Review

Cervicogenic Headache: Diagnostic and Management Approach and Challenges for a Complex Headache Disorder

Tara Sharma *

Posted Date: 30 December 2025

doi: 10.20944/preprints202512.2572.v1

Keywords: cervicogenic headache; migraine; trigeminocervical complex; occipital neuralgia; myofascial pain; cervical facet joint

 doi:10.20944/preprints202512.2572.v1

Review

Cervicogenic Headache: Diagnostic and Management Approach and Challenges for a Complex Headache Disorder

Tara L Sharma

Department of Neurology, University of Washington, Seattle, WA; tlsharma@uw.edu

Abstract

Clinical presentation of headache can be a diagnostic challenge as characteristics and localization of pain allows for multiple headache types. For instance, migraineurs can also have co-existing cervicogenic headache. Characteristics that indicate a cervical cause of headache include referred pain to the head from upper cervical structures, minimal relief from migraine specific therapies, and reduced range of motion (ROM) during the cervical flexion rotation (CFR) test. Diagnostic injection of cervical structures can help determine the source of nociceptive input. Studies have shown that radiofrequency neurotomy targeted at the upper cervical structures can be helpful in treating cervical facet causes of headache. Myofascial causes can be managed through neck physical therapy, alternative therapies, and trigger point injections. Overall, effective diagnosis and management requires knowledge of the anatomy and pain referral patterns of cervical structures. This understanding will assist medical practitioners in knowing when to suspect cervicogenic headache, to apply proper treatments.

Keywords: cervicogenic headache; migraine; trigeminocervical complex; occipital neuralgia; myofascial pain; cervical facet joint

Introduction

Headache is one of the most prevalent neurological disorders, significantly impacting the quality of life and productivity of those afflicted [1]. Of individuals with chronic headache, prevalence of cervicogenic headaches ranges from 15-20% [1,2]. Overlapping symptomatology between migraine and cervicogenic headache leads to misdiagnosis in 50% of cases, leading to inappropriate treatment [3–6]. For instance, presenting symptoms of cervicogenic headaches such as occipital pain, cervicalgia, and neck tension can also be seen in those with migraine and tension type headache [7,8]. Pain may also move, radiate, or spread to different locations making it difficult to determine its exact distribution. Diagnosis and treatment may be further complicated as there are many structures with nociceptive innervations in the cervical spine that can potentially precipitate headache [8]. These include the atlanto-occipital joint (AO), atlanto-axial joints (AA), C2-C3 intervertebral disk, C2-C3 zygapophyseal joints, posterior neck and paravertebral muscles, and upper cervical nerve roots [9,10]. Currently, the third edition of International Classification of Headache Disorders (ICHD-3) has developed criterion for cervicogenic headache, which is outlined in Table 1 [3,5,11]. This criterion has been revised over the years to encompass clinical diagnosis and objective testing (i.e. diagnostic procedures) [5,11,12]. Despite this, diagnosis of cervicogenic headache remains a challenge given intersecting features and co-occurrence with other headache types. The aim of this review is to discuss the pathophysiology of occipital neuralgia, common causes of cervicogenic headaches (i.e. cervical facet joint pain, radiculopathy, and myofascial pain), and the trigeminocervical complex. Clinical findings and diagnostic procedures that may indicate a cervical cause to headache are examined. Finally, based on evidence from the literature, management strategies for different cervical

 doi:10.20944/preprints202512.2572.v1

causes to headache are suggested and clinical exam findings that may potentially differentiate between migraine and cervicogenic headache are considered.

Table 1. ICHD3 criteria for cervicogenic headache.

Cervicogenic Headache
A. Any headache fulfilling criterion C
B. Clinical and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck, known to be able to cause headache
C. Evidence of causation demonstrated by at least 2 of the following:
1. Headache has developed in temporal relation to the onset of cervical spine disorder or appearance of the lesion
2. Headache has significantly improved or resolved in parallel with improvement in or resolution of the cervical disorder or lesion
3. Cervical range of motion is reduced and headache is made significantly worse by provocative maneuvers
4. Headache is abolished following diagnostic blockade of a cervical structure or its nerve supply.
D. Not better accounted for by another ICHD-3 diagnosis

Clinical Features, Diagnosis and Management of Occipital Neuralgia and Other Causes of Cervicogenic Headache

Occipital Neuralgia

Clinical Features/Pathophysiology

The ICHD-3 criteria for occipital neuralgia is presented in Table 2. Occipital neuralgia can be secondary to direct trauma to the occipital nerve, cervical dysfunction, or a primary headache disorder. Briefly, it is defined as unilateral or bilateral paroxysmal shooting or stabbing pain in the posterior scalp in the distribution of the greater, lesser or third occipital nerve [10]. There is usually tenderness, sometimes accompanied by diminished sensation or dysesthesia over the involved nerves. Tapping of the greater or lesser occipital nerves can also lead to shooting pain in the distribution of the nerve knows as tinel's sign. The greater occipital nerve (GON) arises from the dorsal primary ramus of C2 with some contribution from C3 [25]. This nerve courses through the semispinalis capitis and splenius muscle, terminating near the nuchal line through the trapezius muscle or fascia (Figure 1). Tenderness of this nerve is frequently appreciated where it emerges at the base of the skull over the greater occipital notch [10]. The lesser occipital nerve (LON) originates from the ventral primary ramus of C2 and C3 [25]. This nerve penetrates the deep cervical fascia and travels posterior to the insertion of the sternocleidomastoid where it divides into three branches: the auricular, mastoid, and occipital. Entrapment of the GON or LON anywhere along its path can produce occipital nerve pain. Reasons for this pain include trauma, tumors, infection, and surgical interventions, particularly after craniocervical junction surgeries [10]. Occipital pain can also be

 doi:10.20944/preprints202512.2572.v1

referred from the AO, AA, or upper cervical zygapophyseal (facet) joints and the C2-C3 intravertebral disc [19,26,27]. This can be distinguished from other causes of occipital neuralgia if movement of the effected facet joint intensifies occipital pain. Pain can also result from C2 neuralgia, which is caused by lesions effecting the C2 nerve root or dorsal ganglion (i.e. meningioma, neuroma, or vascular malformation), or entrapment/irritation of the C2 root, such as from subluxation from rheumatoid arthritis [10,25]. C2 neuralgia clinically presents as intermittent lancinating occipital pain associated with lacrimation, ciliary injection, and rhinorrhea [10,14]. The diagnosis can be confirmed through elimination of the pain with a selective C2 nerve root block. Given the presence of the trigeminocervical complex at C1-C3, individuals with migraine headaches may experience occipital neuralgia. These individuals can also experience pain in the frontal-orbital area, through the trigeminal spinal nucleus [10].

