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Review Article

Cervicogenic Headache

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Chronic headache is a significant medical and socioeconomic problem resulting in severe disability and impairment. The term "cervicogenic headache" was coined by Sjaastad in 1983, who also proposed criteria for its diagnosis. Cervicogenic headache as described by Sjaastad et al is characterized as recurrent, long lasting, severe unilateral headache arising from the neck.

Exact pathoanatomic and pathophysiologic basis for cervicogenic headache is unclear. Numerous authors have proposed various theories ranging from neurophysiologic basis involving ascending fibers from the C1 and C2 nerve roots to multiple pain generators in pain-sensitive structures involved in head movement. Thus, cervicogenic headache should be considered as a descriptive term rather than a final diagnosis. Because of the numerous potential pain generators, neither uniform clinical findings, nor a pathophysiology has been defined for the entity known as cervicogenic headache. Sequential diagnostic injections may elucidate pain generators and differentiate it from other types of headaches.

This review describes the epidemiological and clinical aspects of cervicogenic headache, pathophysiology, diagnostic strategies to differentiate it from other common headaches and describes various non-operative treatment strategies.

Keywords: Cervicogenic headache, zygapophysial joint, facet joint nerves, medial branches, greater occipital nerve, lesser occipital nerve, radiofrequency neurotomy

Acute and chronic headaches are a significant medical, industrial and socioeconomic problem. While the exact pathophysiology of headaches is frequently unknown, the disability is severe in comparison to the physiologic impairment (1). Josey may have been the first to describe cervical spine-related headaches in 1949 (2). It was not until 1983 that Sjaastad et al (3) coined the descriptive term "cervicogenic headache" and proposed criteria for its diagnosis. Since that time, cervicogenic headache has gained more notoriety (4-7).

Because etiologies of headache are so diverse, it is critical that a correct diagnosis be made prior to initiation of therapeutic plan. This article will review the epidemiological and clinical aspects of cervicogenic headaches, proposed pathophysiology, diagnostic strategies to differentiate it from other common headaches and describe a various non-operative treatment strategies.

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EPIDEMIOLOGY

There is limited epidemiological data on cervicogenic headache. Significant variability in diagnostic criteria makes comparing one prevalence study to another difficult. The prevalence of clinically diagnosed cervicogenic headache among the headache population has been reported at 13.8-35.4% (8-11). Lord et al (12) found headaches associated with whiplash injuries to occur with a frequency of 88%. By using diagnostic blocks in the whiplash population, Lord et al (12) found 54% of these patients had neck pain stemming from a cervical zygapophysial joint. Clearly not all whiplash induced headaches are of cervical origin.

CLINICAL PRESENTATION

The classic cervicogenic headache, as described by Sjaastad et al (3, 13), is characterized as recurrent, long lasting, severe unilateral headache arising from the neck. The headache is always dominant on one side, but may also be present on the contralateral side to a lesser degree. The dominant pain does not alternate from one side to the other (side locked unilaterality). The typical headache

Table 1. *Criteria to include cervicogenic headache in the differential diagnosis*

Typically, the headache:

1. Is recurrent, long-lasting and severe
2. Arises from the neck
3. Has a unilateral dominance (but can bilateral)
4. Is in the low occipital and temporal region (with possible radiation in the face, periorbital, frontal and parietal region and ipsilateral shoulder and arm)
5. Is accompanied with a reduced cervical spine range of motion
6. Can be precipitated with certain neck motions

location is in the low occipital and temporal region and can radiate periorbitally. However, pain in the face, frontal, parietal and upper occipital region as well as the ipsilateral shoulder and arm is not rare. The cervical spine usually has a reduced range of motion, and the headaches can often be precipitated by the patient with certain neck motions. External pressure over the greater occipital nerve can also induce symptoms (Table 1).

Prior to Sjaastad et al's (3) term, some clinicians used the term "occipital neuralgia" for headaches of this nature. Occipital neuralgia suggests the pain is in the occiput, presumably in the territory of the greater occipital nerve. Because cervicogenic headache can often radiates into the frontotemporal region, other pathoanatomic and pathophysiologic mechanisms must be considered. A growing body of literature, beyond the limited scope of the greater occipital nerve, is shedding light on alternative etiologies.

