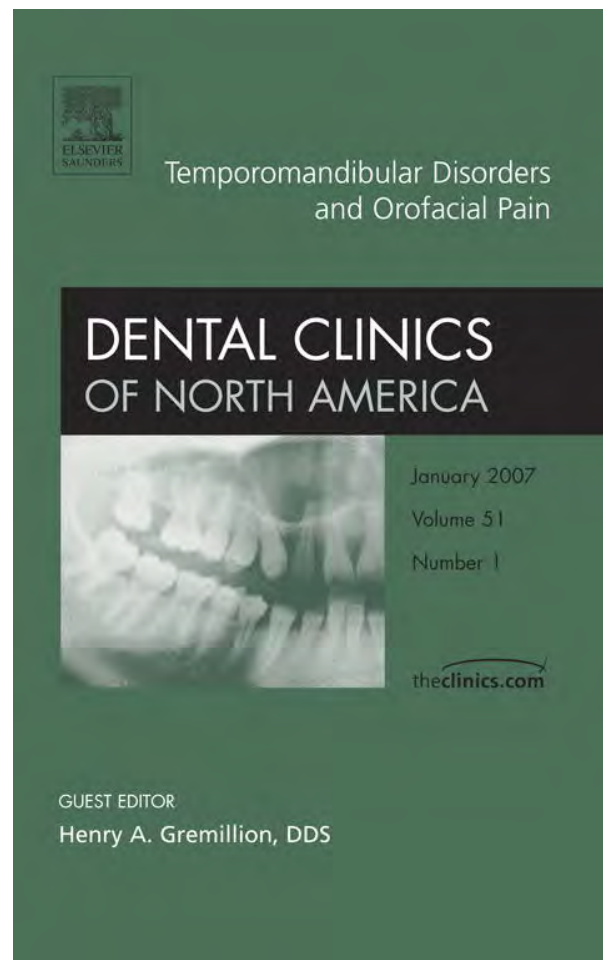


Provided for non-commercial research and educational use only.
Not for reproduction or distribution or commercial use.



This article was originally published in a journal published by Elsevier, and the attached copy is provided by Elsevier for the author's benefit and for the benefit of the author's institution, for non-commercial research and educational use including without limitation use in instruction at your institution, sending it to specific colleagues that you know, and providing a copy to your institution's administrator.

All other uses, reproduction and distribution, including without limitation commercial reprints, selling or licensing copies or access, or posting on open internet sites, your personal or institution's website or repository, are prohibited. For exceptions, permission may be sought for such use through Elsevier's permissions site at:

<http://www.elsevier.com/locate/permissionusematerial>

Temporomandibular Disorders, Head and Orofacial Pain: Cervical Spine Considerations

Steve Kraus, PT, OCS, MTC

2770 Lenox Road, Suite 102, Atlanta, GA 30324, USA

Head and orofacial pain originates from dental, neurologic, musculoskeletal, otolaryngologic, vascular, metaplastic, or infectious disease and is treated by many health care practitioners, such as dentists, oral surgeons, and physicians, who specialize in this pathology. This article's focus relates to the nonpathologic involvement of the musculoskeletal system as a source of head and orofacial pain. The areas of the musculoskeletal system that are reviewed include the temporomandibular joint (TMJ) and muscles of mastication—collectively referred to as temporomandibular disorders (TMDs) and cervical spine disorders [1].

Often, conservative treatment is recommended for most patients who experience TMDs and cervical spine disorders [1,2]. Physical therapists offer conservative treatment in rehabilitation of TMDs and cervical spine disorders. The American Physical Therapy Association (APTA) defines physical therapy as “... the care and services provided by or under the direction and supervision of a physical therapist...” [3]. The position of the APTA is “... only physical therapists provide or direct the provision of physical therapy” [4]. The most valuable contribution that physical therapists make regarding the management of TMDs and cervical spine disorders is in the proper identification of the components in the musculoskeletal system that contribute to a patient's symptoms and functional limitations. This is done by collecting a detailed history from the patient and conducting an appropriate physical assessment based on the history [4]. A properly performed evaluation by a physical therapist determines the type of treatment offered, and results in optimal and meaningful functional outcomes.

Consequently, the validity of research that investigates physical therapy interventions for TMDs and head and orofacial pain should be questioned

E-mail address: stevekraus@mindspring.com

when it is unclear if a physical therapist participated in the evaluation of the patient or provided physical therapy treatment. Referring to physical therapy as only a modality is misleading, and conclusions made about the therapeutic value of physical therapy may be inaccurate [5,6]. The objective of this article is to demonstrate the extent to which a physical therapist who is trained in the specialty of TMDs and cervical spine disorders contributes to the successful management of this condition.

The first part of this article highlights the role of physical therapy in the treatment of TMDs. The second part discusses cervical spine considerations in the management of TMDs and head and orofacial symptoms. The article concludes with an overview of the evaluation and treatment of the cervical spine.

Physical therapy management of temporomandibular disorders

TMD is divided into arthrogenous disorders, which involve the TMJ, and myogenous disorders, which involve the muscles of mastication [1]. An extensive subclassification for arthrogenous and myogenous disorders exists [1]. The common arthrogenous and myogenous disorders that are seen clinically by physical therapists, dentists, oral surgeons, and physicians are addressed in this article (Box 1). The diagnostic criterion for each of the common TMD conditions that follows is referenced in the literature and is not covered in this article [1,7–9]. The objective of this portion of the article is to highlight physical therapy treatment for common TMDs.

Box 1. Common temporomandibular disorders with corresponding *International Classification of Diseases, Ninth Revision (ICD-9)* codes

TMD arthrogenous

Inflammation 524.62

Hypermobility 830.1

Fibrous adhesions 524.61

Disc displacements 524.63

Disc displacement with reduction

Disc displacement without reduction

Chronic disc displacement without reduction

TMD myogenous

Masticatory muscle pain 728.85

Temporomandibular disorders: arthrogenous

Inflammation

Inflammation can originate from TMJ tissues, such as the capsule, medial, and lateral collateral ligaments, TMJ ligament, or posterior attachment. TMJ tissue inflammation can result from blunt trauma and microtrauma that are caused by parafunctional activity. Parafunctional activity is nonfunctional activity, which, when in the orofacial region, includes nail biting, lip or cheek chewing, abnormal posturing of the jaw, and bruxism [1]. Bruxism is diurnal or nocturnal clenching, bracing, gnashing, and grinding of the teeth [1]. Inflammation also can result from arthritic conditions.

Physical therapy treatment for TMJ inflammation involves patient education regarding dietary and oral habits [9]. Iontophoresis, phonophoresis, and interferential electric stimulation are therapeutic modalities that are used to decrease TMJ inflammation [10–12]. Patients who are diagnosed with TMJ inflammation may have altered mandibular dynamics that are due to intracapsular swelling and resultant joint pain. Physical therapists teach patients range of motion exercises that maintain functional mandibular dynamics during the rehabilitation phase without causing more inflammation.

Hypermobility

Hypermobility is excessive translation of the mandibular condyle during opening of the mouth [13]. With condylar hypermobility, the condyle translates anteriorly during opening following the slope of the articular eminence past the articular crest onto the articular tubercle [13]. Hypermobility that occurs unilaterally may be associated with deviation of the mandible, which is observed during mouth opening. Deviation is the mandible moving away from midline, but returning to midline at the end of opening [9]. Although hypermobility may cause disc displacement of the TMJ, the cause and effect relationship has not been established [14,15]. Hypermobility is a common, and, frequently, benign, condition.

Patients who exhibit hypermobility without pain do not require treatment [14]. Controlling hypermobility is necessary only when other TMJ conditions exist. If the patient has TMJ inflammation, hypermobility may perpetuate the inflammation when the patient opens his/her mouth wide during yawning. In the presence of TMJ inflammation, full mouth opening, regardless of whether hypermobility exists, needs to be avoided.

Dislocation of the condyle can result from uncontrolled hypermobility. Diagnosis of condylar dislocation is made if a patient complains that his or her jaw catches on closing from a full, open mouth position. Hypermobility also may be accompanied by palpable joint noises. Palpable joint noises are noises that are heard by the patient and felt by the clinician while palpating over the TMJ during opening and closing movements of the mandible. Joint noises that are associated with hypermobility need to be differentiated

from joint noises that are associated with a disc displacement. Although the patient may not have pain with jaw movement, the experience of joint noise, the feeling of a condyle catching on closing, and an awareness of deviation of the mandible on opening are events that are disconcerting to the patient.

The most important aspect regarding treatment for hypermobility is patient education. Physical therapists should inform their patients that noises and deviations of the jaw are not necessarily signs of significant pathology, and that they can be controlled with proper muscular re-education strategies. When mouth closing is associated with catching, the amount of mouth opening needs to be controlled through neuromuscular coordination exercises that are taught by a physical therapist who is knowledgeable in exercise interventions for TMJ hypermobility [9].

Disc displacement

Disc displacement can be classified into three stages: disc displacement with reduction, disc displacement without reduction, and chronic disc displacement without reduction [16]. Not all disc displacements are painful or interfere with functional movements of the mandible. Treatment is necessary when a patient experiences pain with or without functional limitations of the jaw [17]. Treatment choices for disc displacements that are painful or interfere with function consist of repositioning the disc to the condyle or allowing the disc to remain displaced while improving the function and decreasing the pain in the intra-articular and associated periarticular/myofascial tissues about the TMJ.

When choosing to reposition the disc to the condyle, the options are arthroscopy or an anterior-repositioning appliance. Because of the progressive nature of disc displacement, which is accompanied by increasing pathologic changes in the disc itself and its peripheral attachments, restoring a satisfactory functional disc–condyle relationship may be difficult [17]. Consequently, arthroscopy and anterior-repositioning appliances have led to mixed results in maintaining a normal long-term disc–condyle relationship [18–22].

Arthroscopy is a treatment choice for patients who do not respond to conservative care. Conservative care consists of physical therapy, medication, and a full-coverage acrylic appliance that does not reposition the mandible [23].

An anterior-repositioning appliance, which repositions the mandible, is the most controversial treatment option for repositioning the disc to the condyle [24]. The controversy relates to whether the anterior-repositioning appliance actually recaptures the disc [24]. During the use of an anterior-repositioning appliance, the absence of joint noises and pain with functional mouth opening does not necessarily indicate that the disc has been recaptured [20,24]. Studies using pre- and post-CT and well as MRI showed that permanent long-term disc recapture using an anterior-repositioning appliance was noted in only 10% to 30% of the patients [20]. When an anterior-repositioning appliance is discontinued, some patients may require orthodontics and possible orthognathic surgery. For the most part, an

anterior-repositioning appliance should be considered on a case-by-case basis, and only should be used as an infrequent treatment option for repositioning disc displacements [24].