Table 2. ICHD3 criteria for Occipital Neuralgia.

Occipital Neuralgia

A. Unilateral or bilateral pain in the distribution(s) of the greater, lesser, and/or third occipital nerves and fulfilling criteria B through D.

B. Pain has at least 2 of the following 3 characteristics
   1. Recurring in paroxysmal attacks lasting from a few seconds to minutes
   2. Severe in intensity
   3. Shooting, stabbing or sharp in quality

C. Pain is associated with both of the following:
   1. Dysesthesia and/or allodynia apparent during innocuous stimulation of scalp and/or hair
   2. Either or both of the following:
      a. Tenderness over the affected nerve branches
      b. Trigger points at the emergence of the greater occipital nerve

D. Pain is eased temporarily by local anesthetic block of the affected nerve(s)

E. Not better accounted for by another ICHD-3 diagnosis

 doi:10.20944/preprints202512.2572.v1

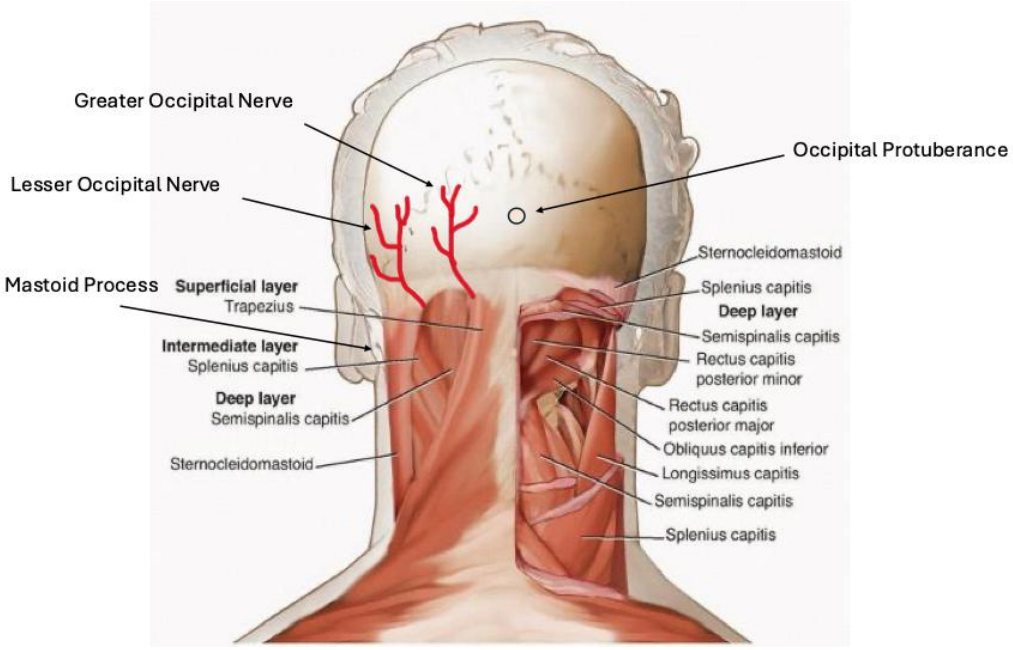


Figure 1. Diagram of occipital nerves, which are involved in occipital neuralgia. The greater and lesser occipital nerves are 2/3 and 1/3 the distance from the mastoid process to the occipital protuberance respectively.

Diagnostic Efficacy and Therapeutic Role of Greater Occipital Nerve Blocks

Occipital nerve blocks are thought to assist in the diagnosis, as some studies have shown that those with a cervicogenic cause to occipital neuralgia will benefit from such injections, experiencing a cessation of symptoms for a month or more compared to other causes [28,29]. For example, one study found that GON blocks resulted in pain reduction in 54.5% of those with cervicogenic headache compared to 14% in those with tension-type headaches and only 6% in those with migraines [2,29]. To the contrary, more recent studies suggest that these blocks produce a neuromodulatory effect on headaches, whether it is of cervical origin or not [14]. For example, these studies indicate that occipital nerve blocks can also relieve headaches, abet temporarily, in those with migraines, cluster headaches, or hemicrania continua [30–32]. The effectiveness of GON blocks in the treatment of acute migraine was not dependent on concentration and dose of anesthetic, which, in one study, compared the following: 0.25% bupivacaine (Volume in ml =mean [range]:6.30 [1.50-20.00]), 0.5% bupivacaine (Volume in ml=mean [range]: 2.5[0.25-5.00]), and 1% lidocaine (Volume in ml=mean: 2.4) [32] This advocates that a positive response to occipital nerve blocks does not confirm a cervicogenic origin to headache [31,33]. Nonetheless, this shows that occipital nerve blocks can be helpful when occipital neuralgia is present in any of the aforementioned headache types.

Cervical Facet Joint Pain

Pathophysiology/Clinical Diagnosis

Nociceptive structures in the facet joint capsules and cervical nerve roots are also suggested to play a role in headache pain [12,14,34,35]. For instance, the lateral AA joint which is innervated by the C2 ventral ramus accounts for approximately 16% of occipital headaches [14,36]. Headaches arising from the lateral AA joint present as occipital or suboccipital pain with little radiation and focal tenderness over the suboccipital area or over the transverse process of C1 [10,37]. Evaluation of the AA joint is established during the cervical flexion-rotation test (CFR). During this test, with the patient supine, the examiner flexes the cervical spine, fully blocking rotational movement below the AA joint [38]. The head is then passively rotated to determine range of motion (ROM). In those with AA joint restriction, AA rotation during the CFR test is restricted to 25-28 degrees or less on the side of the headache (average rotation is 44 degrees) [38]. Pathology of the lateral AA joint pain is usually

 doi:10.20944/preprints202512.2572.v1

degenerative, osteoarthritic or posttraumatic. However, presence of osteoarthritic change on imaging studies of the joint does not always confirm the joint as the cause of head pain. Moreover, pain can still originate from the joint despite normal imaging findings [10].

