Cervicogenic Headache: A Review of Diagnostic and Treatment Strategies

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Cervicogenic headache is a syndrome characterized by chronic hemicranial pain that is referred to the head from either bony structures or soft tissues of the neck. The trigeminocervical nucleus is a region of the upper cervical spinal cord where sensory nerve fibers in the descending tract of the trigeminal nerve (trigeminal nucleus caudalis) are believed to interact with sensory fibers from the upper cervical roots. This functional convergence of upper cervical and trigeminal sensory pathways allows the bidirectional referral of painful sensations between the neck and trigeminal sensory receptive fields of the face and head. A functional convergence of sensorimotor fibers in the spinal accessory nerve (CN XI) and upper cervical nerve roots ultimately converge with the descending tract of the trigeminal nerve and might also be responsible for the referral of cervical pain to the head.

Diagnostic criteria have been established for cervicogenic headache, but its presenting characteristics occasionally may be difficult to distinguish from primary headache disorders such as migraine, tension-type headache, or hemicrania continua.

This article reviews the clinical presentation of cervicogenic headache, proposed diagnostic criteria, pathophysiologic mechanisms, and methods of diagnostic evaluation. Guidelines for developing a successful multidisciplinary pain management program using medication, physical therapy, osteopathic manipulative treatment, other nonpharmacologic modes of treatment, and anesthetic interventions are presented.

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Dr Biondi has a financial interest arrangement or affiliation with the following: Allergan Inc; AstraZeneca; Elan Pharmaceuticals, Inc; GlaxoSmithKline; Merck & Co, Inc; Pfizer Inc; MedPointe Pharmaceuticals; OrthoMcNeil Pharmaceutical, Inc; and Endo Pharmaceuticals.

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Neck pain and cervical muscle tenderness are common and prominent symptoms of primary headache disorders.[1] Less commonly, head pain may actually arise from bony structures or soft tissues of the neck, a condition known as *cervicogenic headache*.[2] Cervicogenic headache can be a perplexing pain disorder that is refractory to treatment if it is not recognized. The condition's pathophysiology and source of pain have been debated,[3-5] but the pain is likely referred from one or more muscular, neurogenic, osseous, articular, or vascular structures in the neck.[6]

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Neck Pain as a Manifestation of Migraine

Neck pain and muscle tension are common symptoms of a migraine attack.[1,7-9] In a study of 50 patients with migraine, 64% reported neck pain or stiffness associated with their migraine attack, with 31% experiencing neck symptoms during the prodrome; 93%, during the headache phase; and 31%, during the recovery phase.[1] In the study by Blau and MacGregor,[1] 7 patients reported that pain was referred into the ipsilateral shoulder and 1 patient reported that pain extended from the neck into the low back region.

In another study of 144 migraine patients from a university-based headache clinic, 75% of patients reported neck pain associated with migraine attacks.[8] Of these patients, 69% described their pain as "tightness", 17% reported "stiffness" and 5% reported "throbbing." The neck pain was unilateral in 57% of respondents, 98% of whom reported that it occurred ipsilateral to the side of headache. The neck pain occurred during the prodrome in 61%; the acute headache phase, in 92%; and the recovery phase, in 41%.

Recurrent, unilateral neck pain without headache is reported as a variant

This continuing medical education publication supported by an unrestricted educational grant from Merck & Co, Inc

Checklist

MAJOR CRITERIA

■ **Point I—Symptoms and Signs of Neck Involvement**
(*listed in a surmised sequence of importance; obligatory that one or more of phenomena are present*)

☐ Precipitation of head pain, similar to the usually occurring (*suffices as the sole criterion for positivity*)*:
— by neck movement and/or sustained awkward head positioning (suffices as the sole criterion for positivity within group, and/or:
— by external pressure over the upper cervical or occipital region on the symptomatic side

(*Provisionally, the combination of the following two points has been set forth as a satisfactory combination within Point 1*)

☐ Restriction of the range of motion (ROM) in the neck*
☐ Ipsilateral neck, shoulder, or arm pain of a rather vague nonradicular nature or, occasionally, arm pain of a radicular nature*

■ **Point II—Confirmatory Evidence by Diagnostic Anesthetic Blockades**

(*This is an obligatory point in scientific works.*)

■ **Point III—Unilaterality of the Head Pain, Without Sideshift**

(*For scientific work, Point III should preferably be adhered to.*)

HEAD PAIN CHARACTERISTICS

■ **Point IV**

(*None of the following points is obligatory*)

