Sanjivan Kandasamy
Charles S. Greene
Donald J. Rinchuse
John W. Stockstill
Editors

TMD and Orthodontics

A Clinical Guide for the Orthodontist

Springer

TMD and Orthodontics

Sanjivan Kandasamy • Charles S. Greene
Donald J. Rinchuse • John W. Stockstill
Editors

TMD and Orthodontics

A Clinical Guide for the Orthodontist

Springer

Editors
Sanjivan Kandasamy
Department of Orthodontics
School of Dentistry
University of Western Australia
Nedlands, WA
Australia

Centre for Advanced Dental Education
Saint Louis University
Saint Louis, MO
USA

Charles S. Greene
University of Illinois
College of Dentistry
Chicago, Illinois
USA

Donald J. Rinchuse
Greensburg, Pennsylvania
USA

John W. Stockstill
Seton Hill University
Greensburg, PA
USA

ISBN 978-3-319-19781-4 ISBN 978-3-319-19782-1 (eBook)
DOI 10.1007/978-3-319-19782-1

Library of Congress Control Number: 2015945966

Springer Cham Heidelberg New York Dordrecht London

Printed on acid-free paper

Springer International Publishing AG Switzerland is part of Springer Science+Business Media (www.springer.com)

This book is dedicated to our troops who sacrifice their lives to provide us with the freedom to study, teach and live freely. They set the foundation and example for clinicians around the world who strive on a daily basis to provide essential evidence-based care to their patients. We hope our book will enable them to accomplish these important goals as they deal with patients in pain.

Foreword

The writing of forewords is something of a cottage industry for retired academics. Regardless of the subject, a few laudatory, largely honest paragraphs are usually easy to craft. A book on TMD for orthodontists, however, has to be approached with caution. My concern about this invitation involves more than an aversion to books for dentists who seek some sort of complex mechanical perfection, seemingly to compensate for childhood difficulties with toilet training. Unfortunately, temporomandibular dysfunction is the red-headed stepchild of many healing arts. Everything seems to work, at least for a while. No wonder so many professions, specialties, and splinter groups claim to be keepers of the flame.

As I write these words, chances are that someone, somewhere is crafting a book on TMD for homeopaths or cranial manipulators or chiropractors or naturopaths, world without end. When challenged with inconvenient evidence (an irritating distraction from their mission of "helping people"), true-believers take refuge in the fact that people are said also to have laughed at Pasteur or Freud or Einstein. True, but most often people laughed at Bozo the Clown.

Given the murky nature of the field, involvement with a TMD book must be approached with care. Does it have a strong evidentiary basis? Is it written by recognized authorities? Are its recommendations consistent with treatments for other types of chronic pain? In the present instance, the answer to these questions is a resounding, reassuring yes! The editors have recruited respected authorities to provide a thorough, evidence-based survey of the various interactions between orthodontics and TMD. Indeed, the authors and editors of this concise but thorough book are the people to whom I look for rational guidance. For example, I once heard Chuck Greene put the problem into perspective with a single short sentence: "TMD is reported; it isn't discovered." Suffice it to say, I am honored to participate in the publication of this meticulous compilation. It is both an antidote for the thought-crime of the past and a rational, evidence-based survey for the present and the foreseeable future of our specialty. Well done!

Lysle E. Johnston Jr., DDS, MS, PhD, FDS RCS, FACD, FICD
Eastport, Michigan, USA

Preface

The orthodontic profession has had long-standing interests in the temporomandibular joint (TMJ). Beginning with the need to understand how the masticatory system develops, all orthodontists can expect to encounter a variety of clinical issues in their practices involving this important joint and the many issues that have evolved related to the TMJs. It is therefore essential that orthodontists should keep up to date on the most current scientific evidence related to these topics.

One major issue that is of concern is the patient who has a temporomandibular disorder (TMD). That patient may present to the orthodontist as a referral from a colleague, with the request for orthodontic treatment as a way to resolve this problem. Alternatively, an orthodontic patient may suddenly develop TMD symptoms during treatment or may return with such complaints following treatment. All of these scenarios demand an appropriate response from the orthodontist, and the nature of that response has changed as new research and data have emerged in the TMD field in the past 25 years.

It is the purpose of this book to bring together a group of experts who are internationally recognized leaders in their field. These experts have come from within and outside the orthodontic profession to address all of the salient topics about orthodontics and the TMDs. We have been extremely fortunate to have several outstanding colleagues join us on this project. This is the first book of its kind to focus exclusively on orthodontics, the TMJs and TMDs, and it is organized to deliver the latest evidence-based information in the ever-changing controversial world of temporomandibular disorders and orofacial pain. Rather than burdening the reader with highly specific detail and basic sciences that can be obtained elsewhere, each chapter is written with a clinical perspective and ends with a large number of useful references. Clinicians, orthodontic residents and faculty will all find this book to be an extremely useful resource providing much needed clarity in an area filled with a great deal of misinformation and confusion.

Sanjivan Kandasamy
Charles S. Greene
Donald J. Rinchuse
John W. Stockstill

Contents

Contributors

Ramesh Balasubramaniam, BDSc, MS, FOMAA School of Dentistry, University of Western Australia, Crawley, WA, Australia

Private Practice, West Leederville, WA, Australia

Charles S. Greene, BS, DDS Department of Orthodontics, University of Illinois at Chicago, College of Dentistry, Chicago, IL, USA

Chester S. Handelman, DMD Department of Orthodontics, University of Illinois at Chicago, College of Dentistry, Chicago, IL, USA

L. Jerrold, DDS, JD NYU-Lutheran Medical Center, Department of Dental Medicine, Division of Orthodontics, Brooklyn, NY, USA

Sanjivan Kandasamy, BDSc, DClinDent, MOrthRCS, FRACDS Department of Orthodontics, School of Dentistry, University of Western Australia, Nedlands, WA, Australia

Centre for Advanced Dental Education, Saint Louis University, Saint Louis, MO, USA

Private Practice, Midland, WA, Australia

Gary D. Klasser, DMD, Cert Orofacial Pain Department of Diagnostic Sciences, Louisiana State University Health Sciences Center, School of Dentistry, New Orleans, LA, USA

D.M. Laskin, DDS, MS Department of Oral and Maxillofacial Surgery, Virginia Commonwealth University, School of Dentistry, Richmond, VA, USA

D. Manfredini, DDS, MSc, PhD Italian Minister of University and Instruction, Padova, Italy

Department of Maxillofacial Surgery, University of Padova, Padova, Italy

Louis G. Mercuri, DDS, MS Department of Orthopedic Surgery, Rush University Medical Center, Chicago, IL, USA

Ambra Michelotti, DDS, BSc, Orthod. Section of Orthodontics, Department of Neuroscience, University of Naples Federico II, Naples, Italy

Norman D. Mohl, DDS, MA, PhD SUNY Distinguished Service, Department of Oral Diagnostic Sciences, University at Buffalo School of Dental Medicine, Buffalo, NY, USA

Richard Ohrbach, DDS, MS, PhD Department of Oral Diagnostic Sciences, University at Buffalo School of Dental Medicine, Buffalo, NY, USA

Jeffrey P. Okeson, DMD Department of Oral Health Science, Orofacial Pain Program, College of Dentistry, University of Kentucky, Lexington, KY, USA

Donald J. Rinchuse, DMD, MS, MDS, PhD Private Practice, Greensburg, PA, USA

John W. Stockstill, DDS, MS Department of Orthodontics, Temporomandibular Disorders/Orofacial Pain, Seton Hill University, Center for Orthodontics, Greensburg, PA, USA

Center for Orthodontics, Graduate Orthodontics Residency Program, Seton Hill University, Greensburg, PA, USA

Static and Functional Anatomy of the Human Masticatory System

1

John W. Stockstill and Norman D. Mohl

1.1 Occlusal Concepts and Terminology

1.1.1 Review of Occlusion Concepts and Definitions

In accordance with the primary intent of this book, part one of this chapter will address the basic and common occlusal concepts and terminology used in dental practice in general, and orthodontic practice in particular. Because so much of the controversy about temporomandibular disorders (TMDs) revolves around these occlusal concepts, the authors will address those relationships wherever appropriate. This also will set up the framework for similar discussions in other chapters throughout this book. Due to the many controversies and "philosophical" explanations reported in the literature regarding occlusal concepts, our intent will be to enlighten the reader rather than to argue about belief systems. Evidence-based explanations and definitions will be offered rather than teleological explanations, and the emphasis will be on physiology rather than philosophy.

According to the *Textbook of Occlusion (Mohl et al. 1988)*, the scope of the subject of occlusion relative to dentistry includes "the relationship between all the components of the masticatory system in normal function, dysfunction, and parafunction, including the morphological and functional features of contacting surfaces of opposing teeth and restorations, occlusal trauma and dysfunction, neuromuscular physiology, the temporomandibular joints and muscle function, swallowing and mastication, psychophysiological status, and the diagnosis, prevention, and treatment of functional disorders of the masticatory system" [1, 2]. Thirty-seven variations of the term "dental occlusion" are found in *Dorland's Illustrated Medical Dictionary, 32nd edition (2012)*, and most or all represent commonly used (often incorrectly) terms for the relationship of the teeth to their environment and to one another specifically [3]. The intent of this section is to offer standardized terminology that satisfies "… the school of thought that is promulgated (as) one that has some basis in objective scientific inquiry or is at least within the mainstream of current thought within dentistry" [2]. It does not represent any particular point of view; instead, it has been framed within the boundaries of

J.W. Stockstill, DDS, MS (✉)
Department of Orthodontics, Temporomandibular Disorders/Orofacial Pain, Seton Hill University, Center for Orthodontics, 2900 Seminary Drive, Building E, Greensburg, PA 15601, USA
e-mail: jstockstill@setonhill.edu

N.D. Mohl, DDS, MA, PhD
Department of Oral Diagnostic Sciences, SUNY Distinguished Service, University at Buffalo-School of Dental Medicine, Buffalo, NY, USA

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_1

evidence-based scientific thinking and clinical application. Controversies regarding the subject of dental occlusion are numerous, and it is not the interest or responsibility of this publication to act as the "decider" for any one of these "schools" or "beliefs."

In a recent publication entitled "Understanding Occlusion" [4], the concepts of occlusion and functional movement of the mandible were described as being confusing and resulting in frustration to the dental profession, but the three "experts" who were interviewed for this article seemed to agree that common ground was available for discussing the three most common occlusal philosophies. These occlusal philosophies include (1) conformational occlusion, (2) neuromuscular-based occlusion, and (3) joint-based occlusion. Briefly, the concept of conformational occlusion holds that one should allow the patient's mandible to function in whatever occlusal scheme they have and with which they are comfortable. The neuromuscular-based occlusion concept theorizes that there is an ideal occlusal position that is determined using electromyography and muscle stimulation devices in order to achieve "muscular physiologic harmony." The third occlusal concept, often referred to as gnathology, implies that the condyle-fossa relationships must be ideal and that occlusal contacts during excursions of the mandible should be in harmony with condylar movements. The ideal temporomandibular joint (TMJ) relationship is generally described as "centric relation (CR)," but it should be noted that this position has been redefined several times over the years.

In any case, these authors agree that there are no clear criteria for deciding which philosophy one must use to "build a healthy masticatory system." But it is interesting to note that, in spite of there being "philosophical differences" between these pronounced theories, "Without scientific evidence, it has not been proved definitely that treatment planning with any one philosophy is better than using the patient's own occlusion" [4]. Therefore, it may be said that the "physiological evidence trumps the philosophical belief" in every instance. Theories aside, the terms and concepts which follow are universally accepted and considered "gold standards" when discussing, diagnosing, and managing the human dentition and occlusion, and also when discussing the static and functional anatomy and biomechanics of the masticatory system [2, 5–8].

1.1.2 Terms and Concepts

Centric Occlusion (Maximum Intercuspation, Habitual Occlusion, Intercuspal Position) The position of the mandible when the relationship of opposing occlusal surfaces provides for maximum planned contact and/or intercuspation. This is a *tooth-determined position.*

Centric Relation Occlusion (Retruded Contact Position, RCP) Is defined as the occlusion of the teeth when the mandible is in centric relation. This is a *tooth-joint determined position.*

Centric Relation (CR) The relationship of the mandible to the maxillae when the mandibular condyles are in their most superior position, with the central bearing area of the articular discs in contact with the articular surface of the condyles and with the articular eminentia. Importantly, the condyles may or may not be in their most retruded position, depending on the degree of restraint provided by the TM ligament. This position is independent of tooth contact and is determined by the structural features of the temporomandibular joint and not the dentition.

Malocclusion Any occlusion in which the structural characteristics are beyond those established for a theoretically ideal occlusion. The term does not necessarily imply that such an occlusion is nonphysiologic or that therapy is indicated. The presence of a malocclusion, particularly in adults, does not mean that therapy is necessary, and the malocclusion may be physiologic.

Physiologic Occlusion Usually in adults, it is an occlusion that deviates in one or more ways from the theoretically ideal, yet is well adapted to that particular environment, is esthetically pleasing to

the patient, and has no pathological manifestations or dysfunctional problems. It does not require intervention.

Nonphysiologic Occlusion An occlusion which presents with signs or symptoms of pathology, dysfunction, or inadequate adaptation of one or more components of the masticatory system that can be attributed to faulty structural relationships or mandibular functional activity. Therapy to improve the malocclusion may be indicated.

Therapeutic Occlusion An occlusion that has been modified by appropriate therapeutic modalities in order to change a nonphysiologic occlusion to one that falls within the parameters of a physiologic occlusion, if not a theoretically ideal occlusion. This occlusion optimizes the health and adaptive potential of the masticatory system.

Theoretically Ideal Occlusion A preconceived theoretical concept of occlusal structural and functional relationships that includes idealized principles and characteristics that an occlusion should have. It does not represent the "norm" and is used as a series of idealized parameters against which variations may be compared.

Muscular Contact Position (MCP) The position of the mandible when it has been raised by voluntary muscular effort to initial occlusal contact with the head erect. This position is consistent with the intercuspal position (CO) in asymptomatic individuals.

Occlusal Vertical Dimension The vertical dimension of the face as determined by a midline vertical measurement of the face between two arbitrary points above and below the mouth when the mandible is in centric occlusion. By convention, vertical dimension is interchangeable with occlusal vertical dimension.

Postural Rest Position The "resting" position of the mandible when an individual is sitting or standing in an upright position. This position is determined by muscles and other structures. A minimal amount of elevator muscle activity is needed to maintain the mandible in this position.

Rest Vertical Dimension The vertical dimension of the face when the mandible is in postural rest position.

Interocclusal Distance The distance (commonly 2–4 mm) between the occluding surfaces of the maxillary and mandibular teeth when the mandible is in postural rest position. It is also referred to as freeway space and is routinely considered to be a space that is "best fit" or averaged throughout the occlusal plane.

1.1.3 Mandibular Movement Terminology

Disclusion The loss of occlusion (nonocclusion) between opposing teeth during tooth-guided movements of the mandible. For example, when the anterior teeth are in an edge-to-edge position, the posterior teeth are said to be in disclusion. The term is appropriate only when some degree of dental contact is occurring.

Hinge Movement Movement in space characterized by two divergent points moving around a central axis of rotation.

Hinge Axis An imaginary line between the mandibular condyles around which the mandible can rotate without translatory movement. This is also referred to as transverse hinge axis.

Translatory Movement Movement in space characterized by linear motion with no axis of rotation. This movement may follow a straight path (rectilinear translation) or a curved path (curvilinear translation).

Protrusion Movement of the mandible forward or in an anterior direction from centric occlusion with an anterior translation of both condyles, either with or without occlusal contacts.

Retrusion Retraction or posterior movement of the mandible from any given point.

Lateral Excursion Sideward movement of the mandible from a median occlusal position, and characterized by a forward, inward, and downward translation of the contralateral condyle. Left lateral excursion results in the left condyle rotating about an axis and the right condyle translating forward, inward, and downward as it tracks the medial wall of the glenoid fossa.

Working Side The lateral segment of the dentition toward which the mandible moves during lateral excursion (functional or ipsilateral or laterotrusive side). Left lateral movement of the mandible results in the left side dentition being designated as the working side and the right side dentition being designated as the nonworking side.

Nonworking Side The side opposite the working side during lateral excursive movement of the mandible (nonfunctioning or contralateral or balancing or mediotrusive side). Left lateral movement of the mandible results in the right side dentition being designated as the nonworking side.

Condylar Guidance The influence on mandibular movements by the direction of condylar movement during translation of the condyles as determined by the anatomical features of the temporomandibular joint. For example, the height and amount of convexity of the articular eminences will dictate the degree of downward movement of the condyles during forward translation (protrusive movement).

Condylar Inclination That part of the condylar guidance formed by the inclination of the condyle path as it translates forward and downward on the articular eminence.

Condylar Angulation That part of the condylar guidance formed by the angulation of the nonworking side condyle path as it translates forward and inward during lateral excursion and tracks the medial wall of the glenoid fossa.

Anterior Guidance The influence on mandibular movements by the relative overlap of the anterior teeth as determined by the lingual surfaces of the maxillary anterior teeth and the incisal edges or labial surfaces of the mandibular anterior teeth. This movement is influenced by the horizontal overlap (overjet) and vertical overlap (overbite) of the anterior teeth. An anterior open bite or negative overlap will reduce or eliminate the downward movement of the mandible during protrusive movement.

Incisal Guidance That part of the anterior guidance that occurs during protrusive movements of the mandible and is influenced by the relative overlap, position, and anatomy of the maxillary and mandibular incisors.

Canine Guidance That part of the anterior guidance that occurs during lateral excursion of the mandible and is influenced by the relative overlap, position, and anatomy of the maxillary and mandibular canines on the working side.

1.1.4 Dental Definitions and Concepts

Plane of Occlusion An imaginary surface that is related anatomically to the cranium and that theoretically touches the incisal edges of the incisors and the tips of the occluding surfaces of the posterior teeth. It is *NOT* a plane in the true sense of the word and represents the mean or best fit of the curvature surface.

Compensating Curve The curvature of the alignment of the occlusal surfaces of the teeth that is present to compensate for the curved movement patterns of the mandible (Monson curve). When viewed laterally, it is referred to as the curve of Spee, and when viewed in the frontal plane, as the curve of Wilson.

Overbite The extension of the maxillary teeth over the mandibular teeth in a vertical direction when the opposing posterior teeth are in

contact in centric occlusion (vertical overlap – see anterior guidance).

Overjet The projection of the maxillary anterior and/or posterior teeth beyond their protagonists in a horizontal direction when the mandible is in centric occlusion (horizontal overlap – see anterior guidance). Anterior guidance is greatly influenced by the relative amounts of overbite and overjet.

1.1.5 Occlusion Concepts and Definitions

Mutually Protected Occlusion An occlusion that provides for maximum occlusal contact on certain teeth or groups of teeth whereas other teeth have light contact or are in disclusion during centric occlusion or excursive movements of the mandible. That is, in CO, the posterior teeth provide maximum occlusal loading and "protect" the anterior teeth from heavy loading. In protrusive movement, the anterior teeth occlude with resulting disclusion or protection for the posterior teeth. On lateral excursive movement of the teeth, working side teeth contact and provide disclusion or protection for the nonworking side teeth.

Canine Protected Occlusion A modification of the mutually protected occlusion in which the canine teeth on the working side serve to disclude all other teeth during lateral excursion of the mandible.

Group Function Occlusion A modification of the mutually protected occlusion in which the canines and one or more adjacent pairs of posterior teeth on the working side are in simultaneous occlusal contact during lateral excursion of the mandible.

Balanced Occlusion An occlusion in which balanced and equal contacts are maintained throughout the entire arch during all excursion of the mandible. This implies simultaneous occlusal contact on both working and nonworking sides during lateral excursion (cross-arch balance).

Angle's "Normal Occlusion" An occlusion in which the mesiobuccal cusp of the maxillary molar occludes in the buccal groove of the mandibular molar AND the teeth are arranged along a smoothly curving line of occlusion.

Angle's Class I Malocclusion An occlusion in which there is a normal relationship of the molars but the line of occlusion is "incorrect" because of malposed teeth, tooth rotations, or other causes.

Angle's Class II Malocclusion An occlusion in which the mandibular molar is distally positioned relative to the maxillary molar, and the line of occlusion may or may not be correct (unspecified).

Angle's Class III Malocclusion An occlusion in which the mandibular molar is mesially positioned relative to the maxillary molar, and the line of occlusion may or may not be correct (unspecified).

The references cited in Part 1 are available at the end of this chapter.

1.2 Masticatory System Anatomy: Concepts and Terminology

Part two of this chapter will address the basic static and functional anatomy of the masticatory system in the context of the most significant clinical issues or problems that orthodontists face when caring for patients who either present with TMD problems, or who develop such problems during their orthodontic treatment. Among these are muscular pain and dysfunction problems, oral habits, joint pain, disc derangements, arthritides, and other pathologies of the TMJ and masticatory system. These important anatomy topics will underlie the contents of this chapter and others in this book relative to the importance of pre-orthodontic screening histories and examinations for the presence of such problems. They also provide the anatomical characteristics and biomechanical principles pertaining to the generalized

masticatory system and temporomandibular joints so essential for performing differential diagnosis using evidence-based approaches.

1.2.1 Masticatory System

The masticatory system is a highly complex system primarily involved in speaking, mastication, swallowing, and functional and parafunctional movement of the mandible. Embryological origin is primarily from first branchial arch with secondary input and influences from the second, third, and fourth branchial arches (diminishing influence from second to fourth). It consists of dental, skeletal, muscular, joint, ligamentous, and tendinous components that operate in highly coordinated, neurosensory-modulated movements essential for nourishment, respiration, and social interaction [14]. Developmental breakdowns in the structures of the masticatory system due to genetic defects, physical trauma, and/or nutritional stressors may result in incomplete or defective growth and development of the head and cervical region, commonly referred to as craniofacial anomalies [17–19].

The following section of this chapter will deal with the anatomical and functional characteristics of the muscles of mastication, temporomandibular joints, and their related structures in functional and parafunctional mandibular movement.

1.2.2 Muscles of Mastication

1.2.2.1 Masseter Muscle

The masseter muscle is one of the major muscles of mastication and is primarily involved in elevation of the mandible (closure) and ipsilateral deviation [9, 20]. This pennated muscle has its origin at the anterior two-thirds of the inferior border of the zygomatic process and projects in an inferior direction to its insertion at the angle of the mandible at its junction with the mandibular ramus. Medial fibers of the masseter have their origin at the inferior border of the zygomatic process and insertion at the central part of the ramus. The deep fibers of the masseter muscle have their origin at a more medial aspect of the zygomatic process and insert into the superior part of the ramus and the mandibular coronoid process. Its fibers are predominantly vertical in alignment and thus give mechanical advantage to mandibular elevation or closure. It has a limited role in lateral excursive movement of the mandible as well as in anteroposterior movement of the mandible. The masseter muscle has both sensory and motor innervation supplied by the mandibular branch of the trigeminal nerve (V3). The masseteric branch of the maxillary artery, the facial artery, and the transverse facial branch of the superficial temporal artery provide vascular support. The masseter muscle, as well as the temporalis and medial pterygoids, have many muscle spindles (sensory receptors) located within extrafusal muscle fibers that convey skeletal muscle length change to the central nervous system. They are active in helping to regulate the excitation-contraction coupling of skeletal muscles during functional movement of the mandible by initiating motoneuron activity as part of the stretch reflex in order to help prevent overextension of the masseter, temporalis, and medial pterygoid muscles during opening [14] (See Fig. 1.1).

1.2.2.2 Temporalis Muscle

The temporalis muscle is a major mandibular elevator of the mandible and consists of three separate muscular components having different force vector orientation and displaying a distinct "fan shape." The temporalis muscle has its origin at the lateral aspect of the skull and envelopes nearly the entire temporal fossa as the anterior, medial, and posterior temporalis fibers. All three fibers then merge and course inferiorly through the medial aspect of the zygomatic process where they converge by way of the temporalis tendon at the coronoid process of the mandible and extend to the anterior border of the mandibular ramus. The anterior fibers are aligned vertically while the posterior fibers are essentially horizontal and the middle fibers are at an oblique or diagonal orientation ("fan shaped"). Functionally, the three distinct fibers are all involved in vertical closure (elevation) of the mandible. The anterior fibers are primarily involved in closure

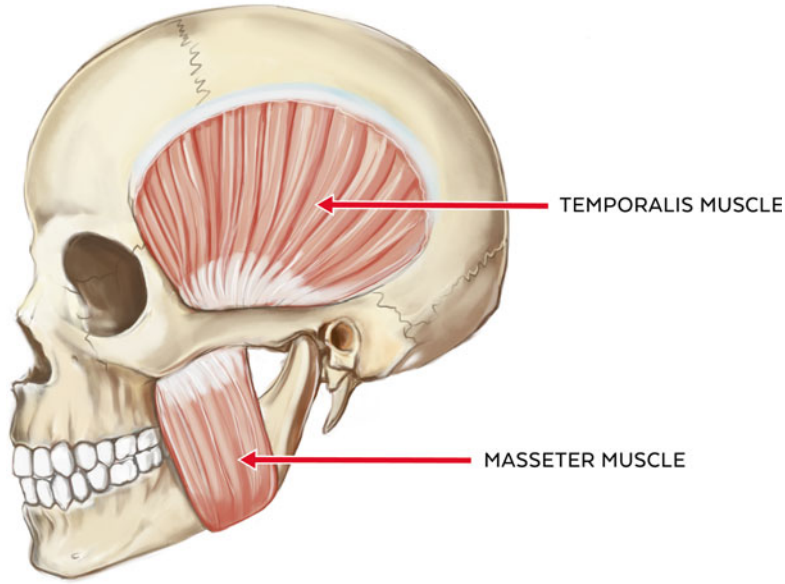


Fig. 1.1 Illustration of the masseter muscle and temporalis muscle

(mandibular elevation to tooth contact). The posterior fibers are involved in closure and seating of the condyle/disc complex during that movement as well as limited retrusion of the mandible after it has been protruded. All three fibers also play a role in lateral excursive movement of the mandible (ipsilateral lateral excursive movement) [9, 20]. The temporalis muscle has both sensory and motor innervation supplied by the deep temporal branches of the trigeminal nerve (V3). Contributions to its vascular supply are from branches of the maxillary artery: the anterior deep temporal artery supplies approximately 20 % of the anterior temporalis, the posterior deep temporal artery supplies approximately 40 % of the posterior temporalis, and the remaining 40 % (middle temporalis muscle) is supplied by the middle temporal artery (See Fig. 1.1).

1.2.2.3 Medial Pterygoid Muscle

The medial pteryoid or "internal pterygoid" muscle is a pennated masticatory muscle having two distinct points of origin. The deep head originates from the medial surface of the lateral pterygoid plate of the sphenoid. A smaller superficial head arises from the maxillary tuberosity and pyramidal process of the palatine bone. These fibers are oriented in a posterior and inferior fashion and share a tendinous insertion at the medial surface of the ramus and angle of the mandible at or near the insertion of the masseter muscle (pterygomasseteric sling). Functionally, its primary roles are in closure of the mandible (mandibular elevation), contralateral deviation, and, in conjunction with the lateral pterygoids, protrusion of the mandible. Contraction of ipsilateral medial pterygoid and lateral pterygoid muscles results in contralateral lateroexcursive movement of the mandible with translatory movement of the ipsilateral condyle and rotational movement of the contralateral condyle or contralateral deviation [9, 20].

Additionally, the medial pterygoid muscle is often described as a "functional analog" of the masseter muscle in terms of function and alignment, and is also closely associated with fibers of the tensor veli palatini muscle. This anatomical relationship of the medial pterygoid muscle and the tensor veli palatini muscle has been the subject of several studies examining the possible role of both muscles in the development of eustachian tube-related functional problems including patulous (open) eustachian tube and auditory tube dysfunction [9, 20, 21]. It has been theorized that the function of the medial pterygoid muscle moderates or influences the opening pressure of the auditory tube, and this in turn has been associated with "ear fullness" complaints in patients reporting with ear-related TMD symptoms in which

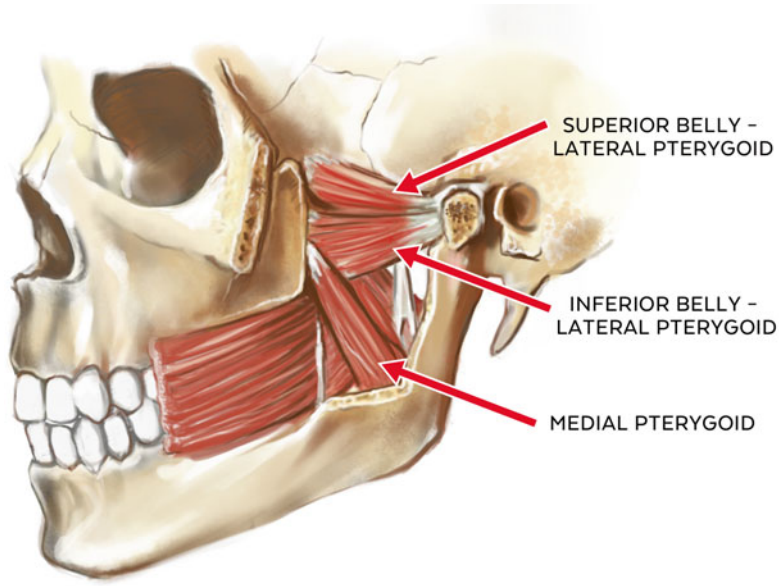


Fig. 1.2 Illustration of the medial pterygoid muscle and lateral pterygoid muscle (superior and inferior bellies)

hyperactivity of the elevator muscles is suspected relative to pain complaints [20, 22, 23]. Vascular supply of the medial pterygoid muscle is from the pterygoid branches of the maxillary artery, and it receives its sensory and motor innervation from the third branch (V3) of the trigeminal nerve (medial pterygoid branch) (See Fig. 1.2).

1.2.2.4 Lateral Pterygoid Muscle

It is a nonpennated muscle of mastication that is involved in both mandibular depression and elevation. It consists of two independent "heads" or "bellies" (superior and inferior) that have independent functional roles in mandibular movement [24]. The superior portion has its origin at the infratemporal surface and infratemporal crest of the greater wing of the sphenoid bone. Its fibers primarily insert at the anterior fovea (pterygoid fovea) of the mandibular condyle, with a variable secondary insertion occurring at the temporomandibular joint disc/capsule complex as well as medial and anterior portions of the disc. Approximately 60–70 % of the fibers insert at the anterior fovea of the condyle while 30–40 % insert at the capsule-disc complex [11, 20]. While it has been posited that the superior lateral pterygoid muscle can pull the disc anteriorly from its contact with the condyle, this does not seem to be anatomically possible. The inferior portion of the lateral pterygoid has a less diverse origin-insertion identity as does the superior portion, and has its origin at the outer surface of the lateral pterygoid plate and insertion at the anterior fovea and neck of the condyle.

Functionally, the lateral pterygoid muscles are considered to play a major role in protrusion and contralateral deviation of the mandible [9]. Specifically, the upper or superior belly is active as a counterbalance or disc/condyle stabilizer during closure of the mandible while the inferior or lower belly of the lateral pterygoid is significantly inhibited during this movement. Conversely, the inferior belly is active in opening and protrusion of the mandible while the superior belly is significantly inhibited during this movement, giving the lateral pterygoid muscle a very unique role in mandibular movement based upon independent functions of the superior and inferior bellies of this masticatory muscle [24].

While the bellies of the lateral pteryoid muscle are active as depressor and elevator muscles in mandibular movement, overall, the lateral pterygoid is considered to be of secondary importance in mandibular opening movement, with the digastric and geniohyoid muscles being the primary mandibular depressors (opening) [20]. Due to the unique anterior and medial alignment of the lateral pterygoid fibers to the bilateral

mandibular condyles, bilateral activation of the inferior bellies of the pterygoids results in a forward or protrusive movement of the mandible having an anterior or horizontal projection, especially during parafunctional activities and heavy mastication [27]. However, unilateral activation of either inferior belly will result in a shift of the mandible to the ipsilateral side and a resulting translational movement of the contralateral side; thus, the terms "condylar translation" and "condylar rotation." This movement is observed in both functional lateral excursion of the mandible (chewing stroke) as in mastication and in parafunctional lateral excursive movement of the mandible as in bruxing and clenching. In conjunction with the digastric and geniohyoid muscles, the lateral pterygoids play a significant role in the three-dimensional functional envelope (Posselt's Envelope of Motion) of mandibular movement in vertical, anterior-posterior, and lateral (transverse) dimensions. Since the origins of lateral pterygoid muscles are medial to their insertions, wide opening of the mandible may result in the mandible being temporarily distorted transversely, with the bilateral posterior lingual borders actually being distorted toward the midline. Thus, taking an impression of the lower dentition with the mouth widely open may result in study casts that are not accurate in the transverse dimension (Mohl, N.D., Verbal communication) [44]. Unlike the other jaw closing muscles, the lateral pterygoids are unique in that they do not contain muscle spindles. The absence of muscle spindles may help to explain why the lateral pterygoids play a secondary role in mandibular depression, where stretch receptors (muscle spindles) are essential for detecting muscle working length change and velocity, and help prevent excessive stretching of the muscle during functional movement [14, 46].

The lateral pteryogid muscles also act in conjunction with the posterior temporalis muscle fibers in controlling anterior and posterior translation of the mandible [9]. For a more detailed explanation of the functional movement of the mandible relative to mastication, the reader may refer to Functional Biomechanics of the Masticatory System: in *Management of Temporomandibular Disorders and Occlusion*, 7th edition, Chapter 1, p. 13, and Mechanics of Mandibular Movement: in *Management of Temporomandibular Disorders and Occlusion*, 7th edition, Chapter 4, pp. 62–72 Okeson, J.

Vascular supply of both heads of the lateral pterygoid muscles is from the pterygoid branches of the maxillary artery and from the ascending palatine branch of the facial artery. Its unique "independent function" mirrors its superior and inferior belly innervation. That is, while the primary innervation is from the third division of the trigeminal nerve (V3 or mandibular nerve), the superior belly and the lateral fibers of the inferior belly receive their innervation from the buccal branch of the mandibular nerve (V3) and the medial fibers of the inferior belly receive their innervation from the anterior trunk of the mandibular nerve (V3) [20] (See Fig. 1.2).

1.2.2.5 Digastric Muscle

The digastric muscle, like the lateral pterygoid muscle, has two distinct components: the anterior and posterior belly, neither of which contains muscle spindles. The anterior belly has its origin at the digastric fossa and submental area near the midline, and its fibers extend inferiorly and posteriorly. The posterior belly has its origin at the mastoid notch of the temporal bone, and its fibers extend inferiorly and forward to join with the anterior belly at a common intermediate tendon attachment at the body and greater horn of the hyoid bone. Both bellies of the digastric muscle act to depress the mandible (opening), and the posterior belly is also involved in elevating the hyoid bone during mastication and swallowing. One important variant in the origin of the anterior belly is that its fibers may cross the anatomical midline at the submental area, and it has been proposed that this variation may play a role in detectable but nonpathologic mandibular deviation on opening [25].Vascular supply to the anterior belly is from the submental branch of the facial artery. The posterior belly receives its blood supply from the posterior auricular and occipital arteries. An interesting and unique feature of the digastric muscle is in its innervation; that is, the anterior belly is innervated by the

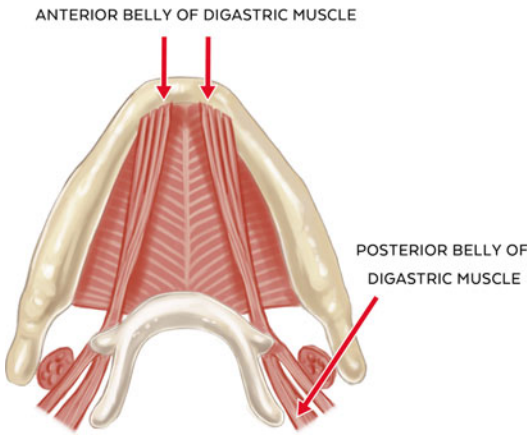


Fig. 1.3 Illustration of the digastric muscle (anterior and posterior belly)

mylohyoid branch of the inferior alveolar nerve (V3) while the posterior belly is innervated by the facial nerve (VII), highlighting their distinct and separate derivations from 1st and 2nd branchial arches [26] (See Fig. 1.3).

1.2.3 Functional Characteristics of the Masticatory Muscles

Coordinated and efficient movement of the mandible is a highly complex function of the neuromuscular system primarily involving muscle fibers, sensory and motor nerves, proprioceptive receptors, and a control "center" within the reticular formation of the midbrain. This coordinated effort has both voluntary as well as involuntary characteristics in terms of stimulation, inhibition, and control, and it is within the context of these phenomena that static, functional, and parafunctional movements of the mandible derive their appropriate descriptions.

The primary skeletal muscles of the masticatory system are designated by their function; that is, elevators/retractors (muscles primarily involved in mandibular closing) and depressors/protruders (muscles primarily involved in mandibular opening). As in all skeletal muscles, their functional relationships are closely coordinated with one another so as to ensure a more efficient and coordinated movement. The actual function of the muscle is dependent upon its origin and insertion, with the origin of the muscle being at a stationary location (musculotendinous anchorage) and the insertion being the attachment of the muscle to the body being moved (bone and joint mechanics). Often the muscles of mastication are labeled as being agonists (primary movers) or antagonists (those that oppose a particular movement) depending upon the functional movement that each is going through at a particular point in time. For example, the elevator muscles may be thought of as being active in closing movement of the mandible while the depressor muscles are relaxed, with the opposite being true during opening movement. However, both groups of muscles are functionally active to some degree at all times rather than being sequentially stimulated or totally inhibited and in opposition to the other during mandibular movement. The three-dimensional functional movement of the mandible requires a high degree of coordinated effort between and among both classes of muscle [9, 15].

Another anatomical factor to address is the architectural arrangement of the muscles of mastication relative to their force and power output. Muscles that have their muscle fibers (fascicles) arranged in an oblique orientation to their origin are considered to be pennated with fibers arranged in some type of "feather-like" or "plume-like" arrangement [9, 10, 28]. Pennated muscles have tendons that extend for most of the muscle's length (origin to insertion), and its fibers are obliquely aligned as they insert into the tendons. Nonpennated muscles have their fascicles arranged in a more parallel-like orientation relative to the origin and insertion of the muscle. The mechanical advantage of the pennated muscles is that they generally possess a greater number of muscle fibers per volume area (physiological cross sectional area or PCSA), resulting in a greater net force production than muscles arranged in parallel orientation. However, while pennated muscles produce more force than parallel muscles, they tend to demonstrate less power output (force x displacement) as a side effect of their orientation as well as less range of motion. Overall, the muscles of mastication have a mixed architectural classification with the masseter,

temporalis and medial pterygoid muscles classified as pennated and the lateral pterygoid muscle classified as a nonpennated skeletal muscle [29].

The term "power stroke of mastication" is often used to describe the elevation of the mandible during closure to an at-or-near tooth contact during mastication ("chewing stroke of mastication") [30]. A single chewing stroke consists of one cycle or loop of mandibular depression, lateral deviation of the mandible, and elevation ("tear drop" configuration in the frontal plane). "Power" is defined as the amount of energy consumed per unit time, is dependent upon the trajectory of the point of force application and torque, is measured in joules per second or watts, and is expressed as "power = work/time." "Force" is defined as a particular amount of energy being exerted in a specific direction (measured in newtons), and is the more accurate expression for mandibular closure movement (bite registration, for example) rather than "power," which, by definition, is velocity or movement/second [13, 16]. Therefore, it is proposed that the term "force closure" be substituted for "power closure" in the discussion of masticatory biomechanics, especially when describing those forces exerted during the chewing stroke (opening and closing) and orthodontic bite registration, for example [34, 35].

Coordination of the masticatory cycle is primarily controlled by the central pattern generator (CPG) of mastication, a neurosensory gait or rhythmicity control system located within the reticular formation of the brainstem. It directs involuntary masticatory function with associated input from the thalamus, hypothalamus, and limbic system. Overriding influences created by emotional stress and/or parafunctional activity may also influence the overall CPG-directed coordination of complex masticatory movements [12, 31, 33].

1.2.4 Anatomy of the Temporomandibular Joints

The temporomandibular joint (TMJ) is a ginglymoarthrodial joint that exhibits a combination of hinge and gliding movements, thus allowing the mandible to slide and rotate in a parasagittal plane during functional and parafunctional movement [28]. It is not a "ball and socket" articulation. By definition, a ginglymoarthrodial joint is "any joint with both uniplanar hinge movement and gliding movement in another plane"; that is, hinging movements in one plane and gliding or sliding movements in another plane. The joint most often used to describe this classification is the knee since it is able to function in three planes or axes of motion (*x, y,* and *z)* [29]. In the human temporomandibular joint, a three-dimensional envelope of motion illustrates these movements during mastication or during parafunctional movements such as lateral bruxing or static clenching of the dentition [30].The human temporomandibular joint, like most joints in the body, acts within the constraints of a Class III lever system. By definition, a lever consists of a rigid body with two externally applied forces and a center of rotation (COR). A Class III lever has its forces (muscle) applied on the same side of the center of rotation (COR), and the muscle forces are closer to the center of rotation than to the external force. In the case of the human TMJ, the forces are represented by muscles of mastication and the COR is the TMJ (fulcrum or condyle/disc loading area). The external forces are those encountered in mastication (third molar anteriorly) or during parafunctional clenching or bruxing. The condyles are loaded to some degree at all times depending upon the position of the occlusal contact and the amount and direction of contraction of the mandibular musculature. Therefore, the human TMJ functions as a Class III lever [15, 31, 32] (See Fig. 1.4).

In the human body, most joints are classified as synovial joints. Joint classification is based upon anatomical criteria including (1) the type of motion in which the joint is involved, (2) shape of their articulating surfaces, and (3) the number of axes that they possess in a full range of motion [33]. Based upon its movement, the human TMJ is classified as a true diarthrodial (synovial) joint or condylar joint in which synovial fluid is secreted by "boundary" synovial villi for lubrication and nutrition of the joint. The bilateral TMJ's

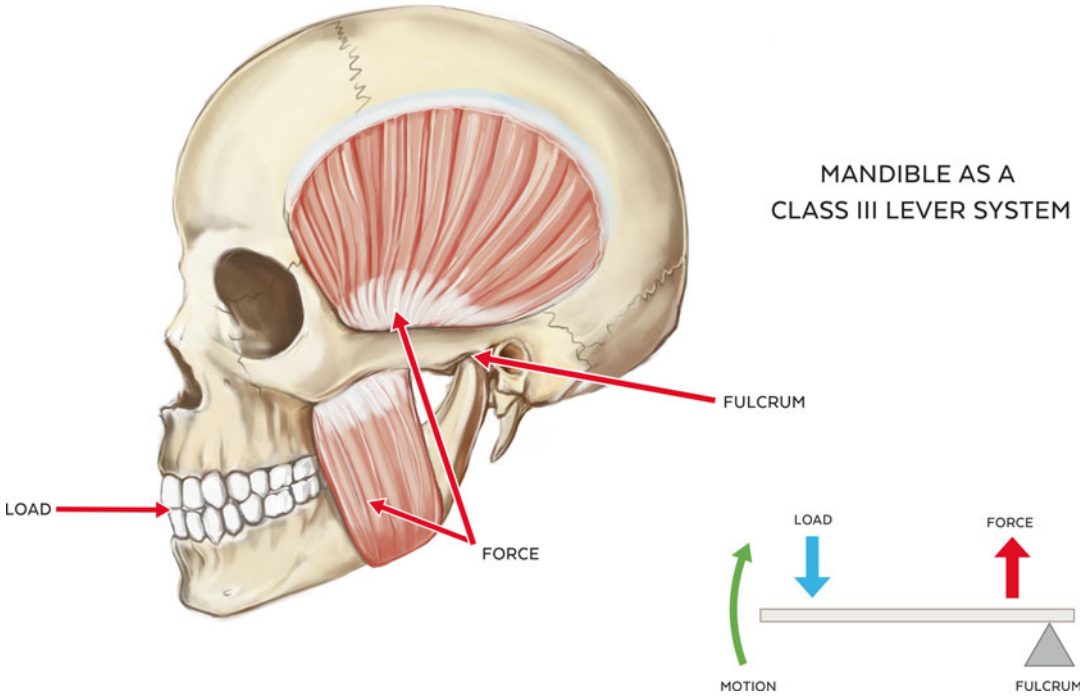


Fig. 1.4 Illustration of the mandible acting as a Class III lever system

are load-bearing joints consisting of an articular disc interposed between the mandibular condyle and articular eminence of the glenoid fossa. All three articular surfaces (condyle, disc, eminence) are composed of avascular, noninnervated dense fibrous connective tissue and fibrocartilage [31].

Capsular ligaments connect one bone to another in synovial joints and provide mechanical reinforcement and joint stability for that joint. The capsular ligament of the temporomandibular joint encapsulates the joint with its chief attachment being at the inferior border of the temporal bone running anteriorly and approximating the articular eminence and glenoid fossa. Its chief functions include stabilizing the joint during three-dimensional movements and maintaining the integrity of the synovial fluid located within the joint capsule "boundaries." The joint capsule (capsular ligament), including the fibrous and synovial layers, defines the functional and anatomical boundaries of the temporomandibular joint. The sphenomandibular ligaments and stylomandibular ligaments (commonly referred to as accessory ligaments) play a less significant role in the stabilization of the mandible, with the stylomandibular ligament acting to help limit excessive mandibular protrusion [11].

As in all diarthrodial joints, synovial tissue is found within the synovial membrane, a vascularized connective tissue that lines the joint capsule. Synovial fluid is expressed at the "boundaries" of the joint's loading area or stress field. The inner layer of the joint capsule is the synovial layer, and the outer fibrous component of the capsule is composed of dense fibrous connective tissue (as in the articular disc). The joint capsule is highly innervated with joint receptors located in the capsule of the joint, tendons, ligaments, and muscles, and these highly innervated structures and their subsequent neurosensory feedback to the CNS help to balance and maintain joint stability and mobility and protect the joint [34] (See Fig. 1.5 and 1.6).

Tendinous attachments (muscle to bone) are found at all muscles of mastication at their points of origin and insertion. In general, tendons connect to muscle at the myotendinous junction, and the muscle and tendinous fibers are interwoven

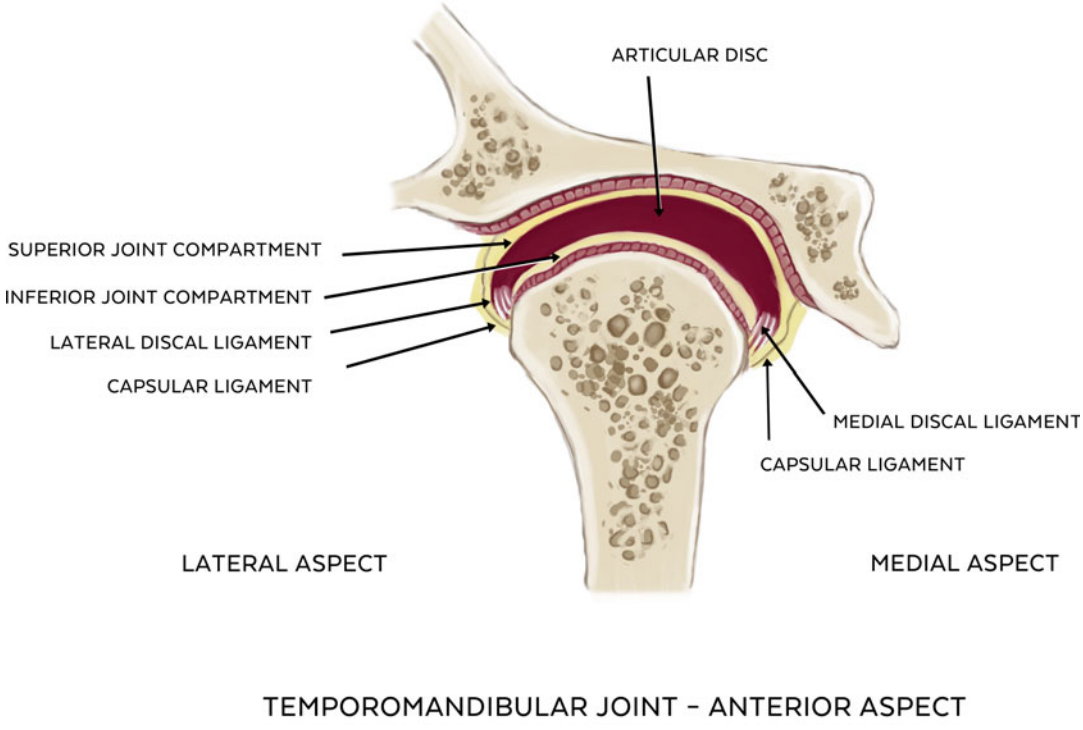


Fig. 1.5 Illustration of the right temporomandibular joint (frontal view)

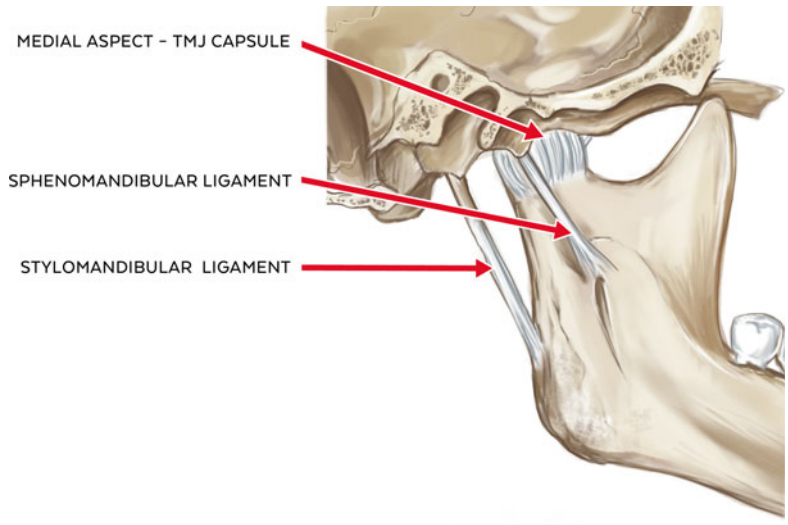


Fig. 1.6 Illustration of the accessory ligaments and capsule (medial view)

together in combination with collagenous fibers for added strength. Tendons transmit muscular force to bone and are critical in maintaining stability and maximum force transduction during biomechanical movement. Like the articular disc, the tendons of the masticatory muscles exhibit some degree of viscoelastic behavior in response to loading, and though composed chiefly of inelastic cords of collagen tissue, they also exhibit some degree of flexibility during muscular activity. The chief tendon encountered during mandibular movement, especially relative to hyperactivity within the temporalis muscle, is the temporalis tendon. It inserts at the coronoid

process of the mandible and transfers force during elevation, lateral excursion, and, to a limited extent, retrusion of the mandible [16, 34, 45].

Fibrous and cartilaginous joints allow linear movement or translatory movement, but the temporomandibular joint is unique as a synovial joint in that it is capable of both rotational and translational movement. Rotational movement is best demonstrated in the early phase or cycle of mandibular opening. Translational movement is observed as the condyle begins its translatory movement against the slope of the articular eminence; this even occurs at the beginning of opening. Rotation in mandibular opening and closing may occur without any significant positional change of the condyles and is observed as occurring in the inferior joint space of the TMJ. Translational movement, or "movement in space characterized by linear motion with no axis of rotation, with that movement describing a straight path (rectilinear translation) or a curved path (curvilinear translation)," occurs in the superior joint space of the TMJ [46]. As a true synovial joint, the TMJ exhibits rotation and translation bilaterally and is classified as having 3° of freedom during mandibular movement per joint, but not functionally independent of one other. Therefore, it is important to note that while the other joints such as the knee can be isolated during range of motion studies without it being influenced by its counterpart, the temporomandibular joint is considered to be a "compound joint" and does not have that "independent counterpart" status. Any movement in one TMJ will be accompanied by some degree of movement in the contralateral TMJ, thus giving the human TMJ's three degrees of freedom in movement per joint [36].

1.2.4.1 Structural and Functional Characteristics of the Temporomandibular Joint

The temporomandibular joint is formed between the condylar process of the mandible and the squamous portion of the temporal bone [10]. The medial-lateral dimension of the condyle is approximately twice that of its anteroposterior dimension. The mandibular (glenoid) fossa is bounded anteriorly by the posterior slope of the articular eminence, medially by a narrow bony wall, and posteriorly by the postglenoid process. Posterior to the postglenoid process, the tympanic plate joins the squamosum to form the squamotympanic fissure. The squamotympanic fissure, in turn, divides into the petrotympanic and petrosquamosal fissures. The chorda tympani nerve and anterior tympanic artery and vein pass through the petrotympanic fissure and are well protected from impingement by the condyle. The roof of the (glenoid) fossa is thin and is not designed to withstand condylar loading. Thus, functional and parafunctional loading by the condyle occurs on the slope of the articular eminence rather than at the superior aspect of the fossa. Unlike the articular tissues of most synovial joints, which are composed of hyaline cartilage, the articular tissues of the temporomandibular joints are, for phylogenic and embryologic reasons, composed of avascular and noninnervated dense fibrous connective tissue [46] (See Fig. 1.7).

As with any synovial joint, the articular capsule defines the anatomical and functional boundaries of the temporomandibular joint. The capsule belongs to the squamous portion of the temporal bone and the tympanic portion of the temporal bone lies completely behind the joint [31].

The capsule completely surrounds the articular eminence and is attached well anterior to its crest. The capsule is strongly reinforced laterally by the temporomandibular (lateral) ligament that consists of obliquely oriented collagen fibers and a narrow band of collagen fibers oriented in a horizontal direction. The primary function of these ligaments is to "check" or limit movements of the condyle-disc complex, particularly retrusion against retrocondylar structures. This "ligamentous position" is considered to be a border position of the mandible and is reproducible, recordable, and is not influenced by transient postural factors. The ligamentous position is akin to "centric relation," which has been defined as "the mandibular jaw position in which the head of the condyle is situated as far anteriorly and superiorly as it possibly can within the mandibular/glenoid fossa." However, when external forces

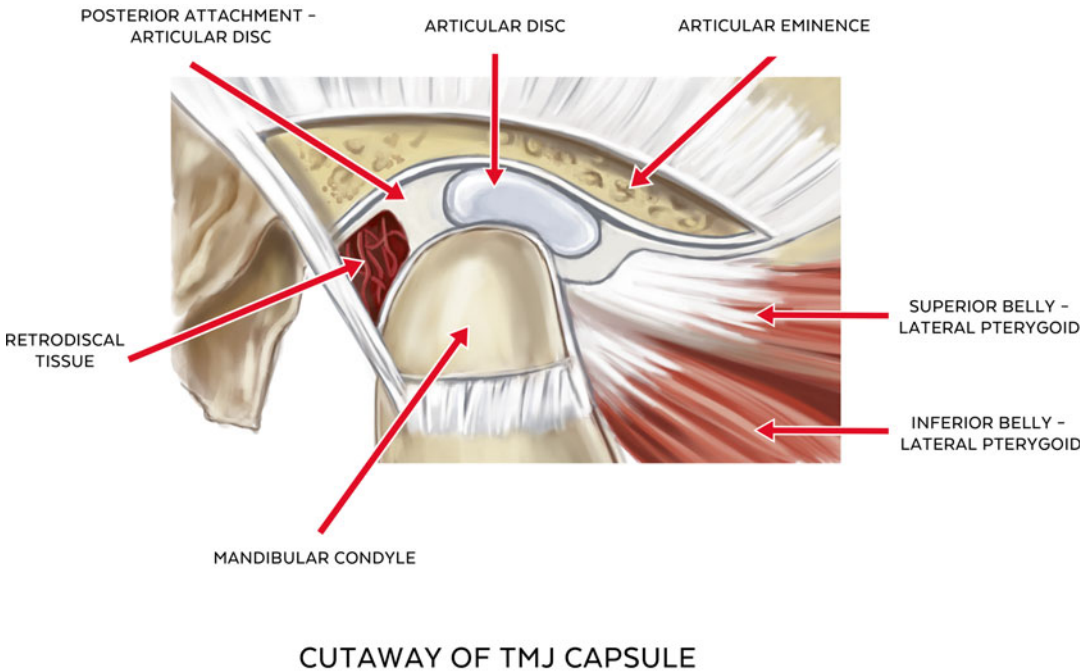


Fig. 1.7 Illustration of the TMJ capsule, disc, condyle, and eminence (cutaway of TMJ capsule)

such as manual manipulation seek this position, the condyles and discs may not necessarily be seated upward and forward against the posterior slope of the articular eminence, and therein lies one aspect of the controversy surrounding the term "centric relation" [31] (See Fig. 1.8).

The articular disc plays a significant role in joint lubrication and stress distribution, and as a result of its physical capacity to alter its shape during loading, it is able to distribute compressive stresses over a large contact area or stress field [37]. Since it exhibits a somewhat amorphous shape, its adaptive capacity to loading is dependent upon several factors including the concentration of proteoglycans at the articular surfaces and its physical shape at the location of loading. Proteoglycans are glycosaminoglycan (GAG) chains covalently attached to a core protein [38]. They are abundant in extracellular matrices (cartilage) and, among other numerous functions, provide hydration and resilience (cushioning) to joints during compressive loading. The major proteoglycan in the articular cartilage is aggrecan (aggregated proteoglycan), a sulfated proteoglycan [38, 39].The degree of resistance to

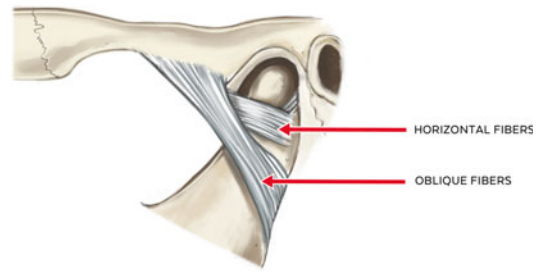


Fig. 1.8 Illustration of the temporomandibular ligament (capsular ligament and fiber orientation)

compressive loading may be directly related to the concentration of GAGs in the loading zone or stress field, so the greater the concentration of GAGs in an area of loading, the greater the resistance to compressive loading within that stress field [40].

1.2.5 Biomechanical Principles in TMJ Loading

The temporomandibular joint may be dynamically loaded during mastication and parafunction

such as bruxing or statically loaded during other parafunctional activities (centric or eccentric tooth clenching). In general, joint structure and the external forces that are applied to the joint will determine the type and quantity of motion that occurs within that joint. Additionally, the point of force application to the joints during normal function determines the "stress-field" loading area of that joint. As these loading surfaces are altered by the magnitude, location, frequency, intensity, and duration of their loads, other physical changes may occur within the articulating surfaces as the joint components attempt to adapt to the loads. Conversely, the same factors that help distribute loading within the joints can be negatively affected by these loads, resulting in a weakening of the articular surfaces, and possibly predisposing the joints to degenerative changes [31, 32, 34, 41].

Two general types of forces, shearing and compressive loading, are generated within the joint during function and parafunction. Shearing or dynamic loading is caused by tractional forces being exerted parallel to the articular surfaces, which result in combinations of frictional and plowing forces. For example, as a hard object (condyle) moves along a softer surface (articular disc), the softer surface is pushed ahead of the hard object, resulting in what is best described as a "plowing" effect.

Plowing is the most common type of force observed in the TM joint, and the magnitude of this force is increased by increases in force intensity and duration. Plowing forces also are affected by overall hydration of the joint disc and the loading surfaces. Since the disc also takes part in lubrication of the joints, any reduction in disc/joint lubrication can result in a subsequent alteration of force distribution within the joint, and eventual weakening of that joint's loading capacity [32, 34, 43]. It is theorized that this weakening effect predisposes the temporomandibular joint to degenerative change commonly seen in TMJ osteoarthritis. Other factors involved in predisposing this joint to degenerative joint disease (DJD) or osteoarthritis include the weakening of its mediolateral collagen cross linkages, which results in surface defects in the disc and propagation of disc degeneration [37, 43]. Similarly, while normal mechanical forces are essential for health of the joints, the presence of excessive cartilage loading, underloading (lack of function or immobility), or static loading can cause proteoglycan depletion, and this can reduce the adaptive capacity of the joint [41, 42].

Finally, stability of the TM joints and health of the discs in particular are highly dependent upon the integrity of the discal polar ligaments (medial discal ligament and lateral discal ligament). Any alteration in the integrity of these discal ligaments (e.g., the stretching that occurs when there is anteromedial disc displacement) may result in stress-field alterations within the disc, thus potentially predisposing the disc and joint to degenerative change. Therefore, while stresses that are imparted upon the articulating surfaces of the joint are important, of equal importance are the integrity and stability of the articulating surfaces and their ability to adapt to changes in loading over time. Compromised surface integrity due to surface incongruities, reduction in extracellular matrix proteoglycan (protein core plus sulfated glucosaminoglycans or GAGs), and reduced synovial lubrication all play a role in the predisposition and progression of degenerative joint disease [32, 41, 43]. There is also sufficient evidence to show that the articular tissues of the temporomandibular joint are capable of adaptation to biomechanical stresses, although this adaptation is slow and unpredictable. In addition to progressive and regressive remodeling, chondrocytes and proteoglycans can be observed in many, if not most temporomandibular joints, especially in those from older individuals. As a result with time the dense fibrous connective tissue takes on the appearance of fibrocartilage, which has a greater capacity to withstand compressive loading, particularly when that loading is cyclical or intermittent [31].

Take Home Messages

1. The human temporomandibular joint is a diarthrodial joint allowing both hinging and gliding movements within a three-dimensional framework.
2. The articular capsule defines the anatomical and functional boundaries of the TMJ.
3. The muscles of mastication are active in the elevation and depression of the mandible, protrusion and retrusion of the mandible, and lateral excursive movement.
4. The articular disc (not a meniscus) consists of a centralized, functional zone of avascular, noninnervated tissue as well as a peripheral portion that is vascular, innervated, and located outside of the functional loading zone.
5. The mandible works within the confines of a Class III lever system.
6. The articular tissues of the TMJ are capable of adaptation to biomechanical stresses, although this adaptation is slow and unpredictable as to its timing and extent.
7. Excessive functional loading (frequency, intensity, duration) of the temporomandibular joint components may result in clinical signs and symptoms including inflammation, pain, reduced mandibular range of motion, and, in some cases, the onset of osteoarthritis.

