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Cervicogenic headaches: a critical review

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Abstract

Background context: The notion that headaches may originate from disorders of the cervical spine and can be relieved by treatments directed at the neck is gaining recognition among headache clinicians but is often neglected in the spine literature.

Purpose: To review and summarize the literature on cervicogenic headaches in the following areas: historical perspective, diagnostic criteria, epidemiology, pathogenesis, differential diagnosis, and treatment.

Study design/setting: A systematic literature review of cervicogenic headache was performed.

Methods: Three computerized medical databases (Medline, Cumulative Index to Nursing and Allied Health Literature [CINAHL], Mantis) were searched for the terms "cervicogenic" and "headache." After cross-referencing, we retrieved 164 unique citations; 48 citations were added from other sources, for a total of 212 citations, although all were not used.

Results: Hilton described the concept of headaches originating from the cervical spine in 1860. In 1983 Sjaastad introduced the term "cervicogenic headache" (CGH). Diagnostic criteria have been established by several expert groups, with agreement that these headaches start in the neck or occipital region and are associated with tenderness of cervical paraspinal tissues. Prevalence estimates range from 0.4% to 2.5% of the general population to 15% to 20% of patients with chronic headaches. CGH affects patients with a mean age of 42.9 years, has a 4:1 female disposition, and tends to be chronic. Almost any pathology affecting the cervical spine has been implicated in the genesis of CGH as a result of convergence of sensory input from the cervical structures within the spinal nucleus of the trigeminal nerve. The main differential diagnoses are tension type headache and migraine headache, with considerable overlap in symptoms and findings between these conditions. No specific pathology has been noted on imaging or diagnostic studies which correlates with CGH. CGH seems unresponsive to common headache medication. Small, noncontrolled case series have reported moderate success with surgery and injections. A few randomized controlled trials and a number of case series support the use of cervical manipulation, transcutaneous electrical nerve stimulation, and botulinum toxin injection.

Conclusions: There remains considerable controversy and confusion on all matters pertaining to the topic of CGH. However, the amount of interest in the topic is growing, and it is anticipated that further research will help to clarify the theory, diagnosis, and treatment options for patients with CGH. Until then, it is essential that clinicians maintain an open, cautious, and critical approach to the literature on cervicogenic headaches.

Keywords: Cervicogenic; Headache; Review

Introduction

It is common in clinical practice to encounter conditions that are widespread and routinely treated but that suffer

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from limited research and lack of consensus among experts. Cervicogenic headache (CGH) is one of these conditions. Although the idea that headaches can originate from structures in the neck and can be treated by interventions directed at the cervical spine is long-standing, it is only in the past two decades that the topic has gained attention in the mainstream headache and pain literature. There are now several associations dedicated solely to studying CGH, including the Cervicogenic Headache International Study Group and the World Cervicogenic Headache Society (WCHS). These societies are comprised mainly of neurologists and pain management and headache specialists. Journals and professional associations devoted to studying the spine, however, have not participated in this development. This is unfortunate, because the majority of spinal pain syndromes are managed by clinicians who treat the spine rather than those who treat headaches.

This problem of headaches related to the cervical spine cannot be underestimated. Up to 80% of patients with cervical acceleration-deceleration injuries report headaches within 2 months of injury [1]. Almost 25% of patients with this form of injury continue to have significant neck pain 2 years later, with the majority also complaining of headaches [2]. These figures do not by themselves confirm that the headaches noted in these studies originate from the cervical spine. Many patients with whiplash injuries to the neck are also under financial and litigation stresses, and their headaches may be the result of muscle tension. Furthermore, these patients commonly take medications with the potential of causing headaches. To make the issue more complicated, many of these patients have also had head injuries that may be the primary cause of their headaches. It is therefore not sufficient to assume that patients with complaints of both neck pain and headaches after injury have headaches that are being caused by the same pathology that is causing the neck pain. It is up to the clinician to be aware of the current literature in order to be able to make a reasonable diagnostic effort to differentiate between the various types of headache that may accompany neck pain.