☐ Moderate to severe, nonthrobbing, and nonlancinating pain, usually starting in the neck
☐ Episodes of varying duration, or
☐ Fluctuating, continuous pain

OTHER CHARACTERISTICS OF SOME IMPORTANCE

■ **Point V**

(*None of the following points is obligatory*)

☐ Only marginal effect or lack of effect of indomethacin
☐ Only marginal effect or lack of effect of ergotamine and sumatriptan succinate
(c) female sex
(d) not infrequent occurrence of head or indirect neck trauma by history, usually of more than only medium severity

OTHER FEATURES OF LESSER IMPORTANCE

■ **Point VI**
☐ **Various attack-related phenomena, only occasionally present:**
— nausea
— phonophobia and photophobia
— dizziness
— ipsilateral "blurred vision"
— difficulties on swallowing
— ipsilateral edema, mostly in the periocular area

*The presence of all three points indicated with asterisk fortifies the diagnosis (but still Point II is an additional obligatory point for scientific work).

Figure 1. *The Cervicogenic Headache International Study Group Diagnostic Criteria. (Modified from Biondi DM: Cervicogenic headache: mechanisms, evaluation, and treatment strategies.* J Am Osteopath Assoc. *2000;100(9 Suppl):S7-14. Source: Sjaastad 0, Fredriksen TA, Pfaffenrath V. Cervicogenic headache: diagnostic criteria.* Headache. 1998;38:442-445.)

of migraine.[10] Careful history gathering in cases of recurrent neck pain discovered that previously overlooked symptoms were either similar or identical to those associated with migraine.

Differences in neck posture, pronounced levels of muscle tenderness, and the presence of myofascial trigger points were observed in subjects with migraine, tension-type headache, or a combination of both, but not in a nonheadache control group.[1,11,12] A comparison of the headache groups demonstrated no significant differences in myofascial symptoms or signs, dispelling the common belief that tension-type headache is associated with a greater degree of musculoskeletal involvement than migraine.[12]

Headache as a Manifestation of Neck Disorders

Head pain that is referred from the bony structures or soft tissues of the neck is commonly called "cervicogenic headache." It is often a sequela of head or neck injury but may also occur in the absence of trauma. The clinical features of cervicogenic headache may mimic those commonly associated with primary headache disorders such as tension-type headache, migraine, or hemicrania continua, and as a result, distinguishing among these headache types can be difficult.

The prevalence of cervicogenic headache in the general population is estimated to be between 0.4% and 2.5%, but in pain management clinics, the prevalence is as high as 20% of patients with chronic headache.[13] The mean age of patients with this condition is 42.9 years, and cervicogenic headache is four times more prevalent in women. Patients with cervicogenic headache have demonstrated substantial declines in quality of life measurements that are similar to those in patients with migraine and tension-type headache when compared with control subjects, but they demonstrate the greatest loss in domains of physical functioning when compared with the groups with other headache disorders.[14]

The Cervicogenic Headache International Study Group developed diagnostic criteria that have provided a detailed, clinically useful description of the condition (*Figure 1*).[15] The diagnosis

Figure 2. *Clinical characteristics of cervicogenic headache. (Modified from Biondi DM: Cervicogenic headache: mechanisms, evaluation, and treatment strategies.* J Am Osteopath Assoc. *2000;100(9 Suppl):S7-14.)* ▶

of cervicogenic headache can often be made without resorting to diagnostic neural blockade by completion of a careful history and physical examination (*Figure 2*).

Diagnostic Testing for Suspected Cervicogenic Headache

Patients with cervicogenic headache will often have altered neck posture or restricted cervical range of motion.[16] The head pain can be triggered or reproduced by active neck movement, passive neck positioning especially in extension or extension with rotation toward the side of pain, or on applying digital pressure to the involved facet regions or over the ipsilateral greater occipital nerve. Muscular trigger points are usually found in the suboccipital, cervical, and shoulder musculature, and these trigger points can also refer pain to the head when manually or physically stimulated. There are no neurologic findings of cervical radiculopathy, though the patient might report scalp paresthesia or dysesthesia.

Diagnostic imaging such as radiography, magnetic resonance imaging (MRI), and computed tomography (CT) myelography cannot confirm the diagnosis of cervicogenic headache but can lend support to its diagnosis.[17] One study reported no demonstrable differences in the appearance of cervical spine structures on MRI scans when 24 patients with clinical features of cervicogenic headache were compared with 20 control subjects.[18] Cervical disc bulging was reported equally in both groups (45.5% vs 45.0%, respectively).