The C2-C3 facet joints are the most common source of cervicogenic headache [10,27], [Figure 2]. This presents with unilateral pain overlying the joint along with occipital pain that can spread to the frontal region and orbit. This distribution of pain is usually reproduced by loading the facet joint on the ipsilateral side [10]. This joint is innervated by the third occipital nerve, which is part of the superficial medial branch of the dorsal ramus of C3 [39]. Because of this, pain arising from the C2-C3 facet joint is named "third occipital headache." This headache type is seen in 27% of patients who experience whiplash injury as about 20% of the facet joint capsule area involves semispinalis multifudus and rotator neck muscles, which undergo excessive contraction during whiplash, amplifying neck and head pain [10,39].

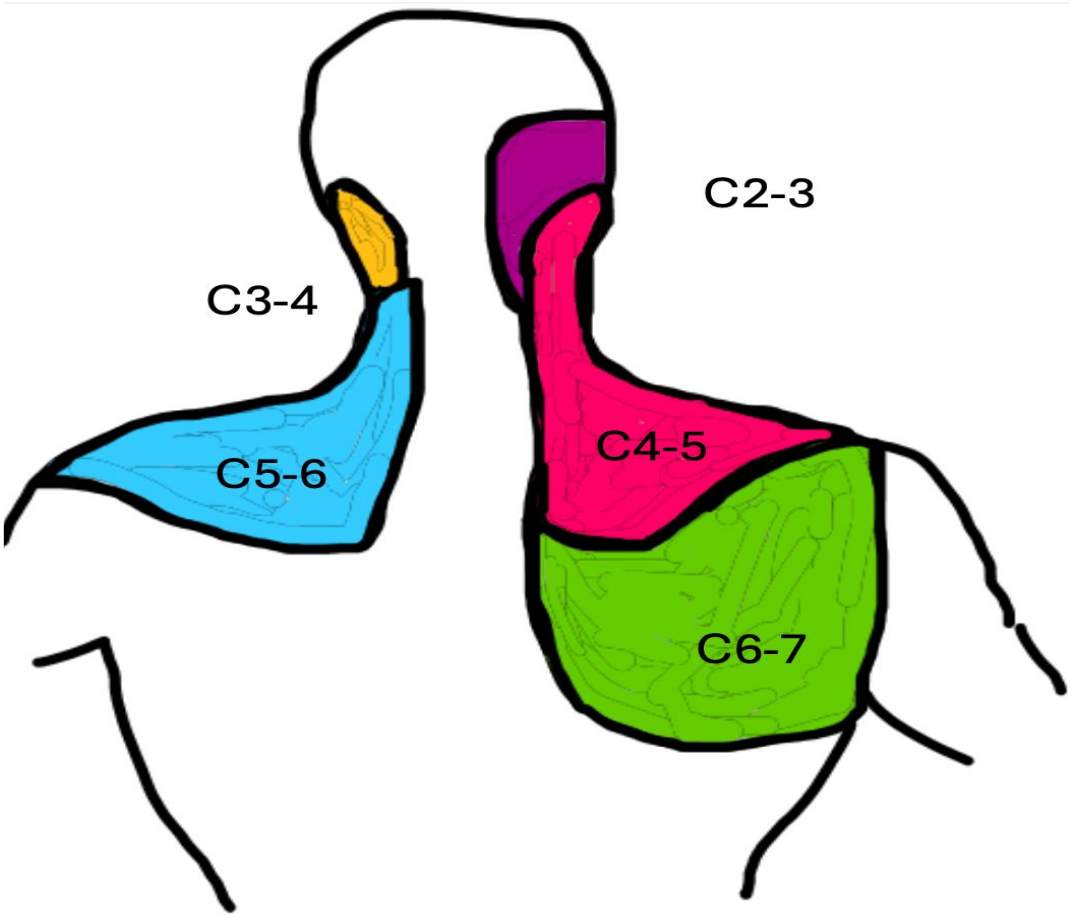


Figure 2. Distributions of cervical facet pain. Cervicogenic headache caused by cervical facet pain most frequently arises from the C2-C3 facet joints.

Interventional Diagnosis

Interventional pain management physicians use diagnostic blocks to evaluate whether a particular cervical structure is the source of headache pain. The only way to establish that the AA joint is a contributing factor is the abolishment of headache with injection of intra-articular steroid and local anesthetic to the joint [37,41]. In one observational study, 26 out of 32 patients obtained immediate relief after intra-articular steroid injection. One in five experienced greater than 50% relief from headache for 3 months and one in eight experienced complete headache relief for 9 months [42]. One limitation to this study is that the placebo effect was not accounted for given that it was not a

 doi:10.20944/preprints202512.2572.v1

randomized controlled clinical trial. Research has shown that the C2-C3 facet joint can be anesthetized by blocking the third occipital nerve where it cross and supplies the joint with articular branches [39,43]. Pain arising from the C3-C4 facet joint can be alleviated by blocking the medial branches of the C3 and C4 dorsal rami [39,43]. Complete relief following these diagnostic blocks strongly supports a cervical facet etiology of headache.

Treatment with Radiofrequency Neurotomy

Radiofrequency neurotomy is the most frequently studied treatment for cervical facet causes of cervicogenic headache. This procedure is suggested if headaches are temporarily relieved by blocking nerves innervating a specific cervical facet joint. Subsequently ablating the nerve can disrupt the pain signal along that nerve [8,14]. This treatment is particularly applicable for treatment of headaches originating from the C2-C3 facet joint.