If the choice is not to reposition the disc to the condyle, the treatment options are arthroscopy (in its simplest format involving lavage/lysis), arthrocentesis, and physical therapy. The therapeutic value common to arthroscopy, arthrocentesis, and physical therapy interventions relates to the facilitation of adaptive responses of the articular tissues to the disc displacement. The human TMJ can adapt or remodel in response to articular disc displacement, regardless of the type of intervention, and often best when there is no intervention. For example, the posterior attachment of the disc (superior and inferior stratum and retrodiscal pad) becomes a pseudo disc that can withstand loading of the condyle during function [17,25]. Restoring a normal disc position is not a necessary component for treating pain and functional resolution [17]. Nonpainful disc displacements are so prevalent in patient and nonpatient populations that they may be considered a normal anatomic variability [26–28]. Because adaptive responses of the articular tissues within the TMJ are common secondary to disc displacement—and in most cases lead to pain-free and functional outcomes—perhaps the most therapeutic intervention should be the least invasive (ie, physical therapy).

Disc displacement without reduction

An article that has reviewed the literature comparing arthrocentesis, arthroscopic surgery, and physical therapy for the treatment of disc displacement without reduction has demonstrated no significant difference in the effects of maximum mandibular opening, pain intensity, or mandibular function [29]. The decision to perform arthroscopy or arthrocentesis instead of physical therapy should be based upon an evidence-based evaluation as well as the needs of the informed patient. When noninvasive treatment is recommended, physical therapy that is performed by a licensed physical therapist with an orthopedic specialty—and preferably a subspecialty in TMDs—should be the first choice in the treatment of disc displacements without reduction.

Physical therapy procedures may be successful in the treatment of pain and limited mouth opening that are associated with disc displacement without reduction [30–33]. Using various active and passive jaw exercises, as well as intraoral mobilization techniques, physical therapists may restore functional mandibular dynamics without pain when the disc is displaced. Inflammation that results from the disc displacement or that coexists with the disc displacement may be treated as identified previously. An oral appliance that is fabricated by a dentist also may facilitate the reduction of inflammation, especially if the patient bruxes. If physical therapy and the use of an oral appliance have not reduced pain to a satisfactory level or regained functional movements of the jaw after 4 to 12 weeks, the patient should consult with an oral surgeon to discuss surgical options.

Disc displacement with reduction and chronic disc displacement without reduction. Patients who experience a disc displacement with reduction or a chronic disc displacement without reduction may have functional movements of the mandible without pain [17]. The first goal of physical therapy consists of educating the patient on the cause of his or her joint noises (ie, reciprocal click or crepitus), so that he or she is aware of the aggravating factors of the condition. If the patient has TMJ pain that is due to inflammation, the goal of physical therapy is to reduce pain and improve mandibular function through manual therapy and exercise interventions, despite the disc displacement. An oral appliance that is fabricated by a dentist also may facilitate the reduction of inflammation, especially if the patient bruxes. A patient who has joint inflammation that does not respond to an oral appliance or 4 to 12 weeks of physical therapy may be referred to an oral surgeon to discuss surgical options.

A physical therapist may attempt to eliminate or decrease joint noises that are associated with a disc displacement with reduction. Clinically, the goal of physical therapy treatment is to have functional mandibular dynamics without pain and without noises, despite the disc being displaced permanently. The following criteria are used for patient selection:

Joint noises are disturbing to the patient

Patient experiences intermittent catching/locking with or without pain during mouth opening

Patient understands that the treatment may (a) cause joint pain or (b) cause limited mouth opening, or (c) result in having TMJ surgery because (a) or (b) could not be resolved.

Patient has consulted with a dentist or oral surgeon previously

Exercises and intraoral manual procedures for treating a reducing disc are not the same as exercises and intraoral manual procedures for increasing limited mouth opening that is associated with a nonreducing disc and fibrous adhesions. Progressing a reducing disc to a nonreducing disc involves the application of exercises and intraoral manual procedures that prevent the disc from reducing on opening. Preventing the disc from reducing on opening elongates the posterior attachment. Once sufficient elongation of the posterior attachment occurs, the patient can achieve functional opening without popping with the disc remaining displaced [9,34,35]. The patient may go through a short period with limited opening and possible pain. In the author's experience, 4 to 12 weeks is a sufficient time to achieve functional mandibular dynamics without pain and with an absence of joint noises with the disc displaced permanently.

Fibrous adhesions

Fibrous adhesions may appear in the capsular-ligament tissues and in the upper joint space of the TMJ [36]. Fibrous adhesions can result from chronic inflammation, blunt trauma, postoperative healing of a capsular

incision, or immobility that occurs with intermaxillary fixation or from limited opening that is associated with a disc displacement without reduction. The physiologic changes that are associated with fibrous adhesions are documented in the literature [37–40]. Physical therapy procedures and modalities for the treatment of fibrous adhesions are similar, but not identical, to those that are used for treating a disc displacement without reduction. Treating fibrous adhesions involves applying an intraoral mobilization technique that is referred to as “lateral glide.” A lateral glide passive intraoral mobilization procedure may be performed at the same time that the patient opens his or her mouth actively. Clinically, this passive/active mobilization force targets the restrictions in the lateral aspect of the capsular–ligament complex of the TMJ. The clinical decisions that are necessary to determine the duration, intensity, frequency, and progression of exercise intervention strategies require skill and experience. The effectiveness of a mobilization technique is related to proper patient selection, appropriate choice of technique, effective execution of the procedure, and making adjustments that are based on tissue response and patient feedback. Inappropriate management of a mechanical dysfunction of the TMJ by untrained personnel may lead to an exacerbation of symptoms and a worsening of the condition.

Temporomandibular disorders: myogenous

Masticatory muscle pain

Masticatory muscle pain is a common clinical finding in patients who experience head and orofacial pain [41]. The relationship between bruxism and masticatory pain is unclear [42]; however, parafunctional activity, such as bruxism, may be a predisposing, precipitating, or perpetuating factor of masticatory muscle pain [43,44]. The common treatment for managing bruxism/masticatory pain is an oral appliance [1]. Oral appliances have been shown to be effective in the treatment of masticatory pain [45,46].

Physical therapists may provide treatments that offer symptomatic relief in masticatory muscle pain through modalities and therapeutic procedures. Modalities, such as iontophoresis, ultrasound, and electric muscle stimulation, may help to reduce muscle pain [9]. Intraoral and extraoral soft tissue mobilization to the muscles of mastication also may provide symptomatic relief [9]. Therapeutic exercises to the mandible that consist of isometric, isotonic, and eccentric contraction have been observed clinically to reduce masticatory muscle pain [30]. Patient education strategies that are related to oral modifications and enhancing self-awareness about aggravating factors also have been shown to provide relief in masticatory muscle pain [47]. Oral modifications consist of diet changes as well as eliminating or limiting oral habits, such as gum chewing and nail, lip, or cheek biting. Self-awareness strategies also include instructing the patient on the proper rest position of the tongue and mandible. Patients who take an active role in making oral

modifications and performing neuromuscular exercises may achieve satisfactory daytime relief from masticatory muscle pain. Decreasing the cumulative loading during the day also may provide relief in nighttime pain that is associated with bruxism. Nocturnal bruxism is more difficult to treat, even when the patient wears an oral appliance. Physical therapists can assist in reducing nocturnal bruxism by addressing head and neck positioning while sleeping. Instructing the patient on proper selection and usage of pillow support that is appropriate for their cervical spine alignment and motion function may help to lessen the tendency for bruxism at night by enabling a more restful mandibular position. Cervical spine disorders that may contribute to bruxism are covered in a later section.

Cervical spine considerations in the management of temporomandibular disorders and head and orofacial pain

The coexistence of neck pain and TMD is common [48–61]. One study found that neck pain is associated with TMD 70% of the time [55]. There also is a high occurrence of neck pain in patients who have facial pain. A study was conducted on 200 consecutive female patients who were referred to a university facial pain clinic. The patients were asked to mark all painful sites on sketches that showed contours of a human body in the frontal and rear views [62]. An analysis of the pain distribution according to the arrangements of dermatomes revealed three distinct clusters of patients: (1) those with pain restricted to the region innervated by the trigeminal nerve ($n = 37$); (2) those with pain in the trigeminal dermatomes and any combination involving the spinal dermatomes C2, C3, and C4, but no other dermatomes ($n = 32$); and (3) those with pain sites involving dermatomes in addition to those listed in (1) and (2) ($n = 131$).

In summary, the pain distribution of the 200 patients who had facial pain is more widespread than commonly assumed [62]. One hundred and sixty-three of 200 patients had pain that extended outside of the head and face to areas that included the C2, C3, and C4 dermatomes [62]. Other studies also have concluded that patients who have head and orofacial pain often experience widespread pain in the neck and shoulder areas [63,64].

A systematic review of the association between cervical posture and TMDs has been conducted [65]. The review examined 12 studies that satisfied the same inclusion criteria for participants. It concluded that an association between TMDs and cervical posture is unclear. The uncertainty of the association between TMDs and cervical posture was related to poor methodologic quality of the 12 studies [65]. Determining the typical resting posture of the head and neck for a study that evaluates upper body positional relationships is difficult, because all individuals assume many different head and neck postures during the course of a day's activities. Perhaps future studies that investigate cervical spine and TMD relationships should

account for the dynamics of the cervical spine, instead of focusing on rest positions. The relationship of mandibular dynamics and the cervical spine needs to be analyzed in future studies by using reliable clinical instrumentation to compare active movements of the cervical spine to mandibular opening and closing or masticatory muscle pain.

The following section highlights cervical spine considerations in the management of TMD; it is followed by a discussion on cervical spine considerations for head and orofacial pain.

Cervical spine considerations with temporomandibular disorders—arthrogenous involvement

The TMJ is a load-bearing joint [1]. TMJ inflammation may be perpetuated by bruxism that loads the joint excessively [66,67]. An oral appliance helps to control bruxism [24]; however, not all patients respond favorably to an oral appliance that is designed to control bruxism. Many variables can contribute to bruxism, which is why an oral appliance may not always be therapeutic in controlling bruxism. One variable is cervical spine involvement. Decreasing the intensity and duration of bruxism by managing cervical spine disorders may reduce pain that originates from arthrogenous involvement. Cervical spine involvement as a cause of masticatory muscle pain or bruxism is discussed later in this article.

Typically, full mouth opening is accompanied by extension of the head, whereas mouth closing typically is accompanied by flexion of the head [68]. A frequently observed abnormal posture involves an extended head–neck position which is a component of “forward head posture.” The forward head posture may facilitate wider mouth opening during functional activities, such as yawning and eating a large sandwich. Increasing patient awareness of forward head posture and instruction in correcting forward head posture during sitting, standing, and walking may control excessive mouth opening that is associated with hypermobility; it should be a part of the conservative management program for every patient who has a TMD.

On the other hand, if the objective is to facilitate mouth opening, physical therapists may position the patient’s head and neck in slight extension during procedures (eg, intraoral mobilization and static–dynamic jaw exercises) that increase mouth opening. When the patient stands for mouth-opening exercises, the patient is instructed to allow his or her head to extend slightly while opening.