This article is an attempt to assist the spine specialist in understanding the current literature on CGH. With this review it is hoped that a clinician will be able to discuss the current state of knowledge and be aware of the controversies concerning CGH, as well as place the multiple theories and treatment approaches for this condition in some perspective.

The literature search

A search was performed with three computerized medical databases (Medline, CINAHL, Mantis) for the terms “cervicogenic” and “headache” for the periods covered by each database (1966 for Medline, 1982 for CINAHL, 1880 for Mantis). The search was limited to articles in English, French, and German. Search results from the different databases were merged using reference-managing software, and duplicates were eliminated. The reference section of each article was then searched for relevant articles not uncovered by the computerized databases. Although no formal search strategy was attempted for books, theses, and presentation abstracts, these were included when articles uncovered by our search made reference to them. This strategy yielded 212 papers, some of which were not relevant to our review. These results encouraged us to pursue our original goal of reviewing the literature pertaining specifically to CGH rather than attempt to cover the much broader topic of headaches and neck pain. Because the term CGH was introduced in 1983, this search was mainly limited to papers published within the last two decades, although additional papers were added when relevant and important to the discussion. Although this may seem too restrictive, we thought that this type of review could reduce some of the confusion surrounding this topic by excluding older literature where it is impossible to determine if the authors are in fact describing CGH or a different condition.

The information gleaned from our literature review was divided into the following six categories for analysis: historical perspective; definition and diagnostic criteria; epidemiology; pathogenesis; differential diagnosis; and treatment.

Historical perspective

Table 1 highlights some of the historical milestones in the evolution of the concept of CGH. The earliest reference we uncovered was a series of lectures given by Hilton in the period 1860–1862, as reported by Pearce [3]. In those lectures, Hilton proposed that pain in the anterior or lateral part of the head may come from the great or small occipital nerve, most likely from disease between the first and second vertebrae. Sixty years later, in 1926, Barré [4] hypothesized a relationship between the cervical spine and neurological symptoms, including headache and vertigo. His collaborator, Lieou [5], stated in 1928 that cervical arthritis should be considered a common cause of these symptoms. Twenty years later, Raney and Raney [6] reported that headache may be a common symptom of cervical disk lesions. The following year, a case series published by Hunter and Mayfield [7] reported that occipital neuralgia, where pain radiated from the occiput to the periorbital and jaw areas, could be an important cause of headaches. This theory, in turn, was used to justify the injection of analgesics into the occipital nerves in an attempt to relieve these headaches. Also in 1949, Bärtschi-Rochaix [8] used the term “cervical migraine” to describe headaches presumed to come from the neck, while Josey [9] published a case series on patients with headaches associated with pathologic changes in the cervical spine. In 1955 Kovacs [10] wrote that motion restriction in the cervical spine could lead to muscle spasm and compromise of the vertebral artery and nerves, causing headaches. This helped popularize osteopathic, chiropractic, and manual treatment of the cervical spine to relieve headaches. Maigne [11] was a prominent advocate of using ma-

