Orofacial Pain

Guidelines for Assessment, Diagnosis, and Management

Fourth Edition

The American Academy of Orofacial Pain

Edited by Reny de Leeuw, DDS, PhD

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American Academy of Orofacial Pain Guidelines for Assessment, Diagnosis, and Management

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Cervicogenic Mechanisms of Orofacial Pain and Headaches

Neuromusculoskeletal structures within the cervical spine can contribute to orofacial pain dysfunction syndromes, separate from or in addition to temporomandibular disorders (TMDs). The clinician should be able to understand the mechanisms of referred cervicogenic pain, perform a screening evaluation so that an accurate diagnosis can be established, and initiate appropriate referral for further evaluation and comprehensive management.

Cervical spine disorders (CSDs) is a term that embraces a number of disorders involving the muscles, facet joints, discs, and nerves of the cervical spine. Symptoms vary with physical activity and/or static positioning but may develop spontaneously or follow trauma. Subclassification of CSDs includes an extensive list of traditional diagnostic terms, such as *cervical sprain/strain*, *discogenic disease*, *facet syndrome*, *myositis*, *fibrositis*, *fibromyositis*, and *articular hypo-* and *hypermobility*.[1] Patients may have two or three diagnoses by different physicians, depending on whether these physicians focused on pathophysiology, radiology, clinical symptoms, physical symptoms, duration of episode, or a combination of these factors.[1] In the absence of disease and fracture, determining the cause of cervical pain is difficult. To facilitate communication among health care professionals, a CSD subclassification scheme is proposed in Box 9-1. The proposal is adapted from guidelines used for low back pain and focuses on signs and symptoms that provide a better description of inclusion and exclusion criteria in a common language.[2,3]

Box 9-1 CSD subclassifications

Category 1	Neck symptoms without musculoskeletal and neurologic signs
Category 2	Neck symptoms without neurologic signs; musculoskeletal signs are present with a decrease in cervical range of motion and/or tension, plus pain on palpation of the cervical muscles
Category 3	Neck symptoms with musculoskeletal and neurologic signs that may include decreased or absent deep-tendon reflexes, weakness, and sensory deficits
Category 4	Radiation of symptoms or cephalic symptoms in addition to symptoms of category 1, 2, or 3; cephalic symptoms may include headaches, dizziness/unsteadiness, nausea/vomiting, tinnitus, visual problems, dysphagia, memory/cognitive problems, and reduced/painful jaw movements

Epidemiology: Prevalence of CSDs Coexisting with TMDs

Between 5% and 6% of the US population visit health care professionals for persistent and severe TMD pain, and 8% seek help for CSDs.[4,5] There appears to be a considerable overlap of signs and symptoms in patients with TMDs and CSDs, especially in those patients with myogenous TMDs.[6,7] Many studies have reported the coexistence of TMD and CSD symptoms.[4,6,8–10] The prevalence of CSDs in TMD patients ranges from 23% to 70%, compared with 5% to 31% in control groups,[11–13] and patients with CSDs report more signs and symptoms of TMDs than healthy controls.[9] For both types of disorders, prevalence generally increases in frequency and intensity with age up to the fifth decade and is higher among women than among men.[8,11,14] Comorbidity of TMDs and CSDs can also be a symptom of a more generalized musculoskeletal disorder. TMD patients regularly meet the criteria for fibromyalgia, and parts of the fibromyalgia syndrome include cervical spinal pain complaints. Studies have shown that 70% to 75% of patients with fibromyalgia syndrome met TMD criteria,[15] whereas 13% to 18% of TMD patients met fibromyalgia syndrome criteria.[16–18] In addition, up to 70% of patients with fibromyalgia met the case definition for chronic fatigue syndrome, and vice versa.[17] It is clear that these disorders frequently co-occur and share key symptoms.[19]

Cervical Spine–Craniomandibular Connection

A close functional coupling between the structures of the neck and the craniomandibular region has been described. In 1950, Brodie[20] suggested a biomechanical model of the musculoskeletal structures of the head and neck.

The cranium and mandible both have muscular and ligamental attachments to the cervical area, forming a functional system called the *craniocervical mandibular* or *stomatognathic system*. A change in the activity

of neck muscles and head position influences the function of the masticatory muscles and jaw function, and vice versa. Therefore, postural or orthostatic stability of the cranium over the cervical spine has been suggested to be a consideration in the diagnosis of cervical and TMD pain.[21,22] Because of these reciprocal functions, the cervical spine and musculature need to be considered in the assessment of the patient with orofacial pain complaints.[22]

Relationship between head posture and mandibular arthrokinematics

When the mandible is at rest, its position is determined by the tone of the masticatory muscles and the associated soft tissues as they react to gravitational pull.[23–25] When the head is held in an upright position, the relaxed mandible maintains a fairly constant distance from the maxilla of approximately 2 to 5 mm.[25] Since cervical and masticatory muscles function reciprocally, there is no movement in the neck that would not be reflected in the jaw musculature. Therefore, in the dental literature, the influence of head and neck posture on mandibular rest position and concomitant masticatory muscle activity has gained much attention.[26–31] For example, it has been suggested that when the head is held in a more forward posture, tension in the supra- and infrahyoid musculature increases, with a consequent depression of the mandible.[26] Others, however, have described an elevation of the mandible when the head is held in a forward posture, and this was later confirmed in an experimental study.[27,28] Moreover, an electromyographic (EMG) study found a positive relationship between extension of the head and activity of the masseter muscles.[29] It is still unclear whether there is an association between abnormal cervical and/or head posture and TMDs.[7,32–36]