Patients often believe that their head and orofacial pain are due entirely to their disc displacement. Many patients believe that the only way to feel better is to have the disc “put back into place.” This may be true, however, in only a small percentage of patients who have a disc displacement. Often, the source of the patient’s pain is independent of the disc displacement. Instead, it originates from TMJ inflammation, overactive masticatory muscles,

and irritation of the pain-sensitive structures of the cervical spine. Cervical spine involvement as a source of head and orofacial pain is discussed later.

Cervical spine considerations with temporomandibular disorders—myogenous involvement

Bruxism is more common in patients who have myofascial pain in the masticatory and cervical spine muscles [51]. Patients who have TMDs report neck symptoms more frequently than do patients who do not have TMDs; patients who have neck pain report more signs and symptoms of TMDs than do healthy controls [58]. Neck and shoulder pain is more prevalent in patients who have a TMD with a myogenous component than in patients who have a TMD with an arthrogenous component [56]. Therefore, the prevalence of neck pain coexisting with masticatory pain may be more than a coincidence. Cervical spine involvement as a predisposing, precipitating, or perpetuating variable to masticatory muscle pain or bruxism is highlighted in the following three theories.

Theory one

The first theory is that afferent input that is associated with neck pain converges onto trigeminal motor neurons in the trigeminocervical nucleus, which results in an increase in masticatory muscle hyperactivity and pain. Motor activity of trigeminal-innervated muscles of mastication increases when tissues that are innervated by upper cervical spine segments are irritated experimentally [69–73]. Little information on human subjects is available regarding the influence of experimental pain in the neck and shoulder muscles on motor activity in the orofacial region. One study was done to clarify the effects of experimental trapezius muscle pain on pain spread and on jaw motor function [74]. Experimental pain was induced in the superior border of the trapezius muscle of 12 subjects, aged 25 to 35 years of age, by injecting 0.5 mL of hypertonic (6%) saline. Results showed pain spread over a wide area to include the temporomandibular region, with pain referral accompanied by a reduction of mouth opening [74]. Afferent nociceptive input from the neck muscles may excite efferent (motor) neurons of cranial V, which results in contraction of masticatory muscles [75,76]. Similar convergences and central excitation phenomena—as seen with cervical and trigeminal sensory neurons—also may exist for trigeminal motor neurons [77,78].

Theory two

The second theory is that masticatory muscles contract in response to the contraction of cervical spine muscles. A neurophysiologic interplay exists that involves a synergistic relationship between the cervical spine and the muscles of mastication under normal circumstances [79–85]. Synergistic

co-contraction can be observed with jaw and neck muscles during activities involving chew, talk, and yawn. Reciprocal innervations of opposing muscles has been demonstrated [82]. The cervical spine muscles and the muscles of mastication can be viewed as agonistic and antagonistic to one another [83]. In overt motor patterns, such as walking, augmentation and diminution of antagonistic muscles contracting concurrently (co-contraction) with agonist muscles contracting has been demonstrated [84,85].

Sometimes common daily events may cause the muscles of mastication to disproportionately contract in response to cervical muscles contracting. Head, neck, shoulder girdle, and upper extremity posture must be positioned precisely during eye–hand coordination activities, such as writing, painting, computer work, and driving. A task that involves a specific head and neck posture requires a constant low-level contraction of the cervical spine muscles. The longer that a subject spends on maintaining a specific head–neck posture, the more likely an exaggerated contraction of the muscles of mastication will occur in response to cervical spine muscles contracting.

Isometric, isotonic, or eccentric contractions of cervical spine muscles occur during lifting, carrying, pushing, pulling, and reaching activities. When cervical spine muscles perform repetitive activity, under load, and over a long duration, the more likely it is that the muscles of mastication will disproportionately contract.

Theory three

The third theory is that the patient bruxes in response to neck pain. Patients start to brux or the intensity and frequency of their bruxing may be exacerbated by their response to acute or chronic neck pain.

Thus, a neurophysiologic interplay exists between the muscles of mastication and the cervical spine, which needs to be addressed in the thorough management of the patient who has a TMD. Although these three theories need further clinical research, physical therapists observe that treating cervical spine pain often decreases masticatory muscle pain. Consequently, neck pain should be added to the list of factors that contribute to bruxism and masticatory muscle pain.

Cervical spine considerations with oral appliances

Common treatments for masticatory muscle pain are medication and application of an oral appliance, both of which can be offered by a dentist or oral surgeon [24]. Physical therapists should be familiar with the different structural designs of splints as well as be able to explain the rationale and therapeutic benefits for oral appliance use [46,86,87].

One common feature of the use of oral appliances and postural re-education/manual therapy intervention of cervical spine dysfunction is that both treatment strategies influence the rest position of the mandible. Rest position of the mandible determines the initial path of closure into

tooth-to-tooth contact or teeth contact onto an appliance [88]. The design of an oral appliance influences the vertical and horizontal positions of the mandibular rest position; this changes the path of mandibular closure and affects how the teeth and oral appliance make contact [89].

Conversely, head and neck posture also influences the vertical and horizontal positions of the mandibular rest position, which subsequently alters the path of closure into teeth-to-teeth contact [90–98]. Mohl [90] stated, “if the rest position is altered by a change in head position, the habitual path of closure of the mandible must also be altered by such a change.” Clinically, physical therapists have recognized that cervical spine motion restrictions and forward head posture affect mandibular closure, which, in turn, alters how the teeth and oral appliance make contact.

Patients may complain that they do not “hit,” “bite,” or “make contact” evenly on their appliance. If the patient’s complaint cannot be explained by interferences that are caused by the appliance design, the dentist should consider a mechanical disorder within the cervical spine that affects the path of closure of the mandible onto the appliance. Patients who do not respond to an oral appliance in a 4-week period may not need more time wearing the appliance or a change in the design of the appliance [1]. Another alternative is to have a physical therapist evaluate the cervical spine to assess for possible dysfunctions that might be interfering with the effectiveness of the oral appliance. Clinically, cervical spine dysfunction with respect to abnormal posture or motion impairment can be treated before, during, or after the use of an oral appliance. Favorable outcomes are more likely to be achieved when cervical spine treatment is rendered concurrently with the use of an oral appliance, according to physical therapists who are experienced in managing masticatory muscle pain.

Cervical spine considerations with head and orofacial pain

Symptoms that originate from the cervical spine and require immediate medical attention secondary to spinal pathology include gross mechanical instability that may affect spinal cord function, primary bone tumor, metastatic disease, infections, fracture, and dislocation [99]. Symptoms also may be referred to the cervical spine from visceral pathology [100]. “Red flags” that suggest a visceral pathology should alert the clinician to a nonmusculoskeletal origin of the patient’s pain (Box 2). Imaging studies and erythrocyte sedimentation rates can help in detecting whether an underlying pathology is present [101].

Most cervical spine–related symptoms are not caused by spinal or visceral pathology [102]. Nonpathologic symptoms may originate from disc disorders, nerve root irritation, spinal cord compromise secondary to spinal stenosis, facet joint dysfunction, and myofascial pain. Common medical diagnoses for each cervical spine tissue are listed in Box 3. Patients

Box 2. Pathologic conditions are suspected with the following “red flags”

Fever
 Unexplained loss of weight
 History of inflammatory arthritis
 History of malignancy
 Osteoporosis
 Vascular insufficiency
 Blackouts
 History of drug abuse, AIDS, or other infection
 Immunosuppression
 Lymphadenopathy
 Severe trauma
 Minor trauma or strenuous lifting in an older patient
 Increasing or unremitting pain

_____ *Data from Jarvik J, Deyo R. Diagnostic evaluation of low back pain with emphasis on imaging. Ann Intern Med 2002;137:586–97.*

frequently have more than one cervical spine–related tissue that is the source of their cervical spine–related symptoms. Multiple cervical spine tissue involvement can be referred to collectively as cervical spine disorders. Cervical spine disorders can cause pain or functional limitations of the cervical spine in which symptoms vary with physical activity or static positioning, which may develop gradually or follow trauma.

The prevalence of nonpathologic neck pain is high. Seventy percent of the general population is affected with neck pain at some time in their lives [103]. Fifty-four percent of the general population has experienced neck pain in the last 6 months [104]. The general population has a point prevalence of neck pain that varies between 9.5% and 22% [105].

Box 3. Common sources of neck symptoms with corresponding *International Classification of Diseases, Ninth Revision (ICD-9) codes*

Disc: 722.6, degeneration; 722.2, herniation
 Nerve root: 723.4, cervical radiculopathy
 Spinal cord: 721.1, cervical myelopathy
 Facet joint: 719.5, hypomobility
 Muscle: 728.5, muscle spasm; 729.1, myalgia

Head and orofacial pain of cervical spine origin

The International Headache Society has created a list of 144 different headache types that fall into one of 13 categories (Box 4) [106]. The cervical spine is listed as a possible causative factor for headaches and is reported as “neck” in classification 11, subclassification 11.2.

The literature is clear that cervical spine tissues refer pain to the head and orofacial areas [77,107]. The neuroanatomic mechanism that explains the referred pain is the convergence between trigeminal afferents and afferents of the upper three cervical nerves [108]. This convergence occurs in an area that is referred to as the trigeminocervical nucleus [109]. The trigeminocervical nucleus is located in the upper cervical spinal cord within the pars caudalis portion of the spinal nucleus of the trigeminal nerve (Fig. 1) [110,111].

Box 4. Classification and diagnostic criteria for headache disorders, cranial neuralgias, and facial pain

1. Migraine headache
2. Tension-type headache
3. Cluster headache and chronic paroxysmal hemicrania
4. Miscellaneous headache, unassociated with structural lesion
5. Headache associated with head trauma
6. Headache associated with vascular disorders
7. Headache associated with nonvascular intracranial disorders
8. Headache associated with substances or withdrawal
9. Headache associated with noncephalic infection
10. Headache associated with metabolic disorder
11. Headache or facial pain associated with disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures
 - 11.1 Cranial bones including the mandible
 - 11.2 Neck
 - 11.3 Eyes
 - 11.4 Ears
 - 11.5 Nose and sinuses
 - 11.6 Teeth and related oral structures
 - 11.7 Temporomandibular joint
 - 11.8 Masticatory muscles
12. Cranial neuralgias, nerve trunk pain, and deafferentation pain
13. Headache not classified

Adapted from International Headache Society, Classification Committee. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalalgia* 1998;8(Suppl 7):9–96.

Primary sources of head and orofacial pain that originate from the cervical spine lie in the structures that are innervated by C1 to C3 spinal nerves [111]. The lower segmental levels, C4 thru C7, also may contribute to head and orofacial pain through the trigeminocervical nucleus [112]. **Box 5** lists the tissues with sensory innervations from the upper three cervical nerves that contribute to referred symptoms to the head and orofacial areas [111].