Table 1
Historical milestones in cervicogenic headaches

Year	Event
1860–1862	Hilton mentions that pain in the anterior or lateral part of the head may come from the great or small occipital nerve, most likely from disease between the first and second vertebrae.
1926	French neurologist Barré describes a relation between the cervical spine and neurological symptoms, including headache and vertigo.
1928	French neurologist Lieou reports that cervical arthritis is a common cause of headache and vertigo.
1948	Raney describes patients with headaches caused by cervical disk lesions.
1949	Hunter and Mayfield publish a series of case reports on occipital neuralgia.
1949	Bärtschi-Rochaix coins the term "cervical migraine."
1949	Josey publishes a case series where headaches are associated with pathologic changes in the cervical spine.
1955	Kovaks postulates that motion restriction in the cervical spine can lead to muscle spasm and compromise of the vertebral artery and nerves causing headaches.
1961	Grillo, a chiropractor, discusses vertebragenous headaches very similar to CGH.
1973	Bogduk discusses third occipital headache with a presentation very similar to CGH.
1981	French physiatrist Maigne reports success in treating headaches with manual medicine.
1983	Sjaastad publishes an article introducing the term cervicogenic headache.
1987	Fredriksen presents a detailed description of the clinical presentation of patients with CGH.
1988	The International Headaches Society introduces a category for headaches associated with disorders of the neck.
1990	Sjaastad publishes diagnostic criteria for CGH.
1994	The International Association for the Study of Pain publishes diagnostic criteria for CGH.
1994	The multidisciplinary World Cervicogenic Headache Society is created.
1995	The Quebec Headache Study Group publishes diagnostic criteria for CGH.
1995	The first randomized controlled clinical trial for CGH is published by Nilsson.
1998	Sjaastad publishes revision of diagnostic criteria for CGH.

CGH = cervicogenic headache.

nipulation to treat headaches, while Bogduk and Marsland [12] advocated surgical intervention to treat what they termed "3rd occipital headache".

The term "cervicogenic headache" was first introduced to the medical literature in 1983 by Sjaastad et al. [13], who described patients with a headache not classified by diagnostic criteria at that time. In 1987 Fredriksen et al. [14] gave a more detailed description of patients they had diagnosed with CGH. In 1988 the International Headache Society (IHS) [15] amended its diagnostic classification system to include a category for headaches associated with disorders of the neck. In 1990 Sjaastad et al. [16] published very specific and detailed diagnostic criteria for CGH. This was followed by the publication of less stringent diagnostic criteria for CGH by the International Association for the Study of Pain (IASP) in 1994, and by the Quebec Headache Study Group in 1995 [11,17]. In 1998, Sjaastad et al. [18] revised their diagnostic criteria for CGH based on more extensive clinical research.

Definition and diagnostic criteria

The term CGH, although adopted by a number of organizations, is not universally accepted, and there remains a great deal of variation in the terminology used to discuss headaches associated with disorders of the cervical spine. This is especially true of literature before 1983, when a number of terms (Table 2) appear to have referred to the same clinical entity. Many of these terms, such as vertebragenous [19], vertebrogenic [20], or spondylotic [21] headaches, can be considered synonymous with CGH, at least in their connotation. Such terms as neuralgia, especially those referring to a specific nerve, assume, with little evidence, that the origin of the headache is known [12,22]. Other terms are more vague, referring simply to a syndrome or a specific location of the headache [23–26].

The confusion seen in CGH terminology is also apparent when examining the diagnostic criteria for CGH. Table 3 summarizes the prominent features of the diagnostic criteria published by various expert groups. The most widely used diagnostic criteria for many years were those proposed by Sjaastad [16] in 1990 and subsequently updated in 1998 [18]. Although the publication of these criteria brought focus to the field of CGH research, certain aspects have proved difficult to embrace. For example, Sjaastad vigor-

Table 2
Conditions similar to cervicogenic headache

Terms used	Reference
Rheumatic headache	[83]
Great occipital-trigeminus syndrome	[84]
Vertebragenous headache	[19]
Greater occipital neuralgia	[22]
Cervical migraine	[24]
Cervical spine syndrome	[23]
Spondylotic headache	[21]
Cervicogenic cephalalgia	[85]
Cervical headache	[25]
Occipital headache	[72]
Occipital myalgia-neuralgia syndrome	[86]
Third occipital headache	[12]
Vertebrogenic headache	[20]
Cervicogenic syndrome	[26]
Spondylogenic headache	[87]

