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Cervicogenic Headache: Diagnostic and Treatment Strategies

ERIC HELM, MD
Assistant Professor, Department of Physical Medicine and Rehabilitation
University of Pittsburgh School of Medicine

ROBERT DONLAN, DO
Resident, Department of Physical Medicine and Rehabilitation
University of Pittsburgh School of Medicine

Clinical Vignette

SK is a 40-year-old male who presents to clinic for post-traumatic headaches. The inciting event was a rear-end motor vehicle collision that resulted in a whiplash movement on impact. SK did not experience a loss of consciousness. His symptoms are right-sided posterior scalp pain and headaches. The headaches start in the right frontal region and radiate to the posterior and lateral scalp, and worsen as the day progresses. There is no aura associated with the headaches. Provocative maneuvers are cardiovascular exercise and computer work. He also endorses mild cognitive fatigue.

He completed an outpatient physical therapy program and recently started taking venlafaxine. He continues to work as a computer engineer, but is no longer able to function as the chief executive officer of his company due to the headaches.

SK wants to return to his high-level job responsibilities and continue physical activity. He is worried that he is becoming a burden on the company. He heard the term "cervicogenic headache" from his daughter, who is studying to be a physical therapist. He is wondering if this condition applies to him and if so, which treatment options could be pursed in order to return to his previous level of intellectual, cognitive, and athletic function.

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Disclosures
Doctors Helm, Donlan, and Munin have reported no relationships with proprietary entities producing health care goods or services.

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Background

Headaches are a common complaint and source of diminished quality of life for many people. Approximately 77% of adults report a headache episode over a one-year time period.[1] Multiple types and causes of headache have been identified, but clinical management remains a significant challenge due to a large overlap in signs and symptoms. The International Headache Society (IHS) has developed a classification system to assist physicians in more accurately diagnosing headaches. There are two broad categories: primary and secondary. Primary headaches are those without a known underlying cause, and include: migraines, tension-type, and cluster headaches. Secondary headaches are those that can be attributed to underlying disease or dysfunction, such as tumors, infections, and structural abnormalities (e.g., temporomandibular arthritis). Cervicogenic headache is a type of secondary headache, defined by the IHS as pain referred to the head and/or face from a bony or soft tissue source in the neck.[2] Estimates reveal that 4.1% of headaches in the general population have a cervicogenic origin,[3] and this type more significantly impairs everyday physical functioning (walking, shopping, cleaning, etc.) when compared to migraine and tension-type headaches.[4]

TABLE 1:
Criteria for Cervicogenic Headache by the International Headache Society[2]

Criteria	Description
A	Pain, referred from a source in the neck and perceived in one or more regions of the head and/or face, fulfilling criteria C and D
B	Clinical, laboratory, and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck known to be or generally accepted as a valid cause of headache
C	Evidence that the pain can be attributed to the neck disorder or lesion based on at least one of the following: • Demonstrations of clinical signs that implicate a source of pain in the neck • Abolition of headache after diagnostic blockade of a cervical structure or its nerve supply
D	Pain resolves within three months after successful treatment of the causative disorder or lesion

The distinction between cervicogenic headache and migraine headache is difficult but important, because they share many similar features. For example, both migraine and cervicogenic headache present with ipsilateral head pain that can radiate to the fronto-orbital region, and both can present with nausea, photophobia, phonophobia, and neck pain to varying degrees.[2,5,6] The distinction becomes easier when migraine headaches present with an aura, which is a set of reversible, transient, and focal neurologic symptoms that occur at the beginning of the migraine headache.[2] Such focal neurologic symptoms may include visual changes, paresthesias, dysphasic speech changes, and less commonly, weakness. Such symptoms are not characteristic of cervicogenic headache.[2]

Pathophysiology

Various structures and injuries can be implicated, including the upper cervical zygapophysial joints, the atlanto-axial joint (AA joint), the suboccipital muscles, and the intervertebral discs. The C2-C3 zygapophysial joint is considered the most common pain generator, followed by the atlanto-axial joint.[7] The mechanism by which these structures refer pain to the head and face is a complex interplay among various neural pathways.

Trigeminocervical Nucleus Convergence Theory

The atlanto-axial joint (C1-C2) and the upper cervical zygapophysial joints (C2-C3, C3-C4) receive sensory innervation via the first three cervical spinal nerves.