The greater occipital nerve (GON) branches off from the C2 nerve root [113]. GON cutaneous branches and their innervations are:

Medial branch: innervates the occipital skin

Lateral branch: innervates the region above the mastoid process and behind the pinna (the projecting part of the ear lying outside of the head)

Intermediate branches: run rostrally and ventrally across the top of the skull as far as the coronal suture. Anastomosis of the GON to the

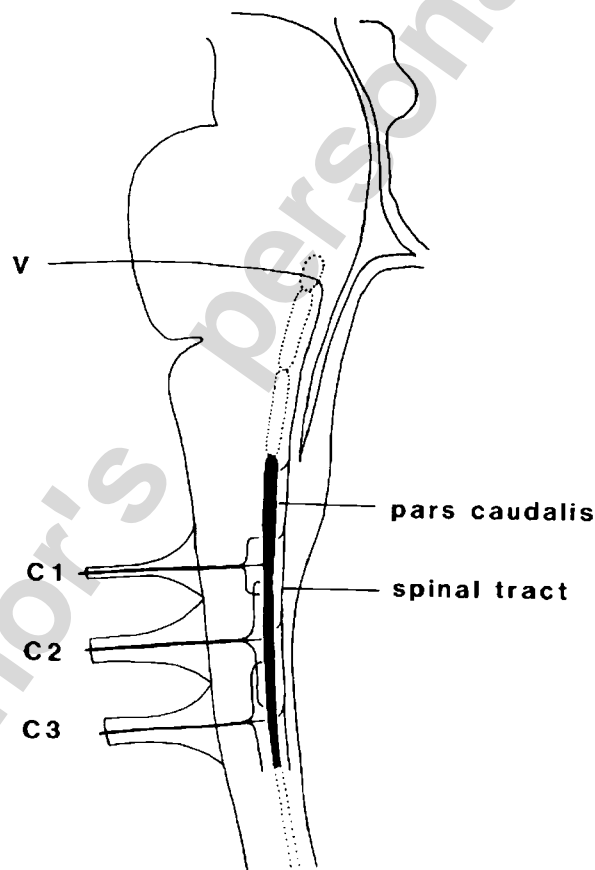


Fig. 1. A sketch of the “trigeminocervical nucleus.” Afferent fibers from the trigeminal nerve (V) enter the pons and descend in the spinal tract to upper cervical levels, sending collateral branches into the pars caudalis of the spinal nucleus of the trigeminal nerve and the gray matter of the C1 to C3 spinal cord segments. Afferent fibers from the C1, C2, and C3 spinal nerves ramify in the spinal gray matter at their segment of entry and at adjacent segments. That column of gray matter that receives trigeminal and cervical afferents constitutes the trigeminal nucleus (*black*). (From Bogduk N. Cervical causes of headache and dizziness. In: Grieve G, editor. Modern manual therapy. 2nd edition. Edinburgh (UK): Churchill Livingstone; 1986. p. 317, with permission.)

Box 5. Sensory innervations from the upper three cervical nerves

C1 sensory innervation

Suboccipital tissues and muscles
 Atlantoccipital and atlantoaxial facet joints
 Paramedian dura of the posterior cranial fossa and dura adjacent to the condylar canal
 Upper prevertebral muscles (longus capitis and cervicis and the rectus capitis anterior and lateralis)

C2 sensory innervation

Skin of the occiput
 Upper posterior neck muscles; semispinalis capitis, longissimus capitis and splenius capitis, the sternocleidomastoid, trapezius, and prevertebral muscles
 Atlantoaxial facet joint
 Paramedian dura of the posterior cranial fossa
 Lateral walls of the posterior cranial fossa

C3 sensory innervation

Multifidus, semispinalis capitis, sternocleidomastoid, trapezius, and prevertebral muscles
 Suboccipital skin
 C2/3 facet joint
 Cervical portion and intracranial branches of the vertebral artery

supraorbital nerve, which is a trigeminal branch, occurs at the coronal suture.

Trauma or suboccipital muscle tightness may involve the GON, referred to as occipital neuralgia [114]. Symptoms that are associated with occipital neuralgia refer to the occipital area, top of the skull, TMJ area, and in or around the ear [115,116].

Cervicogenic headache

The term “cervicogenic headache” was used first in 1983 by Sjaastad and colleagues [117]. Cervicogenic headache refers to head and orofacial pain that originates from the cervical spine tissues. Cervicogenic headache can be a perplexing pain disorder [118]. The following is a clinical presentation of cervicogenic headache as described by Sjaastad et al [117]:

The pain is usually unilateral but when severe can be felt on the opposite side. It is a head pain and not just a neck pain. The main manifestation

of the headache is in the temporal, frontal, and ocular areas. It has fluctuating long-term course with remissions and exacerbations; some patients have a continuous basal headache, others do not. During the headache attack, there may be the following accompanying phenomena; ipsilateral blurring and reduced vision, a “migrainous” phenomena like nausea and loss of appetite; there may even be vomiting. Phonophobia and photophobia occur frequently. Some patients complain of dizziness and of difficulty swallowing during symptomatic periods. Even between attacks, patients may feel stiffness and reduced mobility of the neck.

Prevalence of cervicogenic headache

Cervicogenic headache is one of the three large headache groups; the other two are tension-type headache and common migraine without aura [119]. Cervicogenic headache accounts for 15% to 35% of all chronic and recurrent headaches [119–121].

Although cervicogenic headache has been diagnosed more frequently over recent years, it also has been misdiagnosed because of the considerable overlap in symptoms with more popular causes of headache (tension-type and migraine) [117,122,123]. Cervical pain and muscle tension are common symptoms of a migraine [124,125]. In a study of 50 patients who had migraine, 64% reported neck pain or stiffness associated with their migraine, with 31% experiencing neck symptoms during the prodrome, 93% experiencing neck symptoms during the headache phase, and 31% experiencing neck symptoms during the recovery phase [124]. Other studies show that neck pains often coexist with migraine headaches [126,127]. In addition, cervical muscles may play a role in the pathogenesis of migraine headaches [128]. Patients often suffer several headache types concurrently [129]. Patients may require medications for migraine, application of an oral appliance for tension headache, and physical therapy for cervicogenic headache. In summary, many patients are misdiagnosed to have migraine or tension type headaches, when in fact these patients actually have headaches of cervical origin. Therefore, the appropriate treatment should be targeted to mechanical dysfunction or muscle tension in the cervical spine.

Dizziness

Dizziness and vertigo refer to a false sensation of motion of the body, which patients describe as a spinning or swaying feeling [130,131]. They are synonymous terms that are used to describe spinning, swaying, the subjective accompaniments of ataxia, and a variety of other colloquially described sensations. Dizziness may result from involvement of the eyes, the parietal and temporal lobes, and cerebellum—most commonly as a result of disease affecting the labyrinth or the vestibular nuclei [132,133]. In the absence of disease, the vestibular nuclei can be affected by disorders of the neck in two ways: through ischemic processes or disturbances of neck

proprioceptors [133]. Disturbance of the vestibular nuclei secondary to dysfunctional neck proprioceptors are addressed for this discussion.

Afferent input from neck proprioceptors (ie, facet joints and muscles) is believed to affect the vestibular nuclei activity, which results in a variety of motor and subjective abnormalities [133]. Cervical facet joints and muscles may produce a generalized ataxia, with symptoms of imbalance, disorientation, and motor incoordination [134–139]. Vertigo, ataxia, and nystagmus were induced in animals and man by injecting local anesthetic into the neck [140]. The injections presumably interrupted the flow of afferent information from joint receptors and neck muscles to the vestibular nuclei. Vertigo following a whiplash injury (an extension/flexion movement of the head and neck) may be due to afferent excitation that originates from cervical muscles, ligaments, facet joints, and sensory nerves [141]. Patients who do not respond to treatments for dizziness that is believed to be originating from the eye, inner ear, or sinus should be suspected of having cervicogenic vertigo. Patients who experience cervicogenic vertigo may complain of pain, stiffness, and tightness in the neck; they are good candidates for physical therapy intervention that focuses on the cervical spine [142,143].

Subjective tinnitus and secondary otalgia

Objective tinnitus is characterized by physiologic sounds and represents only 1% of cases of tinnitus. Subjective tinnitus is an otologic phenomenon of phantom sounds. Although 10% of the population suffers from subjective tinnitus, its cause is unknown [144].

Subjective tinnitus has been related to cervical spine involvement. The sensory upper cervical dorsal roots and the sensory components of four cranial nerves (V, VII, IX, X) converge on a region of the brain stem that is known as the medullary somatosensory nucleus [145]. Subjective tinnitus is a neural threshold phenomenon and cervical muscle contraction alters the neural activity that is responsible for tinnitus [146]. One hundred and fifty patients were tested with a series of head and neck maneuvers to assess whether any of the maneuvers changed their subjective tinnitus. Eighty percent of patients had increased tinnitus during the test [146]. A similar study tested 120 patients who had subjective tinnitus and 60 subjects who did not have tinnitus [147]. The findings showed that forceful head and neck contractions, as well as loud sound exposure, were significantly more likely to modulate ongoing auditory perception in people who had tinnitus than in those who did not have tinnitus [147]. This study supports the concept that subjective tinnitus has a neural threshold [147].

Secondary otalgia (ie, earache not caused by primary ear pathology) is common in patients who are suffering from earache [148]. In a standardized examination and interview of 100 subjects, 91 subjects had secondary otalgia and 9 had primary otalgia [149]. An epidemiologic study investigated subjects who had secondary otalgia during a 2-year follow-up period [150].

Subjects who had secondary otalgia had pain with palpation over the masticatory muscles and TMJ, and reported neck and shoulder pain more frequently than did the individuals who did not have secondary otalgia [150]. Kuttilla and colleagues [149] investigated whether secondary otalgia is associated with cervical spine disorder, TMDs, or both [149]. Most of the subjects who reported secondary otalgia also had signs and symptoms of cervical spine and TMD involvement. An examination of the cervical spine and TMD is recommended as a routine diagnostic process for patients who have secondary otalgia.

Cervical spine examination

History

Orthopedic-related cervical spine problems are suspected first during the history. Primary symptoms of cervical spine disorders are neck, shoulder, and upper extremity pain and headaches (cervicogenic). Cervicogenic headaches are described by patients as pain that projects from the neck to the forehead, orbital region, temples, vertex, or ears. The symptoms for cervicogenic headaches as identified by the International Headache Society criteria for cervicogenic headache are listed in **Box 6** [151]. Symptoms, such as dizziness, ear pain (secondary), and subjective tinnitus, also may have a cervicogenic origin. A complete list of cervical spine-related symptoms is shown in **Box 7** [152].

The patient's symptoms can be quantified by documenting frequency, intensity (visual analog scale), and duration of symptoms. This information can be used to monitor the patient's response to treatment. The Copenhagen Neck Functional Disability Scale or the Functional Rating Index can be used to document improvement [153,154]. Duration of sleeping and sitting as well as the patient's ability to reach, pull, and lift are documented in a measurable manner. Change in medication intake also can be used to monitor the patient's response to treatment.

