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 nonpharmacologic modes of treatment, and anesthetic interventions are presented.

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

Cervicogenic headache: mechanisms, evaluation, and treatment strategies

DAVID M. BIONDI, DO

Cervicogenic headache is a 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.3 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-
titioner’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. 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. The development and acceptance of a single diagnostic format for cervicogenic headache has been an item of debate. The International Headache Society (IHS) and the Cervicogenic Headache International Study Group 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 Group 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 aggressive 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 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. In clinical practice, a recent or remote history of head or neck
Biondi • Cervicogenic headache

Occipital neuralgia is a condition that 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 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

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**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, nonthrobbbing, and nonlancinating pain, usually starting in the neck
  - episodes of varying duration, or
  - fluctuating, continuous pain

**Other characteristics of some importance**

- **Point V**
  - 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 periocular area

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

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**Figure 3. Clinical characteristics of cervicogenic headache.**

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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. 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 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...
in several months is common. Temporary relief of pain with treatments of 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 relief of pain is also observed after transection of the greater occipital nerve (neurectomy). 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. Diagnostic imaging such as radiotherapy, MRI, and computed tomographic myelography cannot confirm the presence of cervicogenic headache but can lend support its diagnosis.

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

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

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

Figure 7. Potential treatment interventions for cervicogenic headache.
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 *trigemino-cervical nucleus*. Interneurons within the trigemino-cervical 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 ophthalmic 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 trigemino-cervical 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 1**

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

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

#### Table 2
**Pharmacologic Treatment of Cervicogenic Headache**

<table>
<thead>
<tr>
<th>Drug class and representative medications</th>
<th>Daily dosage range</th>
<th>Typical dosing schedule</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricyclic antidepressants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Amitriptyline hydrochloride,</td>
<td>10 mg to 150 mg</td>
<td>At bedtime</td>
<td>Serum drug levels, monitor complete blood cell count, liver function tests, electrocardiogram</td>
</tr>
<tr>
<td>- Nortriptyline hydrochloride,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Doxepin hydrochloride</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antiepileptic drugs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Divalproex sodium</td>
<td>500 mg to 1500 mg</td>
<td>Two to three times a day</td>
<td>Serum drug levels, monitor complete blood cell count, liver function tests</td>
</tr>
<tr>
<td>- Gabapentin</td>
<td>1800 mg to 3600 mg</td>
<td>Three to four times a day</td>
<td></td>
</tr>
<tr>
<td>- Topiramate</td>
<td>100 mg to 400 mg</td>
<td>Twice a day</td>
<td></td>
</tr>
<tr>
<td>- Carbamazepine</td>
<td>300 mg to 1200 mg</td>
<td>Three times a day</td>
<td>Serum drug levels, monitor complete blood cell count, liver function tests</td>
</tr>
<tr>
<td>Muscle relaxants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Tizanidine hydrochloride</td>
<td>4 mg to 32 mg</td>
<td>Three times a day</td>
<td>Monitor liver function tests</td>
</tr>
<tr>
<td>- Baclofen</td>
<td>30 mg to 60 mg</td>
<td>Three times a day</td>
<td></td>
</tr>
<tr>
<td>- Cyclobenzaprine hydrochloride</td>
<td>10 mg to 40 mg</td>
<td>Three times a day</td>
<td></td>
</tr>
<tr>
<td>- Metaxolone</td>
<td>1200 mg to 2400 mg</td>
<td>Three times a day</td>
<td></td>
</tr>
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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. Side effects may include nausea, dyspepsium, 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 zygapophysal 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 neuromodulation, 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


Suggested reading