Contemporary Management of Temporomandibular Disorders

Fundamentals and Pathway to Diagnosis

S. Thaddeus Connelly
Gianluca Martino Tartaglia
Rebeka G. Silva
Editors

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This book is dedicated to our colleagues, Dr. Rick Katzberg, Dr. Boudewijn Stegenga, and Dr. Patrick Duffy.

Rick was a pioneer in the foundation of imaging of the temporomandibular joint. Even well into his retirement, Dr. Katzberg, an Emeritus Professor in Radiology at UC Davis Medical Center, California, demonstrated an enthusiasm and energy to share his knowledge and continue his work that delighted us all, which included an important contribution to this volume.

Dr. Stegenga was a force in TMJ Pathology and an internationally respected facial and oral pain consultant and surgeon from Groningen in the Netherlands. His chapter encapsulates the knowledge and understanding gained from his brilliant career.

Dr. Patrick Duffy was a fellow oral and maxillofacial surgeon in Northern California and friend to us. A kind and honest man, Patrick was dedicated to his family and his patients, and we regret that we lost him so young.

We miss all three of our friends and solemnly dedicate this text to them.

The Editors

Preface

Have you ever overheard anyone saying, "Wow, I have been really stressed lately and my elbow is really sore!"? Well, of course you haven't, but you may have very well heard someone complaining that their jaw joints are sore when they are in the middle of a very stressful situation. That is because under stress we tend to clench our masticatory muscles and grit our teeth together, an experience almost everyone has had at one point or another in their lives. But this scenario also underlines the uniqueness of this anatomic structure, the temporomandibular joint and indeed the entire stomatognathic apparatus in general. For this anatomy is crucial to so many things we do as humans, including some basic functions such as breathing, eating, speaking, and kissing. However, these structures are also intimately tied into our emotional states; we smile when happy, frown when sad, and yes grit our teeth together when under stress. Because there are so many critical structures and functions in the head and neck region, there is a greater proportion of neuroanatomy allocated for sensory and control, which contributes to an overall greater awareness of our face and associated structures, compared to the rest of our bodies. Thus, when something is abnormal, then not only are we acutely aware of the pain, but it may have a direct effect on our emotional state.

The first volume of this work sets the stage for our current understanding with a rare detailed examination of comparative anatomy of the temporomandibular joint, followed by a concise description of the human anatomy and then a cutting-edge presentation of the neuroanatomy associated with chronic pain. The next chapters include information on how patient data is collected in the clinic through the clinical exam and radiological studies. This data is then analyzed and processed to form a diagnosis and the standardized Diagnostic Criteria of Temporomandibular Disorders is covered and made clinically relevant. This then directly leads into the next volume of the work. It is important to note that this first volume begins with a discussion of how healthcare providers can work best as a team taking the whole patient into consideration; this is a point that the author of the chapter and editors would like the reader to carry on throughout the whole book and be able to incorporate into clinical practice.

San Francisco, CA — S. Thaddeus Connelly

Acknowledgements

The Editor-in-Chief would like to acknowledge first and foremost his family: Pathima, his wife and his two sons Sebastien and Sidney, you all are my world and reason for being.

Additionally, I would like to thank my Co-Editors Rebeka and Gianluca; without their assistance, this book would not have come to full fruition. To Rebeka, in many ways I owe you my professional career, you were the person to call attention to my potential when I first arrived in San Francisco. And, Gianluca, who has become a true lifelong friend, consider me a loyal fellow traveler to the end.

Then my family: Steve, Nancy, Corey, Heather, Adam, Mark, Kelley, Liz, Hannah, Brodey, Ava, and the new boy soon to be. My wife's family: Bernadette, Anthony, Magada, Anne, Chris, Rohsan, Gary, Michael, Gabby, Rachel, Leah, David, JoAnn, Kenny, Matthew, and Nathan.

To all of my past teachers and fellow students and residents, I hope this book is worthy of your many contributions that you have given me.

Life is precious and short and you never know when you are going to be called to go home. I would like to remember and celebrate the lives of three friends, who succumbed to the same hideous disease, cancer. First, Patrick Duffy, he was a good friend, a fellow resident, and a fellow surgeon with a heart of gold. Patrick's life will be carried forward by his three incredible sons and wife Sharice. Then, Rick Katzberg, the author of Chap. 10. He was a giant in his field; his work in this book is testament to that. We only ever met over conversations on the phone and email, but we shared similar visions and I was greatly looking forward to working with him on shared projects. His life will be carried forward by his family and his wife Nancy. Boudewijn was a brilliant colleague who made invaluable contributions to the field of TMJ Pathology. He will be dearly missed by all who knew him.

In honor of Patrick, Rick, and Boudewijn, I am establishing a foundation to support basic and clinical research to defeat cancer. We are at the point in existence where real impact is possible; all we need to do is to put our nose to the grindstone and make it reality. The foundation will be called The Northern California Cancer Initiative in honor of our dear friends who have been called home.

S. Thaddeus Connelly DDS, MD, PhD, FACS

Many years ago during my training in New York, two of my teachers, Dr. Howard Israel and Dr. Arthur Elias, directly inspired me to become interested in temporomandibular joint disorders. In joining the faculty of the San Francisco VA Health Care System near the beginning of my career, I soon became aware that I needed to launch myself into the field as my TMD patients, all US military veterans, depended on me for help. I set out to make the surgical and nonsurgical treatment of TMJ disorders my subspecialty. Therefore, it is with gratitude that I acknowledge my many mentors.

I wish to express my immense gratitude to my family. To my husband, thank you for your compassion and patience. To my daughter, my eternal gratitude for your loving understanding that my career means being of service to others and often entails long hours. To my parents, I am honored that I was raised to value education and hard work. You came from humble emigrant beginnings, and I am astounded by what you accomplished in your lives. My residents deserve mention because your eagerness to master our specialty is incredibly stimulating. To my wonderful colleagues, Drs. Connelly and Tartaglia, I am so proud of you and wish to let you know that your friendship touches me deeply. I know we will continue to work closely in the future and support one another's ideas and projects. Lastly to my TMD patients, my respectful and gentle gratitude goes to you all.

Rebeka G. Silva DMD MS

First, I would like to thank my teacher V.F. Ferrario. He was gracious enough to teach me about research, the methods that are applied in the endeavor of what all of us call "science." In particular, he stressed to me that research should include enthusiasm, strict observance of protocol, intense curiosity, and an overall goal of achieving progress.

There is an equally important person that has made an invaluable contribution to my education; this person has helped to bring me to where I am now. On a daily basis she was there to educate and support my professional and academic activities. Thus, I wish to extend a special thanks to Prof. Chiarella Sforza, an inseparable colleague and friend without whom none of this would have been possible.

For a wonderful dream, I thank my beautiful family…

Additionally, I would like to thank the Editor-in-Chief, Thad, for his competence, enthusiasm, and friendship. He is a really good example for young doctors. Last but not least, I would like to thank my Co-Editor for her medical and surgical skills and patience.

Gianluca Martino Tartaglia, DDS, PhD

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Philosophy, Ethics, Law and Anatomy

Temporomandibular Disorders: Comprehensive Management

1

James Fricton

Abstract

This chapter and indeed the whole of this book summarize a broader more inclusive philosophy in diagnosis and managing TMD that reflects both new conceptual models in understanding chronic illnesses and systematic reviews of therapeutic strategies for successful management of TMD.

1.1 Introduction

The face and associated cranial, oral, and dental structures are among the most complicated areas in the body contributing to an array of orofacial disorders including temporomandibular disorders, orofacial pain disorders, orofacial sleep disorders, oral lesions, dental disorders, and oromotor disorders. Orofacial pain disorders are the most common of these problems and can cause symptoms of orofacial pain, jaw dysfunction, and chronic head and neck pain with a collective estimated prevalence of at least 20% of the general population (Table 1.1) [1–7]. To complicate matters, oral and craniofacial structures have close associations with functions of eating, communication, sight, and hearing as well as form the basis for appearance, self-esteem, and personal expression and, thus, can deeply affect an individual's psychological and functional status [7]. A national poll found more adults working full time miss work from head and face pain than any other site of pain [5].

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Table 1.1 Common orofacial disorders that require special diagnostic and treatment needs with estimated prevalence [1–6]

Orofacial pain disorders	Estimated prevalence (%)
Temporomandibular disorders	5–7
Orofacial pain disorders (burning mouth, neuropathic, atypical pain, neurovascular)	2–3
Headache disorder (tension-type headaches, migraine, mixed, cluster)	20
Orofacial sleep disorders (e.g. sleep apnea, snoring)	3–4
Neurosensory and chemosensory disorders (e.g. taste, paresthesias, numbness)	0.1
Oromotor disorders (e.g. occusal dysethesias, dystonias, dyskinesias, bruxism)	4
Total estimated prevalence in general population	30–40

The high prevalence, personal impact, and poor access to care for these problems have led to an expanded role for dentistry in providing care for them. However, because dentists have most of their patient care focused on treatment of the dentition and related structures, it can be a challenge to understand the broader scope of diagnosis and management of these conditions. Treatment of temporomandibular disorders (TMD), like many pain conditions, is often singular and tends to vary according to the clinicians' favorite theory of etiology. Clinicians tend to see what they treat and treat what they see. Clinicians seeing a stress etiology treat with stress management, surgeons seeing a joint pathology treat with surgery, and dentists seeing a dental etiology treat the teeth. As a result, success of treatment is often compromised by limited approaches that only address part of the problem.

This chapter summarizes a broader more inclusive philosophy in diagnosis and managing TMD that reflects both new conceptual models in understanding chronic illnesses and systematic reviews of therapeutic strategies for successful management of TMD.

1.2 Human Systems Theory: A Comprehensive Model for Understanding Chronic Illness

Humans are complex, multidimensional, and dynamic and live within an ever-changing physical and social environment. Yet, our traditional biomedical model is based on a scientific paradigm that is unidimensional, reductionistic, and inflexible because it is based primarily on understanding the underlying pathophysiology. While distinct pathophysiological mechanisms occur in all chronic conditions, understanding the multitude of factors that play a role in the onset, perpetuation, and progression of the illness is the key to successful management [8]. Thus, traditional scientific protocols often fall short in providing an adequate framework for explaining, predicting, and influencing chronic illness and its outcomes. Scientific and

clinical communities have been searching for a more flexible, holistic, and integrated model that describes the changes that can occur in human biology in response to the circumstances in our lives that contributes to the balance between health and illness.

Human systems theory (*HST*) provides this framework [8]. As originally stated by Aristotle, the Greek philosopher in 300 BC, "The *whole is greater than the sum of its parts*." HST stems from research in general system theory and originated in ecology out of the need to explain the interrelatedness of organisms in ecosystems [8–10]. While conventional biological theories view the subject as a single entity, HST views a person as a whole with an interrelationship between the subparts of their life. These subparts are not "static" but rather are dynamic, evolving, and interrelated processes. The practical application of HST to patient care requires that we understand basic HST principles as they apply to understanding the development and alleviation of illness. These include:

- Seeing the whole patient through the eyes of the *Biopsychosocial Medical Model* [8–10]
- Understanding recursive feedback cycles using *cybernetics* [11, 12]
- Seeing the broad cumulative impact of small changes using *chaos theory* [13–15]
- Understanding the power of positive action through *positive psychology and behavioral medicine* to enhance health as part of treatment of illness [16–19]

These concepts provide a new model for understanding TMD and its management that is well founded in theory and science. It is beyond the scope of this chapter to present an in-depth discussion of each concept. However, for those interested in reading further, the concepts are presented in a more creative format—a murder mystery novel—than traditional academic texts as well as part of the University of Minnesota MOOC (massive open online course) at http://www.coursera.org/course/chronicpain [20].

1.2.1 The Biopsychosocial Medical Model

The Biopsychosocial Medical Model was first proposed by Engel in 1977 suggesting that to understand health and illness, we need to look at the whole person and not simply its physical pathophysiology [8–10]. It suggests that we "see the big picture" of illness. Most studies of risk factors and protective factors suggest that each person has a unique set of interrelated factors that can either perpetuate or protect someone from an illness including TMD. These contributing factors correspond to each realm of our lives including the mind, body, emotions, spiritual, lifestyle, social relationships, and physical environment (Fig. 1.1). By improving them, the strategies for management have greater success than the sum of any singular treatments directed at one realm.

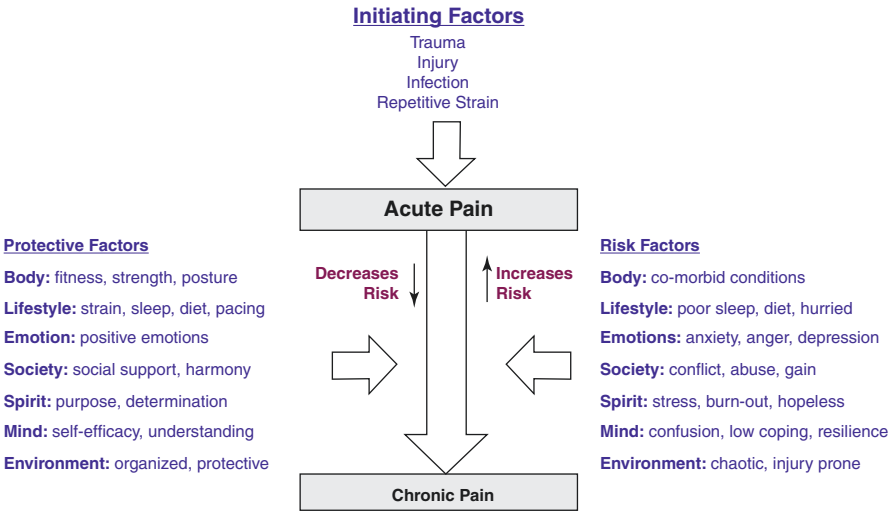

Fig. 1.1 Multiple protective and risk factors play a role on the progression from acute to chronic TMD pain

1.2.2 Cybernetics

Cybernetics, a concept defined in physics, was first applied to human systems by Bateson (1978) [11, 12]. It suggests that "what goes around comes around" and each element of a system generates a change, which causes feedback to the entire system. Positive feedback triggers a continuation of the cycle, while negative feedback leads to a discontinuation of the cycle. This is often referred to as self-reflexive or "circular causation" relationship. Positive and negative feedback cycles play an important role in sustaining a person's illness over time (Fig. 1.2). Patients with an illness often fall into the recursive cycles that perpetuate the illness. Contributing factors to an illness, such as repetitive strain, depression, or poor sleep, are elements that sustain the cycle.

There are several types of change that can influence these cycles (Fig. 1.3). The first-order change is based on "reinforcement" of existing elements that promotes maintenance or escalation of the existing cycle and its related illness. The second-order change involves a "revelation" that makes a significant change from within the system through multimodal education, training, and treatment that lead to a new state. This change may either be toward improved health or escalation of the illness, depending on the direction of change in the element. Finally, the third-order change is based on "enlightenment" which produces a change from outside to achieve a new level of existence distinctly different than the original structure. The basis for significant improvement of a condition to create a new paradigm for health of the individual is through either the second- or third-order change.

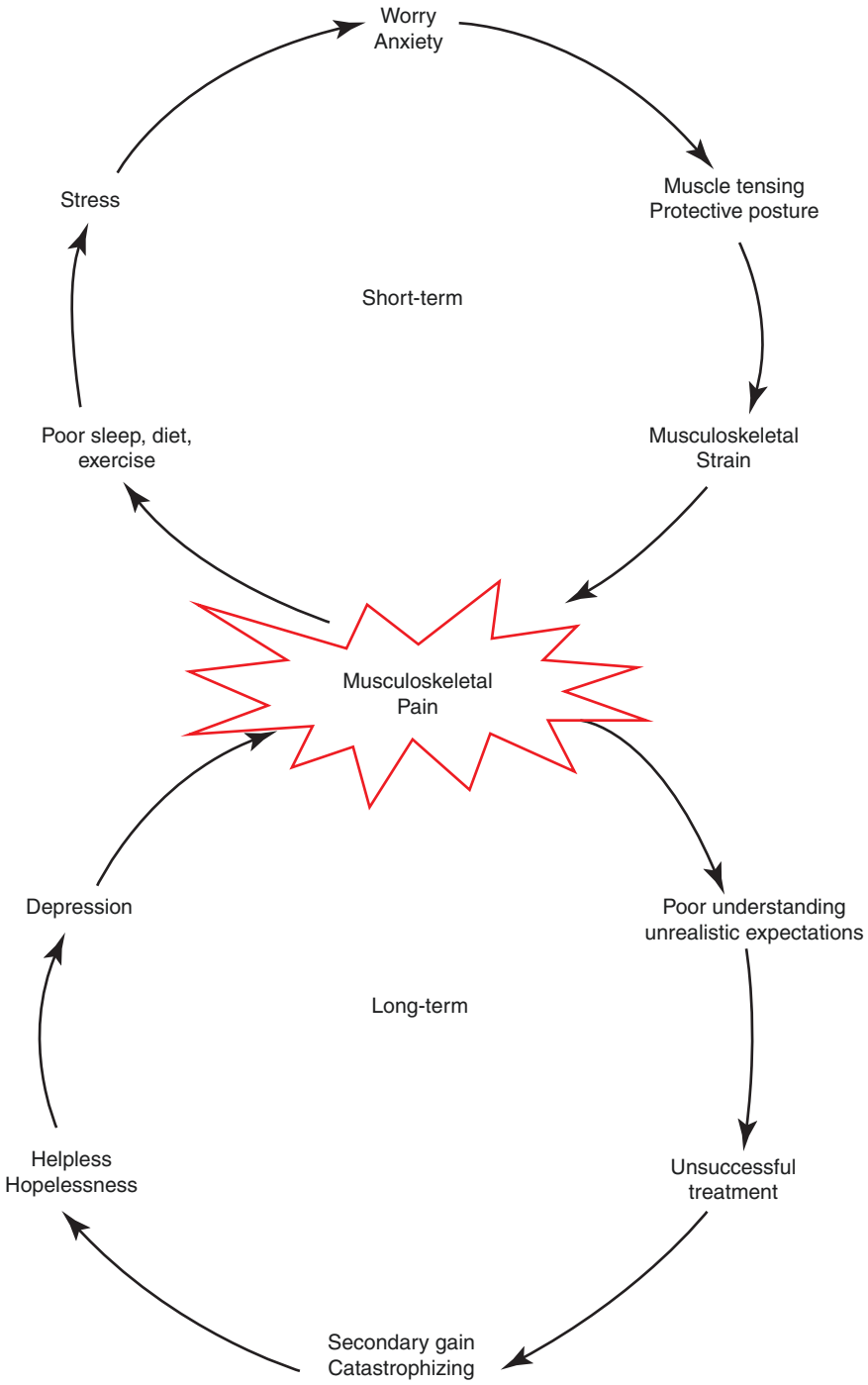

Fig. 1.2 Positive and negative feedback cycles play an important role in sustaining a person's illness over time

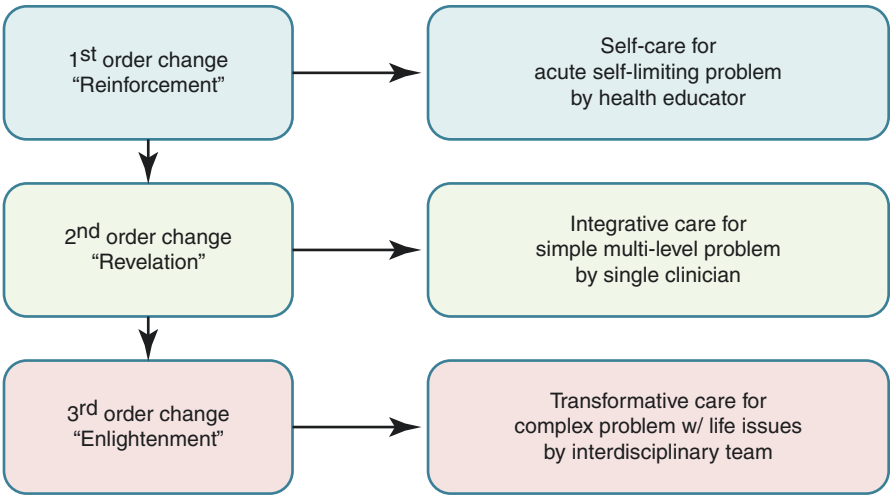

Fig. 1.3 There are three levels of change that matches three levels of care for increasing complexity of the patient

Small first-order compensatory changes made by a patient in response to TMD pain such as reducing use of the jaw, taking an analgesic, or other self-care can improve the illness if it is an acute self-limiting problem, at least in the short term. However, it may also allow a more complex illness to fall into a long-term chronic cycle (Fig. 1.2). If a clinician can help a patient make higher-order changes by understanding the multiple elements in the cycle, and changing those keystone factors that perpetuate it, the illness may change more readily. Integrative care strategies that encourage second-order change within an existing cycle include splints, physical therapy, and behavioral management of oral habits, sleep, and muscle tension. This strategy works quite well for simple to moderate cases but may need a more robust intervention for more complex patients. In this case, transformative care strategies encourage third-order changes that can lead to the most dramatic long-term change. The third-order change involves not only treatment of the TMD pain as noted already but also working with a team to identify all comorbid conditions and contributing factors and help the patient make major changes that may be perpetuating the long-term cycles, such as managing a comorbid medical condition such as fibromyalgia, addressing stressful or abusive relationships, or changing poor work situations. In this way, healthier positive feedback cycles are set up that do not perpetuate the factors that drive the illness.

1.2.3 Chaos Theory

Chaos theory was first popularized by Lorenz (1963) when he presented a paper on the theories of understanding diverse weather patterns entitled "Does the flap of a butterfly's wings in Brazil set off a tornado in Texas?" He presented evidence that found small

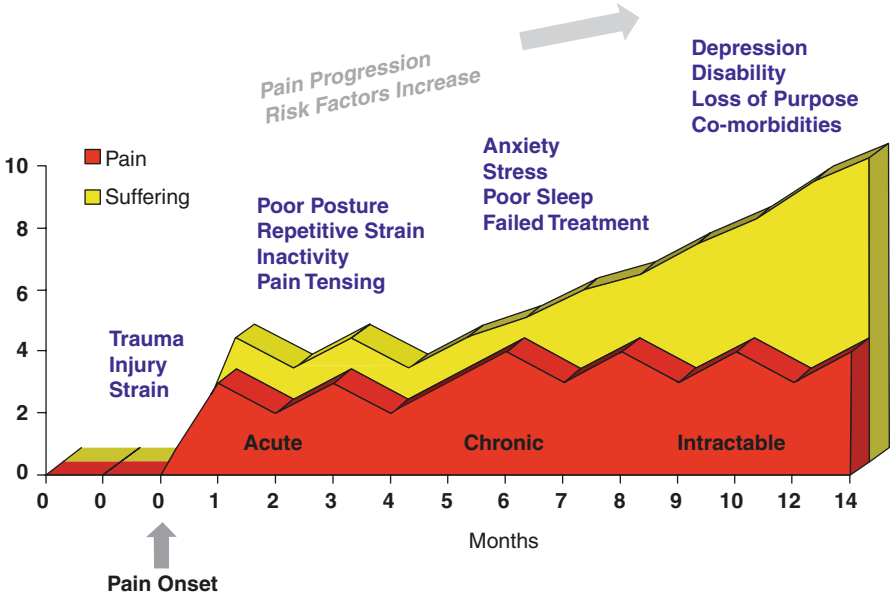

Fig. 1.4 Multiple contributing factors can each play a small role at the early stages of a chronic illness, but when combined they will accelerate the condition dramatically

differences in initial conditions of a system may often yield widely diverging outcomes within dynamic systems. Chaos theory suggests that "It's the little things that matter the most." When applied to health and disease, it suggests that the influence of multiple risk factors can each play a small role at early stages of a chronic illness. However, when these factors are combined, they will accelerate the condition dramatically.

As Fig. 1.4 illustrates, an illness begins with initiating factors such as acute physical injury of the muscles and joints. In most cases, this pain is transient and resolves without complication or persistence. However, if a sufficient number of contributing factors are present, even if small, it can shift the balance from healing of acute pain to delayed recovery and chronic pain (Fig. 1.2) [21–27]. Various underlying neural mechanisms such as peripheral and central sensitization and windup play a role in this process that is difficult to predict. Likewise, the presence of protective factors and early intervention on multiple factors will have the greatest impact in resolving the condition.

1.2.4 Behavioral Medicine

Behavioral Medicine, then, suggests that specific behavioral interventions such as exercise and oral habit reversal can help restore health and wellness and complements theories on *positive psychology* that focuses building health, strength, and positive virtues as much as on correcting illness, problems, and vices [16, 17]. The Aristotle idea that "We are what we repeatedly *do*" is a theory that is supported with much research in achieving health and wellness.

These theories explain the diverse results of placebo-controlled clinical trials for TMD pain and other pain conditions that suggest that many different interventions, from splints and medications to physical and cognitive-behavioral therapies, and even injections and surgery can each be used to successfully improve TMD pain [28–46]. The effect of each of these interventions above the placebo effect may be small, but they are all significant. Furthermore, by integrating these concepts together in a multimodal integrative model of care that is based on a human systems approach to care, the small effects of multiple interventions employed at the same time can result in the greatest positive outcomes. Thus, the evaluation and management proposed in this chapter follow these principles.

1.3 Human Systems Theory Principles of Evaluation

The principles of HST can be applied to evaluating patients with TMD by employing an inclusive problem list, determining the complexity of the patient, and following the decision tree for increasing the potential for successful management.

1.3.1 Determine the Problem List

HST expands the traditional "problem list" to include both the physical diagnoses and the list of contributing factors in each realm. The physical diagnosis is the physical problem that is responsible for the chief complaint and associated symptoms. The orofacial pain disorders noted in Table 1.1 are included in this definition of scope of dental practice because they have characteristics that involve the oral cavity, maxillofacial area, and/or the adjacent and associated structures. Whereas, contributing factors include those factors that initiate, perpetuate, or result from the disorder but in some way complicate the problem. These risk and protective factors are diverse and involve the seven realms of our lives [21–27, 47–63]. This includes the physical (e.g., physiologic, genetic, molecular), lifestyle (repetitive strain, posture, lifestyle, eating, sleep), emotional (depression, fear, anxiety, anger), social (relationships, abuse, secondary gain), cognitive (attitudes, understanding, honesty), spiritual (faith, beliefs, purpose), and the environmental (accidents, pollution, disorganization, hygiene). Specific risk factors for chronic pain may include peripheral factors such as repetitive strain, oral, and postural habits or central mediating factors such as anxiety and depression and comorbid conditions such as fibromyalgia, somatization, and catastrophizing. Protective factors reduce vulnerability to chronic pain. These factors such as the level of coping, self-efficacy, patient beliefs (e.g., perceived control over pain, belief that pain is a sign of damage), and social support can also affect outcomes.

1.3.2 Determining Complexity

The level of care for patients can also vary considerably from simple to complex patients. Patients with complex temporomandibular disorders often present with a

frustrating medical and dental situation, which may include persistent aggravation of pain, multiple clinicians, long-term medications, repeated healthcare visits, and an ongoing dependency on the healthcare system. Thus, successful management is enhanced if the level of complexity is determined and matched to the complexity of the treatment strategy. Singular treatment strategies such as self-care, physical therapy, or splints can be quite successful with simple cases with few contributing factors but often fail in complex patients due to the chronic nature of the disease, central sensitization, and long-standing maladaptive behaviors, attitudes, and lifestyles.

1.3.3 Decision Tree for Triaging Patients

Figure 1.5 outlines the decision tree for sequencing evaluation and management for simple and complex cases. Matching the complexity of a patient with the complexity of the management strategy is the key to success. Once you developed the complete problem list including contributing factors, it can be used as criteria to distinguish simple and complex patients. Complexity of the patient increases with factors such as:

- Presence of multiple comorbid condition
- Persistent pain longer than 6 months in duration
- Significant emotional problems (depression, anxiety)
- Frequent use of healthcare services or medication
- Daily oral parafunctional habits
- Significant lifestyle disturbances

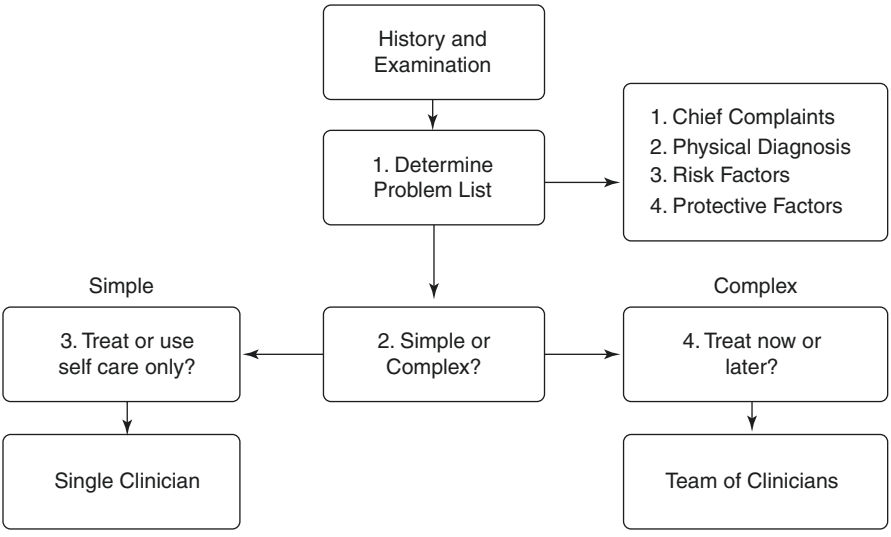

Fig. 1.5 A decision tree for triaging patients and enhancing success

In addition, there are some complex patients that warrant deferral of treatment until more complex problems are addressed. The criteria for not treating until these problems are resolved include factors such as:

- Primary chemical dependency.
- Primary psychiatric disorder.
- Significant litigation.
- Patient overwhelmed with other concerns.
- Patient is not motivated.

Once complexity is determined, the appropriate level of care that matches the complexity of the patient needs to be implemented (Fig. 1.3). For example, acute self-limiting conditions can be managed with a *self-care strategy* by training the patient through a health educator patients with multilevel problems require a second-order change that uses multimodal treatments as implemented by a single clinician. This *integrative care strategy* can include multiple treatments such as splint, exercises, oral habit instruction, medication, and palliative self-care to achieve second-order change with improvement over 2–4 months.

1.3.4 Use of a Healthcare Team

Complex patients with major life issues require a third-order change as implemented by an interdisciplinary team to achieve success. This *transformative care strategy* involves the healthcare team of clinicians such as a dentist, physician, health psychologist, and physical therapist to work together with the patient on different aspects of the problem to achieve success [64–69]. Different aspects of the problem can be addressed by different specialists in order to enhance the overall potential for success. Teams can be interdisciplinary (one setting) or multidisciplinary (multiple settings). The use of a team helps understand and manage the whole patient, allows you to work on multiple aspects of the problem simultaneously, improves patient compliance and outcome, saves time, and is more economical and more enjoyable as you work together.

Treatments may include cognitive-behavioral therapy, counseling, mindfulness meditation, physical medicine treatments, medications, splint, exercises with physical therapy, occlusal therapy, and surgery to change every aspect of the problem. A consistent philosophy and message to the patient are needed including the importance of self-care, self-responsibility, and education using concepts of HST. Success is dependent upon communication, integration among clinicians, and proper patient selection. With complex patients, improvement but rarely resolution is typically achieved in 6 months.

Interestingly, the economics of this model are quite favorable for each of the stakeholders, including the patient, the healthcare provider, and the health plan. The patients receive more comprehensive effective care that is convenient if interdisciplinary in one setting. This not only has a higher potential to achieve success but

also reduces the need for doctor shopping and single sequential trial and error treatments. Thus, the health plan reduces the long-term costs compared to a patient who continues to fail in treatment and bounces from one doctor and intervention to another. Finally, the clinicians within a team practice benefit economically because more clinicians are providing care and generating more income to cover the overhead of the practice. It's a rare win-win-win scenario.

1.4 Principles of Management

Successful management of TMD is focused on treating the diagnosis and reducing the contributing factors to achieve the goals to:

- Reduce or eliminate pain.
- Restore normal jaw function.
- Restore normal lifestyle functioning.
- Reduce the need for future healthcare.

Once complexity is determined, the management options for TMD in general are consistent with treatment of musculoskeletal disorders in other parts of the body. The treatments involve interventions that have been documented with randomized controlled trials and are within the scope of dental practice to deliver or recommend [28–46]. These include both reversible and irreversible treatments. Reversible treatments designed to encourage healing in the muscle and joints include self-care, behavioral therapy, splints, medications, and physical medicine treatment. Irreversible treatments include joint surgery and permanent occlusal treatments within an integrated interdisciplinary team [47–60]. To determine if the problem is self-limiting, self-care should be initiated first. If the problem does not resolve within a few weeks and there is some evidence of progression and/or persistence, treatment can proceed if pain and/or locking is severe enough to affect functioning or quality of life and the patient desires treatment. Each is discussed briefly.

1.4.1 Reversible Treatments

1.4.1.1 Self-Care

A key determinant of success in management of any musculoskeletal disorder is focused on educating the patient about the disorder and the patient's compliance with the self-care aspects of management, including exercises, habit change, and proper use of the jaw (Table 1.2) [37, 38]. Self-care should be provided to all patients, and in some cases, it is the only strategy needed.

1.4.1.2 Behavioral Therapy

Approaches to change maladaptive habits and behaviors need to be addressed and presented as an integral part of the overall treatment program for all patients with

Table 1.2 Characteristics and efficacy of self-management for temporomandibular disorders [37–41]

Intervention	Scientific basis	Implementation
Exercises	Improve range of motion and strength will improve pain	For stretching, gently and gradually increase range of motion by placing two fingers between your front teeth for a count to 10: Rest and repeat six times. When two fingers are comfortable, then increase to two knuckles, then three fingers to full range of motion *To strengthen*, push the jaw to each side without moving it. Repeat six times
Oral habit reversal	Avoid muscle tensing habits that put strain on the muscle and joints	Never touch the upper and lower teeth together except during eating. Use reminders such as stickers or timers. If jaw pain or oral habits are noticed, replace negative habits with positive habits. TATU is Teeth apart, Tongue up in "n" tongue position. Let the jaw relax with lips closed and breathe through nose
Healthy diet habits	Improving diet and sleep will encourage healing	Eat a soft diet to reduce strain. Avoid sugar and simple carbohydrates that cause inflammation. Avoid caffeine that cause headaches and sleep disorders. Avoid tough chewy foods. Cut foods into small bites. Do not chew gum
Healthy sleep habits	Systematic reviews of show efficacy of Improve sleep will encourage healing	Set up a sound sleeping environment with a cool, quiet, and dark sleeping room. Use the bed only to sleep in. Avoid caffeine in coffee, tea, and soft drinks. Get a comfortable semi-firm mattress and squishable pillow. Reduce stimulating activities before bed including computer work, video, TV dramas, and exercising. Sleep on side or back with pillow between or under knees. Accept interruptions as normal and go back to sleep
Daily pauses	Systematic reviews of show efficacy of mindfulness-based stress reduction	Take a few second pauses throughout the day to check in daily on body, lifestyle, thoughts, emotions, purpose, social harmony, and environment without negative judgment
Calming practice	Systematic reviews of relaxation, and guided imagery for pain	Practice deep breathing with relaxation training to relax body and mind and gain insight, understanding, motivation, and compliance

temporomandibular disorders and oral habits [39, 40]. Behavior modification strategies are the most common techniques used to change habits. Although many simple habits will change by making the patient aware of them, changing persistent habits requires a structured program that is facilitated by a clinician trained in behavioral

strategies. Habit change using a habit reversal technique can be accomplished by (1) becoming more aware of the habit, (2) knowing how to correct it (i.e., what to do with the teeth and tongue), and (3) knowing why to correct it.

When this knowledge is combined with a commitment to conscientious monitoring, most habits will change. Progress with changing habits should be addressed at all appointments with the patient. In some cases, patients may have significant psychosocial problems that accompany a temporomandibular disorder and may benefit from counseling or medication with a mental health professional. A decision needs to be made prior to initiating treatment regarding whether the psychological distress is the primary problem. If this is the case, treatment of the psychological problem is best accomplished first and as a problem separate and apart from the TMD.

1.4.1.3 Intraoral Splints

Splint therapy can be effective alone or in combination with other treatments for each stage of TMJ internal derangements and myofascial pain [29]. Although there are many useful types of splints, four types are commonly used for TMD: the full-arch stabilization splint, the anterior repositioning splint, the anterior bite plane, and the posterior bilateral partial coverage splint. Complications that can occur with the use of any splint include caries, gingival inflammation, mouth odors, speech difficulties, and/or psychological dependence upon the splint. The most serious complication is major irreversible changes in the occlusal scheme (open bites), which unintentionally occur as a result of long-term use of partial coverage splints such as the anterior bite plane and the posterior coverage splint. Splints should not be designed to move teeth orthodontically during treatment of a temporomandibular disorder.

1.4.1.4 Pharmacotherapy

The most commonly used medications for pain are classified as nonnarcotic analgesics (nonsteroidal steroidal anti-inflammatories), narcotic analgesics, muscle relaxants, tranquilizers (ataractics), sedatives, and antidepressants [44–46]. Analgesics are used to allay pain; as muscle relaxants for muscle tension and nocturnal activity; as tranquilizers for anxiety, fear, and enhancing sleep; and as antidepressants for pain, depression, and enhancing sleep.

Opioid analgesics have their own problems due the potential for abuse and should be used sparingly only with patients who have intractable chronic pain, have no psychiatric conditions, and have no history of chemical abuse. If prescribed, clinicians need to follow specific opioid prescribing standards such as use of pain contracts, urine toxicology testing, suspension of medications with violation, and other guidelines found at http://www.fsmb.org/pdf/2004_grpol_Controlled_Substances.pdf.

Despite the advantages of medications for pain disorders, there exists an opportunity for problems to occur due to their misuse. For this reason, an important goal of treatment for most patients is to eliminate the need for medications long term. With chronic pain patients, termination of current medications should take precedence over prescribing additional ones. The problems that can occur from the use of medications include chemical dependency, behavioral reinforcement of continuing pain, inhibition of endogenous pain relief mechanisms, side effects, and adverse effects from the use of poly-pharmaceuticals.

1.4.1.5 Physical Medicine

The use of physical medicine techniques follows the same orthopedic and physical therapy guidelines as the evaluation and treatment any musculoskeletal condition [30]. Many exercises and modalities are available to help reduce pain and tenderness and increase range of motion. Exercises are recommended to stretch, strengthen, and relax muscles, to increase joint range of motion, to increase muscle strength, or to develop normal arthrokinematics. They are prescribed in order to achieve specific goals and are changed or modified as the patient progresses. Once the patient has reached the goals of the treatment, a maintenance level of exercise is recommended to assure long-term resolution of the patient's problems. In some cases of structural joint problem limited range of motion and inflammation, ultrasound, iontophoresis, phonophoresis, superficial heat, cryotherapy (cold), and massage have been documented to be helpful for musculoskeletal disorders. Electrotherapies such as electrogalvanic stimulation and transcutaneous electrical stimulation have also been shown to be useful. Muscle and joint injections may also be recommended. However, modalities typically have short-term effects and need to be used with exercises to maintain improvement. For this reason, modalities should be used short term and continued only until there is no longer a change in objective signs and/or improvement in pain.

1.4.2 Irreversible Treatments

In most cases, TMD problems can improve with self-care in combination with reversible treatments that encourage the natural healing processes of the muscles and joints. The use of irreversible treatments has risk and should be used only if specific criteria are met. Both TMJ surgery and permanent dental stabilization are discussed.

1.4.2.1 Surgery

TMJ surgery has also become an effective treatment for structural TMJ disorders [41–43]. However, the complexity of available techniques, the potential for complications, the frequency of behavioral and psychosocial contributing factors, and the availability of nonsurgical approaches make TMJ surgery an approach that should be used in selected cases that meet specific criteria. The decision to treat the patient surgically is dependent upon the degree of pathology present within the joint, the success or failure of appropriate nonsurgical therapy, and the extent of disability that the joint pathology creates for the individual patient. Discussions of individual techniques are found in subsequent chapters. Briefly, surgical management may vary from the closed surgical procedure (arthroscopy) to an open surgical procedure (arthrotomy) depending on the degree of disk deformity and degenerative changes. Each of the following criteria adapted, from the AAOMS criteria, should be taken into consideration before proceeding with TMJ surgery:

1. Documented TMJ internal derangement or other structural joint disorder with appropriate imaging
2. Evidence that suggests symptoms and objective findings are a result of disk derangement or other structural joint disorder
3. Pain and/or dysfunction of such magnitude as to constitute a disability to the patient
4. Prior unsuccessful treatment with a nonsurgical approach that includes a stabilization splint, physical therapy, and behavior therapy
5. Prior management of bruxism, oral parafunctional habits, and other medical or dental conditions or contributing factors that will affect the outcome of surgery
6. Patient consent after a discussion of potential complications, goals to achieve, success rate, timing, postoperative management, and alternative approaches including no treatment

These conditions maximize the potential for a successful outcome but cannot guarantee it. Patients with factors such as fibromyalgia, depression, or resistant nocturnal bruxism present with a complexity that has a poor prognosis. In addition, a full knowledge of complications and the reasons for surgical failure can help clinicians make this decision. Once this information is available, a realistic discussion of the prognosis, the patient's expectations, and any complicating factors can help a patient make a correct decision about surgery.

1.4.2.2 Permanent Dental Stabilization

Permanent dental treatment may be needed in some patients to provide stable occlusal support and function for the dental and temporomandibular structures [47]. These treatments include occlusal adjustment, restorative dentistry, fixed or removable prosthodontics, and orthodontics with or without orthognathic surgery. If needed due to poor stability of the dentition, it is recommended to be completed only after pain is reduced and normal jaw function is restored [47]. The criteria for using secondary dental treatment to maintain comfort and function of the temporomandibular structures include:

1. The function and stability of the occlusion do not provide adequate orthopedic support. This may be due to missing teeth, skeletal malocclusion, or gross interferences in dental function.
2. The lack of stable dental support is demonstrated to be directly related to aggravation or recurrence of the temporomandibular disorder after primary treatment of the disorder is successfully completed.

Permanent dental treatment should proceed with the most conservative approach that will provide adequate function and stability of the occlusion. This ranges from occlusal adjustments to restorative dentistry for changing the teeth to improve the dental occlusion and orthodontics to orthognathic surgery for changing the position of the teeth and skeletal relationships.

1.5 Conclusion

Temporomandibular disorders are common problems that can cause orofacial pain, jaw dysfunction, and chronic head and neck pain with a collective estimated prevalence of at least 20% of the general population (Table 1.1) [1–7]. Because oral and craniofacial structures have close associations with functions of eating, communication, sight, and hearing as well as form the basis for appearance, self-esteem, and personal expression, they can deeply affect an individual's psychological, behavioral, and functional status [8]. Thus, understanding TMD with a conceptual model that reflects an comprehensive and integrated problem list that is inclusive, flexible, and comprehensive can better prepare clinicians to manage the full diversity of patients seen from self-limiting to simple to complex. A human systems approach and its related concepts can provide this.

References

1. Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. J Am Dent Assoc. 1993;124(10):115–21.
2. Petti S. Pooled estimate of world leukoplakia prevalence: a systematic review. Oral Oncol. 2003;39:770–80.
3. Lozada-Nur F, Miranda C. Oral lichen planus: pathogenesis and epidemiology. Semin Cutan Med Surg. 1997;16:290–5.
4. Bailey D, Attanasio R, editors. Sleep disorders: dentistry's role. Dent Clin North Am. 2001;45(4):619–30.
5. Taylor H, Curran NM. The nuprin pain report. New York: Louis Harris and Associates; 1985.
6. Starch R. General population survey of chronic pain. Published by Robert Starch Worldwide; 1999.
7. de Leeuw R, editor. Orofacial pain—guidelines for assessment, diagnosis and management. 5th ed. Berlin: Quintessence Publishing; 2013.
8. Engel GL. The need for a new medical model: a challenge for biomedicine. Science, New Series. 1977;196(4286):129–36.
9. Suls J, Rothman A. Evolution of the biopsychosocial model: prospects and challenges for health psychology. Health Psychol. 2004;23(2):119–25.
10. Borrell-Carrió F, Suchman AL, Epstein RM. The biopsychosocial model 25 years later: principles, practice, and scientific inquiry. Ann Fam Med. 2004;2(6):576–82.
11. Bateson G. (2000 reprint. First published 1972). Steps to an ecology of mind: collected essays in anthropology, psychiatry, evolution, and epistemology. Chicago: University of Chicago Press. ISBN 0-226-03905-6. Retrieved 19 March 2013.
12. Bateson G. Mind and nature: a necessary unity (Advances in systems theory, complexity, and the human sciences). New York: Hampton Press; 1979. ISBN 1-57273-434-5.
13. Lorenz EN. Deterministic nonperiodic flow. J Atmos Sci. 1963;20(2):130–41.
14. Kellert SH. In the wake of chaos: unpredictable order in dynamical systems. Chicago: University of Chicago Press; 1993. p. 32. ISBN 0-226-42976-8.
15. Werndl C. What are the new implications of chaos for unpredictability. Br J Philos Sci. 2009;60(1):195–220. https://doi.org/10.1093/bjps/axn053.
16. Keefe FJ. Behavioral medicine: a voyage to the future. Ann Behav Med. 2011;41:141–51.
17. Feldman MD. Role of behavioral medicine in primary care. Curr Opin Psychiatry. 2012;25(2):121–7.

18. Fredrickson BL. The value of positive emotions: the emerging science of positive psychology is coming to understand why it's good to feel good. Am Sci. 2003;91(4):330–5.
19. Lopez SJ, Snyder CR, editors. The Oxford handbook of positive psychology. Oxford: Oxford University Press; 2011.
20. Fricton J. 2013. The last scroll: a novel. iUniverse. www.thelastscroll.com and http://www.amazon.com/dp/1475975163/ref=rdr_ext_tmb.
21. Turner JA, Dworkin SF, Mancl L, Huggins KH, Truelove EL. The roles of beliefs, catastrophizing, and coping in the functioning of patients with temporomandibular disorders. Pain. 2001;92(1–2):41–51.
22. Turner JA, Aaron LA. Pain-related catastrophizing: what is it? Clin J Pain. 2001;17(1):65–71.
23. Gatchel RJ, Garofalo JP, Ellis E, Holt C. Major psychological disorders in acute and chronic TMD: an initial examination. J Am Dent Assoc. 1996;127(9):1365–70, 1372, 1374
24. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychol Bull. 2007;133(4):581–624.
25. Garofalo JP, Gatchel RJ, Wesley AL, Ellis E III. Predicting chronicity in acute temporomandibular joint disorders using the research diagnostic criteria. J Am Dent Assoc. 1998;129(4):438–47.
26. John MT, Miglioretti DL, LeResche L, Von Korff M, Critchlow CW. Widespread pain as a risk factor for dysfunctional temporomandibular disorder pain. Pain. 2003;102(3):257–63.
27. Litt MD, Shafer DM, Ibanez CR, Kreutzer DL, Tawfik-Yonkers Z. Momentary pain and coping in temporomandibular disorder pain: exploring mechanisms of cognitive behavioral treatment for chronic pain. Pain. 2009;145(1–2):160–8.
28. Fricton JR, Ouyang W, Nixdorf DR, Schiffman EL, Velly AM, Look JO. Critical appraisal of methods used in randomized controlled trials of treatments for temporomandibular disorders. J Orofac Pain. 2010;24(2):139–51.
29. Fricton J, Look JO, Wright E, Alencar F, Chen H, Lang M, Ouyang W, Velly AM. Systematic review of intraoral orthopedic appliance for temporomandibular disorders: 51 RCTs reviewed. J Orofac Pain. 2010;24:237–54.
30. Fricton J, Velly A, Ouyang W, Look J. Does exercise therapy improve headache? A systematic review with meta-analysis. Curr Pain Headache Rep. 2009;13(6):413–9.
31. Bussone G, Grazzi L, D'Amico D, Leone M, Andrasik F. Biofeedback-assisted relaxation training for young adolescents with tension-type headache: a controlled study. Cephalalgia. 1998;18(7):463–7.
32. Loew TH, Sohn R, Martus P, Tritt K, Rechlin T. Functional relaxation as a somatopsychotherapeutic intervention: a prospective controlled study. Altern Ther Health Med. 2000;6(6):70–5.
33. Larsson B, Melin L, Doberl A. Recurrent tension headache in adolescents treated with self-help relaxation training and a muscle relaxant drug. Headache. 1990;30(10):665–71.
34. Blanchard EB, Appelbaum KA, Radnitz CL, Michultka D, Morrill B, Kirsch C, et al. Placebo-controlled evaluation of abbreviated progressive muscle relaxation and of relaxation combined with cognitive therapy in the treatment of tension headache. J Consult Clin Psychol. 1990;58(2):210–5.
35. Komiyama O, Kawara M, Arai M, Asano T, Kobayashi K. Posture correction as part of behavioural therapy in treatment of myofascial pain with limited opening. J Oral Rehabil. 1999;26(5):428–35.
36. Turk DC, Rudy TE, Kubinski JA, Zaki HS, Greco CM. Dysfunctional patients with temporomandibular disorders: evaluating the efficacy of a tailored treatment protocol. J Consult Clin Psychol. 1996;64(1):139–46.
37. Dworkin SF, Huggins KH, Wilson L, Mancl L, Turner J, Massoth D, et al. A randomized clinical trial using research diagnostic criteria for temporomandibular disorders-axis II to target clinic cases for a tailored self-care TMD treatment program. J Orofac Pain. 2002;16(6):48–63.
38. Turner JA, Mancl L, Aaron LA. Brief cognitive-behavioral therapy for temporomandibular disorder pain: effects on daily electronic outcome and process measures. Pain. 2005;117(3):377–87.

39. Flor H, Birbaumer N. Comparison of the efficacy of electromyographic biofeedback, cognitive-behavioral therapy, and conservative medical interventions in the treatment of chronic musculoskeletal pain. J Consult Clin Psychol. 1993;61(4):653–8.
40. Grossman P, Niemann L, Schmidt S, Walach H. Mindfulness-based stress reduction and health benefits: a meta-analysis. J Psychosom Res. 2004;57(1):35–43.
41. Miyamoto H, Sakashita H, Miyata M, Goss AN. Arthroscopic surgery of the temporomandibular joint: comparison of two successful techniques. Br J Oral Maxillofac Surg. 1999;37(5):397–400.
42. Holmlund AB, Axelsson S, Gynther GW. A comparison of discectomy and arthroscopic lysis and lavage for the treatment of chronic closed lock of the temporomandibular joint: a randomized outcome study. J Oral Maxillofac Surg. 2001;59(9):972–7; discussion 977–8.
43. Schiffman EL, Look JO, Fricton JR, Hodges JS, Swift JQ, Decker KL, et al. A randomized clinical trial evaluating four treatment strategies for patients with temporomandibular joint disc displacement without reduction with limited mouth opening. J Dent Res. 2007;86(1):58–63.
44. Ta LE, Dionne RA. Treatment of painful temporomandibular joints with a cyclooxygenase-2 inhibitor: a randomized placebo-controlled comparison of celecoxib to naproxen. Pain. 2004;111(1–2):13–21.
45. Mongini F, Bona G, Garnero M, Gioria A. Efficacy of meclofenamate sodium versus placebo in headache and craniofacial pain. Headache. 1993;33(1):22–8.
46. Ekberg EC, Kopp S, Akerman S. Diclofenac sodium as an alternative treatment of temporomandibular joint pain. Acta Odontol Scand. 1996;54(3):154–9.
47. Fricton J. Current evidence providing clarity in management of temporomandibular disorders: a systematic review of randomized clinical trials for intra-oral appliances and occlusal therapies. J Evid Based Dent Pract. 2006;6(1):48–52.
48. Velly AM, Gornitsky M, Philippe P. Contributing factors to chronic myofascial pain: a case-control study. Pain. 2003;104(3):491–9.
49. Fricton J, Nelson A, Monsein M. IMPATH: microcomputer assessment of behavioral and psychosocial factors in craniomandibular disorders. Cranio. 1987;5(4):372–81.
50. Schiffman E, Fricton J, Haley D. The relationship of occlusion, parafunctional habits and recent life events to mandibular dysfunction in a non-patient population. J Oral Rehabil. 1992;19:201–23.
51. Litt MD, Shafer D, Napolitano C. Momentary mood and coping processes in TMD pain. Health Psychol. 2004;23(4):354–62.
52. Rammelsberg P, LeResche L, Dworkin S, Mancl L. Longitudinal outcome of temporomandibular disorders: a 5-year epidemiologic study of muscle disorders defined by research diagnostic criteria for temporomandibular disorders. J Orofac Pain. 2003;17(1):9–20.
53. Wright AR, Gatchel RJ, Wildenstein L, Riggs R, Buschang P, Ellis E III. Biopsychosocial differences between high-risk and low-risk patients with acute TMD-related pain. J Am Dent Assoc. 2004;135(4):474–83.
54. Fillingim RB, Maixner W, Kincaid S, Sigurdsson A, Harris MB. Pain sensitivity in patients with temporomandibular disorders: relationship to clinical and psychosocial factors. Clin J Pain. 1996;12(4):260–9.
55. Turner JA, Brister H, Huggins K, Mancl L, Aaron LA, Truelove EL. Catastrophizing is associated with clinical examination findings, activity interference, and health care use among patients with temporomandibular disorders. J Orofac Pain. 2005;19(4):291–300.
56. Velly AM, Look JO, Carlson C, Lenton PA, Kang W, Holcroft CA, et al. The effect of catastrophizing and depression on chronic pain—a prospective cohort study of temporomandibular muscle and joint pain disorders. Pain. 2011;152(10):2377–83.
57. Jensen MP, Romano JM, Turner JA, Good AB, Wald LH. Patient beliefs predict patient functioning: further support for a cognitive-behavioural model of chronic pain. Pain. 1999;81(1–2):95–104.
58. Jensen MP, Turner JA, Romano JM. Changes in beliefs, catastrophizing, and coping are associated with improvement in multidisciplinary pain treatment. J Consult Clin Psychol. 2001;69(4):655–62.

59. Jensen MP, Turner JA, Romano JM. Correlates of improvement in multidisciplinary treatment of chronic pain. J Consult Clin Psychol. 1994;62(1):172–9.
60. Jensen MP, Turner JA, Romano JM. Self-efficacy and outcome expectancies: relationship to chronic pain coping strategies and adjustment. Pain. 1991;44(3):263–9.
61. Turner JA, Whitney C, Dworkin SF, Massoth D, Wilson L. Do changes in patient beliefs and coping strategies predict temporomandibular disorder treatment outcomes? Clin J Pain. 1995;11(3):177–88.
62. Turner JA, Holtzman S, Mancl L. Mediators, moderators, and predictors of therapeutic change in cognitive-behavioral therapy for chronic pain. Pain. 2007;127(3):276–86.
63. Jensen MP, Nielson WR, Turner JA, Romano JM, Hill ML. Changes in readiness to self-manage pain are associated with improvement in multidisciplinary pain treatment and pain coping. Pain. 2004;111(1–2):84–95.
64. Gatchel RJ, Stowell AW, Wildenstein L, Riggs R, Ellis E III. Efficacy of an early intervention for patients with acute temporomandibular disorder-related pain: a one-year outcome study. J Am Dent Assoc. 2006;137(3):339–47.
65. Bell IR, Caspi O, Schwartz GER, Grant KL, Gaudet TW, Rychener D, Maizes V, Weil A. Integrative medicine and systemic outcomes research. Issues in the emergence of a new model for primary health care. Arch Intern Med. 2002;162(2):133–40.
66. Mann D. Moving toward integrative care: rationales, models, and steps for conventional-care providers. J Evid Based Complement Altern Med. 2004;9(3):155–72.
67. Grzesiak RC. Psychologic considerations in temporomandibular dysfunction. A biopsychosocial view of symptom formation. Dent Clin N Am. 1991;35(1):209–26.
68. Epker J, Gatchel RJ, Ellis E III. A model for predicting chronic TMD: practical application in clinical settings. J Am Dent Assoc. 1999;130(10):1470–5.
69. Fricton J, Hathaway K, Bromaghim C. The interdisciplinary pain clinic: outcome and characteristics of a long term outpatient evaluation and management system. J Cranio Dis Oral Facial Pain. 1987;1(2):115–22.

Medico Legal Consideration

2

Risk Management of Temporomandibular Joint Therapy

Michael Kowalski

Abstract

As is always the case, patient needs are the priority. Risk management involves implementing strategies for patient care. Risk management strategies reduce the possibility of an adverse outcome, injury, or loss. Good risk management improves the quality of patient care and decreases the probability of poor treatment results or a medical malpractice claim. Such strategies include identifying risks, minimizing those risks, and reducing the impact of injury or loss when untoward outcomes do occur.

The hazards of not preparing for potential treatment complications will have significant long-term effects. Neglecting to have a comprehensive risk management plan in place can compromise patient care, increase liability risks, result in financial losses, and bring about licensing board issues and even potential criminal charges. Many patient risks can be reduced by adequate training of doctors and staff and by encouraging strong communication among the staff members, doctor, and patient.

- Identify the essential elements of a professional negligence claim and some defenses.
- Comply with mandatory state and federal regulations.
- Understand the duties, process, and benefits of obtaining informed consent and informed refusal.
- Adequate record keeping including electronic records, ownership of records, patient access to data, and confidentiality laws.
- When complications do occur, how to sympathetically handle them, the quality of the doctor-patient contact, and the protection of apology laws.
- Avoid inadvertently creating liability with promises, warranties, and guarantees.

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2.1 What Is Risk Management?

Risk management is not just about how not to get sued or brought before the medical or dental board. The fact is that with better risk recognition and management, the doctor will experience increased clinical options. Risk recognition specific to temporomandibular joint (TMJ) therapy commonly includes claims related to diagnosis of temporomandibular dysfunction (TMD), use of 3-D imaging, iatrogenic surgical mishaps, overtreatment of TMD with irreversible therapies permanently changing the occlusion, aggressive treatment of subclinical TMD signs such as asymptomatic intermittent TMJ clicking or mild joint tenderness, and unnecessary treatment such as proposing orthodontics or occlusal adjustments supposedly to prevent TMD.

As far as trends in malpractice claims, the overall frequency is down, which indicates that doctors are indeed doing a better job with risk management. In regard to frequency of current claims, fixed prosthodontics (crown and bridge) and failure to diagnose periodontal disease are continuing with approximately the same frequency. Increasing in frequency are claims related to failure to diagnose infections and nerve injury claims related to extractions and dental implant placement. Decreasing in frequency are claims related to endodontic mishaps and diagnosis and treatment of temporomandibular dysfunction.

In this chapter we will look at the essential elements of a professional malpractice claim and a few legal defenses, informed consent and informed refusal, patient records, handling treatment complications, and, finally, some ways doctors create liability for themselves beyond medical malpractice claims.

With risk recognition and management, the doctor will experience better results and more trusting patients who are more willing to accept treatment recommendations culminating with the best of clinical outcomes.

2.2 Professional Negligence and Defenses

Primum non nocere (first do no harm). Since the fifth century B.C., this phrase has been recognized as one of the most significant admonitions from the Hippocratic Oath. It is as true today as it was in the fifth century B.C. when Hippocrates practiced.[1] In relation to temporomandibular joint (TMJ) treatment, the devastating consequences of violating this tenet of medicine were demonstrated more than two decades ago with the Vitek Proplast-Teflon interpositional jaw implants which injured thousands of patients.

Vitek began general distribution of its Proplast implant in the early 1980s for use for meniscus replacement after discectomy.[2] However, by 1986, patients were

[1] Eugene D. Robin et al., *Cultural Lag and the Hippocratic Oath*, 345 Lancet 1422 (1995).

[2] JN Kent et al., *Pilot Studies of a Porous Implant in Dentistry and Oral Surgery*, 30 J. Oral Surg. 608 (1972); EC Heinds et al., *Use of Biocompatible Interface for Binding Tissues and Prosthesis in Temporomandibular Joint Surgery*, 38 Oral Surg. Oral Med. Oral Pathol. 512 (1974).

experiencing serious irreversible life-altering problems related to the Vitek implant.[3] The implant fragmented under continuous function scattering the Teflon in the joint's soft tissue and transported to regional lymph nodes.[4] In 1991 the Food and Drug Administration (FDA) ordered Vitek to remove the Proplast implants from the market. By then it was too late for many patients who suffered symptoms such as searing pain, immune deficiency disorders, and crippled jaw function. Foreign-body giant cell reaction to the Proplast was also a documented phenomenon even after the implants were removed.[5] Unfortunately, there were no longer-term solutions from most Proplast patients even following multiple surgeries.[6]

When it is alleged that a doctor has harmed a patient by professional negligence, or medical/dental malpractice, a civil lawsuit often follows. A dentist must exercise that degree of skill or care which is usual for the profession in the place where he or she practices.[7] The essential elements of a professional negligence cause of action that must be established are (1) the duty of a professional, (2) a breach of that duty, (3) actual causal connection between the breach and the injury, and (4) damages.[8]

Prior to there being a legal duty to the patient, a doctor-patient relationship must be established. Once the doctor accepts the individual as a patient, the doctor then has fiduciary obligations to the patient to provide appropriate care.

Regarding the element of the standard of care, in rendering professional services, a doctor "must have the degree of learning and skill ordinarily possessed by practitioners of the medical profession in the same locality and he must exercise the ordinary care in applying such learning and skill to the treatment of his patients."[9] The standard of care does not require medical perfection nor is a dentist guarantor of results.[10] A doctor does not impliedly guarantee results merely by undertaking professional services.[11] Nor does a generalized statement that the results of the treatment will be good constitute a promise of a particular result.[12] Furthermore, it is

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[3] Kaplan PA, Tu HK, Williams SM. *Erosive Arthritis of the Temporomandibular Joint Caused by Teflon-Proplast Implants: Plain Film Features*, Am. J. Roentgenol 1988; 151:337–340.

[4] L. Lagrotteria et al., *Patient with Lymphadenopathy Following Temporomandibular Joint Arthroplasty with Proplast*, 4 Cranio. 172 (1986).

[5] Lypka M, Yamashita DR. *Exuberant Foreign Body Giant Cell Reaction to a Teflon/Proplast Temporomandibular Joint Implant: Report of a Case*, J. Oral and Maxillofacial Surgery 2007; Vol. 65, 9:1680–1684.

[6] Sanders et al., *Long-Term Study of Temporomandibular Joint Surgery with Alloplastic Implants Compared with Non-Implant Surgery and Non-Surgical Rehabilitation for Painful Temporomandibular Joint Disc Displacements*, J. Oral and Maxillofacial Surgery 1400 (2002).

[7] 4 Witkin, Summary of California Law, 8th Ed., page 2778.

[8] Hanson v. Grode, (1999) 76 Cal.App.4th 601, 606.

[9] Atienza v. Taub (1987) 194 Cal.App.3d 388, 391.

[10] CACI 505; McKinney v. Nash (1981) 174 Cal.Rptr. 642, 649.

[11] Sanchez v. Rodriquez (1964) 226 Cal.2d 439, 449.

[12] McKinney v. Nash (1981) 120 Cal.App.3d 428. See also Custudio v. Bauer (1967) 251 Cal. App.2d 303 (unless doctor guaranteed sterility, no breach of warranty claim for pregnancy following a tubal ligation).

acceptable for dentists to use alternative methods of treatment.[13] This is true even if only a minority of practitioners utilize that particular method.[14]

Unless the facts present a medical/dental question which is resolved by common knowledge, expert witness testimony is required to establish that the standard of care has been breached.[15] The fact that a treatment may not have produced a good result does not, by itself, establish negligence.

There has to be a causal nexus between the breach of the standard of care and the patient's injury. The plaintiff must prove by reasonable medical probability based on competent expert testimony that a dentist's acts or omissions to act were a substantial factor in causing the injury, in other words, when it is more likely than not that the injury was a result of the negligent act.[16]

Finally, in order to have a viable action for dental malpractice, the patient must have suffered actual damages. The mere breach of a professional duty, causing only nominal damages, speculative harm, or the threat of future harm, that is not yet realized does not suffice to create a cause of action for professional negligence. Damages are an essential element of a professional negligence action.[17] Damages, in all cases, must be reasonable.[18] Moreover, if the plaintiff is found to be comparatively negligent, then both the plaintiff's economic and noneconomic damages must be reduced in proportion to the negligence attributable to the plaintiff,[19] or in a few jurisdictions, it could serve as a complete bar to recovery of damages.

Patients have a maximum time after an alleged injury within which to file a lawsuit. The time limit is known as the statute of limitations. Once the period of time specified in a statute of limitations passes, a claim can no longer be filed or, if filed, may be liable to be struck out if the defense to that claim is, or includes, that it is barred as having been filed after the applicable limitations period. There may be tolling provisions such as fraud or delayed discovery of the injury that will allow the claim to stand after the limitation period has expired. The intention of these laws is to facilitate resolution within a reasonable length of time to avoid the burden of defending a stale claim where evidence is lost, witnesses' memories have faded, or witnesses are no longer available for reasons such as death. What period of time is considered reasonable varies from state to state. Typical statute of limitations period for medical malpractice actions is from 1 to 3 years. Often the limitations period is extended if the patient was a minor at the time of the alleged injury.[20]

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[13] CACI 506; Meier v. Ross General Hospital (1968) 69 Cal.2d 420, 434.

[14] Barton v. Owen (1977) 71 Cal.App.3d 484, 502.

[15] Landeros v. Flood (1976) 17 Cal.3d 399, 410.

[16] Bromme v. Pavitt (1992) 5 Cal.App.4th 1487, 1493; Jones v. Ortho Pharmaceutical Corp. (1985) 163 Cal.App.3d 396, 402.

[17] CACI 500. MacDonald v. United States 767 F. Supp. 1295 (M.D. Pa. 1991).

[18] Bermuderz v. Ciolek (2015) 237 Cal.App. 4th 1311, 1328; California Civ.Code § 3359.

[19] Budd v. Nixen, (1971) 6 Cal.3d 195, 200; CACI 500; CACI 3960.

[20] For example, in California the statute of limitations is 1 or 3 years depending upon the facts of the case. California Code of Civil Procedure Section 340.5. In South Dakota the statute of limitations is 2 years. South Dakota Codified Laws Section 15-2-14.1.

Most state legislators have had the good sense to enact tort reform law for medical malpractice lawsuits.[21] Besides special statute of limitations periods, other reforms include a statutory cap on the amount of money a patient can recover for noneconomic damages. A patient's potential recoverable damages are divided into two large categories: economic or special damages and noneconomic or general damages. Economic damages include items such as the cost of subsequent medical care, lost wages, or loss of earning capacity. Tort reforms place little or no limits on the recovery of economic damages. Noneconomic or general damages primarily include claims for pain and suffering, and many states have placed a reasonable monetary limit on such damages.[22] Another common tort reform is limiting or abrogating of the collateral source rule. The reform allows patients to recover the actual out-of-pocket costs they paid for subsequent medical care rather than the total amount billed for the care as a portion may be covered and paid for by medical or dental insurance.[23]

2.3 Informed Consent and Refusal

Informed consent primarily has to do with the patient's right of self-decision as a measure of the doctor's duty to reveal. The informed consent process should be a dialog between the patient and the doctor that facilitates the patient's ability to make an informed treatment choice and have some control over the direction of care. Likewise, there is a benefit to the doctor. With informed consent the practitioner will have an educated patient. The benefit of an educated patient is that the patient will be trusting and will then more likely follow the dentist's advice. As a side benefit, the doctor will have legal protection should treatment complications arise and things not go well as anticipated.

At a minimum, the dialog must include serious risks of the procedure that are predictable and non-remote, the anticipated benefits of going through the procedure, and the alternatives to the procedure, including what is likely to happen if no treatment is rendered.

How much detail is included in the informed consent dialog and whether informed consent is obtained verbally or in writing are variables for each patient and situation, although there are helpful guidelines. The first consideration is whether the proposed procedure is necessary or elective. For example, in a patient with a partially erupted lower third molar with 12 mm pockets, there is not much debate as to the need to extract the tooth to avoid an inevitable infection and perhaps jaw fracture. Whereas, a patient presenting with a full bony impacted lower third molar

[21] A minority of states, such as Arizona and Washington, have failed to enact tort reform legislation for medical malpractice civil actions.

[22] For example, California has a cap of $250,000.00 for general damages. California Civil Code Section 3333.2(b). In South Dakota the general damages cap is double California's at $500,000.00. South Dakota Codified Laws Section 21-3-11.

[23] California Civil Code Section 3333.2(b); South Dakota Codified Laws Section 21-3-12.

that is asymptomatic requires much more consideration and that more information be conveyed to the patient concerning the risks, benefits, and alternatives to an extraction.

The risk of the procedure for serious bodily injury or death is another consideration as to the detail and documentation of the informed consent dialog. On one extreme, for example, a dental prophylaxis is a low-risk procedure, and no one dies from a cleaning. On the other extreme are procedures performed under conscious sedation or general anesthesia, where written informed consent is required by law in some states.[24]

The informed consent process must be given in the context of who the patient is as a person. For example, the patient's attitude about dentistry and medicine. Some patients are apprehensive or even phobic about dental treatment, while other patients are quite cavalier. The anticipated level of patient cooperation must be assessed: what is the quality of the patient's home life, and will they, or can they, follow instructions and recommendations? Assessment needs to include the patient's ability to understand English, and if less than inadequate, arrangements must be made for a translator, even if the translator is simply a family member. The age and sex of the patient must be considered. The occupation of the patient can be a significant factor in the level of informed consent. For instance, with a patient who is a school teacher or singer, as opposed to an artist or truck driver, a numb tongue or lip could be devastating to a career, and the risk of nerve injury during the operation may have to be discussed and emphasized in much greater detail.

Ideally, the informed consent dialog will be documented in writing. Preprinted consent forms for various treatments are helpful as a start and then can be further customized for specific procedures. However, such consent forms can always be criticized for the holes in information and what is left out. The informed consent dialog must be individualized to the patient and proposed treatment. Even if a consent form is not utilized, the informed consent dialog should be documented in the chart memorializing the relevant risks, benefits, and alternatives discussed. The informed consent process can be verbal. Written informed consent is not required by law with the exception of conscious sedation or general anesthesia.

Technology is another factor in the informed consent process that can be an aid to the dialog. Now, a portion of the informed consent process can take place with a video or DVD presentation. It is essential that what the patient viewed is recorded in the patient chart so that it cannot be denied later.

Lack of informed consent is considered a negligent breach of the standard of care as opposed to battery where a procedure is done without any consent from the patient.[25] There must be a causal relationship between the doctor's failure to inform and the injury to the patient. In other words, the situation in any case of alleged lack of informed consent must be such that the patient would not have consented to the operation had he or she been informed of the risks of such surgery that actually

[24] Business & Professions Code § 1682(e). There are exceptions for nitrous oxide sedation and oral medication sedation, Business & Professions Code § 1647.1.

[25] Cobbs v. Grant (1972) 8 Cal. 229.

occurred. In fact, it would be surprising if the patient/plaintiff did not claim that had he or she been informed of the dangers, he or she would have declined treatment. Therefore, an objective standard applies: if a reasonably prudent person in the patient's position would have consented to the operation had they been adequately informed of all the significant perils, then the lack of disclosure in a particular operation is *not* negligent.[26]

As to what must be disclosed to a particular patient about a specific procedure, an objection standard also applies. The doctor has a duty to disclose all material information a reasonable and prudent person would want to know to enable them to make an informed and educated decision regarding the proposed operation or treatment. The relevant information must include known risks of serious injury or death, as well as alternative treatments. The informed consent dialog does not require a discussion of every conceivable risk no matter how minor or remote.[27]

When a patient refuses the recommended treatment, the informed consent dialog must include informed refusal along with the risks of nontreatment. The doctor has an obligation to explain the risks of refusing a procedure in a language that the patient can understand and give as much information to the patient as a reasonable person needs to make an informed decision about declining treatment. The information must include the risk of death, serious injury, and other complications but not necessarily minor or remote risk.[28] A patient can assert a claim of negligence against a doctor if the doctor did not fully inform the patient about the risks of refusing a specific procedure and the patient suffers injuries from not having the procedure done.[29]

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[26] The Cobbs court in its wisdom stated:

Subjectively he may believe so, with the 20/20 vision of hindsight, but we doubt that justice will be served by placing the physician in jeopardy of the patient's bitterness and disillusionment. Thus, an objective test is preferable: i.e., what would a prudent person in the patient's position have decided if adequately informed of all significant perils. Id. at 245.

[27] The standard set forth in Cobbs is as follows:

First, the patient's interest in information does not extend to a lengthy polysyllabic discourse in all possible complications. A mini-course in medical science is not required; the patient is concerned with the risk of death or bodily harm, and problems of recuperation.

Second, there is no physician's duty to discuss the relatively minor risk inherent in common procedures, when it is common knowledge that such risk inherent in the procedure are of low incidence. Id. at 244.

[28] A [dentist] must explain the risks of refusing a procedure in language that the patient can understanding and give the patient as much information as [he/she] needs to make an informed decision, including any risk that a reasonable person would consider important in deciding not to have [a/an] [*specific medical procedure*]. The patient must be told about any risk of death or serious injury or significant potential complications that may occur if the procedure is refused. A [dentist] is not required to explain minor risks that are not likely to occur. CACI No. 534 (June 2016 Edition).

[29] Plaintiff claims that defendant [dentist] was negligent because [he/she] did not fully inform plaintiff about the risks of refusing the [*specific medical procedure*]. To establish this claim, plaintiff must prove all of the following:

1. That defendant [dentist] did not perform the procedure on plaintiff;
2. That defendant [dentist] did not fully inform plaintiff about the risks of refusing the [*specific medical procedure*];
3. That a reasonable person in plaintiff's position would have agreed to the [*specific medical procedure*] if he or she had been fully informed about these risks; and
4. That plaintiff was harmed by the failure to have the [*specific medical procedure*] performed.

Much of the informed consent process can be delegated, but at some point, the doctor must personally get involved in the dialog and be responsible for having a fully informed patient. When a written consent form is utilized, the doctor should sign the form after the patient has had all their questions satisfactorily answered.

From the risk management perspective, what must be kept in mind is that proper and adequate informed consent acts as a shield providing partial protection rather than a dome providing a complete defense. Doctors are never liable for manifestation of inherent risks (risk that can and do manifest to the best doctors on their best days) of the procedure. However, patients can never consent to negligent treatment. For example, lingual nerve injury is a known and recognized inherent risk during extractions of lower wisdom teeth. Should the lingual nerve be damaged during lower third molar surgery, the surgeon will not be legally answerable unless there was some negligent mishap such as the improper placement of an incision or the misuse of the bur. When a mishap outside of the standard of care occurs, informed consent is not a defense.

Due to varied philosophies and therapeutic modalities in treating temporomandibular dysfunction (TMD), another aspect of risk management as well as informed consent that is essential to understand is a legal affirmative defense known as the two schools of thought doctrine. The doctrine states that, if competent legal authority is divided, a doctor will not be held liable for following treatment approved by one group even if an alternative school recommends another approach.[30] The doctrine is an absolute defense even if the method of treatment at issue was in fact unsuccessful.[31] The doctrine gives physicians some leeway in deviations from customary medical practice. The defendant doctor has the burden of proving that there are indeed two schools of thought concerning the treatment at issue. The defense is generally recognized in virtually all states, although courts may differ somewhat in their approach to the doctrine.

This legal doctrine has existed for at least 170 years.[32] An 1862 Maine case stated the rule that "[w]here there are different schools of practice, all that any physician undertakes is that he understands and will faithfully treat the case according to the recognized rules of his particular school."[33] In 1917 a Washington state court held that each school of medicine is entitled to practice in its own way and that simply not utilizing the methods of another school does not constitute malpractice.[34]

The rational for the two schools of thought doctrine is that courts have acknowledged that doctors may disagree in good faith on what constitutes proper treatment for a particular medical problem because medicine is not a field of absolutes. Therefore, there will always be a need for the practitioner's clinical judgment.[35]

CACI No. 535 (June 2016 Edition).

[30] Jones v. Chidester, 610 A.2d 964, 969 (Pa. 1992).

[31] Meier v. Ross General Hospital, 445 P.2d 519, 529 (Cal. 1968).

[32] Bowman v. Woods, IG, Greene, 441 (Iowa 1848).

[33] Pattern v. Wiggin, 51 Me. 594 (1862).

[34] Ennis v. Banks, 164 P. 58 (Wahs. 1917).