Besides static changes in mandibular rest position and masticatory muscle activity, mandibular kinematic changes have been suggested. Okeson[14] speculated that when the head is extended, the incisal points of the mandibular central incisors traverse a path of closure that is posterior to the same path traversed when the head is in the "natural head posture." And when the head is in a flexed posture, as during eating, the opposite occurs. Recently, in a kinesiographic study, this relationship between head posture and incisal movement paths was confirmed.[37] The same study showed that when the head is held in a forward head posture, the mandibular condyle is pulled slightly downward during opening and closing movements, whereas during movements in a military head position or during lateroflexion of the head, the condyle is pressed against the articular eminence. In addition, others have shown that jaw opening is always accompanied by head-neck extension, and jaw closing by head-neck flexion.[38] Furthermore, EMG recordings indicated active repositioning of the head during jaw movements. The authors concluded that these results provide further support to the concept of a functional trigeminocervical coupling during jaw activities in humans. As a result of this synergistic relationship, it is speculated that various CSDs may influence pain and dysfunction in the craniomandibular complex.

CSDs as a cause of masticatory muscle pain and hyperactivity

The following two theories postulate how CSDs may be a predisposing, precipitating, or perpetuating factor in masticatory muscle pain or hyperactivity.

1. *Convergence and central excitation of cervical sensory and motor neurons stimulates trigeminal motor neurons, causing an increase in masticatory activity.*

Stimulation of tissues innervated by the upper cervical segments has been demonstrated to influence motor activity of the trigeminal-innervated muscles of mastication.[39-42] Electrical stimulation applied to the central end of the ablated first cervical nerve has demonstrated EMG activity in masticatory muscles.[43] Experimental work has demonstrated that patients with an upper trapezius trigger point and ipsilateral masseter pain show decreased discomfort and a reduction in masseteric EMG activity after a single trigger-point injection in the upper trapezius.[40,43] Studies suggest that experimental trapezius pain can spread over a wide area to include the temporomandibular region, with elevation of masticatory activity and reduction of mouth opening.[44] Pain originating from the cervical spine also can cause changes in trigeminal-innervated musculature. It is speculated that a similar convergence and central excitation as seen with trigeminal and cervical sensory neurons may also exist for trigeminal motor neurons.[45,46]

2. *Masticatory muscles (antagonists) are activated in response to contraction of cervical spine muscles (agonists) by the process of cocontraction.*

Coordinated head, neck, and mandibular movements suggest a neurophysiologic and biomechanical interplay involving agonist/antagonist actions between the cervical and masticatory musculature.[47-50] The cervical and masticatory muscles can be viewed as agonistic and antagonistic to one another, and reciprocal innervation may play a role in modifying excitatory and inhibitory levels of appropriate neurons.[49-52] Daily events may cause the muscles of mastication to disproportionately contract in response to cervical 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 long duration, it is more likely that the muscles of mastication will disproportionately contract.[53]

Screening Evaluation of the Cervical Spine

In the last two decades, classification criteria for TMDs and CSDs were proposed, which allow for standardization and replication of the physical examination used to recognize these disorders.[14,54,55] As these criteria evolve, validation of the taxonomic systems remains in process. Moreover, even though both disorders concern musculoskeletal structures, there is no universal diagnostic system that can be applied to both TMDs and CSDs.

Similar to the diagnostic process for TMDs, a chronologic history of treatments, sleep and work positions, functional limitations, and successful pain modifiers is critical to the diagnosis of a CSD. It is beyond the scope of this chapter to describe a thorough examination of the cervical spine.[56-58] The screening clinical examination may include assessment of the active range of motion and recording of responses to palpation of the cervical spine and associated muscles.

The active range of motion of the head and cervical spine may be observed and pain responses noted during extension, flexion, rotation, and side-bending head move-

ments. Passive range of motion of the cervical spine should not be evaluated unless the clinician has had specific training in these techniques. For palpation evaluation, the same procedure used for the muscles of mastication may be used for assessing the cervical muscles. A neurologic examination consisting of muscle, reflex, and sensory testing will complete the cervical spine examination.