References

Occlusal Concepts and Terminology

1. Jablonski S. Illustrated dictionary of dentistry. Philadelphia: W.B. Saunders Co.; 1982.
2. Mohl ND. Introduction to occlusion. In: Mohl ND, Zarb GA, Carlsson GE, Rugh JD, editors. A textbook of occlusion. Chicago: Quintessence Publishing Co.; 1988. p. 15–23.
3. Dorland WAN. Dorland's Illustrated Medical Dictionary. 32nd ed. Philadelphia: Elsevier Saunders. 2012.
4. Syrop J. Understanding occlusion. Inside Dentistry. 2013;9(9):46–58.
5. Okeson JP. Alignment of the occlusion and dentition. In: Okeson JP, editor. Management of temporomandibular disorders and occlusion. St. Louis: Elsevier Mosby; 2013. p. 46–62.
6. Okeson JP. Determinants of occlusal morphology. In: Okeson JP, editor. Management of temporomandibular disorders and occlusion. St. Louis: Elsevier Mosby; 2013. p. 86–100.
7. Proffit WR, Fields HW, Sarver DM. Malocclusion and dentofacial deformity in contemporary society. In: Proffit WR, Fields HW, Sarver DM, editors. Contemporary orthodontics. St. Louis: Elsevier Mosby; 2013. p. 2–18.
8. The Glossary of Prosthodontic Terms. J of Pros Dent. 2005;94(1):10–92.

Masticatory System Anatomy – Concepts and Terminology

9. Pratt N, Oatis C. Mechanics and pathomechanics of the muscles of the TMJ. In: Oatis C, editor. Kinesiology: the mechanics and pathomechanics of human movement. Philadelphia: Lippincott, Williams and Wilkins; 2009. p. 452–65.
10. Infratemporal and pterygopalatine fossae and temporomandibular joint. In: Standring S (editor-in-chief). Gray's anatomy: the anatomical basis of clinical practice, 40th ed London: Churchill Livingstone Elsevier;2008. p. 538.
11. Okeson JP. Functional anatomy and biomechanics of the masticatory system. In: Okeson JP, editor. Management of temporomandibular disorders and occlusion. 7th ed. St. Louis: Elsevier Mosby; 2013. p. 2–20.
12. Morquette P, Lavoie R, Fhima M, Lamoureux X, Verdier D, Kolta A. Generation of the masticatory central pattern and its modulation by sensory feedback. Prog Neurobiol. 2012;96(3):340–55. doi:10.1016/j.pneurobio.2012.01.011.
13. Gorse C, Johnston D, Pritchard M. A dictionary of construction, surveying and civil engineering. 1st ed. Oxford: Oxford University Press; 2012.
14. Okeson JP. Functional neuroanatomy and physiology of the masticatory system. In: Okeson JP, editor. Management of temporomandibular disorders and occlusion. 7th ed. St. Louis: Elsevier Mosby; 2013. p. 21–45.
15. Karduna A. Introduction to biomechanical analysis. In: Oatis CA, editor. Kinesiology: the mechanics and pathomechanics of human movement. Baltimore: Lippincott Williams and Wilkins; 2009. p. 3–20.
16. Pratt N, Oatis C. Biomechanics of skeletal muscle. In: Oatis C, editor. Kinesiology: the mechanics and pathomechanics of human movement. Philadelphia: Lippincott, Williams and Wilkins; 2009. p. 45–68.

17. Congenital anatomic anomalies or human birth defects. In: Moore KL, Persaud TVN, editors. The developing human: clinically oriented embryology. Philadelphia: Saunders Elsevier;2008. p. 457–86.
18. Pharyngeal apparatus, face, and neck. In: Moore KL, Persaud TVN, Torchia MG, editors. The developing human: clinically oriented embryology. Philadelphia: Saunders Elsevier; 2013. p. 159–98.
19. Anatomy of the muscular system. In: Patton KT, Thibodeau GA, editors. Anatomy and physiology. St. Louis: Mosby Elsevier; 2013. p. 301–46.
20. Infratemporal and pterygopalatine fossae and temporomandibular joint. In: Standring S, editor. Gray's anatomy: the anatomical basis of clinical practice. London: Churchill Livingstone Elsevier; 2008. p. 527–46.
21. Oshima T, Ogura M, Kikuchi T, Hori Y, Mugikura S, Higano S, Takahashi S, Kawase T, Kobayashi T. Involvement of pterygoid venous plexus in patulous eustachian tube symptoms. Acta Otolaryngol. 2007;127(7):693–9.
22. Leuwer R, Schubert R, Kucinski T, Liebigt T, Maier H. The muscular compliance of the auditory tube: a model-based survey. Laryngoscope. 2002;112(10):1791–5.
23. McDonald MH, Hoffman MR, Gentry LR, Jiang JJ. New insights into mechanism of eustachian tube ventilation based on cine computed tomography images. Eur Arch Otorhinolaryngol. 2012;269(8):1901–7. doi:10.1007/s00405-011-1829-y.
24. Mahan PE, Wilkinson TM, Gibbs CH, Mauderli A, Brannon LS. Superior and inferior bellies of the lateral pterygoid muscle EMG activity at basic jaw positions. J Prosthet Dent. 1983;50(5):710–8.
25. Stockstill JW, Harn S, Underhill T. Clinical implications of anomalous muscle insertion relative to jaw movement and mandibular dysfunction: the anterior belly of the digastric muscle in a cadaver. J Craniomandib Disord Facial Oral Pain. 1991;5:64–70.
26. Standring S. Neck. In: Standring S, editor. Gray's anatomy: the anatomical basis of clinical practice. London: Churchill Livingstone Elsevier; 2008. p. 441.
27. Murray GM, Phanachet I, Uchida S, Whittle T. The role of the human lateral pterygoid muscle in the control of horizontal jaw movements. J Orofac Pain. 2001;15(4):279–92.
28. Standring S. Infratemporal and pterygopalatine fossae and temporomandibular joint. In: Standring S, editor. Gray's anatomy: the anatomical basis of clinical practice. London: Churchill Livingstone Elsevier; 2008. p. 527–46.
29. The American Heritage Stedman's Medical Dictionary. Boston: Houghton Mifflin Co., Boston, USA; 2002.
30. Posselt U. Studies on the mobility of the human mandible. Acta Odont Scand. 1952;10:1–150.
31. Mohl ND. Functional anatomy of the temporomandibular joint. In: Laskin D, Greenfield W, Gale E, Rugh J, Neff P, Alling C, Ayer W, editors. The president's conference on the examination, diagnosis and management of temporomandibular disorders. American Dental Association publications, Chicago, IL, USA; 1983. p. 3–12.
32. Lockard MA, Oatis CA. Biomechanics of joints. In: Oatis CA, editor. Kinesiology: the mechanics and pathomechanics of human movement. Baltimore: Lippincott, Williams and Wilkins; 2009. p. 103–15.
33. Hamill J, Knutzen KM. Skeletal considerations for movement. In: Hamill J, Knutzen KM, editors. Biomechanical basis of human movement. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2009. p. 27–61.
34. Okeson JP. History and examination for temporomandibular disorders. In: Okeson JP, editor. Management of temporomandibular disorders and occlusion. St. Louis: Mosby Elsevier; 2013. p. 170–221.
35. Dawson PE. Recording centric relation. In: Dawson PE, editor. Functional occlusion – from TMJ to smile design. St. Louis: Mosby Elsevier; 2007. p. 94–5.
36. Hamill J, Knutzen KM. Basic terminology. In: Hamill J, Knutzen KM, editors. Biomechanical basis of human movement. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2009. p. 3–25.
37. de Leeuw R, Boering G, Stegenga B, de Bont LG. TMJ articular disc position and configuration 30 years after initial diagnosis of internal derangement. J Oral Maxillofac Surg. 1995;53(3):234–41.
38. Perrimon N, Bernfield M. Cellular functions of proteoglycans-an overview. Semin Cell Develop Biol. 2001;12:65–7. doi:10.1006/scdb.2000.0237.
39. Mansour J. Biomechanics of cartilage. In: Oatis CA, editor. Kinesiology: the mechanics and pathomechanics of human movement. Baltimore: Lippincott, Williams and Wilkins; 2009. p. 69–83.
40. Yanagishita M. Function of proteoglycans in the extracellular matrix. Acta Pathol Jpn. 1993;43(6):283–93.
41. Palla S, Gallo L. Biomechanics and mechanobiology of the TMJ. In: Greene CS, Laskin DM, editors. Treatment of TMDs: bridging the gap between advances in research and clinical patient management. Chicago: Quintessence Publishing; 2013. p. 101–12.
42. Ramage L, Nuki G, Salter D. Signaling cascades in mechanotransduction: cell- matrix interactions and mechanical loading. Scand J Med Sci Sports. 2009;19:457–69.
43. Braden CF, Hulstyn MJ, Oksendahl HL, Fadale PD. Ligament injury. Reconstruction and osteoarthritis. Curr Opin Orthop. 2005;16(5):354–62.
44. Mohl ND. Verbal communication. 2014.
45. Okeson JP. Alignment of the occlusion and dentition. In: Okeson JP, editor. Management of temporomandibular disorders and occlusion. St. Louis: Elsevier Mosby; 2013. p. 46–62.
46. Mohl ND. Introduction to occlusion. In: Mohl ND, Zarb GA, Carlsson GE, Rugh JD, editors. A textbook of occlusion. Chicago: Quintessence Publishing Co.; 1988. p. 15–23.

Temporomandibular Disorders: Etiology and Classification

2

Jeffrey P. Okeson

2.1 Introduction

Temporomandibular disorders (TMDs) are a group of disorders that have their origin in the musculoskeletal structures of the masticatory system [1]. Therefore, symptoms of TMDs are associated with either the muscles of mastication or the temporomandibular joints (TMJs), or both. Pain can be a common symptom associated with TMDs. These disorders are quite common in the general population. In fact, after dental pains, TMDs are the next most common pain complaint reported by patients in the dental office. Depending on which epidemiologic studies are reviewed the numbers of signs and symptoms associated with TMDs range from 40 % and 60 % of the general population [2, p. 102–28]. However, the numbers of patients requiring professional TMD treatment are reported to be only in the range of 10–15 % [3, 4]. Because TMDs are so common, every dentist needs to have a basic understanding of the etiology, diagnosis, and management of these conditions. This chapter will focus on the etiology and diagnosis of the most common TMD conditions seen in the dental office. Other texts should be reviewed for a more complete overview [2].

J.P. Okeson, DMD (✉)
Department of Oral Health Practice,
Orofacial Pain Program, University of Kentucky
College of Dentistry, Lexington, KY, USA
e-mail: okeson@uky.edu

It needs to be appreciated that all TMDs are not the same. There are a great variety of musculoskeletal conditions associated with disorders of the masticatory system. The two broad conditions are muscle pain disorders and intracapsular disorders. These conditions are quite different in their etiology, pathology, and clinical presentations. Because of these differences, they demand different treatment strategies. The most common types of TMDs are muscle pain disorders, which are found to be almost twice as common as intracapsular pain disorders in a chronic pain clinic. It would therefore be very inappropriate to label these patients as "TMJ patients" when the majority of cases may have nothing to do with their temporomandibular joints. Failing to distinguish between these myogenous and arthrogenous conditions will likely lead to the selection of an ineffective treatment that will ultimately fail.

This chapter will highlight the etiologic factors associated with TMDs and describe a classification for the most common conditions seen in the orthodontic practice. It should be noted that although management is not meant to be a part of this chapter, most of these TMDs can be successfully managed with conservative therapies (see Chap. 8). Orthodontic therapy may be considered as a treatment option in a few of these patients according to their specific diagnosis and etiologic factors, but only after clinical symptoms have been successfully managed.

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_2

2.2 Etiologic Considerations of Temporomandibular Disorders

Over the years there has been significant controversy regarding the etiology of TMDs. Early on dentists were very convinced that temporomandibular disorders were primarily caused by occlusal factors. Many dentists directed their therapies toward changing the patient's occlusion; and if that failed, the operator was thought to be incompetent or the patient was considered to have major psychological problems. By the mid-1980s and 1990s, however, the profession demanded more research evidence, which provided a much broader look at TMDs. Over the last 20–30 years we have learned that there are at least five known TMD etiologic factors that need to be considered. Occlusal factors remain as one of these factors, thereby maintaining TMDs as conditions that need a dental evaluation, but the manner by which the occlusion can affect the onset of a TMD must be revisited. Epidemiologic studies do not reveal a strong association between the static relationship of the teeth, such as Angle Class II or III, and the presence of a TMD [2, p. 102–28]. Therefore, in this chapter a new concept regarding the association between occlusion and TMDs will be presented.

The five etiologic factors that have gained significant research support are the occlusal condition, trauma, emotional stress, deep pain input, and parafunctional activity such as bruxism and clenching. Each will be briefly discussed in this section.

2.2.1 The Occlusal Condition

As mentioned earlier, occlusal factors have been thought to be associated with TMDs for many years. Even today this relationship is continuously debated, with proponents remaining on both sides of the discussion. Recent data do not support the traditional belief that the static relationship of the teeth is strongly associated with TMD (e.g., deep bites, class II, cross bites, eccentric contacts) [5]. Yet to believe that the occlusal condition could not influence masticatory system function and dysfunction seems rather naive. Perhaps instead of studying static occlusion relationships, one needs to investigate some of the dynamic functions of the masticatory system. There appears to be two ways the occlusal relationship of the teeth may be associated with TMD symptoms. The first is related to an acute change in the occlusal condition, and the second is related to loading of the masticatory structures in the absence of TM joint stability. Each will be further explained here.

2.2.1.1 An Acute Change in the Occlusal Condition

Every dentist has observed a situation when a crown or filling is placed and it is left a little high; afterward, the patient will often report back to the office complaining of discomfort. Frequently this discomfort is not only around the sore tooth, but there also is muscle tightness and pain. This occurs because in the presence of injury or even a threat of injury, the muscles protectively co-contract to minimize any damage. This muscle response can lead to pain, especially if the condition is prolonged. Once the offending restoration is corrected, the condition resolves. If it is not corrected in a reasonable amount of time, either the individual will adapt to the change (i.e., by tooth movement, altered biting, or avoidance), or a significant muscle TMD may develop.

2.2.1.2 Orthopedic Instability Coupled with Loading

There is a second mechanism by which the occlusal condition can contribute to a TMD. This relates to the degree of orthopedic stability in the masticatory system. Every mobile joint is designed to be loaded, and this loading comes from the muscles that pull across the joint. Therefore every joint has a musculoskeletally stable position, and in the TMJ this is defined as the condyles resting on the articular eminences with the disks correctly positioned between those articulating surfaces. Orthopedic stability in the masticatory system is present when the teeth are in their stable biting position at the same time the joints are in their stable position. When this is

present, joints and teeth can be loaded without injury or consequence.

However, when the stable joint position is not in harmony with the stable occlusal position, the condition is considered orthopedically unstable. If this were the critical factor leading to TMD, epidemiologic studies should reveal this relationship, but clearly this is not the case. Perhaps the missing element from those studies is the dynamics of loading. When the teeth are loaded by activities such as heavy biting, chewing, or bruxism, the joints need to be in a stable position. When this does not exist, continued loading can result in changes in the joint structures. Common types of changes are fibrous connective tissue breakdown, bony degeneration, clicking, locking, and pain. It is important to appreciate that a lack of orthopedic stability between the stable joint position and the stable occlusal position does not by itself lead to TMD; this only represents a risk factor. However, once this relationship is coupled with excessive loading in a susceptible patient, there is an increased risk of developing intracapsular disorders [2, p. 102–28].

Thus, it is interesting to note that occlusion can affect both muscle disorders and intracapsular disorders, but it does so through different mechanisms. The manner by which occlusion affects TMDs can be summarized by the following two statements: Problems that occur while bringing the teeth into occlusion, such as high restorations, are answered by the muscles. However, once the teeth have reached intercuspation, problems with loading are answered by the joints.

2.2.2 Trauma

Certainly trauma is a known etiology of certain TMDs. A single blow to the face can immediately change the structures of the joint, resulting in an intracapsular issue. Trauma seems to be more related to intracapsular disorders than muscle disorders. It is common to hear a patient report that "ever since I received the blow to my face, my TMJ has been clicking." Once joint pain begins, muscles protectively respond and then it may be difficult to separate the painful conditions. A sudden blow to the face represents macrotrauma. However, microtrauma can also be an issue whereby small but repeated traumas can occur to the joints. The orthopedic instability coupled with loading previously mentioned is an example of microtrauma.

2.2.3 Emotional Stress

There is ample evidence that increased levels of emotional stress can be an etiologic factor associated with TMDs. It has been demonstrated that individuals placed under acute emotional stress show slight increase in EMG activities of their masseter muscles [6]. This is normal, but if the stress is prolonged the muscle may show signs of fatigue, tightness, and pain. Prolonged stressors can result in an increase or upregulation of the autonomic nervous system [7]. When this occurs, the central nervous system can play an active role in maintaining the pain condition, making management more difficult.

2.2.4 Deep Pain Input

Deep pain input refers to any source of neural impulses that originate in the deep structures and lead to a pain experience. This excludes the skin and oral mucosa. Common sources of deep pain input are muscle and joint structures. Pain experienced in the deep structures has the unique characteristic of eliciting a muscle response, which is the same protective co-contraction response already discussed in the section on occlusion. The clinician must appreciate that deep pain input can have its origin in many structures [8]. A common example is cervical pain that elicits a masticatory muscle response. A patient who experiences a whiplash injury initially experiences only cervical pain. However, after a few days the pain will often radiate to the face, eliciting a muscle response that limits mouth opening. The clinical examination will reveal limited mouth opening and pain upon palpation of the muscles of mastication, which in fact is a TMD. However, this

TMD is secondary to another pain disorder and will continue until the primary source of pain is resolved. Clinicians often overlook this relationship and question why their therapies that have been directed to the masticatory structures (such as an occlusal appliance) do not resolve the pain.

2.2.5 Parafunctional Activities

For many years dentists have focused on bruxing and clenching as a significant etiologic factor associated with TMDs. Although this activity can certainly be related, it is not as strongly linked as once believed. We know that bruxing and clenching of the teeth can produce pain [9]. However, sleep studies reveal that most individuals put their teeth together during sleep, often with no pain associated. We have also learned that the patients' occlusal relationships are not strongly related to these parafunctional activities. Instead, they are more correlated with sleep stages and other aspects of the sleep cycle. We have also learned that many individuals clench their teeth during the day with very little awareness. Patients who report that they wake up in the morning with painful muscle are certainly likely to be experiencing sleep related bruxism, and in those cases that can be considered as an etiologic relationship. However, there are other patients who report no pain upon awakening but instead their pain is in the late afternoons or evening. These individuals may be experiencing daytime clenching, or they may have a completely different etiologic basis for their myogenous pain. It is important to appreciate that these activities are different and likely to respond to different treatment strategies [2, p. 291–316].

The five etiologic factors that have been reviewed above reveal that TMDs are a complex group of conditions that are influenced by multiple factors. Making it even more complicated is the fact that more than one of these factors may be involved at any given time, which is often the case. This becomes a real challenge for the clinician attempting to initiate treatment strategies. Furthermore, we need to recognize the fact that all individuals are different in their capacity to adapt to less than ideal circumstances. Most people have less than perfect occlusion, have received some trauma, have some emotional stress, have experienced deep pain, and have some parafunction, and yet they do not develop TMD symptoms. This is likely due to their capacity for adaptability, which is an important clinical consideration since it helps us understand the great variability of patient responses. Clinicians need to appreciate patient adaptability, since it is probably the major reason for our clinical success. Yet, we actually know little about this important issue.

A better understanding of human adaptability would likely lead to better selection of treatment and prediction of outcomes. A more complete understanding of adaptability would be helpful, but investigating this concept is certainly not an easy task. There are likely many variables that contribute to adaptability. A few of the factors may include the individual's biology, learned experiences, psychological conditions (e.g., obsessive compulsive disorders), and genetics.

Some recent genetic studies are offering interesting insights about adaptability, especially with respect to pain. It has been demonstrated that variations in genetic makeup may have significant impact on pain perception [10, 11]. The gene that encodes for catechol-O-methyltransferase (COMT), an enzyme associated with pain responsiveness, varies in patients. It has been shown that for this gene, there are three clusters of individuals who respond differently to painful stimuli. Some individuals are more pain sensitive, while others less pain sensitive. In an interesting prospective cohort study of 186 orthodontically treated females, patients who were genetically in the pain sensitive cluster developed more TMD symptoms than the pain insensitive cluster group [12]. This suggests that the actual orthodontic therapy was not the significant factor in developing TMD; instead, it was performing orthodontic therapy in the patient with a genetically determined pain sensitive haplotype. Perhaps future research will help us recognize the patients who are more vulnerable to develop pain disorders, which may affect our choices of treatment options.

When considering all these issues, assuming that orthodontic therapy is completely unrelated to TMD is a relatively naïve thought. The question that really needs to be asked is how can orthodontic therapy be used to minimize any risk factors that may relate to TMD? In reviewing the known etiologies of TMD, orthodontic therapy routinely affects only one of those factors: occlusion. However, it is clear that occlusion factors are not always related to TMD [2, p. 102–28, 13]. So, where does orthodontic therapy fit in the big picture of TMD? Since occlusal factors may be a potential source of TMD in some patients, it would seem logical that the orthodontist should develop an occlusion condition that will minimize any risk factors that might be associated with TMD. However, developing a sound occlusal relationship does not mean the patient will not develop TMD, because there are at least four other etiologies that are outside the control of the orthodontist. Developing an orthopedically stable occlusal condition should be thought of as minimizing a dental risk factor. It seems logical that since orthodontic therapy will change the patient's occlusal relationships, emphasis should be placed on creating an occlusion condition that will provide the best opportunity for successful masticatory function for the lifetime of the patient.

2.3 Classification of Temporomandibular Disorders

Most temporomandibular disorders fall into one of two broad categories: muscle pain disorders or intracapsular disorders. Muscle pain disorders are by far the more common of these two problems [4, 14]. This is not surprising, since all humans experience some type of muscle pain periodically throughout their lives.

Masticatory muscle pain complaints are very common in patients seeking treatment in the dental office. With regard to orofacial pain, they are second only to odontalgia (i.e., tooth or periodontal pain) in terms of frequency. They are generally grouped in a large category known as masticatory muscle disorders [2, p. 291–316]. The most common symptoms reported by patients with these muscle disorders are pain associated with functional activities (i.e., chewing) and dysfunction (limitation in mouth opening).

2.3.1 Masticatory Muscle Disorders

Patients who experience masticatory muscle pain will describe the pain in terms of ranging from slight tenderness to extreme discomfort. Although muscle pain is common, dentists have generally not been taught well regarding its etiology. In fact, most dentists have been taught that muscle pain is a reflection of a structural problem, such as a poor occlusion or an incorrect joint position. They also associate muscle pain with bruxing and clenching activities. Although some of these thoughts may be true for some patients, they are not the etiologic basis for myogenous TMDs in a large number of patients.

Some muscle pain may arise from increased levels of muscular use. The symptoms are often associated with a feeling of muscle fatigue and tightness. Although the exact origin of this type of muscle pain is debated, some researchers have reported that it is related to vasoconstriction of the relevant nutrient arteries and the accumulation of metabolic waste products in the muscle tissues. Within the ischemic area of the muscle certain algogenic substances (e.g., bradykinins, prostaglandins) are released, causing muscle pain [15–20].

Muscle pain, however, is far more complex than simple overuse and fatigue. In fact, muscle pain associated with most TMD does not seem to be strongly correlated with increased activity such as spasm, or even with the burden of daily activities like hard chewing, cheerleading, singing, etc. [6, 21–24]. It is now appreciated that muscle pain can be greatly influenced by central nervous system mechanisms [16, 25, 26], especially if it has been present for a prolonged period of time.

One of the significant clinical findings with muscle pain disorders is that the pain is increased with function. Therefore, patients often report

that the pain affects their ability to chew and even talk. However, these functional activities are not usually the cause of the disorder, but instead they heighten the patient's awareness of it. More likely some other type of activity or a central nervous system effect has led to the muscle pain [27]. Therefore, directing treatment toward the functional activity itself will not be appropriate or successful. Instead, treatment needs to be directed towards diminishing the CNS effects and/or possibly muscle hyperactivity.

Many clinicians consider all masticatory muscle disorders to be the same. If this were the case it would certainly make treatment considerations quite simple. However, experienced clinicians realize that this is not the case, since all muscle pain disorders do not successfully respond to the same treatment. There are at least five different clinical presentations of muscle pain, and being able to distinguish among them is important because the treatment of each is quite different. The five types are protective co-contraction (muscle splinting), local muscle soreness, myofascial (trigger point) pain, myospasm, and chronic centrally mediated myalgia [2, 27, p. 129–69]. The first three conditions (protective co-contraction, local muscle soreness, and myofascial pain) are commonly seen in the dental office while myospasm and chronic centrally mediated myalgia are less frequently seen. Many of these muscle disorders appear and resolve in a relatively short period of time, sometimes without any type of professional treatment. For most cases a conservative regimen of treatment will be successful. However, when these conditions do not resolve, more chronic pain disorders may result. Chronic masticatory muscle disorders become more complicated and treatment is generally oriented differently than for acute problems. It therefore becomes important that the clinician be able to separate acute muscle disorders from chronic disorders so that proper therapy can be applied.

Since the intent of this chapter is not to review all muscle conditions, only the most common disorders orthodontists will encounter in their practices will be discussed. These are local muscle soreness and myofascial pain.

2.3.1.1 Local Muscle Soreness

Local muscle soreness is the most common type of acute muscle pain seen in the dental practice. It represents a condition that is characterized by changes in the local environment of the muscle tissues. These changes arise from the release of certain algogenic substances (i.e., bradykinin, substance P, histamine [28]) that produce pain. These initial changes may represent nothing more than fatigue. The most likely causes of local muscle soreness are overuse of the muscle or trauma. Overuse may be associated with a protective co-contraction of the muscle secondary to an acute change in sensory input or emotional stress. Trauma may be caused by a direct blow to the muscle, but a more likely reason is simply unaccustomed use of the muscle. When excessive use is the etiology a delay in the onset of muscle soreness can occur [29]. This type of local muscle soreness is often referred to as delayed onset muscle soreness or post exercise muscle soreness [30–34].

Clinically, local muscle soreness TMD patients present with muscles that are tender to palpation and they report increased pain with function. When the elevator muscles are involved, the patient will report limited mouth opening which is secondary to the pain. This means the patient can open wider but is not willing to do this because it increases the pain. The patient may also report muscle weakness [35–37] which usually will be returned to normal strength once the muscle soreness has been resolved [36–38].

2.3.1.2 Myofascial Pain

Myofascial pain is a regional myogenous pain condition characterized by local areas of firm, hypersensitive bands of muscle tissue known as "trigger points." Myofascial pain is common yet not widely appreciated or completely understood. In one study [39] more than 50 % of the patients reporting to a university pain center were diagnosed as having this type of pain.

The trigger points are often felt as taut bands when palpated, which elicit pain. The exact nature of a trigger point is not known. It has been suggested [40–42] that certain nerve endings in the muscle tissues may become sensitized by algogenic substances that create a localized zone of

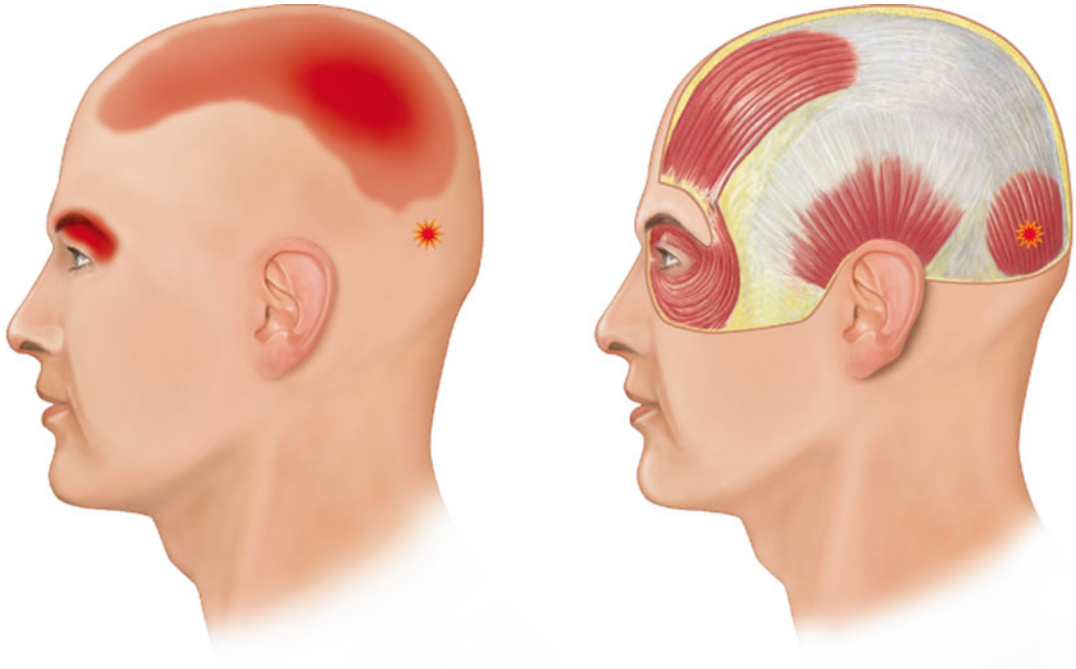


Fig. 2.1 Note how a trigger point (marked with X) in the occipital belly of the occipitofrontalis muscle produces referred headache pain behind the eye (in red) (From Okeson [2], p. 133)

hypersensitivity [43]. There may be a local temperature rise at the site of the trigger point, suggesting an increase in metabolic demand and/or reduction of blood flow to these tissues [44, 45]. A trigger point is a very circumscribed region in which just a relatively few motor units seem to be contracting [46].

The unique characteristic of trigger points is that they are a source of constant deep pain and therefore can produce central excitatory effects. If a trigger point centrally excites a group of converging afferent interneurons, referred pain will often result, generally in a predictable pattern according to the location of the involved trigger point (Figs. 2.1 and 2.2) [2, 47, p. 21–45]. The pain is often reported by the patient as headache pain. In many instances patients may be aware only of the referred pain and not even acknowledge the trigger points. A perfect example is the patient suffering from myofascial trigger point pain in the trapezius muscle that creates referred pain to the temple region (Fig. 2.3) [47–49]. The chief complaint is temporal headache, with very little acknowledgment of the trigger point in the shoulder. This clinical presentation can easily distract the clinician from the source of the problem. The patient will draw the clinician's attention to the site of the pain (the temporal headache) and not the origin of the pain.

2.3.2 Temporomandibular Joint Disorders

Functional abnormalities of the temporomandibular joints are probably the most common findings one observes when examining a patient for masticatory dysfunction. The reason for this is due to the high prevalence of signs, and not necessarily symptoms. (See Chap. 3 for discussion of the significance of signs and symptoms discovered during TMD screening exams). Many of the signs such as joint sounds or deviated opening are not painful, and therefore the patient may not seek treatment. These TM joint disorders generally fall into two broad categories: Internal derangements and inflammatory joint disorders. These conditions will be described separately.

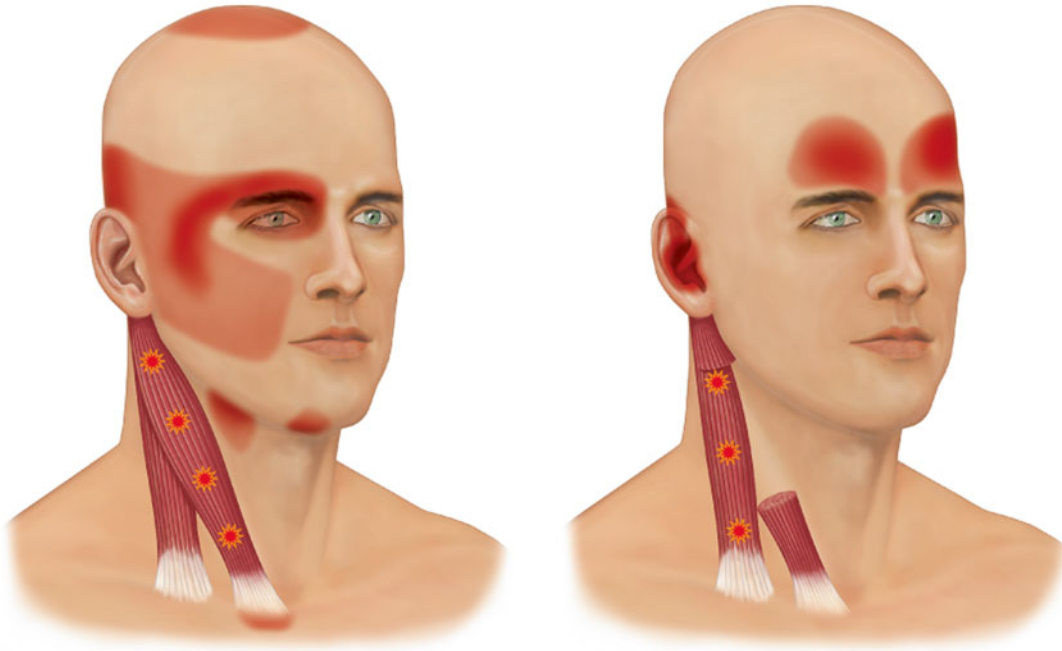


Fig. 2.2 Note how trigger points located in the sternocleidomastoideus refer pain to the preauricular (TMJ) area, the eye, the forehead, and the ear (From Okeson [2], p. 135)

2.3.2.1 Internal Derangements

Internal derangements represent a group of functional disorders that arise from abnormalities in the anatomy and/or positional relationships of the TM joint structures. A review of TMJ anatomy demonstrates that the disc is attached to the poles of the condyle by the medial and lateral collateral ligaments. Many internal derangement disorders arise from alterations of the integrity or lengths of these ligamentous attachments. Once these ligaments become elongated the disc is allowed more freedom to move within the joint. The disc will often begin to assume an anteromedial position in relationship with the condyle (Fig. 2.4). When the disc is in this more forward and medial position, function of the joint can be somewhat altered. As the mouth opens and the condyle moves forward, a short distance of translatory movement can occur between the condyle and the disc until the condyle once again assumes its normal position on the thinnest area of the disc (intermediate zone). Once it has translated over the posterior surface of the disc to the intermediate zone, inter-articular pressure due to joint loading maintains this relationship and the disc is again carried forward with the condyle through the remaining portion of the translatory movement. Upon closing, the disc reassumes its abnormal position on the condyle in the closed joint position. Once in the closed joint position, the disc is again free to move according to the demands of its functional attachments. In this condition, the disc will assume the most anteromedial position allowed by the discal attachments and its own morphology.

As the disc is more chronically repositioned forward and medially by action of the superior lateral pterygoid muscle, the discal ligaments are further elongated. With continuous thinning of the posterior border of the disc and further elongation of the disc ligaments, the disc can move through the discal space and be trapped anterior to the condyle. (Fig. 2.4). When this occurs, the condyle can now function or load the retrodiscal tissues which may be associated with pain.

The important feature of this functional relationship is that the condyle translates across the disc to some degree when movement begins. This type of movement does not occur in the normal joint. During such movement the increased

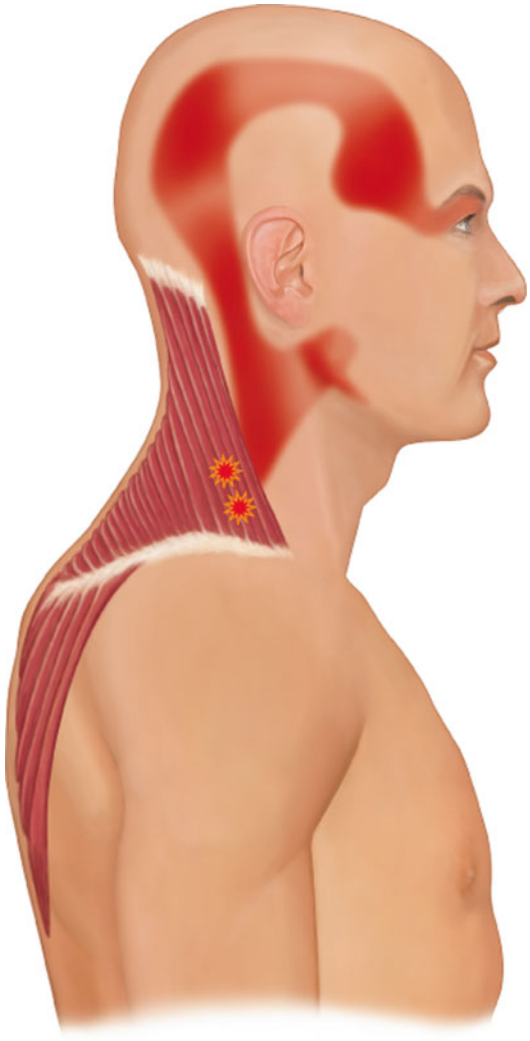


Fig. 2.3 Note how trigger points located in the trapezius muscle (marked with X) refer pain to behind the ear, the temple, and the angle of the jaw (From Okeson [2], p. 134)

interarticular pressure may prevent the articular surfaces from sliding across each other smoothly. The disc can stick or be bunched slightly, causing an abrupt movement of the condyle over it into the normal condyle-disc relationship. A clicking sound often accompanies this abrupt movement. Once the joint has clicked, the normal relationship of the disc and condyle is reestablished and this relationship is maintained during the rest of the opening movement. A second click can occur as the disc is re-displaced during the later stages of closing the mouth. This is called "reciprocal clicking" [50]. As the disc displacement progresses, the condyle actually begins to function behind the disc with loading occurring on the retrodiscal tissues (Fig. 2.4).

As the disc is more chronically repositioned forward and medially, the discal ligaments are further elongated. With continuous thinning of the posterior border of the disc and further elongation of the disc ligaments, the disc can move further forward and be trapped anterior to the condyle. This often is accompanied by folding of the disc into a ball-like shape. When this occurs it will initially lead to a decrease in how far the condyle can move forward. Therefore the patient will have the sensation that he or she cannot open the mouth completely. This has been called a "closed lock" (Fig. 2.5) [50] since the patient feels he or she is locked near the closed mouth position. Patients may report pain when the mandible is moved to the point of limitation, but pain does not always accompany this condition [51–54].

If a closed lock continues, the condyle will be constantly positioned on the retrodiscal tissues. These tissues are not anatomically structured to accept forces, but they often remodel to form a functional pseudodisc. However, as force is applied, some likelihood arises that these tissues may break down in some patients [55–57]. With this breakdown comes tissue inflammation and pain (retrodiscitis).

It is important to appreciate that pain is not always a factor with these conditions. Pain is not generated by the disc, since it is aneural. The structures that are able to produce pain are the connective tissues such as the ligaments and the very highly innervated retrodiscal tissues. If these structures are loaded quickly due to a sudden disc shifting, pain is likely. However, if the changes occur slowly over time, these tissues are often able to adapt and pain may not be associated.

Etiology of Internal Derangements

Any condition or event that leads to elongation of the discal ligaments or thinning of the disc can cause these derangements of the condyle-disc complex disorders. Certainly one of the most common factors is trauma. Two general types of trauma need to be considered: macrotrauma and

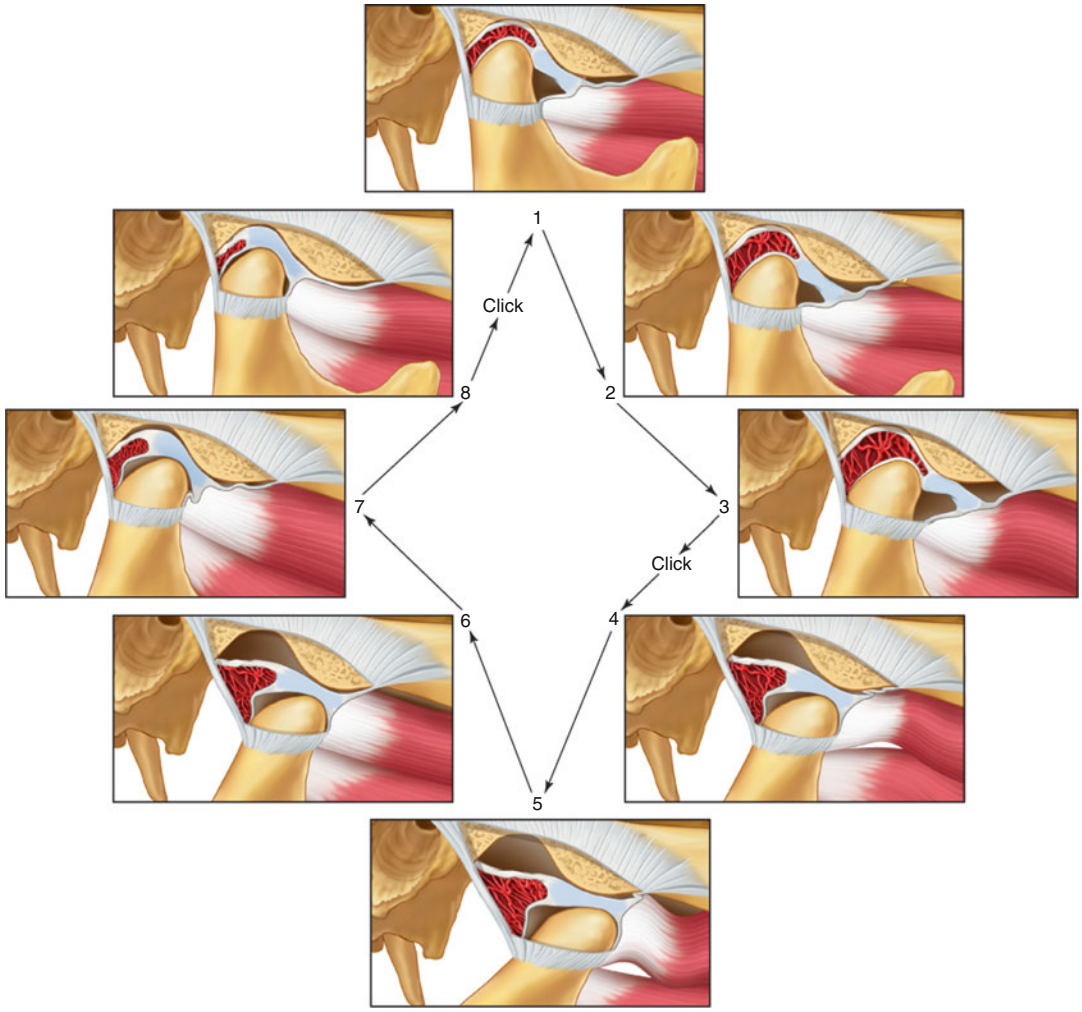


Fig. 2.4 Functional displacement of the disc with reduction. Note that while the mouth is closed (stage 1) the disc is displaced anterior to the condyle. During opening the condyle passes over the posterior border of the disc onto the intermediate area of the disc, thus reducing the displaced disc (stage 4). At this stage a click is felt. During the rest of the opening movement the condyle and disc function normally. During closing the disc is re-displaced (stage 8–1) and a second click is felt (*reciprocal click*) (From Okeson [2], p. 145)

microtrauma. Macrotrauma relates to a sudden blow to the face that can result in a quick elongation of ligaments. This is well documented in the literature [58–71].

Microtrauma represents lower levels of force but repeated over longer periods of time. It can result from joint loading associated with muscle hyperactivity such as bruxism or clenching [72, 73]. This may be especially true if the bruxing activity is intermittent and the tissues have not had an opportunity to adapt. It is likely that if the bruxing is long standing, the articular tissues have adapted to the loading forces and changes will not be seen. In fact, in most patients gradual loading of the articular surfaces leads to an adaptive, more tolerant articular tissue [74–76].

Microtrauma may be the result of mandibular loading in the presence of orthopedic instability, as stated in an earlier section. When orthopedic instability exists, repeated loading, such as clenching the teeth, can lead to slight movements of the condyle resulting in microtrauma to the ligaments. The results can be elongation of these ligaments and eventually

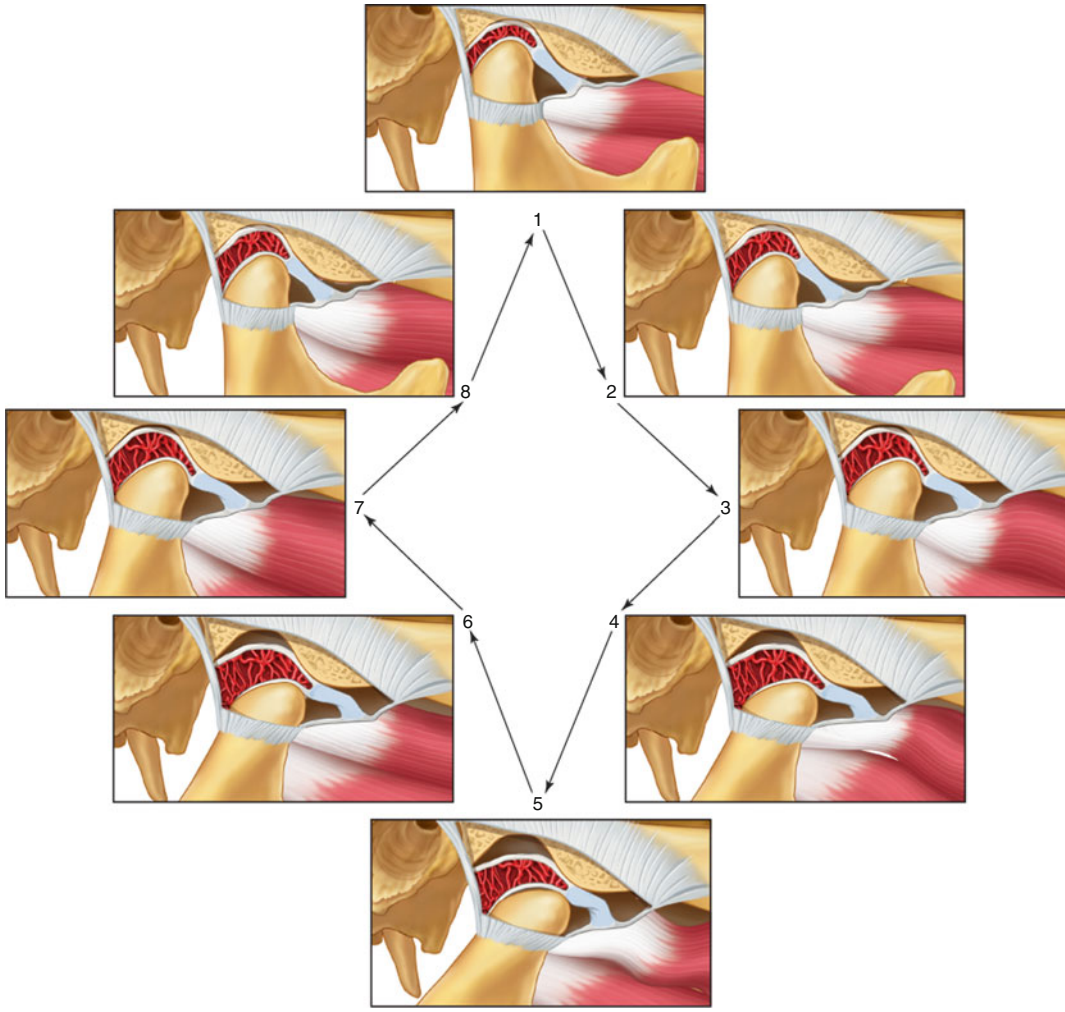


Fig. 2.5 Functional displacement of the disc without reduction Note that during opening the condyle never assumes a normal relationship on the disc but instead causes the disc to move forward ahead of it. This condition limits the distance it can translate forward (*closed lock*). Clicking often resolves when this occurs (From Okeson [2], p. 146)

disc movements. Remember that the amount and intensity of the loading greatly influence whether the orthopedic instability will lead to a disc derangement disorder. Bruxing patients with orthopedic instability, therefore, are more likely to develop problems than non-bruxers with the same occlusion.

An important question that arises in dentistry is "What occlusal conditions are commonly associated with internal derangements?" The most orthopedically stable position of the condyle is in the superior anterior position resting against the posterior slope of the articular eminence. This is referred to as the musculoskeletally stable position [2, p. 291–316] and it is determined by the loading forces of the elevator muscles. It has been demonstrated that when an occlusal condition causes a condyle to be positioned posterior to the musculoskeletally stable position the posterior border of the disc can be thinned [77]. A common occlusal condition that has been suggested by some orthodontists to produce this problem is the skeletal Class II deep-bite malocclusion; advocates of this concept also

believe that this situation may be further aggravated when a Division 2 anterior relationship also exists [78–82]. However, most studies show no relationship between Class II malocclusion and these disorders [13, 83–89]. Other studies show no association between the horizontal and vertical relationship of the anterior teeth and disc derangement disorders [90–94]. While occlusal conditions are not the main etiologic factors for internal derangements, the important feature of an occlusal condition that leads to disc derangement disorders is the lack of joint stability when the teeth are tightly occluded. Therefore, it is likely that some Class II malocclusions provide joint stability (a stable malocclusion) while others do not; but the same can be said for every type of static malocclusion category.

It is obvious that no simple relationship exists between orthopedic instability and intracapsular disorders. It is very important, however, that when orthopedic instability exists, it be identified as a potential etiologic factor. It should be noted that orthodontic therapy can be a viable treatment for orthopedic instability and may need to be considered when this instability has been determined to be a contributing factor to a TMD.

2.3.2.2 Osteoarthritis

When internal derangements of the TMJ occur, the structures affected by these changes often respond. The most common tissues affected are the retrodiscal tissues and the articular surfaces of the condyle and articular eminence. These changes can result in adaptation or destruction, depending upon many factors. Some of these factors are the acuteness of the changes as well as the intensity and duration of the loading. There are also important biologic and genetic factors that may regulate the patient's ability to repair tissues.

If the articular surfaces of the condyle become affected, the subarticular bone receives additional loading and changes can occur. Similar changes also can occur in the absence of internal derangements. These changes represent a group of disorders that are considered joint arthritides. The most common type of TMJ arthritis is osteoarthritis (sometimes called degenerative joint disease). Osteoarthritis represents a destructive process by which the bony articular surfaces of the condyle and fossa become altered. It is generally considered to be the body's response to increased loading of a joint [95]. As loading forces continue and the articular surface becomes softened (chondromalacia), the subarticular bone begins to resorb. Progressive degeneration eventually results in loss of the subchondral cortical layer, bone erosion, and subsequent radiographic evidence of osteoarthritis [96]. It is important to note that radiographic changes are only seen in later stages of osteoarthritis and may not reflect the clinical symptoms accurately.

Osteoarthritis is often painful, and jaw movement accentuates the symptoms. Crepitation (multiple grating joint sounds) is a common finding with this disorder. Osteoarthritis can occur any time the joint is overloaded, but is most commonly associated with disc displacements [97, 98] or perforation [99]. Once the disc is displaced and the retrodiscal tissues break down, the condyle begins to articulate directly with the fossa accelerating the destructive process. In time, the dense fibrous articular surfaces are destroyed and bony changes occur. Radiographically, the surfaces seem to be eroded and flattened. Any movement of these surfaces creates pain, so jaw function usually becomes very restricted. Although osteoarthritis is in the category of inflammatory disorders, it is not a true inflammatory condition. With appropriate treatment and reduction of joint loading, the arthritic condition can become adaptive. The adaptive stage has been referred to as osteoarthrosis [95, 100].

Other types of arthritides can certainly affect the temporomandibular joint. The most common after osteoarthritis is rheumatoid arthritis. This is thought to be an autoimmune disorder and therefore has its origin in systemic factors. The juvenile form of this problem can produce significant joint changes as well as serious occlusal problems. Other common causes of TMJ arthritis are traumatic arthritis, infectious arthritis, psoriatic arthritis, and hyperuricemia (gout). These conditions are not reviewed in this chapter. Other texts can be reviewed for a more thorough review of TMJ arthritides [2, p. 317–61].

2.4 Summary of the Continuum of TMJ Intracapsular Conditions

Disorders of the temporomandibular joints may follow a path of progressive events, i.e., a continuum, from the initial signs of dysfunction to osteoarthritis. They are summarized in Fig. 2.6.

Although this continuum is logical, the question must be asked whether these stages are always progressive for every patient. It is a question of great significance, because if all patients continue to progress in this manner, then there would be a professional obligation to resolve any joint symptoms as soon as they first appear. The sequence of breakdown as summarized in Fig. 2.6 is logical and has clinical support [101–103]. However, clinical longitudinal studies have clearly shown that some intracapsular TMD patients will present in one stage but may not necessarily progress to the next. At any given stage of disc derangement the patient may reach a level of adaptability and no further progression or breakdown will occur [104, 105]. This can be supported by histories of asymptomatic single and reciprocal clicks over many years [106]. Perhaps the key to determining who needs treatment lies in the obvious progression from one stage to the next. Also the presence of pain is important, since it implies continuous breakdown; in any case, the pain should be treated for its own sake. Therefore, it is this

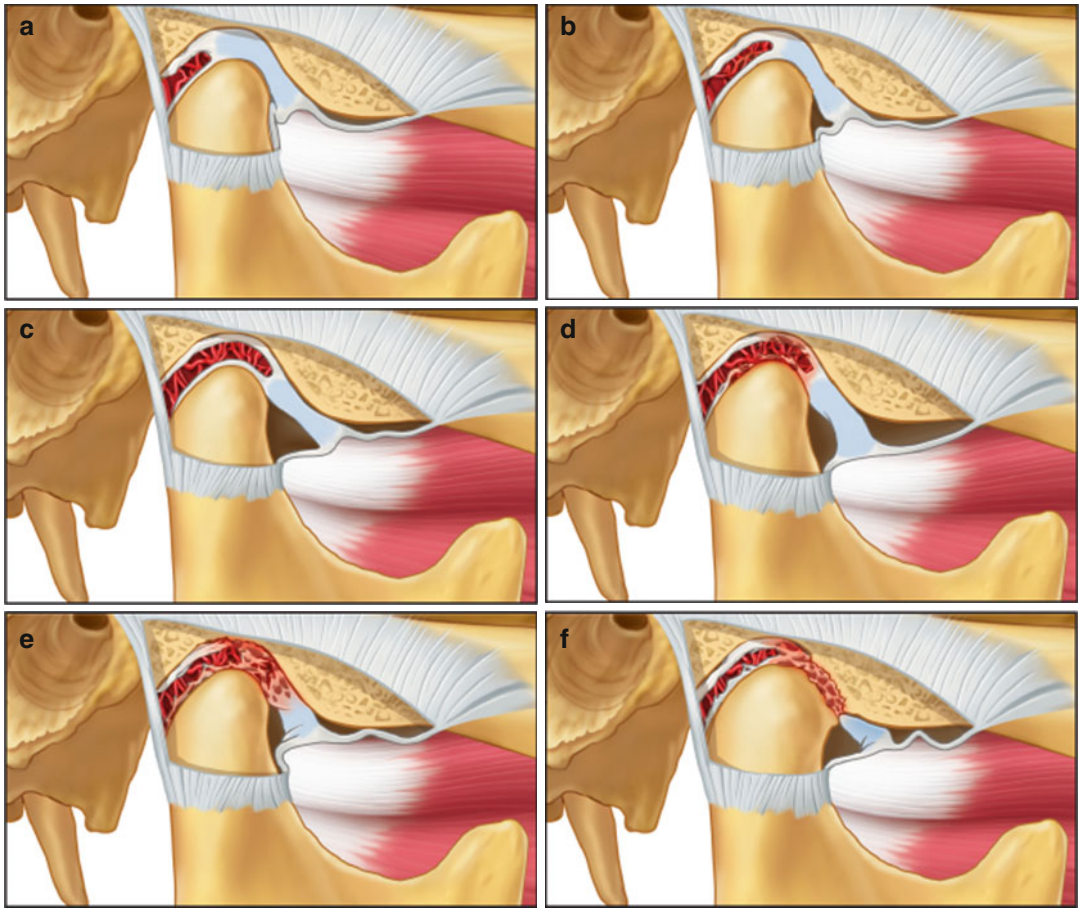


Fig. 2.6 Various states of internal derangement of the TMJ. (**a**) normal joint, (**b**) partial displacement of the disc, (**c**) complete displacement of the disc, (**d**) impingement of retrodiscal tissues, (**e**) retrodiscitis and tissue breakdown, (**f**) osteoarthritis (From Okeson [2], p. 156)

author's opinion that treatment for such patients needs to be instituted when pain is associated with the condition. The treatment should be directed toward controlling pain and changing loading, thereby allowing a better opportunity for the tissues to repair and eventually adapt. Treatment of these conditions is beyond the objectives of this chapter and therefore other sources should be pursued.

Conclusion

Temporomandibular disorders are common conditions that may be encountered by orthodontists: during pre-treatment screening, during their treatment procedures, or during orthodontic retention. Every orthodontist needs to have a basic understanding of these musculoskeletal disorders so that he or she can respond to their patients' needs. The purpose of this chapter was to present the pathophysiology, etiology, and clinical characteristics of the most common temporomandibular disorders. The goal was not to provide detailed therapeutic considerations for TMDs. However, it should be mentioned that most TMDs can be successfully managed by very conservative, reversible treatments. This should always be the initial approach before any irreversible treatments are considered.

Although some patients with TMDs may respond to orthodontic therapy, most will not because their occlusal conditions are not the cause of their symptomatology. Also, the positive responses seen may simply be due to placebo effects, spontaneous improvements, or the passage of time. Therefore, the orthodontist needs to understand which TMD patients will benefit before any orthodontic therapy is begun, and that treatment should not be initiated until acute symptoms of pain and dysfunction have been addressed. A review of the five etiologic factors presented in this chapter reveals that orthodontic therapy potentially affects only one of them, i.e., the occlusal condition. The manner by which orthodontic therapy affects this factor is by providing orthopedic stability, because proper orthodontic therapy can provide a stable occlusal position in the most stable joint position. Accomplishing this provides orthopedic stability in the masticatory structures, which minimizes risk factors associated with TMDs. Since orthodontic treatment will always disrupt the existing occlusal and TMJ relationships, establishing this orthopedic stability at the end of treatment should be the goal for every patient who receives orthodontic therapy.

When a patient presents to the orthodontist with a TMD, the etiology of the TMD needs to be determined before any treatment is begun. Assuming that the patient's malocclusion is the major etiologic factor causing the TMD is a very naïve assumption. Nothing is more discouraging to the patient (and doctor) than to provide excellent orthodontic treatment for 2 years and then hear the patient report that the pain is still present. Although the orthodontic therapy may have been successful, the orthodontist has failed to successfully treat the patient. Therefore, before beginning orthodontic therapy on any symptomatic patient, the clinician needs to confirm that orthopedic instability is an etiology for that patient, because this is the only scenario in which orthodontics can be considered as an appropriate treatment for TMD.

Take Home Messages

- TMD signs and symptoms are common in the general population, but only a small percentage of those require treatment.
- Orthodontists need to be aware how their treatments can affect masticatory function.
- There are five recognized etiologic factors associated with TMD.
- Muscle pain is the most common painful TMD encountered in the orthodontic practice.

- Internal derangements are associated with TMJ conditions and joint sounds are the most common sign.
- Most TMD symptoms can be managed by conservative approaches.
- Treatment goals for all orthodontists should include developing or maintaining orthopedic stability in the masticatory system.

References

1. de Leeuw R, Glasser G. Orofacial pain: guidelines for classification, assessment, and management. 5th ed. Chicago: Quintessence Publ. Co.; 2013.
2. Okeson JP. Management of temporomandibular disorders and occlusion. 7th ed. St. Louis: Elsevier/Mosby Publishers; 2013.
3. De Kanter RJAM, Kayser AF, Battistuzzi PGFCM, Truin GJ, Van T, Hol MA. Demand and need for treatment of craniomandibular dysfunction in the Dutch adult population. J Dent Res. 1992;71:1607–12.
4. Schiffman EL, Fricton JR, Haley DP, Shapiro BL. The prevalence and treatment needs of subjects with temporomandibular disorders. J Am Dent Assoc. 1990;120(3):295–303.
5. Pullinger AG, Seligman DA. The degree to which attrition characterizes differentiated patient groups of temporomandibular disorders. J Orofac Pain. 1993;7(2):196–208.
6. Carlson CR, Okeson JP, Falace DA, Nitz AJ, Curran SL, Anderson DT. Comparison of psychologic and physiologic functioning between patients with masticatory muscle pain and matched controls. J Orofac Pain. 1993;7:15–22.
7. Grassi C, Passatore M. Action of the sympathetic system on skeletal muscle. Ital J Neurol Sci. 1988;9(1):23–8.
8. Okeson JP. Bell's oral and facial pain. 7th ed. Chicago: Quintessence Publishers; 2014. p. 13–40.
9. Kobs G, Bernhardt O, Kocher T, Meyer G. Oral parafunctions and positive clinical examination findings. Stomatologija/issued by public institution "Odontologijos studija" [et al]. 2005;7(3):81–3.
10. Smith SB, Maixner DW, Greenspan JD, Dubner R, Fillingim RB, Ohrbach R, et al. Potential genetic risk factors for chronic TMD: genetic associations from the OPPERA case control study. J Pain Off J Am Pain Soc. 2011;12(11 Suppl):T92–101.
11. Segall SK, Maixner W, Belfer I, Wiltshire T, Seltzer Z, Diatchenko L. Janus molecule I: dichotomous effects of COMT in neuropathic vs nociceptive pain modalities. CNS Neurol Disord Drug Targets. 2012;11(3):222–35.
12. Slade GD, Diatchenko L, Ohrbach R, Maixner W. Orthodontic treatment, genetic factors and risk of temporomandibular disorder. Semin Orthod. 2008;14(2):146–56.
13. McNamara Jr JA, Seligman DA, Okeson JP. Occlusion, orthodontic treatment, and temporomandibular disorders: a review. J Orofac Pain. 1995;9(1):73–90.
14. McCreary CP, Clark GT, Merril RL, et al. Psychological distress and diagnostic subgroups of temporomandibular disorder patients. Pain. 1991;44:29–34.
15. Keele KD. A physician looks at pain. In: Weisenberg M, editor. Pain; clinical and experimental perspectives. St Louis: The CV Mosby Co; 1975. p. 45–52.
16. Svensson P, Graven-Nielsen T. Craniofacial muscle pain: review of mechanisms and clinical manifestations. J Orofac Pain. 2001;15(2):117–45.
17. Mense S. The pathogenesis of muscle pain. Curr Pain Headache Rep. 2003;7(6):419–25.
18. Simons DG. New views of myofascial trigger points: etiology and diagnosis. Arch Phys Med Rehabil. 2008;89(1):157–9.
19. Mense S. Algesic agents exciting muscle nociceptors. Experimental brain research Experimentelle Hirnforschung Experimentation cerebrale. 2009;196(1):89–100.
20. Okeson JP. Bell's oral and facial pain. 7th ed. Chicago: Quintessence Publishers; 2014. p. 41–54.
21. Lund JP, Widmer CG. Evaluation of the use of surface electromyography in the diagnosis, documentation, and treatment of dental patients. J Craniomandib Disord. 1989;3(3):125–37.
22. Lund JP, Widmer CG, Feine JS. Validity of diagnostic and monitoring tests used for temporomandibular disorders [see comments]. J Dent Res. 1995;74(4):1133–43.
23. Paesani DA, Tallents RH, Murphy WC, Hatala MP, Proskin HM. Evaluation of the reproducibility of rest activity of the anterior temporal and masseter muscles in asymptomatic and symptomatic temporomandibular subjects. J Orofac Pain. 1994;8:402–6.
24. Curran SL, Carlson CR, Okeson JP. Emotional and physiologic responses to laboratory challenges: patients with temporomandibular disorders versus matched control subjects. J Orofac Pain. 1996;10(2):141–50.
25. Mense S. Considerations concerning the neurobiological basis of muscle pain. Can J Physiol Pharmacol. 1991;69(5):610–6.
26. Mense S. Nociception from skeletal muscle in relation to clinical muscle pain. Pain. 1993;54(3):241–89.
27. Okeson JP. Bell's oral and facial pain. 7th ed. Chicago: Quintessence Publishers; 2014. p. 287–326.
28. Watanabe M, Tabata T, Huh JI, Inai T, Tsuboi A, Sasaki K, et al. Possible involvement of histamine in muscular fatigue in temporomandibular disorders: animal and human studies. J Dent Res. 1999;78(3):769–75.
29. Christensen LV, Mohamed SE, Harrison JD. Delayed onset of masseter muscle pain in experimental tooth clenching. J Prosthet Dent. 1982;48(5):579–84.
30. Abraham WM. Factors in delayed muscle soreness. Med Sci Sports. 1977;9:11–20.