DIFFERENTIATING CERVICOGENIC HEADACHE

Headaches are inherently a subjective complaint, though often quite debilitating, there is often a relative paucity of corroborating physical examination findings. Radiological studies using cerebral and cervical computer tomography as well as plain radiographs of the spine, cerebral angiography and cervical myelography showed no typical characteristic pathology in cervicogenic headache patient (14). Thus, until recently, differentiation of various forms of headaches often relied on subjective assessments by the clinician and upon clinical diagnostic criteria. Numerous studies have compared and contrasted cervicogenic headache with two common types of headaches-migraine and tension-type.

In contrast to migraine and tension-type headaches, cervicogenic headache can often be induced with neck movements (5, 15-17). Typical symptoms of common migraines such as nausea, vomiting, phonophobia and photophobia are uncommon in cervicogenic headache. Other accompanying phenomena that have also been noted in cervicogenic headache include dizziness, blurred vision and irritability (18).

Anthony (8) reported cervicogenic headache patients were predominantly female and were older (mean age of 49.5 years) than the typical migrainous group (34.7 years). The monthly headache frequency was also much higher in the cervicogenic headache group compared to the migraine group, 18 episodes to 6.9, respectively (8). Women in managerial and professional occupations were found to have significantly higher risk for cervicogenic headache than women working in clerical or blue-collar positions. No similar association was noted in men (19).

The characteristic of side-locked pain unilaterality, however, is not restricted to cervicogenic headaches. This symptom can also be found in migraine with or without aura, tension-type headache, cluster headache, chronic paroxysmal hemicrania, hemicrania continua and short-lasting unilateral neuralgiform attacks with conjunctival injectia and tearing as well. When comparing cervicogenic headache to this differential list, the majority of literature compares cervicogenic headache to migraine and tension-type headache. While unilaterality is a mandatory criterion for cervicogenic headache according to Sjaastad et al (3), it can be found in 10-29% of migraines and 7-12.5% of tension-type headaches (20-22). While there is significant overlap of headache distribution, some tendencies were noted. In common migraines, pain tended to localize anteriorly, particularly at onset, while tension-type headaches were more diffuse. In contrast, cervicogenic headache always began in the occipitonuchal region (20-22). More uncommon and more sinister causes of headache must also be considered (23-25).

PATHOPHYSIOLOGY

The exact pathoanatomic and pathophysiologic basis for cervicogenic headaches is unclear. Numerous authors have proposed a neurophysiologic basis for these types of headaches (26-32). Ascending fibers from the C1 and C2 nerve

roots are intimately involved with the trigeminal nucleus at the level of the brainstem. This may explain how a cervical nociceptive process can affect sensation in the head (33, 34). The nociceptive afferents from the trigeminal nerve and the receptive fields from the first three cervical nerves converge in the trigeminocervical nucleus. Therefore, from the spine, only structures innervated by the first three cervical nerve roots (C1-C3) are capable of causing headache (29). These structures include muscles, joints, ligaments, a portion of the dura mater and the vertebral arteries (29, 35-37). An intimate continuity has also been observed between the posterior cervical spinal dura and the ligamentum nuchae, but again only at the first and second vertebral levels (38). While this well-defined concept is appealing, a case has been reported where a lower cervical spine decompression of the C6-C7 intervertebral disc herniation alleviated a 15-year history of unilateral headache. The surgery was performed for a more recent onset of C7 radicular symptoms (39).

It has been thought that cervicogenic headache could also be a result of various pain generators in pain-sensitive structures involved in head movement. Gronbaek (40) noted that referred pain to the head can occur when cephalic or cervical nociceptive volleys traverse the upper cervical medulla. Cervicogenic headache may also be mediated by a pro-inflammatory process (41, 42). This could suggest potential treatment strategies.

Often there is no specific precipitating event resulting in cervicogenic headaches. However, when an etiology is apparent, whiplash injuries are frequently the culprit. First described by Crowe (43) in 1928, whiplash refers to signs and symptoms from a traumatic extension-flexion acceleration-deceleration injury to the neck, usually following a rear impact motor vehicle accident. Whiplash injuries are a common occurrence with an incidence of perhaps one million per year in the United States (44). Headaches have been reported to occur in 37- 82% of whiplash patients acutely (45, 46). These were reported to be of the muscle contraction type and are often associated with greater occipital neuralgia (44). While the majority of minor head and neck injuries following a motor vehicle accident resolve in a few weeks, a reported 30% continue to have persistent head and/or neck pain (47).