A comprehensive history, review of systems, and physical examination including a complete neurologic assessment will often identify the potential for an underlying structural disorder or systemic disease.[19] Imaging is then primarily used to search for suspected secondary causes of pain that may require surgery or other more aggressive forms of treatment.[20] The differential diagnosis in cases of suspected cervicogenic headache could include posterior fossa tumor, Arnold-Chiari malformation, cervical spondylosis or arthropathy, herniated intervertebral disc, spinal nerve compression or tumor, arteriovenous malformation, vertebral artery dissection, and intramedullary or extramedullary spinal tumors.

A laboratory evaluation may be necessary to search for systemic diseases that may adversely affect muscles, bones, or joints (ie, rheumatoid arthritis, systemic lupus erythematosus, thyroid or parathyroid disorders, primary muscle disease, etc).

Checklist

- ☐ Unilateral head or face pain without sideshift; the pain may occasionally be bilateral
- ☐ Pain localized to the occipital, frontal, temporal or orbital regions
- ☐ Moderate to severe pain intensity
- ☐ Intermittent attacks of pain lasting hours to days, constant pain or constant pain with superimposed attacks of pain
- ☐ Pain is generally deep and nonthrobbing; throbbing may occur when migraine attacks are superimposed
- ☐ Head pain is triggered by neck movement, sustained or awkward neck postures; digital pressure to the suboccipital, C2, C3, or C4 regions or over the greater occipital nerve; valsalva, cough or sneeze might also trigger pain
- ☐ Restricted active and passive neck range of motion; neck stiffness
- ☐ Associated signs and symptoms can be similar to typical migraine accompaniments including:
  - — nausea;
  - — vomiting;
  - — photophobia, phonophobia, and dizziness;
  - — others include ipsilateral blurred vision, lacrimation and conjunctival injection or ipsilateral neck, shoulder or arm pain

Zygapophyseal joint, cervical nerve, or medial branch blockade is used to confirm the diagnosis of cervicogenic headache and predict the treatment modalities that will most likely provide the greatest efficacy. The first three cervical spinal nerves and their rami are the primary peripheral nerve structures that can refer pain to the head.

The suboccipital nerve (dorsal ramus of C1) innervates the atlanto-occipital joint; therefore, a pathologic condition or injury affecting this joint is a potential source for head pain that is referred to the occipital region.

The C2 spinal nerve and its dorsal root ganglion have a close proximity to the lateral capsule of the atlantoaxial (C1–2) zygapophyseal joint and innervate the atlantoaxial and C2–3 zygapophyseal joints; therefore, trauma to or pathologic changes around these joints can be a source of referred head pain. Neuralgia of C2 is typically described as a deep or dull pain that usually radiates from the occipital to parietal, temporal, frontal, and periorbital regions. A paroxysmal sharp or shocklike pain is often superimposed over the constant pain. Ipsilateral eye lacrimation and conjunctival injection are common associated signs. Arterial or venous compression of the C2 spinal nerve or its dorsal root ganglion has been suggested as a cause for C2 neuralgia in some cases.[11,20-23] The third occipital nerve (dorsal ramus C3) has a close anatomic proximity to and innervates the C2–3 zygapophyseal joint. This joint and the third occipital nerve appear most vulnerable to trauma from acceleration-deceleration ("whiplash") injuries of the neck.[24] Pain from the C2–3 zygapophyseal joint is referred to the occipital region but is also referred to the frontotemporal and periorbital regions. Injury to this region is a common cause of cervicogenic headache. The majority of cervicogenic headaches occurring after whiplash resolve within a year of the trauma.[25]

Of interest are reports that patients with chronic headache had experienced substantial pain relief after diskectomy at spinal levels as low as C5–6.[26,27]

Diagnostic anesthetic blockade for the evaluation of cervicogenic headache can be directed to several anatomic struc-

tures such as the greater occipital nerve (dorsal ramus C2), lesser occipital nerve, atlanto-occipital joint, atlantoaxial joint, C2 or C3 spinal nerve, third occipital nerve (dorsal ramus C3), zygapophyseal joint(s) or intervertebral discs based on the clinical characteristics of the pain and findings of the physical examination.[28] Fluoroscopic or interventional MRI-guided blockade may be necessary to assure accurate and specific localization of the pain source.[29-31]

Occipital neuralgia is a specific pain disorder characterized by pain that is isolated to sensory fields of the greater or lesser occipital nerves.[32] The classic description of occipital neuralgia includes the presence of constant deep or burning pain with superimposed paroxysms of shooting or shocklike pain. Paresthesia and numbness over the occipital scalp are usually present. It is often difficult to determine the true source of pain in this condition. In its classic description, the pain of occipital neuralgia is believed to arise from trauma to or entrapment of the occipital nerve within the neck or scalp, but the pain may also arise from the C2 spinal root, C1–2, or C2–3 zygapophyseal joints or pathologic change within the posterior cranial fossa.

