators rather than as a simple physical diagnosis. Whether microtrauma is a factor in the formation of the core findings of myofascial pain, taut bands and trigger points, requires more research as well as perhaps more circumspection in the clinic (see Chapter 8 on exploration of the masticatory musculature for discussion of this topic).

Finally, physical damage is sometimes assumed based on presumed microstrain when overuse behaviors are identified, and what might be a behavioral problem with an unknown mechanism for the reported pain (Glaros & Burton 2004; Glaros et al., 2016) is transformed in the clinician's mind to a diagnosed physical disorder with necessary tissue damage as the source of nociception underlying the pain. This inferential pattern of using any sort of abnormal finding to support the belief that physical damage has occurred (and therefore must be the focus of physical intervention) follows that which has been endemic in the TMD field for decades, and it stems from inadequate assessment as well as not considering levels of diagnosis (Ohrbach & Dworkin, 2016). It is worth noting, however, that microtrauma as a source of pain 'described in terms of such damage' remains a critically important possibility for the TMD pain problems that have onset without known cause (Slade et al., 2016).

With these preliminaries, microtrauma has been an often-stated initial cause of TMDs, particularly for disorders affecting the TMJ, but this appears to be largely based on speculation rather than evidence. Some compelling evidence from one lab points to one pathway for microtrauma to emerge and affect the TMJ. Healthy females (compared to males) have higher energy densities within the TMJ disc during normal closing movement of the jaw (Iwasaki et al., 2017b); women with disc displacements (compared to those without displacements) have higher TMJ energy density (Iwasaki et al., 2017a); and women with disc displacement and pain (compared to those without either) exhibit longer periods of muscle contraction and a higher duty factor during an episode of sleep bruxism (Wei et al., 2017). Collectively, these findings would suggest that TMJ disc displacements and regional pain influence joint biomechanics, such that women may be more susceptible to the repeated forces of sleep bruxism, which would appear to fall into the microtrauma range. These findings may have implications for the potential microtrauma effects of other types of overuse behaviors on TMDs.

Psychological and behavioral factors

It is not hyperbole to state that probably every cross-sectional study examining a set of psychological variables in relation to TMDs has found at least one of the target variables to have a significant association with any of the TMDs, and with the painful TMDs dominating this picture. A comprehensive evaluation of nearly all known TMD-relevant psychological variables confirmed prior associations with chronic painful TMDs, with standardized odds ratios (SOR) ranging from 1.3–2.4 (Fillingim et al., 2011); such a cross-sectional design does not provide any insight into etiology, however. The same variables when examined for predicting who would subsequently develop painful TMDs exhibited significant albeit weak associations, with standardized hazard ratios ranging from 1.1–1.4 with TMD onset (Fillingim et al., 2013). In the chronic cohort, the strongest predictors were physical body symptoms, with SOR: 2.4 distinct from all others; yet, none of the predictors of incident TMDs

were notable in comparison to the others. These findings, taken together, highlight that a psychological etiology of painful TMDs exists primarily as a rubric representing general distress or psychosocial dysfunction, whereas once pain begins, the burden of pain serves to substantially aggravate all psychological processes, some more than others, among subjects with painful TMDs. Consistent with the application of the biopsychosocial model to pain conditions and with special reference to orofacial pain broadly, these findings also indicate that comprehensive multiaxial assessment is essential for both clinical and research subjects in order to better understand the many aspects involved in pain processing (Durham et al., 2015; Ohrbach & Durham, 2017).

A frequently regarded behavioral variable for the etiology of TMDs is parafunctional habits, described previously under the microtrauma section as overuse behaviors. A substantial cross-sectional literature attests to the potential importance of these behaviors in association with TMDs. Experimental studies demonstrate that maintaining a behavioral pattern at a sufficient magnitude, duration, and frequency reliably leads to pain symptoms consistent with a myalgia diagnosis (Glaros, 2007; 2008). Ambulatory studies demonstrate that stress reactivity includes parafunctional behaviors, which lead to pain (Glaros et al., 2016; Glaros et al., 2005). In terms of actual prediction of painful TMDs, individuals with oral parafunctional behaviors with scores above 25 (representing 30% of the total possible score), according to the Oral Behaviors Checklist (Kaplan & Ohrbach 2016; Markiewicz et al., 2006; Ohrbach et al., 2008b) are 75% more likely to develop first-onset painful TMDs, compared to individuals with a score below 17. These findings also indicate that only when a sufficient density of behaviors, based on the number of behaviors and their respective frequency, exists do these behaviors reliably matter; this is consistent with the prior discussion regarding microtrauma.

Alterations in the pain processing system

Considerable evidence across multiple pain disorders exists regarding changes in the pain processing system emerging with pain chronicity. These changes can be measured in multiple domains: pressure pain thresholds, thermal pain thresholds and tolerance, thermal windup and after-sensations, cutaneous pinprick threshold, and cutaneous windup and after-sensations (see Chapter 6 of the current text). These changes in each of these domains are measurable in individuals with chronic painful TMDs (Greenspan et al., 2011). In contrast, alterations in pain processing are less notable prior to painful TMD onset, with smaller changes across fewer measurement domains (Greenspan et al., 2013). In particular, for both chronic and incident TMDs, alterations in pain processing measured by the modality of pressure are most notable, but interestingly changes in pressure pain sensitivity fluctuate with painful TMD onset and do not predict onset (Slade et al., 2014).