[35] Majetich v. Westin (1969) 276 Cal.App.2d 216.

However, another aspect of the two schools of thought doctrine is that the method of treatment followed by the doctor must be supported by at least a respectable minority of expert clinicians.[36] Thus, if respectable experts in the field are in disagreement over the proper course to be pursued by a doctor or surgeon, the jury, with little or no knowledge of medicine and surgery, cannot be called upon to decide the dispute.[37] The principle provides that it is improper to require a lay jury to decide which of the two schools of thought was the proper procedure that should have been followed in a particular case when both schools have their respective and respected advocates and followers in the medical profession. In essence, the jury is not put in a position of choosing one respected body of medical opinion over another when each has a reasonable following among members of the medical community.[38] As to what constitutes a "respectable minor,"[39] courts in various jurisdictions differ. Some hold it to be a "considerable number" even if not a majority.[40] Others are more liberal and hold that as long as there is not an exclusively or uniformly used method by **all** practitioners, the alternative methods of treatment doctrine provide an absolute defense in a professional negligence case.[41] For example, it would be very difficult to apply the two schools of thought doctrine to Sargenti root canal paste since the technique is not taught in a single dental school in the country.[42]

A number of states express the two schools of thought doctrine as the alternative methods of treatment or care jury instruction.[43] For example, California Civil Jury Instruction (CACI) 506, Alternative Methods of Care, states: "[A/An] [*insert type of medical practitioner*] is not necessarily negligent just because [he/she] chooses one medically accepted method of treatment or diagnosis and it turns out that another medically accepted method would have been a better choice."

The requirements of the alternative methods of care defense are as follows: first, the doctor must exercise his or her best clinical judgment in following a given method. Second, the method of diagnosis or treatment must be recognized and approved in relation to the condition of the patient. In other words, a method may be approved in one particular situation for one particular patient, but under different circumstances, the method would not be approved. Third, the method of treatment

[36] Swanson v. Hood, 170 P. 135 (Wash. 1918).

[37] Remley v. Plummer, 79 Pa. Super. 117, 123 (1922).

[38] Jones, 610 A.2d at 966.

[39] Borja v. Phoenix Gen. Hosp., 727 P.2d 355, 357 (Ariz. 1986); Schwab v. Tolley, 345 So.2d 747, 753 (Fla. 1977); Olson v. Weitz, 221 P.2d 537, 538 (Wash. 1950); Hood v. Phillips, 554 S.W.2d 160 (1977).

[40] Jones, 610 A.2d at 969.

[41] Majetich v. Westin, 690 P.2d 726 (Cal.App. 1969).

[42] Dr. Stephen Cohen, et al., PATHWAYS TO THE PULP, 9th Edition, 270–271; 370; 441–442 (2006).

[43] For instance, California CACI 506; Wisconsin Finley v. Culligan, 548 N.W.2d 854, 860 (Wis. 1996); Louisiana McCoy v. Calamia, 653 So.2d 763 (La. App. 1995); Iowa Peters v. Vander Kooj, 494 N.W.2d 708, 712 (Iowa 1993); Arkansas Rickett v. Hayes, 511 S.W.2d 187, 195 (Ark. 1974); Missouri Ladish v. Gordon, 879 S.W.2d 623, 632 (Mo. App. 1994); Connecticut Wasfi v. Maceluch, 588 A.2d 204, 209 (Conn. 1991).

or diagnosis must be viewed in the context of the overall course of conduct followed by the doctor in relation to the particular circumstances of the patient.[44] Finally, the expert support for the method of treatment must be current and not obsolete or irrelevant.[45]

An interesting aspect of the alternative methods of care, or two schools of thought doctrine, in relation to informed consent is that there is no duty for a doctor to inform the patient of the existence of an alternative method articulated by a different school of thought. "What the duty of disclosure requires for the purpose of informed consent is the divulgence of material information, not necessarily the revelation of the existence of various schools of thought."[46] In other words, the doctor has no duty to inform the patient of an alternative school of medical thought that the doctor does not recommend.[47] However, the more controversial, unconventional, or novel the treatment modality or philosophy is or the less it has general acceptance in the medical community, the more important it is to include a discussion of the risks and alternatives during the clinician's informed consent dialog with the patient. With such proposed treatment, the doctor may want to advise treatment alternatives that are considered more traditional and mainstream from a risk management perspective.

2.4 Records

Evaluating a patient as a potential candidate for TMJ treatment, especially for surgery or irreversible therapy, must be done in the context of who the patient is as a person. Patient selection includes evaluating the patient's presentation, attitude, habits, dental history, and medical history.

From a risk management perspective, the patient's medical history cannot be overemphasized. Obtaining a complete medication history is essential. For example, does the patient have a history of being administered corticosteroid medications, and which could have devastating effects on bone metabolism? Staring with a questionnaire in a "yes" or "no" format is an acceptable method in obtaining the patient's medical history. Inquiry regarding medication history and past surgical history or recent hospitalization should also be included in the form. The patient's medical history should be periodically updated and recorded in the chart.

Obtaining a patient's past dental history can also start with a questionnaire in a "yes" or "no" format. History of orthodontic care or periodontal treatment may be important, and relevant records can be obtained from the patient's other treating doctors. Inquiry into the parafunctional habits may reveal the patient clenches or experiences bruxism.

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[44] Barton v. Owen (1977) 71 Cal.App.3d 484, 502–504.

[45] Tesauro v. Perrige, 650 A.2d 1079, 1083 (Pa. Super. 1994); Bonavitacola v. Cluver, 619 A.2d 1363, 1369 (Pa. Super. 1993).

[46] Mathis v. Morrissey (1992) 11 Cal.App.4th 332, 344.

[47] Parris v. Sands (1993) 21 Cal.App.4th 187.

Evaluating the patient's psychiatric history is particularly important in treating TMD. Assessing whether the patient's goals and expectations are reasonable is another essential element in the patient selection process prior to treating the TMD patient. Any unrealistic expectations need to be discovered early on in the doctor-patient relationship.

Initial examination should include obtaining and recording vital signs. Recording missing teeth and the condition of the remaining teeth as well as dental restorations should also be done. Evidence of parafunctional habits, such as wear facets, should be documented.

History and physical examination forms can be customized, or forms specific for TMD evaluation and examination can be obtained from professional liability carriers or state dental associations.

In general, records must include the patient's history, complaint, diagnosis, and treatment. Those elements are often statutorily required to be included by a state's dental practice act, and all entries must be dated and signed (electronic signature is acceptable with electronic medical records).[48] The clinician should always start with the basics with content in the SOAP format, make sure the records are legible, document any educational materials given to the patient, and then expand the records as appropriate. In evaluating the TMD patient, establishing a baseline becomes a valuable resource to gauge progress. Items to document regarding TMD signs and symptoms include pain, jaw motion abnormalities such as opening/closing deviations, joint sounds, occlusal abnormalities and asymmetries in the jaw, occlusion, and jaw range of motion, to mention a few.

Although when 3-D imaging such as a cone beam computer tomography scan (CBCT) is required by the standard of care is still being debated,[49] a CBCT can provide useful information in evaluating the TMD patient. The application of CBCT in evaluating the TMD patient includes assessing growth abnormalities, asymmetries, growth patterns, open bite, as well as the anatomical assessment of the TMJ. When deciding whether or not a CBCT is of clinical value in properly diagnosing the particular patient, it should be kept in mind that juries tend to think, when it comes to the utilization of technology, that if the doctor could have, the doctor should have.

Such specific physical and radiographic evaluation of the TMD patient is needed for proper treatment planning. Whatever treatment modality is ultimately agreed upon between the doctor and patient, the clinician must be able to visualize the end results of the treatment before initiating treatment.

As to the ownership of the chart and radiographs, they are the property of the doctor. Patients are entitled to the data in the records, but they are not entitled to the original records themselves. Doctors may be required to provide patients with a copy of their records and duplicates of their x-rays, but the doctor should never

[48] For example, California Health & Safety Code Sections 123105 and 11,191 and California Business & Professions Code Section 1683.

[49] Deeb G, et al. *Is Cone-Beam Computed Tomography Always Necessary for Dental Implant Placement*, J. Oral and Maxillofacial Surgery, Feb. 2017; Vol. 75, 2:285–289.

release the originals especially when litigation or an inquiry from the medical or dental board is anticipated.

Electronic medical records (EMR) are becoming the normal standard. Producing original records to the patient or co-treating doctor is not an issue with EMR. By transferring the records to a CD or external drive or even e-mail, the patient or co-treater has the original records. To help preserve EMR, they need to be backed up and stored off site. Reasonable efforts need to be made to guard against network attacks and other security threats to the patients' data. Protections against worms, viruses, and Trojans should be in place. Firewalls are needed to prevent unauthorized access to the office's network. Social media is not private and not secure, and it should never be assumed otherwise. With Facebook, MySpace, and Twitter, the doctor and staff should avoid "Friending" patients. A bright line between personal and professional must always be maintained.

Whether records are paper or electronic, the temptation to alter them when litigation is anticipated must be resisted. In defending a civil malpractice action or licensing board matter, it is much easier to deal with poor records, or no records, than altered records. If a correction needs to be made, the entry to be corrected should not be deleted. With a paper chart, a single line should be drawn through the entry and the correct information inserted above or below with the contemporaneous date noted and initialed by the individual making the correction. If a legal action is pending, the alteration of records can result in sanctions, accusations of fraud, and spoliation of evidence. Alteration of records is considered unprofessional conduct by licensing boards.

It is legally acceptable to add information to a chart entry at a later date so long as it is clear that it is a later written addendum to the entry. Such an entry should begin with the notation "Addendum." The date the addendum is being written must be clearly indicated at the beginning of the entry. Never should any portion of the chart be deleted, erased, destroyed, thrown away, or obliterated.

Whatever type of TMJ treatment is rendered to the patient, good record keeping is required. Documentation is primarily done for patient care issues. However, an additional risk management benefit is that well-documented treatment can often allow the doctor to avoid a lawsuit. If there is a lawsuit, good documentation can significantly help with the defense. Lay jurors do tend to believe that what is recorded in the chart happened and is true, and they believe that good doctors keep good records.

2.5 Complications

Treatment complication, therapeutic results less than expected, or prolonged healing and recovery time can and do happen to the best of clinicians. More often than not, it is not the fact that a complication occurs that determines whether the patient will consider legal action but rather how the complication is handled by the doctor. The quality of contact with the doctor is typically more important than what is said by the doctor. The doctor should give the patient full attention, with good eye contact, and not be distracted by writing notes in a chart or discussing the complication

while walking down the hallway to see another patient giving the patient the impression that the doctor is rushed, rude, or unsympathetic. The doctor needs to take the time to explain the nature of the problem the patient is experiencing as well as the plan for resolution. Staff members should be educated to put through patient calls to the doctor or to readily schedule timely follow-up appointments.

When a complication does occur, the doctor must be responsive to the patient. Patients often resort to lawsuits when they feel their problems are not being addressed and cared for and they have no alternative but legal recourse. The doctor must also educate the office staff and make sure they are responsive to patients experiencing treatment problems. If the doctor is too isolated from patients, then patients perceive the doctor as unsympathetic and are more likely to resort to legal action.

A principle that doctors must keep in mind prior to initiating treatment is that clinicians have to be able to fix what they started. Not only does the doctor need to be able to predict potential complications but also to recognize and diagnose complications once they do occur. The doctor must appropriately react to the complication. Either the doctor treats the complication or makes a timely referral to a specialist. Should the patient refuse the specialty referral, then the doctor needs to obtain and document the patient's informed refusal.

With a complex TMD patient, a team approach could be the proper mode of care and help prevent and/or treat complications. The team concept may include an oral and maxillofacial surgeon, orofacial dental and/or medical pain management doctor, orthodontist, dental hygienist or periodontist, prosthodontist or restorative dentist, and laboratory technician. Joint success comes from treatment protocols and division of responsibilities. The diagnosis, patient evaluation, patient selection, consent issues, treatment planning, and follow-up are coordinated among the team members with communication, including in-person meetings with all the team members and documentation. The patient is included in the process with treatment plan presentation and a pretreatment letter so a clear roadmap is presented. Such a risk management joint team concept with more complex cases helps prevent the more avoidable complications where there is no real planning and no real strategy.

With a non-compliant or difficult patient, the clinician may have no alternative but to refer or dismiss the patient. To avoid an allegation of patient abandonment when dismissing a patient, it is important that informed consent and/or informed refusal had been documented in the patient's chart. The patient must be given written notice that care will be terminated. From a risk management perceptive, the patient should be given a 30-day written notice and advised that he or she will be appointed for emergencies only during that time period and then must find a new doctor. Patient abandonment is considered an unprofessional conduct by licensing boards.[50] Although an entire treatment plan does not have to be completed prior to dismissing the patient, the doctor must complete any treatment in progress needed to leave the patient in a stable condition. The patient cannot be dismissed due to financial limitations so finances should be arranged prior to beginning treatment.

[50] California Business & Professions Code Section 1680(u).

One thing that will go a long way in keeping a patient from considering litigation when complications do occur is for the doctor to simply tell the patient "I'm sorry." As long as the doctor does not come out and admit he or she made a negligent mistake, telling the patient that the clinician is sorry for what the patient is experiencing and going through cannot later be used against the doctor should the patient file a lawsuit. There are a number of states that have passed (or are considering passing) immunity known as apology laws. These statutes allow doctors and healthcare providers to apologize and offer expressions of grief without their words being used against them in court. Such laws are intended to allow doctors to be more comfortable and open in communicating with their patients. Moreover, a sympathetic doctor defendant, who has done the right thing, is a lousy target in the court room.[51]

[51] States with Apology Laws:

Colorado Revised Statute 13-25-135 (2003)
Oregon Rev. Stat. 677.082 (2003)
Massachusetts ALM GL ch.233, 23D (1986)
Texas Civil Prac and Rem Code 18.061(1999)
California Evidence Code 1160 (2000)
Florida Stat 90.4026 (2001)
Washington Rev. Code Wash 5.66.010 (2002)
Tennessee Evid Rule 409.1(2003)
Ohio ORC Ann 2317.43 (2004)
Georgia Title 24 Code GA Annotated 24-3-37.1 (2005)
Wyoming Wyo. Stat. Ann. 1-1-130
Oklahoma 63 OKL. St. 1-1708.1H (2004)
Maryland MD Court & Judicial Proceedings Code Ann. 10-920 (2004)
North Carolina General Stat. 8C-1, Rule 413
Hawaii HRS Sec.626-1 (2006)
Maine MRSA tit. 2908 (2005)
South Dakota Codified Laws 19-12-14 (2005)
West Virginia 55-7-11a (2005)
Illinois Public Act 094-0677 Sec. 8-1901, 735 ILL. Comp. Stat. 5/8-1901 (2005)
Arizona A.R.S. 12-2605 (2005)
Louisiana R.S. 13:3715.5 (2005)
Missouri Mo. Ann. Stat. 538.229 (2005)
New Hampshire RSA 507-E:4 (2005)
Connecticut Public Act No. 05-275 Sec.9(2005) amended (2006) Conn. Gen. Stat. Ann. 52-184d
Virginia Code of Virginia 8.01-52.1 (2005)
Vermont S 198 Sec. 1. 12 V.S.A. 1912 (2006)Montana Code Ann.26-1-814 (Mont. 2005)
South Carolina Ch.1, Title19 Code of Laws 1976, 19-1-190 (2006)
Delaware Del. Code Ann. Tit. 10, 4318 (2006)
Indiana Ind. Code Ann. 34-43.5-1-1 to 34-43.5-1-5
Idaho Title 9 Evidence Code Chapter 2 0.9–0.207
Iowa HF 2716 (2006)
Nebraska Neb. Laws L.B. 373 (2007)
Utah Code Ann. 78-14-18 (2006)
North Dakota ND H.B. 1333 (2007).

2.6 Creating Liability

Due to the reality of marketing pressures on many practices, doctors can engage in aggressive marketing and oversell treatment outcomes. Clinicians can create liability for themselves outside of professional malpractice which may not necessarily be covered by their professional liability carrier. Doctors have to take care not to cross the line between expressing an opinion or prediction as to expected treatment outcome and making a promise, guarantee, or warranty of a specific result or treatment outcome. While marketing their practices and services, doctors must not engage in aggressive advertising practices that would violate provisions of state medical and dental practice acts. Keep in mind that marketing one's practice is not fully protected by the right of free speech.

Although the First Amendment of the US Constitution prohibits government interference with the right of free speech, the US Supreme Court made a distinction between political free speech, which is entitled to full First Amendment protection, and commercial free speech, which only has qualified protections.[52]

Commercial free speech has been defined by the US Supreme Court as when the speaker is engaged in commerce where the intended audience are actual or intended consumers. Commercial free speech is protected but less so and can, thus, be regulated by government agencies such as dental boards.[53] For example, advertising of a medical or dental practice cannot be deceptive, false, or misleading. Dentists must take care in calling themselves specialists if they have not successfully completed an ADA-approved specialty program, other recognized board, as well as advertising memberships and credentials. Generally, doctors are prohibited from advertising that their services are superior to other doctors nor can dentists advertise painless dentistry.[54]

Typically, the doctor-patient relationship is not considered a contractual relationship by the law. However, due to the consumer mentality of patients today, doctors can create liability for themselves by going too far in their marketing efforts and cross a line by making actual promises, warranties, or guarantees of specific treatment outcomes. When the final outcome or results are less than promised, the patient may have a viable claim for breach of contract. A nearly 90-year-old case, *Hawkins v. McGee*,[55] is a leading case on damages in contracts handed down by the New Hampshire Supreme Court.

George A. Hawkins, an 11-year-old boy, burned his hand from contact with an electrical wire after turning on the kitchen light in his house, leaving his hand severely

[52] The idea of commercial free speech was first introduced in Valentine v. Chrestensen, 316 U.S. 52 (1942).

[53] Bigow v. Virginia, 412 U.S. 809 (1975); Bolger v. Youngs Drug Products Corp., 463 U.S. 60 (1983).

[54] AAID, et al. v. Parker, et al., No. 16-50157 (5th Cir. 2017); Bingham v. Hamilton, 100 F.Supp.2d 1233 (E.D. Cal. 2000); Potts v. Hamilton, 334 F.Supp.2d 1206 (E.D. Cal. 20040).

[55] 84 N.H. 114, 146 A. 641 (N.H. 1929).

scarred. His father, Charles, was approached by Edward McGee, MD, the local physician, to treat his son's hand. Dr. McGee was anxious to experiment with skin grafting surgery but had no real surgical experience with the proposed surgery. Dr. McGee promised the boy's father that "I will guarantee to make the hand a hundred percent perfect hand." Both the father and the doctor understood that the doctor was guaranteeing a perfect result from the operation. However, Dr. McGee used a technique of skin grafting that he was unfamiliar with, and the operation failed. The surgery was performed in March of 1922. Not only did Dr. McGee fail to remove the scars to give the boy a functional hand, but because he used skin harvested from Hawkins' chest, the graft caused thick matted hair to grow on the palm of the boy's hand. Instead of the promised 100% perfect hand, Hawkins had a deformed hairy hand.

Hawkins sued under a theory of breach of contract and prevailed. The court stated that if Dr. McGee had simply said the surgery would result in a "perfect hand," the court would have characterized that statement as a mere expression of opinion or prediction. The court ruled that the doctor had made a commitment that amounted to a promise. The court's ruling is still the law today in professional liability cases. Only a doctor who expressly guarantees or warrants a cure or a specified result may be liable for breach of contract if that result is not achieved.[56]

An insightful aspect of the case relevant to today's practitioners is the rationale for why the plaintiff made the decision to sue for breach of contract rather than medical malpractice. The choice reflects a change in how the general public views physicians. Had Hawkins sued under the theory of professional negligence, the judge and jury would likely have concluded the doctor did his best, in good faith, to help the boy, and the results simply were not as anticipated. Fifty or sixty years ago, doctors were placed on a pedestal. Every mother wanted her son to be a priest, rabbi, or doctor. Those days are long gone.

Patients today have consumer expectations when it comes to healthcare. They expect dentistry to look good, function well, and last a long time. Any unrealistic expectations need to be discovered early on in the doctor-patient relationship as well as acknowledging the inability of surgery to correct personal problems. Finally, costs can far exceed realistic expectations where the doctor has not taken adequate time to educate the patient.

One last lesson from *Hawkins v. McGee* is having insurance coverage for the defense of a patient's lawsuit denied. After Hawkins prevailed against Dr. McGee, the doctor sought reimbursement of the damages awarded from his professional liability insurance carrier. The company refused to pay, and the doctor sued his carrier.[57] The insurance carrier argued that its professional liability insurance policy insuring Dr. McGee did not cover cases where the physician makes a contract to guarantee the result of treatment provided. Breach of contract and professional negligence are two separate and distinct torts. The court ruled in favor of the company holding that the policy did not cover the insured's liability under a special contract.

[56] Depenbrok v. Kaiser Foundation Health Plan (1978) 79 Cal.3d 167.

[57] McGee v. United States Fidelity & Guaranty Co.
United States Court of Appeals for the First Circuit, 53 F.2d 953 (1931).

Finally, to avoid being involved in litigation, even as a witness in the role of a prior or subsequent treating doctor, it is best to take care about commenting on treatment rendered by another doctor. Lawsuits often come about when doctors or staff members make careless critical comments or blame the prior treating doctor or office. The majority of patients are lay people who do not understand medicine, dentistry, or surgery. At least placing a call to the other doctor to gain an understanding of the prior treatment circumstances may allow for a way to defuse the situation (American Dental Association, Principles of Ethics and Code of Professional Conduct, Section 4.C.1). Such professional courtesy can avoid being deposed as a witness in a malpractice action. It is wise to keep in mind the words of Dr. Oliver Wendell Holmes, the physician father of the former Associate Justice of the US Supreme Court, "never believe what a patient tells you their doctor told them."

2.7 Conclusion

In the Code of Hammurabi, the penalty for medical malpractice was as severe as cutting off the physician's hand.[58] Although doctors today do not face a judgment of that magnitude, defending a malpractice claim in litigation is, nevertheless, just as painful and exceedingly unpleasant. The likelihood of litigation can be reduced with due care.

Risk recognition and management is not about practicing defensive medicine or having doctors feel like they are always looking your shoulder while practicing, but rather it is advantageous as it increases options leading to better results with less mishaps. Educated patients are more trusting patients which allow for more effective diagnosis, a greater degree of patient acceptance of treatment recommendations, and ultimately more excellent clinical outcomes. Most importantly, good risk recognition and management provide the clinician the confidence to simply enjoy being a doctor.

[58] Richard Hunderfund, MAGIC, MYTHS AND MEDICINE, 71–73 (1980).

The Temporomandibular Joint Through the Lens of Comparative Anatomy

3

Boaz Arzi and Carsten Staszyk

Abstract

The temporomandibular joint (TMJ) is a feature that distinguishes mammals from other vertebrates and to a lesser or greater degree have similar anatomic features (J Musculoskelet Neuronal Interact, 3:391–394, 2003). Specifically, the head of the mandible and the squamous temporal bone are covered by a thin fibrocartilage layer and separated by a disc that bisects the joint into two non-communicating compartments. However, despite these general anatomic similarities, the TMJ does exhibit profound functional differences across mammals, which are evolutionary adaptations to the species-specific demands placed on the joint.

3.1 Development

A variety of synonymous terms are currently used for the anatomical description of the TMJ in man and nonhuman mammals. In the following, the official human and veterinary nomenclature will be applied [1].

The TMJ makes a fairly late evolutionary appearance and develops relatively late in the fetal life [2, 3]. This "new joint" functionally replaces the joint between the malleus and incus. The embryologic formation of the TMJ is spread over a longer

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period of time as compared to other joints in the body and is dependent on the growth of two separate blastemata [4]. Meckel's cartilage, the skeletal element of the first branchial arch, provides a template for the development of the inferior bony part of the TMJ, the mandibular head (official Latin term: *Caput mandibulae*, syn. *Condylus mandibulae*) [5]. The cartilage of the squamous temporal bone represents the precursor for the superior bony part of the TMJ, the mandibular fossa (official Latin term: *Fossa mandibularis*) [5]. The gap between the developing mandibular fossa and the mandibular head is completely filled with ectomesenchyme during the first trimester of pregnancy in humans [6].

Several cellular processes occur in a well-orchestrated pattern and lead to the formation of the typical TMJ morphology. Ectomesenchymal cells differentiate into fibroblasts and produce the fibrocartilaginous articular surfaces of the mandibular head and the mandibular fossa as well as the articular disc [6, 7]. Along with the formation of the articular disc, the lateral pterygoid muscle forms. This developmental particularity explains the direct attachment of the superior head of the lateral pterygoid muscle to the articular disc in the mature TMJ [6].

The formation of the TMJ joint cavities is a complex process, which is most likely initiated by apoptosis [6–8]. In certain ectomesenchymal regions superior and inferior to the developmental articular disc, apoptosis results in the formation of the superior and inferior joint cavities [7–10]. Both joint cavities develop a typical articular capsule composed of a peripheral fibrous stratum and an inner synovial stratum. This concludes the formation of the TMJ. However, growth and maturation occur after birth, and the joint is completed only at the second decade of life in human. Finally, the TMJ represents a very unique joint, in both function and structure. Functionally, it is a modified hinge joint allowing a large range of movements including translation and rotation. Structurally it is a synovial joint with fibrocartilaginous articular surfaces, rather than a joint with hyaline cartilage.

3.2 Anatomy

The temporal part of the human TMJ is represented by the *Fossa mandibularis* (also referred to as mandibular fossa or glenoid fossa) [5] which merges anteriorly with the *Tuberculum articulare* (also referred to as articular eminence or articular tubercle) [5]. Posteriorly the *Fossa mandibularis* merges with a small bony protrusion, which is referred to the postglenoid process, although this is not an official anatomical term. The mentioned temporal structures possess fibrocartilaginous articular surfaces and contact the articular disc during normal functional movement of the TMJ.

The mandibular part of the TMJ is represented by the *Caput mandibulae* (mandibular head, syn. mandibular condyle) which is the most anterior structure of the *Processus condylaris* (condylar process) of the mandible [5].

Posterior to the postglenoid process as well as to the mandibular head, the *Meatus acusticus externus* (external acoustic meatus) is present [5]. Its thin inferior wall borders the posterior extension of the TMJ but does not provide any articular

surface. Although not defined by the anatomical nomenclature, the inferior wall of the external acoustic meatus is usually referred to as the tympanic plate (Fig. 3.1a).

Although placental mammals developed an enormous variety of feeding mechanisms, the general features of the TMJ (modified hinge joint, fibrocartilaginous articular surfaces, two synovial joint compartments separated by an articular disc) remained largely constant during evolution. Only a few species with very particular evolutionary adaptions show major changes in their TMJ morphology. Whales and

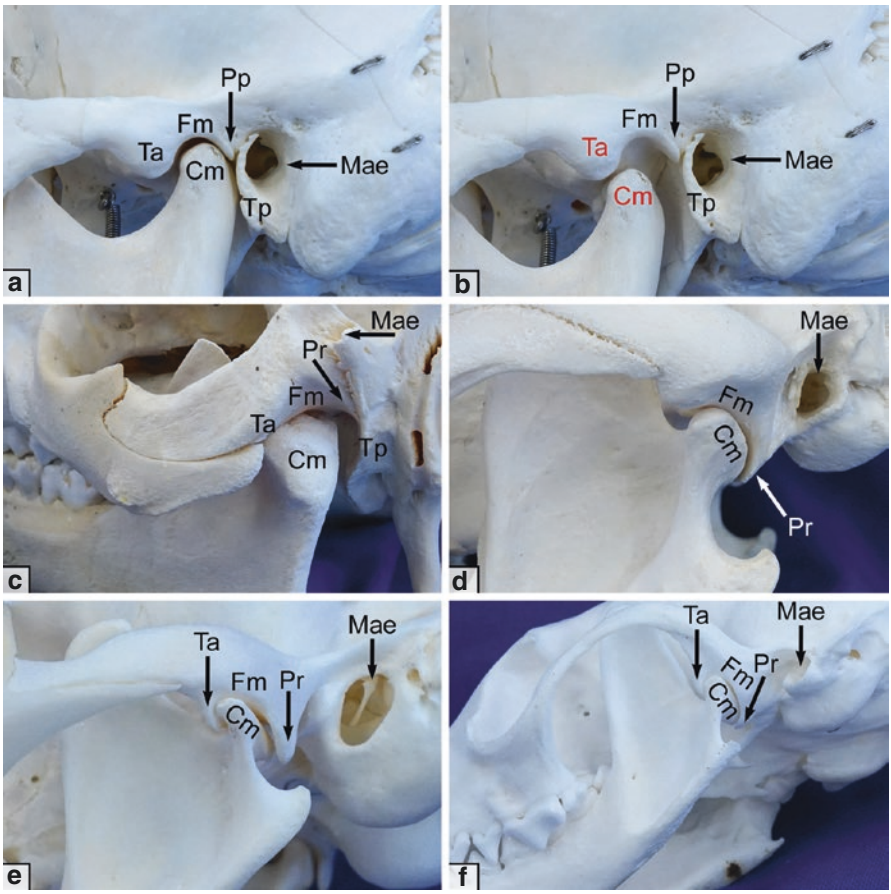

Fig. 3.1 Anatomy of the human TMJ in comparison with omnivore and carnivore species. (**a**) Left human TMJ, mandibular head (Cm) placed in the mandibular fossa (Fm). (**b**) Left human TMJ, mandibular head (Cm) after sliding on the articular tubercle (Ta). (**c**) Left TMJ of a pig. Note the indistinct articular tubercle (Ta) and the short retroarticular process (Pr). (**d**) Left TMJ of a dog. Note the absence of an articular tubercle and the long and marked retroarticular process (Pr). (**e**, **f**) Left TMJ of a cat (**e**) and a beech marten (**f**). Note the almost complete congruent shape of the mandibular head and the corresponding temporal structures. Official anatomical terms: *Ta* Tuberculum articulare, *Fm* Fossa mandibularis, *Mae* Meatus acusticus externus, *Cm* Caput mandibulae, *Pr* Processus retroarticularis. Inofficial terms: *Pp* postglenoid process, *Tp* tympanic plate

dolphins do not feature separated joint compartments, and some edentates (i.e., sloth) as well as marsupials do not develop an articular disc [2].

In other mammals, the individual components of the TMJ demonstrate adaptations in its form and function to the demands of the species such as feeding mechanism, speech and communication, as well as the need for freedom from constraints such as the body weight [2]. Most distinct morphological adaptions are seen in the bony components of the TMJ resulting in almost perfect congruency of the mandibular head and the temporal parts of the joint, as seen in some carnivores, or in almost incongruent joint partners, as seen in some ruminants (Figs. 3.1 and 3.2) [2, 11, 12].

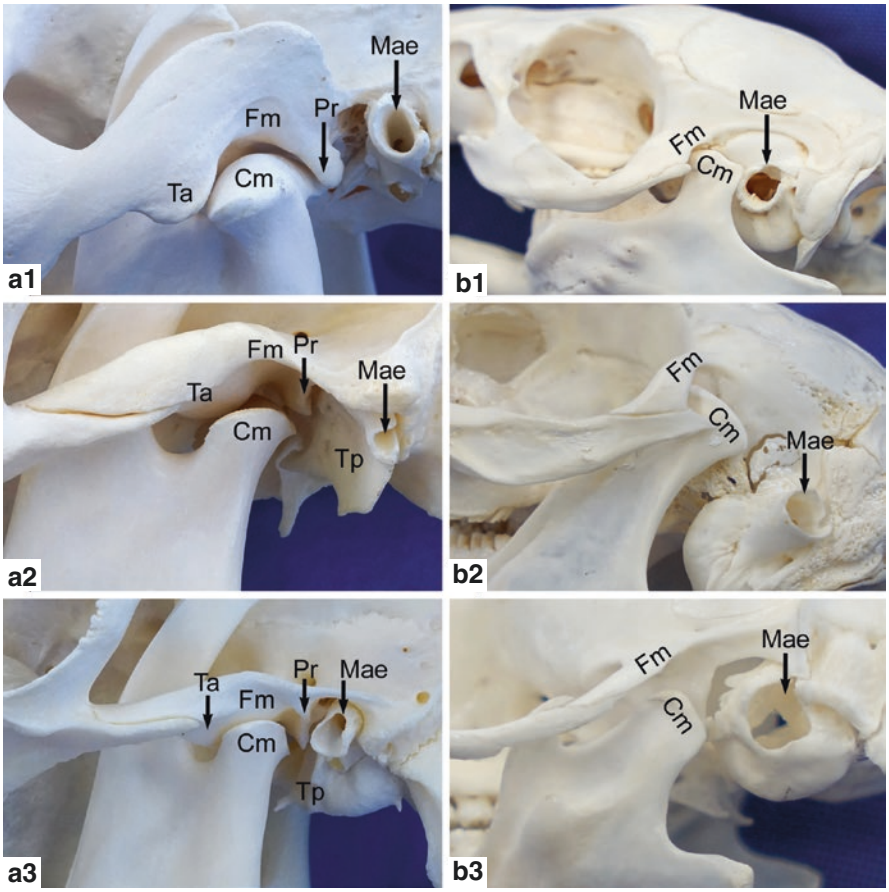

Fig. 3.2 Anatomy of the TMJ of herbivores. (**a1**) Left TMJ of a horse. Note the marked articular tubercle (Ta) and the long retroarticular process (Pr). (**a2**, **a3**) Left TMJ of a cattle (**a2**) and a sheep (**a3**). Note the undistinct articular tubercle (Ta) and retroarticular process (Pr). (**b1**, **b2**, **b3**) Left TMJ of a guinea pig (**b1**), a rabbit (**b2**), and a rat (**b3**). Note the mandibular head (Cm) is orientated in rostrocaudal direction. Official anatomical terms: *Ta* Tuberculum articulare, *Fm* Fossa mandibularis, *Mae* Meatus acusticus externus, *Cm* Caput mandibulae, *Pr* Processus retroarticularis. Inofficial terms: *Tp* tympanic plate

3.2.1 Carnivores

Carnivores possess a bar-shaped *Caput mandibulae* with a marked lateromedial orientation (Fig. 3.1d–f). In contrast to herbivores and omnivores, it is placed at the same horizontal level as the mandibular cheek teeth row. The temporal part of the TMJ is characterized by a pronounced *Processus retroarticularis*, which is analogous to the postglenoid process in man. The rostral margin of the *Fossa mandibularis* is blunt in dogs but features a very distinct rim in the cat and even more pronounced in mustelids. This delicate bony structure corresponds to the *Tuberculum articulare* described in ungulates [13]. As the mandibular head in cats and mustelids is completely surrounded by bony structures of the temporal bone, their TMJ is highly congruent and allows only very minimal, if at all, lateral and translational motion. The TMJ disc is exceptionally thin which is likely due to the limitation of joint motion to mostly sliding movements (i.e., opening and closing the mouth). The joint capsule is strengthened by a strong lateral ligament which becomes tighter as the mouth opens [14].

3.2.2 Omnivores

The most researched omnivore is the pig as it provides a suitable animal model to study human TMJ disorders and therapeutics [18]. Unlike ruminants and carnivores that retain a cartilaginous or fibrous intermandibular joint (i.e., symphysis), the pig has a completely fused *Synchondrosis intermandibularis* similar to humans [15]. The pig's TMJ is similar to human in its movements as it allows movements in all planes and also translational movements [16]. Not only is the movement of the pig's TMJ found to be similar to human, a structure-function relationship exists within the pig's TMJ disc which is fairly analogous to the human TMJ disc in dimension, collagen and glucosaminoglycans content, and mechanical properties [17, 18]. The pig possesses a relatively small retroarticular bony abutment in the form of the *Processus retroarticularis*, which merges with the rostral wall of the tympanic bulla [15]. In this respect, the TMJ of the pig appears to be more similar to the human TMJ than to the TMJ of other mammals. However, the *Tuberculum articulare* is less pronounced in the pig compared to the human TMJ (Fig. 3.1c).

3.2.3 Herbivores

In ruminants such as sheep, cattle, and goats, the bones of the TMJ have a flat mandibular head and an only shallow convex mandibular fossa. The retroarticular process is small and less pronounced, and the articular tubercle is indistinct or even absent [19] (Fig. 3.2c, e). Remarkably, these typical morphological features of the TMJ of ruminants are similar to the changes seen in edentulous humans. In human, the mandibular head flatness is thought to be a response to lack of canine/capsid occlusion [20]. From a comparative perspective, ruminants do not have distinct

bony prominences that limit the rostrocaudal movement of the mandibular head. [20] There is no bone separating the condylar process and the external auditory meatus, and only retrodiscal connective tissue is present in the posterior joint aspect.

In contrast to ruminants, equids develop a very prominent retroarticular process as well as a large articular tubercle (Fig. 3.2a), which features a fibrocartilaginous articular surface [13]. For mandibular movements the concave mandibular head leaves the mandibular fossa and glides along the slope of the articular tubercle. In this position the complex and powerful equine chewing cycle is performed [13, 21, 22].

Rodents (e.g., mice, guinea pigs, and rats) as well as lagomorphs (e.g., rabbits) have a more rounded or ovoid mandibular head which is orientated in rostrocaudal direction [23, 24]. The zygomatic process of the temporal bone features on its ventral aspect a flat and indistinct mandibular fossa (Fig. 3.2b, d, f). Further bony structures of the TMJ like a retroarticular process or an articular tubercle are absent. The bony architecture of the TMJ in rodents allows a wide range of mobility which is a prerequisite for the typical chewing movement in oblique and/or rostrocaudal direction [25].

3.2.4 Other Anatomical Variations and the TMJ

The spatial location of the TMJ among the species is related to the skull configuration and the dissipation and distribution of masticatory loads. This in turn relates to the classification of the species (e.g., herbivore, omnivore, or carnivore). The TMJ is positioned above the occlusal plane in humans, omnivores, and herbivores but approximately at the line of the occlusal plane in carnivores and below the occlusal plane in cetacean such as dolphin and whales. The hypothesis is that selection for TMJ position higher than the occlusal plane is designed to enhance masticatory performance and is associated with masticatory mechanics and bite force [26, 27]. In addition, a relatively higher position of the TMJ above the occlusal plane is sought to enhance masseter and medial pterygoid size and likely moment arm length and even distribution of occlusal forces alone on the premolar and molar teeth [26, 28].

3.3 Mastication

Mastication in mammals is cyclic and rhythmic activity [29]. Masticatory forces are aimed at inciting effective movement of the mouth while avoiding damage to the occlusion and the teeth. This in return requires adjustment of the masticatory forces, the chewing cycle in response to the properties of the materials being chewed [29–31]. The bite strain magnitude is mainly correlated with the bite strain load rate, and the strain magnitude is usually correlated with the strain loading [29]. This means that during rhythmic mastication in mammals, the bite force and strain on the TMJ increased with the rate at which the mandible is loaded [29]. This, in turn, implies

that masticatory forces and TMJ structure-function demand are dependent on the lifestyle, behavior, and sex of the animal [29, 31, 32].

3.4 TMJ Disorders in Nonhuman Mammals

Disorders of the TMJ in human are widespread and affecting between 5 and 15% of the population, and most cases are associated with TMJ disc displacement [18, 33]. TMJ disorders in other mammals are also widespread, but the true occurrence of TMJ disorders among mammals remains elusive. Nevertheless, TMJ disorders in mammals mostly include TMJ arthritis, fractures due to trauma, dysplasia, and ankylosis [33–47]. In addition, as seen in humans, TMJ disorders may become debilitating, necessitating medical or surgical treatments. A striking similarity between human and mammals such as dogs, cats, sea lions, and seals is that a common pathology affecting the TMJ is degenerative joint disease, also known as osteoarthritis or osteoarthrosis. However, displacement of the articular disc or "internal derangement" is not a recognized TMJ disorder in nonhuman mammals. In domestic dogs and cats, TMJ osteoarthritis is typically found in concert with other TMJ disorders such as dysplasia and luxation. Similarly, the common symptoms of TMJ disorders such as painful joints and decreased functionality (range of motion) [48] are seen in human, dogs, and cats in fairly similar occurrence. Specifically 26.6% of the dogs and 50% of cats with solitary TMJ osteoarthritis are demonstrating clinical signs [40], and humans with arthritic changes of the TMJ are seen in 35% of TMJs of asymptomatic peoples [49] and in 63–75% of people with juvenile idiopathic arthritis, the majority of whom were asymptomatic at the time of evaluation [50–52].

As noted earlier, TMJ arthritis also occurs in marine mammals such as the California sea lion and seals. The incidence of TMJ-OA in California sea lions and seals is high and varies in severity with an incidence of approximately 60% in California sea lions and approximately 30% in harbor and fur seals. Finally, herbivores such as the horse have also been found to exhibit age-related changes of the TMJ as well as osteoarthritis of the joint.

3.5 Summary

In summary, humans and most other placental mammals share common features with regard to the development and general anatomy of the TMJ. However, there are striking differences with the spatial position and mechanics of the TMJ among mammals that are associated with the structure-function demand and masticatory demand. This in turn is related to the lifestyle of the animal with regard to its eating preferences (i.e., herbivores vs. carnivores vs. omnivores). Finally, TMJ disorders appear to occur across mammals with various degrees of similarities, occurrence, and severity, and its significance play an important role in quality of life and possibly survival of the species.

References

1. Anonymous. Nomina anatomica Veterinaria. 5th ed. Hannover: Editorial Comittee; 2005.
2. Herring SW. TMJ anatomy and animal models. J Musculoskelet Neuronal Interact. 2003;3(4):391–4.
3. Keith DA. Development of the human temporomandibular joint. Br J Oral Surg. 1982;20(3):217–24.
4. Symons NB. The development of the human mandibular joint. J Anat. 1952;86(3):326–32.
5. Anonymous. Terminologia anatomica: international anatomical terminology. Stuttgart: Thieme; 1998.
6. Sabu C, de Moraes LOC, de Quadros Uzeda-Gonzalez S, et al. Morphology and ultrastracture of the temporomandibular joint disc in human fetuses 21 to 28 weeks old. Braz J Morphol Sci. 2005;22(4):233–8.
7. Ohshima T, Yonezu H, Nishibori Y, et al. Morphological observation of process of mouse temporomandibular joint formation. Bull Tokyo Dent Coll. 2011;52(4):183–90.
8. Yamaki Y, Tsuchikawa K, Nagasawa T, et al. Embryological study of the development of the rat temporomandibular joint: highlighting the development of the glenoid fossa. Odontology. 2005;93(1):30–4.
9. Liang W, Li X, Gao B, et al. Observing the development of the temporomandibular joint in embryonic and post-natal mice using various staining methods. Exp Ther Med. 2016;11(2):481–9.
10. Matsuda S, Mishima K, Yoshimura Y, et al. Apoptosis in the development of the temporomandibular joint. Anat Embryol (Berl). 1997;196(5):383–91.
11. Angelo DF, Morouco P, Alves N, et al. Choosing sheep (Ovis aries) as animal model for temporomandibular joint research: morphological, histological and biomechanical characterization of the joint disc. Morphologie. 2016;100(331):223–33.
12. Bermejo A, Gonzalez O, Gonzalez JM. The pig as an animal model for experimentation on the temporomandibular articular complex. Oral Surg Oral Med Oral Pathol. 1993;75(1):18–23.
13. Adams K, Schulz-Kornas E, Arzi B, et al. Functional anatomy of the equine temporomandibular joint: collagen fiber texture of the articular surfaces. Vet J. 2016;217:58–64.
14. Evans HE, de Lahunta A. Miller's anatomy of the dog. 4th ed. New York: Elsevier; 2013.
15. Nickel R, Schummer A, Wille KH, Wilkens H. Lehrbuch der Anatomie der Haustiere. 8th ed. Berlin: Parey; 2001.
16. Sun Z, Liu ZJ, Herring SW. Movement of temporomandibular joint tissues during mastication and passive manipulation in miniature pigs. Arch Oral Biol. 2002;47(4):293–305.
17. Allen KD, Athanasiou KA. Tissue engineering of the TMJ disc: a review. Tissue Eng. 2006;12(5):1183–96.
18. Kalpakci KN, Willard VP, Wong ME, et al. An interspecies comparison of the temporomandibular joint disc. J Dent Res. 2011;90(2):193–8.
19. Patil AS, Bindra GK. Morphology of the temporomandibular joint (TMJ) of sheep (Ovis aries). Open J Vet Med. 2012;2(4):242–4.
20. Bifano C, Hubbard G, Ehler W. A comparison of the form and function of the human, monkey, and goat temporomandibular joint. J Oral Maxillofac Surg. 1994;52(3):272–5.
21. Bonin SJ, Clayton HM, Lanovaz JL, et al. Kinematics of the equine temporomandibular joint. Am J Vet Res. 2006;67(3):423–8.
22. Staszyk C, Lehmann F, Bienert A, Ludwig K, Gasse H. Measurement of the masticatory forces in the horse. Pferdeheilkunde. 2006;22(1):12–6.
23. Orset E, Chaffanjon P, Bettega G. Temporomandibular joint model: anatomic and radiologic comparison between rat and human. Surg Radiol Anat. 2014;36(2):163–6.
24. Porto GG, Vasconcelos BC, Andrade ES, et al. Comparison between human and rat TMJ: anatomic and histopathologic features. Acta Cir Bras. 2010;25(3):290–3.
25. Hautier L, Lebrun R, Saksiri S, et al. Hystricognathy vs sciurognathy in the rodent jaw: a new morphometric assessment of hystricognathy applied to the living fossil Laonastes (Diatomyidae). PLoS One. 2011;6(4):e18698.

26. Armfield BA, Vinyard CJ. An interspecific analysis of relative jaw-joint height in primates. Am J Phys Anthropol. 2010;142(4):519–30.
27. Vinyard CJ, Wall CE, Williams SH, et al. Comparative functional analysis of skull morphology of tree-gouging primates. Am J Phys Anthropol. 2003;120(2):153–70.
28. Ward SC, Molnar S. Experimental stress analysis of topographic diversity in early hominid gnathic morphology. Am J Phys Anthropol. 1980;53(3):383–95.
29. Ross CF, Dharia R, Herring SW, et al. Modulation of mandibular loading and bite force in mammals during mastication. J Exp Biol. 2007;210(Pt 6):1046–63.
30. Agrawal KR, Lucas PW, Bruce IC, et al. Food properties that influence neuromuscular activity during human mastication. J Dent Res. 1998;77(11):1931–8.
31. Anderson K, Throckmorton GS, Buschang PH, et al. The effects of bolus hardness on masticatory kinematics. J Oral Rehabil. 2002;29(7):689–96.
32. Buschang PH, Hayasaki H, Throckmorton GS. Quantification of human chewing-cycle kinematics. Arch Oral Biol. 2000;45(6):461–74.
33. Reston JT, Turkelson CM. Meta-analysis of surgical treatments for temporomandibular articular disorders. J Oral Maxillofac Surg. 2003;61(1):3–10.
34. Beam RC, Kunz DA, Cook CR, et al. Use of three-dimensional computed tomography for diagnosis and treatment planning for open-mouth jaw locking in a cat. J Am Vet Med Assoc. 2007;230(1):59–63.
35. Dickie AM, Schwarz T, Sullivan M. Temporomandibular joint morphology in Cavalier King Charles Spaniels. Vet Radiol Ultrasound. 2002;43(3):260–6.
36. Gatineau M, El-Warrak AO, Marretta SM, et al. Locked jaw syndrome in dogs and cats: 37 cases (1998–2005). J Vet Dent. 2008;25(1):16–22.
37. Maas CP, Theyse LF. Temporomandibular joint ankylosis in cats and dogs. A report of 10 cases. Vet Comp Orthop Traumatol. 2007;20(3):192–7.
38. Meomartino L, Fatone G, Brunetti A, et al. Temporomandibular ankylosis in the cat: a review of seven cases. J Small Anim Pract. 1999;40(1):7–10.
39. Roza MR, Silva LA, Januario AL, et al. Cone beam computed tomography in the diagnosis of temporomandibular joint alterations in cats. J Feline Med Surg. 2011;13(6):393–8.
40. Arzi B, Cissell DD, Verstraete FJ, et al. Computed tomographic findings in dogs and cats with temporomandibular joint disorders: 58 cases (2006-2011). J Am Vet Med Assoc. 2013;242(1):69–75.
41. Aalderink MT, Nguyen HP, Kass PH, et al. Dental and temporomandibular joint pathology of the Eastern Pacific Harbour Seal (Phoca vitulina richardii). J Comp Pathol. 2015;152(4):335–44.
42. Aalderink MT, Nguyen HP, Kass PH, et al. Dental and temporomandibular joint pathology of the Northern Fur Seal (Callorhinus ursinus). J Comp Pathol. 2015;152(4):325–34.
43. Arzi B, Winer JN, Kass PH, et al. Osteoarthritis of the temporomandibular joint in southern sea otters (Enhydra lutris nereis). J Comp Pathol. 2013;149(4):486–94.
44. Arzi B, Leale DM, Sinai NL, et al. The temporomandibular joint of California sea lions (Zalophus californianus): part 2-osteoarthritic changes. Arch Oral Biol. 2015;60(1):216–22.
45. Arzi B, Murphy MK, Leale DM, et al. The temporomandibular joint of California sea lions (Zalophus californianus): part 1—characterisation in health and disease. Arch Oral Biol. 2015;60(1):208–15.
46. Winer JN, Arzi B, Leale DM, et al. Dental and temporomandibular joint pathology of the polar bear (Ursus maritimus). J Comp Pathol. 2016;155(2–3):231–41.
47. Winer JN, Arzi B, Leale DM, et al. Dental and temporomandibular joint pathology of the walrus (Odobenus rosmarus). J Comp Pathol. 2016;155(2–3):242–53.
48. Tanaka E, Detamore MS, Mercuri LG. Degenerative disorders of the temporomandibular joint: etiology, diagnosis, and treatment. J Dent Res. 2008;87(4):296–307.
49. Brooks SL, Westesson PL, Eriksson L, et al. Prevalence of osseous changes in the temporomandibular joint of asymptomatic persons without internal derangement. Oral Surg Oral Med Oral Pathol. 1992;73(1):118–22.

50. Muller L, Kellenberger CJ, Cannizzaro E, et al. Early diagnosis of temporomandibular joint involvement in juvenile idiopathic arthritis: a pilot study comparing clinical examination and ultrasound to magnetic resonance imaging. Rheumatology (Oxford). 2009;48(6):680–5.
51. Stoll ML, Good J, Sharpe T, et al. Intra-articular corticosteroid injections to the temporomandibular joints are safe and appear to be effective therapy in children with juvenile idiopathic arthritis. J Oral Maxillofac Surg. 2012;70(8):1802–7.
52. Weiss PF, Arabshahi B, Johnson A, et al. High prevalence of temporomandibular joint arthritis at disease onset in children with juvenile idiopathic arthritis, as detected by magnetic resonance imaging but not by ultrasound. Arthritis Rheum. 2008;58(4):1189–96.

Detailed Anatomy of the Temporomandibular Joint

4

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Abstract

The temporomandibular joint is one of the most complex structures in the human body. Understanding the form is a prerequisite to understanding the function. This chapter is devoted to presenting the details of TMJ anatomy.

4.1 Macroscopic Features of the Temporomandibular Joint

The temporomandibular joint (TMJ) is a bilateral synovial articulation between the mandible and the two temporal bones with several intra- and interindividual morphological variants [1]. Its complex anatomy is characterized by highly incongruent skeletal surfaces that are thus separated by a fibrous disc in two independent but cooperating cameras: the temporo-discal space superiorly and condylar-discal space inferiorly. This division aims to increase joint stability by enlarging the contact area and provides the TMJ a unique dynamic and kinematic behavior, allowing movement of the lower jaw with six degrees of freedom under the heaviest load in the body joints (50–80 kpa) [2].

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4.2 Mandibular Articular Surface

The condyle represents the mandibular articular component located on top of the condylar process by means of a neck, showing peculiar shape and spatial orientation. It is geometrically similar to an ovoid with a rounded medial pole in contact with the entoglenoid area and a lateral pole which is extra-articular. It has a minor anteroposterior axis of about 1 cm and major axis of about 2 cm directed backward and medially in order to form an anterior 145° angle with the contralateral at the basion (landmark located at the anterior edge of the foramen magnum) (Fig. 4.1).

The landmarks used to identify the anatomical limits of the condylar surface for diagnostic, surgical, or reconstructive purposes are reported in Fig. 4.2 [3].

Both shape and dimensions of the mandibular condyle show high variability and change with increasing age. In the embryonic period, the condyle is small and approximately spherical and progressively moves to a more elongated shape under the morphogenetic regulation induced by the contraction of the lateral pterygoid muscle which inserts on the pterygoid fossa at the anterior base of the condylar head [4, 5]. However, recent studies using cone beam computed tomography (CBCT) have reported a high prevalence of round-shaped condyles in a Caucasian young adult and adolescent population. Also flattened and spiked forms have been found with less frequency [6]. New information about the condylar morphology and its three-dimensional (3D) relationships with the temporal head in different populations are continuously enriching the literature thanks to the increasingly precise and sophisticated volumetric reconstructions from CBCT recordings [7]. Also, some authors found that condyles with reduced dimensions, both in the mediolateral and anteroposterior directions, have greater propensity for disc displacement due to a possible insufficient anatomo-functional stability of the disc, which ends up

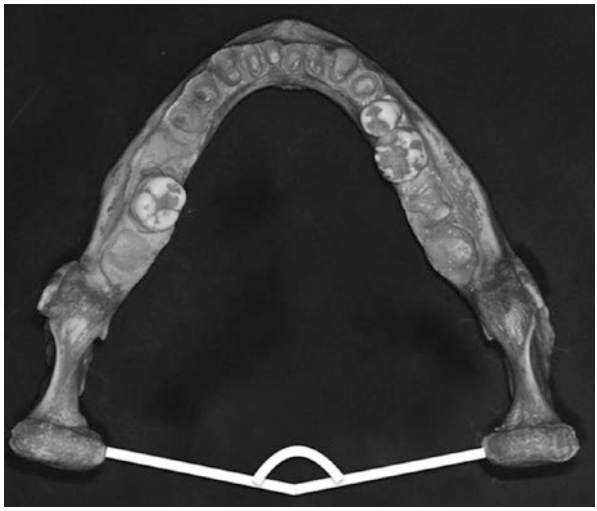

Fig. 4.1 Angle formed by the major axes of the right and left mandibular condyles

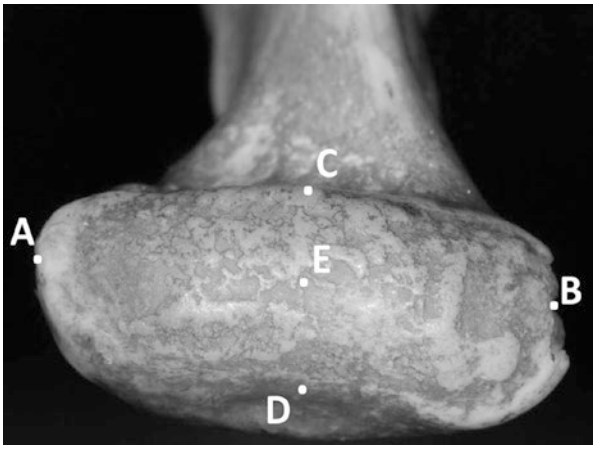

Fig. 4.2 Landmarks used for condyle identification: (**a**) and (**b**) correspond, respectively, to the most medial and to the most lateral point on the articular surface of the mandibular condyle; (**c**) and (**d**) correspond, respectively, to the most anterior and to the most posterior point on the articular surface of the mandibular condyle. The major (mediolateral) and minor (anteroposterior) axes are traced together with their intersection, i.e., the midpoint of the mediolateral and anteroposterior curvatures (**e**)

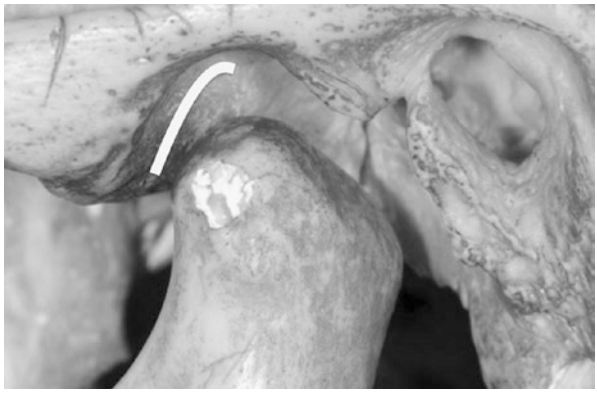

Fig. 4.3 Position of the condyle into the articular fossa and eminence plane angulation (white line) illustrating a S-shaped condylar path

becoming displaced more commonly in the anterior direction, favored by the relationship between the articular components and articular mechanics [8].

The articular surface of the condyle consists of a fibrocartilage covering its anterosuperior portion which deals with the articular eminence of the temporal bone by the interposition of a biconcave disc or meniscus, and thus the extension of the cartilage reflects those parts of the temporomandibular joint that are subject to the highest load (Fig. 4.3).

Under the surface, the head of the condyle of growing subjects harbors a primary intra-articular compensatory growth center that allows frictionless sliding and evenly transmits compressive forces to the subchondral bone. This nucleus is

composed of fibrous cartilage with isogenous groups of chondrocytes randomly distributed in all directions, unlike the metaphyseal cartilage of long bones that have a columnar organization to sustain compressive loads during growth. The proliferative chondrogenic layer persists until the end of the ossification process and then is gradually replaced by a fibrous layer rich in chondrocytes that provide elasticity and resilience to allow adaptive processes to withstand mechanical stress.

In adults despite the drastic reduction in activity, the proliferative layer of condylar head may respond to the compressive loads by differentiation of mesenchymal cells into fibroblasts or chondrocytes [9]. The fibrocartilage thickens by interstitial growth under low loads and thins under higher-pressure forces. These structural changes in the tissues interposed between the articular fibrous layer surface and the bone determine shape modifications in the condylar head called "deviations in form." For example, in case of posterior displacement of the head of the condyle, there is a flattening of the posterior surface of the head associated with volumetric expansion of the front surface. Even in the elderly where most of the cartilage layer beneath the fibrous tissue undergoes a calcification process, the head of the condyle may react to the load because of the presence of residues of secondary cartilage [10].

4.3 Temporal Articular Surface

The articular surface of the temporal bone has a saddle morphology since it is composed of a concavity named "glenoid fossa" which continues with a convex angulated "eminence plane" (Fig. 4.3). The articular fossa represents the anterior portion of the mandibular fossa and belongs to the horizontal exocranial face of the squamous portion which corresponds to the floor of the middle cranial fossa. It is a very thin bony layer that can be easily eroded in case of a middle ear disease with a rapid spread into the neurocranium [11]. The thickness of the roof of the glenoid fossa (RGF) was found to be unaffected by the coronal condyle head morphology and the number of remaining teeth while altered by osteoarthritic process [12]. In addition, no significant gender differences were reported for the thickness of the RGF as well as for condylar length, even if condylar volume, width, height, and the joint spaces were significantly greater among males [13].

The posterior limit of the fossa is the tympanosquamous suture, while anteriorly it is in continuity with the transverse root of the zygomatic process of temporal bone forming the articular eminence continuous with the preglenoid plane. Some individuals have also a rounded conic preglenoid tubercle on the anterior portion of articular fossa at the anterior extent of the capsule and articular tubercle. It is considered as the insertion point of the short tendinous bunches of the superior head of the lateral pterygoid muscle, but its functional role is not yet clear and may be linked to the specific diet or masticatory activity of a given population, as proposed by a group of anthropologists [14]. On the medial aspect, the temporal articular surface presents an entoglenoid process close to the infratemporal surface of the sphenoid spine.

4.3.1 The Eminence Plane

The articular eminence is an angulated plane extending from the most superior point to the most inferior point of the glenoid fossa with a mean inclination of a 45° to the horizontal plane in the adult life. The slope is almost flat in newborns and increases dynamically with age by loading stimulation until the completion of deciduous dentition, attaining approximately the half of the adult angle value at 2 years of age and the full inclination by the age of 30 years [15, 16]. Further changes occur by aging under the influence of the articular loads, so that old subjects have a thin and nearly horizontal eminence plane, thus being more flexible and prone to dislocate than that of younger adults [17–19]. This remodeling pattern is highly variable between individuals and genders (higher in men than in women over 60 years old) being genetically determined but dramatically altered by the remodeling related to functional modifications such as the tooth loss and the consequent alveolar atrophy [20, 21]. Similar changes may be observed in all the TMJ components due to the continuous remodeling throughout life, so that in the elderly the morphology tends to come back to the child appearance. For example, in newborns the articular tubercle is absent, the tympanic bone is incomplete, the shape of condyle is almost round and smaller compared to glenoid fossa, the condylar neck is less shrunken compared to the condylar process, and the condylar axis inclination is smaller than in adults, thus predisposing children to intracapsular fracture of the condyle when a serious trauma transfers to the condyle [5].

The temporal articular surface is coated by fibrocartilage extending only from the crest of the roof of the fossa, down and across the articular eminence. This tissue is deformable during loading and has a reactivity similar to that of the periosteum. Therefore, the surface may respond to higher-compressive forces with large movements of the skeletal components compared to the hyaline cartilage that covers the other diarthrodial joints of the human body [22]. The layer of the cortical bone that covers the articular eminence has been reported to be much thicker than the relevant condylar one. In fact, while the glenoid fossa has a delicate roof, the eminence is rather thick because the biomechanical stresses applied to the condyle are directed obliquely upward and forward during the physiological function. During masticatory movements, the condyle disc complex slides over the eminence according to the slope and to the guiding planes of the teeth. The chewing surfaces of the teeth are crucial codeterminants of some joint shapes and movements, thus conferring additional unique features to the TMJ. Since the flatness or steepness of the articular eminence dictates the path of condylar movements and the degree of rotation of the disc over the condyle, a steep eminence plane has been mentioned as a predisposing factor to TMJ dysfunction, but to date no scientific evidence exists of a relationship between the inclination of articular eminence and the risk to develop a condylar-discal incoordination [16, 23].

It has also been hypothesized that the articular eminence ideally develops to minimize joint loads. In the study by Iwasaki et al. [24], a larger range of steepness values and a higher prevalence of asymmetry of the eminence plane were found in subjects with disc displacement than in those with normally positioned disc. The

analysis of the condylar path shape with minimum contact between TMJ structures during jaw opening revealed that in the first 3 mm of condylar protrusion, joint load minimization was optimized. In contrast, in cases where a condylar protrusion larger than 3 mm was required to produce bite force, eminence shapes were expected to be inconsistent with joint load minimization. Thus, in certain individuals joint load combined with stress concentration may lead to fatigue in the fibrous connective tissue of the joint [24].

According to the mathematical model of the human masticatory system by Tuijt et al. [25], larger joint reactions might be predicted during unloaded mouth opening compared to unloaded jaw closing due to a more cranial condylar path along the temporal surface in the mandibular protrusion. Unfortunately, the biomechanical models obviously differ from natural motion, and an actual assessment of condylar movements is still lacking so that only hypotheses about joint mechanical behavior can be raised. However, the current technology with the imaging systems and three-dimensional motion analyzers provide mathematical and geometrical calculations useful as necessary background to formulate hypotheses [24–29].

4.4 Condyle-Fossa Spatial Relationships

The physiological 3D position of the condyle in the articular fossa is still a matter of intense debate among international researchers. Most studies in the 1960s reported a posterior position of the condyle versus the glenoid fossa, while the publications of the following two decades located the condyle in the center of the fossa in asymptomatic subjects, i.e., geometric centricity. A published literature review concluded that normal condyle-fossa position at the closed intercuspal position is approximately concentric, but there is a lack of agreement because both normal and disc derangements have a wide distribution range and thus would fail in univariate prediction value [30]. With the increased application of imaging techniques such as magnetic resonance imaging and CBCT, it emerged the evidence that there is more than one anatomic model that is compatible with normal function and more than one for disc displacement with and without reduction. The contribution of a variable may be bidirectional and splits samples into subsets. Therefore, no ideal position of the condyle in its glenoid fossa does exist, but a range of "normal" positions in harmony with the neuromuscular complex nonconcentric condyle positions can be compatible with normal function although a prospective outcome study has not been done [31]. There is a predictive value in the osseous anatomic and orthopedic organization of the TM joint for normal versus disc derangement sub-diagnoses and between sub-diagnoses. This is manifested as significant interactions between condyle-fossa position, fossa shape and size variation, and ratios of joint space. However, multifactor analysis and modeling are required for statistical significance and to understand and build clinically recognizable and potentially usable models. The basic biological tenet of a relationship between shape and function is therefore upheld, but the prediction value should not be overstated [31].

4.5 Joint Capsule

The articular capsule envelops completely the mandibular condyle by inserting at the inferior limit of its neck, but its contour is well recognizable only laterally where it is reinforced by the temporomandibular ligament, since the other walls are in continuity with the disc attachments on the temporal and condylar surfaces. In addition, the lateral wall of the joint capsule participates in the superficial fascial lining of the masseter and temporal regions, thus involving the proprioception sensitivity from two muscles neuromuscular spindles in the neural control of the position of the skeletal heads [4].

The capsule is attached medially to the sphenoid spine, laterally to the longitudinal root of the zygomatic process, ventrally to the front edge of the articular tubercle where it seems to continue with the lateral pterygoid muscle, and dorsally to the front edge of the petrosquamous suture (in some patients, a postglenoid tubercle has also been observed giving attachment to the superior retrodiscal lamina) [18]. The tympanosquamous fissure has two parts: (1) laterally, it is an undivided suture between the tympanic and squamous parts of the temporal bone; (2) medially, it is divided by the lower extension of the tegmen tympani, which belongs to the petrous portion of the temporal bone and represents the roof of the tympanic cavity, in an anterior petrosquamous fissure and in a posterior petrotympanic Glaserian fissure [32].

The joint capsule resembles a wide sleeve providing the condyle with large moving space and thus forming villi and folds all around and particularly in the posterior portion, which corresponds to the bilaminar zone of the articular disc. All structures arranged in the retroarticular region are extracapsular and are filled with loose connective tissue through which the anterior tympanic artery and its branches run, as detailed in the following paragraphs concerning the disc [33].

4.5.1 The Synovial Membrane

From a histologic point of view, the TMJ capsule consists of the outer fibrous covering composed of collagen and elastic fibers and the inner synovial membrane, which produces synovial fluid. The synovia consists of a superficial layer rich in synoviocytes and a deeper layer containing vessels, fibroblasts, macrophages, and elastic fibers. The synoviocytes produce synovial fluid necessary for the normal lubrication of the joint heads during the movement and for the tissue trophism, acting as a double-acting pump system in the superior and inferior compartments of the joint. The synovial fluid is a viscous complex composition of hyaluronic acid and the glycoprotein lubricin, able to stream freely along the disc, and the effect of the pump system is reduced in order to allow the joint to work as a "free flying," as well as a force transmitting articulation, especially in statics [34]. The deep connective tissue layer adjusts the volume and composition of the synovial fluid and keeps the negative pressure into the articular cavity, aiming at withstanding high mechanical

loads and therefore preserving the integrity of joint tissue. Differences in the structure of the synovial membrane have been reported in the various areas of normal subjects in relation to mechanical loads so that three main types of synoviae—areolar, fibrous, and mixed—were described. In the upper joint compartment, the synovial membrane is predominantly made of areolar connective tissue rich in collagen and elastic fibers. Indeed, adaptive processes to load alterations may modify the molecular and histological features of the synovia with a transition toward a fibrotic aspect in the retrodiscal portion in patients with disc displacement [35].

The synovial membrane contains some nociceptors for capsaicin and free endings associated with slowly conductive fibers, while the external capsular layer presents many Ruffini-type mechanoreceptors with a low threshold and high threshold and several Pacinian corpuscles. Also, Golgi tendon organs are located in the proximity of the insertion of the temporomandibular ligament so that an anesthesia of the lateral wall of the capsule may determine a 10% increment of mouth opening [36].

4.6 Ligaments

4.6.1 The Temporomandibular Ligament

The temporomandibular ligament is the only functional ligament of the TMJ that limits the physiological range of the condylar excursions. It consists of a superficial bundle that is obliquely angulated (with about 70° inclination to the horizontal plane) from the articular tubercle to the posterior face of condylar neck and of a deep beam that is directed horizontally to the lateral pole of the condyle. It thus restrains the retrusive and downward movements of the mandibular condyle beyond the articular tubercle, and its laxity may predispose to meniscal displacement [37].

Several anatomical and histological variants concerning the thickness and inclination of the ligamentous bundles were reported. In particular the deep component seems to be well recognizable only in some individuals, while in others it represents a simple reinforcement of the joint capsule that tends to ensure the mandible a security block posture. This joint position has been proposed by dentists as a reference position for recording posture of alveolar arches in edentulous patients and is known as “centric relation.” The centric relation can be obtained by a dental clinic maneuver in those subjects that have a well recognizable horizontal ligament, and in these cases it may help the clinicians in the treatment planning. However, it has to be unlighted that this position, when recordable, is a not physiological position [38].

4.6.2 Other Ligaments

Additional ligaments medially run past the capsule in the proximity of the articular components in order to inhibit the protrusive movement of the mandible but without playing a significant role in masticatory movements: the stylomandibular and the

sphenomandibular ligaments [37]. The stylomandibular ligament connects the styloid process to the mandibular angle fusing with the thickening of the superficial cervical fascia that envelops the medial pterygoid muscle. It represents an important landmark for the external carotid artery exposure in the retromandibular space during maxillofacial surgery.

The sphenomandibular ligament runs from the spine of the sphenoid to the Spix spine close to the mandibular foramen. Also this ligament serves as an anatomic landmark separating the internal maxillary artery with its inferior alveolar branch (which passes between the ligament and the medial surface of the mandibular ramus) from the inferior alveolar nerve which remains medial. The inferior alveolar artery enters vertically the mandibular foramen, while the inferior alveolar nerve enters obliquely in a medial-lateral direction. Therefore, a possible risk of damaging the inferior alveolar artery may occur during anesthesia of inferior alveolar nerve. The sphenomandibular ligament may influence the spread of anesthetic liquid. The sphenomandibular ligament originates from the perichondrium of Meckel's cartilage and continues into the anterior mallear ligament (AML) that crosses the petrotympanic fissure in Hugier's canal to reach the malleus. The AML does not insert on the disc, thus representing an independent entity from the discomallear ligament (Pinto ligament—DML) which is a capsular structure belonging to the retrodiscal upper lamina. The DML is the remnant tendon of the primitive lateral pterygoid muscle on the primitive quadrato-articular joint, and it forms by the convergence of fibroelastic bundles from the superoposterior and medial edge of the articular disc to the malleus after passing through the petrotympanic fissure [32]. Both the AML and DML should be considered as intrinsic retrodiscal ligaments of the TMJ with no (apparent) important function.

4.6.3 TMJ-Tympanic Cavity Relationships

In the past, the presence of such ligaments raised the question of a close functional relationship between TMJ and structures of the middle ear, possibly causing otologic symptoms as tinnitus or plugged ear in patients with temporomandibular dysfunctions (TMD). However, experimental functional tests and histological evidences showed that no movement of the disc was seen when overstretching the AML which is totally composed of collagen fibers. In contrast, the abundant elastic fibers found in the DML may be responsible for compensation of strain forces on this ligament and may reflect minor forces to the mallear head. Anterior disc displacement is a condition caused by a disunity of the disc-condyle complex and not by an altered tension of the DML which tends only to limit jaw movements (maximum opening and protrusion). A physiologic mallear movement is possible in newborns where the Glaserian fissure is widely open. The fissure undergoes progressive closure in the first years of life and leaves the Hugier's canal for the passage of the chorda tympani nerve, the tympanic artery, and the two abovementioned ligaments. In fetuses and newborns, movements of the articular disc are accompanied by movements of the chain of the auditory ossicles until closure of the petrotympanic fissure [39].

The explanation of the otologic disturbances in TMD patients is therefore of other origin, i.e., neurological as a consequence of trigeminal nerve irritation. It seems to be related to the spasm of the tensor tympani muscle after peripheral lesions of the masticatory muscles or after compression of the auriculotemporal, masseter, and posterior deep temporal nerves in case of disc displacement and capsule deformation. These mechanisms may trigger a trigeminal neuropathy altering the performance of the tensor tympani and palate muscles with dysfunction of the tympanic membrane and the opening of auditory tube [32].

4.7 TMJ Disc

At birth the temporomandibular disc is flat and takes an S shape after the formation of the articular tubercle, thus assuming a biconcave fibrous lamina with a prevalent medial-lateral extension that provides a large load-bearing capacity over the entire joint range of motion. In the anteroposterior direction, it is composed of three portions: an expanded anteroinferior part of about 2 mm, a 3-mm-thick posterosuperior tract, and a thin central portion of about 1 mm—called intermediate zone—which is subjected to functional loads (Fig. 4.4). The anterior and posterior bands of the disc consist of a 3D network of collagen fibers, more dense and robust in the posterior one, while the intermediate zone is characterized by a well-defined anteroposterior alignment of the collagen bundles reflecting the major orientation of forces distribution. Indeed, high compressive loads seem to alter the behavior of the disc, which responds to the tensional forces with changes in the alignment of the collagen fibers. The disc structure presents the additional content of some elastic fibers, essential for the shape and position of the disc once stopped loading, so the content of elastin is an indicator of regions of the disc more prone to stretching and to morphological recovery during mandibular movements. This peculiar texture gives the disc elevated viscoelastic properties responsible for its morphologic deformation and thickness variation according to the circadian rhythms of vascularization and water content and to the rate of shock absorbance. Hence, the relationship between condyle and glenoid fossa is dynamically variable and different from individual to individual [37].

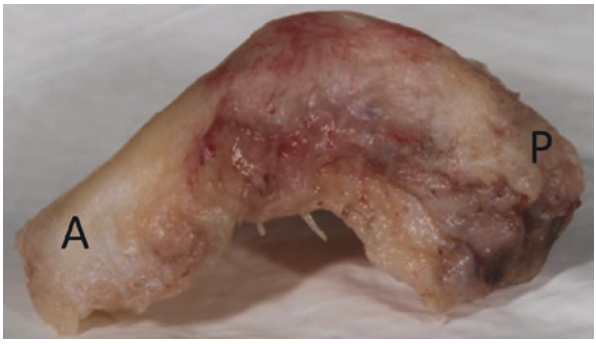

Fig. 4.4 Lateral view of the temporomandibular disc (A, anterior; P, posterior)

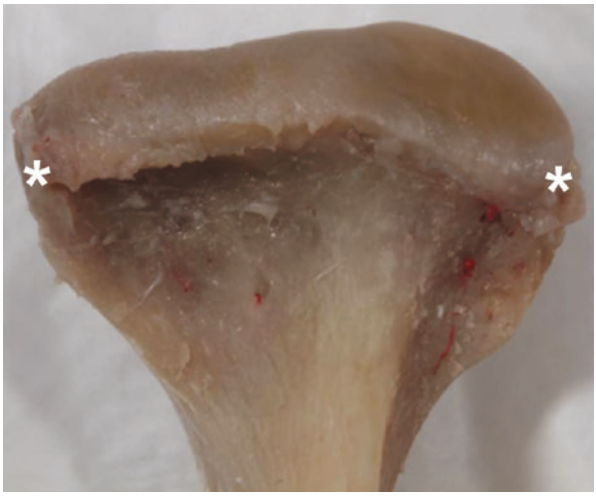

Fig. 4.5 Landmarks on the medial and lateral poles of the condyle for the attachment of the condylar-disc ligaments

The disc adheres to the articular capsule in the anterior aspect, medially and laterally, in order to completely divide the joint into a superior and an inferior compartment. On the anterior border, the tendon ends of the lateral pterygoid muscle medially radiate to the disc so that about 10% of the fibers of its superior belly attach to the capsule and continue with the meniscus. However, the extent of this muscular portion in relation to the portion that attaches directly to the condylar neck is highly variable and thus functionally negligible [40].

Also, it attached to the poles of the condyle by medial and lateral condylar-disc ligaments (Fig. 4.5) that represent fibrous connection systems independent from the capsule, thus allowing the condyle to perform rotational movements around the meniscus together with the translation movement of condylar-disc complex along the eminence plane. Therefore, the mandibular head possesses no fixed turning point, and the joint forms a gear unit with the "interincisal joint."