31. Tegeder L, Zimmermann J, Meller ST, Geisslinger G. Release of algesic substances in human experimental muscle pain. Inflamm Res Off J Europ Histamine Res Soc [et al]. 2002;51(8):393–402.
32. Evans WJ, Cannon JG. The metabolic effects of exercise-induced muscle damage. Exerc Sport Sci Rev. 1991;19:99–125.
33. Byrnes WC, Clarkson PM. Delayed onset muscle soreness and training. Clin Sports Med. 1986;5(3):605–14.
34. Bobbert MF, Hollander AP, Huijing PA. Factors in delayed onset muscular soreness of man. Med Sci Sports Exerc. 1986;18(1):75–81.
35. Bakke M, Michler L. Temporalis and masseter muscle activity in patients with anterior open bite and craniomandibular disorders. Scand J Dent Res. 1991;99(3):219–28.
36. Tzakis MG, Dahlstrom L, Haraldson T. Evaluation of masticatory funciton before and after treatment in patients with craniomandibular disorders. J Craniomandib Disord Facial Oral Pain. 1992;6:267–72.
37. Sinn DP, de Assis EA, Throckmorton GS. Mandibular excursions and maximum bite forces in patients with temporomandibular joint disorders. J Oral Maxillofac Surg. 1996;54(6):671–9.
38. High AS, MacGregor AJ, Tomlinson GE. A gnathodynanometer as an objective means of pain assessment following wisdom tooth removal. Br J Maxillofac Surg. 1988;26:284.
39. Fricton JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral Surg Oral Med Oral Pathol. 1985;60(6):615–23.
40. Simons DG, Travell J. Myofascial trigger points, a possible explanation [letter]. Pain. 1981;10(1):106–9.
41. Mense S, Meyer H. Bradykinin-induced sensitization of high-threshold muscle receptors with slowly conducting afferent fibers. Pain. 1981;(Suppl 1):S204.
42. Simons DG, Mense S. Understanding and measurement of muscle tone as related to clinical muscle pain. Pain. 1998;75(1):1–17.
43. McMillan AS, Blasberg B. Pain-pressure threshold in painful jaw muscles following trigger point injection. J Orofac Pain. 1994;8(4):384–90.
44. Travell J. Introductory comments. In: Ragan C, editor. Connective tissues transactions of the fifth conference. New York: Josiah Macy, Jr; 1954. p. 12–22.
45. Simons DG, Travell JG, Simons LS. Travell & Simons' myofascial pain and dysfunction: a trigger point manual. 2nd ed. Baltimore: Williams & Wilkins; 1999. p. 67–78.
46. Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. Spine. 1993;18(13):1803–7.
47. Fernandez-de-Las-Penas C, Galan-Del-Rio F, Alonso-Blanco C, Jimenez-Garcia R, Arendt-Nielsen L, Svensson P. Referred pain from muscle trigger points in the masticatory and neck-shoulder musculature in women with temporomandibular disorders. J Pain Off J Am Pain Soc. 2010;11(12):1295–304.
48. Giunta JL, Kronman JH. Orofacial involvement secondary to trapezius muscle trauma. Oral Surg Oral Med Oral Pathol. 1985;60(4):368–9.
49. Wright EF. Referred craniofacial pain patterns in patients with temporomandibular disorder. J Am Dent Assoc. 2000;131(9):1307–15.
50. Farrar WB, McCarty Jr WL. The TMJ dilemma. J Ala Dent Assoc. 1979;63(1):19–26.
51. Roberts CA, Tallents RH, Espeland MA, Handelman SL, Katzberg RW. Mandibular range of motion versus arthrographic diagnosis of the temporomandibular joint. Oral Surg Oral Med Oral Pathol. 1985;60(3):244–51.
52. Tallents RH, Hatala M, Katzberg RW, Westesson PL. Temporomandibular joint sounds in asymptomatic volunteers. J Prosthet Dent. 1993;69:298–304.
53. Dibbets JMH, van der Weele LT. The prevalence of joint noises as related to age and gender. J Craniomandib Disord Facial Oral Pain. 1992;6:157–60.
54. Katzberg RW, Westesson PL, Tallents RH, Drake CM. Anatomic disorders of the temporomandibular joint disc in asymptomatic subjects. J Oral Maxillofac Surg. 1996;54:147–53.
55. Isberg A, Isacsson G, Johansson AS, Larson O. Hyperplastic soft-tissue formation in the temporomandibular joint associated with internal derangement. A radiographic and histologic study. Oral Surg Oral Med Oral Pathol. 1986;61(1):32–8.
56. Holumlund AB, Gynther GW, Reinholt FP. Disk derangement and inflammatory changes in the posterior disk attachment of the temporomandibular joint. Oral Surg Oral Med Oral Pathol. 1992;73:9.
57. Taskaya-Yylmaz N, Ogutcen-Toller M. Clinical correlation of MRI findings of internal derangements of the temporomandibular joints. Br J Oral Maxillofac Surg. 2002;40(4):317–21.
58. Harkins SJ, Marteney JL. Extrinsic trauma: a significant precipitating factor in temporomandibular dysfunction. J Prosthet Dent. 1985;54(2):271–2.
59. Moloney F, Howard JA. Internal derangements of the temporomandibular joint. III. Anterior repositioning splint therapy. Aust Dent J. 1986;31(1):30–9.
60. Weinberg S, Lapointe H. Cervical extension-flexion injury (whiplash) and internal derangement of the temporomandibular joint. J Oral Maxillofac Surg. 1987;45(8):653–6.
61. Pullinger AG, Seligman DA. Trauma history in diagnostic groups of temporomandibular disorders. Oral Surg Oral Med Oral Pathol. 1991;71(5):529–34.
62. Westling L, Carlsson GE, Helkimo M. Background factors in craniomandibular disorders with special reference to general joint hypermobility, parafunction, and trauma. J Craniomandib Disord. 1990;4(2):89–98.
63. Pullinger AG, Seligman DA. Association of TMJ subgroups with general trauma and MVA. J Dent Res. 1988;67:403.
64. Pullinger AG, Monteriro AA. History factors associated with symptoms of temporomandibular disorders. J Oral Rehabil. 1988;15:117.

65. Skolnick J, Iranpour B, Westesson PL, Adair S. Prepubertal trauma and mandibular asymmetry in orthognathic surgery and orthodontic paients. Am J Orthod Dentofacial Orthop. 1994;105:73–7.
66. Braun BL, DiGiovanna A, Schiffman E, et al. A cross-sectional study of temporomandibular joint dysfunction in post-cervical trauma patients. J Craniomandib Disord Facial Oral Pain. 1992;6:24–31.
67. Burgess J. Symptom characteristics in TMD patients reporting blunt trauma and/or whiplash injury. J Craniomandib Disord. 1991;5(4):251–7.
68. De Boever JA, Keersmaekers K. Trauma in patients with temporomandibular disorders: frequency and treatment outcome. J Oral Rehabil. 1996;23(2):91–6.
69. Yun PY, Kim YK. The role of facial trauma as a possible etiologic factor in temporomandibular joint disorder. J Oral Maxillofac Surg Off J Am Assoc Oral Maxillofac Surg. 2005;63(11):1576–83.
70. Arakeri G, Kusanale A, Zaki GA, Brennan PA. Pathogenesis of post-traumatic ankylosis of the temporomandibular joint: a critical review. Br J Oral Maxillofac Surg. 2012;50(1):8–12.
71. Okeson JP. Bell's oral and facial pain. 7th ed. Chicago: Quintessence Publishers; 2014. p. 327–69.
72. Israel HA, Diamond B, Saed Nejad F, Ratcliffe A. The relationship between parafunctional masticatory activity and arthroscopically diagnosed temporomandibular joint pathology. J Oral Maxillofac Surg Off J Am Assoc Oral Maxillofac Surg. 1999;57(9):1034–9.
73. Nitzan DW. Intraarticular pressure in the functioning human temporomandibular joint and its alteration by uniform elevation of the occlusal plane. J Oral Maxillofac Surg. 1994;52(7):671–9.
74. Milam SB, Schmitz JP. Molecular biology of temporomandibular joint disorders: proposed mechanisms of disease. J Oral Maxillofac Surg. 1995;12:1448–54.
75. Monje F, Delgado E, Navarro MJ, Miralles C, Alonso Jr dH. Changes in temporomandibular joint after mandibular subcondylar osteotomy: an experimental study in rats. J Oral Maxillofac Surg. 1993;51:1221–34.
76. Shaw RM, Molyneux GS. The effects of induced dental malocclusion on the fibrocartilage disc of the adult rabbit temporomandibular joint. Arch Oral Biol. 1993;38:415–22.
77. Isberg A, Isacsson G. Tissue reactions associated with internal derangement of the temporomandibular joint. A radiographic, cryomorphologic, and histologic study. Acta Odontol Scand. 1986;44(3):160–4.
78. Wright Jr WJ. Temporomandibular disorders: occurrence of specific diagnoses and response to conservative management. Clin Observ Craniol. 1986;4(2):150–5.
79. Seligman DA, Pullinger AG. Association of occlusal variables among refined TM patient diagnostic groups. J Craniomandib Disord. 1989;3(4):227–36.
80. Solberg WK, Bibb CA, Nordstrom BB, Hansson TL. Malocclusion associated with temporomandibular joint changes in young adults at autopsy. Am J Orthod. 1986;89(4):326–30.
81. Tsolka P, Walter JD, Wilson RF, Preiskel HW. Occlusal variables, bruxism and temporomandibulae disorder: a clinical and kinesiographic assessment. J Oral Rehabil. 1995;22:849–956.
82. Celic R, Jerolimov V. Association of horizontal and vertical overlap with prevalence of temporomandibular disorders. J Oral Rehabil. 2002;29(6):588–93.
83. Williamson EH, Simmons MD. Mandibular asymmetry and its relation to pain dysfunction. Am J Orthod. 1979;76(6):612–7.
84. DeBoever JA, Adriaens PA. Occlusal relationship in patients with pain-dysfunction symptoms in the temporomandibular joint. J Oral Rehabil. 1983;10:1–7.
85. Brandt D. Temporomandibular disorders and their association with morphologic malocclusion in children. In: Carlson DS, McNamara JA, Ribbens KA, editors. Developmental aspects of temporomandibular joint disorders. Ann Arbor: University of Michigan Press; 1985. p. 279.
86. Nilner M. Functional disturbances and diseases of the stomatognathic system. A cross-sectional study. J Pedod. 1986;10(3):211–38.
87. Stringert HG, Worms FW. Variations in skeletal and dental patterns in patients with structural and functional alterations of the temporomandibular joint: a preliminary report. Am J Orthod. 1986;89(4):285–97.
88. Gunn SM, Woolfolk MW, Faja BW. Malocclusion and TMJ symptoms in migrant children. J Craniomandib Disord. 1988;2(4):196–200.
89. Dworkin SF, Huggins KH, LeResche L, Von KM, Howard J, Truelove E, et al. Epidemiology of signs and symptoms in temporomandibular disorders: clinical signs in cases and controls. J Am Dent Assoc. 1990;120(3):273–81.
90. Ronquillo HI, et al. Comparison of internal deranagements with condyle-fossa relationship, horizontal and vertical overlap, and angle class. J Craniomandib Disord Facial Oral Pain. 1988;2:137.
91. 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.
92. Pullinger AG, Seligman DA. Overbite and overjet characteristics of refined diagnositic groups of temporomandibular disorders patients. Am J Orthod Dentofacial Orthop. 1991;100:401.
93. Hirsch C, John MT, Drangsholt MT, Mancl LA. Relationship between overbite/overjet and clicking or crepitus of the temporomandibular joint. J Orofac Pain. 2005;19(3):218–25.
94. John MT, Hirsch C, Drangsholt MT, Mancl LA, Setz JM. Overbite and overjet are not related to self-report of temporomandibular disorder symptoms. J Dent Res. 2002;81(3):164–9.
95. Stegenga B, de Bont L, Boering G. Osteoarthrosis as the cause of craniomandibular pain and dysfunction: a unifying concept. J Oral Maxillofac Surg. 1989;47(3):249–56.
96. Stegenga B, de Bont LG, Boering G, van Willigen JD. Tissue responses to degenerative changes in the temporomandibular joint: a review. J Oral Maxillofac Surg. 1991;49(10):1079–88.

97. DeBont LGM, Boering G, Liem RSB, Eulderink F, Westesson PL, et al. Osteoarthritis and internal derangement of the temporomandibular joint: a light microscopic study. J Oral Maxillofac Surg. 1986;44:634–43.
98. Mills DK, Daniel JC, Herzog S, Scapino RP. An animal model for studying mechanisms in human temporomandibular joint disc derangement. J Oral Maxillofac Surg. 1994;52(12):1279–92.
99. Helmy E, Bays R, Sharawy M. Osteoarthrosis of the temporomandibular joint following experimental disc perforation in Macaca fascicularis. J Oral Maxillofac Surg. 1988;46(11):979–90.
100. Boering G. Temporomandibular joint arthrosis: a clinical and radiographic investigation [thesis]. Groningen: University of Groningen; 1966.
101. Farrar WB, McCarty WL. A clinical outline of temporomandibular joint diagnosis and treatment. 7th ed. Montgomery: Normandie Publications; 1983. p. 191.
102. McCarty WL, Farrar WB. Surgery for internal derangements of the temporomandibular joint. J Prosthet Dent. 1979;42(2):191–6.
103. Wilkes CH. Arthrography of the temporomandibular joint in patients with the TMJ pain-dysfunction syndrome. Minn Med. 1978;61(11):645–52.
104. Akerman S, Kopp S, Rohlin M. Histological changes in temporomandibular joints from elderly individuals. An autopsy study. Acta Odontol Scand. 1986;44(4):231–9.
105. Kircos LT, Ortendahl DA, Mark AS, Arakawa M. Magnetic resonance imaging of the TMJ disc in asymptomatic volunteers. J Oral Maxillofac Surg. 1987;45(10):852–4.
106. Magnusson T, Egermark I, Carlsson GE. A longitudinal epidemiologic study of signs and symptoms of temporomandibular disorders from 15 to 35 years of age. J Orofac Pain. 2000;14(4):310–9.

Screening Orthodontic Patients for Temporomandibular Disorders

3

Charles S. Greene and Gary D. Klasser

3.1 Introduction

Patients who present for orthodontic diagnosis and treatment must have a thorough oral examination before beginning the orthodontic evaluation process. The traditional oro-dental screening includes three major components: (1) caries history and current dental situation, (2) periodontal history and current findings of concern, and (3) oral cancer screening and soft tissue exam.

However, there is a fourth item that needs to be included in the aforementioned list, i.e., an evaluation of orofacial region with emphasis regarding the temporomandibular joints (TMJs) and associated musculoskeletal structures. The main focus of such an evaluation is to ascertain whether the patient has a temporomandibular disorder (TMD). The American Association of Orthodontists has only a brief recommendation about this subject in its Clinical Guidelines [1]:

An evaluation [should be done] of the temporomandibular joint and associated musculature to assess function and disease.

C.S. Greene, BS, DDS (✉)
Department of Orthodontics,
University of Illinois at Chicago,
College of Dentistry, Chicago, IL, USA
e-mail: cgreene@uic.edu

G.D. Klasser, DMD, Cert Orofacial Pain
Department of Diagnostic Sciences,
Louisiana State University Health Sciences Center,
School of Dentistry, New Orleans, LA, USA

Obviously, this limited suggestion does not even begin to inform the orthodontic practitioner about what actually needs to be done in order to perform a proper screening exam for TMDs. Furthermore, it does not address what should be done if positive signs and symptoms of TMD are discovered during the screening. In this chapter, the authors will address the challenge of performing a proper initial screening examination for TMDs on prospective orthodontic patients. Screening protocols and forms that have been recommended in the past by the American Dental Association (ADA) and other groups will be discussed. Recommendations will be made for reacting in an appropriate manner to the various minor or major findings obtained during the screening exam.

In addition to the need for routine screening of all prospective orthodontic patients for the presence of a TMD, the orthodontist also must be prepared to deal with patients who present with orofacial pain symptoms. The following three situations can occur in every orthodontic practice:

1. The orthodontist may have a patient referred specifically for TMD issues.
2. TMD signs and symptoms may arise during orthodontic treatment.
3. A completed patient may develop TMD after orthodontic treatment.

In order to deal with these possibilities, the orthodontist needs to know how to obtain a proper

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_3

history and conduct a clinical examination of a patient who presents for any of the aforementioned reasons with symptoms that fall within the broad category of orofacial pain. There are many different types and categories of orofacial pain, including various types of headache disorders. However, for the purposes of this chapter and this book we will be focusing on the TMDs, because these are the disorders that orthodontists will most commonly have to deal with in their practices. The diagnosis of some form of TMD requires a careful process of differential diagnosis, because there are many medical and dental problems that produce orofacial pain symptoms; some of these overlap considerably with TMD symptoms. Even if the orthodontist does not want to manage TMD problems in his or her office, it is important to establish a presumptive diagnosis of the patient's complaints. This process will lead to more appropriate referrals for each situation encountered.

There are important differences between the differential diagnosis procedures for individuals reporting possible TMD pain and the procedures used in screening for those conditions. In addition, the skills needed for screening should be used every day with new patients, while more complete assessments will only be needed when individuals initially present with these problems or when problems arise during the course of treatment. In the next section, we provide a brief review of the complete examination protocol for assessing a patient complaining of pain. This will be followed by a comprehensive discussion of TMD screening protocols and how to deal with the findings obtained in that process.

3.2 Assessment of Patients Presenting with or Who Develop TMD Symptoms

The American Academy of Orofacial Pain (AAOP) defines TMD as "a group of musculoskeletal and neuromuscular conditions that involve the TMJs, the masticatory muscles, and all associated tissues" [2]. As previously stated, the signs and symptoms associated with these disorders are often similar to those that arise from other non-musculoskeletal sources (neurologic, neurovascular, neoplastic, and glandular). These commonalities may create a confusing scenario for the orthodontist who has not been trained in the diagnosis of such problems. Unfortunately, the possibility for misinterpretation, misdiagnosis, and mistreatment with the potential for morbidity and mortality may exist, so it is important for the orthodontist to be able to perform this preliminary differential diagnosis [3]. A good reference for how practitioners should conduct this type of diagnostic exam can be found in the AAOP Guidelines [2].

Therefore, if patients present with signs/symptoms of a TMD condition, orthodontists will be faced with two choices. They can either manage the TMD problem for this patient prior to initiating orthodontic interventions, or they may refer to a colleague with expertise in the field of TMD and orofacial pain. If the choice is made to manage this individual, then it should be done in accordance with currently accepted guidelines for TMD diagnosis and treatment [2]. Accepting this responsibility should be done with the understanding that, according to recent systematic reviews, orthodontic treatment is neither the cause nor cure for TMD (see Chap. 6), so the appropriate management will involve using reversible and conservative modalities such as medications, physical therapy, oral appliances, and self-care.

The importance of adhering to a complete and thorough assessment process cannot be overstated, since it is only by following this protocol that the orthodontist can deliver a proper diagnosis and provide appropriate management strategies. The history portion of a TMD assessment should be similar to that conducted by our medical colleagues. The sequencing should be performed in a logical manner commencing with the chief complaints as presented in the patient's own words. These complaints should be documented in the order of severity as expressed by the patient, and details of each complaint are elicited in a systematic manner. This is then followed by the history of the chief complaint which should include such information as the location of the pain(s), date of onset, event onset (spontaneous or stimulus induced), quality, frequency, duration, and intensity (based upon a numeric rating scale of 0=no pain to 10=the most extreme pain, or a visual analog scale using a 10-cm line labeled at one end with "no pain" and at the other

end with "most extreme pain"). Questions should be asked about factors that alleviate, aggravate, or precipitate the pain; changes over time; previous treatment results; and any associated issues.

Finally, it is essential to ask if the patient has any of the well-known comorbid conditions that are frequently found in TMD patients such as certain headaches, affective disorders (anxiety and depression), and nonorganic (functional) disorders such as fibromyalgia, irritable bowel syndrome, interstitial cystitis/bladder pain syndrome, chronic pelvic pain, and vulvodynia [4, 5].

The next line of questioning should be directed toward the medical, dental, and psychosocial history. The medical history should inquire about previous surgery, hospitalizations, trauma, illness, developmental and acquired anomalies, sleep disorders and sleep-related breathing disorders, allergies, and medication usage (including prescribed, over the counter, herbal and vitamin supplements, and illicit drug use). The dental history should include information regarding previous dental disease, treatment, and habit history (awake and asleep). This should then be followed by taking a psychosocial history which includes a discussion of social, behavioral, and psychological issues; occupational, recreational, and family status; litigation, disability, or secondary gain issues.

Next, a comprehensive physical examination of the region must be performed. This consists of a general inspection of the head and neck; orthopedic evaluation of the temporomandibular joint including intracapsular sounds; assessment of the cervical spine; masticatory and cervical muscle evaluation; evaluation of the cranial nerves for neurovascular, neurosensory, and motor problems; and finally an intraoral assessment (hard and soft tissues).

Adjunctive tests may be required if the yield from doing so would enhance the ability of the orthodontist to develop a definitive diagnosis and/or provide appropriate management. One of the adjunctive tests to be considered is dental imaging (bitewing, periapical, and panoramic radiographs) and/or medical imaging (computerized tomography, cone-beam computed tomography, magnetic resonance imaging, radionucleotide, and ultrasonography) [6].

TMJ imaging is warranted when the history or examination, or both, are indicative of a recent or progressive pathological joint condition; significant dysfunction or alteration in range of mandibular movements; or significant and often sudden changes in occlusion (anterior open bite, posterior open bite, and mandibular shift). Other adjunctive tests to be considered are diagnostic anesthesia and serologic testing.

The authors have provided a protocol for managing patients who either present with or develop TMD during treatment or after completion of treatment (Table 3.1). For a comprehensive

Table 3.1 Protocol for the management of TMD signs and symptoms within an orthodontic practice

At time of presentation	1. If patient has signs and symptoms of TMD, then the patient should be informed that orthodontic treatment will not resolve those problems.
	2. Current TMD signs and symptoms should be noted, and a full TMD history and clinical examination should be undertaken and recorded.
	3. If the existing TMD is acute and severe, the commencement of orthodontic treatment should be *postponed* until the condition is either resolved or stabilized.
During treatment	1. Acknowledge and recognize the signs and symptoms of TMD.
	2. Reassure and educate the patient that TMD is not necessarily a progressive problem and in most cases symptoms will improve over time with conservative treatment.
	3. Active orthodontic treatment should be *postponed* and TMD signs and symptoms should be managed by either the orthodontist or an expert TMD colleague.
	4. Once signs and symptoms have been alleviated or controlled, active orthodontic treatment may be resumed with consideration to modification of treatment (reduction of forces on headgear, remove or lighten elastics, use of oral TMD treatment appliance).
After treatment	The patient should be *monitored* for signs and symptoms throughout the retention period. If symptoms arise, appropriate management should be provided.

review of the history taking and clinical examination process, the reader is referred to other sources [7–9].

3.3 Previous and Current TMD Screening Forms or Recommended Protocols

The first formal attempt to present a structured questionnaire for screening all dental patients for the presence of a TMD appeared at the end of the book summarizing the proceedings of the ADA President's Conference that was held in 1982, and it also was cited in the ADA journal [10]. The recommended history questions and examination procedures from that conference are presented in Table 3.2. While some of the questions might have been helpful for screening purposes, others were so broad (ever had injury? ever had arthritis?) as to be practically meaningless. This form was not widely distributed or accepted by the profession, mainly due to its limited publication, and therefore it was not widely used by clinical dentists. Subsequent attempts to present recommended approaches to TMD screening have met with mixed success. In 1986 the TMJ Scale, a commercially developed questionnaire for screening TMD in private dental offices, was described in a popular journal; however, this 97-question form was far too cumbersome for routine use in dental practices [11]. In that same year, Kleinknacht et al. [12] presented a 14-question approach to screening, but their questionnaire had many shortcomings: the interexaminer reliability (percentage agreement) ranged from 50 to 92 %, there was a lack of reference to any standard diagnosis, and no psychometric properties were presented.

Table 3.2 Recommended (1982) protocol for screening patients for temporomandibular disorders

Screening history for temporomandibular disorders	Screening examination for temporomandibular disorders
Do you have difficulty opening your mouth?	Inspection for facial asymmetry
Do you hear noises from the jaw joints?	Evaluation of jaw movements
Does your jaw get "stuck, locked, or go out?"	Palpation for muscle or joint tenderness
Do you have pain in or about your ears or cheeks?	Palpation for clicking, crepitus, abnormal movements (incoordination)
Do you have pain on chewing? Wide opening?	
Does your bite feel uncomfortable/ unusual?	
Have you had injury to jaw, head, or neck?	
Have you ever had arthritis?	
Have you previously been treated for TMD?	

From the Report of the President's Conference on examination, diagnosis, and management of temporomandibular disorders, 1982 [10]

Similar problems afflicted several subsequent attempts to develop a useful screening instrument for detecting TMD problems in ordinary dental patient populations. These past attempts are well summarized in Table 3.1 in an excellent article by Gonzalez et al. [13] so they will not be discussed further here. In that 2011 paper, these authors presented both short (three-item) and long (six-item) versions of a newly developed TMD screening form (Fig. 3.1). By using psychometric methods for item selection, they developed these questionnaires and evaluated them for validity among 504 participants. They concluded that the selected items exhibited excellent content validity. The excellent levels of reliability, sensitivity, and specificity demonstrate the validity and usefulness of this instrument in any clinical office setting.

In addition to these TMD screening forms presented by various authors in the dental literature, there have been other approaches recommended by several dental organizations (academies, consortiums, institutes, etc.). The American College of Prosthodontists formed a committee that developed a 15-item form, which appears in an article in the inaugural issue of their journal [14]. In 2008, the European Academy of Craniomandibular Disorders (EACD) published a 4-item questionnaire that is quite minimal, with the instruction that any positive answer should lead to more in-depth investigations [15]. Meanwhile, various

Temporomandibular pain disorder screening instrument.

1. In the last 30 days, on average, how long did any pain in your jaw or temple area on either side last?

   a. No pain

   b. From very brief to more than a week, but it does stop

   c. Continous

2. In the last 30 days, have you had pain or stiffness in your jaw on awakening?

   a. No

   b. Yes

3. In the last 30 days, did the following activities change any pain (that is, make it better or make it worse) in your jaw or temple area on either side?

   A. Chewing hard or tough food

   a. No

   b. Yes

   B. Opening your mouth or moving your jaw forward or to the side

   a. No

   b. Yes

   C. Jaw habits such as holding teeth together, cleaning, grinding or chewing gum

   a. No

   b. Yes

   D. Other jaw activities such as talking, kissing or yawning

   a. No

   b. Yes

Items 1 through 3A constitute short version of the screening instrument, and items 1 through 3D constitute the long version. An "a" response receives 0 points, a "b" response 1 point and a "c" response 2 points.

Fig. 3.1 TMD screening instrument (Gonzalez et al. [13]). Copyright © 2011 American Dental Association. All rights reserved. Reprinted by permission)

occlusally oriented institutes and study clubs have developed in-house protocols for detecting TMD problems in newly presenting patients. The Pankey and Dawson groups in Florida emphasize a manipulative methodology in which the mandible is placed in centric relation and occlusal relationships are observed. Also, the mandible is "loaded" by pushing horizontally backward and laterally to see how the TMJs respond to such forces [16]. Other groups (Spear, Kois) use so-called "de-programming splints" to allow the mandible to drift into a "relaxed" muscular position. Based on the outcome of this procedure, judgments are made about the need to change the TMJ relationship via permanent occlusal treatment [17]. The Las Vegas Institute, however, utilizes electronic diagnostic instrumentation to analyze mandibular and occlusal relations; their concept is described as "neuromuscular dentistry." Based on a combination of electrical stimulators, jaw trackers, electromyographic recorders, and sound recorders, they determine who needs to have occlusal therapy as either a preventive or therapeutic treatment for TMD [18]. However, numerous papers have been published about the flaws and problems associated with the use of these electronic devices [19–23].

It should be obvious that all of these parochial in-house procedures are biased by the underlying philosophy of each organization; therefore, it is hard to accept the outcomes as providing meaningful analyses of good or bad mandibular positions, let alone as a screening method to detect TMDs. In addition, the invasiveness of the irreversible occlusal procedures that follow such analyses demands a much higher level of scientific evidence than is provided by these arbitrary diagnostic practices.

3.4 What to Do If Positive Findings Are Obtained During a Screening Exam?

In the course of screening new orthodontic patients for the presence of TMDs, there will inevitably be some positive answers to clinical questions as well as some positive "findings" from the physical examination of stomatognathic structures. The important question will be: when are those positive findings significant enough to establish a clinical diagnosis of TMD? This dilemma was first confronted by some of the early epidemiologic studies of TMD, in which researchers wanted to establish the prevalence of TMDs in the general population. The numbers reported in early studies by Helkimo and others who used the Index he developed were surprisingly high, often exceeding 50 % of the surveyed population [24]. This was largely due to the inclusion of various minor pain complaints and certain "objective" findings like TMJ clicking or deviated opening. Over time, this approach was criticized by Greene and Marbach [25] as well as several others, and later de Kanter in Holland presented survey numbers for that entire country which were much more reasonable [26]. Terms like "clinically significant" or "requiring treatment" became the threshold for determining whether people had actual TMD problems, or if they merely had variations from the expected ideal or normal findings. As a result, most modern epidemiologic studies of TMD agree that less than 10 % of the general population has a clinical problem that requires professional attention [27–29].

A screening exam for TMD should begin by asking questions about symptoms, both past and present. The first question should be: "Have you ever been diagnosed and/or treated for a TMD problem?" Assuming the answer is NO, the patient should be asked if any type of nondental facial pain has been occurring; however, this question should be elaborated so that the answers to it are meaningful for establishing a TMD diagnosis. What kind of pain has occurred? How often? Where did it seem to be? Did it affect jaw functions like chewing? Was it happening only after extreme functions like long dental appointments, extended gum chewing, etc.? Many people will report having a minimal experience of some type of facial pain history, often related to dento-alveolar problems. Therefore, a positive response to a pain question is far from being enough to classify the person as having a TMD.

The next symptom to ask about is TMJ clicking or popping. If there is a positive response, once again some important qualifying questions should be asked: When did the clicking start? Has it become more frequent or louder? Is it associated with any pain? Does the jaw ever get "stuck" in trying to open or close? Did the patient ever report it to a physician or dentist?

Some questions about functional difficulty should be part of the symptom history. Patients should be asked if they have noticed a limitation in their ability to open widely; however, it is important to ask whether that has always been true, or if it has been developing over time. They should be asked if normal functions like chewing hard food, singing in a choir, yawning widely, chewing gum, or sitting through a long dental appointment produce fatigue and pain; if so, does this symptom linger afterward or go away fairly quickly? Once again, there is a possibility that this is merely a situational problem, but it also is possible that these functional limitations are significant.

After the history portion of the exam is completed, the clinician needs to perform a physical examination to look for signs of TMD (and to correlate those with symptoms if possible). The first phenomenon to look for is clicking, popping, or other TMJ noises. This can be done by manual

palpation or by auscultation with a stethoscope; clinicians may be surprised by how often a sound is discovered during the exam, but it was not reported by the patient. If the joint sound is a single click, it often will be louder on opening and softer on closing (reciprocal click) as the condyle goes beyond the posterior band of the articular disk. If the sound is a grating (crepitus) noise, the patient should be asked about a history of arthritis in other joints; if the TMJ is the only joint, questions can be raised about previous painful episodes in that area. Imaging is not required either medico-legally or clinically to document this type of finding in the absence of significant pain and dysfunction.

Continuing with manual palpation, the masticatory muscles and both TMJs can be palpated for tenderness. Once again, however, caution must be exercised so that every positive response does not become an important "finding" of muscle or joint problems. Some sensible questions like "Are you surprised this area is tender? Have you noticed pain or tenderness before in this area?" can be helpful in sorting out the significance of such findings. It is wise to palpate some adjacent structures that should not ever be tender in order to decide whether the patient is simply a strong responder to these kinds of provocations.

The next objective assessment to perform is the observation and measurement of mouth opening, lateral excursions, and mandibular protrusion. One might see *deviation* during opening, but the mandible ends up in the midline at maximum opening, or there may be *deflection* of the mandible to one side as wide opening is reached. A finding of limited opening requires questioning the patients about their awareness of this fact; many subjects will report that they either were not aware of it or this is how it always has been for them, while others may say that this limitation has become an increasing problem for them. This is a crucial distinction, since the latter finding may be a sign of serious trouble and will need further investigation.

After gathering all of the aforementioned information, the clinician has to make a decision about whether the positive findings from an individual patient are minor or major in significance. While there is no absolutely clear line to be drawn between these two categories, there are some general biologic points to consider. First, it should be recognized that many people have transient orthopedic discomforts and dysfunctions in various parts of the body, most of which are likely to be self-resolving; the TMJ system is no different in this regard, so complaints of minor transient jaw fatigue or pain episodes should not be classified as clinically significant. On the objective side, phenomena like painless TMJ clicking or crepitus, deviated opening, tenderness in certain areas, or nonprogressive limited jaw opening cannot be resolved in most cases by any reasonable treatment; therefore, they should simply be regarded as imperfections that do not reach the threshold of being significant clinical problems.

On the other hand, major TMD symptomatology that is either discovered in screening or reported by the patient should be dealt with before embarking on any orthodontic evaluation or treatment protocol as discussed previously *[see "Assessment of patients presenting with or who develop orofacial pain symptoms"].* If the orthodontist is not comfortable in managing such problems in his/her office, an appropriate referral will be necessary. In some cases, even with expert care, the patient may continue to have low-level or recurrent TMD symptoms, so a decision must be made about proceeding with the ideal orthodontic treatment plan or needing to compromise. In any case, an informed consent note should be placed in the record and signed by the patient, in which the orthodontist lists all positive findings from the screening exam and specifies what action (if any) needs to be taken before starting orthodontic treatment.

3.5 What to Do If the Orthodontist Is Consulted Specifically for TMD Issues?

Every orthodontist can expect to have some patients referred to them specifically for treatment of a TMD. The most common reason for this is the observation by a referring dentist that the patient has some type of morphologic or

functional malocclusion, leading to a presumption that this finding is the basis for developing TMD symptoms. Since many people in the general population have "untreated occlusal problems," the likelihood of that coincidental finding occurring in a TMD patient is rather high. In a recent survey by the authors of this chapter it was found that many dental schools in the USA and Canada do not provide adequate predoctoral training in the area of orofacial pain and TMDs [30]. Therefore, a general dentist who has not been exposed to contemporary concepts about the etiology and management of TMDs might expect an orthodontist to deal with such problems by performing orthodontic treatment.

However, the idea that occlusal dysharmonies and certain maxillo-mandibular relationships are responsible for the development of TMD symptoms has largely been discredited in the orthodontic literature as well as in the TMD literature. The reader is urged to look at the comprehensive orthodontic/TMD review papers cited here for confirmation of this important fact [31–33]. Instead, it is now widely recognized that TMDs are complex musculoskeletal pain disorders that share many characteristics with other somatic pain disorders, and the etiologic basis for developing them is complex and multifactorial [34, 35]. Therefore, an orthodontist who receives a TMD-based referral must be able to respond appropriately to both the patient and the referring doctor. The patient needs to know what is the best course to follow (rather than the proposed orthodontic treatment), and the referring doctor needs to understand why the orthodontist will not be providing that expected orthodontic treatment. All of this has to be accomplished without alienating any of the parties involved, so it is obvious that the orthodontist needs to have a good understanding of all the issues surrounding this type of situation. If an orthodontist does decide to provide primary care for patients with TMD symptoms within his/her office, this can be a valuable service to the local community of dentists as well as for the patients themselves. However, as discussed earlier, it is essential that treatment provided should be evidence-based conservative care that conforms to the current standards and protocols in the TMD field [2]. If this is not feasible, then an appropriate referral needs to be made.

3.6 What to Do If TMD Symptoms Arise During Orthodontic Treatment?

The fact that a majority of orthodontic patients are adolescents becomes important when talking about TMDs and orthodontic treatment. Since the onset of many TMD problems begins during the adolescent years, the probability of coincidental development of TMD symptoms becomes a challenge for the orthodontist. The first issue to consider is whether the reported pain and/or dysfunction are indeed coincidental, or whether this might be a response to the orthodontic treatment forces. In either case, the recommended protocol is to *STOP* all active mechanics and treat the symptoms conservatively. Then, if the symptoms return after resuming orthodontic treatment, it can be assumed they are likely to be related to the orthodontic procedures. If so, this may require a change in the orthodontic strategy and a compromise in the outcome of the case.

If the TMD symptoms are serious enough to warrant referral to another practitioner, the orthodontist will be confronted by the dilemma of choosing an appropriate doctor. Fortunately, there currently are 13 postgraduate programs in orofacial pain and about the same number of oral medicine programs in the USA and Canada, and the graduates from those programs are generally well-qualified to handle most TMD problems. If the teaching center for such programs is located in your community, it would be prudent to refer your patients there. However, the small number of graduates and the limited geographic distribution of these teaching centers make it unlikely that the average patient will have access to that level of care.

Instead, orthodontists will probably need to identify other qualified practitioners near them, and that is not an easy task these days. The combination of Web-based promotional layouts and papers advertising by various dentists,

all claiming to be "TMD experts" of some kind, is overwhelming and confusing for both patients and dentists. Since the ADA does not recognize a specialty in this area, there are no clear criteria for choosing one person over another. Some useful guidelines for doing this are as follows:

- Look for people with advanced training in oral medicine, oral pathology, or orofacial pain mini-programs that have been presented in university settings. Beware of dentists who have taken short courses in various continuing education (CE) settings or study clubs that claim to provide a quick training in these complex areas.
- Consider referring to an oral surgeon or GPR-trained general dentist in your area; these people have had hospital experience and some medical training in managing pain.

Ultimately, the orthodontist entering a new practice community should take some time to find and interview these kinds of colleagues. It is important to know whether the person tends to overtreat TMD problems by doing extensive jaw-repositioning or occlusion-changing procedures. Similarly, it is important to know if an oral surgeon tends to perform an inordinate number of intracapsular procedures rather than following a conservative medical treatment approach (See Chap. 9).

3.7 What to Do If a Patient Develops TMD After Orthodontic Treatment?

The laws of probability guarantee that every orthodontist will have some patients develop TMD symptoms at some point in time after the completion of orthodontic treatment. Since the annual incidence rate is estimated to be at 2 % and with many other people experiencing various combinations of transient TMD symptoms from time to time, the orthodontist is sure to be exposed to this situation [36–38]. As stated earlier, many adolescents (especially females) comprise the population of first-onset TMD problems. If these patients have had previous orthodontic treatment, the orthodontist as well as the referring dentist may be led to believe that there is a connection. But even adults who develop TMD symptoms are often asked by their dentists whether they had orthodontic treatment as a teenager, because many dentists believe there is a causal relationship between those phenomena. Searching the Internet for information on this matter is guaranteed to produce even more confusion, since there are a plethora of opinions.

Therefore, the orthodontist will have the dual challenge of communicating properly with both the patient and the dentist while also trying to assist the patient in obtaining appropriate TMD care. The various possibilities for obtaining such care are discussed in the previous section, so that will not be repeated here. However, the communication part can be tricky for several reasons. In speaking with the patient's dentist, the orthodontist may have to overcome a variety of negative beliefs or opinions. In addition to generally believing that orthodontic treatment is a likely cause of TMD problems, that dentist may have acquired various unscientific concepts about TMD-orthodontic relationships through some training at an institute or CE course, or through membership within a particular dental study club. If the dentist believes that TMD problems can be attributed to extraction of teeth, or failure to finish the orthodontic treatment properly (e.g., in centric relation or in neuromuscular centric), or failure to develop posterior disclusion of teeth, those ideas may be communicated to his or her patients.

Thus, the orthodontist needs to be knowledgeable about all of these controversies that exist both within the orthodontic specialty and throughout some parts of the general dental community. Assuming that the treatment protocol for the patient was within normal standards of practice, and the final result was within those parameters, the literature on orthodontic-TMD relationships is very clear: orthodontic treatment generally does not cause or cure TMD problems, so the random develop-

ment of symptoms cannot be attributed to that treatment [31–33, 39].

Take Home Messages

- At a minimum, a screening evaluation for TMDs must be a component of the initial orthodontic evaluation process.
- Orthodontists need to discriminate between major (significant) and minor (insignificant) signs and symptoms of TMD if they are discovered during the screening.
- If the patient has significant TMD issues, the orthodontist must decide whether to take on the responsibility for managing them prior to initiating orthodontic treatment. If not, an appropriate referral must be made.
- Orthodontists must respond appropriately when a patient is referred specifically for the treatment of TMD issues. Communication with both the patient and the referring dentist must be in line with current scientific concepts about TMD-orthodontic relationships.
- Orthodontists must be cognizant of proper procedures to follow when TMD signs and symptoms arise during orthodontic treatment.
- Because there is some potential for the development of TMD issues after orthodontic treatment in a segment of their population, it is important for orthodontists to react appropriately in these circumstances.

References

1. American Association of Orthodontists. Clinical Practice Guidelines for Orthodontics and Dentofacial Orthopedics. 2008 .https://www.aaoinfor.org/practice/clinical-practice guidelines. Accessed 13 July 2014.
2. American Academy of Orofacial Pain. Diagnosis and management of TMDs. In: De Leeuw R, Klasser GD, editors. Orofacial pain: guidelines for assessment, diagnosis, and management. 5th ed. Chicago: Quintessence; 2013. p. 129–30.
3. Klasser GD, Epstein JB, Utsman R, Yao M, Nguyen PH. Parotid gland squamous cell carcinoma invading the temporomandibular joint. J Am Dent Assoc. 2009;140:992–9.
4. Klasser GD, Bassiur J, de Leeuw R. Differences in reported medical conditions between myogenous and arthrogenous TMD patients and its relevance to the general practitioner. Quintessence Int. 2014;45:157–67.
5. de Leeuw R, Klasser GD, Albuquerque RJ. Are female patients with orofacial pain medically compromised? J Am Dent Assoc. 2005;136:459–68.
6. 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.
7. American Academy of Orofacial Pain. General assessment of the orofacial pain patient. In: De Leeuw R, Klasser GD, editors. Orofacial pain: guidelines for assessment, diagnosis, and management. 5th ed. Chicago: Quintessence; 2013. p. 25–46.
8. Okeson JP. History of and examination for temporomandibular disorders. In: Management of temporomandibular disorders and occlusion. 7th ed. St. Louis: Mosby; 2013. p. 170–221.
9. Schiffman E, Ohrbach R, Truelove E, Look J, Anderson G, Goulet JP, 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;28:6–27.
10. Griffiths RH. Report of the President's Conference on examination, diagnosis, and management of temporomandibular disorders. J Am Dent Assoc. 1983;106:75–7.
11. Lundeen TF, Levitt SR, McKinney MW. Discriminative ability of the TMJ scale: age and gender differences. J Prosthet Dent. 1986;56:84–92.
12. Kleinknecht RA, Mahoney ER, Alexander LD, Dworkin SF. Correspondence between subjective report of temporomandibular disorder symptoms and clinical findings. J Am Dent Assoc. 1986;113:257–61.
13. Gonzalez YM, Schiffman E, Gordon SM, Seago B, Truelove EL, Slade G, et al. Development of a brief and effective temporomandibular disorder pain screening questionnaire: reliability and validity. J Am Dent Assoc. 2011;142:1183–91.
14. Nassif NJ, Hilsen KL. Screening for temporomandibular disorders: history and clinical examination. American Dental Association. J Prosthodont. 1992;1:42–6.
15. De Boever JA, Nilner M, Orthlieb JD, Steenks MH. Recommendations by the EACD for examination, diagnosis, and management of patients with temporomandibular disorders and orofacial pain by the general dental practitioner. J Orofac Pain. 2008;22:268–78.

16. Pankey Institute. A Comprehensive Look at TMD Patients from Diagnosis to Restorative: Part 2. http://www.pankey.org/curriculum/courses/focus-courses/a-comprehensive-look-at-tmd-patients-from-diagnosis-to-restorative. Accessed 10 July 2014.
17. Kois Deprogrammer. http://www.generalfamilydentistry.net/KoisDep.pdf. Accessed 10 July 2014.
18. Las Vegas Institute. What is Neuromuscular Dentistry? http://www.lviglobal.com/what-is-nmd-neuromuscular-dentistry. Accessed 10 July 2014.
19. Klasser GD, Okeson JP. The clinical usefulness of surface electromyography in the diagnosis and treatment of temporomandibular disorders. J Am Dent Assoc. 2006;137:763–71.
20. Al-Saleh MA, Armijo-Olivo S, Flores-Mir C, Thie NM. Electromyography in diagnosing temporomandibular disorders. J Am Dent Assoc. 2012;143:351–62.
21. Manfredini D, Cocilovo F, Favero L, Ferronato G, Tonello S, Guarda-Nardini L. Surface electromyography of jaw muscles and kinesiographic recordings: diagnostic accuracy for myofascial pain. J Oral Rehabil. 2011;38:791–9.
22. Gonzalez YM, Greene CS, Mohl ND. Technological devices in the diagnosis of temporomandibular disorders. Oral Maxillofac Surg Clin North Am. 2008;20:211–20.
23. Sharma S, Crow HC, McCall WD, Gonzalez YM. Systematic review of reliability and diagnostic validity of joint vibration analysis for diagnosis of temporomandibular disorders. J Orofac Pain. 2013;27:51–60.
24. Helkimo M. Studies on function and dysfunction of the masticatory system. II. Index for anamnestic and clinical dysfunction and occlusal state. Sven Tandlak Tidskr. 1974;67:101–21.
25. Greene CS, Marbach JJ. Epidemiologic studies of mandibular dysfunction: a critical review. J Prosthet Dent. 1982;48:184–90.
26. De Kanter RJ, Kayser AF, Battistuzzi PG, Truin GJ, Van't Hof MA. Demand and need for treatment of craniomandibular dysfunction in the Dutch adult population. J Dent Res. 1992;71:1607–12.
27. Drangsholt M, LeResche L. Temporomandibular disorder pain. In: Crombie IK, editor. Epidemiology of pain. Seattle: IASP Press; 1999. p. 203–33.
28. Schiffman EL, Fricton JR, Haley DP, Shapiro BL. The prevalence and treatment needs of subjects with temporomandibular disorders. J Am Dent Assoc. 1990;120:295–303.
29. Dworkin SF, Huggins KH, LeResche L, Von Korff M, Howard J, Truelove E, et al. Epidemiology of signs and symptoms in temporomandibular disorders: clinical signs in cases and controls. J Am Dent Assoc. 1990;120:273–81.
30. Klasser GD, Greene CS. Predoctoral teaching of temporomandibular disorders: a survey of U.S. and Canadian dental schools. J Am Dent Assoc. 2007;138:231–7.
31. McNamara JA, Seligman DA, Okeson JP. Occlusion, orthodontic treatment, and temporomandibular disorders: a review. J Orofac Pain. 1995;9:73–90.
32. Michelotti A, Iodice G. The role of orthodontics in temporomandibular disorders. J Oral Rehabil. 2010;37:411–29.
33. Luther F, Layton S, McDonald F. Orthodontics for treating temporomandibular joint (TMJ) disorders. Cochrane Database Syst Rev. 2010;(7):CD006541.
34. Greene CS. The etiology of temporomandibular disorders: implications for treatment. J Orofac Pain. 2001;15:93–105; discussion 06–16.
35. Klasser GD, Greene CS. The changing field of temporomandibular disorders: what dentists need to know. J Can Dent Assoc. 2009;75:49–53.
36. Von Korff M, Le Resche L, Dworkin SF. First onset of common pain symptoms: a prospective study of depression as a risk factor. Pain. 1993;55:251–8.
37. Luz JG, Oliveira NG. Incidence of temporomandibular joint disorders in patients seen at a hospital emergency room. J Oral Rehabil. 1994;21:349–51.
38. De Kanter RJ, Truin GJ, Burgersdijk RC, Van't Hof MA, Battistuzzi PG, Kalsbeek H, et al. Prevalence in the Dutch adult population and a meta-analysis of signs and symptoms of temporomandibular disorder. J Dent Res. 1993;72:1509–18.
39. Leite RA, Rodrigues JF, Sakima MT, Sakima T. Relationship between temporomandibular disorders and orthodontic treatment: a literature review. Dental Press J Orthod. 2013;18:150–7. See #10.

Psychological Considerations

4

Richard Ohrbach and Ambra Michelotti

4.1 Introduction and Overview

The standard definition of temporomandibular disorder (TMD) – a collective term embracing a number of clinical problems that involve the masticatory muscles, the temporomandibular joints (TMJs), and the associated structures – clearly places the emphasis onto the underlying structures. From a physical diagnostic perspective, this emphasis is of course appropriate and essential. Because TMDs are the most prevalent clinical conditions afflicting the masticatory apparatus, and because they are associated with both pain and limitations in masticatory function, emphasis on structural problems and etiologies can understandably dominate at the time of the clinical consultation. However, current texts [1, 2] emphasize that structural perspectives of TMDs alone are insufficient. Moreover, recent publications focusing on disease classification systems are highlighting the process aspect of pain. These pain classification systems are anchoring the "location" of pain disorders at a broad systems level, involving brain, mind, and the person in addition to pointing specifically at the masticatory system for TMDs [3, 4]. While this broad perspective has been advocated for decades as part of the biopsychosocial model of disease [5–7], new approaches to disease classification are leading to the same conclusions with greater rigor [8].

This chapter provides an overview of the biopsychosocial perspective of TMD, and then it focuses on aspects of the biopsychosocial model that are particularly challenging within the context of malocclusions and orthodontic treatment. Hopefully, this approach can begin to explain how perceived occlusion, behavior, and psychosocial status are perhaps directly linked. A major point in the present chapter, which we will return to repeatedly, is that a dominant source of the many controversies surrounding the relationship between structure (e.g., orthodontic malocclusion) and TMD is the persistent keyhole view of considering only structural factors when discussing TMDs. As a result, the psychological and behavioral cofactors that are necessarily involved in TMD (as they are in all pain disorders) are consistently omitted from most of the research as well as clinical decision making in the orthodontic community. We believe that the current evidence can be improved, and thereby make the psychosocial realm yet more applicable to the orthodontic consulting room, by incorporating a better theoretical model that has been missing

R. Ohrbach, DDS, MS, PhD (✉)
Department of Oral Diagnostic Sciences,
University at Buffalo School of Dental Medicine,
355 Squire, Buffalo, NY 14214, USA
e-mail: ohrbach@buffalo.edu

A. Michelotti, DDS, BSc, Orthod
Section of Orthodontics,
Department of Neuroscience, School of Dentistry,
University of Naples Federico II, Naples, Italy

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_4

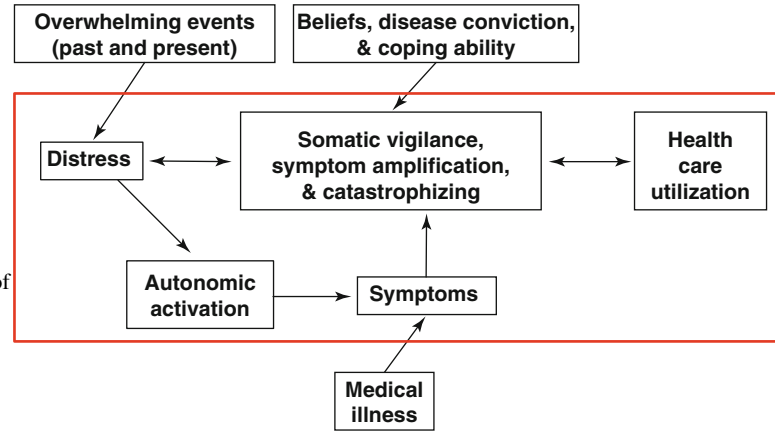


Fig. 4.1 General model for functional disorders. Physical and psychological processes directly associated with functional disorders are shown inside the larger box. Outside the large box are exogenous processes that represent life events, trait characteristics in the form of beliefs and coping style, and medical illness. All of these are illustrated as existing separate from the functional disorder (Modified from Sullivan and Katon [12])

from the discussions to date. As this chapter will explore, behavior and structure need to be and can be better integrated.

4.2 TMD as a Biopsychosocial Disorder

Pain disorders are often characterized by nonpredictable response to usual treatment, recurrence of symptoms, behavioral complications, and transition to chronicity. The severity and type of the physical condition has little association with the impact of these factors. Consequently, the prevailing model for properly understanding all pain disorders is the biopsychosocial or biobehavioral model. Both terms essentially highlight the same mechanisms in that the intersections of biology, mind, and social environment shape disease and illness. It is generally accepted today that certain affective and cognitive behavioral factors contribute to differences in individual pain or discomfort perception [9, 10]. For instance, and specifically relevant to the medical and dental settings, pain perception is influenced by factors such as somatosensory amplification (described below) and anxiety [11]. Figure 4.1 illustrates how psychological constructs and bodily processes interact to create functional disorders; these relationships, as modified from Sullivan and Katon [12], are central to how the biopsychosocial perspective becomes relevant in the clinical setting.

It is noteworthy that TMDs, through the publication of the Research Diagnostic Criteria for TMD (RDC/TMD) [13], have led the pain field in terms of explicit recognition and assessment of the psychosocial aspects of pain disorders, with other disease domains now building related approaches [14]. As science progresses, the nature and number of psychosocial constructs deemed relevant to a pain disorder may change, but the critical clinical issue is that the psychosocial aspect of the patient must be assessed in some form, rather than being sidestepped because the clinician (or researcher) believes that this particular pain does not have any psychosocial significance. When a pain disorder is regarded as purely a physical process, that is, a disease and associated nociception, the clinical view is, most often, one of regarding only the body as relevant. As a result, various kinds of tests, imaging, and the like are requested because they are believed to be critical, whereas assessing the biobehavioral status of the person or requesting a pain psychology consultation is considered irrelevant, too expensive, or both. But we now know that how a person functions – before the pain disorder emerges, or as a consequence of pain, or in relation to different aspects of physically oriented treatment – has been shown to be critical [15–21].

The obvious tendency in the orthodontic consulting room is to regard a malocclusion as a purely physical aberration of the body; yet, the evidence from many studies clearly demonstrates that the majority of individuals seek orthodontic

Table 4.1 Recommended psychosocial domains for patient assessment

Domain	Suggested instrument	# items	Screening evaluation	Comprehensive evaluation
Pain intensity and pain-related disability	Graded Chronic Pain Scale (GCPS)	7	✓	✓
Pain locations	Pain drawing	1	✓	✓
Limitation	Jaw Functional Limitation Scale-short form (JFLS)	8 or 20	✓	✓
Distress	Patient Health Questionnaire-4 (PHQ-4)	4	✓	
Depression	Patient Health Questionnaire-9 (PHQ-9)	9		✓
Anxiety	Generalized Anxiety Disorder-7 (GAD-7)	7		✓
Physical symptoms	Patient Health Questionnaire-15 (PHQ-15)	15		✓
Parafunction	Oral Behaviors Checklist (OBC)	21	✓	✓

A suggested psychological assessment instrument is listed for each domain, based on the DC/TMD, and the number of questions in each form (# items) is listed. Total items for a full *screening* evaluation is 43, while an abbreviated screening evaluation contains 12 items (see text). Total number of items for a *comprehensive* evaluation is 80. Any of these approaches is readily managed by patients in a clinic setting for completion of the questionnaires

treatment for reasons of aesthetics and not functional limitation. [22, 23] A case series by one of the present authors (AM) revealed that individuals with severe skeletal malocclusions also seek care mostly for aesthetics, not functional limitation [24]. Equally striking is that restoration of those patients to a functional Angle's Class I (mal) occlusion generally satisfies their chief complaint regarding aesthetics while simultaneously not providing any notable functional improvement. The latter absence of treatment effect, given the extreme physical impairment of such malocclusions prior to the treatment, remains a mystery. One salient point remains: malocclusions of all types are primarily aesthetic problems, and most individuals functionally adapt to severe malocclusions in an apparently sufficient manner, at least as indicated by the available data. One conclusion from this observation about structural problems versus aesthetics is that structural factors from malocclusions are clearly less important for understanding TMDs in comparison to psychosocial factors related to pain.

An assessment of TMD that incorporates the biopsychosocial domain can be as open ended as the practitioner wishes, and of course the direction and scope of such an evaluation will be determined by the nature of the chief complaint and what emerges in a symptom history as well as past history. From a pragmatic clinical perspective, where time in the consultation room is limited, and cognitive resources for gathering a history and putting the information together are equally limited, some type of structured evaluation for the psychosocial domain is generally both more efficient and clearly more reliable. The present authors have found that the structured format of the biobehavioral axis of the DC/TMD (as an update on the previously used RDC/TMD) is a very good place to start. Specifically, self-report instruments exist for each of the constructs of pain intensity, pain-related disability, functional limitation of the jaw, functional physical symptoms of the body, anxiety symptoms, depressive symptoms, and overuse oral behaviors. Collectively, these instruments adequately assess the current Diagnostic Criteria for TMD (DC/TMD) biobehavioral domain. Catastrophizing, not included in the DC/TMD at this stage, is the remaining pain-relevant construct that should be considered. These constructs are listed in Table 4.1, along with suggested instruments for their assessment.

Body dysmorphic disorder (BDD) is a particular psychosocial disorder of substantial relevance to both the orthodontic consulting room and this chapter. BDD is characterized by the belief that one's own appearance is particularly defective, and those concerns interfere with normal functioning [25]. Even if only a slight defect is present, the concern is markedly excessive. Clinically, the individual is noted to have pervasive thoughts about the defect, which tend to be intrusive and are thereby distressing. Patients with a malocclusion (of any magnitude) who present with an obsession regarding an imagined or a greatly exaggerated defect in their appearance may be more accurately diagnosed as having a BDD. These individuals can be easily misdiagnosed in terms of the source of the distress (i.e., the belief about the malocclusion rather than the malocclusion itself). This becomes particularly true if pain is an accompanying symptom, because that pain may automatically be attributed to a nociceptive response to a physical stimulus. Once nociception is inferred, then a structural or physical cause for the nociception becomes the clinical focus for identification.

Consequently, if the orthodontic consultation overemphasizes the significance of the particular features of the malocclusion and contrasts those features with the importance of having an "ideal" occlusion, the orthodontist unintentionally can create the perception of the malocclusion as a serious defect in the person's appearance, and therefore deserving of the patient's concerns. This may then contribute to the iatrogenic development (or worsening) of BDD [26–28].

4.3 Why Behavior Matters in the Differential Diagnosis of TMD?

The best way to appreciate why behavioral assessment should be a part of diagnosing a TMD patient is to describe a prototypical individual presenting with a malocclusion to an orthodontist. That patient could have a masticatory muscle myofascial pain disorder simultaneous with a clinically important TMJ disc disorder. These may arise originally as two separate disorders, stemming from separate causes at two unrelated points in time, or they may exist as interrelated disorders emerging at the same time from a single cause. The distinction in the time course and potentially different etiologies may or may not be important for clinical management – the clinician must make an assessment from the history. Simultaneous with these two Axis I diagnoses (physical categories in the DC/TMD), there might be clinically important Axis II symptoms (psychosocial categories in the DC/TMD). These could include depression and anxiety, catastrophizing, and poor coping skills, which might be pain related or, as previously illustrated, could be focused on the malocclusion. There also may be ongoing and likely enduring life stressors as well as substantial interference in function from the myofascial pain disorder, and these problems would be revealed as part of an Axis II evaluation.