There is evidence both supporting and refuting this procedure in treatment of cervicogenic headache. In studies showing no benefit of this procedure, neurotomy was done at levels C2-C6. In one study, only one person out of 15 had complete relief from pain [44]. In another small randomized controlled study, outcomes were no different in those who received C2-C6 radiofrequency neurotomy ipsilateral to the pain (n=6) versus sham treatment (n=6) [45]. A third study showed that outcomes from neurotomy at the C3-C6 facet joints in 30 participants were no different than those who received greater occipital nerve blocks [46]. These studies were not without limitations. One is that neurotomy included lower cervical spine segments, which rarely is a source of cervicogenic headache. Also, they did not adequately ascertain the source of pain. Conversely, studies that support use of the procedure adequately established the origin of the pain at the C2-C3 facet joint through diagnostic blocks. The joint was then denervated by radiofrequency neurotomy of the third occipital nerve. This procedure led to complete relief of headache in 88% of patients with median duration of relief lasting 297 days [11,47,48]. If headache reoccurred the procedure was then repeated, which led to sustained relief from headache for more than 2 years [14,47,48]. This shows that radiofrequency neurotomy can lead to long-term pain relief and should be trialed in patients who had a positive response to diagnostic nerve blocks.

C3 Cervical Radiculopathy

Cervical radiculopathy at C2-C3 can present similarly to C2-C3 facet disease with side-locked, sharp, or achy pain in the temporal, retro-auricular, occipital and/or suboccipital regions [40]. Findings on magnetic resonance images (MRI) of the cervical spine in cervical radiculopathy show foraminal stenosis in the C2-C3 level, while arthropathy or degenerative changes of the C2-C3 facet joints are common with C2-C3 facet joint pain [40]. Per the ICHD-3 criteria, diagnosis can be confirmed if headache is temporarily relieved with a selective C3 nerve root block (Table 3) [40]. Decompressive foraminotomy of the C2-C3 spine at the side of the pain can lead to lasting headache relief if there is a positive response to a selective nerve root block and significant foraminal stenosis [40].

Table 3. The ICHD3 criteria in the appendix for cervical causes to headache.

Headache attributed to Upper cervical radiculopathy
A. Headache and/or neck pain fulfilling criterion C
B. Clinical, electrodiagnostic or radiological evidence of a C2 or C3 radiculopathy
C. Evidence of causation demonstrated by both of the following:
1. At least two of the following
a) Pain has developed in temporal relationship to the radiculopathy

 doi:10.20944/preprints202512.2572.v1

b) Pain has significantly improved or worsened in parallel with improvement or worsening of the radiculopathy

c) Pain is temporarily halted by local anesthesia to the nerve root

2. Headache is ipsilateral to the radiculopathy

Not better accounted for by another ICHD-3 diagnosis

Headache attributed to cervical myofascial pain

A. Head and/or neck pain fulfilling criterion C

B. A source of myofascial pain the muscles of the neck, including trigger points

C. Evidence of causation demonstrated by at least two of the following:

1. Either or both of the following:

a) Pain has developed in temporal relation to the onset of cervical myofascial pain disorder

b) Pain has significantly improved in parallel with improvement in the cervical myofascial pain disorder

2. Tenderness with pressing on the implicated cervical muscles

3. Pain is halted by local anesthetic injections into trigger points or by trigger point massage

Not better accounted for by another ICHD-3 diagnosis

Cervical Myofascial Pain

Myofascial trigger points are thought to be secondary to increased acetylcholine (Ach) levels, leading to excessive muscle contraction causing a release of pro-inflammatory cytokines and the formation of trigger points [49]. One small study showed that individuals with headache attributed to cervical myofascial pain are more likely to experience upper trapezius muscle tension and reduced function of deep neck flexor muscles than those without headache [50]. Tender and trigger points in the posterior neck muscles, particularly the trapezius, sternocleidomastoid, and the splenius capitis are also common myofascial sources of headache [51,52]. This finding, however, is non-specific as myofascial pain can be seen in those with tension headaches with pericranial tenderness and in migraineurs [10]. Furthermore, tender points may encompass the facet joints making it difficult to differentiate from joint pain [10,53]. Because of the difficulty in diagnosing cervical myofascial pain, ICHD-3 includes a dedicated criterion (Table 3). Massage and neck physical therapy may assist in relaxing suboccipital, trapezius, and cervical paraspinal muscles. These modalities can also be a useful initial treatment approach for other causes of cervicogenic headache. One systemic review showed that combined interventions such as dry needling with spinal manipulation and muscle energy with exercise resulted in the greatest reduction of short-term headache intensity [54]. Caution should be taken with spinal manipulation techniques as studies have only shown short-term benefit

 doi:10.20944/preprints202512.2572.v1

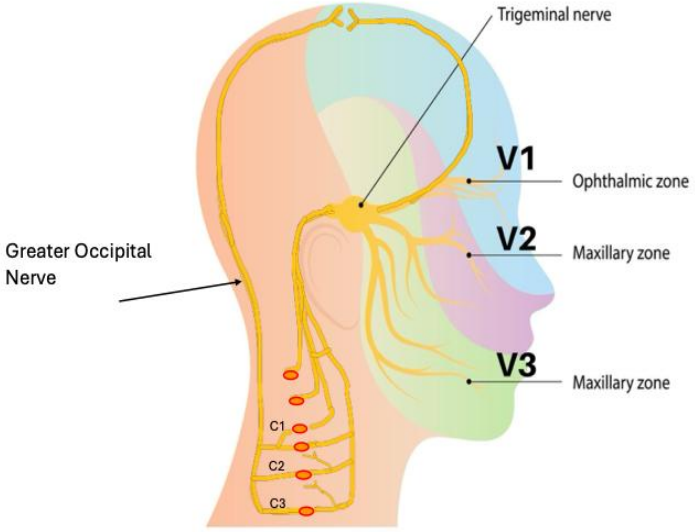
and there is a risk of vertebral artery dissection and subsequent stroke [55]. Other modalities that showed benefit in reducing pain intensity include soft tissue techniques (i.e. myofascial release, trigger point therapy), and dry needling with exercise [56]. Trigger point injections may be effective for cases less responsive to conservative therapies, but more studies are needed to evaluate if any of these treatments have long-term benefit for this type of pain [54]. Alternative therapies, such as transcutaneous electric nerve stimulation (TENS), osteopathic manipulative therapies, and acupuncture may also be beneficial. onabotulinumtoxinA can be considered in cases of severe cervical myofascial hypertonicity, and pain resistant to trigger point injections, however, its efficacy is still debatable. Although helpful in a subset of patients, more evidence-based research is needed to assess its efficacy in treating myofascial neck pain [10,56–58].

