



Cervicogenic headache: mechanisms, evaluation, and treatment strategies

DAVID M. BIONDI, DO

Cervicogenic headache is a chronic, hemicranial pain syndrome in which the sensation of pain originates in the cervical spine or soft tissues of the neck and is referred to the head. The trigeminocervical nucleus is a region of the upper cervical spinal cord where sensory nerve fibers in the descending tract of the trigeminal nerve converge with sensory fibers from the upper cervical roots. This convergence of nociceptive pathways allows for the referral of pain signals from the neck to the trigeminal sensory receptive fields of the face and head as well as activation of the trigeminovascular neuroinflammatory cascade, which is generally believed to be one of the important pathophysiologic mechanisms of migraine. Also relevant to this condition is the convergence of sensorimotor fibers of the spinal accessory nerve (CN XI) and upper cervical nerve roots, which ultimately converge with the descending tract of the trigeminal nerve. These connections may be the basis for the well-recognized patterns of referred pain from the trapezius and sternocleidomastoid muscles to the face and head. Diagnostic criteria have been established for cervicogenic headache, but presenting characteristics of this headache type may be difficult to distinguish from migraine, tension-type headache, or paroxysmal hemicrania. This article reviews the clinical presentation of cervicogenic headache, its proposed diagnostic criteria, pathophysiologic mechanisms, and methods of diagnostic evaluation. Guidelines for developing a successful multidisciplinary pain management program using medication, osteopathic manipulative treatment, other non-pharmacologic modes of treatment, and anesthetic interventions are presented.

(Key words: cervicogenic headache, trigeminal nerve, zygapophyseal joints, cervical facet, occipital neuralgia, osteopathic manipulative treatment)

Chronic daily as well as refractory intermittent head or face pain can be a perplexing medical disorder, especially in those cases in which it is not recognized that the pain actually originates in the cervical spine or soft tissues of the neck. Head pain referred from the neck

has been designated cervicogenic headache.¹ It is often a sequela of head or neck injury, but it may just as well occur in the absence of trauma. Cervicogenic headache is a diagnosis that lacks expert consensus with respect to its acceptance as a distinct medical disorder.² Although its existence is generally accepted, the condition's pathophysiology and source of pain are debated.¹⁻⁴ Muscular, neurogenic, osseous, articular, and vascular sources for pain referred to the head have been described in the medical literature.⁵ In clinical practice, referred head pain appears to originate from more than one anatomic source, not only from patient to patient but also in the same patient.

Further confounding the medical prac-

Dr Biondi is Director, Head Pain Division, Michigan Head-Pain & Neurological Institute, Ann Arbor, and an assistant clinical professor of medicine (Neurology), Michigan State University—College of Osteopathic Medicine, East Lansing.

Correspondence to David M. Biondi, DO, Michigan Head-Pain & Neurological Institute, 3120 Professional Dr, Ann Arbor, MI 48104-5199.