Physical examination

A physical examination of the cervical spine involves tests that incriminate nerve involvement. Often, neurologic signs are the result of nerve root compromise and are referred to as cervical radiculopathy, whereas spinal cord compromise is referred to as cervical myelopathy. Aside from physical tests that evaluate nerve function (manual muscle tests, sensory tests, reflex responses, and nerve tension tests), the physical therapy examination assesses for motion impairments of the cervical spine that influence gross range of motion or result in abnormal segmental vertebral motion that corresponds to the patient's symptoms and functional limitations. Palpatory tests evaluate for myofascial pain and dysfunction with respect to tenderness, and tightness. Pain also can be accessed upon

Box 6. International Headache Society criteria for cervicogenic headache

- A. Pain localized in the neck and occipital region. May project to the forehead, orbital region, temples, vertex, or ears.
- B. Pain is precipitated or aggravated by special neck movements or sustained neck posture.
- C. At least one of the following occurs:
 - a. Resistance to or limitation of passive neck movements
 - b. Changes in neck muscle contour, texture, tone or response to active and passive stretching and contraction
 - c. Abnormal tenderness in neck muscles
- D. Radiologic examination reveals at least one of the following:
 - a. Movement abnormalities in flexion/extension
 - b. Abnormal posture
 - c. Fractures, congenital abnormalities, bone tumors, rheumatoid arthritis, or other distinct pathology (not spondylosis or osteochondrosis)

Adapted from International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalalgia* 1998;8(Suppl 7):9–96.

contraction of the muscle. Manual muscle and neuromotor tests are used to assess strength and coordination. A postural analysis is included to evaluate for possible areas of stress concentration. Physical therapists often determine the patient's response to manual traction during the initial examination to evaluate the need for mechanical cervical traction treatment. Physical examination procedures are listed in [Box 8](#). Imaging studies may be needed if the history and physical examination findings are questionable or vague.

Treatment strategies for cervical spine and related symptoms*Invasive procedures*

Treatment guidelines, such as the Scientific Monograph of the Quebec Task Force on Whiplash-Associated Disorders and Evidence-based Practice Guidelines for Interventional Techniques in the Management of Chronic Spinal Pain, recommend a noninvasive approach in the treatment of cervical spine symptoms with or without neurologic signs [152,155]. Only after unsuccessful conservative treatment should invasive procedures be considered [156]. Invasive procedures include epidural injections, nerve root

Box 7. Symptoms that may originate from cervical spine disorders

Neck/shoulder pain
Reduced/painful neck movements
Numbness, tingling or pain in arm or hand
Reduced/painful jaw movement
Headaches
Dizziness/unsteadiness
Nausea/vomiting
Difficulty swallowing
Ringing in the ears
Vision problems
Numbness, tingling, or pain in leg or foot
Lower back pain
Memory problems
Problems concentrating

Data from Spitzer WO, Skovron ML, Salmi LR, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining "whiplash" and its management. Spine 1995;20(8 Suppl):1S-73S.

injections, facet joint denervation, myofascial trigger point injections, and surgery (ie, cervical fusion).

Unless neurologic signs suggest otherwise, patients who have symptoms of radiculopathy or myelopathy should be considered for surgery after conservative care has failed. 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. Two studies were prospective, randomized studies that evaluated a total of 130 patients; the other study was a randomized study that involved 68 participants [157–159]. No differences were found in sensation or motor strength between the patients who were treated surgically and those who were managed conservatively in follow-up examinations at 24 and 36 months. Therefore, patients need to be informed that the long-term outcomes for conservative treatment of minimal to moderate cervical radiculopathy or myelopathy may be the same as having surgical intervention, and in some cases, the only reason for selecting a surgical approach may be to achieve faster pain relief.

Conservative care

Patients who have neck pain can choose from several complementary/alternative treatments that may be part of a physical therapist's knowledge

Box 8. Procedures used to diagnose cervical spine disorders (disc, nerve root, spinal cord, facet joint, and muscle)*Neurologic testing for nerve function*

- Deep tendon reflex
- Sensation
- Strength
- Spurlings test
- Hoffman's reflex
- Lhermitte's test
- Nerve tension tests

Active range of motion

- Passive range of motion*
- Cardinal plane movement
- Intersegmental movement

*Muscle contraction (isometric/isotonic/eccentric)**Palpation*

- Muscles
- Facet joints
- Greater occipital nerve

*Manual traction**Posture*

and skill base. Complementary and Alternative Medicine (CAM) is a diverse group of health-related professionals that have not documented the therapeutic value of their alternative treatments (eg, magnet therapy, crystal application) through randomized clinic trials [160]. Physical therapy, however, is not CAM. Physical therapists offer evidence-based treatments for TMDs and cervical spine disorders with data that are well documented in peer-reviewed journals [161–167]. Physical therapists follow evidence-based guidelines using a multimodal conservative treatment approach for cervical spine symptoms that consists of manual therapy, exercise, patient education, and mechanical cervical traction.

A multicenter, randomized, controlled trial with unblinded treatment and blinded outcome measures was conducted to investigate the efficacy of physical therapy management of cervicogenic headache [168]. A group of 200 participants who met the diagnostic criteria for cervicogenic headache was randomized into four treatment groups: manipulative therapy, exercise therapy, combined therapy, and no treatment. The primary outcome measured was a change in headache frequency. Other outcomes evaluated included

changes in headache intensity and duration, improvement in the Northwick Park Neck Pain Index, reduction in medication intake, and patient satisfaction. The physical outcomes evaluated included pain on neck movement, upper cervical joint tenderness, a craniocervical flexion muscle test, and a photographic measure of posture. The treatment period was 6 weeks with follow-up assessment after treatment, then at 3, 6, and 12 months. At the 12-month follow-up assessment, manipulative therapy and specific exercise had reduced headache frequency and intensity and neck pain significantly, and effects were maintained ($P < .05$ for all). In summary, manipulative therapy and specific therapeutic exercise reduce the symptoms of cervicogenic headache in the short and long term [168].

Manual therapy

Manual therapy techniques consist of a continuum of skilled passive movements to joints or related soft tissues that are applied at varying speeds and amplitudes, including a small-amplitude/high-velocity therapeutic movement [169]. Mobilization (nonthrust) or manipulation (thrust), when used with exercise, is effective for alleviating persistent pain and improving function when compared with no treatment. When compared with each other, neither mobilization nor manipulation is superior [161]. The psychologic, neurophysiologic, and mechanical benefits of manual therapy have been covered adequately in the literature [170,171].

Exercise

Exercises may be effective in treating and preventing neck pain [172]. Specific exercises combined with manual therapy may be effective in the treatment of subacute and chronic neck pain, with or without headache, in the short and long term [155,173]. Physical therapists can identify muscles of the cervical, shoulder, and thoracic areas that are tight, weak, and have difficulty in regulating tension levels. Physical therapists instruct patients in exercise programs that consist of stretching, strengthening, conditioning, and coordination that are specific to the patient's needs. Modification of the exercise program frequently is necessary after re-evaluation of the patient, and is dependent upon the changes in the patient's signs and symptoms. A successful home exercise program is a function of proper patient performance and diligence. The skill of the physical therapist in teaching correct exercise form, making modifications in the exercises based on patient's response, and motivating the patient to perform his or her home program are critical in obtaining an optimal outcome.

Patient education

Patient education focuses on many elements of patient care, and often involves instructing the patient on proper sitting and sleep postures. Support and encouragement of patients also is important to help them overcome fear, anxiety, and misconceptions about their condition. Frequently, well-

meaning advice from friends or family members may interfere with recovery because of misbeliefs or incorrect information. In some cases, incorrect information is being received from online computer resources that the patient has read. Frequently, physical therapists must dispel myths that the patient may have obtained from different sources to alleviate anxiety-fear and manage pain [174,175].

Patients are educated about the meaning of their diagnosis by physical therapists because physical therapists typically spend more time with the patient than do medical professionals. Patients often perceive that “something is wrong” (ie, irreversible) from a medical diagnosis, such as degenerative joint disease, when degenerative joint disease in itself is neither predictive of, nor strongly correlated with, the patient’s symptoms. In this way, a medical diagnosis may enhance the feelings of fear and anxiety, which can intensify symptoms and lead the patient to believe that a cure is not available [176]. Patients can become preoccupied with their diagnosis and often seek invasive treatment in an attempt to “fix” the condition.

The health practitioner must understand that a patient’s fear, misunderstanding, and beliefs about the meaning of pain may determine whether he or she progresses from acute to chronic neck pain [177]. A patient is less likely to develop a chronic pain mentality when he or she is educated about the condition secondary to the knowledge obtained about the medical diagnosis and symptoms. The physical therapist plays a major role in reducing patient anxiety and fear by keeping the patient focused to functional goals.

Mechanical cervical traction

Traction is a treatment that is based on the application of a longitudinal force to the axis of the spinal column. Medically accepted uses for spinal traction include soft tissue tightness, joint stiffness, cervical radiculopathy, and cervical myelopathy that are caused by disc degeneration or disc herniation [178]. The therapeutic value of traction was demonstrated in a trial of 30 patients who had unilateral C7 radiculopathy [179]. Patients were assigned randomly to a control group or an experimental group. The application of cervical traction, combined with electrotherapy and exercise, produced an immediate improvement in the hand-grip function in patients who had cervical radiculopathy compared with the control group that received electrotherapy/exercise treatment [179]. Although this is only one study that provides support for the use of mechanical traction, it does demonstrate its potential for radicular signs and symptoms.

The benefits of neck traction are optimal when performed with the patient in a supine position. The traction unit should not pull through the mandible, but only through the base of the skull/mastoid process areas. Guidelines are available that recommend angle of pull, poundage, and duration of pull [178]. A physical therapist considers the patient’s signs and symptoms to adjust the force and duration of stretch to get the desired results.

Summary

Physicians, dentists, oral surgeons, and physical therapists need to work together to achieve the best outcomes for patients who experience TMDs and head and orofacial pain. Physical therapists play an important role in the conservative care of TMDs and cervical spine disorders that cause head and orofacial pain. Physicians and dentists should keep in mind that not all physical therapists have specialty practices that focus on TMDs and cervical spine disorders. Therefore, referral to an orthopedic physical therapist who specializes in TMDs and cervical spine disorders is important for the appropriate management of the patient.

Physical therapists treat TMDs that are secondary to inflammation, hypermobility, disc displacements, fibrous adhesions, and masticatory muscle pain and bruxism. Studies have shown that masticatory muscle pain and bruxism may be perpetuated by cervical spine involvement. Research evidence suggests a neurophysiologic interplay between the muscles of mastication and the cervical spine muscles. The cervical spine should be evaluated and treated when patients' TMD symptoms do not respond to medication and an oral appliance.