Some of the Axis II characteristics in this hypothetical patient might be intrinsically related to the current Axis I problems, and others, for example, anxiety, may exist due to wholly separable aspects of the person's life and may have no impact on the pain. Alternatively, the anxiety may impact the patient's pain greatly; the clinician needs to take a history to make this determination. While the extent of these Axis II symptoms can be readily assessed with standardized self-report instruments (see Table 4.1), the relevance to the chief complaint or to a differential diagnosis must be determined from the history. In addition, the individual may exhibit sleep bruxism as an enduring aspect of a sleep parasomnia. This behavior might aggravate the disc disorder and, because there is a malocclusion, the impact of the sleep bruxism upon the disc disorder may be interpreted to be occlusion-mediated. Finally, the malocclusion might be fully functional and adaptive for the individual but aesthetically is unacceptable. Given the other regional problems (pain, interference in functioning from the joint, and tooth grinding), all of which have specific diagnoses based on current knowledge, the significance of the malocclusion is not likely to be great in this patient.

In this example, part of the differential diagnosis is to place each complaint, each disease, and each illness characteristic into context. Initially, the patient presents with the chief complaints of a malocclusion (notably highlighted here as problems in aesthetics) and a pain problem, so the clinician needs to use assessment and diagnostic criteria that are presumably validated for each respective characteristic. For example, the anxiety disorder is assessed based on the validated features of anxiety, not because the person "seems" anxious, and not because someone with all of the other problems would surely be anxious. A validated and standardized self-report assessment instrument for anxiety will rapidly indicate the extent of any anxiety symptoms, and an interview will identify their context and relation to the complaints and clinical problems under consideration. The clinician might say, "I notice that you are reporting a number of symptoms of anxiety. Does your pain get worse when you are more anxious? And, when you are more anxious, do you tend to focus more on how your teeth appear?"

Similarly, a malocclusion is diagnosed based on its own parameters, and not because the person has symptoms of a TMD which according to the biopsychosocial model could exist as a result of a variety of presumed causes. In summary, differential diagnosis requires consideration of all measurable and classifiable problems, placed into a context, and ranked according to plausible mechanisms. In a case scenario like the one described above, the present authors would place the functional but unaesthetic malocclusion at the bottom of a problem list in terms of priorities, to be addressed only after adequately resolving all of the other complaints and identified problems.

4.3.1 Parafunctional Behaviors

Oral parafunction takes many forms, including tooth-to-tooth behaviors (such as clenching, grinding, and pressing of the teeth together), teeth-separated behaviors (such as bracing or guarding the jaw), soft tissue behaviors (such as cheek biting or tongue posturing), and others (such as musical instrument placement or telephone cradling with the jaw). Among these parafunctions, teeth clenching and grinding, nail and object biting, and gum chewing are the most commonly reported [29–32]. While all oral parafunctions were once regarded as a result of malocclusion (e.g., sleep bruxism was the body's response to correcting an occlusal discrepancy; bracing the jaw was due to not having a comfortable centric occlusion), no causal relationship between any occlusal feature and oral parafunction has ever been supported by any evidence. In contrast, stress reactivity and habitual behavior have varying but sufficient levels of support as important features of oral parafunctions, and each of these belongs to the domain of psychosocial factors [33–36]. In addition, nocturnal bruxism has been identified as a sleep-related movement disorder with a multifactorial etiology.

The importance of oral parafunctional behaviors to both TMD and orthodontic treatment is becoming increasingly supported by research. Multiple studies have found significant associations between daytime oral parafunction (typically, clenching, and grinding) and myofascial pain [33, 35–37]. In addition, diurnal parafunctional activities can be a risk factor for disc displacement as well [37]. One proposed mechanism for parafunctional behaviors contributing to TMD is that such behaviors may overload the dentition and masticatory system [38, 39]. More specifically, the mechanism has been postulated to involve damage of muscle fibers [40] or to a reduction of blood supply [41]. Because most oral parafunction occurs at a low intensity level, but often for prolonged periods, the hypotheses of "overload" to the masticatory system and "damage" to muscle fibers clearly need better evidence and explanation regarding mechanism; at present, however, we do not know how parafunction exerts its apparently pathologic effects. A malocclusion coupled with a particular pattern of clenching or grinding might aggravate a disc displacement with reduction. However, not everyone with such malocclusions engages in parafunctional behaviors as described here, and those behaviors generally emerge independent of the malocclusion. The presence of the particular

occlusal features will often be seen by some clinicians as causing the patients to do the particular parafunctional behavior, but this represents attribution bias, not causation.

The mechanism by which parafunction may affect the dentition and masticatory system (whether by "overload" or other mechanism) is equally relevant for orthodontic diagnosis, treatment, and posttreatment retention. According to the belief of an "occlusion-centered" etiology for masticatory system overload, an orthodontist may focus on the evaluation of centric condylar position, dental or skeletal discrepancies, and occlusal interferences [42, 43]. However, during orthodontic treatment, teeth are moved considerably, and this induces a stream of constantly changing occlusal interferences and continuous bouts of occlusal instability. As a consequence of the continuous change in the occlusal pattern, the achievement of any "ideal centric" condylar position cannot be guaranteed.

Finally, during posttreatment retention, attention is given again to the potential presence of occlusal interferences, and sometimes a limited occlusal adjustment is suggested to finalize and stabilize the orthodontic treatment results [44, 45]. This entire process often has the unfortunate side effect of inducing patients and their dentists to focus their attention on the necessity of an ideal and perfect intercuspation and on checking for potential occlusal interferences. The increased attention of the patients on their teeth can be detrimental if, during the posttreatment retention phase, even a little relapse of the achieved "ideal" occlusion occurs, inducing worries and fears. Interestingly, empirical data exist in support of this observation; a recent study showed that individuals with a history of TMD developed symptoms and adapted less well to the introduction of an active occlusal interference when compared to subjects without a TMD history [46].

In summary, oral parafunctions are behaviors that are not caused by malocclusion of any type. Rather, they are a function of mental states such as anxiety, stress reactivity, or simple habit; in the case of sleep bruxism, it is part of the sleep disorder phenomenology. Therefore, it is not the kind of malocclusion a person has but rather what the person does with it (e.g., parafunction) that matters in terms of symptoms that appear to be caused by the occlusion but are not.

4.3.2 Psychological Traits

Trait anxiety, somatosensory amplification, and hypervigilance are three psychological constructs with specific relevance to the focus of this chapter. While trait anxiety has been studied for decades with respect to its strong relationship to health and disease, somatosensory amplification and hypervigilance were identified more recently as core constructs involved in functional disorders. Functional disorders are also known as idiopathic pain syndromes when pain is the primary symptom, and as medically unexplained symptoms in other contexts. A primary characteristic of such disorders is that the extent of illness greatly exceeds the extent of objectively measured disease.

Trait anxiety refers to a general pattern of physical dysregulation and worry that is an inherent characteristic of an individual [47–49]. Somatosensory amplification refers to the tendency of the individual to perceive a given somatic sensation as intense, noxious, and disturbing [50]. And, hypervigilance is an increased awareness of the discrepancy between the perceived sensation vs. what is expected as "normal", with a heightened attention typically focused on weak sensations. While each construct can occur independently of the others, together they appear to constitute a substantial triad linking a variety of psychosocial states with a variety of clinical disorders that overlap with TMD and problematic occlusions.

Trait anxiety might predispose to somatosensory amplification [51]. A number of studies have shown that somatosensory amplification is correlated with several indices of general distress including anxious and depressive symptoms [52–54]. Anxiety appears to influence the perception of orthodontic pain [55, 56], and individuals with prolonged pain during orthodontic treatment exhibit higher anxiety scores as compared to individuals with pain of short duration [57]. Orthodontic pain perception has been found to be

significantly greater in individuals with high trait anxiety and somatosensory amplification [58]. Perhaps either of these processes could underlie pain complaints during orthodontic treatment that are misdiagnosed as TMD.

Moreover, anxiety and somatosensory amplification could jointly contribute to a more pronounced attention bias toward a potentially threatening stimulus represented by occlusal interferences. It is likely that individuals with high anxiety and/or somatosensory amplification are also hypervigilant against a perceived discrepancy in perceived vs. expected sensation. Such individuals may exhibit an increased awareness of their occlusion, reacting to those somatic sensations with emotional affect and cognitions that intensify them and transform them into more alarming, threatening, and disturbing sensations [51].

Individuals with high trait anxiety also exhibit greater amounts of parafunction [59]. Indeed, high levels of trait anxiety indicate an anxious personality disposition, which can be associated with a high rate of environmental scanning and reduced ability to switch attention away from the threatening stimulus. So, it could be hypothesized that patients presenting with high-frequency parafunctional activity are *occlusally hypervigilant* and are more disturbed by the occlusal interferences and changes induced by orthodontic therapy, resulting in pain and dysfunction. Occlusal hypervigilance may be explained by the Generalized Hypervigilance Hypothesis, according to which hypervigilance is a "perceptual habit" that involves subjective amplification of a variety of aversive sensations, not just painful ones [60]. According to this hypothesis, if attention is habitually focused on sensations of a particular type, their amplification increases and became autonomous [61]. Moreover, it has also been reported that some myofascial pain patients have high levels of somatosensory amplification that is characterized by a general bodily hypervigilance to unpleasant sensations [62]. This hypothesis could explain why, during the orthodontic treatment process, which creates several occlusal interferences throughout a long period, some patients do not adapt to the occlusal changes. These individuals may develop TMD signs and symptoms, which are then misdiagnosed as being "caused" by the constantly changing occlusion during orthodontic treatment.

The general model for functional disorders in Fig. 4.1 can be applied more specifically to the orofacial region, as shown in Fig. 4.2; the lower section (in regular font) is modified after Ohrbach & McCall [63]. Indeed, an occlusal interference in individuals who exhibit somatic hypervigilance, symptom amplification, or catastrophizing can induce a muscle hyperactivity that becomes persistent, which in turn leads to additional hypervigilance, symptom amplification, or catastrophizing, and ultimately more distress. Health care utilization that results in a focus on structure as the source of the individual's distress will reinforce the hypervigilance, symptom amplification, catastrophizing, and distress. Collectively, these processes can lead to TMD pain, masticatory dysfunction, or both. Different levels of these factors might account for the high interindividual variability in perception of pain or discomfort observed in orthodontic patients during treatment [64, 65]. As a consequence, orthodontic practitioners should be aware of the psychological characteristics of their patients, and they should try to recognize those individuals who may represent yellow flags for irreversible dental treatments. This is especially true when patients report having multiple bad experiences with previous orthodontists, but now they have selected you to solve their problems.

4.4 Recommendations for Clinical Best Practice

The orthodontic treatment plan should always be tailored according to the chief complaint, to the problem list of the patient, and to evidence-based dentistry principles, all integrated within appropriate clinical decision-making principles. As a standard of practice, it is advisable before starting orthodontic treatment to always perform a screening examination for the presence of TMD (see Chap. 3). This examination should be coupled with at least a screening evaluation of psychosocial factors as presently defined by the Axis II of

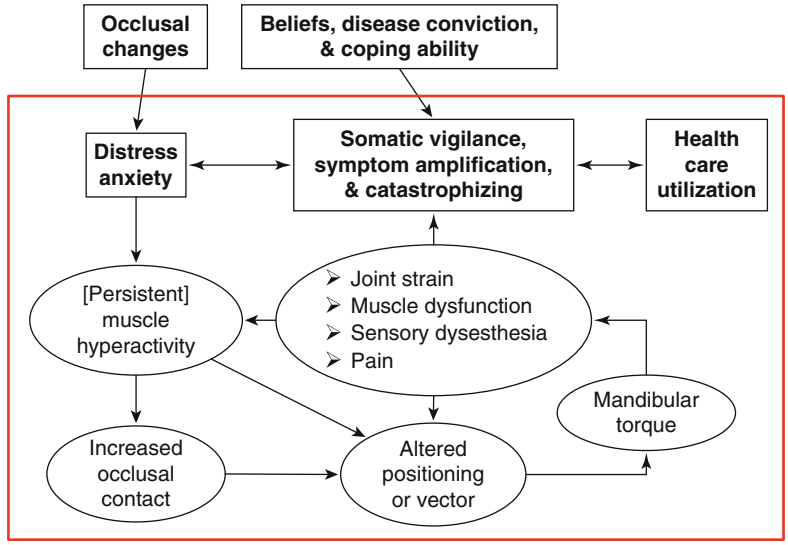


Fig. 4.2 Parafunction and clinical structural problems. Cyclic model in the lower part of the figure (regular font) illustrates how muscle hyperactivity (parafunctional behavior) causes altered positioning of the mandible. This change probably is mediated through occlusal contact, which results in torque and a potential range of symptoms that contribute to ongoing hyperactivity. This hyperactivity becomes persistent as a response (reactive behavior), or an attempted adaptive response may occur via more alteration in habitual mandibular positioning. Acute changes in occlusion as well as beliefs, disease conviction, and coping are exogenous processes that contribute to the resultant self-maintaining vicious cycle (Modified after Sullivan and Katon [12] and Ohrbach and McCall [63])

the DC/TMD (see Table 4.1). The complete instrument set listed in Table 4.1 is intended for a specialty pain practice. The screening set of instruments listed in the table is intended for routine clinical settings (including orthodontic consulting rooms); yet, we recognize that even this screening set may seem too burdensome in some treatment settings. Consequently, an even more abbreviated approach to screening could be based on using the Graded Chronic Pain Scale for pain intensity and disability, the patient health questionnaire(PHQ), PHQ-4 for distress, and a pain drawing to assess the presence of other pain disorders (which is one of the more significant red-flag indicators for further assessment). Collectively, these three instruments can be completed in less than one minute, and they provide a very good snapshot of a person's functional and pain status.

As described in this chapter, the identified Axis II constructs are relevant for not only TMD but for the differential diagnosis of other conditions relevant to the orthodontist. Depending on the severity of any findings from a TMD screening examination, a more comprehensive examination should be considered; similarly, positive findings from an Axis II screening evaluation should be followed by a more comprehensive Axis II instrument-based assessment, clinical interview, or referral to a mental health specialist. For medico-legal reasons, any positive findings from the TMD screening exam or from the assessment of psychosocial status should be recorded and updated at 6-monthly intervals [66, 67]. Guidelines for a structured examination were recently published at the International RDC/TMD Consortium Network [68], and the Axis II instruments are available at that same source.

In general, TMD treatment in the context of orthodontic practice still conforms to the general standards for TMD treatment, which are described in greater detail elsewhere in this book (See Chap. 9) as well as in other sources [1, 2]. Treatments should address not only the physical diagnosis but also the psychological distress and the psychosocial dysfunction [69]. The first stage in TMD treatment is symptom focused and behavioral [70], and it includes (as determined by the problem list) patient education, thermal

packs, home exercises, physiotherapy (to improve movements and function), pharmacotherapy (e.g., analgesics, anti-inflammatory agents, and antidepressants), control of overuse behaviors, and intraoral TMD appliances (See Chap. 9). In addition, psychological therapy (e.g., cognitive behavioral therapy, stress-management, and self-regulatory skills) should be part of the initial plan for the orthodontic patient with TMD and integrated as appropriate to the patient's readiness and symptom pattern.

4.4.1 A Behavioral Perspective on Usual TMD Treatment

Related to the present chapter, we wish to emphasize how the treatment procedures mentioned in the previous paragraph can have a specific psychosocial relevance. Patient education is regarded as a fundamental component of treatment within a biopsychosocial illness model. An explanation of the disorder and its supposed etiology as well as the good prognosis of this benign disorder is generally reassuring to the patient. Topics such as normal jaw muscle function, characteristics of a muscle or joint disorder, and a rehabilitative model of treatment should be explained. A key component is to emphasize the importance of avoiding overloading of the masticatory system, which could be a major cause of the complaints; the implication for the patient is that he or she has to try to control oral parafunctional behaviors. In particular for orthodontic situations associated with TMD, the task of clearly distinguishing the putative etiologic factors for each patient's TMD situation from concerns or beliefs about malocclusion is an essential part of patient education.

Behavioral therapy is focused on controlling parafunctional behaviors, as described earlier in this chapter regarding the pathogenesis of musculoskeletal pain. In order for behavioral therapy to succeed in the control of parafunction, sufficient time (typically, months) as well as continuous feedback and reinforcement from the clinician for this type of behavioral change must be provided. Patients must learn to keep the muscles relaxed by holding the mandible in a neutral position with the teeth separated rather than keeping the teeth in occlusion, because this fully closed jaw position requires "unintentional" muscle contraction [71]. A reliable convenience position of the mandible with a sufficiently low level of masticatory muscle activity can be obtained by asking the patients to pronounce several times the letter "N." This will locate the tongue in a neutral position, and then the patient should separate the teeth and relax the masticatory muscles while maintaining the lips in slight contact. Note that for patients with vertical maxillary excess, it may be difficult for them to maintain the lips in contact simultaneous with the teeth being separated and the masticatory (and facial) muscles relaxed. In this situation, additional patient education is needed in order to bridge the current structural situation with the orthodontic treatment goal (which is ultimately the correction of the maxillary excess and absence of lip patency).

Psychosocial factors generally play a role in the triggering of parafunctional behaviors, and consequently the control of the parafunctional behaviors may require more than simple behavioral therapy. The clinician, therefore, needs to monitor progress by the patient with respect to behavioral control and recognize when to refer for additional psychological treatment. For example, a persistent anxiety disorder (which would have been probably detected at the initial consultation via the PHQ-4 instrument) may emerge as a substantial barrier as the patient tries to control any overuse behaviors. If anxiety bouts overwhelm the patient, then the self-control of masticatory motor activity is likely to be displaced. In this instance the anxiety state needs further assessment, and probably specific treatment for it will be required.

One additional aspect of the behavioral program suggested for TMD patients with muscle pain and/or limited mouth opening includes relaxation exercises with diaphragmatic breathing. It is possible for the orthodontist or office staff to teach these skills [17], or a referral may be indicated to the appropriate mental health provider.

In summary, TMD treatment should include behavioral therapies ranging from auto massage

of the masticatory muscles to diaphragmatic breathing, and patients need the orthodontist to provide careful instruction, monitoring, and reinforcement in order for them to achieve sufficient compliance and adherence [72]. The key to success in TMD management depends a lot on the success in educating the patients about their role in managing the disorder. This will enhance the self-care aspects that allow the patients to better understand their symptoms and to independently manage them.

4.5 Summary

We wish to stress three main principles that have been discussed in this chapter:

The *first* involves symptom perception associated with occlusal structures. Indeed, cognitive (e.g., catastrophizing), attentional (e.g., hypervigilance), and perceptual (symptom amplification) factors clearly contribute to psychophysiologic reactivity and treatment seeking. Of these, one factor in particular (hypervigilance) merits investigation in association with occlusal variables. Hypervigilance can be considered a "perceptual habit," in which attention is focused on sensations of a particular type with subjective amplification of perceptions. Hypervigilance is hypothesized to account for reported outcomes of occlusal treatment in patients with TMD where complaints associated with the occlusion (e.g., the bite is not right, my teeth don't fit together, I can't find the right position on closing, etc.) are predominant. This situation has been labeled as occlusal dysesthesia, occlusal hyperawareness, and phantom bite [73, 74]. This condition arises subsequent to any modifications performed on the occlusion, and upon clinical examination there are no abnormal findings in regard to its anatomical or functional status. It can be speculated that hypervigilance, together with other psychological states such as anxiety or catastrophizing, could be a risk factor in patients with TMD when the planned therapeutic management (i.e., orthodontics) includes a modification of the occlusion. Any alteration of the existing occlusal pattern (even if minimally invasive) may, especially in these patients, trigger a "bodily distress disorder" (e.g., an occlusal dysesthesia disorder) leading to further distress.

The *second* involves the adaptive capability or resilience of the individual which could influence the definition of "normal" occlusion as well as the reaction to any typical occlusal interventions provided to patients with dental restorative or aesthetic needs. It has been shown that some TMDs can cause irreversible degenerative alterations in the temporomandibular joints. Consequently, the masticatory system reacts with adaptive changes, in some cases also irreversible, in an attempt to regain the functional equilibrium. In this clinical situation, any occlusal modification procedure performed, even if appropriately indicated for occlusal rehabilitation purposes and technically correct, may exceed the adaptive capability of the system, or the patient, or both. Adaptive capability is important for all aspects of dentistry, in that procedures regarded by the profession as routine may not be routine for a given individual. Therefore, occlusal modification performed as part of an extensive plan of restorative treatment may exceed that person's capability and trigger the onset of an iatrogenic "TMD." A complete understanding of the patient, from the level of the masticatory system to the level of the person, is essential in order to assess the complex dynamics involved in adaptive capacity; ultimately, this analysis will determine what the clinician should or should not do.

The *third* involves the clinical significance of oral parafunctional behaviors. Hyperactivity of the masticatory muscles may include both functional (chewing, e.g., habitual food, gum, tobacco, and sunflower seeds) and "parafunctional" oral behaviors. The latter group includes but is not restricted to nonfunctional tooth contacts; clenching and grinding of the teeth; biting of objects such as nails, fingers, or lips; and other behaviors such as bracing or

thrusting the jaw. The current literature does not support the belief that occlusion per se is the reason for the occurrence of nonfunctional tooth contacts. Before starting orthodontic treatment, the patient should be evaluated in order to determine (i) whether the patient already has increased muscle activity related to parafunctional behaviors, and (ii) whether the patient has TMD. The presence of parafunction increases the risk for symptoms associated with changing occlusion; the joint presence of parafunction and TMD increases the risk for TMD flare-ups associated with changing occlusion. Awareness of the consequences of these factors can reduce the likelihood of a bad doctor–patient interaction or poor treatment outcome, so the orthodontist should consider the psychosocial aspects of both TMD and occlusion-related complaints before finalizing an orthodontic diagnosis and initiating mechanical therapy for tooth movement.

Take Home Messages

Before or during orthodontic treatment:

- Screen for signs and symptoms of TMD using both physical and psychosocial assessments.
- If a TMD problem is present, screen for the biopsychosocial characteristics relevant to differential diagnosis in an orthodontic setting: the minimum includes pain intensity, pain disability, distress, and other pain disorders.
- Make an assessment for the presence of anxiety, hypervigilance, somatosensory amplification, and catastrophizing. Also, assess patient characteristics that interact with a changing occlusion during orthodontic treatment.
- Carefully evaluate for the presence of oral parafunctional behaviors.
- Utilize a symptom-focused and behavioral treatment protocol for managing the TMD condition.

References

1. de Leeuw R, Klasser GD, editors. Orofacial pain: guidelines for assessment, diagnosis, and management. 5th ed. Hanover Park: Quintessence Publishing; 2013.
2. Ohrbach R, Blasberg B, Greenberg MS. Temporomandibular disorders. In: Glick M, editor. Burket's oral medicine. 12th ed. Shelton: PMPH-USA, LTD.; 2015. p. 263–308.
3. Diatchenko L, Nackley AG, Slade GD, Fillingim RB, Maixner W. Idiopathic pain disorders – pathways of vulnerability. Pain. 2006;123:226–30.
4. 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.
5. Engel GL. The need for a new medical model: a challenge for biomedicine. Science. 1977;196:129–36.
6. Dworkin SF. Illness behavior and dysfunction: review of concepts and application to chronic pain. Can J Physiol Pharmacol. 1991;69:662–71.
7. Dworkin SF, Von Korff MR, LeResche L. Epidemiologic studies of chronic pain: a dynamic-ecologic perspective. Ann Behav Med. 1992;14:3–11.
8. Ceusters W, Smith B. A unified framework for biomedical technologies and ontologies. Stud Health Technol Inform. 2010;160(Part 2):1050–4.
9. Meana M, Lykins A. Negative affect and somatically focused anxiety in young women reporting pain with intercourse. J Sex Res. 2009;46:80–8.
10. Sturgeon JA, Zautra AJ. Psychological resilience, pain catastrophizing, and positive emotions: perspectives on comprehensive modeling of individual pain adaptation. Curr Pain Headache Rep. 2013;17:319–25.
11. Baeza-Velasco C, Gély-Nargeot MC, Bulbena Vilrasa A, Bravo JF. Joint hypermobility syndrome: problems that require psychological intervention. Rheumatol Int. 2011;31:1131–11346.
12. Sullivan M, Katon W. Somatization: the path between distress and somatic symptoms. APS J. 1993;2(3):141–9.
13. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomandib Disord Fac Oral Pain. 1992;6(4):301–55.
14. Fillingim RB, Bruehl S, Dworkin RH, Dworkin SF, Loeser JD, Turk DC, et al. The ACTTION-American Pain Society Pain Taxonomy (AAPT): an evidence-based and multidimensional approach to classifying chronic pain conditions. J Pain. 2014;15(3):241–9.
15. Epker J, Gatchel RJ, Ellis EI. A model for predicting chronic TMD: practical application in clinical settings. JADA. 1999;130:1470–5.
16. Gatchel RJ, Peng YB, Peters M, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychol Bull. 2007;133:581–624.
17. 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;16:48–63.
18. Rudy TE, Turk DC, Kubinski JA, Zaki HS. Differential treatment responses of TMD patients as a function of psychological characteristics. Pain. 1995;61:103–12.
19. Turk DC, Rudy TE. The robustness of an empirically derived taxonomy of chronic pain patients. Pain. 1990;43:27–35.
20. Fillingim RB, Slade GD, Diatchenko L, Dubner R, Greenspan JD, Knott C, et al. Summary of findings from the OPPERA baseline case-control study: implications and future directions. J Pain. 2011;12((11, Suppl 3)): T102–7.
21. Slade GD, Fillingim RB, Sanders AE, Bair E, Greenspan JD, Ohrbach R, et al. Summary of findings from the OPPERA prospective cohort study of incidence of first-onset temporomandibualr disorder: implications and future directions. J Pain. 2013;14(12, Suppl 2):T116–24.
22. Rusanen J, Silvola A-S, Tolvanen M, Pirttiniemi P, Lahti S, Sipilä K. Pathways between temporomandibular disorders, occlusal characteristics, facial pain, and oral health-related quality of life among patients with severe malocclusion. Europ J Orthod. 2012;34(4): 512–7.
23. Silvola A-S, Rusanen J, Tolvanen M, Pirttiniemi P, Lahti S. Occlusal characteristics and quality of life before and after treatment of severe malocclusion. Europ J Orthod. 2012;34(6):704–9.
24. Michelotti A, Iodice G. The role of orthodontics in temporomandibular disorders. J Oral Rehabil. 2010;37: 411–29.
25. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. In: Text revision (DSM-IV-TR). 4th ed. Arlington: APA; 2000.
26. Polo M. Body dysmorphic disorder: a screening guide for orthodontists. Am J Orthod Dentofacial Orthop. 2011;139(2):170–3.
27. Hepburn S, Cunningham S. Body dysmorphic disorder in adult orthodontic patients. Am J Orthod Dentofacial Orthop. 2006;130(5):569–74.
28. Leon-Salazar V, Morrow L, Schiffman EL. Pain and persistent occlusal awareness: what should dentists do? JADA. 2012;143(9):989–91.
29. Velly AM, Gornitsky M, Philippe P. Contributing factors to chronic myofascial pain: a case-control study. Pain. 2003;103:491–500.
30. Ohrbach R, Markiewicz MR, McCall Jr WD. Waking-state oral parafunctional behaviors: specificity and validity as assessed by electromyography. Eur J Oral Sci. 2008;116:438–44.
31. Feiteih RM. Signs and symptoms of temporomandibular disorders and oral parafunctions in urban Saudi Arabian adolescents: a research report. Head Face Med. 2006;2:25–31.
32. Farsi NM. Symptoms and signs of temporomandibular disorders and oral parafunctions among Saudi children. J Oral Rehabil. 2003;30:1200–8.
33. Glaros AG, Tabacchi KN, Glass EG. Effect of parafunctional clenching on TMD pain. J Orofacial Pain. 1998;12:145–52.
34. Glaros AG, Williams K, Lausten L. The role of parafunctions, emotions and stress in predicting facial pain. JADA. 2005;136:451–8.
35. Ohrbach R, Bair E, Fillingim RB, Gonzalez Y, Gordon SM, Lim PF, et al. Clinical orofacial characteristics associated with risk of first-onset TMD: the OPPERA prospective cohort study. Journal of Pain. 2013;14(12, Suppl 2):T33–50.
36. Ohrbach R, Fillingim RB, Mulkey F, Gonzalez Y, Gordon S, Gremillion H, et al. Clinical findings and pain symptoms as potential risk factors for chronic TMD: Descriptive data and empirically identified domains from the OPPERA case-control study. Journal of Pain. 2011;12(11, Suppl 3):T27–45.
37. 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.
38. Miyake R, Ohkubo R, Takehara J, Morita M. Oral parafunctions and association with symptoms of temporomandibular disorders in Japanese university students. J Oral Rehabil. 2004;31(6):518–23.
39. Winocur E, Littner D, Adams I, Gavish A. Oral habits and their association with signs and symptoms of temporomandibular disorders in adolescents: a gender comparison. Oral SurgOral Med Oral Pathol Oral Radiol Endod. 2006;102:482–7.
40. Larsson S-E, Bengtsson A, Bodegard L, Henriksson KG, Larsson J. Muscle changes in work-related chronic myalgia. Acta Orthop Scand. 1988;59:552–6.
41. Larsson B, Bjork J, Kadi F, Lindman R, Gerdle B. Blood supply and oxidative metabolism in muscle biopsies of female cleaners with and without myalgia. C J Pain. 2004;20:440–6.
42. Roth RH, Rolfs DA. Functional occlusion for the orthodontist. Part II. J Clin Orthod. 1981;15:100–23.
43. Martin D, Cocconi R. Orthodontic dental casts: the case for routine articulator mounting. Am J Orthod Dentofacial Orthop. 2012;141(1):8–14.
44. Kirveskari P, Alanen P, Jämsä T. Association between craniomandibular disorders and occlusal interferences in children. J Prosthet Dent. 1992;67(5):692–6.
45. Kirveskari P, Le Bell Y, Salonen M, Forssell H, Grans L. Effect of elimination of occlusal interferences on signs and symptoms of craniomandibular disorder in young adults. J Oral Rehabil. 1989;16(1):21–6.
46. Le Bell Y, Jamsa T, Korri S, Niemi PM, Alanen P. Effect of artificial occlusal interferences depends on previous experience of temporomandibular disorders. Acta Odontol Scand. 2002;60(4):219–22.
47. McEwen BS. Mood disorders and allostatic load. Biol Psychiatry. 2003;54:200–7.
48. Spielberger CD, Gorsuch RL, Lushene RE. Manual of the state-trait anxiety inventory. Palo Alto: Consulting Psychologists Press; 1970.
49. Spielberger CD, Sydeman SJ, Owen AE, Marsh BJ. Measuring anxiety and anger with the State-Trait Anxiety Inventory (STAI) and the State-Trait Anger

Expression Inventory (STAXI). In: Maruish ME, editor. The use of psychological testing for treatment planning and outcomes assessment. 2nd ed. Mahwah: Lawrence Erlbaum Associates, Publishers; 1999. p. 993–1021.
50. Barsky AJ. The amplification of somatic symptoms. Psychosom Med. 1988;50:510–9.
51. Barsky AJ. Amplification, somatization, and somatoform disorders. Psychosomatics. 1992;33:28–34.
52. Aronson KR, Barrett LF, Quigley KS. Feeling your body or feeling badly: evidence for the limited validity of the Somatosensory Amplification Scale as an index of somatic sensitivity. J Psychosom Res. 2001; 51:387–94.
53. Duddu V, Isaac MK, Chaturvedi SK. Somatization, somatosensory amplification, attribution styles and illness behaviour: a review. Int Rev Psychiatry. 2006;18:25–33.
54. Mantar A, Yemez B, Alkin T. The validity and reliability of the Turkish version of the anxiety sensitivity index-3. Turkish J Psychiatry. 2010;21:225–34.
55. Krishnan V. Orthodontic pain: from causes to management–a review. Eur J Orthod. 2007;29:170–9.
56. Minor V, Marris CK, McGorray SP, Yezierski R, Fillingim R, Logan H, et al. Effects of preoperative ibuprofen on pain after separator placement. Am J Orthod Dentofacial Orthop. 2009;136:510–7.
57. Bergius M, Broberg AG, Hakeberg M, Berggren U. Prediction of prolonged pain experiences during orthodontic treatment. Am J Orthod Dentofacial Orthop. 2008;133:e1–8.
58. Beck VJ, Farella M, Chandler NP, Keiser JA, Thomson WM. Factors associated with pain induced by orthodontic separators. J Oral Rehabil. 2014;41(4):282–8.
59. 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;26:168–75.
60. McDermid AJ, Rollman GB, McCain GA. Generalized hypervigilance in fibromyalgia: evidence of perceptual amplification. Pain. 1996;66:133–44.
61. Hollins M, Harper D, Gallagher S, Owings EW, Lim PF, Miller V, et al. Perceived intensity and unpleasantness of cutaneous and auditory stimuli: an evaluation of the generalized hypervigilance hypothesis. Pain. 2009;141(3):215–21.
62. Raphael KG, Marbach JJ, Klausner J. Myofascial face pain: clinical characteristics of those with regional vs widespread pain. JADA. 2000;131:161–71.
63. Ohrbach R, McCall Jr WD. The stress-hyperactivity-pain theory of myogenic pain: proposal for a revised theory. Pain Forum. 1996;5:51–66.
64. Cioffi I, Piccolo A, Tagliaferri R, Paduano S, Galeotti A, Martina R. Pain perception following first orthodontic archwire placement–thermoelastic vs superelastic alloys: a randomized controlled trial. Quintessence Int. 2012;43:61–9.
65. Wang J, Jian F, Chen J, Ye NS, Huang YH, Wang S, et al. Cognitive behavioral therapy for orthodontic pain control: a randomized trial. J Dent Res. 2012; 81:580–5.
66. Machen DE. Legal aspects of orthodontic practice: risk management concepts. Excellent diagnostic informed consent practice and record keeping make a difference. Am J Orthod Dentofacial Orthop. 1990; 98:381–2.
67. Machen DE. Legal aspects of orthodontic practice: risk management concepts. Disposing of your orthodontic practice: be careful. Am J Orthod Dentofacial Orthop. 1991;99:486–7.
68. Ohrbach R, Gonzalez Y, List T, Michelotti A, Schiffman E. Diagnostic criteria for temporomandibular disorders (DC/TMD) Clinical Examination Protocol International RDC/TMD Consortium Network2014 [March 14, 2014]. Available from: http://www.rdc-tmdinternational.org/Portals/18/protocol_DC-TMD/DC-TMDProtocol - 2013_06_02.pdf.
69. Ohrbach R. Disability assessment in temporomandibular disorders and masticatory system rehabilitation. J Oral Rehabil. 2010;37:452–80.
70. List T, Axelsson S. Management of TMD: evidence from systematic reviews and meta-analyses. J Oral Rehabil. 2010;6:430–51.
71. Michelotti A, Farella M, Vollaro S, Martina R. Mandibular rest position and electrical activity of the masticatory muscles. J Prosthet Dent. 1997;78(1):48–53.
72. Turk DC, Rudy TE. Neglected topics in the treatment of chronic pain patients – relapse, noncompliance, and adherence enhancement. Pain. 1991;44:5–28.
73. Bartilotta BY, Galang-Boquiren MT, Greene CS. Nonpainful phantom sensations in dentistry: an update on etiologic concepts. 2014; September/October:2–4, General Dentistry, Academy of General Dentistry. http://www.agd.org/publications-media/publications/general-dentistry/general-dentistry-archives.aspx
74. Ligas BB, Galang MTS, BeGole EA, Evans CA, Klasser GD, Greene CS. Phantom bite: a survey of US orthodontists. Orthodontics. 2011;12:38–47.

Sleep Bruxism: What Orthodontists Need to Know?

5

Gary D. Klasser and Ramesh Balasubramaniam

5.1 Definition of Sleep Bruxism

The American Academy of Sleep Medicine defines general bruxism in the International Classification of Sleep Disorders (ICSD-3 available only on website at http://www.aasmnet.org/library/default.aspx?id=9) as the following: A repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Furthermore, bruxism has been divided into two distinct categories based upon a 24 h circadian cycle as to when this activity occurs: sleep bruxism (SB – occurring during sleep) and awake bruxism (AB – occurring during wakefulness) [1].

5.2 Classification of Sleep Bruxism

According to the ICSD-3, the clinical criteria for classification as SB include the following: (A) presence of regular or frequent tooth grinding sounds occurring during sleep; (B) presence of one or more of the following clinical signs: (1) abnormal tooth wear consistent with above reports of tooth grinding during sleep; (2) transient morning jaw muscle pain or fatigue; and/or temporal headache; and/or jaw locking upon awakening consistent with above reports of tooth grinding during sleep. It should be noted that although polysomnography (PSG) is not required for the diagnosis of SB, it is ideally recorded with masseter and/or temporalis muscle activity along with audio-video signal to increase diagnostic reliability [1].

SB may be classified according to etiology into two distinct categories: (A) primary or idiopathic/essential SB which is without an identifiable cause or any associated medical problem and (B) secondary SB which is related to a medical condition (e.g., movement or sleep disorder, sleep disordered breathing, neurologic or psychiatric condition, drug/chemical related). Orthodontists should be aware that SB may be concomitant with many other sleep disorders such as sleep epilepsy, REM (rapid eye movement) behavior disorder, and sleep breathing disorders due to upper airway resistance or apnea-hypopnea events [2, 3].

SB motor events may also be classified according to motor activity based upon stringent criteria (Table 5.1). Using PSG and audio-video recordings (either ambulatory or from the sleep laboratory), motor activity pattern types based on

G.D. Klasser, DMD (✉)
Department of Diagnostic Sciences,
School of Dentistry, Louisiana State University
Health Sciences Center, New Orleans, LA, USA
e-mail: gklass@lsuhsc.edu

R. Balasubramaniam, BDSc, MS, FOMAA
School of Dentistry, University of Western Australia,
Crawley, WA, Australia

Private Practice, West Leederville, WA, Australia

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_5

Table 5.1 Criteria for classification of bruxism according to motor activity pattern types as recorded by electromyography (EMG)

Phasic (rhythmic) – more than three EMG bursts (masseter or temporalis muscles) at a frequency of 1 Hz, separated by two inter-burst pauses with each burst lasting between 0.25 and 2.0 s
Tonic (sustained) – one EMG burst lasting >2.0 s
Mixed events – combination of phasic/tonic

EMG electromyographic
Note: For each burst, EMG is 10–20 % or more of the voluntary contraction and each burst must last for at least 0.25 s

Table 5.2 Diagnostic grading system of bruxism, for clinical and research purposes [1]

Possible – based upon self-report using a questionnaire and/or the anamnestic part of the clinical examination
Probable – based upon self-report *plus* the inspection report of the clinical examination
Definite – based upon self-report, a clinical examination and a polysomnographic recording preferably containing audio/visual recordings

electromyographic (EMG) signals of masseter and/or temporalis muscles referred to as rhythmic masticatory muscle activity (RMMA) can be subdivided into phasic (rhythmic), tonic (sustained), and mixed events [4, 5]. The majority of these EMG events (88 %) are of the phasic or mixed variety while rarely do we observe the tonic type that characterizes clenching; these EMG events occur at a mean frequency of 5.4 to 5.8 episodes per hour of sleep [4–6].

Another classification system for SB recently developed by consensus among an international group of experts employs a novel diagnostic grading system for both clinical and research purposes using the terms possible, probable, and definite (Table 5.2) [1].

5.3 Epidemiology

The prevalence of SB is difficult to establish as most of the studies are based on self-report of bruxism and do not distinguish between SB and AB. It has been found that SB peaks during childhood and decreases with age without gender differences [7]. Based on self-report of tooth grinding awareness, SB affects about 8 % of the adult population [7–9]. In children and adolescents, however, there is high variability reported (4–46 %) due to the different age groups under investigation [10–15].

5.4 Risk Factors

There are a number of risk factors for SB including cigarette smoking (Odds Ratio, OR = 1.3), caffeine (OR = 1.4), alcohol (OR = 1.8), and recreational drugs such as ecstasy, cocaine, or amphetamines; medications such as selective serotonin reuptake inhibitors or haloperidol; and sleep disordered breathing (SDB) problems such as snoring (OR = 1.4) and obstructive sleep apnea (OSA; OR = 1.8) [16–22].

On the other hand, SB is a risk factor for tooth wear, damage and fracture, muscle fatigue and pain (primarily in the morning), headache, and temporomandibular disorders (TMD). Of interest, there is an increased risk for tooth wear, jaw muscle fatigue and difficulty with wide mouth opening among children with SB [16].

Orofacial pain has been reported in 66–84 % of SB patients [23, 24]. Contrary to popular belief, increased frequency of SB events is not associated with greater presence or intensity of pain [25, 26]. Rather, a low level of SB activity (between 2 and 4 episodes/h of sleep) increases the risk for orofacial pain and headache complaints among SB patients compared to those with a high level of SB activity (>4 episodes/h of sleep) [26].

5.5 Comorbidities

There are some medical disorders that may be comorbid with SB. Among these are certain sleep disorders including parasomnias such as sleep walking and sleep talking; enuresis; restless leg syndrome; and SDB [8, 22, 27–32]. Also, other medical disorders such as attention deficit hyperactivity disorder (ADHD) [33, 34], Parkinson's disease [35], epilepsy [36–38], and gastroesophageal reflux [39] may be comorbidities of SB.

5.6 Pathophysiology

5.6.1 Sleep Architecture

Normal sleep comprises two distinct states: NREM (non-rapid eye movement), which, based upon electroencephalography (EEG), is subdivided into three distinct stages (N1-3) and REM (rapid eye movement). A typical normal sleep pattern is where individuals progress from wakefulness to the NREM state, followed by the REM state and then cyclically alternating between REM and NREM stages. Overall, a night of sleep consists of approximately 75–80 % of NREM sleep and 20–25 % of REM sleep. Humans typically cycle through NREM/REM sleep stages at a rate of four to six times per sleep period with duration of each cycle being 90 to 110 min. NREM allows for physiological restoration and REM accommodates psychological restoration.

Young adult SB patients (20 to 40 years of age) without coexisting medical problems such as chronic pain or those experiencing OSA exhibit a normal sleep architecture [40]. When investigating the occurrence of SB during the sleep cycles at night, it has been found that SB events are higher in the second and third transition from NREM to REM sleep cycles as compared to the first and fourth cycles [41]. SB events are most frequently identified in the ascending period within a sleep cycle where there is a shift from deep NREM toward REM sleep associated with arousal activity and increase in sympathetic tone [42, 43]. Furthermore, it is important to appreciate that the manifestation of tooth grinding is preceded by a cascade of complex and well timed physiologic events (Table 5.3). Evidence regarding the pathophysiology of rhythmic masticatory muscle activity (RMMA) supports the hypothesis that this activity is associated with autonomic sympathetic cardiac activity and sleep arousals [6, 41, 44, 45]. Arousals are the response of the sleeping brain to external (environmental) and internal (physiological or pathological) stimuli [46]. The purpose of these arousals or active periods are that they are "windows" whereby the sleeping individual can readjust his/her body position, reset body temperature, and if any harmful event is perceived, can become fully awake, i.e., a fight or flight reaction could be triggered [47]. In normal healthy adults, sleep arousals occur between 6 and 14 times per hour of sleep and tend to occur at the end of a NREM period [48]. Approximately 80 % of SB events, i.e., repetitive jaw muscle contractions with or without tooth grinding, are observed during such recurrent arousal periods while the source of the genesis of the other 20 % is under investigation [49]. Evidence that SB and RMMA are associated with sleep arousal is supported by the observation that tooth grinding and RMMA can be

Table 5.3 Sequence of physiological events preceding the oromotor activity of rhythmic masticatory muscle activity/sleep bruxism (RMMA/SB) [44, 169]

Time (prior to RMMA or tooth grinding episode)	Physiologic event
−8 to −4 min	Increase in sympathetic cardiac activity Reduction in parasympathetic activity
−4 s	Increase in cortical – brain activity (sleep arousal) Presence of alpha and delta waves recorded on the EEG
−1 s	Increase in suprahyoid muscle (jaw opening muscles) tone (possibly involved in mandibular protrusion or airway patency) Increase in respiratory and cardiac frequency (tachycardia)
−0.8 s	Initiation of two large inspirations Modest but significant rise in blood pressure
Onset of RMMA	Initiation of phasic or tonic contraction of masseter and temporal muscles (jaw closing muscles), with or without tooth grinding. This is followed in about 60 % of SB episodes by swallowing activity
Note of importance	Approximately 80 % of RMMA events are associated with sleep arousals with or without accompanying leg or body movements
Note of importance	Over 90 % of RMMA/SB events could be predicted by an increasing heart rate of 110 %

RMMA rhythmic masticatory muscle activity, *EEG* electroencephalography, *SB* sleep bruxism

evoked experimentally through manipulations that trigger arousal [2, 6, 22, 23, 50]. Interestingly, there does not appear to be any presence of arousals during RMMA events in normal adult volunteers who do not experience SB [45].

5.6.2 Catecholamines and Neurochemistry

Catecholamines such as dopamine, norepinepherine, and serotonin have been suggested as being involved in SB pathophysiology [20, 40, 51]. Studies have reported that SB patients have elevated levels of catecholamines in their urine compared to controls, thus suggesting a link between stress and SB [52, 53]. In a pilot imaging study [54] involving dopamine, it was found that there was an asymmetric distribution of striatal dopamine binding sites in the brains of SB patients. However, the overall density of the striatal dopamine receptors was found to be within normal range in young adults with SB. In a clinical trial using L-dopa (a dopamine precursor), the results indicated an inhibitory effect on SB; however, when bromocriptine (a dopamine receptor agonist) was administered it did not result in any effect on SB events, and it failed to restore the imbalance of the striatal dopamine binding sites [55, 56].

The observation that smoking exacerbates tooth grinding provides indirect evidence for the role of the cholinergic system mediated through the nicotinic receptors as a mechanism for SB [57–59]. However, it remains to be determined if this occurrence is indeed due to the effect of nicotine receptor activation (increased vigilance and brain arousal), or if it increases the risk of SB as an oral habitual behavior.

5.6.3 Stress and Psychosocial influences

There is a common belief that stress and psychosocial variables contribute to SB. Studies suggest that children and adults reporting self-awareness of tooth grinding are more anxious, aggressive, and hyperactive [13, 17, 60–65]. However, the majority of these studies had methodological limitations resulting in rather weak evidence [66]. SB patients diagnosed by PSG displayed similar reaction times to vigilance as normal controls under an attention motor test condition [67]. Interestingly, the SB patients scored higher than the normal controls on anxiety regarding successful test performance. There is a suggestion among some studies that SB patients are more likely to deny the impact of life events due to their coping styles or personality [68, 69]. Additionally, in some case studies, masseter EMG activity increased during sleep following days with emotional or physical stressors; [70, 71] however, these findings were not consistent in all studies [72–74]. From these studies it can be concluded that there might exist a subgroup of SB patients whose response to life stressors includes excessive jaw motor activity and this reaction differs from that of normal individuals [66, 69, 75].

5.6.4 Genetic and Familial Predisposition

A genetic or familial predisposition for SB has been suggested by studies utilizing a questionnaire format or tooth wear examinations [76]. Twenty to 50 % of SB patients may have a family member who also reports tooth grinding during childhood [77–79]. Analyzing twin studies, it has been revealed that tooth grinding has greater concordance among monozygotic than dizygotic twins [80, 81]. Furthermore, the presence of SB in childhood persists in 86 % of adults [80]. In a large population-based cohort of young adult twins, it was reported that genetic factors accounted for 52 % of the total phenotypic variance [82]. In contrast, Michalowicz et al. [83], on the basis of a combined questionnaire and clinical study with almost 250 pairs of twins, concluded there was a lack of genetic correlation with SB. To date, no genetic variants or genetic inheritance patterns have been associated with SB. Yet, in a recent case-control study involving a Japanese population (non-related participants) it was found that the C allele carrier of the serotonin receptor 2A single nucleotide polymorphism

(rs6313) was associated with an (OR = 4.25) increased risk of SB [84]. This finding is the first to identify a specific genetic component contributing to the etiology of SB. Despite this finding, it must be understood that SB is a multi-factorial disorder in which many other factors including other candidate genes are most likely involved in the etiology of this oral motor behavior or activity.

5.6.5 Local Factors Including Dental Occlusion

Historically, the dental profession was quite convinced that SB was directly related to occlusal factors, and early studies seemed to indicate that occlusal corrections diminished or stopped this activity [85–87]. However, later studies challenged the concept that occlusal factors such as occlusal disharmony or premature tooth contacts could be considered as principal initiating factors, while other studies showed that SB activity was not reduced by occlusal therapy [88–91]. There has also been a lack of correlation between dental morphology (dental arch, occlusion) and SB events among SB adult patients assessed by PSG [92]. Furthermore, the average tooth contact time, including meals, in healthy individuals is approximately 17.5 min/day [93]. Usually tooth contact is absent during sleep without motor activity, whereas it does occur in association with arousal, swallowing, and motor activity [94, 95]. Tooth contacts seem to occur in clusters approximately every 90 to 120 min during the night, suggesting that tooth contact is a consequence of jaw closing muscle activation within a sequence following arousal rather than a cause [95–97]. Interestingly, patients who are edentulous exhibit RMMA when they sleep while not wearing their dentures [98, 99]. In a study by Manfredini et al. [100], it was concluded that the role of various occlusal features such as interferences and centric slides, bite relationships, horizontal overlap, and midline discrepancies in the pathogenesis of SB is very minor and the contribution of occlusion to the differentiation between bruxers and non-bruxers is negligible.

5.6.6 Salivary Flow, Airway Patency, and Jaw Motor Activity During Sleep

Swallowing is a normal physiologic oropharyngeal motor activity occurring five to ten times/hour during sleep, which is a much lower rate as compared to wakefulness (60 times/hour during non-eating periods) [101]. This decreased rate of swallowing during sleep may be related to a decrease in salivary secretion and/or reflex sensitivity. Swallowing seems to occur predominantly in light NREM sleep in relation to arousals [44, 101]. Swallowing has also been found to occur with approximately 60 % of RMMA events in both SB patients and normal adult individuals [102]. Masseter bursts associated with RMMA occur when esophageal pH decreased in SB patients who did not experience sleep-related gastroesophageal reflux [39]. The relationship between swallowing, esophageal pH, microarousals, and salivation requires further investigation as it relates to sleep.

There appears to be an interaction between airway patency and jaw motor activity during sleep. During sleep, due to a decrease in oropharyngeal muscle tonicity, the jaw is open for 90 % of the total sleep time [94]. Narrowing of the upper airway during sleep occurs as the mandible and the tongue collapse into the pharynx [103]. The reduction in this space is exacerbated when sleeping in the supine position as a result of gravitational forces. Intriguingly, 75 % of RMMA events also occur in the supine position [102]. Khoury et al. [104] reported that an increase in the amplitude of respiration was observed with a simultaneous and significant increase in the activation of the suprahyoid (jaw opening) muscles when RMMA events occur. This increase in respiratory amplitude preceding RMMA, however, seems more likely to be associated with an autonomic drive during arousals rather than to function as an opening of the upper airway after an apneic event. Furthermore, studies have shown that RMMA events rarely present after apneic events [105]. Therefore, it remains to be demonstrated whether or not SB is a reactive-protective mechanism of the upper airway to overcome upper airway collapse.

5.7 Clinical Features of Sleep Bruxism

5.7.1 Tooth Grinding Reports

A primary feature of SB is tooth grinding noise. When clinically assessing the presence of SB it is imperative to differentiate tooth grinding noise due to SB from that of other oral sounds emitted from the mouth and throat during sleep such as snoring, grunting, groaning, vocalization, tongue clicking, lip smacking, or temporomandibular joint noise [106]. Additionally, sounds made from the bed itself due to movements and sleeping position changes also must be taken into account. Clearly, it is very difficult for a tooth grinding history to be reliably elicited from the patients who do not have a sleep partner or who are edentulous. In certain individuals, fluctuation in grinding history may be associated with jaw muscle symptoms or other risk factors such as stressors and medication use [58, 107, 108]. Therefore, tooth grinding noise should not be used as the sole determinant of SB activity.

5.7.2 Tooth Wear

The severity of tooth wear can be assessed according to published criteria [109, 110]. However, it is not possible to separate patients with SB from those without by observing tooth wear factors [111], as tooth wear may be produced by other etiologic factors (oral habits, food consistency, acid reflux, alimentary disorders, etc.); therefore, occlusal attrition cannot be considered an accurate indicator of this habit being currently performed [112]. Menapace et al. [113] reported that tooth wear was present in 100 % of SB patients but also in 40 % of asymptomatic individuals. Abe et al. [114] determined that SB patients (young adults) present with greater tooth wear as compared to controls (no report of any history of tooth grinding or sleep laboratory evidence of SB) but tooth wear was not able to discriminate between different sub-groups (moderate/high versus low) of SB patients. Furthermore, SB cannot be assumed to exist if there is no current report of tooth grinding as witnessed by a sleep partner, since the tooth wear may have occurred years before the SB activity.

5.7.3 Jaw Muscle Symptoms

Muscle pain (myalgia) and dysfunction symptoms related to SB may be quite different than those related to concomitant disorders. SB patients most frequently report myalgia on awakening in the morning, whereas masticatory myofascial pain intensifies as the day progresses [115, 116]. Other orofacial symptoms associated with TMD such as limitation in opening, TMJ noise, and arthralgia can be present concomitantly [117]. Although studies have suggested an association between self-reported SB and TMD, causation has not been clearly established [116, 118]. Furthermore, PSG studies have been unable to confirm such a link [119–121]. Raphael et al. [122] in a case-control study (124 vs. 46; all females) investigating the association between SB and myofascial TMD, using two-night laboratory PSG monitoring, found no statistically significant differences in SB rates among cases (9.7 %) compared to controls (10.9 %). They concluded there was no relationship between SB and myofascial TMD, but their study did not address the possibility that SB could be involved in the initial onset or triggering of myofascial TMD. Their findings merely emphasized that treatment aimed at reducing SB among those who already have chronic myofascial TMD may be inappropriate, since myofascial TMD patients do not brux at excessive rates while asleep. Other studies, using PSG and masseter EMG recordings, have reported that SB patients with orofacial pain report significantly less bruxism episodes per hour of sleep and less EMG activity in the masticatory muscles during sleep than pain free controls [123, 124]. It appears the association between orofacial pain symptoms and SB may be somewhat dependent on poor sleep, as pain and sleep have a bidirectional association [116, 125, 126].

5.7.4 Muscle Hypertrophy

Masseter muscle hypertrophy may be bilaterally manually palpated. If these muscles are hypertrophic, the volume of muscle tissue increases approximately two times while the teeth are clenched in comparison to a relaxed state [2]. However, masseter muscle hypertrophy does not strictly imply sleep muscle activity as it can also occur as a result of awake clenching [127].

5.7.5 Awake Clenching

As previously discussed, awake bruxism or AB is considered a distinct nosologic entity from SB. AB, based upon self-report studies, tends to be mainly a reactive process and is induced or exaggerated by stressors and/or anxiety or hyperactivity [107, 128]. SB patients often report an awareness of AB, with patients who have mild SB more often being cognizant of AB and stress than those with severe SB [26]. Physiologic recordings in subjects with and without orofacial pain while experiencing natural stress (before an examination) or during experimental stress (mental calculations) revealed increases in muscle tone, heart rate and/or voluntary chewing/clenching [129–131]. The clinical consequences associated with AB may deleteriously impact dental structures (natural dentition and prosthetic devices) and/or involve pain and dysfunction of the jaw musculature and joints [120, 132–134].

5.7.6 Headaches

Headache is a common finding in the general adult population with a lifetime prevalence of 85–95 % [135]. Headache is also a problem in children, with as many as 70 % of children being affected at least once in childhood [136, 137]. The prevalence of reported headache-related complaints among SB patients is also high (60–90 % of SB patients) [138–140]. Children who have migraine headaches have been shown to have a high prevalence of sleep disturbances, including snoring and SB [141]. Furthermore, it has been reported that 30–50 % of SB adult patients complain of headache either in the morning (most frequently) or during the day [142]. In a descriptive PSG study, it was reported that within a SB patient population spanning from 23 to 67 years of age, 65 % reported morning headaches [143]. The exact mechanisms underlying the possible interactions between SB and headache requires further investigation, but this is a difficult challenge due to the high prevalence of headaches in general.

SB may be a possible cause of tension-type headaches if patients wake with facial and/or temporal skull area pain, with pain typically subsiding as the day progresses [24, 71, 121]. These morning headaches may be explained as a post-exercise soreness in the temporalis muscles [144]. SB patients may report waking up in the middle of the night with pain and tension in facial and cranial areas following sustained SB events. In a study by Kampe et al. [62], 14 % of SB patients reported pain at night, while 31 % reported pain during both at night and daytime. It is important to recognize that nocturnal pain and headaches that may be induced by SB can be confused with similar symptoms experienced by fibromyalgia patients, which include muscle tenderness areas and morning stiffness, fatigue, and poor sleep [145, 146].

5.7.7 Sleep Disordered Breathing (SDB)

A cause and effect relationship between SB and SDB, which is a combination of upper airway resistance syndrome and OSA, has yet to be established despite frequent claims of an association among these entities [17]. However, other studies have shown a correlation between habitual snoring and SB [147]. In a PSG study, increased masticatory EMG activity including RMMA was detected in approximately 50 % (10/21) of adult patients) with OSA [22]. In another PSG study investigating sleep disorders among a group of 53 myofascial pain patients

(75 % met self-report criteria for SB, but only 17 % met PSG criteria for active SB), two or more sleep disorders were diagnosed in 43 % of those patients; insomnia disorder (36 %) and OSA (28.4 %) demonstrated the highest frequencies [119]. In another PSG study involving 119 patients between the ages of 2–16 years referred to a pediatric sleep center for snoring, SB was identified in 70 patients [148]. There have been clinical observations and some studies that have provided indirect evidence of a relationship between SB and SDB by reporting a decrease in SB after the patients have undergone treatments (adenotonsillectomy and continuous positive airway pressure) for the underlying sleep disorder [149–151]. These findings support the hypothesis that RMMA may be a sleep oromotor activity that assists in reinstating airway patency following a respiratory obstruction [104, 152]. It is important to note that the association between apnea/hypopnea and arousals is opposite to the association between SB and arousals; apneic events trigger arousals, while RMMA is triggered during arousals [105]. Nonetheless, several studies failed to show a temporal association between apneic events and RMMA; instead, tonic masseter muscle activity is frequently found at the termination of apneic events [22, 29, 153]. Overall, the factors responsible for the induction of increased RMMA frequency in patients with SB require further investigation.

5.7.8 Gastroesophageal Reflux

In a study of healthy young adults, it was reported that a significant relationship between decreased esophageal pH and RMMA, short EMG bursts and tooth clenching seems to occur when the person is sleeping mainly in a supine position. Of note, only about 10 % of the episodes of decreased esophageal pH (defined as a rapidly decreasing intraesophageal pH with a decrease of more than 0.4 per 2 s) included clenching episodes and the number of clenching episodes was independent of various sleep positions [154]. More specifically, it was found that RMMA is a secondary event to gastroesophageal reflux occurring via sleep arousal and often associated with swallowing [39]. Furthermore, RMMA events including SB were induced by esophageal acidification [155]. It has been proposed that preventing gastroesophageal reflux and avoiding sleeping in a supine position might be effective in decreasing the frequency of SB [154]. Overall, the physiologic link between SB, the increase in salivation and the association with gastroesophageal reflux requires further investigation.

5.8 Diagnostic Considerations

5.8.1 Clinical Assessment

SB is frequently reported to dentists or physicians by the patient and/or bed partner and parents. Given a positive report about tooth grinding, the diagnosis of SB is usually clinical, based on the observation of the following signs and symptoms: abnormal tooth wear, hypertrophy of masseter muscles, fatigue, discomfort or pain of jaw muscles [156]. However, none of these clinical findings is a direct proof of current SB activity. Tooth wear for example, although widely reported as the distinctive dental sign of bruxism in general may be related to many other factors that can influence the presence of attrition and erosion on dental surfaces.

There is an intraoral appliance (Bruxocore™) that indirectly assesses the mechanical impact of SB on the dentition [157, 158]. This appliance covers the upper dentition and is worn for a few weeks while the patient is sleeping, and the surface area and volume of attrition on the appliance are evaluated. When this technique is employed, it has been found that jaw muscle activities during sleep are not always correlated with the degree of wear. Therefore, to reliably and accurately diagnose SB, electronic recording and documenting devices are utilized with strict criteria to detect and classify SB activity. It is also important that the presence of other conditions such as orofacial pain, headache, and SDB be assessed in patients with SB by questionnaire at the time of initial examination.

5.8.2 Ambulatory Monitoring

Attempts have been made to monitor SB activity in natural home settings using ambulatory monitoring. Despite the obvious benefits of these devices such as lower cost and being used in the natural environment, the specificity of SB motor activity assessment remains a limitation [2]. In the absence of simultaneous audio-visual recording, it is difficult to exclude the presence of non-SB-specific orofacial movements during sleep such as swallowing and scratching [159]. A novel portable EMG device (Grindcare®) has been designed to provide online recording of EMG activity, online processing of EMG signals to detect a particular oromotor activity (tooth grinding/tooth clenching), and also for use as a biofeedback device. Encouraging results have been reported from several studies where this device has been utilized due its ability to detect EMG events associated with SB, and to exclude orofacial movements unrelated to SB (grimaces, swallowing, etc.) [160, 161]. In a systematic review assessing the diagnostic accuracy of ambulatory monitoring devices compared to PSG in the measurement of SB, it was concluded that the validity of portable instrumental diagnostic approaches is not sufficient to support any non-PSG techniques employed as a stand-alone diagnostic method in the research setting, with the possible exception of the Bruxoff® device which needs to be further confirmed with future investigations [162].

5.8.3 Sleep Laboratory Recording

Although a variety of tools have been developed to assess jaw muscle activity during sleep, the gold standard for SB diagnosis remains a full night PSG audio-video recording (highly controlled but in an unnatural environment). This is the only protocol, which allows the simultaneous monitoring of sleep electroencephalographic, electrocardiographic, electromyographic, and respiratory signals during sleep. However, PSG recordings are not routinely performed for clinical SB diagnosis, as they are both costly and time consuming. A PSG investigation may be indicated in cases of SB associated with other signs and symptoms suggestive of other sleep disorders, especially SDB. In these cases, the patient should be referred to a sleep physician for further investigations and diagnosis.

5.9 Management of Sleep Bruxism

Treatment of SB is primarily based on managing the harmful consequences of SB. Currently there are three strategies available for the management of SB, namely: (1) behavioral measures; (2) occlusal therapies; and (3) pharmacologic therapies (Table 5.4). Prior to treatment, SB patients need to be questioned about other comorbid medical conditions (e.g., SDB, insomnia, ADHD, depression, mood disorders, gastroesophageal reflux), especially when considering a pharmacotherapeutic approach. This provides an opportunity for management of SB and associated comorbidities, but it should be recognized that some management strategies may aggravate associated comorbidities.