Important cervical muscles or muscle groups to evaluate are the sternocleidomastoid (SCM), suboccipital, paravertebral (scalenes), posterior deep cervical, and upper trapezius muscles. If further evaluation of the cervical region is indicated, referral to an appropriately trained clinician is recommended. Suboccipital pain syndromes are usually evaluated by physical therapists or spine specialists, who perform postural assessment as well as palpation for the presence of myalgic bands, trigger points, or tender facet joint pillars.[56,59-64] Craniofacial pain may either be triggered, reduced, or enhanced by manual segmental movement (compression/distraction), static and dynamic pain tests, or postural provocation testing techniques such as foraminal encroachment (Spurling test) and maximization of the forward head posture.[56,64,65]

Radiographs provide an ideal means by which mobility of the cervical spine is evaluated. Conventional radiography forms the basis of examination of the patient with acute cervical spine pathology, and a minimum of two views, preferably obtained in perpendicular planes, is essential. Typically, films are obtained with natural head position in neutral lateral, flexed, extended (retroflexion), oblique, and anteroposterior directions.[66-69]

If findings indicate nerve root involvement, further study with magnetic resonance imaging (MRI) or computerized tomography (CT) is recommended. Both MRI and CT techniques require the subject to be in a supine position, however, which eliminates the weight-bearing compression forces that are more pronounced by everyday postural situations and thus MRI and CT results may not be truly indicative of the effects of those functional and compressive forces. Obtaining, reading, or even requesting such imaging generally falls beyond the scope of the orofacial pain specialist. Patients not responding to physical therapy may need to have their source of pain verified and evaluated by the use of short-acting local anesthetic blocks to the suspected region or structure.[70-73]

Craniofacial Pain Syndromes of Cervical Origin

Pain disorders of the cervical spine can involve a variety of structures, such as fascia, muscles, ligaments, joints, bones, and vascular and neural tissues. Symptoms such as dizziness, gastrointestinal complaints, and visual, memory, and cognition problems may accompany some of these disorders, and the presence of these symptoms may contribute to misdiagnosis.[74,75]

Knowledge of the anatomy and innervation of the various structures that make up the suboccipital spine is required to understand the myriad pain syndromes that originate from this region and often mimic forms of headache and orofacial pain. Also, understanding the referral patterns and characteristics of pain and dysfunction can provide the dental practitioner and physical therapist with the means by which to delineate the structure that is causing the pain.

Innervation of the cervical spine

The cervical spine comprises the suboccipital and mid-lower cervical sections.The suboccipital spine is considered the switching station for all afferent and efferent transmission to the cranium and orofacial region. The first three cervical nerves (C1-C3) mediate pain at the suboccipital spine and may refer pain to the craniofacial region. These nerves innervate the ligaments and joints of the suboccipital spine, the anterior, posterior, and lateral suboccipital muscles, as well as the SCM and upper trapezius. They also innervate the posterior dura mater, tentorium and falx cerebelli, vertebral arteries, and the lateral walls of the posterior cranial fossa.

At the base of the occiput, the foramen magnum contains the meningeal branches of C1-C3, the vertebral and spinal arteries, and the spinal components of the spinal accessory nerve. The posterior cranial fossa contains the confluence of sinuses, the roots of the fifth to twelfth cranial nerves, the first two cervical nerves via the hypoglossal canal, the branches of C2-C3 through the foramen magnum, and the ascending branches of C1-C3, thus comprising several potential pathways for cervicogenic referral to the craniofacial region.[76-79] The greater occipital nerve branches off from the C2 dorsal root ganglia and thus may represent a source for occipital pain with or without retroorbital referral. Suboccipital spinal neurons have been shown to be excited by ipsilateral vagal input and correspond to dermatomal receptive fields of the upper cervical segments. They may represent another referral mechanism to the neck and jaw.[80,81]

The spinal accessory nerve, which arises from the C2-C4 levels, innervates the SCM and upper trapezius, both of which commonly refer pain to the craniofacial region. Spinal accessory fibers also cross the midline, providing implications for contralateral or bilateral frontal headache.[82-85]

Another source of cranial pain is transmitted via the C1-C3 sinuvertebral nerves, which innervate the cranial membranes, dura mater of the posterior cranial fossa, and epidural vasculature.[76,77] From the C3 level and below, the sinuvertebral nerves also provide innervation to the outer third of the intervertebral annular fibers as well as the cruciate, posterior longitudinal, and atlantoaxial ligaments.[76,78,79,86] The occipitoatlantal and atlantoaxial levels are devoid of intervertebral discs; however, sinuvertebral nerves are present at these two levels.

The SCM and upper trapezius are involved in positional control of the head in space along with the ligaments, facet joints, and capsules of the atlas and axis. Proper head position, balance, and equilibrium depend on a composite of visual and vestibular signals in addition to mechanical input from the suboccipital spine. Abnormal suboccipital afferent input can therefore contribute to torticollis, dizziness, nystagmus, vertigo, and disequilibrium in conjunction with craniofacial pain.[82,85,87,88]

Common CSDs

While it is important for the dental practitioner to be aware of the cervical etiologic factors in orofacial pain, cervical disorders are not primarily managed in the dental setting. Patients with cervical disorders should be referred to a knowledgeable physical therapist or cervical spine specialist for evaluation and treatment.

