Headache associated with cervical spine dysfunction

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Introduction

This article includes discussion of headache associated with cervical spine dysfunction, cephale cervicale, cervical headache, cervical pain syndrome, cervicogenic headache, and cervical myofascial pain. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

The author reviews the variety of headaches associated with cervical spine dysfunction. There are many pain generators located in the cervical spine. In this article, headaches with a potential origin in the neck are reviewed, including cervicogenic headache, taking into account the accepted criteria found in the International Classification of Headache Disorders. Sjaastad’s concept of cervicogenic headache is also presented. Rare, but well-defined, potential causes of headache of cervical origin, such as developmental or acquired lesions of the craniovertebral junction as well as other more controversial cervical entities, are also discussed. Concepts on anatomy, pathophysiology, treatments, and mechanisms underlying cervicogenic headache have been updated.

Key points

• Headaches caused by disorders of the neck can present with different clinical features, making it difficult to describe a set of chief complaints to define them.
• Side-locked pain, provocation of typical headache by digital pressure on neck muscles and by head movement, and posterior-to-anterior radiation of pain are features that help distinguish cervicogenic headache from other primary headaches.
• The term “cervicogenic headache” was first introduced to the medical literature by Sjaastad and colleagues in 1983, and their diagnostic criterion continues to be a debate among specialists in the field.
• The two main diagnostic criteria for cervicogenic headache, one proposed by the International Headache Society and the other by the Cervicogenic Headache International Study Group (CHISG), differ from each other.

Historical note and terminology

Headache is one of the most common reasons for visits to the emergency room and out-patient treatment. Primary headache, such as migraine and tension-type headache, is a disorder unto itself; no underlying disease process is present. Secondary headache is a manifestation of an underlying disease process (Biller 2009). A variety of headaches are frequently associated with the occurrence of neck pain (Antonacci et al 2001).

The International Classification of Headache Disorders (ICHD) is a detailed hierarchical classification of all headache-related disorders published by the International Headache Society (IHS). This schematic headache classification is divided into 3 parts containing 14 sections. The first part identifies the primary headache disorders. The second part describes headaches attributed to an underlying (secondary) condition. It is under this section of the ICHD that headaches attributed to disorders of cervical structures, including cervicogenic headache, are described. The third part characterizes painful cranial neuropathies, other facial pains, and other headaches. The ICHD, 3rd edition (ICHD-3 beta), which is the most updated version, defines “headache attributed to a disorder of the neck” in section 11.2 as a “headache caused by a disorder involving any structure in the neck, including bony, muscular and other soft tissue elements.” Section 11.2 further subdivides into (1) cervicogenic headache, (2) headache attributed to retropharyngeal tendonitis, and (3) headache attributed to craniocervical dystonia. The Appendix diagnosis “headache attributed to cervical myofascial pain (A11.2.5) awaits further evidence that this type of headache is more closely related to other cervicogenic headaches than to tension-type headache.” Some overlap does exist between these 2 categories. In the context of cervicogenic headache, prolonged nociceptive inputs from peripheral structures play an important role in the development of sensitization mechanisms of the central nervous system. The neurophysiological basis of the convergence between trigeminal nociception and upper cervical afferents onto neurons in the brainstem is now well understood.
understood. The ICHD-3 beta states that headache caused by cervical radiculopathy is a logical cause of headache. Headaches caused by head and neck trauma are classified separately under section 5 (Headache Classification Subcommittee of the International Headache Society 2013).

Bärtschi-Rochaix reported what seems to have been the first clinical description of cervicogenic headache (Bartschi-Rochaix 1968), but it was not until 1983 that Sjaastad and his school defined diagnostic criteria for this syndrome (Sjaastad et al 1983; Antonaci et al 2005). There is lack of agreement between the ICHD classification and the one proposed by Sjaastad's group. The International Headache Society recognizes cervicogenic headache as a distinct disorder whereas the Sjaastad school stipulates that cervicogenic headache is not a “disease” or entity sui generis but a reaction pattern (Sjaastad et al 1983; Sjaastad et al 1990; Sjaastad et al 1998; Sjaastad 1992). Both criteria are described in this article.

**Clinical manifestations**

**Presentation and course**

The accepted cervical causes of headache are listed in Table 1 (Gobel and Edmeads 2006). We will first describe each one of those causes that includes retroparyngeal tendonitis and craniocervical dystonia, and then focus on cervicogenic headache.

**Table 1. Cervical Causes of Headache**

Note: Sjaastad cervicogenic headache and Bogduk third occipital headache are not specific headache entities but rather syndromes or reaction patterns said to result from a variety of lesions.

I. Well-demonstrated causes:
   A. Developmental anomalies of the craniocervical junction and upper cervical spine with or without neural anomalies (mainly Chiari type I malformation)
   B. Acquired lesions:
      1. Tumors of craniovertebral junction and upper cervical spine
      2. Paget disease of the skull with secondary basilar invagination
      3. Osteomyelitis of the upper cervical vertebrae
      4. Rheumatoid arthritis of the upper cervical spine
      5. Ankylosing spondylitis of the upper cervical spine
      6. Traumatic subluxation of the upper cervical vertebrae
      7. Retroparyngeal tendonitis
      8. Craniocervical dystonias
      9. Neck-tongue syndrome

II. Controversial causes:
   A. Cervical disc disease
   B. Spondylosis
   C. “Whiplash” injuries

III. Unaccepted causes:
   A. Posterior cervical syndrome of Barré
   B. Migraine cervicale syndrome of Bartschi-Rochaix

**Well-demonstrated causes of headache.**

*Developmental anomalies of the craniovertebral junction.* Developmental anomalies of the craniovertebral junction and upper cervical spine frequently cause headaches (MacRae 1969). More than 3 decades ago, headache was reported as the presenting complaint in a quarter of patients with anomalies such as basilar invagination, congenital atlantoaxial dislocation, or separate odontoid (Edmeads 1988). With the advent of MRI, we now know that most of these patients also have soft tissue anomalies, such as Chiari type I malformation (Pascual et al 1992).{embed="pagecomponents/media_embed" entry_id="9346"}