There are many behavioral measures such as cognitive behavioral therapy and biofeedback available for the management of SB with only weak to moderate evidence. However, these strategies are typically cost effective and safe.

Similarly, there are occlusal therapies which are mostly reversible and with good short-term evidence for the management of SB [163]. As these therapies are without significant side effects, they also may be used in the long term. However, there are now studies, which have reported aggravation of snoring and OSA with the use of a stabilization-type maxillary occlusal splint for the management of SB. Therefore, clinicians considering oral appliance therapy for SB should screen patients for snoring and OSA. The effect of the mandibular occlusal splint on snoring and OSA is yet to be investigated [164, 165].

There are several drugs with probable centrally-acting mechanisms involving the dopaminergic, serotoninergic, and adrenergic systems for the management of SB [20]. The evidence on

Table 5.4 Management strategies for sleep bruxism

	Strategy	Comment
Behavioral [160, 170–173]	Avoidance of risk factors: smoking, alcohol, caffeine, drug use	Weak evidence
	Relaxation techniques	Weak evidence
	Good sleep hygiene	Weak evidence
	Hypnotherapy	Weak evidence
	Biofeedback	Moderate evidence in short term
	Cognitive behavioral therapy	Moderate evidence in short term
Occlusal therapies	Occlusal adjustments/removal of occlusal interference	No evidence
	Occlusal appliance [6, 173–178]	Decrease SB activity for 2 weeks only, but able to protect dentition from wear
	Anterior appliance (e.g., Hawley anterior platform or mini-anterior type) [179–185]	No better than full coverage occlusal appliance No evidence of long-term efficacy or safety
	Mandibular advancement appliance [186]	Decrease SB activity (up to 70 % reduction) during sleep, especially when worn in advanced positions (50–75 % of the maximal protrusion of the patients). No evidence of long-term efficacy or safety
Pharmacologic	Clonazepam [187]	40 % decrease in SB activity in the short term with risk for tolerance and dependency.
	Buspirone [188]	Weak evidence
	Clonidine [189]	Reduced SB by 60 %; however associated with severe hypotension in the morning
	Gabapentin [190]	Decrease in jaw muscle EMG and improved sleep. Need larger studies to reproduce this finding
	Botulinum toxin [191, 192]	Decrease in jaw muscle EMG activity during sleep. Its effect is short term

their efficacy and safety is quite minimal, so they should only be considered in severe symptomatic patients and only as a short-term therapy [166].

5.10 The Effects of Sleep Bruxism on Orthodontic Procedures

Currently there are no available data on the prevalence of SB during orthodontics. Also, the effect of orthodontic treatment on SB is unknown. Similarly, the effect of SB on orthodontic treatments or outcomes is unknown. Theories proposing that the attainment of an "ideal occlusion" after orthodontics may negate SB and TMD have largely been debunked. One study reported a decrease in anterior teeth wear by patient report alone after orthodontic treatment was performed on 296 children and adolescent patients [167], suggesting that orthodontic treatment may have a similar effect as oral appliance therapy. However, this study could not exclude AB activity, nor did it study SB utilizing PSG. Hence, the suggestion that orthodontic treatment may temporarily interrupt or permanently reduce parafunctional activities is unsubstantiated. In another study, it was reported that previous orthodontic treatment did not alter the presence of current bruxism (i.e., no better or worse) [168].

Based on a rational approach and clinical experience, SB is not a contraindication for orthodontic treatment. However, if a patient has clinically significant TMD symptoms related to SB, it is prudent that the TMD should be managed prior to embarking on orthodontic treatment to minimize the likelihood of interruption or alteration of the orthodontic treatment plan. Similarly, if TMD related to SB occurs during active

orthodontic treatment, it will be necessary to interrupt that process and treat the pain and dysfunction prior to continuation of orthodontic treatment (see Chap. 3).

Once orthodontic treatment has been completed in a patient with SB, the fabrication of an occlusal splint to protect the dentition and provide retention may be appropriate. The utilization of standard removable or lingual bonded orthodontic retainers is unlikely to withstand the forces of SB and probably will require frequent replacement, so other retention strategies should be considered.

Take Home Messages

- Sleep bruxism is mainly regulated by the central nervous system rather than being initiated peripherally.
- Sleep bruxism is not a simple jaw movement like chewing, but rather there are rhythmic movements with intense jaw muscle contractions.
- Management of sleep bruxism should focus on protecting the stomatognathic system from its harmful consequences, and interventions to do so should be conservative and reversible.
- Orthodontists should screen for sleep bruxism and sleep disordered breathing prior to initiating orthodontic treatment.
- Orthodontists should realize that establishing an "ideal" occlusion is not a preventive or curative measure for sleep bruxism.

References

1. Lobbezoo F, Ahlberg J, Glaros AG, Kato T, Koyano K, Lavigne GJ, et al. Bruxism defined and graded: an international consensus. J Oral Rehabil. 2013;40:2–4.
2. Lavigne GJ, Manzini C, Kato T. Sleep bruxism. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 4th ed. Philadelphia: Elsevier; 2005. p. 946–59.
3. Saito M, Yamaguchi T, Mikami S, Watanabe K, Gotouda A, Okada K, et al. Temporal association between sleep apnea-hypopnea and sleep bruxism events. J Sleep Res. 2014;23:196–203.
4. Lavigne GJ, Rompre PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res. 1996;75:546–52.
5. Lavigne GJ, Rompre PH, Poirier G, Huard H, Kato T, Montplaisir JY. Rhythmic masticatory muscle activity during sleep in humans. J Dent Res. 2001;80:443–8.
6. Macaluso GM, Guerra P, Di Giovanni G, Boselli M, Parrino L, Terzano MG. Sleep bruxism is a disorder related to periodic arousals during sleep. J Dent Res. 1998;77:565–73.
7. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep. 1994;17:739–43.
8. Ohayon MM, Roberts RE. Comparability of sleep disorders diagnoses using DSM-IV and ICSD classifications with adolescents. Sleep. 2001;24:920–5.
9. Maluly M, Andersen ML, Dal-Fabbro C, Garbuio S, Bittencourt L, de Siqueira JT, et al. Polysomnographic study of the prevalence of sleep bruxism in a population sample. J Dent Res. 2013;92(7 Suppl):S97–103.
10. Simola P, Niskakangas M, Liukkonen K, Virkkula P, Pitkaranta A, Kirjavainen T, et al. Sleep problems and daytime tiredness in Finnish preschool-aged children-a community survey. Child Care Health Dev. 2010;36:805–11.
11. Petit D, Touchette E, Tremblay RE, Boivin M, Montplaisir J. Dyssomnias and parasomnias in early childhood. Pediatrics. 2007;119:e1016–25.
12. Cheifetz AT, Osganian SK, Allred EN, Needleman HL. Prevalence of bruxism and associated correlates in children as reported by parents. J Dent Child (Chic). 2005;72:67–73.
13. Laberge L, Tremblay RE, Vitaro F, Montplaisir J. Development of parasomnias from childhood to early adolescence. Pediatrics. 2000;106(1 Pt 1):67–74.
14. Strausz T, Ahlberg J, Lobbezoo F, Restrepo CC, Hublin C, Ahlberg K, et al. Awareness of tooth grinding and clenching from adolescence to young adulthood: a nine-year follow-up. J Oral Rehabil. 2010;37:497–500.
15. Manfredini D, Restrepo C, Diaz-Serrano K, Winocur E, Lobbezoo F. Prevalence of sleep bruxism in children: a systematic review of the literature. J Oral Rehabil. 2013;40:631–42.
16. Carra MC, Huynh N, Morton P, Rompre PH, Papadakis A, Remise C, et al. Prevalence and risk factors of sleep bruxism and wake-time tooth clenching in a 7- to 17-yr-old population. Eur J Oral Sci. 2011;119:386–94.
17. Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. Chest. 2001;119:53–61.
18. Wise M. Citalopram-induced bruxism. Br J Psychiatry. 2001;178:182.
19. Baylen CA, Rosenberg H. A review of the acute subjective effects of MDMA/ecstasy. Addiction. 2006; 101:933–47.
20. Winocur E, Gavish A, Voikovitch M, Emodi-Perlman A, Eli I. Drugs and bruxism: a critical review. J Orofac Pain. 2003;17:99–111.

21. Dinis-Oliveira RJ, Caldas I, Carvalho F, Magalhaes T. Bruxism after 3,4-methylenedioxymethamphetamine (ecstasy) abuse. Clin Toxicol (Phila). 2010;48:863–4.
22. Sjoholm TT, Lowe AA, Miyamoto K, Fleetham JA, Ryan CF. Sleep bruxism in patients with sleep-disordered breathing. Arch Oral Biol. 2000;45:889–96.
23. Bader G, Lavigne G. Sleep bruxism; an overview of an oromandibular sleep movement disorder. REVIEW ARTICLE. Sleep Med Rev. 2000;4:27–43.
24. Camparis CM, Siqueira JT. Sleep bruxism: clinical aspects and characteristics in patients with and without chronic orofacial pain. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006;101:188–93.
25. Nagamatsu-Sakaguchi C, Minakuchi H, Clark GT, Kuboki T. Relationship between the frequency of sleep bruxism and the prevalence of signs and symptoms of temporomandibular disorders in an adolescent population. Int J Prosthodont. 2008;21:292–8.
26. Rompre PH, Daigle-Landry D, Guitard F, Montplaisir JY, Lavigne GJ. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res. 2007;86:837–42.
27. Bruni O, Fabrizi P, Ottaviano S, Cortesi F, Giannotti F, Guidetti V. Prevalence of sleep disorders in childhood and adolescence with headache: a case–control study. Cephalalgia. 1997;17:492–8.
28. Sforza E, Zucconi M, Petronelli R, Lugaresi E, Cirignotta F. REM sleep behavioral disorders. Eur Neurol. 1988;28:295–300.
29. Inoko YSK, Morita O, Kohno M. Relationship between masseter muscle activity and sleep-disordered breathing. Sleep Biol Rhyth. 2004;2:67–8.
30. Kato T, Yamaguchi T, Okura K, Abe S, Lavigne GJ. Sleep less and bite more: sleep disorders associated with occlusal loads during sleep. J Prosthodont Res. 2013;57:69–81.
31. Okeson JP, Phillips BA, Berry DT, Cook YR, Cabelka JF. Nocturnal bruxing events in subjects with sleep-disordered breathing and control subjects. J Craniomandib Disord. 1991;5:258–64.
32. Phillips BA, Okeson J, Paesani D, Gilmore R. Effect of sleep position on sleep apnea and parafunctional activity. Chest. 1986;90:424–9.
33. Silvestri R, Gagliano A, Arico I, Calarese T, Cedro C, Bruni O, et al. Sleep disorders in children with Attention-Deficit/Hyperactivity Disorder (ADHD) recorded overnight by video-polysomnography. Sleep Med. 2009;10:1132–8.
34. Herrera M, Valencia I, Grant M, Metroka D, Chialastri A, Kothare SV. Bruxism in children: effect on sleep architecture and daytime cognitive performance and behavior. Sleep. 2006;29:1143–8.
35. Tan EK, Jankovic J, Ondo W. Bruxism in Huntington's disease. Mov Disord. 2000;15:171–3.
36. Meletti S, Cantalupo G, Volpi L, Rubboli G, Magaudda A, Tassinari CA. Rhythmic teeth grinding induced by temporal lobe seizures. Neurology. 2004;62:2306–9.
37. Bisulli F, Vignatelli L, Naldi I, Licchetta L, Provini F, Plazzi G, et al. Increased frequency of arousal parasomnias in families with nocturnal frontal lobe epilepsy: a common mechanism? Epilepsia. 2010;51:1852–60.
38. Tinuper P, Provini F, Bisulli F, Vignatelli L, Plazzi G, Vetrugno R, et al. Movement disorders in sleep: guidelines for differentiating epileptic from non-epileptic motor phenomena arising from sleep. Sleep Med Rev. 2007;11:255–67.
39. Miyawaki S, Tanimoto Y, Araki Y, Katayama A, Fujii A, Takano-Yamamoto T. Association between nocturnal bruxism and gastroesophageal reflux. Sleep. 2003;26:888–92.
40. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil. 2008;35:476–94.
41. Huynh N, Kato T, Rompre PH, Okura K, Saber M, Lanfranchi PA, et al. Sleep bruxism is associated to micro-arousals and an increase in cardiac sympathetic activity. J Sleep Res. 2006;15:339–46.
42. Halasz P, Terzano M, Parrino L, Bodizs R. The nature of arousal in sleep. J Sleep Res. 2004;13:1–23.
43. Terzano MG, Parrino L, Boselli M, Smerieri A, Spaggiari MC. CAP components and EEG synchronization in the first 3 sleep cycles. Clin Neurophysiol. 2000;111:283–90.
44. Lavigne GJ, Huynh N, Kato T, Okura K, Adachi K, Yao D, et al. Genesis of sleep bruxism: motor and autonomic-cardiac interactions. Arch Oral Biol. 2007;52:381–4.
45. Kato T, Rompre P, Montplaisir JY, Sessle BJ, Lavigne GJ. Sleep bruxism: an oromotor activity secondary to micro-arousal. J Dent Res. 2001;80:1940–4.
46. Carra MC, Rompre PH, Kato T, Parrino L, Terzano MG, Lavigne GJ, et al. Sleep bruxism and sleep arousal: an experimental challenge to assess the role of cyclic alternating pattern. J Oral Rehabil. 2011;38:635–42.
47. Terzano MG, Parrino L. Origin and significance of the Cyclic Alternating Pattern (CAP). REVIEW ARTICLE. Sleep Med Rev. 2000;4:101–23.
48. Terzano MG, Parrino L, Rosa A, Palomba V, Smerieri A. CAP and arousals in the structural development of sleep: an integrative perspective. Sleep Med. 2002;3:221–9.
49. Kato T, Montplaisir JY, Guitard F, Sessle BJ, Lund JP, Lavigne GJ. Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. J Dent Res. 2003;82:284–8.
50. Reding GR, Zepelin H, Robinson Jr JE, Zimmerman SO, Smith VH. Nocturnal teeth-grinding: all-night psychophysiologic studies. J Dent Res. 1968;47:786–97.
51. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil. 2001;28:1085–91.
52. Clark GT, Rugh JD, Handelman SL. Nocturnal masseter muscle activity and urinary catecholamine levels in bruxers. J Dent Res. 1980;59:1571–6.
53. Vanderas AP, Menenakou M, Kouimtzis T, Papagiannoulis L. Urinary catecholamine levels and

bruxism in children. J Oral Rehabil. 1999;26: 103–10.
54. Lobbezoo F, Soucy JP, Montplaisir JY, Lavigne GJ. Striatal D2 receptor binding in sleep bruxism: a controlled study with iodine-123-iodobenzamide and single-photon-emission computed tomography. J Dent Res. 1996;75:1804–10.
55. Lavigne GJ, Soucy JP, Lobbezoo F, Manzini C, Blanchet PJ, Montplaisir JY. Double-blind, crossover, placebo-controlled trial of bromocriptine in patients with sleep bruxism. Clin Neuropharmacol. 2001;24:145–9.
56. Lobbezoo F, Lavigne GJ, Tanguay R, Montplaisir JY. The effect of catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. Mov Disord. 1997;12:73–8.
57. Lavigne GL, Lobbezoo F, Rompre PH, Nielsen TA, Montplaisir J. Cigarette smoking as a risk factor or an exacerbating factor for restless legs syndrome and sleep bruxism. Sleep. 1997;20:290–3.
58. Ahlberg J, Savolainen A, Rantala M, Lindholm H, Kononen M. Reported bruxism and biopsychosocial symptoms: a longitudinal study. Community Dent Oral Epidemiol. 2004;32:307–11.
59. Madrid G, Madrid S, Vranesh JG, Hicks RA. Cigarette smoking and bruxism. Percept Mot Skills. 1998; 87(3 Pt 1):898.
60. Manfredini D, Landi N, Fantoni F, Segu M, Bosco M. Anxiety symptoms in clinically diagnosed bruxers. J Oral Rehabil. 2005;32:584–8.
61. Pingitore G, Chrobak V, Petrie J. The social and psychologic factors of bruxism. J Prosthet Dent. 1991; 65:443–6.
62. Kampe T, Tagdae T, Bader G, Edman G, Karlsson S. Reported symptoms and clinical findings in a group of subjects with longstanding bruxing behaviour. J Oral Rehabil. 1997;24:581–7.
63. Kampe T, Edman G, Bader G, Tagdae T, Karlsson S. Personality traits in a group of subjects with longstanding bruxing behaviour. J Oral Rehabil. 1997; 24:588–93.
64. Manfredini D, Ciapparelli A, Dell'Osso L, Bosco M. Mood disorders in subjects with bruxing behavior. J Dent. 2005;33:485–90.
65. Restrepo CC, Vasquez LM, Alvarez M, Valencia I. Personality traits and temporomandibular disorders in a group of children with bruxing behaviour. J Oral Rehabil. 2008;35:585–93.
66. Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. J Orofac Pain. 2009;23:153–66.
67. Major M, Rompre PH, Guitard F, Tenbokum L, O'Connor K, Nielsen T, et al. A controlled daytime challenge of motor performance and vigilance in sleep bruxers. J Dent Res. 1999;78:1754–62.
68. Ahlberg K, Ahlberg J, Kononen M, Partinen M, Lindholm H, Savolainen A. Reported bruxism and stress experience in media personnel with or without irregular shift work. Acta Odontol Scand. 2003;61: 315–8.
69. Schneider C, Schaefer R, Ommerborn MA, Giraki M, Goertz A, Raab WH, et al. Maladaptive coping strategies in patients with bruxism compared to nonbruxing controls. Int J Behav Med. 2007;14:257–61.
70. Funch DP, Gale EN. Factors associated with nocturnal bruxism and its treatment. J Behav Med. 1980; 3:385–97.
71. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. Adv Neurol. 1988;49:329–41.
72. Makino M, Masaki C, Tomoeda K, Kharouf E, Nakamoto T, Hosokawa R. The relationship between sleep bruxism behavior and salivary stress biomarker level. Int J Prosthodont. 2009;22:43–8.
73. Pierce CJ, Chrisman K, Bennett ME, Close JM. Stress, anticipatory stress, and psychologic measures related to sleep bruxism. J Orofac Pain. 1995;9:51–6.
74. Watanabe T, Ichikawa K, Clark GT. Bruxism levels and daily behaviors: 3 weeks of measurement and correlation. J Orofac Pain. 2003;17:65–73.
75. Giraki M, Schneider C, Schafer R, Singh P, Franz M, Raab WH, et al. Correlation between stress, stress-coping and current sleep bruxism. Head Face Med. 2010;6:2.
76. Lobbezoo F, Visscher CM, Ahlberg J, Manfredini D. Bruxism and genetics: a review of the literature. J Oral Rehabil. 2014;41:709–14.
77. Abe K, Shimakawa M. Genetic and developmental aspects of sleeptalking and teeth-grinding. Acta Paedopsychiatr. 1966;33:339–44.
78. Kuch EV, Till MJ, Messer LB. Bruxing and nonbruxing children: a comparison of their personality traits. Pediatr Dent. 1979;1:182–7.
79. Reding GR, Rubright WC, Zimmerman SO. Incidence of bruxism. J Dent Res. 1966;45:1198–204.
80. Hublin C, Kaprio J, Partinen M, Koskenvuo M. Sleep bruxism based on self-report in a nationwide twin cohort. J Sleep Res. 1998;7:61–7.
81. Lindqvist B. Bruxism in twins. Acta Odontol Scand. 1974;32:177–87.
82. Rintakoski K, Hublin C, Lobbezoo F, Rose RJ, Kaprio J. Genetic factors account for half of the phenotypic variance in liability to sleep-related bruxism in young adults: a nationwide Finnish twin cohort study. Twin Res Hum Genet. 2012;15:714–9.
83. Michalowicz BS, Pihlstrom BL, Hodges JS, Bouchard Jr TJ. No heritability of temporomandibular joint signs and symptoms. J Dent Res. 2000;79:1573–8.
84. Abe Y, Suganuma T, Ishii M, Yamamoto G, Gunji T, Clark GT, et al. Association of genetic, psychological and behavioral factors with sleep bruxism in a Japanese population. J Sleep Res. 2012;21:289–96.
85. Ramfjord SP. Bruxism, a clinical and electromyographic study. J Am Dent Assoc. 1961;62:21–44.
86. Ash MM, Ramfjord SP. Occlusion. 4th ed. Philadelphia: W B Saunders; 1995.
87. Guichet NE. Occlusion: a teaching manual. Anaheim: The Denar Corporation; 1977.
88. Rugh JD, Barghi N, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxism. J Prosthet Dent. 1984;51:548–53.

89. Kardachi BJ, Bailey JO, Ash MM. A comparison of biofeedback and occlusal adjustment on bruxism. J Periodontol. 1978;49:367–72.
90. Tsukiyama Y, Baba K, Clark GT. An evidence-based assessment of occlusal adjustment as a treatment for temporomandibular disorders. J Prosthet Dent. 2001; 86:57–66.
91. Clark GT, Tsukiyama Y, Baba K, Watanabe T. Sixty-eight years of experimental occlusal interference studies: what have we learned? J Prosthet Dent. 1999;82:704–13.
92. Lobbezoo F, Rompre PH, Soucy JP, Iafrancesco C, Turkewicz J, Montplaisir JY, et al. Lack of associations between occlusal and cephalometric measures, side imbalance in striatal D2 receptor binding, and sleep-related oromotor activities. J Orofac Pain. 2001;15:64–71.
93. Graf H. Bruxism. Dent Clin North Am. 1969; 13:659–65.
94. Miyamoto K, Ozbek MM, Lowe AA, Sjoholm TT, Love LL, Fleetham JA, et al. Mandibular posture during sleep in healthy adults. Arch Oral Biol. 1998;43:269–75.
95. Powell RN. Tooth contact during sleep: association with other events. J Dent Res. 1965;44:959–67.
96. Baba K, Clark GT, Watanabe T, Ohyama T. Bruxism force detection by a piezoelectric film-based recording device in sleeping humans. J Orofac Pain. 2003;17:58–64.
97. Powell RN, Zander HA. The frequency and distribution of tooth contact during sleep. J Dent Res. 1965;44:713–7.
98. Okeson JP, Phillips BA, Berry DT, Cook Y, Paesani D, Galante J. Nocturnal bruxing events in healthy geriatric subjects. J Oral Rehabil. 1990;17:411–8.
99. von Gonten AS, Palik JF, Oberlander BA, Rugh JD. Nocturnal electromyographic evaluation of masseter muscle activity in the complete denture patient. J Prosthet Dent. 1986;56:624–9.
100. Manfredini D, Visscher CM, Guarda-Nardini L, Lobbezoo F. Occlusal factors are not related to self-reported bruxism. J Orofac Pain. 2012;26:163–7.
101. Lichter I, Muir RC. The pattern of swallowing during sleep. Electroencephalogr Clin Neurophysiol. 1975;38:427–32.
102. Miyawaki S, Lavigne GJ, Pierre M, Guitard F, Montplaisir JY, Kato T. Association between sleep bruxism, swallowing-related laryngeal movement, and sleep positions. Sleep. 2003;26:461–5.
103. Kato TLG. Sleep bruxism: a sleep-related movement disorder. Sleep Med Clin. 2010;5:9–35.
104. Khoury S, Rouleau GA, Rompre PH, Mayer P, Montplaisir JY, Lavigne GJ. A significant increase in breathing amplitude precedes sleep bruxism. Chest. 2008;134:332–7.
105. Kato T. Sleep bruxism and its relation to obstructive sleep apnea–hypopnea syndrome. Sleep Biol Rhythms. 2004;2:1–15.
106. Kato T, Thie NM, Montplaisir JY, Lavigne GJ. Bruxism and orofacial movements during sleep. Dent Clin North Am. 2001;45:657–84.
107. Egermark I, Carlsson GE, Magnusson T. A 20-year longitudinal study of subjective symptoms of temporomandibular disorders from childhood to adulthood. Acta Odontol Scand. 2001;59:40–8.
108. Lavigne GJ, Guitard F, Rompre PH, Montplaisir JY. Variability in sleep bruxism activity over time. J Sleep Res. 2001;10:237–44.
109. Johansson A, Haraldson T, Omar R, Kiliaridis S, Carlsson GE. A system for assessing the severity and progression of occlusal tooth wear. J Oral Rehabil. 1993;20:125–31.
110. Lobbezoo F, Naeije M. A reliability study of clinical tooth wear measurements. J Prosthet Dent. 2001;86: 597–602.
111. Pergamalian A, Rudy TE, Zaki HS, Greco CM. The association between wear facets, bruxism, and severity of facial pain in patients with temporomandibular disorders. J Prosthet Dent. 2003;90:194–200.
112. Lavigne GJ, Goulet JP, Zuconni M, Morrison F, Lobbezoo F. Sleep disorders and the dental patient: an overview. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999;88:257–72.
113. Menapace SE, Rinchuse DJ, Zullo T, Pierce CJ, Shnorhokian H. The dentofacial morphology of bruxers versus non-bruxers. Angle Orthod. 1994;64:43–52.
114. Abe S, Yamaguchi T, Rompre PH, De Grandmont P, Chen YJ, Lavigne GJ. Tooth wear in young subjects: a discriminator between sleep bruxers and controls? Int J Prosthodont. 2009;22:342–50.
115. Dao TT, Lund JP, Lavigne GJ. Comparison of pain and quality of life in bruxers and patients with myofascial pain of the masticatory muscles. J Orofac Pain. 1994;8:350–6.
116. Svensson P, Jadidi F, Arima T, Baad-Hansen L, Sessle BJ. Relationships between craniofacial pain and bruxism. J Oral Rehabil. 2008;35:524–47.
117. Okeson JP. Occlusal appliance therapy. In: Management of temporomandibular disorders and occlusion. 6th ed. St. Louis: Mosby; 2008. p. 468–97.
118. Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? J Orofac Pain. 1997;11:15–23.
119. Smith MT, Wickwire EM, Grace EG, Edwards RR, Buenaver LF, Peterson S, et al. Sleep disorders and their association with laboratory pain sensitivity in temporomandibular joint disorder. Sleep. 2009;32:779–90.
120. Rossetti LM, Pereira de Araujo Cdos R, Rossetti PH, Conti PC. Association between rhythmic masticatory muscle activity during sleep and masticatory myofascial pain: a polysomnographic study. J Orofac Pain. 2008;22:190–200.
121. Camparis CM, Formigoni G, Teixeira MJ, Bittencourt LR, Tufik S, de Siqueira JT. Sleep bruxism and temporomandibular disorder: clinical and polysomnographic evaluation. Arch Oral Biol. 2006; 51:721–8.
122. Raphael KG, Sirois DA, Janal MN, Wigren PE, Dubrovsky B, Nemelivsky LV, et al. Sleep bruxism and myofascial temporomandibular disorders: a laboratory-based polysomnographic investigation. J Am Dent Assoc. 2012;143:1223–31.

123. Arima T, Arendt-Nielsen L, Svensson P. Effect of jaw muscle pain and soreness evoked by capsaicin before sleep on orofacial motor activity during sleep. J Orofac Pain. 2001;15:245–56.
124. Lavigne GJ, Rompre PH, Montplaisir JY, Lobbezoo F. Motor activity in sleep bruxism with concomitant jaw muscle pain. A retrospective pilot study. Eur J Oral Sci. 1997;105:92–5.
125. Okifuji A, Hare BD. Do sleep disorders contribute to pain sensitivity? Curr Rheumatol Rep. 2011;13: 528–34.
126. Merrill R. Orofacial pain and sleep. Sleep Med Clin. 2010;5:131–44.
127. Kato T, Dal-Fabbro C, Lavigne GJ. Current knowledge on awake and sleep bruxism: overview. Alpha Omegan. 2003;96:24–32.
128. Glaros AG. Incidence of diurnal and nocturnal bruxism. J Prosthet Dent. 1981;45:545–9.
129. Ruf S, Cecere F, Kupfer J, Pancherz H. Stress-induced changes in the functional electromyographic activity of the masticatory muscles. Acta Odontol Scand. 1997;55:44–8.
130. Carlson CR, Okeson JP, Falace DA, Nitz AJ, Curran SL, Anderson D. Comparison of psychologic and physiologic functioning between patients with masticatory muscle pain and matched controls. J Orofac Pain. 1993;7:15–22.
131. Rao SM, Glaros AG. Electromyographic correlates of experimentally induced stress in diurnal bruxists and normals. J Dent Res. 1979;58:1872–8.
132. Carlsson GE, Egermark I, Magnusson T. Predictors of signs and symptoms of temporomandibular disorders: a 20-year follow-up study from childhood to adulthood. Acta Odontol Scand. 2002;60:180–5.
133. van Selms MK, Lobbezoo F, Visscher CM, Naeije M. Myofascial temporomandibular disorder pain, parafunctions and psychological stress. J Oral Rehabil. 2008;35:45–52.
134. Velly AM, Gornitsky M, Philippe P. A case–control study of temporomandibular disorders: symptomatic disc displacement. J Oral Rehabil. 2002;29:408–16.
135. Biondi DM. Headaches and their relationship to sleep. Dent Clin North Am. 2001;45:685–700.
136. Laurell K, Larsson B, Eeg-Olofsson O. Prevalence of headache in Swedish schoolchildren, with a focus on tension-type headache. Cephalalgia. 2004;24: 380–8.
137. Zwart JA, Dyb G, Holmen TL, Stovner LJ, Sand T. The prevalence of migraine and tension-type headaches among adolescents in Norway. The Nord-Trondelag Health Study (Head-HUNT-Youth), a large population-based epidemiological study. Cephalalgia. 2004;24:373–9.
138. Molina OF, Dos Santos Jr J, Nelson SJ, Grossman E. Prevalence of modalities of headaches and bruxism among patients with craniomandibular disorder. Cranio. 1997;15:314–25.
139. Hamada T, Kotani H, Kawazoe Y, Yamada S. Effect of occlusal splints on the EMG activity of masseter and temporal muscles in bruxism with clinical symptoms. J Oral Rehabil. 1982;9:119–23.
140. Yustin D, Neff P, Rieger MR, Hurst T. Characterization of 86 bruxing patients with long-term study of their management with occlusal devices and other forms of therapy. J Orofac Pain. 1993;7:54–60.
141. Miller VA, Palermo TM, Powers SW, Scher MS, Hershey AD. Migraine headaches and sleep disturbances in children. Headache. 2003;43:362–8.
142. Lavigne G, Palla S. Transient morning headache: recognizing the role of sleep bruxism and sleep-disordered breathing. J Am Dent Assoc. 2010;141:297–9.
143. Bader GG, Kampe T, Tagdae T, Karlsson S, Blomqvist M. Descriptive physiological data on a sleep bruxism population. Sleep. 1997;20:982–90.
144. Lund JP. Pain and the control of muscles. In: Fricton JR, Dubner R, editors. Advances in pain research and therapy, Orofacial pain and temporomandibular disorders, vol. 21. New York: Raven; 1995. p. 103–15.
145. Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. Arthritis Rheum. 1990;33:160–72.
146. Moldofsky HK. Disordered sleep in fibromyalgia and related myofascial facial pain conditions. Dent Clin North Am. 2001;45:701–13.
147. Ng DK, Kwok KL, Poon G, Chau KW. Habitual snoring and sleep bruxism in a paediatric outpatient population in Hong Kong. Singapore Med J. 2002;43: 554–6.
148. Sheldon SH. Obstructive sleep apnea and bruxism in children. Sleep Med Clin. 2010;5:163–8.
149. DiFrancesco RC, Junqueira PA, Trezza PM, de Faria ME, Frizzarini R, Zerati FE. Improvement of bruxism after T & A surgery. Int J Pediatr Otorhinolaryngol. 2004;68:441–5.
150. Eftekharian A, Raad N, Gholami-Ghasri N. Bruxism and adenotonsillectomy. Int J Pediatr Otorhinolaryngol. 2008;72:509–11.
151. Oksenberg A, Arons E. Sleep bruxism related to obstructive sleep apnea: the effect of continuous positive airway pressure. Sleep Med. 2002;3:513–5.
152. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med. 2003;14:30–46.
153. Yoshida K. A polysomnographic study on masticatory and tongue muscle activity during obstructive and central sleep apnea. J Oral Rehabil. 1998;25: 603–9.
154. Miyawaki S, Tanimoto Y, Araki Y, Katayama A, Imai M, Takano-Yamamoto T. Relationships among nocturnal jaw muscle activities, decreased esophageal pH, and sleep positions. Am J Orthod Dentofacial Orthop. 2004;126:615–9.
155. Ohmure H, Oikawa K, Kanematsu K, Saito Y, Yamamoto T, Nagahama H, et al. Influence of experimental esophageal acidification on sleep bruxism: a randomized trial. J Dent Res. 2011;90:665–71.
156. American Academy of Sleep Medicine. Sleep related bruxism. In: American Academy of Sleep Medicine, editors. ICSD-2 International classification of sleep disorders: diagnosis and coding man-

ual. Westchester: American Academy of Sleep Medicine; 2005. p. 189–192.
157. Ommerborn MA, Giraki M, Schneider C, Schaefer R, Gotter A, Franz M, et al. A new analyzing method for quantification of abrasion on the Bruxcore device for sleep bruxism diagnosis. J Orofac Pain. 2005; 19:232–8.
158. Pierce CJ, Gale EN. Methodological considerations concerning the use of Bruxcore Plates to evaluate nocturnal bruxism. J Dent Res. 1989;68:1110–4.
159. Dutra KM, Pereira Jr FJ, Rompre PH, Huynh N, Fleming N, Lavigne GJ. Oro-facial activities in sleep bruxism patients and in normal subjects: a controlled polygraphic and audio-video study. J Oral Rehabil. 2009;36:86–92.
160. Jadidi F, Castrillon E, Svensson P. Effect of conditioning electrical stimuli on temporalis electromyographic activity during sleep. J Oral Rehabil. 2008; 35:171–83.
161. Jadidi F, Castrillon EE, Nielsen P, Baad-Hansen L, Svensson P. Effect of contingent electrical stimulation on jaw muscle activity during sleep: a pilot study with a randomized controlled trial design. Acta Odontol Scand. 2013;71:1050–62.
162. Manfredini D, Ahlberg J, Castroflorio T, Poggio CE, Guarda-Nardini L, Lobbezoo F. Diagnostic accuracy of portable instrumental devices to measure sleep bruxism: a systematic literature review of polysomnographic studies. J Oral Rehabil. 2014;4:836–42.
163. Huynh NT, Rompre PH, Montplaisir JY, Manzini C, Okura K, Lavigne GJ. Comparison of various treatments for sleep bruxism using determinants of number needed to treat and effect size. Int J Prosthodont. 2006;19:435–41.
164. Gagnon Y, Mayer P, Morisson F, Rompre PH, Lavigne GJ. Aggravation of respiratory disturbances by the use of an occlusal splint in apneic patients: a pilot study. Int J Prosthodont. 2004;17:447–53.
165. Nikolopoulou M, Ahlberg J, Visscher CM, Hamburger HL, Naeije M, Lobbezoo F. Effects of occlusal stabilization splints on obstructive sleep apnea: a randomized controlled trial. J Orofac Pain. 2013;27:199–205.
166. Huynh N, Manzini C, Rompre PH, Lavigne GJ. Weighing the potential effectiveness of various treatments for sleep bruxism. J Can Dent Assoc. 2007; 73:727–30.
167. Hirsch C. No increased risk of temporomandibular disorders and bruxism in children and adolescents during orthodontic therapy. J Orofac Orthop. 2009; 70:39–50.
168. Fujita Y, Motegi E, Nomura M, Kawamura S, Yamaguchi D, Yamaguchi H. Oral habits of temporomandibular disorder patients with malocclusion. Bull Tokyo Dent Coll. 2003;44:201–7.
169. Nashed A, Lanfranchi P, Rompre P, Carra MC, Mayer P, Colombo R, et al. Sleep bruxism is associated with a rise in arterial blood pressure. Sleep. 2012;35:529–36.
170. Shulman J. Teaching patients how to stop bruxing habits. J Am Dent Assoc. 2001;132:1275–7.
171. Lobbezoo F, van der Zaag J, van Selms MK, Hamburger HL, Naeije M. Principles for the management of bruxism. J Oral Rehabil. 2008;35:509–23.
172. Wieselmann-Penkner K, Janda M, Lorenzoni M, Polansky R. A comparison of the muscular relaxation effect of TENS and EMG-biofeedback in patients with bruxism. J Oral Rehabil. 2001;28:849–53.
173. Ommerborn MA, Schneider C, Giraki M, Schafer R, Handschel J, Franz M, et al. Effects of an occlusal splint compared with cognitive-behavioral treatment on sleep bruxism activity. Eur J Oral Sci. 2007;115:7–14.
174. Nascimento LL, Amorim CF, Giannasi LC, Oliveira CS, Nacif SR, Silva Ade M, et al. Occlusal splint for sleep bruxism: an electromyographic associated to Helkimo Index evaluation. Sleep Breath. 2008;12: 275–80.
175. Harada T, Ichiki R, Tsukiyama Y, Koyano K. The effect of oral splint devices on sleep bruxism: a 6-week observation with an ambulatory electromyographic recording device. J Oral Rehabil. 2006;33:482–8.
176. van der Zaag J, Lobbezoo F, Wicks DJ, Visscher CM, Hamburger HL, Naeije M. Controlled assessment of the efficacy of occlusal stabilization splints on sleep bruxism. J Orofac Pain. 2005;19:151–8.
177. Dube C, Rompre PH, Manzini C, Guitard F, de Grandmont P, Lavigne GJ. Quantitative polygraphic controlled study on efficacy and safety of oral splint devices in tooth-grinding subjects. J Dent Res. 2004;83:398–403.
178. Macedo CR, Silva AB, Machado MA, Saconato H, Prado GF. Occlusal splints for treating sleep bruxism (tooth grinding). Cochrane Database Syst Rev. 2007;(4):CD005514.
179. Daif ET. Correlation of splint therapy outcome with the electromyography of masticatory muscles in temporomandibular disorder with myofascial pain. Acta Odontol Scand. 2012;70:72–7.
180. Stapelmann H, Turp JC. The NTI-tss device for the therapy of bruxism, temporomandibular disorders, and headache - where do we stand? A qualitative systematic review of the literature. BMC Oral Health. 2008;8:22.
181. Jokstad A, Mo A, Krogstad BS. Clinical comparison between two different splint designs for temporomandibular disorder therapy. Acta Odontol Scand. 2005;63:218–26.
182. Baad-Hansen L, Jadidi F, Castrillon E, Thomsen PB, Svensson P. Effect of a nociceptive trigeminal inhibitory splint on electromyographic activity in jaw closing muscles during sleep. J Oral Rehabil. 2007;34:105–11.
183. Jokstad A. The NTI-tss device may be used successfully in the management of bruxism and TMD. Evid Based Dent. 2009;10:23.
184. de Tommaso M, Shevel E, Pecoraro C, Sardaro M, Divenere D, Di Fruscolo O, et al. Intra-oral orthosis vs amitriptyline in chronic tension-type headache:

a clinical and laser evoked potentials study. Head Face Med. 2006;2:15.
185. Scrivani SJ, Keith DA, Kaban LB. Temporomandibular disorders. N Engl J Med. 2008;359:2693–705.
186. Landry-Schonbeck A, de Grandmont P, Rompre PH, Lavigne GJ. Effect of an adjustable mandibular advancement appliance on sleep bruxism: a crossover sleep laboratory study. Int J Prosthodont. 2009;22:251–9.
187. Saletu A, Parapatics S, Anderer P, Matejka M, Saletu B. Controlled clinical, polysomnographic and psychometric studies on differences between sleep bruxers and controls and acute effects of clonazepam as compared with placebo. Eur Arch Psychiatry Clin Neurosci. 2010;260:163–74.
188. Ranjan SCP, Prabhu S. Antidepressant-induced bruxism: need for buspirone? Int J Neuropsychopharmacol. 2006;9:485–7.
189. Carra MC, Macaluso GM, Rompre PH, Huynh N, Parrino L, Terzano MG, et al. Clonidine has a paradoxical effect on cyclic arousal and sleep bruxism during NREM sleep. Sleep. 2010;33:1711–6.
190. Madani AS, Abdollahian E, Khiavi HA, Radvar M, Foroughipour M, Asadpour H, et al. The efficacy of gabapentin versus stabilization splint in management of sleep bruxism. J Prosthodont. 2013;22:126–31.
191. Lee SJ, McCall Jr WD, Kim YK, Chung SC, Chung JW. Effect of botulinum toxin injection on nocturnal bruxism: a randomized controlled trial. Am J Phys Med Rehabil. 2010;89:16–23.
192. Shim YJ, Lee MK, Kato T, Park HU, Heo K, Kim ST. Effects of botulinum toxin on jaw motor events during sleep in sleep bruxism patients: a polysomnographic evaluation. J Clin Sleep Med. 2014;10:291–8.

Orthodontics and TMD

6

Sanjivan Kandasamy and Donald J. Rinchuse

6.1 Introduction

In 1987, a landmark court case entitled Brimm versus Malloy [1] in the USA prompted an in-depth examination on the issue of whether or not orthodontic treatment causes temporomandibular disorders (TMDs). The Brimm case resulted in a million-dollar judgment against a Michigan orthodontist for allegedly causing TMD in a 16-year-old girl. The orthodontic treatment involved the extraction of two maxillary first premolar teeth and the use of a headgear to address the patient's Class II Division I malocclusion. The TMD symptoms experienced by the plaintiff were temporomandibular joint pain and headaches following the removal of the appliances. The argument regarding the cause of these TMD symptoms was that the orthodontic treatment carried out resulted in the over-retraction of the upper incisors, leading to the distal displacement of the mandible, and thereby causing temporomandibular joint (TMJ) internal derangements. Regardless of the lack of scientific evidence behind such an argument, the jury awarded the plaintiff US$850,000 at the initial court trial.

The dental profession in the USA acknowledged the importance of TMD in 1982 with the first TMD Conference held by the American Dental Association (held in June 1982 and results published in January 1983). However, it was not until this famous TMD court case in 1987 that the orthodontic community was prompted to investigate the subjects of occlusion, condyle position, and orthodontics related to TMD; clearly, this much needed research had to be carried out from an evidence-based perspective. The numerous studies that followed the Brimm case have provided invaluable information in regard to the understanding of relationships between occlusion, condyle position, orthodontics, and TMD.

The purpose of this chapter is to examine the key issues related to occlusion and malocclusion, condyle position, and orthodontics as they might relate to TMD. Functional occlusion, internal derangements, imaging, and the role of articulators in orthodontics are also discussed.

6.2 Orthodontics and TMD: An Evolution of Controversy

The modern history of TMD essentially starts in 1934. An otolaryngologist, Dr James Costen, described a syndrome (Costen's syndrome)

S. Kandasamy, BDSc, DClinDent, MOrthRCS, FRACDS (✉)
Department of Orthodontics, School of Dentistry, University of Western Australia, Nedlands, WA, Australia

Centre for Advanced Dental Education, Saint Louis University, Saint Louis, MO, USA

Private Practice, Midland, WA, Australia
e-mail: sanj@kandasamy.com.au

D.J. Rinchuse, DMD, MS, MDS, PhD
Private Practice, Greensburg, PA, USA

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_6

related to the TMJs and ears based on the analysis of 11 cases [3, 4]. The etiology was believed to be overclosure of the mandible due to loss of dental vertical dimension subsequent to tooth loss. Symptoms of Costen's syndrome included TMJ sounds, pain in and around the jaw, limited mandibular opening, and myofascial tenderness/pain, as well as ear symptoms such as dizziness, tinnitus, pain, and impaired hearing. The close anatomical proximity of the TMJ to the external auditory meatus and related structures was believed to contribute to the ear symptoms. Almost a decade later, the famous anatomist Dr. Harry Sicher proved that Costen's syndrome was fallacious from an anatomical viewpoint. [5, 6] Even though Costen's etiologic proposals were disproved, they formed an initial baseline framework for a variety of dentally based theories of TMD etiology. These included trapped mandibles, reduced vertical dimension, condylar malposition, occlusal interferences or disharmonies, and malalignments of the mandible with the skull. All of these initiated great interest, awareness, and involvement of dentists to begin assessing and treating these problems. Dentists were also stimulated to look more closely at occlusion as the major causative factor in TMDs.

During this same time period, Dr. Alan Brodie, Chair of the Orthodontic Department at the University of Illinois (and student of Dr. Edward H. Angle), wrote about the differential diagnosis of TM joint conditions in orthodontics [7, 8]. In the 1940s, 1950s, and 1960s, several prominent orthodontists such as Thompson [9–11], Moyers [12], Ricketts [13], and Perry [14, 15] petitioned orthodontists to consider mandibular kinematics, occlusion, and the TMJ as important elements in their practices. It was not until the early 1970s that the "gnathologic-prosthodontic" view made its way into orthodontics, led by Dr. Ronald H. Roth [16–20]. Roth rationalized that orthodontics was analogous to prosthodontics/restorative dentistry such as full-mouth rehabilitation, with the difference being that the orthodontists did not "cut away" at teeth. Consistent with the traditional gnathologic-prosthodontic view, Roth believed that disharmonies of the occlusion and improper condyle position would cause temporomandibular joint (TMJ) disorders [16–20]. Therefore, in addition to attaining an optimal static occlusion as outlined by Angle [21, 22] and Andrews [23], orthodontists were obliged to attain gnathologically optimal functional occlusal and condyle relationships. The gnathologic goals were (are) as follows:

- Attain a canine-protected (mutually protected) occlusion
- Attain coincidence of a patient's centric occlusion (maximum intercuspation) with a then posterior-superior (presently an anterior-superior) centric relation condyle position
- Analyze the discrepancy between a patient's occlusion and centric relation position after obtaining a particular centric bite registration (Power-Bite) followed by the articulator mounting of the patient's dental casts

When these gnathologic objectives were not achieved with orthodontic treatment, it was believed that patients would be predisposed to TMD. An extension of this thinking was that the orthodontist could mitigate or cure TMD by correcting an existing malocclusion as well as the associated functional disharmony and improper CR position [16–20]. It was also contended that orthodontic treatment would cause TMD when orthodontists ignored the functional occlusion/CR goals of the gnathologic orthodontists [16–20].

The gnathologic/orthodontic view of the past, however, was not evidence based, and the scientific evidence accumulated over the years has contradicted much, if not all, of it. Intra-oral telemetry studies [24–27], as well as a large amount of subsequent scientific data, have supported the current concepts that occlusion and/or condyle position (CR) are not the primary cause of TMD [2, 28–35]. Of importance, the modern evidence-based view does not argue that occlusion and condyle position have no relevance to the considerations of TMD, but they at most play a secondary role. The gross evaluation of a patient's occlusion is important in the diagnosis and treatment of TMD – "…assessment of the occlusion is necessary as part of the initial oral examination to identify and eliminate gross

occlusal discrepancies…" [34]. *Gross* occlusal interferences causing or contributing to tooth mobility, fremitus, and deviations or deflections on mandibular closure and movement, should be evaluated for possible treatment.

6.3 What Are TMDs? (See Chap. 2)

TMDs comprise a group of musculoskeletal and neuromuscular conditions that involve the TMJs, the masticatory muscles, and associated tissues. TMDs are essentially divided into joint/ disk disorders and masticatory muscle disorders. The masticatory muscle disorders include muscle pain, inflammation, contracture, hypertrophy, neoplasms, and movement disorders. TM joint disorders include joint pain, inflammation, degeneration, neoplasms, disc displacements, hypomobility, hypermobility, congenital or developmental disorders and fractures [2, 29, 32]. (see Chap. 2). Due to the limited knowledge of the etiology and natural progression of the majority of these disorders, the classification of TMDs is still a constantly evolving topic. Given the difficulties associated with defining the etiologies of TMD, contemporary TMD diagnoses and treatments are based on addressing the symptoms rather than the cause; this is an approach that requires little attention to individual etiologic factors. Similar to the treatments of other musculoskeletal disorders, management is typically palliative and symptomatic, primarily targeted at decreasing pain, decreasing loading on the muscles and joints, and facilitating the restoration of function and quality of life of patients. TMD treatments in most cases should be conservative, reversible, and based on scientific evidence [36] (see Chaps. 3 and 8).

Orthodontic treatment in general has not been found to cause TMD [37–43]. Orthodontics is generally described as TMD "neutral," in that it neither causes nor cures (or mitigates) TMD [37]. Orthodontics does not prevent the development of TMD in patients who have malocclusion [32]. Therefore, it is not evidence based for orthodontists or others in the dental profession to advise patients and parents of young children that orthodontic treatment is indicated to address a child's malocclusion to mitigate the risk of TMD developing in the future. The utilization of any specific type of orthodontic treatment, or appliance, such as headgear, elastics, chin cup, or whether extractions are performed, has not been shown to lead to any increased risk for TMD [35–53].

6.4 Occlusion, Malocclusion, and TMD

Dentists have vigorously debated the role of occlusion or malocclusion in causing TMD for many years. Numerous studies have investigated the relationship between both functional and morphologic aspects of occlusion and TMD; some have showed statistically significant associations (not cause-effect), while others have reported no such relationship. The differences in the findings can be explained by problems in the study designs of many of these investigations. The main problems have been: relating symptoms to disease states such as joint sounds with no pain, failing to establish a differential diagnosis, small and heterogeneous samples with lack of controls or poorly matched comparison or control groups, issues with sample selection bias, inter- and intra-examiner variation, failing to isolate contributing factors, or failing to neutralize confounding variables [42, 44].

Several functional and morphological occlusal relationships have been investigated, and a small number of them have been purported to cause people to develop TMD [42, 44]. Some of the occlusal factors are open bite, overbite, overjet greater than 7 mm, centric slides (greater than between 2 and 4 mm), unilateral posterior crossbites with and without lateral functional mandibular displacement, and missing posterior teeth. Current understanding and evidence-based literature fail to demonstrate a *causal* relationship between these occlusal factors and TMD signs and symptoms; thus, the relationship is only an association [42, 44, 54]. Therefore, with regard to TMDs, it is clear that occlusion today is not believed to be as important as it once was

thought to be. This is largely a reflection of the significant amount of research that has taken place in the last two decades. TMD has moved from a dental and mechanical-based model to a biopsychosocial and medical model that integrates a host of biologic, behavioral, and social factors to the onset, maintenance, and progression of TMDs. These disorders are considered to have multiple associated etiologic factors, including both extrinsic and intrinsic patient factors that may contribute to the development of symptoms. Factors such as parafunction, trauma, psychosocial disorders, emotional stress, gender, genetics, and centrally mediated mechanisms are currently considered to be most important.

6.5 Centric Relation Dilemma

Centric relation (CR) has been a topic of much debate in dentistry for more than a century. The definition as well as the concept of CR has changed over the years. For more than a half century, the definition of CR within the prosthodontic community has evolved from a retruded, posterior, and for the most part, superior condyle position to an anterior-superior condyle position [55]. Dr. Ronald Roth in the early 1970s advocated a posterior-superior (retruded CR) position, and then he changed his view in the early to mid-1980s in favor of the more current anterior-superior CR position. Those arguing for an anterior-superior CR position were motivated by findings from TMJ imaging of the era, initially TMJ arthrography followed by magnetic resonance imaging (MRI), demonstrating many TMJ internal derangements with the disc often located (or displaced) anteromedially. They also realized that the pull of the elevator masticatory muscles was in an upward and forward direction, typically seating the condyles in an anterior and superior position position.

CR has been defined in so many ways that today it has lost credibility [56]. The changes in the definition of CR appear to have been quite arbitrary rather than based on evidence, spurring Dr. Lysle Johnston to sarcastically write in 1990: "It could be argued that the progressive modifications in the definition of CR have done more to eliminate centric slides than 20 years of grudging acquiescence of the precepts of gnathology." [57] Johnston also followed with: "I know of no convincing evidence that condyles of patients with intact dentitions should be placed in CR, or that once having been placed there, the resulting improvement on nature will be stable." [57]

There is little evidence to support the gnathologic view that centric occlusion (CO) position or maximum intercuspation (MI) should be coincident with an arbitrary CR position. In addition, centric slides greater than 4 mm that have been found associated with TMJ arthropathies are most likely the result of TMD rather than the cause [42, 44]. It is still important, however, for orthodontists to check for centric slides and to take some note if they are greater than 2 mm in order to discern whether there is a marked slide between CO/MI and centric relation occlusion or CRO, commonly referred to as a "Dual Bite" or "Sunday Bite." This is especially important in determining the true extent of a dental and skeletal malocclusion in three planes for orthodontic treatment planning. Kandasamy et al. [58] recently demonstrated via an MRI study that irrespective of the centric bite registration used, including the Roth Power Bite Registration, clinicians cannot accurately and predictably position condyles into specific locations in the glenoid fossae.

Further, the evidence suggests that there is a range of acceptable condylar positions and not one position that is optimal for all individuals. There is a particular optimal position for each person, with the anterior to mid-condyle positions more commonly found than retruded (posterior) CR positions. Nonetheless, there is evidence that individuals with healthy TMJs can have a retruded condylar position [31, 37, 53, 59, 60]. Further, the condyle-fossa relationship in every person may change very slightly throughout the day depending upon various factors including fatigue of the facial and masticatory muscles, parafunction, posture, tongue pressure, hydration of the disc, and so on. There is no optimal three-dimensional position/location of the TMJ condyles in the glenoid fossa [2, 32, 34].

Various orthodontic treatments such those involving extractions, headgear, inter-arch elastics, chin cups, and so forth do not necessarily cause the posterior displacement (or positioning) of the mandibular condyles nor do they necessarily predispose patients to developing TMD [35–53].

6.6 Functional Occlusion and Orthodontics

Criteria for an optimal "static occlusion" have found universal support based primarily on the work of Angle [21, 22] and Andrews [23]. It is important to be cognizant of the fact that malocclusion is not a disease and there is no persuasive evidence to demonstrate that deviations from Angle's normal/class I relationship will predispose patients to TMD or periodontal disease [61]. Nonetheless, there is less of a consensus as to what constitutes the optimal "functional occlusion"; this refers to the contact relationship of the upper and lower teeth within the functional range of mandibular movement. This subject has been debated for over a century and views are based more so on conjecture rather than evidence. Clark and Evans stated: "The criteria that denote an 'ideal' functional occlusion have not been conclusively established." [62]

There is a long-standing belief that the optimal functional occlusion for all dental patients, including orthodontic patients, is "canine-protected occlusion" (CPO or mutually protected occlusion). Canine-protected occlusion refers to contact occurring only at the canine teeth on the working side with no occlusal contact(s) on the non-working (balancing) side, during lateral or side-to-side mandibular movements. That is, the canine teeth disclude the entire dentition on laterotrusive movements out of centric occlusion. The contention by some is that failure to establish CPO during orthodontic treatment could predispose patients to TMD, as well as orthodontic relapse [16–18, 20, 63]. When discussing functional occlusion, one must qualify the difference between the terms balancing side contact and balancing side interference. A balancing side contact is a very light occlusal contact and this is considered benign. On the other hand, a balancing side interference is a gross occlusal disharmony that can cause deflection of the mandible, tooth mobility, fremitus, and so forth. Balancing side contacts are acceptable for a physiologic functional occlusion, irrespective of what tooth contact occurs on the working side. A balancing side interference is not acceptable and deviates from a healthy functional occlusion.

To regard CPO as the optimal functional occlusion for orthodontic patients is arbitrary and not supported by the evidence [64]. The routine selection and attainment of CPO as the optimal functional occlusion type for all patients ignores the importance of each person's unique stomatognathic and neuromuscular functional status. CPO might be one of several functional occlusion schemes that are acceptable for orthodontic patients. Further, not all subjects actually function in the extreme lateral side-to-side border movements governed by the functional occlusion paradigm of CPO. Studies have also shown that the functional occlusion which exists as the mandible moves immediately laterally out of centric occlusion is not typically CPO, and individuals move in and out of one functional occlusion type to another as the mandible moves from centric occlusion to the extreme cusp to cusp lateral border movement.

No one single type of functional occlusion predominates in nature. As Woda and coworkers found back in the mid-1970s, "Pure canine protected or pure group function rarely exists and balancing contacts seem to be the general rule in the population of contemporary civilizations." [65] Even if a particular functional occlusion is achieved, it will not necessarily be stable or retained over the patient's life time [41, 62]. If CPO is established, one must take in to consideration that over time the functional occlusion will typically evolve into group function followed by balanced occlusion as a result of tooth attrition, changes in the oral environment, demands on the dentition with growth and aging, and occlusal settling, all affecting the vertical level and position of the canines.

CPO is difficult to achieve in an orthodontically treated population because there is less canine rise and disclusion when the canines are in

an Angle's Class I/normal relationship; the canines never come together in contact in a cusp tip to cusp tip relationship in lateral border movements like they do in a Class II relationship [66]. In a Class I canine relationship, rather than cusp *tip* to cusp *tip* laterotrusive functional relationship typical of functioning Class II canines, the canines disclude in a cusp *incline plane* to cusp *incline plane* functional relationship. This provides less canine rise and therefore more chance of having balancing side contacts/interferences. So to achieve CPO, an orthodontist would have to either establish a canine relationship that is between a Class I and a full unit Class II relationship or deliberately extrude patients' canines past their physiologic and normal contact point/area and/or restoratively build up the canine tips to achieve CPO. This not only tends to produce a non-consonant smile arc but also produces an unesthetic "vampire look." Achieving canine-protected occlusion routinely in orthodontics is not evidence based and is generally mutually exclusive to achieving a consonant smile arc and ideal smile esthetics [64].

In summary, none of the traditional functional occlusion schemes are inherently bad, but it does seem that group function occlusion and balanced occlusion (with no interferences and only balancing side contacts/light) appear to be the most practical for orthodontic patients over a lifetime. It is not evidence based to simply advocate one type of functional occlusal scheme over another for all patients. A patient's static occlusion type, craniofacial morphology, parafunctional habits, chewing kinematics (vertical versus horizontal chewing patterns), and current oral health status are only some of the myriad factors that contribute to establishing the best and most practical functional occlusion scheme for each individual patient.

6.7 Articulators for Orthodontic Diagnosis

Articulators are mechanical devices which aim to crudely simulate mandibular movements and occlusal relationships. There are a number of different types of articulators such as arcon, nonarcon, fully adjustable, semi-adjustable, polycentric hinge, and so forth. Articulators are useful for involved prosthodontic treatments and orthognathic surgical procedures to at least maintain a certain vertical dimension, while laboratory procedures are being performed. Early on, the need to mount cases was related mostly to detecting "sagittal" discrepancies, particularly "Sunday bites." Later on it became more about finding transverse and vertical discrepancies. Nonetheless, the utility of articulators in orthodontics to improve patient diagnoses has been the subject of much debate ever since the early 1970s, when Dr. Ronald Roth introduced the classic gnathological-prosthodontic philosophy to the orthodontic profession [16–20]. Roth believed that mounting dental study casts on articulators would aid the orthodontist in diagnosing three-dimensional condylar (CR) discrepancies. He also believed that he could position and seat condyles in a more ideal position within the glenoid fossa, and then he could base his orthodontic treatments around this condylar position (i.e., CR) with the aim of curing a pre-existing TMD or mitigating the risk of TMD developing in the future.

It is has been argued that articulator mountings with the appropriate centric bite registration will improve the orthodontic diagnostics (Angle's classification) in 18.7–40.9 % of cases [67, 68]. Whether all cases need to be mounted is the subject of much debate. Some gnathologic orthodontists take the position that not all cases need to be mounted, so they only mount in cases needing orthognathic surgery or in TMD patients. Others have stated that they like to mount models in cases involving most adults, or those with multiple missing teeth, functional shifts and/or midline deviations, and those with deviations on opening or closing. Nonetheless, Dr. Frank Cordray, a contemporary Roth supporter, believes all cases need to be mounted because a practitioner would not be able to determine beforehand which cases will become the most challenging [63]. Ellis and Benson found that mounting of study casts in CR instead of CO did not make any difference in the eventual diagnosis and treatment planning decisions [69].

As mentioned previously, in the past, the views on the causes and treatments for TMD

were centered on a mechanical dental-based model and this involved a detailed analysis of occlusion and condyle position (CR). As mentioned previously, the contemporary TMD model has moved away from focusing on these issues and has embraced a medical and biopsychosocial model. So the question is, do we need to even debate the issue of the utility of articulators in orthodontics in general, or in relation to TMDs, when there is only a minimal influence of occlusion and condyle position on the development of TMDs? In our view, the answer is a simple no. Regardless, we will still discuss the key issues in relation to articulators in orthodontics below, as there still exists a vocal and substantial proportion of the dental profession that still propagates this philosophy as the standard of care.

Let us assume that seating the condyle in the prescribed "ideal" position within the fossa and then rehabilitating the occlusion or malocclusion to this position with either orthodontics, prosthodontics and/or orthognathic surgery is critical to preventing or curing TMD. If this is the case, then in order to be able to mount a set of models properly on an articulator, one has to be able to obtain an accurate bite registration that seats the condyle in the so-called ideal CR position within the fossa. The critical issue here is whether these bite registrations are reliable and more importantly valid. Orthodontic gnathological records such as the Roth power centric bite registration and the articulator mounting instrumentation appear to be reliable (repeatability and consistency of the records/techniques) in controlled laboratory conditions [63, 70, 71]. However, are these centric bite registrations valid? That is, do any of these recording methods, including the Roth power bite registrations, actually anatomically seat the condyles in an anterior-superior position within the fossa according to imaging findings? The evidence-based data supports the view that clinicians are not able to estimate the position and location of patients' condyles via certain bite registrations taken chairside. Kandasamy et al [58] have provided MRI data demonstrating that patient condyles are not located where gnathologists believe they would be. Based on their study, they concluded that given the small changes and the extremely unpredictable nature of condylar positioning associated with centric relation and Roth power bite registrations, advocating this modality routinely in clinical practice as a prophylactic or curative measure for TMD is an invalid and unjustified procedure [58].