Cervicalgia (ICD-9 *723.1*)

Cervicalgia is a broad term meaning pain in the neck and represents the most common neck pain complaint. Although the pain may originate from any cervical structure, discomfort is primarily felt in the suboccipital area, SCM, and upper trapezius, with possible referral to the frontal, temporoparietal, occipital, vertex, and orbital regions. In addition, auditory, gastrointestinal, vestibular, and visual symptoms can occur as a result of vertebral artery involvement or convergence at the suboccipital region via neural interaction at the trigeminocervical complex with the spinal accessory and vagus nerves. Suboccipital discomfort is frequently associated with headache, nausea, vomiting, diplopia, dysphagia, and respiratory distress, which may cause the inexperienced clinician to immediately consider a primary vascular component. Dizziness can result from altered proprioception due to increased nociceptive input to cervical facet mechanoreceptors or to hyperactivity of the SCM.[41,56,74,75,88] Gastrointestinal disturbances may be due to involvement of the vagus nerve. The presence of these red flags should prompt the clinician to seek additional evaluation and care for such patients, which may include referral to an experienced physical therapist (Box 9-2).

Delayed referral in the presence of red flags and associated cervical hypomobility and/or postural dysfunction may lead to adaptive soft tissue shortening and joint restriction. Complications may progress to intervertebral disc bulging/herniation (*ICD-9* 722.0) as well as spondylosis (*ICD-9* 721.0/721.1). Intervertebral disc bulging or herniation may also occur as a result of acute trauma, whereas spondylosis and/or disc involvement can also be the result of cumulative microtrauma due to longstanding postural dysfunction. The association between cervicalgia and headache is significant.[89,90]

Box 9-2 Symptoms requiring referral

- Gradual worsening of symptoms
- Constant upper quadrant pain
- Upper extremity paresthesias
- Marked postural changes
- Marked limitation of cervical mobility
- Disorientation
- Respiratory distress
- Dizziness, vertigo, nystagmus
- Nausea, vomiting, dysphagia, globus
- Visual deficits
- Unrelenting headache

Cervical strain (ICD-9 *847.0*)

The most frequent traumatic spinal injury encountered in medical practice is a sprain or strain of the cervical spine following a motor vehicle accident.[91] Other terms used to describe this disorder are *flexion-extension injury*, *acceleration-deceleration injury*, and *whiplash*. The location and nature of the injuries that occur as a result of head and neck strain vary depending on the severity of the trauma and the structures involved.[91–96] The incidence of soft tissue injury to the cervical spine and resultant neck pain along with postural changes is significant.[91,93–96] Depending on the mechanism of injury, a cervical strain can affect the anterior, posterior, or lateral structures of the cervical spine, as well as the shoulder girdle in whole or in part. Therefore, the Quebec Task Force suggests use of the term *whiplash-associated disorder*.[94] Recent studies show that patients

with whiplash-associated disorder have significantly more signs and symptoms of TMDs than controls.[97–99]

The upper cervical spine usually sustains the greatest injury in flexion-extension injuries, because it is a pivot point. Acute suboccipital flexion and concomitant extension of the lower cervical spine may also result in increased dural tension and increased tension of the C1-C3 roots.[92] Postural factors such as a head-rest that is placed too low or a posteriorly inclined seat back may result in hyperextension of the head past the limit of stretch of the soft tissues of the neck, which can lead to a compression fracture of the posterior arch of the atlas.[96] Lateral whiplash can result in tractional injuries to the brachial plexus that may even cause nerve root avulsion, leading to Erb palsy.[91,93,96] In patients with preexisting cervical spondylosis, flexion-extension injuries may cause acute intervertebral disc herniation, giving rise to upper extremity myelopathy and radiculopathy.[95]

The onset of neck pain following acute trauma may be immediate or delayed for 24 to 48 hours, or in some instances, for several days. The onset of pain may occur as an "ache" or "stiffness" in the neck that is localized to the cervical paraspinal muscles depending on the mechanics of injury. Beside the common muscle guarding, stiffness, and local tenderness, pain referral to sites distant from the original injury is common. Headache, dizziness, tinnitus, dysphagia, and visual disturbances may occur with involvement of the suboccipital muscles, the SCM, and the upper trapezius.[74,75,91,96] Following the initial injury, neck pain is generally accompanied by limitations in range of motion secondary to involvement of the paraspinal musculature from either direct damage to the muscles (rupture or tear) or by reflex response (splinting). Such injuries usually cause damage to both muscles and ligaments, and healing is often aggravated or delayed by sensory hyperactivity and abnormal function of the cervical spine.[73,100,101]

Patients with cervical strain injuries often present with subjective symptoms that are much greater than the objective signs. They often report a history of poor response to conventional therapeutic interventions and may present with psychologic and behavioral aspects common to chronic pain disorders. Consequently, these patients are often misclassified as hysterics or malingerers. Nevertheless, clinical and experimental evidence leaves little doubt that most cervical strain injuries can be explained on firm physiologic grounds. Evidence suggests that most patients with whiplash injuries recover within 2 months, but some may suffer from chronic cervical pain indefinitely.[94]

A history of trauma to the cervical spine is not always indicative of cervical strain. Other disorders that need to be considered in the differential diagnosis are degenerative osteoarthritis (OA), joint and ligamentous laxity, inflammatory diseases of muscle, vascular insufficiency, and neural compression syndromes. In addition, pathologic processes in other areas, such as the head, shoulders, or diaphragm, can cause pain that is referred to the neck. Such referred pain may be difficult to distinguish from primary cervical pathology.