4.7.1 Retrodiscal Space

Posteriorly, the disc passes to a retroarticular "bilaminar zone" consisting of two laminae independent of each other, the stratum superius extending from the disc to the mandibular fossa and reinforced by the discomalleolar ligament and the stratum inferius extending from the articular disc to the mandibular condyle. It was reported that the superior retrodiscal layer is made up of predominant elastic fibers, while the inferior consists partly of elastic and mainly of collagen fibers [41]. The *genu vasculosa* located in the interlaminar space is made up of collagenous connective and fat tissue containing many nerve endings and a retrodiscal venous plexus in continuity with the pterygoid venous plexus without interposition of a posterior capsular wall. Both elastic laminae are physiologically not in tension, and they do not prevent the meniscus to move forward with the condyle.

The retrodiscal space hosts branches of the deep auricular, anterior tympanic, and middle meningeal arteries, temporomandibular veins, and auriculotemporal nerve and acts as a hydropneumatic pad that absorbs mechanical stresses and compensates for volume changes, thus protecting the tympanic wall during mouth closure [37, 41]. The finding of a condylar retrusion with decreased retrodiscal space can be a sign of bone compression of the bilaminar area [33, 42].

The disc does not contain blood vessels and nerves but has numerous receptors such as Pacinian corpuscles, Golgi tendon organs, and muscle-Ruffini endings with maximum density at the periphery and decreasing toward the center of the disc. Also, at the periphery of the disc of rats, TRPV1 nociceptors for capsaicin associated with small arterioles were observed [36]. The coactivation of many Ruffini nerve endings and neuromuscular spindles could provide useful information during mastication in patients wearing implant-retained dental prostheses where periodontal mechanoreceptors are missing.

4.8 Disc-Condyle Spatial Relationships

Physiologically, the posterior band of the disc is located over the head of the condyle, with the disc-condyle complex in the anterior-superior position against the slope of the articular eminence.

The anatomical situations in which the thick posterior paortion of the disc is placed anterior, anteromedial, anterolateral, medial, or lateral to the upper limit of the condylar head have been described as “condylar-disc displacements” [43]. Studies performed with new imaging techniques and standardized protocols (e.g., adjusting the angle of the condyle in the horizontal plane) have reported the anterior condylar-disc displacement as the most frequent displacement followed by the anterolateral displacement both in pathologic patients and in asymptomatic subjects.

A reduction in the space above the condylar head is suggestive of meniscal displacement, while an increase suggests a condylar distraction [44].

4.9 Topographic Anatomy of Temporomandibular Joint

The temporomandibular joint is quite superficial, and it is located in front of the tragus of the ear corresponding to the upper and posterior part of the parotideomasseteric region.

Excluding endoscopic techniques and intraoral, retromandibular, and retroauricular approaches, most of the surgical approaches of the temporomandibular joint like preauricular, endaural, and inverted hockey stick incisions involved this region. Therefore, the skin incision involves also the inferior part of the temporal region [45] (Fig. 4.6).

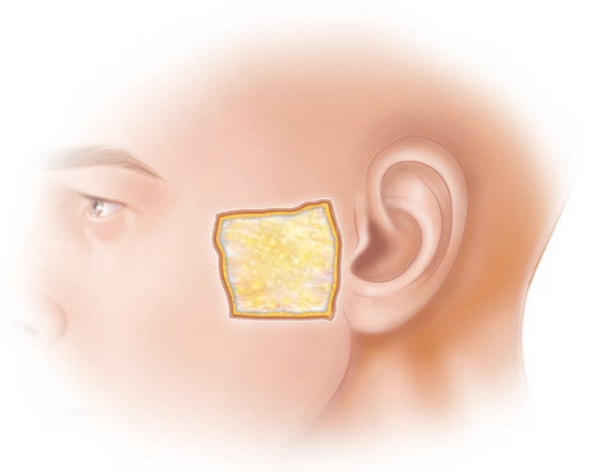

Fig. 4.6 Subcutaneous tissue in TMJ region after removing the skin

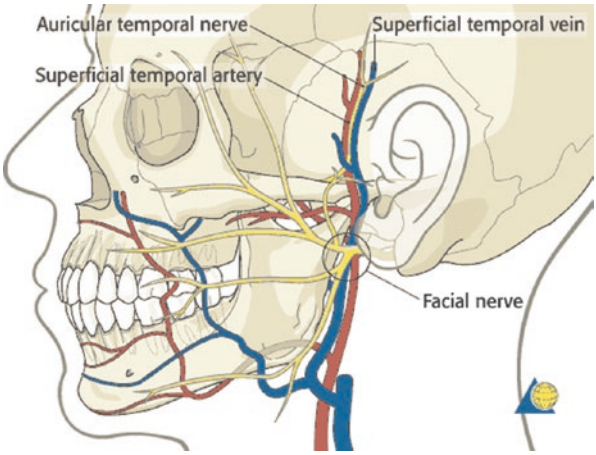

Fig. 4.7 Schematic rappresentation of nerves and vessels in the TMJ region

Some important anatomical structures including superficial temporal vessels, auriculotemporal nerve, parotid gland, and facial nerve must be considered in this dissection (Fig. 4.7).

To dissect the TMJ using the preauricular approach that permits the access to the superior portion of mandibular condylar process, a vertical incision should be performed in front of the external acoustic meatus, crossing the zygomatic arch and extending into the upper part of the parotideo-masseteric region and the lower part of the temporal region.

The dissection involves the following structures: the skin, subcutaneous tissue and temporoparietal fascia, parotid and temporal fasciae, and TMJ capsule.

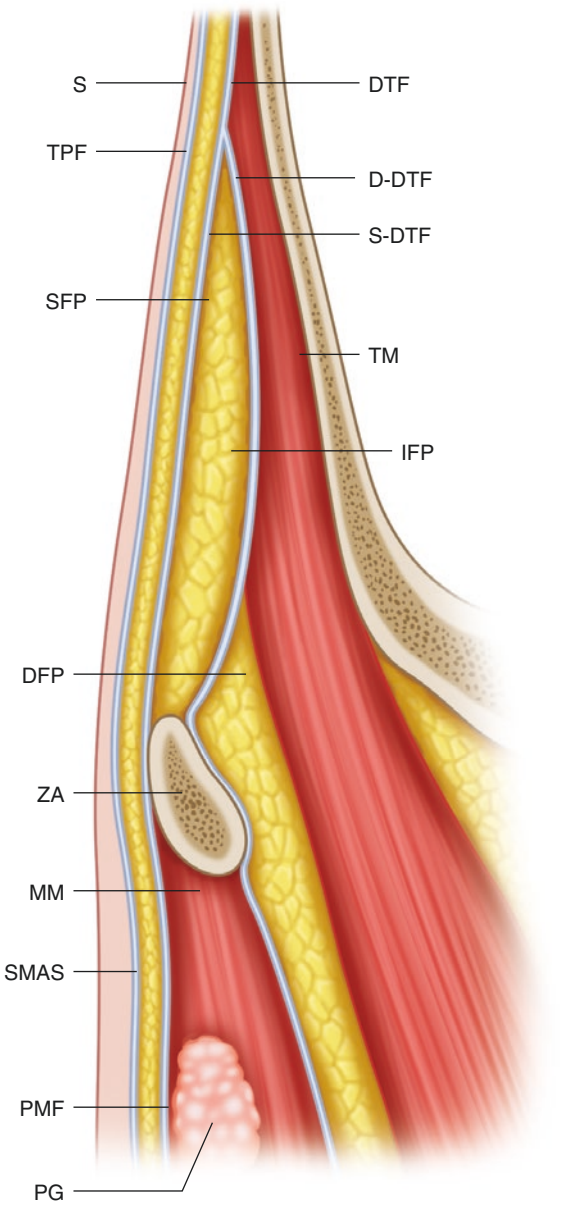

Fig. 4.8 Schematic rappresentation of the connective fasciae in temporal region

The skin is relatively thin and frequently presents with vertical oriented preauricular wrinkles. Very thin fibers of the mandibular branch of the trigeminal nerve innervate the skin.

The subcutis contains a thin layer of adipose tissue and the temporoparietal fascia (Fig. 4.8).

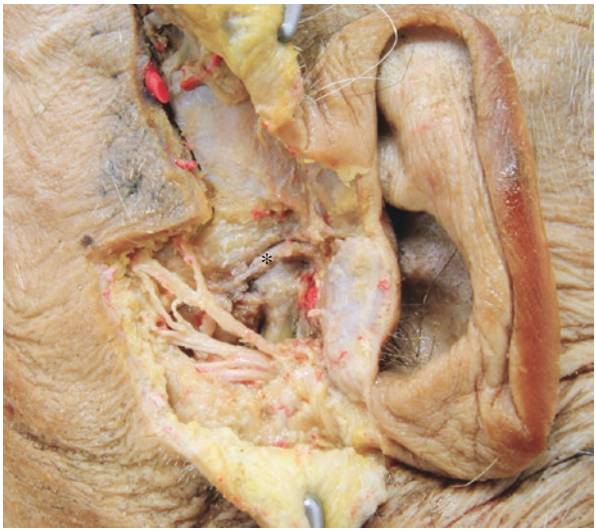

Fig. 4.9 Laterl view of TMJ. The articular disk (*) is well evident between mandibular condile and temporal articular surface

In the upper portion of the dissected area, the superficial temporal vessels and the auriculotemporal nerve may be found.

The fascial organization of this region is very complex, and also the nomenclature of the fasciae in the literature is variable [46].

The temporoparietal fascia is well evident in the temporal region. This fascia has no attachment to the zygomatic arch, and it descends in the parotid region losing its fascial structure and becomes a foamy tissue continuing in the same layer of the superficial musculoaponeurotic system (SMAS).

Deep to the temporoparietal fascia, it is possible to identify the superficial layer of the deep temporal fascia and its continuation that covers the parotid gland and the masseter muscle: the parotideo-masseteric fascia.

At the level of the parotid, we can observe several fibrous connections between the temporoparietal fascia and the parotideo-masseteric fascia without the recognition of a defined dissection plane. Therefore, the SMAS, the parotideo-masseteric fascia, and the superficial layer of deep temporal fascia are in continuity.

Above the zygomatic arch, the superficial layer of the deep temporal fascia covers the intermediate fat pad. The deep layer of the deep temporal fascia separates the intermediate fat pad from temporal muscles. More cranially the superficial and the deep layers of the deep temporal fascia merge (Fig. 4.9).

The superficial temporal artery and vein and the auriculotemporal nerve emerge from the superior aspect of the parotid gland. The vein is more superficial and lies posterior to the artery. The nerve is posterior and superficial to the artery, few millimeters in front to the cartilaginous portion of the external auditory meatus. A dissection very close to this last structure is needed to minimize the damage to the nerve (Figs. 4.10 and 4.11).

Deep to the parotideo-masseteric fascia, the TMJ capsule is partially covered by the parotid gland (Figs. 4.10 and 4.11).

Some important structures are present within the parotid gland:

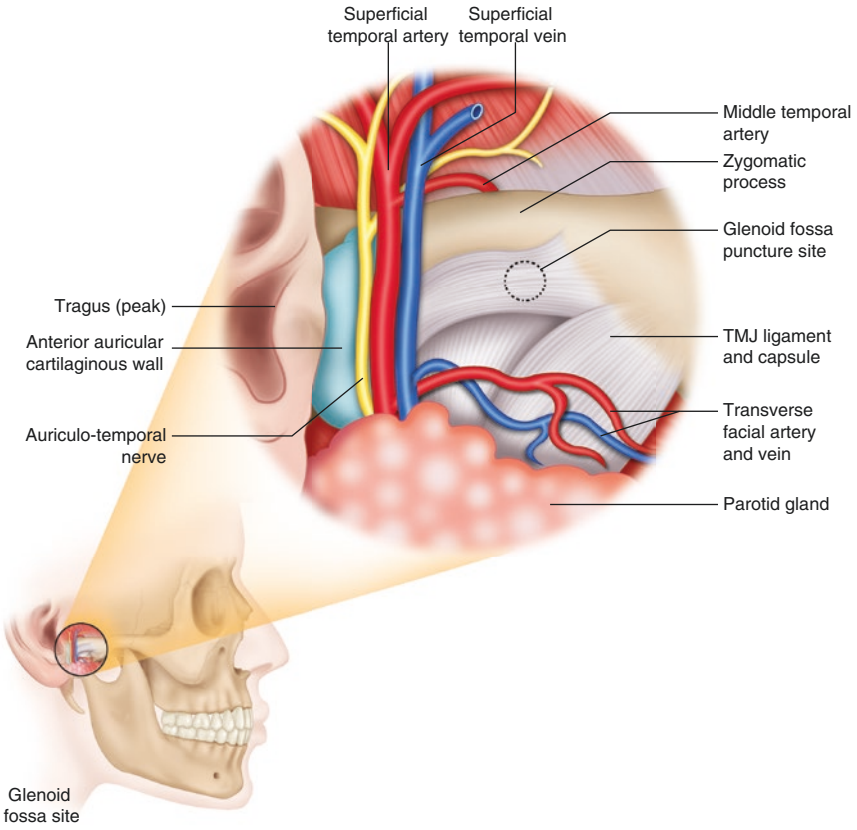

Fig. 4.10 Auriculotemporal nerve and temporal vessels running close the TMJ

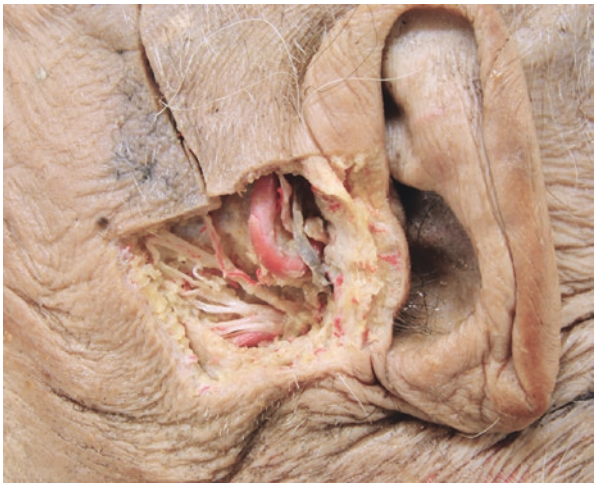

Fig. 4.11 Superficial temporal artery and their ralationships with TMJ

– The facial nerve forms the parotid facial plexus within the gland and partially separates the glandular tissue into superficial and deep portion. The terminal branches of the facial nerve emerge from anterior, superior, and inferior border of the gland.
– The superficial temporal and maxillary veins merge in the glands to form the retromandibular vein.
– The external carotid artery divides into maxillary and superficial temporal arteries into the gland. Transverse facial artery originates from temporary artery within the parotid and runs anteriorly inferior to the zygomatic arch.

Removing a little portion of the parotid gland, it is visible the lateral aspect of the TMJ capsule that receives its vascularization from the superficial temporal artery, frequently evident (Fig. 4.12).

Opening the capsule, the details of the internal organization of the TMJ can be observed (Figs. 4.13 and 4.14).

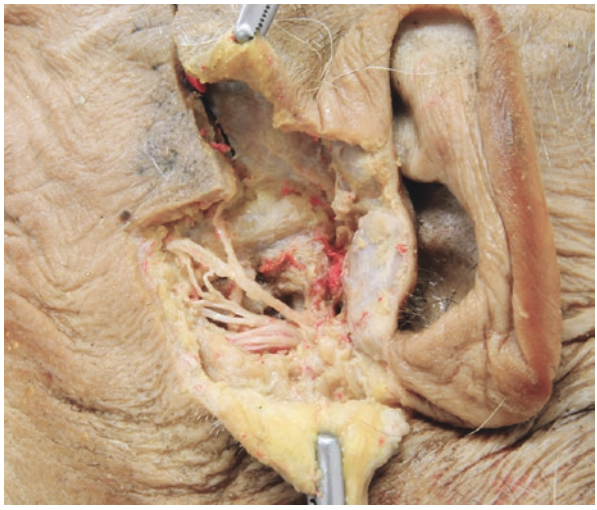

Fig. 4.12 Lateral view of TMJ with intact joint capsule

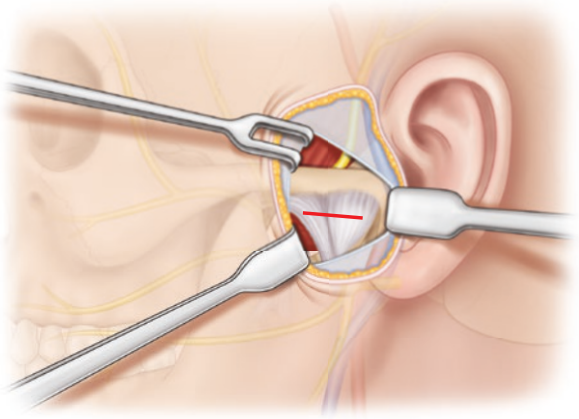

Fig. 4.13 Shematic rappresentation of the lateral view if TMJ with intact joint capsule

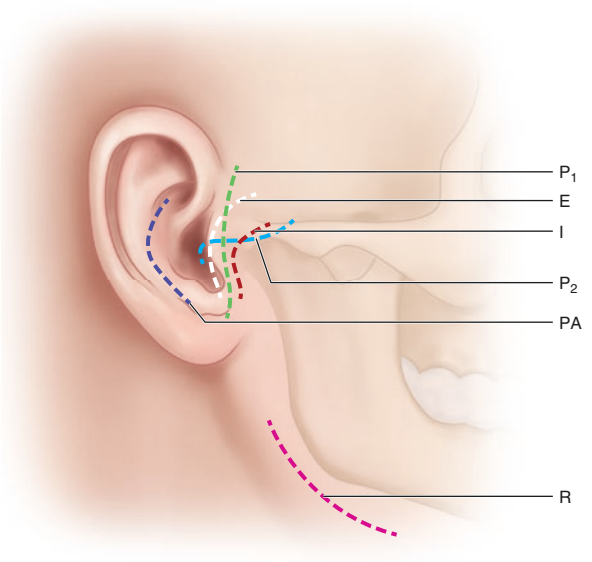

Fig. 4.14 Surgical incision for the access to TMJ

References

1. Stamm T, Hohoff A, Van Meegen A, Meyer U. On the three dimensional physiological position of the temporomandibular joint. J Orofac Orthop. 2004;65:280–9.
2. Koolstra JH. Dynamics of the human masticatory system. Crit Rev Oral Biol Med. 2002;13:366–76.
3. Terhune CE. How effective are geometric morphometric techniques for assessing functional shape variation? An example from the great ape temporomandibular joint. Anat Rec. 2013;296(8):1264–82.
4. Carranza ML, Carda C, Simbron A, Quevedo MC, Celaya G, de Ferraris ME. Morphology of the lateral pterygoid muscle associated to the mandibular condyle in the human prenatal stage. Acta Odontol Latinoam. 2006;19:29–36.
5. Meng F, Liu Y, Hu K, Zhao Y, Kong L, Zhou S. A comparative study of the skeletal morphology of the temporo-mandibular joint of children and adults. J Postgrad Med. 2008;54:191–4.
6. Caruso S, Storti E, Nota A, Ehsani S, Gatto R. Temporomandibular joint anatomy assessed by CBCT images. Biomed Res Int. 2017;2017:2916953.
7. Ikeda R, Oberoi S, Wiley DF, Woodhouse C, Tallman M, Tun WW, McNeill C, Miller AJ, Hatcher D. Novel 3-dimensional analysis to evaluate temporomandibular joint space and shape. Am J Orthod Dentofac Orthop. 2016;149(3):416–28.
8. Torres MG, Crusoé-Rebello IM, Rosário M, Albuquerque MC, Campos PS. Morphometric features of the mandibular condyle and association with disk abnormalities. Oral Surg Oral Med Oral Pathol Oral Radiol. 2016;121(5):566–72.
9. Mao JJ, Nah HD. Growth and development: hereditary and mechanical modulations. Am J Orthod Dentofac Orthop. 2004;125:676–89.
10. Paulsen HU, Thomsen JS, Hougen HP, Mosekilde L. A histomorphometric and scanning electron microscopy study of human condylar cartilage and bone tissue changes in relation to age. Clin Orthod Res. 1999;2:67–78.
11. Ahuja AT, Yuen HY, Wong KT, Yue V, van Hasselt AC. Computed tomography imaging of the temporal bone-normal anatomy. Clin Radiol. 2003;58:681–6.

12. Ejima K, Schulze D, Stippig A, Matsumoto K, Rottke D, Honda K. Relationship between the thickness of the roof of glenoid fossa, condyle morphology and remaining teeth in asymptomatic European patients based on cone beam CT data sets. Dentomaxillofac Radiol. 2013;42(3):90929410.
13. Al-Koshab M, Nambiar P, John J. Assessment of condyle and glenoid fossa morphology using CBCT in South-East Asians. PLoS One. 2015;10(3):e0121682.
14. Zeitoun V. High occurrence of a basicranial feature in Homo erectus: anatomical description of the preglenoid tubercle. Anat Rec B New Anat. 2003;274:148–56.
15. Baqaien MA, Al-Salti FM, Muessig D. Changes in condylar path inclination during maximum protrusion between the ages of 6 and 12 years. J Oral Rehabil. 2007;34:27–33.
16. Katsavrias EG. Changes in articular eminence inclination during the craniofacial growth period. Angle Orthod. 2002;72:258–64.
17. Csadò K, Martòn K, Kivovics P. Anatomical changes in the structure of the temporomandibular joint caused by complete edentulousness. Gerodontology. 2012;29:111–6.
18. Katsavrias EG, Dibbets JMH. The postglenoid tubercle: prevalence and growth. Ann Anat. 2002;184(2):185–8.
19. Reicheneder C, Gedrange T, Baumert U, Faltermeier A, Proff P. Variations in the inclination of the condylar path in children and adults. Angle Orthod. 2009;79:958–63.
20. Raustia AM, Pirttiniemi P, Salonen MA, Pyhtinen J. Effect of edentulousness on mandibular size and condyle-fossa position. J Oral Rehabil. 1998;25:174–9.
21. Sa SC, Melo SL, Melo DP, Freitas DQ, Campos PS. Relationship between articular eminence inclination and alterations of the mandibular condyle: a CBCT study. Braz Oral Res. 2017;31:e25.
22. Herring SW, Decker JD, Liu ZJ, Ma T. Temporomandibular joint in miniature pigs: anatomy, cell replication, and relation to loading. Anat Rec. 2002;266:152–66.
23. Sato S, Kawamura H, Motegi K, Takahashi K. Morphology of the mandibular fossa and the articular eminence in temporomandibular joints with anterior disk displacement. Int J Oral Maxillofac Surg. 1996;25:236–8.
24. Iwasaki LR, Crosby MJ, Marx DB, Gonzalez Y, McCall WD Jr, Ohrbach R, et al. Human temporomandibular joint eminence shape and load minimization. J Dent Res. 2010;89:722–7.
25. Tuijt M, Koolstra JH, Lobbezoo F, Naeije M. Differences in loading of the temporomandibular joint during opening and closing of the jaw. J Biomech. 2010;43:1048–54.
26. Curtis N. Craniofacial biomechanics: an overview of recent multibody modelling studies. J Anat. 2011;218(1):16–25.
27. Mapelli A, Galante D, Lovecchio N, Sforza C, Ferrario VF. Translation and rotation movements of the mandible during mouth opening and closing. Clin Anat. 2009;22:311–8.
28. Seemann R, Czerny C, Schicho K, Undt G, Piehslinger E, Ewers R, et al. Pseudodynamic MRI differs from natural opening of the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;105:371–8.
29. Sforza C, Tartaglia GM, Lovecchio N, Ugolini A, Monteverdi R, Giannì AB, et al. Mandibular movements at maximum mouth opening and EMG activity of masticatory and neck muscles in patients rehabilitated after a mandibular condyle fracture. J Craniomaxillofac Surg. 2009;37:327–33.
30. Gonzalez B. The not-so-controversial issue of condylar position. Int J Orthod Milwaukee. 2007;18:17–26.
31. Pullinger A. Establishing better biological models to understand occlusion. I: TM joint anatomic relationships. J Oral Rehabil. 2013;40(4):296–318.
32. Cheynet F, Guyot L, Richard O, Layoun W, Gola R. Discomallear and malleomandibular ligaments: anatomical study and clinical applications. Surg Radiol Anat. 2003;25:152–7.
33. Mérida-Velasco JR, Rodríguez JF, de la Cuadra C, Peces MD, Mérida JA, Sánchez I. The posterior segment of the temporomandibular joint capsule and its anatomic relationship. J Oral Maxillofac Surg. 2007;65:30–3.
34. Kubein-Meesenburg D, Nägerl H, Fialka-Fricke J, Hahn W, Weber S, Hönig J, Hansen C, Fanghänel J, Thieme KM, Ihlow D. Functional states of mandibular movements and synovial

pumps of the temporomandibular joint. Is it possible to provide a biomechanically correct replacement for the TMJ? Ann Anat. 2012;194(2):200–7.
35. Israel HA, Langevin CJ, Singer MD, Behrman DA. The relationship between temporomandibular joint synovitis and adhesions: pathogenic mechanisms and clinical implications for surgical management. J Oral Maxillofac Surg. 2006;64:1066–74.
36. Ioi H, Kido MA, Zhang JQ, Yamaza T, Nakata S, Nakasima A, Tanaka T. Capsaicin receptor expression in the rat temporomandibular joint. Cell Tissue Res. 2006;325:47–54.
37. Fanghänel J, Gedrange T. On the development, morphology and function of the temporomandibular joint in the light of the orofacial system. Ann Anat. 2007;189(4):314–9.
38. Rinchuse DJ, Kandasamy S. Centric relation: a historical and contemporary orthodontic perspective. J Am Dent Assoc. 2006;137:494–501.
39. Sencimen M, Yalçin B, Doğan N, Varol A, Okçu KM, Ozan H, et al. Anatomical and functional aspects of ligaments between the malleus and the temporomandibular joint. Int J Oral Maxillofac Surg. 2008;37(10):943–7.
40. Koolstra JH, Tanaka E. Tensile stress patterns predicted in the articular disc of the human temporomandibular joint. J Anat. 2009;215:411–6.
41. Morales H, Cornelius R. Imaging approach to temporomandibular joint disorders. Clin Neuroradiol. 2016;26:5–22.
42. Siéssere S, Vitti M, Semprini M, Regalo SC, Iyomasa MM, Dias FJ, et al. Macroscopic and microscopic aspects of the temporomandibular joint related to its clinical implication. Micron. 2008;39:852–8.
43. Rao VM, Bacelar MT. MR imaging of the temporomandibular joint. Neuroimaging Clin N Am. 2004;14:761–75.
44. Haiter-Neto F, Hollender L, Barclay P, Maravilla KR. Disk position and the bilaminar zone of the temporomandibular joint in asymptomatic young individuals by magnetic resonance imaging. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;94:372–8.
45. Kempers KG, Quinn PD, Silvestein K. Surgical approach to mandibular condylar fractures: a review. J Craniomaxillofac Trauma. 1999;5:25–30.
46. Babakurban ST, Cakmak O, Kendir S, Elhan A, Quatela VC. Temporal branch of the facial nerve and its relationship to fascial layers. Arch Facial Plast Surg. 2010;1:16–23.

Functional Anatomy and Biomechanics of the Temporomandibular Joint

5

L. M. Gallo and V. Colombo

Abstract

Evolving from pure anatomy, this chapter focuses on TMJ motor function and dysfunction. Functional anatomy and its morphological structures are the limits where the mandibular movements and its central and peripheral neurological inputs can govern TMJ motion.

- The masticatory system as a functional unit
- Definition of mandibular positions and limitations of its analysis
- Descriptions of mandibular dynamics in function and dysfunction
- TMJ static and dynamic loading

5.1 The Masticatory System as a Functional Unit

The healthy and intact masticatory system consists of a complex biological mechanism controlled both consciously by the central nervous system (CNS) and unconsciously by the brain stem. Among the functions of the masticatory system are not only *food processing and intake* (chewing and swallowing) but also *phonation* and—partially—facial *expression* (mimicry). These functions are based on neural control of the masticatory muscles for force development and movement production. Humans ingest food and communicate verbally and nonverbally with the environment by dynamically adjusting tissues and parts of the masticatory system, airflow, and forces of the jaw adductors. A dysfunction of the masticatory system not only affects fundamental biological aspects of life but also psychological well-being and social communication (*biopsychosocial model*).

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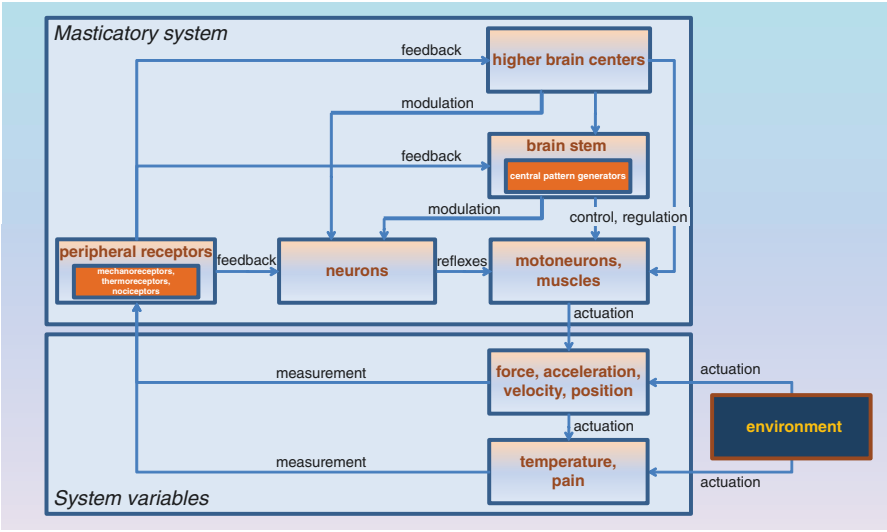

Fig. 5.1 The masticatory system as a functional unit. *Higher brain centers* as well as the *brain stem* with *central pattern generators* (CPGs) activate via *motoneurons*, the musculature of the masticatory system. *Muscles* act on the mandible and develop *forces* and other related mechanical variables (*accelerations*, *velocities*, *positions*) which are measured by mechanoreceptors and fed back to higher brain centers or the brain stem. Further stimuli of mechanical, thermal, or noxious nature from the *environment* are also sensed by *peripheral receptors* and fed back. This information is processed for system control and can also trigger *reflexes* over shorter or longer pathways

Muscles of the masticatory systems develop forces that influence other mechanical variables such as acceleration, velocity, and position of the mandible. The complex activation and interaction of the system components is achieved automatically by means of specific neural circuits in the brain stem (*central pattern generators*, CPG) that are clocked by biological oscillators [1–5]. Thus, masticatory muscles mechanically load structures of the mandible and of its counterpart, the maxilla. Other mechanical, thermal, and noxious stimuli originating from the environment including food mechanical resistance can further affect the masticatory system, which reacts accordingly. In order to do so, it relies on biological sensors that return the state of its components via neural paths (*feedback*) (Fig. 5.1). These sensors consist of receptors, which respond to mechanical (*mechanoreceptors*) but also to thermal (*thermoreceptors*) as well as pain stimuli (*nociceptors*). Information from peripheral receptors is directed to higher brain centers, brain stem, and reflex routes via different pathways [6].

Biological systems that support vital functions must guarantee error robustness, so that they can remain as functional as possible throughout lifetime. This can be achieved by failure compensation in the form of *redundancy* (e.g., the presence of multiple components). This is provided not so much by paired structures but rather by the multiple presences of receptors and other subordinate units. Feedback from peripheral receptors serves to recognize the actual state of the individual system parts (e.g., mandibular position or bite forces) and to finely adjust them (*regulation*). This is advantageous over pure *control*—in which neural impulses send information

blindly to the musculature (*feed-forward*)—but entails a *time delay* and possibly a slowdown. This is the case, e.g., when a system variable such as bite force needs to be held constant over a period of time in spite of external mechanical influences (e.g., food resistance).

System variables are not only measured internally by peripheral receptors in order to provide masticatory function but to a lesser extent can also be the object of biomechanical experimental analysis. Historically, first biomechanical analyses were performed by prosthodontists interested in restoring occlusion by empirical static and dynamic simulations of mandibular movement: articulators have been developed for a realistic yet simplified and averaged mechanical replication of mandibular function. However, researchers from a much wider range of disciplines are currently interested in mandibular biomechanics. With the advancement of imaging and measurement techniques as well as computer science, it is now possible to analyze mandibular biomechanics statically and dynamically in three dimensions with high precision. A general important finding is the great intra- and interindividual variability of human data. This variability is strongly influenced by the interaction between craniomandibular morphology and function [7], whereby these change continuously in the course of life.

5.2 Statics

The most common position of the mandible is the relaxed rest position without tooth contact (*mandibular postural position* or "physiologic rest position") [8]. In this position, the tone of jaw adductors (masseter, temporal, and medial pterygoid) and jaw abductors (suprahyoid muscles) is balanced. The thick posterior disc band is located on the cranial aspects of the condylar head; the anterior portion of the condylar head lies against the intermediate zone and the anterior band of the disc as well as the dorsal slope of the articular tubercle; and the bilaminar zone is cranially relaxed and caudally taut. In this situation, the condylar position is inter- and intraindividually indeterminate, since it mainly depends on the variable muscle tone and can be made more caudal by relaxation exercises through biofeedback [9].

Because of this imprecision, all mandibular movements examined in scientific investigations start from the position of the mandible in which cusps and sulci of maxillary and mandibular teeth are in greatest contact and the mandible is in its most closed position (*intercuspal position*, ICP). It is to be noted that in this initial position, the condylar position is codetermined by the nature of the dentition and is not necessarily coincident with the position achieved with dorsally directed mandibular manipulation (*retruded contact position*, RCP). These and other boundary positions are often shown in the so-called Posselt diagram (its sagittal section appears in Fig. 5.2) [10]: further boundary positions are *maximum protrusion* (MP) (when mandibular incisors slide under their maxillary counterparts ending most anteriorly), *retruded opening* (R) (reached by means of a retruded hinge movement centered in the condyles), as well as *maximum opening* (MO), which is not determined by rigid anatomic structures. This is also the case—as said before—for the *mandibular postural position* (X). It is important to note, however, that this scheme does not fully take into account individual anatomical diversity, tissue structure, and

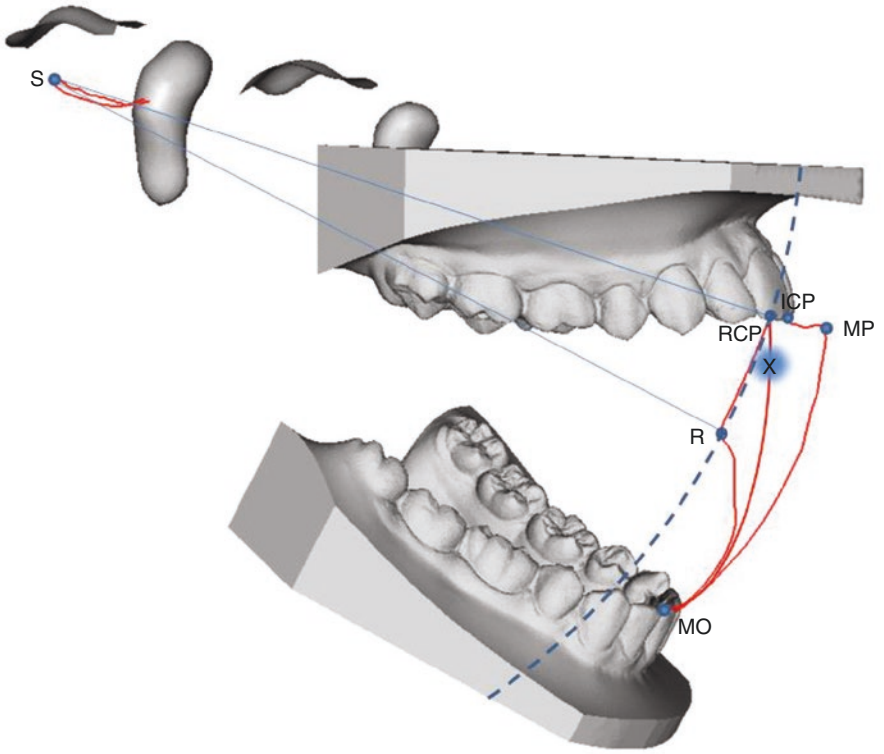

Fig. 5.2 Sagittal section of Posselt diagram. *RCP* retruded contact position; *ICP* intercuspal position; *MP* maximum protrusion; *MO* maximum opening; *R* retruded opening; *S* hinge axis; *X* mandibular postural position; *RCP→R* circular arc centered in S; *ICP→MO* habitual opening; *RCP, MP, MO* sagittal border positions; *RCP→MP, MP→MO, RCP→MO* sagittal border movements

physiology of various parts such as bones, cartilage, ligaments, muscles, and nerves. Therefore, its form has no absolute universality. In addition, the lateral border movements of the mandible are hardly reached during function (Sect. 3.2). In most cases they may be detected during parafunction (e.g., tooth grinding).

5.3 Dynamics

The mandible can be opened and closed, protracted and retracted, and laterally displaced by means of the *temporomandibular joint* (TMJ). Its compartmentation in two parts by the disc plays an important role for mandibular mechanics since this provides a whole spectrum of combined hinge and sliding movements against the temporal bone. Mandibular movements are also guided by the individual joint shape, the teeth, and especially the masticatory muscles. The lateral pterygoid has a special role as a guiding muscle since it is involved in almost all movements. The muscles usually do not work individually but always as coordinated groups.

5.3.1 Jaw Opening/Closing

During *jaw opening*, condylar motion consists of two coordinated components: a rolling and a sliding movement (rotation and translation). In the upright position, a slight degree of opening of the mouth occurs through gravity after tone loss of jaw adductors. The upper mandibular muscles (anterior belly of the digastric, mylohyoid, and geniohyoid) pull the mandible actively downward for a stronger jaw opening. This hinge movement occurs in the lower compartment of the joint around a transverse axis of rotation at the level of the lingula. This movement is facilitated by the lower tubercles (sternohyoid and thyrohyoid) which fix the tympanic bone. Since the space in the retromandibular fossa is too narrow for a pure downward/backward hinge movement, the lateral pterygoid also contracts at jaw opening. By combining rotation of the condyle-disc complex and pulling on the condylar neck, this muscle displaces disc and condylar head along the fossa or the articular eminence forward and downward over the articular tubercle. This movement, occurring mainly in the upper compartment, is supported by the lateral ligament, which is strained while securing the joint. Given that the lateral pterygoid can insert laterally both on the condyle and the disc, its active influence on disc position is controversial. Since both structures are mobile, it is difficult to differentiate the tractional force effect.

Depending on joint anatomy, the proportion of rotational and translational components can vary. On average, the joint head rotates about 2° per mm of ventral translation. Measurements from 30 subjects with a maximum jaw opening of 55 ± 6 mm resulted in an average sliding movement of the joint heads ranging 13–15 mm and a rotation ranging 26–30° [11]. Normal values of jaw opening during growth and in adulthood are described in the literature [12–14].

Jaw closing is performed by the bilateral contraction of the temporalis (anterior and middle parts), the masseter (superficial and deep parts), and the medial pterygoid and includes a rearward displacement of joint components. The lateral pterygoid controls this movement. While it is clear that jaw adductors return the mandible to the fossa, it is not conclusively clear which forces are responsible for the return of the disc. It could passively follow condylar motion and/or be repositioned by elastic retrodiscal tissue components [15].

TMJs as well as their motion are mostly shown in a sagittal plane, which does not render the three-dimensionality of mandibular structures. This simplified representation neglects, in particular, the diversity of the condyles, the variable angulation between the condylar main axes, as well as the asymmetrical fossa concavity. This leads to the erroneous idea that the TMJ is a sliding hinge joint and that its axis of rotation passes through both condyles [16, 17]. Modern recording and analysis techniques have now shown that the mandibular axis of rotation is not fixed in space [18]. During jaw opening/closing, the rotation axis does not pass through the condyles, but rather lies in the region of the mandibular angle and follows the direction of translation of the mandible (Fig. 5.3a). If a particular condylar point is followed in the course of the movement, it is obvious that the opening and closing traces are not exactly coincident [19]. The same is true for the pathways of the axes of rotation [20, 21]. This non-coincidence of the paths is probably the result of different muscle activations or their nonreciprocal force vectors when opening and closing. Detailed

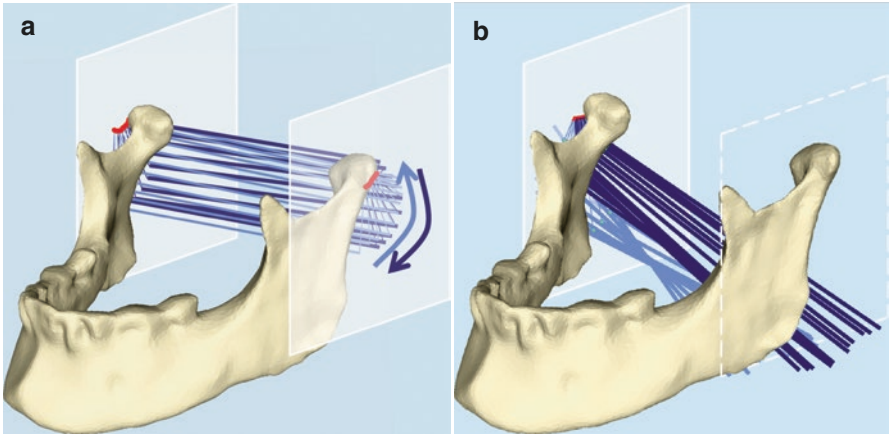

Fig. 5.3 Mandibular axis of rotation during jaw opening/closing. (**a**) Perspective view of the axis pathway of a normal TMJ during jaw opening (blue lines) and closing (red lines). The traces of the lateral condylar poles are yellow. (**b**) Corresponding view of a right clicking joint: Although the condylar trace is not relevantly different from the normal case, mandibular motion is strongly disturbed

descriptions of the course pattern of the mandibular rotation axis for different mandibular movements, such as chewing soft and tough food or smaller or larger food bolus, and joint conditions, such as disc displacement, can be found in the literature [20, 21]. In particular, when TMJ discs are displaced, the axis of rotation fluctuates more strongly than for asymptomatic joints, as shown in Fig. 5.3b. This is also the case when a TMJ is unilaterally replaced by a prosthesis.

Note: In jaw opening/closing, condylar motion consists of combined rotational and translational components, and their proportions vary depending on joint anatomy. The axis of rotation of the mandible lies in the region of the mandibular angle and follows its direction of translation.

5.3.2 Border Movements

Mandibular motions to the front are defined as protrusion (the return movement is called retraction), backward as retrusion, and laterally as laterotrusion. As a rule, again, border movements (the mandibular teeth slide along their maxillary antagonists) are recorded and analyzed starting from maximum intercuspation (ICP).

Protrusion is mainly due to the combined symmetrical action of lateral and medial pterygoids as well as superficial parts of the masseters. Condyles, including the disc, are displaced forward from the fossa toward the articular tubercle. Movements are guided by the teeth rows. Since this movement necessarily forces the joint downward, protrusion occurs with a slight lowering of the mandible.

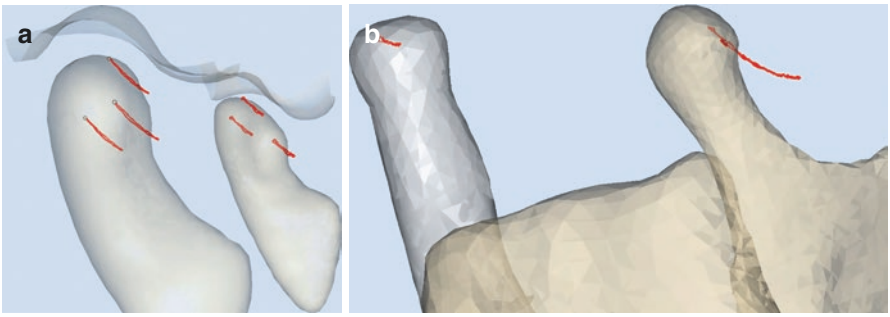

Fig. 5.4 Protrusion. (**a**) Protrusive phase of a healthy right TMJ. The condyle slides along the fossa. The paths of the poles as well as of the apex are practically identical since the rotation component is missing. (**b**) Protrusive phase of a prosthetic right TMJ. The right prosthetic condyle is hardly displaced, since the lateral pterygoid muscle is no longer connected (lateral pole trajectories in yellow)

During *retraction*, the said joint elements are displaced back into the fossa. This is mainly due to the contraction of the horizontally extending fibers of the posterior temporal muscle with the support of deep portions of the masseter, as well as the digastric and the geniohyoid. Similarly, *retrusion* is mainly determined by the symmetrical activation of the posterior parts of the temporal muscles. Figure 5.4a illustrates the trajectories of condylar poles and apex during a protrusion in a healthy person. The condyle slides along the fossa, and the paths of the poles as well as of the apex are practically identical since the rotation component is missing. The consequence of the lack of function of the lateral pterygoid is visible in a patient with a TMJ prosthesis, the lateral pterygoid being absent or disconnected on the prosthetic side (Fig. 5.4b) [22].

Laterotrusions are performed under activation of the medial and lateral pterygoid. They consist of a unilateral displacement of the condyle-disc complex, while on the opposite side the condyle stays in the fossa, rotates around a vertical axis, and displaces laterally to the front [23]. Mandibular incisors move to the side of the respective axis of rotation. The one-sided temporalis contraction causes a local rotation of the joint head about a vertical axis (*working side*) with simultaneous forward-downward movement of the opposite side to the articular tubercle (*balancing side*) due to one-sided contraction of the lateral pterygoid and masseter. An oblique motion component is caused especially by the medial pterygoid muscle. The guide through the teeth row also plays an important role. A movement sequence of balancing and working condyle during laterotrusion to the left is visible in Fig. 5.5.

Due to different fossa inclinations in dorsoventral and mediolateral direction and variable condylar shapes, it is evident that the traces of the balancing condyle differ between protrusion and laterotrusion. For the sake of completeness, the concept of the *Bennett angle*, which is produced in the transverse projection between the protrusive and the laterotrusive path, may be mentioned [24, 25]. Finally, the still unclear role of TMJ mechanoreceptors – due to scarce research in this area – appears

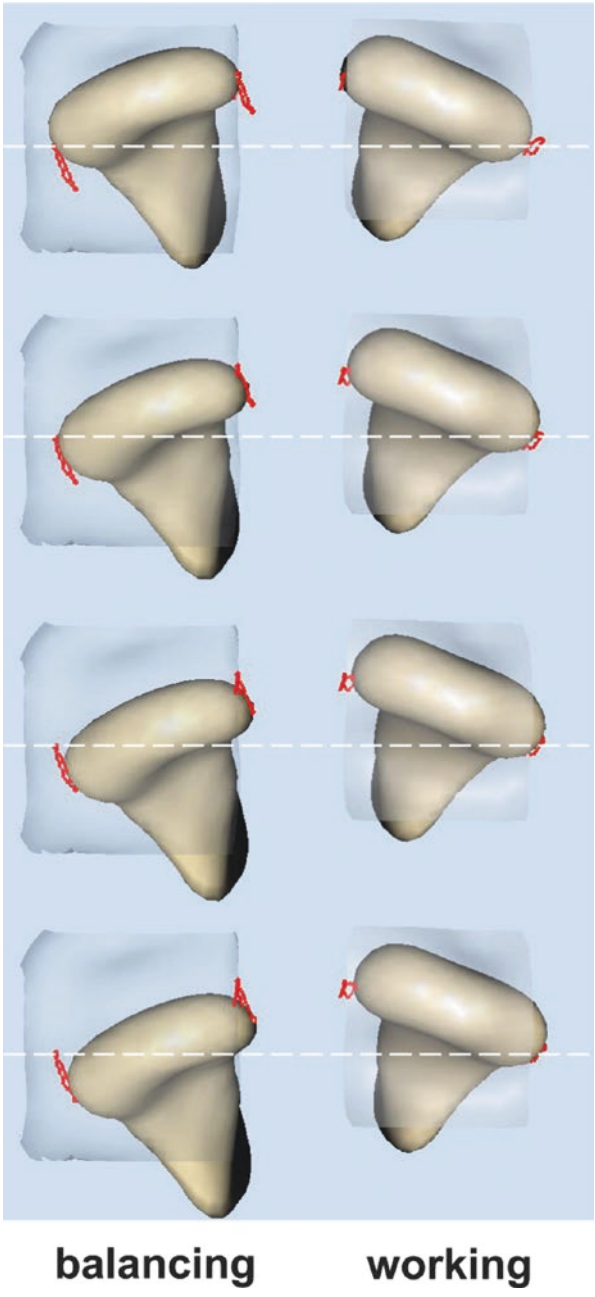

Fig. 5.5 Laterotrusion. Top view of four condyle positions of healthy jaw joints during a laterotrusive phase to the left. The balancing side is on the right and the working side on the left of the subject. Note the different movements of the medial and lateral condylar poles on the working side (the lateral pole displaces slightly dorsally)

to be mostly of proprioceptive nature: they seem to provide with a sensitivity of around 3 mm the detection of extreme positions of the mandible, thus possibly preventing its dislocation [26–31].

Note: In mandibular protrusion/retraction, retrusion, and laterotrusion, the symmetrical or asymmetrical activation of the lateral pterygoid muscle plays an important role. In the case of asymmetrical mandibular movements (laterotrusions and chewing movements), a working side and a balancing side are distinguished.

5.3.3 Mastication

Masticatory movements represent an asymmetrical and alternating combination of protrusion, retraction, laterotrusions, opening, and closing. They follow a rhythmic pattern, similar to breathing and gait or other learned physical behaviors. Knowledge about the mechanisms underlying the rhythmogenesis is still incomplete. Neurons with the property of generating amplitude accumulations were observed in subpopulations of the trigeminal mesencephalic and main sensory nuclei as well as in supratrigeminal neurons [1]. They are considered as components of a CPG network. The alternating activation pattern of agonistic and antagonistic muscles is essentially regulated by decreasing cortical pathways of the CPG network, but also by the feedback of peripheral neural receptors (among others the periodontal, mucosal, and temporomandibular receptors as well as muscle spindles) [32]. Therefore, chewing patterns largely differ inter- and intraindividually, and age, sex, and food-related qualities also play a role [33–37].

Based on observations performed mainly in animals, the chewing cycle is subdivided into an opening phase, an initial fast, and a slowly closing phase (*power stroke*) (Fig. 5.6). Toward the end of the vertical opening, the mandible deviates toward the bolus side (working side). From this position begins the fast closing phase, which ceases as soon as the masticatory system senses food resistance. This is followed by the power stroke, during which force is generated for food comminution [38, 39]. It is important to know that mandibular teeth approach their maxillary antagonists from the lateral side. Intercuspation is rarely achieved during the initial chewing cycles because the bolus is between the teeth. The asymmetrical movement of the two condyles during the closing phase is illustrated in the example of a right chewing cycle in Fig. 5.7. Views of the dynamic displacement of the axis of rotation of the mandible during a chewing cycle are given in Fig. 5.8. As mentioned above, position and orientation of the axis of rotation are determined by the size and consistency of the food particles: the more consistent the food or the larger the bolus, the stronger the deflections of the axis [21]. Activation and coordination of the masticatory muscles during chewing is

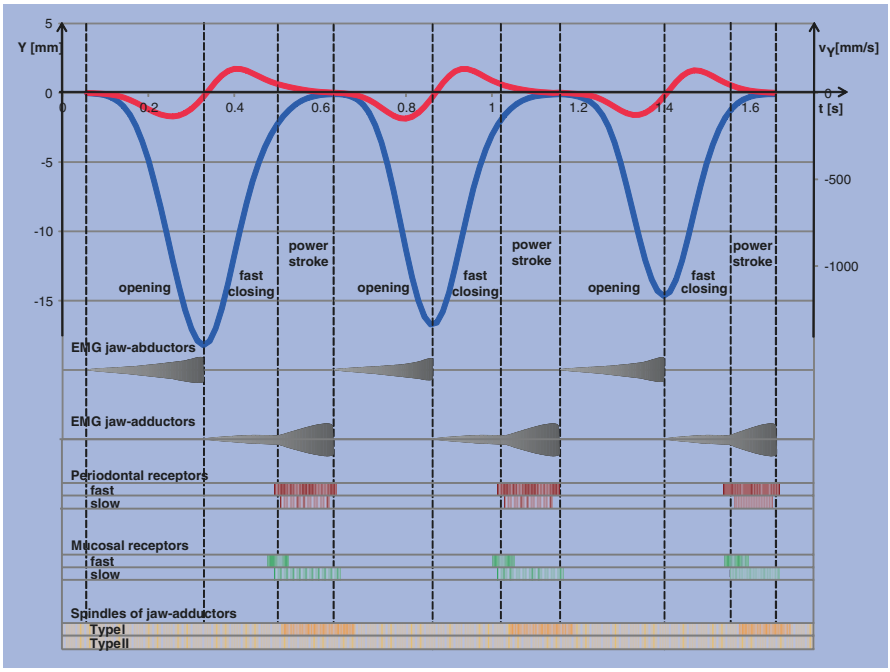

Fig. 5.6 Chewing timing. Vertical position y [mm] and velocity component v_y [mm/s] of the incisal point during three chewing cycles plotted vs. time [s]. Cycles are divided into three phases: opening, closing, and occlusal phase (*power stroke*). EMG signals of jaw abductors (mainly suprahyoid musculature as well as the lower belly of the lateral pterygoid muscle) are present during the opening phases. During the whole jaw closing phase, EMG signals of jaw adductors can be detected, but during occlusal phases, signals increase as force is developed for food crushing. Fast and slow periodontal and mucosal receptors fire almost exclusively during occlusal phases. Spindle signals of jaw adductors are present during all chewing phases since they are used to regulate muscle contractions; only during power strokes those of type I increase

strongly modulated by periodontal mechanoreceptors which are involved in most functional physiological and pathological processes of the masticatory system [40–43].

5.3.4 Dysfunctional Activities

Parafunctions (or oral habits) is a collective term for a mostly unconscious use of the masticatory system in opposition to normal functions (such as food intake or phonation). These include, among others, lip and cheek biting, pin and fingernail chewing, thumb-sucking, and bruxism (involuntary hyperactivity of the masticatory muscles). Stress-related parafunctions do not necessarily cause TMJ problems, but are often associated with them.

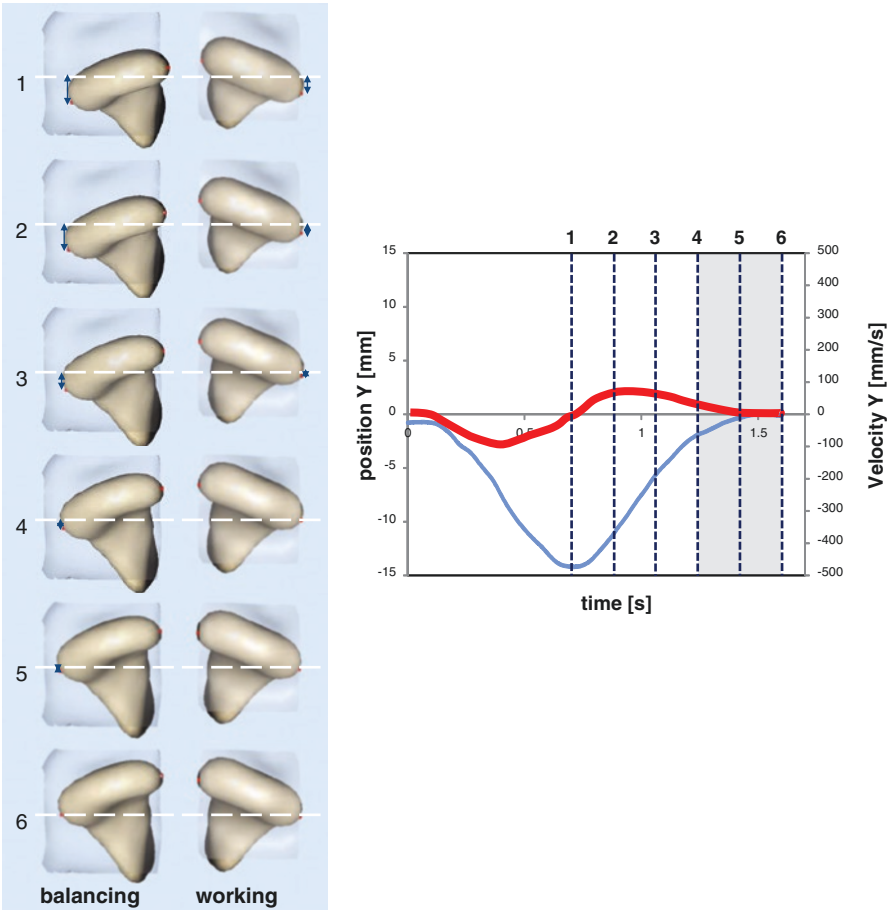

Fig. 5.7 Condylar movement during chewing. Top view of both condyles during the fast and slow (occlusal) closing phase (six positions). The bolus is located on the left side of the subject (working side, right in the picture). The working condyle moves faster dorso-cranially than the balancing condyle (left side of the subject) and reaches much earlier the most cranial position in the fossa. The posterior end position of the condyles is indicated by the blue dashed line. The different positions between the working and balancing sides can be viewed using the green distance arrows

Note: During mastication, a central pattern generator in the brain stem coordinates masticatory muscle activation. During the occlusal part of the closing phase, force is generated which serves for food comminution by grinding movements on the working side.

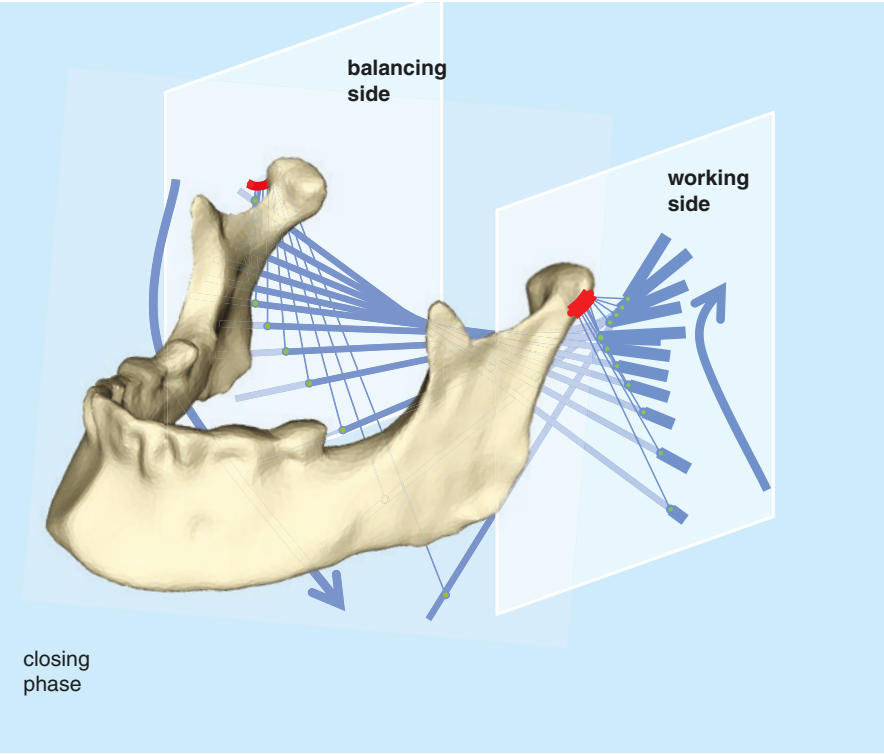

Fig. 5.8 Axis of rotation during chewing. Views of the typical pathways of the instantaneous rotation axis of the mandible during the closing and occlusal phase of a chewing cycle. This view was taken obliquely from the left/front side and shows 12 steps. On the working side, the rotation axis at the beginning of the closure is located dorso-caudally to the condyle. At the end of the closure, it is near or sometimes above it. On the balance side, on the other hand, the rotation axis is located close to the beginning of the closure or cranial to the condyle. At the end of the closure, it is dorso-caudal. The working condyle reaches its uppermost position temporally before the balancing condyle while this is still translating dorsally and cranially. The working condyle rotates almost exclusively

Bruxism is an involuntary hyperactivity of the masticatory muscles that determine mandibular position. This hyperactivity can occur during wake time or during sleep (*sleep bruxism* (SB)). Predominant hyperactivity of lateral pterygoids causes eccentric movements resulting in tooth grinding, whereas jaw-adductor hyperactivity (masseter and temporalis) manifests itself as *tooth clenching* (tonic) or *tooth rattling* (rhythmic). Frequently, the term *rhythmic masticatory muscle activity* (RMMA) is used reductively in the context of the concept of bruxism. In addition to hyperactivity, there is also an *increased base activity* of the masticatory muscles, which manifests itself as increased tooth contact [44]. First definitions of bruxism originate from sleep research, and the current terms and definitions are found in the *International Classification of Sleep Disorders* (ICSD) where bruxism is classified among behavioral problems during sleep (*parasomnia*) [45]. In general, ICSD bruxism efinitions are based on very vague clinical and instrumental parameters without

clear quantitative criteria. Therefore, instrumental methods such as *polysomnography* (PSG) including *electromyography* (EMG) for the quantification of muscle activity and indirectly bite force are needed [46, 47].

The masticatory frequency set by the CPG network appears to be an individual feature of every human being [48]. Unconscious lower jaw movements during sleep are less well documented than chewing movements. It is known that RMMA, measured by EMG, occurs in 60% of normal sleepers. On the basis of indirect evidence (bevels on the teeth, wear facets, etc.), it can be assumed that predominantly sliding movements occur in the horizontal plane. In persons with sleep bruxism, contraction episodes are found to be about three times more frequent than in non-bruxers [49]. The hypothesis that nocturnal bruxism and chewing activity are the basis of a common pattern generator could not be confirmed in a first pilot study on 13 subjects [50]. In fact, it was observed that the frequency of RMMA during sleep is not correlated with masticatory frequency and that they have a duration and intensity of only half as long.

Note: Among dysfunctional activities of the masticatory system, sleep bruxism can only be quantified by means of instrumental methods. The literature shows inconsistent data regarding self-reports as well as associations between RMMA and TMJ disorders.

5.4 TMJ Loading and Unloading

By means of measurements of the TMJ space, it can be inferred indirectly on soft tissue loads, which can be calculated using the material properties of the disc and its deformation [51–54]. Two force components can be distinguished: one perpendicular (mostly axial) and one tangential to the cartilage surface. The difference of repeated intra-articular distance measurements allows conclusions to be drawn on the perpendicular (*normal*) load. The tangential (*shear*) load is determined by the dynamic displacement of the minimum intra-articular distances and defined as *stress field* [55].

During jaw opening/closing, the shear load acts mainly in the lateral joint part. In normal disc position, it moves synchronously with the condyle-disc complex and has a mediolateral component (Fig. 5.9a). In two thirds of the TMJs, the shear stress shifts from medial to lateral, whereas in 20% it moves from lateral to medial (the residual cases are without a clear direction) [56]. This might explain why, in MR imaging, partial disc displacement appears to occur predominantly laterally [57]. An additional factor that affects the pathways of the shear strain is the condyle and fossa morphology [58, 59]. In contrast to normal joints, shear stress in TMJs with disc displacement with reduction[1] is quite different. In this case, the stress-field paths diverge

[1] "Anterior disc displacement with reduction," the disc is totally or partially displaced ventrally when the mouth is closed and the condyle is situated cranially to the disc at mouth opening.

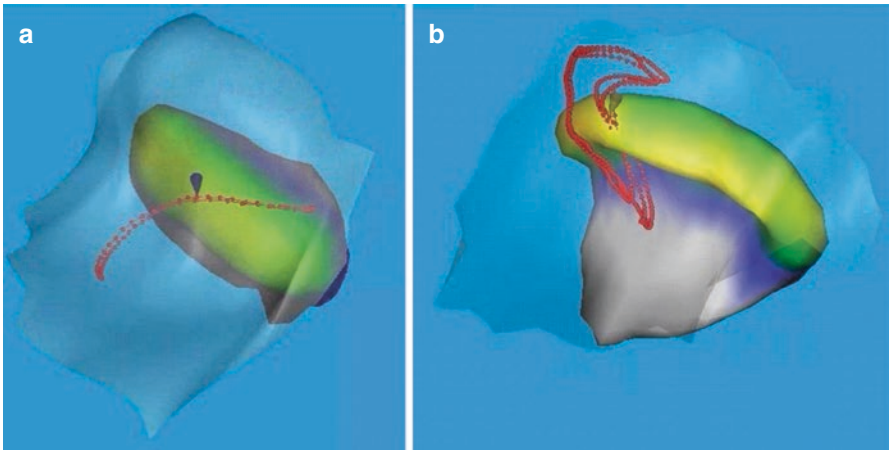

Fig. 5.9 Dynamic TMJ loading. (**a**) Stress-field paths during jaw opening/closing in an asymptomatic TMJ (red lines) almost perfectly coincident for both phases and located in the lateral part of the joint. (**b**) Analogous example in a TMJ with disc displacement (black lines): The paths do not match during the opening and closing phases

significantly between the opening and the closing phase. They move mainly in the medial joint area during jaw opening and above all in the lateral joint area during closing (an example shown in Fig. 5.9b). Whether this discrepancy of the shear load between the two joint conditions is the cause or consequence of the disc position is unclear.

As mentioned above, border movements such as laterotrusion with tooth contact occur primarily in tooth grinding (bruxism). Motion analysis reveals that the shear stress in the balancing joint has a mediolateral component significantly larger than on the working side. Protrusive movements follow a similar pattern as in the balancing joint during laterotrusion [60].

During chewing, the joint load in the power stroke (slow closing phase) is of interest, since in this phase the axial load increases in importance compared to the slightly loaded opening and fast closing phases. First measurements in healthy individuals show that the minimum condyle-fossa distance during the power stroke on the working side is less reduced on chewing than on the opposite side [61, 62]. This result agrees with models which describe a greater load on the balancing than on the working side during the final phase of chewing [63]. However, patients with inflammatory jaw disorders still have to clarify whether chewing on the affected side is more painful than on the opposite side.

In case of TMJ disorders, unloading of joint structures is often sought after. The use of occlusal appliances has been one of the noninvasive therapies of choice, although their mechanism is still unclear [64]. Michigan-type splints appear to slightly reduce intra-articular distance and thus mechanical axial loading, which however can also depend on joint morphology and the actual joint reaction force.

Other joint unloading strategies can be developed by means of biofeedback for jaw-adductor relaxation, which appears to be consistently applicable [9, 65]. Finally, diversity in durations and intensities of jaw function between TMD diagnostic groups—besides variability in joint morphology—could further differentiate the amount of mechanical work imposed on TMJ tissues and explain their fatigue failure [7, 66].

Note: The (tangential) shear stress of the disc tissue arises from the incongruence of the surfaces of the fossa and condyle and mainly acts in the lateral joint part. During the slow (occlusal) portion of the closing phase of the chewing cycle, an axial TMJ load is produced in the balance joint more strongly than in the working TMJ.

In a nutshell: Modern measurement technology allows accurate three-dimensional, static, and dynamic acquisition and analysis of biomechanics of the jaw joint. The large intra- and interindividual variability of jaw morphology and function is caused by their interaction, which changes continuously in the course of life.

References

1. Morquette P, Lavoie R, Fhima M-D, Lamoureux X, Verdier D, Kolta A. Generation of the masticatory central pattern and its modulation by sensory feedback. Prog Neurobiol. 2012;96:340–55.
2. Harris-Warrick RM. General principles of rhythmogenesis in central pattern generator networks. Prog Brain Res. 2010;187:213–22.
3. Daun S, Rubin JE, Rybak IA. Control of oscillation periods and phase durations in half-center central pattern generators: a comparative mechanistic analysis. J Comput Neurosci. 2009;27:3–36.
4. Barlow SM, Estep M. Central pattern generation and the motor infrastructure for suck, respiration, and speech. J Commun Disord. 2006;39:366–80.
5. Lund JP, Kolta A. Generation of the central masticatory pattern and its modification by sensory feedback. Dysphagia. 2006;21:167–74.
6. Türker KS. Reflex control of human jaw muscles. Crit Rev Oral Biol Med. 2002;13:85–104.
7. Iwasaki LR, Gonzalez YM, Liu Y, et al. Mechanobehavioral scores in women with and without TMJ disc displacement. J Dent Res. 2017;96:895–901.
8. The glossary of prosthodontic terms: ninth edition. J Prosthet Dent. 2017;117:e1–105.
9. Michelotti A, Farella M, Vollaro S, Martina R. Mandibular rest position and electrical activity of the masticatory muscles. J Prosthet Dent. 1997;78:48–53.
10. Posselt U. Range of movement of the mandible. J Am Dent Assoc. 1958;56:10–3.

11. Salaorni C, Palla S. Condylar rotation and anterior translation in healthy human temporomandibular joints. Schweizer Monatsschrift fur Zahnmedizin = Revue mensuelle suisse d'odonto-stomatologie = Rivista mensile svizzera di odontologia e Stomatologia. 1994;104:415–22.
12. Ferrario VF, Sforza C, Lovecchio N, Mian F. Quantification of translational and gliding components in human temporomandibular joint during mouth opening. Arch Oral Biol. 2005;50:507–15.
13. Mapelli A, Galante D, Lovecchio N, Sforza C, Ferrario VF. Translation and rotation movements of the mandible during mouth opening and closing. Clin Anat. 2009;22:311–8.
14. Gallagher C, Gallagher V, Whelton H, Cronin M. The normal range of mouth opening in an Irish population. J Oral Rehabil. 2004;31:110–6.
15. Wilkinson TM, Crowley CM. A histologic study of retrodiscal tissues of the human temporomandibular joint in the open and closed position. J Orofac Pain. 1994;8:7–17.
16. Bowley JF, Pierce CJ. Reliability and validity of a transverse horizontal axis location instrument. J Prosthet Dent. 1990;64:646–50.
17. Hayashi T, Itoh K, Miyakawa M. Determination of the kinematic axis point of the temporomandibular joint regardless of cyclic mandibular movement data. Front Med Biol Eng. 1994;6:199–208.
18. Ferrario VF, Sforza C, Miani A, Serrao G, Tartaglia G. Open-close movements in the human temporomandibular joint: does a pure rotation around the intercondylar hinge axis exist? J Oral Rehabil. 1996;23:401–8.
19. Gallo LM, Gössi DB, Colombo V, Palla S. Relationship between kinematic center and TMJ anatomy and function. J Dent Res. 2008;87:726–30.
20. Gallo LM, Airoldi GB, Airoldi RL, Palla S. Description of mandibular finite helical axis pathways in asymptomatic subjects. J Dent Res. 1997;76:704–13.
21. Gallo LM, Brasi M, Ernst B, Palla S. Relevance of mandibular helical axis analysis in functional and dysfunctional TMJs. J Biomech. 2006;39:1716–25.
22. Leiggener CS, Erni S, Gallo LM. Novel approach to the study of jaw kinematics in an alloplastic TMJ reconstruction. Int J Oral Maxillofac Surg. 2012;41:1041–5.
23. Palla S, Gallo LM, Gössi D. Dynamic stereometry of the temporomandibular joint. Orthod Craniofac Res. 2003;6(Suppl 1):37–47.
24. Holste T. Untersuchungen über den Bennett-Winkel. Deutsche zahnarztliche Zeitschrift. 1980;35:315–7.
25. Fanucci E, Spera E, Ottria L, et al. Bennett movement of mandible: a comparison between traditional methods and a 64-slices CT scanner. Oral Implantol. 2008;1:15–20.
26. Klineberg I. Influences of temporomandibular articular mechanoreceptors in functional jaw movements. J Oral Rehabil. 1980;7:307–17.
27. Ayesh EE, Ernberg M, Svensson P. Effects of local anesthetics on somatosensory function in the temporomandibular joint area. Exp Brain Res. 2007;180:715–25.
28. Macefield VG. Physiological characteristics of low-threshold mechanoreceptors in joints, muscle and skin in human subjects. Clin Exp Pharmacol Physiol. 2005;32:135–44.
29. Kawamura Y, Abe K. Role of sensory information from temporomandibular joint. Bull Tokyo Med Dent Univ. 1974;21(Suppl):78–82.
30. Suzuki O, Tsuboi A, Tabata T, Takafuji Y, Sakurai T, Watanabe M. Response properties of temporomandibular joint mechanosensitive neurons in the trigeminal sensory complex of the rabbit. Exp Brain Res. 2012;222:113–23.
31. Tsuboi A, Takafuji Y, Itoh S, Nagata K, Tabata T, Watanabe M. Response properties of trigeminal ganglion mechanosensitive neurons innervating the temporomandibular joint of the rabbit. Exp Brain Res. 2009;199:107–16.
32. Olsson KA, Sasamoto K, Lund JP. Modulation of transmission in rostral trigeminal sensory nuclei during chewing. J Neurophysiol. 1986;55:56–75.
33. Proschel P. An extensive classification of chewing patterns in the frontal plane. Cranio. 1987;5:55–63.
34. Pröschel P. Zum Einfluss der Okklusalflächenform auf den Bewegungsablauf des Unterkiefers bei der Kaufunktion. Deutsche zahnarztliche Zeitschrift. 1988;43:1099–103.

35. Proschel P, Hofmann M, Ott R. Zur Orthofunktion des Kauorgans. Deutsche zahnarztliche Zeitschrift. 1985;40:186–91.
36. Pröschel P, Hofmann M. Zur Problematik der Interpretation von funktionellen Unterkieferbewegungen. Teil I: Die Auswirkungen multifaktorieller Einflüsse auf die Interpretierbarkeit von Kaubewegungsaufzeichnungen. Deutsche zahnarztliche Zeitschrift. 1987;42:696–700.
37. Pröschel P, Hofmann M. Frontal chewing patterns of the incisor point and their dependence on resistance of food and type of occlusion. J Prosthet Dent. 1988;59:617–24.
38. Ottenhoff FA, van der Bilt A, van der Glas HW, Bosman F. Control of elevator muscle activity during simulated chewing with varying food resistance in humans. J Neurophysiol. 1992;68:933–44.
39. Ottenhoff FA, van der Bilt A, van der Glas HW, Bosman F. Control of human jaw elevator muscle activity during simulated chewing with varying bolus size. Exp Brain Res. 1993;96:501–12.
40. Johansson RS, Trulsson M, Olsson KA, Abbs JH. Mechanoreceptive afferent activity in the infraorbital nerve in man during speech and chewing movements. Exp Brain Res. 1988;72:209–14.
41. Johansson RS, Trulsson M, Olsson KA, Westberg KG. Mechanoreceptor activity from the human face and oral mucosa. Exp Brain Res. 1988;72:204–8.
42. Trulsson M. Sensory-motor function of human periodontal mechanoreceptors. J Oral Rehabil. 2006;33:262–73.
43. Piancino MG, Isola G, Cannavale R, et al. From periodontal mechanoreceptors to chewing motor control: a systematic review. Arch Oral Biol. 2017;78:109–21.
44. Raphael KG, Janal MN, Sirois DA, et al. Validity of self-reported sleep bruxism among myofascial temporomandibular disorder patients and controls. J Oral Rehabil. 2015;42:751–8.
45. American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
46. Gonzalez Y, Iwasaki LR, McCall WD, Ohrbach R, Lozier E, Nickel JC. Reliability of electromyographic activity vs. bite-force from human masticatory muscles. Eur J Oral Sci. 2011;119:219–24.
47. Khoury S, Carra MC, Huynh N, Montplaisir J, Lavigne GJ. Sleep bruxism-tooth grinding prevalence, characteristics and familial aggregation: a large cross-sectional survey and polysomnographic validation. Sleep. 2016;39:2049–56.
48. Po JMC, Kieser JA, Gallo LM, Tésenyi AJ, Herbison P, Farella M. Time-frequency analysis of chewing activity in the natural environment. J Dent Res. 2011;90:1206–10.
49. Lavigne GJ, Rompré PH, Poirier G, Huard H, Kato T, Montplaisir JY. Rhythmic masticatory muscle activity during sleep in humans. J Dent Res. 2001;80:443–8.
50. Po JMC, Gallo LM, Michelotti A, Farella M. Comparison between the rhythmic jaw contractions occurring during sleep and while chewing. J Sleep Res. 2013;22:593–9.
51. Nickel JC, McLachlan KR. In vitro measurement of the frictional properties of the temporomandibular joint disc. Arch Oral Biol. 1994;39:323–31.
52. Nickel JC, McLachlan KR. In vitro measurement of the stress-distribution properties of the pig temporomandibular joint disc. Arch Oral Biol. 1994;39:439–48.
53. Schmolke C. The relationship between the temporomandibular joint capsule, articular disc and jaw muscles. J Anat. 1994;184(Pt 2):335–45.
54. Schmolke C, Hugger A. The human temporomandibular joint region in different positions of the mandible. Ann Anat. 1999;181:61–4.
55. Gallo LM, Nickel JC, Iwasaki LR, Palla S. Stress-field translation in the healthy human temporomandibular joint. J Dent Res. 2000;79:1740–6.
56. Gössi DB, Gallo LM, Bahr E, Palla S. Dynamic intra-articular space variation in clicking TMJs. J Dent Res. 2004;83:480–4.
57. Chen Y-J, Gallo LM, Palla S. The mediolateral temporomandibular joint disc position: an in vivo quantitative study. J Orofac Pain. 2002;16:29–38.
58. Colombo V, Palla S, Gallo LM. Temporomandibular joint loading patterns related to joint morphology: a theoretical study. Cells Tissues Organs. 2008;187:295–306.