There are several other problems with gnathologic bite registrations and articulators:

- The basic premise of the use of articulators dates back more than a half century ago to Posselt and the "terminal hinge axis" [74]. The concept of the terminal hinge axis argues that in the initial phase of opening of the mandible (first 20 mm) there is only rotation and not translation. If this is true than articulators need not have the ability to calculate the possible effects of both rotation and translation and the different degrees of each due to individual variability. However, in 1995, Lindauer et al [75] demonstrated that both rotation and translation of the condyles occur even the first few millimeters of opening and closing, proving that there is no "terminal hinge axis" and that the basic premise of articulators is invalid. Further, the terminal hinge axis is difficult to achieve given that condyles are generally irregular, individually suspended and asymmetrically angulated to the midsagittal plane [76]. It is interesting to note that when Posselt posed his theory on the terminal hinge axis, CR was regarded as a posterior-superior retruded condyle position and CR was recorded by applying significant distal chin pressure on the mandible resulting in the obvious reason for observing a terminal hinge axis-type movement.
- In children, the TMJ condyle-glenoid fossa complex changes location with growth; the fossae are displaced posteriorly and inferiorly and the condyles grow posteriorly and superiorly. To maintain any particular CR position, gnathologists would need to take new CR bite registrations, facebow transfers, and mountings periodically during treatment to evaluate TMJ growth changes during orthodontic treatment [77].
- There are errors associated with the taking of bite registrations including the deformation

and shrinking of the bite registration material and errors with transferring the registration onto the articulator. There also exist errors associated with the mounting process, including orientation of the casts, the taking and transferring of the facebow record, flexing of the bite fork when mounting the maxillary cast, plaster expansion and contraction as well as using average settings on the articulator.

- For the Roth Power Bite registration, the bite is recorded at 2 mm separation of the most posterior teeth [72] and not in occlusion. What determines the final dental relationship as set on the articulator when the pin of the articulator is dropped to allow full occlusal interdigitation? When bite registrations are taken with the vertical dimension open, this will result in errors in occlusal interdigitation following the removal of the bite registration, because the centers of rotation of the patient's TMJs and the articulator are different [73].
- CR bite registrations are static recordings which do not record any dynamics of mandibular movement.
- Articulators do not furnish any biologic information about human TMJs. Articulators cannot simulate the real life dynamics of occlusion or evaluate chewing kinematics and masticatory movements that are dependent upon muscle patterns and the resilience of the soft tissues and the joint disc.
- There also exists the issue of added costs, both financial and time, in performing articulator mountings as well as the issue of storing these records, even if they are temporary.

To eliminate or reduce the errors and limitations associated with articulators, there is a current trend toward using three-dimensional computer-aided treatment planning incorporating data from multi-slice computed tomographic (CT) or cone-beam CT (CBCT) scans, especially for orthognathic surgical procedures [78–82]. This modality allows the clinicians to visualize the craniofacial anatomy in all three dimensions and to simulate surgical procedures. Clinicians have the ability to carry out accurate jaw positioning with reduced errors associated with vertical dimensional and condylar rotational changes, as well as less errors with inter-occlusal splint fabrication and seating in comparison to the traditional two dimensional work-up using articulators and plain films. The virtual planning is also significantly less labor intensive and affords greater efficiency and accuracy than traditional methods.

Given the countless number of limitations and errors associated with the bite registration and mounting process, articulators, the lack of validity of centric relation bite registrations, the lack of emphasis of condyle position and occlusion in the role of TMD and the evolution of the medical and biopsychosocial model for TMD diagnosis and management, the whole discussion about the routine use of articulators orthodontics becomes an essentially futile exercise. Given the evidence-based literature available today, there is no justification for the routine mounting and condylar positioning in orthodontics.

6.8 TMJ Sounds, Internal Derangements, and Orthodontics

In the past, TMJ sounds have been described as being both a symptom and a sign of TMD that relates to some form of internal derangement of the articular disk inside the TMJ. It was believed at one time that this condition will inevitably progress to limited opening (locking) or degenerative joint disease (arthritis), but this viewpoint has changed as more information has emerged. It is now widely known and appreciated that TMJ sounds are not always diagnostic of pathology/dysfunction. In fact, there is a type of "soft" sound described by Watt [83] that is subtle and heard in normal joints, and which has a number of possible causes. These include phenomena such as sudden movement of the TMJ ligaments, the separation of the articular surfaces, the sucking of loose connective tissue behind the condyle as it moves forward on the articular surface, surface irregularities on the condyle or eminence, or an alteration in mandibular position related to

hypertonicity of one of the lateral pterygoid muscles.

Most often TMJ sounds such as popping and clicking that are present without any other TMJ symptoms such as pain, muscle tenderness, and limitation of mandibular movement are not indicative of a TMD, and in current taxonomies they are not classified as clinical disorders. Studies have shown that clicking alone is a benign condition that is found in over a third of the population, and which only rarely progresses to more serious clinical dysfunction or disease. Furthermore, even in the presence of other TMJ symptoms, patients with clicks do not necessarily progress to more advanced TMD [84–87].

Crepitus, or a grating/grinding sound, is different than the typical clicking or popping and usually represents an underlying osteoarthrosis or osteoarthritis of the TMJ. The finding of TMJ crepitus without pain or dysfunction does not mean that a patient needs immediate treatment, but the patient should be made aware of this finding and reviewed regularly. Treatment is generally not indicated when there is no joint pain or dysfunction (also see Chap. 3).

6.9 Internal Derangements, Recapturing Discs, and Orthodontics

With better imaging of the TMJ disc that was done initially with arthrography followed by magnetic resonance imaging in the early to mid-1980s, it was found that a significant number of TMD patients had TMJ internal derangements. A similar finding was observed in the TM joints of over a third of the normal population. Most often these internal derangements are anterior and medially displaced TMJ discs. This information prompted the dental profession to question the definition of centric relation at that time and contributed to the change in the definition of CR from a posterior-superior condyle position to an anterior-superior condyle position. This further led to the practice (which is still prevalent today) of attempting to "recapture" anterior displaced discs with so-called repositioning splints [88–90]. After the TMJ discs were believed to be recaptured, the mandible (and discs) would typically need to be stabilized at the occlusal level with the help of orthodontics, prosthodontics, or orthognathic surgery.

It has been reported that TMJ disc abnormalities are also associated with a reduction in the forward growth of the maxilla and mandible; and for adolescents, a reduction in the growth of the mandibular ramus [91–93]. Based on this information, it is conjectured that untreated or inadequately treated TMJ internal derangements (IDs) can lead to degenerative joint disease (DJD), pain, compromised mandibular growth, and several other adverse conditions [94, 95]. It is believed that these asymptomatic ID patients need treatment, typically involving an occlusal stabilizing splint followed by orthodontic treatment. It is also argued that the best time to treat these individuals is early and when they are young [94], because this would be a time before significant changes of the disc, skeleton, and occlusion occurs, when there is optimal capacity for repair and adaptation. Treatment rendered at a later time, after growth, is believed to be less effective since the TMJs would have progressed to having a nonreducing disc displacement and degenerative joint disease [94, 95].

However, even if we acknowledge that there are some biologic effects of TMJ internal derangements on the growth and development of patients, there are serious questions to consider regarding what needs to be done about that (if anything). First, are these individuals at a higher risk of developing TMD in the future? Secondly, if these individuals are at a higher risk, do they need treatment at this time to mitigate the future development of TMD? Thirdly, is treatment carried out to "recapture" displaced discs evidence based and has it been clinically successful?

The opposing view is to leave this cohort of individuals alone and let sleeping dogs lie [96, 97]. As pointed out earlier, it has been shown that more than 30 % of the population who are asymptomatic exhibit discernable MRI disc displace-

ments, and many of them have palpable clicking sounds [98–101]. There is no evidence that this group would be more prone to develop TMD in the future than a group who do not have TMJ discs located in an "abnormal" position. Further, there is no evidence that treating patients with "abnormal" TMJ disc locations without TMD symptoms would either prevent or mitigate future TMD. It has also been shown that patients with moderate to severe TMD with associated disc displacement without reduction will improve with minimal, or no, treatment [87, 102].

Originally it was believed that anteriorly displaced discs can be recaptured with anterior repositioning appliances, thereby resulting in the "walking back" of the disc-condyle relationship to the normal physiological position. What actually occurs is that by positioning the mandible in a forward position, the retrodiscal tissues are allowed to recover, allowing adaptive and reparative changes to occur. The retrodiscal tissues become avascular and fibrotic, allowing the condyles to eventually function "off the disc" as they articulate on the newly adapted retrodiscal tissues with no pain [103–106]. The validity of use of repositioning splints is speculative and not supported by the evidence and may lead to irreversible changes in the occlusion.

Furthermore, there is no clear linear relationship between disc displacement and pain, mandibular dysfunction, osteoarthritis, and/or growth disturbances. Not all patients with growth deficiencies have disc displacements, and not all growing patients with disc displacement grow abnormally. Trying to administer treatment for an internal derangement involving splint therapy followed by orthodontics, prosthodontics, and surgery, especially when there is no pain or dysfunction present, is not evidence-based care and a gross disservice to the patients.

6.10 TMJ Imaging for Orthodontics

The standard radiographs usually taken as part of a routine orthodontic examination include a panoramic and a lateral cephalometric radiograph. With regard to the TMJs, these two dimensional plain-film or digital radiographs can be used as a screening tool to only crudely assess basic "bony" elements such as mandibular and condylar asymmetries, developmental skeletal anomalies, and fractures. Today, there are more sophisticated imaging techniques available, such as MRIs, computed tomography (CT), and cone beam CT (CBCT) scans. MRI is a non-invasive soft tissue imaging technique capable of providing excellent high contrast ratios and diagnostic information of the soft tissues of the TMJs and disc-condyle relationship. CT and CBCT are excellent tools for the three-dimensional imaging of hard tissues. There is a growing trend for using CBCTs instead of conventional radiographs for many dental applications. These include pre-surgical dental implant planning, airway assessment, and orthognathic surgery planning, as well as the orthodontic assessment of skeletal structures and the localization of impacted, malformed, and supernumerary teeth. With regard to the TMJs, CBCTs provide excellent osseous detail of the TMJs that is impossible to achieve with conventional radiographs.

Given the contemporary medical and biopsychosocial model for TMD, routine imaging of the TMJs for the assessment of condyle position and the diagnosis of TMD is not evidence based or justified. Imaging should only be performed following a thorough clinical examination that indicates the need for more diagnostic information, (see Chap. 3). The British Orthodontic Society Guidelines argue against the use of conventional radiographs of the TMJs for the diagnosis of TMDs in orthodontic patients [107]. A recent systematic review found that there was no high-quality evidence found for the benefits of routine CBCT use in orthodontics [108]. In addition, the increased risk of radiation exposure from CBCTs as opposed to two-dimensional conventional digital imaging greatly outweighs their justification to be used routinely in orthodontics [108, 109]. With significant improvements in CBCT technology today, especially in terms of reducing the radiation exposure to the point where it is below conventional radiographs, their use will become more common and justified [110].

6.11 TMD Informed Consent in Orthodontics

Informed consent for orthodontic treatment is a necessary requirement prior to the commencement of treatment. Part and parcel of obtaining informed consent for orthodontic treatment is obtaining informed consent from the patient (parent) in relation to orthodontics and TMD. Various orthodontic organizations such as the American Association of Orthodontics have sections in their published informed consent document(s) on the current and evidence-based view on the topic of orthodontics and TMD. One point that must be stressed here is that informed consent is not a one-time event; patients/parents must be constantly reminded about the salient elements of risk factors that are applicable for each individual patient/family. This topic is dealt with in greater detail in Chap. 10. The key issues that are important to discuss with the patient and accompanying parent/guardian are summarized in the Take-Home Messages section of this chapter.

Conclusion

The term TMD encompasses a number of clinical problems of multifactorial etiology that involve the masticatory musculature and the TMJs. The historic mechanical and dental-based model has been gradually replaced by a medical model used in the treatments of TMD and other acute and chronic musculoskeletal disorders. The contemporary biopsychosocial approach to TMD management focuses on the integration of biologic, clinical, and behavioral factors that may ultimately account for the onset, maintenance, and remission of TMD. Genetics (vulnerabilities related to pain), endocrinology, behavioral risk-conferring factors, and psychosocial traits and states appear to be the variables currently being researched and receiving the most attention. Despite the compelling current evidence, some professionals in orthodontics are reluctant to change and continue to still hold onto past unscientific beliefs that lead to the use of outdated treatment approaches. It is critical that orthodontists continually pay attention to the new research developments so that they can ultimately provide their patients with the best and most appropriate possible care.

Take Home Messages

- TMDs are a group of musculoskeletal and neuromuscular conditions that involve the TMJs, the masticatory muscles, and associated tissues.
- Current understanding and evidence-based literature fail to demonstrate a relationship between various occlusal factors and TMD signs and symptoms.
- TMD has moved from a dental and mechanical-based model to a biopsychosocial and medical model that integrates a host of biologic, behavioral, and social factors to the onset, maintenance, and progression of TMD.
- Management of TMD is typically palliative and symptomatic, aimed at decreasing pain, decreasing loading on the muscles and joints, and facilitating the restoration of function and quality of life of patients.
- Orthodontics is generally described as TMD "neutral," in that it neither causes nor cures (or mitigates) TMD.
- There is no evidence that early orthodontic treatment of patients with malocclusions will prevent the development of TMD in the future.
- The evidence suggests that there is a range of acceptable condylar positions and not one CR position that is optimal for all individuals.
- Irrespective of the centric bite registration used, clinicians cannot accurately and predictably position condyles into specific locations in the glenoid fossae.
- The practice of condylar positioning associated with centric relation and Roth power bite registrations in clinical practice as a prophylactic or curative measure for TMD is an invalid and unjustified procedure.
- Given the countless number of limitations and errors associated with CR bite registrations, articulators, and the mounting process, the routine use of articulators in orthodontics is not justified.

- Advocating and establishing canine-protected occlusion as the optimal functional occlusion for orthodontic patients is arbitrary and not supported by the evidence.
- Group function occlusion and balanced occlusion (with no interferences and only light balancing side contacts) appear to be the most practical outcomes for orthodontic patients over a lifetime.
- Most often TMJ sounds (e.g., popping, clicking) that are present without any other TMJ symptoms such as pain, muscle tenderness, and limitation of mandibular movement are not indicative of a TMD.
- There is no evidence that asymptomatic individuals with disc displacements will be more prone to develop TMD in the future than those who do not have TMJ discs located in an "abnormal" position.
- There is an unclear relationship between disc displacement and pain, mandibular dysfunction, osteoarthritis, and growth disturbances.
- Not all patients with growth deficiencies have disc displacements, and not all growing patients with disc displacement grow abnormally.
- Anteriorly displaced discs cannot reliably be recaptured with anterior repositioning appliances.
- Routine imaging of the TMJs for the assessment of the condyle position and the diagnosis of TMD is not evidence based or justified. Imaging should only be performed following a thorough clinical examination that indicates the need for more diagnostic information.

References

1. Pollack B. Cases of note: Michigan jury awards $850,000 in ortho case: a tempest in a teapot. Am J Orthod Dentofacial Orthop. 1988;94:358–60.
2. Griffiths RH. Report of the president's conference on the examination, diagnosis and management of temporomandibular disorders. J Am Dent Assoc. 1983;106:75–7.
3. Costen JB. Syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. Ann Otol Rhinol Laryngol. 1934;43:1–15.
4. Costen JB. Some features of the mandibular articulation as it pertains to medical diagnosis, especially in otolaryngology. J Am Dent Assoc. 1937;24:1507–11.
5. Sicher H. Temporomandibular articulation in mandibular overclosure. J Am Dent Assoc. 1948;36:131–9.
6. Zimmerman AA. An evaluation of Costen's syndrome from an anatomic point of view. In: Sarnat BG, editor. The temporomandibular joint. Springfield: Charles C Thomas; 1951.
7. Brodie AG. Differential diagnosis of joint conditions in orthodontia. Angle Orthod. 1934;4:160–76.
8. Brodie AG. The temporo-mandibular joint. III. Dent J. 1939;8:2–12.
9. Thompson JR. The rest position of the mandible and its significance to dental science. J Am Dent Assoc. 1946;33:151–80.
10. Thompson JR. Anatomical and physiological considerations for positions of the mandible. Dent J Aust. 1951;23:161–6.
11. Thompson JR. Concepts regarding function of the stomatognathic system. J Am Dent Assoc. 1954;48:626–37.
12. Moyers RE. An electromyographic analysis of certain muscles in temporomandibular movement. Am J Orthod. 1950;36:481–515.
13. Ricketts RM. Laminography in the diagnosis of temporomandibular joint disorders. J Am Dent Assoc. 1953;46:620–48.
14. Perry Jr HT. Principles of occlusion applied to modern orthodontics. Dent Clin North Am. 1969;13:581–90.
15. Perry HT. Temporomandibular joint and occlusion. Angle Orthod. 1976;46:284–93.
16. Roth RH. Temporomandibular pain-dysfunction and occlusal relationships. Angle Orthod. 1973;43:136–53.
17. Roth RH. The maintenance system and occlusal dynamics. Dent Clin North Am. 1976;20:761–88.
18. Roth RH. Functional occlusion for the orthodontist. J Clin Orthod. 1981;15:32–51.
19. Roth RH. Treatment mechanics for the straight-wire appliance. In: Graber TM, Swain BK, editors. Orthodontics: current principles and techniques. St. Louis: Mosby; 1985. p. 655–716.
20. Roth RH. Functional occlusion for the orthodontist. Part III. J Clin Orthod. 1981;15:174–9.
21. Angle EH. Classification of malocclusion. Dent Cosmos. 1899;41:246–64.
22. Angle EH. Malocclusion of teeth. 7th ed. Philadelphia: SS White Mtg Co.; 1907.
23. Andrews LF. The six keys to normal occlusion. Am J Orthod. 1972;62:296–309.
24. Graf H, Zander HA. Functional tooth contacts in lateral and centric occlusion. J Prosthet Dent. 1963;13:1055–66.

25. Pameijer JH, Glickman I, Roeber FW. Intraoral occlusal telemetry. 3. Tooth contacts in chewing, swallowing, and bruxism. J Periodontol. 1969;40:253–8.
26. Pameijer JH, Brion M, Glickman I, Roeber FW. Intraoral occlusal telemetry, V. Effect of occlusal adjustment upon tooth contacts during chewing and swallowing. J Prosthet Dent. 1970;24:492–7.
27. Glickman I, Martigoni M. Haddad A, Roeber FW. Further observation on human occlusion monitored by intraoral telemetry (Abstract #612), IADR. 1970:201.
28. Gesch D, Bernhardt O, Kirbshus A. Association of malocclusion and functional occlusion with temporomandibular disorders (TMD) in adults: a systematic review of population-based studies. Quintessence Int. 2004;35:211–21.
29. Mohl ND. Temporomandibular disorders: role of occlusion, TMJ imaging, and electronic devices – a diagnostic update. J Am Coll Dent. 1991;58:4–10.
30. Seligman DA, Pullinger AG. The role of intercuspal occlusal relationships in temporomandibular disorders: a review. J Craniomandib Disord. 1991;5:96–106.
31. LeResche L, Truelove EL, Dworkin SF. Temporomandibular disorders: a survey of dentists' knowledge and beliefs. J Am Dent Assoc. 1993;124: 90–105.
32. McNeill C, Mohl ND, Rugh JD, Tanaka TT. Temporomandibular disorders: diagnosis, management, education, and research. J Am Dent Assoc. 1990;120:253–60.
33. Rinchuse DJ, McMinn J. Summary of evidence-based systematic reviews of temporomandibular disorders. Am J Orthod Dentofacial Orthop. 2006;130:715–20.
34. National Institutes of Health Technology Assessment Conference Statement. Management of temporomandibular disorders. J Am Dent Assoc. 1996;127: 1595–606.
35. DeKanter RJ, Truin GJ, Burgersdijk RC, Van't Hop MA, Battistuzzi PG, Kalsbeek H, et al. Prevalence in the Dutch adult population and a meta-analysis of signs and symptoms of temporomandibular disorders. J Dent Res. 1993;72:1509–18.
36. American Academy of Orofacial Pain. Diagnosis and management of TMDs. In: De Leeuw R, Klasser GD, editors. Orofacial pain: guidelines for assessment, diagnosis, and management. 5th ed. Chicago: Quintessence; 2013. p. 130.
37. Gianelly AA. Orthodontics, condylar position, and TMJ status. Am J Orthod Dentofacial Orthop. 1989;95:521–3.
38. Reynders RM. Orthodontics and temporomandibular disorders: a review of the literature (1066–1988). Am J Orthod Dentofacial Orthop. 1990;97:463–7.
39. Kim MR, Graber TM, Viana MA. Orthodontics and temporomandibular disorders: a meta-analysis. Am J Orthod Dentofacial Orthop. 2002;122:438–46.
40. Luther F. Orthodontics and the temporomandibular joint. Where are we now? Part 1. Orthodontics and temporomandibular disorders. Angle Orthod. 1998; 68:295–304.
41. Luther F. Orthodontics and the temporomandibular joint. Where are we now? Part 2. Functional occlusion, malocclusion, and TMD. Angle Orthod. 1998;68:305–18.
42. McNamara JA, Seligman DA, Okeson JA. Occlusion, orthodontic treatment, temporomandibular disorders: a review. J Orofac Pain. 1995;9:73–90.
43. Macfarlane TV, Kenealy P, Kingdon HA, Mohlin BO, Pilley JR, Richmond S, Shaw WC. Twenty-year cohort study of health gain from orthodontic treatment: temporomandibular disorders. Am J Orthod Dentofacial Orthop. 2009;135:692.e1–8.
44. Michelotti A, Iodice G. The role of orthodontics in temporomandibular disorders. J Oral Rehabil. 2010; 37:411–29.
45. Gianelly AA, Hughes HM, Wohlgemuth P, Gildea C. Condylar position and extraction treatment. Am J Orthod Dentofacial Orthop. 1988;96:428–32.
46. Gianelly AA, Cozzani M, Boffa J. Condylar position and maxillary first premolar extraction. Am J Orthod Dentofacial Orthop. 1991;99:473–6.
47. Mohlin BO, Derweduwen K, Pilley R, Kingdon A, Shaw WD, Kenealy P. Malocclusion and temporomandibular disorder: a comparison of adolescents with moderate to severe dysfunction with those without signs and symptoms of temporomandibular disorders and their further development to 30 years of age. Angle Orthod. 2004;74:319–27.
48. Deguchi T, Uematsu S, Kawahara Y, Mimura H. Clinical evaluation of temporomandibular joint disorders (TMD) in patients treated with chin-cup. Angle Orthod. 1998;68:91–4.
49. Dibbets JMH, van der Weele LT. Orthodontics treatment in relation to symptoms attributed to dysfunction of the temporomandibular joint – a 10 year report of the University of Groningen study. Am J Orthod Dentofacial Orthop. 1987;91:193–9.
50. Dibbets JMH, van der Weele LT. Extraction, orthodontic treatment, and craniomandibular dysfunction. Am J Orthod Dentofacial Orthop. 1991;99:210–9.
51. Carlton KL, Nanda RS. Prospective study of posttreatment changes in the temporomandibular joint. Am J Orthod Dentofacial Orthop. 2002;122:486–90.
52. Luppanapornlap S, Johnston Jr LE. The effects of premolar extraction: a long-term comparison of outcomes in "clear cut" nonextraction and extraction class II patients. Angle Orthod. 1993;63:257–72.
53. Beattie JR, Paquette DE, Johnston Jr LE. The functional impact of extraction and nonextraction treatments: a long term comparison in patients with "borderline", equally susceptible class II malocclusions. Am J Orthod Dentofacial Orthop. 1994;105: 444–9.
54. Farella M, Michelotti A, Iodice G, Milani S, Martina R. Unilateral posterior crossbite is not associated with TMJ clicking in young adolescents. J Dent Res. 2007;86:137–41.
55. Rinchuse DJ, Kandasamy S. Centric relation: a historical and contemporary orthodontic perspective. J Am Dent Assoc. 2006;137:494–501.

56. Gilboe DB. Centric relation as the treatment position. J Prosthet Dent. 1983;50(5):685–9.
57. Johnston Jr LE. Fear and loathing in orthodontics: notes on the death of theory. In: Carlson DS, Ferara AM, editors. Craniofacial growth theory and orthodontic treatment. Ann Arbor: Center for Human Growth and Development, University of Michigan; 1990. p. 75–91.
58. Kandasamy S, Boeddinghaus R, Kruger E. Condylar position assessed by magnetic resonance imaging after various bite position registrations. Am J Orthod Dentofacial Orthop. 2013;144:512–7.
59. Kircos LT, Ortendahl DA, Arakawa M. Magnetic resonance imaging of the TMJ disc in asymptomatic volunteers. J Oral Maxillofac Surg. 1987;45:852–3.
60. Bean LR, Thomas CA. Significance of condylar positions in patients with temporomandibular disorders. J Am Dent Assoc. 1987;114:76–7.
61. Rinchuse DJ, Rinchuse DJ. Orthodontics justified as a profession. Am J Orthod Dentofacial Orthop. 2002;121(1):93–6.
62. Clark JR, Evans RD. Functional occlusion-1. A review. J Orthod. 2001;28:76–81.
63. Cordray FE. Centric relation treatment and articulator mountings in orthodontics. Angle Orthod. 1996;66:153–8.
64. Rinchuse DJ, Kandasamy S, Sciote J. A contemporary and evidence-based view of canine protected occlusion. Am J Orthod Dentofacial Orthop. 2007; 132:90–102.
65. Woda A, Vigneron P, Kay D. Non-functional and functional occlusal contacts: a review of the literature. J Prosthet Dent. 1979;42:335–41.
66. Scaife RR, Holt JE. Natural occurrence of cuspid guidance. J Prosthet Dent. 1969;22:225–9.
67. Cordray FE. Three-dimensional analysis of models articulated in the seated condylar position from a deprogrammed asymptomatic population: a prospective study. Part 1. Am J Orthod Dentofacial Orthop. 2006;29:619–30.
68. Utt TW, Meyers Jr CE, Wierzba TF, Hondrum SO. A three-dimensional comparison of condylar position changes between centric relation and centric occlusion using the mandibular position indicator. Am J Orthod Dentofacial Orthop. 1995;107:298–308.
69. Ellis PE, Benson PE. Does articulating study casts make a difference to treatment planning? J Orthod. 2003;30:45–9.
70. Lavine D, Kulbersh R, Bonner P, Pink FE. Reproducibility of the condylar position indicator. Semin Orthod. 2003;9:96–101.
71. Schmitt ME, Kulbersh R, Freeland T, Bever K, Pink FE. Reproducibility of the Roth power centric in determining centric relation. Semin Orthod. 2003; 9:2–8.
72. Freeland TD. Articulators in orthodontics. Semin Orthod. 2012;18:51–62.
73. Rekow ED, Worms FW, Erdman AG, Speidel TM. Treatment-induced errors in occlusion following orthognathic surgery. Am J Orthod. 1985;88:425–32.
74. Posselt U. Studies in the mobility of the human mandible. Acta Odontol Scand. 1952;10 suppl 10:1–160.
75. Lindauer SJ, Sabol G, Isaacson RJ, Davidovitch M. Condylar movement and mandibular rotation during jaw opening. Am J Orthod Dentofacial Orthop. 1995;105:573–7.
76. Nuelle DG, Alpern MC. Centric relation or natural balance. In: Alpern SB, editor. The ortho evolution—the science and principles behind fixed/functional/splint orthodontics. New York: GAC International; 2003. p. 37–47.
77. Buschang P, Santos-Pinto A. Condylar growth and glenoid fossa displacement during childhood and adolescence. Am J Orthod Dentofacial Orthop. 1998; 113:437–42.
78. Haas Jr OL, Becker OE, de Oliveira RB. Computer-aided planning in orthognathic surgery- systematic review. Int J Oral Maxillofac Surg. 2015;44:329–42.
79. Zinser MJ, Sailer HF, Ritter L, Braumann B, Maegele M, Zoller JE. A paradigm shift in orthognathic surgery? A comparison of navigation, computer-aided designed/computer-aided manufactured splints, and "classic" intermaxillary splints to surgical transfer of virtual orthognathic planning. J Oral Maxillofac Surg. 2013;71:2151. E1–21.
80. Uribe F, Janakiraman N, Shafer D, Nanda R. Three-dimensional cone-beam computed tomography-based virtual treatment planning and fabrication of a surgical splint for asymmetric patients: surgery first approach. Am J Orthod Dentofacial Orthop. 2013;144:748–58.
81. Farrell BB, Franco PB, Tucker MR. Virtual surgical planning in orthognathic surgery. Oral Maxillofac Surg Clin North Am. 2014;26:459–73.
82. Maestre-Ferrin L, Romero-Millan J, Penarrocha-Oltra D, Penarrocha-Diago M. Virtual articulator for the analysis of dental occlusion: an update. Med Oral Patol Oral Cir Bucal. 2012;17:e160–3.
83. Watt DM. Temporomandibular joint sounds. J Dent. 1980;8:119–27.
84. Greene CS, Laskin DM. Long-term status of TMD clicking in patients with myofascial pain and dysfunction. J Am Dent Assoc. 1988;117:461–5.
85. Vincent SD, Lilly GE. Incidence and characterization of temporomandibular joint sounds in adults. J Am Dent Assoc. 1988;116:203–6.
86. Rinchuse DJ, Abraham J, Medwid L, Mortimer R. TMJ sounds: are they a common finding or are they indicative of pathosis/dysfunction? Am J Orthod Dentofacial Orthop. 1990;98:512–5.
87. De Leeuw R. Internal derangements of the temporomandibular joints. Oral Maxillofac Surg Clin North Am. 2008;20:159–68.
88. Farrar WB. Differentiation of temporomandibular joint dysfunction to simplify treatment. J Prosthet Dent. 1972;28:629–36.
89. Farrar WB. Disk derangement and dental occlusion: changing concepts. Int J Periodontics Restorative Dent. 1985;33:713–21.
90. Farrar WB, McCarty Jr WL. The TMJ dilemma. J Ala Dent Assoc. 1979;63:19–26.

91. Flores-Mir C, Nebbe B, Heo G, Major PW. Longitudinal study of temporomandibular joint disc status and craniofacial growth. Am J Orthod Dentofacial Orthop. 2006;130:324–30.
92. Nebbe B, Major PW, Prasad NG. Female adolescent facial pattern associated with TMJ disk displacement and reduction in disk length. Part I. Am J Orthod Dentofacial Orthop. 1999;116:167–76.
93. Nebbe B, Major PW, Prasad NG. Male adolescent facial pattern associated with TMJ disk displacement and reduction in disk length. Part II. Am J Orthod Dentofacial Orthop. 1999;116:301–7.
94. Hall HD. Intra-articular disc displacement. Part I: its significant role in temporomadibular joint pathology. J Oral Maxillofac Surg. 1995;53:1073–9.
95. Hall HD, Nickerson JW. Is it time to pay more attention to disc position? J Orofac Pain. 1994;8:90–6.
96. Larheim TA, Westesson PL, Sano T. Temporomandibular joint disk displacement: comparison in asymptomatic volunteers and patients. Radiology. 2001;218:428–32.
97. Dolwick LF. Intra-articular disc displacement. Part I: its questionable role in temporomandibular joint pathology. J Oral Maxillofac Surg. 1995;53:1069–72.
98. Tallents RH, Katzberg RW, Murphy W, Proskin H. Magnetic resonance imaging findings in asymptomatic volunteers and symptomatic patients with temporomandibular disorders. J Prosthet Dent. 1996;75:529–33.
99. Kircos L, Ortendahl D, Mark AS, Arakawa M. Magnetic resonance imaging of the TMJ disc in asymptomatic volunteers. J Oral Maxillofacial Surg. 1987;45:852–4.
100. Emshoff R, Brandlmaier I, Bertram S, Rudisch A. Comparing methods for diagnosing temporomandibular joint disk displacement without reduction. J Am Dent Assoc. 2002;133:442–51.
101. Emshoff R, Brandlmaier I, Gerhard S, Stobl H, Bertram S, Rudisch A. Magnetic resonance imaging predictors of temporomandibular joint pain. J Am Dent Assoc. 2003;134:705–14.
102. Kurita K, Westesson PL, Yuasa H, Toyama M, Machida J, Ogi N. Natural course of untreated symptomatic temporomandibular joint disc displacement without reduction. J Dent Res. 1998;77:361–5.
103. Klasser GD, Greene CS. Role of oral appliances in the management of sleep bruxism and temporomandibular disorders. Alpha Omegan. 2007;100:111–9.
104. Choi BH, Yoo JH, Lee WY. Comparison of magnetic resonance imaging before and after nonsurgical treatment of closed lock. Oral Surg Oral Med Oral Pathol. 1994;78:301–5.
105. Clark GT. The TMJ, repositioning appliance: a technique for construction, insertion, and adjustment. J Cranio Pract. 1986;4:37–46.
106. Clark GT. Classification, causation and treatment of masticatory myogenous pain and dysfunction. Oral Maxillofac Surg Clin North Am. 2008;20:145–57.
107. Isaacson KG, Thom AR, Horner K, Whaites E. Orthodontic radiographs: guidelines for the use of radiographs in clinical orthodontics. 3rd ed. London: British Orthodontic Society; 2008.
108. van Vlijmen OJC, Kuijpers MAR, Berge SJB, Schols JGJH, Maal TJJ, Bruening H, Kuijpers-Jagtman AM. Evidence supporting the use of cone-beam computer tomography in orthodontics. J Am Dent Assoc. 2012;143:241–52.
109. American Academy of Oral and Maxillofacial Radiology. Clinical recommendations regarding use of cone beam computed tomography in orthodontics. Position statement by the American Academy of Oral and Maxillofacial Radiology. Oral Surg Oral Med Oral Pathol Oral Radiol. 2013;116:238–57.
110. Carlson SK, Graham J, Mah J, Molen A, Paquette DE, Quintero JC. Let the truth about CBCT be known. Am J Orthod Dentofacial Orthop. 2014;145:418–9.

7 Idiopathic/Progressive Condylar Resorption: An Orthodontic Perspective

Chester S. Handelman and Louis Mercuri

7.1 Introduction

Idiopathic condylar resorption (ICR), which is alternatively called progressive condylar resorption (PCR), is an uncommon aggressive form of degenerative disease of the temporomandibular joint (TMJ). It is usually encountered in adolescent and young females, although it has also been observed in males. Pathognomonic features of this condition include a loss of condylar mass, thereby decreasing the height of the ramus and length of the mandible, and an opening rotation of the mandible resulting in a Class II open bite.

The purpose of this chapter is to provide the orthodontist with practical knowledge about the diagnosis of this disease and an approach to its management and treatment based on an update of our previous publications. We will also present material from long-term records of some idiopathic condylar resorption (ICR) patients to illustrate the problems the orthodontist might face. We also report findings from a survey of a group of orthodontists to determine their experience with this troubling disease [1, 2]. We will discuss the orthodontists' responsibility in managing ICR/PCR cases, including how to avoid legal liability sometimes associated with these patients. Finally we will discuss a rationale for total alloplastic joint replacement for cases that may be refractory to TMJ or orthognathic surgery.

The orthodontist is likely to have contact with patients afflicted with ICR/PCR in the following two contexts. The first are patients who spontaneously exhibit ICR/PCR independent of surgical intervention. The most troubling are those who develop ICR/PCR during orthodontic treatment or in retention. These patients are almost always young adolescent females, while others are afflicted in their late teens or early 20s. Their clinical records show that their occlusion was acceptable in the past, but has deteriorated within a relatively brief time period. The second group includes patients who have undergone orthognathic surgery for correction of any one or more of the following conditions: anterior open bite, mandibular retrognathia, or long anterior face height. Following surgery the intermaxillary correction appears to be successful, but by the 3rd to 6th month afterward the correction starts to fail to a variable extent [3, 4].

Do these two groups of patients have different problems, either clinically or pathophysiologically? Or are they the same, with ICR/PCR manifesting itself at somewhat different ages and under different circumstances? In both cases, the vast majority of

C.S. Handelman, DMD (✉)
Department of Orthodontics,
University of Illinois at Chicago,
College of Dentistry, Chicago, IL 60612, USA
e-mail: cshortho@prodigy.net

L. Mercuri, DDS, MS
Department of Orthopedic Surgery,
Rush Medical University Center,
Chicago, IL 60612, USA

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_7

these patients share similarities in sex, age, malocclusion, skeletal pattern, and condylar pathology. With the exception of subjects with known medical or traumatic causality, these patients are described as having an "idiopathic" problem (i.e., ICR) [5].

7.2 Sex and Age

ICR/PCR is a disease of young females in their teens or early 20s. Many diseases have a higher incidence in either males or females; for example, a greater number of females are reported to suffer from temporomandibular disorders [6]. It is unusual for a disease to be clustered almost completely within one gender when the sexual organs are not directly involved. Gunson et al. have made a case for low serum 17 beta-estradiol as a major factor in progressive condylar resorption [7]. They state that the use of oral contraceptives and abnormal menstrual cycles are often seen in females with severe condylar resorption. Milam states that estrogen receptors are present in the female TMJ; and low levels of estrogen can have a negative influence on joint tissues. He also has observed that there may be a predisposition to degenerative joint disease in certain females including even ordinary osteoarthritis [8].

But why should there be a preponderance of adolescent and young females in the ICR/PCR population? The premenstrual female may have insufficient circulating estrogen to initiate condylar pathology, but then there is the onset of menses. The age of expression of ICR/PCR is in adolescent and young females up to the third decade of life [9]. Interestingly, there appears to be an unexplained "burnout" of the resorption process in afflicted individuals sometime in the mid 20s, although resorption can extend into the early 30s. It should be remembered that ICR/PCR is a subset of the more inclusive "degenerative joint disease," but it is uniquely aggressive with dramatic facial and occlusal changes occurring in adolescent and in young adults.

Can the preadolescent facial type and occlusion predict later ICR/PCR? Studies have shown that the orthognathic surgery patient most susceptible to ICR/PCR has a Class II open bite type malocclusion with a high mandibular plane angle [3]. Can we assume that the ICR/PCR patient have these tendencies early in life, for example in the mixed dentition? Not necessarily (See Fig 7.3a).

7.3 Mechanical Loads

The healthy TMJ that undergoes natural remodeling can withstand and adapt to heavy mechanical loads that are frequently experienced, including parafunctional habits like nocturnal bruxism, orthodontic procedures like wearing elastics, and orthopedic appliances such as Herbst or chin cup [10]. Facial trauma as well as orthognathic surgical procedures can also produce increased loads on the TMJs [10]. However, certain adolescent and young adult females appear to be susceptible to developing degenerative joint disease that progresses to condylar resorption when their TMJs are exposed to excessive mechanical loading. Essentially, the intrinsic adaptive capacity of their joints to withstand mechanical loads is exceeded by their functional (or parafunctional) demands, which is just another way of saying they are a vulnerable subset of the population [10, 11].

Even healthy joints cannot withstand extreme mechanical loads that exceed their adaptive capacity. Studies have shown that this can be the result of severe trauma or a surgical mandibular advancement over 5 mm. For example, Scheerlink et al. have shown that surgical mandibular advancement of less than 5 mm resulted in only 2 % of patients developing significant condylar resorption, while 10 % of cases with 5–10 mm advancement had resorption, and in cases with greater than 10-mm advancement 67 % of cases had resorption [12]. Apparently, the forces produced by the stretched musculature and soft tissues exceeded the adaptive capacity of the joints in those cases.

7.4 Orthognathic Surgery as a Risk Factor

Orthognathic surgery for the correction of the Class II open bite malocclusion usually involves maxillary impaction via a LeFort I osteotomy to induce mandibular closing rotation, combined

with mandibular advancement via bilateral sagittal split osteotomies (BSSO). Both surgeries will cause a sudden repositioning of the condyles in the fossae and thereby alter both the direction and the magnitude of the mechanical load in the TM joints [3, 10, 13]. In most patients the joints will remodel and adapt to this change; but in some patients the remodeling capacity of their TMJs is exceeded by the functional demands of these sudden anatomical changes, and their condyles resorb.

Arnett et al. have demonstrated that the use of bicortical screws to fixate the mesial and distal segments during BSSO can rotate the condylar segments either laterally or medially in the glenoid fossa. This torquing of the condyle could initiate condylar resorption [10]. To minimize torquing they suggest using titanium bone plates adapted to the outer cortical surfaces of the two segments with unicortical screw fixation to minimize this problem. They also point out that overseating the condyle in the fossa during BSSO can cause compression resulting in dysfunctional remodeling of the joint [13].

Surgical procedures to correct the Class II open bite and associated mandibular retrognathia were in place by the mid 1970s. In the 1980s to 1990s, there was an increasing number of patients undergoing orthognathic surgery. A number of Dutch surgeons observed that some patients with satisfactory initial responses to surgery subsequently would experience variable amounts of relapse. They realized this was mostly due to condylar resorption. Two factors contributed to their hypothesis: (1) they had extensive documentation on a large number of surgical patients pre- and postsurgery as well as during long-term follow-up; and (2) they understood that the condyle was sensitive to pathological resorption because of the pioneering research of their mentor Dr. G. Boering, who had described "arthrosis deformans" in the 1960s [14]. In a series of important papers, these surgeons reported on postsurgical follow-up for subjects who had undergone orthognathic surgery [15–18]. In the combined patient groups, the incidence of ICR/PCR was found to be between 1.2 and 5.8 %. When the surgeons investigated further, they found the relapse was concentrated in the Class II high angle group. For this select group, the ICR/PCR-induced relapse was between 19 and 31 % [3, 15, 17, 19–24]. This condylar resorption was labeled "idiopathic," since it was difficult to determine the exact cause or which patients would have a successful or unsuccessful outcome.

Re-operation on the ICR/PCR patients whose first orthognathic surgery was unsuccessful had close to a 50 % failure rate [3, 18, 23, 25]. Patients with pre-existing signs of ICR/PCR prior to orthognathic surgery also had unfavorable results. Arnett and Tumborello reported 4 of 9 of these patients had postoperative condylar resorption [23], while Huang et al. reported 4 of 18 had a postsurgical relapse [25].

In a study by Hoppenreijs et al., the incidence for condylar resorption in ICR/PCR patients following maxillary surgery for correction of Class II open bite malocclusions was less than after surgeries involving both jaws (9 % compared to 23 %) [21]. However, most severe Class II open bite patients will benefit from having bimaxillary surgery as well as advancement genioplasty in order to maximize facial aesthetics (Fig. 7.1). Hoppenreijs et al. also demonstrated that the initial correction can relapse due to condylar resorption by the 6th month, and this resorption can continue for up to 3 years [21].

7.5 Pathology of the Condyle Undergoing Resorption

The TMJ condyle is covered with a layer of fibrocartilage. During ICR/PCR this tissue breaks down, and then the outer osseous cortex of the condyle starts to resorb [8, 9]. This is seen on imaging as resorption lacunae, with the disappearance of the dense outer cortical layer. Other changes include narrowing as well as shortening of the condylar process and ultimately there are changes in the length of the ramus [8]. There is demineralization of the bone below the cortex of the condyle. This may result in the collapse of articular surface bone, which is clinically manifested by a rapid opening of the bite and opening rotation of the mandible [9]. Although the disease is described as condylar resorption, there may also be resorptive changes occurring in the articular eminence which tends to flatten [3, 9]. ICR/PCR

can affect either one or both condyles; if unilateral, the result is significant facial asymmetry. However, in most cases both condyles are involved, although one joint may demonstrate more advanced pathology than the other. This more common bilateral pattern may indicate a genetic predisposition as well as the humoral nature of the etiology. Hatcher has illustrated both the resorptive stages and the eventual healing stages of condylar resorption visualized on CBCT imaging (Fig. 7.2) [26].

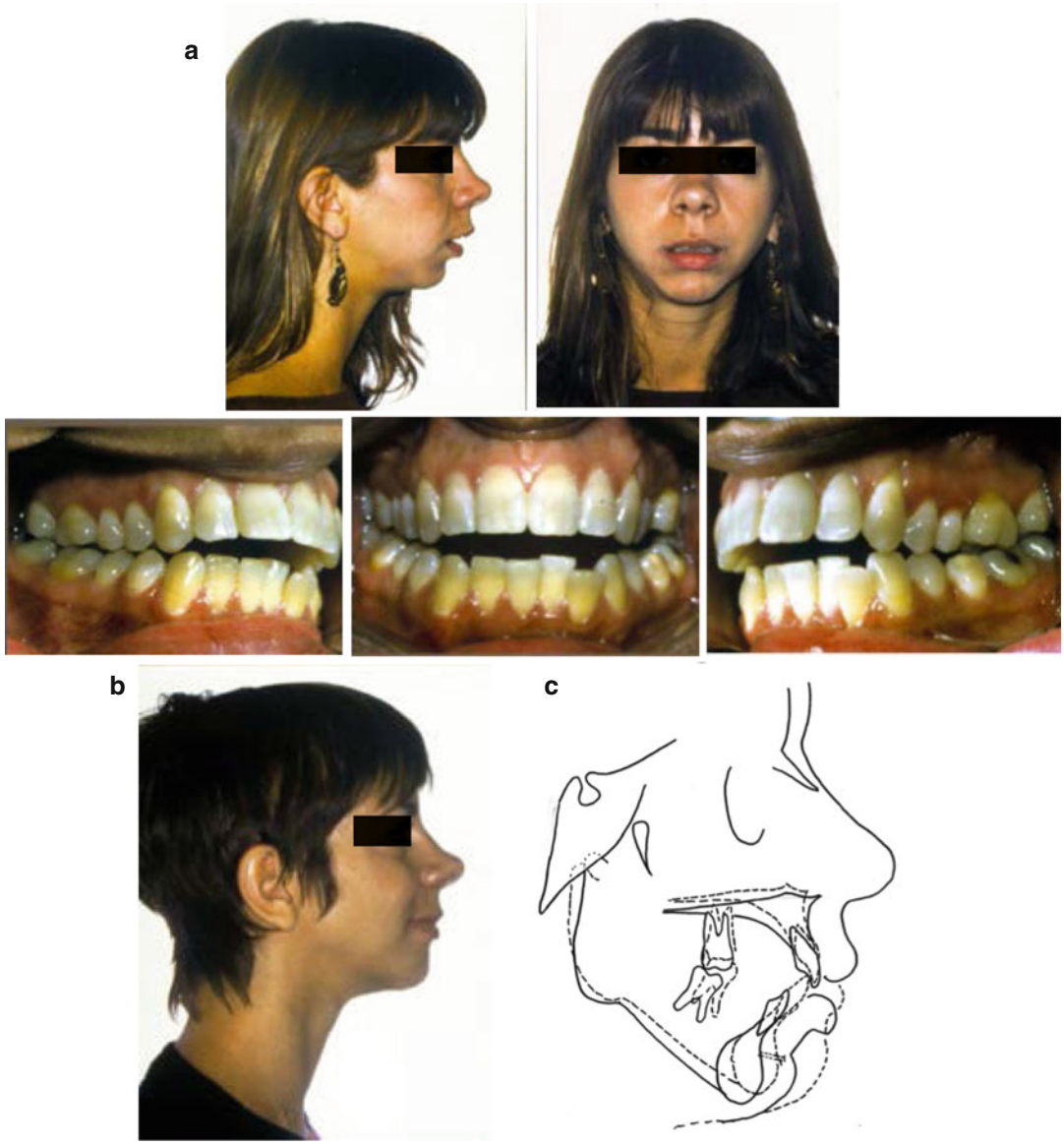
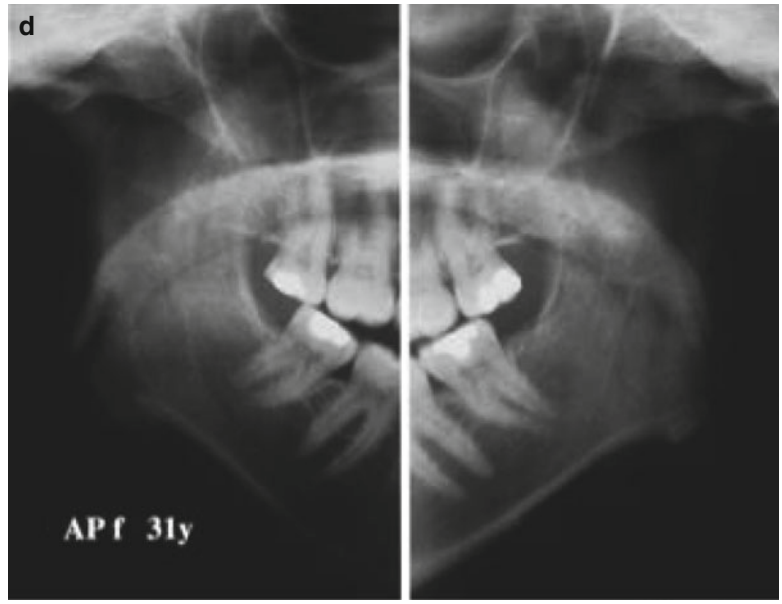


Fig. 7.1 (**a**) Left portrait: age 34 years 8 months pretreatment (**b**). Patient portrait: 35 years 3 months postsurgery. The surgical improvement was dramatic and involved maxillary impaction, mandibular autorotation, and bilateral sagittal split osteotomy for mandibular advancement as well as advancement genioplasty (Reprinted with permission from Handelman and Greene [2]). (**c**) Cephalometric superimposition on Nasion-Basion line at Basion. Presurgery: 34 years 8 months solid line and 35 years 3 months dotted line. Note: soft tissue advancement of lower lip and chin. This improvement would not be possible limited surgery to the maxilla. (**d**) Panorex: age 34 years 8 months. Note: minimal size of condyles [2]

Fig. 7.1 (continued)



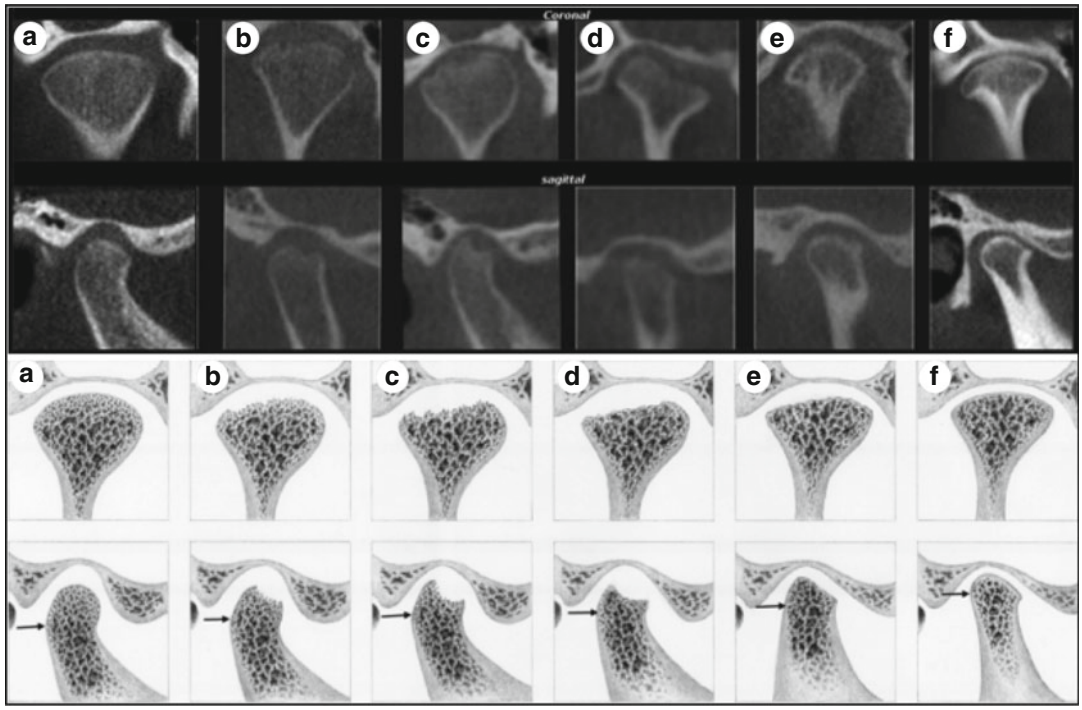


Fig. 7.2 Imaging stages of ICR/PCR; CBCT TMJ images at top and associated anatomic illustrations below. Image *A* – normal TMJ: Band *C*, the destructive stage; *B* and *E*, the repair stage; *F*, the stable stage (Reprinted with permission from Hatcher [26])

7.6 The Role of the TMJ Disc in Development of ICR/PCR

The TMJ disc acts as a shock absorber during load bearing mandibular movements, while also providing lubricated surfaces over which translation and rotation movements occur [11, 27]. The role of the disc in the etiology of ICR/PCR is controversial in terms of "chicken-and-egg" explanations. The functional surfaces of both the condyle and the articular eminence can demonstrate degenerative changes when the disc is perforated or displaced without reduction [28, 29]. Link and Nickerson studied patients referred for orthognathic surgery and reported that all their open bite cases and 88 % of their Class II malocclusions had bilateral disc displacement [30]. On the other hand, the condyles undergoing resorption simply may not be able to maintain normal disc position, so the discs may become displaced. Wolford and Cardenas recommend joint surgery to attach the displaced disc to the head of the condyle prior to or during orthognathic surgery for ICR/PCR to prevent reoccurrence of disc displacement or changes in disc morphology [31]. Other surgeons do not agree that disc surgery is necessary. They do not enter the joint when performing orthognathic surgery on ICR/PCR patients, especially if the joint is functional – even if the condyle and ramus are severely affected by the disease (Figs. 7.1 and 7.3) [3, 13]. It should be remembered that TMJ disc displacement occurs in about 1/3 of asymptomatic individuals in the random population, while ICR/PCR is a relatively rare occurrence [2, 32]. Also, patients with degenerative joint disease of the TMJ do not necessarily have abnormally positioned discs [32].

7.7 Survey of Orthodontists About ICR/PCR

Author CH mailed a questionnaire in 1998 regarding ICR/PCR experience to a group of orthodontists who were members of the Midwest component of the Edward H. Angle Society and faculty of the orthodontic department at the University of Illinois, Chicago [2]. From a total of 69 mailings there were 57 responses, and these practitioners reported seeing only 56 cases of ICR/PCR during their years in practice. An incidence of ICR/PCR of approximately 1 per 5,000 patients was derived by estimating the number of new patients examined over the years in each practice. Another way of looking at the rarity of the incidence was that each provider saw an average of 1.3 ICR cases in his/her career up to the point of the survey. This incidence may be low due to failure to recognize the disease, and small sample size, but it does demonstrate the infrequency of ICR/PCR seen in an orthodontic practice. Surgery centers that perform a large number of orthognathic procedures have reported an incidence of 2–5 %, which is still infrequent, but more common than reported by the orthodontists [15–19].

Of the 56 cases of ICR/PCR recorded in this survey, 35 (62.5 %) were spontaneous with no history of surgery, while 21 (37.5 %) developed the problem following surgery. The following data were derived from 40 of the questionnaires where there was adequate reporting:

- The ICR/PCR group was comprised of 38 (95 %) females and 2 males who were brothers. There were 37 (92.5 %) whites, 3 Asians, and no blacks in the group.
- Of the 40 cases, 33 (82.5 %) were reported to be bilateral and 7 were unilateral.
- The incidence of TMJ or facial pain was 12 (30.7 %) out of 39 cases.
- The malocclusions before ICR/PCR were diagnosed, 14 (35 %) Class I, 1 (2.5 %) Class I open bite, 12 (30 %) Class II, 12 (30 %) Class II open bite, and 1 unclassified.
- Severity of the ICR/PCR as rated by the respondents was one case mild, while 39 were described as severe or extreme.

From the responses, it is clear that these were patients that the orthodontist remembered well. This survey should be repeated with a greater number of orthodontic offices participating. However, even these limited findings confirm the anecdotal

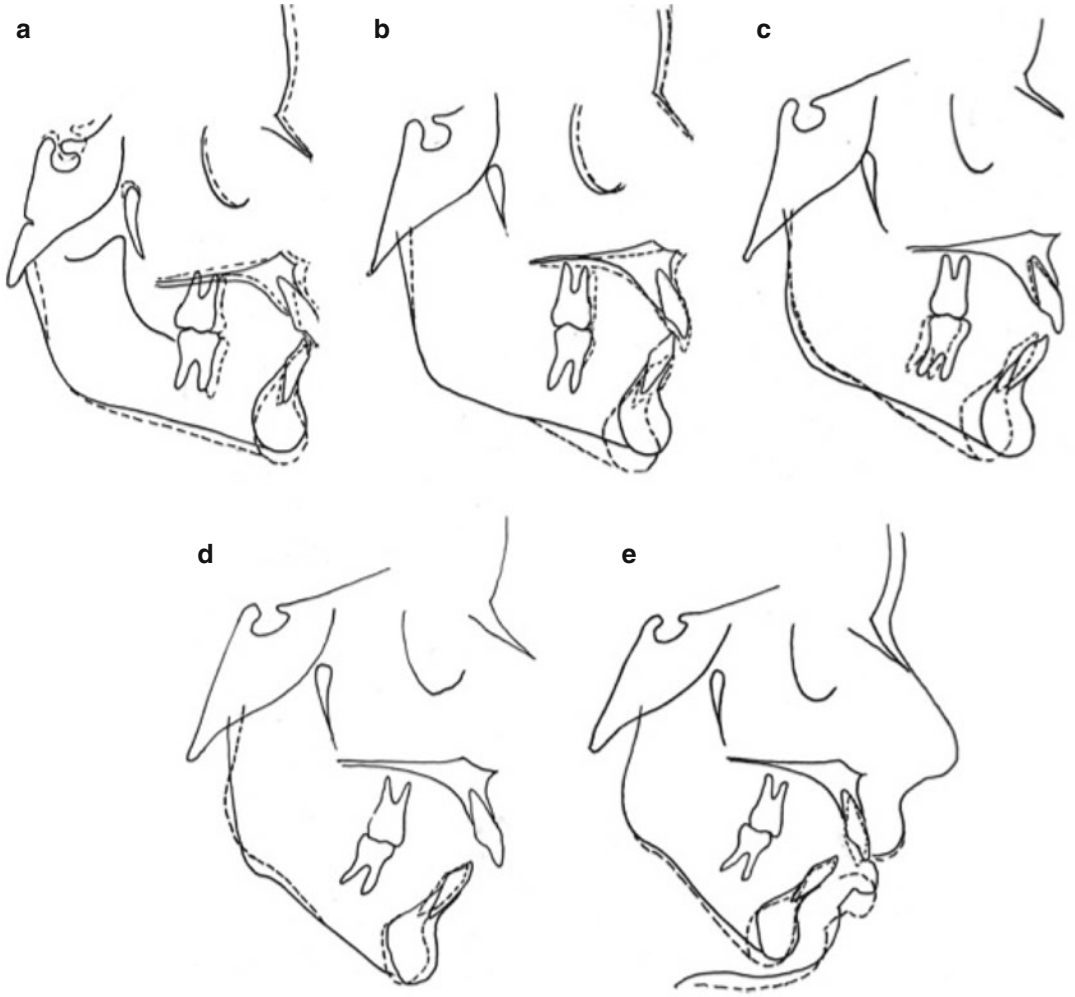


Fig. 7.3 Series of cephalometric images of a patient with severe ICR/PCR. All x-rays are superimposed on the Nasion-Basion line at Basion. Articulare moves forward due to resorption on the anterior aspect of the condyle. (**a**) Cephalometric superimposition from pre-orthodontic treatment 10 years, 3 months (*solid line*) to postorthodontic treatment at 13 years (*dashed line*). Note at age 10 years 3 months future ICR/PCR could not be predicted. The maxilla grew normally, while the mandible grew vertically but not anteriorly. The mandible rotated down and back, and articulare moved mesially. (**b**) Cephalometric superimposition of postorthodontic treatment from age 13 (*solid line*) to 14 years (*dashed line*). The maxilla grew normally, while the mandible rotated downward and backward, causing an open bite with an increased overjet. Articulare moved forward. (**c**) Cephalometric superimposition from age 14 years (*solid line*) to 15 years, 10 months (*dashed line*). The maxilla has stabilized as growth has ceased. However, the mandible continued its downward and backward rotation and regression, with shortening of the ramus. (**d**) Cephalometric superimposition from 15 years, 10 months (*solid line*) to 30 years, 6 months (*dashed line*). The condylar resorption has continued with shortening of the ramus and mesial movement of articulare. (**e**) Cephalometric superimposition from 18 years, 6 months (*solid line*) to 30 years, 10 months (*dashed line*). The condyle continued to resorb with a downward and backward rotation of the mandible

reports in the literature that ICR/PCR is a disease affecting young females of Caucasian or Asian descent, less than half presenting with TMJ pain, and with variable occlusions prior to developing this problem, and when ICR/PCR does occur it presents as a severe management problem.

7.8 Diagnosis of the ICR/PCR Patient

7.8.1 History

A careful history can reveal the probable diagnosis of ICR/PCR. A report of a sudden change in occlusion is nearly pathognomonic of ICR/PCR. Unfortunately, this change may occur during orthodontic treatment, when it easily can be misinterpreted as being due to unfavorable growth, tongue thrust, or poor cooperation. Or it might initially be regarded as an adverse response to treatment mechanics.

A history of autoimmune and collagen diseases should be part of the historical questioning [5]. Referral to a physician for rheumatoid factor serology should be considered, although this is usually negative in patients with ICR/PCR. A history of TMJ discomfort and disc displacement is an important factor, since a number of ICR/PCR patients report pain or other TMD symptoms and have displaced discs on imaging [2, 30]. When pain is reported, this may be an indication that the disease is active.

A family history of rheumatoid disease, TMD, and open bite malocclusion should be recorded even though there has been no report of familial incidence of ICR/PCR. A history of facial trauma, especially when the TMJ is involved, is important as a possible cause of condylar resorption [5]. Fortunately, only rarely does a trauma case evolve into ICR/PCR.

Orthodontic treatment and third molar extractions have been indicted as possible causes of ICR/PCR [10, 13]. Statistics indicate that this is a very rare disease and a large percentage of the young have had orthodontic treatment and/or third molar removal. Therefore, it is problematic to assign etiology of a rare disease to a common experience. However, in those rare subjects whose condyles are undergoing an early, often undetected, stage of ICR/PCR, orthodontic treatment, and/or third molar extractions may overwhelm the adaptive capacity of their vulnerable condyles.

Young female orthognathic surgery patients who have developed ICR/PCR prior to surgery have been reported to exhibit a history of irregular menstrual cycles, amenorrhea, or oral contraceptive use according to Gunson et al. [7] They suggest that mid-cycle serum levels of 17 beta-estradiol should be measured, because low levels were associated with postsurgical development of severe condylar resorption.