Valsalva-induced headaches are headaches that are typically provoked by activities—coughing, sneezing, laughing, crying, heavy lifting, straining at stool, or bending over—that incorporate a Valsalva maneuver (VanderPluym 2015). It is referred as benign or primary Valsalva-induced headache when no underlying organic cause is found although...
imaging is mandatory to exclude a variety of medical conditions, especially posterior fossa pathology that can present with a similar clinical picture (Pascual 2005). In one series of 97 patients evaluated for Valsalva-induced headaches, 45% were found to have an underlying intracranial abnormality, with Arnold-Chiari type I malformation being the one most commonly found (Pascual et al 2008). Clinically, it is described as sudden in onset, rapidly reaching peak intensity, and then either disappearing or fading to a dull ache that may remain for several hours (Williams 1976; Williams 1980). The Valsalva maneuver increases intrathoracic and intra-abdominal pressure, which is transmitted to epidural veins and produces a pressure wave that moves cerebrospinal fluid rostrally. The headache may be caused by the temporary impaction of the cerebellar tonsils with traction on the pain-sensitive dura. This sudden obstruction of the free flow of cerebrospinal fluid in the subarachnoid space has been confirmed in Chiari type I patients with cough headache versus controls before surgery. Cough headache disappeared after surgery when intrathecal pressure became normalized (Sansur et al 2003).

As the name indicates, cough is the main and sometimes only Valsalva trigger in patients suffering from cough headache. Pain is usually bilateral, of moderate to severe intensity (with a sharp or stabbing quality), and not associated with nausea or vomiting. The major affected locus is the occipital-suboccipital regions, but vertex or frontal radiation is commonly seen. These patients are usually pain-free between attacks (Pascual et al 1996). Some families have a genetic basis for anomalies of the craniocervical junction (Speer et al 2000). Secondary cough headache has been noted to begin earlier in life, occur more frequently, and last longer than primary cough headache. Primary cough headache is found almost exclusively in older men and is not associated with upper cervical symptoms or signs (Pascual et al 1996).

Acquired lesions of the craniocervical junction. Acquired disorders also produce occipital headaches that are triggered and worsened by neck movements or straining (Gobel and Edmeads 2000; Gobel and Edmeads 2006; Edmeads 2001). These disorders include primary tumors (schwannoma, meningioma, ependymoma), multiple myeloma, metastatic tumors, Paget disease of the skull with secondary basilar invagination, and osteomyelitis of the upper cervical column. (embed="pagecomponents/media_embed" entry_id="9347") All of the aforementioned conditions may produce headache by erosion of the pain-sensitive structures or traction on the upper cervical nerve roots. Rheumatoid arthritis and ankylosing spondylitis of the upper cervical spine can also induce inflammation of the synovial atlantoaxial and atlantooccipital joints and stretching of the upper cervical ligaments and nerve roots caused by atlantoaxial subluxation. Blows to the head, or even forceful sneezing, may produce rotatory subluxation of the atlas, which, through irritation of the synovial joints, can cause daily occipital headache. When performing a physical examination on patients with rheumatoid arthritis and headache, the clinician should exercise extreme caution while having the patient flex the neck, as fatalities have resulted from compression of the medulla by the odontoid, which, no longer bound to the atlas by the transverse ligament, fails to move away from the brainstem on anteflexion of the cervical spine (Sharp and Puser 1961; Conlon 1966; Bland 1967).

Headache attributed to retropharyngeal tendonitis. Retropharyngeal tendonitis, also called retropharyngeal calcific tendonitis, is a common form of calcific periartthritis in the tendon of the longus colli muscle (Kaplan and Eacey 1984). International Headache Society criteria for headache attributed to retropharyngeal tendonitis include unilateral or bilateral nonpulsating pain in the back of the neck, radiating to the back of the head or to the whole head (Headache Classification Subcommittee of the International Headache Society 2013). The radiologic diagnosis can be made from finding enlargement of the retropharyngeal space due to fluid collection and, subsequently, from calcifications present in the superior longus colli tendons. Retropharyngeal calcific tendonitis may occasionally mimic a retropharyngeal abscess, presenting with symptoms of sore throat, low-grade fever, mild leukocytosis, and elevated sedimentation rate. Headache is caused by inflammation or calcification of retropharyngeal soft tissues and is aggravated severely by extension of the neck, rotation of the head, or swallowing. In a literature review of 71 case reports of patients with retropharyngeal calcific tendonitis, the most common symptom was neck pain (94%), followed by limited range of motion (45%) (Park et al 2010). Upper carotid dissection should be ruled out (Headache Classification Subcommittee of the International Headache Society 2013).

Headache attributed to craniocervical dystonia. Dystonia is a neuromuscular disorder defined as continuous muscle contractions that lead to repetitive movements and abnormal posturing. Craniocervical dystonia affects muscles of the neck innervated by the cranial nerves. Pain is thought to originate from muscle hyperactivity and persistent contraction as well as secondary changes in sensitization. Within the nervous system, craniocervical dystonia is thought to originate from abnormality of the basal ganglia-thalamo-cortical circuitry (Colosimo 2010) as well as from chemical imbalances of the GABA-releasing cerebellar Purkinje cells, particularly due to dysfunction of the Na+/K+
Headache attributed to craniocervical dystonia should develop in close temporal relation to the onset of the craniocervical dystonia, or there should be clinical signs that implicate a source of pain in the hyperactive muscle (e.g., pain is precipitated or exacerbated by muscle contraction, movements, sustained posture, or external pressure). Both conditions are expected to worsen in parallel, and pain should improve or resolve with treatment of the abnormal movements (Headache Classification Subcommittee of the International Headache Society 2013).

Controversial causes of headache.

Cervical disc disease and spondylosis. The presence of cervical spondylosis or cervical disc disease should not necessarily be taken as the cause of a patient's headaches. Their almost ubiquitous existence in people over 40 years old makes it difficult to establish causality or even strong association. As they involve the lower cervical discs and vertebrae, a pathophysiologic referral of pain to the head would be difficult to explain. Hypothetically, the restriction of movement in these lower cervical regions would lead to excessive work in the C2-4 joints, and this could refer pain to the head (Edmeads 1988; Edmeads 2001; Bogduk 1997; Gobel and Edmeads 2000).