Segmental hypomobility has also been implicated in cervicogenic headache. A computer-based technique found impaired overall mobility of the cervical spine, particularly from the occiput to C5, with the greatest hypomobility at the craniocervical joints C0-C2 in patients with cervicogenic headache (48). Meloche et al (49) and Jull (50) commented that a dysfunctional spinal segment can induce pain radiating in the dermatome of that segment, and that the most frequent dysfunctional segment is at C2-C3. Jensen et al (51) found reduced C1 through C7 flexion-extension motion in patients with post-traumatic headaches. In particular, significant reduction of C2-3, C5-6 and C6-7 motion was noted.

Some authors believe that the neck and upper back muscles play an important role in headaches. A low incidence of headaches has been described in patients with cervical spondylosis. Because there are no qualitative symptom or features distinguishing cervicogenic headache from tension-type headaches, Iansek et al (52) has suggested that muscle contractions are the basis of the cervicogenic headache. Myofascial trigger points (53) along with segmental cervical dysfunction (54) have also been implicated in playing an important role in cervicogenic headache.

Entrapment or irritation of the occipital nerves has also been postulated in the pathogenesis of cervicogenic headache (8, 55-58). The medial branch of the dorsal ramus of the C2 spinal nerve becomes the greater occipital nerve while the lesser occipital nerves arises from the cervical plexus formed by the ventral rami of C2 and C3. The greater occipital nerve classically innervates the occiput medially and the lesser occipital nerve laterally, though the literature has little consensus concerning the sensory innervation of the scalp. Neither the greaer occipital nerve nor the lesser occipital nerve directly innervates the frontal or periorbital region. However, experimental cat models have documented afferents from the C2 ganglion to the brainstem. These findings could explain the referral of pain from the neck to the fronto-ocular region (34, 59). The C2 ganglion itself can be injured in several different ways, including whiplash (55). The C2 dorsal root ganglia occupy most of the neuroforamen height, rendering the C2 ganglion vulnerable to entrapment (60). It can be compressed between the posterior arch of the atlas and the lamina of C2 (61) or entrapped by the atlantoepistrophic ligament (62, 63). Chronic vascular irritation of the C2 nerve root has also been reported to cause cervicogenic headache (64).

In humans, the C2 and C3 pain dermatomes, or dynatome, have been mapped by symptom resolution following surgical root decompression, dysesthesias from electrical stimulation and hypalgesia by anesthetic root block. Poletti (65) describes the C2 dynatome 6-8 cm wide ascending paramedially from the subocciput to the vertex. The C3

dynatome includes the ear, pinna, lateral cheek and angle of the jaw as well as the anterolateral upper neck. The C2 and C3 dermatomes appear to overlap these dynatomes, which are also smaller.

Previous clinical and morphologic studies have not proven the existence of an entrapment mechanism in cases of unilateral head pain. Significant inter-individual and intra-individual variability has been noted in the greater occipital nerve. Becser et al (66) could not identify any anatomic structures that posed imminent risk to greater occipital nerve entrapment. Possible compression of the greater occipital nerve was found in 27% of cadavers with no known history of headache. This casts doubt on greater occipital nerve entrapment since it seems to exist in the absence of headaches (56). Furthermore, greater occipital nerve entrapment has largely been discredited by Bogduk (67). His cadaveric dissections demonstrated that the greater occipital nerve pierces the semispinalis and innervates the scalp as it passes under an aponeurotic sling formed by the trapezius and sternocleidomastoid. Contraction of the trapezius muscle actually increases the diameter of the tunnel through which the greater occipital nerve travels. This makes the claim of compression of the greater occipital nerve by trapezius spasm (57, 58) highly unlikely. Indeed, a plethora of literature exists suggesting cervicogenic headache may result from more than just the greater occipital nerve.

Another of the proposed causes of cervicogenic headache is pain from the C2-C3 zygapophysial joint. In a study of normal volunteers, Dwyer et al (68) evoked pain patterns by distending the cervical zygapophysial joint capsules. They found that the C2-C3 zygapophysial joint could cause unilateral pain in the occiput and upper cervical spine and were able to generate typical composite pain patterns from each joint tested. Dreyfus et al (69), using a similar technique, also demonstrated that pain from the atlanto-occipital and atlanto-axial joints is referred to the upper cervical and occipital region unilaterality. The pain referral patterns of the atlanto-axial joints were more consistently localized than the atlanto-occipital joints.