7.8.2 Orthopantogram and Lateral Cephalometric Radiographs

The orthopantogram (OPG) can be used for the gross examination of condylar anatomy. The condyle will appear to have lost mass relative to the rest of the mandible, and appear thin or shortened with flattening of the superior and/or anterior curvature [3]. In many cases, the condylar neck will have a distal inclination [3, 19, 21].

Lateral cephalometric imaging will show mandibular divergence relative to the cranial base and maxilla, shortened posterior facial height, and increased anterior facial height with an increase in the overjet and negative overbite. Sagittal measures for the skeletal Class II relationship will be increased. Serial cephalometric imaging is diagnostic for active ICR. There are many choices for lateral cephalometric superimposition of the films, but the method used in the tracings in this chapter superimposes on the Basion-Nasion plane at Basion (Fig. 7.3). The posterior shadow of the condyle on this plane is defined as articulare. Its location will reposition medially when ICR is active. The mandible on the succeeding cephalometric images will reveal a clockwise rotation, a shortened posterior height (articulare to gonion) and length (Fig. 7.4). One limitation of cephalometric imaging is that it is frequently difficult to visualize the shadow of the head of the condyle in advanced ICR/PCR subjects because of the resorption, thus the more convenient use of articulare.

7.8.3 Cone Beam Computed Tomography (CBCT)

While CBCT has increased radiation and cost relative to OPG and cephalometric imaging, it is

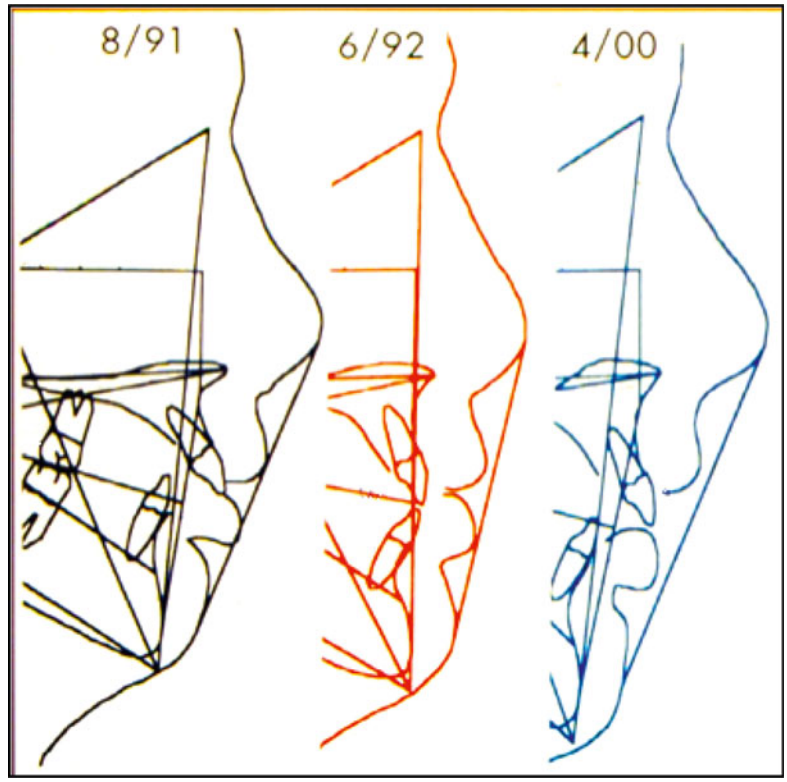


Fig. 7.4 Cephalometric tracing of postsurgical relapse. The *left*, 15 years 7 months, presurgery; the *middle* 16 years 5 months showing mandibular advancement for correction; the *right* 23 years 4 months showing 100 % relapse of the correction (Courtesy of Dr. John Russell, Mobile AL)

clearly justified because of its clarity and the avoidance of superimposition of adjacent structures. This enhances the clinician's ability to see detailed pathologic features of the condyle such as disappearance of the dense outer cortical layer, erosions, sclerosis, flattening and sub-cortical cyst formation (Fig. 7.2) [26, 34]. Prior to CBCT, tomograms of the joint were useful, but they were not three-dimensional, providing only sagittal cuts in one or more planes of space. Both tomograms and CBCT are made in the open and closed mouth position. Both can be useful to follow the progress of ICR/PCR and to determine when the disease is in remission. Radiologists look for the healing of the outer cortical layer [26, 34]. A second CBCT scan taken 1 year after the first should demonstrate a healed and stable joint prior to considering surgery. Also, superimposition of cephalometric images should demonstrate no change in mandibular position during this period.

Unfortunately, a healed joint does not necessarily mean the disease will not reactivate. It has been demonstrated that orthognathic surgery can reactivate the resorption processes [3, 4, 21, 25]. Figure 7.3 illustrates a case carefully followed with tomograms. ICR onset was at age 13 and the condyles were healed by age 16. However, orthognathic surgery performed at this age reactivated the ICR/PCR, resulting in 100% bony relapse.

The appearance of an osteophyte on the anterior aspect of the condyle is associated with ICR/PCR and other degenerative joint diseases (Fig. 7.2). However, this finding is thought to represent the healing of the condyle as it attempts to increase its bearing surface to distribute the joint loads [26, 34].

7.8.4 Radioisotope Diagnosis

The value of performing a radioisotope study as part of the diagnosis of ICR/PCR is disputed. Some believe that the resultant technicium-99 nuclear medicine scan is difficult to interpret, since various other TMJ conditions will also

demonstrate high levels of isotope uptake, while others are of the opinion it does have some diagnostic value [25, 33].

7.8.5 Magnetic Resonance Imaging (MRI)

MRI is useful in the examination of the soft tissues of the TMJ such as the cartilaginous integrity of the condylar head surface, articular disc position and condition, joint effusion and marrow edema [35]. However, it does not provide diagnostic images of the bony cortices of the condyle or eminence that are clearly defined by a tomogram or a CBCT scan.

7.8.6 Occlusal Splints: Diagnostic Aspects

Occlusal orthotic splints are suggested as a joint stabilizing modality in ICR/PCR cases when there is joint pain and dysfunction as well as before orthognathic surgery [36]. An often overlooked use is its potential as a diagnostic tool for determining cessation of the resorptive process. Patients suspected of having ICR/PCR should be fitted with a maxillary occlusal splint with contact registered on all the mandibular teeth. If the lower incisors no longer register contact at a future evaluation, this indicates further joint degeneration and active disease [1, 2]. A maxillary occlusal splint should routinely be placed following orthognathic surgery/orthodontic treatment at the time of retention for TMJ comfort, reduction of the forces on the joint, and to evaluate stability of the correction [1, 2, 36].

7.9 Timing and Surgical Options for Correction of ICR/PCR

The role of the orthodontist prior to orthognathic surgery is to prepare upper and lower teeth to an ideal arch form that will maximize occlusal contact in a normal Class I occlusion following surgery. This is true no matter which surgical approach is taken. A limited number of ICR/PCR cases can be successfully treated following remission of the disease by orthodontic means, without performing surgery. This is especially true when the resorption starts in the 20s rather than in the early teens (Fig. 7.5). These patients may have acceptable aesthetics, but this is not the situation in many cases. The tremendous loss of condylar mass and the resulting facial deformity in most cases requires comprehensive surgical management (Fig. 7.1).

ICR/PCR typically first appears in young adolescents, although Dibbets et al. has shown deformed condyles and growth disturbances in a younger age group [37]. The period from the teen years to the early 20s appears to be the most active time for ICR/PCR. Optimistically, the disease goes into remission and eventually "burns out" by the mid 20s. However, some patients continue to have active ICR/PCR into their 30s (Fig. 7.3). As a result, teenagers diagnosed with ICR/PCR should be advised to postpone orthognathic surgery, young girls with severe facial deformity and/or their parents often insist on correction earlier due to social and peer related pressures.

Another option available is to advance the mandible slowly by distraction osteogenesis, thereby allowing the soft tissue and musculature to adapt to the large mandibular advancements often required in such cases. Schendel et al. reported a case of 15.6 mm vertical elongation and 13.4 mm horizontal advancement using a curved distractor [40].

Gunson and Arnett have taken a more biomedical and pharmacologic approach in the management of patients with ICR/PCR or who might be at risk of postsurgical ICR/PCR [36]. They prescribe an occlusal splint employed for 6 months prior to as well as following orthognathic surgery to reduce the mechanical loads on the joint. Also, before and after surgery they place the patient on a comprehensive series of drugs to relax the musculature, decrease bruxism, decrease inflammation and reduce the patient's inherent bone resorption capacity [10, 36, 41]. They reported stable postorthognathic surgery results at 24-month follow-up in 24 ICR/PCR patients (American Society of TMJ Surgeons, May 2,3, 2014, Chicago, IL).

The approach to the management of ICR/PCR cases should be individualized and based on the

extent of the disease process. Arnett's and Gunson's approach appears appropriate for most early cases of ICR/PCR requiring orthognathic surgical management. For cases requiring extreme mandibular advancements, distraction osteogenesis might be considered [38].

If condylar resorption is still active, replacement of the joint with an autologous costochon-

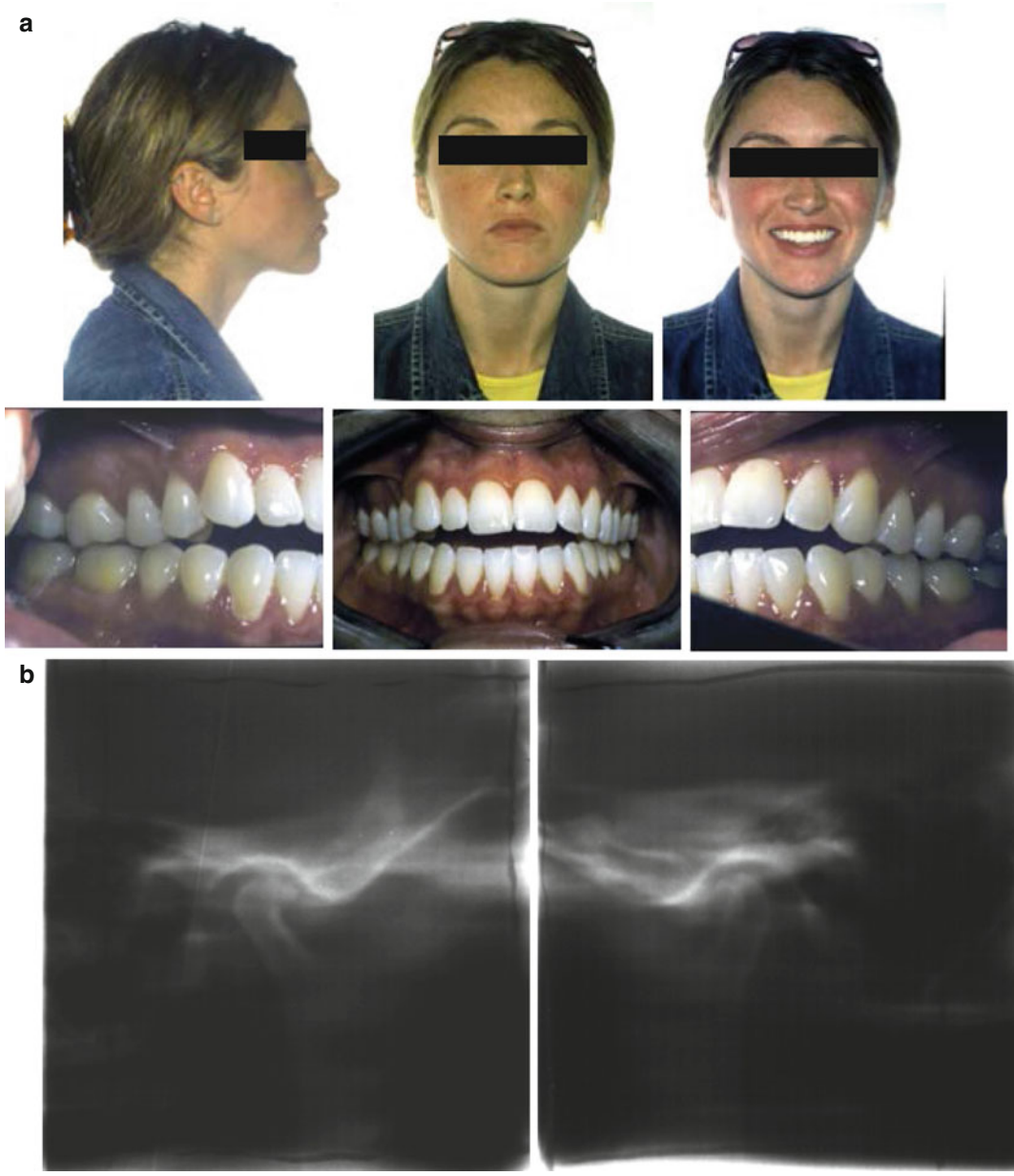


Fig. 7.5 Patient developed ICR/PCR at age 20 years and treated by orthodontic mechanics only. (**a**) Pretreatment portrait and intraoral photographs at age 26 years 4 months. Note favorable profile despite anterior open bite. (**b**) Tomograms at age 26 years 11 months demonstrate reduced size of condyles due to resorption from the superior surfaces, signs of flattening and erosions. A small osteophyte extends from the anterior surfaces. The radiologist diagnosed degenerative joint disease that was no longer active, so orthodontic treatment was started. (**c**) Postorthodontic treatment portrait and inter oral photographs were taken at age 33 years 2 months with the patient 5 years into retention. Note stability of the closure of the anterior open bite

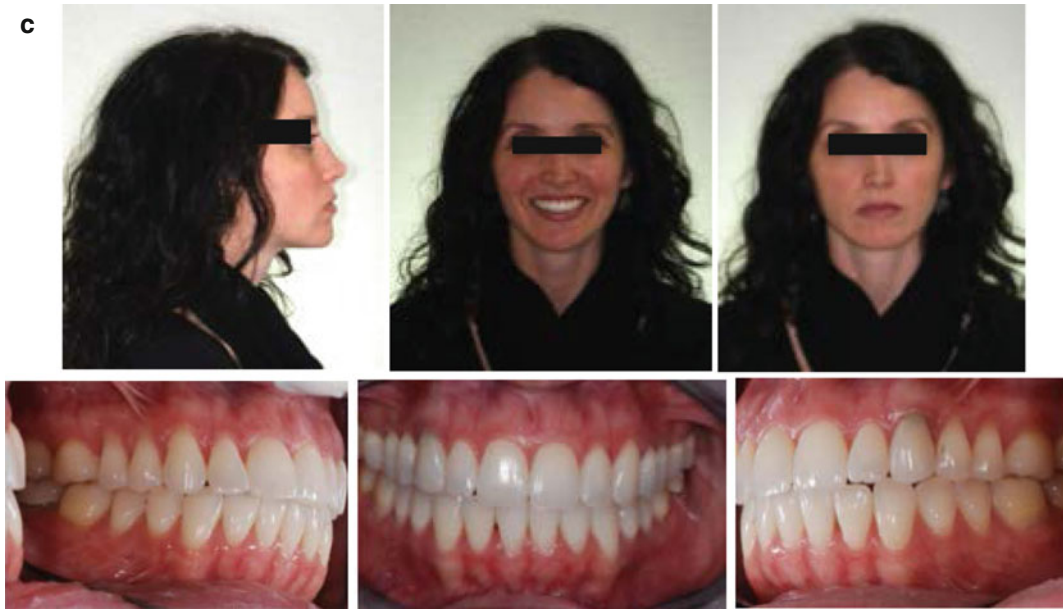


Fig. 7.5 (continued)

dral graft or total alloplastic temporomandibular joint replacement prosthesis is possible [25, 40–45]. There are a number of ICR/PCR patients who present with one of the following problems, who are best treated with synthetic total joint replacement (TJR). They are individuals with:

- Compromised function as seen in severely limited movement of the joints
- Failure of previous orthognathic surgery
- Poor prognosis for orthognathic surgical correction [41–45]

Patients with active ICR/PCR may also be candidates for total joint replacement as the artificial joint cannot resorb. The next section will discuss this option in detail.

7.10 Rationale for the Use of TMJ TJR in the Surgical Management of End-Stage ICR/PCR

End-stage disease is the most pernicious condition of an organ or disease state; in this state the organ can barely function. Examples include end-stage renal disease where the kidneys have shut down and the patient requires dialysis, or end-stage cardiac disease where the heart is functioning so poorly that it requires mechanical support or transplantation for the patient to survive [45].

End-stage joint disease portends a joint so architecturally devastated by disease or injury that it results in a severe functional impairment for the patient. In the ICR/PCR patient, the local adaptive capacity of the TMJ condyle has been mechanically and biologically tested in vivo by the disease process and it has failed. This failure is demonstrated by the load-compromised bony architecture having end-stage pathological changes as seen on imaging. Clinically, the articular end-stage pathology is manifested by the patient's signs and symptoms [41–45].

In cases where the surgeon selects costochondral grafting, orthognathic surgery, or distraction osteogenesis, the long-term outcomes rely on either the ability of an avascular graft or an end-stage diseased compromised condylar remnant to withstand potentially greater functional forces and loading. This is further complicated by the question of whether that compromised end-stage diseased TMJ complex will adapt despite what is possibly a local manifestation of systemic pathology [41–45].

Placing already functional-load-compromised articular bone into a mechanically stressful environment may account for the less than satisfactory outcomes cited with orthognathic surgery, distraction osteogenesis, and autogenous tissue replacement surgical options. This kind of mechanically and biologically unfavorable environment can occur as a result of many factors that are inherent in various surgical procedures. For example, it is well known that re-orientation of positional, muscular and/or rigid internal fixation (RIF) loading forces invariably develop after orthognathic and/or distraction surgical manipulation of the maxillofacial skeleton, and also after autogenous TMJ reconstruction. Therefore, an ICR/PCR management option not dependent on the compromised mechanical and biological adaptive capacity of the TMJ and surrounding tissues should be considered.

Total alloplastic joint replacement provides such an option because it is a biomechanical rather than biological solution to the management of joints that are anatomically distorted, end-stage diseased and dysfunctional. It has been recognized for decades that without TJR for the management of end-stage joint disease, the modern practice of orthopedic surgery would be inconceivable. TMJ TJR has likewise demonstrated a long-term safe and effective option for the reconstructive maxillofacial surgeon managing end-stage TMJ disease [43, 44].

The goals of any TMJ replacement, whether autogenous or alloplastic, are to: (1) improve mandibular function and form; (2) reduce suffering and disability; (3) contain excessive treatment and cost; and (4) prevent morbidity [44]. TMJ TJR as a management option for end-stage ICR/PCR meets all of them.

Autogenous tissue TMJ (e.g., costochondral graft) replacement options for ICR/PCR have not achieved these goals as evidenced by outcomes requiring at least one and sometimes two re-operations with increased disability, morbidity and costs [41–45].

The advantages of TMJ TJR are: (1) physical therapy can begin immediately; (2) there is no need for a secondary donor site and decreased surgery time; and (3) they are able to mimic normal anatomy [42]. In the ICR/PCR patient, there is the added advantage that the materials from which these devices are constructed are not susceptible to the pathophysiology of the disease process.

Autogenous tissue TMJ replacement involves harvesting an avascular-free bone graft (rib), using rigid internal fixation (RIF) to fixate and stabilize it against the host ramus, cortex-to-cortex. Maxillomandibular fixation is required while the free bone graft vascularizes and integrates into the host bone and surrounding soft tissues. Reitzik reported that despite ideal healing circumstances, the return to full strength cortex-to-cortex healing took 20 weeks in a monkey; therefore, he postulated it would probably require 25 weeks in man [46]. This would rule out early masticatory muscle rehabilitation, because any graft-to-host bone mobility leads to graft failure [47].

Long-term functional immobility is contrary to all of the principles of physical rehabilitation after joint surgery of any kind. Salter definitely demonstrated with his continuous passive motion (CPM) theory that early active physical therapy after orthopedic joint surgery is essential to successful long-term improved functional outcomes [48]. Early physical therapy after conventional surgical procedures can lead to mechanically unfavorable stresses and strains on the host bone in a compromised biological environment, resulting in failure and relapse in ICR/PCR cases. On the other hand, because of its inherent immediate fixation stability, TMJ TJR insertion permits immediate active physical therapy leading to improved long-term functional outcomes.

Individually customized patient-fitted TMJ TJR fossa and ramus (condyle) components are designed and manufactured from a stereolaser model generated from the patient's CT scan data to mimic the normal anatomical contours of the structures they are intended to replace. At implantation, these TMJ TJR components are adapted and fixed in a stable and close fashion to the bony surfaces of the temporal bone and mandibular ramus [49, 50].

There is always a component of counter-clockwise mandibular rotation in the surgical management of ICR/PCR. This fact has negative implications for the outcome of most traditional

ICR/PCR treatments, because one cannot predictably expect an avascular autogenous rib graft or a compromised condyloid process to withstand the muscle and other soft tissue forces generated in these new anatomic relationships. Under functional loading in the short-term as well as the long-term, there is a significant potential for relapse if one considers the effects of muscle forces on bone. However, the long-term stability using a customized patient-fitted TMJ TJR in such cases is well documented [51–54].

The relative disadvantages of TMJ TJR are: (1) cost of the device; (2) material wear and failure; (3) uncertainty about long-term stability; and (4) alloplastic implants not following a patient's growth [42].

Considering the demographics of ICR/PCR, longevity of any TMJ TJR must be an important consideration. Since this is a biomechanical rather than a biological solution, future planning must be made for revision surgery to remove scar tissue from the articulating components of the implant. Eventually replacement of the implant over time due to material wear and/or failure may be required. At present, patients are advised that these devices may have a functional life-span of 10–15 years based on the orthopedic experience in total joint arthroplasty and recent results of TMJ TJR long-term outcomes [42, 44, 48, 49, 52–63].

In orthopedics, advancement in surgical techniques, implant materials, and designs have led to excellent long-term function and quality of life improvement results. Survival rates of an orthopedic joint replacement device have been reported to exceed 90 % after 10 years in younger patients [64, 65]. Orthopedists are therefore no longer deterred from replacing end-stage diseased joints in younger patients in order to provide these patients with an improved function and quality of life. The same consideration should be given to ICR/PCR patients when making the decision for how to manage the skeletal consequences of their end-stage TMJ disease process. This is an especially important point in regard to these patients, because their average age as a group is quite low and most are likely to live into their geriatric years.

When calculating cost, it is critical that not only is the cost of the device to be considered, but also all factors associated with its implantation. The major advantage of TMJ TJR over autogenous reconstruction is that there is no need for a secondary surgical donor site (rib). Donor site morbidity is therefore eliminated, as are the expenses associated with prolonged intra-operative and postoperative hospitalization soft tissue stretching procedures. Despite the fact that these devices may be considered expensive, the relative overall cost involved with TMJ TJR is either equal to or eventually less than autogenous reconstruction [48].

Between 1985 and 2010, 14 ICR/PCR patients presented to the author (LM) for management. All were females between the ages of 13 and 34 years (mean age = 23.4, s.d. = 7.1). At their initial visit, all of them had a history, clinical and imaging characteristics of ICR/PCR, and they also had negative rheumatoid factor serology. All but one (YF) were taking oral contraceptive pills (OCPs) either to control menstrual dysfunction or prevent conception [38].

7.11 Case

TC, a 26-year-old female (Fig. 7.6), presented for consultation in 2009 with chief complaints of increasing bilateral TMJ pain and dysfunction, headaches, progressive mandibular retrusion, and obstructive sleep apnea (OSA) for which she required nocturnal continuous positive airway pressure (CPAP). Her maximum incisal opening (MIO) was 25 mm. She denied any history of prior orthodontics, arthritic disease, micro- or macro-mandibular trauma, clenching, or bruxism. A review of systems was unremarkable except for a history of OSA and dysmenorrhea managed by OCPs. Rheumatoid factor and serology were negative.

Imaging and cephalometric analysis revealed bilateral severe end-stage degenerative condylar changes (Fig. 7.6), a compromised upper airway and microgenia. Therefore, the working diagnosis was ICR/PCR, OSA, and microgenia.

The management plan included bilateral TMJ TJR using a patient-fitted TMJ replacement

system (TMJ Concepts, Ventura, CA), LeFort I osteotomy, and advancement genioplasty.

In 2010, TC underwent uneventful surgery. Immediately postimplantation she was able to discontinue the use of the CPAP device and reported no further significant TMJ pain or headaches.

At 4 years postoperatively, she has maintained a stable, repeatable, and functional occlusion. Her MIO was 35 mm. Imaging demonstrated excellent integration of the fixation screws, no evidence of osteolysis around the device fossa or ramus/condyle components, increased upper airway dimension, and acceptable facial esthetics (Fig. 7.7).

By combining the pathophysiologic mechanisms of ICR/PCR with the reported outcomes of past surgical procedures for treating that condition, the frequent failure of those procedures is now understood much better. Since it is unlikely that altering those traditional procedures will do much to change the predictability of outcomes, the use of an alloplastic joint replacement should be considered as a feasible choice. This is especially true if there has been skeletal relapse of an ICR case managed by either orthognathic or autogenous reconstruction surgery.

While the track record for TMJ TJR is shorter than that for other joints, the same general principles apply. In cases of ICR/PCR, orthodontists and surgeons should consider utilizing the technologic advances made by patient-fitted (custom) TMJ TJR device design and load-bearing materials over what can be achieved with autogenous tissue or orthognathic surgery alone.

7.12 Management of the ICR/PCR Patient and Medico-legal Implications

We are aware of a few cases of patients who developed ICR/PCR during orthodontic treatment, and subsequently the families of those patients who have brought legal action against the orthodontist. These patients developed severe malocclusions while they were wearing active orthodontic appliances, and in addition they had TMJ pain and would require future orthognathic (and possibly TMJ) surgery. The allegations in

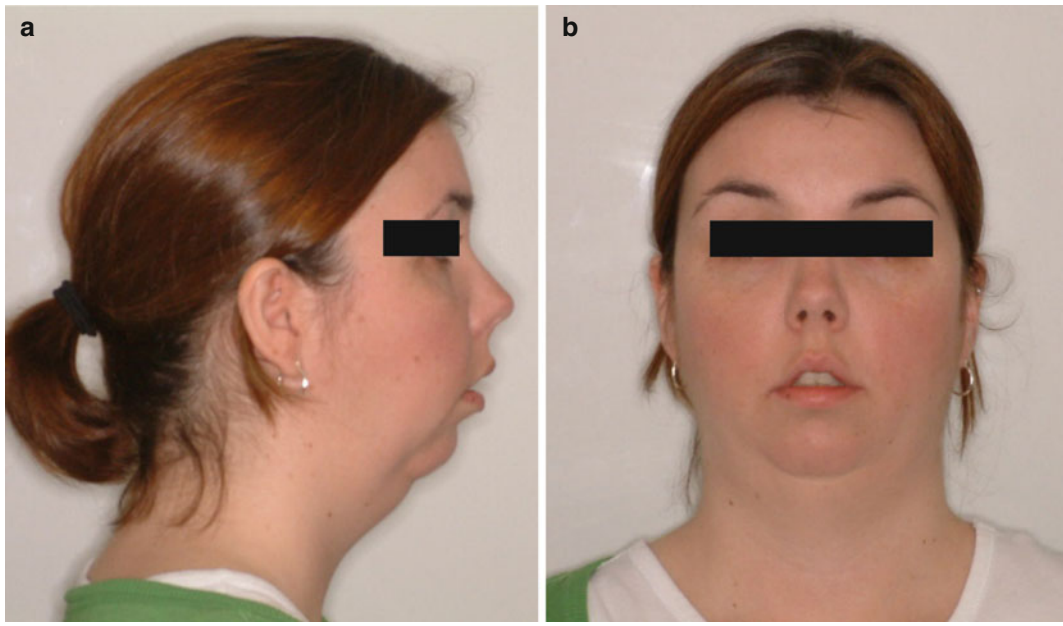


Fig. 7.6 TC presurgical 2009. (**a**) Right lateral profile. (**b**) Anterior profile. (**c**) Right cone beam image. (**d**) Cephalometric analysis. (**e**) Orthopantomogram. (**f**) Right occlusion. (**g**) Left occlusion

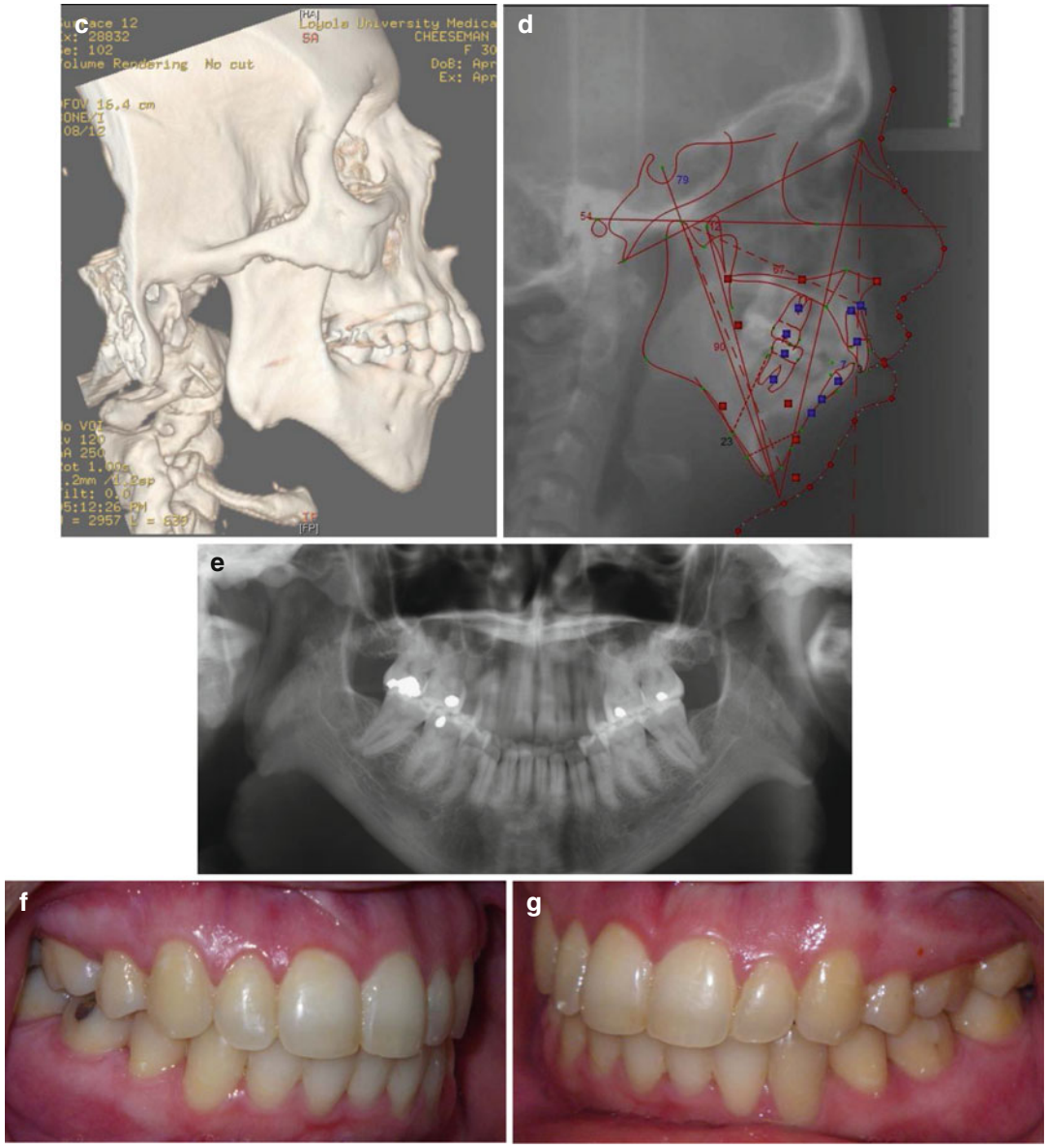


Fig. 7.6 (continued)

those lawsuits were failure to diagnose, failure to advise the family of the risks of orthodontic treatment in the informed consent procedures, and failure to discontinue treatment when the diagnosis of ICR/PCR could have or should have been made. Obviously, these cases raise questions about what kind of informed consent should be provided to prospective orthodontic patients.

Even though ICR/PCR is a serious disease, it is not medico-legally required to inform every patient that ICR/PCR may occur prior to starting routine orthodontic treatment, since its incidence is so rare – about one case per 5,000 patients [2]. Once the orthodontist is suspicious that ICR/PCR may be present however, a discussion of the risks of continued orthodontic treatment becomes mandatory. This is especially critical if orthognathic

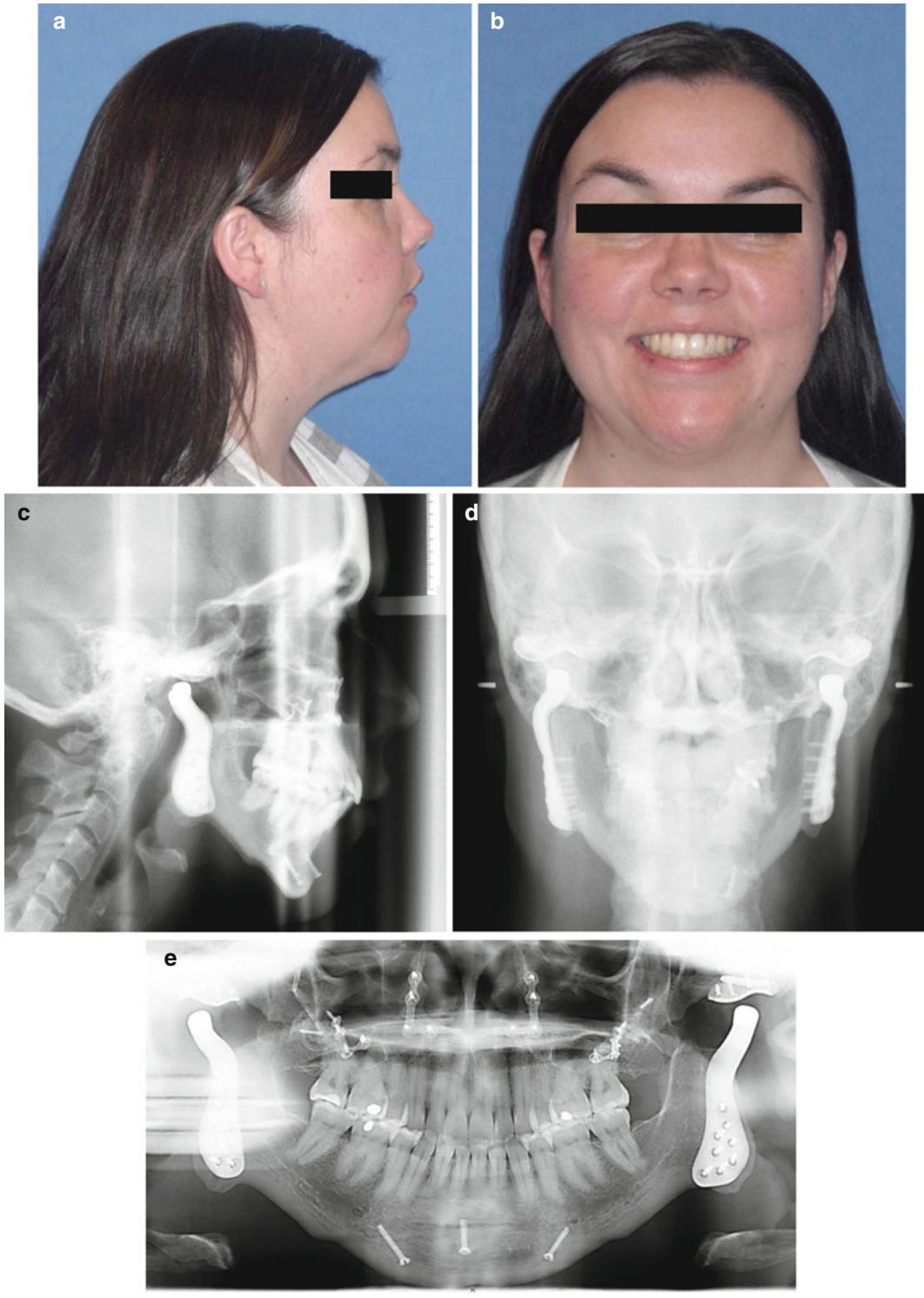


Fig. 7.7 TC postsurgical 2014. (**a**) Right lateral profile. (**b**) Anterior profile. (**c**) Lateral cephalometric image.* (**d**) Anterior-posterior cephalometric image.* (**e**) Orthopantomogram.* (**f**) Right occlusion. (**g**) Left occlusion. (**h**) Incisal opening. *Note that the articulating component of the TMJ Concepts (Ventura, CA) patient-fitted TJR fossa is composed of ultra-high molecular weight polyethylene, therefore radiolucent

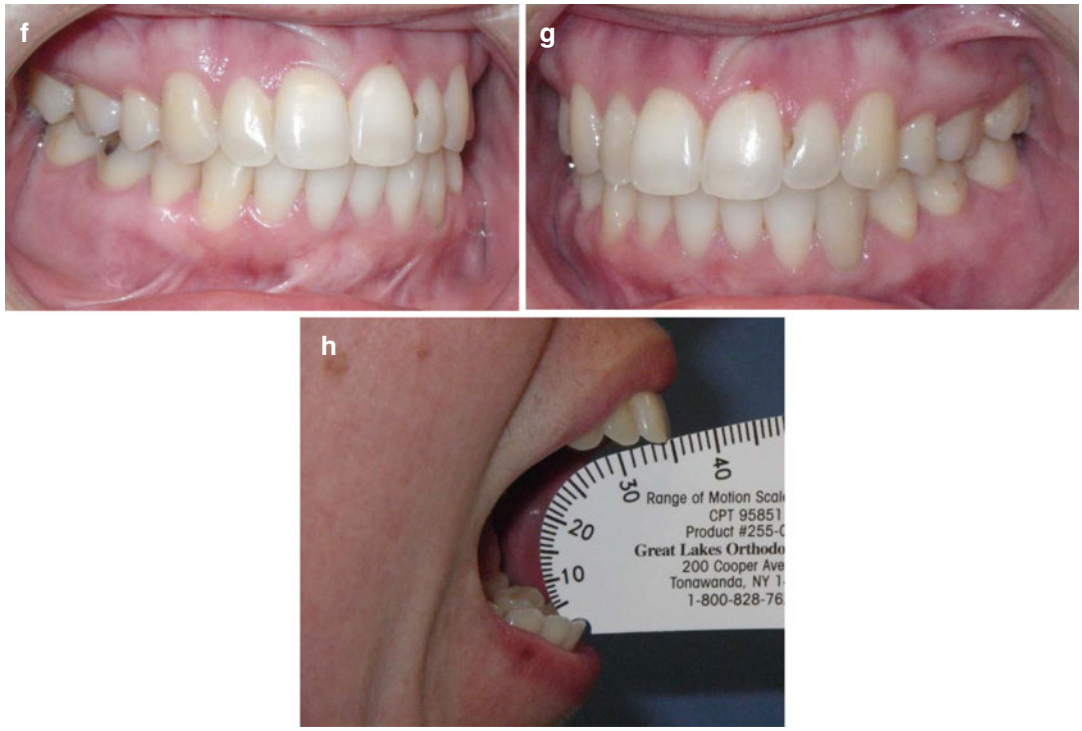


Fig. 7.7 (continued)

surgery is contemplated. The incidence of ICR/PCR for all surgery patients is between 1.2 and 5 % and this rises to between 20 and 25 % for the Class II high angle patient [3, 16–19].

If patients (especially females) either present with or develop any of the features of ICR/PCR while you are treating them, some very specific procedures should be considered. The clinical features to watch for include various combinations of divergent mandible, mandibular retrognathia, short ramus, open bite, and TMJ distress. If these are present, the patient and family should be informed that ICR/PCR is a possible diagnosis. All discussions should be summarized in the patient's record and dated. Suspicion of ICR/PCR requires that a history should be taken followed by certain clinical procedures. Study models should either be mounted or stabilized with an appropriate bite registration to record the open bite. Obviously, cephalometric x-rays should be taken both before and during treatment, and the superimpositions should be analyzed. The OPG may be suggestive of ICR, but for a more detailed record of the bony/joint architecture, CBCT may be indicated [33, 34].

When confronted with the possibility that your patient has ICR/PCR, the orthodontist should rely on collaborating with a team of experts:

- An oral maxillo-facial radiologist (to submit a written report on the CBCT scans)
- An oral maxillo-facial surgeon with extensive experience with orthognathic surgery, ideally with experience in dealing with ICR/PCR cases
- An oro-facial pain specialist
- A rheumatologist to rule out autoimmune and rheumatoid diseases

The record taking, the consultations and the discussions should educate the patient and family that this is not a problem limited to "crooked teeth," but instead the patient may have a medical problem involving the pathologic loss of tissue, possible pain and dysfunction, and abnormal occlusal and skeletal morphology of a serious nature.

What should the orthodontist do when the diagnosis of ICR is made and the patient is still in orthodontic appliances? The number-one rule that has emerged from both clinical and medico-legal experience is this: *discontinue treatment and retain*. Continue to then monitor the patient every 6 months and take annual cephalometric x-rays and CBCT scans and construct an occlusal splint. All of this should be done in collaboration with your professional colleagues who will also be involved with this case. Once the condyle has healed and the occlusion is stable, the discussion of orthodontic treatment alone or the available surgical options should be conducted with all parties, with the patient being a key part of the decision-making process. Both emphatic communication "that you really care" and careful record keeping will minimize the chance of any legal ramifications/liability.

Take Home Messages

- Idiopathic condylar resorption (ICR) is a relatively rare condition involving mainly female adolescents.
- Loss of condylar mass results in an open bite, Class II malocclusion, shortened mandibular condyle and ramus, and the clockwise rotation of the mandible.
- ICR/PCR may occur prior to, during, or postorthodontic treatment; and can be seen after orthognathic surgical procedures.
- It is believed a condyle affected by ICR/PCR has a diminished adaptive capacity to withstand increased or even normal mandibular mechanical loading demands.
- The pathology of ICR/PCR involves resorption of the outer dense cortical layer of the condyle, narrowing and shortening of the condyle, and flattening of the anterior and/or superior articular surface.
- A survey of orthodontists indicated an incidence of ICR/PCR of approximately 1 in 5,000 patients or about 1–2 in each orthodontic practice. 30.5 % of the patients complained of facial pain . 82.7 % of the cases were reported to be bilateral.
- Diagnosis requires taking a careful history with special focus on sudden change in occlusion resulting in an open bite and/or Cl II. Rheumatoid serology and estrogen levels should be evaluated. Irregular menstrual cycles and use of oral contraceptives may be associated with condylar resorption.
- Serial cephalometric imaging superimposing on Nasion-Basion at Basion will show that as the ramus shortens, articulare moves forward. CBCT scans should be made in open and closed condylar position.
- Consultation with an experienced oral and maxillofacial surgeon familiar with ICR/PCR cases should be obtained when orthognathic surgery is deemed an option.
- A full coverage occlusal splint should be prescribed for use over 6 months to both decrease the mandibular mechanical loading forces on the TMJ, and to test if the anterior occlusion on the splint remains stable. If the incisors move off the splint, the disease is still active.
- If ICR/PCR occurs during orthodontic treatment, it is best to remove the appliances and monitor the condition clinically and with images until the condyle heals.
- Reactivation of ICR can develop following orthognathic surgery. It is best to wait until the mid 20s where the disease may be in remission, but this may not always occur.
- The ICR patient with a severe malocclusion and facial deformity has three choices after it has been determined that the resorptive process has stopped.
  1. No treatment or minor orthodontic alignment of the anterior teeth and acceptance of the malocclusion and facial deformity.

2. Orthognathic surgery, ideally following the detailed surgical protocol proposed by Gunson and Arnett which also includes the use of occlusal splints to reduce joint loading and a comprehensive series of medications to control the resorption process.
3. Placement of a total joint prosthesis as part of orthognathic surgical correction. These devices will not be affected by the disease process and they can bear the increased functional and mechanical loading that result from the large surgical movements required to correct the occlusion and establish more ideal facial aesthetics.

References

1. Handelman CS. Ask us: condylar resorption. Am J Orthod Dentofacial Orthop. 2004;125:16A.
2. Handelman CS, Greene CS. Progressive/idiopathic condylar resorption: an orthodontic perspective. Semin Orthod. 2013;19:55–70.
3. Hoppenreijs TJM, Stoelinga PJW, Grace KL, et al. Long-term evaluation of patients with progressive condylar resorption following orthognathic surgery. Int J Oral Maxillofac Surg. 1999;28:411–8.
4. Kobayashi T, Izumi N, Kojima T, et al. Progressive condylar resorption after mandibular advancement. Br J Oral Maxillofac Surg. 2012;50:176–80.
5. Sarver D, Janyavula S. Condylar degeneration and diseases – local and systemic etiologies. Semin Ortho. 2013;19:89–96.
6. Osterberg T, Carlsson GE. Symptoms and signs of mandibular dysfunction in 70 year old men and women in Gothenburg, Sweden. Community Dent Oral Epidemiol. 1973;7:315–21.
7. Gunson MJ, Arnett GW, Formby B, et al. Oral contraceptive pill use and abnormal menstrual cycles in women with severe condylar resorption: a case for low serum 17 beta-estradiol as a major factor in progressive condylar resorption. Am J Orthod Dentofacial Orthop. 2009;136:772–9.
8. Milam SB. TMJ osteoarthritis. In: Laskin DM, Greene CS, Hylander WL, editors. Tempomandibular disorders: an evidence-based approach to diagnosis and treatment. Chicago: Quintessence; 2006. p. 105–23.
9. Stegenga B, De Bont LGM, Boering G, et al. Tissue responses to degenerative changes in the temporomandibular joint: a review. J Oral Maxillofac Surg. 1991;49:1079–88.
10. Arnett GW, Milam SB, Gottesman L. Progressive mandibular retrusion-idiopathic condylar resorption. Part I. Am J Orthod Dentiofacial Orthop. 1996;110:8–15.
11. Haskin CL, Milam SB, Cameron IL. Pathogensis of degenerative joint disease in the human temporomandibular joint. Crit Rev Oral Biol Med. 1995;6:248–77.
12. Scheerlink JPO, Stoelinga PJW, Blijdorp PA, et al. Sagittal split advancement osteotomies stabilized with miniplates: 2–5 years follow-up. Int J Oral Maxillofac Surg. 1994;23:127–31.
13. Arnett GW, Milam SB, Gottesman L. Progressive mandibular retrusion idiopathic condylar resorption, Part II. Am J Orthodont Dentofacial Orthop. 1996; 110:117–27.
14. Boering G. Arthrosis deformans van het kaakgewricht. Een klinisch en rontgenobgrach onderzoek. Thesis University of Groningen: Van Denderen; 1966 [In Dutch]
15. Kerstens HC, Turnizing DB, Golding RP, et al. Condylar atrophy and osteoarthrosis after bimaxillary surgery. Oral Surg Oral Med Oral Pathol. 1990;69:274–80.
16. Moore KE, Gooris PJ, Stoelinga PJ. The contributing role of condylar resorption to skeletal relapse following mandibular advancement surgery: report of five cases. J Oral Maxillofac Surg. 1991;49:448–60.
17. Bouwman JP, Kerstens HC, Tanzing DB. Condylar resorption in orthognathic surgery. The role of intermaxillary fixation. Oral Surg Oral Med Oral Pathol. 1994;78:138–41.
18. Merkx MAW, Van Damme PA. Condylar resorption after orthognathic surgery. Evaluation of treatment in 8 patients. J Craniomaxillofac Surg. 1994;22:53–8.
19. Hwang SJ, Haers PE, Seifert B, et al. Non-surgical risk factors for condylar resorption after orthognathic surgery. J Craniomaxillofac Surg. 2004;32:103–11.
20. De Clercq CAS, Neyt LF, Mommaerts MY, et al. Condylar resorption in orthognathic surgery: a retrospective study. Int J Adult Orthodon Orthognath Surg. 1994;9:233–40.
21. Hoppenreijs TJM, Freihofer HPM, Stoelinga PJW, et al. Condylar remodeling after LeFort I and bimaxillary osteotomies in patients with anterior open bite. A clinical and radiological study. Int J Oral Maxillofac Surg. 1998;27:81–91.
22. Miguel JA, Turvey TA, Phillips C. Long –term stability of two-jaw surgery for treatment of mandibular deficiency and vertical maxillary excess. Int J Adult Orthodon Orthognath Surg. 1995;10:235–45.
23. Arnett GW, Tumborello JA. Progressive class II development: female idiopathic condylar resorption. Oral Maxillofac Surg Clin North Am. 1990;2: 699–716.
24. Crawford JG, Stoelinga PJW, Blijdorp PA, et al. Stability after reoperation for progressive condylar resorption after orthognathic surgery: report of seven cases. J Oral Maxillofac Surg. 1994;52:460–6.

25. Huang YL, Pogrel MA, Kaban LB. Diagnosis and management of condylar resorption. J Oral Maxillofac Surg. 1997;55:114–9.
26. Hatcher DC. Progressive condylar resorption: pathologic processes and imaging considerations. Semin Orthod. 2013;19:97–105.
27. Osborn JW. The disc of the human temporomandibular joint: design, function and failure. J Oral Rehabil. 1985;12:279–93.
28. Scapino PR. Histopathology associated with malposition of the human temporomandibular joint disc. Oral Surg Oral Med Oral Pathol. 1983;55:382–97.
29. Nebbe B, Major PW, Prasad NGN. Adolescent female craniofacial morphology associated with advanced bilateral TMJ disc displacement. Eur J Orthod. 1998; 20:701–12.
30. Link JJ, Nickerson JW. Temporomandibular joint internal derangements in an orthognathic surgery population. Int J Adult Orthodon Orthognath Surg. 1992; 7:161–9.
31. Wolford LM, Cardenas L. Idiopathic condylar resorption: diagnosis, treatment protocol and outcomes. Am J Orthod Dentofacial Orthop. 1999;116:667–77.
32. Dolwick MF. Intra-articular disc displacement, part I: its questionable role in temporomandibular joint pathology. J Oral Maxillafac Surg. 1995;53:1069–72.
33. Hoppenreijs TJM, Maal T, Tong Y. Evaluation of condylar resorption before and after orthognathic surgery. Semin Orthod. 2013;19:106–15.
34. Hatcher DC, McEvoy SP, Mah RT, et al. Distribution of local and general stresses in the stomatognathic system. In: McNeill C, editor. Science and practice of occlusion. Chicago: Quintessence; 1997. p. 259–70.
35. Progrel MA, Chigurupati R. Management of idiopathic condylar resorption. In: Laskin DM, Greene CS, Hylander WL, editors. Temporomandibular disorders: an evidence-based approach to diagnosis and treatment. Chicago: Quintessence; 2006. p. 105–23.
36. Arnett GW, Gunson MJ. Risk factors in the initiation of condylar resorption. Semin Orthod. 2013;19:81–8.
37. Dibbets J, Muller B, Krop F, et al. Deformed condyles and craniofacial growth: findings of the Groningen longitudinal temporomandibular disorders study. Semin Orthod. 2013;19:71–80.
38. Schendel SA, Tulasno J-F, Link III DW. Idiopathic condylar resorption and micrognathia: the case for distraction osteogenesis. J Oral Maxillofac Surg. 2007;65:1610–6.
39. Gunson MJ, Arnett GW, Milam SB. Pathophysiology and pharmacologic control of osseous mandibular condylar resorption. J Oral Maxillofac Surg. 2012;70: 1918–34.
40. Mercuri LG. A rationale for total alloplastic temporomandibular joint reconstruction in the management of idiopathic/progressive condylar resorption. J Oral Maxillofac Surg. 2007;65:1600–9.
41. Mercuri LG. Alloplastic total joint replacement: a management option in temporomandibular joint replacement. Semin Orthod. 2013;19:116–26.
42. Mercuri LG, Edibam NR, Giobbie-Hurder A. 14-Year follow-up of a patient fitted total temporomandibular joint reconstruction system. J Oral Maxillofac Surg. 2007;65:1140–8.
43. Wolford LM, Mercuri LG, Schneiderman ED, Movahed R, Allen W. Twenty-Year Follow-up Study on a Patient-Fitted Temporomandibular Joint Prosthesis: The Techmedica/TMJ Concepts Device. J Oral Maxillofac Surg 2015;73:952–60.
44. Mercuri LG. The use of alloplastic prostheses for temporomandibular joint reconstruction. J Oral Maxillofac Surg. 2000;58:70–5.
45. Mercuri LG. Chapter 52. End-stage TMD and TMJ reconstruction. In: Miloro M, Ghali G, Larsen P, Waite P, editors. Peterson's principles of oral & maxillofacial surgery. 3rd ed. Shelton, CT 2012. p. 1173–86.
46. Reitzik M. Cortex-to-cortex healing after mandibular osteotomy. J Oral Maxillofac Surg. 1983;41:658–63.
47. Lienau J, Schell H, Duda GN, et al. Initial vascularization and tissue differentiation are influenced by fixation stability. J Orthop Res. 2005;23:639–45.
48. Salter RB. Continuous passive motion. Baltimore: Williams and Wilkins; 1993.
49. Mercuri LG. Alloplastic TMJ replacement. Rationale for custom devices. Int J Oral Maxillofac Surg. 2012;41:1033–40.
50. Mercuri LG. The role of patient-fitted devices in total temporomandibular joint replacement. Rev Esp Cir Oral Maxillofacial. 2013;35:1–10.
51. Dela Coleta KE, Wolford LM, Gonçalves JR, et al. Maxillo-mandibular counter- clockwise rotation and mandibular advancement with TMJ Concepts total joint prostheses: part I–skeletal and dental stability. Int J Oral Maxillofac Surg. 2009;38:126–38.
52. Coleta KE, Wolford LM, Gonçalves JR, et al. Maxillomandibular counter- clockwise rotation and mandibular advancement with TMJ Concepts total joint prostheses: part II– airway changes and stability. Int J Oral Maxillofac Surg. 2009;38:228–35.
53. Coleta KE, Wolford LM, Gonçalves JR, et al. Maxillomandibular counter- clockwise rotation and mandibular advancement with TMJ Concepts total joint prostheses: part IV– soft tissue response. Int J Oral Maxillofac Surg. 2009;38:637–46.
54. Pinto LP, Wolford LM, Buschang PH, et al. Maxillomandibular counter- clockwise rotation and mandibular advancement with TMJ Concepts total joint prostheses: part III– pain and dysfunction outcomes. Int J Oral Maxillofac Surg. 2009;38:326–31.
55. Wolford LM, Cottrell DA, Henry CH. Temporomandibular joint reconstruction of the complex patient with the Techmedica custom-made total joint prosthesis. J Oral Maxillofac Surg. 1994;52:2–10.
56. Mercuri LG, Wolford LM, Sanders B, et al. Custom CAD/CAM total temporomandibular joint reconstruction system: preliminary multicenter report. J Oral Maxillofac Surg. 1995;53:106–15.
57. Mercuri LG. Considering total alloplastic temporomandibular joint replacement. Cranio. 1999;17: 44–8.

58. Mercuri LG. Subjective and objective outcomes in patients reconstructed with a custom-fitted alloplastic temporomandibular joint prosthesis. J Oral Maxillofac Surg. 1999;57:1427–30.
59. Donlon WC, editor. Total temporomandibular joint reconstruction. Oral Maxillofac Surg Clin N Am, vol 12. Philadelphia: Saunders; 2000.
60. Mercuri LG, Wolford LM, Sanders B, et al. Long-term follow-up of the CAD/CAM patient fitted alloplastic total temporomandibular joint reconstruction prosthesis. J Oral Maxillofac Surg. 2002;60:1440–8.
61. Wolford LM, Dingworth DJ, Talwar RM, et al. Comparison of 2 temporomandibular joint prosthesis systems. J Oral Maxillofac Surg. 2003;61:685–90.
62. Mercuri LG, Giobbe-Hurder A. Long-term outcomes after total alloplastic temporomandibular joint reconstruction following exposure to failed materials. J Oral Maxillofac Surg. 2004;62:1088–96.
63. Mercuri LG, Swift JQ. Considerations for the use of alloplastic temporomandibular joint replacement in the growing patient. J Oral Maxillofac Surg. 2009;67:1979–90.
64. Salvati E, Wilson P, Jolley MA. A ten-year follow-up study of our first one hundred consecutive Charnley total hip replacements. J Bone Joint Surg. 1981;63A:753–76.
65. Schulte KR, Callaghan JJ, Kelley SS, et al. The outcome of Charnley total hip arthroplasty with cement after a minimum of twenty-year follow-up. The results of one surgeon. J Bone Joint Surg. 1993;75A:961–75.

8 Management of TMD Signs and Symptoms in the Orthodontic Practice

Charles S. Greene, Donald J. Rinchuse, Sanjivan Kandasamy, and John W. Stockstill

Like all other dentists, orthodontists are likely to encounter some patients with TMD signs and symptoms in their practices that require some form of professional treatment. These patients may come into their office as referrals from other dentists, or they may develop TMD problems while under the orthodontist's care. While some benign TMD signs and symptoms may be present in new patients, or may arise in patients under treatment, not all of these need to be treated (see Chap. 3). However, as discussed in Chap. 2, there are a number of significant TMD conditions that need to be properly diagnosed and appropriately treated.

The topic of TMD treatment can be quite complex, and indeed whole books have been devoted to that subject. In this chapter, we will try to present a commonsense approach to TMD management that a practicing orthodontist can readily incorporate into his or her practice. As repeatedly mentioned in earlier chapters, the emphasis today is on conservative treatment for the vast majority of TMD conditions, especially during the early and acute stages. Also, since orthodontic procedures per se are not generally indicated as treatment modalities for TMD patients, this chapter will not include any discussion about those approaches.

8.1 General Considerations

The two major clinical features of most temporomandibular disorders are pain and dysfunction. While other signs and symptoms may also be present and require attention, these two are the main reason most people seek professional care. As Lund and others have pointed out [1, 2], the dysfunction usually is a consequence of the pain rather than its cause, so primary therapeutic attention should be directed at the pain. When pain is relieved, improved function can be anticipated. If an orthodontist needs to provide a patient with basic TMD treatment, i.e., conservative management of their pain and dysfunction, then the therapies administered should be supported by science and evidence.

C.S. Greene, BS, DDS (✉)
Department of Orthodontics, University of Illinois at Chicago, College of Dentistry, Chicago, IL, USA
e-mail: cgreene@uic.edu

D.J. Rinchuse, DMD, MS, MDS, PhD
Private Practice, Greensburg, PA, USA

S. Kandasamy, BDSc, DClinDent, MOrthRCS, FRACDS
Department of Orthodontics, School of Dentistry, University of Western Australia, Nedlands, WA, Australia

Centre for Advanced Dental Education, Saint Louis University, Saint Louis, MO, USA

Private Practice, Midland, WA, Australia

J.W. Stockstill, DDS, MS
Department of Orthodontics, Temporomandibular Disorders/Orofacial Pain, Seton Hill University, Center for Orthodontics, 2900 Seminary Drive, Building E, Greensburg, PA 15601, USA

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_8

TMD treatments are now based on a biopsychosocial model rather than the historical, dental-based model [3–5]. That is, the field of TMD management has moved away from treatments related to conventional dentistry *via* altering the occlusion and realigning jaw relationships to treatments based on the biomedical and psychosocial sciences. The contemporary biopsychosocial model attempts to integrate the host of biologic, clinical, and behavioral factors that may account for the onset, maintenance, and remission of TMD [6]. The factors that are receiving the most attention and research in the understanding of TMD today are genetics (vulnerabilities related to pain), imaging of the pain-involved brain, endocrinology, behavioral risk factors, sexual dimorphism, and psychosocial traits and states [7]. There also is considerable interest in the issue of comorbid pain conditions, which are found in a large number of TMD patients, and a significant amount of research is focused on the problem of chronicity (who is at risk, and why?).

TMD signs and symptoms can develop in any individual at any time. In many cases, these can be transient phenomena like a sore jaw muscle, a painful joint following a minor trauma, or limited opening after a dental appointment. Also, many patients observed during a screening exam (see Chap. 3) may have occasional jaw pain, or a painless TMJ click or an odd opening and closing pattern, but these do not rise to the level of being a clinical case of TMD. When actual TMD problems do arise, that group of patients is often in their mid to late teens or they are young or middle-aged adults rather than children and the elderly. Prevalence of TMDs in women is twice more common than in men [8]. Based on these facts, the likelihood of an orthodontic patient developing TMD signs or symptoms before, during, or after treatment is definitely a possibility. It has been shown that over the long term most cases of TMD pain and dysfunction generally tend to resolve or improve [9, 10]. This however does not obviate an orthodontist's professional obligation to recognize TMD problems when they do arise, to inform and educate those patients about the conservative treatment protocols, and if needed either engage in the treatment or effect the necessary referral to an appropriate specialist to manage the patient's TMD.

There are conservative and reversible TMD treatments that orthodontists can provide for patients, or at least understand their use by other practitioners. These include patient self-directed care, physical therapies, cognitive-behavioral therapies, biofeedback, pharmacologic agents, and oral occlusal appliances [11].

It is important to understand that TMDs are generally cyclic in nature, so symptoms often gradually progress from mild to moderate to severe, and then they can move toward a downward phase which ends up as mild to no symptoms. Therefore, practitioners may provide some form of treatment during the downward side of the cycle and get symptom relief. The practitioner may then incorrectly assume that the treatment rendered was responsible for this symptom improvement, but in fact it is possible that the patient was getting better on his or her own due to the cyclic nature of TMD [12, 13].

8.2 Patient Self-Directed Care and Education

It is well known that patients experiencing TMD-related pain and dysfunction frequently are anxious about what is happening to them, especially if they have been led to believe that they have a structural problem requiring irreversible treatment procedures. Assuming that a preliminary diagnosis of some type of TMD has been established, it is important for the orthodontist to reduce that anxiety by communication with the patient. For this phase of interaction to proceed smoothly, however, the orthodontist must be knowledgeable about current concepts of TMD. Whereas previous concepts about these disorders have included a heavy emphasis on structural mal-alignments and functional bite disharmonies, most modern authorities regard TMD problems as benign musculoskeletal conditions that are likely to be addressed successfully by simple and reversible measures [14]. Therefore, it is not necessary to provide extensive (and oftentimes expensive) structural corrections in most cases. Long-term studies have shown that 80–90 % of these patients can expect good

short-term results with little or no long-term problems after conservative orthopedic therapy to reduce pain and restore normal function [15, 16].