Pathophysiology of the Trigeminocervical Complex

Individuals with migraine can present with occipital and neck pain through the trigeminocervical complex. This is formed by the convergence of nociceptive sensory transmission from the C1-C3 nerve roots (cervical afferents) and trigeminal afferents at the trigeminal nucleus caudalis located in the brainstem and upper cervical spine [8,10,12,13], [Figure 3A]. Convergence between cervical afferents allows for referral of pain to regions of the head innervated by the cervical nerves. Convergence of trigeminal afferents leads to referred pain in the parietal, occipital, and orbital regions [14]. Ascending pain pathways project via the spinothalamic tract to the thalamus and higher cortical structures [15], [Figure 3B]. Neurosurgical observations have shown that stimulation of supratentorial dura mater and large cranial arteries causes pain in both the ophthalmologic division of the trigeminal nerve and dermatomes supplied by the upper cervical nerve roots [16–18]. Stimulation of the posterior fossa or upper cervical roots elicits not only occipital pain but also pain in trigeminal innervated regions such as the frontal regions and the orbit [7,19]. On the other hand, stimulation of lower cervical spine structures only elicited pain in the neck with some referral to the occipital regions [14]. There has also been evidence to suggest that neurostimulation of peripheral pain sources such as the greater occipital nerve can temper migraine pain [20]. In one study, neurostimulation of structures near the greater occipital nerve led to decreased migraine pain through activation of the anterior cingulate cortex, which is known to modulate migraine pain [15,21,22]. The effects of neurostimulation are postulated to be secondary to modulation of rostral pain structures in the dorsal rostral pons, periaqueductal gray (PAG), and thalamus [14,20,21,23,24]. This illustrates evidence of the connection between nociceptive dural and cervical afferents in the greater occipital nerve onto the trigeminocervical complex. Patients with chronic headaches disorders, such as chronic migraine, can also experience scalp allodynia and occipital nerve tenderness from over-activation of nociceptors leading to central sensitization [10,15,16]. This exemplifies the complex bidirectional transmission of sensory information between the trigeminal nerve and structures of the neck innervated by the upper cervical nerve roots [10,15,16].

 doi:10.20944/preprints202512.2572.v1

A.



B.

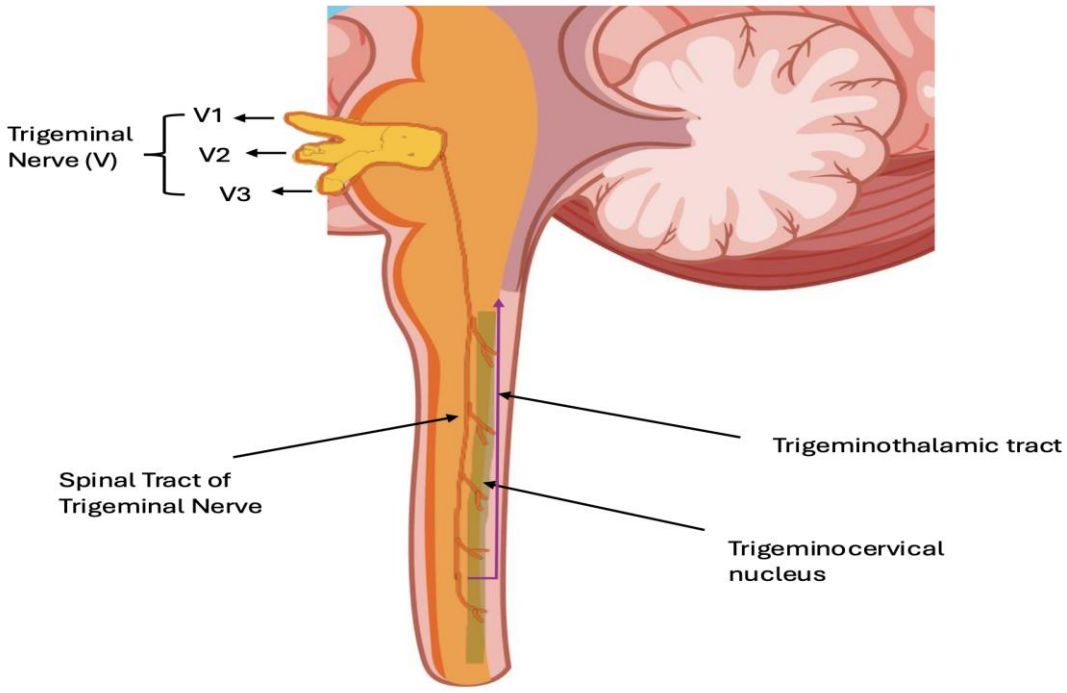


Figure 3. 3A. Diagram of the connection between the trigeminal and cervical afferents. **3B.** Diagram of the trigeminocervical complex illustrating pain transmission from the cervical spinal nerves through the spinal tract of the trigeminal nerve and the trigeminothalamic tract.

 doi:10.20944/preprints202512.2572.v1

Migraine Versus Cervicogenic Headache

Because many other cervicogenic headache types can overlap with migraine, an algorithm has been constructed to assist in the diagnosis and treatment of cervicogenic headache (Figure 1). Those with cervicogenic headache usually have a profound response to interventional treatments, but efficacy is dependent on identifying and treating the specific cervicogenic origin of pain (Figure 4). Interventional treatment may be insufficient if overlaying migraine is not treated adequately. Symptoms that suggest coexisting migraine include light and sound sensitivity or nausea. Patients with migrainous symptoms and suspected cervicogenic headache should be treated with migraine-specific therapies in conjunction with interventional procedures. Other than migraine, presence of centralized pain disorder, i.e. fibromyalgia, can further complicate treatment. Consequently, it is recommended that any centralized pain should be treated first, as prematurely introducing interventional treatments can be minimally beneficial, or potentially worsen symptoms. Chronic centralized pain is best treated with a multidisciplinary approach, incorporating physical therapy, pharmacotherapy, and psychotherapy (biofeedback and meditation) [10,59].