59. Nickel JC, McLachlan KR. An analysis of surface congruity in the growing human temporomandibular joint. Arch Oral Biol. 1994;39:315–21.
60. Gallo LM, Chiaravalloti G, Iwasaki LR, Nickel JC, Palla S. Mechanical work during stress-field translation in the human TMJ. J Dent Res. 2006;85:1006–10.
61. Palla S, Krebs M, Gallo LM. Jaw tracking and temporomandibular joint animation. In: McNeill C, editor. Science and practice of occlusion. Chicago, IL: Quintessence Publishing; 1997. p. 365–78.
62. Fushima K, Gallo LM, Krebs M, Palla S. Analysis of the TMJ intraarticular space variation: a non-invasive insight during mastication. Med Eng Phys. 2003;25:181–90.
63. Rues S, Lenz J, Türp JC, Schweizerhof K, Schindler HJ. Muscle and joint forces under variable equilibrium states of the mandible. Clin Oral Investig. 2011;15:737–47.
64. Ettlin DA, Mang H, Colombo V, Palla S, Gallo LM. Stereometric assessment of TMJ space variation by occlusal splints. J Dent Res. 2008;87:877–81.
65. Capuozzo R, Farella M, Gallo LM, Palla S. Effects of low-level clenching on TMJ intra-articular distances. J Oral Rehabil. 2011;38:E2–E22.
66. Iwasaki LR, Gonzalez YM, Liu H, Marx DB, Gallo LM, Nickel JC. A pilot study of ambulatory masticatory muscle activities in temporomandibular joint disorders diagnostic groups. Orthod Craniofac Res. 2015;18(Suppl 1):146–55.

Neuroanatomical Signatures of Acute and Chronic Orofacial Pain

6

M. Bruegger

Abstract

The more fully we understand chronic pain, the more adept we as providers will be able to deliver effective care to the patient with TMD. There have been significant advances in our current understanding of the neuroanatomical and neurochemical elements that underlie chronic pain, but the picture of how it is established and maintained is by no means complete. This chapter presents a short synopsis of our current appreciation of pain in general as well as a discussion of the research that contributes to the basis of our contemporary knowledge and theories that help us understand TMD-associated chronic pain.

6.1 Neuroanatomy of Pain: A General View

Wilder Penfield and colleagues lead the way to our current understanding regarding the principles of the cortical representation of somatosensory input. Their spectacular discoveries led to the famous somatosensory homunculus, a distorted scaled model of the human body neurally arranged at the postcentral gyrus [1, p. 1721]. Later on, cortical motor, sensory, and speech areas were discovered based on electrical stimulation of respective brain areas within the context of presurgical examination in epileptic patients [2]. Interestingly, those pioneering works revealed no distinct pain responses. Penfield and Jasper noted that some patients expressed feeling sensations best described as prickling or tingling and slightly unpleasant, but not painful at all [3].

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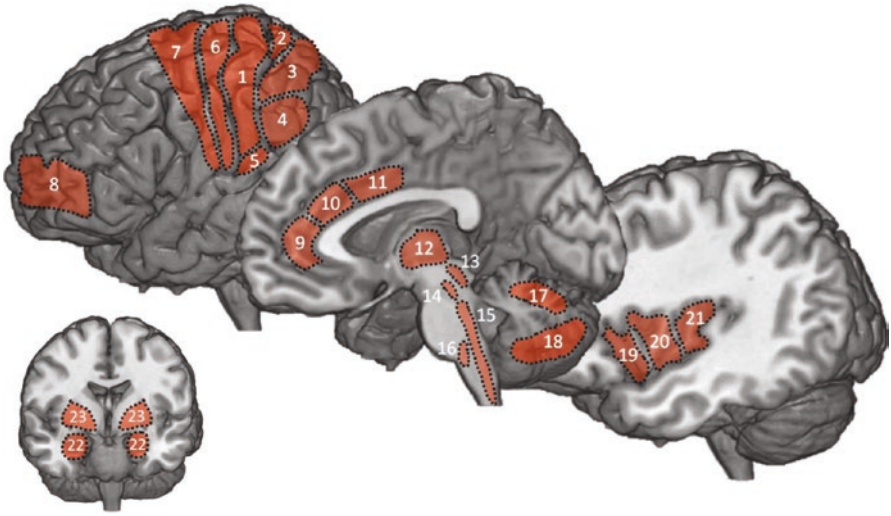

Fig. 6.1 Schematic illustration of cortical and subcortical areas found to be incorporated in the processing of experimental and chronic pain (see corresponding Table 6.1 for anatomical and functional description)

With the advent of noninvasive neuroimaging techniques, this spectacular initial work was extended and resulted in extensive knowledge incorporating the whole signaling cascades from peripheral somatosensory transduction mechanisms to their repository in different cortical and subcortical areas. Over the decades, profound explorations regarding neuroanatomical and functional aspects have led to the identification of a pain-associated neural network often denoted as a "pain matrix" [4, p. 883] or in more recent publications as a "neurologic signature of physical pain" [5, p. 1072]. This network can be summarized as a confluence across a huge amount of clinical and basic research with a main focus on pain. Fig. 6.1 and Table 6.1 provide a schematic according to brain areas observed to be involved in coding the whole experience of pain.

For quite some time, a rather deductive approach leads clinicians and scientists to believe that pain was primarily a nociceptive phenomenon and thus assignable to just a few distinct cortical areas across the pain matrix, with the primary somatosensory cortex playing the major role. This obvious simplification was on the one hand attributable to methodological constraints but also due to a primary "sensory-guided" view of pain processing. After all, sensory input is sensory by nature and thus should be processed within somatosensory areas. Thus, it logically followed that pain, as the strong(est) sensory sensation, must be localized and most pronounced within those sensory regions [6, p. 913, 7, p. 1145].

Since then, our knowledge regarding the underlying principles of brain-related pain processing have broadened substantially. In particular, the importance of a multifaceted perspective on the topic became evident. Today, even a simple definition of pain has become challenging. Currently, the International Association for the

Table 6.1 Corresponding anatomical and functional descriptions of areas within the "cortical functional pain circuit" delineated in Fig. 6.1

Nr	Anatomical description	Primary functions within a pain experience
1	Postcentral gyrus (S1)	Primary somatosensory "Somatosensory homunculus"
2	Superior-parietal area	Somatosensory association
3	Superior-parietal area	Somatosensory association
4	Supramarginal area	Somatosensory association
5	Subcentral area/parietal operculum	Somatosensory association, somatosensory awareness, intensity coding
6	Precentral gyrus (M1)	Motor reactions and planning "Motor homunculus"
7	Extend precentral areas	Supplementary motor reactions, motor anticipation
8	Prefrontal/frontopolar areas	Somatosensory/pain-related attention and evaluation Memory/meta-memory regarding pain and/or thread linked with pain Pain memory/reference Pain chronification
9	Pregenual anterior cingulate	Emotional integration/partly visceral integration Anticipation "Suffering component" of pain
10	Anterior mid-cingulate	Cognitive-evaluative processing linked with avoiding of potentially pain evoking situations Anticipation "Suffering component" of pain
11	Posterior mid-cingulate	Cognitive-evaluative processing linked with motor reactions
12	Thalamus	Relay station for all spino-cortical and corticospinal signaling cascades
13	Periaqueductal gray (PAG)	Modulating functions of somatosensory input, can be mitigating or amplifying
14	Nucleus cuneiformis (NCF)	Primary pain inhibitory function Recent work point to a more complex involvement/modulation of pain signals
15	Spinothalamic, spinoreticular, and spinomesencephalic paths with embedded nuclei	Stimulus conduction periphery-thalamus
16	Rostroventral medulla (RVM)	Primary pain inhibitory function Recent work point to a more complex involvement/modulation of pain signals
17	Anterior cerebellum	Stimulus sensory and cognitive processing Anticipation
18	Posterior cerebellum	Rather cognitive and emotional processing Anticipation
19	Anterior insula	Chiefly involvement in a variety of cognitive-evaluative aspects regarding pain processing Anticipation
20	Middle insula	Complex involvement in a variety of different pain-related processes, its subclassification not entirely clear
21	Posterior insula	Chiefly involved in a variety of direct sensory-related pain processes The only region pain can be induced by intracranial stimulation
22	Amygdala and hippocampus areas	Fear of pain, pain anxiety, pain memory Probably involved in several key mechanisms to chronify pain
23	Putamen and pallidum (basal ganglia)	Motor-and anticipation related pain processing

Study of Pain (IASP) suggests the following: *An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage* (http://www.iasp-pain.org/terminology). Of course, there are lots of additional contents listed, but the key element is evident by focusing on the term *experience*.

Indeed, pain has to be interpreted as a "global experience," being hardly definable by anyone single, distinct neural constituent. Current views and explanations regarding human pain take into account multiple, complexly intertwined systems including sensory, motor, attentional, and cognitive neural processes [8, p. 1874, 9, p. 887]. Further, there is little doubt that pain has a strong and unique attentional activation quality that channels feelings, emotions, and thoughts in a specific direction, preserving the negative thoughts related to the possible events and consequences surrounding a specific pain experience [10, p. 1820]. But despite pain being a mostly unpleasant experience, it is probably the most important aspect of somatosensation [11, p.1667, 12, p. 958]. We need pain in order to be protected from injury and tissue damage. Without an intact, functioning pain system, it's challenging for an individual to stay healthy and free from injury. Indeed, reports on people suffering from congenital insensitivity to pain quite remarkably demonstrate the consequences of this statement. Several cases describe affected individuals who learned to live with their handicap and long-term outcomes were worsened by severe orthopedic complications mostly as a result of untreated skeletal injuries sustained in childhood [7, p. 1145, 13, p. 2017, 14, p. 2018].

Based on the above reasoning, when one considers the areas delineated in Fig. 6.1/Table 6.1, it now makes sense why there are numerous brain regions that participate in the experience of pain. There is a complex interplay of sensory, vegetative, emotional, motor, and cognitive aspects of pain, and it is now evident that the brain as a whole is challenged to adequately deal with such a global experience.

6.2 Acute Versus Chronic Pain

A healthy somatosensory system is one that is optimally equipped to accurately process pain, meaning that temporally, the pain diminishes either simply through the passage of time or by administration of medication. But some people are less "lucky"; in some cases, the pain remains and turns into a disruptive chronic entity, sometimes accompanied by severe comorbidities that become very difficult to treat [15, p. 613, 16, p. 1803, 17, p. 2021]. There are several open questions regarding this maladaptive development, and the associated risk factors remain poorly understood [18, p. 2027]. The rather simplified assumption was that chronic pain results from either constant nociceptive activation or central/peripheral somatosensory system damage which was generally summarized as neuropathic pain [19, p. 2028]. Others suggested that the chronicity derives from structural reorganization within the spinal cord and associated brain regions due to either the stimulation of long-lasting intense pain or severe psychologically/environmentally coincident stress

experienced when the injury was sustained [20, p. 980, 21, p. 1091, 22, p. 1157]. More recent views suggest a complex intermingling of structural alterations in combination with disturbances of default mode networks (DMN) and connectivity patterns, which are probably interdependent on each other [23, p. 1820, 24, p. 1917]. Additionally, cortically localized "risk factors" have been theorized to exist in the form of deviant circuits incorrectly connecting relevant cerebral areas that could underlie a person's vulnerability to develop the manifestations of chronic pain. Additionally, brain regions not classically considered to be associated with pain processing, structures located within the corticolimbic system (dorsal medial prefrontal cortex-amygdala-nucleus accumbens, ventral medial prefrontal cortex-amygdala, orbitofrontal cortex-amygdala), have been hypothesized to be key regions involved in the development of an individuals increased risks or susceptibility to chronic pain [18, p. 2027].

6.2.1 Example

A very interesting investigation by Mutso et al. [25, p. 2019] took a look at the structural and functional mechanisms underlying the transition from acute to chronic back pain. Based on the results of previous animal research, they assumed that the hippocampus would be the locus where structural and connective deviations would lead to the generation of the maladaptive circuitry, which is ultimately responsible for the switch from subacute to chronic pain. Indeed, they observed a significant involvement of hippocampal and prefrontal areas during the transformation from subacute (1–4 months) to chronic back pain (>10 years). The most severe alterations were observed in the structural reorganization of the hippocampus itself in addition to unbalanced connectivity patterns between the hippocampus/amygdala and prefrontal areas. Strikingly, experimentally driven acute pain studies very rarely report the hippocampal area as being activated.

However, another hypothesis favors the existence of a dynamic pain connectome, which exists as a spatiotemporal neural signature involving a variety of brain networks that communicate in a distinct fashion to integrate all the aspects of the pain experience. This model seems to represent the most accurate view we have to date (adapted from [10, p. 1820]). Table 6.2 summarizes the suggested networks and assigned functions to the areas involved in this theory.

It is not entirely clear which underlying mechanisms drive the alterations in network connectivity and increases or decreases in neural center activity. There are indications of aberrant DMN (default mode network) characteristics in several chronic pain states, but caution is advised in terms of conclusive causal interpretations of the reported observations. Also, alterations in connectivity strengths between respective areas/networks are discussed in both directions (amplification or mitigation) again in several chronic pain states [25, p. 2019, 26, p. 266, 27, p. 135, 28, p. 2118]. The underlying basic systems—sensorimotor, default mode, salience, and nociceptive—can further be interpreted as being involved in other daily behavior regulation processes we only begin to understand in detail [29, p. 1472]. Thus,

Table 6.2 A possible dynamic connectome regarding cortical pain processing including suggested networks, supposed functions, and associated areas

Network/system	Function	Areas involved
Sensory-motor (SM)	Sensory and motor-related fundamental states in regard to pain events	Primary somatosensory area Primary motor area
Salience (SN)	Pain-related interoceptive and sustained attention	Anterior insula Dorsolateral prefrontal area Posterior insula Temporoparietal junction Orbitofrontal area
Default mode network (DMN)	Most likely suppressed when concentrating on pain or when ruminate toward pain	Posterior cingulate areas Medial dorso-/anterior prefrontal Medial temporal lobe
Antinociceptive system (AS)	Descending pain modulation, amplified under acute pain, mitigated under chronic pain	Medial prefrontal Thalamus Brainstem substructures PAG/NCF/RVM

despite the concept of a dynamic pain connectome representing an intelligible approach for explaining the proneness to pain chronification and its neural manifestations, it is still challenging to allocate the different facets of pain specifically to a single areas connectivity changes in resting state network architecture or structural changes in areas belonging to the neural signature of pain.

Thus, to summarize this short general sketch, based on what we know from brain studies, both human and animal, chronic pain seems rather maintained by cortical areas, networks, and circuits whose functions are assigned to all sensory, vegetative, as well as emotional and cognitive processes. This might be a result of a maladaptive learning and association process [30, p. 987] or the amplification within prevulnerable systems possibly manifested in aberrant fronto-limbic structures and related processing [18, p. 2027].

6.3 Neuroanatomy of TMJ-Related Pain

The neural arrangement of the temporomandibular joint is—from a non-nociceptive somatosensory perspective—quite unequivocal: the locus is within the face area of the primary somatosensory cortex, tightly adjoining the hand area. Aside from the hand/finger area, the face area is the largest representation in this part of the brain, correlated with the associated peripheral receptor densities reflected in the sensitivity of the tongue, teeth, lips, nose, eye, and the skin of the human face in general. Thus, this system constitutes the main afferent pathways for all somatosensory processing regarding these structures, including the mandibular joint as well as associated muscles and tendons. Considering the pain-related neural signature delineated in Fig. 6.1, it's important to note that trigeminally mediated nociceptive input is underrepresented compared to pain evoked at other body sites [31, p. 1506, 32, p. 1950]. This is quite astonishing as trigeminal pain and associated burden,

suffering, and costs would justify enormous effort toward examination of underlying brain processing. In principle, there are two options to pursue to unravel this paradox, either one can study chronic TMJ conditions or focus on studying healthy volunteers in experimental orofacial pain models.

6.3.1 Experimental Approaches

Investigating healthy human subjects in experimental orofacial pain models in a standardized setting represents an important branch of research aimed to elucidate fundamental mechanisms of associated cortical pain processing. In this vein, toothache can be utilized as an acute form of orofacial pain, thus providing the researcher with an ideal experimental orofacial pain paradigm to evaluate trigeminally mediated cortical activation and response patterns. Indeed, several reports have been published applying either painful or painless stimuli to a least one tooth, while concomitantly recording brain responses. The modalities range from tactile/vibrotactile, electric, and air stimuli. An overview is given in Table 6.3.

To summarize, brain response patterns in response to tooth stimulation strongly resemble those from experimental pain applied to extra-trigeminal sites, especially stimulation at painful levels. This means that the associated neurological signature encompasses the areas illustrated in Fig. 6.1, however, with a number of obvious peculiarities.

Focusing firstly on somatotopic cortical organization aspects, the study by Jantsch et al. [34, p. 683] demonstrated S1 activity contralateral to hand pain compared to bilateral activity during tooth pain. This bilateral activity pattern might be related to the fact that the stimulated left incisor tooth is close to the body midline, whereas the hand is clearly more distal. It is critical to be cautious about this result as ideally, both incisor teeth should be stimulated to conclusively prove this assumption. The work conducted by Brügger et al. [31, p. 1506] addresses this issue by stimulation of the left/right maxillary canines and central incisors. A direct comparison between central incisor and canine stimulation revealed a more prominent tendency toward contralateral S1 activity for canines compared to central incisors. This finding implies a certain cortical lateralization scheme related to the distance from the body midline. Both findings support the concept of somatotopic organization within S1 also for teeth, however, with the limitation that this somatotopic pattern was induced by pain and not by painless somatosensory stimulation.

Utilizing MEG (Magnetic Encephalography) as an alternative method, the work by Kubo et al. [36, p. 1074] compared painless stimulation of the right maxillary first premolar with the median nerve of the right wrist. The findings also revealed bilateral activity in a region the authors termed “parieto-temporal” area. But when looking at the sources of this activity, a contralateral main focus located in the central sulcus (S1) was observed for tooth and wrist stimulation with a slight posterior/superior shift of tooth stimulation. Further fMRI-based evidence investigating possible somatotopy of the intraoral area is further supported by results of Miyamoto et al. [35, p. 1075]; their protocol also involves applying painless stimuli to the right

Table 6.3 Studies combining tooth stimulation and neuroimaging

Authors	Imaging technology magnetic field strength	N subjects	Stimulus modality Pain/painless/both	Stimulated tooth/teeth
Ettlin et al. [33]	fMRI 3 T	5 (2 male)	Mechanical vibrotactile Painless	Central incisor/canine/second premolar and second molar of each jaw quadrant
Jantsch et al. [34]	fMRI 1.5 T	8 (4 male)	Electric Weak and strong pain	Left maxillary central incisor
Miyamoto et al. [35]	fMRI 3 T	14 (8 male)	Tactile, rubber tip Painless	Right central upper incisor (beside lower lip/tongue)
Kubo et al. [36]	MEG	7 (7 male)	Electric Painless	Right maxillary first premolar
Brügger et al. [37]	fMRI 3 T	14 (8 male)	Electric Graded from painless to painful	Left/right maxillary canine
Trulsson et al. [38]	fMRI 3 T	10 (10 male)	Mechanical vibrotactile Painless	Left central upper incisor
Weigelt et al. [39]	fMRI 1.5 T	13 (8 male)	Electric pain	Left mandibular and maxillary canine
Brügger et al. [31]	fMRI 3 T	21 (13 male)	Electric pain (150% of pain perception thresholds)	Left/right maxillary canine and central incisor
Gutzeit et al. [40]	fMRS 3 T	10 (10 male)	Electric pain (4–5 out of 10 on a VAS)	Right maxillary canine
Brügger et al. [41]	fMRI 3 T	13 (13 male)	Electric 5 graded stimulus strengths from painless to painful	Right maxillary canine
Meier et al. [42]	fMRI 3 T	10 (2 male)	Air puffs 5 graded stimulus strengths from painless to painful	Right and left maxillary canine and molars
Gutzeit et al. [43]	fMRS 3 T	16 (16 male)	Electric pain (4–5 out of 10 on a VAS)	Right maxillary canine
Meier et al. [44]	fMRI 3 T	14 (14 male)	Electric pain (5 out of 10 on a NRS) Modulated by the anesthetic articaine	Left mandibular canine
De Matos et al. [45]	fMRS 3 T	13 (13 male)	Electric pain (4–5 out of 10 on a VAS)	Right maxillary canine

VAS means Visual Analog Scale, NRS means Numeric Rating Scale

upper central incisor tooth, the lower lip, and the tongue. They used a rubber tip and administered the stimuli manually. Clearly overlapping S1 activity was observed with a lip-tooth-tongue gradient from superior to inferior in rostral S1 subareas of their activity cluster. Important to note only the contralateral S1 region was investigated; therefore it is not possible to compare their results with the Jantsch/Brügger/Kubo reports as they observed a general bilateral activation pattern but with lateralization tendencies. Importantly, the report by Brügger et al. [31, p. 1506] demonstrated a robust bilateral activation pattern with further contralateral tendencies in the thalamus, in the posterior insula, around the parietal operculum (BA 43), and surprisingly in the amygdala. The finding of increased contralateral activation of the amygdala has to be specifically brought to attention as it is the only experimental human pain report demonstrating such a pattern. For example, a review by Baas et al. [46, p. 1102], summarizing 54 studies with amygdala activity, revealed no clear lateralization effect and highlighted the main functional contribution of the amygdala in processing primarily negative affective states such as fear and anxiety but no somatosensory encoding properties. On the other hand, Neugebauer [47, p. 1104] and Neugebauer [48, p. 1103] found evidence in rats that this structure consists of a so-called nociceptive amygdala located in the latero-capsular division of the central nucleus which directly processes sensory input. Yet, in humans, this has yet to be clarified although the study by Brügger and colleagues opened the window toward the amygdala's possible direct involvement in decoding somatotopic information. A possible explanation of this finding might be that tooth pain induces higher levels of threat/anxiety than pain originating from other parts of the human body, requiring the aberrant recruitment of additional brain structures to somato-topically encode the afferent sensory signals. A recent report by Meier et al. [49, p. 1436] substantiated this presumption by demonstrating enhanced conditioned fear induced by a short tooth pain stimulus compared to pain administered to the tibia. However, it must not be forgotten that functional measurements of such small subareas require specific imaging strategies especially when the amygdala is targeted. Mainly, this is due to its central localization, the surrounding vasculature and bordering cerebrospinal fluid. Those facts are accompanied by strong phase-encoding susceptibility inferences leading to false positive and negative activation patterns unrelated to a specific stimulation or task [50, p. 1101, 51, p. 1578].

Besides the information of "where" does it hurt and the "how much" does it hurt is—at least—of comparable importance. There is one report addressing this question directly by applying five different stimulus strengths to a right maxillary canine, whereas two were painless, and the remaining three were painful [41, p. 84]. Also, the study of Jantsch et al. [34, p. 683] can be interpreted as intensity coding as they applied "weak" and "strong" pain, however, no painless stimuli were used. An alternative approach was used by Trulsson et al. [38, p. 1073] also focusing on "intensity coding" by applying tactile stimulation of different frequencies to the left maxillary incisor, but no painful stimulation specifically. Cortical correlates of somatosensory intensity coding have been demonstrated across the literature, most particularly in the subareas of the insular and cingulate cortices (i.e., [52, p. 916, 53, p. 1144, 54, p. 919]). Beginning with the study of Brügger et al. [41, p. 84] which applied the whole range of perception

from painless to painful, the anterior insula together with two cingulate cortex subareas, namely, the anterior mid- and pregenual anterior cingulate cortex, demonstrated a significant linear relation between applied stimulus strengths and activity levels. The observation that the insular cortex plays a crucial role in intensity coding was also demonstrated by the Jantsch study, however; the insula also showed a stronger activity pattern during the hand stimulation, which may be due to the varying modalities used in their paradigm (electrical for tooth and mechanic for the index finger). Interestingly, the Trulsson study revealed generally stronger activity when applying higher tactile stimulus frequencies (100 Hz) and additional insular-opercular subarea activity, but only contralateral to the stimulation site. The same pattern was observed in the right cerebellar cortex. Generally, these results fit well into known cortical response patterns to stimuli of extra-trigeminal origin. But recent elaborations of cortical systems coding specifically for different strengths of somatosensory input suggest a multisensory magnitude—instead of a specific pain-related assessment module, particularly within the insular cortex [21, p. 1091, 55, p. 417, 56, p. 920, 57, p. 1415, 58, p. 206]. In our opinion, this line of reasoning is understandable, as somatosensory stimulation of, for example, the human back induces a multitude of perceptions due to a variety of different receptor types transmitting the whole range of sensory modalities. On the other hand, teeth are unique "organs" consisting of hard mineralized material surrounding densely innervated and vascularized soft tissue located within the tooth pulp. The pulp itself is predominantly innervated by C and A-delta fibers, implying that neural inputs are (1) of mostly nociceptive characteristic and (2) of rather homogenous perceptive quality [31, p. 1506, 59, p. 897]. This physiological specificity makes the tooth an ideal stimulation target in order to investigate more thoroughly the "pure pain perception" and probably also the processing of the intensity level of pain. Two studies can be considered in this vein: [42, p. 46] and [44, p. 1504]. The first investigated patients suffering from dental hypersensitivity in response to an application of an air stimulus sufficiently strong to evoke pain. A sensitive as well as an insensitive tooth were stimulated and patients were required to focus selectively on their intensity perception. Surprisingly, intensity coding related activity was observed in a multitude of areas, including anterior insular and mid-cingulate subareas (see results section for details). Those two regions were also found to specifically code for the sensitive tooth with clearly stronger activity, providing the evidence that these areas seem to have pain specific functions beside the intensity coding properties. To substantiate this finding, a follow-up study by the same group addressed this issue with an elegant approach [44, p. 1504]. Using electric stimuli at a constant intensity applied to the left mandibular canine, they injected an anesthetic drug (articaine) to block afferent signaling transmission while the stimulation continued at the same intensity level. Over time a gradual pain decrease was perceived despite the ongoing painful stimulation. The specific brain response pattern in reaction to the articaine-induced dental pain relief was observed in a small portion of the left posterior insula, reiterating the critical role of this brain area in coding pain specificity and related intensity coding.

From a neurochemistry perspective, there is strong evidence subserving the idea of the insula is key structure within the cortical dental pain circuitry derived from two studies using fMRS (functional magnetic resonance spectroscopy) [40, p. 882, 43, p. 1152]. In the first attempt, the whole left insular cortex was measured during

continuous stimulation of the right maxillary canine. Significant increases were measureable in the levels of glutamine (Gln) and the glutamine-glutamate complex (Glx) together with a significant drop in myo-inositol (mI). The second report investigated the insular cortex bilaterally, and they subclassified the insula into an anterior and posterior portion using the same paradigm. Comparable to the first study, Glx, Gln, and also glutamate (Glu) showed a significant increase during the pain stimulation phase, whereas mI significantly dropped. This pattern was observed in all four subareas. An interesting effect was found in significant differences between left and right insular subareas irrespective of stimulation or rest. As the insular cortex is incorporated in a manifold of different cortical functions, this fundamental disparity might support the suggestion that there are inherent functional differences between the subareas as pointed out by several investigations [20, p. 980, 60, p. 976] or [54, p. 919]. Neurochemical alterations within subareas of the right insular cortex have been shown by applying heat pain to an inner left forearm area. However, they measured a dorsal-anterior area, thus a rather evaluative-cognitive region than sensory encoding. Regardless, they revealed partly comparable reaction patterns with respect to increased Glu levels. None of the other metabolites demonstrated a pattern related with the stimulation. The stimulus related measurement of neurochemistry (event-related MRS or functional MRS) is complementary to fMRI in brain imaging. To date, only four studies have applied experimental pain while measuring changes in neurochemical compositions, thus, a lot of ambiguity remains that has to be investigated in more details.

The group of De Matos et al. [45] attempted a closer investigation into the fundamental nociceptive processing within the CNS. Applying the paradigm used in other studies by this group, the brainstem trigeminal nuclear complex (BTNC) was targeted while administering painful electrical stimuli to the right maxillary canine and neurochemical alterations during pain vs baseline were assessed. The BTNC constitutes the first CNS relay along the peripheral-central signaling cascade and therefore enables the investigation of pain-related processing at a very initial level. As the main result, a significant decrease in NAA and GABA during experimental orofacial stimulation was found. To date, a conclusive summary regarding this neurochemical pattern is not advocated by the authors as the results need clarification by further investigations. While of particular interest regarding early nociceptive processing, the study demonstrated above all, the possibility to measure the human brainstem neurochemistry with high accuracy, thus paving the way to a better understanding of this important brain area in the context of acute pain processing as well as pain related chronification mechanisms.

6.4 Summary

Pain is a multidimensional experience incorporating sensory, motor, affective and cognitive components. This applies to pain in general, as well as to orofacial pain in particular. This chapter provided an overview of neural signatures based on experimental acute orofacial pain up to explanatory approaches possibly underlying chronification mechanisms and chronic pain.

Summarized, brain responses of acute orofacial pain are quite well characterized based on sophisticated experimental models combined with imaging methods such as fMRI, fMRS, MEG and EEG. This strategy is important as such experiments allow standardized and controlled application of pain stimuli. In this way it is possible to understand the fundamental neural processes of experiencing pain, which in turn is the prerequisite for understanding the much more complex chronic pain. Concluded, these experiments revealed that several brain areas, often termed "Pain Matrix" or "Neurological Signature of Pain", representing the neural framework regarding the multidimensional facets of an acute pain experience.

Still a challenge to understand are neural underpinnings of chronic pain and associated mechanisms that facilitates the transition from acute to subacute and finally chronic pain. Recent investigations suggest that chronic pain involves additional areas not known as classic pain areas (hippocampus) or propose a highly "Dynamic Pain Connectome" linking attention and pain related brain areas such as Salience-, Default Mode and Antinociceptive networks.

Recent years of intensive basic and clinical research has not yet brought the solution, but we are on good terms to comprehend the basic processes of pain chronification better and better. Including multimodal approaches that measure and quantify different facets of brain function, together with improved analytical methods (i.e. deep learning and big data management), a much better understanding of this highly significant global health problem is closer than ever before.

References

1. Penfield W, Boldrey E. Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. London: Macmillan; 1937.
2. Penfield W. Engrams in the human brain: mechanisms of memory. Proc Roy Soc Med. 1968;61:831–40.
3. Penfield W, Jasper H. Epilepsy and the functional anatomy of the human brain. Boston: Brown L; 1954.
4. Peyron R, Laurent B, García-Larrea L. Functional imaging of brain responses to pain. A review and meta-analysis (2000). Neurophysiol Clin. 2000;30:263–88.
5. Wager TD, Atlas LY, Lindquist MA, Roy M, Woo C-W, Kross E. An fMRI-based neurologic signature of physical pain. N Engl J Med. 2013;368:1388–97.
6. Bushnell MC, Duncan GH, Hofbauer RK, Ha B, Chen JI, Carrier B. Pain perception: is there a role for primary somatosensory cortex? Proc Natl Acad Sci U S A. 1999;96:7705–9.
7. Denk F, McMahon SB, Tracey I. Pain vulnerability: a neurobiological perspective. Nat Neurosci. 2014;17:192–200.
8. Moayedi M, Davis KD. Theories of pain: from specificity to gate control. J Neurophysiol. 2013;109:5–12.
9. Tracey I, Mantyh PW. The cerebral signature for pain perception and its modulation. Neuron. 2007;55:377–91.
10. Kucyi A, Davis KD. The dynamic pain connectome. Trends Neurosci. 2015;38:86–95.
11. Haggard P, de Boer L. Oral somatosensory awareness. Neurosci Biobehav Rev. 2014;47:469–84.
12. Scholz J, Woolf CJ. Can we conquer pain? Nat Neurosci. 2002;5:1062–7.
13. Minde J, Svensson O, Holmberg M, Solders G, Toolanen G. Orthopedic aspects of familial insensitivity to pain due to a novel nerve growth factor beta mutation. Acta Orthop. 2006;77:198–202.
14. Losa M, Scheier H, Rohner P, Sailer H, Hayek J, Giedion A, Boltshauser E. Langzeitverlauf bei kongenitaler Analgesie. Schweiz Med Wochenschr. 1989;119:1303–8.

15. Baliki MN, Chialvo DR, Geha PY, Levy RM, Harden RN, Parrish TB, Apkarian AV. Chronic pain and the emotional brain: specific brain activity associated with spontaneous fluctuations of intensity of chronic back pain. J Neurosci. 2006;26:12165–73.
16. Brown RS, Arm RN, Epstein JB. Diagnosis and treatment of chronic orofacial pain, 2nd edn. In: Clinician's guide. Hamilton, ON: BC Decker; 2008.
17. Apkarian AV, Baliki MN, Geha PY. Towards a theory of chronic pain. Prog Neurobiol. 2009;87:81–97.
18. Vachon-Presseau E, Tetreault P, Petre B, Huang L, Berger SE, Torbey S, Baria AT, Mansour AR, Hashmi JA, Griffith JW, Comasco E, Schnitzer TJ, Baliki MN, Apkarian AV. Corticolimbic anatomical characteristics predetermine risk for chronic pain. Brain. 2016;139:1958–70.
19. Treede R-D, Jensen TS, Campbell JN, Cruccu G, Dostrovsky JO, Griffin JW, Hansson P, Hughes R, Nurmikko T, Serra J. Neuropathic pain—redefinition and a grading system for clinical and research purposes. Neurology. 2008;70:1630–5.
20. Craig ADB. How do you feel—now? The anterior insula and human awareness. Nat Rev Neurosci. 2009;10:59–70.
21. Legrain V, Iannetti GD, Plaghki L, Mouraux A. The pain matrix reloaded: a salience detection system for the body. Prog Neurobiol. 2011;93:111–24.
22. van Ryckeghem DML, Crombez G, Eccleston C, Legrain V, van Damme S. Keeping pain out of your mind: the role of attentional set in pain. Eur J Pain. 2013;17:402–11.
23. Kucyi A, Davis KD. The neural code for pain: from single-cell electrophysiology to the dynamic pain connectome. Neuroscientist. 2017;23(4):397–414.
24. Davis KD, Kucyi A, Moayedi M. The pain switch: an "ouch" detector. Pain. 2015;156:2164–6.
25. Mutso AA, Petre B, Huang L, Baliki MN, Torbey S, Herrmann KM, Schnitzer TJ, Apkarian AV. Reorganization of hippocampal functional connectivity with transition to chronic back pain. J Neurophysiol. 2014;111:1065–76.
26. Napadow V, LaCount L, Park K, As-Sanie S, Clauw DJ, Harris RE. Intrinsic brain connectivity in fibromyalgia is associated with chronic pain intensity. Arthritis Rheum. 2010;62:2545–55.
27. Baliki MN, Baria AT, Apkarian AV. The cortical rhythms of chronic back pain. J Neurosci. 2011;31:13981–90.
28. Kucyi A, Moayedi M, Weissman-Fogel I, Goldberg MB, Freeman BV, Tenenbaum HC, Davis KD. Enhanced medial prefrontal-default mode network functional connectivity in chronic pain and its association with pain rumination. J Neurosci. 2014;34:3969–75.
29. Hutchison RM, Womelsdorf T, Gati JS, Everling S, Menon RS. Resting-state networks show dynamic functional connectivity in awake humans and anesthetized macaques. Hum Brain Mapp. 2013;34:2154–77.
30. Farmer MA, Baliki MN, Apkarian AV. A dynamic network perspective of chronic pain. Neurosci Lett. 2012;520:197–203.
31. Brügger M, Ettlin DA, Meier M, Keller T, Luechinger R, Barlow A, Palla S, Jäncke L, Lutz K. Taking sides with pain—lateralization aspects related to cerebral processing of dental pain. Front Human Neurosci. 2011;5:12.
32. Lin C-S. Brain signature of chronic orofacial pain: a systematic review and meta-analysis on neuroimaging research of trigeminal neuropathic pain and temporomandibular joint disorders. PLoS One. 2014;9:e94300.
33. Ettlin DA, Zhang H, Lutz K, Järmann T, Meier D, Gallo LM, Jäncke L, Palla S. Cortical activation resulting from painless vibrotactile dental stimulation measured by functional magnetic resonance imaging (FMRI). J Dent Res. 2004;83(10):757–61.
34. Jantsch HHF, Kemppainen P, Ringler R, Handwerker HO, Forster C. Cortical representation of experimental tooth pain in humans. Pain. 2005;118:390–9.
35. Miyamoto JJ, Honda M, Saito DN, Okada T, Ono T, Ohyama K, Sadato N. The representation of the human oral area in the somatosensory cortex: a functional MRI study. Cereb Cortex. 2006;16:669–75.
36. Kubo K, Shibukawa Y, Shintani M, Suzuki T, Ichinohe T, Kaneko Y. Cortical representation area of human dental pulp. J Dent Res. 2008;87:358–62.
37. Brügger M, Ettlin DA, Keller T, Luechinger R, Jäncke L, Palla S, Barlow A, Gallo LM, Lutz K. Interindividual differences in the perception of dental stimulation and related brain activity. Eur J Oral Sci. 2009;117:27–33.

38. Trulsson M, Francis ST, Bowtell R, McGlone F. Brain activations in response to vibrotactile tooth stimulation: a psychophysical and fMRI study. J Neurophysiol. 2010;104:2257–65.
39. Weigelt A, Terekhin P, Kemppainen P, Dörfler A, Forster C. The representation of experimental tooth pain from upper and lower jaws in the human trigeminal pathway. Pain. 2010;149:529–38.
40. Gutzeit A, Meier D, Meier ML, von Weymarn C, Ettlin DA, Graf N, Froehlich JM, Binkert CA, Brügger M. Insula-specific responses induced by dental pain. A proton magnetic resonance spectroscopy study. Eur Radiol. 2011;21:807–15.
41. Brügger M, Lutz K, Brönnimann B, Meier ML, Luechinger R, Barlow A, Jäncke L, Ettlin DA. Tracing toothache intensity in the brain. J Dent Res. 2012;91:156–60.
42. Meier ML, Brügger M, Ettlin DA, Luechinger R, Barlow A, Jäncke L, Lutz K. Brain activation induced by dentine hypersensitivity pain—an fMRI study. J Clin Periodontol. 2012;39:441–7.
43. Gutzeit A, Meier D, Froehlich JM, Hergan K, Kos S, V Weymarn C, Lutz K, Ettlin D, Binkert CA, Mutschler J, Sartoretti-Schefer S, Brügger M. Differential NMR spectroscopy reactions of anterior/posterior and right/left insular subdivisions due to acute dental pain. Eur Radiol. 2013;23:450–60.
44. Meier ML, Widmayer S, Abazi J, Brügger M, Lukic N, Lüchinger R, Ettlin DA. The human brain response to dental pain relief. J Dent Res. 2015;94(5):690–6.
45. de Matos NMP, Hock A, Wyss M, Ettlin DA, Brügger M. Neurochemical dynamics of acute orofacial pain in the human trigeminal brainstem nuclear complex. Neuroimage. 2017;162:162–72.
46. Baas D, Aleman A, Kahn RS. Lateralization of amygdala activation: a systematic review of functional neuroimaging studies. Brain Res Rev. 2004;45:96–103.
47. Neugebauer V, Li W. Differential sensitization of amygdala neurons to afferent inputs in a model of arthritic pain. J Neurophysiol. 2003;89:716–27.
48. Neugebauer V, Li W, Bird GC, Han JS. The amygdala and persistent pain. Neuroscientist. 2004;10:221–34.
49. Meier ML, de Matos NMP, Brügger M, Ettlin DA, Lukic N, Cheetham M, Jäncke L, Lutz K. Equal pain-unequal fear response: enhanced susceptibility of tooth pain to fear conditioning. Front Hum Neurosci. 2014;8:526.
50. Mathiak KA, Zvyagintsev M, Ackermann H, Mathiak K. Lateralization of amygdala activation in fMRI may depend on phase-encoding polarity. Magma. 2012;25:177–82.
51. Boubela RN, Kalcher K, Huf W, Seidel E-M, Derntl B, Pezawas L, Našel C, Moser E. fMRI measurements of amygdala activation are confounded by stimulus correlated signal fluctuation in nearby veins draining distant brain regions. Sci Rep. 2015;5:10499.
52. Vogt BA. Pain and emotion interactions in subregions of the cingulate gyrus. Nat Rev Neurosci. 2005;6:533–44.
53. Nieuwenhuys R, Voogd J, van Huijzen C. The human central nervous system. 4th ed. Berlin: Springer; 2008.
54. Kurth F, Zilles K, Fox PT, Laird AR, Eickhoff SB. A link between the systems: functional differentiation and integration within the human insula revealed by meta-analysis. Brain Struct Funct. 2010;214:519–34.
55. Baliki MN, Geha PY, Apkarian AV. Parsing pain perception between nociceptive representation and magnitude estimation. J Neurophysiol. 2009;101:875–87.
56. Mazzola L, Isnard J, Peyron R, Mauguière F. Stimulation of the human cortex and the experience of pain: Wilder Penfield's observations revisited. Brain. 2012;135:631–40.
57. Pomares FB, Faillenot I, Barral FG, Peyron R. The 'where' and the 'when' of the BOLD response to pain in the insular cortex. Discussion on amplitudes and latencies. NeuroImage. 2013;64:466–75.
58. Mouraux A, Diukova A, Lee MC, Wise RG, Iannetti GD. A multisensory investigation of the functional significance of the "pain matrix". NeuroImage. 2011;54:2237–49.
59. Sessle BJ. Peripheral and central mechanisms of orofacial inflammatory pain. Int Rev Neurobiol. 2011;97:179–206.
60. Craig ADB. The sentient self. Brain Struct Funct. 2010;214:563–77.

Part II

Pathophysiology, Clinical Evaluation, Imaging and Diagnosis

Pathophysiology of Temporomandibular Disorders

7

B. Stegenga*

Abstract

Temporomandibular disorder is the term used for the musculoskeletal disorders of the jaw system, which comprises the temporomandibular joints and its associated musculature. In the past decades, several concepts on the pathology, diagnosis, and management of temporomandibular disorders have been proposed, which have resulted in several classifications of these disorders. The most commonly used is the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) (Schiffman et al., J Oral Facial Pain Headache 28:6–27, 2014). In this classification, TMJ disorders are distinguished from masticatory muscle disorders, although these categories commonly coexist. Within the category of TMJ disorders, pain (arthralgia, arthritis) and disorders (internal derangements, including disc interferences, adhesions, ankylosis, hypermobility) usually represent manifestations of TMJ disease, which include arthritic diseases and growth disorders. This chapter focuses on the pathophysiologic processes occurring in the most common group of joint disorders, i.e., TMJ degenerative diseases (Stegenga, J Oral Rehabil 37:760–765, 2010).

7.1 Conceptual Approaches

The temporomandibular joint is classified as a complex synovial joint. It is termed "complex" due to the presence of an articular disc, which separates the intra-articular space into two compartments. Essentially it consists of several

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interdependent connective tissues, each with its specific adaptive capacity, which is the capacity to adapt to functional changes by remodeling. Pathologic changes may affect all joint tissues, including the articular cartilage, the disc, the subchondral bone, and the synovial membrane.

The appreciation of the importance of specific joint structures has considerable influence on the approach toward classification of TMJ disorders and consequently on their diagnosis and management. The presence of the disc has focused the attention on the joint's mechanical aspects. In this approach the primary focus is on a joint that is not working properly, in terms of impaired gliding and obstructions, especially by the disc in internal derangements. In this conceptual approach, degenerative changes are frequently regarded as the result of the mechanical derangement.

Based on the idea that the temporomandibular joint is affected by the same pathologic processes and diseases as the other synovial joints and supported by clinical observations in a large group of patients [1] and their long-term follow-up [2] and detailed observations of pathologic changes in articular cartilage [3] and the synovial membrane [4], we conceptually described TMJ osteoarthritis as a "whole joint" disease back in 1989 [5]. The focus was placed on pathological changes that begin whenever the connective tissues making up the joint have not successfully adapted to the demands imposed to them, eventually resulting in pain and function impairment. During the past 25 years, there has been a massive expansion of our understanding regarding the various pathologic events that take place in the development of synovial joint degenerative diseases, in general, and of TMJ degenerative diseases in particular. These findings support the adoption of the "joint is an organ" concept [6].

Osteoarthritis is the clinical and pathological outcome of a range of disorders and conditions that lead to pain, disability, and structural failure in synovial joints. Therefore, usually primary osteoarthritis (which may be localized or generalized when three or more joint sites are involved) is distinguished from secondary osteoarthritis, which follows a clearly defined predisposing disorder or disease. Throughout the years, besides the genetic background, mechanical and psychological stresses have been consistently connected to pain conditions and function impairment associated with the temporomandibular joints and masticatory muscles. An important notion that may unify the general thinking about synovial joint diseases is that there is a dynamic balance between the loads imposed on a tissue or system and its adaptive capacity, which results in ongoing structural changes aimed at enabling the tissue to optimally withstand the loads and functional demands [7].

7.2 Etiology and Risk Factors

The development of all types of TMJ degenerative diseases is associated with multiple etiological and risk factors. The primary etiological factor is usually unknown, but it is likely that one or more of the risk factors mentioned in Table 7.1 play a role in TMJ degenerative disease, supporting that this is a complex and probably multifactorial joint condition. Progress has been made in identifying mutations in

Table 7.1 Risk factors for TMJ degenerative disease

Trauma
Parafunctions
Functional overloading
Aging
Systemic disease
Hormonal factors

collagen genes that are associated with different types of bone and cartilage dysplasia where osteoarthritis is part of a more complex phenotype, but none of the single-gene mutations that code for structural matrix proteins appears to be important in determining the susceptibility to the common types of osteoarthritis [8].

Whatever primary factors may be involved, it is essential to note that they result in a disturbance of the balance between synthesis and breakdown. The adaptive capacity is insufficient to withstand the loads, which is expressed in a relative surplus of breakdown products. The balance may become disturbed due to excessive physical stress, such as a traumatic event, or due to reduction of the adaptive capacity of the tissues involved, for example, due to a systemic disease. This explains that a joint may become overloaded even when the amount of loading is normal [9, 10].

Although many etiologic factors may be important in the etiopathogenesis of the disease, mechanical stress appears to play a critical role in the events that lead to the initiation and progression of osteoarthritic diseases. There are at least two mechanisms through which mechanical loads can trigger molecular events that may lead to degenerative disease in susceptible individuals. The first is the production of reactive free radicals [11, 12]; the second is the stimulation of sensory neurons resulting in release of neuropeptides [13, 14].

7.3 Biochemical Responses

Compressive loading not only may lead to direct damage of the loaded tissues but also may disturb synovial capillary perfusion. This induces a relative hypoxia, which on reperfusion is followed by the generation of highly reactive free radicals (oxidative stress). Normally, scavengers neutralize these radicals to prevent damage to occur. When the free radicals exceed the concentration of scavengers, chondrocyte apoptosis may be the result, and damage to the articular tissues and to the molecules in the synovial fluid may occur [15]. Further research is needed to support (or reject) that this hypoxia-reperfusion injury actually occurs in TMJ osteoarthritis [16]. The other mechanism concerns mechanically stimulated sensory nerves releasing neuropeptides, such as substance P and calcitonin gene-related peptide, which produce several cytokines, nitric oxide, and other molecules that contribute to an inflammatory response. In a recent study, it was suggested that hyaluronic acid may inhibit substance P and CGRP expression in the TMJ [17].

Since pain is one of the cardinal symptoms of TMJ degenerative disease, the importance of inflammation in the progression of the disease has received considerable attention. In painful joints, prostaglandin E_2 and COX-2, which are important

for the production of prostaglandins, appear to be detectable [18]. However, concentrations of prostaglandin E_2 and several other markers were not significantly increased in the synovial fluid of patients with TMJ osteoarthritis compared with healthy controls [16]. It has been suggested that celecoxib (which is a selective COX-2 inhibitor) has protective effects on condylar chondrocytes [19].

A growing number of inflammatory cytokines (e.g., interleukin (IL)-1-beta, IL-6, IL-12, and tumor necrosis factor-alpha) that are produced by macrophages and synovial cells have been identified in the synovial fluid of patients with TMJ degenerative diseases [20, 21]. Synovial fluid levels of IL-1-beta and IL-6 appear to correlate with the degree of pain and synovitis. IL-1 and TNF-alpha play an important role in the upregulation and activation of matrix-degrading enzymes [22–24].

The most important matrix-degrading enzymes found in osteoarthritic joints include aggrecanases and collagenases, which are members of the matrix metalloproteinase (MMP) family. The enzyme activity is not only controlled by various cytokines but also by steroid hormones and by specific inhibitory molecules (the so-called tissue inhibitors of metalloproteinases). Excessive mechanical stress has been shown to activate the plasminogen activator system, which may lead to proteolysis of extracellular matrix components [25]. Type I collagen is the primary component of TMJ articular cartilage, which can be degraded by several types of MMPs and by cathepsin K. Aggrecan is a proteoglycan which is critical in imbibing water into the matrix, thereby giving the joint surface the ability to withstand compressive forces. Degradation of aggrecan (by several MMPs and aggrecanases, such as ADAM-TS-4 and ADAM-TS-5) has been shown to occur early in the osteoarthritic process [26]. With the degradation of cartilage matrix proteins, fragments are produced that can stimulate the production of inflammatory cytokines and MMPs and further matrix destruction (Fig. 7.1).

Several authors have shown an increase in vascular endothelial growth factor (VEGF) expression in diseased TMJs following mechanical overloading and hypoxia [27, 28]. It is known that angiogenesis is stimulated by metabolic stress (e.g., due to hypoxia), mechanical stress, inflammation, and alteration in hormonal levels, and these are all factors that play a role in the susceptibility for or in the etiopathogenesis of osteoarthritis.

An increasing number of studies have focused on the significance of subchondral bone in the pathogenesis of TMJ degenerative disease. The chondrocytes of degraded cartilage influence osteoclastogenesis by affecting the ratio of receptor activator of nuclear factor kappaB ligand (RANKL) and osteoprotegerin (OPG), resulting in subchondral bone loss and turnover [29, 30]. Transforming growth factor (TGF) beta-1 has been suggested to play an initiating role in decreasing bone mineral density and increasing subchondral bone turnover [31], which is frequently observed in early stages of in TMJ degenerative disease.

The female preponderance and the occurrence of TMJ degenerative diseases mainly during the reproductive years [32] suggest a possible role of female hormones in the pathogenesis. In a rat model, Wang et al. [33] showed that estrogen aggravates the degradation of cartilage and destruction of subchondral bone, which could be inhibited by an estrogen receptor antagonist. Studies on the role of estrogen and effects of other female hormones should be further evaluated.

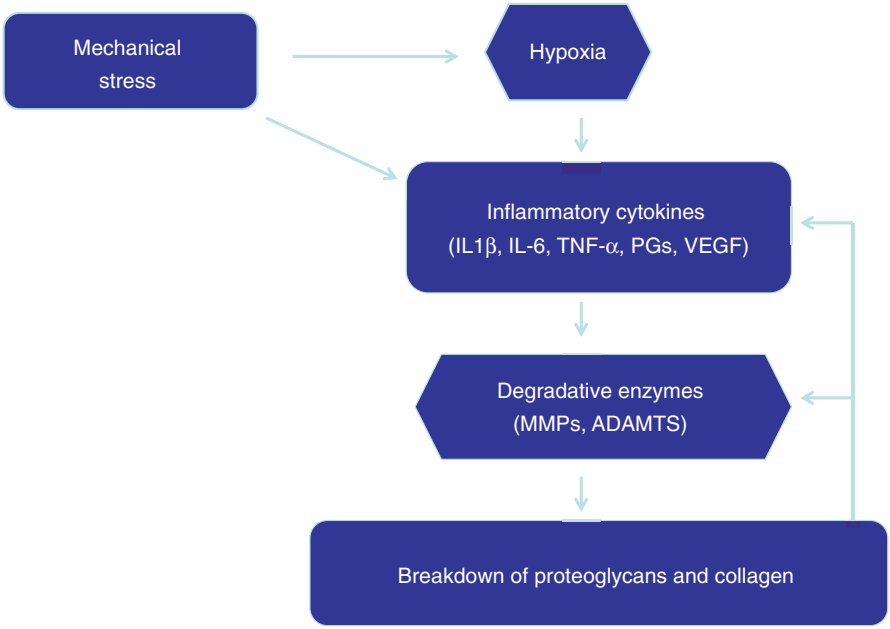

Fig. 7.1 Biochemical responses involved in TMJ osteoarthritis disease. *IL* interleukin, *TNF* tumor necrosis factor, *PG* prostaglandin, *VEGF* vascular endothelial growth factor, *MMP* matrix metalloproteinases, *ADAMTS* a disintegrin and metalloproteinase with thrombospondin motifs. To summarize, several key cytokines and degradative enzymes have been identified, setting up a biochemical cascade that represents the complex processes that occur in a pathologic joint, and sets up a vicious cycle that is potentially destructive for the joint. Despite the identification of these events, the number of properly controlled studies is still limited. Nevertheless, we seem to be on the threshold of the identification of TMJ degeneration biomarkers that can be easily detected in saliva, blood and urine. Theoretically, the explosion of knowledge is also interesting from a therapeutic perspective, although trials involving anticytokine therapy have produced disappointing results in osteoarthritis so far. I will come back to this issue later

To summarize, several key cytokines and degradative enzymes have been identified, setting up a biochemical cascade that represents the complex processes that occur in a pathologic joint and sets up a vicious cycle that is potentially destructive for the joint. Despite the identification of these events, the number of properly controlled studies is still limited. Nevertheless, we seem to be on the threshold of the identification of TMJ degeneration biomarkers that can be easily detected in saliva, blood, and urine.

The relative increase of breakdown products within the joint leads to natural attempts at repair, which have to compete with ongoing damaging events. Initially chondrocyte activation and proliferation of clusters of chondrocytes are associated with anabolic responses with increased synthesis and turnover of matrix collagens and proteoglycans. Anabolic mediators include growth factors (e.g., insulin-like growth factor, fibroblast growth factor, transforming growth factor β) and bone morphogenetic proteins (BMPs): the anti-inflammatory cytokine interleukin-4 and proteinase inhibitors such as TIMPs and plasminogen activator inhibitor. In many cases

these processes reach a state of nonprogressive equilibrium. However, catabolism of cartilage matrix proteins may outstrip the capacity for cartilage repair, leading to decreased cartilage thickness and chondrocyte apoptosis. Catabolic mediators include nitric oxide, prostaglandins, and the pro-inflammatory cytokines IL-1β, tumor necrosis factor α, IL-6, and IL-7, as well as metalloproteinases and aggrecanases (ADAM-TS-4 and ADAM-TS-5). When damaging events are allowed to go on and repair and adaptive attempts are insufficiently successful, the disease gradually progresses from local damage to an interactive combination of degenerative changes and inflammatory responses and, eventually, to adhesion formation and radiographically visible degenerative changes. So, the vicious cycle of breakdown has to be interrupted to promote healing and to prevent further damage to occur.

7.4 Pathological Changes

Pathological changes may become obvious within all the tissues making up the joint. Microscopic breakdown of the articular cartilage in the early stages of osteoarthritis starts with clustering of chondrocytes. As the disease progresses, further matrix depletion occurs, affecting the proteoglycans and altering the collagen fiber architecture. This results in softening of the cartilage, which loses its normal resilience and capability of absorbing loading. The articular surface subsequently undergoes vertical and horizontal splitting, fibrillation, and thinning. Figure 7.2a shows a microscopic picture of a TMJ with the disc in a normal position. The enlarged view (Fig. 7.2b) shows signs of cartilage splitting, as well as early degenerative changes within the subchondral bone, i.e., fibrosis in the bone marrow spaces. Thus, early changes occur not only within the cartilage (especially in the deeper layers) but also within the subchondral bone. This can be explained by the changes occurring at the cartilage-subchondral bone interface. Chondrocytes become hypertrophic and produce more growth factors, such as vascular endothelial growth factor, and less sulfated glycosaminoglycans. The thickness of the cartilage reduces as a result and is increasingly depleted from proteoglycans. Cytokines produced by osteoblasts may

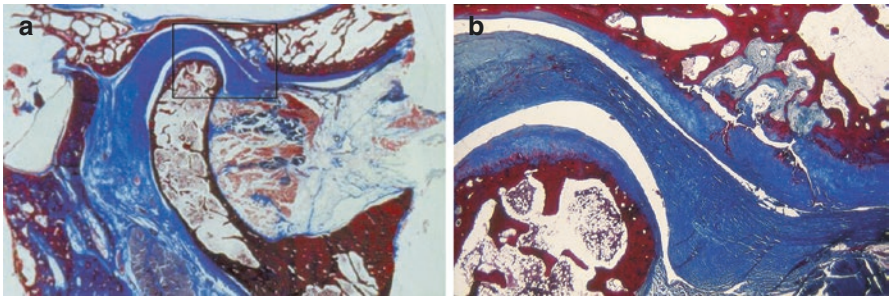

Fig. 7.2 (**a**) Histologic sagittal coupe of a right temporomandibular joint. (**b**) Enlargement of the area depicted in (**a**), showing signs of osteoarthritis in cartilage (vertical and horizontal splitting of cartilage) and subchondral bone (fibrosis of bone marrow)

diffuse to the cartilage region, while osteoclast activity and vascular ingrowth are increased due to growth factors that are produced by chondrocytes and diffuse to the bone marrow region [34].

The biomechanical properties of the cortical and subchondral bone play an important role in protecting articular cartilage following impact loading. The pathogenesis of osteoarthritis may in some cases be initiated by an increase in the density and stiffness of the subchondral bone following the healing of microfractures caused by unprotected loading of joints [35]. The consequent loss of bone viscoelasticity results in steep stiffness gradients in the bone. This in turn results in stretching and fibrillation of the overlying articular cartilage as well as focal osteonecrosis and the formation of bone cysts.

In inflamed joints, the compressive and tensile moduli of the TMJ disc significantly decrease, indicating that the disc becomes relatively softer [36]. This results in greater strain under the same stress, contributing to overloading and subsequent tissue breakdown. These changes are opposite to age-related biomechanical changes of TMJ discs, which imply stiffening of discs [37]. Thickening and increased stiffness of the disc with a loss of the characteristic biconcave shape may represent a defense mechanism to maintain the smoothness of the joint by compensating for the degeneration of the cartilage surface [38]. The integrity of the disc maintains the homeostasis of the joint; degenerative changes in the disc, including perforation, lead to disruption of the joint [39]. There appears to be interdependency between the integrity of the cartilage and the integrity of the disc, and that changes in either structure will have an effect on the health of the joint [38].

Important changes also occur within the synovial membrane and synovial fluid. First of all, recruited T-cells and B-cells contribute to local synovitis and neovascularization, signs of which may be observed during arthroscopy. Hyaluronic acid and lubricin have been shown to be deficient in osteoarthritic joints [40, 41]. Synovial fluid lubricates the joint and protects the articular cartilage surfaces from erosion and protein deposition. Damage of important synovial fluid molecules lowers the fluid's viscosity, which not only has consequences for the fluid as a lubricant but also disturbs the important function of the synovial fluid in nutrition as well as protection of the articular cartilage. In a study by Asakawa and co-workers [42], it was demonstrated that a compromised lubrication in the TMJ is associated with altered frictional properties, and Koolstra [43] showed that surface wear mainly occurred at the bone-supported cartilage surfaces but hardly in the articular disc.

Especially lubricin has been shown to protect against glycosaminoglycan depletion, collagen degradation, and loss of cells in the cartilage superficial zone [38, 44]. Lubricin is a large proteoglycan encoded by the gene proteoglycan 4 (Prg4) and is essential in the boundary lubrication to maintain joint integrity. It has been shown to prevent adhesion and regulate synoviocyte cell proliferation in the knee joint [45], and recently this has been shown to occur in the TMJ as well [38]. Hyaluronic acid does not have these specific protective effects, but the therapeutic benefit from lubricin appears to be enhanced by the addition of exogenous hyaluronic acid [46, 47].

7.5 Clinical Symptoms

Obviously, these pathologic changes eventually may become clinically manifest. Pain is the presenting symptom in the majority of patients, usually insidious in onset and intermittent at first, typically aching in character. Initially it is provoked by loading or movement of the joint and relieved by rest, but as the disease progresses, the pain may be more prolonged and experienced at rest and may become severe enough to wake the patient at night. A few minutes of early morning stiffness and transient stiffness (gelling) after rest are common. Pain may result from low-grade synovitis, inflammatory effusions, capsular distension, increased pressure or microfractures in the subchondral bone, hyperemia in subchondral bone (nocturnal aching), and tendinitis, myalgia, or muscle spasm.

As the disease progresses and the ligaments and the disc become involved, mechanical changes may occur, resulting in derangements within the joint that may interfere with or even interrupt smooth joint motion. Patients may develop painful or painless functional impairment due to restricted movements. Common physical signs include clicking, restriction of range of movement of the joint (due to obstruction of movement by a displaced disc, capsular fibrosis, or blocking by osteophytes), joint crepitus, periarticular tenderness, deformity, and muscle weakness and wasting. In the late stages, gross bony changes may eventually result in the loss of height of the mandibular ramus, which become manifest as asymmetry, tilting of the occlusal plane, and radiographically visible changes.

Kalladka et al. [48] summarized the etiopathogenesis of TMJ degenerative disease and the corresponding clinical features in different slow-progressive phases (Fig. 7.3). The early phase, in which there is evolution of the disease, may take 2.5–4 years on average and is clinically associated with clicking joints and intermittent locking. The intermediate phase, associated with TMJ destruction resulting in joint pain and functional limitations, lasts 0.5–1 year on average. The late (burned out) phase is the stage at which the joint tends to stabilize to a steady state.

7.6 Diagnosis

The most commonly occurring pathologic changes fit the diagnosis osteoarthritis or degenerative joint disease, which is a whole-organ disease involving biological mechanisms giving rise to pathological tissue changes and subsequent clinical manifestations. The diagram shown in Fig. 7.4 from a recent review, beautifully, summarizes the major biological mechanisms [49]:

- Cytokines such as IL-1β, TNF, and IL-6 are produced by chondrocytes and macrophages.
- Pro-MMPs, released by synoviocytes and macrophages, are cleaved into MMPs and further contribute to tissue damage.

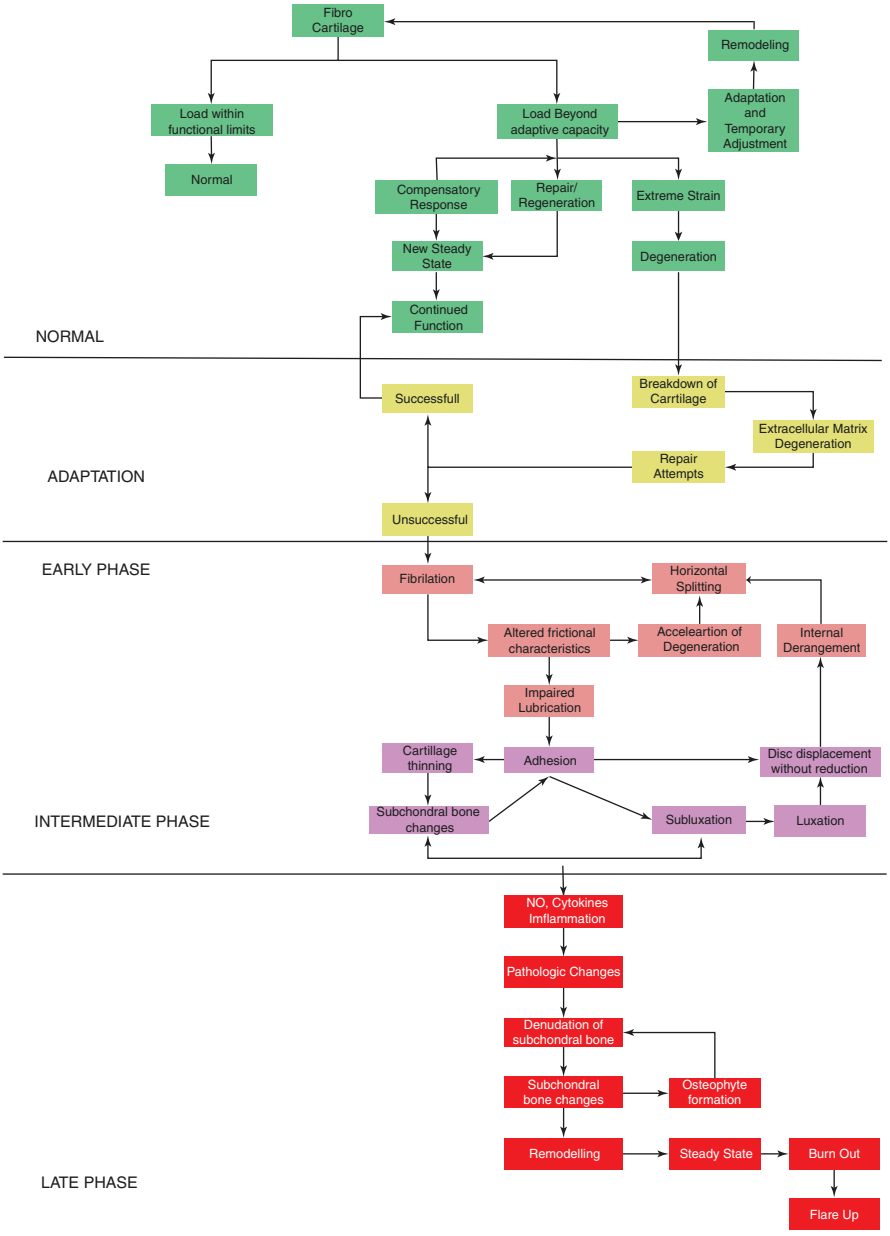

Fig. 7.3 Flowchart showing the etiopathogenesis of osteoarthritis in different phases (modified from [48])

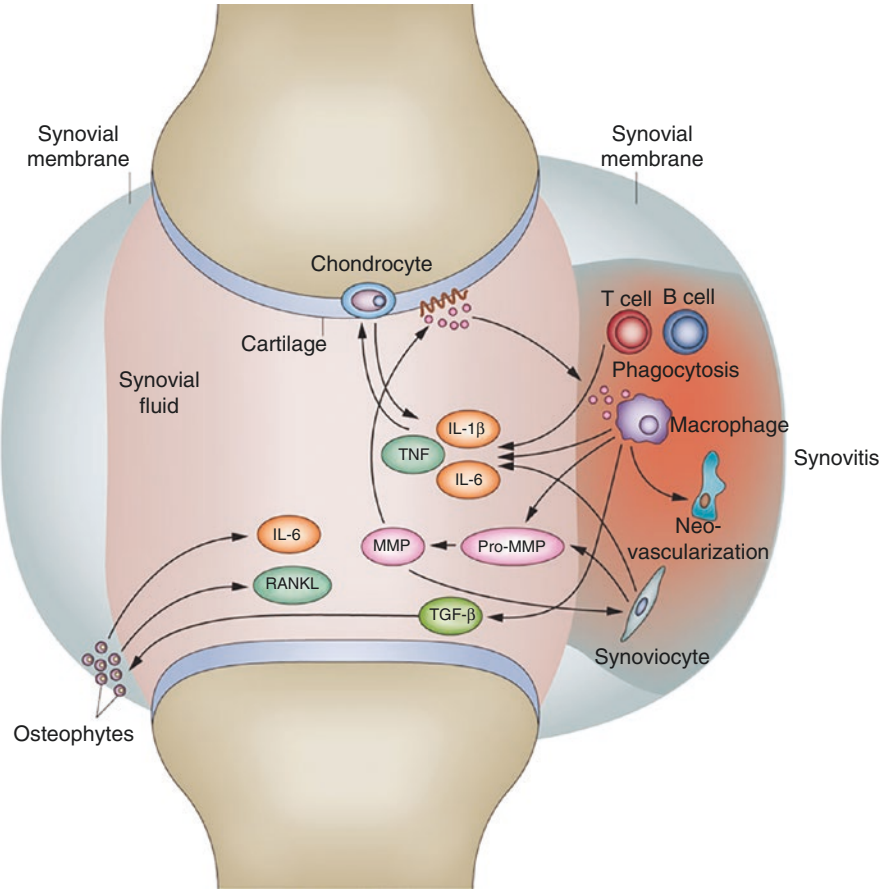

Fig. 7.4 Osteoarthritis as a whole-organ disease involving cytokine production by the cartilage, synovial membrane, and bone. Cytokines such as TNF, IL-1β, and IL-6 are produced by chondrocytes, macrophages, T-cells, and osteophytes in response to tissue damage. Pro-MMPs, released by synoviocytes and macrophages, are cleaved into MMPs and further contribute to tissue damage. T-cells and B-cells are recruited by the cytokine milieu in the synovial fluid and contribute to local synovitis. Bone cells release several cytokines, most notably IL-6 and RANKL. *MMP* matrix metalloproteinase, *RANKL* receptor activator of nuclear factor κB ligand (also known as tumor necrosis factor ligand superfamily member 11), *TGF-β* transforming growth factor β (ref. [49])

– T-cells and B-cells are recruited by the cytokine milieu in the synovial fluid and contribute to local synovitis.
– Bone cells release IL-6, and receptor activator of nuclear factor κB ligand (also known as tumor necrosis factor ligand, or RANKL) stimulates osteoclastogenesis, while growth factors stimulate osteophyte formation.

Commonly, the pathological changes include degeneration of cartilage and bone, as well as inflammatory changes. These changes lead to clinical signs and

symptoms, such as pain, interferences with smooth movement, and gross deformities. The impact on the patient may vary and depends on the extent of damage and inflammation, his or her coping skills, cognitions, emotional stability, social environment, and support. The net result is reflected by the extent of physical and psychosocial function impairment and impairment of the quality of life.

The primary conceptual focus inevitably has implications for diagnosis of temporomandibular joint diseases. When the mechanical changes are the primary focus, the position of the disc is a major concern. Most of the classification systems that have been proposed in the past emphasize the importance of disc position and are primarily designed to differentiate between anterior disc displacement with and without reduction, mechanical bone changes, accompanying joint sounds, and restriction of motion due to obstruction of the condyle by the displaced disc. During the past decades, there has been much discussion with regard to the course of internal derangements in several consecutive stages, reflecting a progression from disc displacement with reduction to a joint in which reduction does not take place anymore. This course was mainly based on retrospective and cross-sectional studies. In prospective controlled studies, it appeared that reducing disc displacement may remain unchanged throughout many years and also that a permanent disc displacement can occur without a preceding stage of disc displacement with reduction [50, 51]. In addition, it has been repeatedly established that the disc is in an anterior position in about 1/3 of asymptomatic persons, while in patients with clicking joints or with restricted movement, the disc is in a normal (i.e., nondisplaced) position [52, 53]. Moreover, clinically successful nonsurgical and minimally invasive treatment modalities do not influence the position of the disc [54, 55]. Therefore, there is a growing doubt with regard to the significance of disc position in TMJ afflictions, which makes it noteworthy that the emphasis in diagnosis is so often put on the position of the disc.

Focusing on the pathological events described in this chapter implies that the diagnostic workup is directed to changes in the articular cartilage, bone, and synovial fluid. Although cartilage degradation products (hyaluronan, keratin sulfate, cartilage oligomeric protein) and cartilage synthesis markers (collagen c-pro-peptide) have been shown to be increased in the plasma, synovial fluid, or urine of patients with osteoarthritis, there are currently no biochemical markers that have clinical utility for diagnosis, monitoring the progress of structural changes or assessing the prognosis of osteoarthritis in clinical practice. Biologic agents may have dramatic effects in rheumatic inflammatory diseases, and they were hoped to have similar effects in the treatment of degenerative joint diseases. Chevalier et al. [49] reviewed the results of several types of cytokine blockers, targeting IL-1β, TNF, and nitrogen oxide production mainly in osteoarthritic knee and hand joints. These results have been repeatedly negative in clinical trials. The same was the case for growth factor therapy, which not only showed very limited beneficial effects but also local effects (in the form of excessive formation of osteophytes) and systemic adverse effects [56, 57].

To date, synovial fluid analysis in osteoarthritis is indicated only to exclude bacterial joint infection or gout. Research efforts are ongoing to investigate the significance of potential risk factors and markers in the synovial fluid or other easily accessible body fluids.

7.7 Implications for Management

Management should not be focused on restoring the position of the disc but should primarily be aimed at restoring the balance between synthesis and breakdown, i.e., by load reduction and controlling other stress factors and by removing damaging products in an attempt to increase the tissue's adaptive capacity and provide for a favorable environment to allow tissue healing to occur. In the TMJ, arthrocentesis has been shown to be an effective therapeutic modality in terms of clinical improvement, aimed at reducing inflammation by removing damaging molecules and inflammatory mediators from the joint [58–61]. In many clinics, it is common to flush the joint with anti-inflammatory medications, or leaving hyaluronic acid as viscosupplementation.

Currently, it is widely advised to start the management of patients with temporomandibular joint disorders with conservative treatment modalities and to consider invasive treatment only in cases where noninvasive treatments have failed. A serious limitation of this strategy is the time it takes to try conservative treatment modalities first, without being able to predict their success. The degenerative process is allowed to persist, which might result in less favorable circumstances for subsequent invasive treatment measures.

In a controlled study from our group, we clinically and economically compared arthrocentesis as *initial* treatment to the usual conservative approach that is advised in current textbooks [61]. This study not only confirmed that arthrocentesis is an effective treatment modality for painful osteoarthritis but also suggests that early timing of this procedure appears to be more cost-effective than the usual approach. Early arthrocentesis might interrupt the degenerative process and prevent the disease from getting worse, leaving better conditions for additional conservative treatments.

We all recognize that patients with the same disorder, when given the same treatment, do not tend to respond in the same way. This implies that besides the pathologic process and the resulting clinical manifestations, the disorder's impact on the patient's physical and psychosocial well-being should be incorporated in both the diagnostic workup and management approach. In fact, the axis II diagnosis appears to be very important indeed, as was shown once more in a recent study by Manfredini's group [62]. Moreover, there is increasing evidence from the psychological literature [63, 64] that supports the importance of the calming care system in emotion regulation. We tend to enter the *danger* mode as soon as external and internal threats elicit mechanisms of self-protection, while we enter the *competitive* mode when we hunt for achievements in life. In both systems, the level of stress is relatively high as sympathetic activity is dominating, ensuring a necessary state of readiness (Fig. 7.5). These systems are opposed to the "care system," giving rise to a mode of compassion, which brings the body in a state of tranquility and relaxation. Here the parasympathetic system dominates, supporting an environment allowing for growth, repair, and healing. So, we not only have a responsibility to accurately assess the extent of physical pathology and its clinical manifestations in order to determine possible

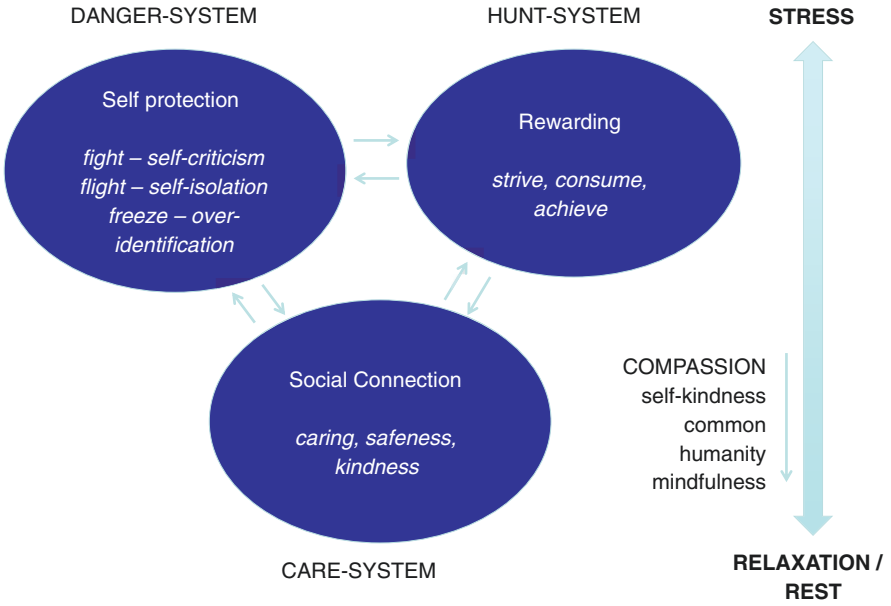

Fig. 7.5 Care system as opposed to the danger and hunt systems. Moreover, there is increasing evidence from the psychological literature that supports the importance of the calming care system in emotion regulation. We tend to enter the *danger mode* as soon as external and internal threats elicit mechanisms of self-protection, while we enter the *competitive mode* when we hunt for achievements in life. In both systems, the level of stress is relatively high as sympathetic activity is dominating, ensuring a necessary state of readiness. These systems are opposed to the care system, giving rise to a mode of compassion, which brings the body in a state of tranquility and relaxation. Here the parasympathetic system dominates, supporting an environment allowing for growth, repair, and healing

treatment options, but we must also incorporate the impact of the disease in order to establish the management option that fits the individual patient best as well as a third axis addressing compassionate care which is crucial for healing to occur. Each of the three axes is necessary, but not sufficient on its own, to enable optimal care for our patients.