Clinically, these patients may complain of attacks that last days or even weeks and of dull, aching pain in the neck and occiput on one or both sides that may radiate to the vertex or the frontoparietal area. Headache is common on awakening and diminishes spontaneously within hours. Unilaterality is not an essential feature, but some may complain of more obvious hemicranial radicular pains precipitated by coughing or sneezing. A prospective study has shown an increased prevalence of headache in patients with lower cervical disc prolapse as compared to patients with lumbar disc prolapse. Most of these patients with lower cervical disc prolapse were reported to be pain-free after operation (Diener et al 2007).

Whiplash injuries. Whiplash is defined as sudden and inadequately restrained acceleration and deceleration movements of the head with flexion and extension of the neck. It may occur after either high- or low-impact forces (Headache Classification Subcommittee of the International Headache Society 2013). In the ICHD-3 beta, acute and persistent headache attributed to whiplash are described under “headache attributed to trauma or injury to the head and/or neck.” When a headache occurs for the first time in close temporal relation to trauma or injury of the head or neck, it is considered a secondary headache attributed to the trauma or injury. This is true even when the headache has the characteristics of a primary headache. In other words, headache attributed to trauma can present as any type of primary headache as long as it happens in close temporal relation to the trauma. To facilitate the diagnosis, the headache must develop within an arbitrary 7-day interval of the trauma or injury. If loss of consciousness is involved, headache should be reported within 7 days after regaining ability to sense or report pain. During the first 3 months, it is considered acute; if the headache continues beyond that period, it is designated as persistent. Headache may occur as part of a postconcussion syndrome that includes other symptoms, such as dizziness, fatigue, psychomotor slowing, decreased concentration, anxiety, irritability, mild memory impairment, or insomnia.

Although whiplash is a controversial cause, it is a clinical reality that many people with an extension-flexion injury of the neck do complain of occipitofrontal dull or shooting pain that usually subsides within 3 to 6 weeks. Headache seems to be present in 50% to more than 75% of cases in the acute stage and in 20% to 30% of cases in the early chronic stage. Stretch or injury to the upper cervical painful structures including long neck muscles and interspinous ligaments is a reasonable explanation for this transient headache. Some studies suggest that shearing injuries of long axons in the brainstem and upper cord may disrupt central pain and other regulatory mechanisms. Previous history of headache, female gender, and presence of comorbid psychiatric conditions can all be considered risk factors for the development of headaches after a trauma. The patient's expectations of headache, litigation issues, and sleep disruption can certainly contribute to persistence of pain (Hawkins 1962; Berry 1976; Weiss et al 1991; Zwart 1997; Vincent and Luna 1999). “Persistent headache attributed to whiplash” is classified as pain that persists for more than 3 months after the whiplash and is associated with increased risk for medication overuse (Headache Classification Subcommittee of the International Headache Society 2013).

 Syndromes not generally accepted.

Third occipital headache. Bogduk third occipital headache is an entity of uncertain status (Bogduk and Marsland 1986). This nerve, the superficial medial branch of the C3 dorsal ramus, supplies the C3 dermatome, part of the semispinalis capitis muscle, and the C2-3 zygapophyseal joint. Bogduk and Marsland blocked this nerve and recorded pain relief in
two thirds of patients who had occipital headaches that radiated frontally, and they recorded at least 1 feature that could suggest a cervical origin for the pain (eg, history of neck injury, triggering of pain by neck movement). They interpreted their results as suggesting that disease of the C2-3 zygapophyseal joints may produce headache by irritating or compressing the third occipital nerve. The small number of patients and the absence of radiological evidence of local anomalies, however, are disturbing. Alternatively, the nerve blocks could relieve headache in a nonspecific fashion by cutting impulses to the central trigeminal system (Edmeads 1988; Edmeads 2001; Bogduk 1997; Gobel and Edmeads 2000; Gobel and Edmeads 2006).

**Barré syndrome and cervical migraine syndrome.** In Barré posterior cervical sympathetic syndrome, symptoms of headache, neck discomfort, dizziness, visual blurring, psychological disturbances, and impaired hearing are believed to be caused by osteophytes irritating the sympathetic nerve plexus that invests the vertebral arteries (Sjaastad and Fredriksen 2000). In the cervical migraine syndrome of Bartschi-Rochaix, similar symptoms are believed to be due to actual compression of the vertebral artery by osteophytes or trauma (Bartschi-Rochaix 1968).

Trauma is considered a frequent precipitant of these syndromes. Physical findings in both syndromes are suboccipital tenderness, palpable spasm of neck muscles, limitation of neck movement, and reproduction or intensification of these symptoms by neck movements. Sensory changes in the territory of C2 dermatome have occasionally been described. The similarities of these syndromes and the whiplash syndrome are striking. Impeding the acceptance of these syndromes is the failure to demonstrate that stimulation of the posterior sympathetic syndrome can indeed induce these symptoms.

**Other studied causes.** Other sources of cervicogenic headache have been proposed through case reviews and retrospective studies. There are many pain generators in the cervical spine, which include cervical facet joints, cervical intervertebral discs, skeletal muscles, and neurovascular structures (Ng and Wang 2015). It would be appropriate to list these as controversial causes as there are either limited sample sizes or inherent flaws when using accepted criteria.

**Atlas spina bifida occulta.** Atlas spina bifida occulta, a developmental anomaly characterized by defective closure of the posterior arc of the first cervical vertebra of the spine, has been reported as a probable source of cervicogenic headache (Adigo et al 2015). Although Chiari I malformations have well-demonstrated links to cervicogenic headaches, there seems to be mixed supporting evidence of a direct link between atlas spina bifida occulta as a pure source for cervicogenic headache based on the demand for unilaterality as a criteria and ipsilateral arm/neck discomfort (Pascual et al 1992; Sjaastad and Bakkeiteig 2008b). A study with a small sample size showed a bilateral predominance in 12 of 17 reviewed cases, with less than 30% having ipsilateral arm discomfort. The arguments in favor included successful C2 nerve blocks in 16 of 17 patients (Adigo et al 2015). There was also postero-anterior irradiation of pain, which was considered an important criterion, the elimination of migraine attack and tension headache diagnoses, and a female predominance (International Headache Society (2013); Fredriksen et al 2015).