Other than response to interventional treatments, there have been other physical exam findings that might indicate a cervicogenic component to headache. For example, one review suggests that those with cervicogenic headaches are more likely to have restricted range of motion in flexion and extension, and reduced neck flexion strength compared to those with migraines [1]. They were also found to have reduced cervical range of motion (ROM) during the CFR test compared to migraine patients [1,60,61], [see Table 4 for comparisons]. There were, however, limitations to these evaluations. Diagnostic blocks confirming cervicogenic headache were not performed, so it cannot be said if these patients definitively had cervicogenic headache or another pathology, such as centralized pain disorder. The sample size for comparison of participants in the study was also small, which limits the generalizability of these results. Despite these limitations, regular testing of cervical ROM in patients suspected of having concurrent cervicogenic headache may help in obtaining diagnostic clarity.

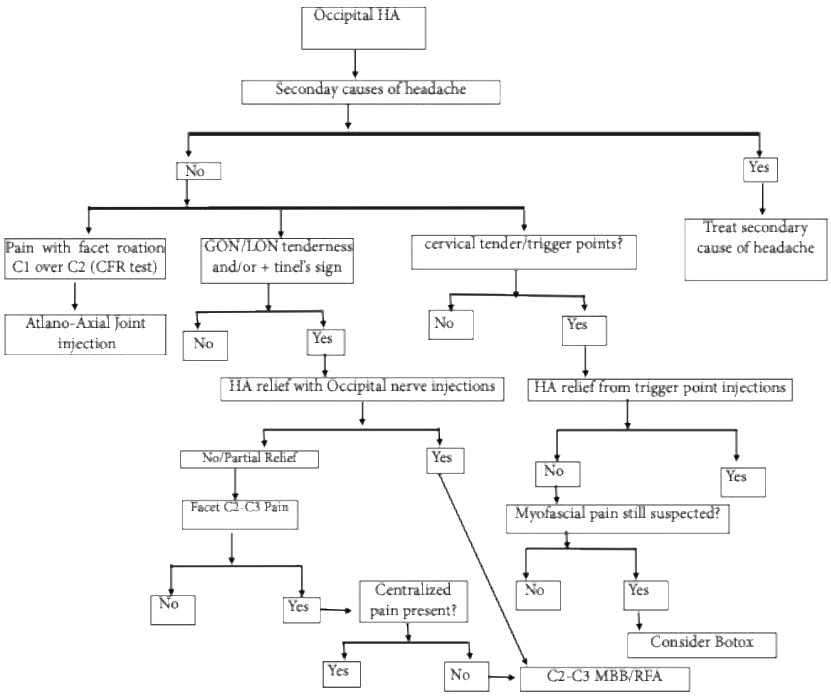


Figure 4. Algorithm for diagnosis and treatment of cervicogenic headache. *Abbreviations:* HA=headache, GON= greater occipital nerve, LON= lesser occipital nerve, CFR= cervical facet rotation, MBB= medial branch block, RFA= radiofrequency ablation.

 doi:10.20944/preprints202512.2572.v1

Table 4. Distinguishing Characteristics of Migraine and Cervicogenic Headache.

	Migraine	**Cervicogenic Headache**
Pain Location	Unilateral that switches sides, but may vary	Unilateral, fixed side
Associated symptoms: Aura, Nausea, light and sound sensitivity,	Common	Rare
Cervical ROM	Normal or mildly reduced	Often significantly reduced, particularly with CFR test
Response of Diagnostic Blocks	Limited Improvement	Often dramatic improvement

Of note, some patients might have both migraine and cervicogenic headache.

Discussion

Diagnosis of cervicogenic headache can be a challenge in those with underlying migraine, as both can present with musculoskeletal impairments. History, physical exam findings, and interventional procedures can assist with diagnosis, and are summarized for each cause of cervicogenic headache. Physical exam findings that support the diagnosis of cervicogenic headache include posterior-to-anterior radiation of pain with loading cervical facet joints, side-locked pain, reduced ROM on CFR test, and reduced neck flexion strength [10,60,61]. Treatment can be complicated by the presence of the trigeminocervical complex [3,7,20]. This can lead to sensitization during headache resulting in hypersensitivity and spread of referred pain to trigeminal and cervical dermatomes. In these cases, the addition of a multidisciplinary treatment plan along with migraine-specific medications can be beneficial.

Conclusion

In summary, it is essential to accurately diagnose cervicogenic headache to avoid inadequate and costly abortive and prophylactic therapies, and opioid or analgesic rebound from medication overuse [2]. This can be accomplished by identifying the source of pain through history and physical exam findings outlined in the previous sections, utilizing the diagnostic criteria established by the ICHD-3, and performing diagnostic blocks if cervicogenic headache is suspected (Table 1, Figure 1) [10,12]. Adhering to these steps can assist providers in recognizing the precise cervicogenic cause, leading to treatment of this type of headache. Larger, controlled studies are needed to further elucidate the distinguishing factors between cervicogenic headache and migraine, and to evaluate the efficacy of interventional treatments for cervical facet causes of cervicogenic headache in those with migraine.