References

1. Boering G. Osteoarthrosis of the temporomandibular joint. Thesis. Groningen, University of Groningen; 1966 (reprinted in English, 1994).
2. de Leeuw R, Boering G, Stegenga B, de Bont LGM. Clinical signs of TMJ osteoarthrosis and internal derangement of the temporomandibular joint 30 years after non-surgical management. J Orofac Pain. 1994;8:18–24.
3. de Bont LGM, Boering G, Liem RSB, et al. Osteoarthrosis and internal derangement of the temporomandibular joint. A light microscopic study. J Oral Maxillofac Surg. 1986;44:634–43.
4. Dijkgraaf LC, Liem RSB, de Bont LGM. Synovial membrane involvement in osteoarthritic temporomandibular joints. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997;83:373–86.

5. Stegenga B, de Bont LGM, Boering G. Osteoarthrosis as the cause of craniomandibular pain and dysfunction. A unifying concept. J Oral Maxilliofac Surg. 1989;47:249–56.
6. Stegenga B. Nomenclature and classification of temporomandibular joint disorders. J Oral Rehabil. 2010;37:760–5.
7. Stegenga B, de Bont LGM, Boering G, van Willigen JD. Tissue responses to degenerative changes in the temporomandibular joint. J Oral Maxillofac Surg. 1991;49:1079–88.
8. Luqmani R, Robb J, Porter D, Joseph B. Textbook of orthopaedics, trauma and rheumatology. 2nd ed. Edinburgh: Elsevier Mosby; 2013.
9. Stegenga B. Osteoarthritis of the temporomandibular joint organ and its relationship to disc displacement. J Orofac Pain. 2001;15:193–205.
10. Tanaka E, Detamore MS, Mercuri LG. Degenerative disorders of the temporomandibular joint: etiology, diagnosis, and treatment. J Dent Res. 2008;87:296–307.
11. Haskin CL, Milam SB, Cameron IL. Pathogenesis of degenerative joint disease in the human temporomandibular joint. Crit Rev Oral Biol Med. 1995;6:248–77.
12. Milam SB, Schmitz JP. Molecular biology of temporomandibular joint disorders: proposed mechanisms of disease. J Oral Maxillofac Surg. 1995;53:1448–54.
13. Alstergren P, Appelgren A, Appelgren B, et al. Co-variation of neuropeptide Y, calcitonin gen-related peptide, substance P and neurokinin A in joint fluid from patients with temporomandibular joint arthritis. Arch Oral Biol. 1995;40:127–35.
14. Holmlund A, Ekblom A, Hansson P, et al. Concentrations of neuropeptides substance P, neurokinin A, calcitonin gene-related peptide, neuropeptide Y and vasoactive intestinal polypeptide in synovial fluid of the human temporomandibular joint. A correlation with symptoms, signs and arthroscopic findings. Int J Oral Maxillofac Surg. 1991;20:228–31.
15. Milam SB, Zardaneta G, Schmitz JP. Oxidative stress and degenerative temporomandibular joint disease: a propose hypothesis. J Oral Maxillofac Surg. 1998;56:214–23.
16. Vos LM, Huddleston Slater JJR, Leijsma MK, Stegenga B. Does hypoxia-reperfusion injury occur in osteoarthritis of the temporomandibular joints? J Orofac Pain. 2012;26:233–9.
17. Liu Z, Peng YJ, Long X, et al. Mutual effects between neuropeptides and inflammatory cytokines in neurogenic SMSCs of human temporomandibular joint. J Huazhong Univ Sci Technol. 2014;34:602–7.
18. Alstergren P, Kopp S. Prostaglandin E2 in temporomandibular joint synovial fluid and its relation to pain and inflammatory disorders. J Oral Maxillofac Surg. 2000;58:180–6.
19. Su SC, Tanimoto K, Tanne Y, et al. Celecoxib exerts protective effects on extracellular matrix metabolism of mandibular condylar chondrocytes under excessive mechanical stress. Osteoarthr Cartil. 2014;22:845–51.
20. Cevidanes LH, Walker D, Schilling J, et al. 3D osteoarthritic changes in TMJ condylar morphology correlates with specific systemic and local biomarkers of disease. Osteoarthr Cartil. 2014;22:1657–67.
21. Vernal R, Velasquez E, Gamonal J, et al. Expression of proinflammatory cytokines in osteoarthritis of the temporomandibular joint. Arch Oral Biol. 2008;53:910–5.
22. Goldring MB, Otero M. Inflammation in osteoarthritis. Curr Opin Rheumatol. 2011;23:471–8.
23. Kubota E, Imamura H, Kubota T, Shibata T. Interleukin 1β and stromelysin (MMP3) activity of synovial fluid as possible markers of osteoarthritis in the temporomandibular joint. J Oral Maxillofac Surg. 1997;55:20–7.
24. Kubota E, Kubota T, Matsumoto J, Shibata T. Synovial fluid cytokines and proteinases as markers of temporomandibular joint disease. J Oral Maxillofac Surg. 1998;56:192–8.
25. Chen W, Tang Y, Zheng M, et al. Regulation of plasminogen activator activity and expression by cyclic mechanical stress in rat mandibular condylar chondrocytes. Mol Med Rep. 2013;8:1155–62.
26. Li W, Wu M, Jiang S, et al. Expression of ADAMTs-5 and TIMP-3 in the condylar cartilage of rats induced by experimentally created osteoarthritis. Arch Oral Biol. 2014;59:524–9.
27. Shirakura M, Tanimoto K, Eguchi H, et al. Activation of the hypoxia-inducible factor-1 in overloaded temporomandibular joint, and induction of osteoclastogenesis. Biochem Biophys Res Commun. 2010;393:800–5.

28. Tanaka E, Aoyama J, Miyauchi M, et al. Vascular endothelial growth factor plays an important autocrine/paracrine role in the progression of osteoarthritis. Histochem Cell Biol. 2005;123:275–81.
29. Embree M, Ono M, Kilts T, et al. Role of subchondral bone during early-stage experimental TMJ osteoarthritis. J Dent Res. 2011;90:1331–8.
30. Jiao K, Niu LN, Wang MQ, et al. Subchondral bone loss following orthodontically induced cartilage degradation in the mandibular condyle of rats. Bone. 2011;48:362–71.
31. Jiao K, Zhang M, Niu L, et al. Overexpressed TGF-beta in subchondral bone leads to mandibular condyle degeneration. J Dent Res. 2014;93:140–7.
32. Zhao YP, Zhang ZY, Wu YT, et al. Investigation of the clinical and radiographic features of osteoarthrosis of the temporomandibular joints in adolescents and young adults. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2011;111:e27–34.
33. Wang XD, Kou XX, Meng Z, et al. Estrogen aggravates iodoacetate-induced temporomandibular joint osteoarthritis. J Dent Res. 2013;92:918–24.
34. Weinans H, Siebelt M, Agricola R, et al. Pathophysiology of peri-articular bone changes in osteoarthritis. Bone. 2012;51:190–6.
35. Radin EL, Paul IL, Rose RM. Role of mechanical factors in pathogenesis of primary osteoarthritis. Lancet. 1972;1:519–22.
36. Wang XD, Cui SJ, Liu Y, et al. Deterioration of mechanical properties of discs in chronically inflamed TMJ. J Dent Res. 2014;93:1170–6.
37. Tanaka E, Hirose M, Yamano E, et al. Age-associated changes in viscoelastic properties of the bovine temporomandibular joint disc. Eur J Oral Sci. 2006;114:70–3.
38. Hill A, Duran J, Purcell P. Lubricin protects the temporomandibular joint surfaces from degeneration. PLoS One. 2014;9:e106497.
39. Lang TC, Zimny ML, Vijayagopal P. Experimental temporomandibular joint disc perforation in the rabbit: a gross morphologic, biochemical, and ultrastructural analysis. J Oral Maxillofac Surg. 1993;51:1115–28.
40. Hui AY, McCarty WJ, Masua K, et al. A systems biology approach to synovial joint lubrication in health, injury, and disease. Wiley Interdiscip Rev Syst Biol Med. 2012;4:15–37.
41. Leonardi R, Perrotta RE, Almeida L-E, et al. Lubricin in synovial fluid of mild and severe temporomandibular joint internal derangements. Med Oral Pathol Cir Bucal. 2016;21:e793–9.
42. Asakawa-Tanne Y, Su S, Kunimatsu R, et al. Effects of enzymatic degradation after loading in temporomandibular joint. J Dent Res. 2015;94:337–43.
43. Koolstra JH. Biomechanical analysis of the influence of friction in jaw joint disorders. Osteoarthr Cartil. 2012;20:43–8.
44. Koyama E, Saunders C, Salhab I. Lubricin is required for the structural integrity and postnatal maintenance of TMJ. J Dent Res. 2014;93:663–70.
45. Rhee DK, Marcelino J, Baker M, et al. The secreted glycoprotein lubricin protects cartilage surfaces and inhibits synovial cell overgrowth. J Clin Invest. 2005;115:622–31.
46. Guo H, Fang W, Li Y, et al. Up-regulation of proteoglycan 4 in temporomandibular osteoarthritic synovial cells by hyaluronic acid. J Oral Pathol Med. 2015;44:622–7.
47. Teeple E, Elsaid KA, Jay GD. Effects of supplemental intra-articular lubricin and hyaluronic acid on the progression of posttraumatic arthritis in the anterior cruciate ligament-deficient rat knee. Am J Sports Med. 2011;39:164–72.
48. Kalladka M, Quek S, Heir G, et al. Temporomandibular joint osteoarthritis: diagnosis and long-term conservative management: a topic review. J Indian Prosthodont Soc. 2014;14:6–15.
49. Chevalier X, Eymard F, Richette P. Biologic agents in osteoarthritis: hopes and disappointments. Nat Rev Rheumatol. 2013;9:400–10.
50. Könönen M, Waltimo A, Nyström M. Does clicking in adolescence lead to painful temporomandibular joint locking? Lancet. 1996;347:1080–1.
51. Sato S, Goto S, Nasu F, Motegi K. Natural course of disc displacement with reduction of the temporomandibular joint: changes in clinical signs and symptoms. J Oral Maxillofac Surg. 2003;61:32–4.
52. Kircos LT, Ortendahl DA, Mark AS, Arakawa M. Magnetic resonance imaging of the TMJ disc in asymptomatic volunteers. J Oral Maxillofac Surg. 1987;45:852–4.

53. Pereira FJ Jr, Lundh H, Westesson PL. Clinical findings related to morphologic changes in TMJ autopsy specimens. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1994;78:288–95.
54. Sato S, Kawamura H, Nagasaka H, Motegi K. The natural course of anterior disc displacement without reduction in the temporomandibular joint: follow-up at 6, 12, and 18 months. J Oral Maxillofac Surg. 1997;55(3):234–8.
55. Kurita K, Westesson PL, Yuasa H, et al. Natural course of untreated symptomatic temporomandibular joint disc displacement without reduction. J Dent Res. 1998;77:361–5.
56. Hunter DJ, Pike MC, Jonas BL, et al. Phase 1 safety and tolerability study of BMP-7 in symptomatic knee osteoarthritis. BMC Musculoskelet Disord. 2010;11:232.
57. McPherson R, Flechenshar K, Hellot S, Eckstein F. A randomized, double blind, placebo-controlled multicenter study of FGF 18 administered intra-articularly using single or multiple ascending doses in patients with primary knee osteoarthritis, not expected to require knee surgery within a year. Osteoarthr Cartil. 2011;19(suppl 1):S35–6.
58. Guarda-Nardini L, Olivo M, Ferronato G, et al. Treatment effectiveness of arthrocentesis plus hyaluronic acid injections in different age groups of patients with temporomandibular joint osteoarthritis. J Oral Maxillofac Surg. 2012;70:2048–56.
59. Manfredini D, Rancitelli D, Ferronato G, Guarda-Nardini L. Arthrocentesis with or without additional drugs in temporomandibular joint inflammatory-degenerative disease: comparison of six treatment protocol. J Oral Rehabil. 2012;39:245–51.
60. Nitzan DW, Price A. The use of arthrocentesis for the treatment of osteoarthritic temporomandibular joints. J Oral Maxillofac Surg. 2001;59:1154–9.
61. Vos LM, Huddleston Slater JJR, Stegenga B. Arthrocentesis as initial treatment for temporomandibular joint arthropathy: a randomized controlled trial. J Cranio-Maxillofac Surg. 2014;42:e134–9.
62. Manfredini D, Favero L, Del Giudice A, et al. Axis II psychosocial findings predict effectiveness of TMJ hyaluronic acid injections. Int J Oral Maxillofac Surg. 2013;42:364–8.
63. Germer CK, Neff KD. Self-compassion in clinical practice. J Clin Psychol. 2013;69:856–67.
64. Neff KD. Self-compassion, self-esteem, and well-being. Social Personal Psychol Compass. 2011;5:1–12.

Clinical Evaluation

8

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Abstract

For each temporomandibular disorder (TMD) patient, every diagnosis is preceded by a thorough data gathering process, which begins with the standardized history and targeted clinical evaluation. A clinical assessment should flow naturally from the initial components of the history and follow a consistent pattern so that nothing is left out. If done in such a systematic manner, the information obtained will help ascertain the patient's current functional status, diagnosis, and necessary imaging studies to obtain and to help begin to formulate treatment strategies. Lastly, this chapter will introduce the value of including electromyography combined with motion-capture technology as standard evaluations in the TMD clinical exam.

8.1 Introduction

The clinical evaluation provides information for determining the following: current functional status, diagnosis, and potential treatment strategies for a temporomandibular disorder (TMD). This chapter will cover the relevant clinical assessment procedures and findings as well as indications for imaging of the

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temporomandibular joints (TMJs) for any underlying pathology; see Chaps. 8 and 9 regarding imaging. The clinical assessment is also the foundation for incorporating additional information from imaging, in that clinical correlates must be considered in the interpretation of imaging findings. The information from clinical assessment serves not only for clinical diagnosis (Chap. 10) but also for understanding the full spectrum of symptoms and associated factors accompanying the patient's complaint such as the possible presence of a chronic pain disorder (see Chaps. 1 and 6, Vol. 1 and Chap. 8, Vol. 2). To restate, all of this information must be viewed within the context of the patient's complaint. In addition to improving the reader's understanding of the value as well as limitations of standard clinical examination techniques, this chapter will introduce how devices that combine electromyography and motion-capture technology can further contribute to our understanding of the functional status of the stomatognathic system in both normal and disease states and how it could be morphologically and functionally changed after surgery.

Clinical assessment and associated imaging are now considered classical methods due to their extensive history and empirical support. The history of clinical assessment includes procedures with obvious face validity (e.g., measure jaw range of motion (ROM) with a ruler) as well as procedures that purport to have the same face validity but have been accompanied by substantial controversy due to inadequate research (e.g., measure jaw range of motion with optical tracking or measure masticatory "rest" with nonstandardized electromyographic (EMG) analysis). While there are many clinical methods advocated for assessment of any part or the whole of the stomatognathic system, particularly within the context of TMDs, few have been assessed using the current methodological standards, STARD (Standards for the Reporting of Diagnostic Accuracy Studies), required for diagnostic research [1]. A notable emphasis within STARD, as applied to TMD, is that diagnostic methods research must include adequate controls in order to develop appropriate normative values, which, in turn, allow clinical decision-making to correctly assign a "diagnosis" to individuals who have corresponding pain or functional complaints. In contrast, the commonly used measurements of jaw ROM with optical tracking or nonstandardized EMG produce a wide range of statistics that have been used to classify individuals as pathologic in the absence of clinical complaints [2]. Part of the extensive history of the classical methods now includes appropriate diagnostic research [3] complying with standards such as STARD. STARD guidelines indicate that diagnostic validity is based on the statistics of sensitivity and specificity within the context of appropriate reference standard diagnoses. For this chapter, we rely upon the values listed within STARD, which emphasize reliability, validity, and utility of a test as core attributes.

As previously noted [4], development of our understanding of valid criteria for the TMD pain disorders has vastly exceeded that of the disorders specifically affecting the TMJ. The clinical-only assessment within the DC/TMD has insufficient validity for formal diagnosis of most of the intracapsular and degenerative disorders, serving instead as initial screening diagnoses pending the addition of imaging

(e.g., MRI, CT) for formal diagnoses. But joints are dynamic and static images do not capture the functional demands of the joint. Therefore, we discuss new tools that will extend what the present DC/TMD (inclusive of imaging) can provide. We then provide recommendations for best clinical practice.

8.2 Clinical Assessment

8.2.1 Overview

For clinical assessment of the stomatognathic system, we rely upon the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) [3] for the most common disorders and upon the expanded TMD [5] for the less common disorders, as further discussed in Chap. 11, for identification of essential information to be obtained by history and by examination. While our recommended methods for clinical assessment follow well-established procedures within the validated DC/TMD, clinical procedures for the less common (and nonvalidated) TMDs have only been defined to date and are therefore, pending further development, currently without specific operationalized guidelines that underlie reliable assessment. For both most common and less common TMDs, the DC/TMD taxonomy represents a comprehensive and well-organized depiction of the TMDs, and for painful disorders, the clinical assessment methods underlying the DC/TMD are particularly useful [6]. The evolution of the DC/TMD is described elsewhere [4], and it highlights the fundamental rationale for why such a system is preferred to ad hoc approaches and, in particular, preferred to methods that have no documented or inadequate reliability or validity. Consequently, for the core clinical assessment of TMDs, we use the current DC/TMD for its breadth, comprehensive development, reliability, validity, and empirical basis, and we use other clinical assessment methods to augment the examination as indicated by the patient complaint, history, and findings from core procedures.

The DC/TMD formally specifies essential information by history and by clinical examination using basic tools and skills. These procedures are well-described, and the instructions are readily available [6]. The procedures are reliable, well-published, and produce clinical findings that contribute to diagnostic classification. DC/TMD diagnoses rely on both history and clinical examination; for the common disorders, both history and examination are formally implemented via self-report questionnaire and specific procedures, respectively. However, history of complaint typically requires more detail than the standardized symptom questions can provide or is intended to provide. Consequently, the intent behind the DC/TMD is that a history of a pain complaint is obtained via both standardized instruments and interview and in conjunction with the other components of the DC/TMD protocol. In this section, we address the following: (1) history of complaint, (2) reliability and validity, (3) description of each of the core examination procedures, and (4) systemic and general contributions to TMD (see Table 8.1).

Table 8.1 Clinical assessment procedures and associated signs and symptoms to be considered during a standard evaluation of a patient with suspected TMD

Procedure	Signs and symptoms to be considered
Pain history	All
Jaw range of motion (ROM)	Any change in vertical mobility from that lifelong normal in an individual patient; deviation in frontal plane >2 mm
TMJ status	Noise detection
Joint overloading	Muscular hypotension or proprioceptive inhibition
Palpation for pain	1 kg load on extraoral masticatory muscles for 2 s for identifying local pain and at least 5 s for identifying potential referral pathways. For the TMJ, 1 kg rotary loading around the lateral TMJ pole. Goal is to identify purported sources of nociception vs reported pain locations
Gross muscle examination	Muscle bellies during contraction are compared across the two sides as well as to what is considered normal
Occlusion	Parafunctional behaviors based on habit, pain-avoidance, or psychological status
Cervical muscles/ occlusion/posture and their relation to TMJ	Whiplash injury, cervical spine dysfunction or injury, cervical area muscles alterations
Trigger points	To identify putative sources of nociception
Instrumental tools to support the TMJ diagnosis	To confirm a suspected TMJ diagnosis
Systemic and general factors	Systemic illness, hormonal factors, trauma, psychological and/or psychiatric-related problems

8.2.2 Pain History

Disorders characterized by pain as their primary feature and which lack clear pathognomonic findings are diagnosed almost exclusively by the history of complaint. Adjunctive tests, such as imaging for neoplasm or laboratory tests for alterations in specific metabolic parameters, primarily serve the purpose for ruling out other primary disorders (e.g., Lyme disease vs myofascial pain disorder). The 15 fundamental components of a pain history are well-described by Blau [7], and these consist of the following, by domain:

1. Time (onset, frequency, and duration)
2. Site (origin, travel, and deep vs superficial)
3. Cofactors (initiating, aggravating, and alleviating)
4. Quality
5. Intensity
6. Associated symptoms
7. Previous treatment
8. Patient's explanatory model
9. And why consulting now

See Blau [7] for further details about each component and about relationships among the components. In addition, it is important to connect the history of the pain complaint to a patient's life course, at both a daily and across the life-span level.

This will lead to a better understanding of how behavior versus any physical condition contributes to the overall complaint as well as to treatment options [8]. For example, behaviors contributing to the symptoms associated with a TMJ condition warranting surgery must be identified and addressed in order to improve the prognosis for the surgical outcome.

As previously noted, diagnostic criteria and our understanding of pain disorders including myofascial pain (also known as myalgia, particularly when the pain is localized) and arthralgia are well developed, and reliance upon the DC/TMD [3] for the common painful TMDs is appropriate and empirically supported as a best practice. In contrast, our understanding of how pain interacts with the more common TMJ conditions such as intracapsular disorders and degenerative joint disease of the TMJ is less well developed; the general approach is to assign priority to a coexisting myofascial pain or arthralgia disorder (because these two pain disorders are well understood), but this may be suboptimal for patients within a tertiary setting with more focal complaints associated with the TMJs.

Intracapsular disorders with pain as a prominent feature specifically associated with function in the joint and separated from pain of muscle origin or of more general joint origin (e.g., hyperalgesia—provocation and replication of pain via standardized palpation of the joint) require greater attention in the history. While there is no current diagnostic category, pain specifically associated with clicking or locking and associated cofactors should be identified.

The central point of a pain history is captured by the formal definition of pain as established by the International Association for the Study of Pain: "Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" [9]. A patient history of pain must address the experience of pain within the context of this definition: pain is a complex experience, and pain is whatever the patient says it is, as long as the reported experience is tied to the body and with the exclusion of clear evidence of malingering or fabrication. Various types of pain have been defined based on careful analysis of the IASP definition as the following: pain with concordant tissue damage, pain without concordant tissue damage, neuropathic nociception, pain behavior without pain, and tissue damage without pain [10]. The formal definition of pain and the types of pain revolve around the presence or absence of tissue damage, and tissue damage is proximally caused by either injury or disease. A necessary goal in the evaluation of a patient's symptom complaint in a medical context is to carefully evaluate for and rule out a physical basis (i.e., ongoing tissue damage) for the symptoms. When injury or disease is determined to be responsible for pain via identified nociception, then pain with concordant tissue damage is present. However, in many pain disorders, especially those that become chronic, tissue damage or potential tissue damage is absent, yet the symptom of pain is described by the patient in terms of such damage (as per the IASP definition), in such a manner that the patient links the reported experience to body structure and consequently that type of pain is pain without concordant tissue damage.

What exactly is pain without concordant tissue damage? In general, we cannot eliminate the possibility that tissue damage exists at a more granular level than our detection methods permit us to identify, and therefore, the identified location of pain

is regarded as a source for peripheral nociception and thereby giving rise to "pain… described in terms of such damage." For example, research using microcatheters inserted into areas of muscle, putatively the cause of nociception within a myofascial pain disorder, has yielded a range of algesic and inflammatory substances not found in control areas of the same muscle [11]. Despite such findings, whether myofascial pain disorder exists remains a controversial question [12–15]. As another example of the complex relationship that pain has with tissue damage, injury is itself equally complex but consistent with the IASP definition as Chap. 12 notes. Two extremes are illustrative: external trauma-like events to the body often result in no observable tissue damage and pain that is temporary, contrasted with injury associated with observable tissue damage which heals but leads to persistent pain in the absence of any subsequent evidential tissue damage. Is the contrast caused by our limited means of detecting tissue damage at the granular level, or is the contrast related to alterations in central pain processing? As it is very often impossible to know the clinical answer to this question, the clinician must remain skeptical about both "obvious" sources of nociception as well as inferred sources of nociception, consider both options viable until pain has sufficiently resolved via treatment, and acknowledge that for many patients we never know, and therefore, the injunction to "do no harm" should be in the foreground to guide the clinician's decisions and behaviors.

In summary, pain is a complex experience, and the presence of tissue damage as a source of nociception must be very carefully considered for both acute pain and chronic pain. The absence of evidential tissue damage may hide some type of lesion that did not adequately heal, and it is the responsibility of the clinician to carefully consider that possibility. Contrariwise, there may be no tissue damage, and its absence could point to the role of behavioral factors as well as changes in central pain processing; it is the responsibility of the clinician to carefully consider this possibility as well. The history remains the best tool currently available for exploring these alternative hypotheses for better understanding, diagnosing, and treating a person's complaint of pain.

8.2.3 Reliability and Validity

Reliability refers to consistency, and validity refers to truth. In a clinical setting, we assess the reliability of our symptom history by repeat questions from different perspectives and by repeated evaluations across time. The resultant details in the history of a particular patient's pain are compared against known pain patterns and are assessed for plausibility and coherency, leading to the clinician's degree of confidence in this particular history of this particular patient. Requiring this level of consistency in the obtained history is an essential safeguard for preventing inappropriate escalation of treatment. Validity in a history emerges from matching the core elements in the patient's history to known patterns associated with the putative diagnosis for the given patient, and from insisting that the obtained information creates a sense of coherence also on the part of the examiner taking the history. As clinicians,

our understanding and informal assessment of the reliability and validity of a given history are also informed by available information from standardized assessment tools. For example, pain intensity assessment is less reliable and valid when obtained by a single measure on a single occasion but is improved via a formal set of questions about intensity which take known variability in pain intensity across time into account [16] or via pain diaries [17] for an even more detailed assessment. The insightful clinician will augment the history with a standardized self-report tool for assessing intensity, for example, as needed and not solely rely on a single item with low specificity. For example, the interview question "What is your pain intensity on a 0–10 scale?" is open as to time, circumstance, and episode course.

For any clinical examination procedures, reliability is readily determined by utilizing two or more examiners and conducting an appropriate reliability assessment for the procedure, thereby determining how well the procedure has been defined and operationalized and how much training is required for a given examiner to obtain consistent data. Validity of most clinical findings (as well as instrumental approaches) is typically assessed by comparing groups: cases vs non-cases, for example, in relation to whether the finding relates to the disorder or not.

8.3 Description of Core Examination Procedures

Fundamental clinical signs from clinical evaluation procedure checklist are summarized in Table 8.1.

8.3.1 Jaw Range of Motion

While jaw range of motion (ROM) is not part of the diagnostic criteria for muscle pain or joint pain disorders, jaw ROM is used to distinguish disc displacement without reduction, with limitation, from disc displacement without reduction, without limitation. Jaw ROM is also a key measure of physical status of the jaw, often used clinically as a reference for understanding patient-reported outcomes (PRO). For either purpose, the measurement must be made in a reliable manner in order to yield commensurate data across persons (e.g., to distinguish the extent from the shape of the movements) and across time (e.g., before and after surgical intervention, in order to monitor progress). To create reliable clinical measurement of jaw ROM, the ruler must be linked to stable landmarks, held in a standard manner, and measure jaw movement that is controlled via standardized instructions to the patient. The instructions to "open as wide as you can, without any pain or increasing your current pain" for establishing the extent of pain-free jaw opening and "open as wide as you can, even if it is painful" for establishing the extent of maximal unassisted jaw opening yield reliable measurements of mobility; in addition, maximal assisted jaw opening is measured with a similar instruction. To use any instruction, such as "open as wide as you can, even if uncomfortable" (for maximal unassisted opening) will yield noncomparable results. How pain (or other

potential barriers that may affect mobility) is operationalized is critical in order to capture the intended phenomenon. During jaw ROM measurement, the ruler is held vertically with one edge against a central incisal edge, and the extent of opening (as the space through which food is ingested) is read relative to the opposing incisal edge. The measured extent of opening can be augmented by adding vertical overlap (negative overlap when open bite and positive otherwise), thereby yielding a true measure of extent of actual mandibular movement during opening. Normal minimal opening is typically considered around 40 mm of inter-incisal space; individuals with lifelong deviations from that normal threshold should be identified during the history: normal for some people will be 60 mm, while for others it might be 32 mm. The presence of restricted jaw movement is thus often interpreted from both the examination and the history. The PRO of jaw ROM can also be obtained via standardized self-report instrument.

During jaw opening, the jaw may deviate from side to side in the frontal plane, with the midpoint of the mandible aligning (±2 mm) with the midsagittal plane at maximal opening; such a pattern of movement is considered a corrected deviation. This pattern arises from three causes (that may also act in combination):

1. Disc displacement of one or both TMJs; a displaced disc is often associated with altered TMJ mechanics (e.g., rotational vs translational component of the movement is altered) which then requires a different acceleration pattern of movement in order for the condyle to negotiate the displaced disc and effect reduction.
2. Neurologic cause, such as from a dyskinesia disorder.
3. Incoordination of the masticatory muscles [18] during functional tasks.

If the jaw deviation in the frontal plane results in the midpoint of the mandible being displaced from the midsagittal plane at maximal opening, then the pattern of movement is considered an uncorrected deviation. This pattern arises from two causes (that may also occur in combination). The first cause is acute disc displacement, such that translation of the affected condyle is reduced to very little since the displaced disc anterior to the condyle cannot be negotiated by the translating condyle. Uncorrected deviation at maximal jaw opening of about 35 mm is a classic presentation for the acutely displaced anterior disc without reduction, but the classic presentation does not always occur with the acutely displaced nonreducing disc. The other cause for uncorrected deviation, particularly well identified when the jaw movement is kept slow, is unilateral tightness or restriction of an elevator muscle (usually, the masseter). When this occurs, the joint is not usually responsible (though this extracapsular problem can, when a disc is anteriorly displaced, exacerbate the disc disorder which can then contribute to contracture state in the muscle), and treatment is oriented toward restoring normal opening pattern. Of course, even without a disc problem, a unilateral acute restriction in muscle length can, if not treated, develop into a contracture. When a strong history of joint clicking coexists with the finding of uncorrected deviation, automatically concluding that the deviation is due to a disc displacement without reduction is common. However, uncorrected deviation due to contracture ipsilateral to the side of deviation may coexist with disc

displacement without reduction (which may be secondary to the limitation in vertical ROM induced by the contracture) and must be carefully identified.

Similar to uncorrected deviation in the vertical plane of the ROM is restricted lateral movement to one side compared to the other, with the restriction contralateral to the side of the problematic TMJ. In addition, the performance of these movements and their measurement are less reliable, compared to vertical ROM. Like uncorrected deviation as the classic hallmark of acute disc displacement, limitation in contralateral movement of the mandible occurs less commonly than believed. Rather, only with acute disc displacements may one encounter these patterns, whereas with recurrent or chronic disc displacements, these limitations in movement during opening or lateral movements are far less common.

8.3.2 TMJ Status

Clicking of the TMJs, as disclosed via clinical examination, has often been considered pathognomonic for disc displacements, joint instability, risk factor for TMJ disease progression, or joint dysfunction. However, TMJ clicks may occur with disc displacements, either with or without reduction, and may also occur with normally positioned discs, and consequently TMJ clicking has poor diagnostic sensitivity for any TMJ diagnosis. In addition, clinical assessment of clicking, whether by palpation or auscultation, has low reliability, in part due to difficulty in feature detection (is this vibration a click or not?) [19] and in part due to low temporal stability of the phenomenon. Consequently, current evidence indicates that TMJ clicking, if not accompanied by pain, interference in function, or locking, is benign and should be regarded as a benign condition [20]. For clinical assessment, auscultation allows detection of too many sounds leading to many false-positive classifications, whereas palpation for joint sounds is more useful in terms of both sensitivity and specificity and is therefore the preferred detection method for clinical examination [21]; the procedure for joint palpation for noise detection is described well in the DC/TMD protocol manual.

Given the poor diagnostic validity of the clinical assessment for TMJ internal derangements, imaging is often necessary to establish the diagnoses of both disc displacement with reduction and disc displacement without reduction. Particularly, if the history is positive for problematic TMJ clicking accompanied by pain associated specifically with the click, recurrent locking (i.e., disc displacement with reduction, with intermittent locking), or interference with function, and the clicking-relevant complaints have not sufficiently improved within an 8-week period of compliant self-care methods for TMD, then imaging should be considered. Most often, such imaging does not provide any further information; that is, the imaging confirms that the intracapsular problem is of the well-known type, indicating that continued self-management care for pain and mobility is appropriate. If the imaging indicates that the disc or other intracapsular structure is not of the normal expected configuration (e.g., torn disc, or abnormally large posterior band), then intervention focused moreso on the joint would be appropriate.

Clinical examination may indicate that a click in the TMJ is very late in the opening cycle. While the problems of reliability of detection and stability of the phenomenon remain with this type of click, the timing of the click is readily apparent. Linking the pattern of mandibular movement with the timing of the click can be, however, difficult; a slight lateral or vertical mandibular shift coincident with the late click is often present but difficult to reliably detect. When such a noise is present, it is called a subluxation click and is frequently associated with open locking, also termed subluxation or luxation. These two terms have variable usage within the medical literature pertaining to the TMJ, with luxation (formally, displacement of a bone from a joint) referring to the condyle at maximal opening and unable to engage in the closing movement (either because of discal interference or because of anterior slope of the eminence blocking the movement) and subluxation (formally, partial dislocation) referring to the condyle in either a partial luxation position or only momentarily in the luxation position. The important distinction appears to be the length of time of the blockage of the condylar movement when the subluxation click occurs. At one extreme, there is simply a mandibular shift but no effect on further movement, and the next level of severity is a momentary hesitation (often called a "catch" by patients) whereby the mandible is stuck in the fully open position for a moment and the joint self-reduces and resumes closure in a timely manner in the movement cycle (the threshold for a subluxation). Careful clinical observation over several open and close movements of the mandible can lead to accurate classification if the problem is active at the time of clinical assessment. Imaging for this condition is unreliable since the phenomenon is for most individuals intermittent. When the phenomenon is consistent, clinical evaluation is generally easy, and while imaging is typically not needed, in specific situations imaging may provide further information. When automatic or self-reduction is not possible, a luxation is present. This use of the term luxation should be contrasted with formal displacement of the condyle from the TM fossa, such as from severe injury, and which would also be termed luxation.

8.3.3 Joint Overloading

Joint overload is emerging as an increasingly important aspect of TMJ status. Strong evidence indicates that the TMJs are subject to considerable amount of loading, which occurs across the articular surface with the eminence, mediated by the intervening disc or disc-like structure [22]. Growing evidence suggests that functional overload with subsequent microtrauma is a crucial event for the development of TMJ internal derangement and osteoarthrosis. TMJ overloading during both static and functional mandibular tasks can be caused from different physiological or pathological reasons that have to be considered during patient interview and clinical evaluation. A history of joint overload due to habits (excessive gum chewing, unilateral chewing) or parafunction (bruxism, clenching, whether during sleep or waking periods) has to be recorded. At the same time, clinicians need to consider that any correlation between the TMJ signs and symptoms of disease and history and physical findings may still be lacking.

Low-inflammatory arthritic conditions often involve the TMJ. These conditions can be easily treated and seldom require invasive surgical intervention. Early treatment with simple biomechanical occlusal splints able to move the occlusal center of gravity position as backward as possible during static or functional task is often sufficient [23].

8.3.4 Palpation for Pain

The final component of the standardized DC/TMD examination is palpation for pain of both masticatory muscles and TMJs. Current clinical procedure is based on multiple strands of evidence and is simply to apply a load via palpation with the tip of one finger to the area under examination. This appears to be both sufficiently reliable and appropriate as a threshold to distinguish reported pain from muscle or TMJ as a positive finding. The load has been measured to be approximately 1 kg to the masseter and temporalis and 0.5 kg to the lateral pole of the TMJ. In addition, a 1.0 kg load should be applied in a rotary manner around the TMJ lateral pole over approximately 5 s. For identifying hyperalgesia or allodynia, a load for 2 s, from onset to offset, appears to be sufficient, whereas for identifying common patterns of referred pain or spreading pain, sustaining the load for 5 s appears to produce reliable findings [21]. The duration of load for palpation remains controversial; longer durations of up to 20 s are often advocated within the clinical world and from a pain physiology perspective. Such increased duration of load is perhaps consistent with known facts about temporal summation. Yet, a duration of 5.0 s remains the only method that presently has been shown to be reliable for detection of referred pain patterns [21]. There is considerable critique regarding the existence of purported trigger point phenomena (see above), and consequently the clinical relevance of referred or spreading pain remains unclear. At present, its relevance is certainly well-established for differential diagnosis in order to identify purported sources of nociception and not treat evident locations of reported pain but without nociceptive origins [3].

8.3.5 Gross Muscle Examination

Muscles should also be examined for hypertrophy as evidence for overuse and as an indication of the load or stresses put on the TMJ complex. The clinical detection method is simple but has no empirical support. Fingers are placed on the skin overlying the bilateral masseter muscles, and the patient is asked to maximally contract. The muscle bulk formed during contraction is compared across the two sides as well as to what is considered normal (i.e., minimal increase in muscle bulk with maximal contraction). This exam maneuver is learned and improved by experience. The clinical implication of this finding is speculative; we assume that increased muscle bulk is due to increased “exercise” of the masseter muscles with such exercise

stimulating muscle hypertrophy. The exercise is in the form of parafunctional behaviors such as sleep bruxism or waking clenching. Such behaviors can be identified via self-report, such as with the Oral Behaviors Checklist. The clinical examination may provide additional evidence in support of a behavioral etiology for a patient's complaints.

8.3.6 Occlusion

Perfect morphological occlusion occurs naturally in less than 5% of the US population [24]; one implication of this is that the so-called malocclusion is poorly defined in that mild departures from perfect are not well distinguished from substantial departures. Rather, all departures are called malocclusion, which is typically non-informative. The spectrum of possible types of malocclusion is large, and malocclusion that may truly represent a disorder (i.e., to have clear operational terms and to have some type of consequence) has yet to be reliably defined and identified. In contrast, common views consider important occlusal aberrations to include patterns of lateral disclusion, balancing interferences, and extent of overbite. Yet, none of these characteristics have reliable associations with TMD. However, unilateral cross-bites, excessive overjet, and substantial lateral deviations between most retruded condylar position and maximal occlusion do appear to contribute to joint instability [25]. The evidence, however, supports at best only a modest contribution from such structural aspects of occlusion. Stronger evidence supports the mediating role of parafunctional behaviors stemming from hypervigilance toward the occlusion and driven by anxiety as more important factors [26–28].

Occlusion has a stronger role as a marker of joint change or remodeling. This is illustrated in such cases where there is a recurrent episodes of a unilateral posterior open bite or progressive anterior open bite as reflective of either an unstable disc or condylar degeneration, respectively. In these instances, the occlusal change is the consequence, not the cause, of the change in the TMJ structures. Consequently, monitoring occlusal stability or pattern of tooth contact at the full arch level is an important part of routine examinations.

Dental wear patterns (to be differentiated from dental erosion), scalloped tongue, and tori are also often interpreted as evidence of parafunction. However, wear patterns have two notable limitations: clinical assessment of wear has very poor reliability [29], and dental wear occurs over a lifetime, and its time course is generally unknown. Serial facial photographs clearly showing the maxillary incisors may be helpful in determining if excessive incisal wear is recent. There are other oftenmentioned clinical findings such as scalloped tongue which has a long history of anecdotal association with bruxism or tongue habits; however, supporting evidence is poor. Further, there is also poor evidence connecting the existence of bony exostoses with parafunctional behaviors, although they may be strongly associated with tooth wear [30]. See Chap. 11 for more information regarding bruxism assessment and its reliable markers.

8.3.7 Cervical Muscles/Occlusion/Posture and Their Relation to TMJ

Neurologically, the trigeminocervical nucleus and upper cervical nociceptive neurons are the point of convergence between the cervical spine and jaw nociceptive neurons, and this provides a neuroanatomic underpinning for the interrelation between TMJ and neck problems. Consequently, signs and symptoms of TMD can often overlap with those related to cervical spine dysfunction or pathology. It is important to appreciate this point in formulating a diagnosis and treatment plan that considers the whole person. Head movement is controlled by more than 20 pairs of cervical muscles. These muscles work simultaneously to stabilize and control the head as a stable foundation for mandibular movements, which also consequently affects the TMJ. The interconnectedness of these functional muscles groups is illustrated during forceful kinetic movements, where the head needs to be stabilized against the shoulder girdle to allow the coordinated actions of the masticatory muscles to occur, such as during mastication. These mostly involuntary muscle activations are controlled at both a motor and sensory level by various feedforward and feedback loops (see Chap. 5). Additionally, during swallowing, stabilization of the upper teeth with the lower ones is necessary to hold the mandible so that the suprahyoid muscles can elevate the pharynx complex and permit the closure of the larynx complex to complete this highly coordinated movement. The counter-forces applied by the cervical muscles thus stabilize the mandibular condyle into the temporal fossa, providing a balancing force to this automatic dynamic mechanism. Overall, it is apparent that alterations in muscular activities in the craniocervical area play a significant role in the physiological and pathophysiological relationship between occlusion, TMJ, and the neck muscles.

For instance, cervical spine dysfunction or injury may precede the onset of TMD symptoms and cervical problems or abnormal posture may result in the inability of the masticatory muscles to adapt to influences originating from the cervical spine. But that which is thought of in theoretical terms needs to be measured clinically to make it relevant. Historically, the literature does not provide an adequate explanation of these functional relationships. However, emerging evidence from the beginning of this century is growing [31–34]. The clinical use of standardized EMG is shedding some light on the question and has been introduced to provide evidence through quantitative instrumental measurements in order to better understand the interactivities of these muscle groups. Correlations between occlusion/TMJ and the other body parts inferior to the craniocervical complex are not possible because there is currently no method of measuring such connections.

8.3.8 Trigger Points

Changes in muscle tissue, including nodules, tight bands, and the so-called trigger points, have been well-described in the literature of many medical specialties. The clinical phenomena of muscle pain resistant to treatment and of pain referral are

well known and noncontroversial. The purported changes in muscle tissue underlying these phenomena and whether muscle should be directly treated, however, are very controversial due to poor examiner reliability [35], problems in short-term stability of the clinical characteristics [36], poor development of the purported pathophysiology [12, 13, 15], and insufficient evidence for specific efficacy of targeted treatments. While these concerns with the concepts and clinical assessment of the trigger point phenomenon have been countered [14], the existence of alternative models regarding pain persistence deserve careful clinical consideration; see Chap. 10 regarding chronic pain syndromes. Certainly, the activation of referred pain can be performed clinically without recourse to trigger points as the mechanism; this activation can be reliably achieved at least within a clinical session. The importance of evaluation for referred pain is to better identify putative sources of nociception. For example, masseter muscle tissue can be responsible for pain that is perceived within the TMJ, and if that TMJ has a intracapsular problem described by the patient as painful with function, but the pain is itself replicated via examination of the masseter muscle (including activation of pain referred to the TMJ) and not via pain provocation from the clicking, then the intracapsular problem may not be the source of the pain. Treatment of the TMJ intracapsular problem may not only not resolve the complaint of pain but may, if the treatment is interventional, worsen the pain.

8.4 Systemic and General Contributions to TMDs

8.4.1 Systemic Illness

Refer to Chap. 14, which well-addresses this topic. In brief, systemic illness may also influence fibrocartilage metabolism and could affect the adaptive capacity of the TMJ. These types of illnesses may include autoimmune disorders (lupus erythematosus, psoriasis, ankylosing spondylitis, ulcerative colitis), endocrine disorders, nutritional disorders, metabolic diseases (gout), infectious disease (Reiter's syndrome), and generalized joint hypermobility [37].

8.4.2 Hormonal Factors

Hormonal factors and changes associated with advanced age may also have a marked influence on remodeling of the mandibular condyle. From an epidemiologic point of view, women seek TMD treatment three times more frequently than men. The presence of higher testosterone levels may be a plausible explanation for low treatment seeking by males [38]. Painful symptoms increase by 30% among patients on menopause treatment with estrogen replacement therapy and by 20% in women using oral contraceptives [39]. Initial evidence suggests that estrogens and relaxin could contribute to the degeneration of cartilage homeostasis inducing activation of metalloproteinases (MMP) that disorderly degrade cartilage matrix macromolecules (collagen and proteoglycans) of the TMJ [40].

8.4.3 Trauma

The acute or chronic effects of trauma and resultant maxillofacial skeletal discrepancies, osteochondroma, unstable occlusion, and increased joint friction have to be carefully considered during patient interview and clinical evaluation in order to consider TMJ surgery a viable treatment option. These factors may occur alone or may be interrelated, interdependent, and/or coexistent in the mechanism of delayed condylar resorption or deformation.

Although these disorders may be histologically and chemically different, clinical findings and management are often similar. A sensory nerve supplying a joint also supplies the muscles moving the joint and the skin overlying the insertions of these muscles, a fact well codified as HILTON'S JOINT. Loss of joint function or late stage ankylosis, joint instability, and facial deformity due to loss of posterior mandibular vertical dimension, as pathologic osteolysis could be considered for correction in a surgical treatment.

8.4.4 Psychological and/or Psychiatric Related Problems

The biopsychosocial model expresses a variety of biological, psychological, and social factors that can contribute to pain. The interactions among these three major factors vary considerably across individuals and over time. Stress, anxiety, and other psychological or psychiatric conditions can induce a number of physiological consequences, often referred to as medically undiagnosed disorders or functional disorders due to how the symptoms and physical manifestations can mimic other disorders. The classic example is heart palpitations that can result from either cardiovascular disease or anxiety. In the masticatory system, muscle hyperactivity, fatigue, or both are typical consequences from psychosocial factors that can result in contracture, functional occlusal disharmony, or neuromuscular imbalance. Depressive symptoms and physical symptom reporting are higher in those with TMD pain than in those diagnosed with disc displacements [41].

8.5 Instrumental Tools to Support the TMD Diagnosis

The prevalence of severe TMJ arthropathy is certainly lower than for the painful TMDs, yet while the painful TMDs can respond, albeit unreliably, to currently available treatments, severe TMJ arthropathy has a notable morbidity, and patient histories disclose a pernicious vicious cycle of worsening with each treatment attempt. Consequently, there is a need for better diagnostic methods for the TMJ arthropathies. Previous and widely used attempts to extend that diagnostic understanding have focused on several methods. One method has been to relate static occlusion to TMJ arthropathy, but the associations have been very weak if at all [42, 43]. Another method has focused on joint vibration analysis or movement tasks during function, but the literature currently cannot provide evidence to support the

diagnostic reliability and validity of such methods [44]. More recently, the role of joint movement and associated muscle activity has been conceptualized differently and thereby offers a new opportunity to perhaps better assess joint function in light of how surgical methods may be more appropriately used to address the clinical problems that directly involve TMJ arthropathy.

During the last century and the previous decade, many tools were developed to discriminate between patients who have TMJ problems requiring treatment and those who do not. Unfortunately, for many years, dentistry has used these instruments to "scientifically" support a specific clinical approach or philosophy, rather than more appropriately as adjunctive clinical tools, keeping in mind their basic limitations and sources of error. Medical instruments should not be used as a standalone diagnostic tool, but rather they should simply provide part of the data set that is used to formulate an overall clinical assessment.

Again, being mindful of the quantitative value and inherent limitations, there are mainly four types of technologies that have been implemented to evaluate the patient with TMD:

1. Standardized EMG waveform analysis and its relation to occlusal stability and joint overload
2. Nocturnal EMG/heart rate frequency and its relation to evidence of bruxism or high trigeminal tone
3. Motion capture technology and its relation to clinical normal range of mandibular and neck motion and its significance of deviation
4. Plain radiography, MRI, and ultrasound-based technology

The term occlusal stability has assumed varied clinical significance throughout the years. Regardless, occlusal stability should be considered in the context of functional significance. In other words, for example, a "normal" morphological relationship between the dental arches could be associated with an abnormal neuromuscular balance both in terms of mediolateral, anteroposterior positions and coupling of the forces acting on the mandible and consequently the TMJ. Joint overload caused from muscular inhibition could be a primary effect of this functional altered proprioception. Interactions vary considerably across patients and over time.

Bruxism is well covered in this book in Chap. 12. From a diagnostic perspective, it is important to differentiate between asymptomatic bruxism (the patients who exhibit progressive tooth wear but without any symptoms or functional disturbances) and symptomatic clenching (the patients who do not exhibit tooth wear yet normally suffer from TMJ pain). In this case polysomnography or EMG correlated with heart rate frequency [45] could be helpful for the diagnosis. Three-dimensional (3D) kinematic analysis has been suggested as a useful, accurate, and noninvasive supporting method to deepen the understanding of oral motor control and TMJ function [46, 47]. Investigations found that acute muscular pain has only minor effects on chewing patterns, probably because the function does not exacerbate pain [5], and few changes in jaw kinematics occur in non-chronic TMD [48].

Functional impairment may be a consequence of the chronicity. Sensory inputs derived from the orofacial tissues are essential for motor control mechanisms [49], and previous experiences, including pain, may influence the sensorial processing and the motor output programming [50]. Mandibular and condylar kinematics in individuals with disc displacement with reduction specifically exhibit asymmetry and asynchrony of condylar movements, which can be detailed by TMJ dynamic behavior, focusing on the assessment of the relative contribution of jaw rotation (condyle-disc compartment) and translation (mandibular fossa-disc compartment) [51]. Alterations in their reciprocal magnitude have been identified as important indicators of TMJ dysfunction [52].

8.6 Differential Diagnosis

The onset and persistence of TMD pain is the result of multiple risk determinants. Consequently, a sufficiently deep and broad patient history coupled with clinical examination is required. The general concepts underlying the different diagnoses, including the common DC/TMDs, are shown in Table 8.2. After obtaining the history of the pain and the physical exam, a decision on advanced imaging or supplementary tests has to be taken. Images should be considered only if this expensive modality will incrementally aid in enhanced diagnosis and improved management for the patient. Taking into consideration the reliability and validation of some simple, well-studied, and published recommendations from the DC/TMD, the same clinical outcomes are likely with less expense for the patients and community. Anxiety, depression, sleep disturbance, and generalized decreased quality of life should be discussed with the patient with chronic conditions and prior to any surgical intervention.

8.7 Conclusions and Recommendations

Clinical assessment is comprised of comprehensive pain history, routine medical history, standardized self-report instruments, and clinical examination appropriate to the differential diagnosis and using standardized clinical methods as a foundation to diagnose or rule out common TMDs. Additional tests may be ordered, but only if the expected information will make a difference in diagnosis, prognosis, or treatment. Since common TMDs occur commonly, standardized assessment approach which will reliably diagnose at least 90% of TMD patients in a typical tertiary setting should be considered mandatory and not optional. Appropriate level of psychosocial assessment is necessary to adequately assess the person with pain. Good clinical practice dictates that comprehensive assessment is necessary before interventional treatment and that treatment should be escalated in a patient-specific manner, insuring that the complexity of pain has been adequately incorporated into the clinical assessment at each stage of assessment.

Table 8.2 General algorithm applicable in clinical evaluation and differential diagnosis of TMDs and related disorders

DC/TMD taxonomy		Pain history	Core examination findings	Sensitivity	Specificity	Additional tests
	Intracapsular disorders					
	Arthralgia	+		0.89	0.98	
	Disc displacement with reduction	+/–	+	0.34	0.92	MRI
	Disc displacement with reduction with intermittent locking	+/–	+	0.38	0.98	MRI
	Disc displacement without reduction with limited opening	+/–	+	0.80	0.97	MRI Joint play is not preserved
	Disc displacement without reduction without limited opening	+/–	+	0.54	0.79	MRI Joint play is preserved
	Degenerative joint disease	+/–	+	0.55	0.61	CT Crepitus detected
	Trauma/fracture	+	+			CT
	Extracapsular disorders					
	Trauma/fracture	+	+			CT
	Muscular disorders					
	Myalgia	+		90%	99%	Local hyperalgesia
	Myofascial pain	+		86%	98%	Local hyperalgesia AND spread or referral of the pain
	Headaches attributed to TMD	+		89%	87%	

	Myositis	+		N/A	N/A	
	Spasm	+		N/A	N/A	
	Contracture	+/−		N/A	N/A	
	Hypertrophy	−		N/A	N/A	
	Hypotrophy	−		N/A	N/A	
	Waking parafunction	+/−		N/A	N/A	
	Tumors	+/−	+/−			CT, MRI
Not included into DC/TMD taxonomy						
	Sleep disorders: bruxism	+/−		N/A	N/A	
	Psychological TMD-related symptoms	+/−	+/−	N/A	N/A	Identification of psychological symptomatology (depressive, anxiety, stress, and post-traumatic stress disorder)

Legend: signs are indicating the patient answer to the specific complaint. + positive; − negative. *N/A* not available or not applicable. *CT* Computed tomography scan, *MRI* magnetic resonance imaging

References

1. Bossuyt PM, Reistsma JB, Bruns DE, Gatsonis PP, Glasziou PP, Irwig LM, et al. Toward complete and accurate reporting of studies of diagnostic accuracy: the STARD initiative. Standards for Reporting of Diagnostic Accuracy. Clin Chem. 2009;49(1):1–6.
2. Feine JS, Hutchins MO, Lund JP. An evaluation of the criteria used to diagnose mandibular dysfunction with the mandibular kinesiograph. J Prosthet Dent. 1988;60:374–80.
3. Schiffman E, Ohrbach R, Truelove E, Look J, Anderson G, Goulet J-P, et al. Diagnostic Criteria For Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. J Oral Facial Pain Headache. 2014;28(1):6–27.
4. Ohrbach R, Dworkin SF. The evolution of TMD diagnosis: past, present, future. J Dent Res. 2016;95(10):1093–101.
5. Peck CC, Goulet J-P, Lobbezoo F, Schiffman EL, Alstergren P, Anderson GC, et al. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. J Oral Rehabil. 2014;41(1):2–23.
6. Ohrbach R, Gonzalez Y, List T, Michelotti A, Schiffman E. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) Clinical Examination Protocol International RDC/TMD Consortium Network 2014. http://www.rdc-tmdinternational.org/Portals/18/protocol_DC-TMD/DC-TMD Protocol - 2013_06_02.pdf.
7. Blau JN. How to take a history of head or facial pain. Br Med J. 1982;285:1249–51.
8. Johnson P. Psychological factors influencing headache. In: Tollison CD, Satterthwaite J, Tollison J, editors. Handbook of pain management. Philadelphia: Williams & Wilkins; 1994.
9. IASP. IASP taxonomy. 2011. http://www.iasp-ain.org/AM/Template.cfm?Section=Pain_Definitions2011 [updated 2011].
10. Smith B, Ceusters W, Goldberg LJ, Ohrbach R. Towards an ontology of pain. In: Okada M, editor. Proceedings of the conference on logic and ontology. Tokyo: Keio University Press; 2011. p. 23–32.
11. Shah JP, Phillips TM, Danoff JV, Gerber LH. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. J Appl Physiol. 2005;99(5):1977–84.
12. Cohen M, Quintner J. The clinical conversation about pain: tensions between the lived experience and the biomedical model. In: Fernandez J, editor. Making sense of pain. Clinical and interdisciplinary perspectives. Oxford: Inter-Disciplinary Press; 2010.
13. Cohen M, Quintner J. The horse is dead: let myofascial pain syndrome rest in peace. Pain Med. 2008;9(4):464–5.
14. Dommerholt J, Gerwin RD. A critical evaluation of Quintner et al: missing the point. J Bodyw Mov Ther. 2015;19(2):193–204.
15. Quintner JL, Bove GM, Cohen ML. A critical evaluation of the trigger point phenomenon. Rheumatology. 2015;54(3):392–9.
16. Ohrbach R, Turner JA, Sherman JJ, Mancl LA, Truelove EL, Schiffman EL, et al. Research diagnostic criteria for temporomandibular disorders. IV: Evaluation of psychometric properties of the Axis II measures. J Orofac Pain. 2010;24(1):48–62.
17. Aaron LA, Turner JA, Mancl L, Brister H, Sawchuk CN. Electronic diary assessment of pain-related variables: is reactivity a problem? J Pain. 2005;6(2):107–15.
18. Ferrario VF, Sforza C. Coordinated electromyographic activity of the human masseter and temporalis anterior muscles during mastication. Eur J Oral Sci. 1996;104(5–6):511–7.
19. Leader JK, Boston JR, Rudy TE, Greco CM, Zaki HS, Henteleff HB. Quantitative description of temporomandibular joint sounds: defining clicking, popping, egg shell crackling and footsteps on gravel. J Oral Rehabil. 2001;28(5):466–78.
20. Ohrbach R, Greene C. Temporomandibular joint diagnosis: striking a balance between the sufficiency of clinical assessment and the need for imaging. Oral Surg Oral Med Oral Pathol Oral Radiol. 2013;116(1):124–5.
21. Schiffman EL, Ohrbach R, Truelove EL, Tai F, Anderson GC, Pan W, et al. The research diagnostic criteria for temporomandibular disorders. V: methods used to establish and validate revised Axis I diagnostic algorithms. J Orofac Pain. 2010;24(1):63–78.

22. Nickel J, Spilker R, Iwasaki L, Gonzalez Y, McCall WD, Ohrbach R, et al. Static and dynamic mechanics of the temporomandibular joint: plowing forces, joint load and tissue stress. Orthod Craniofac Res. 2009;12(3):159–67.
23. Ferrario VF, Sforza C, Tartaglia GM, Dellavia C. Immediate effect of a stabilization splint on masticatory muscle activity in temporomandibular disorder patients. J Oral Rehabil. 2002;29(9):810–5.
24. Proffit WR, Fields HW, Sarver DM. Contemporary orthodontics. New York: Elsevier Health Sciences; 2014.
25. Pullinger AG, Seligman DA, Solberg WK. Temporomandibular disorders. Part II: occlusal factors associated with temporomandibular joint tenderness and dysfunction. J Prosthet Dent. 1988;59(3):363–7.
26. Michelotti A, Farella M, Gallo LM, Veltri A, Palla S, Martina R. Effect of occlusal interference on habitual activity of human masseter. J Dent Res. 2005;84(7):644–8.
27. Michelotti A, Cioffi I, Festa P, Scala G, Farella M. Oral parafunctions as risk factors for diagnostic TMD subgroups. J Oral Rehabil. 2010;37(3):157–62.
28. Michelotti A, Cioffi I, Landino D, Galeone C, Farella M. Effects of experimental occlusal interferences in individuals reporting different levels of wake-time parafunctions. J Orofac Pain. 2012;26(3):168–75.
29. Dworkin SF, LeResche L, DeRouen T, Von Korff M. Assessing clinical signs of temporomandibular disorders: reliability of clinical examiners. J Prosthet Dent. 1990;63(5):574–9.
30. Bertazzo-Silveira E, Stuginski-Barbosa J, Porporatti AL, Dick B, Flores-Mir C, Manfredini D, et al. Association between signs and symptoms of bruxism and presence of tori: a systematic review. Clin Oral Investig. 2017;21(9):2789–99.
31. Häggman-Henrikson B, Eriksson P-O. Disturbed jaw behavior in whiplash-associated disorders during rhythmic jaw movements. J Dent Res. 2002;81(11):747–51.
32. Ferrario VF, Sforza C, Dellavia C, Tartaglia GM. Evidence of an influence of asymmetrical occlusal interferences on the activity of the sternocleidomastoid muscle. J Oral Rehabil. 2003;30(1):34–40.
33. Häggman-Henrikson B, Eriksson P-O. Head movements during chewing: relation to size and texture of bolus. J Dent Res. 2004;83(11):864–8.
34. Sforza C, Rosati R, De Menezes M, Musto F, Toma M. EMG analysis of trapezius and masticatory muscles: experimental protocol and data reproducibility. J Oral Rehabil. 2011;38(9):648–54.
35. Gerwin RD, Shannon S, Hong C-Z, Hubbard D, Gevirtz R. Interrater reliability in myofascial trigger point examination. Pain. 1997;69(1):65–73.
36. Ohrbach R, Gale EN. Pressure pain thresholds, clinical assessment, and differential diagnosis: reliability and validity in patients with myogenic pain. Pain. 1989;39(2):157–69.
37. De Coster PJ, Van den Berghe LI, Martens LC. Generalized joint hypermobility and temporomandibular disorders: inherited connective tissue disease as a model with maximum expression. J Orofac Pain. 2005;19(1):47–57.
38. Fischer L, Clemente JT, Tambeli CH. The protective role of testosterone in the development of temporomandibular joint pain. J Pain. 2007;8(5):437–42.
39. LeResche L, Saunders K, Von Korff MR, Barlow W, Dworkin SF. Use of exogenous hormones and risk of temporomandibular disorder pain. Pain. 1997;69(1–2):153–60.
40. Wang W, Hayami T, Kapila S. Estrogen/relaxin induce while progesterone represses MMP expression in TMJ fibrochondrocytes. J Dent Res. 2007;86:1279.
41. Ferrando M, Andreu Y, Galdon MJ, Dura E, Poveda R, Bagan JV. Psychological variables and temporomandibular disorders: distress, coping, and personality. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004;98(2):153–60.
42. Fricton J. Current evidence providing clarity in management of temporomandibular disorders: summary of a systematic review of randomized clinical trials for intra-oral appliances and occlusal therapies. J Evid Based Dent Pract. 2006;6(1):48–52.
43. Lindenmeyer A, Sutcliffe P, Eghtessad M, Goulden R, Speculand B, Harris M. Oral and maxillofacial surgery and chronic painful temporomandibular disorders—a systematic review. J Oral Maxillofac Surg. 2010;68(11):2755–64.

44. Sharma S, Crow HC, McCall WD Jr, Gonzalez YM. Systematic review of reliability and diagnostic validity of joint vibration analysis. J Orofac Pain. 2013;27(1):51–60.
45. Castroflorio T, Bargellini A, Rossini G, Cugliari G, Deregibus A, Manfredini D. Agreement between clinical and portable EMG/ECG diagnosis of sleep bruxism. J Oral Rehabil. 2015;42(10):759–64. https://doi.org/10.1111/joor.12320. Epub 2015 Jun 7. PMID: 26059761.
46. Gallo LM, Brasi M, Ernst B, Palla S. Relevance of mandibular helical axis analysis in functional and dysfunctional TMJs. J Biomech. 2006;39(9):1716–25.
47. Lötters FJ, Zwijnenburg AJ, Megens CC, Naeije M. Relationship between condylar and incisor point displacement during habitual maximum open-close movements. J Oral Rehabil. 1996;23(8):548–54.
48. De Felıcio CM, Mapelli A, Sidequersky FV, Tartaglia GM, Sforza C. Mandibular kinematics and masticatory muscles EMG in patients with short lasting TMD of mild-moderate severity. J Electromyogr Kinesiol. 2013;23(3):627–33.
49. Sessle BJ, Avivi-Arber L, Murray GM. Motor control of masticatory muscles. In: McLoon LK, Andrade F, editors. Craniofacial muscles: a new framework for understanding the effector side of craniofacial muscle control. New York: Springer; 2013. p. 111–30.
50. Bhaskaracharya M, Memon SM, Whittle T, Murray GM. Jaw movements in patients with a history of pain: an exploratory study. J Oral Rehabil. 2015;42(1):18–26.
51. Mapelli A, Galante D, Lovecchio N, Sforza C, Ferrario VF. Translation and rotation movements of the mandible during mouth opening and closing. Clin Anat. 2009;22(3):311–8.
52. Miyawaki S, Tanimoto Y, Inoue M, Sugawara Y, Fujiki T, Takano-Yamamoto T. Condylar motion in patients with reduced anterior disc displacement. J Dent Res. 2001;80(5):1430–5.

CBCT Evaluation of the TMJ

9

Dania Tamimi and Elnaz Jalali

Abstract

The temporomandibular joint is a complex structure that affects the growth, development, and maintenance of many of the structures of the oral and maxillofacial complex. In order to evaluate this joint and the changes that occur in the rest of the facial skeleton due to its dysfunction and pathology, the appropriate imaging needs to be procured, such as a large field of view cone beam CT scan that includes not only the TMJs but also the entire facial skeleton and upper respiratory tract. This chapter discusses the osseous radiographic anatomy of the TMJ and how different types of pathology change that anatomy and subsequently change the facial skeleton. Many of the TMJ osseous pathologies as they would be viewed on osseous imaging (CBCT or CT) are reviewed and discussed.

9.1 2D vs. 3D Imaging

Evaluation of the temporomandibular joint (TMJ) form and function can be achieved through different types of 2D radiographic techniques such as panoramic, lateral transcranial, transpharyngeal, and transmaxillary anteroposterior (AP) views [1, 2]. As a preliminary "screening" projection for TMJ evaluation, the panoramic radiograph is often used in combination with other hard tissue imaging modalities to detect gross osseous abnormalities within the joint [3, 4]. Its high availability,

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low-radiation dose, and low cost make panoramic radiography a popular imaging tool for diagnosis of sclerosis, osteophytes of condyle, and other large abnormalities [5]. However, diagnosis of early lesions, evaluation of soft tissue components of the joint, and even accurate assessment of the morphology and the spatial relationships of the TMJ osseous components cannot be achieved through panoramic imaging [3]. Condylar position cannot be evaluated reliably based on panoramic projection because the patient is placed in a protrusive and slightly open position. In addition, due to the angle of projection, the articular surfaces of the condyles are distorted, and clear imaging of the glenoid fossa is limited.

Conventional tomography is superior to panoramic radiography in disclosing more of the structural changes, evaluation of joint spaces, and providing the clinician with more information related to the precise morphology of the osseous components of the TMJ [3]. Furthermore, with conventional tomography, anatomic structures can be studied more accurately than transcranial radiography [6–8]. However, just like the panoramic technique, conventional tomography has limited capacity in the detection of early arthritic changes, progressive changes within the fossa, and imaging the soft tissue components of the joint [5]. Also, superimposition of the surrounding structures on the image in the plane of interest results in some inherent blurring of the image. With the advent of CBCT imaging, conventional tomography is now rarely employed for TMJ imaging.

TMJ arthrography is a mildly invasive technique for radiographic assessment of the joint space and surrounding soft tissue, cartilage, and lesions of the disc [3], where a radiopaque iodine-based contrast material is injected into the synovial space of the joint followed by radiography of the joint. The flow of the contrast material and function of the disc during opening and closing is monitored through fluoroscopy. Arthrography is a sensitive procedure which demands substantial prowess and experience. Due to the presence of the radiopaque contrast agent, arthrography is not capable of providing much accurate information regarding the osseous components of the TMJ. Other disadvantages of this technique include postoperative discomfort, risk of postoperative infection, and allergy to the contrast agent [3–5]. In addition, use of fluoroscopy in this technique can provide significant radiation exposure to the patient [5]. Arthrography is the best available imaging technique to identify perforations between the joint compartments and adhesions of the disc to the temporal component of the joint.

With three-dimensional imaging, accurate evaluation of the subarticular osseous components of the TMJ can be accomplished through multiplanar reconstruction (MPR) which facilitates not only 3D reconstruction of the image but simultaneously also provides series of cross-sectional slices of the tissue of interest in different orientation planes. With 3D imaging there is no superimposition of structures outside the area of interest. However, 3D imaging modalities such as magnetic resonance (MRI) and computed tomography (CT) demand large footprint, expert skill, and high radiation dosage and are costly [9–12]. Although MRI is considered as the best modality for evaluation of soft tissue pathology of TMJs [13] such as disc displacement and joint effusion, production of strong magnetic fields during imaging makes this technique hazardous for patients with metallic implants, pacemakers, or intracranial vascular clips [14].

Cone beam CT (CBCT) reduces the effective radiation dose to the patient by using a large cone-shaped beam to provide high-resolution volumetric images of the area of interest in only one rotation [10, 15–17]. Unlike CT's heavy helical scanners, CBCT scanners are much smaller and only require less radiation than MDCT [18]. In addition, less scatter radiation than conventional CT is produced [19]. Although CBCT dosage is much greater than conventional tomography, this technique provides undistorted, accurate evaluation of the osseous components of the TMJs, including assessment of TMJ pathology, bony ankylosis, fractures, dislocations, arthritis [5], as well as condylar position within the glenoid fossa in closed and open mouth positions [20], all of which may not be well imaged with 2D techniques [21, 22].

9.2 CBCT Imaging Protocols

When obtaining a CBCT imaging study of the TMJs, it is important to realize that many of the radiographic clues to diagnosis of TMJ disorders lie not in the TMJs themselves but in how the disorder affects the rest of the craniofacial complex. The TMJs are intricately connected and related to the jaws and the dentition as well as the upper respiratory tract and the skull base, and changes in one of these structures may affect the others. The growth and development of the mandible mirrors that of the TMJ, and if TMJ growth is stunted or if there are changes in the dimensions of the TMJ, the mandible will reflect these changes in an alteration of its osseous morphology, and these alterations are, in some cases, specific to certain TMJ disorder diagnoses. Changes in the occlusion, such as open bites, cross bites, cants, and inclination of teeth that may lead to a functional shift, can provide a clearer picture of the nature of the TMJ disorder. Thus, collimation of the field of view to only include the TMJs can result in an incomplete diagnosis due to exclusion of the important clues in the rest of the craniofacial complex.

Another important consideration is the position of the mandible during imaging. As soft tissue changes of the TMJs cannot be visualized on CBCT imaging, the condylar position in the maximum intercuspal position offers valuable clues to the TMJ soft tissue condition. Habitual changes in mandibular posture may indicate underlying airway patency deficiencies as well. A complete and accurate CBCT evaluation of the TMJs necessitates the inclusion of the entire mandible in the field of view with the teeth in maximum intercuspation. If a second view is required in any other position, such as open mouth, rest position, or with a splint in place, the second scan can be collimated to include just the TMJs to decrease the radiation dose to the patient.

9.3 Post-processing the CBCT Data of the TMJs

After acquiring the CBCT scan, the CBCT volume should be oriented correctly at the observer's workstation to align the skull anatomy to the axial, coronal, and sagittal plane (Fig. 9.1). This step is the first and most important step and should be performed on every scan reviewed. The patient's midline should be aligned with the sagittal plane, the structures that line up with the coronal plane (such as

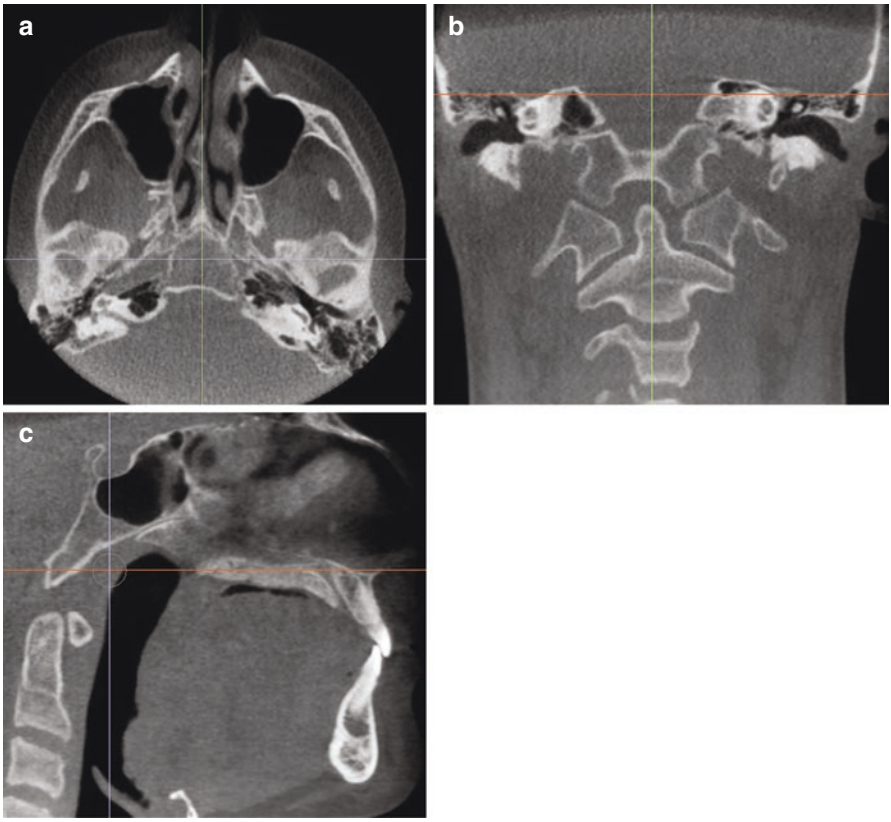

Fig. 9.1 Prior to evaluating a CBCT volume, anatomic orientation is necessary to increase efficiency and efficacy. In the axial view (**a**), orientation of the coronal plane (blue line) with relatively consistent anatomy, such as the foramen ovale, as shown here, will orient the skull anatomically in coronal plane. In the coronal view (**b**), orientation of the axial plane (red line) with, for example, the tegmen tympani (roof of the middle ear cavity) will orient the skull anatomically mediolaterally in the axial plane. In the sagittal view (**c**), orientation of the axial plane (red line) with the hard palate (from the anterior nasal spine to the posterior nasal spine) or with the Frankfort plane will orient the skull anatomically mediolaterally in the axial plane

foramen ovale and external auditory canals) should be aligned with the coronal plane and the structures that normally lie in the axial plane (such as the External auditory canals, ossicles or tegmen tympani) should be aligned with the axial plane. This allows for accurate and efficient evaluation the anatomy of the head and is very useful in detecting asymmetries in the patient's anatomy, many of which commonly occur with TMJ disorders. After orientation is complete, the data can be evaluated for pathology and reformatted to obtain the diagnostic cross sections, panoramic reformation, and 3D reconstruction (Fig. 9.2). The most helpful

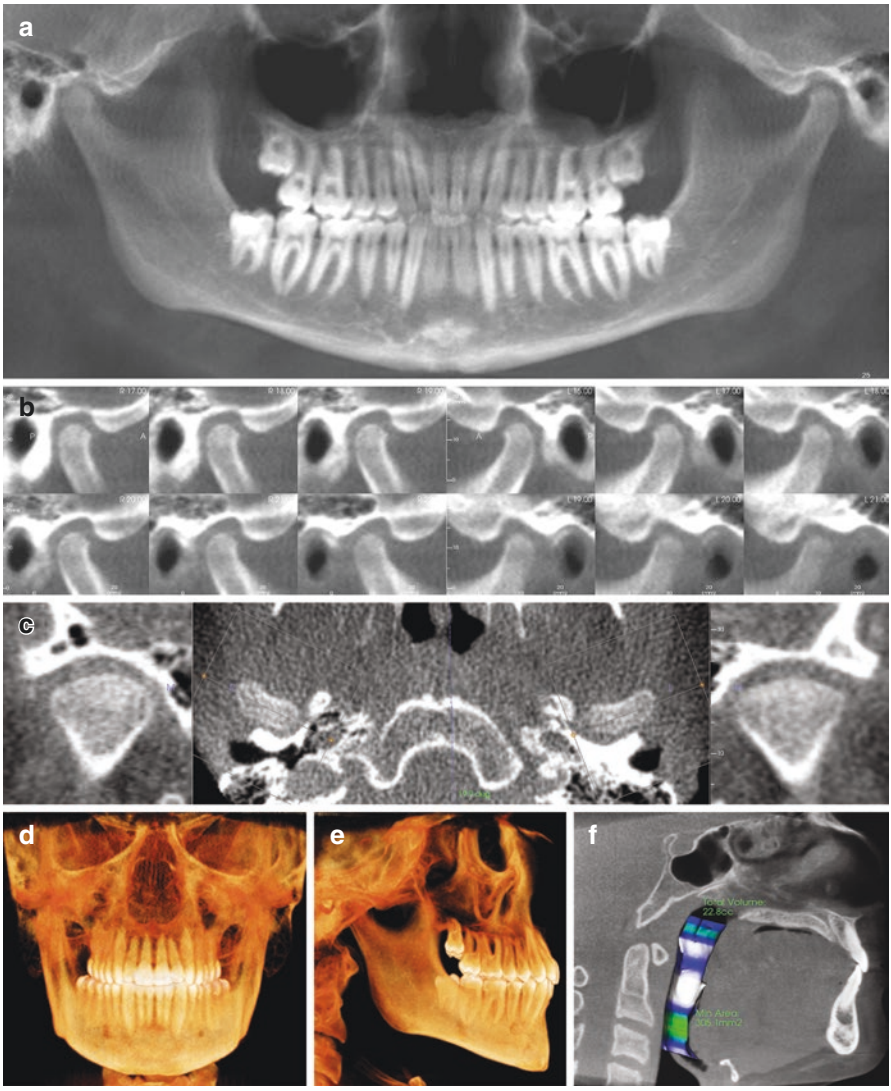

Fig. 9.2 Following the orientation of the CBCT volume, cross sections specific for TMJ evaluation and diagnosis can be produced: (**a**) panoramic reformat, to give an overall view of the jaws; (**b**) sagittal oblique cross sections to show the anteroposterior morphology and spatial relationship of the TMJs; (**c**) coronal oblique and axial cross sections to show the mediolateral morphology and spatial relationship of the TMJs; (**d**) frontal 3D rendering to evaluate for asymmetry and transverse discrepancies which may be caused by the TMJ condition; (**e**) lateral 3D rendering to evaluate for asymmetry and sagittal changes to the jaws; and (**f**) airway analysis, which allows the evaluation of the effects of the TMJ condition on the oropharyngeal airway

reformations are the axially corrected sagittal oblique and coronal oblique cross sections (used to evaluate condylar and fossa morphology and spatial relationships), the panoramic reformation (to allow a visualization of the entire maxillofacial complex in a plane familiar to the dental practitioner), and the frontal and lateral 3D renderings (helpful in detecting mandibular asymmetries and occlusal changes). Other reformations can be created at the discretion of the observer for specific diagnostic tasks. Upper respiratory tract narrowing should be observed with these reformations as a possible consequence of TMJ disorders, and the inadequate of growth of the jaws or posterior rotation of the mandible may result in inferior and posterior displacement of the tongue into the oropharynx, narrowing the airway. Other observable changes are narrowing of the transverse dimension of the maxilla as a result of inferior positioning of the tongue and narrowing of the transverse dimension of the nasal cavity (which shares its floor with the palate). With these reformats in hand, the observer can appreciate the TMJs and their effect on the craniofacial complex.