**Vertebral artery dissection.** A single case review did demonstrate headache and transient unilateral vision loss as presenting symptoms in a 33-year-old male with vertebral artery dissection (Yvon et al 2016). However, the lack of description of the headache quality and the sample size still warrant further investigation into causality.

**Spinal cord tumors.** In a case report, an intradural extramedullary tumor of the middle cervical spine induced cervicogenic headache, which arose from the fifth cervical nerve root. Previously reported cases of “headache attributed to upper cervical radiculopathy” described intradural extramedullary tumors in the upper cervical spine, whereas this report is the first to describe this lesion occurring in the middle-lower cervical spine, causing cervicogenic headache. The diagnostic criteria listed in the appendix of the ICHD-3 beta currently describe the presence of clinical or radiological evidence of a C2 or C3 radiculopathy (Gondo et al 2016).

**Cervical myofascial pain.** A current review article presents “an overview of the role of muscle pain and how referred pain from myofascial trigger points in the posterior cervical, head, and shoulder muscles can be the source of head pain by contributing to the development and/or maintenance of sensitization mechanisms in some headaches such as tension-type, cervicogenic, or migraine” (Fernández-de-Las-Peñas 2015). Myofascial trigger points are defined as “a hypersensitive spot within a taut band of skeletal muscle that is painful on mechanical stimulation and causes a referred pain that is perceived distant from the spot.” The evidence suggests that the referred pain elicited by trigger points reproduces at least part of the headache pattern. This peripheral nociceptive mechanism results in sensitization of nociceptive pain pathways in the central nervous system. Further clinical trials are needed to clarify the role of...
referred pain patterns, which are generated by active trigger points, in the development of a cervicogenic headache disorder. Postural misalignment is a common musculoskeletal pain disorder of the cervical spine. In a cross-sectional study by Ferracini and colleagues, a bidirectional relationship was proposed between cranio cervical postural misalignment and facilitation of active trigger points in associated muscles of the head and neck (suboccipital, upper trapezius, sternocleidomastoid, temporalis, and masseter) (Ferracini et al 2016). Correction of posture and inactivation of active trigger points by physical modalities (such as local anesthetic injection, deep and superficial needling, electrotherapy, and stretching exercise) leads to restoration of muscle balance and reduction in muscle tension.

Arteriovenous fistula and hemodynamic flow. A 43-year-old female with a recent renal transplant and still functioning left arteriovenous fistula presented with nonthrobbing unilateral headache along with vertigo and lateropulsion (Pereira et al 2016). Neuroimaging and neurologic exam were normal, but color flow Doppler demonstrated subclavian steal syndrome in the left vertebral artery with internal jugular flow reversal. The symptoms resolved after ligation of the fistula. The proposed etiology of headache was the flow reversal in the left internal jugular vein causing increasing intracranial pressure. The headache characteristics, however, changed over a 6-month course prior to the ligation, and the proposed mechanism involved constant stimulation to the dural sinus with sensitization of the trigeminovascular system in addition to the intracranial hypertension. A literature review from the same group determined little consistency, with either reports of headaches in similar cases or lack of qualifying description.

Cervical cord arteriovenous malformations. Neuroradiology reviews and case reports have been published over the years, and spinal dural arteriovenous malformations generally present in middle-aged males rather than in pediatric populations (Inci et al 2012). In a case that involved a 13-year-old child, headache in the occipital region with occasional radiation of pain along the neck was the presenting chief complaint (Saini et al 2015). A review of systems was fairly uneventful, but on physical exam the child had generalized hyperreflexia. MRI of the cervical spine demonstrated an arteriovenous malformation at the C3-C4 level with multiple collaterals. It has been hypothesized that 2 mechanisms can incite cervicogenic headache (Bassuk et al 2003). The first is cervical dermatomal referred pain to the occipital region, and the other is related to intermittent bleeding that causes obstruction of cerebrospinal fluid flow causing a clinical stenosis with myelopathy-type presentations.

Cervicogenic headache. Rather than a disease entity, cervicogenic headache is thought to be the clinical manifestation of a disorder of the cervical spine and its component bone, disc, or soft tissue elements—usually but not invariably accompanied by neck pain (Gobel and Edmeads 2006). There should be clinical, laboratory or radiological evidence of a disorder or lesion within the cervical spine or soft tissues of the neck capable of causing headaches. Evidence of causation is the motivation behind the revised ICHD-3 beta criteria for cervicogenic headache. Headache is demonstrated by the presence of at least 2 of the following: (1) headache has developed in temporal relation to the onset of the cervical disorder or appearance of the lesion; (2) headache has significantly improved or resolved in parallel with improvement in or resolution of the cervical disorder or lesion; (3) cervical range of motion is reduced or significant worsening of the headache by provocative maneuvers; (4) headache is abolished following diagnostic blockade of a cervical structure or its nerve supply (Headache Classification Subcommittee of the International Headache Society 2013). The criteria for cervicogenic headache are listed in Table 2.

This headache is usually unilateral, beginning in the neck and eventually spreading to the oculo frontotemporal area where the maximum pain is. Pericranial tenderness and radiation of pain from the occipital area to frontal or periorbital regions is suggestive but neither specific or necessary for the diagnosis. Prevalence estimates of cervicogenic headache in the general population range from 0.4% to 4% and up to 20% of patients evaluated for chronic headaches in pain management clinics (Haldeman and Dagenais 2001; Ng and Wang 2015). This condition is 4 times more common in women than in men, with a mean age of 42.9 years old.

Table 2. ICHD-3 Beta Criteria For Cervicogenic Headache

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

Comments:
Features that tend to distinguish 11.2.1 Cervicogenic headache from 1. Migraine and 2. Tension-type headache include side-locked pain, provocation of typical headache by digital pressure on neck muscles and by head movement, and posterior-to-anterior radiation of pain. However, although these may be features of 11.2.1 Cervicogenic headache, they are not unique to it, and they do not necessitate causal relationships. Migrainous features such as nausea, vomiting, photo/phonophobia may be present with 11.2.1 Cervicogenic headache, although to a generally lesser degree than in 1. Migraine, and may differentiate some cases from 2. Tension-type headache.