Often, cervical spine involvement is a misdiagnosed or unrecognized source of head and orofacial pain (ie, headache), dizziness, subjective tinnitus, and secondary ear pain. Head and orofacial pain that originates from the cervical spine is referred to as cervicogenic headache. Cervicogenic headache symptoms can be similar to other common headache disorders, such as migraine or tension-type headache.

Cervical spine disorders that are treated by physical therapists using evidence-based interventions, such as manipulation/mobilization and therapeutic exercise, can decrease the protracted course of costly treatment and reduce the patient's pain. Physical therapists, therefore, have an important role in the management of head-neck and orofacial pain. Patients who present with TMD and cervical spine disorders many times can be effectively treated by a physical therapist that has specialized skills and experience. Consequently, physical therapists should be an important member of the group of health practitioners who work with patients who have head, neck, and orofacial pain.

References

- [1] Differential diagnosis and management considerations of temporomandibular disorders. In: Okeson JP, editor. *Orofacial pain; guidelines for assessment, diagnosis, and management*. Carol Stream (IL): Quintessence Publisher Co., Inc.; 1996. p. 45–52.
- [2] Hoving JL, Gross AR, Gasner D, et al. A critical appraisal of review articles on the effectiveness of conservative treatment for neck pain. *Spine* 2001;26(2):196–205.
- [3] A guide to physical therapist practice. Volume I: A description of patient management. *Phys Ther* 1995;75:70756.
- [4] APTA House of Delegates Policies HOD #06–93–22–43. Alexandria (VA): American Physical Therapy Association; 1996.

- [5] Clark G, Seligman D, Solberg W, et al. Guidelines for the treatment of temporomandibular disorders. *J Craniomandib Disord* 1990;4:80–8.
- [6] Feine J, Widmer C, Lund J. Physical therapy: a critique. Presented at the National Institutes of Health Technology Assessment Conference on Management of Temporomandibular Disorders. Bethesda (MD), April 29–May 1, 1996.
- [7] McNeill C, editor. Temporomandibular disorders. Guidelines for classification, assessment and management. 2nd edition. Carol Stream (IL): Quintessence Publishing Co., Inc; 1993.
- [8] Dworkin S, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examination and specifications critique. *J Craniomandib Disord* 1992;6: 301–55.
- [9] Kraus SL. Temporomandibular disorders. In: Saunders HD, Saunders Ryan R, editors. Evaluation, treatment and prevention of musculoskeletal disorders, vol. 1 - Spine. 4th edition. Chaska (MN): The Saunders Group, Inc.; 2004. p. 173–210.
- [10] Schiffman E, Braun B, Lindgren J, et al. Temporomandibular joint iontophoresis: a double-blind randomized clinical trial. *J Orofac Pain* 1996;10:157–65.
- [11] Shin S-M, Choi J-K. Effect of indomethacin phonophoresis on relief of TMJ pain. *J Craniomandibular Pract* 1997;15(4):345–8.
- [12] Watson T. The role of electrotherapy in contemporary physiotherapy practice. *Man Ther* 2000;5(3):132–41.
- [13] Dijkstra PU, de Bont LGM, Leeuw R, et al. Temporomandibular joint osteoarthritis and temporomandibular joint hypermobility. *J Craniomandibular Pract* 1993;11:268–75.
- [14] Westling L, Mattiasson A. General joint hypermobility and temporomandibular joint derangement in adolescents. *Ann Rheum Dis* 1992;51:87–90.
- [15] Dijkstra PU, de Bont LGM, Stegenga B, et al. Temporomandibular joint osteoarthritis and generalized joint hypermobility. *J Craniomandibular Pract* 1992;10:221–7.
- [16] Moffett BC. Definitions of temporomandibular joint derangements. In: Moffett BC, Westesson P-L, editors. Diagnosis of internal derangements of the temporomandibular joint, vol 1. Double-contrast arthrography and clinical considerations. Proceedings of a Continuing Dental Education Symposium. Seattle, 1984.
- [17] Milam S. Pathophysiology of articular disk displacements of the temporomandibular joint. In: Fonseca RJ, editor. Oral & maxillofacial surgery: temporomandibular disorders, vol. 4. 1st edition. Philadelphia: W.B. Saunders Company; 2000. p. 46–72.
- [18] Montgomery MT, Gordon SM, Van Sickels JE, et al. Changes in signs and symptoms following temporomandibular joint disc repositioning surgery. *J Oral Maxillofac Surg* 1992; 50(4):320–8.
- [19] Assael LA. Arthrotomy for internal derangements. In: Kaplan AS, Assael LA, editors. Temporomandibular disorders: diagnosis and treatment. Philadelphia: W.B. Saunders Company; 1991. p. 663–79.
- [20] Orenstein ES. Anterior repositioning appliances when used for anterior disk displacement with reduction—a critical review. *J Craniomandib Pract* 1993;11(2):141–5.
- [21] Chen CW, Boulton J, Gage JP. Splint therapy in temporomandibular joint dysfunction: a study using magnetic resonance imaging. *Aust Dent J* 1995;40(2):71–8.
- [22] de Leeuw R. Clinical signs of TMJ osteoarthritis and internal derangement 30 years after nonsurgical treatment. *J Orofac Pain* 1994;8:18–24.
- [23] Bays R. Surgery for internal derangement. In: Fonseca RJ, editor. Oral & maxillofacial surgery: temporomandibular disorders, vol. 4. 1st edition. Philadelphia: W. B. Saunders Company; 2000. p. 275–300.
- [24] Sollecito T. Role of splint therapy in treatment of temporomandibular disorders. In: Fonseca RJ, editor. Oral & maxillofacial surgery: temporomandibular disorders, vol. 4. 1st edition. Philadelphia: W. B. Saunders Company; 2000. p. 145–60.
- [25] Blaustein DI, Scapino RP. Remolding of the temporomandibular joint disc and posterior attachment in disc displacement specimens in relation to glycosaminoglycan content. *Plast Reconstr Surg* 1986;79:756–64.

- [26] Turell J, Ruiz HG. Normal and abnormal findings in temporomandibular joints in autopsy specimens. *J Craniomandib Disord* 1987;1:257–75.
- [27] Kircos LT, Ortendahl DA, Mark AS, et al. Magnetic resonance imaging of the TMJ disc in asymptomatic volunteers. *J Oral Maxillofac Surg* 1987;45:852–4.
- [28] Westesson PL, Eriksson L, Kurita K. Reliability of a negative clinical temporomandibular joint examination: prevalence of disk displacement in asymptomatic temporomandibular joints. *Oral Surg Oral Med Oral Pathol* 1989;68:551–4.
- [29] Kropmans TJ, Dijkstra PU, Stegenga B, et al. Therapeutic outcome assessment in permanent temporomandibular joint disc displacement. *J Oral Rehabil* 1999;26:357–63.
- [30] Kraus SL. Physical therapy management of temporomandibular disorders. In: Fonseca RJ, editor. *Oral & maxillofacial surgery: temporomandibular disorders*, vol. 4. 1st edition. Philadelphia: W. B. Saunders Company; 2000. p. 161–93.
- [31] Segami N, Murakami K-I, Iizuka T. Arthrographic evaluation of disk position following mandibular manipulation technique for internal derangement with closed lock of the temporomandibular joint. *J Craniomandib Disord* 1990;4:99–108.
- [32] Van Dyke AR, Goldman SM. Manual reduction of displaced disk. *J Craniomandib Pract* 1990;8:350–2.
- [33] Minagi S, Nozaki S, Sato T, et al. A manipulation technique for treatment of anterior disk displacement without reduction. *J Prosthet Dent* 1991;65:686–91.
- [34] Scapino RP. The posterior attachments: its structure, function, and appearance in TMJ imaging studies: Part 1. *J Craniomandib Disord* 1991;5(2):83–94.
- [35] Scapino RP. The posterior attachments: its structure, function, and appearance in TMJ imaging studies: Part 2. *J Craniomandib Disord* 1991;5(3):155–66.
- [36] Holmlund AB. Arthroscopy. In: Fonseca RJ, editor. *Oral & maxillofacial surgery: temporomandibular disorders*, vol. 4. 1st edition. Philadelphia: W. B. Saunders Company; 2000. p. 255–74.
- [37] Hardy MA. The biology of scar formation. *Phys Ther* 1989;69(12):22–32.
- [38] Akeson WH, Amiel D, Woo S. Immobility effects of synovial joints the pathomechanics of joint contracture. *Biorheology* 1980;17:17–95.
- [39] Salter RB. The biologic concept of continuous passive motion of synovial joints: the first 18 years of basic research and its clinical application. *Clin Orthop Relat Res* 1989;242:12–25.
- [40] Frank C, Akeson WH, Woo SL, et al. Physiology and therapeutic value of passive joint motion. *Clin Orthop Relat Res* 1984;185:113–25.
- [41] Moss R, Rum M, Sturgis E. Oral behavioral patterns in facial pain, headache, and non-headache populations. *Behav Res Ther* 1984;6:683–97.
- [42] Dao TTT, Lund JP, Lavigne GJ. Comparison of pain and quality of life in bruxers and patients with myofascial pain of the masticatory muscles. *J Orofac Pain* 1994;8:350–6.
- [43] Faulkner KDB. Bruxism: a review of the literature. Part I. *Aust Dent J* 1990;35:266–76.
- [44] Faulkner KDB. Bruxism: a review of the literature. Part II. *Aust Dent J* 1990;35:355–61.
- [45] Boero RP. The physiology of splint therapy: a literature review. *Angle Orthod* 1989;59(3):165–80.
- [46] Al-Ani MZ, Davies SJ, Gray RJM, et al. Stabilisation splint therapy for temporomandibular pain dysfunction syndrome. *Cochrane Database Syst Rev* 2004;(1):CD002778.
- [47] Molina OF, Santos JD, Mazzetto M, et al. Oral jaw behavior in TMD and bruxism: a comparison study by severity of bruxism. *J Craniomandib Pract* 2001;19:114–22.
- [48] Clark GT. Examining temporomandibular disorder patients for craniocervical dysfunction. *J Craniomandib Pract* 1984;2:56–63.
- [49] Clark GT, Green EM, Doman MR, et al. Craniocervical dysfunction levels in a patient sample from a temporomandibular joint clinic. *J Am Dent Assoc* 1987;115:251–6.
- [50] Kirveskari P, Alanen P, Karskela V, et al. Association of functional state of stomatognathic system with mobility of cervical spine and neck muscle tenderness. *Acta Odont Scand* 1988;46:281–6.