9.4 CBCT TMJ Anatomy Review

The hard tissue and soft tissue anatomy of the TMJ has been reviewed elsewhere in this book. This section focuses on TMJ anatomy as visualized on CBCT axially corrected sagittal and coronal oblique cross sections (Fig. 9.3).

When the TMJ is viewed on CBCT axially corrected sagittal oblique sections, one should observe the morphology of the cortical outline and trabecular pattern of the condyle and the fossa. The cortical outline of the condyle in the neck of the condyle is thick and gradually tapers into a posterior height of contour posteriorly and the anterior height of contour (above the pterygoid fovea) anteriorly. The articular surface should be convex with a continuous cortex that is very thin in an adult (indicating the health of the overlying fibrocartilage) and non-apparent in a growing child (indicating rapid bone turnover during growth). The presence of articular cortical thickening in an adult suggests a response to increase biomechanical loading. An articular surface cortex in a child is often seen with end-stage degenerative changes. The trabecular pattern should be uniform with no areas of sclerosis or low density, which indicate abnormality. The fossa should have a thick, continuous, and uniform cortical outline, and the posterior slope of the eminence should be rounded and smooth and gently sloping toward the crest of the eminence. The marrow spaces should have a uniform trabecular pattern. The mastoid air cells can pneumatize the roof of the fossa and the eminence, and this is within normal limits.

When viewing the TMJ in the coronal oblique view, the observer can once again notice the tapering of the cortices of the condylar neck toward the medial and lateral poles of the condyle, with a very thin cortex overlying the condylar articular surface in adults and no articular cortex in children but a convex and uniform articular surface. The spatial relationships of the condyle with the fossa are described below.

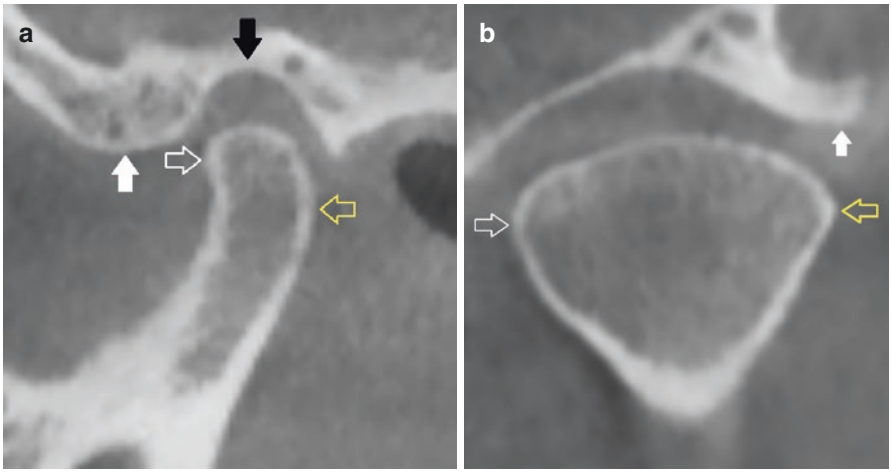

Fig. 9.3 The osseous morphology of the TMJ as seen on CBCT axially corrected sagittal oblique (**a**) and coronal oblique (**b**) cross sections. The arrows point to important diagnostic landmarks that help the observer determine whether the condyle has normal contours or has been altered due to disease. On (**a**), the yellow hollow arrow points to the posterior height of contour, and the white hollow arrow points to the anterior height of contour of the condyle. The black solid arrow points to the roof of the fossa, and the white solid arrow points to the crest of the eminence. On (**b**), the white solid arrow points to the lateral rim of the fossa. The yellow and white hollow arrows point to the lateral and medial poles of the condyle, respectively

9.5 Deciphering Closed-Mouth Condylar Position on CBCT

If soft tissue information provided with MRI imaging is not available, the spatial relationships of the TMJ osseous components can offer the observer some clues to the condition of the joint space soft tissues. It is impossible to visualize the disc on CBCT, but a condyle that is not in the normal position may indicate disc displacement or other abnormalities. Changes in condylar position can also indicate problems with the occlusion and the presence of lesion in or around the joint space. Thus it is imperative to obtain the closed-mouth scan in maximum intercuspation. The normal condylar position in the sagittal oblique view is when the condyle is positioned slightly anterior and slightly superior to the center of the fossa. If the observer were to imagine a biconcave disk interposed between the osseous components, with the thick posterior band of the disk located at superior to the condyle, and the thin intermediate zone located between areas of maximum curvature of the anterior aspect of the condyle and the posterior slope of the eminence (Fig. 9.4a, b). On the coronal oblique view, the observer should image that a crescent-shaped thick posterior band is located superior to the condyle and that the medial and lateral aspects of the disc evenly taper toward the poles of the condyle (Fig. 9.4c, d). If the condyle is shifted and is narrowing the joint space in any direction, one can assume that there are underlying soft tissue changes, although the nature of these changes and the direction of disc displacement—if present—cannot be

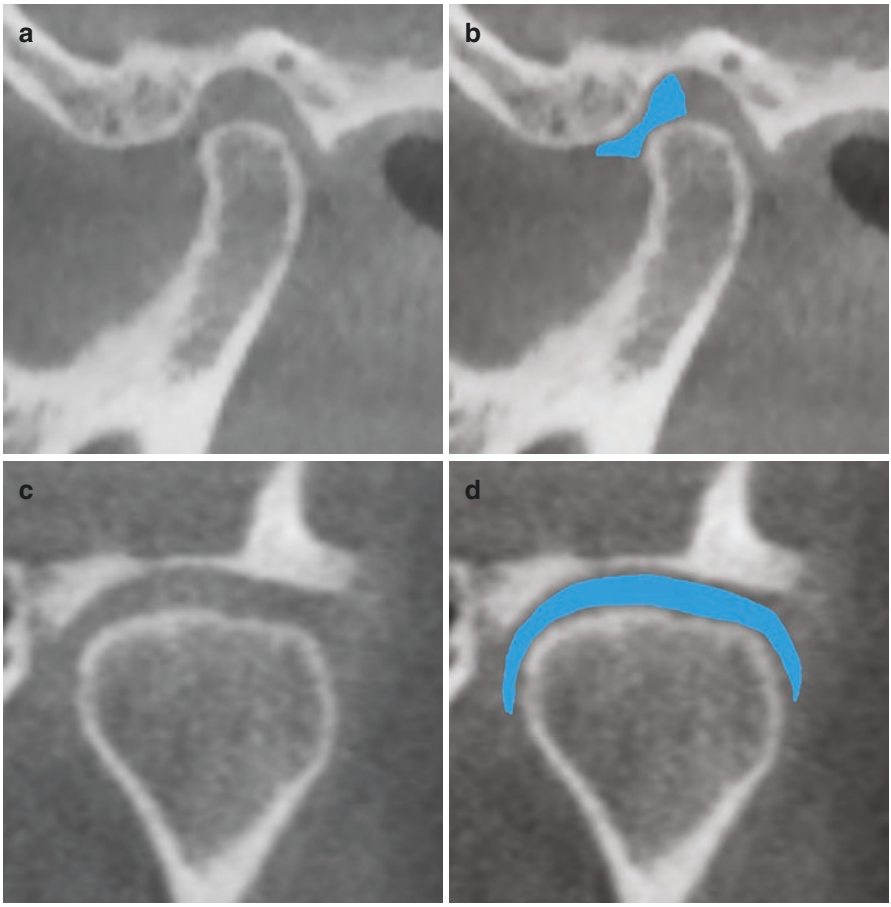

Fig. 9.4 These cross sections illustrate a visual exercise that can be done when evaluating the entire set of cross sections to determine whether the condyle is in the correct position in relationship to the fossa. One can imagine that a biconcave disc can fit in the joint space of the sagittal cross sections (**a** and **b**) and a crescent-shaped disc in the coronal cross section (**c** and **d**)

ascertained. The following are some of the commonly encountered abnormal condylar positions on CBCT and a brief discussion of their differential diagnosis.

9.5.1 Anterior Condylar Position

An anterior condylar position in an acquired position that is most likely an orthopedically unstable position—that is to say that there is a chance that the condyle will eventually seat back into the fossa, resulting in bite changes, the most common being an anterior open bite due to the posterior rotation of the mandible around a fulcrum created on the last molar teeth.

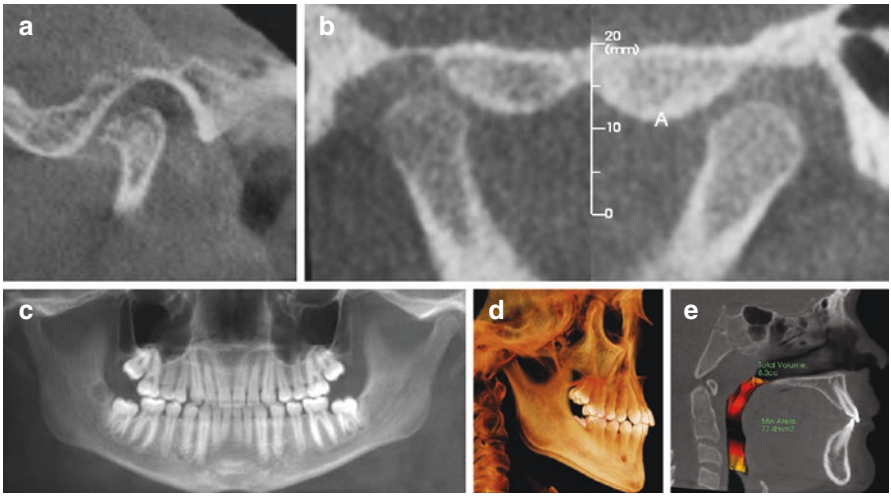

Fig. 9.5 CBCT reformats for common diagnosis for anterior condylar position: (**a**) end-stage degenerative joint disease and (**b**–**e**) dual bite formation in order to increase oropharyngeal airway dimensions. (**b**) Cross sections show anterior position of normal contoured condyles; (**c**) panoramic reformat shows the teeth in maximum intercuspation with the condyles anteriorly positioned, a hallmark of dual bite formation; (**d**) lateral 3D reformat shows the flattening of the maxillary curve of Spee, indicating that the posterior teeth have supererupted after chronic anterior posturing of the mandible; and (**e**) airway reformation shows a narrow oropharyngeal airway, which may indicate that the reason for the anterior posturing of the mandible was to increase patency of the airway

When the observer encounters an anteriorly positioned condyle, the differential diagnosis should include adult or juvenile degenerative joint disease, where the anterior condyle position is caused by reduction of condylar and eminence volume (Fig. 9.5a). A dual bite is another relatively common diagnosis for an anterior condylar position, but in this case, the condylar height and volume are normal, and the anterior position of the condyle is a result of a chronic acquired position of the mandible, either due to habitual anterior mandibular posturing (as a result of habit or increasing airway patency) or anterior repositioning devices, such as functional appliances and anterior repositioning splints (Fig. 9.5b–e). Anterior position of the condyle can also be observed in cases of skeletal Class III growth patterns; there could be a functional shift of the mandible due to the locking out of the mandible anteriorly due to a complete anterior cross bite relationship. End-stage inflammatory changes, which like degenerative disorders, can result in reduction of condylar and eminence volume and can also result in an anterior position of the condyle. The least common diagnosis would be of a space-occupying mass posterior to the condyle, which would displace the affected condyle anteriorly (a unilateral appearance) with corresponding changes in mandibular position and occlusion. The space-occupying mass may be soft tissue (completely radiolucent), in which case the only clue to its presence may be the position of the condyle and, if large enough, the remodeling of the condyle and the fossa. If the

space-occupying mass is of mixed or high density, the diagnosis becomes easier on CBCT.

9.5.2 Posterior Condylar Position

A posterior condylar position is often associated with regressive remodeling of the posterior aspect, either appearing as a flattening, a broad concavity, and/or reduction of the anteroposterior dimension of the condyle without loss of condylar height (Fig. 9.6c). These may or may not progress to degenerative joint disease.

The posterior condylar position is often associated with anterior disc displacement (a) but can also be due to a functional shift and posterior displacement of the mandible due to retroclination of the maxillary central incisors (Fig. 9.6b–d). When these teeth close in maximum intercuspation, there is a posterior and inferior displacement of the condyle, which is not an orthopedically stable position. Bilateral mandibular sagittal split osteotomies with bicortical stabilization have a tendency to torque, rotate, and displace the proximal osteotomy segments of the mandible, resulting in posterior position of the condyle and often present with regressive remodeling. Most displaced fractures of the condylar neck result in anterior, inferior, and medial displacement of the condyle due to the action of the lateral pterygoid muscle. When the neck sustains trauma and a non-displaced fracture occurs, the medial pterygoid will elevate the angle of the mandible slightly, causing a posteriorly angles condylar fragment. Lastly, when the contralateral condyle is enlarged (such as in cases of osteochondroma and condylar hyperplasia), the unaffected condyle can become displaced posterior due to shifting of the mandible to the unaffected side.

9.5.3 Superior Condylar Position

The superior joint space is usually the largest of the joint space areas on CBCT as it contained the thickest part of the disc—the posterior band. If there are structural changes to the disc or if it no longer occupies the superior joint space area, the superior joint space is narrowed. In general, a small superior joint space is only considered normal in skeletal Class III growth patterns (Fig. 9.7a, b).

Disc displacement, soft tissue thinning, and perforation are the most common causes of the superior joint space narrowing appearance. Without the soft tissue thickness in this area, the condyle displaces superiorly (Fig. 9.7c). On the coronal oblique view, one may observe either a uniform mediolateral thinning of the joint space between the condyle and the fossa or a shifting of the condyle medially and superiorly or laterally and superiorly (Fig. 9.7d). This sideways shift of the condyle in this plane may indicate an anterior rotational disc displacement or a true sideways disc displacement, but this cannot be evaluated with certainty without MRI correlation. The reduction of condylar height due to degenerative or inflammatory disorders and possible subsequent seating of the condyle often results in a condyle

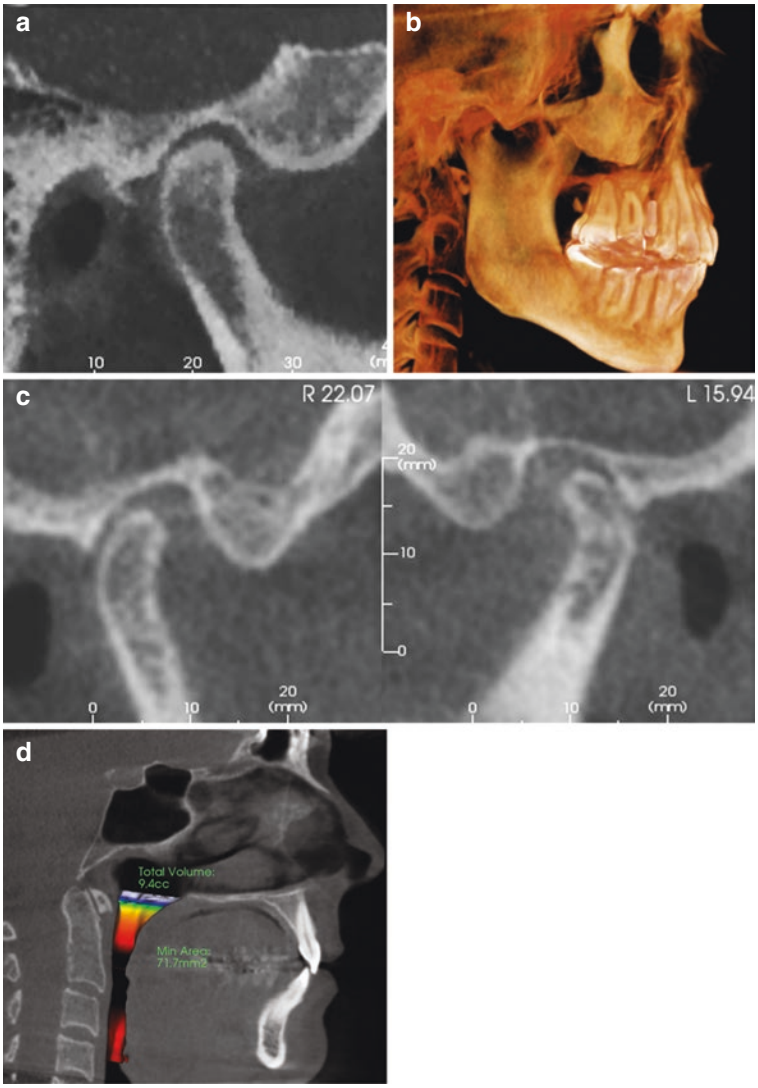

Fig. 9.6 CBCT reformats for common diagnoses for posterior condylar position: (**a**) disc displacement (MRI verified) and (**b–d**) functional shift of the mandible. The maxillary central incisors are vertically oriented (**b**), and this has shifted the mandible and condyles posteriorly (**c**), which may also contribute to the narrowing of the oropharyngeal airway (**d**) due to posterior repositioning of the tongue along with the mandible

position more superior than the relatively more centered position of the condyle in a normal TMJ. Finally, in rare cases of mandibular trauma, fracture of the glenoid fossa roof can occur, resulting in a superior displacement of the condyle into the middle cranial fossa.

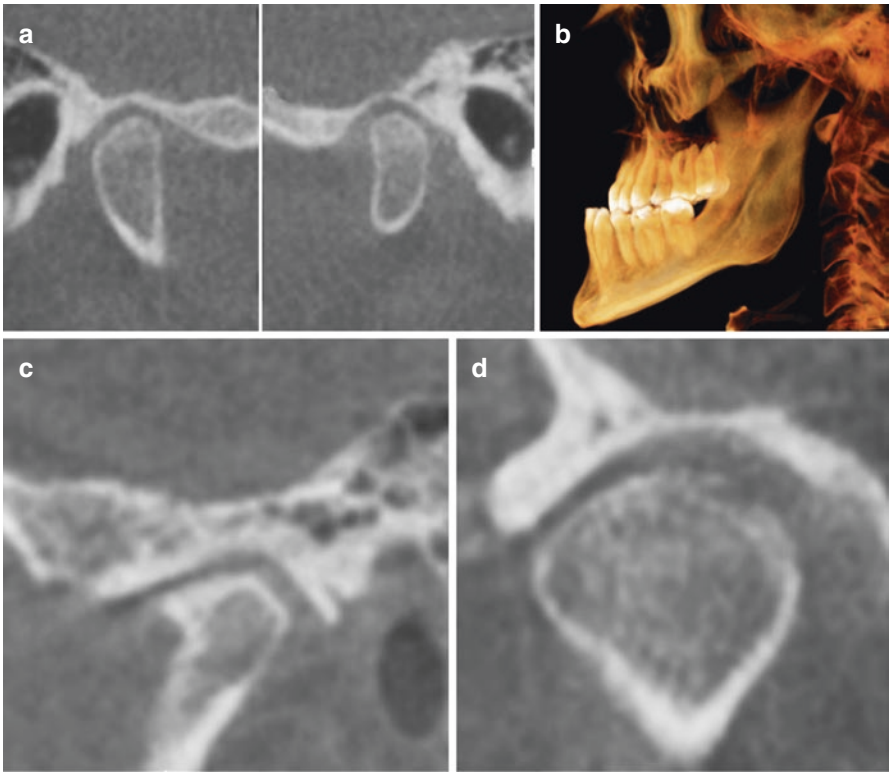

Fig. 9.7 CBCT reformats of some of the more common differential diagnoses for superior condylar position: (**a** and **b**) Class III skeletal malocclusion is often associated with superior condylar position, (**c**) disc displacement, and end-stage DJD, and (**d**) disc displacement off of one side of the condyle may lead to repositioning of the condyle to that side

9.5.4 Inferior Condylar Position

An inferior condylar position is usually a result of a fracture of either the condyle itself or fracture of the neck with displacement due to lateral pterygoid muscle action (Fig. 9.8a); loss of condylar height due to degenerative or inflammatory disease; inferior displacement of the condyle due to presence of a space-occupying mass superior to the condyle such as synovial chondromatosis, tenosynovitis, or TMJ neoplasia (Fig. 9.8b, c); or displacement due to overgrowth of the condyle (hyperplasia or acromegaly). Very rarely, malignancy or surgical procedure affecting the trigeminal nucleus, trigeminal ganglion, or the CNV3 at or post-foramen ovale can denervate the muscles of mastication, resulting in muscle weakness or paralysis. This results in loss of muscle tone, atrophy, and subsequent fatty replacement of the mandibular elevator muscles which causes the appearance of the condyle dropping out of its fossa.

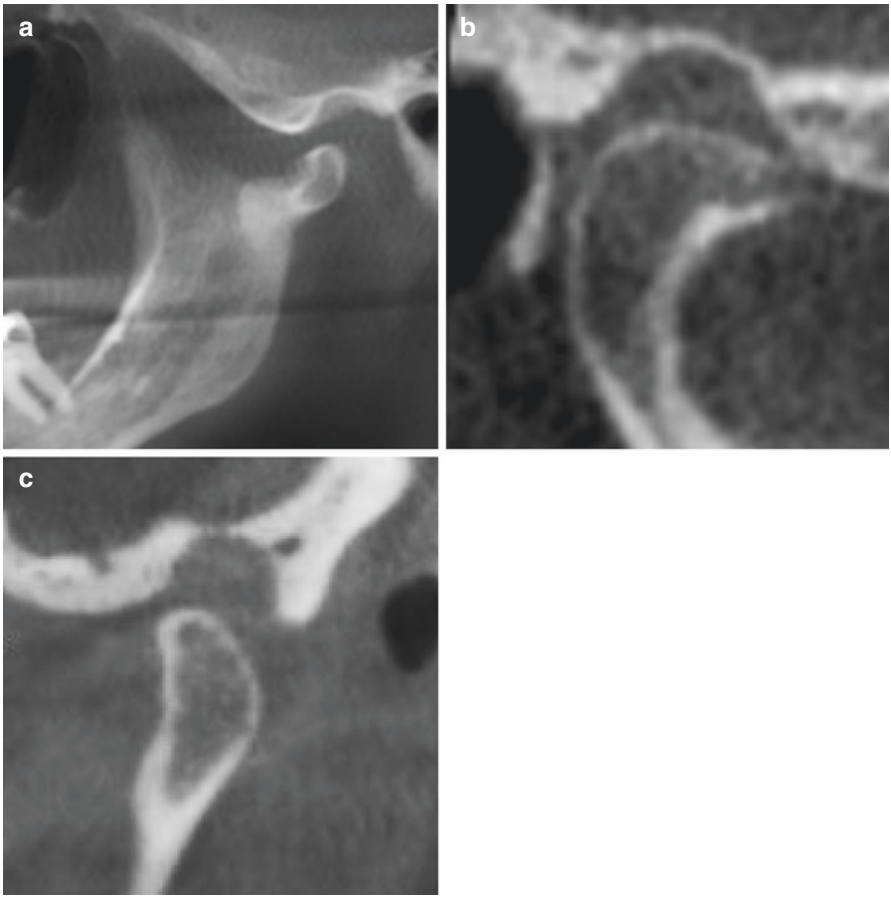

Fig. 9.8 CBCT reformats of some of the differential diagnoses for inferior condylar position: (**a**) condylar fracture, (**b**) osteochondroma (similar to an osteophyte but displaces condylar out of the fossa), and (**c**) space-occupying masses in the joint space (the mass itself may not be visualized radiographically, but if it is large enough its effect on the fossa and condyle will) (Courtesy, M. Bourgeois, DDS)

9.6 Deciphering Open-Mouth Condylar Position on CBCT

In the absence of MRI imaging, the range of motion of the condyle can be evaluated clinically and on an open-mouth CBCT scan. The normal range of motion for the condyle in the open position would be to be inferior to the crest of the eminence, slightly posterior and inferior to the crest of the eminence or slightly anterior and inferior to the crest of the eminence (Fig. 9.9a) and of course would have to be correlated to clinical maximum opening. If the condyle is positioned posterior and superior to the crest of the eminence, it is restricted (Fig. 9.9b, c),

although the reason for the restriction cannot be visualized on CBCT unless it is calcified. The mandible should be evaluated for signs of coronoid hyperplasia. If the condyle is positioned anterior and superior to the crest of the eminence, it may indicate hypermobility or subluxation (Fig. 9.9d). The distance between the condyle and eminence can be evaluated to see if a bone-on-bone relationship occurs, which indicate perforation of the soft tissues in this area. A relatively open mouth joint space between the condyle and eminence is suggestive of a disc interference.

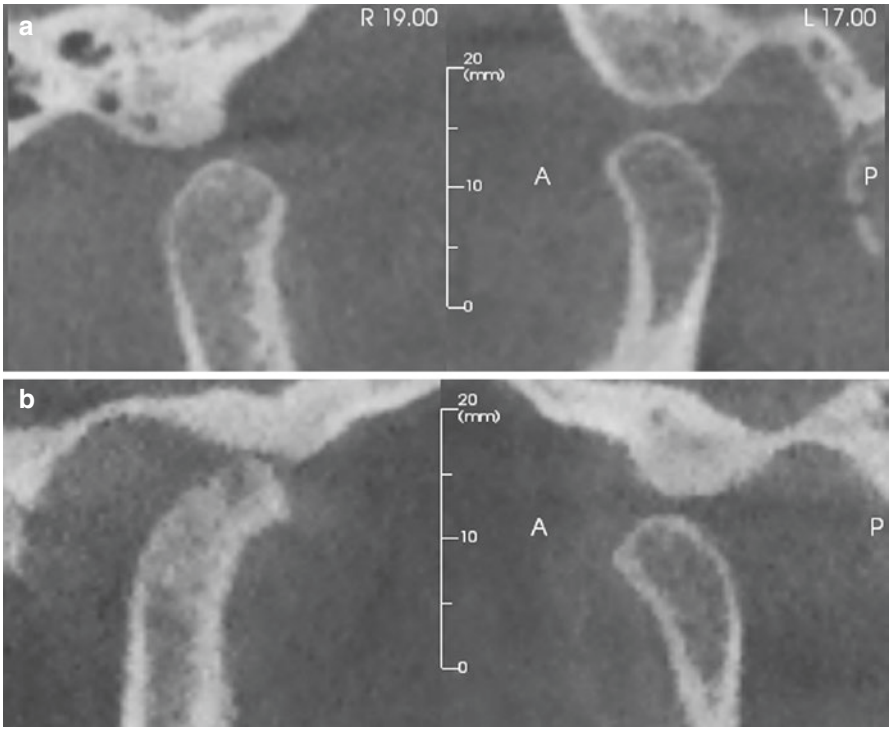

Fig. 9.9 CBCT reformats show different ranges of motion: (**a**) normal range of motion (at the level of the crest of the eminence); (**b**) unilateral limitation of condylar translation on the right; (**c**) bilateral limitation of condylar motion, with the condyles located posterior and superior to the crest of the eminence and in contact with the posterior slope of the eminence; and (**d**) hypermobility of the left condyle, with the condyle located anterior and superior to the crest of the eminence

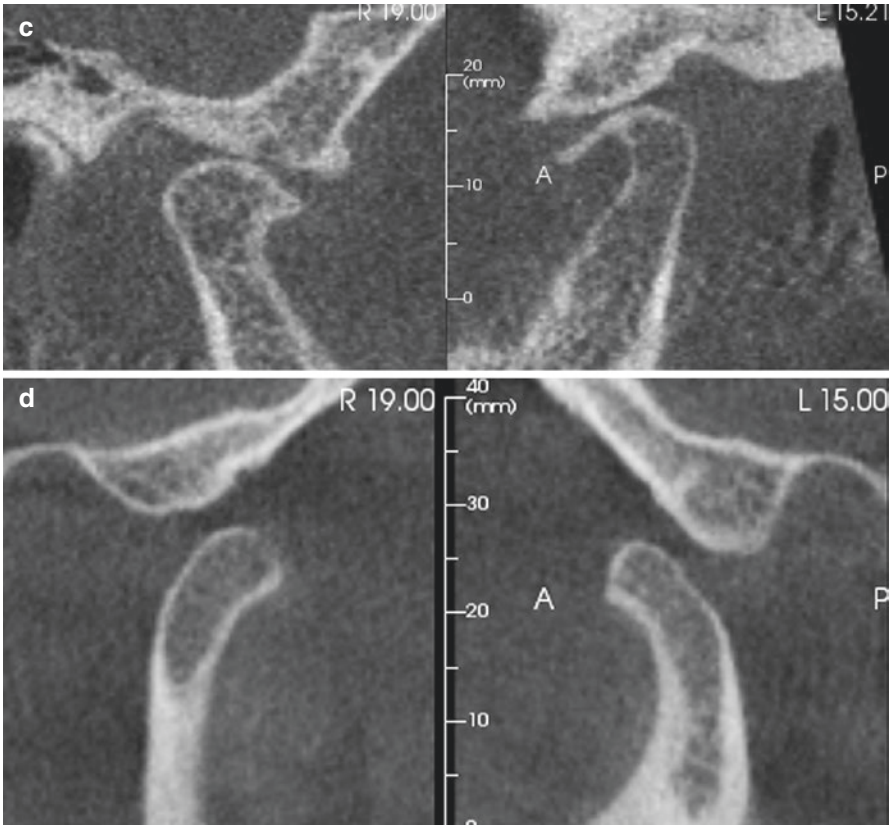

Fig. 9.9 (continued)

9.7 Radiographic Evaluation of TMJ Disorders on CBCT

CBCT imaging is ideal in evaluation of structural changes of the osseous morphology of the TMJs. While not all TMJ conditions are covered in this chapter, the CBCT radiographic appearance of some of the more commonly encountered pathosis will be discussed. Those are the degenerative changes (degenerative changes in adults and in children), the inflammatory diseases (rheumatoid arthritis and juvenile idiopathic arthritis), developmental changes (hemifacial microsomia, condylar hypoplasia, and condylar hyperplasia), and tumors (osteochondroma, osteoma, malignancies) and tumor-like lesions (synovial chondromatosis and calcium pyrophosphate dihydrate (CPPD) deposition disease).

9.7.1 Degenerative Disorders of the TMJ

9.7.1.1 Adult Degenerative Joint Disease (DJD)

Also known as osteoarthritis, this is the breakdown of the osseous articular surfaces of the TMJs and is the sequela of the loss integrity of the soft tissues of the disc, which was discussed in previous chapters. The biomechanical threshold of these articular surfaces is met and exceeded without the compressive and tensile properties of the disc absorbing the loading forces. This results in the structural disintegration of the underlying bone. The bone undergoes active degenerative changes, which manifest radiographically initially as non-corticated erosions (Fig. 9.10a) that eventually repair and recorticate as the biomechanical condition of the TMJ stabilizes. The surface area of the condyle increases to distribute the load, resulting in flattening, reduction of condylar height, sclerosis, and anterior osteophyte formation (Fig. 9.10b). A frequently seen end-stage finding is the subchondral bone "cyst" which is a round low density in the subarticular surface area (Fig. 9.10c). These "cysts" communicate with the articular surface through a short and thin radiolucent

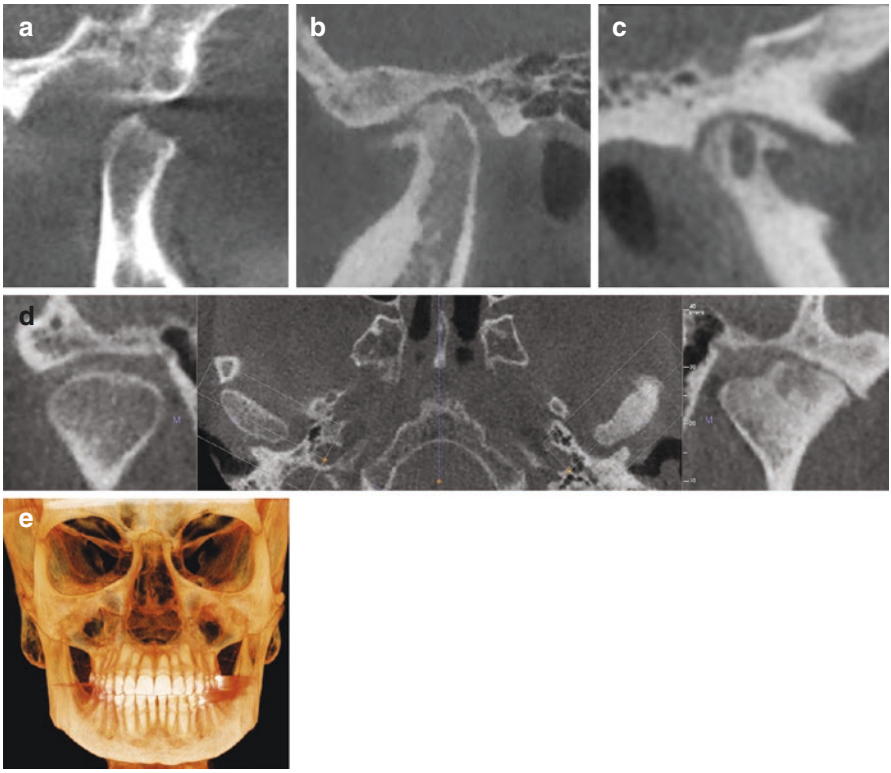

Fig. 9.10 CBCT reformats show (**a**) active DJD, with an erosion noted on the articular surface, (**b**) stable DJD with flattening and sclerosis of the articular surfaces and anterior osteophyte formation, (**c**) stable DJD with a large subchondral bone cyst connecting with the articular surface, (**d** and **e**) minimal mandibular asymmetry in unilateral adult DJD

tract, pathognomonic for this finding. These degenerative changes are often coupled with joint space narrowing, indicative of the disc displacement that preceded the degenerative joint disease. As the condylar height reduction occurs in adulthood (after the growth and development of the mandible is complete), these changes manifest in very mild if any mandibular asymmetry (Fig. 9.10d, e).

9.7.1.2 Juvenile Degenerative Joint Disease

Also known as progressive condylar resorption (PCR) and idiopathic condylar resorption (ICR), these are changes that are similar to adult degenerative joint disease in that they are preceded with the soft tissue changes of disc displacement and breakdown followed by lysis and repair of the osseous articular surfaces, but they differ in the morphology of the condyle as it undergoes degeneration (Fig. 9.11a, b). The end-stage product appears different in juvenile cases and does not usually manifest as sclerosis, large osteophytes, and subchondral bone cysts. More profoundly, because these juvenile degenerative changes occur in a growing mandible (most occur peripuberty), the changes in the mandibular skeleton is more pronounced. If the condition occurs in one TMJ only, the result is mandibular asymmetry (Fig. 9.11c). If it occurs in both TMJs, a symmetric but small and posteriorly rotated mandible with a steep mandibular plane and high gonial angle often occurs (Fig. 9.11d), and an open bite can occur (Fig. 9.11e). There could be narrowing of the oropharyngeal airway observed on the CBCT due to the posterior rotation of the mandible (Fig. 9.11f). If these changes occur closer to the end of the growth of the mandible, the mandibular changes are not as pronounced as when they occur earlier in the active growth phase.

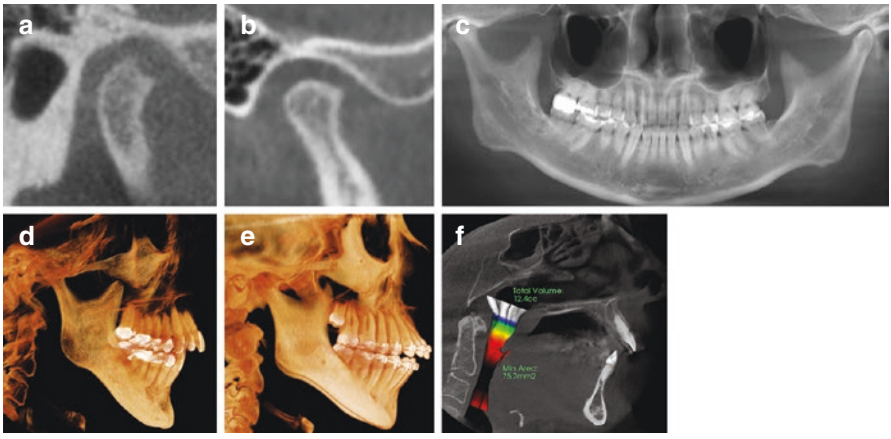

Fig. 9.11 CBCT reformats for different patients with juvenile degenerative joint disease in the (**a**) active phase and (**b**) stable phase (note the location of the condylar heights of contours in relation to one another). Some of the skeletal and soft tissue changes that may occur with any condition that reduced the size of the condyle, including this one, are (**c**) marked mandibular asymmetry (if unilateral), (**d**) small posteriorly rotated mandible with high gonial angle and steep mandibular plane (if bilateral), (**e**) anterior open bite (can occur with posterior rotation of the mandible), and (**f**) small oropharyngeal airway (with small, posteriorly rotated mandible)

9.7.2 Inflammatory Disorders of the TMJ

9.7.2.1 Rheumatoid Arthritis (RA)

This is a chronic inflammatory disease that manifests synovial membrane inflammation in several joints and is characterized by progressive bony erosions and cartilage destruction. While no radiographic signs are seen on CBCT in the early phase of the disease, the disease may manifest in the osseous components of the TMJs as generalized osteopenia of the condyle and the eminence. Erosions occur on the articular surfaces that can destroy the entire condyle and articular eminence (Fig. 9.12a), and there may be late fibrous or bony ankylosis (Fig. 9.12b). As there is condylar height reduction bilaterally, it is common to see an anterior open bite develop due to the posterior rotation of the mandible. It is sometimes difficult to distinguish from degenerative joint disease, but the erosions in RA occur on both the posterior and the anterior aspects of the condyle, giving it the "sharpened pencil" appearance. Other joints may be affected, and lesions may occur on the cervical spine and craniovertebral junction.

9.7.2.2 Juvenile Idiopathic Arthritis (JIA)

This is an autoimmune musculoskeletal inflammatory disease of childhood. On CBCT, the condyles are flat and deformed, sometimes referred to as "condylar stumps," and the fossa is wide and the eminence flat (Fig. 9.13a, b). Due to the flattening of the eminence, the condyles are often repositioned anteriorly. This disease can occur bilaterally or unilaterally, and the bilateral presentation is usually more common. The mandibular changes are usually more profound than RA due to the younger population it occurs in, and these changes include a small mandible, a steep mandibular plane, a high gonial angle, and a deepened antegonial notch. There are other observable changes on the CBCT, such as an elongated coronoid process, a small oropharyngeal airway (due to posterior rotation of the

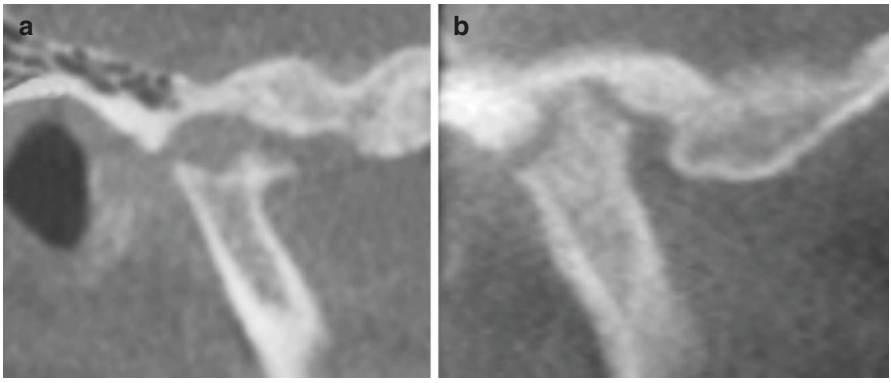

Fig. 9.12 A TMJ affected by rheumatoid arthritis (RA) may be difficult to differentiate from degenerative joint disease radiographically and should be assessed clinically as well. Some of the radiographic clues for RA are irregular and pronounced destruction of the articular surfaces (**a**). End-stage RA may result in fibrous or bony ankylosis of the joint (**b**) (Courtesy, D, Hatcher, DDS)

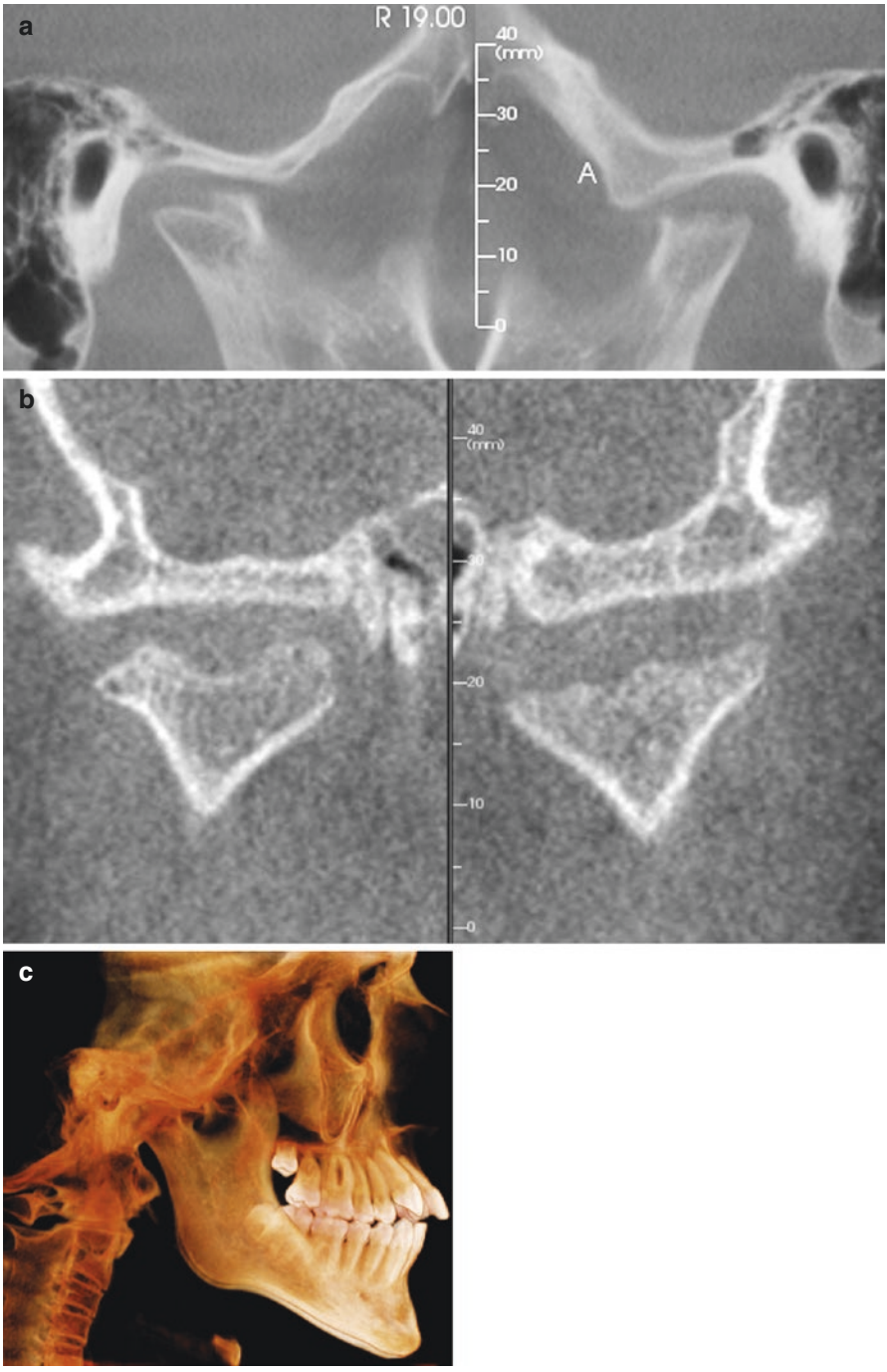

Fig. 9.13 CBCT images of a 15-year-old male with juvenile idiopathic arthritis (JIA) show pronounced destruction of the articular surfaces (**a** and **b**), fusion of the posterior elements of the spine, and coronoid process elongation (**c**) (Courtesy, D. Hatcher, DDS)

small mandible), and fusion of the posterior elements of the cervical spine (Fig. 9.13c).

9.7.3 Developmental Disorders

9.7.3.1 Hemifacial Microsomia (HFM)

This is a developmental anomaly of unknown origin unilaterally affecting the derivatives of the first and second branchial arched, including the ear, facial soft tissues, mandible, zygoma, and orbit. The condyle is small or absent. The fossa is relatively undeveloped. There is decreased ipsilateral ramus and body development, and the occlusal plane is elevated on the affected side. The zygoma is small, and the zygomatic arch often has a defect, and the orbit is displaced inferiorly. Middle and external ear defect or aplasia is often seen (Fig. 9.14).

9.7.3.2 Condylar Hypoplasia

This involves a developmentally small condyle which limited growth of the ipsilateral half of the mandible, leading to mandibular asymmetry (Fig. 9.15). The shape of the condyle is normal, with all of the cortices and contours within normal limits, but the condyle is either small or short, resulting in a developmentally smaller mandibular

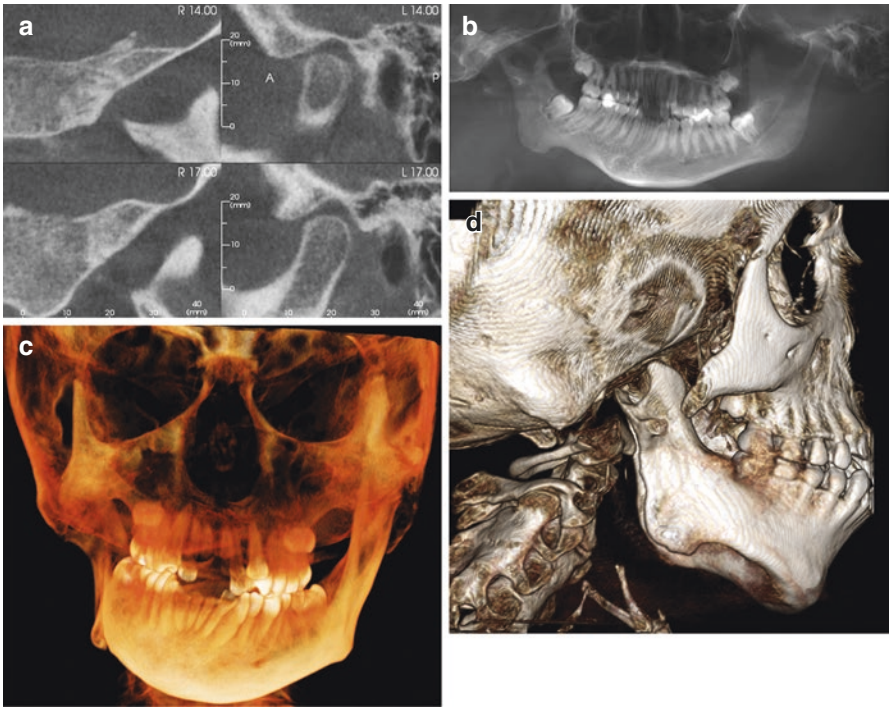

Fig. 9.14 CBCT reformations show some of the features of hemifacial microsomia. The right affected side shows a lack of TMJ development (**a**) and a poorly developed mandible, resulting in asymmetry (**b**–**d**). The zygomatic arch is absent (**d**) (Courtesy, F. Eraso, DDS)

ramus and body on that side. The antegonial notch is usually deepened, the ramus is often medially bowed or positioned, and an occlusal cant with the smaller side being more elevated is often seen. The chin is deviated to the affected side. The degree of asymmetry depends on the age at which the condyle was affected, and degenerative changes can occur as a long-term sequela. Hypoplasia may occur secondary to a TMJ insult, such as a trauma or displaced disc during development.

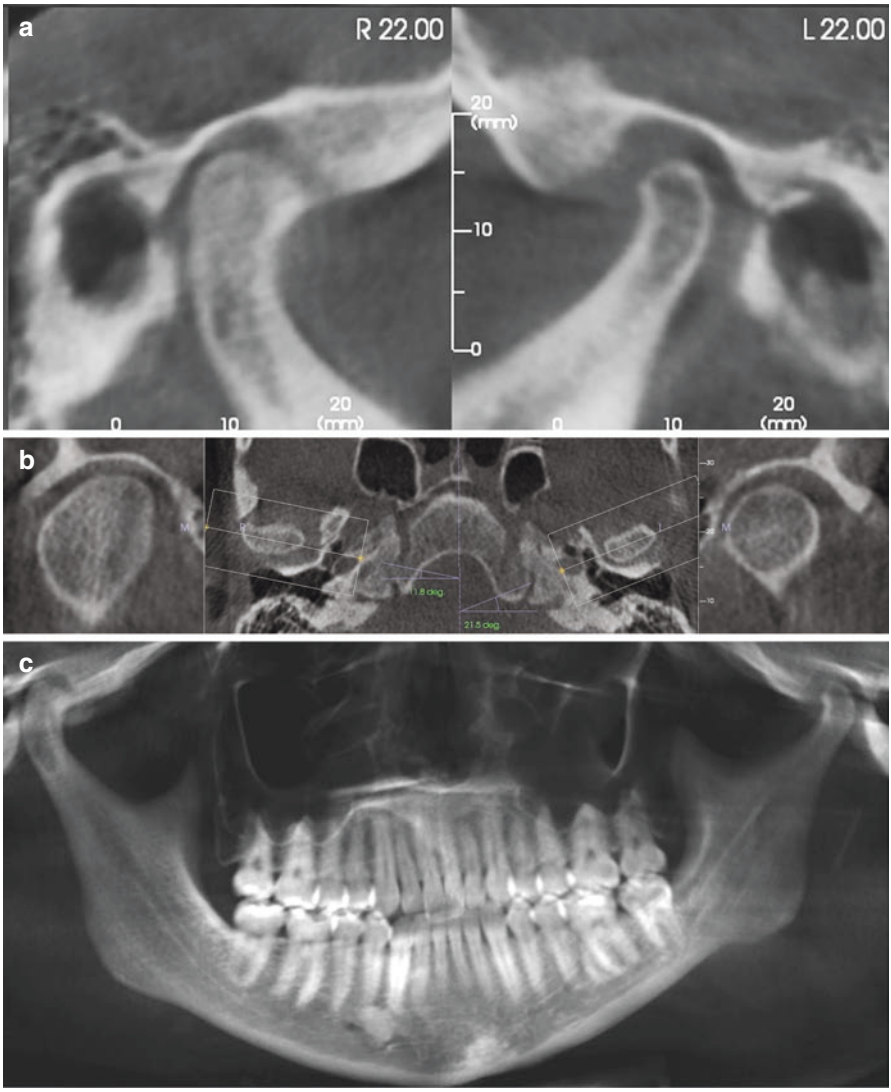

Fig. 9.15 CBCT reformations in a patient with condylar hypoplasia shows reduction of condylar size on the left (**a** and **b**) with preservation of the position of the contours of the condyle previously discussed in Fig. 9.3, indicating that this is a developmental change and not reduction due to destruction. The result of the small condyle on the left is a small left side of the mandible (**c**–**e**). The differential diagnosis includes condylar hyperplasia on the right, but the morphology of the left side of the mandible indicate growth changes consistent with condylar hypoplasia

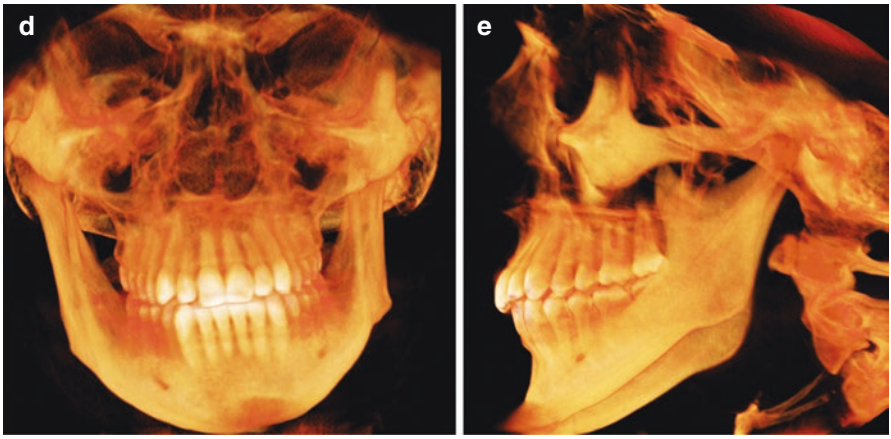

Fig. 9.15 (continued)

9.7.3.3 Condylar Hyperplasia (Hemimandibular Hyperplasia)

This is a slowly developing overgrowth of the mandibular condyle which leads to a regionally overgrowth of the ipsilateral half of the mandible proportional to the condylar growth (Fig. 9.16). The onset of the enlargement can occur as early as the age of 9 and continue to grow beyond normal somatic growth and up to the age of 30. The condyle has normal contours and subchondral bone. The shape changes can remodel the fossa. The antegonial notch is flattened or bowed inferiorly. The lateral border of the ramus is laterally bowed. There is an occlusal cant with the affected side being more depressed with or without ipsilateral open bite.

9.7.3.4 Hemimandibular Elongation

A condition where elongation of the condyle results in horizontal elongation of the mandible without vertical elongation of the ramus (Fig. 9.17). This results in a mandibular and dental midline shift to the unaffected side and a Class III molar occlusion on the affected side. The shape of the condyles is almost symmetrical. There is often a contralateral and anterior cross bite. The affected condyle stops growth at the completion of somatic growth.

9.7.4 Tumors

9.7.4.1 Benign Tumors

The most common of benign tumors are the osteochondromas and the osteomas. *Osteochondromas* are cartilaginous-capped high-density exophytic lesions arising from bone which can arise from the condyle and the coronoid process in the jaws. The size can vary from a small exophytic lesion simulating an osteophyte or can grow very large (Fig. 9.18). If the lesion becomes large, it can displace the

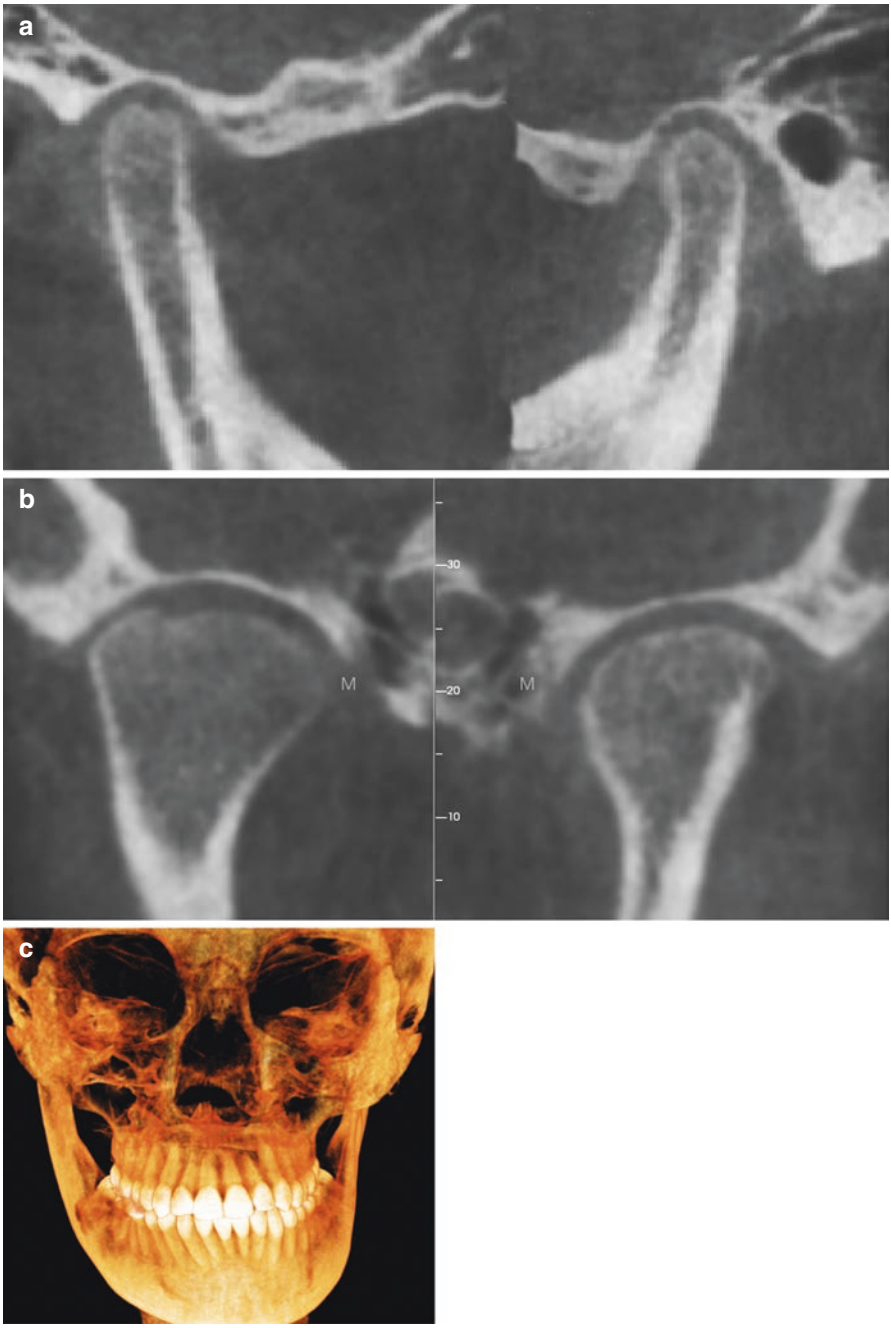

Fig. 9.16 CBCT reformations in a patient with condylar hyperplasia show increase in size of the right condyle with preservation of the position of the condylar contours. The condylar neck is elongated (**a** and **b**), and the mandible on the right side is larger with deviation of the mandible to the unaffected side (**c**) (Courtesy, S. Brooks, DDS)

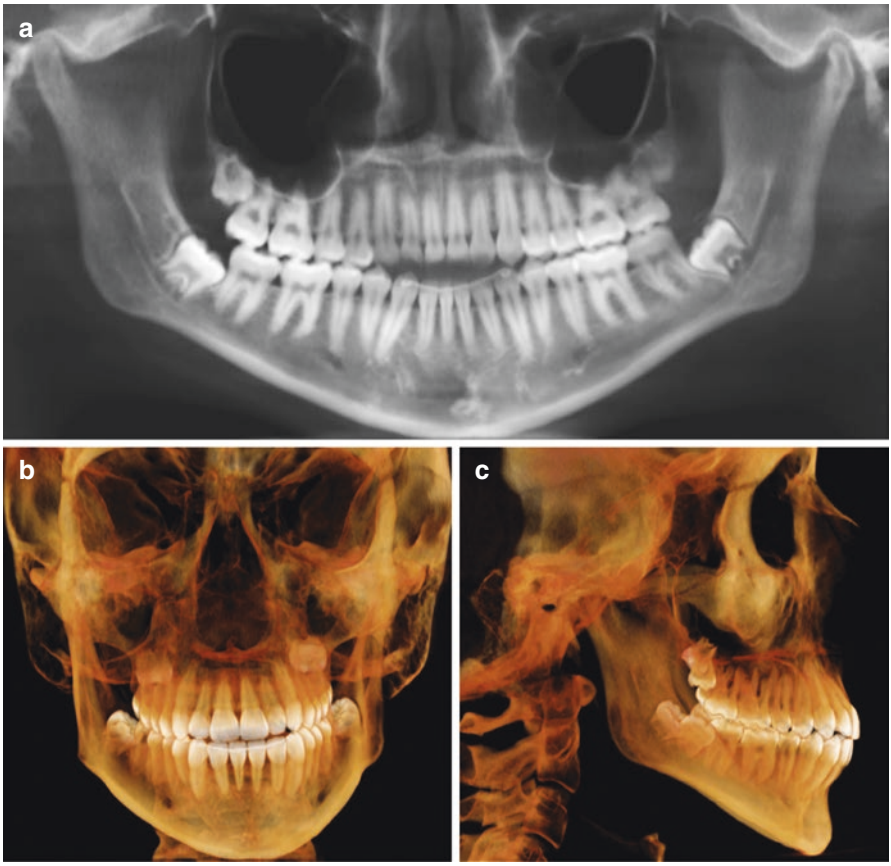

Fig. 9.17 CBCT reformats show elongation of the right condyle without vertical lengthening of the ramus (**c**), right Class III molar relationship (**a**), and deviation of the mandibular midline to the left (**b**), suggestive of hemimandibular elongation

condyles inferiorly and anteriorly and also deviate the mandible to the unaffected side and change the occlusion (unilateral posterior open bite or contralateral cross bite). As this is not a developmental entity and as it occurs in adults, there is no ipsilateral mandibular enlargement seen as with condylar hyperplasia. Secondary degenerative changes may occur, making the differentiation from an osteophyte more difficult. If the aforementioned mandibular and occlusion changes occur, then it is most likely a tumor as an osteophyte will not displace the mandible. *Osteomas* are benign, slow-growing bone-forming tumors that are characterized by proliferation of either compact or cancellous bone (Fig. 9.19). In the TMJ, they are rare but tend to occur in areas covered by the periosteum (condylar neck,

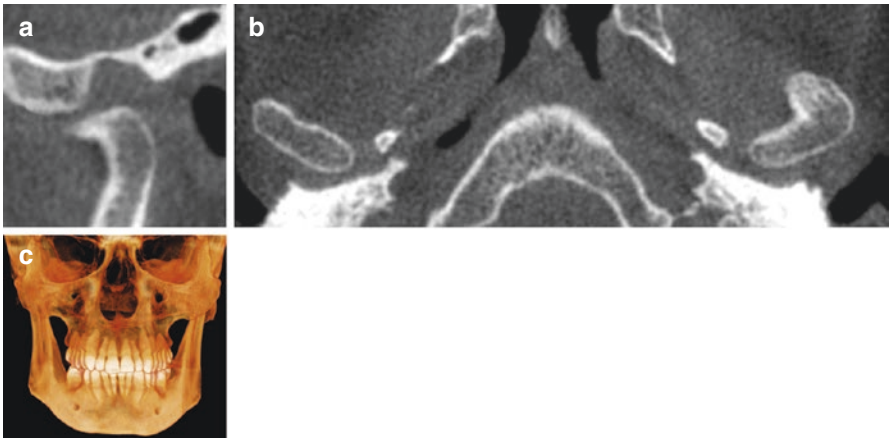

Fig. 9.18 CBCT reformations show an osteochondroma presenting as an osteophyte-like projection on the anterior surface of the condyle that is directed along the muscle fibers of the lateral pterygoid (**a** and **b**). The condyle is inferiorly displaced, and this has resulted in mandibular displacement and midline shift to the right (**c**) (Courtesy, S. Brooks, DDS)

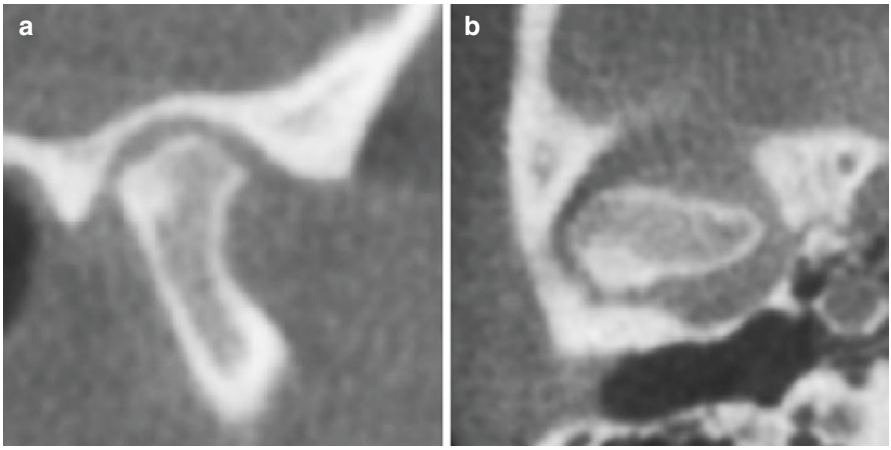

Fig. 9.19 CBCT cross sections show a high-density exophytic bony lesion on the posterior surface of the condyle (**a** and **b**), suggestive of an osteoma. This currently has not caused anterior displacement of the condyle but could over time

posterior aspect of the condyle, and the mandibular ramus). It presents as a well-defined pedunculated or sessile high-density mass similar in density to compact or cancellous bone. These are slow growing and can displace the condyle and subsequently the mandible.

9.7.4.2 Malignant Tumors

Malignant tumors of the TMJ are very rare. When evaluating these, it is important to note that CBCT should not be the only modality used due to the soft tissue extent of these lesions, possible nodal metastases, distant metastases, and perineural spread of malignancy. On CBCT imaging, the affected osseous tissues should be evaluated for characteristics of these lesions, such as osseous extent, other areas of demineralization, calcifications, periosteal reactions, and widening of the skull base foramina of the nerves innervating the affecting structures. These findings should be followed up by either MDCT with contrast, MRI with contrast, or PET-CT, as indicated.

9.7.4.3 Chondrosarcoma

This is a rare malignant lesion that can occur centrally in the bone and parosteally or peripherally in the soft tissue. The radiographic pattern is a ringlet pattern of cloud-like calcification in and around the condyle with or without speculated periosteal reaction (Fig. 9.20a). The condyle may appear enlarged. Destruction of the condyle and fossa may or may not occur, but there is widening of the joint space, and intracranial extension can occur (Fig. 9.20b). This is often difficult to distinguish from calcium pyrophosphate dihydrate deposition disease. *Osteosarcoma* is a malignant tumor of the bone in which osteoid is produced by the malignant stroma. There is often bone destruction with aggressive periosteal reaction ("hair-on-end" or "sunray spicules") perpendicular to the bone surface with or without tumor formation. *Metastases* to the TMJ are very rare, but when it occurs, it has the characteristics of a malignancy destructive lesion that effaces the bony architecture of the TMJ. Of note is the similarity of active erosions in the active phase of degenerative joint disease, but these erosions occur on the articular surface, whereas those seen with metastases are not limited to the articular surface.

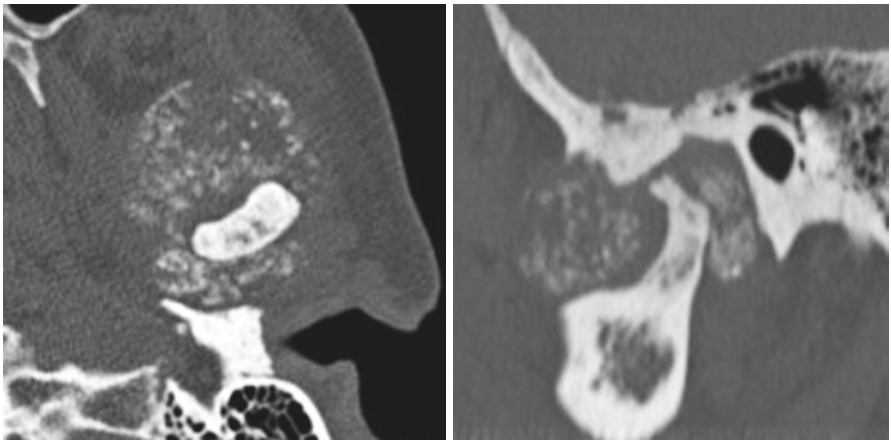

Fig. 9.20 Axial and coronal CTs of an 84-year-old female with grade 2 chondrosarcoma show cloud-like calcification surrounding the condyle with minimum destruction of the condyle and fossa and erosion of the articular eminence (Courtesy, Christine Glastonbury)

9.7.5 Tumor-Like Lesions

9.7.5.1 Synovial Chondromatosis

This is a nonneoplastic process characterized by the development of cartilaginous nodules within the sub-synovial connective tissue that subsequently detach, may ossify, and form loose articular bodies in the joint space. These articular bodies can be calcified or not, and when they are not calcified, they are not visible on CBCT. The calcified loose articular bodies are easily identifiable, and the diagnosis is relatively easier than the non-calcified variety. Condylar displacement and remodeling in a hydraulic manner should alert the observer to the presence of a space-occupying mass, and the differential diagnosis should include synovial chondromatosis. There is another type of synovial chondromatosis that is secondary to degenerative changes which results in the production of loose articular bodies without the presence of a hydraulic mass effect. The joint space will not be widened on CBCT in this type of synovial chondromatosis and will most likely be narrowed (Figs. 9.21 and 9.22).

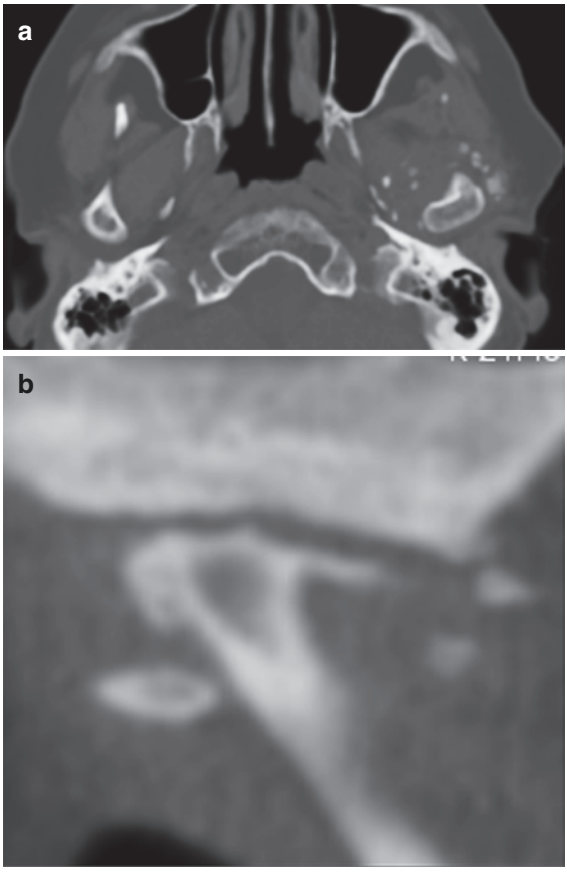

Fig. 9.21 CBCT cross sections show two different types of synovial chondromatosis. (**a**) Shows primary synovial chondromatosis, with multiple calcified loose articular bodies. (**b**) Shows synovial chondromatosis secondary to degenerative joint disease

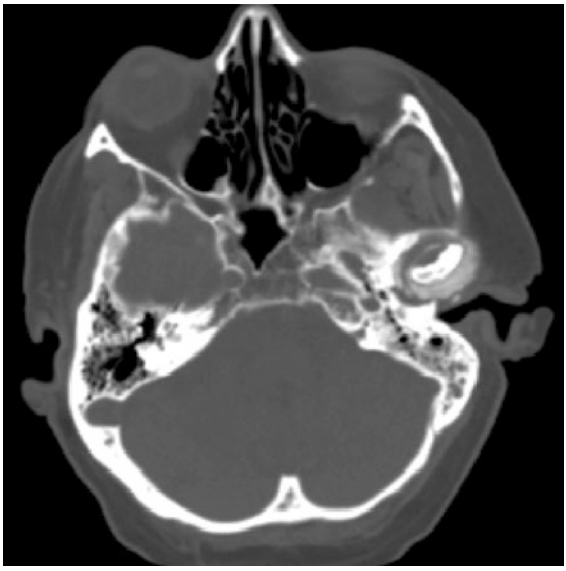

Fig. 9.22 CT axial view shows a cloud-like calcification in the joint space surrounding the left condyle. This is one of the radiographic features of CPPD deposition disease, but chondrosarcoma can mimic this appearance and thus should be ruled out

9.7.5.2 Calcium Pyrophosphate Dihydrate (CPPD) Deposition Disease (Aka Pseudogout)

This is a metabolic disease where calcium pyrophosphate crystals deposited in the synovial fluid result in calcification of articular cartilage, leading to acute arthritis in some patients. It presents as a calcified TMJ mass that has fine, cloud-like synovial calcifications with even distribution in the joint space in the early phase, but as the disease progresses, the calcifications become a chunky and diffuse calcified mass that may have a ground-glass appearance. There is associated remodeling, erosion, or mass effect on the osseous components of the TMJ which may mimic malignancy due to extensive bone destruction [23].

9.8 Summary

CBCT evaluation of the TMJs requires a comprehensive knowledge of TMJ anatomy, biomechanics, and function. The effect of the TMJ on the structures of the oral and maxillofacial complex can be studied with specialized reformats of CBCT data, such as the axially corrected sagittal and coronal oblique cross sections, panoramic reformation, and 3D reformations. The closed-mouth scan must be with the teeth in maximum intercuspation and should involve the entire oral and maxillofacial complex to enable the evaluation of the skeletal clues of TMJ disorders and aid in their diagnosis.