Occipital nerve blockade, as it is typically done in the clinic setting, often results in a nonspecific regional blockade rather than a specific nerve blockade and might result in a misidentification of the occipital nerve as the source of pain. This "false localization" might lead to unnecessary interventions aimed at the occipital nerve, such as surgical transection or other neurolytic procedures.[5]

A regional myofascial pain syndrome (MPS) affecting cervical, pericranial, or masticatory muscles can be associated with referred head pain. Sensory afferent nerve fibers from upper cervical regions have been observed to enter the spinal column by way of the spinal accessory nerve before entering the dorsal spinal cord.[33,34] The close association of sensorimotor fibers of the spinal accessory nerve with the spinal sensory nerves is believed to allow for a functional exchange of somatosensory, proprioceptive, and nociceptive information from the trapezius, sternocleidomastoid, and other cervical muscles to converge in the trigeminocervical nucleus and ultimately resulting in the referral of pain to trigeminal sensory fields of the head and face.

Muscular trigger points, a hallmark of MPS, are discreet hyperirritable regions of contracted muscle that have a lowered pain threshold and refer pain to distant sites in predictable and reproducible patterns.[35,36] Anesthetic injections into trigger point regions can assist in the diagnostic evaluation and therapeutic management of referred head or face pain from cervical muscular sources.[35]

Checklist

Pharmacologic

(*None of the listed medications are given an indication for this condition by the US Food and Drug Administration [FDA]*)

- ☐ Tricyclic antidepressants (amitriptyline hydrochloride, nortriptyline hydrochloride, doxepin hydrochloride, desipramine hydrochloride, and others)
- ☐ Antiepileptic drugs (gabapentin, carbamazepine, topiramate, divalproex sodium, and others)
- ☐ Muscle relaxants (tizanidine hydrochloride, baclofen, cyclobenzaprine hydrochloride, metaxalone, and others)
- ☐ Nonsteroidal, anti-inflammatory drugs
  - — nonselective cyclooxygenase (COX) inhibitors (indomethacin, ibuprofen, naproxen, and others)
  - — COX-2 selective inhibitor (celecoxib)

Nonpharmacologic

- ☐ Osteopathic manipulative treatment or manual modes of therapy
- ☐ Physical therapy
- ☐ Transcutaneous electrical nerve stimulation (TENS)
- ☐ Biofeedback/relaxation therapy
- ☐ Individual psychotherapy

Interventional

- ☐ Anesthetic blockade
  - — spinal roots, nerves, rami, or branches
  - — muscular trigger points
- ☐ Neurolytic procedure
  - — radiofrequency thermal neurolysis
- ☐ Botulinum toxin injections (*not given an indication for this condition by the FDA*)
- ☐ Occipital nerve stimulator

Surgical

- ☐ Neurectomy
- ☐ Dorsal rhizotomy
- ☐ Microvascular decompression
- ☐ Nerve exploration and "release"
- ☐ Joint fusion

Figure 3. *Potential treatment interventions for cervicogenic headache. (Modified from Biondi DM: Cervicogenic headache: mechanisms, evaluation, and treatment strategies.* J Am Osteopath Assoc. *2000;100(9 Suppl):S7-14.)*

Treatment of Cervicogenic Headache

The successful treatment of cervicogenic headache usually requires a multifaceted approach using pharmacologic, nonpharmacologic, manipulative, anesthetic, and occasionally surgical interventions[37] (*Figure 3*). Medications alone are often ineffective or provide only modest benefit for this condition.

Anesthetic injections can temporarily reduce pain intensity but have their greatest benefit by allowing greater participation in physical treatment modalities. The success of diagnostic cervical spinal nerve, medial branch, or zygapophyseal joint blockade can predict response to radiofrequency thermal neurolysis.[38] Developing an individualized treatment plan enhances successful outcomes.