E-mail: dbiondi@pol.net

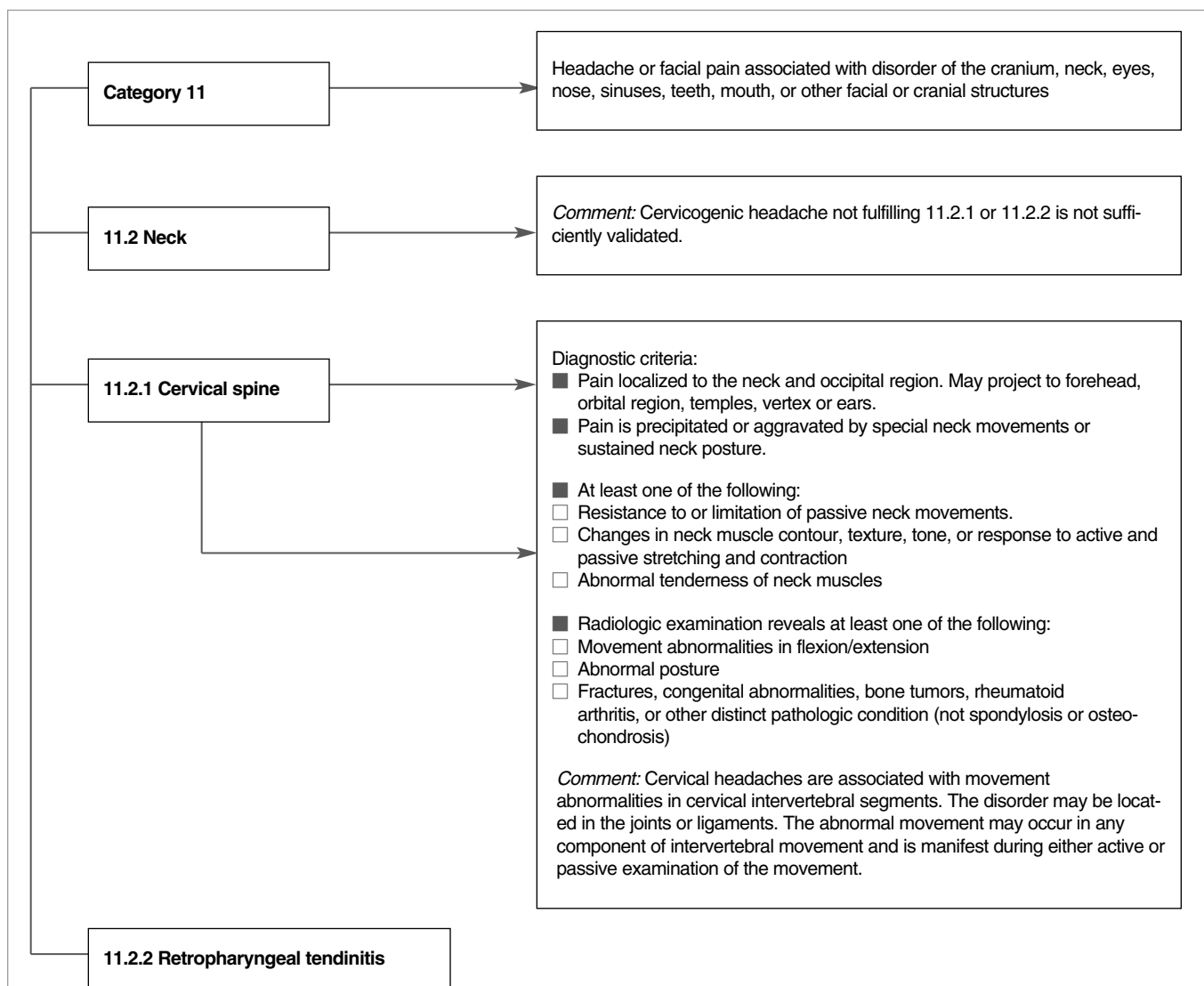


Figure 1. International Headache Society criteria for headache associated with disorder of the neck.⁷

itioner's ability to derive a diagnosis of cervicogenic headache is the similarity of its clinical characteristics to primary headache disorders such as migraine, cluster, benign paroxysmal hemicrania, hemicrania continua, and tension-type headache. Primary headache disorders may present with coexisting neck pain and tenderness as an epiphenomenon of their pathophysiology.⁶ Therefore, neck pain associated with headache is not pathognomonic for the diagnosis of cervicogenic headache.

Diagnostic criteria

Efforts have been made to develop a set of diagnostic criteria for head or face pain associated with disorders of the neck.^{7,8} The development and acceptance of a sin-

gle diagnostic format for cervicogenic headache has been an item of debate. The International Headache Society (IHS) and the Cervicogenic Headache International Study Group⁸ have each proposed diagnostic guidelines that have differed in their specific criteria to define this condition. *Figures 1 and 2* outline and contrast these two sets of proposed guidelines.

The diagnostic criteria of the Cervicogenic Headache International Study Group⁸ provide a more detailed description of the condition in a way that is useful to the clinical practitioner. Radiographic evidence is not required in these criteria, although imaging procedures are necessary on a practical basis to elucidate any underlying pathologic condition that may require surgery or other such aggres-

sive interventions. The need for confirmatory anesthetic blockade is suggested to be obligatory for scientific work. In daily practice, the diagnosis of cervicogenic headache can be applied on clinical grounds by taking a careful history and doing a thorough physical examination (*Figure 3*) without anesthetic blockade, but diagnostic blockade procedures are often necessary because their outcome will help to direct a specific therapeutic course of action.

Differential diagnosis

Several pathologic conditions may present similarly or contribute to structural changes that ultimately cause cervicogenic headache (*Figure 4*).⁹⁻¹³ In clinical practice, a recent or remote history of head or neck



Checklist

Major criteria of cervicogenic headache

■ **Point I**—Symptoms and signs of neck involvement (*in a surmised sequence of importance*):

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

(A combination of the following two symptoms is satisfactory for the diagnosis.)