Most minor cervical strains and spasms can be managed with rest, immobilization, anti-inflammatory drugs, and muscle relaxants until the patient is pain free and has regained full mobility of the cervical spine. Functional restoration may require an individualized and comprehensive physical therapy program, which may include short-term immobilization via a cervical collar.[102,103] Al-

though the cervical collar is the most frequently used orthotic, it is recommended that it be used only until the acute pain has subsided for fear of creating hypomobility. A recent study did not find any difference in pain and disability scores between patients who wore a soft cervical collar for 2 versus 10 days.[103] Tendons can be injured as well, and may slow down recovery and necessitate physical therapy with postural reeducation that includes unloading and gradual reloading.[101]

Cervical osteoarthritis (ICD-9 721.0)

Direct or indirect cumulative microtrauma to the weight-bearing joints of the cervical spine may lead to progressive degenerative arthritic changes. These changes may occur when the normally well-hydrated intervertebral discs lose their ability to withstand loading forces or are affected by abnormal postural factors.[104,105] There also may be degenerative changes to the vertebral body, adjacent uncinate processes (joints of Luschka), and posterior facet areas.[104] The cervical spine supports 10 to 15 pounds of weight through daily motions of the head. The most common load-bearing sites are in the area of C5-C6 and C6-C7, which are common regions of intervertebral disc disease.[106] Degenerative changes include inflammation of the joint linings with osteophyte formation, along with bony and cartilaginous exostoses. OA of the synovial joints is more usual in the more mobile upper cervical segments.[104]

OA is common in individuals aged 50 years and older, and may be associated with genetic predisposition. By the seventh decade of life, 75% of individuals display signs and symptoms of OA, and it is generally considered that 100% will develop it during the course of a normal lifetime.[105] As the elasticity of tissues decreases with age, there is a concomitant loss of range of motion, the neck becomes less resilient, and muscle strength declines. Early subjective complaints of OA include occasional episodes of neck pain triggered by activity, exertion, minor trauma, or weather changes, but postural factors should also be considered. Often these episodes will resolve in several days or a week with little more than rest. More advanced symptoms include stiffness, limitation of movements, crepitation, local pain, tenderness, and myalgia.

Early cervical OA may present without radiographic changes, while advanced cases frequently reveal radiographic evidence, such as alteration in curvature of the spine, loss of lordosis, narrowing of the intervertebral disc spaces, and anterior or posterior osteophytes.[69,106] Lateral neck movements, rotation, and extension are generally more limited than flexion. Progressive degeneration may lead to narrowing of the intervertebral spaces, and disc displacements may result in radiculopathy. Both sensory and motor roots may be involved, but sensory symptoms are more common and include pain, paresthesia, hypoesthesia, and hyperesthesia. With involvement of the C1-C3 nerve roots, pain may be referred to the head, neck, and shoulder girdle, and may be accompanied by suboccipital or occipital headaches, blurred vision, tinnitus, and dysphagia.[56,67-69] Involvement of the C4-T2 roots can cause interscapular, arm, and finger symptoms, all of which can be associated with painless or painful crepitation on active range of motion.[104,105,107] In advanced cervical OA, osteophytes and exostosis of neural foramina can lead to stenosis and nerve root compression, with subsequent cervical spondylotic myelopathy.

Mild and moderate stages of cervical OA will normally respond to comprehensive physical therapy management with or without medication. However, once osteoarthritic changes have reached the point of neural compression and radiculitis or myelopathy ensues, remission of symptoms is more difficult. Management at this stage may be accomplished by compliance with postural corrective guidelines, home use of a suboccipital traction device, and transcutaneous electrical nerve stimulation with or without medication.[108] Traction is preferably performed intermittently and in the supine position to avoid compressive forces on the temporomandibular joint (TMJ).[109]

Compression, irritation, or distortion of upper cervical roots by structural lesions (IHS 13.12)

Entrapment or impingement of cervical rootlets, roots, ganglia, and peripheral nerves can occur throughout the cervical spine. Entrapment of C1-C3 may cause ipsilateral or contralateral headaches, facial pain, and associated sensory deficits. Compression, irritation, or distortion of the C1 root may produce orbital, frontal, and vertex pain.[86,110] C1-mediated pain may arise from irritation of the atlantooccipital joint, which may result in occipitofrontal headache.[86] C1-mediated pain may also be caused by vertebral dissection of the C1 horizontal segment, entrapment from posterior fossa tumors, or compression of the vertebral artery.[110,111]

The C2 root exits between the atlas and axis, and its peripheral distribution forms the greater occipital nerve, supplying sensation to the scalp from the occiput to the vertex. Disorders that affect the C2 dorsal rootlets, root, ganglion, or the peripheral branch may cause neuralgic pain, numbness, and dysesthesia, accompanied by a sensory deficit. The ventral ramus of the C2 root has meningeal branches to the hypoglossal and vagus nerves, which may account for throat and gastrointestinal symptoms.[112,113] Dull pain, primarily in the suboccipital, occipital, and frontal regions, may be due to lesions in anatomic structures innervated by peripheral branches of C2.[86,114] C3 and its distribution may refer pain in the preauricular region, with associated sensory deficits. Irritation or compression of the root of C3 may cause referred pain to the pinna, the angle of the jaw, the TMJ, and the retroorbital area.[86,114] Structures innervated by C3, such as the zygoapophyseal joints of C2-C3, may cause pain in the occipital region.[80,114] Space-occupying processes such as a tumor, vascular lesions (eg, arteriovenous malformation or aneurysm), or bony changes causing lesions of C2-C3 can also produce cephalic pain.

