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Physical Therapy Management of Temporomandibular Disorders With Cervical Spine Considerations

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Key Terms

Active myofascial trigger point: Produces spontaneous pain and/or referred pain and always evokes symptoms.

Anterior guidance (protrusive guidance or canine guidance): Is the relationship of any of the anterior 6 mandibular teeth maintaining contact with any of the anterior 6 maxillary teeth during protrusive and lateral excursions. When a person moves their jaw forward, the posterior teeth should separate. When a person moves their jaw into lateral excursion, the posterior teeth on the contralateral side should separate.

Behavioral modification: The process of changing unwanted behaviors that contribute to an overuse, disuse, and/or abuse of the muscles of mastication, temporomandibular joint (TMJ), and/or cervical spine.

Bruxism: A repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible.

Cervicogenic headache (CGH): A headache whose origin is the cervical spine.

Click: A distinct sound (eg, click, pop, snap) that is of a brief and very limited duration with a clear beginning and end that emanates from the TMJ.

Crepitus: A noise, like sandpaper moving over a surface, that is longer in duration that emanates from the TMJ.

Dislocation: Joint is outside its physiological and anatomical boundary.

Familiar symptom: A symptom that the patient has experienced in the past 30 days and not a symptom that was experienced only as a result of the physical examination (false positive).

Latent myofascial trigger point: Is asymptomatic and can be quiescent for months or years but becomes painful when stretched or compressed (palpated) during the physical examination.

Modify: To reproduce, increase, or decrease the patient's symptoms.

Occlusion: How the teeth come together.

Provocation test: Tests performed to modify the patient's familiar symptom(s)

Psychosocial distress: Involves, but is not limited to, fear, anxiety, anger, and depression.

Wallmann HW, Donatelli R, eds. *Foundations of Orthopedic Physical Therapy* (pp 403-435).

Subluxation: Joint is outside its physiological boundary but inside its anatomical boundary.

Temporomandibular disorders (TMD): A cluster of symptoms and signs involving masticatory muscles, the TMJs, or both.

Chapter Questions

Answers to questions can be found at https://www.tmd-stevekraus.com/. Go to tab "Links."

1. What percent of the US population have a malocclusion?
   A. Less than 25%
   B. 25% to less than 50%
   C. 50% to less than 75%
   D. 75% to 100%
2. Which of the following disk displacements is always associated with pain?
   A. Disk displacement with reduction (DDwR)
   B. Disk displacement without reduction with limited opening (DDwoR wLO)
   C. Disk displacement without reduction without limited opening (DDwoR woLO)
   D. None of the above
3. Which of the following findings of the clinical examination of TMD requires treatment?
   A. Click greater than 30 mm without pain
   B. Crepitus greater than 30 mm without pain
   C. Deflection equal to or less than 30 mm without pain
   D. Deflection greater than 30 mm without pain
4. Which of the following clinical findings will help to diagnose subluxation?
   A. Late opening click with mouth opening and late closing click with mouth closing
   B. Early opening click with mouth opening and late closing click with mouth closing
   C. Late opening click with mouth opening and early closing click with mouth closing
   D. Early opening click with mouth opening and early closing click with mouth closing
5. Using the kinesiograph can _____.
   A. Make and accurate diagnosis of TMD
   B. Separate normal patients from patients with TMD
   C. Analyze mandibular movements three dimensionally
   D. Use the data to make an evidence based oral appliance
6. Which one of the following is a common finding with patients with a displaced disk?
   A. Facial asymmetry on the side of the displaced disk
   B. Clicking with or without locking related to a displaced disk
   C. Neck pain on the opposite side of the displaced disk
   D. Posterior open bite on side of the displaced disk
7. Which of the following should be included routinely as part of a TMD examination?
   A. Imaging studies
   B. Occlusal studies
   C. Sleep apnea studies
   D. None of the above
8. Which diagnostic subset of TMD is always associated with a click?
   A. Disk displacement without reduction (DDwoR)
   B. Degenerative joint disease (DJD)
   C. DDwR
   D. Subluxation
9. Which of the following applies to the Diagnostic Criteria of Temporomandibular Disorders (DC/TMD)
   A. Sensitivity and specificity values unknown
   B. Does not account for concurrent diagnostic subsets
   C. Is seldom used in academic and clinical research
   D. Unable to differentiate between myalgia and arthralgia
10. According to the DC/TMD, familiar symptom(s) are symptoms that the patient has experienced in the past _____.
    A. 30 days
    B. 60 days
    C. 90 days
    D. 120 days
11. Which of the following is true about the DC/TMD?
    A. Does consider latent myofascial trigger points
    B. Does not consider latent myofascial trigger points
    C. Does not consider active myofascial trigger points
    D. Does not consider active or latent myofascial trigger points
12. The probability that a malocclusion can cause TMD problems:
    A. Very high
    B. High
    C. Moderate
    D. Low
13. To be clinically meaningful, a noise associated with a DDwR must be _____.
    A. Palpated by the clinician but not heard by the patient
    B. Palpated by the clinician and heard by the patient
    C. Not palpated by the clinician and not heard by the patient
    D. Not palpated by the clinician but heard by the patient

14. Which of the following is not an indication for wearing an oral appliance?
    A. To reduce morning jaw muscle pain and/or headache triggered by sleep bruxism
    B. To establish a centric relation and centric occlusion of the condyle to minimize sleep bruxism
    C. To reduce morning locking/catching associated with a disk displacement related to sleep bruxism
    D. To protect occlusal surfaces of teeth and dental restorations from sleep bruxism
15. Reducing TMJ loading over time is best achieved by _____.
    A. Intraoral joint distraction grade 3
    B. Normalize head and neck posture
    C. Reduce parafunctional activity
    D. Patient wears a pivotal appliance
16. The cause of nocturnal bruxism is _____.
    A. Malocclusion
    B. Sleep apnea
    C. Psychosocial distress
    D. Multifactorial
17. The trigeminocervical nucleus is located in the upper cervical spinal cord within the _____.
    A. Par oralis
    B. Pars cranialis
    C. Pars caudalis
    D. Pars intermedius
18. Subjective tinnitus, subjective fullness, and subjective hearing loss may be related to an increase in activity of which of the following muscles?
    A. Auricular levator and labii superioris
    B. Tensor veli palatine and tensor tympani
    C. Musculus uvulae and palatopharyngeus
    D. Palatoglossus and laryngopharynx
19. What percentage of asymptomatic participants greater than 40 years old have positive imaging findings of cervical spondylitis and cervical disk herniation?
    A. 20%
    B. 40%
    C. 60%
    D. 80%
20. The American Board of Physical Therapy Specialties was established to provide formal recognition for physical therapists with advanced clinical knowledge, experience, and skills in 9 specialty areas of practice. The field of TMDs and orofacial pain falls into which specialty area?
    A. Neurology
    B. Orthopedics
    C. Clinical electrophysiology
    D. None of the above

Introduction

The International Classification of Headache Disorders (ICHD) identified more than 284 sources for headache and facial pain (HFP), making the evaluation and treatment of HFP a daunting task.[1] Dental, neurologic, musculoskeletal, otolaryngologic, vascular, metaplastic, infectious disease, neuropathic, and neurogenic are potential sources for HFP. The management of HFP can involve several health care professionals, which may include a dentist, oral surgeon, physical therapist, and physicians of different specialties. The ICHD lists TMD and neck pain as 2 sources for HFP. TMD and neck pain are musculoskeletal disorders that will be the focus of this chapter and best managed by a physical therapist.

TMD is divided into myogenous disorders involving the muscles of mastication and arthrogenous disorders involving the TMJ.[2] Myogenous and arthrogenous each have additional diagnostic subsets that are listed in Appendix A. It is estimated that up to 12% of the population experience signs and symptoms of TMD.[3] Common sources for neck pain consist of any one or combination of cervical disks, nerve roots, facet joints, and associated muscles. Generic terms used to describe involvement of the previous cervical tissues are *chronic uncomplicated neck pain*, *nonspecific neck pain*, *mechanical neck pain*, *cervical spine disorders (CSD)*, and *neck pain*. In this chapter, neck pain and CSD will be interchanged to indicate symptoms originating from the cervical spine. It is estimated that up to 14% of the population experience signs and symptoms related to neck pain.[4]

Little information is available for costs of services related to TMD. In 1995, the annual cost for treating TMD and orofacial pain was estimated to be $32 billion.[5] In 1994, neck and back pain estimated medical expenditures was $33.6 billion.[6] One can only imagine what the annual costs for TMD and neck pain treatment may be composed of in today's dollars. In contrast to other medical conditions, the National Heart, Lung, and Blood Institute estimated in 2002 that the cost of treatment for diabetes was $98.1 billion, and in 2007, the cost of treatment for cancer was $89.0 billion.[7]

The emphasis of this chapter is on physical therapists as a health care provider that offers a conservative, evidence-based, and cost-effective treatment for TMD and CSD. The first part of this chapter discusses the diagnostic criteria and management for TMD followed by a discussion on predoctoral education for health care professionals pertaining to TMD. The last part of the chapter is an overview of cervical spine considerations for patients with TMD and HFP. Mechanisms by which the cervical spine contributes to TMD myalgia, headache, and ears symptoms will be reviewed. Cervical spine evaluation and management will be highlighted.

Table 14-1

Red Flags

Suspect pathology with any of the following red flags:
• Blunt trauma to jaw and/or neck
• Osteoporosis with minor or no trauma
• Neurologic signs (cranial nerve examination positive)
• Dysarthria, dysphagia, diplopia, drop attacks
• Less than 17 years old with stiff neck, headache, and fever in the absence of trauma suggest meningitis
• Unremitting night pain, fever, unexplained weight loss within 6 months or night sweats
Imaging studies and erythrocyte sedimentation rate are highly sensitive and specific to diagnose fracture, instability, infections, and cancer to rule out such pathology.[10]

Temporomandibular Disorders

Diagnosing Temporomandibular Disorders

Imaging

Imaging studies of TMJ include, but are not limited to, a panoramic radiograph, computed tomography, and magnetic resonance imaging (MRI).[2] The panoramic radiograph is the most widely used imaging in the dental office. The panoramic radiograph provides a broad view of the mandible, teeth, sinuses, nasal area, and TMJ. Computed tomography scanning provides detail of bony structures. The cone beam allows for viewing the condyle in multiple planes so all surfaces can be visualized. Cone beam tomography can image both hard and soft tissues.[8] MRI evaluates the soft tissue of TMJ. MRI has become the gold standard to diagnose TMJ disk displacements.[9]

The primary advantage of imaging studies is to rule out fractures and disease that are suspected with red flags (Table 14-1).[10] Pertinent to TMD, imaging cannot differentiate patients who are in pain from those who are not in pain.[9] Technology has allowed us to see in detail TMJ and adjacent tissues, but seldom does it change the initial treatment plan or lead to a better treatment outcome. Routine imaging of TMJ will overdiagnose arthrogenous conditions that may not be related to the patient's pain and would therefore not require treatment.[9] An example is a disk displacement, which is a common TMJ diagnosis. MRI studies have repeatedly been shown to identify one-third of asymptomatic volunteers with disk displacement of their TMJ.[11,12] Imaging of TMJ is mainly indicated when red flags (see Table 14-1) are present or if the patient's pain stemming from the TMJ has not responded to conservative care and TMJ surgery is being considered.[12]

Computerized Instruments

Computerized instruments include sonography to measure vibration stemming from TMJ, electromyography to measure the activity of jaw muscles, and electronic tracking instruments to record mandibular position and movement 3-dimensionally.[13] Data produced from the computerized instruments are used to make a TMD diagnosis and the structural design of an oral appliance.[13-15] Unfortunately, computerized instruments overdiagnose patient's with TMD and any subsequent treatments would likely be unnecessary.[13-15] According to the American Association for Dental Research's Policy Statement on Temporomandibular Disorders:

> [T]he consensus of recent scientific literature about currently available technological diagnostic devices for TMDs is that except for various imaging modalities, none of them shows the sensitivity and specificity required to separate normal subjects from TMD patients or to distinguish among TMD subgroups.[16]

Occlusal Studies

Occlusion as it relates to TMD is one of the most debated topics in dentistry. In 1934, Costen made an observation that certain symptoms, such as loss of hearing, tinnitus, dizziness, headache, and a burning sensation of the throat and tongue was the result of a dental malocclusion.[17] Ninety five percent of the population have some form of malocclusion (eg, crowding, malalignment, structural abnormality).[18] Costen's fundamental observation of cause and effect was extended by many dentists to include malocclusion as an etiology for TMD, to justify occlusal equilibration, prosthodontics, and orthodontics in the diagnosis, prevention, and treatment of TMD.[19-25] Studies have concluded that a malocclusion is not an etiology of TMD; malocclusion cannot diagnose TMD, and occlusal treatment cannot prevent or treat TMD.[20-26] Although a very limited number of patients diagnosed with

TMD could benefit from occlusal treatment, the author of this chapter is not aware of any published guidelines that have a broad consensus among academic and clinical dentists that clearly identifies the criteria for when occlusal therapy should be offered for patients suffering from TMD. Okeson summarizes the state of the occlusion and TMD in the following way:

> One might conclude that if occlusion were the major etiologic factor in TMD, the profession would have confirmed this many years ago. On the other hand, if occlusion has nothing to do with TMD, the profession would have also likewise already confirmed this conclusion. Apparently neither of these conclusions is true. Instead, the confusion and controversy concerning the relationship between occlusion and TMD continues. The general message is that there is no simple cause-and-effect relationship explaining the association between occlusion and TMD.[27]

Box 14-1.