- ☐ 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, this point should preferably be adhered to.)

Head pain characteristics

■ Point IV

- moderate-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 following single points is obligatory.)

- only marginal effect or lack of effect of indomethacin
- only marginal effect or lack of effect of ergotamine and sumatriptan
- female sex
- 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

(None of following single points is obligatory.)

- ☐ Various attack-related phenomena, only occasionally present:
 - nausea
 - phonophobia and photophobia
 - dizziness
 - ipsilateral “blurred vision”
 - difficulties on swallowing
 - ipsilateral edema, mostly in the periorcular area

Figure 2. Symptoms and signs of neck involvement. Major criteria of cervicogenic headache as set forth by the Cervicogenic Headache International Study Group.⁸

injury is commonly encountered. A comprehensive history, review of systems, and physical examination including a complete neurologic and directed musculoskeletal assessment will most often suggest an underlying structural or systemic disease if present, but diagnostic testing is usually required for confirmation.¹⁴

Occipital neuralgia is a condition that is characterized by pain occurring in the distribution of the greater or lesser occip-

ital nerves.¹⁵ The pain is typically deep or burning with superimposed paroxysms of shooting or lancinating pain. Paresthesia and numbness over the occipital scalp are usually present. In the clinical assessment of this condition, it is difficult to determine the cause or source of pain. The pain may arise from trauma or entrapment of the occipital nerve in the neck or scalp, but its source may be just as likely from “deeper” anatomic structures or tissues



Checklist

- ☐ Unilateral head or face pain without sideshift; the pain may occasionally be bilateral (unilateral on both sides)
- ☐ 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's maneuver, cough, or sneeze may 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

Figure 3. Clinical characteristics of cervicogenic headache.

such as the C2 spinal nerve, C1-2 or C2-3 zygapophyseal joints or pathologic lesion within the posterior cranial fossa.

In general clinical practice, many cases that are given a diagnosis of “occipital neuralgia” are pain syndromes characterized by occipital pain referred from cervical structures or tissues other than



Checklist

- ☐ Posterior fossa tumor
- ☐ Chiari malformation
- ☐ Arteriovenous malformation (intracranial or perispinal)
- ☐ Vasculitis (giant cell arteritis)
- ☐ Vertebral artery dissection
- ☐ Cervical spondylosis or arthropathy
- ☐ Herniated cervical disk
- ☐ Spinal nerve compression or tumor

Figure 4. Selected pathologic conditions in the differential diagnosis of cervicogenic headache.



Checklist

- ☐ Greater occipital nerve (dorsal ramus C2)
- ☐ Atlanto-occipital joint
- ☐ Atlantoaxial joint
- ☐ C2 or C3 spinal nerve
- ☐ Third occipital nerve (dorsal ramus C3)
- ☐ C2-3 zygapophyseal joint
- ☐ Cervical epidural blockade

Figure 5. Diagnostic anesthetic blockade for the evaluation of cervicogenic headache.

the occipital nerve. Occipital nerve blockade, as performed in the typical medical office setting, is a “nonspecific” regional blockade that often results in a “false localization” of the source of pain to the occipital nerve. Greater occipital nerve release, which is the surgical liberation of the occipital nerve from “entrapment” in the trapezius muscle and surrounding connective tissues, can provide relief of pain in up to 75% of cases, but the return of pain within several months is common. Temporary



Checklist

- ☐ Atlanto-occipital joint
- ☐ Lateral atlantoaxial joint (C1-2)
- ☐ Ligaments of the median atlantoaxial joint
- ☐ C2-3 zygapophyseal joint
- ☐ Suboccipital and upper posterior neck muscles
- ☐ Upper prevertebral muscles
- ☐ Upper cervical spinal dura mater
- ☐ Vertebral arteries
- ☐ C2-3 intervertebral disk
- ☐ Trapezius muscle
- ☐ Sternocleidomastoid muscle

Figure 6. Structures and tissues innervated by C1, C2, and C3 spinal nerves.

relief of pain is also observed after transection of the greater occipital nerve (neurectomy).¹⁶ The short duration of pain relief afforded by these surgical procedures may suggest that the source of pain was “deeper” than the “peripheral” surgical site.