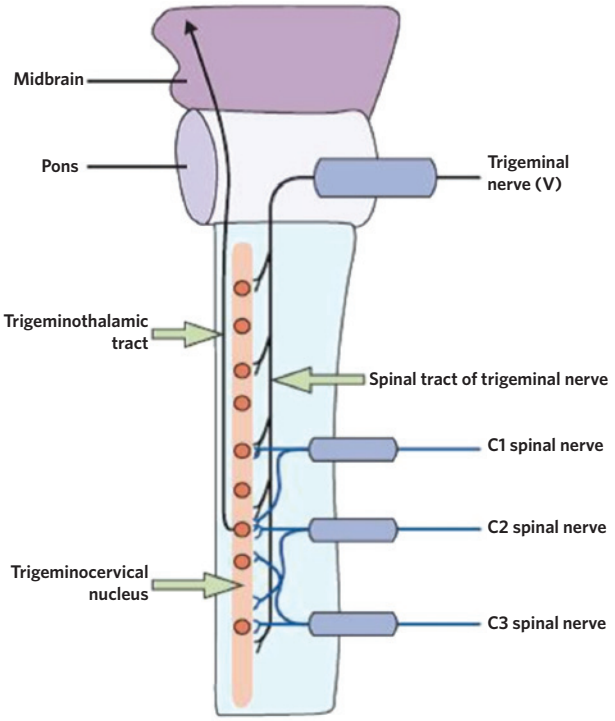

FIGURE 1: **Mechanism of pain referral from the cervical spine to the head.**[8] *Reproduced with permission from Elsevier.*

In particular, the AA joint is innervated by all three spinal nerves (recurrent meningeal branches of C1, C2, C3, and the ventral ramus of C2). The C2-C3 zygapophysial joint is innervated by the superficial medial branch of the C3 dorsal primary ramus, also known as the third occipital nerve (TON). The C3-C4 zygapophysial joint is innervated by the medial branches of the C3 and C4 dorsal rami.[7] A summary is provided in Table 1.

The sensory afferents from the C1-C3 spinal nerves ultimately synapse in the trigeminocervical nucleus in the upper cervical spinal cord. This sensory nucleus extends cephalad along the length of the brainstem and receives input from the trigeminal nerve via caudal projections (see Figure 1). The convergence of cervical spinal afferents and trigeminal afferents in this nucleus allows for referral of pain generated in the neck to the head, most notably the fronto-parietal, orbital, and occipital areas.[8]

Craniocervical Junction

The craniocervical junction comprises the area of transition between the skull and cervical spine and encompasses two major joints: the occipito-atlantal (OA) joint and the atlanto-axial (AA) joint. Flexion and extension occur primarily at the OA joint, which derives its stability from bony configuration. Axial rotation occurs primarily at the AA joint, which derives stability from a strong complex of ligaments, namely the transverse and alar ligaments (see Figure 2). The transverse ligament prevents anterior subluxation of the odontoid process on the body of C1. The alar ligament limits axial rotation and lateral bending, and is often injured by the anterior shear forces created by whiplash events. As mentioned before, the AA joint receives innervation from the C1-C3 spinal nerves, so the resulting hypermobility/instability at the joint can cause neck pain and headache via the trigeminocervical nucleus. In severe cases, vertebral artery and spinal accessory nerve injury also can occur as they traverse the foramen magnum.[13]

Despite being controversial, whiplash-associated disorder (WAD) has frequently been associated with cervicogenic headaches.[9] During whiplash injuries, large anterior shear forces are generated across the upper cervical spine,

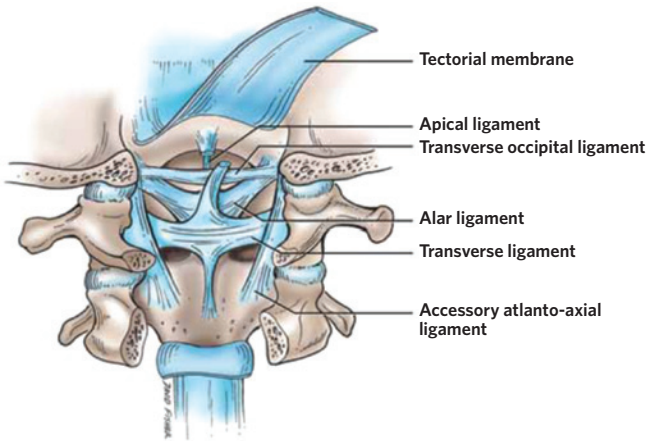

FIGURE 2: **Ligaments of the craniocervical junction.**[13] *Reproduced with permission from the Journal of Neurosurgery Publishing Group (JNSPG).*