Cervical entrapment disorders should be referred to the proper health care professional for evaluation and management. If space-occupying lesions or malignant processes have been ruled out as the source of the pain, comprehensive noninvasive treatment may include definitive manual physical therapy, with education and instructions in proper postural corrective techniques and therapeutic exercises.

Cervicogenic headache (IHS 11.2.1; ICD-9 *723.2 or* ICD-9 *784.0)*

Cervicogenic headache is defined as "referred pain perceived in any part of the head caused by a primary nociceptive source in the musculoskeletal tissues innervated by cervical nerves."[115-117] While there may be no single entity called *cervicogenic headache* it is reasonable to refer to headaches that originate in

the cervical region as *cervicogenic* or *cervically mediated*, or as a *cervical-cranial syndrome*.[118]

Cervicogenic headache is characterized by a moderately severe, dull, dragging, unilateral headache without side-shift. The pain is provoked or aggravated by neck movements. The headache may last from 1 to 3 days and may be accompanied by myriad symptoms, including lacrimation, conjunctival hyperemia, dizziness, nausea, vomiting, and sensitivity to light and noise.[117] The IHS diagnostic criteria state that clinical signs need to imply that the source of the pain is a cervical structure and that the headache is abolished by diagnostic blockade of the source or its innervation.[119]

There are numerous structures in the region of the cervical spine that are pain sensitive and refer pain to the head. Craniofacial pain is mediated by the first three cervical nerves, the C1-C3 sinuvertebral nerves, and cranial nerves V, VII, IX, X, XI, and XII. Additional innervation is provided by sympathetic afferents that course with the first two thoracic nerves, synapsing in the trigeminal nucleus, as well as parasympathetic afferents traveling with cranial nerves VII and IX.[20,78] Irritating forces on these nerves at sites of neural compression can mediate craniofacial referral. The density of the suboccipital musculature along with the SCM and upper trapezius are prime sources of cervicogenic headache of myofascial origin. The myriad neural pathways that course through, connect with, or originate from the C1-C3 levels provide fertile ground for the development of discomfort in the suboccipital fossa, with referral to the occiput and vertex of the head. Anastomosis of the occipital to the supraorbital nerve, which is a trigeminal branch, provides a distinct neural pathway for headache in this region.[78] In addition, the myofascial connection between the occipitalis and frontalis muscles represents another mechanism behind the common occipital-frontal headache.[85] A decrease in the suboccipital space by occipitoatlantal approximation due to a forward head posture, which may be compounded by posterior cranial rotation, presents a mechanical mechanism for compression or irritation of the musculature, vasculature, and neural innervating components that compose the entire trigeminocervical complex.

Pain referred from the cervical muscles can be similar to that of a vascular or tension-type headache.[89,90,120–122] Referral of pain to the eye is quite common in headache of cervicogenic origin.[70] Pain can also be perceived in more than one area of the head and face.[85] A prime example is referral to the occipital, temporoparietal, and lateral orbital regions caused by hyperactivity of the upper trapezius, which in addition refers pain to the angle of the mandible in the masseteric region.[85]

The comorbidity of cervical and craniofacial pain is commonly seen by physical therapists, and this relationship has been well documented.[6,9–12,48,123–125] A definitive physical therapy evaluation of the upper quarter and temporomandibular complex is required to delineate the origin of headache as stemming from the cervical as opposed to the craniofacial region.

Occipital neuralgia (IHS 13.8)

Occipital neuralgia is characterized by paroxysms of jabbing pain in the distribution of the greater or lesser occipital nerves, with the occasional persistence of aching between attacks. There may be a reduction of sensation or dysesthesia in the area, and the affected nerve is tender on palpation.

The pathogenesis is not always clear but may be related to trauma of the nerve. The differential diagnosis includes occipital referral of pain from the atlantoaxial or upper zygapophyseal joints, tender trigger points in neck muscles, and neoplasia or other lesions affecting the spine or occiput.[126]

A decrease in the suboccipital space by occipital-atlantal approximation due to a forward head posture, which may be compounded by posterior cranial rotation, presents a mechanical mechanism for compression or irritation of the musclulature, vasculature, and neural innervating components that comprise the entire trigeminocervial complex. Manual or intermittant mechanical suboccipital traction or injection of local anesthetics and corticosteroids may provide temporary and even longlasting relief.[126]