The clinical features of cervicogenic headache may overlap or mimic those of primary headache disorders such as migraine, cluster, chronic paroxysmal hemicrania, hemicrania continua, or tension-type headache. One or more clinical characteristics can be used to differentiate each of these headache types from one another, but the distinction may at times be difficult (Table 1).

Diagnostic testing

Cervical nerve or zygapophyseal joint blockade is often required to confirm a diagnosis of cervicogenic headache (Figure 5). Fluoroscopic or interventional magnetic resonance imaging (iMRI)-guided blockade is necessary to assure accurate and specific localization of the source of pain. Trigger point injections can assist in evaluating the presence of pain referred to the head or face from muscular sources.¹⁷ Diagnostic imaging such as radiography, MRI, and computed tomographic myelography cannot confirm the



Checklist

Pharmacologic

- ☐ Tricyclic antidepressants (amitriptyline hydrochloride, nortriptyline hydrochloride, doxepin hydrochloride, and others)
- ☐ Antiepileptic drugs (gabapentin, topiramate, carbamazepine, divalproex sodium)
- ☐ Muscle relaxants (tizanidine hydrochloride, baclofen, cyclobenzaprine hydrochloride, metaxalone, and others)
- ☐ Nonsteroidal, anti-inflammatory drugs

Nonpharmacologic

- ☐ Osteopathic manipulative treatment¹⁹
- ☐ Physical therapy
- ☐ Transcutaneous electrical nerve stimulation (TENS)
- ☐ Biofeedback/relaxation therapy
- ☐ Individual psychotherapy

Interventional

- ☐ Anesthetic blockade
 - spinal roots, nerves, rami, or branches
 - zygapophyseal joints
 - trigger points¹⁷
- ☐ Neurolytic procedures
 - radiofrequency thermal neurolysis
 - cryoneurolysis
- ☐ Botulinum toxin (Botox) injections²⁰

Surgical

- ☐ Neurectomy
- ☐ Dorsal rhizotomy
- ☐ Microvascular decompression
- ☐ Nerve exploration and “release”
- ☐ Zygapophyseal joint fusion

Figure 7. Potential treatment interventions for cervicogenic headache.

presence of cervicogenic headache but can lend support its diagnosis.

Imaging is primarily used to search for secondary causes of pain that may require surgery or other more aggressive forms

Table 1
Characteristics of Several Headache Types

Variable	Cervicogenic Headache	Migraine	Cluster	Hemicrania continua	Tension headache
■ Female to male ratio	F>M to F=M	F>M	M>F	F>M	F>M
■ Laterality	Unilateral without sideshift	Unilateral with sideshift	Unilateral without sideshift	Unilateral without sideshift	Bilateral
■ Location	Occipital to frontoparietal and orbital	Frontal, orbital, temporal, hemicranial	Orbital, temporal	Frontal, temporal, orbital, hemicranial	Frontal, occipital, circumferential
■ Duration	Intermittent or constant with attacks	4 to 72 h	15 to 180 min several per day	Constant with attacks	Days to weeks
■ Triggers	Neck movement, Valsalva's maneuver, pressing over C 1-3	Multiple but neck movement not typical	Alcohol, headaches occur at predictable times of day	None typical	Multiple but neck movement not typical
■ Associated symptoms	Usually absent or similar to migraine but milder, decreased neck movement	Nausea, vomiting, phonophobia and photophobia, visual scotoma	Autonomic symptoms: tearing, rhinorrhea, ptosis, miosis all ipsilateral to pain	Autonomic symptoms may occur	Occasionally decreased appetite, photophobia or phonophobia
■ Treatment	Anesthetic blockade, typical migraine treatment usually not effective; can try AEDs,* TCAs, NSAIDs	Typical migraine treatment, (ie, ergot, triptans, usual preventives)	Oxygen, ergots, triptans, usual preventives	Excellent response to indomethacin	Simple analgesics, muscle relaxants, medications used in migraine treatment

*AEDs = antiepileptic drugs; TCAs = tricyclic antidepressants; NSAIDs = nonsteroidal anti-inflammatory drugs.

of treatment. Imaging procedures to examine the brain, craniocervical junction, and cervical spine are usually recommendable. A laboratory evaluation may be necessary to search for systemic diseases that may adversely affect muscles, bones, or joints.

Pathophysiology

Pain of the head or face referred from a cervical source may originate from one or more pain-sensitive anatomic structures or soft tissues in the neck (Figure 6). The pain most often originates in the upper cervical regions but may also originate from the middle to lower cervical regions. An anatomic basis exists that explains these patterns of referred pain.