Pain comorbidity

Distinguishing a pain condition local to one system (e.g., TMD within the masticatory system with an otherwise negative medical history for relevant factors) versus a pain condition existing within a set of general health factors or other pain disorders is critical and necessary based on substantial empirical data. General health factors exhibit strong association with chronic TMDs and also strongly predict new TMD onset (Aggarwal et al., 2010; Ohrbach et al., 2011; Sanders et al., 2013). These factors include other pain disorders, neurosensory disorders, respiratory disorders, and tobacco use for both new onset and chronic TMD (Ohrbach et al., 2011; Sanders et al., 2013). In addition, increasingly poor sleep over time is a substantial predictor of new onset TMDs (Sanders et al., 2016). Among these general health factors, other pain disorders (such as low back pain, irritable bowel syndrome, headache, and genital pain) are perhaps the most investigated and substantiated risk factors for painful TMDs. Early insights into the

compounding effects of two or more co-occurring pain disorders informed the subsequent gate control theory and, later, neuromatrix theory of pain (Livingston, 1998; Melzack, 1989; Melzack & Wall, 1965), with the implication that multiple-pain disorders do not have an additive effect but have a multiplicative effect on the consequences of pain, including the intensity of pain experienced, psychological variables such as mood and catastrophizing, pain-related disability, and risk for subsequent onset of yet another pain disorder (Aggarwal et al., 2006; Creed et al., 2012; Macfarlane et al., 2003; McBeth et al., 2002; Raphael et al., 2000). These findings also suggest that TMD as a local disorder, existing in isolation from other comorbid pain disorders, and TMD as a disorder mixed with other pain disorders would respond differently to condition-specific treatment. And, indeed, strong evidence points to oral appliances, for example, as treatment for sleep bruxism having notably lower efficacy when widespread pain is also present (Raphael & Marbach, 2001).

TMD, as a musculoskeletal pain condition, occurs in the facial region situated between two other major structures, each with its own complex pain disorders: the head and headache, and the cervical spine and neck pain. In addition to the link with other pain disorders via more general CNS mechanisms associated with the pain processing system broadly, TMDs share specific mechanism-overlap with both headache and cervical problems (Ballegaard et al., 2008; Häggman-Henrikson et al., 2016; Häggman-Henrikson et al., 2002; Wiesinger et al., 2013). The mechanism overlap includes peripheral nerve convergence in the trigeminal nucleus caudalis, mechanical activity-based motor control strategies subserving basic functions such as mastication, and gross structure overlap such as the temporal region that is home to the temporalis muscle of jaw function and is the site of a large proportion of headaches.

Conclusion

In summary, musculoskeletal conditions as a group are exemplified by nonspecific back pain, neck pain, and TMD pain, and the conditions have in common two main risk factors, psychological factors and injury, and one probable risk factor of smoking, conclusions nicely summarized elsewhere (McLean et al., 2010; Taylor et al., 2014; Sharma, 2017). Herewith, we take a broader perspective on painful TMD and regard it as only one among other musculoskeletal conditions. Of the two main risk factors, it appears that psychological characteristics have played the most robust role in not only initiating the development of such conditions, but also in the persistence of pain symptoms and the development of chronic pain. This stronger role for psychological characteristics may also be a function of more and better research examining those characteristics in contrast to the more limited research focusing on injury. Smoking is a common risk factor for back pain and TMDs; while it is often regarded as a potential proxy for poorer social or health conditions, the evidence also suggests direct effects on pain. Occupational factors play a major role in back pain, whereas occupational factors have a much weaker or nonexistent role in TMDs and an unclear role at present in neck pain. Similarly, education, socioeconomic status, and physical activity have shown stronger associations with back pain than with neck pain or TMDs.

The role of case definitions and its impact on research has differed not only for TMDs, but across these other two musculoskeletal pain types, and with the same effect. In contrast to the recent extremely well-operationalized and reliable definitions for TMDs, the varying level of validity in the case definitions for back pain and for neck pain may have impeded the respective research programs. It is notable that the well-standardized definitions for TMDs that have now been developed have allowed research into TMD pain to move forward at a relatively rapid pace (Ohrbach & Dworkin, 2016).

Furthermore, taken together the available information suggests that risk factors for musculoskeletal pain conditions may have a more complex dynamic

relationship over time, which also includes reciprocal causation. Musculoskeletal pain conditions are now considered complex diseases, and a collective picture of complex causation emerges. Only recently has one aspect of the complexity become clearer through the OPPERA studies, and that is that one single risk factor alone is not sufficient to cause a pain disorder. While that understanding about musculoskeletal pain has so far been restricted to TMDs, there is no evidence to suggest that TMDs are unique among musculoskeletal pain problems. However, to date, mechanisms underlying the transition from risk factors to pain disorders remain poorly understood. Lung cancer provides a good example of what is missing in the pain literature. For lung cancer, risk factors such as smoking and asbestos are sufficient and readily explainable in biological terms, for which a tissue-level correspondence can be found and which makes for clearer public health implications. Although the characteristics of individual risk factors have been presented based on the existing studies on TMDs, there are major neurobiological processes underlying the multivariable predictors at play in the development of musculoskeletal pain conditions in general, and the nature of the causal processes are complex indicating a higher level interaction across these factors. We close this chapter with the emphasis on the importance of multiple-risk determinants, acting together and over time, in the development of painful TMDs and their persistence into chronicity.

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