Conflict: of Interest Dr. Sharma declares receiving grants from ITHS at UW outside the scope of this work. She also has stock in Profunds and JP Morgan.

 doi:10.20944/preprints202512.2572.v1

References

1. Racicki S, Gerwin S, Diclaudio S, Reinmann S, Donaldson M. Conservative physical therapy management for the treatment of cervicogenic headache: a systematic review. J Man Manip Ther. 2013; 21(2):113-24. doi: 10.1179/2042618612Y.0000000025. PMID: 24421621; PMCID: PMC3649358.
2. Yi X, Cook AJ, Hamill-Ruth RJ, Rowlingson JC. Cervicogenic headache in patients with presumed migraine: Missed diagnosis or misdiagnosis? Journal of Pain. 2005; 6(10), 700–703.
3. Anarte-Lazo E, Carvalho GF, Schwarz A, Luedtke K, Falla D. Differentiating migraine, cervicogenic headache and asymptomatic individuals based on physical examination findings: a systematic review and meta-analysis. BMC Musculoskeletal Disorders. 2021; 22(1), 1–18.
4. Becker WJ. Cervicogenic headache: evidence that the neck is a pain generator. Headache. 2010; 50(4):699–705. https://doi.org/10.1111/j.1526-461 0.2010.01648.x.
5. Blumenfeld A, Siavoshi S. The challenges of cervicogenic headache. Curr Pain Headache Rep. 2018; 22(7):47. https://doi.org/10.1007/s11916-018-0699- z.
6. PfaffenrathV, Kaube H. Diagnostics of cervicogenic headache. Funct Neurol. 1990; 5(2):159–64.
7. Bartsch T, Goadsby PJ. Anatomy and physiology of pain referral patterns in primary and cervicogenic headache disorders. Headache Currents. 2005; *2*(2), 42–48. https://doi.org/10.1111/j.1743-5013.2005.20201.x
8. Grandhi RK, Kaye AD, Abd-Elsayed A. Systematic Review of Radiofrequency Ablation and Pulsed Radiofrequency for Management of Cervicogenic Headaches. Current Pain and Headache Report. 2018; *22*(3), 109–130. https://doi.org/10.1007/s11916-018-0673-9
9. Yu M, Wang SM. Anatomy, Head and Neck, Occipital Nerves. [Updated 2022 Oct 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.
10. Narouze SN. Interventional management of head and face pain: Nerve blocks and Beyond. Springer New York; 2014. p1-463.
11. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018; 38 (1):1-211.
12. Antonaci F, Ghirmai S, Bono G, Sandrini G, Nappi G. Cervicogenic headache: evaluation of the original diagnostic criteria. Cephalalgia. 2001; 21(5):573–83. https://doi.org/10.1046/j.0333-1024.2001.00207.x
13. Akerman S, Holland PR, Hoffmann J. Pearls and pitfalls in experimental in vivo models of migraine: Pearls and pitfalls in experimental in vivo models of migraine: Dural trigeminovascular nociception. Cephalalgia. 2013; 33:577-592.
14. Bogduk N Govind J. Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests, and treatment. The Lancet Neurology. 2009; *8*(10), 959–968. https://doi.org/10.1016/S1474-4422(09)70209-1
15. Bartsch T, Goadsby PJ. Increased responses in trigeminocervical nociceptive neurons to cervical input after stimulation of the dura mater. Brain. 2003; 125(Pt 8): 1801-13.
16. Bartsch T, Goadsby PJ. The trigeminocervical complex and migraine: current concepts and synthesis. Curr Pain Headache Rep. 2003; 7(5);371-6.
17. Wolff H. Headache and Other Head Pain. New York: Oxford University Press; 1963.
18. Penfield W, McNaughton F. Dural headache and innervation of the dura mater. Arch Neurol Psychiatry. 1940; 44:43–75.
19. Wirth FP, Jr., Van Buren JM. Referral of pain from dural stimulation in man. J Neurosurg.1971;34(5):630–642.
20. Bartsch T, Goadsby PJ. Anatomy and physiology of pain referral patterns in primary and cervicogenic headache disorders. Headache Currents. 2005; *2*(2), 42–48. https://doi.org/10.1111/j.1743-5013.2005.20201.
21. Bahra A, Matharu MS, Buchel C, Frackowiak RS, Goadsby PJ. Brainstem activation specific to migraine headache. Lancet. 2001; 357(9261):1016–1017.
22. Weiller C, May A, Limmroth V, Juptner M, Kaube H, Schayck RV, Coenen HH, Diener HC. Brain stem activation in spontaneous human migraine attacks. Nat Med. 1995;1(7):658–660.
23. Lindblom U, Tapper DN, Wiesenfeld Z. The effect of dorsal column stimulation on the nociceptive response of dorsal horn cells and its relevance for pain suppression. Pain. 1977; 4(2):133– 144.