The trigeminal nucleus caudalis

descends as low as the C3 or C4 segments of the spinal cord. This nucleus is contiguous with the gray matter of the spinal dorsal horn at these levels. This column of gray matter has been called the *trigeminocervical nucleus*. Interneurons within the trigeminocervical nucleus allow for an exchange of sensory information between the upper cervical spinal nerves and the trigeminal nerve. It is through this exchange of sensory information that nociceptive signals from the anatomic structures and soft tissues of the upper region of the neck can be referred to the sensory receptive fields of the trigeminal nerve in the head and face. The topographic arrangement of the trigeminal nucleus caudalis allows the greatest interchange of nociceptive information with the oph-

thalmic division of the trigeminal nerve (CN V); therefore, it is most common for pain from a cervical source to be referred to the forehead, temple, or orbit. There is also some interchange of sensory signals with the maxillary division of CN V that allows referral of neck pain to the face.

Afferent sensory signals ascend or descend up to three spinal cord segments in the dorsolateral tract and substantia gelatinosa before entering the spinal dorsal horn. This can allow nociceptive signals from spinal segments as low as C6 or C7 the potential to interact with interneurons in the trigeminocervical nucleus, and thereby, the referral of pain from anatomic structures or soft tissues in the middle and lower portion of the neck to the head and face.

Table 2
Pharmacologic Treatment of Cervicogenic Headache

Drug class and representative medications	Daily dosage range	Typical dosing schedule	Comments
■ Tricyclic antidepressants ☐ Amitriptyline hydrochloride, nortriptyline hydrochloride, doxepin hydrochloride	10 mg to 150 mg	At bedtime	Serum drug levels, monitor complete blood cell count, liver function tests, electrocardiogram
■ Antiepileptic drugs ☐ Divalproex sodium ☐ Gabapentin ☐ Topiramate ☐ Carbamazepine	500 mg to 1500 mg 1800 mg to 3600mg 100 mg to 400 mg 300 mg to 1200 mg	Two to three times a day Three to four times a day Twice a day Three times a day (twice a day for extended-release formulations)	Serum drug levels, monitor complete blood cell count, liver function tests Serum drug levels, monitor complete blood cell count, liver function tests
■ Muscle relaxants ☐ Tizanidine hydrochloride ☐ Baclofen ☐ Cyclobenzaprine hydrochloride ☐ Metaxalone	4 mg to 32 mg 30 mg to 60 mg 10 mg to 40 mg 1200 mg to 2400 mg	Three times a day Three times a day Three to four times a day Three times a day	Monitor liver function tests

Pain originating from muscles in the neck can also be referred to the head and face. Predictable patterns for referred pain from muscles in the neck and shoulders to the head and face have been identified.¹⁸ Trigger points are discreet areas of contracted muscle that have a lowered pain threshold and are hyperirritable.¹⁷ Trigger points, when manually compressed, refer pain to distant regions. An active trigger point is able to elicit spontaneous pain or pain after physical stimulation that is referred to distant sites in predictable and reproducible patterns. A latent trigger point can also produce a pattern of referred pain when it is manually compressed or when the involved muscle is stretched or stressed in some way. Pain can be elicited directly through an activation of sensory afferents in the upper spinal nerves or referred through an exchange of mechanical and nociceptive signals between the spinal accessory nerve (CN XI) and upper cervical sensory afferent nerves. This interchange and convergence of sensory information would allow

for the referral of nociceptive sensory signals from the trapezius, sternocleidomastoid, and other cervical muscles to regions of the head and face.

Treatment

The successful treatment of cervicogenic headache requires a multifaceted approach using pharmacologic, nonpharmacologic, manipulative, anesthetic, and, occasionally, surgical interventions (*Figure 7*). Medications alone are often ineffective or provide only modest benefit. Many patients with cervicogenic headache overuse and become dependent on simple and opioid analgesics because they are often desperate to find relief of pain. The multidisciplinary treatment team will employ a primary treating physician or pain specialist to manage administration of medications and, if appropriately trained, provide manipulative treatment; an anesthesiologist with experience in pain management; a psychologist; and possibly a physical or occupational therapist.