Pharmacologic Treatment

Pharmacologic treatment modalities for cervicogenic headache include many medications that are used for the preventive or palliative management of tension-type headache, migraine, and "neuropathic" pain syndromes. The listed medications have neither been approved by the US Food and Drug Administration (FDA) nor rigorously studied in controlled clinical trials for the treatment of cervicogenic headache and are only suggested as potential treatments based on the anecdotal experiences of clinicians who treat this condition or similar pain disorders. The side effects and laboratory monitoring guidelines provided are not intended to be comprehensive, and consultation of standard references or product package inserts are recom-

mended before prescribing any of these medications.

Many patients with cervicogenic headache overuse or become dependent on analgesics. Medication when used as the only mode of treatment for cervicogenic headache does not generally provide substantial pain relief in most cases. Despite this observation, the judicious use of medications can provide enough pain relief to allow greater patient participation in a physical therapy and rehabilitation program. To improve compliance, medications are initially prescribed at a low dose and increased over 4 to 8 weeks as necessary and tolerated.

The cautious combining of medications from different drug classes or with complementary pharmacologic mechanisms may provide greater efficacy than using individual drugs alone (eg, an antiepileptic drug combined with a tricyclic antidepressant [TCA]). Frequent follow-up visits for medication dosage adjustments, monitoring of serum drug levels, and evidence of medication toxicity are recommended.

■ **Antidepressants**—The TCAs have long been used for management of various neuropathic, musculoskeletal, head, and face pain syndromes. Analgesic dosages are typically lower than those required for the treatment of patients with depression. The serotonin and norepinephrine reuptake inhibitors (SNRIs) such as venlafaxine hydrochloride and duloxetine hydrochloride have been anecdotally observed helpful in the prophylactic management of migraine. Similar observations have been reported for venlafaxine in the treatment of painful diabetic neuropathy, fibromyalgia, and regional myofascial pain syndromes, while duloxetine is indicated for the management of painful diabetic neuropathy.

The selective serotonin reuptake inhibitors (SSRIs) are generally ineffective for pain control.

■ **Antiepileptic Drugs**—The antiepileptic drugs (AEDs) are believed to be modulators or stabilizers of peripheral and central pain transmission and are commonly used for the management of neuropathic, head, and face pain syndromes. Divalproex sodium is indicated for the preventive management of migraine headache and may be effective for cluster headaches as well as other neurogenic pain syndromes. Serum drug levels can be used as a therapeutic dosing guide. Monthly monitoring of liver transaminase levels and of complete blood cell (CBC) counts for evidence of toxicity is recommended, especially during the first 3 to 4 months of treatment or whenever dosages are escalated.

Gabapentin is indicated for the management of postherpetic neuralgia and has been used for management of other neuropathic pain syndromes and migraine. No specific laboratory monitoring is usually necessary.

Topiramate is indicated for migraine prophylaxis and has been anecdotally reported effective in the management of painful diabetic neuropathy and cluster headache. Intermittent monitoring of serum electrolyte levels might be needed because of this medication's diuretic effect through carbonic anhydrase inhibition.

Carbamazepine is an effective medication in the treatment of patients with trigeminal neuralgia and central neuropathic pain. Serum drug levels can be used as a therapeutic dosing guide. Monthly monitoring of liver transaminase levels and of CBC counts is recommended, especially during the first 3 to 4 months of treatment or whenever dosages are increased.

Several of the other newer AEDs might be used when other treatments are ineffective.

■ **Analgesics**—Simple analgesics such as acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs) may be used as regularly scheduled medications for round-the-clock management of chronic pain or as needed for the management of acute pain.

The selective cyclooxyenase-2 (COX-2) antagonist celecoxib might have less gastrointestinal toxicity than nonselective NSAIDs, but renal toxicity after long-term use remains as a concern. Recent reports have linked the long-term use of selective COX-2 antagonists with an increased risk of cardiovascular and cerebrovascular events; therefore, the risk-benefit ratio of their use requires strong consideration. It is recommended that prescribers review the safety information and warnings found in the product package inserts.

Narcotic analgesics are not generally recommended for the long-term management of cervicogenic headache[39] but may be cautiously prescribed for temporary pain relief to expedite the advancement of manual modes of therapy or improve tolerance for anesthetic interventions.

Migraine-specific abortive medications such as ergot derivatives or triptans are not effective for the chronic head pain of cervicogenic headache but may relieve the pain of episodic migraine attacks that can occur in some patients with cervicogenic headache.

■ **Other Medications**—Muscle relaxants, especially those with central activity such as tizanidine hydrochloride and baclofen, may provide some analgesic efficacy. Botulinum toxin, type A injected into pericranial and cervical muscles is a promising treatment for patients with migraine and cervicogenic headache,[37,40,41] but further clinical and scientific study is needed.