Tumors, fractures, infections, and rheumatoid arthritis of the upper cervical spine have not been validated formally as causes of headache but are nevertheless accepted as valid causes when demonstrated to be so in individual cases.

Cervical spondylosis and osteochondritis may or may not be valid causes fulfilling criterion B, depending on the individual case.

When cervical myofascial pain is the cause, the headache should probably be coded under tension-type headache. However, awaiting further evidence, an alternative diagnosis is included in the Appendix as A11.2.5 Headache attributed to cervical myofascial pain.

Headache caused by upper cervical radiculopathy has been postulated and, considering the now well-understood convergence between upper cervical and trigeminal nociception, this is the logical cause of headache. Pending further evidence, this diagnosis is found in the Appendix as A11.2.4 Headache attributed to upper cervical radiculopathy.

(Headache Classification Subcommittee of the International Headache Society 2013)

Sjaastad cervicogenic headache. The true debate on the concept of “cervicogenic headache” was provoked by Sjaastad when he proposed that the neck is the origin of a somewhat uniform pain profile frequently experienced by headache patients (Sjaastad et al 1983). The term “cervicogenic headache” was first introduced to the medical literature by Sjaastad and colleagues in 1983. In 1987, Sjaastad set up the Cervicogenic Headache International Study Group (CHISG), with its diagnostic criteria first published in 1990 (Sjaastad et al 1990) and revised in 1994 (Merskey and Bogduk 1994). This revised criteria is listed in Table 3.

Table 3. Sjaastad's Diagnostic Criteria For Cervicogenic Headache

Major (mandatory) criteria:
   I. Symptoms and signs of neck involvement
A. Precipitation of head pain, similar to the usually occurring one:
   1. By neck movement or sustained awkward head positioning and/or:
   2. By cervical pressure over the upper cervical or occipital region on the symptomatic side
B. Restriction of the range of motion in the neck
C. Ipsilateral neck, shoulder, or arm pain of a rather vague nonradicular nature or, occasionally, arm pain of a radicular nature

II. Confirmatory evidence by diagnostic anesthetic blockades
III. Unilaterality of the head pain, without sideshift

Head pain characteristics:
I. Moderate-severe, nonthrobbing, and non-lancinating pain, usually starting in the neck
II. Episodes of varying duration, or
III. Fluctuating, continuous pain

Other characteristics of some importance:
I. Only marginal effect or lack of effect of indomethacin
II. Only marginal effect or lack of effect of ergotamine and sumatriptan
III. Female sex
IV. Not infrequent occurrence of head or indirect neck trauma by history, usually of more than only medium severity

Other features of lesser importance:
I. Various attack-related phenomena, only occasionally present, or moderately expressed when present:
   A. Nausea
   B. Phonophobia and photophobia
   C. Dizziness
   D. Ipsilateral "blurred vision"
   E. Difficulties on swallowing
   F. Ipsilateral edema, mostly in the periocular area

As mentioned before, the criteria proposed by the International Headache Society classification differs from the one proposed by CHISG (Antonaci et al 2005). For Sjaastad’s group the concept of cervicogenic headache reiterates that some headaches may result from neck dysfunction or disease and that these headaches may show clinical characteristics suggesting a neck origin.

Prognosis and complications

Prognosis varies with specific etiology and, when there is no obvious etiology, with the patient's age, occupation, and treatment response. Excluding other primary and secondary headache causes is crucial for a correct diagnosis so appropriate management can be established.

Comorbid depression and anxiety, commonly found in chronic pain sufferers, can contribute to treatment refractoriness when not addressed properly. Resultant medication overuse, decreased productivity, and work absenteeism are potential socioeconomic problems.

Clinical vignette

A 42-year-old woman who worked as a secretary complained of left-sided head pain. She had no migraine antecedents and related the beginning of the pain to an innocent cervical injury when playing with her child. The pain started suboccipitally, spread over the temporal and frontal areas or, rarely, to ipsilateral neck and shoulder, and was moderate, usually described as a dull ache or as a strain feeling, sometimes accompanied by dizziness and nausea. The duration of the headache was variable, ranging from a few hours to a few days. The pain could be precipitated or aggravated by external pressure over the left upper cervical region, by rapid neck movements to the left, or by looking continuously at the computer at work. She tried sodium naproxen, ibuprofen as symptomatic treatment, and then amitriptyline for 1 month, with only partial relief. Examination disclosed cervical tenderness, more marked on the left.
Neck x-rays and cervical MRI showed spondylosis, without signs of disc herniation. An anesthetic blockade relieved her symptoms. The pain reappeared after 1 week and disappeared again after a new infiltration with local anesthetic plus depot steroid.

**Biological basis**

**Etiology and pathogenesis**

Pain sensation from the front of the head and face as well as anterior skull is mainly carried by the 3 divisions of the trigeminal nerve (CN V): ophthalmic, maxillary, and mandibular (Levin 2008). Unmyelinated C fibers that innervate those pain-sensitive structures pass through the trigeminal ganglion and enter the pons to reach the trigeminal nucleus caudalis, which extends caudally to connect with the first 3 cervical segments of the spinal cord. This “brainstem integrator,” often referred to as the trigeminocervical complex and extensively studied in animal models as detailed below, provides a pathophysiologic explanation for pain referral patterns seen in conditions affecting the head, face, or upper neck (Goadsby and Zagami 1991a; Goadsby and Zagami 1991b; Goadsby et al 1997).

Below the tentorium, there is more complex innervation with contribution from the facial nerve (CN VII), glossopharyngeal nerve (CN IX), and vagal nerve (CN X) as well as the C2 and C3 nerve roots. The upper cervical roots, primarily C2 and C3, carry pain sensation from posterior portions of the head as well as the dura in the posterior fossa. Nociceptive cervical structures include joints, peristeum, ligaments of the cervical spine, muscles around the cervical spine, cervical roots, nerves, and arteries (Gobel and Edmeads 2006). C1 is essentially a motor nerve innervating several suboccipital muscles. Lesions or dysfunction of these nerves may produce pain localized to the innervated area and are often referred to different areas given the above-mentioned overlap of central pathways (Farooq and Williams 2008). Rostrally, the trigeminal nucleus caudalis sends fibers to the thalamus, autonomic nucleus in the pons (superior salivatory nucleus), and hypothalamus.