The C2-C3 zygapophysial joint is innervated by the third occipital nerve. Bogduk and Marsland (30) reported subjects with suspected cervical headache were found to suffer from pain mediated by the third occipital nerve. Lord et al (12) subsequently reported the prevalence of third occipital neuralgia at 27% among 100 whiplash patients, and among those with dominant headache, the prevalence was as high as 53%. Using comparative local anesthetic blocks, a prevalence of 50-53% of patients with dominant headaches had pain stemming from the C2-C3 zygapophysial joint. If the C2-C3 zygapophysial joint was not the pain generator, placebo-controlled blocks identified the prevalence of lower cervical zygapophysial joint mediated pain to be 49%. The overall prevalence of cervical zygapophysial joint mediated pain was 60% (12, 70). Those with positive diagnostic injections were significantly more likely to be tender of the C2-C3 zygapophysial joints (50).

Not only have cervical zygapophysial joints been implicated in cervicogenic headache, cervical discogenic disease as a pain generator of cervicogenic headache must also be considered. Schellhas et al (71) correlated MRI findings and cervical discography in both asymptomatic and chronic neck pain sufferers. He found evidence of painless annular tears by cervical discography in 17 of 20 discs deemed normal by MRI. He also described the pain pattern. The pain distribution from provoking the C2-3 disc occurred consistently further cephalad in the occiput than pain from the C3-4 disc, which was located in the lower occipital region and upper posterior neck. Considerable overlap of pain distribution was noted between C2-3 and C3-4. These findings were consistent with the pain patterns subsequently described by Grubb and Kelly (72) in 173 cervical discograms over a 12-year period. Among patients with headache, discography at the C3-C4 disc and lower never provoked headaches. Similar results were noted in a prospective multi-center study in which referred pain patterns were mapped during cervical discography. The results showed, in order of frequency, that the C2-C3, C3-C4 and C4-C5 discs commonly referred pain to the head. No disc below C4-C5 ever referred pain to the head (73). It should be noted that 50% of positive concordant provocative discs caused unilateral symptoms.

Grubb and Kelly (72) also described the proportion of examined discs with concordant pain to be slightly more unilateral than bilateral. A midline structure causing unilateral pain is not altogether surprising. In the lumbar spine, Slipman et al (74) found that the side of pain did not correlate to the side of the annular tear. That is, unilateral pain in the lumbar spine also occurs from discogenic disease. While no correlation between the side of pain and the side of the annular tear has been reported in the cervical spine, Slipman et al's (74) findings at least suggest the possibility that a unilateral headache could occur from the midline disc.

DIAGNOSIS

Determining the etiology of cervicogenic headaches is often frustrating for both the patient and physician. Differentiating between cervicogenic headache and other forms of headache is difficult because not only is there variability in headache presentation, but there is also considerable symptom overlap. Many authors have suggested varying methods to diagnose and differentiate cervicogenic headache from other various forms of headaches. Use of clinical criteria has been strongly advocated (3, 13, 75). Significant variations in the definition of cervicogenic headache among clinicians have clouded not only the incidence and prevalence of cervicogenic headache, but also its treatment recommendations and outcomes (22).

Other diagnostic tools have also been proposed. Since significant pain pressure threshold differences were noted in patients with cervicogenic headache, migraine, tension headaches and controls, the use of pressure algometers has been advocated (76, 77). Fredriksen et al (78) has advocated for pupillometric testing with mydriatic agents to differentiate cervicogenic headache from cluster headaches. He notes fundamental autonomic differences between cluster headaches and cervicogenic headache. The skin roll test has also been proposed since asymmetrical tenderness in the upper trapezius can be found in cervicogenic headache. However, because trapezius tenderness can also be found in migraine and tension headache groups, the skin roll test is felt to contribute little to making the diagnosis of cervicogenic headache (79).

Given these limitations in diagnosing cervicogenic headache, it is evident that a more reliable and accurate means of establishing this condition is needed. Indeed, in the absence of clear clinical findings and corroborating imaging, diagnostic injections have a role in not only differentiating between various types of headaches, but also identifying the pain generator.

Refinements of Sjaastad's criteria have since been published, emphasizing the use of diagnostic nerve blocks in patients with cervicogenic headache as important confirmatory evidence (80). Patients selected based on their adherence to two fundamental criteria: (i) side-locked unilaterally of pain; and (ii) pain starting in the neck and spreading to the fronto-orbital area may better discriminate between cervicogenic headache and other types of headache (Table 1). These simple criteria can contribute to a preliminary identification of possible cervicogenic headache cases that may then undergo a sequence of clinical and minimally invasive procedures to confirm the diagnosis. These criteria may also be used to localize the level(s) of dysfunction in the cervical spine, which may be the target for therapeutic investigations, whether invasive or non-invasive (81).