The following forms can be obtained by going to www.tmdstevekraus.com/tmd-course-forms/

- TMD History
- TMD Examination
- TMD Pain Screener
- Jaw Functional Limitation Scale (JFLS)
- Oral Behaviors Checklist
- Patient Health Questionnaire (PHQ-9)
- Two-item Graded Chronic Pain Scale (GCPS)
- Soft Diet
- Home Care for Jaw and Neck Related Symptoms

Clinical Examination

In 1992, the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) was established and has become the most widely used diagnostic criterion for TMD in academic and clinical research.[28] RDC/TMD diagnostic criterion is divided into axis I, which consists of the history and physical examination, and axis II, which assesses psychosocial distress.[28] In 2014, the RDC/TMD was updated and renamed to the DC/TMD.[29] The 12 diagnostic subsets of TMD consist of 5 myogenous and 7 arthrogenous diagnostic subsets. For the purpose of this chapter, local myalgia, myofascial pain, and myofascial pain with referral will be placed under the diagnostic subset of myalgia. Myalgia and headache attributed to temporomandibular disorder (HTMD) will be the 2 myogenous diagnostic subsets discussed in this chapter. History and physical examination along with sensitivity and specificity values for all myogenous and arthrogenous diagnostic categories can be found in Appendix A.

Many patients present with one or more TMD diagnostic subsets.[2,30,31] The DC/TMD does not account for patients having concurrent diagnostic subsets of TMD. Clinicians may need to modify the DC/TMD, using their clinical experience and clinical reasoning to arrive at a valid diagnostic subset of TMD. With several exceptions, the author of this chapter closely follows the DC/TMD.[30,32] Clinical Examination forms used by the author to document the history and physical examination for TMD can be found in Box 14-1.

History

Common TMD symptoms are:

- Facial, jaw, and/or ear symptoms
- Headache of any type located in the temple(s)
- The following symptoms may or may not be accompanied by pain:
  - Joint sounds with jaw movement
  - Intermittent locking on opening
  - Intermittent locking on closing from a wide-open mouth position
  - Unable to bring back teeth into full occlusion
  - Unable to close from a wide-open mouth position
  - Limited mouth opening but not severe enough to interfere with opening of the mouth to eat and yawn
  - Limited mouth opening that is severe enough to interfere with opening of the mouth to eat and yawn

Pain and symptom will be interchanged throughout this chapter. The history portion of axis I documents functional or parafunctional activities that may modify common TMD symptoms. Symptom frequency (constant, daily, or weekly) and pain intensity, using the Visual Analog Scale 0 to 10, will be documented. Documentation will include, but is not limited to, functional limitations associated with chewing, talking, and/or yawning. Functional limitations can be documented by completing the JFLS (see Box 14-1).[33] The JFLS assesses the patient's limitation in mastication, mobility, and communication. On subsequent visits, reassessing patient's symptoms (change in frequency and intensity) and functional limitations can provide information regarding patient's progress with treatments.

The history portion of the examination will also include axis II. Axis II assesses psychosocial distress. Psychosocial distress involves, but is not limited to, fear, anxiety, anger, and depression. Psychosocial distress can affect treatment outcomes by enhancing a patient's perception of pain contributing to an allodynia or hyperalgesia response to nociceptive input.[34] The PHQ is a short, reliable, and valid screening instrument for detecting "psychological distress"–related anxiety and/or depression (see Box 14-1).[35] The GCPS measures pain intensity and disability (see Box 14-1).[36] Kotiranta et al[37] did a study of 399 patients diagnosed with TMD to determine how often pain disability was magnified by psychosocial distress. The majority (61%) of patients fell into

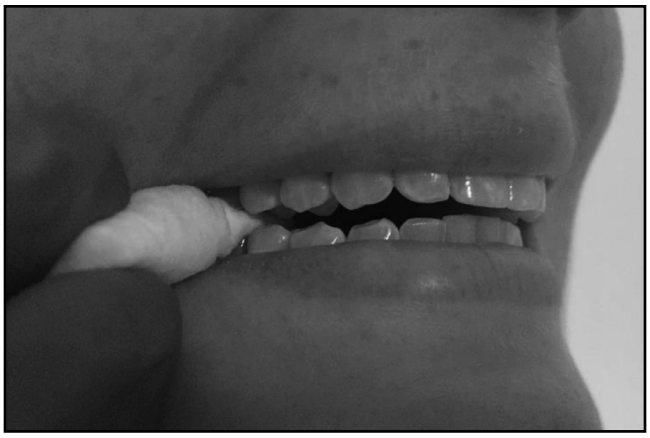

Figure 14-1. Bite Test. Patient bites on a dental roll or rolled up gauze that is placed between 2 opposing mandibular and maxillary molars. Biting with a firm force results in joint loading of the contralateral side. Joint loading does occur on the ipsilateral side but not to the same degree. A positive response is reproduction of the patient's familiar pain on the contralateral side stemming from the TMJ. Figure depicts joint loading of the left TMJ. Bite test requires jaw-closing muscles to contract. Although the bite test is used to assist in diagnosing arthralgia, if myalgia is also present, the bite test may elicit a myalgia response. Location of the familiar pain along with other tests used to diagnose arthralgia and myalgia will assist in making the diagnosis of arthralgia and/or myalgia.

the no-disability group, 27% to the low-disability group, with only 12% in the high-disability group. Myalgia is associated with bruxism, and bruxism is often found to be associated with psychosocial distress.[38] Patients with no disability to low disability often respond well to a conservative treatment program offered by a physical therapist and if indicated, an evidence-based oral appliance from a dentist. Patients with high psychosocial distress may not reach full potential regardless of treatments offered. Psychosocial distress is a complex topic. Incorporating the PHQ and GCPS or other methods to assess psychosocial distress can assist the clinician to identify patients with high psychosocial distress who may need to be referred to the appropriate health care professional.

Physical Examination

The physical examination consists of 3 parts: provocation tests, assessing mandibular dynamics, and assessing joint noises. The primary focus of the physical examination is to modify common familiar symptoms of TMD.

Provocation Tests. Provocation tests are done to modify the patient's familiar symptom(s). Provocation tests include palpation of the masseter and temporalis muscles, palpation over the lateral pole of the TMJ, maximum unassisted and maximum assisted mouth opening, mandibular protrusive and lateral excursions, and bite test. Active or latent myofascial trigger points can only be diagnosed with palpation. Active myofascial trigger points are a common source of TMD symptoms to include, but are not limited to, facial, jaw, ear, and headache pain. When an active trigger point is palpated, the patient's familiar symptoms would be modified (ie, increased).[39,40] An exception of reproducing familiar pain during the examination are latent myofascial trigger points. A latent myofascial trigger point is asymptomatic and can be quiescent for months or years but only becomes painful when stretched or compressed (palpated) during the physical examination.[39] Latent myofascial trigger points may be a source of referred pain and/or can perpetuate adjacent active myofascial trigger points (satellite myofascial trigger points).[40] Active satellite myofascial trigger points may become resistive to treatment unless the latent myofascial trigger points are first treated.[40] The definition of familiar pain would not apply to latent myofascial trigger points because latent myofascial trigger points are not painful at the time of the examination unless palpated. The DC/TMD does not account for latent myofascial trigger points in their diagnostic criterion for myalgia.

Bite test is described in Figure 14-1.[41] Knowledge of the anatomy will assist the clinician to determine what tissue is provoked and whether the involved tissue correlates to the patient's familiar symptoms and functional limitations. There are other provocation tests that the reader may find useful,[42] but at this time, they have not been incorporated into the DC/TMD. Regardless of the provocation tests used, the clinician needs to understand the strengths and limitations of each test.

Mandibular Dynamics. Mouth opening or interincisal opening (IO) is measured using a millimeter (mm) ruler. IO is the distance from the tip of an upper central incisor to the tip of the lower central incisor (Figure 14-2). DC/TMD includes overbite in their IO measurement. The author of this chapter does not add vertical incisal overlap (overbite) to the IO measurement. The rationale for not including overbite is that this relationship between the central incisors is constant and does not change during the time that a patient is receiving treatment. Measuring IO is a useful clinical measurement because it is the variable that is expected to change in response to intervention.[32] Not correcting for overbite reduces reliability concerns when measuring IO.[32]

Functional maximum unassisted IO ranges from greater than 30 mm to 40 mm or more. Maximum unassisted IO that is 30 mm or less represents a significant limitation in mouth opening.[30,32] Objectives for measuring maximum unassisted IO and maximum assisted IO mouth opening are listed in Figures 14-2 and 14-3. Opening of the mouth can occur in midline or the mandible can deflect (movement away from midline but does not return) or deviate (movement away from midline but then returns to midline). Boney asymmetries are common in the TMJ. Boney asymmetries consist of, but are not limited to, the shape of the mandibular condyles, variations of the long axis of the condyles, and angle of the articular eminences. If IO is functional, any associated deflection or deviation of the mandible may be the result of boney asymmetry and would not require treatment.

The DC/TMD recommends using a millimeter ruler to measure lateral and protrusive mandible movements. However, there are also reliability concerns in obtaining

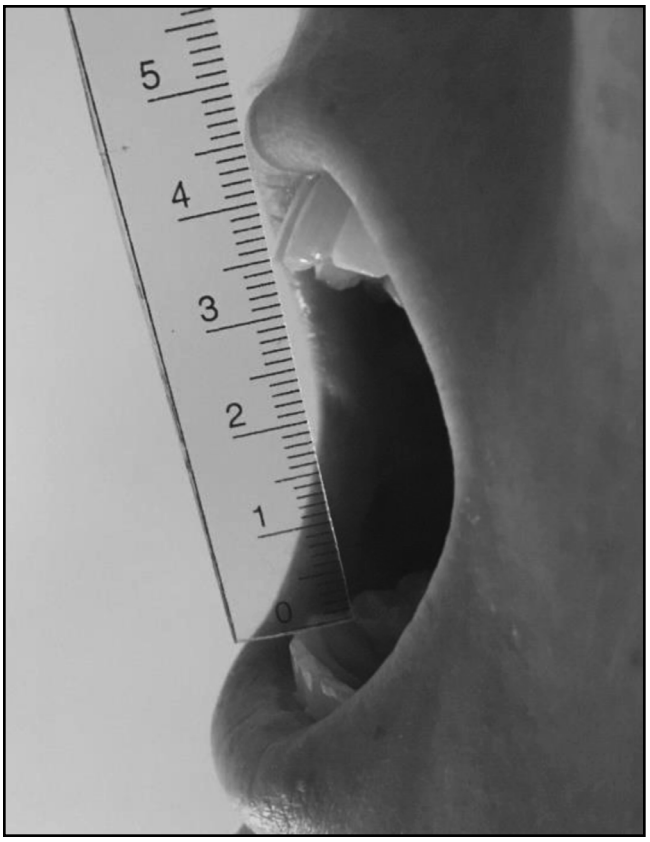

Figure 14-2. Measuring Maximum Unassisted IO. Objectives for measuring maximum unassisted IO: (1) to document maximum unassisted IO without pain, (2) to document maximum unassisted IO with pain, and (3) if pain on opening is present, assess if #2 modifies patient's familiar pain. Objectives 1, 2, and 3 are used as a baseline documentation to reassess the patient's response to treatments.

such measurements using a millimeter ruler.[30] The author of this chapter prefers to assess functional lateral excursions and functional protrusion by observing how the mandibular canines and central incisors move in relationship to the maxillary canines and central incisors (Figures 14-4 and 14-5). Ultimately, what the patient believes and feels with mouth opening, lateral excursions, and protrusion is as important as what the clinician can measure and observe.