Pharmacologic treatment

Pharmacologic treatment modalities for cervicogenic headache include medications that are often used for the preventive management of migraine (*Table 2*). The medications reviewed in this section have neither been approved by the Food and Drug Administration (FDA) nor rigorously studied in controlled clinical trials for efficacy in the treatment of cervicogenic headache. The medications are suggested as treatment for cervicogenic headache based on office- and clinic-based experiences of the author and other practitioners of pain management. The side effects and laboratory monitoring guidelines provided are not intended to be a comprehensive review, and the reader is cautioned to consult standard references or the medication package inserts before prescribing any medication.

Medications used as the sole treatment for cervicogenic headache do not tend to provide substantial relief of pain in many cases, but they can often provide enough benefit to allow the patient to be more

actively involved in a physical rehabilitation program. When medications are prescribed for this condition, they are initially prescribed at a low dose and increased as necessary and tolerated over 4 to 8 weeks. The cautious combining of medications from different drug classes may provide more efficacy than either drug used alone, such as an antiepileptic drug combined with a tricyclic antidepressant (TCA). Frequent office follow-up visits are often necessary for adjustments of medication dosage and monitoring of serum drug levels and for evidence of medication toxicity (Table 2).

The TCAs have long been used for management of neuropathic, musculoskeletal, head, and face pain syndromes. Serum drug levels may be used as a therapeutic guide. Levels of liver transaminase and complete blood cell counts should be monitored intermittently for evidence of toxicity. Sedation, confusion, weight gain, and anticholinergic side effects (xerostomia, blurred vision, constipation, urinary retention, orthostatic lightheadedness) may become a problem for some patients. Because the TCAs may cause cardiac conduction block at the atrioventricular node and intraventricular pathways, intermittent monitoring by electrocardiography is advisable while the TCA dosage is adjusted upward.

The antiepileptic drugs (AEDs) are neuromodulators of pain transmission in the central nervous system and are 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 neurogenic and cervicogenic head pain. Serum drug levels may be used as a therapeutic guide. Levels of liver transaminase and complete blood cell counts should be monitored regularly (that is, monthly) for evidence of toxicity, especially during the first 4 months of treatment or whenever dosages are adjusted upward. Potential side effects include nausea, sedation, dysequilibrium, tremor, weight gain, and hair loss. Gabapentin has been used for management of neuropathic and migraine pain. No specific laboratory monitoring is usually necessary. Side effects may include sedation, dysequilibrium, agitation, peripheral edema, and elevated blood pressure. Topiramate has demonstrated its effectiveness as a migraine preventive in early clinical studies. Electrolytes should be monitored inter-

mittently because of this medication's ability to inhibit carbonic anhydrase. Side effects may include tingling of the face and extremities, sedation, appetite suppression, weight loss, cognitive disturbances, and renal lithiasis. Carbamazepine has been an effective medication in the treatment of neuropathic pain syndromes such as trigeminal neuralgia. Serum drug levels may be used as a therapeutic guide. Levels of liver transaminase and the complete blood cell count should be monitored regularly (that is, monthly) for evidence of toxicity, especially during the first 4 months of treatment or whenever dosages are adjusted upward. Side effects may include nausea, dysequilibrium, sedation, cognitive disturbances, and diplopia.

Muscle relaxants and nonsteroidal anti-inflammatory drugs may be used as scheduled medications for preventive management of chronic pain or as necessary for management of acute pain.

Migraine abortive medications such as ergots or triptans are not typically effective in providing relief from cervicogenic head pain. Narcotic analgesics are also not typically effective and should be avoided if possible.

Osteopathic manipulative treatment

Osteopathic manipulative treatment can be a very important therapeutic modality when properly applied by an experienced provider and used as an integral part of a multidisciplinary pain rehabilitation program. The pain of cervicogenic headache has a tendency to worsen after manual or physical treatment modalities that are not carefully performed. A slow-paced progression of manipulation should begin with gentle muscle stretching and manual cervical traction. Osteopathic manipulative treatment techniques such as the muscle energy, craniosacral and strain/counterstrain techniques can provide significant pain relief. It is equally important to train the patient how to correctly participate in a regularly scheduled home exercise and rehabilitation program.

Anesthetic blockade

If a diagnostic nerve or zygapophyseal joint blockade is successful in providing pain relief, treatment will be directed toward proceeding with longer-acting neurolytic procedures such as radiofrequency thermal neurolysis or cryoneurolysis. Cervical epidural steroid injections may be indicated in cases of multilevel

disk or spine degeneration. Trigger point injections with local anesthetic may provide temporary pain relief and surrounding muscle relaxation. Anesthetic and neurolytic procedures can often provide enough pain relief to allow for a more comfortable and expedient course of manipulative treatment and physical rehabilitation. The routine use of botulinum toxin in this condition will require further clinical and scientific study.