Bartsch and Goadsby stimulated the supratentorial parietal dura and the greater occipital nerve of anesthetized rats and recorded the response in the C2 spinal dorsal horn with the aim of better understanding the physiology of trigeminocervical input and the mechanisms of pain referral. They found a 48% convergence of both dural and greater occipital nerve input (Bartsch and Goadsby 2002). Immunohistochemical activity was seen in the trigeminal nucleus caudalis and dorsal horn at the C1 and C2 levels after meningeal and superior sagittal sinus stimulation (Nozaki et al 1992; Kaube et al 1993).

Burstein and Jakubowski proposed that peripheral information from meningeal nociceptors, once reaching the above-mentioned trigeminothalamic pathway, could be modulated further by inhibitory and facilitatory neurons in the brainstem (Burstein and Jakubowski 2005). The evolution of functional neuroimaging revealed the existence of a nociceptive neural circuit often referred to as “the pain matrix.” The main components of this complex network are the primary and secondary somatosensory cortices, thalamus, anterior and posterior parts of the insula, anterior cingulate cortex, and prefrontal cortex (Apkarian et al 2005). Activity within this matrix can affect how pain is perceived.

There is no known physiologic basis for cephalic referral of pain originating from cervical segments below C4 (Gobel and Edmeads 2006).

**Epidemiology**

The prevalence of cervicogenic headache varies considerably as a result of the heterogeneity of diagnostic criteria. The prevalence of cervicogenic headache was 2.5% in the Danish general population when applying criteria of the International Headache Society (Nilsson 1995). In headache centers, prevalence estimates range from 0.4% to 80% (Haldeman and Dagenais 2001). In the general population, prevalence rates have varied from 0.4% to 2.5% whereas studies looking at all patients with a complaint of headache reported estimates of 15% to 20% (Evers 2008; Nilsson 1995).

In 2008, Sjaastad published a large epidemiological study in Vaga, Norway; they reviewed the records of 1838 patients with ages ranging from 18 to 65 years with the goal of describing the prevalence and various clinical characteristics of cervicogenic headache (Sjaastad and Bakkeiteig 2008a). The authors used the CHISG diagnostic criteria and found a prevalence of 4.1%, with 97% of the cases reporting pain exacerbation beginning in the neck or occipital region. The Vaga study showed a male preponderance (F/M: 0.71) although the majority of studies reveals a clear female predominance (4:1), mean age of onset 43 years, and a mean duration of symptoms of around 7 years (Sjaastad and
Prevention

Ergonomics is applied to a limited extent in occupations where the head and neck are held in position for long periods (e.g., bench workers, typists, computer or visual display unit operators, musicians). Flat-topped desks are the rule, but if such desks were inclined, neck flexion would be reduced. Only architects and draftsmen use sloping drawing boards, which are variable. Too often, individuals seek help only after they develop neck and head pains. Many music academies have Alexander relaxation or other therapists to advise, but faulty postures that were developed in the formative years are difficult to correct.

Long-distance driving is likely to cause neck stiffness and pain. Car accidents and diving into shallow water are also causes of neck injury. In clinical practice, those with long, slender necks, which are often associated with joint hypermobility, are more prone to develop musculoskeletal neck pains resulting in headaches; such patients form a younger age group (in their 20s and 30s) than the middle-aged and elderly who are more commonly affected by cervical spondylosis.

Differential diagnosis

Based on their experience, Sjaastad and Bakketeig believe that cervicogenic headache is, in principle, a strict unilateral headache, but it may also be bilateral ("unilaterality on the 2 sides") (Sjaastad and Bakketeig 2008b). Otherwise, this headache will be confused with tension-type headache or migraine without aura. The duration of the solitary attack—or an exacerbation—varies from a few hours to a few weeks. In the initial phase, the headache is usually episodic; later, it frequently becomes chronic-fluctuating. Symptoms and signs referable to the neck, such as a reduced range of motion in the neck or mechanical precipitation of attacks, are essential. When present, "migrainous" symptoms, like nausea and photophobia, are generally not marked. A positive response to appropriate anesthetic blockades is essential.

According to Sjaastad's group, no specific radiological abnormalities have been identified, contradictory to the International Headache Society classification, in which radiological abnormality is an obligatory criterion (Sjaastad and Bovim 1991).

The scarcity of autonomic symptoms and signs distinguishes this headache from cluster headache, as do other features, such as the temporal pattern, severity, and female preponderance. Hemicrania continua and cervicogenic headache have many traits in common. Both disorders frequently begin with a remitting headache, which may eventually develop into a chronic type and predominate in females. However, precipitation mechanisms are not an integral part of hemicrania continua, and the response to indomethacin is a decisive factor in the differential diagnosis. Sjaastad's group states that the differential diagnosis of migraine is also possible, taking into account that migraine attacks are more commonly accompanied by photophobia, phonophobia, pulsating pain, and aggravation on minor physical activity (Sjaastad et al 1998). Migraine pain usually starts in the anterior parts of the head, not infrequently shifts side, and is not abated by local anesthetic blockades of cervical nerves. It does not appear that any specific clinical finding or test can be used to define patients with cervicogenic headache versus migraine without aura or even tension-type headache. We agree with the proposal that cervicogenic headache versus tension-type headache and migraine without aura should only be considered when a pattern of unilateral pain without sideshift exists, when the initial pain is located at the posterior area, and when the headache cannot be classified by diagnostic criteria for other headaches. In a small case series, Sjaastad and colleagues demonstrated the coexistence of migraine without aura and cervicogenic headache (Sjaastad et al 1999). Each headache was successfully addressed separately, migraine attacks treated with triptans and cervicogenic headaches managed invasively.