Joint Noises. A noise originating from the TMJ will be heard by the patient. The clinician may or may not hear the noise. The clinician will palpate over the lateral pole of the condyle as the patient moves their mandible. If a noise is present, the clinician will feel a vibration created by friction between the tissues causing the noise. A click or crepitus are 2 noises associated with TMD. A click is required to diagnose a DDwR (Figure 14-6) and crepitus is required to diagnose DJD. Without a click or crepitus, a DDwR and DJD cannot be diagnosed by the clinical examination (Appendix A). A click may or may not occur with subluxation (see Appendix A). A noise that cannot be associated with a DDwR, DJD, or subluxation is an unclassified noise. An unclassified noise is likely related to a *deviation in form*, referring to any change in the articular surfaces of the TMJ that may result in a noise.[26]

In summary, imaging studies, computerized instruments, and occlusal studies are not necessary to make the diagnosis or determine treatment for all common TMD diagnostic subsets. These previous procedures will overdiagnose TMD, resulting in treatments that are not necessary and will only drive up the cost of care. What is available to medical, dental, and physical therapy professionals is a reasonably reliable and valid clinical examination that can diagnose all common diagnostic subsets of TMD (see Appendix A).[28,30,32] An accurate diagnosis that factors in psychosocial distress often leads to a better treatment plan and treatment outcomes.[28,43,44]

Treatment Guidelines for Temporomandibular Disorder

The first treatment guideline for TMD was published in 1982 by The President's Conference on the Examination, Diagnosis, and Management of Temporomandibular Disorders.[45] Since 1982, the American Academy of Oral Facial Pain (AAOP) has published 7 treatment guidelines for orofacial pain. The most recent guideline was published in 2023.[2] All guidelines agree that treatments are to be conservative and cost effective for all common diagnostic subsets of TMD. Conservative care consists of medication, oral appliance, behavioral modification, and physical therapy. However, this author believes that physical therapy is underrepresented in the AAOP guidelines, position papers, policy statements, and other scientific literature.

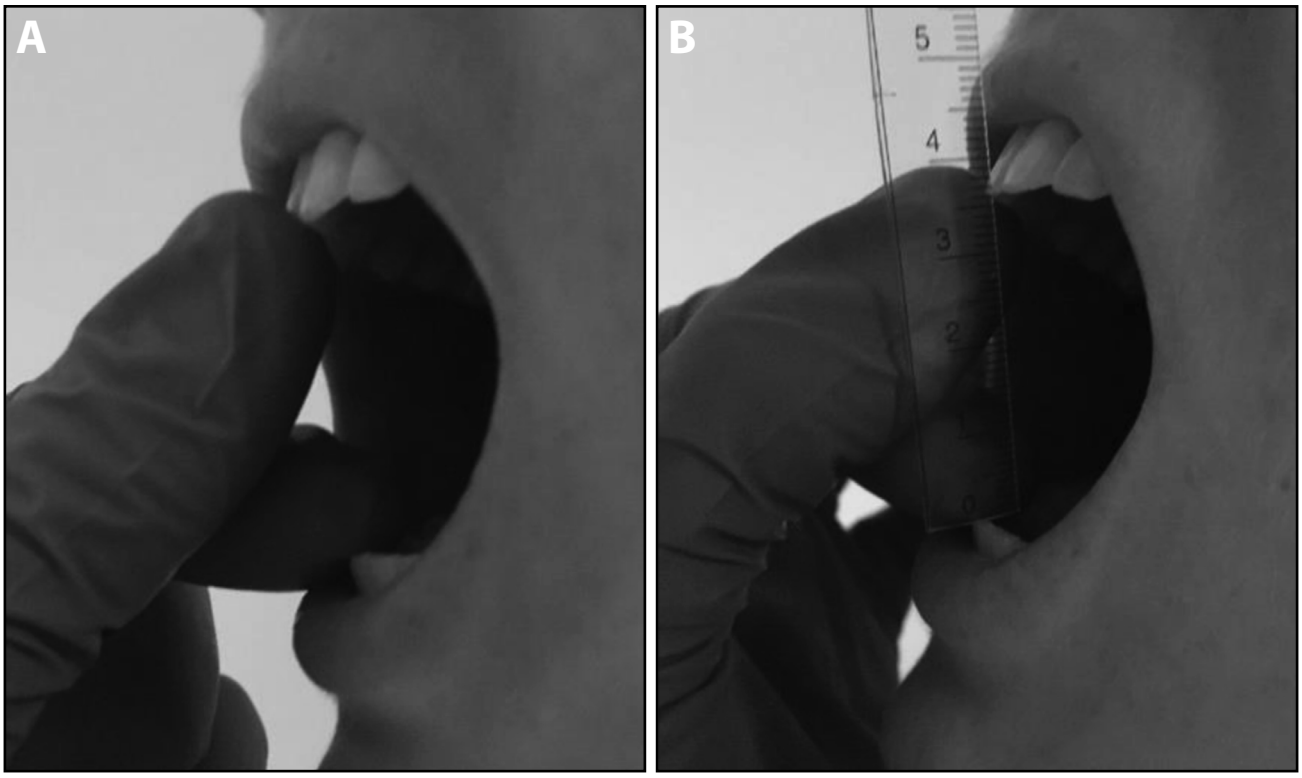

Figure 14-3. Maximum Assisted Mouth Opening. (A) Objectives for maximum assisted mouth opening (passive stretch): (1) Assess if this procedure modifies the patient's familiar pain and (2) assess end-range feel (ie, firm or springy). (B) With passive stretch, measure the maximum assisted IO using a millimeter ruler. Objective for measuring maximum assisted mouth opening: (1) to document IO. Passive stretch: Patient actively opens as wide as possible followed by the clinician pressing down on the patient's mandibular central incisors with the index finger as the thumb presses up on the maxillary central incisors. Objectives listed in A and B are used as baseline documentation to reassess the patient's response to treatments. Clinical points: (1) Maximum assisted opening should only be done if the patient has limited opening as determined by measuring maximum unassisted opening, and (2) maximum assisted opening should not be done if TMJ surgery or orthognathic surgery was recently done (depending on the surgery, this may vary between 1 to 3 months).

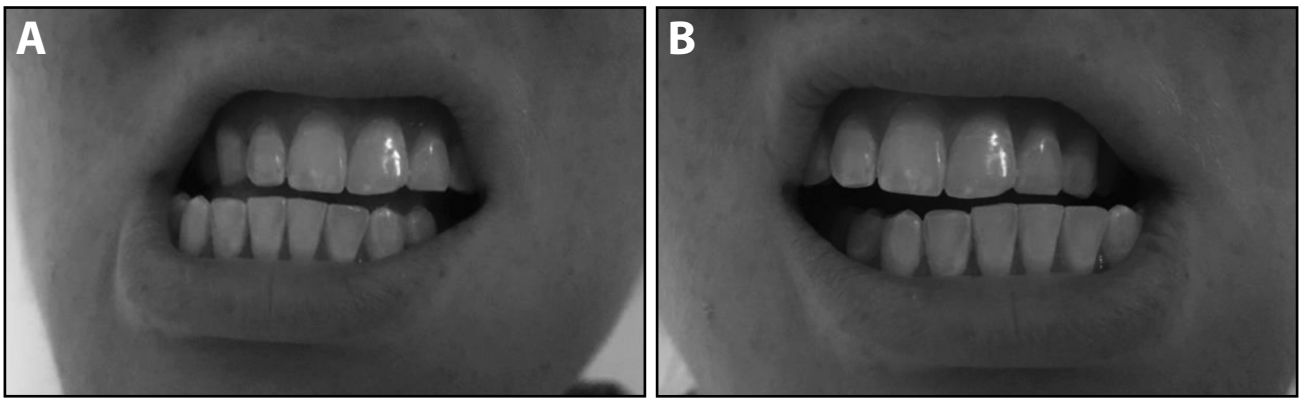

Figure 14-4. Functional Lateral Excursions. (A) Functional right lateral excursion. (B) Functional left lateral excursion. Functional lateral excursions are assessed by observing if the patient can move their mandibular canine past their maxillary canine on the ipsilateral side. Functional lateral excursions are necessary to chew food. If the patient cannot achieve an end-to-end position of their mandibular and maxillary canines, this is considered limited lateral excursion. Objectives for observing lateral excursions: (1) assess if lateral excursion(s) modify patient's familiar pain and (2) assess if patient has limited lateral excursion(s). Objectives are used as baseline documentation to reassess patient's response to treatments.

Clinical Point of the Clinical Examination

The DC/TMD requires pain to be present to make the diagnosis for myalgia and arthralgia. The DC/TMD does not require pain to be present to make the diagnosis for all other TMD arthrogenous diagnoses (ie, all disk displacements, DJD, dislocation, and subluxation). Knowing that arthrogenous diagnosis subsets may not be painful (exception is arthralgia) will guide treatment options and provide realistic expectations for treatment outcomes. Myalgia and/or arthralgia may represent separate mutually independent manifestations from other coexisting TMD arthrogenous conditions.[28,30]

Medication

Over-the-counter medications are available to patients. Although cost effective, over-the-counter medication taken for pain control has associated complications. Prolonged use of a nonsteroidal anti-inflammatory drug can cause gastrointestinal irritation.[46] Excessive use of acetaminophen, especially coupled with alcohol, can lead to liver damage.[46] Prescription medication such as a muscle relaxant have known complications such as dependency or can have severe interactions if taken with antihistamines or alcohol. Narcotics should seldom be given, and if given, the clinician must take ownership of overseeing the distribution, complications, abuse, and addiction that may arise. For more information on medication for TMD and HFP, the reader is referred to Kraus et al.[46]

Oral Appliance

The primary treatment offered by the dental profession for TMD is an oral appliance. An oral appliance is referred to by many different names with many different designs. Regardless of design, all oral appliances have some evidence as to their effectiveness but for reasons that are not known.[47] Evidence to justify the use of any oral appliance for any diagnostic subset of TMD is mixed.[48-50] All oral appliances have potential complications, which may include an increase in pain and/or movement of teeth.[47] Should a patient respond to wearing an oral appliance, it should not be assumed that a malocclusion was the etiology of the patient's symptoms and occlusal treatment is required.

Indications for an oral appliance are[50]:

- To reduce morning jaw muscle pain and/or headache (temples) related to myalgia and/or arthralgia triggered by sleep bruxism

Figure 14-5. Functional Protrusion. Functional protrusion is assessed by observing if the patient can move their mandibular central incisors past the maxillary central incisors. Functional protrusion is necessary to bite and for phonetics. If the patient cannot achieve an end-to-end position of their mandibular and maxillary central incisors, this is considered limited protrusion. Objectives for observing protrusion: (1) assess if protrusion modifies patient's familiar pain and (2) assess if protrusion is limited. Objectives are used as baseline documentation to reassess patient's response to treatments.