 doi:10.20944/preprints202512.2572.v1

24. Garrison D, Foreman R. Decreased activity of spontaneous and noxiously evoked dorsal horn cells during transcutaneous electrical nerve stimulation (TENS). Pain. 1994; 58(3):309–315.
25. Poletti CE, Sweet WH. Entrapment of the C2 root and ganglion by the atlantoepistrophic ligament: Clinical syndrome and surgical anatomy. *Neurosurgery*. 1990; 27:288-91.
26. Dreyfuss P, Michaelsen M, Fletcher D. Atlanto-occipital and lateral atlanto-axial joint pain patterns. Spine. 1994; 19: 1125–31.
27. Dwyer A, Aprill C, Bogduk N. Cervical zygapophysial joint pain patterns I: a study in normal volunteers. Spine 1990; 15: 453–57. Schellhas KP, Smith MD, Gundry CR, Pollei SR. Cervical discogenic pain: prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers. Spine. 1996; 21: 300–12.
28. Anthony M. Headache and the greater occipital nerve. Clin Neurol Neurosurg. 1992; 94:297–301.
29. Bovim G, Sand T. Cervicogenic headache, migraine without aura and tension-type headache. Diagnostic blockade of greater occipital and supra-orbital nerves. Pain. 1992; 51:43-48.
30. Afridi SK, Shields KG, Bhola R, Goadsby PJ. Greater occipital nerve injection in primary headache syndromes—prolonged effects from a single injection. Pain. 2006; 122: 126–29.
31. Goadsby PJ, Lipton RB, Ferrari MD. Migraine—current understanding and treatment. N Engl J Med. 2002; 346: 257–70.
32. Allen SM, Mookadam, F, Cha SS, Freeman JA, Starling AJ, Mookadam, M. Greater occipital nerve block for acute treatment of migraine headache: A large retrospective cohort study. Journal of the American Board of Family Medicine 2018. *31*(2), 211–218. https://doi.org/10.3122/jabfm.2018.02.170188.
33. Leone M, Cecchini AP, Mea E, Tullo V, Bussone G. Epidemiology of fixed unilateral headaches. Cephalalgia. 2008; 28.
34. Kamins, J. Models for Treating Post-traumatic Headache. Current Pain and Headache Reports. 2021; *25*(8), 1–9. https://doi.org/10.1007/s11916-021-00970-3.
35. Grubb SA, Kelly CK. Cervical discography: clinical implications from 12 years of experience. Spine 2000; 25: 1382–89.
36. Fukui S, Ohseto K, Shiotani M, Ohno K, Karasawa H, Naganuma Y, Yuda Y. Referred pain distribution of the cervical zygapophyseal joints and cervical dorsal rami. Pain. 1996; 68(1):79–83.
37. Aprill C, Axinn MJ, Bogduk N. Occipital headaches stemming from the lateral atlanto-axial (C1-C2) joint. Cephalagia. 2002; 22 (1):15-22.
38. Page, P. Clinical Suggestion Cervicogenic Headaches : an Evidence Led Approach to Clinical Management. The International Journal of Sports Physical Therapy. 2011;6(3), 254–266. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3201065/
39. Lord S, Barnsley L, Wallis B, Bogduk N. Third occipital headache: A prevalence study. J Neurol Neurosurg Psychiatry. 1994; 57: 1187-90.
40. Ko HC, Son, BC. Chronic Unilateral Temporo-Occipital Headache Attributed to Unilateral C3 Radiculopathy. Asian J Neurosurg. 2018; Oct-Dec;13(4):1229-1232. doi: 10.4103/ajns.AJNS_197_17. PMID: 30459902.
41. McCormick CC. Arthrography of the atlanto-axial (C1–C2) joints: technique and results. J Intervent Radiol. 1987; 2: 9–13. Busch E, Wilson PR. Atlanto-occipital and atlanto-axial injections in the treatment of headache and neck pain. Reg Anesth 1989; 14 (suppl 2): 45.
42. Narouze SN, Casanova J, Mekhail N. The longitudinal effectiveness of lateral atlantoaxial intra-articular steroid injection in the treatment of cervicogenic headache. Pain Med 2007; 8: 184–88.
43. International Spine Intervention Society. Cervical medial branch blocks. In: Bogduk N, ed. Practice Guidelines for Spinal Diagnostic and Treatment Procedures. San Francisco: International Spinal Intervention Society. 2004; 112–37.
44. van Suijlekom HA, van Kleef M, Barendse GAM, Sluijter ME, Sjaastad O, Weber WEJ. Radiofrequency cervical zygapophyseal joint neurotomy for cervicogenic headaches: a prospective study of 15 patients. Funct Neurol. 1998; 13: 297–303.
45. Stovner LJ, Kolstad F, Helde G. Radiofrequency denervation of facet joints C2–C6 in cervicogenic headache: a randomised, double-blind, sham-controlled study. Cephalalgia 2004; 24: 821–30.

 doi:10.20944/preprints202512.2572.v1

46. Haspeslagh SR, van Suijlekom HA, Lame IE, Kessels AG, van Kleef M, Weber WE. Randomised controlled trial of cervical radiofrequency lesions as a treatment for cervicogenic headache. BMC Anesthesiol. 2006; 6: 1.
47. Govind J, King W, Bailey B, Bogduk N. Radiofrequency neurotomy for the treatment of third occipital headache. J Neurol Neurosurg Psychiatr. 2003; 74: 88–93.
48. Lord SM, Barnsley L, Wallis BJ, McDonald GJ, Bogduk N. Percutaneous radio-frequency neurotomy for chronic cervical zygapophysial-joint pain. N Engl J Med. 1996; 335: 1721–26.
49. Bordoni B, Sugumar K, Varacallo MA. Myofascial Pain. [Updated 2023 Aug 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK535344.
50. Jull G, Barrett C, Magee R, Ho P. Further characterisation of muscle dysfunction in cervical headache. Cephalalgia. 1999; 19(22), 179–185.
51. Graff-Radford SB, Reeves JL, Jaeger B. Management of chronic head and neck pain effectiveness of altering factors perpetuating myofascial pain. Headache 1987;27:186-90.
52. Jaeger B. Are cervicogenic headaches due to myofascial pain and cervical spine dysfunction. Cephalalgia. 1989; 9:157-64.
53. Bodguk N. The neck and headache. Neurol Clin. 2004; 22 (1):151-71.
54. Jung, A, Carvalho, GF, Szikszay, TM, Pawlowsky, V, Gabler, T, Luedtke, K. Physical Therapist Interventions to Reduce Headache Intensity, Frequency, and Duration in Patients With Cervicogenic Headache: A Systematic Review and Network Meta-Analysis. Physical Therapy. 2024; 104(2), 1–15. https://doi.org/10.1093/ptj/pzad154.
55. Ernst, E. Adverse effects of spinal manipulation: A systematic review. Journal of the Royal Society of Medicine. 2007; 100(7), 330–338. https://doi.org/10.1258/jrsm.100.7.330.
56. Cheshire, WP, Abashain, SW, Mann, JD. Botulism toxin in the treatment of myofascial pain syndrome. Pain.1994; 59:65-9.
57. Freud BJ, Schwartz M. Treatment of whiplash-associated neck pain with botulinum toxin-A: a pilot study. J Rheumatol. 2000; 27: 481-4.
58. Wheeler AH, Goolkasian P, Gretz SS. A randomized, double-blind, prospective pilot study of botulism toxin injection for refractory, unilateral, cervicothoracic, paraspinal, myofascial pain syndrome. Spine. 1998; 23:1662-6.
59. Dydyk AM, Givler A. Central Pain Syndrome. [Updated 2023 Feb 19]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK553027/
60. Hall TM, Brikka K, Hooper D, Robinson K. Comparative analysis and diagnostic accuracy of the cervical flexion-rotation test. J Headache Pain. 2010;11(5):391–7. https://doi.org/10.1007/s10194-010-0222-3.
61. Ogince M, Hall, T, Robinson K, Blackmore AM. The diagnostic validity of the cervical flexion-rotation test in C1/C2-related cervicogenic headache. Man Ther. 2007; 12(3):256–62. https://doi.org/10.1016/j.math.2006.06.016.