stressing the transverse and alar ligaments.[10] However, some studies have also shown whiplash to generate compression and distraction forces exceeding the physiologic limits of the cervical zygapophysial joints (the same joints implicated in cervicogenic headache).[11] Furthermore, investigations have found cervical zygapophysial joint pathology to be implicated in at least 50% of whiplash-associated pain.[12]

WAD can also involve the muscles of the craniocervical junction. The deep suboccipital muscle group comprises four muscles: the rectus capitis posterior major and minor, and the oblique capitis superioris and inferioris. These muscles add to craniocervical junction stability primarily through postural control, although they can produce small extension and rotation movements at the OA and AA joints, respectively. Patients with WAD have been shown to have increased fatty infiltration of these muscles compared to healthy controls.[14]

Diagnosis

Cervicogenic headache is defined as headaches and neck pain referred from a source in the upper cervical spine.

TABLE 2:

Potential Headache and Neck Pain Generators[7]

Pain Generator	Innervation
Atlanto-occipital joint	Ventral ramus of C1
Atlanto-axial (AA) joint	Medial AA joint: Recurrent meningeal branch (ventral ramus) of C1, C2, C3 Lateral AA joint: Ventral ramus of C2
C2-C3 zygapophysial joint	Third occipital nerve (superficial medial branch of C3 dorsal primary ramus)
C3-C4 zygapophysial joint	C3 and C4 medial branch of dorsal primary ramus
C2-C3 disc	Sinuvertebral branch of superficial medial branch of C3 dorsal ramus

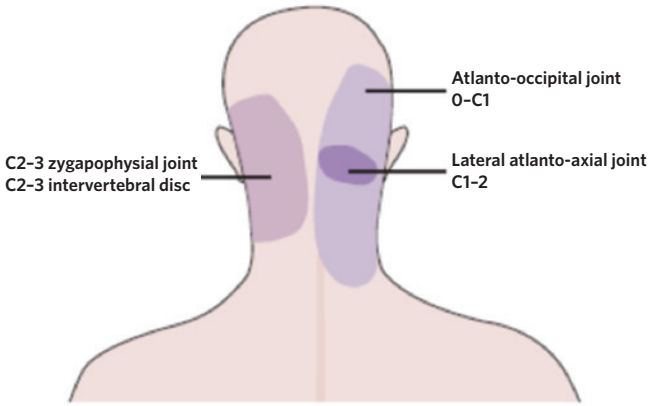

FIGURE 3: **Referral patterns from upper cervical spine structures that are implicated in neck pain and headaches.**[8] *Reproduced with permission from Elsevier.*

Our outpatient clinics evaluate a large subset of patients that have suffered some etiology of head trauma. These patients may be suffering from a constellation of disorders, including concussion, cervical spine disorders, or myofascial pain syndrome. However, when patients endorse symptoms of both headaches and neck pain concurrently, the diagnosis of cervicogenic headache must be considered.

There is controversy regarding the diagnosis of cervicogenic headache. Various medical societies have published diagnostic criteria, including the IHS (see Table 1 on Page 2). There is significant debate across multiple medical disciplines concerning which criteria should be accepted. Controversy also exists in establishing the "true" pain generator in cervicogenic headache. Multiple generators that reside in the upper cervical spine (see Table 2) and exhibit a general pattern of pain referral to the head and neck (see Figure 3) have been implicated.

The C2-C3 zygapophysial joint is the most implicated pain generator, and the atlanto-axial joint is the second most commonly implicated. These joints and the third occipital nerve are often targeted with interventional spine procedures. Current literature supports the role for diagnostic medial branch blocks and not intra-articular zygapophysial joint injections as the criterion for diagnosis

of pain emanating from or mediated by the cervical zygapophysial joints.[20]

With a wide overlap of surrounding neurovascular structures, cervicogenic headache needs to be distinguished from other recognized medical disorders (see Table 3).