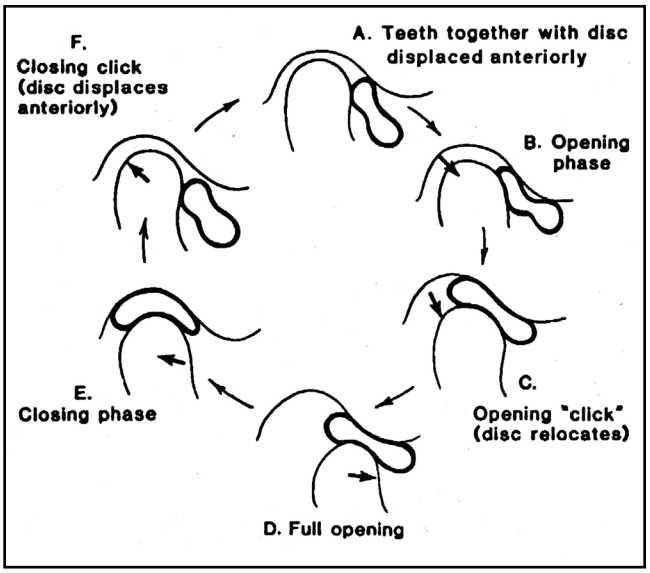

Figure 14-6. DDwR. The disk is displaced when the back teeth are together. As the mouth opens, an opening click occurs, indicating the disk has relocated on the condyle. As the mouth closes, bringing back teeth together, a closing click occurs. The closing click indicates the disk is displacing anterior or anteromedially to the condyle. (Reproduced with permission from Steve L Kraus. Evaluation and Management of Temporomandibular Disorders. Copyright 1993 by Steven L Kraus PT OCS. Reprinted with permission from: Evaluation, Treatment and Prevention of Musculoskeletal Disorders by H Duane Saunders, MS PT and Robin Saunders, MS PT. The Saunders Group Minneapolis, MN 55439.)

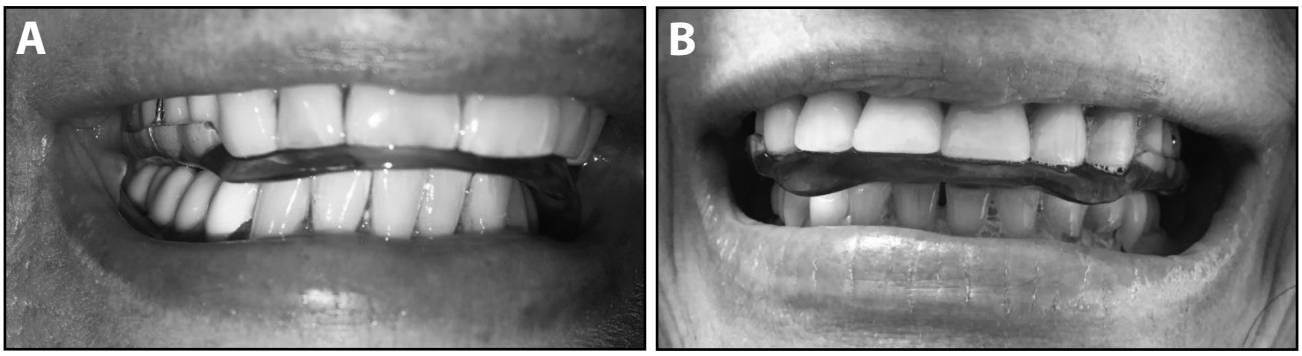

Figure 14-7. Examples of 2 oral appliances with evidence-based features.

- To reduce limited mouth opening in the morning due to myalgia or intermittent locking/catching associated with a disk displacement, both triggered by sleep bruxism
- To protect occlusal surfaces of teeth and dental restorations from sleep bruxism forces

To choose one oral appliance design over another may be based on minimizing complications from wearing an oral appliance than the effectiveness from wearing the oral appliance. An oral appliance with the following features has the best evidence for reducing myalgia and minimizing complications from prolong use (Figure 14-7)[47]:

- Made of hard acrylic
- Full coverage (can be maxillary or mandibular)
- Thin posteriorly (molar region)
- During unassisted closing, all opposing teeth hit evenly on the appliance with slightly heavier contact posterior than anterior
- Smooth (flat)
- Anterior guidance: As the anterior teeth (one or more) slide along the anterior portion of the appliance during protrusive and lateral excursions, the posterior teeth separate from the appliance

For patients wearing an oral appliance who consult with a physical therapist, the physical therapist needs to assess if the oral appliance has the features previously described. If the evidence-based features are not present, the physical therapist will need to call the dentist to discuss other options of an oral appliance design. Prior to calling the dentist, the physical therapist needs to assess how long the appliance has been worn, if the appliance is providing relief, and does the patient believe the appliance is changing their bite.

Physical Therapy

Treatment for all common diagnostic subsets for TMD should begin with a physical therapist, unless red flags are present or the examination is inconclusive. Physical therapy is cost effective and conservative.[30] Patient's TMD signs and symptoms often improve with physical therapy alone. After 4 to 6 physical therapy treatments, if the patient's signs and symptoms are not improving and there are indications for an oral appliance, the patient can be referred to a dentist for an oral appliance. Pain that is confined to the joint and is refractory to both physical therapy treatment and an oral appliance, a referral to an oral surgeon would be indicated.

Physical therapy treatment objectives for TMD are to eliminate or reduce pain with a return to unrestricted mandibular function regardless of joint noise, DJD, and disk displacement. Conventional physical therapy treatments for TMD are listed in Appendix C. Indications of modalities and procedures can be found in the references.[43,51-53] The following highlights several of the conventional physical therapy treatments.

Patient Education

Patients will be educated on their TMD diagnosis, treatment objectives, other treatment options, and treatment expectations. Patient education will address inaccurate or misleading information regarding the etiology, diagnosis, and treatment of TMD. Misinformation may come from family members, friends, the internet, and, unfortunately, from health care professionals. Patient's psychosocial distress may be unnecessarily enhanced or be the result of misinformation.[48] It is essential that the physical therapist addresses misinformation and provides a "biopsychosocial message" of encouragement to the patient that will assure and reassure that a positive treatment outcome can be achieved.[54]

Behavioral Modification

Behavioral modification is defined as the process of changing unwanted behavior that contributes to an overuse, disuse, and/or abuse of the muscles of mastication and/or TMJ. Treatment success is largely dependent on reducing or eliminating oral parafunctional activity. Oral parafunctional activity includes habitual use of the mouth unrelated to eating, drinking, yawning, or talking. A significant oral parafunctional activity is bruxism. Bruxism is believed to be the most common trigger for TMD myalagia. Bruxism can be diurnal or nocturnal that consists of repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible.[55] The etiology of bruxism is unknown.[55]

Diurnal Bruxism. Diurnal bruxism is best managed by educating the patient to be mindful that their teeth should not come into contact during their waking hours unless they are chewing or swallowing. The patient is instructed on keeping their tongue up and back teeth apart (TUTA).[51] TUTA is a very simple and effective self-awareness exercise to control diurnal bruxism. There continues to be discussions pertaining to the rest position of the tongue. Is the resting tongue position up against the palate of the mouth or down on the floor of the mouth? Wherever the resting position of the tongue is located, it should be a position that is maintained by the patient without effort and with back teeth slightly apart.

Bruxism also consists of bracing or thrusting of the mandible. This unwanted behavior is avoided by the "wiggle at will" (WW) exercise.[51] WW is performed by the patient moving their mandible left and right in a low amplitude movement. Only a few repetitions are necessary but performed many times throughout the day. Any pain or repetitive clicking is to be avoided. WW will be discontinued for patients who are not coordinated moving their mandible from side to side.

Patients must recognize triggers that may lead to bruxism. Common triggers consist of physical triggers (eg, lifting, reaching, pushing, pulling), focus triggers (eg, computer, driving, reading), emotional stress (eg, mental demands of work and home, interpersonal relationships, work pace), and psychosocial distress triggers (eg, fear, anxiety, anger, depression). When confronted with triggers, patients must focus on TUTA and WW.

Patients must eliminate oral parafunctional activities consisting of, but not limited to, nail biting, biting the inside of the cheek, or chewing gum or ice. Other activities to reduce, modify, or eliminate include chin leaning, eating hard/chewy food, singing, playing a musical instrument that involves pressure on the mandible, and sucking thick liquid through a straw.

Patients with TMD may concurrently have sleep apnea. Sleep apnea is considered to be one of many comorbidities of TMD myalgia. Sleep apnea may be treated by a sleep apnea appliance that repositions the mandible forward or by wearing a full-face continuous positive airway pressure (CPAP) mask that may apply retrusive pressure on the mandible. The sleep apnea appliance, or CPAP, can trigger bruxism and/or joint pain. Such devices may have to be modified or discontinued until the TMD symptoms become more manageable. Patients diagnosed with a disk displacement may be treated with an anterior repositioning appliance (ARA). As the name implies, an ARA repositions the mandible in a protrusive position. Like the sleep apnea appliance, an ARA can trigger bruxism and/or joint pain. If an ARA is worn for an extended period of time, the ARA can change the patient's occlusion, committing the patient to having orthodontic treatment.

Nocturnal Bruxism. Nocturnal bruxism is a challenge to control because the patient performs this unwanted behavior at night. Awake and sleep bruxism are not independent but interact additively.[56] Reducing diurnal pain and bruxism may help to reduce nocturnal bruxism.[56] Patients must avoid stomach sleeping and placing their hands under their mandible when sleeping on their side. Patients are encouraged to use a soft yet supportive pillow. A reduction in nocturnal bruxism may be helped if the patient were to increase aerobic exercise,[57] reduce caffeine, improve diet,[58] and avoid irregular sleep patterns due to lifestyle.[59] Although managing daytime pain and bruxism and improving sleeping postures may help reduce nocturnal bruxism, patients often require an evidence-based oral appliance (see Figure 14-7).

Manual Therapy, Therapeutic Exercise, and Modalities

There are a number of systematic and meta-analysis publications summarizing the evidence for physical therapy treatment for TMD patients.[60-64] A universal conclusion reached by the reviewers is that most randomized clinical trials reviewed were of poor methodological quality, investigated more than one physical therapy procedure or modality, investigated other treatments (oral appliance and medication) at the same time a physical therapy modality or procedure was investigated, and the definition of physical therapy in some studies included chiropractic, osteopathic, and massage therapists. The reviewers of this literature suggested conclusions be interpreted with caution. A generalization of all these studies suggests that active and passive exercise for the mandible, manual therapy, postural exercises, and neck exercises appear to have favorable effects for patients with TMD in the reduction of pain and improved IO.[60-64]

A systematic review and meta-analysis article published in 2016 looked at interventions of manual therapy, dry needling, and exercise therapy that were administered only by physical therapists.[65] The search identified 7 articles that met the inclusion criteria that were used in the analysis. The conclusion was manual therapy, dry needling, and exercise performed by physical therapists are more effective than other treatment modalities and sham treatments in reducing TMD pain and improving active mandibular ROM.[65] Conclusions were not definitive and should be reviewed with caution due to a small number of studies and variability of instruments used to assess the outcomes.

Clinical Points Pertaining to Temporomandibular Disorder Treatment

Point One

The TMJ is a load bearing joint.[66] Excessive and prolonged joint loading places stress on all components of the TMJ. Joint loading is reduced by eliminating or reducing parafunctional activities. Reducing TMJ loading often has a positive effect on concurrent arthrogenous diagnostic subsets. Reducing joint loading often results in arthralgia being reduced or eliminated, clicking related to a DDwR or subluxation becomes faint or undetectable, crepitus associated with DJD becomes faint or undetectable, and intermittent locking associated with a DDwR decreases in frequency or is eliminated. At the risk of oversimplification, focusing on reducing joint loading by managing parafunctional activities will help many patients with concurrent arthrogenous disorders.