Regarding the differential diagnosis with secondary headaches, cervicogenic headache must be differentiated from anterior and posterior circulation artery dissection, which can present as neck and facial pain. Usually, the pain is ipsilateral to the dissected vessel and may be a warning symptom preceding a stroke. A constellation of neurologic findings, including ipsilateral Horner syndrome, painful tinnitus of sudden onset, or painful facial palsy, are highly suggestive of carotid dissection. Diagnosis is based on cervical MRI with fat suppression, carotid Doppler ultrasound, magnetic resonance angiogram, computed tomography angiogram, and in doubtful cases, conventional angiography. The onset of focal symptoms suggestive of vascular damage in the vertebrobasilar territory is an indication of this diagnosis (Stahmer et al 1997). If this differential diagnosis is not considered, there is risk that a potential cervical
manipulation treatment for suspected cervicogenic headache aggravates the dissection. Other secondary headaches to be considered in the differential diagnosis of cervicogenic headaches are neck-tongue syndrome and C2 neuralgia (Bogduk and Govind 2009; Antonaci and Sjaastad 2011).

As previously discussed, patients presenting with Valsalva-induced headaches, including cough headaches, should undergo magnetic resonance imaging to exclude posterior fossa lesions, especially Chiari type-I malformation (Pascual 2005).

**Diagnostic workup**

Performing an expensive battery of exams on all patients with headache is neither cost-effective nor appropriate. Failure to perform diagnostic tests in certain patients, however, may result in missing life-threatening, yet treatable conditions (Biller 2009). In addition to obtaining a thorough history and physical examination, the clinician should be attentive to signs and symptoms suggestive of secondary headaches. An easy mnemonic “SNOOPP” can be used as a reminder of the most important signs and symptoms:

“S” for systemic signs and symptoms as well as for secondary risk factors. For example, the presence of fever and meningismus should be concerning for meningitis. Weight loss, fatigue, or an underlying history of cancer should require investigations for intracerebral metastatic disease. Other risk factors include immunodeficiency (HIV).

“N” stands for neurologic signs and symptoms. Altered mental status or a focal neurologic complaint or finding on exam, especially if abrupt in onset and persistent, should raise concerns.

“O” stands for onset. Abrupt onset (split-second, thunderclap) headaches can be a clinical presentation of ruptured aneurysm and subarachnoid hemorrhage.

“O” also stands for older age. New onset of headaches after the age of 50 years in a patient with no underlying history of headaches requires further investigation.

“P” stands for pattern change and postural component. Any changes in frequency or quality of primary headaches can be suggestive of development of a secondary concomitant condition. Headaches that are influenced by vertical versus horizontal position may suggest intracranial hyper- or hypotension. The presence of papilledema can indicate increase intracranial pressure. Pregnancy can also be included within this category given the risk of preeclampsia, eclampsia, idiopathic intracranial hypertension, subarachnoid hemorrhage, tumor, pituitary apoplexy, cerebral venous thrombosis, and reversible cerebral vasoconstriction syndrome.

<table>
<thead>
<tr>
<th>Stands for...</th>
<th>Example...</th>
<th>Think of...</th>
</tr>
</thead>
<tbody>
<tr>
<td>S Systemic Secondary risk factors</td>
<td>Fever, weight loss, fatigue, HIV, cancer, immune suppression</td>
<td>Infection, inflammation, metastatic cancer, carcinomatous meningitis</td>
</tr>
<tr>
<td>N Neurologic symptoms and signs</td>
<td>Altered consciousness, focal deficits</td>
<td>Encephalitis, mass lesion, stroke</td>
</tr>
<tr>
<td>O Onset</td>
<td>Thunderclap, abrupt</td>
<td>SAH, IPH, RCVS</td>
</tr>
<tr>
<td>O Older</td>
<td>New after age 50</td>
<td>Temporal arteritis</td>
</tr>
<tr>
<td>P Positional Pattern change Papilledema</td>
<td>Change upright vs laying Change with neck position Different in quality Visual obscurations</td>
<td>Intracranial hypotension, dysautonomia, cervicogenic headache, intracranial hypertension Posterior fossa pathology</td>
</tr>
</tbody>
</table>

Magnetic resonance imaging is mandatory for patients who have a posterior pain that is triggered by Valsalva maneuver or who exhibit upper cervical neurologic signs, such as weakness, paresthesia, or gait changes. In particular cases, electromyography or nerve conduction studies may be necessary for further evaluation. Computed tomography myelography or CSF flow MRI can be performed if there is suspicion of altered spinal cerebral fluid pressure. As previously mentioned, cervical MRI with fat suppression, carotid Dopplers, magnetic resonance angiogram, computed tomography angiogram, and in doubtful cases, conventional angiography are diagnostic tests available when carotid or vertebrobasilar dissection is suspected.
Response to cervical structure blockade is helpful when considering the diagnosis of cervicogenic headache and, thus, can be used as a diagnostic tool. Greater occipital nerve blocks should not be used to diagnose cervicogenic headache as response to these blocks do not necessarily establish a cervical source of pain (Bogduk and Govind 2009). This procedure seems to have a partial neuromodulatory action on headache mechanisms, whether the headaches have a cervical source or not, and as such, patients with other types of headaches, including migraine and cluster headaches, may also obtain transient pain relief (Afridi et al 2006). Interventionist pain physicians use fluoroscopically-guided, controlled diagnostic blocks to test whether particular structures are the source of pain in patients with suspected cervicogenic headache. Studies have focused on the following structures: the lateral atlantoaxial joint, which can be anesthetized by intra-articular blocks; the C2-3 zygapophyseal joint, which can be blocked by anesthetizing the third occipital nerve, in addition to C2 and C3 spinal rami; and the C3-4 zygapophyseal joint, which can be anesthetized by blocking the medial branches of the C3 and C4 dorsal rami. According to those specialists, complete relief of headache after such blocks, under controlled conditions, provides a more objective evidence of a cervical source of pain. The authors further observe that patients with primary headaches, such as migraine and tension-type headache, do not respond to diagnostic blocks of cervical joints (Bogduk and Govind 2009; Ng and Wang 2015).

Management

The management of headaches attributed to disorders of the neck will depend on their underlying cause. Pharmacologic treatment with analgesics, anti-inflammatories, and muscle relaxants may provide benefit in certain cases although it is usually short-lived and long-term use is precluded by side effects. There is little information available from clinical trials to support many of the conservative treatments for mechanical neck pain (Aker et al 1996). In a systematic review, Racicki and colleagues found that the combination of therapist-driven cervical manipulation and mobilization with cervico-scapular strengthening was most effective for decreasing pain outcomes in patients with cervicogenic headache (Racicki et al 2013).