The orthodontist who is aware of these positive data can easily allay the anxiety of TMD patients by reassuring the patients that:

1. Most TM problems are extracapsular (myofascial) rather than intra-capsular (derangement and/or arthritis). Therefore, they can be expected to respond to the same kinds of conservative muscle treatment modalities that are used elsewhere in the body (e.g., for lower back pain, sore shoulder, and others).
2. Even when intracapsular changes have occurred (such as a displaced disk or degenerative changes), a good response to conservative treatment is likely [17]. For the few patients who require actual treatment inside the joint, there often are simpler measures available today (arthrocentesis or arthroscopy, see Chap. 9) rather than the traditional open-joint surgical operations.
3. Neither of these conditions will require irreversible treatment procedures in most cases.

While nobody can guarantee that things will work out easily and favorably for any individual patient, it is nice to be able to say that very positive treatment outcomes often will result when appropriate and conservative measures are taken. Some studies have reported that patients informed of this prognosis sometimes get better without any professional treatment simply because they were relieved by the explanation, went home and let some time pass [15].

8.3 Home Care Instructions

Patient self-directed care for TMD includes actions that the patient can take to limit jaw function and parafunctional activities. Relying once again on the orthopedic medicine analogy, the orthodontist should advise and instruct the patient on home care practices [13]. Patients should limit or stop such activities as chewing gum, yawning, yelling, singing, cheerleading, and so on. They can support their mandible to limit opening when yawning, and they should avoid unnecessary clicking maneuvers. In addition, patients should maintain good posture by following good orthopedic practices. This may involve using a headset when speaking on the phone, making sure their workstation is properly "fitted" for the body allowing for adequate posture, keeping their head in a neutral position while sitting and using an orthopedic pillow at night. For acute symptoms which include limited mandibular opening, patients can perform "scissor jaw" exercises with their fingers or a clothespin. Depending on their signs and symptoms, they can be advised to temporarily change their diet as follows: eat soft foods; avoid hard or chewy foods; avoid wide opening during meals; and grind or finely chop meats and other tough foods.

Also, TMD patients should be advised to relax their jaws and keep the teeth apart. Because stress and tension often are associated with musculoskeletal pain, patients should be informed about this connection, and instructed about relaxation procedures that can be practiced at home [17]. In more complex cases, professional help in this area may be required from psychologists or stress management specialists (see Chap. 4). Home physical therapy procedures can be taught, such as using ice for acute pain and heat for more chronic pain. Self-massage and jaw manipulation (controlled exercises) can be encouraged, hopefully supplemented with a printed instruction sheet to improve patient compliance. In general, hot showers, saunas, or steam baths are known to be helpful for dealing with all types of musculoskeletal pain.

Finally, non-prescription medications taken continuously around the clock for 2–4 weeks may be very effective in breaking a cycle of pain and inflammation. The most likely ones are acetaminophen or non-steroidal anti-inflammatory drugs (NSAIDs) including aspirin; the NSAIDs should be taken with stomach acid-prevention products. For acute-onset pain, especially if related to some form of trauma, a tapering dosage of steroids can be prescribed over a 5–7 day period. Other medications requiring a prescription are beyond the scope of this chapter.

8.4 Psychological Approaches to Treatment

While stress and tension may play a role in the onset or continuance of TMD symptoms, these patients do not necessarily need to be referred to a professional psychologist. Instead, consideration should be given to using a simpler method referred to as cognitive behavioral therapy (CBT) [18] which can be used by all types of medical providers. The purpose of cognitive behavioral therapies is to increase the patient's awareness about the mind-body connection through education about stress management and the body's reaction to stress. Studies have shown that compliant patients can be taught about relaxation techniques, use of distraction and pleasant activity scheduling, cognitive restructuring, self-instructional training, and various maintenance skills. However, some patients may need a referral for professional counseling, which also can include the use of hypnosis, biofeedback, guided imagery training, etc. Patients often are given workbooks with reading assignments, as well as homework assignments. See Chapter 4 for more detailed information regarding psychological considerations in the etiology of various TMD conditions and in the management of TMD patients.

8.5 Oral Appliances (Splints)

Most orthodontists feel very comfortable about prescribing and using splints for the management of both bruxism and TMD patients. However, they may not all realize that splints are capable of being both the best treatment modality and the worst thing ever to appear in the TMD field. In one form or another, splints have been around for more than 75 years, and certainly many thousands of patients have been helped by their use. Unfortunately, the potential for serious negative outcomes is very high for splints, because they can produce irreversible occlusal and jaw position changes, altered vertical dimension, major dentoalveolar discrepancies, and extreme dependency. This occurs when they are either designed improperly or worn full time for extended periods or both. In some cases, these outcomes are worse than a bad surgical result.

In some circles, these changes were deemed to be desirable because they meant that the purported "wrong" jaw/occlusal relationships had been "corrected." By describing an oral appliance as a deprogrammer, or a centric splint, or a neuromuscular splint, the proponents of such devices are saying that they expect to obtain a more ideal jaw position by utilizing splint therapy. Proponents of this viewpoint generally speak of Phase I and Phase II treatments, with the splint being Phase I, and then either occlusal equilibration or more extensive occlusion-changing treatments (orthodontics, orthognathic surgery, or full mouth reconstruction) being described as Phase II "stabilization" procedures [19, 20].

A more reasonable interpretation of these events, however, would be to call that Phase I approach "unnecessary iatrogenic change," with Phase II being required to fix the occlusal problems created by improper splint design and usage [21]. A tremendous number of studies from around the world have shown that this mechanistic approach to TMD therapy (which obviously is quite invasive, expensive, and irreversible) is also generally unnecessary, because patients simply can get well without it. Therefore, most modern authorities recommend splint therapy as a temporary orthopedic modality, with the therapeutic goals being relaxation of muscles, reduction of oral habits, altering joint loading, and general relief of symptoms [22–24].

A proper rule of thumb for the use of oral appliances is: "Do no harm," which translates in dental terms to: "Produce no irreversible changes." At worst, the failure to respond positively to splint therapy should be the only downside risk. Even if prolonged splint wear is required (e.g., to control nocturnal bruxism or to treat recurrent symptoms), no irreversible occlusal changes or alterations of TMJ relationships should occur. The most important aspect of this conservative viewpoint is avoiding any protocols involving 24-hour wearing of splints. With rare exceptions, the proper protocol for an oral appliance is nighttime usage only, so that normal occlusal relationships can be maintained in the daytime.

Take Home Messages

- The contemporary approach to TMD treatment is centered on conservative and reversible therapies for the vast majority of TMD conditions, especially during the early and acute stages.
- Orthodontic procedures per se are not generally indicated as treatment modalities for TMD patients.
- Communicating with the patients about their TMD issues is the first step in the effective management of these conditions; this step plays an important role in reducing their anxiety, improving their compliance with treatment, and in some cases improving their symptoms without professional treatment.
- Patient self-directed care for TMD includes actions that the patients can take to limit jaw function. This may include supporting their jaws in function, limiting jaw movement, soft diet, massage, relaxation, stress management, and medication to reduce the pain and/or inflammation.
- Cognitive behavioral therapy is a highly effective modality in facilitating stress reduction and enhancing self-management. This involves educating the patient about the mind-body connection and the body's reaction to stress, and equipping the patients with techniques and skills to reduce both their stress and their symptoms.
- Oral appliances have been shown to be effective in some TMD patients. The key to effective splint therapy is its short term use as well as nighttime wear rather than 24 hour application. Also, no irreversible occlusal changes or alterations of TMJ relationships should occur following splint wear.

References

1. Lund JP, Donga R, Widmer CG, Stohler CS. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol. 1991;69:683–94.
2. Murray GM, Peck CC. Orofacial pain and jaw muscle activity: a new model. J Orofac Pain. 2007;21:263–78; discussion 279–88.
3. Greene CS. The etiology of temporomandibular disorders: implications for treatment. J Orofac Pain. 2001; 15:93–105.
4. Rinchuse DJ, Kandasamy S. Chapter 29. Orthodontics and TMD management. In: Manfredini D, editor. Current concepts on temporomandibular disorders. Chicago: Quintessence Publishing; 2010. p. 429–46.
5. Suvinen TI, Reade PC, Kemppainen P, Könönen M, Dworkin SF. Review of aetiological concepts of temporomandibular pain disorders: towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. Eur J Pain. 2005;9:613–33.
6. Dougall AL, Jimenez CA, Haggard RA, Stowell AW, Riggs RR, Gatchel RJ. Biopsychosocial factors associated with the subcategories of acute temporomandibular joint disorders. J Orofac Pain. 2012;26:7–16.
7. Slade GD, Fillingim RB, Sanders AE, Bair E, Greenspan JD, Ohrbach R, Dubner R, Diatchenko L, Smith SB, Knott C, Maixner W. Summary of findings from the OPPERA prospective cohort study of incidence of first-onset temporomandibular disorder: implications and future directions. J Pain. 2013; 14(12 Suppl):T116–24.
8. American Academy of Orofacial Pain. Diagnosis and management of TMDs. In: De Leeuw R, Klasser GD, editors. Orofacial pain: guidelines for assessment, diagnosis, and management. 5th ed. Chicago: Quintessence; 2013. p. 130.
9. Mohlin BO, Derweduwen K, Pilley R, Kingdon A, Shaw WC, Kenealy P. Malocclusion and temporomandibular disorder: a comparison of adolescents with moderate to severe dysfunction with those without signs and symptoms of temporomandibular disorders and their further development to 30 years of age. Angle Orthod. 2004;74:319–27.
10. De Kanter RJ, Truin GJ, Burgerdijk RC, Van 't Hop MA, Battistuzzi PG, Kalsbeek H, et al. Prevalence in the Dutch adult population and a meta-analysis of signs and symptoms of temporomandibular disorder. J Dent Res. 1993;72:1509–18.
11. Greene CS. Concepts of TMD etiology: effects on diagnosis and treatment. In: Laskin DM, Greene CS, Hylander WL, editors. TMDs – an evidence-based approach to diagnosis and treatment. Chicago: Quintessence Publ. Co; 2006. p. 219–28.

12. Skeppar J, Niilner M. Treatment of craniomandibular disorders in children and young adults. J Orofac Pain. 1993;7:362–9.
13. Garefis P, Grigoriadou E, Zarif A, Koidis PT. Effectiveness of conservative treatment for craniomandibular disorders: a two-year longitudinal study. J Orofac Pain. 1994;8:309–14.
14. Murakami K, Kaneshita S, Kanoh C, Yamamura I. Ten-year outcome of nonsurgical treatment for the internal derangement of the temporomandibular joint with closed lock. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;94:572–5.
15. Yatani H, Kaneshima T, Kuboki T, Yoshimoto A, Matsuka Y, Yamashita A. Long-term follow-up study on drop-out TMD patients with self-administered questionnaires. J Orofac Pain. 1997;11:258–69.
16. Michelotti A, de Wijer A, Steenks MH, Farella M. Home-exercise regimens for the management of non-specific temporomandibular disorders. J Oral Rehabil. 2005;32:779–85.
17. Ohrbach R. Biobehavioral therapy. In: Laskin DM, Greene CS, Hylander WL, editors. TMDs: an evidence based approach to diagnosis and treatment. Chicago: Quintessence; 2006. p. 391–403.
18. Oakley ME, McCreary CP, Clark GT, Holston S, Glover D, Kashima K. A cognitive-behavioral approach to temporomandibular disorder treatment failures: a controlled comparison. J Orofac Pain. 1994;8: 397–401.
19. Gelb ML, Gelb H. Gelb appliance: mandibular orthopedic repositioning therapy. Cranio Clin Int. 1991;1: 81–98.
20. Simmons HC. Temporomandibular joint orthopedics with anterior repositioning appliance therapy and therapeutic injections. J Calif Dent Assoc. 2014;42: 537–47.
21. Greene CS. Managing TMD, patients: initial therapy is the key. J Am Dent Assoc. 1992;123:43–5.
22. Turp JC, Komie F, Hugger A. Efficacy of stabilization splints for the management of patients with masticatory muscle pain: a qualitative systematic review. Clin Oral Investig. 2004;8:179–95.
23. Schmitter M, Zahran M, Duc MJ, Henschel V, Rammelsberg P. Conservative therapy in patients with anterior disc displacement without reduction using 2 common splints: a randomized clinical trial. J Oral Maxillofac Surg. 2005;63:1295–303.
24. 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.

9 Surgical Management of Temporomandibular Joint Problems

D.M. Laskin

It is generally recommended that medical management should be the first-line approach to treating most diseases and disorders of the temporomandibular joint (TMJ). However, surgery can play a role when medical means of management are either not indicated or when such treatments have failed. Although orthodontists are usually not directly involved in these forms of therapy, they do encounter patients with a wide variety of temporomandibular disorders (TMDs) in their practices and so it is important for them to understand what surgical treatments are available, when they are indicated, and what they are designed to accomplish. This chapter will focus on two major areas of concern to the orthodontist: surgical versus non-surgical management of internal derangements of the TMJ and the use of orthognathic surgery as a treatment for TMDs.

9.1 Surgical Management of Internal Derangements

Internal derangements of the TMJ can be divided into two categories – anteromedial disc displacement (commonly referred to as anterior disc displacement) with reduction to normal position on mouth opening (accompanied by a clicking or popping sound) and anterior disc displacement without reduction to normal position on mouth opening (locking) (see Chap. 2). The initial treatment of painful clicking or popping in the TMJ is always medical and involves such modalities as an analgesic for the pain, a soft non-chewy diet, and a bite appliance to control any parafunctional activity. The objective is to control the pain and not stop the clicking, because the disc generally can only be returned permanently to its normal position surgically. However, this is not recommended unless proper medical management fails to relieve the pain.

There is some evidence that patients with TMJ locking will also improve with medical management and jaw-stretching exercises [1, 2]. However, this can take a considerable time to occur and may not be successful in up to 36 % of such patients [3, 4]. Therefore, if these patients do not show decreased pain and improvement in mouth opening within a reasonable period, a different approach is indicated. At one time, the only answer was to open the joint and surgically replace the disc into its normal position (discoplasty) or to remove the disc (discectomy) if it was severely damaged and irreparable (Fig. 9.1). However, with the introduction of arthroscopic surgery all of this changed, because it is now possible to operate within the joint in a much less invasive manner. The arthroscopic approach allows the surgeon to lavage the joint and remove

D.M. Laskin, DDS, MS
Department of Oral and Maxillofacial Surgery,
Virginia Commonwealth University,
School of Dentistry, Richmond, VA, USA
e-mail: dmlaskin@vcu.edu

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_9

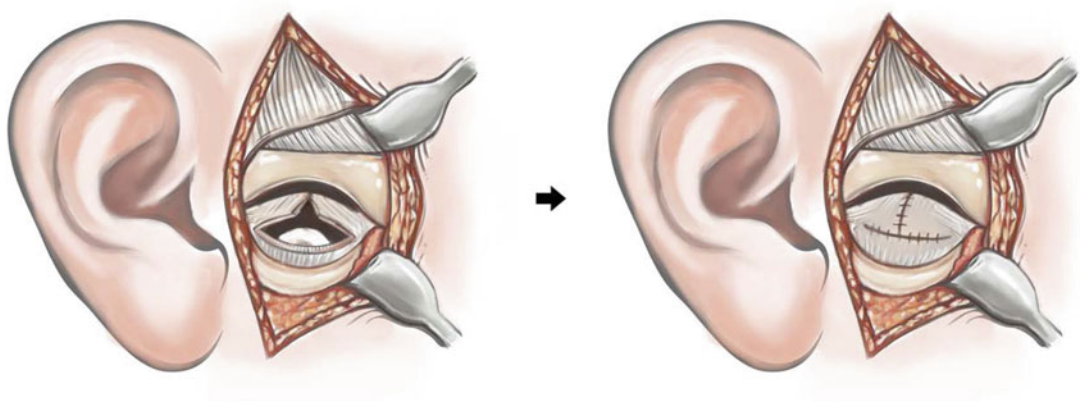


Fig. 9.1 Discoplasty for correcting an anteriorly displaced disc. After exposure of the joint and isolating the disc, a wedge of retrodiscal tissue is excised. When this area is sutured, it places the disc back into its normal anatomic position

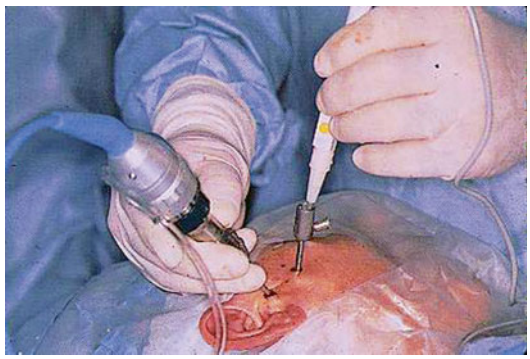


Fig. 9.2 Arthroscopy of the temporomandibular joint. The arthroscope, which has a camera attached to a TV monitor, is inserted through a cannula into the joint. This permits visualization while using surgical instruments inserted via the second cannula

tissue breakdown products and inflammatory cytokines and to surgically break up any adhesions and improve joint mobility (Fig. 9.2). However, it is extremely difficult to surgically reposition the disc arthroscopically and so it is generally allowed to remain in its anterior position. Despite this, most patients had little or no pain and an improved range of motion postoperatively. With the realization that improving joint mobility and not disc position was the important factor in successfully treating patients with painful TMJ locking, arthroscopic surgery became the primary treatment modality for this condition.

However, all of this changed in 1991 with the introduction of arthrocentesis [5]. This involved the placement of two hypodermic needles into the upper joint space through which the joint could be lavaged, as in arthroscopic surgery, and the adhesions were eliminated and joint mobility was improved by manual manipulation of the mandible (Fig. 9.3). Thus, the same goals as for arthroscopic surgery were accomplished in an even less invasive manner. Moreover, a comparison of the two procedures showed similar success, with arthrocentesis being simpler, less expensive, and having fewer complications [6–9]. The only difference was not being able to actually visualize the joint, but this is generally not necessary. Therefore, arthrocentesis has now become the initial treatment of choice for TMJ locking. If it fails, arthroscopic surgery, discoplasty or discectomy may then be indicated.

9.2 Orthognathic Surgery and the TMJ

Orthognathic surgery is one of the most common procedures performed by oral and maxillofacial surgeons. Because most of these procedures involve changes in the position of one or both jaws, it has been suggested that this could affect the relationship of the articulating components of the TMJ. This raises two questions: Can orthognathic surgery cause TMJ problems and can orthognathic surgery cure TMJ problems?

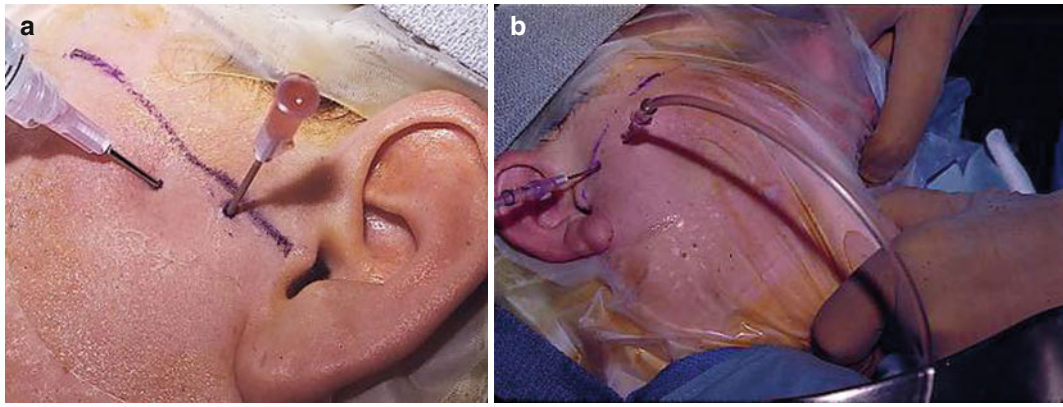


Fig. 9.3 Arthrocentesis of the temporomandibular joint. (**a**) Placement of two hyperdermic needles into the upper joint space to allow irrigation of the joint. (**b**) Manual manipulation of the mandible during joint irrigation to break up adhesions and increase joint mobility

9.2.1 Orthognathic Surgery as a Cause of TMJ Problems

There have been numerous reports on the development of various signs and symptoms such as clicking, crepitation, persistent joint pain and tenderness, limitation of mouth opening and condylar resorption arising in previously asymptomatic patients following orthognathic surgery [10–21]. Their prevalence has ranged anywhere from 4 to 60 %, depending on the conditions studied (Table 9.1). Thus, there is no doubt that orthognathic surgery can cause TMJ problems in some patients. The question then is: Are there things that can be done to prevent, or at least reduce, such problems?

There are four groups of patients that are at the highest risk for developing postoperative TMJ symptoms, especially internal derangements and condylar resorption, as a result of changes in condylar position during orthognathic surgery. These include high angle mandibular retrognathic patients, particularly young females; patients with an associated anterior open bite; patients requiring superior repositioning of the maxilla, which results in an autorotation of the mandible; patients with mandibular asymmetry who will require axial rotation of the mandible; and patients needing a mandibular setback of more than 9 mm. Thus, it is important that excessive changes in condylar position be avoided in such patients, especially following a bilateral sagittal split osteotomy (BSSO). There are a number of factors that can affect proper manual repositioning of the condyles during this operation. These include the supine position of the patient, the reduced muscle tonus, the direction of the surgical mandibular movement, and whether the condyles are positioned in centric occlusion or centric relation. Once the condyles are manually positioned, how they are fixed in that position is also a crucial factor. Because of the u-shape of the mandible, both advancement and setback produce a changed axial relationship between the proximal and distal segments and a resultant lateral condylar rotation can occur as a result of the improper application of rigid fixation (Fig. 9.4).

Table 9.1 Prevalence of new TMJ symptoms in previously asymptomatic orthognathic surgery patients

Friehofer and Petosevic [10]	45 %
Hackney et al. [11]	60 %
De Clerq et al. [12]	12 %
Dervis and Tuncer [13]	10 %
Karabouta and Martis [14]	11.5 %
White and Dolwick [15]	8 %
Panula et al. [16]	13 %
Westermark et al. [17]	21 %
Dujoncquoy et al. [18]	4 %
Aoyama et al. [19]	24 %
Oland et al. [20]	10 %
Wolford et al. [21]	36 %

The desire to place the condyles back in their exact preoperative position following orthognathic surgery has led to the use of condylar positioning devices by some surgeons [22] (Fig. 9.5). However, others have questioned the necessity to have such accurate positioning, arguing that anecdotally most patients have minimal or no dysfunction with closed reduction of displaced subcondylar fractures. Also, the procedure known as condylotomy, which changes the position of the condyle, has actually been used to treat some TMJ disorders [23]. In a comparison study, Gerressen et al. [24] treated 28 patients by mandibular advancement, of which 18 had manual condylar repositioning and 10 had a repositioning device used, and 21 patients by mandibular setback, of which 10 also had a repositioning device used. They found that in the advancement patients the manually positioned group had fewer signs of temporomandibular disorders (TMDs), and in the setback patients there was no significant difference. Furthermore, in a review of 11 studies on the use of condylar positioning devices in orthognathic surgery, Costa et al. [25] found no scientific evidence to support the routine use of such devices. It can therefore be concluded that although excessive malposition of the condyle can lead to postoperative relapse, and therefore should be avoided, lack of exact condylar positioning is not a factor in causing TMDs in orthognathic surgery patients.

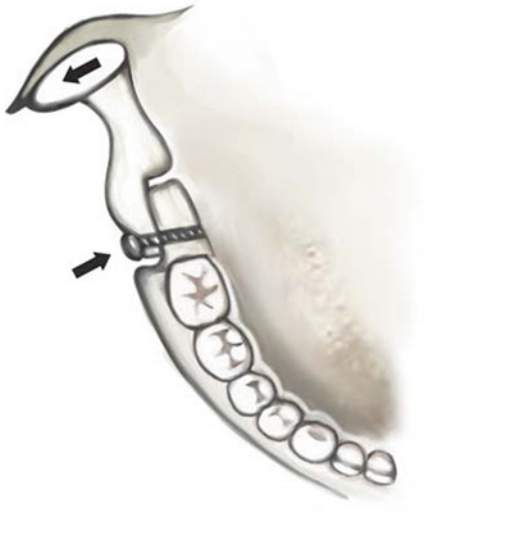


Fig. 9.4 Potential lateral displacement of the condyle following a sagittal split osteotomy caused by improper fixation screw placement

9.2.2 Orthognathic Surgery as a Cure for TMJ Problems

Studies have shown a higher incidence of TMDs in patients undergoing orthognathic surgery than in the general population [12, 15–17, 26–28],

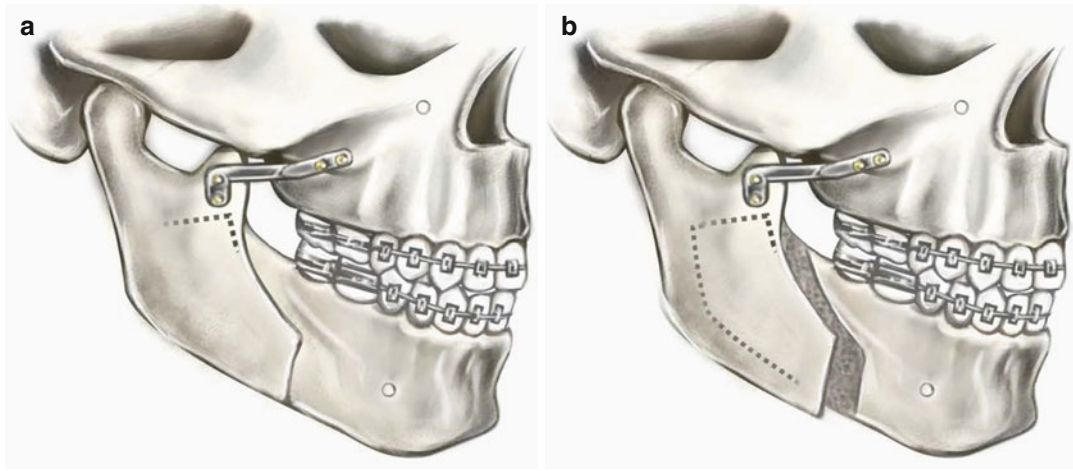


Fig. 9.5 Condylar positioning technique. (**a**) The bone plate is adapted prior to making the bone cuts. (**b**) Application of the bone plate after completion of the osteotomies repositions the condyle in its original position (Reprinted with permission from Ellis [22])

Table 9.2 Prevalence of TMJ signs and symptoms in preoperative orthognathic surgery patients

De Clercq et al. [12]	26 %
White and Dolwick [15]	49 %
Panula et al. [16]	73 %
Westermark et al. [17]	43 %
Upton et al. [26]	53 %
Link and Nickerson [27]	90 %
Schneider and Witt [28]	80 %

(Table 9.2) and this has led to the idea that malocclusions and skeletal deformities can predispose to such problems. For example, it has been claimed anecdotally that there are increased compressive forces on the mandibular condyle in Class II patients with a high mandibular plane angle and an anterior open bite or in those with a low angle deep bite. Although the literature often cites such malocclusions and skeletal deformities as contributing to TMDs, this information is based mainly on opinion and weak correlative data; no controlled studies have proven this relationship. Nevertheless, orthognathic surgery is often recommended as a treatment for TMDs [15–17].

There are numerous reasons why it is difficult to accurately evaluate and compare the various studies that have been published on the ability of orthognathic surgery to effectively treat various TMDs. Most studies include different types and severities of malocclusion and skeletal deformity rather than relate to a single clinical entity. They also vary in the criteria that were used preoperatively to establish the presence of a TMD. The type of TMD and the preoperative duration of the TMD symptoms are often not considered, and there are different follow-up periods postoperatively. Also, the signs and symptoms evaluated vary between studies. Finally, the samples studied are heterogenous and evaluated subjectively based on the patients' findings rather than being carried out by blinded observers. As an example, in a positive retrospective review, which included probably the largest number of post-orthognathic surgery patients ever evaluated in a single study [17] it was noted that 43 % of the patients reported subjective symptoms preoperatively and that only 28 % reported such symptoms postoperatively. It was therefore concluded that "This difference indicates an overall beneficial effect of orthognathic surgery on TMD signs and symptoms." However, an analysis of this study shows that it had a number of flaws that raise serious doubts about such a conclusion. The data combined rather than separated the various types of skeletal deformities; two different types of mandibular operations (BSSO and vertical ramus osteotomy) were performed, and four different types of osteosynthesis were used. The clinical outcomes evaluated included only joint pain, chewing pain, joint noise, unspecified type of "grinding" and headache, and the findings were based on the patients' own subjective evaluation of these variables.

In a more recent study [18] the conclusion was that "Most patients with preoperative TMJ signs and symptoms can improve TMJ function and pain levels can be reduced by orthognathic surgery." Yet, this conclusion was based on a subjective questionnaire to which only 57 of 176 patients responded. Moreover, there was no indication of the types of malocclusion or skeletal deformity treated, and postoperatively there were actually 15 new cases of joint sounds, 10 new cases of TMJ pain, 8 new cases of joint locking, and 9 new cases of clicking.

In a recent systematic review of 23 studies on the use of orthognathic surgery to relieve chronic painful TMDs [29], 16 studies showed no change or a worse outcome and 7 showed mostly insignificant improvement. Thus, on the basis of these various studies, it can be concluded that orthognathic surgery is not a reliable treatment approach for TMDs, and since it is such an invasive procedure, it should be performed only when indicated for other biological or psychological reasons.

Conclusions

Orthodontic patients presenting with painful clicking and popping sounds in the temporomandibular joint should always be managed medically at first. The objective is to eliminate the pain, and this is accomplished by allowing the retrodiscal tissues to heal and function as a new "disc". The clicking and popping can only be eliminated surgically, and this is indicated only if medical management does not stop the pain.

Persistent locking may also respond favorably to medical management and jaw exercises in some patients. For those who do not respond to such treatment in a reasonable period, arthrocentesis should be the initial therapy; arthroscopic surgery, discoplasty, or discectomy should only be done if this fails.

It is evident that orthognathic surgical treatment can sometimes produce TMDs in patients who did not have such problems presurgically. Such problems can be minimized by an understanding of the various contributing factors, but are probably not completely preventable. Therefore, patients need to be informed of these potential risks as well as of the benefits of orthognathic surgery. It is also evident that orthognathic surgery is not a predictable treatment approach for TMDs [30]. Thus, there must always be indications for performing orthognathic surgery in these patients other than the treatment of a TMD.

Take Home Messages

- It is generally recommended that medical management (rather than surgery) should be the first-line approach in treating most diseases and disorders of the temporomandibular joint (TMJ).
- The initial treatment of painful clicking or popping in the TMJ is always medical and involves modalities such as an analgesic for the pain, a soft non-chewy diet, and a bite appliance to control any parafunctional activity.
- For most TMDs, arthrocentesis appears to be the best surgical approach.
- Does orthognathic surgery cause TMD? Although excessive malposition of the condyle can lead to postoperative relapse and steps need to be taken to minimize this, the lack of exact condylar positioning is not a factor in causing TMDs in orthognathic surgery patients.
- Can orthognathic surgery be a cure for TMD? Orthognathic surgery is not a reliable treatment approach for TMDs.

References

1. Kirk Jr WS, Calabrese DK. Clinical evaluation of physical therapy in the management of internal derangement of the temporomandibular joint. J Oral Maxillofac Surg. 1989;47:113–9.
2. Nicolakis P, Erdogmus B, Kopt A, Ebenbichler G, Kollmizer J, Piehslinger E, et al. Effectiveness of exercise therapy in patients with internal derangement of the temporomandibular joint. J Oral Rehabil. 2001; 28:1156–64.
3. Kurita K, Westesson P-L, Yuasa H, Toyama J, Machida J, Ogi N. Natural course of untreated symptomatic temporomandibular joint disc displacement without reduction. J Dent Res. 1998;77:361–5.
4. Manfredini D, Favero L, Gregorini F, et al. Natural course of temporomandibular disorders with low pain-related impairment: a 2-to-3-year follow-up study. J Oral Rehabil. 2013;40:436–42.
5. Nitzan DW, Dolwick MF, Martinez GA. Temporomandibular joint arthrocentesis. A simplified treatment for severe, limited mouth opening. J Oral Maxillofac Surg. 1991;49:1163.
6. Kropmans TJ, Dijkstra PU, Stegenga B, De Bont LGM. Therapeutic outcome assessment in permanent temporomandibular joint disc displacement. J Oral Rehabil. 1999;26:357–63.
7. Goudot P, Jaquinet AR, Hugonnet S, Haefliger W, Richter M. Improvement of pain and function after arthroscopy and arthrocentesis of the temporomandibular joint: a comparative study. J Craniomaxillofac Surg. 2000;28:39–43.
8. Sanroman JF. Closed lock (MRI fixed disc): a comparison of arthrocentesis and arthroscopy. Int J Oral Maxillofac Surg. 2004;33:344–8.
9. Hobeich JB, Salamejh ZA, Ismail E, Sadig WM, Hokayem NE, Almas K. Arthroscopy versus arthrocentesis. A retrospective study of disc displacement management without reduction. Saudi Med J. 2007;28:1541–4.
10. Freihofer DM, Petresevic D. Late results after advancing the mandible by sagittal splitting of the rami. J Maxillofac Surg. 1975;3:230–7.
11. Hackney FL, Van Sickels JE, Nummikoski PV. Condylar displacement and temporomandibular joint dysfunction following bilateral sagittal split osteotomy and rigid fixation. J Oral Maxillofac Surg. 1989;47:223–7.
12. De Clercq C, Neyt L, Mommaerts M, Abeloos J. Orthognathic surgery: patients' subjective findings with focus on the temporomandibular joint. J Craniomaxillofac Surg. 1998;26:29–34.
13. Dervis E, Tuncer E. Long-term evaluations of temporomandibular disorders in patients undergoing orthognathic surgery compared with a control group. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;94:554–60.
14. Karabouta J, Martis C. The TMJ dysfunction syndrome before and after sagittal split osteotomy of the rami. J Oral Maxillofac Surg. 1985;13:185–8.
15. White CS, Dolwick MF. Prevalence and variance of temporomandibular dysfunction in orthognathic surgery patients. Int J Adult Orthodon Orthognath Surg. 1992;7:7–14.

16. Panula K, Somppi M, Finne K, Oikarinen K. Effects of orthognathic surgery on temporomandibular joint dysfunction: a controlled prospective 4-year follow-up study. Int J Oral Maxillofac Surg. 2000;29:183–97.
17. Westermark A, Shayeghi F, Thor A. Temporomandibular dysfunction in 1,516 patients before and after orthognathic surgery. Int J Adult Orthodon Orthognath Surg. 2001;16:145–51.
18. Dujoncquoy J-P, Ferri J, Raoul G, Kleinheinz J. Temporomandibular joint dysfunction and orthognathic surgery. Head Face Med. 2010;6:27. Published online 17 Nov 2010. doi:10.1186/1746-160X-6-27.
19. Aoyama S, Kino K, Kobayashi J, Yoshimasu H, Amagasa T. Clinical evaluation of the temporomandibular joint following orthognathic surgery-multiple logistic regression analysis. J Med Dent Sci. 2005;52:109–14.
20. Oland J, Jensen J, Melsen B. Factors of importance for the functional outcome in orthognathic surgery patients: a prospective study of 118 patients. J Oral Maxillofac Surg. 2010;68:2221–31.
21. Wolford LM, Reiche-Fischel O, Mehra P. Changes in temporomandibular joint dysfunction after orthognathic surgery. J Oral Maxillofac Surg. 2003;61:655–60.
22. Ellis III E. Condylar positioning devices for orthognathic surgery: are they necessary? J Oral Maxillofac Surg. 1994;52:536–52.
23. Hall HD, Navarro EZ, Gibbs SJ. Prospective study of modified condylotomy for treatment of nonreducing disk displacement. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000;89:147–58.
24. Gerressen M, Zadeh MD, Stockbink G, Riediger D, Ghassemi A. The functional long-term results after bilateral sagittal split osteotomy (BSSO) with and without a condylar positioning device. J Oral Maxillofac Surg. 2006;64:1624–30.
25. Costa F, Robiony M, Toro C, Sembronio S, Polini F, Politi M. Condylar positioning devices for orthognathic surgery: a literature review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;106:179–90.
26. Upton LG, Scott RF, Hayward JR. Major maxillomandibular malrelations and temporomandibular joint pain-dysfunction. J Prosthet Dent. 1984;51:686–90.
27. Link JJ, Nickerson JW. Temporomandibular joint internal derangements in the orthognathic surgery population. In J Adult Orthodon Orthognath Surg. 1992; 7:151–9.
28. Schneider S, Witt E. The functional findings before and after a combined orthodontic and oral surgical treatment of Angle Class III patients. Fortschr Kieferorthop. 1991;52:51–9.
29. Lindenmeyer A, Sutcliffe P, Eghtessad M, Goulden R, Speculand B, Harris M. Oral and maxillofacial surgery and chronic painful temporomandibular disorders – a systemic review. J Oral Maxillofac Surg. 2010;68:2755–64.
30. Al-Riiyami S, Cunningham SJ, Moles DR. Orthognathic treatment and temporomandibular disorders: a systematic review. Part 2. Signs and symptoms and meta-analysis. Am J Orthod Dentofacial Orthop. 2009;136:626. e1–e16.

TMD and Its Medicolegal Considerations in Contemporary Orthodontic Practice

10

L. Jerrold, Sanjivan Kandasamy, and D. Manfredini

10.1 The Standard of Care

The diagnosis and management of patients exhibiting temporomandibular disorders (TMD) in the orthodontic setting is fraught with great debate, confusion, diverse opinions, and philosophies. Because TMD has a multifactorial etiology and is layered in nature, it currently is viewed as reflecting more of a medical and psychosocial model regarding its diagnosis and subsequent management. As such, it is critical that orthodontists understand their limitations, the need for adequate training in this continually evolving area, and the need for thorough and well-documented records [1, 2].

L. Jerrold, DDS, JD (✉)
NYU-Lutheran Medical Center,
Department of Dental Medicine,
Division of Orthodontics, 5800 3rd Ave,
Brooklyn, NY 11220, USA
e-mail: drlarryjerrold@gmail.com

S. Kandasamy, BDSc, DClinDent, MOrthRCS, FRACDS
Department of Orthodontics, School of Dentistry,
University of Western Australia, Nedlands, WA,
Australia

Centre for Advanced Dental Education,
Saint Louis University, Saint Louis, MO, USA

Private Practice, Midland, WA, Australia

D. Manfredini, DDS, MSc, PhD
Italian Minister of University and Instruction,
Padova, Italy

Department of Maxillofacial Surgery,
University of Padova, Padova, Italy

Given these caveats and concerns one must first ask, are there medicolegal considerations specific to the diagnosis and treatment of TMD associated with the practice of orthodontics? The simple answer is no; the medicolegal considerations are the same across the board in all of the healing arts. However, from a risk management perspective, one of the first concerns is addressing the elements that a potential plaintiff needs to prove for an orthodontist to be found liable regarding any treatment he or she has rendered. Those elements are that there was a duty to conform to a given standard of care and that the breach of this duty was the direct or proximate cause of any injury suffered.

It is only through the existence of a doctor-patient relationship that orthodontists are required to conform to a certain standard of care; which will vary slightly throughout the world. This "conformance" is the duty to which we are held. Three elements comprise this duty. First, regardless of the general or specialty area of dentistry in which you practice, you are bound to possess the degree of SKEE (skill, knowledge, education, and expertise) as is possessed by the average practitioner in good standing, within the same area of practice, acting under the same or similar circumstances. Second, you must exercise this degree of SKEE in a reasonable manner. Third, you must use your best judgment as you go about administering the care you render to a patient.

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1_10

At this point, a certain distinction needs to be made. A plaintiff can initiate a lawsuit against you based on your failing to conform to a given standard of care as described above; but it is here that one must be aware that a suit can also be filed claiming that the practitioner did not obtain adequate informed consent even though the care itself may have been adequate. In other words, there are different legally recognized "causes of action" to which a practitioner may become exposed. You can be sued for one or the other or both. The standard of care is the same. Did you divulge whatever information was required in order for the patient to adequately grant you their informed consent to proceed with whatever treatment you are proposing to render. This is discussed in detail further on in this chapter.

10.2 Defining the Duty of Care

10.2.1 Ascertaining the Patient's Chief Complaint

In the practice of orthodontics, including the treatment of any signs or symptoms of TMD, one of the more common breaches of the duty that we owe our patients is not conducting an adequate comprehensive examination. Whenever we see a new patient, the first thing we need to do after we introduce ourselves is to ask the patient "What can I do for you?" "How can I help you?" "Why are you here?" What you are attempting to do is to elicit the patient's chief complaint. This is the first step in conducting a comprehensive examination of the patient. You may find out that one of the patient's chief complaints does or does not include symptoms that may relate to TMD. Attempting to elicit not only what the patient's concerns are but also integrating this information with your impressions is important because it will guide you to have to make the first in a number of decisions, that being whether or not you want to treat this patient's particular problem.

The standard of care requires that you MANAGE every patient appropriately. You are not obligated to treat every patient that enters your office nor are you required to treat every type of clinical problem. If you are comfortable treating patients presenting with TMD, fine; if you are not, the standard of care requires that you must either refuse to treat the patient or you can offer a referral to someone else who has more SKEE in this particular area. Referral is an appropriate way to manage a particular problem.

As the title of this chapter relates to TMD in the orthodontic patient, let us develop a hypothetical patient. She is in her early 30s, single, and employed in a middle management position. Her chief complaints are that she does not like the crowding of her upper and lower anterior teeth, nor the look of her smile, and she states that her jaw sometimes clicks on opening which she finds annoying at times. Assuming that you believe you possess the requisite SKEE in both orthodontics and TMD to treat the patient, what is next?

10.3 The Comprehensive Clinical Examination

10.3.1 The Patient's Medical, Dental, and Social History

As with any examination, the critical risk management concerns are twofold. First, that the exam was appropriately conducted given the totality of the circumstances attached to any particular patient; and second, and of equal importance, is that the results of your examination were adequately documented. All findings, both positive and negative, need to be documented. This is a cardinal risk management principle. If a finding is negative, meaning it was not found to be present, then that negative finding is what needs to be documented. If the negative finding is not documented as such, it will not be presumed that the lack of documentation was because the finding was negative; rather it will be presumed that that part of the exam was not performed.

The first step after obtaining the patient's chief complaint is to perform a comprehensive clinical examination and the first part of this activity involves obtaining adequate prior medical, dental, and social histories. This may or may not provide you with any information that impacts on

your diagnosis and treatment of any given patient but one thing is certain, if you do not obtain this information, you will never know whether or not it was important. As there are numerous medical and psychological factors that can relate to treating an orthodontic patient who also happens to present with TMD symptomatology, not obtaining an adequate history is a breach of the standard of care owed to every patient.

The past dental history may also elicit relevant information such as whether or not the patient has undergone prior orthodontic treatment and if so what treatment was performed. In addition, you would want to know whether the TMD sign or symptom is acute or long standing as this may have some bearing on how you will manage a particular problem. Again, obtaining this information certainly falls within the standard of care.

A comprehensive clinical exam also includes a social history that encompasses looking into any habits or lifestyle activities that may relate to both the etiology of the malocclusion and the TMD symptomatology presented. In a TMD patient, a social history could also include the possibility of referring the patient for psychological assessment depending upon the circumstances of the patient's presentation.

These three aspects, medical, dental, and social, of a patient's history should not be ignored as they are core elements of formulating a differential diagnosis and an appropriate treatment plan for every patient. It relates to what we were taught at the beginning of our dental education – never treat a stranger.

10.3.2 The Examination

The remainder of the clinical exam consists of a functional and extra oral examination, and an intraoral examination of the patient's hard and soft tissues, and a radiologic examination. This is discussed in more detail in Chapter Three.

The functional examination is just that, an examination of the functional status of a soon to be orthodontic patient. Function includes an evaluation of the joints and an evaluation of the occlusion both static and dynamic.

Let us go back to our hypothetical patient. The first thing you would want to know concerning the click is if there is any pathology associated with this symptom. Pathology is defined as the study of disease, the process in which the disease manifests itself, and its degree of abnormality. When you query the patient about the click you learn that it happens every now and then but that she does not admit to experiencing any pain or dysfunction such as an obvious limited range of motion or difficulties in opening or closing her mouth. This finding, assuming no other diagnostic findings are found, may lead one to conclude that at this point in time, observation is the most viable and conservative of the several treatment options for the click. On the other hand, if there is pain, a limitation in lateral excursions or in opening, or occasional episodes of locking your evaluation should also include obtaining measurements relating to any limitation of movement, and, in some selected cases, specific imaging studies. All of these diagnostic evaluations are detailed in other chapters of this book.

When it comes to the extra oral examination you are essentially looking for relative facial symmetry or evidence of asymmetry that might indicate the presence of a functional shift or skeletal asymmetry. In addition, if the patient has complained of headaches, tightness in the jaw, clenching, etc., simple palpation of the muscles of mastication may elicit a positive response that may lead toward recommending one form of treatment over another.

With regard to the intraoral examination all hard and soft tissues must be thoroughly examined. Of particular importance to the relationship between orthodontic therapy and TMD is an examination of the occlusal and incisal surfaces of the teeth as well as scalloping of the lateral borders of the tongue as these findings may indicate the presence of clenching and/or bruxism, a habit/condition that has been one of the factors possibly associated with TMD symptomatology. As for occlusion, despite its diminished role in TMD etiology, its evaluation still remains important albeit more so for orthodontic purposes.

When undertaking this aspect of the orthodontic examination, the patient's occlusion must be

evaluated both statically and dynamically. There is no question that TMD is multifactorial in terms of etiology. The current literature reveals that there is very little direct cause and effect regarding occlusion and its role in TMD, and the same can also be said for the role of the condyle's position in the fossa. A simple static occlusal exam takes note of excessive deep bites, open bites, and crossbites that are associated with a functional shift. A dynamic exam notes balancing interferences in protrusive and lateral excursions as well as ascertaining any limitations of mandibular movement in lateral excursion or in opening.

Why is all this important? The reality of orthodontic practice is that one needs to have baseline information in order to evaluate the efficacy of any treatment rendered particularly if the maxillo-mandibular relationship will be changed as a result of the orthodontic treatment rendered. Remember, that at the end of the day, behind every smile we create, there needs to be a working, functioning stomatognathic system. The bottom line from a medicolegal perspective is that not performing these basic examinations and noting any positive findings, or in the alternative, not noting that everything appears to be within normal limits, can easily be interpreted as falling below the standard of care.

Why is this so? Our hypothetical patient has reported a symptom of intermittent clicking that may be of possible clinical relevance. The duty of any treating health care provider is once informed of a symptom, to first verify its existence or nonexistence. If it exists, the duty extends to establish the degree of deviation or abnormality. Next is the requirement to discover or attempt to discover the cause(s) of the sign/symptom. As discussed in previous chapters, with most TMD cases, the cause is idiopathic and the management is usually palliative and symptomatic. Next is to ascertain the clinical impact, if any, of this symptom. Finally, the practitioner is required to make a recommendation on managing the finding via observation, treatment, or appropriate referral. If you are not going to treat the TMD, at least your findings will provide documentation for the basis of the referral. It is these steps that constitute the duty we have to conform to a given standard of care. Once again, if your findings are not noted it will not be assumed that they were negative. Rather it will be presumed that the exam in question was not performed.

10.3.3 Radiographic and Photographic Examination

The most routine images associated with the provision of orthodontic therapy are the panoramic and lateral cephalometric radiographs, and the intra and extra oral photographs. Relative to TMD, panoramic films can be used to see the condyles and fossa to the extent that any gross condylar asymmetry or abnormal morphology may often be clearly visualized. By our basic dental training, we are sufficiently trained and astute enough to ascertain when facial or cranial structures appear to be abnormal in shape or size. We can see that either "everything appears within normal limits," "something is wrong," or "something is not right." Our duty is not necessarily to be able to diagnose whatever the abnormality is, nor how best to treat it, but rather to recognize that something is amiss and to make an appropriate referral. Once again, it comes down to managing the problem appropriately.

As one cannot assess or diagnose TMD based on standard orthodontic radiographs in an asymptomatic patient the next question arises: Are transcranial films, magnetic resonance imaging (MRI), computer-assisted tomography (CAT), or cone beam computed tomography (CBCT) of the joint the standard of care? Well, they might be within the standard of care if you are going to treat a specific TMD or temporomandibular joint (TMJ) problem and you have determined that these additional radiographs will actually augment the data gleaned from the clinical examination. A key point is that these images are not part and parcel of a routine orthodontic examination; even to a patient who exhibits mild TMD symptomatology so long as that patient is without discomfort, pain, limitation in range of motion, or other dysfunction.

10.4 Diagnostic Considerations

Having performed a comprehensive examination, you are required to arrive at a diagnosis followed by a creation of a problem list listing the aberrations from normal; skeletal, functional, dental, and aesthetic.

Again, let us go back to our hypothetical patient. You have determined that she presented with irregular alignment of her upper anterior teeth, a slight (2–3 mm) off of Class I toward a Class II molar and canine relationship bilaterally, and mild (3 mm) lower anterior crowding. The mandible appears to be slightly retrusive. The TMD part of the examination revealed an opening and closing reciprocal click on the left side which the patient reports is intermittent. The patient also reported no history of locking, and there was no limitation of lateral movement or in opening. There was no history of pain or dysfunction. What is your treatment plan? Are you able to plan different strategies for the orthodontic treatment and the TMJ diagnoses? Would your strategy differ if the patient was functionally symptomatic?

In our asymptomatic patient, some of us would find a way to distalize the upper posterior segments. As we all know, there are a lot of ways to do that. If that approach is chosen some could make the argument that it does not address the mandibular retrusion. Some might simply just treat the crowding, align the upper and lower anterior teeth essentially treating the aesthetic zone, and leave the mild Class II relationship the way it is. After all, the patient was not really concerned with the slight overjet (she did not even really know she had one) but was more concerned with the lower crowding and the slight upper anterior irregularities. Again, that approach addresses some of the problems found but not the retrognathism. Some would opt to use Class II mechanics to address the overjet. Again there are a lot of ways to effectuate mandibular propulsive mechanics. Whether you are merely getting dentoalveolar changes, mandibular repositioning or growth, or a combination of both is a whole different discussion. What about the reciprocal click? In an asymptomatic patient who has an occasional click eliciting no pain or dysfunction of any type, one can probably proceed directly with orthodontic therapy making sure to monitor the status of the click throughout treatment. The same cannot be said for someone who exhibits pain, limitations of movement, or locking during function.

10.5 Informed Consent

Regardless of which approach you prefer, you now have to have a conference with the patient. It is here that we obtain the patient's informed consent. It should be noted that we do not give informed consent; we get it. Depending upon what State or for that matter, what country one practices in, this requirement will vary. In the USA, the jurisdictions are divided between those that require a practitioner to divulge whatever information would be divulged by the average practitioner under the same or similar circumstances with those States that require patients be provided with that degree of information that they would deem material in the decision-making process in order to accept or reject a contemplated course of therapy. Either way, they can only make this decision when they are told in a language they can understand what their particular problem is, and the various ways in which it can be treated. The only way to choose between various treatment plans is to be told the benefits, risks, limitations, and compromises associated with each viable treatment approach. Patients should also be informed of any necessary secondary treatment associated with undergoing the proposed treatment. They have to be informed of the prognosis associated with each approach as well as the option of no treatment. Once they have had the chance to ask and have answered all of their questions we can then proceed with the option that the patient has chosen. Let us look a little closer at each of these elements.

If there are language barriers or comprehension challenges that prevent the patient from understanding what their problem is or what is

being proposed to correct these problems, any consent ostensibly given may easily be contested. "Dentalese" is never an acceptable alternative to basic English or the patient's native tongue.

Occasionally, there is one preferred way to treat a given condition. More often, there are several viable alternatives to address a given problem. The patient must be apprised of all viable treatment alternatives, each one accompanied by the expected prognosis, and any risks, compromises, or limitations that attach to a particular treatment approach.

Patients also need to be informed of why treatment is necessary or being recommended and what the prognosis will be if they decline treatment. They have to be given the opportunity to ask and have answered all of their questions. In addition, there have been instances where how long treatment will take, scheduling concerns, financial responsibilities etc., come into play as these considerations fall within the specter of obtaining informed consent.

Necessary secondary treatment must also be divulged as patients will often claim that had they known that "XYZ" was required after treatment, and that there are additional fees that accompany this secondary treatment, they would not have agreed to the treatment proposed or might have opted for a different treatment plan. Two common examples are post treatment prosthetics and lifetime fixed retention.

Going back to our hypothetical patient, from the diagnostic perspective, you should explain that the irregular line-up of her top front teeth is detracting from her smile, that she has a slight horizontal "overbite" (patients think of overjet as overbite), that she has some mild crowding of her lower front teeth, and the tissues comprising her jaw joint on the left side are in such a position with one another that they make a clicking sound on opening and closing which does not appear to have affected the ability to move her jaw from side to side or limit her ability to open and close.

Addressing her treatment, she should be told that there are essentially three ways to address her orthodontic problem. The first would be to correct her "overbite" by pushing her top teeth further back in her mouth however there is a slight chance that it may negatively affect her upper lip profile and overall facial profile. The second way is to leave the "overbite" and just line up her upper front teeth and take care of the crowding in her lower front teeth (if there are periodontal concerns related to the expansion therapy they should be stated here). Finally, her "overbite" can also be corrected by wearing rubber bands between the jaws but there is a slight chance that they may make the clicking worse. You should then relate your experience with each and provide a recommendation but the choice is hers to make.

As to her TMD symptoms, as the patient is essentially asymptomatic except for the occasional reciprocal click, she would only need to be advised that you will monitor her TMJ status throughout treatment and that if changes are discerned, based on the mechanics employed or the patient's response to treatment, you will manage whatever the clinical findings are in both a timely and appropriate manner. As the majority of the literature shows that orthodontics is basically TMD neutral, this is a reasonable approach.

However, let us say our patient was symptomatic. As TMD symptoms, such as pain, locking, and limitations of movement, are indicative that something is wrong or in the alternative, the recognition that something is not right, it is not appropriate to begin orthodontic therapy until these symptoms are adequately addressed. Appropriate management of this patient would entail either treating the TMD yourself (if you choose to treat this yourself you must have the requisite SKEE regardless of how it is obtained) or referring the patient to someone with more SKEE in this area. Once the TMD is under control, it is at this point in time that orthodontics can begin. This is analogous to the patient who has active periodontal disease and a malocclusion. It is below the standard of care to initiate orthodontic therapy until the periodontal condition is stabilized and maintained. All of the other elements noted above also need to be discussed with the patient in the same simple language.

The last consideration would be if our hypothetical patient developed increased TMD symptoms during treatment. What then? TMD

symptomatology can develop in any patient at any time. A recent systematic review which included only studies which utilized the Research Diagnostic Criteria for TMD (RDC/TMD) reported a prevalence of up to 16 % for disc disorders, up to 13 % for masticatory muscle pain and up to 9 % for TMJ pain disorders in the general population [3]. Only 3.6–7 % of individuals with TMD are estimated to be in need of treatment [4]. This group of patients is often in their mid to late teens or young adults and middle-aged adults rather than children and the elderly. Prevalence of TMD in women is twice more common than in men [4]. Based on this, the likelihood of an average orthodontic patient developing signs or symptoms is more than possible. Unfortunately, this is commonly misinterpreted as the orthodontic treatment causing TMD. We must tell all patients, especially those like our hypothetical one, that there is the possibility that TMD may occur during orthodontic treatment. Notice we did not say that TMD occurred because of the orthodontics. As we already discussed, orthodontic therapeutic intervention is essentially TMD neutral. While some jaw and or muscular tenderness may occur as a result of the mechanotherapy employed, at least until musculoskeletal adaptation is achieved, a variety of TMD issues may occasionally occur during orthodontic treatment. It is this fact that patients need to be aware of. Further clinical evidence as well as the literature supports the role exerted by psychosocial factors in the multifactorial etiology of TMD and therefore we cannot ignore educating our patient regarding this issue. It is the acceptance of the possibility that something negative can occur during treatment and the acquiescence to proceed that forms the basis for the doctrine of informed consent.

There is no "one way" to provide this information. Videos, pamphlets, and other patient educational tools are viable methods. Proprietary forms are another. Some prefer a good old fashioned conversation. A patient's verbal consent is acceptable but documentation of what was discussed and the patient's assent needs to be documented. There are numerous methods of documentation available today.

It also does not matter who interacts with the patient. It can be the doctor, the treatment coordinator, an assistant, and so on. The bottom line is that practitioners need to transmit this information and then just as importantly, they need to document that this responsibility was completed.

10.6 To Whom Does One Refer?

TMD has gradually undergone the transition from a purely orofacial/dental problem to a medically based psychosocial model because of its multifactorial etiology and its multifaceted approach to treatment. This model integrates a plethora of biologic, clinical, and behavioral factors that are inter-related in the onset, diagnosis, treatment, maintenance, and hopefully the remission or resolution of the problem at hand. In situations where one makes a referral, either because of a lack of SKEE to treat the patient's problem or because of the need for interdisciplinary treatment to be rendered by other concurrent treating practitioners, the risk management concern is how to ensure that one does not make a negligent referral that could result in liability being imposed.

Negligent referral liability occurs when a referral is made to someone who does not possess the requisite SKEE to adequately manage or treat the patient; essentially this is a question of the referred to doctor's competency. Merely because one is a self-proclaimed guru in the diagnosis and management of TMD is not a basis on which an acceptable referral can be made. A negligent referral can also result from referring to an impaired practitioner who for whatever reason is incapable of adequately treating the patient. The good news is that the referrer can only be found liable if he or she knew or reasonably should have known that the referred to doctor was incompetent (lack of SKEE) or impaired.

You should be making your referral for managing a patient's TMD in the same manner you would make any other referral. In other words, you are aware of the practitioner's competency and or reputation either first hand or vicariously, and you are not aware of any mitigating factors that would compromise his ability to treat the

patient. The fact that the referred to doctor was your brother-in-law, classmate, golfing buddy, fellow church member, or what have you, is not a sufficient basis on which to generate a referral. In most countries the field of TMD and orofacial pain is an unrecognized specialty, so the best you can do is to base your referral on the same type of criteria you use when you refer a patient to any other dental specialist. Once again, as a referral is a form of management and any act of managing a patient is a form of treatment, all referrals require adequate documentation.

10.7 Records Management

Clinical record keeping is a requirement of every practitioner in the healthcare arena. Many doctors do not appreciate the scope of the dental record. The patient's record is comprised of all intra, inter, and extra office communications relating to the care and treatment of that patient. This includes all billing forms, financial records, appointment books, daily schedules, all forms and form letters used, all correspondence with the patient and all relevant third parties, all diagnostic material, and last but not least, the clinical chart. The two most important reasons for keeping accurate and timely records of the treatment rendered is to serve first, as a basis for diagnosing and treatment planning purposes; and second, to maintain the continuity of patient care.

On the clinical side, patient records provide documentary evidence of your evaluation, diagnosis, and treatment plan for that patient, the informed consent obtained, the treatment rendered as well as all referrals made, interactions with other concurrent treating practitioners, and all follow up treatment rendered. On the administrative side, all communications with the patient, relevant third parties, and third party payers, also comprise a patient's dental record. From a legal perspective, the dental record serves to protect the legal interests of all of these parties. Finally, a patient's dental record provides data for continuing education, research, outcomes assessment, quality assurance, other administrative functions such as productivity evaluations, and billing.

The only requisites are that the dental record must accurately reflect the treatment rendered, they must be completed by a person with knowledge of the content appearing therein, the data entered was done so at or near the time that the treatment was rendered, and that the records have the indicia of credibility. This last item is quite important as all healthcare records, in order to be viewed as trustworthy, have to be made and kept in such a fashion as to preclude the ability of alteration. They must be genuine and they must be able to be unequivocally believed.

Conclusion

At the beginning, we discussed a hypothetical patient. Various risk management concerns relative to each step along the doctor-patient relationship time line were discussed. Prudent attention to risk management procedures and protocols are a requirement today if practitioners are to conform within the standards of care relating to both the administrative and clinical aspects of orthodontic practice particularly when TMD symptomatology accompanies the orthodontic problem. The bottom line is that by adhering to good risk management activities, the doctor-patient relationship is enhanced and strengthened, and this fact alone, tends to lessen the possibility of winding up in the malpractice arena.

Take Home Messages

- Always complement an orthodontic exam with an examination of the TMJ and the associated structures.
- Documenting negative findings are equally important as documenting positive findings.
- Always maintain clear, thorough, and timely records.
- Orthodontics is TMD neutral. This however will not prevent patients and some dentists from believing that your orthodontic treatment has caused TMD signs

or symptomatology to appear during or after treatment.
  - Educate both your patients and other concurrent treating practitioners accordingly.
  - Monitor your patients regularly especially if they exhibited TMD symptoms with or without pain or limitations of movement before treatment.
- If you choose to not treat TMD within your orthodontic practice, refer your patients to someone with the requisite SKEE; if possible someone with additional and/or formal training in the field of orofacial pain.

References

1. Manfredini D, Bucci MB, Montagna F, Guarda-Nardini L. Temporomandibular disorders assessment: medicolegal considerations in the evidence-based era. J Oral Rehabil. 2011;38(2):101–19.
2. Reid KI, Greene CS. Diagnosis and treatment of temporomandibular disorders: an ethical analysis of current practices. J Oral Rehabil. 2013;40(7):546–61.
3. Manfredini D, Guarda-Nardini L, Winocur E, Piccotti F, Ahlberg J, Lobbezoo F. Research diagnostic criteria for temporomandibular disorders: review, criteria, examination and specifications critique. J Craniomandib Disord. 1992;112:453–62.
4. American Academy of Orofacial Pain. Diagnosis and management of TMDs. In: De Leeuw R, Klasser GD, editors. Orofacial pain: guidelines for assessment, diagnosis, and management. 5th ed. Chicago: Quintessence; 2013. p. 130.

Index

S. Kandasamy et al. (eds.), *TMD and Orthodontics: A Clinical Guide for the Orthodontist*,
DOI 10.1007/978-3-319-19782-1