References

1. Quintero JC, Trosien A, Hatcher D, Kapila S. Craniofacial imaging in orthodontics: historical perspective, current status, and future developments. Angle Orthod. 1999;69:491–506.
2. Okeson J. Management of temporomandibular disorders and occlusion. St. Louis: Mosby-Year Book, Inc.; 1993.
3. Dixon DC. Radiographic diagnosis of temporomandibular disorders. Semin Orthod. 1995;1:207–21.
4. Pharoah MJ, Petrokowski CG. Imaging temporomandibular joint disorders. Oral Maxillofac Surg Clin. 2001;13:623–38.
5. Brooks SL, Brand JW, Gibbs SJ, Hollender L, Lurie AG, Omnell KA, et al. Imaging of the temporomandibular joint: a position paper of the American Academy of Oral and Maxillofacial Radiology. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997;83:609–18.
6. Eckerdal O, Lundberg M. The structural situation in temporomandibular joints. A comparison between conventional oblique transcranial radiographs, tomograms and histologic sections. Dentomaxillofac Radiol. 1979;8:42–9.
7. Lindvall AM, Helkimo E, Hollender L, Carlsson GE. Radiographic examination of the temporomandibular joint. A comparison between radiographic findings and gross and microscopic morphologic observations. Dentomaxillofac Radiol. 1976;5:24–32.
8. Bean LR, Omnell KA, Oberg T. Comparison between radiologic observations and macroscopic tissue changes in temporomandibular joints. Dentomaxillofac Radiol. 1977;6:90–106.
9. Sukovic P. Cone beam computed tomography in craniofacial imaging. Orthod Craniofac Res. 2003;6(Suppl 1):31–6; discussion 179–82.
10. Hilgers ML, Scarfe WC, Scheetz JP, Farman AG. Accuracy of linear temporomandibular joint measurements with cone beam computed tomography and digital cephalometric radiography. Am J Orthod Dentofac Orthop. 2005;128:803–11.
11. Mah JK, Danforth RA, Bumann A, Hatcher D. Radiation absorbed in maxillofacial imaging with a new dental computed tomography device. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003;96:508–13.
12. Honda K, Larheim TA, Maruhashi K, Matsumoto K, Iwai K. Osseous abnormalities of the mandibular condyle: diagnostic reliability of cone beam computed tomography compared with helical computed tomography based on an autopsy material. Dentomaxillofac Radiol. 2006;35:152–7.
13. Honda K, Matumoto K, Kashima M, Takano Y, Kawashima S, Arai Y. Single air contrast arthrography for temporomandibular joint disorder using limited cone beam computed tomography for dental use. Dentomaxillofac Radiol. 2004;33:271–3.
14. Ericson S, Kurol J. Incisor resorption caused by maxillary cuspids. A radiographic study. Angle Orthod. 1987;57:332–46.
15. Lascala CA, Panella J, Marques MM. Analysis of the accuracy of linear measurements obtained by cone beam computed tomography (CBCT-NewTom). Dentomaxillofac Radiol. 2004;33:291–4.
16. Marmulla R, Wortche R, Muhling J, Hassfeld S. Geometric accuracy of the NewTom 9000 Cone Beam CT. Dentomaxillofac Radiol. 2005;34:28–31.
17. Broadbent B Jr. A new X-ray technique and its application to orthodontia. Angle Orthod. 1931;1:2–24.
18. Hatcher DC, Aboudara CL. Diagnosis goes digital. Am J Orthod Dentofac Orthop. 2004;125:512–5.
19. Kau CH, Richmond S, Palomo JM, Hans MG. Three dimensional cone beam computerized tomography in orthodontics. J Orthod. 2005;32:282–93.
20. Tsiklakis K, Syriopoulos K, Stamatakis HC. Radiographic examination of the temporomandibular joint using cone beam computed tomography. Dentomaxillofac Radiol. 2004;33:196–201.

21. White S, Pharoah M. Oral radiology principles and interpretation. St. Louis: Mosby; 2000.
22. Petrokowski CG. Disorders of the temporomandibular joint. In: Oral radiology-principles and interpretation, white and pharoah. Mosby; 2004. pp. 538–75.
23. Tamimi D, Hatcher D. Specialty imaging: temporomandibular joint. Philadelphia: Elsevier; 2016.

Imaging Internal Derangement: State-of-the-Art MR and Prospects for Ultrasonography

10

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Abstract

Diagnostic imaging of the TMJ has evolved rapidly since the early days of plain film radiography. This chapter is devoted to soft tissue imaging techniques that do not involve ionizing radiation, MR, and ultrasound.

10.1 Background

Diagnostic imaging of the TMJ has evolved rapidly since the early days of plain film radiography. This chapter is devoted to soft tissue imaging techniques that do not involve ionizing radiation, MR, and ultrasound.

The first systematic approach to soft tissue imaging was arthrography, developed in the 1970s. This technique involved fluoroscopically guided percutaneous injection of an opaque, water-soluble contrast medium into the lower or lower and upper joint spaces for an indirect depiction of the disc position, shape, and motion with opening and closing jaw maneuvers (Fig. 10.1a–c). Diagnostic characteristics of disc displacement were correlated with clinical signs and symptoms of joint pathology. Groundbreaking clinical insights of disc displacement corroborated by arthrography and surgical observations led to systematic concepts of

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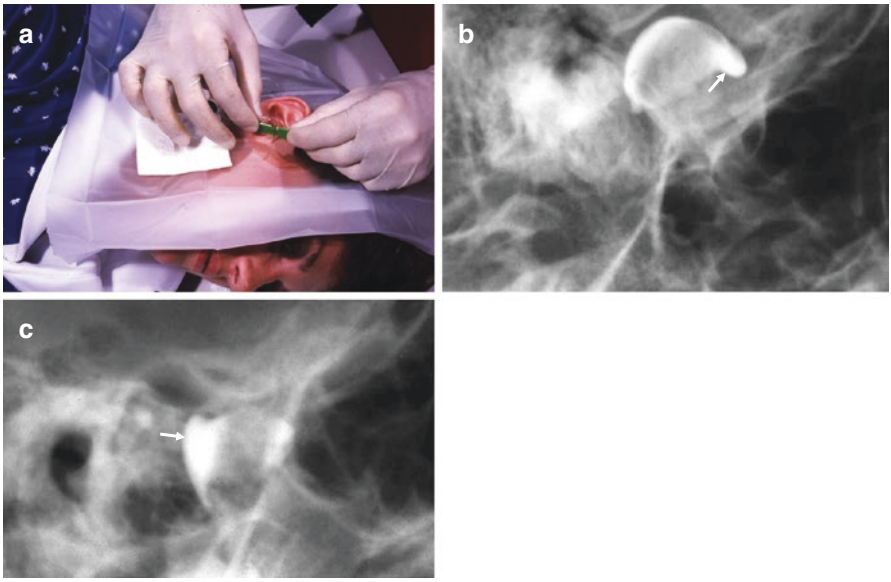

Fig. 10.1 (**a**) Joint puncture for contrast injection into the lower joint space, using a 23-gauge butterfly needle. The direction of the needle is guided by fluoroscopic guidance of the location of the osseous anatomy versus the needle direction. The needle is actually advanced into the joint space by feel, rather than direct observation. (**b**) Closed jaw, transcranial spot radiograph with contrast in the normal lower joint space. The anterior recess is well shown (arrow). (**c**) Opened jaw, normal lower joint space arthrogram. The disc position and function are identified indirectly by the configuration and movement of the contrast material into the posterior recess (arrow) on the open-mouth image

TMJ internal derangement, evolved through the pioneering works of Farrar [1, 2] and Wilkes [3, 4].

CT imaging appeared on the scene in 1982, demonstrating the capability to depict the disc without the need for the invasive injection of contrast material using needles and infusion catheters. CT employs a computer to map and construct cross-sectional images of the body from x-ray transmission through thin slices of patient tissue [5]. The x-ray beam is attenuated by absorption and scatter as it passes through the patient but exposes the patient to potential risks from ionizing radiation. Cone beam CT technology has rapidly developed for dental imaging as shown in Chap. 9 but cannot depict soft tissues such as the TMJ disc.

In the early 1980s, the remarkable technology of MR imaging was developed, and the first surface coil images of the TMJ were reported in 1985. MR is a technique that produces tomographic images by means of magnetic fields and radio waves [5]. The complicated physics is beyond the scope of this chapter; however, in its simplest terms, MR is based on the ability of protons in the body to absorb and emit radio waves when the body is placed in a strong magnetic field, most commonly at 1.5 and 3.0 T. These magnetic field strengths are 30,000–60,000 times that of the Earth's magnetic field strength.

The advantages of MR are exquisite soft tissue contrast resolution, ability to provide images in any anatomic plane, and absence of ionizing radiation. However, MR is limited by inability to demonstrate dense bone detail or calcifications, long imaging times, limited accessibility, and high expense. Because of the confining internal imaging space for the patient, a number of patients experience symptoms of claustrophobia and require sedation or are unable to tolerate the exam altogether. Even though there are contraindications such as pacemakers and aneurysm clips, there is no biologic risk inherent in MR image generation.

Ultrasound is a multipurpose medical imaging technique that also does not employ ionizing radiation that can also produce cross-sectional images of the body [5]. The ultrasound transducer transmits a brief pulse of high-frequency sound energy that is transmitted into the patient tissues. The ultrasound transducer then becomes a receiver, detecting echoes of sound energy reflected from tissue. Ultrasound units operate sufficiently rapidly to produce real-time images of moving patient tissue that can, for example, assess cardiac movement, vascular pulsations, bowel peristalsis, and the moving fetus. The ultrasound transducer is placed directly onto the patient's skin or mucous membranes using a water-soluble gel as a coupling agent to assure adequate contact. Anatomic images can be produced in an infinite variety of anatomic planes by adjusting the angulation of the transducer. However, visualization of anatomic structures is severely limited by bone or gas containing structures such as the lung or bowel. Ultrasound is now commonly used as a dynamic extension of the physical examination such as in sports medicine, urgent care, cardiology, and obstetrics and gynecology. There are no biological risks for this ubiquitous imaging technology.

10.2 MR TMJ Protocol Strategy

Successful imaging of the TMJ is best performed with high field strength magnets at 1.5 or 3.0 T and using high-resolution surface coils (Fig. 10.2a, b). The fundamental characteristics of the MR image acquisition are (1) the anatomic plane of the image, (2) the slice thickness of each anatomic plane, (3) the matrix (pixel) size, (4) the number of signal averages, (5) the pulse sequence, and (6) the pulse time intervals [6]. Table 10.1 demonstrates the parameters that are specified to perform an MR scan. For imaging planes, the axial plane is used for localizing the condyle and programming the imaging angles for the sagittal and coronal planes. The sagittal plane is standard, and images are obtained perpendicular to the long axis of the condyle and in closed- and open-mouth positions. Coronal images are parallel to the horizontal long axis of the condyle. Coronal images are useful for depicting medial and lateral disc displacements.

Slice thicknesses of 3 mm are recommended. A typical matrix size is 192 × 256. The finer the matrix size, the better the resolution but with the sacrifice of longer imaging times. The number of excitations (NEX) are typically 0.5 or 1.0. As for the matrix size, more averages improve image quality, but with the sacrifice of longer imaging times and the risk of patient motion. Pulse sequences frequently employed for TMJ imaging include T1-weighted or proton density acquired using a spin echo sequence and T2-weighted images for joint effusion and bone marrow edema.

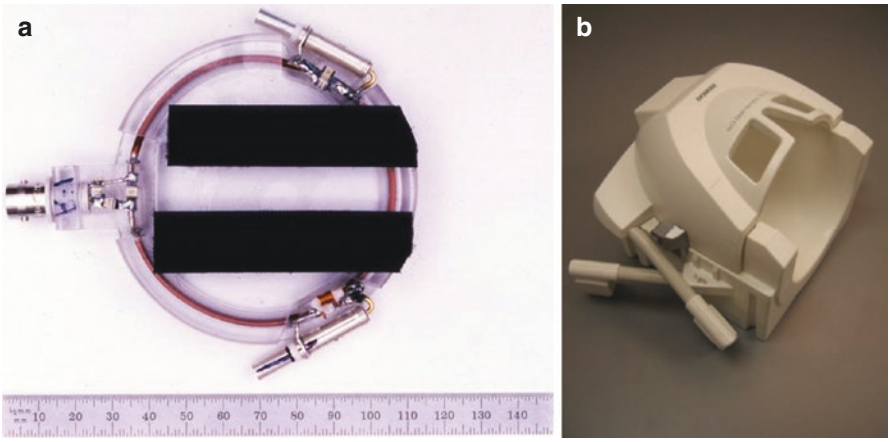

Fig. 10.2 (**a**) First-generation prototype 6.5 cm surface coil embedded in Plexiglas. This is the first TMJ surface coil developed by General Electric for a 1.5 T MR system in the early 1980s. The copper loop serves as an antenna to increase signal-to-noise of the small TMJ when placed in front of the ear. This surface coil had to be hand-tuned with an oscilloscope prior to each imaging session. (**b**) State-of-the-art Siemens MR 32-channel head coil used for a 3 T magnet and bilateral TMJ imaging

Table 10.1 TMJ anatomy and MR parameters

TR	TE	Effect	Terminology	Results
Short	Short	Highlights fat	T1-weighting	Lateral pterygoid fat pad accentuated Posterior disc ligament accentuated Disc signal low Marrow signal of condyle accentuated Excellent anatomic detail
Long	Short	Highlights fat	Proton density	Similar effects as T1-weighting
Long	Long	Highlights water	T2-weighting	Joint effusion accentuated Bone marrow edema accentuated Signal of fat pad, posterior ligament, and marrow decreased

Table 10.2 shows the relationships between the signal intensity of TMJ anatomy and MR parameters. A short TR and a short TE represent a T1-weighted parameter which highlights fat, especially the lateral pterygoid fat pad, the posterior disc attachment, and the marrow signal of a normal condyle. A proton density pulse sequence has both a long TR and a short TE. The effects are similar to a T1-weighted image. T2-weighting has a long TR and a long TE, and this highlights water as well as joint effusion and bone marrow edema. The grayscales of body tissues seen on the typical T1- or proton-weighted spin echo sequence are as follows: fat is white, muscle is gray, the TMJ disc is gray to black, and cortical bone and air are black. The advantage of a shorter imaging time, especially when the jaw is open, is to minimize patient's discomfort and minimize collection of saliva in the posterior pharynx which can lead to motion degradation.

Table 10.2 Parameters specified to perform an MR scan

Parameters	Typical options	Comments
Imaging plane	Axial	Axial plane is used for localizing the condyle and programming the imaging angles for the sagittal and coronal scans
	Sagittal	Sagittal is the standard plane of imaging, and images are obtained at closed- and open-mouth positions. Oblique images perpendicular to the horizontal long axis of the condyle are preferable to straight sagittal images
	Coronal	Coronal images should be parallel to the horizontal long axis of the condyle. Coronal images are important for studying medial and lateral disc displacement. Coronal images are usually obtained only at the closed-mouth position
Slice thickness	3 mm	Slices as thin as possible are desirable, because these provide better anatomic detail. The disadvantage of thin slices is a lower signal-to-noise ratio. This is a proven optional slice thickness. Thinner slices with lower signal-to-noise end with no diagnostic advantage
Matrix size	192 × 256	The finer the matrix, the better the resolution, but the longer imaging time
Number of averages (NEX)	0.5, 0.075, 01.0, 2.0	More averages improve image quality but require longer scanning time and increase risk for motion artifacts. Between 0.5 and 1 is usually employed for the TMJ
Pulse sequence	Spin echo	Spin echo is most frequently used for TMJ imaging
Pulse time intervals (in ms)		TR = 600–2000 TE = 10–200

A negative aspect of the advent of MR imaging is the lack of joint motion studies with this technology. However, there are pseudo-motion capabilities and investigators are developing true kinematic techniques to reestablish this imaging capability.

10.3 Normal Disc Position and Function

The TM joint is compartmentalized by the interposed disc into upper and lower joint compartments, which normally do not communicate [6]. The disc is a flexible but firm plate of dense collagenous connective tissue that merges around its periphery with the surrounding capsule. The central part is typically considerably thinner than its periphery, and its posterior band is thicker (Fig. 10.3). The under aspect of the disc is concave and sits on top of the rounded condyle. The anterior and posterior bands of the disc are leading edges. The undersurface of the disc and the top of the condyle fit well together and move smoothly with opening and closing jaw function (Fig. 10.4).

The peripheral attachment to the capsule binds the disc firmly to the lateral and medial poles of the condyle. Anteriorly there is no direct connection between the disc and the mandibular condyle. Thus, the disc can rotate relatively freely over the condyle in an anteroposterior direction, but it can move relatively little in the

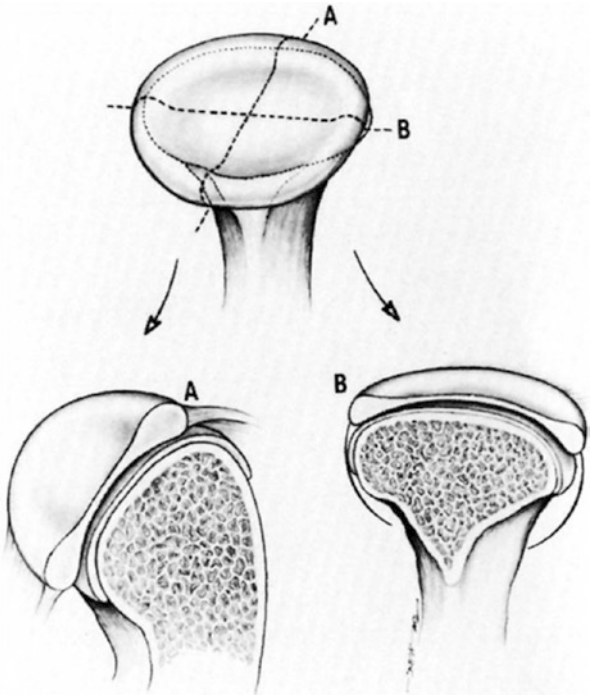

Fig. 10.3 Sagittal (**a**) and coronal (**b**) diagrams of the normal disc and condyle. In the sagittal plane, the posterior band of the disc is at the 12 o'clock position relative to the condylar head. In the coronal plane of imaging, the lateral and medial margins of the disc are adjacent to the lateral and medial poles of the condyle

medial-lateral direction unless the attachments to the capsule and condyle have been torn or elongated. Anterior movement of the disc is limited by the length of the undersurface of the posterior disc attachment. This extends from the posterior band of the disc down to the back of the condyle and prevents the disc from moving anteriorly over the condyle. This surface consists of fibrous tissue. If it is damaged, the disc can prolapse anteriorly relative to the condyle, resulting in disc displacement.

In the sagittal plane, the disc is biconcave; that is, it is thickest anteriorly and posteriorly (Fig. 10.4). The thickness of the disc is 1 mm in its central part, 3 mm posteriorly, and 2 mm anteriorly. There is great variation in the configuration of the disc; generally its shape is well adapted to the shape of the condyle and the temporal component.

The normal position of the disc has generally been described as a 12 o'clock relationship between the superior aspect of the condyle and posterior band of the disc. However, there is some variability in this relationship. Alternative landmarks for the determination of the normal disc position have been proposed. When the posterior band is not in exactly the 12 o'clock position, the next aspect is to examine the relationship between the anterior prominence of the condyle and the central thin zone of the disc itself. This can be used as an additional criterion for normal disc position. In the example shown in Fig. 10.5, the posterior band of the disc is anterior to the 12 o'clock position, while the anterior prominence of the condyle opposes the central thin part of the disc. This is also considered a normal disc position since the function in these joints is smooth without friction or evidence of internal arrangement.

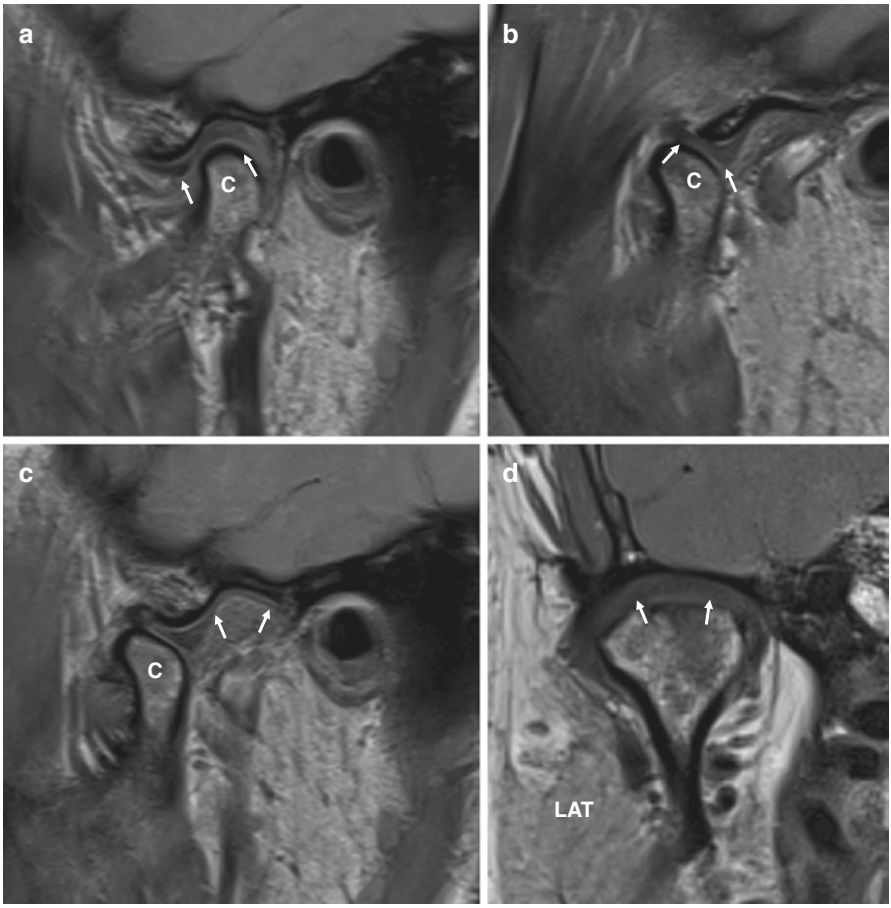

Fig. 10.4 Images of a normal asymptomatic volunteer acquired with the 32-channel head coil shown in Fig. 10.2b at 3 T. (**a**) Sagittal MR image with the jaw closed. The biconcave, intermediate signal intensity disc (arrows) is situated in the normal relationship superior or 12 o'clock position relative to the condylar head and being between the anterior convexity of the condyle (C) inferiorly and the posterior convexity of the temporal bone. (**b**) Sagittal MR image with the jaw maximally opened shows the biconcave lenslike ("Bowtie") of the disc (arrows) in the normal position between the convex surface of the condyle (c) interiorly and the convex surface of the tubercle of the temporal bone superiorly. The condyle shows normal translation just beyond the tubercle. (**c**) The normal temporal posterior attachment (arrows) is well depicted in the opened jaw position. The posterior attachment expands on opening. (**d**) Coronal MR image of the right TMJ of the same subject with the jaw in the closed position. The disc (arrows) is in a normal position and has an arc-like configuration

Additional criteria of normal disc position have been reported [7]. Drace and Enzmann [8] described a junction of the bilaminar zone and posterior band of the disc to be within 10% of the vertical of the central condyle and to be within 95 percentile of the normal. Katzberg and Westesson [6] have defined a posterior band of the disc to be superior or slightly anterior to the thin zone to articulate with the anterior surface of the condyle and posterior surface of the eminence and

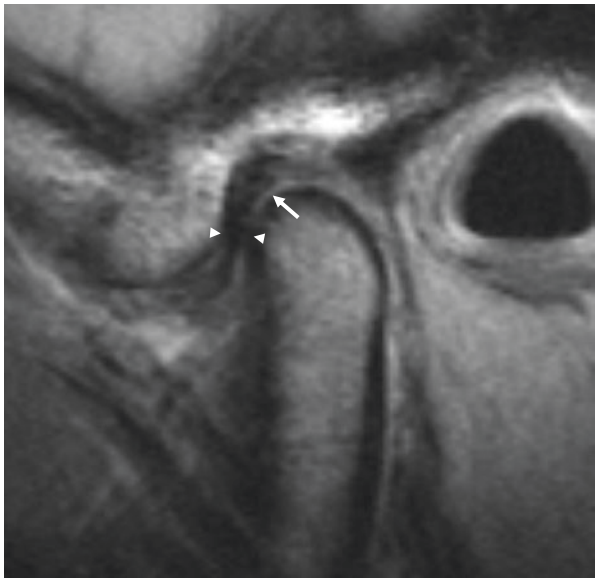

Fig. 10.5 Normal disc position and configuration with the posterior band (arrow) being anterior to the 12 o'clock position relative to the condyle, but the relationship between the central thin zone to the anterior prominence of the condyle (arrowhead) and the posterior prominence of the articular tubercle (arrowhead) is normal. These and all subsequent MR images were acquired at 1.5 T field strength and are derived from the VA UCSF patient archive

being associated with the functional aspects of disc and condyle motion and a smooth motion and without hindrance. Ahmad et al. [9] as part of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) defined the borders of the disc in the posterior attachment to be located between the 11:30 and 12:00 o'clock positions and the intermediate zone (thin zone) to be located between the anterior-superior aspect of the condyle and posterior-inferior aspect of the eminence.

In MR images, the normal TMJ demonstrated by MR imaging in the sagittal and coronal closed-jaw positions is depicted in Fig. 10.4a–d. The low signal intensity of the fibrous disc is clearly demonstrated because of the relatively bright signal intensity emanating from the surrounding soft tissues and the lateral pterygoid fat pad. The cortex of the condylar head has an absence of signal but is well depicted because of the relatively bright signal intensity of the contiguous fibrocartilage and synovial tissues superiorly and the bright signal of the fatty bone marrow inferiorly. The disc has a "bow tie"-like configuration with maximal jaw opening (Fig. 10.4b) and maintains its position interposed between the convexity of the condyle inferiorly and the convexity of the tubercle superiorly. The posterior disc attachment has a bright signal owing to the rich network of fatty tissue (Fig. 10.4b). This contrasts extremely well with the low signal intensity of the fibrous disc. The insertion of the superior belly of the lateral pterygoid muscles often demonstrated on MR imaging is a low intensity threadlike structure attaching to the anteromedial aspect of the disc and condyle. In the coronal plane, the disc has an arc-shaped configuration with the medial margin of the disc attaching just inferior to the medial pole and to the neck of the condyle (Fig. 10.4d). The lateral margin is attached just underneath the lateral pole and to the lateral capsular wall.

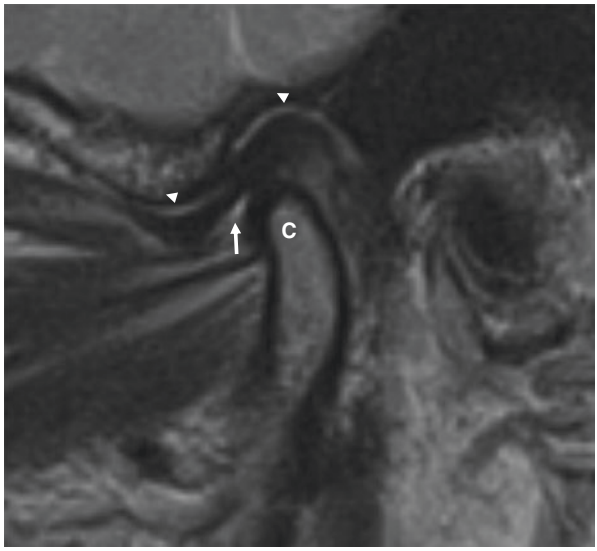

Fig. 10.6 Normal T2-sagittal image showing a small amount of synovial fluid in both the lower (arrow) and upper (arrowheads) joint spaces creating an arthrogram effect. Note the much larger articular surface of the temporal compartment (joint space) in comparison with the lower joint surface. Compare with the arthrogram in Fig. 10.1b

The intra-articular osseous parts of the mandibular condyle and the temporal component are covered by a thin layer of dense collagenous connective tissue that can become cartilaginous. The thickness of the articular soft tissue cover has been measured in postmortem studies of normal and pathologic joints and varies between 0.1 and 1 mm. The total area of the articulating surface of the temporal compartment of the joint is two to three times greater than that of the mandibular condyle. This difference is to a large extent a function of the larger anterior recess of the upper joint compartment, which extends about 8–10 mm anterior to the apex of the articular tubercle (Fig. 10.6). The large articular surface on the temporal component is necessary to accommodate the condyle when it translates anteriorly on jaw opening. In the lower joint space, there is less translation and more rotation. The extensive articular surface in the lower joint space is on the back of the condyle. This surface is there to provide an articulating surface for the disc when the condyle rotates forward, as the posterior band of disc articulates against the posterior aspect of the condyle. The TMJ capsule originates from the periphery of the articulating surface of the temporal bone. It extends inferiorly in the shape of a funnel, encloses the disc and condyle, and attaches to the lower part of the condyle and the upper part of the condylar neck. Medially and laterally the capsule is firm to stabilize the mandible during movement.

The superior belly of the lateral pterygoid muscle rises from the infratemporal crest of the greater wing of the sphenoid bone and passes through the anteromedial wall of the TMJ capsule. The anatomy of the lateral pterygoid muscle is variable in humans. A study of human autopsy material showed that 65% of specimens have two heads to this muscle, while 20% actually have three heads, and 15% have only a single head. The same study showed that in about 30% of

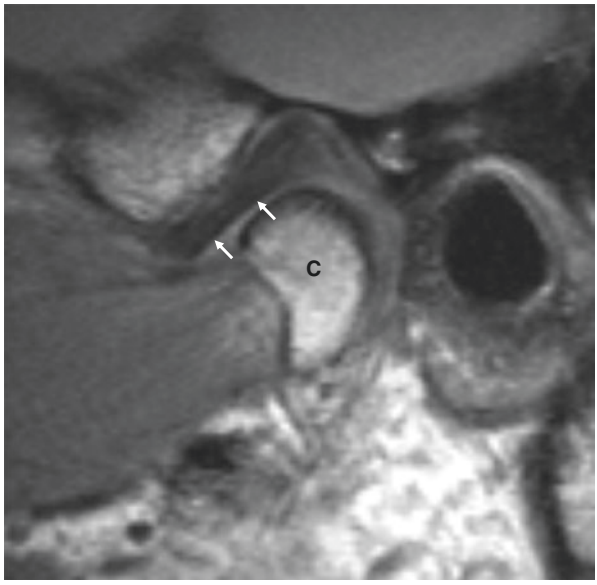

Fig. 10.7 This is a normal variation in disc configuration which is thick and biplanar (arrows). Normal disc position in this closed-jaw sagittal image. *C* condyle

specimens, the superior head of the lateral pterygoid muscle inserts into the disc. From a review of the literature and examination of cadaver material, it is assumed that the attachment of the lateral pterygoid muscle is not only to the condyle and not only to the disc but to both in some joints and to only one in other joints [6].

The TMJ is supplied by the superficial temporal and maxillary arteries, which arise from the external carotid artery [7]. The blood vessels surround the joint in a network of fine branches. Venous drainage is via the superficial temporal, the maxillary, and the pterygoid plexus of veins. The joint capsule and attachments of the disc originally are vascularized during growth but are not vascularized in the adult. As the disc continues posteriorly, it merges into the tissue of the posterior disc attachment which has also been called the bilaminar zone or retrodiscal pad. This tissue is composed of a loosely organized network of collagen fibers intermixed with a branching system of large elastic fibers, fat, and numerous blood vessels and nerves. In contrast to the disc, which is firm, the posterior disc attachment is readily deformable, and when the mouth is open widely, it becomes substantially expanded (Fig. 10.4a, b). The posterior attachment has been divided into three parts: the temporal PA (TPA) (also called the superior lamina), the intermediate part of the PA (IPA), and the condylar PA (CPA) (also called the inferior lamina). The TPA of the posterior disc attachment is more frequently clearly delineated in the normal joint and less consistent in joints with chronic disc displacement. However, there is no proven specificity in applying this for diagnostic grading.

Variations in normal disc thickness and configuration are shown in Figs. 10.7 and 10.8.

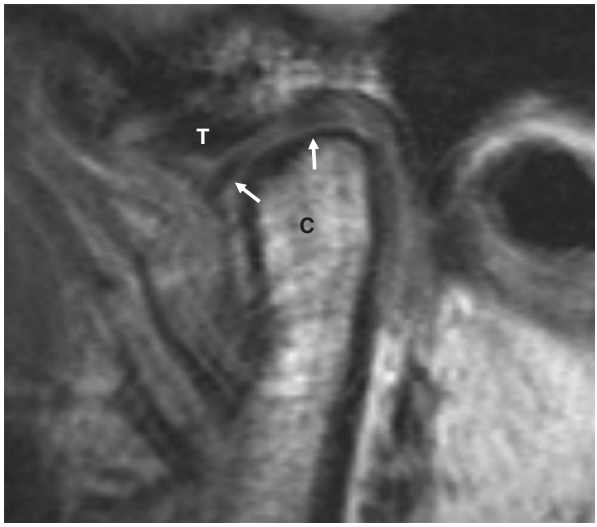

Fig. 10.8 This is another normal variation in disc configuration which is markedly thinner than in Fig. 10.7. The disc is biconcave with small anterior and posterior bands (arrows). There is flattening of both the condyle (C) and tubercle (T) indicating remodeling of both. The disc is in a normal position

10.4 Abnormal Disc Position and Function

TMJ disorders are defined as intra-articular morphologic abnormalities, such as different forms of disc displacement, degenerative joint disease (arthrosis), inflammatory arthritis, cellulitis, and congenital and neoplastic abnormalities. When studying patients with signs and symptoms of TMJ disorders, the most common findings are different forms of disc displacement causing internal derangement and degenerative joint disease. Internal derangement is defined as an abnormal positional and functional relationship between the disc, the mandibular condyle, and the articulating surfaces of the temporal bone. Arthrosis is a deterioration of the articular soft tissue cover of the joint components with exposure of bone. Remodeling represents an alteration in the form of the articular joint compartments with an intact articular soft tissue cover. Disc displacement is an abnormal position of the disc relative to the condyle and separation of the anterior joint surface of the condyle and the inferior articular surface of the disc in the region of the central thin zone (Fig. 10.9) [6].

Disc displacement with reduction is the condition whereby the disc is displaced in the closed-mouth position but assumes its normal position between the condyle and the tubercle on opening. This is frequently associated with clicking. During closing of the jaw, the disc again becomes anteriorly displaced which is associated with a softer click. This type of repetitive clicking has been termed as reciprocal clicking, stressing the interdependence of the closing click and the opening click [1, 2].

Disc displacement without reduction occurs when the disc becomes displaced and does not reduce. This is generally considered a more advanced phase of internal derangement and is associated with restricted jaw opening and deviation of the mandible toward the affected side.

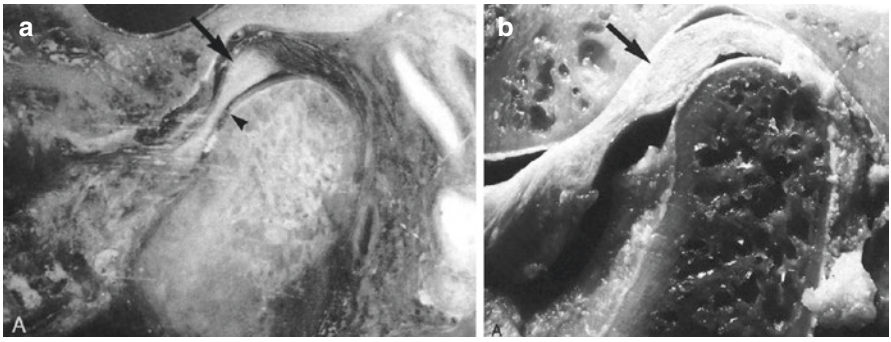

Fig. 10.9 Normal versus anterior disc displacement in cadaver specimens. (**a**) Even though the posterior band of the disc is located anterior to the 12 o'clock position (arrow), the central thin zone (arrowhead) is in a normal relationship between the anterior prominence of the condyle (arrowhead). The position of the disc is normal. (**b**) This sagittal cadaver section shows early anterior disc displacement. As in (**a**), the posterior band of the disc (arrow) is anterior to the 12 o'clock position of the condyle. However, the thin zone of the disc is below and anterior to the anterior prominence of the condyle

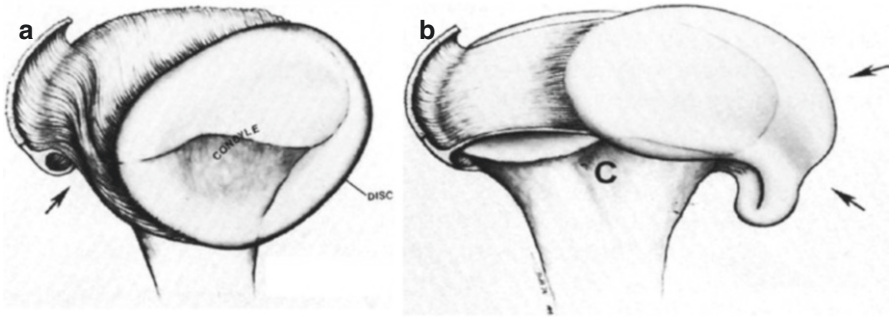

Fig. 10.10 Rotational and sideways disc displacement. (**a**) Diagrammatic depiction of a rotational displacement with one posterior edge being anterior the condyle while the other posterior edge being normally positioned. (**b**) This diagrammatic depiction shows a side-to-side displacement of the disc (arrow) relative to the condyle (C). It is termed as sideways displacement as there is no anterior component to the abnormal position

Disc displacement with or without reduction occurs in a variety of anatomic directions (Fig. 10.10a) [10]. However, the four general categories are anterior, anterior rotational (anteromedial and anterolateral), sideways (medial, lateral) (Fig. 10.10b), and posterior. Displacement of the disc may also be complete or partial. Partial disc displacement implies that one part of the disc is still interposed between the condyle and the glenoid fossa, while another part of the disc has been displaced out of its normal position. Complete disc displacement implies that the entire disc has been displaced out of its normal position.

Anterior and anterolateral displacements are generally of equal frequencies, and these are approximately two times the frequency of anteromedial displacement. Lateral and medial sideways displacements are similarly encountered, but being

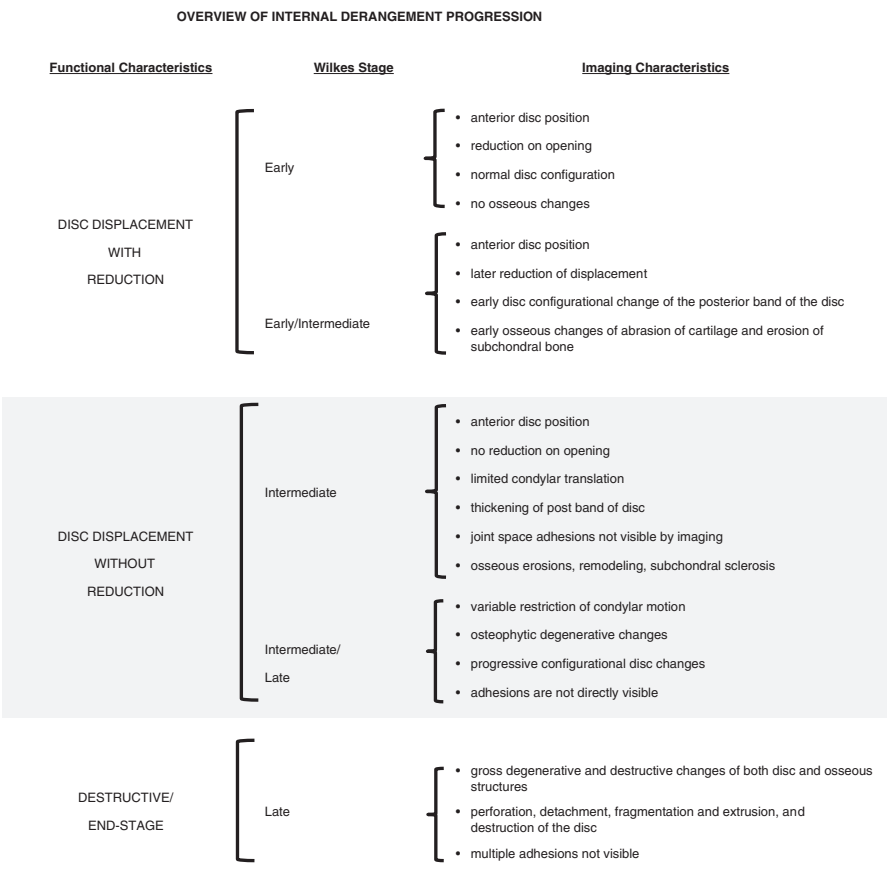

Fig. 10.11 Overview of internal derangement progression

only about 5% in frequency of the total number of disc displacement types. Posterior displacement is extremely rare.

Internal derangement of the TMJ due to disc displacement is generally believed to be a progressive disorder. A commonly used classification of internal derangement has been proposed by Wilkes [11]. An overview of internal derangement progression and relationships to functional and imaging characteristics followed in this chapter is detailed in Fig. 10.11.

10.4.1 Disc Displacement with Reduction

Temporomandibular clicking is among the most frequent finding in patients with TMJ pain and dysfunction. According to epidemiologic studies, clicking has been recognized to occur in between 14 and 44% of the general population. Clicking sounds have been ascribed to a variety of mechanisms including disc displacement, condylar subluxation, deviations in the form or shape of any of the articulating

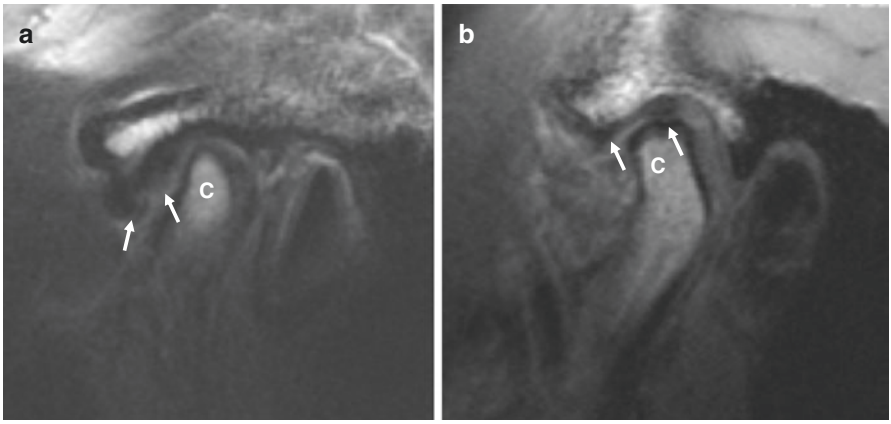

Fig. 10.12 Disc displacement with reduction and false suggestion of disc configurational changes. (**a**) Closed-mouth image shows apparent thickening and deformity of the disc (arrows) which is also anteriorly displaced. (**b**) Jaw opening beyond the click with disc reduction shows a normal biconcave configuration (arrows). The stage of internal derangement can be misclassified from intermediate to late if the images are not acquired properly. *C* condyle

surfaces, loose joint bodies, and fibrous bands or adhesions within the joint spaces. Less commonly encountered are joints with clicking that actually have disc displacement without reduction which represents a potential pitfall in clinical staging. A prior study demonstrated that 15% of patients with clicking were proven to have disc displacement without reduction. In these joints, the clicking was due to deformation of the disc and/or articulating surfaces. By the Wilkes classification (Fig. 10.11), disc displacement with reduction is in the early stage to early/intermediate stage of internal derangement, and the morphology of the disc is normal; however, deformations may occur. On the other hand, morphologic changes of the disc might be incorrectly diagnosed if MR imaging is not acquired beyond the opening click (Fig. 10.12a, b). This may lead to an overstaging of the severity of the internal derangement. The actual configuration of the disc is more accurately assessed by examining its morphology in the open-jaw position and after it has been reduced to a normal anatomic relationship to the condyle and tubercle.

With disc displacement, the displacements can be purely anterior (Fig. 10.13a–c); anterior rotational in medial or lateral directions (Fig. 10.14a–d); sideways, medial (Fig. 10.15a–d), or lateral; and very rarely true posterior.

10.4.2 Disc Displacement Without Reduction

Disc displacement without reduction specifies that the disc remains in the displaced position during all mandibular movement (Fig. 10.16). This means that attempts by the patient to manipulate the jaw so that the disc position is normal will be unsuccessful. Initially, this condition was termed as “closed lock,” which is a more descriptive term. However, with time the jaw opening can eventually become

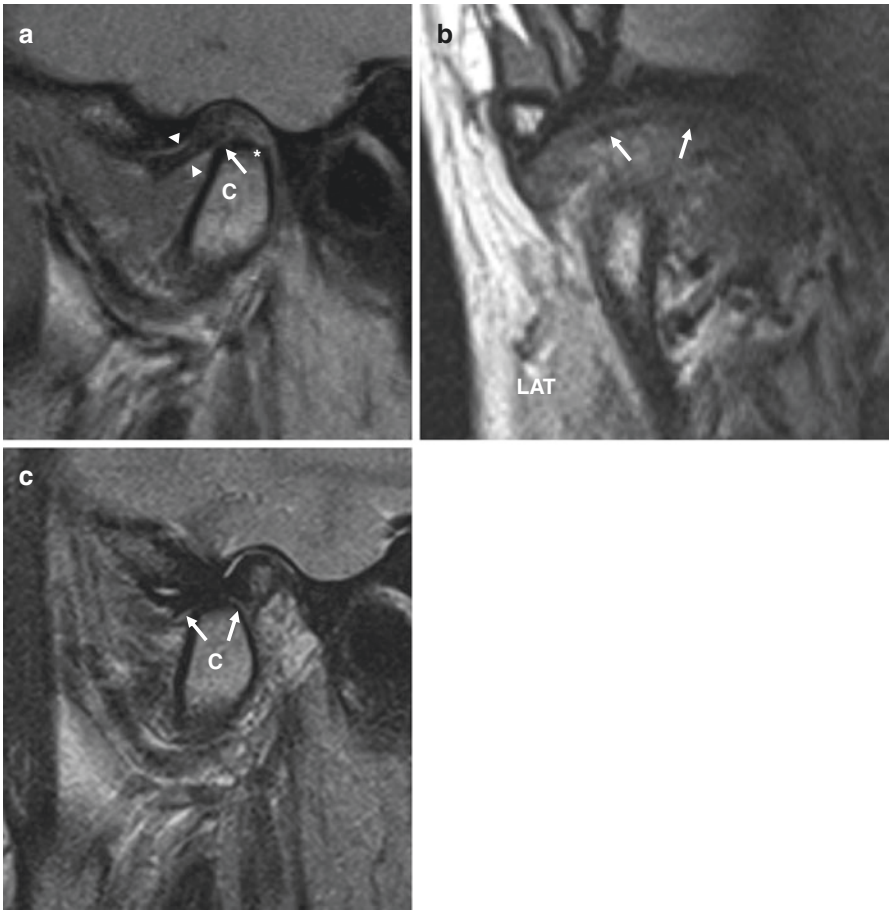

Fig. 10.13 Disc displacement with reduction that is anterior without rotation. (**a**) Closed-mouth MR image showing anterior disc (arrow) displacement. The disc has a normal configuration, and the thin zone (arrowheads) is also anterior to the articulating surfaces of the condyle (C) and tubercle. Small erosion (asterisk) is noted on the posterior condylar surface secondary to abrasion of the condyle against the posterior band of the disc associated with reduction occurring with forward condylar rotation and translation. (**b**) Closed-mouth MR image. (**b**). Coronal image with the jaw closed confirming no rotational component of the disc (arrows). (**c**) On opening the disc (arrows) is now reduced (recaptured) with the thin zone of the disc articulating with the superior surface of the condyle (C) and tubercle. This is a T2-weighted image and shows synovial fluid in the upper joint space and the normal "bow tie" disc configuration. *LAT* lateral

relatively normal again in spite of the disc being anteriorly displaced. For this reason, the term "closed lock" was later replaced by anterior disc displacement without reduction. With acute disc displacement, there can be a history of a click that suddenly stopped in association with limited jaw opening to less than 25 mm interincisal dimension. There are often multiple episodes of joint pain and temporal headaches with transient catching, locking, and sustained jaw locking. As

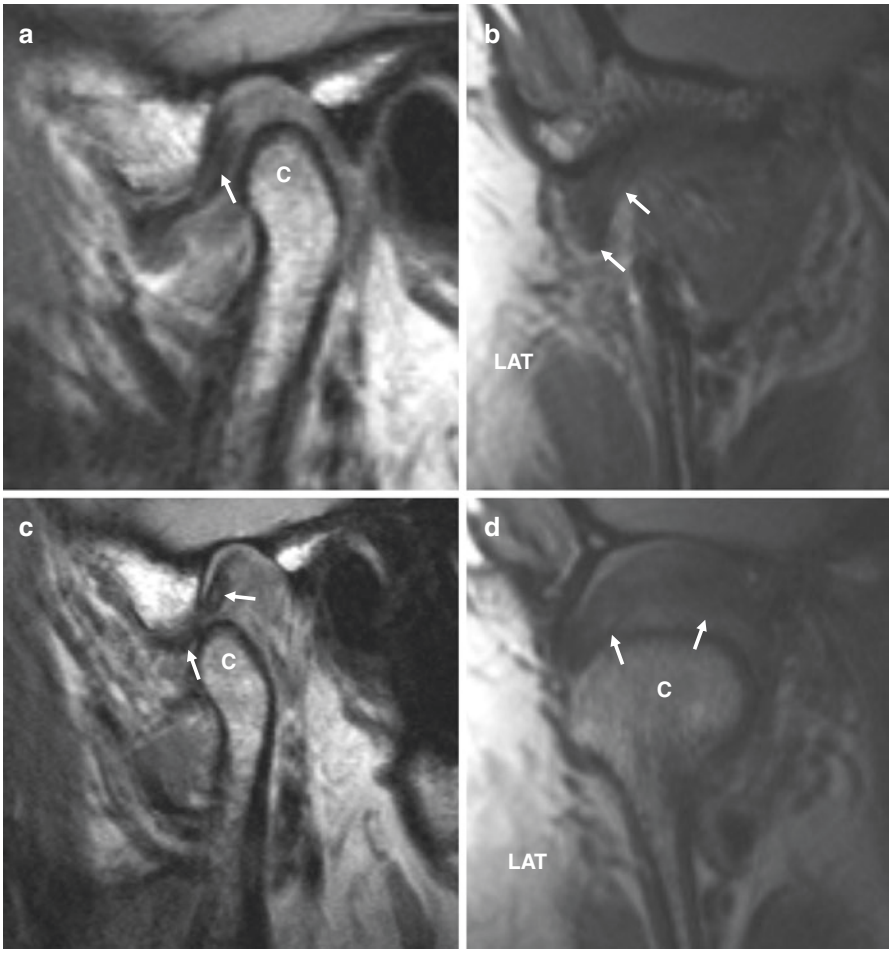

Fig. 10.14 Anterolateral rotational disc displacement with complete reduction. (**a**) Closed-mouth sagittal image shows anterior disc (arrow) displacement. (**b**) The closed-mouth coronal image shows a lateral rotational aspect to the disc (arrows) displacement. (**c**) Opened jaw position in the sagittal image shows reduction of the disc (arrows) displacement. The condyle (C) has translated anteriorly and downward along the posterior slope of a prominent tubercle. (**d**) Open-mouth coronal image shows the disc (arrows) to be reduced into a central position relative to the condyle (C) which has translated downward and anteriorly. *C* condyle, *LAT* lateral

previously noted, the jaw deviates to the involved side on opening. This is a significant progression and is Wilkes classification as intermediate (Fig. 10.11).

With chronic disc displacement, jaw opening can eventually become relatively normal, even though the disc is completely displaced. There is restoration of jaw motion with progressive increase in anterior condylar translation. There can be reduction in pain to normal values. Some patients may continue to experience clicking due to multiple irregular surfaces that provide frictional restrictions such in

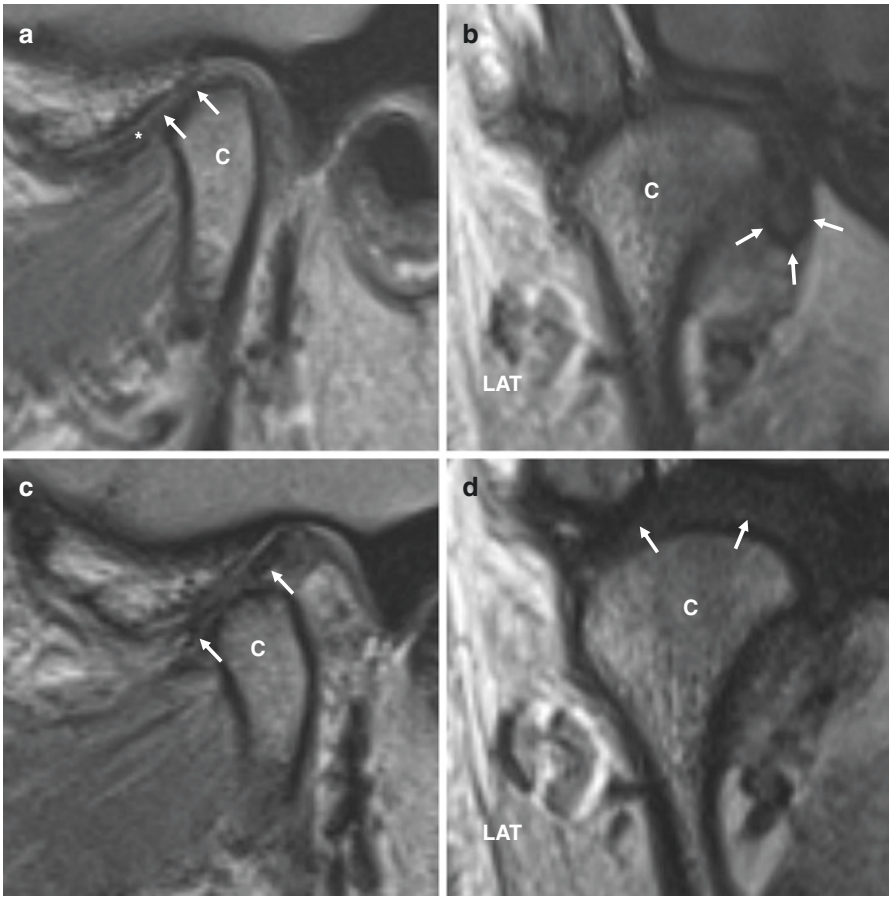

Fig. 10.15 Medial sideways disc displacement with reduction. (**a**) Closed-mouth sagittal image does not show the disc to be in the articular fossa (arrows). This is called the "empty fossa" sign. A common pitfall is to mistake thickening of the lateral pterygoid fascia (asterisk) as the disc. (**b**) Closed-mouth coronal image shows a sideways medial disc displacement (arrows) having a hairpin configuration. (**c**) Open-mouth sagittal image shows the disc (arrows) to now be visible in the fossa in a normal position and with a normal configuration. (**d**) Open-mouth coronal image confirms that the disc (arrows) to be in a normal (reduced) position above the condyle (C). The clinical significant of purely sideways disc displacement is not well understood. *C* condyle, *LAT* lateral

cases of adhesions and advanced degenerative changes in the condyle, disc, and fossa (crepitus is also heard). The clinical assessment without imaging of disc displacement without reduction that is associated with limited jaw opening has a high sensitivity and specificity. The clinical assessment when there is no limitation of jaw opening has a lower sensitivity and specificity.

Deformation of the disc is a salient characteristic in intermediate- to late-stage internal derangement (Fig. 10.11). Deformation most commonly begins with thickening and enlargement of the posterior band of the disc (Fig. 10.16) [12]. Thus, the

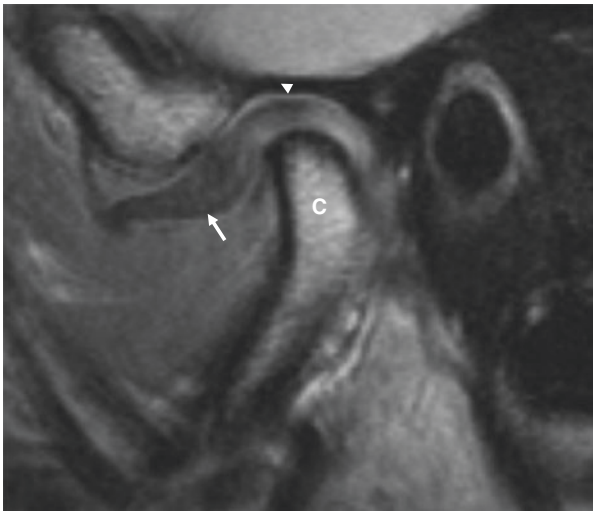

Fig. 10.16 Anterior disc displacement without reduction, disc deformation, and pseudodisc. Closed-mouth MR image showing disc (arrow) displacement without reduction. There is enlargement and thickening of the posterior band (arrow) and fibrosis (low signal intensity) of the anterior part of the posterior attachment termed as "pseudodisc." The central thin zone and anterior third of the disc are diminished. Histologically, the deformed disc frequently shows layers of proliferating cells and mucinous degeneration. There is early osteophyte formation on the anterior condylar (C) surface. This is Wilkes stage, intermediate to late

superoinferior thickness as well as the anteroposterior dimension of the posterior band gradually increases. The thickening of the posterior band takes place essentially on the undersurface of the disc. The upper surface remains relatively flat. The anterior one-third of the disc becomes merged with the anterior capsule and regresses in size. The central thin zone of the disc, which in this condition is nonfunctional, also becomes smaller, and sometimes folds on opening, ending with a biconvex configuration of the disc (Fig. 10.16).

In conjunction with disc deformation, the posterior disc attachment appears stretched and becomes longer and thinner. This partially explains the increasing opening capacity of the jaw that many patients experience after initially suffering from limitation of opening with disc displacement without reduction. Loosening or tearing of the joint capsule, resolution of muscle spasm, and detachment or perforation of the posterior disc attachment probably are also responsible for the increased jaw mobility that frequently occurs over time. With the chronically displaced disc that is associated with thickening of the posterior band, histologically the deformed disc frequently shows layers of proliferating cells on the surface of the abnormally thickened posterior band. There is also an increase in the number of transverse fibers encroaching upon the central thin zone of the disc and mucinous degeneration of the posterior band, as well (Figs. 10.17 and 10.18) [12]. Other histologic alterations include hyalinization, calcification, and cartilaginous metaplasia of the disc [6].

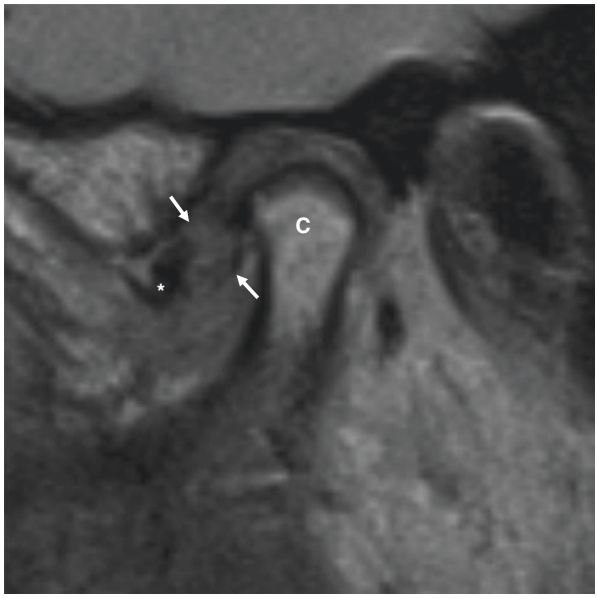

Fig. 10.17 Disc displacement without reduction and late-stage internal derangement with thickened posterior band of the disc (arrows). The thickened displaced disc is flexed downward, and there is near obliteration of the thin zone (asterisk), now with a notch-like appearance. The condyle (C) is flattened and there is anterior osteophytosis

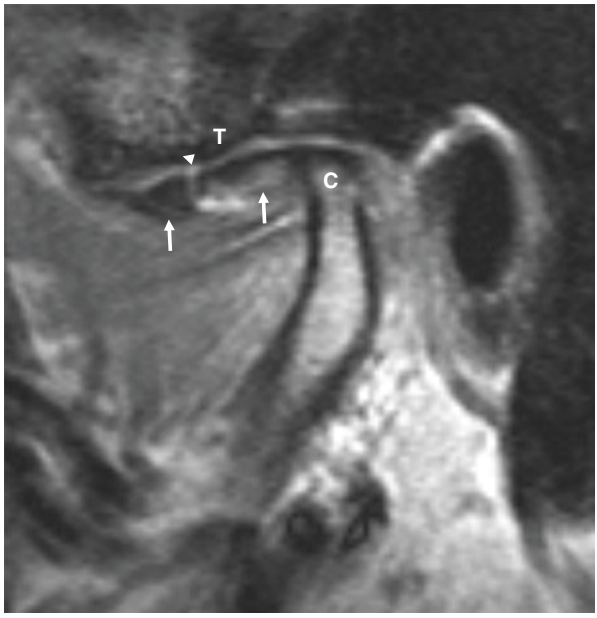

Fig. 10.18 Chronic disc displacement without reduction and with disc deformation. The disc (arrows) is markedly deformed with a thickened posterior band. There is a cleft (arrowhead) in the disc itself. The tubercle (T) is markedly flattened and sclerotic (dark signal intensity) that is typical for disc posterior attachment perforation. The condyle (C) is thinned, showing regressive remodeling. This represents late-stage disease

In some circumstances with chronic internal derangement, the anterior part of the posterior attachment near the posterior band of the disc develops a fibrotic character (Fig. 10.16). The normal pattern of collagen fibers at the union of the posterior attachment and the posterior band is replaced by a compact mass of fibers that

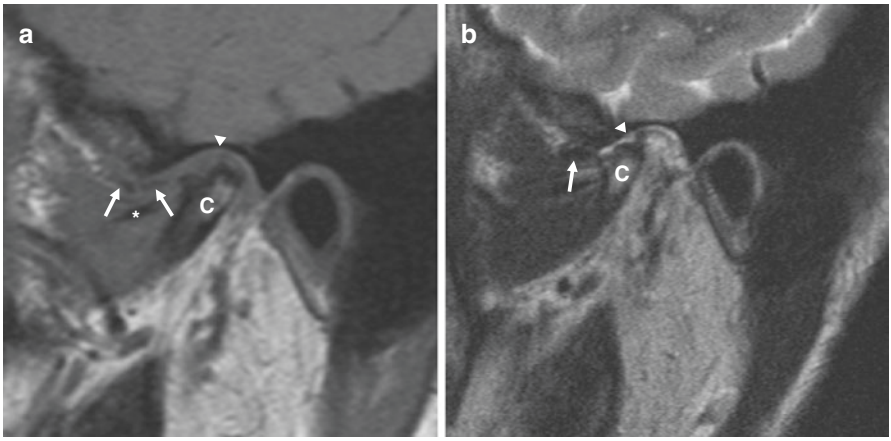

Fig. 10.19 End-stage, Charcot-like destructive joint. (**a**) Closed-mouth sagittal image showing "broken-glass" spiculated surface of the condyle (C). There is near complete fragmentation and autolysis of the extruded disc (arrows). There is apparent discontinuity of the temporal posterior attachment (arrowhead). Chronic thickening of the lateral pterygoid fascia is apparent (asterisk). (**b**) Open-mouth sagittal image employing a T2-weighted pulse sequence showing a single joint space (arrow) that is compatible with an MR-arthrographic demonstration of posterior disc attachment perforation or detachment. Spiculation of the articulating surface of the condyle (C) and tubercle is demonstrated. The disc (arrows) is anteriorly displaced and deformed

contains fewer or none of the small vessels normally present. When this fibrosis develops, the configuration of the posterior disc attachment sometimes also changes to resemble the posterior band of the disc, which has been likened to a "pseudodisc" (Fig. 10.16).

Perforations are seen in approximately 5–15% of the joints with disc displacement at autopsy. Perforations may also occur both in the disc and the disc attachment. Perforations associated with disc displacement are more commonly seen in the posterior attachment, rather than in the disc itself, and in the late to end stages which are associated with arthrosis (Fig. 10.19a, b). Perforation of the disc proper is also seen, especially in elderly individuals. These probably have a different etiology, such as simple wearing down over time, compared with perforations of the posterior disc attachment associated with disc displacement.

As for displacement with reduction, rotational and sideways disc displacement occurs in chronic displacement without reduction. An example of late- to end-stage medial sideways displacement is shown in Fig. 10.20a–c. Posterior disc displacement may sometimes actually represent disc fragmentation and retraction of the posterior disc behind the condyle (Fig. 10.21).

Arthrosis or degenerative joint disease has been defined as deterioration of the articular soft tissue cover and exposure of bone. Degenerative joint disease of the TMJ is frequently associated with disc displacement without reduction, and it has been documented in more than 50% of patients with this condition. Arthrosis of the bony structures of the TMJ associated with internal derangement fits into the

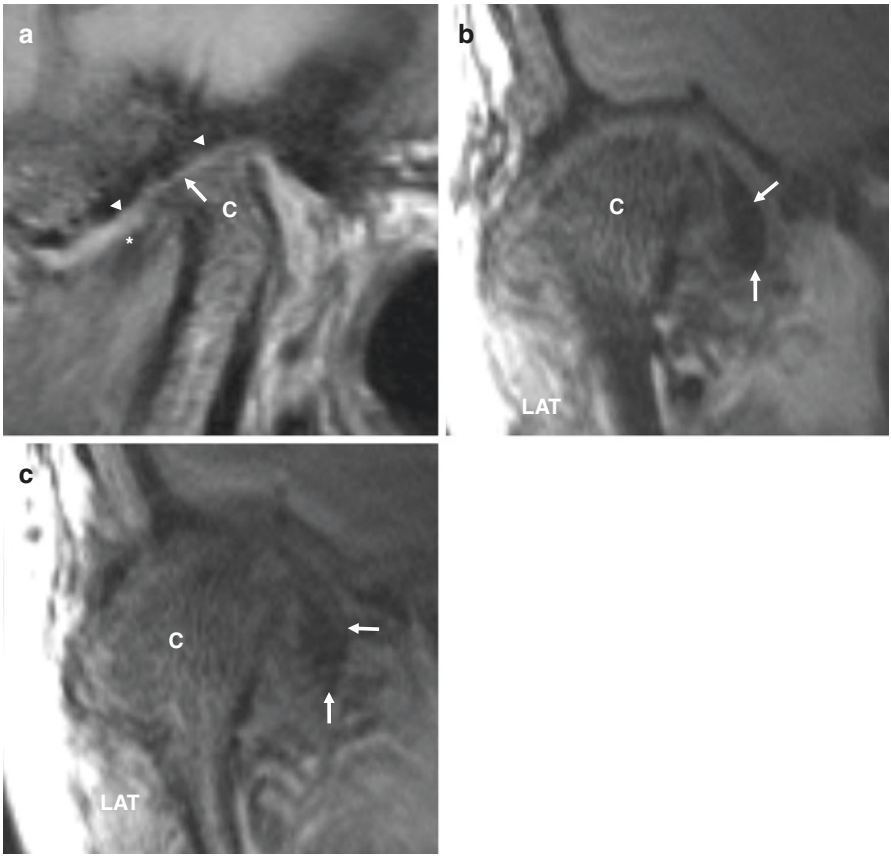

Fig. 10.20 Medial sideways disc displacement without reduction in association with advanced osseous disease. (**a**) Closed-mouth image in the sagittal plane. The disc is not visible in the fossa. There is marked flattening and roughening of the condyle (arrow) and tubercular articulating surface (arrowheads) which is typical for disc perforation. Thickening of the pterygoid fascia (asterisk) should not be mistaken for the disc. Low signal intensity of the tubercular surface represents sclerosis. Compare with Fig. 10.15a–d. (**b**) Closed-mouth coronal image shows medial sideways disc (arrows) displacement. (**c**) Open-mouth sagittal image shows no reduction of the sideways disc (arrows) displacement. This is classified as late to end-stage disease. *C* condyle, *LAT* lateral

intermediate- to late-stage classification scheme (Fig. 10.11). Examples of intermediate- to late-stage internal derangement are shown in the figures.

An inflammatory component of internal derangement can be manifested by the occurrence of joint fluid and bone marrow alterations. A minimal to moderate amount of synovial fluid can be demonstrated in the TMJ by MR without the association of a painful joint. Marked collections of joint fluid are more likely to be associated with joint pain, though even in this circumstance, the correlation is not great. Examples of joint fluid collections in the TMJ, assessed by T2-weighted imaging, are shown in the figures. An additional value of joint fluid collections

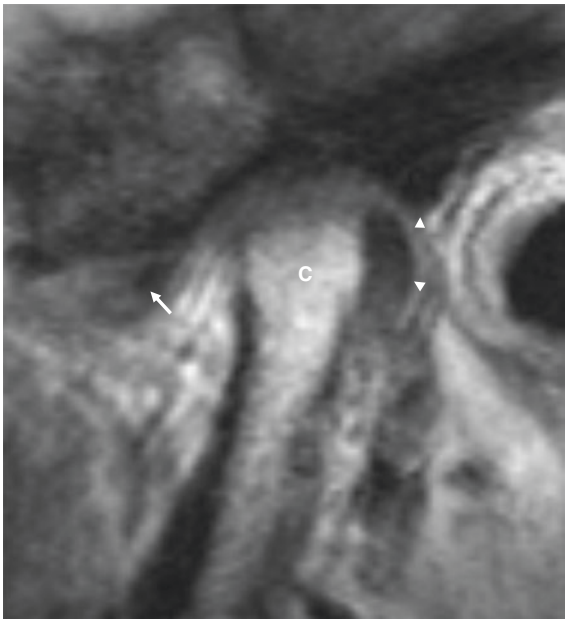

Fig. 10.21 Posterior disc displacement versus posterior disc retraction. Closed-mouth sagittal image showing a low signal intensity structure (arrowheads) posterior to the condyle (c). There appears to be a structure (arrow) anterior to the condyle typical for anterior disc displacement as well. This could be interpreted as posterior disc displacement as a primary consideration

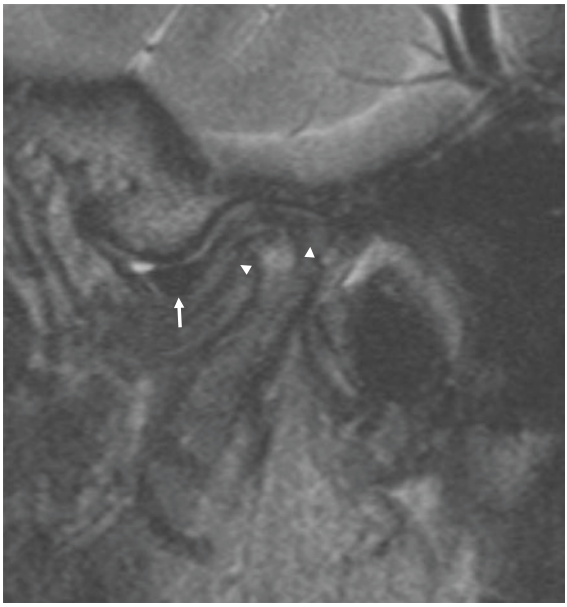

Fig. 10.22 Bone marrow edema is demonstrated by a focal region (arrowheads) of high signal intensity on this T2-weighted sagittal image of the joint in the mouth-closed position. The disc (arrows) is thickened, deformed, and anteriorly displaced without reduction

within the TMJ is the ability to better depict the morphology of the disc and, occasionally, disc perforations.

The normal bone marrow by MRI is bright on T1- or proton-weighted MR pulse sequences. With T2-weighted images, the fat components of the bone marrow become gray or darkened. Bone marrow edema, being of fluid content, converts to a bright signal with T2-weighted images (Fig. 10.22). In some circumstances the

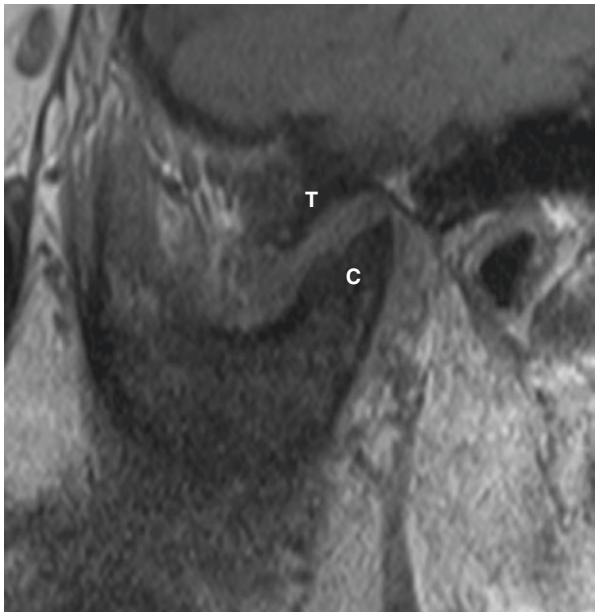

Fig. 10.23 Mouth-closed position MR image showing dark signal intensity of the boney structures on this proton-weighted image. The bone marrow should be bright on this pulse sequence. The condyle (c) and tubercle (T) are flattened and speculated. The disc is not visualized. This represents co-existing diffuse osseous sclerosis (fibrosis) and destructive internal derangement

bone marrow signal intensity is blackened on either T1- or T2-weighted images. This represents conversion of the normal marrow components to fibrous tissue or all bone (Fig. 10.23).

The hallmarks of osteoarthritis (degenerative joint disease) in weight-bearing joints such as the hip and knee are narrowing of the joint space, subchondral sclerosis, osteophytosis cyst or pseudocyst formation, and lack of pronounced osteoporosis [13]. These manifestations are similar to those encountered in the TMJ. Remodeling of bone is often seen with chronic disc displacement (Fig. 10.24). Chronic and advanced TMJ internal derangement also has parallel manifestations to destructive neuropathic (Charcot) arthropathy with the same degenerative changes as osteoarthritis, but seen in the most severe form (Figs. 10.19a, b and 10.20a–c). Hallmarks are fragmentation of bone and cartilage, chronic synovitis with joint effusion, and joint instability with subluxation or dislocation. Bone spiculation is also commonly seen in advanced internal derangement (Fig. 10.19a, b). Osteophyte change in the bony margins is also a common feature of advanced internal derangement, representing some element of remodeling. Subchondral bone cysts are sometimes encountered (Fig. 10.25). These are more common in weight-bearing joints than in the TMJ. They represent hydraulic intrusion of synovial fluid through surface erosion into subchondral bone.

Differences between osteoarthritis and inflammatory arthritis is that in the latter, there are marginal or central erosions, absent or minimal subchondral sclerosis, lack of osteophytes, and presence of osteoporosis. Osteonecrosis (ischemic or avascular) is a condition where there is cellular death of bone tissue when the bone is deprived of sufficient supply of arterial blood. Etiologies include sickle cell disease, steroid treatment in renal transplant recipients, lupus, and radiation exposure. These rarely manifest in the TMJ.

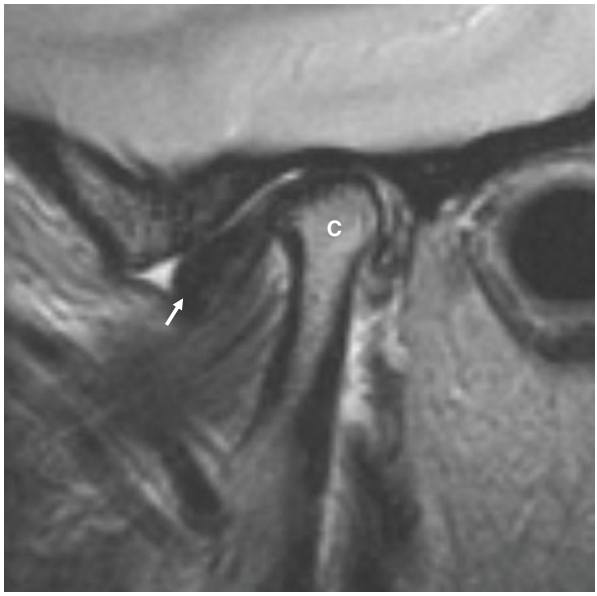

Fig. 10.24 Late-stage internal derangement with remodeling. Closed-mouth sagittal T2-weighted image shows disc (arrow) displacement without reduction and with marked deformity of the disc. There is synovial fluid in the upper joint space as manifested by a bright signal intensity band above the disc. The condylar surface appears intact, but the condyle is deformed, probably representing remodeling. The temporal boney surfaces are of low signal intensity representing sclerosis or fibrosis. There is anterior osteophytosis of the deformed condyle

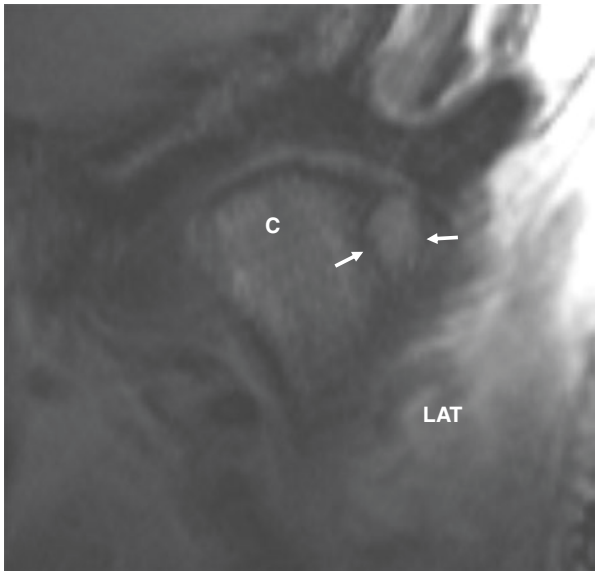

Fig. 10.25 MR proton-weighted coronal image in the mouth-closed position showing a cystic structure (arrows) showing an increased signal intensity. This is a typical appearance of a subchondral cyst associated with disc displacement. *C* condyle, *LAT* lateral

Postoperative changes in the TMJ are usually not optimally followed using MR when any metallic materials are used as these components create signal void artifacts (Fig. 10.26) and image distortion. Nonmetallic materials, though without signal void distortion, have no innate image creation. Plain film and CT are preferred for these circumstances.