Physical and Manual Modes of Therapy

Physical and manual modes of therapy are important therapeutic modalities for the acute rehabilitation of cervicogenic headache.[42] A controlled trial testing the effectiveness of therapeutic exercise and manipulative treatment for cases of cervicogenic headache found that efficacy was not substantially affected by age, gender, or headache chronicity in patients with moderate to severe pain intensity.[43] This finding suggests that all patients with cervicogenic headache could benefit from manual modes of therapy and physical conditioning.

Another study comparing an exercise program with manipulative therapy for cervicogenic headache reported substantial and sustained reductions of headache frequency and intensity that were similar in both treatment groups but with a trend toward greater efficacy when the treatment modalities are combined.[44]

A review of the medical literature suggested that the efficacy of physical treatment modalities for the long-term prevention and control of headaches

appears greatest in patients who are involved in ongoing exercise and physical conditioning programs.[45]

Osteopathic manipulative techniques such as craniosacral, strain-counter strain, and muscle energy techniques are particularly well suited for the management of cervicogenic headache. High velocity, low amplitude manipulation can be carefully used in some patients, though it is not unusual to observe an increase in headache intensity after manual modes of therapy of this type, especially if it is delivered too vigorously. Physical treatment modalities are generally better tolerated when initiated with gentle muscle stretching and manual cervical traction. Therapy can be slowly advanced as tolerated to include strengthening and aerobic conditioning. Using anesthetic blockade and neurolytic procedures for temporary pain relief can enhance the efficacy and advancement of physical modes of therapy.

Psychological and Behavioral Treatment

Psychological and nonpharmacologic interventions such as biofeedback, relaxation, and cognitive-behavioral therapy are important adjunctive treatments in the comprehensive management of pain.[46] Ongoing intensive, individual psychotherapy is often required if the patient with chronic pain has a prominent affective or behavioral component and the pain persists despite aggressive treatment.

Anesthetic Blockade and Neurolysis

Cervical epidural steroid injections may be indicated in patients with multilevel disc or spine degeneration.[47] Greater and lesser occipital nerve blockade may provide temporary, but substantial, pain relief in some cases.[48] A published report suggested that repeated greater occipital nerve blockade provided efficacy similar to repeated blockade of the C2 and C3 nerves.[49] This finding suggests that repeated greater occipital nerve blockade in the office setting is a reasonable treatment option before considering referral for more invasive or more expensive interventions.

Trigger point injections with a local anesthetic may also provide temporary pain relief and relaxation of local muscle spasm. If diagnostic blockade of cervical nerve, medial branch, or zygapophyseal joint blockade is successful in providing substantial, but temporary, pain relief, the treatment algorithm can then proceed to consideration for a longer-acting neurolytic procedure such as radiofrequency thermal neurolysis.[38,50,51]

A course of physical therapy and rehabilitation is recommended after anesthetic blockade and neurolytic procedures to enhance functional restoration and effect a longer-lasting analgesic benefit.

Surgical Treatment

A variety of surgical interventions have been done for presumed cases of cervicogenic headache.[3] Surgical liberation of the occipital nerve from "entrapment" in the trapezius muscle or surrounding connective tissues can provide substantial, but temporary, pain relief in some patients.[52] Similarly, only temporary pain relief is observed after surgical transection of the greater occipital nerve.[52] Intensification of pain or anesthesia dolorosa is a potential adverse outcome that must be seriously considered when contemplating the use of surgical interventions.

There have been preliminary reports of efficacy in reducing headache frequency, intensity, and associated disability in cases of chronic migraine after surgical implantation of occipital or spinal nerve stimulators.[53] Based on pathogenic models of cervicogenic headache, neurostimulation would appear to be a reasonable option for the management of cervicogenic headache, but its safety and efficacy have not yet been determined. Overall, surgical procedures such as neurectomy, dorsal rhizotomy, and microvascular decompression of nerve roots or peripheral nerves are not generally recommended without compelling radiologic evidence for a surgically correctable pathologic condition or a history of refractoriness to all reasonable nonsurgical treatment modalities.

Comment

Cervicogenic headache is a relatively common cause of chronic headache that is often misdiagnosed or unrecognized. Its presenting symptom complex can be similar to that of the more commonly encountered primary headache disorders such as migraine or tension-type headache. Early diagnosis and management by way of a comprehensive, multidisciplinary pain treatment program can significantly decrease the protracted course of costly treatment and disability that is often associated with this challenging pain disorder.

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