Point Two

If reducing joint loading does not help to reduce the frequency or eliminate intermittent locking associated with a DDwR, a change in treatment protocol is required. Therapeutic exercises and intraoral mobilization techniques can be offered for the purpose of increasing mouth opening while preventing the disk to relocate during mouth opening. The objective of these techniques is to progress a DDwR with intermittent locking to a DDwoR woLO. This is acceptable for the following reasons:

- MRI studies of the TMJ have identified one-third of asymptomatic volunteers have unilaterally or bilaterally a DDwoR woLO.[10,11]
- In their simplest application, arthrocentesis and arthroscopy are successful in the reduction of TMJ pain and improving mouth opening in patients diagnosed with a disk displacement. However, the success of these 2 procedures is not dependent on relocating a displaced disk. Following these 2 surgical procedures, patients often have a DDwoR woLO[67,68]

Point Three

Patients with a DDwoR wLO (Figure 14-8) can, over time, improve their mouth opening without any treatment.[69,70] Patients often pursue treatment because this condition severely limits mouth opening while brushing teeth, eating, and yawning. A systematic review examined 20 studies that utilized different treatment options for a DDwoR wLO.[71] Treatment options were divided into 3 groups: noninvasive, consisting of patient education, self-management, physical therapy, and splint therapy; minimally invasive, consisting of arthrocentesis; and invasive, consisting of arthroscopy and arthrotomy. The comparable therapeutic effects of the 3 groups of interventions identified physical therapy as one of the simplest, least costly, and least invasive interventions for the management of a DDwoR wLO. Kraus and Prodoehl[72] investigated physical therapy treatment outcomes for 97 patients diagnosed with a DDwoR wLO. This study concluded that physical therapy treatment provided a positive trend for long-term benefits of pain reduction, improved active IO, and patient satisfaction for patients diagnosed with a DDwoR wLO.[72]

Point Four

DJD (osteoarthrosis) has been identified in patients whose disks are displaced and in patients whose disks are not displaced.[73,74] Degenerative changes of the TMJ may be dependent on the balance between a patient's adaptive capacity and functional loading or excessive loading with or without a disk displacement.[74] The message to communicate to patients is that the disk does not have to be in place to have successful treatment outcomes of pain reduction and a return to function.[75]

Pre- and Postdoctoral Education of Health Care Providers for Temporomandibular Disorders

Clinician's knowledge of anatomy, physiology, mechanics, etiology, and pathophysiology will help in all aspects of the examination and treatment for TMD. A clinician's knowledge is dependent on their educational experiences. Unfortunately, at the time of this writing, there is no standardized curricula to guide predoctoral teaching across the dental, physical therapy, and medical professions. A 2007 study of predoctoral dental education on TMD found that only 3 dental schools described their TMD-related teaching situation as ideal.[76] The study concluded that dental professionals may not receive any entry-level training on diagnosing or managing TMD. It was not until 2020 that the Commission for Dental Accreditation (CODA) approved the

teaching of TMDs in the predoctoral curriculums of all US dental schools by 2022.[77,78] The American Dental Association (ADA) did not recognize orofacial pain (to include TMD) as one of the 12 dental specialties until 2020.[79] With these changes made only recently by CODA and the ADA, it should not be a surprise there is a shortage of trained dental professionals who are competent in the management of TMD.[80]

A recent study examined self-perceived adequacy of entry-level TMD education of physical therapists from Florida.[81] This pilot study shows the lack of confidence of physical therapists in Florida to treat patients with TMD. A recent study investigated the status of entry level physical therapy education in the United States related to the diagnosis and management of TMD.[82] An email survey was sent to 224 accredited, entry-level US physical therapy programs. Of the programs that responded, this survey identified several barriers of the entry-level physical therapy programs suggesting not all entry-level physical therapists are ready to work with patients with TMD.[82]

Post-graduation, there are various avenues by which physical therapists can obtain professional development. Physical therapists can review the American Physical Therapy Association *Guide to Physical Therapy Practice*. The guide mentions TMD as a musculoskeletal condition causing pain but it does not provide any specific protocols for diagnosis or management of TMD.[83] The Orthopedic Clinical Specialist (OCS) certification is recognized by The American Board of Physical Therapy Specialties.[84] The OCS status is achieved by passing a multiple-choice format with no oral or practical testing. Only 3% of the questions on the OCS examination are related to the head, maxillofacial, or craniomandibular areas.[84] Orthopedic residency and fellowship programs in the United States and internationally provide limited educational experiences on the diagnosing and management of TMD.[85,86] There are a few postdoctoral educational opportunities for the physical therapist that leads to specialization in TMD.[87] The most common avenue physical therapists rely on for professional development are continuing education courses to maintain their clinical competency.[88] Peterson et al concluded that most continuing education courses (regardless of topic) do not incorporate current best evidence but may instead teach outdated or misaligned concepts, riddled with inefficiencies.[88] Physical therapists incorporating continuing education courses should take note of the concerns raised by Peterson et al when choosing continuing education courses on TMD management. To help in this short coming of professional development opportunities on TMD management, the American Physical Therapy Association is sponsoring the first TMD clinical practice guidelines to be published early in 2024.[89]

The consumer should know what to expect of the medical and other health care providers regarding their education on TMD. Medical doctors receive little to no training on

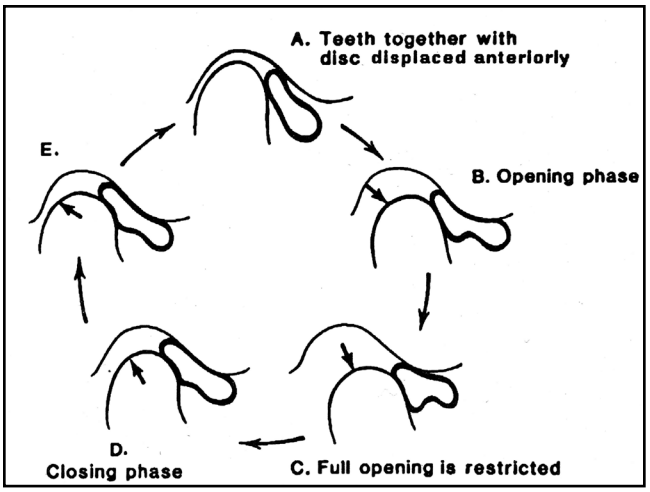

Figure 14-8. DDwoR. The disk is displaced when the back teeth are together. As the mouth opens, the disk remains displaced during full mouth opening and closing. Active IO less than or equal to 30 mm is associated with a DDwoR wLO. Active IO greater than 30 mm is associated with a DDwoR woLO. See Appendix A for additional diagnostic criteria. (Reproduced with permission from Steve L Kraus. Evaluation and Management of Temporomandibular Disorders. Copyright 1993 by Steven L Kraus PT OCS. Reprinted with permission from: Evaluation, Treatment and Prevention of Musculoskeletal Disorders by H Duane Saunders, MS PT and Robin Saunders, MS PT. The Saunders Group.)

the classification and management of TMD.[90,91] Greene and Bertagna[92] in their web search, found no evidence that TMD is taught as part of the regular curriculum in all medical and allied practitioners with professional degrees and clinicians with training in complementary alternative therapy. Greene and Bertagna concluded "physical therapists emerged as the only ancillary health care professionals with some positive aspects with regard to their training as well as their treatment approaches for TMDs."[92]

Predoctoral education on TMD for health care professionals is sobering. Postdoctoral TMD education becomes the responsibility of the dentist, physician, and physical therapist to be kept informed by attending scientific forums with an inquisitive mind and to read current scientific publications in peer-reviewed journals and textbooks on TMD. Despite the lack of continuity of the health care professional's education on TMD, the majority of patients with acute and chronic TMD do respond well to an evidence-based diagnostic and treatment approach offered by physical therapy and dental professionals. However, some patients continue to experience TMD and HFP. For nonresponding patients, investigating TMJ pathology, high pain disability, psychosocial distress, comorbidities, and other diagnoses contributing to HFP need to be considered. However, a very common missed or misdiagnosis for patients suffering from TMD and HFP is a CSD. Cervical spine influences on TMD and HFP will be discussed next.

Cervical Spine Considerations in Patients With Temporomandibular Disorders

Seventy percent of the population with TMD have neck pain.[30,93] Women experience both TMD and neck pain more than men.[30] The more pain and dysfunction due to TMD, the more pain and dysfunction exist in the cervical spine.[94] Bruxism is more common in patients who have myofascial pain in the masticatory and cervical spine muscles.[95] Neck and shoulder pain is more prevalent in patients who have a TMD myogenous involvement, than in patients who have a TMD arthrogenous involvement.[96] The previous studies and clinical observations suggest that cervical spine myalgia and masticatory myalgia may interact with each other and not just coexist with each other.

Cervical Neck Reflexes

The trigeminal cervical reflex (TCR) and asymmetric/symmetric tonic neck reflexes (TNR) are an elaborate set of reflexes between trigeminal and cervical areas. The TCR is mediated by a pathway comprising the trigeminal nerve and trigeminal spinal tract, which projects to motor neurons of the sternocleidomastoid muscle and posterior neck muscles.[97] The reflex is a defensive withdrawal of the head by contraction of neck muscles in response to facial stimuli.[98] Neck muscles are constantly providing orientation of the face in response to posture and movement.[99]

The TNR is a primitive reflex that exists in newborn babies and is still present in adults.[100] The origin of the TNR is in the upper 3 cervical segments (muscles and facet joints).[101] The TNR, in response to movement of the head and neck, modifies masticatory muscle activity. Funakoshi and Amano[101] observed the effects of the TNR on the tone of jaw muscles in rats. Muscle tone of the jaw muscles varied with flexion, extension, and rotation of the neck. When the first 3 cervical nerves were cut, the effects of head/neck movement on the jaw activity were abolished.[101] Other studies have demonstrated the TNR effects on jaw muscle activity.[102,103]

Functional Jaw Movements

A synergistic relationship exists between the cervical spine muscles and the muscles of mastication. Movement of the head-neck occurs with oral functions involving chewing, talking, swallowing, and yawning.[104] The TCR and TNR likely play an integral role in this synergistic relationship. Coordinated mandibular and head-neck movements during jaw opening/closing activities has been observed during chewing.[105] Each chewing cycle was accompanied not only by mandibular movements but also by head extension-flexion movements suggesting a close functional linkage between the jaw and the neck regions.[105] Zafar and colleagues completed 2 studies that found a high degree of temporal coordination between concomitant mandibular and head and neck movements during maximum jaw opening and closing tasks at both fast and slow speeds.[106,107] Ericksson and colleagues[108] describe this synergistic relationship between the cervical spine and muscles of mastication as "functional jaw movements" (FJMs). Functional jaw movements are the result of activation of jaw and neck muscles, leading to simultaneous movements in the temporomandibular, atlanto-occipital, and cervical spine joints.[108] When the head is fixated, reduced mandibular movements and shorter duration of jaw opening/closing cycles were observed.[109] The findings suggest a recruitment of neck muscles during jaw activities exists and head fixation can impair jaw function.[109,110] In a non-patient population, different head and neck postures have been shown to affect genioglossus muscle activity during swallow.[111] In a pilot study of 8 partipants, a change in head and neck posture affects the rest position of the mandible.[112] Other studies have shown that head and neck posture can affect the rest position of the mandible, resulting in a change in the trajectory of jaw closure and subsequently a change in the initial tooth/teeth contact.[113-115] Clinically, altered masticatory muscle activity occurs in response to diminished cervical movement and changes in head and neck posture.[116]