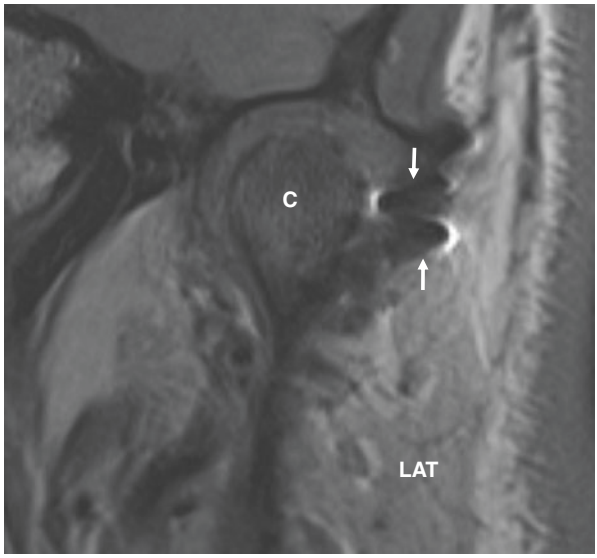

Fig. 10.26 Coronal image showing metal artifact in the lateral soft tissues of the joint. Metal creates a signal void (arrows) and image distortion. Consideration of nonmetallic surgical materials can decrease this type of artifact and opportunity for postsurgical follow-up imaging. *C* condyle, *LAT* lateral

10.5 Ultrasonography of Temporomandibular Joint Internal Derangement

Ultrasound imaging is a highly sophisticated medical technology that could minimize or eliminate many of the negatives currently found in temporomandibular joint diagnostics. The potential for TMJ imaging includes the characteristics of being noninvasive, not requiring ionizing radiation, 3-D and 4-D imaging of joint function and mechanics, accessibility and ease of portability, interactive, point-of-care clinical adjunct, and low expense (Table 10.3) [14, 15]. Until the early 2000s, ultrasound imaging was dominated by cart-based systems priced in the $100,000–$200,000 range. Physicians, or specialized imaging technicians, in hospital radiology and cardiology departments operated these instruments. More recently, ultrasound imaging has proliferated in the form of handheld and laptop-size instruments that are finding use in a range of settings beyond conventional large-hospital imaging-focused departments. A combination of portability, low cost, and ease of use makes ultrasound imaging a tool becoming more readily available for professionals who need to obtain imaging diagnosis or guide therapeutic interventions quickly and efficiently. Ultrasound combines excellent ability for deep penetration into soft tissues with very good spatial resolution, with only a few exceptions, those involving overlying bone or gas. Real-time imaging (up to hundreds and thousands of frames per second) enables guidance of therapeutic procedures and biopsies, characterization of the mechanical properties of the tissues, and ability to deposit energy locally for the potential for localized intervention encompassing tissue ablation [5]. Overall, ultrasound has become the most widely used imaging modality in modern medicine; and it will continue to grow and expand, developing more rapidly than any other existing medical imaging technology, including CT and MRI.

Table 10.3 Ultrasound potential for TMJ imaging

• Noninvasive
• No ionizing radiation
• 3-D and 4-D imaging of joint functioning and mechanics
• Accessible and ease of portability
• Interactive, point-of-care adjunct
• Low expense

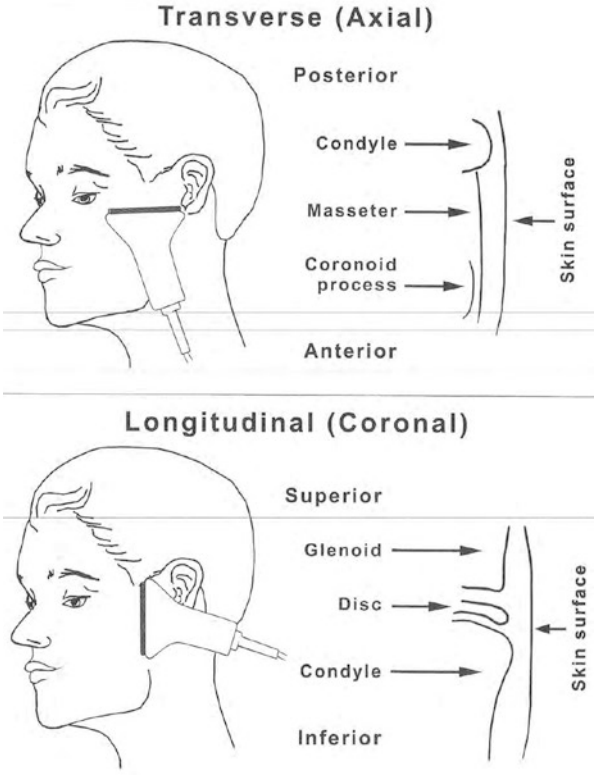

Fig. 10.27 Diagramatic depictions of ultrasound probe placement using an external facial approach for transverse (axial) and longitudinal (coronal) images of the temporomandibular joint. Top, in the axial plane, there is only demonstration of the outer third of the soft tissue anatomy, including the disc. Bottom, in the coronal plane, there is also only demonstration of the outer third of the joint anatomy

There is currently no imaging modality that can depict dynamic joint function, an integral characteristic of routine medical ultrasound imaging and currently unavailable with the abandonment of TMJ arthrography because of its invasive nature. The availability of TMJ imaging in the clinical setting, as could be achieved with ultrasound, beyond primary clinical diagnosis, could expand the possibility of screening protocols to rule out disc displacement in other dental patients.

Prior attempts at developing ultrasound of the TMJ have been suboptimal because of the use of large-sized imaging probes that are a constraint to imaging approaches of only the axial and coronal planes because images only can be acquired externally from the side of the face and the front of the ear (Fig. 10.27) [14, 15]. Thus, the external approach has not gained clinical acceptance because (1) sound penetration is severely limited by the markedly contoured anatomy of the bony

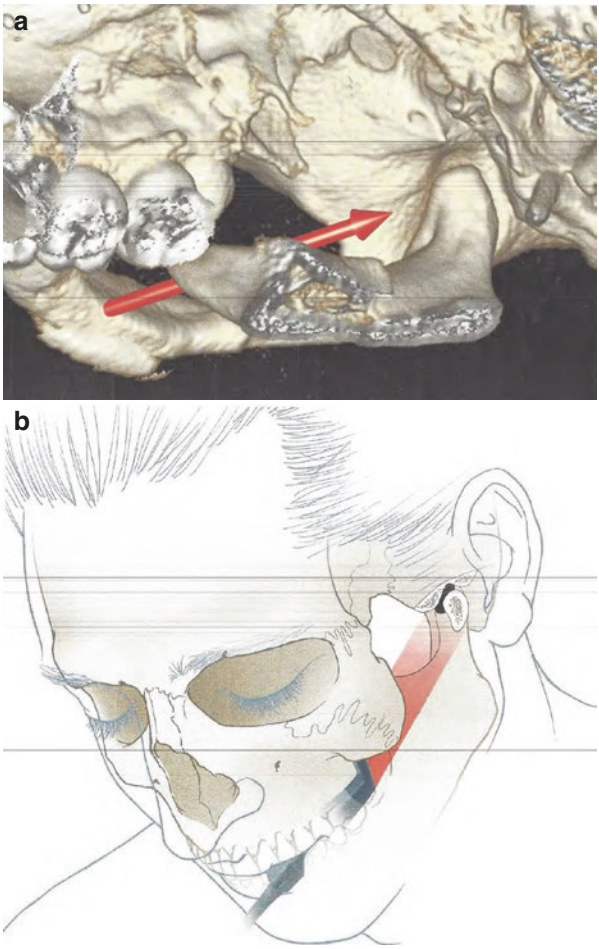

Fig. 10.28 Acoustic window for sagittal imaging without bone obstruction. (**a**) Oblique axial three-dimensional CT showing a bony window (arrow) for direct sagittal ultrasound imaging. (**b**) Diagrammatic depiction of probe orientation between the cheek and gum of the upper maxillary arch to acquire sagittal ultrasound images of the condyle and disc through a soft tissue acoustic window that is without bone hindrance

condyle and glenoid fossa and (2) restricted acquisition is limited to only the axial (transverse) and coronal (longitudinal) planes of imaging. Bone barriers restrict sound penetration to only the superficial aspect of the entire TMJ soft tissue anatomy. Even if ultrasound sound energy had the penetrating ability to image the entire axial and coronal TMJ anatomy, these two imaging planes are not adequate to diagnose disc displacement. On the other hand, literature and extensive clinical experience derived from TMJ imaging, including arthrotomography, CT, and MR, have shown that the sagittal plane of imaging is necessary for the effective and accurate depiction of the TMJ disc, condyle, and fossa. As suggested previously, an examination of the facial skeletal anatomy by three-dimensional CT shows the possibility of a true sagittal (longitudinal) soft tissue imaging window into the TMJ through an intraoral route (Fig. 10.28a). A diagrammatic depiction of the soft tissue acoustic window is shown in Fig. 10.28b.

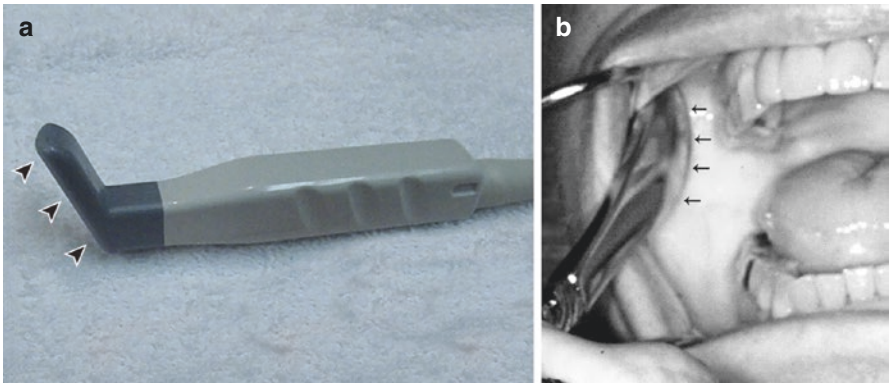

Fig. 10.29 Intraoral probe and probe positioning. (**a**) Philips skeletal hockey-stick probe used for transoral sagittal ultrasound probe. The probe imaging aperture (arrowheads) is placed vertically between the cheek and gum of the upper maxillary arch and directed cephalad toward the external auditory canal. (**b**) Spatula demonstrating placement of probe aperture for intraoral imaging. The hockey-stick probe is ergonomically suboptimal but is what is currently available. Arrows delineate the edge of the spatula where the probe aperture would be located

The author and colleagues have shown that high-resolution sonography with ultrasonic probe configurations (Fig. 10.29a) that are in routine use, though not optimal for ergonomics, can acquire the necessary imaging depth and sagittal plane of imaging through an intraoral route [15]. Imaging is with the patient in either a supine or laterally recumbent position and with the imaging side upward. The probe is placed in a condom-like cover filled with an ultrasound gel. A high-resolution linear probe, in this case a linear skeletal hockey-stick probe, is placed between the cheek and gingiva of the upper maxillary arch. The probe is directed toward the external auditory canal (about 30–50° cephalad) and parallel to the maxillary arch and ipsilateral molars (Fig. 10.29b). The probe is swept vertically and from side to side until the condylar head is visualized by its echogenic, arc-shaped subcondylar cap (Fig. 10.30). Images are then acquired in both static and dynamic modes with jaw opening and closing. Sagittal transoral sonographic imaging of the TMJ is shown in the figures in both normal and abnormal subjects. The condyle is vertically oriented and hypoechoic (dark), which is typical for bone. The arc-shaped condylar surface or "cap" is hyperechoic, which is typical for all subcondylar bone. The biconcave disc is above the condylar cap and is slightly hypoechoic. The pterygoid muscles are noted to be hypoechoic, the muscle bundles typically being separated by hyperechoic fat and fascial planes.

Images from another asymptomatic, normal subject are shown in Fig. 10.31a, b. These images were acquired in the closed-mouth position with typical images in the sagittal imaging plane. The posterior band of the disc is at the 12 o'clock position relative to the condylar head, and the thin zone is articulating with the anterior condylar prominence. The disc is hypoechoic. This is in comparison with sonographic and MR images in a 65-year-old female patient showing slight anterior disc

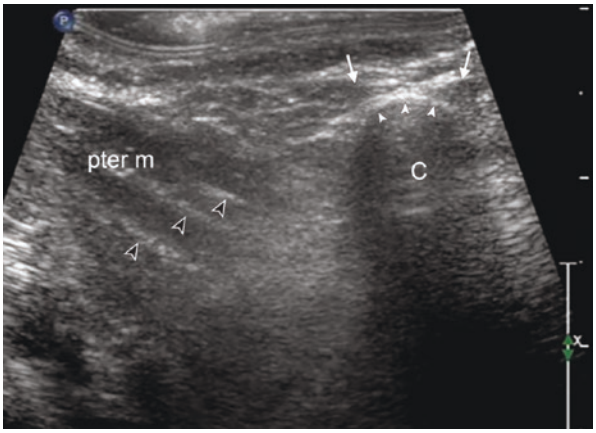

Fig. 10.30 Sagittal sonographic image in a 35-year-old male volunteer asymptomatic for temporomandibular joint pain and dysfunction shows an anatomic overview. The condyle (C) is vertically oriented and hypoechoic (dark). The arc-shaped condylar surface, or "cap," is hyperechoic (small arrowheads), typical for subcondylar bone. The disc (arrows) is above the condylar cap, is hypoechoic, and is in a normal anatomic position in association with slight anterior condylar translation. The pterygoid muscles (pter m) are noted to the left (anterior), with hypoechoic muscle bundles separated by hyperechoic (large arrow heads) fat and fascial planes

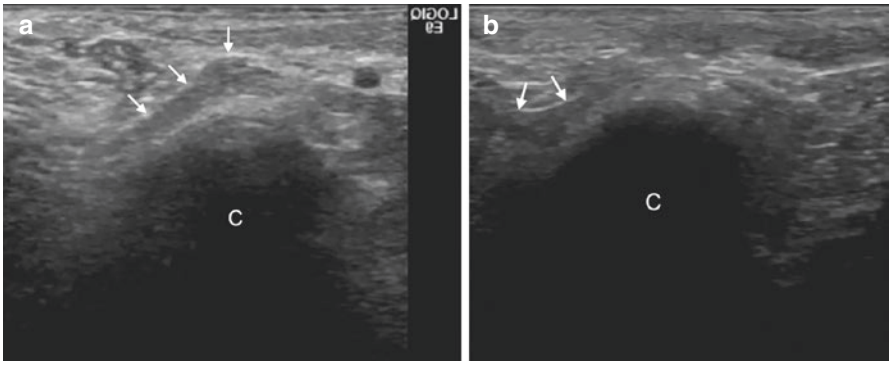

Fig. 10.31 Sagittal transoral sonographic imaging of the TMJ in an asymptomatic volunteer in the closed-mouth position (**a**) The condyle (C) and the posterior band and thin zone of the disc (arrows) are in a normal anatomic position. (**b**) The anterior disc band (arrows) is shown along the anterior condylar surface. *C* condyle

displacement of the right TMJ (Fig. 10.32a, b) and a left-sided pseudo-articulation secondary to a healed subcondylar fracture (Fig. 10.33a, b).

Sagittal transoral sonographic images (Fig. 10.34a–c) of the left temporomandibular joint in a 41-year-old female patient with bilateral long-standing TMJ pain and dysfunction are shown with MRI comparison. The first sagittal sonogram (Fig. 10.34a) was obtained both with a wider field of view of 8 MHz and

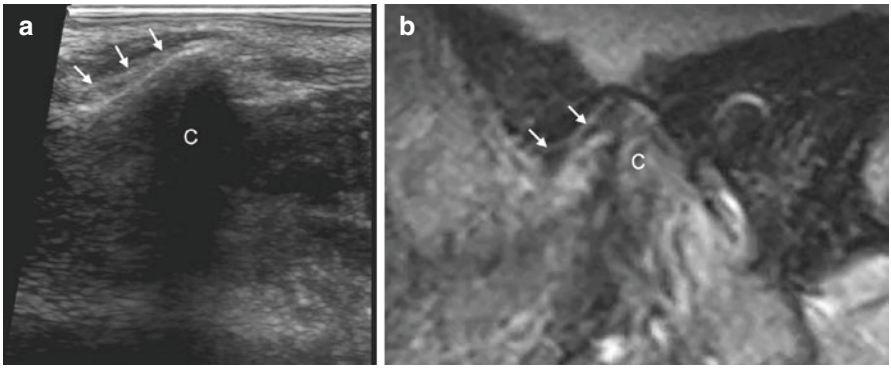

Fig. 10.32 Sagittal sonogram and MR in a 65-year-old female with minimal anterior disc displacement. (**a**) Mouth-closed sonogram shows minimal anterior disc displacement (arrows) as manifested by the thin zone being anterior to the anterior condylar prominence. (**b**) MR comparison confirms slight anterior disc displacement (arrows). *C* condyle

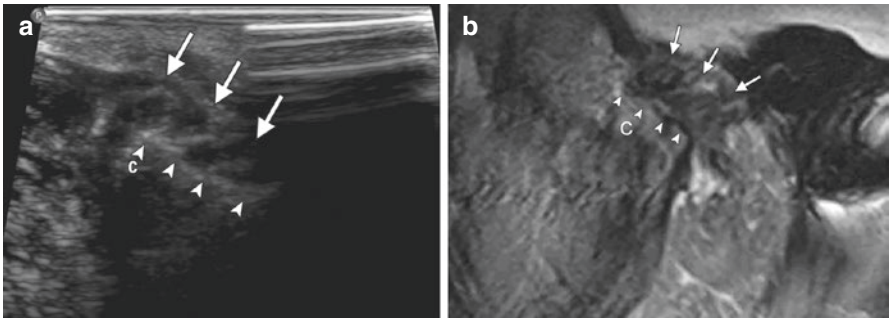

Fig. 10.33 Condylar neck pseudo-articulation in the same patient as shown in Fig. 10.32 following a healed left subcondylar fracture. (**a**) Sagittal sonogram shows a subcondylar stump (C) and pseudo-articulation (arrowheads) of the stump with the articular eminence that is partially encased in a fibrous ankylosis (arrows) that is hypoechoic. (**b**) The MR image shows comparable findings. Arrowheads show the pseudo-articulation and subcondylar stump (C). The fibrous ankylosis (arrows) is of low signal intensity by MR

low-resolution neonatal head probe and shows an anteriorly displaced, deformed disc with low echogenicity. A second sagittal sonogram is shown (Fig. 10.34b) using the hockey-stick probe. These findings are confirmed by a sagittal MRI image showing anterior disc displacement and disc deformation (Fig. 10.34c).

Future studies will be needed to encompass a wide variety of internal derangement types that are known to occur to develop better ergonomic probe designs. A prototype, dedicated TMJ imaging system is currently under development. Beyond the differentiation of a normal disc versus disc displacement on sonography, further advancements in technology may be required to diagnose rotational and sideways disc displacements. One possibility would be advancement into three-dimensional imaging probes.

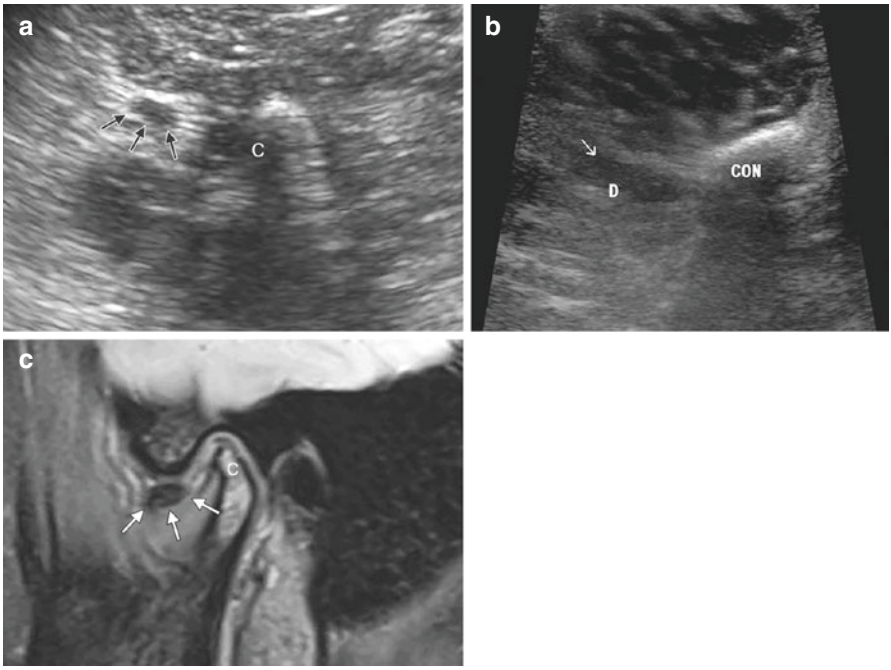

Fig. 10.34 Late-stage internal derangement with anterior disc displacement without reduction and disc deformation. (**a**) Wide-field of view 8 MHz low-resolution neonatal head probe inserted intraorally for sagittal sonography shows an anteriorly displaced, deformed disc (arrows) with low (dark) echogenicity. (**b**) Higher-resolution hockey-stick probe sonogram also shows disc (D) displacement (arrow) and deformation. (**c**) Sagittal MR shows anterior disc displacement (arrows) and disc deformation. *C* condyle, *CON* condyle

It is not suspected that TMJ sonography will achieve the high spatial resolution obtained by current MR technology. However, there is a reasonable possibility that sonography can assist the clinical examination and a screening role, as has already been achieved in other critical settings such as cardiology, obstetrics and gynecology, emergency department medicine, and many others.

In summary there is high promise for ultrasound imaging to be a new approach to TMJ screening in clinical diagnostics with the potential as a point-of-care office procedure.

References

1. Farrar WB. Diagnosis and treatment of painful temporomandibular joints. J Prosthet Dent. 1968;20:345–51.
2. Farrar W. Diagnosis and treatment of anterior dislocation of the articular disc. N Y J Dent. 1971;41:348–51.
3. Wilkes CH. Structural and functional aspects of the temporomandibular joint. Northwest Dent. 1978;57:287–94.

4. Wilkes CH. Arthrography of the temporomandibular joint in patients with the TMJ pain-dysfunction syndrome. Minn Med. 1978;61:645–52.
5. Brant WE, Helms CA, editors. Fundamentals of diagnostic radiology. Baltimore: Williams and Wilkins; 1994.
6. Katzberg RW, Westesson P-L. Diagnosis of the temporomandibular joint. Philadelphia: W.B. Saunders Company; 1993.
7. Tamimi D, Hatcher D, editors. Specialty imaging—temporomandibular joint. Philadelphia: Elsevier; 2016.
8. Drace JE, Enzmann D. Temporomandibular joint meniscus and bilaminar zone: MR imaging of the structure—diagnosis landmarks and pitfalls of interpretation. Radiology. 1990;177:73–6.
9. Ahmad M, Hallender L, Anderson Q, et al. Research diagnostic criteria for temporomandibular disorders (RDC/TMJ): development of image analysis criteria and examiner reliability for image analysis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;107:844–60.
10. Katzberg RW, Westesson P-L, Tallents RH, et al. Temporomandibular joint: MR assessment of rotational and sideways disk displacement. Radiology. 1988;169:741–8.
11. Wilkes CH. Internal derangement of the temporomandibular joint. Pathological variations. Arch Otolaryngol Head Neck Surg. 1989;115:469–77.
12. Scapino RP. Histopathology associated with malposition of the temporomandibular joint disc. Oral Surg Oral Med Oral Pathol. 1983;55:382–97.
13. Greenspan A. Orthopedic radiology. 2nd ed. New York and London: Gower Medical Publishing; 1992.
14. Katzberg RW. Is ultrasonography of the TMJ ready for prime time? Is there a “window” of opportunity? J Oral Maxillofac Surg. 2012;70:1310–5.
15. Katzberg RW, Conway WF, Ackerman SJ, et al. Pilot study to show the feasibility of high-resolution sagittal ultrasound imaging of the temporomandibular joint. J Oral Maxillofac Surg. 2017;75(6):1151–62.

Diagnosis of Temporomandibular Disorders Using DC/TMD Criteria

11

Ambra Michelotti, Stefano Vollaro, and Roberta Cimino

Abstract

It is strongly recommended that the gold standard for the diagnosis of TMD according to the DC/TMD and the differential diagnosis of orofacial pain conditions should be based primarily on information obtained from a comprehensive history together with a clinical examination and, when indicated, TMJ imaging (Schiffman and Ohrbac, JADA 147(6):438–445, 2016). Indeed for the TMJ disorders, the reference standard was established by board-certified radiologists using bilateral TMJ magnetic resonance imaging (MRI) and computed tomography (CT). Together with the physical diagnosis (Axis I), questionnaires for the evaluation of psychosocial factors were identified to assess a reliable and valid Axis II profile [Ohrbach, J Oral Rehabil 37:784–798, 2010). When the clinician uses the Axis I, diagnoses must first exclude odontogenic disease and other pain disorders that can occur in the masticatory system.

Temporomandibular disorder (TMD) represents a heterogeneous musculoskeletal disorder that is characterized by regional acute or persistent pain in the facial and/or preauricular areas, limitation or interference in jaw functions, and/or noises from the TMJs during jaw movements [1, 2]. Frequently there are also findings of hyperalgesia or symptoms from other painful disorders (comorbidities).

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The development of diagnostic criteria for TMD (DC/TMD) was necessary to ensure a common language among clinicians and establish an appropriate treatment plan or management for the diseases. This was the reason why a team of experts developed a diagnostic TMD classification, producing a dual-axis framework for the assessment and diagnosis of an Axis I physical diagnosis (for most common TMD) and an Axis II self-report psychosocial and behavioral screening questionnaire [1, 3, 4].

It is strongly recommended that the gold standard for the diagnosis of TMD according to the DC/TMD and the differential diagnosis of orofacial pain conditions should be based primarily on information obtained from a comprehensive history together with a clinical examination and, when indicated, TMJ imaging [1]. Indeed for the TMJ disorders, the reference standard was established by board-certified radiologists using bilateral TMJ magnetic resonance imaging (MRI) and computed tomography (CT). Together with the physical diagnosis (Axis I), questionnaires for the evaluation of psychosocial factors were identified to assess a reliable and valid Axis II profile [5]. When the clinician uses the Axis I, diagnoses must first exclude odontogenic disease and other pain disorders that can occur in the masticatory system.

The recently published DC/TMD is reliable and valid. These criteria cover the most common types of TMD, which include pain-related disorders (e.g., myalgia, headache attributable to TMD, and arthralgia) as well as disorders associated with the TMJ (primarily disc displacements and degenerative disease) [2]. Besides the DC/TMD, the expanded taxonomy for all TMDs has been developed to classify other diagnostic subtypes of TMD (Table 11.1) [6].

11.1 Temporomandibular Joint Disorders

11.1.1 Joint Pain

(a) *Arthralgia* is defined like a *pain of joint origin* that is affected by jaw movement, function, or parafunction and is replicated during the provocation TMJ test [4].

It is difficult to distinguish "arthralgia" from "arthritis" for their frequent overlap.

Arthralgia is perhaps more of a clinical finding of joint pain, whereas "arthritis" may comprise pain and can cause arthralgia [7]. At the same time, arthralgia may be due to overstretching of the joint and peripheral or central sensitization. One problem is certainly the absence of a reference standard for arthritis. We know that TMJ pain on palpation or TMJ movement pain has a high specificity for inflammatory activity [8] and is strongly related to the degree of inflammatory activity in the TMJ.

The reported values of sensitivity and specificity for the diagnosis of arthralgia are 0.89 and 0.98, respectively [4].

Table 11.1 Expanded taxonomy for temporomandibular disorders [6]

I. Temporomandibular joint disorders			
1. Joint pain	A. Arthralgia		
	B. Arthritis		
2. Joint disorders	A. Disc disorders	1. Disc displacement with reduction	
		2. Disc displacement with reduction with intermittent locking	
		3. Disc displacement without reduction with limited opening	
		4. Disc displacement without reduction without limited opening	
	B. Hypomobility disorders other than disc disorders	1. Adhesions/adherence	
		2. Ankylosis	a. Fibrous
			b. Osseous
	C. Hypermobility disorders	1. Dislocations	a. Subluxation
			b. Luxation
3. Joint diseases	A. Degenerative joint disease	1. Osteoarthrosis	
		2. Osteoarthritis	
	B. Systemic arthritides		
	C. Condylysis/idiopathic condylar resorption		
	D. Osteochondritis dissecans		
	E. Osteonecrosis		
	F. Neoplasm		
	G. Synovial Chondromatosis		
4. Fractures			
5. Congenital/ developmental disorders	A. Aplasia		
	B. Hypoplasia		
	C. Hyperplasia		
II. Masticatory muscle disorders			
1. Muscle pain	A. Myalgia	1. Local myalgia	
		2. Myofascial pain	
		3. Myofascial pain with referral	
	B. Tendonitis		
	C. Myositis		
	D. Spasm		
2. Contracture			
3. Hypertrophy			
4. Neoplasm			
5. Movement Disorders	A. Orofacial dyskinesia		

(continued)

Table 11.1 (continued)

	B. Oromandibular dystonia		
6. Masticatory muscle pain attributed to systemic/central pain disorders	A. Fibromyalgia/ widespread pain		
III. Headache			
1. Headache attributed to TMD			
IV. Associated structures			
1. Coronoid hyperplasia			

Arthralgia

Pain of joint origin that is affected by jaw movement, function, or parafunction, and replication of this pain occurs with provocation testing of the TMJ.

History of the patient must be positive for:
1.Pain in the jaw, ear, or in frontofear; AND
2. pain modified with jaw movement, functionor parafunction

Exam must be positive for:
1. confirmation of painlocation in the area of TMJ(s); AND
2. report of familiar paini n the TMJ with at least one of the following provocation test:
a. palpation of the lateral pole or around the lateral pole; OR
b. maximum unassisted or assisted opening, right/left lateralor protrusive movement(s)

Sensitivity 0.89; specificity 0.98.

SchiffmanE. et all, 2014

(b) *Arthritis*: represents pain of joint origin with clinical characteristics of inflammation or infection over the affected joint: edema, erythema, and/or increased temperature. Associated symptoms can include dental occlusal changes. This disorder is also referred to as synovitis or capsulitis, although these terms limit the sites of nociception. This is a localized condition; there should be no history of systemic inflammatory disease [6].

11.1.2 Joint Disorders

11.1.2.1 Disc Disorders

1. *Disc displacement with reduction*: represents an intracapsular biomechanical disorder involving the condyle-disc complex. In the closed-mouth position, the disc is in an anterior position relative to the condylar head, and the disc reduces

upon opening of the mouth. Medial and lateral displacement of the disc may also be present. Clicking, popping, or snapping noises may occur with disc reduction [1, 4, 6]. Without imaging the sensitivity is 0.34 and the specificity is 0.92. TMJ magnetic resonance imaging is the reference standard for this diagnosis.

Disc displacement with reduction

An intracapsular biomechanical disorder involving the condyle-disc complex. In the closed mouth position the disc is in an anterior position relative to the condylar head and the disc reduces upon opening of the mouth.

Medial and lateral displacement can be also present. Clicking, popping, or snapping noises may occurr with disc reduction. An history of prior locking in the closed position coupled with interference in mastication precludes this diagnosis

History of the patient must be positive for at least one of the following:

1. TMJ noise(s) will be presentin the past 30 days during jaw movement or function; ***OR***

2. Patient report any noise present during the exam

Exam must be positive fort he following:

1.Clicking, popping, or snapping noise during both opening or closing movement, detected with palpation during at least one/three repetitions of jaw opening and closing; ***OR***

2.a. Clicking, popping, or snapping noise detected with palpation during at least one/three repetitions of opening and closing movement(s); ***AND***

2b. Clicking, popping, or snapping noise detected with palpationd uring at least one/three repetitions of right or left lateral or protrusive movement(s);

Imaging is the reference standard for this diagnosis
sensitivity 0.33; specificity 0.94

Schiffman E. et al, 2014

2. *Disc displacement with reduction with intermittent locking*: represents an intracapsular biomechanical disorder involving the condyle-disc complex. In the closed-mouth position, the disc is in an anterior position relative to the condylar head, and the disc intermittently reduces with opening of the mouth. When the disc does not reduce with opening of the mouth, intermittent limited mandibular opening occurs. When limited opening occurs, a maneuver may be needed to unlock the TMJ. Medial and lateral displacement of the disc may also be present. Clicking, popping, or snapping noises may occur with disc reduction [1, 6]. Without imaging the sensitivity is 0.38 and the specificity is 0.98. Imaging is the reference standard for this diagnosis.

Disc displacement with reduction with intermittent locking

An intracapsular biomechanical disorder involving the condyle-disc complex. In the closed mouth position the disc isin anterior position relative to the condylar head and the disc intermittently reduces with opening of the mouth.

When the disc does not reduce with opening of the mouth, ***intermittent limited mandibular opening*** occurs. When limited opening occurs, a maneuver may be needed to unlock the TMJ. Medial and lateraldisplacement of the disc can be also present. Clicking, popping, or snapping noises may occurr with disc reduction.

History of the patient must be positive for both the following:

1a. TMJ noise(s) are present in the past 30 days during jaw movement or function; ***OR***

1b. Patient report any noise present during the exam; ***AND***

2. Jaw locks with limited mouth opening was present, in the last 30 days, even for a moment, and then unlocks.

Exam must be positive for the following:

1.Clicking, popping, or snapping noise detected during both opening and closing movements, detected with palpation during at least one/three repetitions of opening and closing; ***OR***

2.a. Clicking, popping, or snapping noise detected with palpation during at least one/three repetitions of opening and closing movement(s); ***AND***

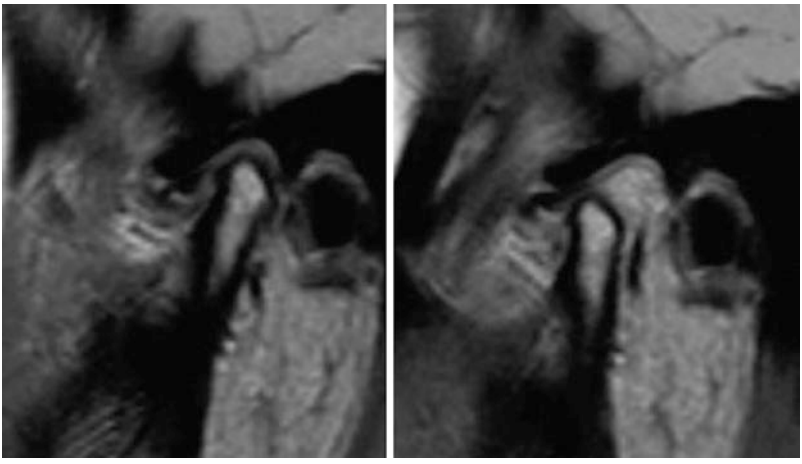
2b. Clicking, popping, or snapping noise detected with palpation during at least one/three repetitions of right or left lateral or protrusive movement(s);

Imaging is the reference standard for this diagnosis

Without imaging: Sensitivity 0.38; specificity0.98

Schiffman E et al, 2014

3. *Disc displacement without reduction with limited opening*: represents an intracapsular biomechanical disorder involving the condyle-disc complex. In the closed-mouth position, the disc is in an anterior position relative to the condylar head, and the disc does not reduce with opening of the mouth. Medial and lateral displacement of the disc may also be present. This disorder is associated with persistent limited mandibular opening that does not reduce with the clinician or patient performing a manipulative maneuver. This is also referred to as "closed lock." Limitation in jaw opening is severe enough to interfere with the ability to eat with maximum assisted opening (passive stretch) movement including vertical incisal overlap <40 mm [1, 4, 6]. Without imaging the sensitivity is 0.80 and the specificity is 0.97. Imaging is the reference standard for this diagnosis.



Disc displacement without reduction with limited opening

An intracapsular biomechanical disorder involving the condyle-disccomplex. In the closed mouth position the disc is in an anterior position relative to the condylar head and the disc does not reduce with opening of the mouth.

Medial and lateral displacement of the disc can be also present. This disorder is associated with persistent limited mandibular opening that does not reduce with a manipolative maneuver of the clinician or of the patient. This is also refererred to as ***"closed lock".***

History of the patient must be positive for boththe following:
1. Jaw locked so that the mouth would not open all the way; ***AND***
2. Limitation in jaw opening severe enough to limit jaw opening and interfere with ability to eat.

Exam must be positive for the following:
1. Maximu assisted opening (Passive stretch) movement including vertical incisor overlap <40mm

to confirm diagnosis TMJ some MRI criteria need to be respected
Without imaging: Sensitivity 0.80; specificity 0.97

Schiffman E et al, 2014

4. *Disc displacement without reduction without limited opening*: represents an intracapsular biomechanical disorder involving the condyle-disc complex. In the closed-mouth position, the disc is in an anterior position relative to the condylar head, and the disc does not reduce with opening of the mouth. Medial and lateral displacement of the disc may also be present. This disorder is *NOT* associated with current limited opening with maximum assisted opening (passive stretch) movement including vertical incisal overlap ≥40 mm [1, 4, 6]. Without imaging the sensitivity is 0.54 and the specificity is 0.79. Imaging is the reference standard for this diagnosis.

Disc displacement without reduction without limited opening

An intracapsular biomechanical disorder involving the condyle-disc complex. In the closed mouth position the disc is in an anterior position relative to the condylar head and the disc does not reduce with opening of the mouth.

Medial and lateral displacement of the disc can be also present. This disorder is not associated with current limited opening .

History of the patient must be positive for both the followingin the past:

1. Jaw locked so that the mouth would not open all the way; ***AND***
2. Limitation in jaw opening severe enough to limit jaw opening and interfere with ability to eat.

Exam must be positive for the following:
1. Maximu assisted opening (Passive stretch) movement including vertical incisor overlap ≥40mm

Imaging is the reference standard for this diagnosis
With outi maging: Sensitivity 0.54; specificity 0.79

Schiffman E et al, 2014

For the diagnosis of temporomandibular disorders, a diagnostic algorithm was proposed that starts with a good anamnesis, a specific clinical exam, and finally, where necessary, a definitive diagnosis, with TMJ MRI for DD and TMJ TC for DJD [1].

For the TMJ intra-articular disorders, there is a good accuracy for sensitivity and specificity. Consequently these diagnostic algorithms can be used only for screening purposes to obtain a preliminary diagnosis of disc displacement (DDs) or degenerative joint diseases (DJD). The only exception is the DD without reduction with limited opening that has a sensitivity of 80% and specificity of 97%. Subluxation is diagnosed on the basis of history and has acceptable diagnostic validity for clinical use [6].

TMJ noise by history is a recommended criterion for the intra-articular disorders of DD with reduction and DJD. This history criterion may be met by the patient's report of any joint noise (click or crepitus) during the 30 days prior to the examination or by the patient's detection of any joint noise with jaw movements during the clinical examination.

A diagnosis of DD with reduction requires examiner detection of clicking, popping, or snapping noises during the examination. Establishing a diagnosis of DJD necessitates examiner detection of crepitus (e.g., crunching, grinding, or grating noises) during the examination. For DJD, no distinction between fine versus coarse crepitus is made.

DD with reduction with intermittent locking and TMJ subluxation are included as new disorders.

11.1.3 Hypomobility Disorders Other than Disc Disorders

(a) *Adhesions/adherence*. Intra-articular fibrous adhesions/adherence and ankylosis are characterized by a restricted mandibular movement with deflection to the affected side on opening. In the case of bilateral involvement, asymmetries in mandibular movements during clinical examination will be less pronounced or absent. The condition is not usually associated with pain [6].

Adhesions may occur secondary to joint inflammation that results from direct trauma, excessive loading, or systemic conditions such as a polyarthritic disease and are typically associated with disc disorders. The adhesion is realized in the superior articular space, between disc and fossa, or in the inferior compartment between disc and condyle. An inferior adhesion limits the condylar rotation, during opening of the mouth; there isn't a limitation of this movement, but there is only a sensation of movement less fluid than normal. The adhesion realized in the upper articular compartment [9–11] is accompanied by a limitation of condylar translation with a consequently limited mouth opening. The disc adhesion can be temporary, and it is characterized by an intermittent clicking, or it is "persistent" like in the anchored disc phenomenon (ADP) [9].

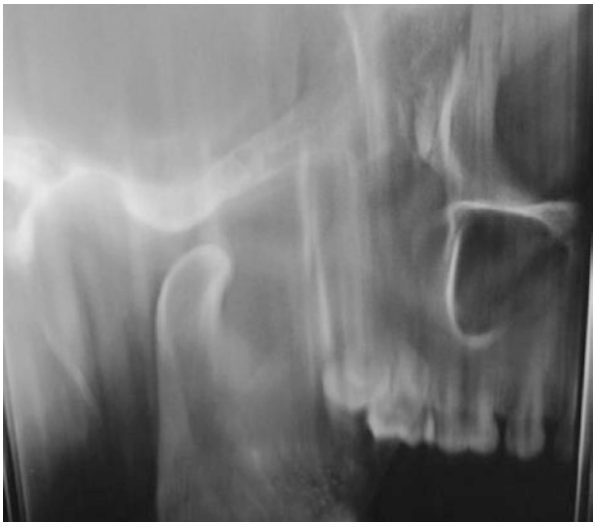
(b) *Ankylosis*. The most frequent cause of TMJ *ankylosis* is macrotrauma; less frequent causes are infection of the mastoid or middle ear, systemic disease, and

inadequate surgical treatment of the condylar area. Distinction is requested between intra-articular fibrous and osseous ankylosis.

1. *Fibrous ankylosis*: There are no gross bony changes, and the predominant radiographic finding is absence of ipsilateral condylar translation on opening. Note that fibrous ankylosis may be considered a more severe form of TMJ adhesions/adherence. It is characterized by a severe limited mouth opening capacity, in the absence of pain or joint sounds, but accompanied by marked deflection to the affected side and marked limitation of movement to the contralateral side.
2. *Osseus ankylosis* is characterized by a radiographic evidence of bone proliferation with marked deflection to the affected side and marked limited laterotrusion to the contralateral side. Sensitivity and specificity have not been established [6].

11.1.4 Hypermobility Disorders

(a) *Subluxation*: represents a hypermobility disorder involving the disc-condyle complex and the articular eminence. In the open mouth position, the disc-condyle complex is positioned anterior to the articular eminence and is able to return to a normal closed-mouth position without a manipulative maneuver. The duration of dislocation may be momentary or prolonged.



(b) *Luxation*: Luxation or complete dislocation represents a condition in which the condyle is positioned anterior to the articular eminence and is unable to return to a closed position, without a specific maneuver by the clinician. Pain may occur at the time of dislocation with residual pain following the episode. This disorder is also referred to as "open lock" [6].

The sensitivity and specificity have been established for only subluxation [4]. Without imaging and based only on history, the sensitivity is 0.98 and the specificity is 1.00.

Subluxation

A hypermobility disorder involving the disc-condyle complex and the articular eminene.In the open mouth position the disc-condyle complex is positioned anterior to the articular eminence and is unable to return to a normal closed mouth position without a manipulative maneuver.

The duratio of the dislocation may be momentary or prolonged.
When the patient can reduce the dislocation by him/herself , this is referred as a ***"Subluxation".***
When the patient need the assistance of the clinician to reduce the dislocation and normalize jaw movement, this is referred as a ***"Luxation".***

Historyofthe patientmustbepositive forboththe following:

1. *Jaw locking or catching in a wide open mouth position, even for a moment, so could not close from this position;*

AND

2. *Inability to close the mouthf rom a wide open position withouta self-maneuver.*

Althout no exam findings are required, when this disorde is present clinically, examination is positive for inability to return to a normal closed mouth position without the patient performing a manipulative maneuver.

With out imaging and based onlyon the history: Sensitivity 0.98; specificity 1.00

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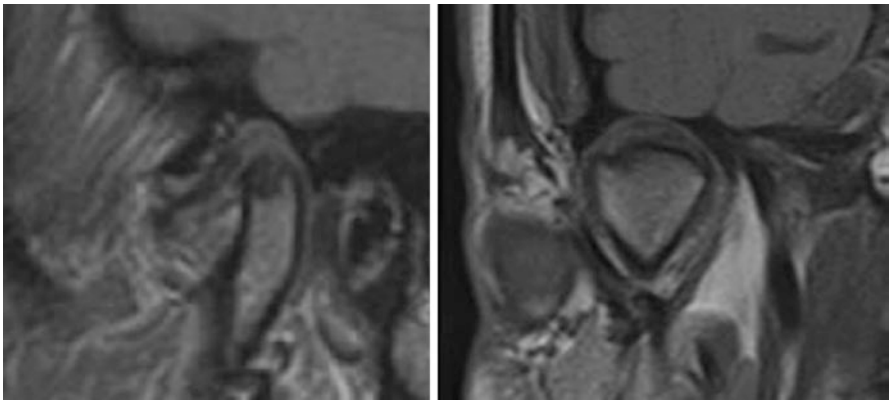
11.1.5 Joint Diseases

11.1.5.1 Degenerative Joint Diseases

1. *Osteoarthrosis/osteoarthritis* represents a degenerative disorder involving the joint characterized by deterioration of articular tissue with concomitant osseous changes in the condyle and/or articular eminence. During clinical examination crepitus is detected with palpation during at least one of the following movements: opening, closing, right or left lateral, or protrusive [6]. The difference between osteoarthrosis and osteoarthritis is the presence of arthralgia in the second one.

   Without imaging, the sensitivity is 0.55 and the specificity is 0.61 [4]. TMJ computed tomography imaging is the reference standard for this diagnosis. A rheumatologic consultation, when needed, is negative.



Degenerative joint Disease

A degenerative disorder involving the joint characterized by deterioration of articular tisue with concomitant osseous changes in the condyle and/or aticular eminence.

History of the patient must be positive for both the followingi n the past:

1. TMJ noise(s) are present in the past 30 days during jaw movement or function; ***OR***
2. Patient report any noise present during the exam;

Exam must be positive for the following:

1.Crepitus detected with palpation during at least one of the following: opening, closeing, righ or left lateral, or protrusive movement(s).

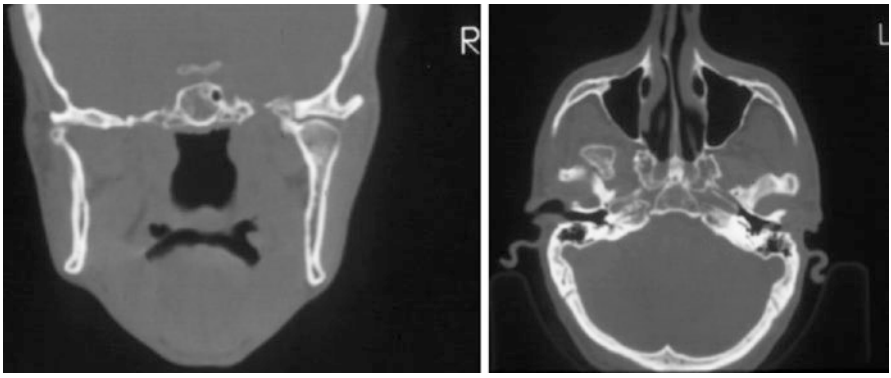
Imaging is the reference standard for this diagnosis
Without imaging: Sensitivity 0.55; specificity 0.61

Schiffman E et all, 2014

2. *Systemic arthritides*: include joint inflammation resulting in pain or structural changes caused by a generalized systemic inflammatory disease, including rheumatoid arthritis, juvenile idiopathic arthritis, spondyloarthropathies (ankylosing spondylitis, psoriatic arthritis, infectious arthritis, Reiter's syndrome), and crystal-induced disease (gout, chondrocalcinosis). Other rheumatologically related diseases that may affect the TMJ include autoimmune disorders and other mixed connective tissue diseases (scleroderma, Sjögren's syndrome, lupus erythematosus). This group of arthritides includes multiple diagnostic categories that are best diagnosed and managed by rheumatologists regarding the general/systemic therapy. Clinical signs and symptoms of ongoing chronic (TMJ) inflammation are variable among patients and often over time for a single patient. They can vary from no sign/symptom to only pain, or swelling/exudate, or tissue degradation, or growth disturbance. Resorption of condylar structures may be associated

with malocclusion such as a progressive anterior open bite. Imaging in early stages of the disease may not demonstrate any osseous findings [6].

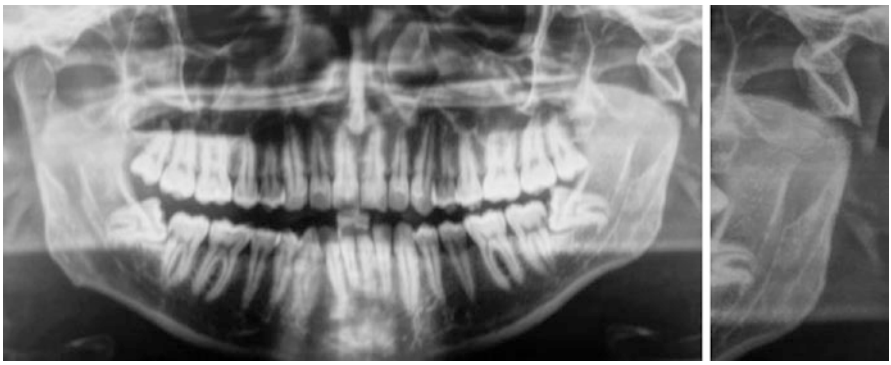
3. *Condylysis/idiopathic condylar resorption*: represents resorption of the condyles, leading to the idiopathic loss of condylar height, and a progressive anterior open bite. The condition is almost always bilateral and predominantly occurs in adolescent and young adult females. The presence of pain or articular sounds is variable. In early stages, dental occlusal changes may not be evident, but imaging findings would be positive. The cause is unknown, although it has been suggested that estrogen may be implicated [6].
4. *Osteochondritis dissecans*: represents a joint condition in which there are loose osteochondral fragments within the joint. The pathophysiology is unclear. It occurs usually in the knee and elbow and is often related to sports. The clinical presentation may be a combination of pain, swelling, joint noises, and limitation of jaw movements [6].
5. *Osteonecrosis*: represents a painful condition that is found in the mandibular condyle on MRI as decreased signal in T1-weighted or proton density images and on T2-weighted images (sclerosis pattern) and can be combined with increased signal on T2 images (edema). This condition has also been referred to in the literature as avascular necrosis (AVN) [6].
6. *Neoplasms*: result from tissue proliferation with histologic characteristics and may be benign (e.g., chondroma or osteochondroma) or malignant (e.g., primary or metastatic). They may present with swelling, pain during function, limited mouth opening, crepitus, occlusal changes, and/or sensory-motor changes. Facial asymmetry with a midline shift may occur as the lesion expands. Diagnostic imaging, typically using CT/CBCT and/or MRI, and biopsy are essential when a neoplasm is suspected [6].



7. *Synovial chondromatosis*: cartilaginous metaplasia of the mesenchymal remnants of the synovial tissue of the joint. Its main characteristic is the formation of cartilaginous nodules that may be pedunculated and/or detached from the synovial membrane becoming loose bodies within the joint space. The disease may be associated with malocclusion, such as a progressive ipsilateral posterior open bite. Imaging is needed to establish the diagnosis [6].

11.1.6 Fractures

(a) *Fracture*: includes non-displaced or displaced break in bone involving the joint (i.e., temporal bone and/or mandible). The fracture may include the cartilage. The most common is the subcondylar fracture. The condition may result in a malocclusion (e.g., contralateral posterior open bite) and impaired function (e.g., uncorrected ipsilateral deviation with opening, restricted contralateral jaw movement) and typically results from a traumatic injury.



11.1.7 Congenital/Developmental Disorders

(a) *Aplasia*: is typically a unilateral absence of condyle and incomplete development of the articular fossa and eminence, resulting in facial asymmetry. It is commonly associated with other congenital anomalies (Goldenhar syndrome, hemifacial microsomia, and Treacher Collins syndrome). It is occasionally bilateral, and in such cases, asymmetry is not present, but micrognathia is the dominant clinical manifestation. The condition may be associated with malocclusion, which may include open bite.
(b) *Hypoplasia*: is an incomplete development or underdevelopment of the mandibular condyle. It can be secondary to facial trauma, as well as the same congenital anomalies associated with aplasia. Facial asymmetry or micrognathia occurs, and the condition may be associated with malocclusion (e.g., non-horizontal occlusal plane and contralateral posterior open bite in unilateral cases or anterior open bite in bilateral cases).
(c) *Hyperplasia*: is an overdevelopment of the cranial bones or mandible. There is a non-neoplastic increase in the number of normal cells. Hyperplasia is typically unilateral as a localized enlargement such as condylar hyperplasia or as an overdevelopment of the entire mandible or side of the face.

11.2 Masticatory Muscle Disorders

11.2.1 Muscle Pain

(a) *Myalgia*: represents pain of muscle origin localized in the jaw, in the temple, in the ear, or in front of the ear that is affected by jaw movement, function, or parafunction. Replication of this pain (i.e., familiar pain) occurs with provocation testing of the masticatory muscles (i.e., muscle palpation and/or mouth opening). It has a sensitivity of 0.90 and a specificity of 0.99. The pain is not better accounted for another pain diagnosis.

Myalgia

Pain of muscle origin that is affected by jaw movement, function, or parafunction , and replication of this pain occurs with provocation testing of the masticatory muscles.

History of the patient must be positive for:
1.Pain in the jaw, temple, in the ear, or in front of ear; AND
2. pain modified with jaw movement, function or parafunction
Exam must be positive for:
1. confirmation of pain location(s) in the temporalis or masseter muscle(s); AND
2. report of familiar pain in the temporalis or masseter muscle(s) with at least one of the following provocation test:
a. palpation of the temporalis or masseter muscle(s); OR
b. maximum unassisted or assisted opening movement(s)

Sensitivity 0.90; specificity 0.99.

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There are three subclasses of myalgia: local myalgia, myofascial pain, and myofascial pain with referral [4].

Local myalgia

Pain of muscle origin as described for myalgia with localization of pain only at the site of palpation when using myofascial examination protocol.

History of the patient must be positive for:
1. pain in the jaw, temple, in the ear, or in front of ear; AND
2. pain modified with jaw movement, function or parafunction

Exam must be positive for:
1. confirmation of pain location(s) in the temporalis or masseter muscle(s); AND
2. report off amiliar pain in the temporalis or masseter muscle(s) AND
3. report of pain localized to the site of palpation

Sensitivity and specificity have not been estabilshed

Schiffman E. et all, 2014

1. *Local myalgia* is muscle soreness with pain in the masticatory muscles during function. It is usually bilateral and described as a cramp-like feeling. It has been thought to be associated with prolonged nonfunctional jaw activities, which lead to delayed-onset muscle soreness, e.g., after prolonged activation of the masticatory muscles as that exerted by masseter and temporal muscles during jaw-clenching activities. It is difficult to distinguish from other differential diagnoses of muscle pain [4].

Myofascial pain

Pain of muscle origin as described for myalgia with pain spreading beyond the site of palpation but with in the boundary of the muscle when using myofascial examination protocol.

History of the patient must be positive for:
1.Pain in the jaw, temple, in the ear, or in front of ear; AND
2. pain modified with jaw movement, function or parafunction

Exam must be positive for:
1. confirmation of pain location(s) in the temporalis or masseter muscle(s); AND
2. report of familiar pain with palpation of the temporalisor masseter muscle(s) AND
3. report of pain spreading beyond the site of palpation but with in the boundaryof the muscle.

Sensitivity and specificity have not been estabilshed

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2. *Myofascial pain* is characterized by a regional, dull, aching pain within the muscle. With respect to local myalgia, myofascial pain is characterized by pain at rest and pain aggravation with provocation of trigger points [4].

Myofascial pain with referral

Pain of muscle origin as described for myalgia with referral of pain beyond the boundary of the muscle when using myofascial examination protocol.

History of the patient must be positive for:
1.Pain in the jaw, temple, in the ear, or in front of ear; AND
2. pain modified with jaw movement, function or parafunction

Exam must be positive for:
1. confirmation of pain location(s) in the temporalisor masseter muscle(s); AND
2. report of familiar pain with palpation of the temporalis or masseter muscle(s) AND
3. report of pain at a site beyond the boundary of the muscle being palpated.

Sensitivity 0.86; specificity 0.96.

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3. *Myofascial pain with referral* is diagnosed on using only the examination findings from palpation with the palpation pressure being held over the site being palpated for 5 s compared to 2 s for myalgia and the patient reporting pain at a site beyond the boundary of the muscle being palpated [4].

(b) *Tendonitis*: is pain of tendon origin affected by jaw movement, function, or parafunction, and replication of this pain occurs with provocation testing of the masticatory tendon. Limitation of mandibular movement secondary to pain may be present. The temporalis tendon may be a common site of tendonitis and refer pain to the teeth and other nearby structures. Tendonitis could also apply to other masticatory muscle tendons. Presence of pain of masticatory muscle tendon, including the temporal tendon and the report of the familiar pain with at least one of the following tests provocation: palpation of the tendon or maximum unassisted or assisted opening [12].

(c) *Myositis:* is pain of muscle origin with clinical characteristics of inflammation or infection. Myositis causes can be divided into several categories:

1. *Inflammatory conditions*. Conditions causing inflammation throughout the body may affect the muscles, causing myositis. Many of these causes are autoimmune conditions, in which the body attacks its own tissues. Inflammatory conditions causing potentially severe myositis include dermatomyositis, polymyositis, lupus, scleroderma, and rheumatoid arthritis. Inflammatory conditions are often the most serious myositis causes, requiring long-term treatment [13].
2. *Infection*. Viral infections are the most common infections causing myositis. Rarely, bacteria, fungi, or other organisms can cause myositis as well. Viruses or bacteria may invade muscle tissue directly or release substances that damage muscle fibers. Common cold and flu viruses, as well as HIV, are just a few of the viruses that can cause myositis [14].
3. *Drugs*. Many different medications and drugs can cause temporary muscle damage. Because inflammation in the muscles is often not identified, the muscle problem may be called myopathy rather than myositis. Drugs causing myositis or myopathy include statins, colchicine, Plaquenil (hydroxychloroquine), alpha interferon, cocaine, and alcohol. Myopathy may occur right after starting a medication or may occur after taking a drug for months or years. Sometimes it is caused by an interaction between two different medications. Severe myositis caused by medications is rare [15].
4. *Trauma*. It generally arises acutely following direct trauma of the muscle.

The main symptom of myositis is muscle weakness. The weakness may be noticeable or may only be found with testing. Muscle pain (myalgias) may or may not be present. Limitation of unassisted mandibular movements secondary to pain is often present. Calcification of the muscle can occur (i.e., myositis ossificans) [16].

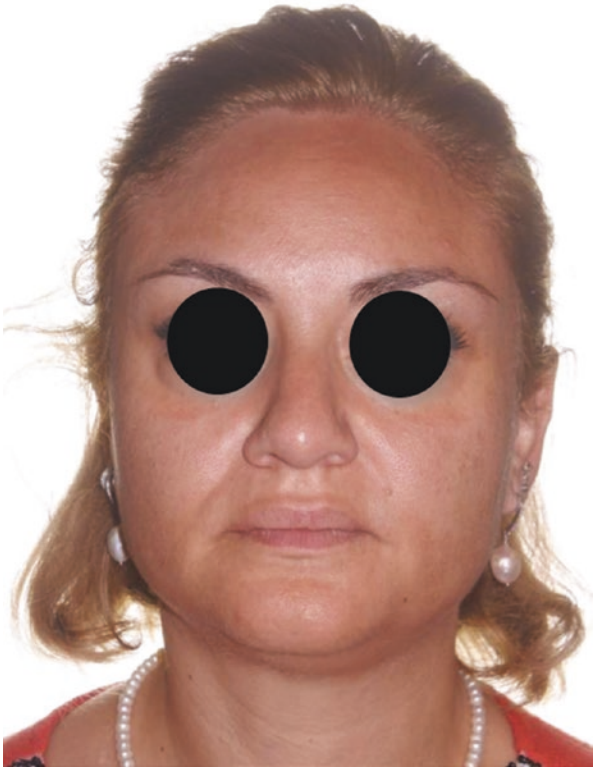
(d) *Spasm*: Muscle spasm is sudden, involuntary, reversible tonic contraction of a muscle that can cause a great deal of pain. Spasm may affect any of the masticatory muscles. Acute malocclusion may be present. It can cause immediate report of limited range of the yaw motion.

11.2.2 Contracture

Contracture: is the shortening of a muscle due to fibrosis of tendons, ligaments, or muscle fibers. It is usually not painful unless the muscle is overextended. A history of radiation therapy, trauma, or infection is often present. It is more commonly seen in the masseter or medial pterygoid muscle [12].

11.2.3 Hypertrophy

Hypertrophy: is an enlargement of one or more masticatory muscles usually not associated with pain and can be secondary to overuse and/or chronic tensing of the muscle(s). Some cases are familial or genetic in origin. Diagnosis is based on clinician assessment of muscle size and needs consideration of craniofacial morphology and ethnicity [12].



11.2.4 Neoplasms

Neoplasms: result from tissue proliferation with histologic characteristics and may be benign (e.g., myoma) or malignant (e.g., rhabdomyosarcoma or metastatic). They are uncommon. They may present with swelling, spasm, pain during function,

limited mouth opening, and/or sensory/ motor changes (e.g., paresthesia, weakness). Diagnostic imaging, typically using CT/CBCT and/or MRI, and biopsy are essential when a neoplasm is suspected [12].

11.2.5 Movement Disorders

1. *Orofacial dyskinesia or Tardive dyskinesia:* is an involuntary, mainly choreatic, (dance-like) movements that may involve the face, lips, tongue, and/or jaw. In most cases, they occur in older psychotic patients who are in institutions and in whom long-term treatment with antipsychotic drugs of the phenothiazine and butyrophenone groups is being carried out. These dyskinesias are frequent in occurrence and characteristically are irreversible. Several biochemical mechanisms have been proposed as causes, including hypersensitivity or partially enervated brain dopamine receptors and low affinity of the offending drugs for brain muscarinic cholinergic receptors. Clinical therapy has been attempted primarily with drugs that antagonize dopamine receptors or deplete brain dopamine. The benefits of drug treatment have been variable, and lack of consistent improvement has been discouraging. Early recognition of dyskinesia should be attempted and the dose reduced or the drug omitted at the first sign.
2. *Oromandibular dystonia* is a focal dystonia characterized by forceful contractions of the face, lips, jaw, and/or tongue causing difficulty in opening and closing the mouth, often affecting chewing and speech. Oromandibular dystonia symptoms usually begin later in life, between the ages of 40 and 70 years, and appear to be more common in women than in men [17]. Another word used to describe dystonia of this kind is cranial dystonia. Cranial dystonia is a broad description for dystonia that affects any part of the head. Dystonia that affects the facial muscles and lips of musicians who play wind instruments is called embouchure dystonia. Dystonia that specifically affects the tongue is called lingual dystonia. Oromandibular dystonia may be primary (meaning that it is the only apparent neurological disorder, with or without a family history) or be brought about by secondary causes such as drug exposure or disorders such as Wilson's disease. Oromandibular dystonia is often associated with dystonia of the neck muscles (cervical dystonia/spasmodic torticollis), eyelids (blepharospasm), or larynx (spasmodic dysphonia). The combination of upper and lower dystonia is sometimes called cranial-cervical dystonia. Sometimes symptoms of oromandibular are task-specific and occur only during activities such as speaking or chewing. Paradoxically, in some people, activities like speaking and chewing reduce symptoms. Difficulty in swallowing is a common aspect

of oromandibular dystonia if the jaw is affected, and spasms in the tongue can also make it difficult to swallow. Drug-induced dystonia often manifests as symptom in the facial muscles. Secondary oromandibular dystonia may persist during sleep [18].

11.2.6 Masticatory Muscle Pain Attributed to Systemic/Central Pain Disorders

1. *Fibromyalgia:* Fibromyalgia is a disorder characterized by widespread musculoskeletal pain accompanied by fatigue, sleep, memory, and mood issues. Researchers believe that fibromyalgia amplifies painful sensations by affecting the way your brain processes pain signals. It is a widespread pain condition with concurrent masticatory muscle pain.

11.3 Headache

11.3.1 Headache Attributed to TMD

Headache is one of the most common painful conditions; few people are spared during their lifetime by at least one episode of headache: it is estimated that about 90% of the general population in a year suffer from at least a headache episode [19]. Temporomandibular disorders (TMDs) and headache are closely related pathologies; prevalence of headache in the dysfunctional population varies between 48% and 77%, while in the general population, the prevalence of headache is around 45% [20–22]. Primary headaches as migraine, ETTH (episodic tension-type headaches), and CDH (chronic daily headaches) are more common in patients with TMD symptoms compared to individuals without headache [23]. According to several studies, there is a strong correlation between headache and other dysfunctional symptoms, such as joint noise, pain during mandibular movement, pain in the temporomandibular area, depression, anxiety, and poor sleep quality [24].

Headache attributed to TMD represents headache in the temple area secondary to pain-related TMD that is affected by jaw movement, function, or parafunction. Replication of this headache (i.e., familiar headache) occurs with provocation testing of the masticatory system (i.e., palpation of temporalis muscle and/or mouth opening) [4]. “Headache attributed to TMD” is included as a new disorder type to replace “headache or facial pain attributed to temporomandibular joint (TMJ) disorder” as described in the International Classification of Headache Disorders II (ICHD-2) and has been incorporated into the beta version of the ICHD-3, however, with minor discrepancies in the exact criteria [25].

Headache attributed to TMD

A Headache in the temple area secondary to pain-related TMD that is affected by jaw movement, function, or parafunction, and replication of this headache occurs with provocation testing of the masticatory system.

Historyofthe patient must be positive for both the following:
1. Headache of any type in the temple; AND
2. Headache modifed with jaw movement, function or parafunction..

Exam must be positive for:
1. Confirmation of headache location in the area ofthe temporalis muscle(s): AND
2. Report of familiar headache in the templearea with at least one of the following provocation tests:
A. Palpation of the temporalis muscle(s): OR
B. Maximum unassisted or assisted opening, right or left lateral, or protusive movement(s)

Sensitivity 0.89; specificity 0.87

The headache is not better accounted for another headache diagnosis
A diagnosis of pain-related TMD (eg, myalgiaor TMJ arthralgia) must be present and is established using valid diagnostic criteria.

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Headache attributed to temporomandibular disorder (TMD) is usually most prominent in the preauricular areas of the face, masseter muscles, and/or temporal regions. Pain generators include disc displacements, joint osteoarthritis, joint hypermobility, and regional myofascial pain. Headache attributed to temporomandibular disorder (TMD) tends to be unilateral when the temporomandibular complex is the generator of pain but may be bilateral when muscular involvement is present. Pain referral to the face is common.

There is some overlap between headache attributed to temporomandibular disorder (TMD) due to muscular tension and tension-type headache. When the diagnosis of TMD is uncertain, the headache should be coded as tension-type headache or one of its subtypes (presumably with pericranial muscle tenderness).

11. Headache or facial pain attributed to disorder of the cranium, neck eyes, ears, nose sinuses, teeth, mouth or facial or cervical structure

11.7 Headache attributed to temporomandibulardisorder (TMD)
Description:
Headache caused by a disorder involving structures in the temporomandibularregion.

Diagnostic criteria:
A. Any headache fulfilling criterion C

B. Clinical and/or imaging evidence of a pathological process affecting the temporomandibularjoint (TMJ), muscles of mastication and/or or associated structures

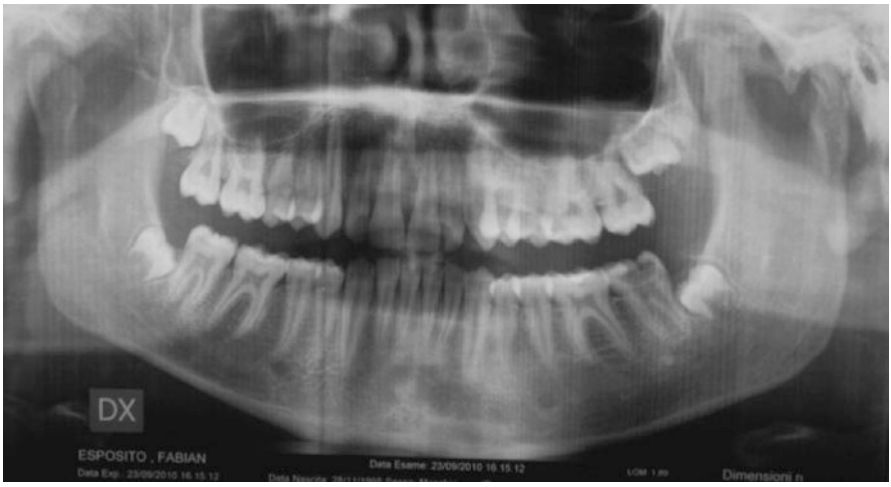
C. Evidence of causation demonstrated by at least two of the following:
1. headache has developed in temporal relation to the onset of the temporomandibulardisorder
2. either or both of the following:
   a) headache has significantly worsened in parallel with progression of the temporomandibulardisorder
   b) headache has significantly improved or resolved in parallel with improvement in or resolution of the temporomandibulardisorder
3. the headache is produced or exacerbated by active jaw movements, passive movements through the range of motion of the jaw and/or provocative manœuvresapplied to temporomandibularstructures such as pressure on the TMJ and surrounding muscles of mastication
4. headache, when unilateral, is ipsilateralto the side of the temporomandibulardisorder

D. Not better accounted for by another ICHD-3 diagnosis.

HIS CLASSIFICATION ICHD-3 BETA

11.4 Associated Structures

(a) *Coronoid hyperplasia*: represents a progressive enlargement of the coronoid process that impedes mandibular opening when it is obstructed by the zygomatic process of the maxilla [6].



It is also a rare cause of mouth opening limitation. The hyperplasia can be a consequence of trauma, but sometimes the bilateral condition begins spontaneously about puberty. Bilateral coronoid hyperplasia is a relatively rare condition in the pediatric population and yet may be an unrecognized cause of limited mouth opening in children [26]. The patients maximal interincisal mouth opening varied from 10 to 15 mm [27].

References

1. Schiffman E, Ohrbach R. Executive summary of the Diagnostic Criteria for Temporomandibular Disorders for clinical and research applications. JADA. 2016;147(6):438–45.
2. List T, Jensen RH. Temporomandibular disorders: old ideas and new concepts. Cephalalgia. 2017;37(7):692–704.
3. Ohrbach R, Gonzales Y, List T, Michelotti A, Schiffmann E. Diagnostic criteria for temporomandibular disorders (DC/TMD). Clinical examination protocol. 2014: protocol website in IADR INfORM.
4. Schiffman E, Ohrbach R, Truelove E, Look J, Anderson G, Goulet JP, List T, Svensson P, Gonzalez Y, Lobbezoo F, Michelotti A, Brooks SL, Werner E, Ceusters W, Drangsholt M, Ettlin D, Gaul C, Goldberg LJ, Haythornthwaite JA, Hollender L, Jensen R, John MT, De Laat A, de Leeuw R, Maixner W, van der Meulen M, Murray GM, Nixdorf DR, Palla S, Arne Petersson A, Pionchon P, Smith B, Visscher CM, Zakrzewska J, Dworkin SF. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: Recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Group. J Orofac Pain. 2014;28(1):6–27.
5. Ohrbach R. Assessment and further development of RDC/TMD axis II biobehavioural instruments: a research programme progress report. J Oral Rehabil. 2010;37:784–98.
6. Peck C, Goulet JP, Lobbezoo F, Schiffman EL, Alstergren P, Anderson GC, De Leeuw R, Jensen R, Michelotti A, Orbach R, Petersson A, List T. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. J Oral Rehabil. 2014;41:2–23.
7. Michelotti A, Alstergren P, Goulet JP, Lobbezoo F, Ohrbach R, Peck C, Schiffmann E, List T. Next steps in development of the diagnostic criteria for temporomandibular disorders (DC/TMD): recommendations from the International RDC/TMD Consortium Network workshop. J Oral Rehabil. 2016;43(6):453–67.
8. Alstergren P, Kopp S, Theodorsson E. Synovial fluid sampling from the temporomandibular joint: sample quality criteria and levels of interleukin-1 beta and serotonin. Acta Odontol Scand. 1999;57(1):16–22.
9. Nitzan DW, Marmary Y. The "anchored disc phenomenon": a proposed etiology for sudden-onset, severe, and persistent closed lock of the temporomandibular joint. J Oral Maxillofac Surg. 1997;55:797–802.
10. Yura S, Totsuka Y, Yoshikawa T, Inoue N. Can arthrocentesis release intracapsular adhesions? Arthroscopic findings before and after irrigation under sufficient hydraulic pressure. J Oral Maxillofac Surg. 2003;61:1253–6.
11. Nitzan DW, Etsion I. Adhesive force: the underlying cause of the disc anchorage to the fossa and/or eminence in the temporomandibular joint—a new concept. Int J Oral Maxillofac Surg. 2002;31:94–9.
12. Bakke M. Iaw muscle disorder. In: Klineberg I, Eckert S, editors. Functional occlusion in restorative dentistry and prosthodontics. St Louis: Elseiver Mosby; 2015. p. 173–86.
13. Plotz P, Christopher-Stine L. Adult inflammatory myopathies. Best Pract Res Rheumatol. 2004;18(3):331–44.

14. Firestein G. Kelley's textbook of rheumatology. Philadelphia: W.B. Saunders Company; 2008.
15. Gold R, Dalakas MC, Toyka KV. Immunotherapy in autoimmune neuromuscular disorders. Lancet Neurol. 2003;2(1):22–32. Review 2003.
16. Hansen KE, Hildebrand JP, Ferguson EE, Stein JH. Outcomes in 45 patients with statin-associated myopathy. Arch Intern Med. 2005;165(22):2671–6.
17. Geyer HL, Bressma S. Diagnosis of dystonia. In: Warner T, Bressma S, editors. Clinical diagnosis and management of dystonia. London: Informa Healthcare; 2007. p. 1–14.
18. Thompson P, Obeso J, Delgado G, Gallego J, Marsden C. Focal dystonia of the jaw and differential diagnosis of unilateral jaw and masticatory spasm. J Neurol Neurosurg Psychiatry. 1986;49:651–6.
19. Zanchin G, Maggioni F. Cefalee e Nevralgie. In: Angelini C, Battistin L, editors. Neurologia Clinica. Bologna: Società Editrice Esculapio; 2014. p. 542–79.
20. Glaros A, Urban D, Locke J. Headache and temporomandibular disorders: evidence for diagnostic and behavioural overlap. Cephalalgia. 2007;27:542–9.
21. Mitrirattanakul S, Merrill L. Headache impact in patients with orofacial pain. J Am Dental Assoc. 2006;137:1267–74.
22. Stovner L, Hagen K, Jensen R. The global burden of headache: a documentation of headache prevalence and disability worldwide. Cephalalgia. 2007;27:193–210.
23. Gonçalves D, Bigal M, Jales L, Camparis M, Speciali J. Headache and symptoms of temporomandibular disorder: an epidemiological study: research submission. Headache. 2010;50:231–41.
24. Caspersen N, Hirsvang J, Kroell L. Is there a relation between tension-type headache, temporomandibular disorders and sleep? Pain Res Treat. 2013;2:845684–6.
25. Olesen J. ICHD-3 beta. Cephalalgia. 2013;33:629–808.
26. Jaskolka MS, Eppley BL, van Aalst JA. Mandibular condylar hyperplasia in pediatric patients. J Craniofac Surg. 2007;18:849–54.
27. Khandavilli SD, Pattni N, Naredla PR, Williams R. First case of bilateral coronoid hyperplasia in monozygotic twin sisters—a new aetiological perspective? Oral Maxillofac Surg. 2016;20(4):441–3.