Diagnostic greater occipital nerve and even lesser occipital nerve injections have been recommended to diagnose and treat cervicogenic headache. From a theoretical standpoint, a C2 nerve block should be able to alleviate pain in all patients who responded to greater occipital nerve blocks. The C2 blockade should anesthetize the dorsal rami of C2 greater occipital nerve as well as the ventral rami of C2 (a portion of the lesser occipital nerve). In a small study, Bovim et al (82) found that if the simpler greater occipital nerve block was effective, a C2 blockade may not be necessary. However, if the greater occipital nerve block had no response, they recommended a C2 blockade (82).

Because no strong correlation could be made for findings on physical examination and cervicogenic headache, many authors recommend using the accepted diagnostic criteria and successful anesthetic blockade of the C2 root and ganglion. No specific prognostic factors could be established (83, 84). Some have concluded that greater occipital nerve and/or lesser occipital nerve blockades will help distinguish chronic paroxysmal hemicrania, hemicrania continua, migraine and tension-type headaches from cervicogenic headache with only the latter responding to greater occipital nerve blocks (84, 85).

Because third occipital headache may be indistinguishable clinically from tension or other forms of headache, third occipital nerve blocks have also been advocated as means of establishing this largely unrecognized diagnosis (30, 84). Use of the double-block paradigm for the third occipital nerve blocks are essential to make this diagnosis (86). The use of a comparative double-block paradigm is believed to minimize the placebo effect and is frequently employed during the work-up of potential joint mediated pain (87, 88).

The classic double block paradigm uses an inert injectate or a sham procedure as the control. In this model, two injections on separate occasions are necessary. One injection is with a local anesthetic; the control is typically normal saline. A true neurogenic pain generator should respond to the local anesthetic. However, one must be cautious about a response to normal saline when performed intra-articularly. The saline may dilute or wash chemical inflammatory mediators, which may alleviate pain. Small

volumes of injectate are typically used to avoid a volume effect.

Some authors have even advocated a triple block paradigm to eliminate false positive responses and reconfirm the pain generator (89). In this protocol, an initial injection of local anesthetic is used to identify a potential pain generator. Two subsequent injections are then injected on separate occasions. One injection is normal saline and the other is local anesthetic. The patient is blinded to which injectate used. A true positive result occurs when local anesthetic provides pain relief and the normal saline does not. Due to ethical and logistic considerations, many clinicians instead use two local anesthetics that offer different duration of effect (89). The patient receives two injections, both of which temporarily alleviate pain. This method requires two comparative positive responses. A short acting anesthetic should provide pain relief of approximately one hour or less if the pain generator is correctly identified. Subsequently, pain relief of longer duration caused by an injection of a longer acting local anesthetic confirms the result.

NON-OPERATIVE TREATMENT

Successful treatment of cervicogenic headache is entirely dependent on the accuracy and specificity of the etiologic diagnosis. As noted above, many potential pain generators have been implicated in cervicogenic headache. Likewise, numerous treatments have been advocated (90). Perhaps the greatest problem with defining and comparing successful treatments and outcomes is the method by which cervicogenic headache was diagnosed. Depending on whether cervicogenic headache was diagnosed by clinical criteria, manual techniques, systematic diagnostic injections or a combination thereof, treatment strategies and outcomes will differ greatly (30, 91-93).

Some proposed treatment options have been shown to have limited efficacy such as intracutaneous sterile water injections (94). Responses to treatments for other forms of headaches, such as nitroglycerin, oxygen, ergotamines and morphine, have had limited efficacy in treating cervicogenic headache (95). Epidural corticosteroid injections in cervicogenic headache have also been endorsed (96, 97). The immediate (12 hours) and medium-term (4 - 8 weeks) results noted significant clinical improvement compared to a control group with tension type headaches (96, 97). However, long-term results of cervical epidural steroid injections failed to demonstrate statistically significant findings in cervicogenic headache population (98).

Botulinum toxin A (Botox) has also been recommended in patients with muscularly induced cervicogenic headache. Botox produces dose dependent prolonged muscle relaxation and can be easily targeted to the involved muscles. Early in this treatment paradigm, the Botox group showed a significant improvement in pain and cervical range of motion whereas the placebo control group demonstrated no statistically significant changes at any time (99, 100).

Another commonly recommended treatment option in the literature is cervical spine manipulation. Cervical spine manipulation, and its benefits and inherent risks, have been described (101-108). There is some literature to suggest that cervical spine manipulation and mobilization can provide short-term relief for some patients with neck pain and headaches. However, spinal manipulation has also been shown to provide short-term relief for patients with tension-type headache (109, 110) as well as those with post-traumatic headaches (104). A case report also suggested treating chronic post-traumatic cervicogenic headache with supine cervical traction and exercise (111).