Cervicogenic headache is a clinical diagnosis. In our clinic, the primary symptom is pain that starts in the neck and radiates to the fronto-temporal region, and is exacerbated with certain provocative neck maneuvers, including the supine cervical facet glide examination and lateral shear test. Typically, when performing the supine cervical facet glide examination, the elicitation of headaches and scalp pain usually occurs during palpation of the upper cervical zygapophysial joints (ex. C1-C2 and C2-C3).[15] In the absence of any cervical spine injury or instability, the remainder of the physical examination should be normal. We also evaluate patients for instability at the occipito-atlantal (OA) and the atlanto-axial (AA) joint with the Sharp-Purser and lateral shear tests. To view a slide presentation featuring demonstrations and descriptions of each test, please visit this issue of *Rehab Grand Rounds* at **UPMCPhysicianResources.com/RGRFall14**. Radiographically, we obtain anterior-to-posterior open-mouth films with the head in neutral and right/left lateral flexion positions to evaluate the alar ligaments (see Figure 4). If instability exists, we would typically see a lateral shift of C1 on C2. Our clinic considers a shift of 2 mm or more indicative of instability. If more specific diagnostic imaging is required to exclude other diagnoses, MRI of the brain or cervical spine should be obtained.

TABLE 3:

Differential Diagnosis of Headache and Neck Pain

Cervicogenic headache
Concussion
Migraine headache with or without aura
Tension-type headache
Paroxysmal hemicranias
Space occupying lesion (e.g., brain tumor, intracranial hemorrhage)
Chiari malformation
Cerebral venous thrombosis
Vertebrobasilar insufficiency
Temporal arteritis
Viral meningitis
Psychological syndromes

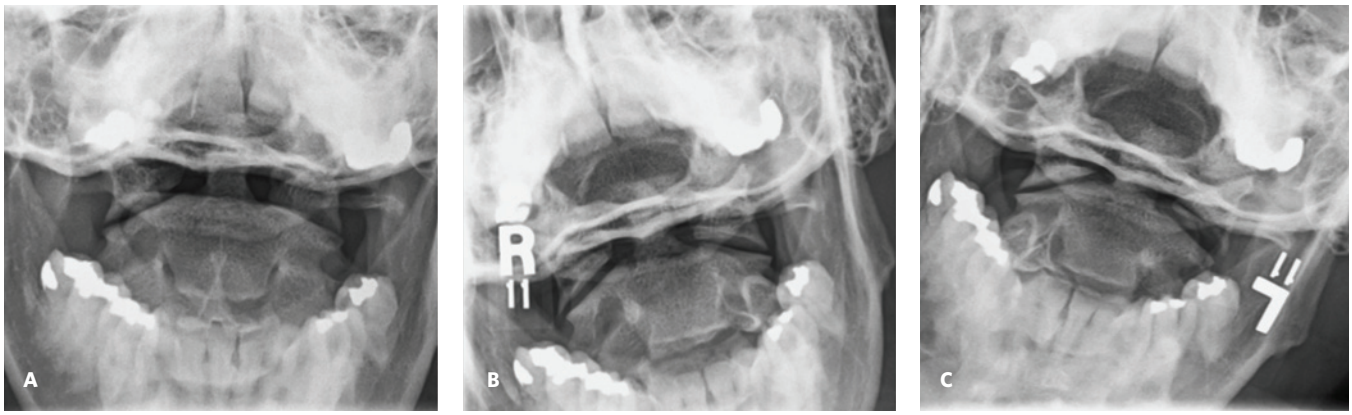

FIGURE 4: **Normal open-mouth AP radiographs with the head in (a) neutral, (b) right lateral flexion, and (c) left lateral flexion. The lateral borders of C1 and C2 remain congruent with one another during both side-bending movements. A lateral shift of C1 on C2 is concerning for instability.**

Treatment

Physical Therapy

Joint mobilization techniques have been effective in reducing both headache and neck pain frequency. Mobilization is a manual therapy technique that applies passive movements at varying speeds and amplitudes in order to reduce tension and restore movement. Certain manual therapy protocols have been utilized with success in hypo-mobility of the cervical and thoracic spine. The most advocated techniques include mobilization of the craniocervical junction, occipital traction, and occipital glide mobilization. There is not an established precise estimate of the dose-response relationship in patients with cervicogenic headache.[16] Emphasis should be on postural exercises focusing on cervicoscapular stabilizers, scapular mechanics, and upper body ergometry.[17] We typically include our vestibular therapists in treatment for this reason.