Fredriksen summarized some of the available invasive procedures for the treatment of cervicogenic headache (Table 5). Greater occipital nerve blocks can be used as a treatment tool although should not be used diagnostically as response to these blocks do not necessarily establish a cervical source of pain (Bogduk and Govind 2009). Cryo procedures (cryo analgesia) directed at the greater occipital nerve may reach success rates around 60%. In a survey population study Knackstedt and colleagues found that patients self-reported a 90% effectiveness with greater occipital nerve blockage and cryotherapy compared to other procedures (Knackstedt et al 2010). Radiofrequency procedure is still controversial as to its efficiency and durability, with reported results ranging from no statistical difference compared with placebo to success rates of up to 80% and durability up to more than 10 years (Stovner et al 2004).

Rarely, well-selected patients in a precarious situation nonresponsive to other treatment options, may benefit from more invasive procedures, such as decompression of the spinal dura followed by stabilization and fusion (Jansen 2008). Jansen and Sjaastad published a series of 28 patients with bilateral cervicogenic headache submitted to the above-mentioned procedure and followed in a prospective fashion (Jansen and Sjaastad 2006). Among the 28 patients, 9 reported pain freedom after 5 months of follow up. Even though the numbers seem promising, the placebo response to surgical intervention is very high, and its use in treating cervicogenic headache requires controlled studies.

A retrospective study compared the efficacy of the 3 surgical interventions listed below, and the authors concluded that no surgical technique is better than the other for the treatment for cervicogenic headache associated with a cervical spondylotic myelopathy or cervical radiculopathy (Sun et al 2015).

1. Anterior cervical discectomy and fusion
2. Total disc replacement
3. Open door laminoplasty

Physiotherapy, relaxation techniques, and psychotherapy are other modalities that can be selectively considered.

Table 5. Invasive Procedures for Cervicogenic Headache

- Nerve blocks
- Cryo analgesia
- Radiofrequency procedure
- Operative procedure
- Electrical stimulation
In a randomized double-blind, placebo-controlled crossover study by Linde and colleagues, 28 adult patients with long-standing and treatment-resistant cervicogenic headaches were treated with injections of either onabotulinum toxin A or placebo given in fixed sites in the neck muscles on the painful side (Linde et al 2011). There were no significant differences favoring the use of onabotulinum toxin A in neck muscles in cervicogenic headache. However, onabotulinum toxin A is effective in chronic migraine with neck pain (Dodick et al 2010).

Patients suffering from primary cough headache tend to respond to indomethacin. Patients with Chiari I malformations who have minimal or equivocal symptoms without syringomyelia can be treated conservatively with analgesics, muscle relaxants, and occasional use of a soft collar. Frankly symptomatic patients should be offered surgical treatment aiming to decompress the cervicomedullary junction and restore normal CSF flow in the region of the foramen magnum (Pascual 2005). Reconstructive suboccipital craniectomy can relieve pain in cough headache due to Chiari type I malformation (Pascual et al 1992; Pascual et al 1996).

Treatment options for dystonia currently include physical, medicinal, and surgical intervention. Occupational therapies can be used to improve daily living activities, such as swallowing or speaking (ie, in the case of lingual or laryngeal dystonia). Anticholinergics can be administered to limit hyperactivity in the muscles. Local injections of botulinum toxin have brought a dramatic improvement in this condition, with success in more than two thirds of patients with segmental dystonia (Adam and Jankovic 2007). In severe cases, surgical interventions, such as deep brain stimulation surgery, have proven effective and work by altering the stimulation of the basal ganglia circuitry (Bittar 2005). By improving muscle pain and pressure caused by cranio cervical dystonia, the subsequent headache should presumably subside.

Retropharyngeal tendonitis is managed with nonsteroidal anti-inflammatory medications. Surgical drainage may be attempted, but is difficult and often not needed (Eastwood et al 1998). Additionally, steroids may be prescribed as well as opiate analgesics while the pain is present (Park et al 2010). These therapies are prescribed to decrease the inflammation and retropharyngeal space, leading to a decrease in neck pain and pressure, and subsequent headache.

Patients with acute whiplash usually respond to ice for 24 hours followed by heat applications, analgesics or NSAIDs, and muscle relaxants. Cervical collars, active physiotherapy, and traction should be avoided in the acute phase (Bogduk 1997; Gobel and Edmeads 2000; Gobel and Edmeads 2006; Edmeads 2001; Haldeman and Dagenais 2001). Physical therapy, local steroids, botulinum toxin, acupuncture, and transcutaneous nerve stimulators can be added for pain control (Freund and Schwartz 2000; Haldeman and Dagenais 2001).

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**References especially recommended by the author or editor for general reading.

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**ICD and OMIM codes**

**ICD codes**

ICD-9:
- Headache: 784.0

ICD-10:
- Cervical myofascial pain: M79.1
- Cervicogenic headache: R51

**Profile**

**Age range of presentation**

- 13-18 years
- 19-44 years
- 45-64 years
65+ years

**Sex preponderance**
Female > male

**Family history**
family history may be obtained

**Heredity**
heredity may be a factor

**Population groups selectively affected**
none selectively affected

**Occupation groups selectively affected**
Musicians
Truck or bus drivers
Computer operators

**Differential diagnosis list**
tension-type headache
migraine without aura
cluster headache
hemicrania continua
migraine
anterior and posterior circulation artery dissection
neck-tongue syndrome
C2 neuralgia
posterior fossa lesions
Chiari type-I malformation
cervical radiculopathy
cervical myofascial pain

**Associated disorders**
Cervical myofascial pain
Cervical spondylosis
Migraine
Occipital neuralgia
Postconcussion headache
Syringomyelia
Tension-type headache
Whiplash injury

**Other topics to consider**
Cervical disc disease
Chiropractic manipulation: neurologic complications
Cough headache
Indomethacin-responsive headache syndromes
Migraine
Neuroimaging of headache
Neurologic complications of chiropractic manipulation