Psychologic treatment

Psychologic and nonpharmacologic interventions such as biofeedback, relaxation, and cognitive-behavioral therapy are an important adjunctive treatment. Ongoing intensive, individual psychotherapy is often required if the patient has a prominent affective or behavioral component associated with the pain syndrome and the head and neck pain persists despite aggressive treatment.

Surgical treatment

Surgical procedures such as neurectomy, dorsal rhizotomy, and microvascular decompression of nerve roots or peripheral nerves are not generally recommended unless compelling circumstances such as radiologic evidence of a surgically correctable pathologic condition or refractoriness to all reasonable nonsurgical treatment modalities warrant their consideration.

Comment

Cervicogenic headache is a 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 the primary care physician can significantly decrease the protracted course of costly treatment and disability that is often associated with cervicogenic headache. By virtue of training in manual diagnostic skills and manipulative treatment, osteopathic physicians are well equipped to identify and manage this challenging pain disorder.

References

1. Sjaastad O, Saunte C, Hovdahl H, Breivik H, Gronbaek E. Cervicogenic headache. A hypothesis. *Cephalalgia* 1983;3:249-256.
2. Edmeads J. The cervical spine and headache. *Neurology* 1988;38:1874-1878.

3. Pollmann W, Keidel M, Pfaffenrath V. Headache and the cervical spine: a critical review. *Cephalalgia* 1997;17:501-516.
4. Leone M, D'Amico D, Grazzi L, Attanasio A, Bussone G. Cervicogenic headache: a critical review of the current diagnostic criteria. *Pain* 1998;78:1-5.
5. Bogduk N. The anatomical basis for cervicogenic headache. *J Manipulative Physiol Ther* 1992;15:67-70.
6. Blau JN, MacGregor EA. Migraine and the neck. *Headache* 1994;34:88-90.
7. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Headache Classification Committee of the International Headache Society. *Cephalalgia* 1988;8 Suppl 7:1-96.
8. Sjaastad O, Fredriksen TA, Pfaffenrath V. Cervicogenic headache: diagnostic criteria. *Headache* 1998;38:442-445.
9. Kerr FWL. A mechanism to account for frontal headache in cases of posterior fossa tumors. *J Neurosurg* 1961;18:605-609.
10. Stovner LJ. Headache associated with the Chiari type I malformation. *Headache* 1993;33:175-181.
11. Sturzenegger M. Headache and neck pain: the warning symptoms of vertebral artery dissection. *Headache* 1994;34:187-193.
12. Chiba S, Nishioka H, Saitoh M, Imai T, Tanabe S, Matsumoto H. Cervical dural arteriovenous malformation presenting with right-sided occipitalgia: before and after successful treatment by embolization. *Headache* 1994;34:234-236.
13. Goldhammer L. Second cervical root neurofibroma and ipsilateral migraine headache. *Cephalalgia* 1991;11:75-85.
14. Pfaffenrath V, Dandekar R, Pollmann W. Cervicogenic headache-the clinical picture, radiological findings and hypotheses on its pathophysiology. *Headache* 1987;27:495-499.
15. Bogduk N. The anatomy of occipital neuralgia. *Clin Exp Neurol* 1980;17:167-184.
16. Bovim G, Fredriksen TA, Stolt-Nilsen A, Sjaastad O. Neurolysis of the greater occipital nerve in cervicogenic headache. A follow-up study. *Headache* 1992;32:175-179.
17. Jaeger B. Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 1989;9:157-164.
18. Travell JG. Referred pain from skeletal muscle. *NY State J Med* 1955;55:331-340.
19. Nilsson N, Christensen HW, Hartvigsen J. The effect of spinal manipulation in the treatment of cervicogenic headache. *J Manipulative Physiol Ther* 1997;20:326-330.
20. Hobson DE, Gladish DF. Botulinum toxin injection for cervicogenic headache. *Headache* 1997;37:253-255.

Suggested reading

Bogduk N. Headache and the neck In: Goadsby PJ, Silberstein SD, eds. *Headache*. Boston, Mass: Butterworth-Heinemann; 1997.