Dural headache (ICD-9 *784.0)*

The pain quality of dural headache is achy and can exist bilaterally or on alternate sides.[127] Dural pain is commonly triggered by abnormal movements, postures, or positions. Dural tension testing, such as in the form of the slump or the long-sitting test may recreate or increase the pain. It is therefore imperative that patients be asked about their sitting, working, and sleep postures, which may create tension on the spinal dura that emanates to the cranial region.[56,109] Posterior disc bulging or herniation at the C2-C3 or C3-C4 levels can also cause the outer annular fibers to press against the posterior longitudinal ligament and thus put pressure on the anterior dura, giving rise to craniofacial pain.[127-129] The anatomic relationship between the rectus capitis posterior minor in the suboccipital fossa and its attachment to the dura mater may also represent a source of dural headache.[130-134] Chemical stimulation of the intracranial dura has demonstrated that all dura-sensitive neurons have cutaneous receptive fields, including one or more trigeminal divisions, the most common being the ophthalmic branch.[130-134] This evidence has implications for the origin of dural as well as vascular headache and the related eye pain that often accompanies these entities.

Eagle syndrome

Eagle syndrome, which consists of either elongation or calcification of the stylohyoid ligament, has a variable presentation that may include sore throat, dysphagia, otalgia, glossodynia, headache, or vague orofacial pain, predominantly along the neck.[135,136] The patient may report pain on swallowing, yawning, or turning of the head. The pain is usually unilateral and has a neuralgic quality that may mimic glossopharyngeal neuralgia. Examination should include palpation of the stylohyoid area and tonsillar fossa, and provocation tests such as turning the head in an ipsi- as well as contralateral direction. Carotidynia is often confused with Eagle syndrome because of the close approximation of the styloid process and carotid artery. Spasm of the carotid artery can occur as a result of a traumatic hyperextension injury or contact irritation from an elongated styloid process. The SCM also courses in the same vicinity, and carotidymia or styloid compression may therefore excite the SCM and create a secondary occipital, frontal, or orbital headache. Radiographic examination will reveal the elongated/calcified stylohyoid process.[137] Treatment may consist of pharmacologic management with analgesics or anti-inflammatory agents and/or surgical excision via either an intraoral or extraoral approach.[135,136,138]

Torticollis (ICD-9 723.5)

Torticollis, also known as *wry* or *stiff neck*, can occur from congenital absence of one SCM; musculoskeletal trauma; metabolic, infectious, or neurologic factors; and emotional triggers.[87,88,139] The resultant muscular rigidity, adversely affecting ipsilateral sidebending and contralateral rotation of the head, is caused by hyperactivity of the SCM and/or the upper trapezius, both of which cause myotomal referral to the craniofacial region. Bilateral SCM involvement will cause posterior cranial rotation.[140] The pain distribution from SCM referral depends in part on involvement of one or both heads of this muscle and can include the occiput, ear, forehead, and orbital region. Referral to the eye involves the superior, lateral, and inferior orbit, which may mimic migraine.[85] Frontal pain can be unilateral on the side of the involved SCM, contralateral, or bilateral due to the contralateral referral pattern of the SCM. The levator scapulae may also be involved, further contributing to the ipsilateral sidebending. Since these muscles also control positioning of the head, dizziness and visual disturbances may become evident. Congenital absence of one SCM is rare, but acute torticollis after a throat or glandular infection is seen in childhood. A cumulative involvement can occur in the adolescent stage by a slowly increasing C2-C3 or C3-C4 posterolateral intervertebral nuclear migration.[69,122]

The idiopathic spasmodic variety accounts for only 5% to 10% of all cases and usually has a familial neurologic origin that may cause concomitant facial dystonia.[69] Postencephalitis produces a paroxysmal twisting movement of the head, while a spastic scenario presents a fixed position of ipsilateral sidebending and contralateral rotation of the head. A fixed forward head posture with posterior cranial rotation can also occur due to bilateral involvement of the SCMs, giving rise to antecollis with concomitant dysphagia and vocal disturbance.[87] It is important to understand that the addition of posterior cranial rotation causes increased approximation of the occiput on the atlas/axis, therefore maximizing suboccipital compression. Torticollis may also be caused by hypothyroidism, alcoholism, and emotional stress disorders. Physical therapy intervention in the form of manual soft tissue release techniques, postural correction, and EMG biofeedback have demonstrated effectiveness in the management of torticollis.[141,142] Botox injections can be beneficial for recalcitrant torticollis or headache associated with torticollis.[143]

Neck-tongue syndrome (IHS 13.9)

Neck-tongue syndrome is a rare disorder characterized by infrequent attacks of unilateral pain in the upper neck that last from 15 seconds to several minutes. The pain radiates toward the ear, with simultaneous numbness, paresthesia, or the sensation of involuntary movement involving the ipsilateral half of the tongue. A sudden rotational movement of the head will elicit the attack contralateral to the side of rotation.[144] Additional findings have been described, including upper extremity tingling or pain (usually on the same side), sensory changes of the oropharynx with subsequent dysphagia, and a sensation of choking.[145]

The proposed mechanism is compromise of the C2 ventral ramus by subluxation of the lateral atlantoaxial joint, which produces occipital pain. Numbness of the tongue arises because afferent fibers from the tongue pass from the ansa hypoglossi into the C2

ventral ramus.[114] Most individuals with this syndrome have significant pathology of the atlantoaxial joint, and it has also occurred in patients with rheumatoid arthritis or congenital joint laxity. Hypomobility in the contralateral atlantoaxial joint may be a predisposing factor.[144]

In the absence of pathologic findings the disorder appears benign, and conservative management with cervical collars,[146] manipulation,[147] analgesics, antiepileptic drugs, antidepressants,[146] steroids, muscle relaxants, and injections of local anesthetics may be effective.