Neck Pain and Masticatory Muscle Activity

Myalgia is the most common diagnostic subset for acute or chronic TMD and CSD.[117] Patients with jaw and neck pain share common risk factors. Psychosocial distress is one of the most common risk factors contributing to an increase in myalgia of the cervical spine and jaw.[118,119] Another risk factor, often overlooked, that increases masticatory muscle activity is neck pain. Komiyama and colleagues induced experimental pain by injecting 0.5 ml of hypertonic (6%) saline in the trapezius muscle of 12 participants, 25 to 35 years of age.[120] In addition to pain referral over a wide area to include the temporomandibular region, there was a reduction in mouth opening.[120] Hu and colleagues[121] injected an inflammatory irritant into deep neck muscles surrounding C1-C3 of 19 anaesthetized rats, resulting in a sustained and reversible activation of both jaw and neck muscles. Carlson and colleagues[122] investigated 20 patients with upper trapezius myofascial trigger points and ipsilateral masseter muscle pain. Each patient received a single trigger point injection of 2% lidocaine solution in the upper trapezius muscle. After trapezius injection, there was a significant reduction in pain intensity and electromyography activity of the masseter muscle.[122] Myofascial trigger points in the muscles of mastication may be satellite trigger points and may be resistive to treatment until the primary myofascial trigger points in the cervical spine muscles are first treated.[40,123] A recent study using rats showed that by activating nociceptors of the trapezius muscle increased the level of calcitonin gene-related peptide in the trigeminal ganglion, which led to sensitization of the trigeminal system, concluding neck pain is a risk for masticatory muscle pain.[124]

Considering the previous studies, it should come as no surprise that there is a strong association between jaw disability and neck disability. Using the Jaw Functional Scale and the Neck Disability Index, Olivo and colleagues demonstrated that patients having more disability in the neck also have more jaw disability and vice versa.[125] Other studies have shown patients with TMD that had more limited mouth opening and tender points on palpation also exhibited significantly more cervical spine limitations and more tender points upon palpation of neck and shoulder muscles.[126-128]

Patients with myalgia of the muscles of mastication can no longer be viewed as a local disorder of the jaw. The cervical spine must be evaluated in patients with simple to complex symptoms stemming from the muscles of mastication and/or TMJ. Postural reeducation, manual therapy, and exercise directed at the cervical spine improves pain intensity, pressure pain sensitivity, as well as limited mouth opening in patients with masticatory muscle pain.[129-131]

Cervical Spine and Headache and Facial Pain

The ICHD identifies the cervical spine as a second of 2 musculoskeletal sources for HFP.[1] A high occurrence of neck pain has been found in patients with HFP. Two hundred consecutive female patients referred to a university facial pain clinic were asked to mark all painful sites on sketches that showed contours of a human body in frontal and rear views.[132] Analysis of the pain distribution according to the arrangements of dermatomes revealed pain was not confined only to the region innervated by the trigeminal nerve. One hundred thirty-one patients had pain in the trigeminal area but also had widespread pain to include spinal dermatomes C2, C3, and C4. Patients with HFP often experience pain in the neck and shoulder areas.[133,134]

Cephalic symptoms such as headache, ear, and jaw symptoms can originate from the cervical spine. This referral mechanism is based on the established neurophysiological relationship of the upper 3 cervical nerves converging onto neurons of cranial nerve V as well as neurons from cranial nerves VII, IX, and X.[135-138] This region of convergence is in the upper cervical spinal cord within the pars caudalis portion of the spinal nucleus of the trigeminal nerve and is referred to as the *trigeminocervical nucleus* (Figure 14-9).[139] All pain and temperature originating from the head and face terminates in the trigeminocervical nucleus.[140] Cervical spine tissues innervated by C1, C2, and C3 spinal nerves that are responsible for cephalic symptoms are listed in Figure 14-10.[141]

Cervicogenic Headache

Sjaastad et al first identified CGH in 1983.[142] Cervicogenic symptoms are symptoms originating from the cervical spine and are felt by the patient in the head, face, jaw, and ear areas.

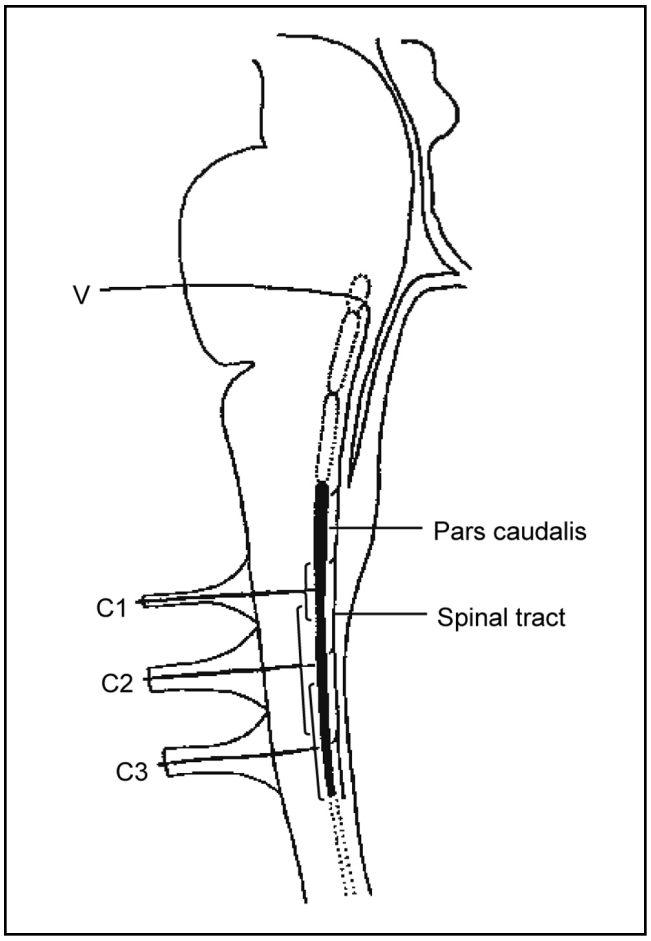

Figure 14-9. Afferent fibers from the trigeminal nerve (V) enter the pons and descend within the spinal tract of the trigeminal nerve to the upper cervical levels C1, C2, and C3. Trigeminal and cervical afferents constitute the trigeminocervical nucleus. (Reproduced with permission from Haldeman S, Dagenais S. Cervicogenic headaches: a critical review. *Spine J.* 1[2001]:31-46.)

Tissues innervated by C1, C2, and/or C3 can be the source of CGH. The prevalence of a CGH estimates range from 0.4% to 2.5% of the general population to 15% to 20% of patients with chronic headaches.[139] CGH is 1 of the 3 large headache groups; the other 2 are tension-type headache (TTH) and common migraine without aura.[143]

The clinical examination used to diagnose CGH is[144]:

1. Pain can be unilateral, but can be bilateral, localized to neck and occipital region. May project to forehead, orbital region, temples, vertex, or ears. ICHD indicates that "Migrainous features such as nausea, vomiting and photo/phonophobia may be present with [CGH]"[1]
2. Headache modified by at least one of the following:
   A. Neck movement and/or sustained head position
   B. Palpation of neck muscles (upper cervical, trapezius, and sternocleidomastoid muscles)
3. Restricted active or passive cervical ROM

Structure	Innervation		
	C1	C2	C3
Joints	Atlanto-occipital	Median atlantoaxial Lateral atlantoaxial	C2-3 zygapophyseal C2-3 disc
Ligaments	Transverse atlantoaxial and alar; membrana tectoria		
Muscles	Suboccipital	Prevertebral; sternocleidomastoid, trapezius Semispinalis, splenius	Multifidus; semispinalis
Dura	Upper spinal cord; posterior cranial fossa		
Arteries	Vertebral; internal carotid		

Figure 14-10. Possible sources of CGH listed according to innervation and type of structures. (Reproduced with permission from Bogduk N. Cervicogenic headache: anatomic basis and pathophysiologic mechanisms. *Curr Pain Headache Rep*. 2001;5:382-386.)

Cervicogenic Headache and Headache Attributed to Temporomandibular Disorder

There is considerable overlap of symptoms associated with CGH and HFP. Cervical spine tissues listed in Figure 14-10 can refer pain cephalically. The cervical spine tissue responsible for the majority of HFP originates from myofascial trigger points.[139] Myofascial trigger points located in the semispinalis capitis, longissimus and splenius capitis, sternocleidomastoid, and trapezius can all refer pain into the temple area.[145] As stated earlier, HTMD is a headache originating from the temporalis muscle and felt by the patient in the temporalis muscle.[28] A differential diagnosis will be required to know if the patient's headache located in the temple area is referred from the cervical spine or originating from the temporalis muscle. Treatment would then be directed toward the cervical spine muscles or temporalis muscle or both.

The next section highlights the overlap of CGH and HTMD with TTH and migraine

Tension-Type Headache

TTH is the most common of all headaches.[1] In 1972, Wolff believed that TTH was due to sustained contraction of the pericranial muscles contributing to ischemia of the muscle, and pain causing a pressure or band-like tightness in the head.[146] The ICHD states "[i]ncreased pericranial tenderness recorded by manual palpation is the most significant abnormal finding in patients with [TTH]."[1] To diagnose TTH, the ICHD recommends the following muscles be palpated: frontalis, temporalis, masseter, pterygoid, and cervical muscles to include sternocleidomastoid, splenius, and trapezius muscles.[1] Patient's description of a pressure or band-like tightness and increased tenderness on palpation of jaw and neck/shoulder muscles resemble the descriptions of referred pain originating from myofascial trigger points.[146] Clinically, the majority of TTH are reproduced by palpating myofascial trigger points located in key muscles such as the temporalis, sternocleidomastoid, splenius, and trapezius.[147]

Tension-Type Headache and Cervical Spine. Sakai and colleagues[148] diagnosed 60 patients with TTH, and Ashina and colleagues[149] diagnosed 20 patients with TTH. Both studies concluded that tension in the trapezius muscle of patients with TTH was significantly greater than that in age-matched normal participants. In a controlled study of 20 participants diagnosed with TTH, all participants had myofascial trigger points in the suboccipital muscles.[150] Active myofascial trigger points in suboccipital muscles of 13/20 (65%) were associated with referred pain that reproduced the participants' TTH. Latent myofascial trigger points were identified in 7/20 (35%) participants with TTH. Headache intensity and frequency was greater in participants with TTH who had active myofascial trigger points compared to those with only latent myofascial points.[150]

Tension-Type Headache and Headache Attributed to Temporomandibular Disorder. There is a significant overlap between TTH and HTMD.[151,152] Forty percent of patients diagnosed with TTH will have a TMD myalgia (temporalis) diagnosis.[153] HTMD and/or TMD myalgia may be a pathogenesis for TTH.[154] Management of TTH will need to incorporate treatment of the myofascial trigger points located in the muscles of mastication, especially the temporalis.[153]

Migraine

Woman are diagnosed more with migraine than men.[155] Migraine with or without aura is an episodic headache lasting 4 to 72 hours with aura as the defining feature of migraine.[1] Migraine can be diagnosed with a certain degree of confidence if the patient responds positively to 2 or more of the following 3 items[156]:

1. Has a headache limited your activities for a day or more in the last 3 months? (disability)
2. Are you nauseated or sick to your stomach when you have a headache? (nausea)
3. Does light bother you when you have a headache? (photophobia)

Migraine and Cervical Spine. As stated earlier, "Migrainous features such as nausea, vomiting and photo/phonophobia may be present with [CGH]."[1] Up to 75% of patients with migraine report having neck pain or stiffness associated with their migraine attack.[157] Regardless of the intensity of an episodic or chronic migraine, the migraine is more likely to be accompanied by neck pain than by nausea.[158] Myofascial trigger points located in cervical spine muscles can trigger a migraine and have been considered to be a pathogenesis for migraine.[124,159-167] Patients with migraine should have their cervical spine evaluated and if indicated treated.

Migraine and Headache Attributed to Temporomandibular Disorder. Tenderness of the pericranial muscles (temporalis) during a migraine attack has been known for more than 70 years.[168] TMD myalgia (HTMD) is a risk factor for increased frequency of migraine and often leads to chronic migraine, whereas TMD arthrogenous diagnostic subsets are not a risk factor to migraine.[169] Although they may be separate problems, TMD myalgia and migraine might aggravate or sustain each other.[170] Garrigos-Pedron and colleagues[171] looked at 45 participants diagnosed with chronic migraine and TMD. Patients were randomized in 2 groups: a cervical group that received only physical therapy to the cervical region, and the orofacial group that received physical therapy to both the cervical and orofacial regions. Results showed that both groups reported significant improvement in pain and disability, but cervical and orofacial treatment was more effective than cervical treatment alone. Clinical implication is patients with migraine may benefit by treating coexisting TMD myalgia and cervical myalgia by a physical therapist.