Dynamic strengthening exercises are incorporated later in the plan of care.[18] Some of these exercises include: latissimus dorsi pull-downs, upright rows, open and closed-chain scapular punches, and prone strengthening of middle and lower trapezius muscles. Cervical stabilization and dynamic proprioceptive protocols have shown more sustained benefit than mobilization techniques.[19] There is limited data to support trunk stabilization protocols. Myofascial release techniques can be applied to the suboccipital muscle group (e.g., rectus capitus posterior major and minor, superior and inferior oblique) throughout the treatment course. No definite functional benefit has been reported with this approach.

Medication

Pharmacologic treatment includes antidepressants, membrane stabilizers, and analgesics. No substantial efficacy from clinical trials exists for their use in cervicogenic headache. There is limited evidence for efficacy in primary headache disorders, such as migraine and tension-type headaches. Antidepressants, such as venlafaxine, are commonly used in primary headache disorder prophylaxis, but have limited clinical data efficacy.

Interventional

Procedures targeting the third occipital nerve, upper cervical medial branches, and C1-C2 and C2-C3 zygapophysial joints are common treatments. The third occipital nerve is the most targeted structure in our clinic. The third occipital nerve is thicker than the corresponding cervical medial branches and is usually embedded in the periscapular fascia of the C2-C3 zygapophysial joint. It can be visualized 96% of the time and blocked accurately in 82% of cases under ultrasound guidance.[21] In our opinion, the most accurate third occipital nerve block technique uses both ultrasound and fluoroscopic guidance. We perform two diagnostic third occipital nerve blocks with 0.25% preservative-free bupivacaine to determine if the third occipital nerve is the pain generator, because the patient should have greater than 50% reduction in their typical headache and neck pain. This benefit should last for the duration of the anesthetic used. If two diagnostic injections to the third occipital nerve are effective, radiofrequency neurotomy is recommended for long-lasting treatment. To see a video demonstration of this technique, please visit this issue of *Rehab Grand Rounds* at **UPMCPhysicianResources.com/RGRFall14**.

Percutaneous radiofrequency neurotomy destroys the somatic afferent nerve supply of this joint. There is good evidence that third occipital nerve radiofrequency neurotomy is effective in 88% of patients that have confirmatory-controlled diagnostic blocks, with a 297-day median duration of relief.[20] C1-C2 and C2-C3 zygapophysial joint injections have been described as effective treatments, but there is limited evidence to support their use in this patient population.[22,23]

Clinical Outcome

SK was diagnosed with cervicogenic headache. We obtained anterior-to-posterior open-mouth and lateral bending radiographs to evaluate for craniocervical instability. After ruling out any instability, he underwent a more comprehensive physical therapy program, with emphasis on upper cervical zygapophysial joint mobilization, cervical stabilization exercises, and dynamic proprioceptive training.

His symptoms were recalcitrant to the physical therapy program. A cervical spine MRI was obtained that did not show any gross abnormalities. We performed two diagnostic right third occipital nerve blocks under ultrasound and fluoroscopic guidance. He demonstrated complete resolution of the scalp pain and headaches for the duration of the anesthetic. The decision was made to pursue radiofrequency neurotomy to the right third occipital nerve. When seen at six-month follow-up, he was symptom-free of the posterior scalp pain and headaches. We discussed continuation of the cervical and thoracic stabilization protocol at home. When asked regarding return to high-level work, we allowed him to increase responsibility with computer projects, but emphasized frequent rest breaks to avoid prolonged static positioning.

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ADDRESS CORRESPONDENCE TO:

Michael C. Munin, MD
Senior Editor and Vice Chairman
Clinical Program Development
Department of Physical Medicine and Rehabilitation

Kaufmann Medical Bldg.
Suite 201
3471 Fifth Ave.
Pittsburgh, PA 15213
T: 412-648-6848
F: 412-692-4410
Email: muninmc@upmc.edu

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