Many others have written about the utility of and personal experience with occipital nerve blocks (112-114). The pain reduction after greater occipital nerve blockade was noted to be significantly more marked in the cervicogenic headache group than for other headache categories. Moreover, pain reduction in the forehead was generally only found in the cervicogenic headache patients (77%). Peri-orbital pain reduction also was significantly more marked following the greater occipital than the supra-orbital nerve blockade (116).

In a study by Anthony (8), corticosteroid injections into the region of the greater occipital nerve and lesser occipital nerve produced complete relief of headache in 169 out of 180 patients with cervicogenic headache. However, similar results were also achieved in patients with unilateral migraine or cluster headache attacks. They suggested that steroid injections may have an effect at the cervicotrigeminal relay, which can arrest other forms of unilateral headache.

Slipman et al (116) reported the results following C2-C3 corticosteroid zygapophysial injections with a positive response to a single diagnostic C2-C3 injection. In this post-traumatic (whiplash) population with symptom duration averaging 34 months, 61% had good or excellent results. The number of headache days, headache duration, headache intensity, disability indices and narcotic medication usage all had significant statistical and clinical improve-

ment.

The use of radiofrequency neurotomy procedures to relieve cervicogenic headache has been described (117-120). It has been shown that local anesthetic injections around the medial branch of the dorsal ramus innervating a painful cervical zygapophysial joint can adequately anesthetize pain emanating from that joint (121). Therefore, radiofrequency neurotomy of the superficial medial branch of the C3 sinuvertebral nerve, which innervates the C2-C3 joint for more long-lasting denervation has been utilized. Likewise, ablation of the C3 and C4 medial branches denervates the C3-C4 joint. In the treatment of chronic neck pain, long-term efficacy of percutaneous radiofrequency medial branch neurotomy has been demonstrated. In selected patients, using controlled diagnostic blocks, complete relief of pain was obtained in 71% of patients after an initial procedure. Success was also seen with repeat radiofrequency neurotomy if pain recurs (122). In a placebo study using triple-block paradigm, Lord et al (123) was able to show clinical efficacy of radiofrequency neurotomy for chronic cervical zygapophysial pain from C3-C4 to C6-C7. C2-C3 zygapophysial joint mediated pain was excluded because of technical difficulties. This population, however, was not specific for headache sufferers. Subsequently, an unblinded non-randomized trial in patients with cervicogenic headache demonstrated both short-term (8 weeks) and long-term (mean 16.8 months) clinical efficacy of radiofrequency cervical zygapophysial joint neurotomy. These patients reported decreased pain level, decreased number of days per week with headache and decreased oral analgesic usage (117, 118).

Radiofrequency neurotomy of the sinuvertebral nerves to the upper cervical discs has also been described to treat cervicogenic headache (119). In this technique, provocative discography is used to appropriately identify discogenic causes of cervicogenic headache. The C2-C3 and C3-C4 discs are the typical offenders. Radiofrequency neurotomy to the outer layers of the C3 or C4 nerve roots, including sympathetic nerve fibers from the nearby sympathetic chain, has been reported with considerable success in eliminating or significantly reducing cervicogenic headache (120).

Successful outcomes using radiofrequency neurotomy to the greater occipital nerve, the C2 medial rami, and the cervical discs has also been reported following appropriate diagnostic injection work-up (120). Radiofrequency neurotomy to the periosteum of the occipital bone (planum nuchale) is yet another location for treatment. Radiofrequency neurotomy to the symptomatic side demonstrated 71% improvement at 4.5-year follow-up (124).

CONCLUSION

Cervicogenic headache should be considered a descriptive term rather than a final diagnosis. Because of the numerous potential pain generators, neither uniform clinical findings nor a pathophysiology has been defined for the entity known as cervicogenic headache. There is considerable overlap of cervicogenic headache symptoms with other headache diagnoses. Several potential nociceptive sources may converge into a final common neuroanatomic pathway, which is manifested by headache symptoms. A high index of suspicion, together with Sjaastad et al's (124) criteria, should be used to suggest the possibility of cervicogenic headache. Sequential diagnostic injections may elucidate pain generators and differentiate it from other types of headaches. Only when a precise nociceptive source is identified can a focused and comprehensive treatment regimen be implemented to attend to this prevalent, debilitating, yet often elusive and under-recognized diagnosis.

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