Clinical Points

Peripheral and central mechanisms have been suggested as important components of TTH and migraine. Evidence suggests a strong myofascial component associated with TTH and migraine. What has yet to be determined is if the masticatory and cervical spine muscles are the result of a TTH or migraine or the pathogenesis of a TTH or migraine. If the latter is true, this would have strong implications for treatment directed toward the muscles of mastication and cervical spine for patients suffering from TTH or migraine.

Cervicogenic Somatosensory Ear Symptoms

Ear symptoms include ear pain, tinnitus, fullness, and subjective hearing loss. Ear symptoms can be primary otalgia (ear pathology present) or secondary otalgia (ear symptom not caused by primary ear pathology).[172] A source for secondary otalgia is the cervical spine. Once otolaryngologic diseases have been ruled out as the cause of ear symptoms, the focus needs to be on CSD and TMD as sources of secondary ear symptoms.[173] Kuttila and colleagues did an interview and examination of 100 participants with otalgia.[174] In this study, 91 participants had secondary otalgia and 9 participants had primary otalgia. It was determined that the 91 particpants with secondary symptoms had more signs and symptoms related to CSD than of TMD. Neck pain may be a predictor for secondary ear symptoms.[173] Patients with secondary otalgia should routinely have an examination of the cervical spine and TMD.[173,174] Ear symptoms originating from the cervical spine are referred to as *cervicogenic somatosensory ear symptoms (CSES).*[175] A common source for CSES is myofascial trigger points in the cervical spine muscles, especially the sternocleidomastoid muscle.[176-178]

The pathophysiology for subjective tinnitus, subjective fullness, and subjective hearing loss is more complex. Primary tinnitus (a pathology is present) represents only 1% of cases of tinnitus.[179] Ten percent of the population suffers from subjective tinnitus with a multifactorial etiology.[179] Subjective tinnitus, fullness, and hearing loss may be related to an increase in activity of the tensor veli palatine and tensor tympani. These are muscles of the middle ear and are innervated by the trigeminal nerve. One function of the tensor veli palatine is to open and close the eustachian tubes.[180] This muscle allows air pressure to equalize between the tympanic cavity and the outside air.[180] The tensor tympani originates from the cartilaginous portion of the eustachian tube and crosses the middle ear by a slender tendon and attaches itself to the manubrium of the malleus.[180] This muscle tenses the tympanic membrane by drawing the tympanic membrane medially to dampen noise.[180] Regarding the tensor veli palatine and tensor tympani, Ramirez et al states, "[t]hese are muscles of the middle ear although they are really muscles of mastication because they are modulated by motoneurons coming from the trigeminal motor nucleus."[181] The trigeminal motor neuron system may be modulated by cervical nociceptive afferent information resulting in an increase in muscle activity of the tensor veli palatine and tensor tympani. A high prevalence of somatosensory tinnitus is associated with the cervical spine, which infers cervical spine treatment may reduce tinnitus.[181-183] Abel and Levine demonstrated that patients with subjective tinnitus could change their tinnitus with forceful head and neck contractions.[184] In the same study, about 80% of nonclinical participants, who had ongoing tinnitus at the time of testing, could modulate their tinnitus with head and neck contractions. These findings support the concept of a neural threshold for tinnitus. The cervical spine may be a source for ear pain, subjective tinnitus, subjective fullness, and subjective hearing loss.

Cervical Spine Examination

A negative proviso is not a part of the diagnostic criteria for neck pain. However, if the patient's TMD and HFP (TTH or migraine) is not responding to medications, injections, or an oral appliance and the HFP is not better accounted for by another condition, the cervical spine needs to be considered. Clearly, if the patient is complaining of neck pain, the cervical spine will need to be evaluated and treated. Some patients may not complain of neck pain but instead complain

of stiffness and tightness in their neck. The objective of the cervical spine examination is to modify the patient's familiar symptoms (neck, upper extremity, headache, jaw, facial, and/or ear symptoms) through tests of provocation consisting of palpation for active/latent myofascial trigger points, active and passive ROM testing. The clinical examination described in Appendix B is only a portion of a more comprehensive clinical examination that can be performed by the physical therapist. Michiels and colleagues assessed the diagnostic value of passive segmental testing, adapted Spurling test, strength and endurance of the deep flexor neck muscles, and 16 muscle palpation sites for myofascial trigger points to diagnose cervicogenic tinnitus.[185] A group of 17 international physical therapists completed several surveys to arrive at a consensus of 11 most useful physical examination tests to identify cervical musculoskeletal impairments in patients with headaches.[186] Patients with neck pain should have an assessment for psychosocial distress. The PHQ and the GCPS questionnaires recommended in the TMD evaluation would be appropriate to use in a comprehensive evaluation for patients with neck pain.[35,36]

Imaging of the cervical spine is not necessary to diagnose neck pain. Asymptomatic patients have positive imaging findings of cervical spondylitis and cervical disk herniation in 25% of patients less than 40 years old and in 60% of patients greater than 40 years old.[187] Imaging studies of the cervical spine are required if cervical radiculopathy or myelopathy signs and symptoms are present or if red flags are identified (see Table 14-1).

Clinician's should not be quick to conclude a patient has anatomic alignment problems of their cervical vertebrae from visually observing forward head posture or from using surface measurements of head and neck posture. When participants judged to have extreme head and neck posture based on external appearance or surface measurements were then imaged, participants showed vertebral positioning in the upper cervical spine were within the normal population distribution.[188] There are not adequate studies available comparing patients with neck pain to the asymptomatic population in order to make conclusions about any specific physical dimensions of posture related to neck pain (ie, forward head posture).[189,190] Maintaining good posture in a sitting, standing, or sleeping position is important. However, patients are to be encouraged to move frequently their head, neck, and shoulders throughout the day (especially if the patient's occupation involves sitting) vs staying in one static correct posture. Future studies should investigate the dynamics of posture as it relates to neck pain, CGH, CSES, TMD, and HFP.

Treatment Guidelines for Neck Pain

Conservative care for acute or chronic neck pain consists of several treatment strategies tailored to the individual patient. Treatments to the cervical spine will reduce or eliminate CGH and CSES symptoms, and if related, a reduction or elimination of symptoms may be seen in patients diagnosed or misdiagnosed with TTH and migraine.

Conservative care consists of patient education and behavioral modification, especially in sitting and sleeping postures. Manual therapy includes soft tissue massage, facet joint mobilization/manipulation, and dry needling. Therapeutic exercise (strength, condition, and flexibility), modalities, and cervical traction will complete the conventional treatment approach for neck pain. It is beyond the scope of this chapter to discuss in detail, indications, and method of application of all interventions available to the physical therapist. The evidence suggests a combination of the previous treatment strategies provides the best relief for patients suffering from neck pain and associated cephalic symptoms originating from the cervical spine.[191-197]

Should the patient not respond to conventional physical therapy treatments, there are other nonsurgical treatment options available. These treatments may include medication, injections to include lidocaine and botulinum toxin, epidural injections, nerve root injections, facet joint denervation, stellate ganglion block, and sphenopalatine block. These procedures lack Level I and Level II evidence in achieving a positive treatment outcome for neck pain with or without radiculopathy.[198-205] Cervical spine surgery includes laminectomy and fusion. Long-term benefits of surgery when compared to conservative care have similar results. Three studies examined the effects of surgery and conservative care on pain for sensory loss and weakness in patients who had minimal to moderate cervical radiculopathy or myelopathy.[206-208] No differences were found in sensation or motor strength between patients who were treated surgically and those who were managed conservatively in follow-up examinations at 24 and 36 months.[206-208]

Conclusion

Many conditions listed by ICHD contribute to HFP.[1] This chapter focused on TMD and CSD as 2 musculoskeletal disorders contributing to HFP. The evidence clearly indicates a conservative and a cost-effective approach is required for nearly all patients diagnosed with common TMD and CSD. Physical therapists, with the necessary knowledge and skills, are best equipped to manage all common diagnostic subsets of TMD and CSD. Patients not responding to physical therapy will require management from the physician, dentist, and/or oral surgeon. Health care professionals working together and recognizing what each profession has to offer will achieve the best outcomes for patients suffering from TMD and CSD.

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Appendix A

History and Physical Examination for Common Temporomandibular Disorder Diagnostic Subsets

TEMPOROMANDIBULAR DISORDER MYOGENOUS DIAGNOSTIC SUBSETS

MYALGIA

Sensitivity 0.90 and specificity 0.99

History

- Patient reports pain located in the jaw, temple, and/or ear areas.
- Patient reports pain modified with jaw movement, function, or parafunction.

Physical Examination

Familiar pain is modified by

- Palpation of the temporalis and/or masseter muscles (palpation identifies active or latent myofascial trigger point[s]).
- Maximum unassisted active opening (opening may or may not be limited; see Figures 14-2 and 14-3).

HEADACHE ATTRIBUTED TO TEMPOROMANDIBULAR DISORDER

Sensitivity 0.89 and specificity 0.87

History

- Patient reports headache of any type in the temple.
- Patient reports headache, modified with jaw movement, function, or parafunction.

Physical Examination

Familiar pain (headache located in temple area) is modified by

- Positive examination for myalgia (headache felt in the temporalis muscle).
- Positive examination for arthralgia (during the examination, pain is referred from TMJ to temple area).

Clinical Points

- To make a diagnosis of HTMD, requires the patient to complain of a headache in the temple area confirmed by the diagnosis of myalgia of the temporalis muscle(s).
- HTMD can also be referred to the temple area from the TMJ. This requires that the patient's headache, located in the temple(s) area, be modified by the examination directed toward the TMJ and the diagnosis of arthralgia is confirmed. Arthralgia is not as common of a source for HTMD as is myalgia.
- Headache is not better accounted for by another headache diagnosis.

TEMPOROMANDIBULAR DISORDER ARTHROGENOUS DIAGNOSTIC SUBSETS

ARTHRALGIA

Sensitivity 0.89 and specificity 0.98

History

- Patient reports pain located in the jaw, temple, ear, or in front of ear.
- Patient reports pain modified with jaw movement, function, or parafunction.

(continued)

Appendix A (continued)

History and Physical Examination for Common Temporomandibular Disorder Diagnostic Subsets

Physical Examination
Familiar pain is modified by • Palpation of the lateral pole or around the lateral pole (done with back teeth together and slightly apart). • Maximum unassisted or assisted opening, protrusion, and/or right or left lateral excursion (see Figures 14-2 through 14-5). • Bite test (see Figure 14-1). *(continued)*

Appendix A (continued)

History and Physical Examination for Common Temporomandibular Disorder Diagnostic Subsets

Clinical Point

The following diagnostic subsets, *dislocation* and *subluxation,* are frequently misused. See Key Terms for the definitions of dislocation and subluxation. DC/TMD uses the term subluxation when their definition is actually describing dislocation.[29] This author has added his version of what the history and physical examination should consist of to diagnosis subluxation.

DISLOCATION

Sensitivity 0.98 and specificity 1.00

History

- Patient reports jaw locking in a wide-open mouth position even for a moment, so they could not close from a wide-open position. Closing could only be done by a self-maneuver or with assistance.

Physical Examination

- No physical examination is necessary if at the time of the examination, the patient is unable to close from a wide-open position. Locking that occurs intermittently can only be diagnosed by what the patient recalls in the history.

SUBLUXATION

No sensitivity or specificity values have been established.

History

- Patient reports when closing from a wide-open mouth position, their jaw catches and/or the patient may report that their jaw feels like it is "going out of place" toward the end of a wide-open mouth position and/or on closing from a wide-open mouth position.
- Patient may report a noise toward the end of a wide-open mouth position and/or a noise at the beginning of closing from a wide-open mouth position.