- [51] Isaccsson G, Linde C, Isberg A. Subjective symptoms in patients with temporomandibular joint disc displacement versus patients with myogenic craniomandibular disorders. *J Prosthet Dent* 1989;61:70–1.
- [52] Cacchiotti DA, Plesh O, Bianchi P, et al. Signs and symptoms in samples with and without temporomandibular disorders. *J Craniomandib Disord* 1991;5:167–72.
- [53] Braun BL, DiGiovanna A, Schiffman E, et al. A cross-sectional study of temporomandibular joint dysfunction in post-cervical trauma patients. *J Craniomandib Disord* 1992;6:24–31.
- [54] De Laat A, Meuleman H, Stevens A. Relation between functional limitations of the cervical spine and temporomandibular disorders [abstract]. *J Orofac Pain* 1993;1:109–10.
- [55] Padamsee M, Mehta N, Forgione A, et al. Incidence of cervical disorders in a TMD population [abstract]. *J Dent Res* 1994;186.
- [56] Lobbezoo-Scholte AM, De Leeuw JRJ, Steenks MH, et al. Diagnostic subgroups of craniomandibular disorders. Part 1: self-report data and clinical findings. *J Orofac Pain* 1995;9:24–36.
- [57] de Wijer A, Steenks A, de Leeuw MH, et al. Symptoms of the cervical spine in temporomandibular and cervical spine disorders. *J Oral Rehabil* 1996;23(11):742–50.
- [58] de Wijer A, de Leeuw JRJ, Steenks MH, et al. Temporomandibular and cervical spine disorders: self-reported signs and symptoms. *Spine* 1996;21:1638–46.
- [59] De Laat A, Meuleman H, Stevens A, et al. Correlation between cervical spine and temporomandibular disorders. *Clin Oral Investig* 1998;2:54–7.
- [60] Ciancaglini R, Testa M, Radaelli G. Association of neck pain with symptoms of temporomandibular dysfunction in the general adult population. *Scand J Rehab Med* 1999;31:17–22.
- [61] Visscher CM, Lobbezoo F, de Boer W, et al. Clinical tests in distinguishing between persons with or without craniomandibular or cervical spinal pain complaints. *Eur J Oral Sci* 2000;108:475–83.
- [62] Turp JC, Kowalski CJ, O’Leary N, et al. Pain maps from facial pain patients indicate a broad pain geography. *J Dent Res* 1998;77(6):1465–72.
- [63] Hagberg C, Hagberg M, Koop S. Musculoskeletal symptoms and psychological factors among patients with craniomandibular disorders. *Acta Odontol Scand* 1994;52:170–7.
- [64] Sipila K, Ylostalo P, Joukamaa M, et al. Comorbidity between facial pain, widespread pain, and depressive symptoms in young adults. *J Orofac Pain* 2006;20:24–30.
- [65] Olivo SA, Bravo J, Magee DJ, et al. The association between head and cervical posture and temporomandibular disorders: a systematic review. *J Orofac Pain* 2006;20(1):9–23.
- [66] Molina OF, dos Santos J, Nelson SJ, et al. Prevalence of modalities of headache and bruxism among patients with craniomandibular disorders. *J Craniomandib Pract* 1997;15:314–25.
- [67] Trenouth MJ. The relationship between bruxism and temporomandibular joint dysfunction as shown by computer analysis of nocturnal tooth contact patterns. *J Oral Rehabil* 1979;6:81–7.
- [68] Eriksson PO, Zafar H, Nordh E. Concomitant mandibular and head-neck movements during jaw opening-closing in man. *J Oral Rehab* 1998;25:859–70.
- [69] Hu JW, Yu XM, Vernon H, et al. Excitatory effect on neck and jaw muscle activity of inflammatory irritant applied to cervical paraspinal muscles. *Pain* 1993;55:243–50.
- [70] McCouch G, Deering I, Ling T. Location of receptors for tonic neck reflexes. *J Neurophysiol* 1951;14:191–6.
- [71] Sumino R, Nozaki S, Katoh M. Trigemino-neck reflex. In: Kawamura Y, Dubner R, editors. *Oral-facial sensory and motor functions*. Tokyo: Quintessence Books Publishing Co Inc.; 1981. p. 81–8.
- [72] Wyke BD. Neurology of the cervical spinal joints. *Physiotherapy* 1979;65:72–6.

- [73] Funakoshi M, Amano N. Effects of the tonic neck reflex on the jaw muscles of the rat. *J Dent Res* 1973;52:668–73.
- [74] Komiyama O, Arai M, Kawara M, et al. Pain patterns and mandibular dysfunction following experimental trapezius muscle pain. *J Orofac Pain* 2005;19:119–26.
- [75] Svensson P, Arendt-Nielsen L. Muscle pain modulates mastication: an experimental study in humans. *J Orofac Pain* 1998;12:7–16.
- [76] Komiyama O, Arai M, Kawara M, et al. [Effects of experimental pain induced in trapezius muscle on mouth opening]. *J Jpn Soc TMJ* 2003;15:173–7 [in Japanese].
- [77] Sessle BJ, Hu JW, Amano M, et al. Convergence of cutaneous, tooth pulp, visceral, neck and muscle afferents onto nociceptive and nonnociceptive neurons in trigeminal subnucleus caudalis and its implications for referred pain. *Pain* 1986;27:219–35.
- [78] Carlson CR, Okeson JP, Falace DA, et al. Reduction of pain and EMG activity in the masseter region by trapezius trigger point injection. *Pain* 1993;55:397–400.
- [79] Clark GT, Browne PA, Nakano M, et al. Co-activation of sternocleidomastoid muscles during maximum clenching. *J Dent Res* 1993;72:1499–502.
- [80] Ehrlich R, Garlick D, Ninio M. The effect of jaw clenching on the electromyographic activities of 2 neck and 2 trunk muscles. *J Orofac Pain* 1999;13:115–20.
- [81] Eriksson PO, Haggman-Henrikson B, Nordh E, et al. Co-ordinated mandibular and head-neck movements during rhythmic jaw activities in man. *J Dent Res* 2000;79:1378–84.
- [82] Sherrington CS. *The integrative action of the nervous system*. 2nd edition. New Haven (CT): Yale Press; 1906.
- [83] Kapandji IA. *The physiology of the joint*. The Trunk and Vertebral Column 1974;3:170–251.
- [84] Ralston HJ, Libet B. The question of tonus in skeletal muscle. *Am J Phys Med* 1953;32:85–92.
- [85] Smith AM. The coactivation of antagonist muscles. *Can J Physiol Pharmacol* 1981;59:733–47.
- [86] Major PW, Nebbe B. Use and effectiveness of splint appliance therapy: review of literature. *J Craniomandibular Pract* 1997;15:159–66.
- [87] Clark GT. A critical evaluation of orthopedic interocclusal appliance therapy: design, theory and overall effectiveness. *J Am Dent Assoc* 1984;108:359–64.
- [88] Posselt U. *Studies on the mobility of the human mandible*. *Acta Odontol Scan* 1952;10:1–50.
- [89] Lawrence ES, Razook SJ. Nonsurgical management of mandibular disorders. In: Kraus SL, editor. *Temporomandibular disorders*. 2nd edition. New York: Churchill Livingstone Inc.; 1994. p. 125–60.
- [90] Mohl N. *Head posture and its role in occlusion*. *New York State Dent J* 1976;42:17–23.
- [91] McLean LF. *Gravitational influences on the afferent and efferent components of mandibular reflexes* [doctoral dissertation]. Philadelphia: Thomas Jefferson University of Philadelphia; 1973.
- [92] Lund P, Nishiyama T, Moller E. Postural activity in the muscles of mastication with the subject upright, inclined, and supine. *Scand J Dent Res* 1970;78:417–24.
- [93] Eberle WR. A study of centric relation as recorded in a supine position. *J Am Dent Assoc* 1951;42:15–26.
- [94] Mclean LF, Brenman HS, Friedman MGF. Effects of changing body position on dental occlusion. *J Dent Res* 1973;52:1041–5.
- [95] Goldstein DF, Kraus SL, Williams WB, et al. Influence of cervical posture on mandibular movement. *J Prosthet Dent* 1984;52:421–6.
- [96] Darling DW, Kraus SL, Glasheen-Wray MB. Relationship of head posture and the rest position of the mandible. *J Prosthet Dent* 1984;52:111–5.
- [97] Root GR, Kraus SL, Razook SJ, et al. Effect of an intraoral appliance on head and neck posture. *J Prosthet Dent* 1987;58:90–5.

- [98] Mohl ND. The role of head posture in mandibular function. In: Solberg WK, Clark GT, editors. *Abnormal jaw mechanics diagnosis and treatment*. Chicago (IL): Quintessence Publishing; 1984. p. 97–111.
- [99] Mausner JS, Kramer S. Screening in the detection of disease. In: Mausner, Baum, editors. *Epidemiology: an introductory text*. Philadelphia: WB Saunders Co.; 1985. p. 214–37.
- [100] Ness TJ, Gebhart GF. Visceral pain: a review of experimental studies. *Pain* 1990;41:167–234.
- [101] Jarvik J, Deyo R. Diagnostic evaluation of low back pain with emphasis on imaging. *Ann Intern Med* 2002;137:586–97.
- [102] Spitzer WO, LeBlanc FE, Dupuis M, et al. Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for Clinicians Report of the Quebec Task Force on Spinal Disorders. *Spine* 1987;12:S4–59.
- [103] Cote P, Cassidy JD, Carroll L. The Saskatchewan Health and Back Pain Survey: the prevalence of neck pain and related disability in Saskatchewan adults. *Spine* 1998;23:1689–98.
- [104] Andersson HI, Ejlertsson G, Leden I, et al. Chronic pain in a geographically defined general population: studies of differences in age, gender, social class, and pain localization. *Clin J Pain* 1993;9:174–82.
- [105] Bovim G, Schrader H, Sand T. Neck pain in the general population. *Spine* 1994;19:1307–9.
- [106] International Headache Society Classification Committee. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalalgia* 1998;8(Suppl 7):9–96.
- [107] Bogduk N. The neck and headaches. *Neurol Clin* 2004;22:151–71.
- [108] Kerr FWL. Structural relation of the trigeminal spinal tract to upper cervical roots and the solitary nucleus in cat. *Exp Neurol* 1961;4:134–48.
- [109] Bogduk N. The anatomical basis for cervicogenic headache. *J Manipulative Physiol Ther* 1992;15:67–70.
- [110] Bogduk N. Anatomy and physiology of headache. *Biomed Pharmacother* 1995;49:435–45.
- [111] Bogduk N. Cervical causes of headache and dizziness. In: Grieve G, editor. *Modern manual therapy*. Edinburgh (UK): Churchill Livingstone. 2nd edition. 1994. p. 317–31.
- [112] Michler RP, Bovim G, Sjaastad O. Disorders in the lower cervical spine. A cause of unilateral headache? A case report. *Headache* 1991;31:550–1.
- [113] Bogduk N. The clinical anatomy of the cervical dorsal rami. *Spine* 1982;7(4):319–29.
- [114] Bogduk N. The anatomy of occipital neuralgia. *Clin Exp Neural* 1980;17:167–84.
- [115] Hildebrandt J, Jansen J. Vascular compression of the C2 and C3 roots—yet another cause of chronic intermittent hemicrania? *Cephalalgia* 1984;4:168–70.
- [116] Rosenberg W, Swearingen B, Poletti C. Contralateral trigeminal dysaesthesias associated with second cervical nerve compression: a case report. *Cephalalgia* 1990;10:259–62.
- [117] Sjaastad O, Saunte C, Hovdahl H, et al. “Cervicogenic” headache. An hypothesis. *Cephalalgia* 1983;3:249–56.
- [118] Biondi DM. Cervicogenic headache: a review of diagnostic and treatment strategies. *J Am Osteopath Assoc* 2005;105(4):16S–22S.
- [119] Nilson AN. The prevalence of cervicogenic headache in a random population sample of 20–59 year olds. *Spine* 1995;20:1884–8.
- [120] Pfaffenrath V, Kuabe H. Diagnostics of cervical spine headache. *Funct Neurol* 1990;5:157–64.
- [121] Balla J, Lansek R. Headache arising from disorders of the cervical spine. In: Hopkins A, editor. *Headache: problems in diagnosis and management*. London: Saunders; 1988. p. 241–67.
- [122] Vernon H, Steiman I, Hagino C. Cervicogenic dysfunction in muscle contraction headache and migraine: a descriptive study. *J Manipulative Physiol Ther* 1992;15:418–29.
- [123] Yi X, Cook AJ, Hamill-Ruth RJ, et al. Cervicogenic headache in patients with presumed migraine: missed diagnosis or misdiagnosis? *J Pain* 2005;6(10):700–3.
- [124] Blau JN, MacGregor EA. Migraine and the neck. *Headache* 1994;34:88–90.