Vertebral artery syndrome (ICD-9 435.1)

Vertebral artery involvement can also contribute to various cervicogenic headaches. Conditions with similar symptoms include vertebral artery compression syndrome (*ICD-9* 721.1), vertebral basilar syndrome (*ICD-9* 435.3), and benign positional vertigo (*ICD-9* 386.11). The major extracranial region of the vertebral artery is protected by the vertebral canal and the surrounding soft tissue structures, but the area from C2 to the foramen magnum is vulnerable. Vertebral artery injury in the suboccipital region can result from severe cervical spine trauma, such as a fracture of the atlas, whereas vertebral artery compression may be associated with Barré-Liéou syndrome.[148] Red flags, such as disorientation, nausea, vomiting, visual disturbances, dizziness, or vertigo that occur with change from a non–weight-bearing (supine) to a weight-bearing (sitting or standing) position, should immediately necessitate further testing to rule out vertebral artery involvement.[148-151] An experienced physician or physical therapist should perform the assessment, which may be curtailed in favor of neurologic and radiologic testing if any of the above red flags recur or intensify during the evaluation.

Active cervical spine range of motion and provocation testing in positions of rotation, sidebending, and extension can be used to stretch, narrow, or kink the ipsilateral and/or contralateral vertebral artery, but this testing must be performed with great caution, and the reliability is suspect unless performed by an experienced clinician.[148-151] The delineation between labyrinthine and vertebral artery involvement should also be considered. The trained clinician can auscultate the carotid and subclavian arteries for bruits with the patient in a seated position and the head in a neutral position, but further testing in positions of cervical rotation and extension to each side should be designated to others.[148]

Barré-Liéou syndrome (ICD-9 723.2)

Irritation of the vertebral artery or posterior cervical sympathetic network via stretching or compression forces can give rise to Barré-Liéou syndrome, also known as *posterior cervical sympathetic syndrome*.[152] This rare syndrome, characterized by intracranial vasoconstriction, may cause widespread facial and cranial symptoms that can mimic migraine, tension-type headache, sinusitis, and craniofacial dysautonomia, due to involvement of the trigeminal spinal tract, upper cervical roots, posterior sympathetic fibers, and the vertebral artery. Head and neck pain that falls into the Barré-Liéou category is usually continuous but variable, with qualitative characteristics that consist of throbbing, burning, stinging, or pinching.[152-154] Tinnitus, decreased auditory perception, a feeling of dust in the eye, blurred vision,

tearing, nasal irritation, and hoarseness may also be present. One or more of these symptoms in addition to pain may become evident or exacerbated by active range of motion and positional or manual suboccipital testing. It is imperative that any positive findings be further evaluated by a definitive neurologic examination. This syndrome is very controversial, as it may simply fall into the category of a vertebral artery syndrome associated with other compressive forces within the suboccipital fossa.[154]

Arnold-Chiari syndrome (ICD-9 348.4)

Herniation of the cerebellar tonsils into the foramen magnum of 3 to 5 mm or distally to the C2 level represents a structural malformation of the brainstem and dura known as *Arnold-Chiari syndrome*.[155] This syndrome is commonly delineated by the type of abnormality and may include hydrocephalus, myelomeningocele, syringomyelia, spinal cord cavitations (syrinx), as well as the components of the posterior fossa. Headaches, hemifacial spasm, coughing with or without symptoms of sleep apnea, inability to speak, dysphagia, and nystagmus may be associated symptoms.[155-160] An Arnold-Chiari malformation causes traction or compression of one or more cranial nerves and is confirmed with a definitive neurologic assessment as well as MRI evaluation. Surgical decompression is often required.

Summary

The composite of neural elements that converge at the trigeminocervical complex can cause, mimic, or contribute to TMDs, orofacial pain, or headaches as well as associated gastrointestinal, aural, laryngeal, pharyngeal, and equilibrium disturbances. Determination of the level and structures that give rise to the perceived nociception and associated symptoms requires a comprehensive upper-quarter physical therapy evaluation.[56] It is imperative for the clinician to understand that disorders of the suboccipital spine refer pain proximally and are often described as headache variants. Associated sensory deficits are common in these disorders and help establish the proper diagnosis.

Current evidence has shown that specific and individualized intervention consisting of early and comprehensive posture correction, ergonomic adaptation, therapeutic exercise, and manual therapy by experienced physical therapists can assist the clinician in effectively managing cervicogenic factors to reduce pain, restore function, and prevent recurrence.[100,109,161-173]

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