Physical Examination

Palpating over the lateral pole

- Opening wide and/or on closing from a wide-open mouth position, a judder* is detected.
- An eminence click** is felt at the end of a wide-open mouth position and/or the beginning of closing from a wide-open mouth position.

* Judder is a sudden change in mandibular movement toward the end of a wide-open mouth position and/or from the beginning of closing from a wide-open mouth position. Patient may state they feel their "jaw is going out of place." The more acute the angle is between the articular eminence and the articular tubercle, the more pronounced the judder may be.

** An eminence click occurs as the condyle translates past the articular crest on opening and/or on closing. The articular crest is the anatomical point on the temporal bone between the articular eminence and articular tubercle. The more acute the angle is between the articular eminence and the articular tubercle, the more pronounced the click may be.

Clinical Point

- Without a judder and/or eminence click, subluxation can still occur, but the examination is not sensitive enough to make the diagnosis.

(continued)

Appendix A (continued)

History and Physical Examination for Common Temporomandibular Disorder Diagnostic Subsets

DISK DISPLACEMENT WITH REDUCTION

Sensitivity 0.34 and specificity 0.92

History

- Patient reports a noise (click) emanating from the TMJ with jaw movement or function.

Physical Examination

- Palpating over the lateral pole of the condyle, a click is detected during jaw opening and/or closing movements (see Figure 14-6).
- Palpating over the lateral pole of the condyle, a click may or may not be detected during protrusive and/or right or left lateral excursion.
- If the first bullet point was positive, the elimination test* is positive.

* Elimination test. Patient opens past the open click, then closes in a protrusive position to bring central incisors end to end. With repeated opening and closing in a protruded position, the opening and closing clicks are eliminated (a positive elimination test).

Clinical Points

- An opening click can be early, intermediate, or late. Regardless where in range the opening click occurs, the closing click (often difficult to detect) occurs toward the end of closing.
- A click detected during protrusion and/or right or left lateral excursions without an opening click seldom occurs. It is the opening click with a closing click and a positive elimination test that confirms the diagnosis of a DDwR.

DISK DISPLACEMENT WITH REDUCTION WITH INTERMITTENT LOCKING

Sensitivity 0.38 and specificity 0.98

History

- Patient reports a noise with jaw movement or function.
- Patient reports jaw intermittently locks closed. Patient is unable to open wide without pressing on their jaw or moving their jaw in a lateral or protrusive manner to get it to unlock.

Physical Examination

- Positive findings for DDwR.
- Intermittent locking on opening is observed. If not observed, a patient will need to report having had intermittent locking within the last 30 days.

DISK DISPLACEMENT WITHOUT REDUCTION WITH LIMITED OPENING

Sensitivity 0.80 and specificity 0.97

History

- Patient reports having had TMJ noise (clicking) with or without intermittent locking.
- Patient reports being limited in mouth opening severe enough to interfere with ability to eat, yawn, brush teeth, etc. Patient may refer to their jaw being locked closed (see Figure 14-8).

Physical Examination

- Maximum active unassisted IO 30 mm or less (see Figures 14-2 and 14-3).

(continued)

Appendix A (continued)

History and Physical Examination for Common Temporomandibular Disorder Diagnostic Subsets

Clinical Points
What may or may not be observed during active jaw movement • Deflection during opening to side of the involved joint. • Deflection during protrusion to side of the involved joint. • Decrease in lateral excursion to the opposite side of the involved joint.

DISK DISPLACEMENT WITHOUT REDUCTION WITHOUT LIMITED OPENING
Sensitivity 0.54 and specificity 0.79
History
• Patient reports having had a history of TMJ noise (clicking) with or without intermittent locking. • Patient reports having had limited mouth opening severe enough to interfere with ability to eat, yawn, brush teeth, etc. Over time, their mouth opening improved.
Physical Examination
• Maximum active unassisted IO greater than 30 mm (see Figure 14-2).
Clinical Point
• This diagnosis relies on the patient's memory. This diagnosis cannot be made by the clinical examination if the patient cannot recall having a history as described earlier. Without a clear history, imaging would be required to make the diagnosis. However, imaging is not necessary because this stage of disk displacement is often not painful or limits jaw function.

DEGENERATIVE DISK DISEASE
Sensitivity 0.55 and specificity 0.61
History
• Patient reports a "grinding" noise (crepitus) emanating from their TMJ with jaw movement or function.
Physical Examination
• Palpating over the lateral pole of the condyle, crepitus is detected during jaw opening and closing movements and/or protrusion and/or right or left lateral excursion.
Clinical Point
• Without crepitus being detected, this diagnosis cannot be made, and imaging would be required to make the diagnosis. However, imaging would only be necessary if patient's TMJ pain (diagnosis of arthralgia with or without a diagnosis of a disk displacement) is not responding to physical therapy and an oral appliance. Then imaging with a referral to an oral surgeon is indicated.
Sensitivity and specificity values for TMD diagnostic subsets are from Schiffman et al.[29] History and physical examination for TMD diagnostic subsets, except for subluxation, are based in part on but with modifications from Schiffman et al.[29]

Appendix B

History and Physical Examination for Cervical Spine Disorder Contributing to Headache and Facial Pain Symptoms

CLINICAL EXAMINATION FOR CERVICAL SPINE DISORDERS

History

- Patient reports neck symptoms to include pain, stiffness, and/or tightness in the occipital, neck, shoulder, and/or upper extremity areas.
- Patient reports neck symptoms are modified by any one or combination of
  - Static activities involving sitting (eg, computer, driving) and sleeping.
  - Dynamic activities to include pushing, pulling, lifting, and reaching.

Physical Examination

Familiar symptoms are modified by

- Active and/or passive cervical flexion, extension, rotation, and/or side-bending.
- Palpation of the sternocleidomastoid, trapezius, levator scapulae, suboccipitals, and/or posterior midline cervical muscles.

CLINICAL EXAMINATION FOR CERVICOGENIC HEADACHE

History

- Patient reports pain localized in the neck and occipital region to project to the forehead, orbital region, temples, vertex, or ears.
- Patient reports headache is modified by any one or combination of
  - Static activities involving sitting (eg, computer, driving) and sleeping.
  - Dynamic activities to include pushing, pulling, lifting, and reaching.

Physical Examination

- Headache is modified by provocation tests as described in the physical examination for CSD.

Clinical Points

- Depending on tissue irritation, a reproduction or an increase in the CGH may or may not occur during the physical examination, but the patient reports headache has occurred in the past 30 days with the headache being modified by what was described above in the history.
- The headache is not better accounted for by another headache diagnosis.

(continued)

Appendix B (continued)

History and Physical Examination for Cervical Spine Disorder Contributing to Headache and Facial Pain Symptoms

CLINICAL EXAMINATION FOR CERVICOGENIC SOMATOSENSORY EAR SYMPTOMS
History
• Patient reports having ear symptoms (pain, tinnitus, fullness, hearing loss) that coincide with neck symptoms and/or a CGH. • Patient reports ear symptoms are modified by any one or combination of ◦ Static activities involving sitting (eg, computer, driving) and sleeping. ◦ Dynamic activities to include pushing, pulling, lifting, and reaching.
Physical Examination
• Ear symptoms are modified by provocation tests described in the physical examination for CSD.
Clinical Points
• Depending on tissue irritation, a reproduction or an increase in ear symptoms may or may not occur during the physical examination, but the patient reports ear symptoms have occurred in the past 30 days with ear symptoms modified by what was described above in the history. • The ear symptoms are not better accounted for by another diagnosis.

Appendix C

Intervention Strategies Used in the Individualized Treatment Plan for Patients With Temporomandibular Disorder

TREATMENT STRATEGY	DESCRIPTION
Patient education	Patient education involves many facets. Fundamental to patient education is an explanation of their diagnosis, treatment objectives, expectations of treatment, frequency, cost, and expected number of treatments. Education to reduce unnecessary psychosocial distress due to misinformation is essential to achieve optimal treatment outcomes. The patient is educated on other treatment options that are available for TMD. This includes medications, oral appliances, and surgical interventions. Pros and cons of all treatments are discussed based on the available scientific evidence.
Behavioral modification	Behavioral modification is defined as the direct changing of unwanted behavior. Behavioral modification incorporates cognitive awareness exercises such as TUTA and WW. Being mindful to focus on the relaxation of the muscles of mastication will help to reduce diurnal bruxism. TUTA and WW are applied all day, especially when experiencing triggers for bruxism. Triggers include physical, focused, emotional, and psychosocial distress triggers. Eliminating harmful parafunctional activities including gum chewing, chewing ice, and fingernail biting is essential. The patient is to avoid hard/chewy food but to focus on a nonpainful diet. Behavioral modification also includes the correction of poor sitting and sleeping postures.
Therapeutic exercise	Therapeutic exercise is defined as any exercise performed with the aim of improving a single parameter, such as strength, range of motion (ROM), flexibility, or endurance. Depending on the TMD diagnostic subset, therapeutic jaw exercise can consist of active, active assistive, and/or passive jaw exercises with the goal of improving ROM to achieve functional mandibular dynamics. Jaw strengthening exercises are seldom, if ever, needed.
Neuromuscular reeducation	Neuromuscular reeducation is defined as the reeducation of movement, balance, kinesthetic sense, posture, and proprioception. Mandibular kinesthetic and proprioceptive exercises are used to enhance self-awareness of jaw movement and position.
Manual therapy	Manual therapy consists of soft tissue mobilization, joint mobilization, and dry needling. Soft tissue mobilization: The movement of contractile or inert tissues in such a way as to effect change in that structure or its related elements. Targeted tissues are the muscles of mastication. Joint mobilization: The act of moving articular structures generally performed passively by the physical therapist, with appropriate positioning to facilitate the intended movement. Intraoral techniques directed toward the head of the condyle consist of arthrokinematic techniques to create movement of joint distraction, condylar translation, and lateral glide (a joint play movement). Intraoral joint mobilization may be used for disk displacements or for capsular tightness due to trauma to the mandible and/or immobilization that occurs post-TMJ or post-orthognathic surgery. Dry needling: Is the insertion of a solid filiform needle into a latent or active myofascial trigger point.

(continued)

Appendix C (continued)

Intervention Strategies Used in the Individualized Treatment Plan for Patients With Temporomandibular Disorder

TREATMENT STRATEGY	DESCRIPTION
Modalities	Continuous ultrasound energy is absorbed in tissues with high collagen content. Used to heat tissue that has shortened or scarred down. Stretching can be done during or after to improve flexibility. Primary use is for chronic capsular tightness due to trauma and/or immobilization that occurs after trauma to the TMJ, TMJ arthrotomy, or orthognathic surgery. Pulsed ultrasound facilitates healing in the inflammatory and proliferative phase and is used for transdermal transport of anti-inflammatory medications (ketoprofen) referred to as *phonophoresis*. Primary use is for TMJ arthralgia or arthralgia after TMJ arthrocentesis, arthroscopy, and arthrotomy. Iontophoresis is the process by which drugs, usually anti-inflammatory in nature, are introduced to a small body part via direct electrical current. It is noninvasive, painless, and eliminates potential side effects and adverse reactions that can occur with medications delivered orally or by injection. Iontophoresis is used primarily for TMJ arthralgia but can be used to treat myalgia of the masseter muscle. Interferential stimulation is a type of electrical stimulation used for the control of pain. Interferential stimulation is believed to penetrate to deeper tissues than other forms of electrical stimulation such as transcutaneous electrical nerve stimulation. At higher frequencies, there is a decrease in skin resistance with interferential stimulation allowing the patient to tolerate interferential current better than transcutaneous electrical nerve stimulation, especially when applied over the masseter and/or TMJs. To avoid the crossover effect as with true interferential, premodulated interferential stimulation is used. When applied over the masseter muscles, a premodulated interferential stimulation with an intermittent setting of 10 to 15 seconds on and 10 to 15 seconds off is preferred by the author of this chapter. The intermittent current cues the patient when to perform active, active assistive, passive, and/or cognitive awareness exercises at the same time receiving the benefits from premodulation interferential stimulation of a reduction in pain, edema, and myalgia.