- [125] Bartch T, Goadsby PJ. The trigeminocervical complex migraine: current concepts and synthesis. *Curr Pain Headache Rep* 2003;7:371–6.
- [126] DeNarinis M, Accornero N. Recurrent neck pain as a variant of migraine: description of four cases. *J Neurol Neurosurg Psychiatry* 1997;62:669–70.
- [127] Marcus D, Scharff L, Mercer MA, et al. Musculoskeletal abnormalities in chronic headache; a controlled comparison of headache diagnostic groups. *Headache* 1999;39:21–7.
- [128] Shevel E, Spierings E. Cervical muscles in the pathogenesis of migraine headache. *J Headache Pain* 2004;5(1):12–4.
- [129] Lance J. Mechanism and management of headache. 5th edition. Oxford (UK): Butterworth-Heinemann; 1993.
- [130] Brown J. A systemic approach to the dizzy patient. *Neurol Clin* 1990;8:209–24.
- [131] Fisher CM. Vertigo in cerebrovascular disease. *Arch Otolaryngol* 1967;85:529–34.
- [132] Hoffman RM, Einstadter D, Kroenke K. Evaluating dizziness. *Am J Med* 1999;107:468–78.
- [133] Reker V. Cervical nystagmus caused by proprioceptors of the neck. *Laryngologica Rhinol Otol Stuttgart* 1983;62:312–4.
- [134] Cohen L. Role of eye and neck proprioceptive mechanisms in body orientation and motor coordination. *J Neurophysiol* 1961;24:1–11.
- [135] Abrahams VC. The physiology of neck muscles; their role in head movement and maintenance of posture. *Can J Physiol Pharmacol* 1977;55(3):332–8.
- [136] Manzoni D, Pompeiano O, Stampacchia G. Tonic cervical influences on posture and reflex movements. *Arch Ital Biol* 1979;117(2):81–110.
- [137] Cope S, Ryan GMS. Cervical and otolith vertigo. *J Laryngol Otol* 1959;73:113–20.
- [138] Gray LP. Extra labyrinthine vertigo due to cervical muscle lesions. *J Laryngol Otol* 1956;70(6):352–61.
- [139] Weeks VD, Travell J. Postural vertigo due to trigger areas in the sternocleidomastoid muscle. *J Pediatr* 1955;47(3):315–27.
- [140] De Jong PTVM, De Jong JMBV, Cohen B, et al. Ataxia and nystagmus induced by injection of local anesthetics in the neck. *Ann Neurol* 1977;1:240–6.
- [141] Hinoki M. Vertigo due to whiplash injury: a neurotological approach. *Acta Otolaryngol (Stockh) Suppl* 1985;419:9–29.
- [142] Norre ME. Neurophysiology of vertigo with special reference to cervical vertigo. A review. *Acta Belg Med Phys* 1986;9:183–94.
- [143] Phillipszoon A. Neck torsion nystagmus. *Pract Otorhinolaryngol (Basel)* 1963;25:339–44.
- [144] Rubinstein B. Tinnitus and craniomandibular disorders: is there a link? *Swed Dent J Suppl* 1993;95:1–46.
- [145] Young ED, Nelken I, Conley RA. Somatosensory effects on neurons in dorsal cochlear nucleus. *J Neurophysiol* 1995;73:743–65.
- [146] Levine RA. Somatic (craniocervical) tinnitus and the dorsal cochlear nucleus hypothesis. *Am J Otolaryngol* 1999;20:351–62.
- [147] Abel MD, Levine RA. Muscle contractions and auditory perception in tinnitus patients and nonclinical patients. *J Craniomandibular Pract* 2004;22(3):181–91.
- [148] Paparella MM, Jung TTK. Odontalgia. In: Paparella MM, Shumrik DA, Gluckman JL, editors. *Otolaryngology*. Philadelphia: Saunders; 1991. p. 1237–42.
- [149] Kuttilla S, Kuttilla M, Le Bell Y, et al. Characteristics of subjects with secondary otalgia. *J Orofac Pain* 2004;18(3):226–34.
- [150] Kuttilla S, Kuttilla M, Le Bell Y, et al. Aural symptoms and signs of temporomandibular disorder in association with treatment need and visits to a physician. *Laryngoscope* 1999;109:1669–73.
- [151] Olsen J, editor. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. 1st ed. Copenhagen (Denmark): The International Headache Society; 1990.
- [152] Spitzer WO, Skovron ML, Salmi LR, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining “whiplash” and its management. *Spine* 1995;20(8 Suppl):1S–73S.

- [153] Jordan A, Manniche C, Mosdal C. The Copenhagen Neck Functional Disability Scale: a study of reliability and validity. *J Manipulative Physiol Ther* 1998;21(8):520–7.
- [154] Feise RJ, Micheal MJ. Functional Rating Index: a new valid and reliable instrument to measure the magnitude of clinical change in spinal conditions. *Spine* 2001;26(1):78–87.
- [155] Manchikanti LS, Peter SS, Vijay S, et al. Evidence-based practice guidelines for interventional techniques in the management of chronic spinal pain. *Pain Phys* 2003;6:3–81.
- [156] Abenhaim L, Rossignol M, Valat J-P, et al. International Paris Task Force on Back Pain. *Spine* 2000;25(4 Suppl):1S–31S.
- [157] Persson LCG, Carlsson C-A, Carlsson JY. Long-lasting cervical radicular pain managed with surgery, physiotherapy, or a cervical collar. *Spine* 1997;22:751–8.
- [158] Bednarik J, et al. The value of somatosensory- and motor-evoked potentials in predicting and monitoring the effect of therapy in spondylotic cervical myelopathy. Prospective randomized study. *Spine* 1999;24(15):1593–8.
- [159] Kadanka Z. Approaches to spondylotic cervical myelopathy: conservative versus surgical results in a 3-year follow-up study. *Spine* 2002;27(20):2205–10.
- [160] Raphael KG, Klausner JJ, Nayak S, et al. Complementary and alternative therapy use by patients with myofascial temporomandibular disorders. *J Orofac Pain* 2003;17:36–41.
- [161] Gross AR, Hoving JL, Haines TA, et al. Cervical overview group manipulation and mobilisation for mechanical neck disorders. *Cochrane Database Syst Rev* 2004;(1):CD004249.
- [162] Aker PD, Gross AR, Goldsmith CH, et al. Conservative management of mechanical neck pain: systematic overview and meta-analysis. *Br Med J* 1996;313(7068):1291–6.
- [163] Hurwitz EL, Aker PD, Adams AH, et al. Manipulation and mobilization of the cervical spine: a systematic review of literature. *Spine* 1996;21:1746–60.
- [164] Jordan A, Bendix T, Nielsen H, et al. Intensive training, physiotherapy, or manipulation for patients with chronic neck pain: a prospective single-blinded randomized clinical trial. *Spine* 1998;23:311–9.
- [165] Nelson BW, Carpenter DM, Dreisinger TE, et al. Can spinal surgery be prevented by aggressive strengthening exercises? A prospective study of cervical and lumbar patients. *Arch Phys Med Rehabil* 1999;80:20–5.
- [166] Rodriguez AA, Bilkey WJ, Agre JC. Therapeutic exercise in chronic neck and back pain. *Arch Phys Med Rehabil* 1992;73:870–5.
- [167] Swezey RL, Swezey AM, Warner K. Efficacy of home cervical traction therapy. *Am J Phys Med Rehabil* 1999;78(1):30–2.
- [168] Jull GA, Trott P, Potter H, et al. A randomized, controlled trial of exercise and manipulative therapy for cervicogenic headache. *Spine* 2002;27(17):1835–43.
- [169] Farrell JP, Jensen GM. Manual therapy: a critical assessment of role in the profession of physical therapy. *Phys Ther* 1992;12(2):11–20.
- [170] Goldstein M. The research status of spinal manipulative therapy 1975. US Department Of Health, Education, and Welfare Publication No. (NIH) 76–998, NINCDS Monograph No. 15.
- [171] Korr IM. The neurobiologic mechanisms in manipulative therapy. New York: Plenum Press; 1978.
- [172] O’Leary S, Falla D, Jull G. Recent advances in therapeutic exercise for the neck: implications for patients with head and neck pain. *Aust Endod J* 2003;29(3):138–42.
- [173] Kay TM, Gross A, Santaguida PL, et al. Cervical Overview Group. Exercises for mechanical neck disorders. *Cochrane Database Syst Rev* 2005;(3):CD004250.
- [174] Al-Obaidi S, et al. The role of anticipation and fear of pain in the persistence of avoidance behavior in patients with chronic low back pain. *Spine* 2000;25(9):1126–31.
- [175] Wrubel J, et al. Social competence from the perspective of stress and coping. In: Wine J, Smye M, editors. *Social competence*. New York: Guilford Press; 1981. p. 61–99.
- [176] Pincus T. A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine* 2002;27(5):E109–20.

- [177] Waddell G, Newton M, Henderson I, et al. A fear-avoidance beliefs questionnaire (FABQ), the role of fear-avoidance belief in chronic low back pain and disability. *Pain* 1993;52: 157–68.
- [178] Wong A, et al. The traction angle and cervical intervertebral separation. *Spine* 1992;17(2): 136–8.
- [179] Joghataei MT, Arab AM, Khaksar H. The effect of cervical traction combined with conventional therapy on grip strength on patients with cervical radiculopathy. *Clin Rehabil* 2004; 18(8):879–87.

Author's personal copy