Table 3
The characteristics and definitions of cervicogenic headache

Criteria/group	International Headache Society [15]	Cervicogenic Headache International Study Group [18]	Quebec Headache Study Group [11]	International Association for the Study of Pain [17]	World Cervicogenic Headache Society [28]
Subjective					
Location of pain (region)	Neck, occipital	Starts in neck Ipsilateral, vague, nonradicular neck, shoulder, arm pain or radiculopathy	Occipital	Starts in neck/occiput Forehead Temporal Whole hemicranium	Neck, occipital Parietal-temporal Frontal Orbital
Pain characteristics	—	Unilateral without sideshift Moderate–severe Nonthrobbing Nonlancinating	Unilateral or bilateral	Unilateral without sideshift Becomes more continuous Moderately severe Varying duration	Unilateral or bilateral Dull, aching Nonlancinating
Pain increased with	Neck movement Posture	Neck movement Awkward head positioning Pressure over ipsilateral cervical/occipital area	—	Neck movement	Neck movement
Objective					
Cervical spine range of motion	Decreased passive range of motion	—	—	—	Impaired
Palpation findings	Tender neck muscles Change in neck muscle properties	—	Pain on C2/C3 facet palpation C2/C3 dermatome cellulalgia	—	Neck tenderness Identification of neck source of pain
Response to blockade	—	Occipital nerves, facets, or nerve roots abolish pain	—	Occipital nerves or nerve roots relieve pain	Relief of pain after blockade of cervical nerves
Radiologic findings	Flexion/extension abnormalities Fracture Congenital anomaly Tumor/rheumatoid arthritis, not spondylosis	—	Normal or arthrosis	—	—
Neck trauma	—	Yes	—	—	Yes
Other	—	Nausea/vomiting Edema/flushing Dizziness Phono/photophobia Blurred vision Dysphagia No effect with indomethacin, ergotamine, or sumatriptan	—	—	—

ously advocated the position that these headaches should be strictly unilateral, whereas others have accepted that these headaches may be unilateral or bilateral. Sjaastad also included a number of accompanying symptoms, such as nausea, vomiting, flushing, dizziness, phono- and photophobia, blurred vision, and dysphagia, making his criteria too specific and detailed for general practice.

Lack of consensus is also evident in the criteria of the three other main expert groups. For example, the IHS included radiological abnormalities in their diagnostic criteria, despite a failure to identify radiographic abnormalities specific to CGH [15,27]. The IASP and WCHS focused their attention on the relief of pain by the injection of analgesics into cervical structures with no convincing clinical trials to support this position and no consensus regarding the various injection techniques [17,28].

Despite these differences, certain features are common to the majority of the diagnostic criteria for CGH. There is

agreement that these headaches start in the neck or occipital area and can then spread to other areas of the head, including the frontal, temporal, and periorbital regions. The pain tends to be dull, nonthrobbing, and nonlancinating, and can become moderate to severe in intensity. Examination reveals tenderness and abnormal palpatory findings in the cervical paraspinal tissues, as well as possible decreased cervical range of motion. The other reported findings and characteristics of CGH appear to be less well defined.

Epidemiology

There is a great deal of variation in the perceived prevalence of CGH. The published prevalence rate estimates uncovered through our literature search are presented in Table 4. These ranged from 0% of patients with migraine headache (MH) [29] to 80% of patients with headache [30]. There are several reasons for this wide range of published estimates, including vastly different populations of subjects. In the general population, for example, prevalence rates ranged from 0.4% to 2.5% [31,32], whereas studies looking at all patients with a complaint of headache reported estimates of 15% to 20% [15,32–34]. The highest variation was among headache center patients, with prevalence estimates of 0.4% to 80%. Part of this variation can be attributed to the different methodology used in these studies (i.e., prospective cohort, retrospective analysis, etc.), as well the different diagnostic criteria used to define CGH. Several studies did not specify the criteria used to define CGH, making direct comparisons impossible; even studies using the same criteria varied in the stringency with which these were applied (i.e., patient must fulfill a minimum of *x* criteria to be included). The study reporting the highest prevalence for CGH was by Rothbart [30], a clinician from a pain management center, who estimated that 80% of the patients with headache in his clinic had CGH. As founder of the WCHS, he may have spent considerably more time than other physicians in seeking patients with this syndrome. An incidental finding in a study by Loh et al. [35] reported that 10% of patients with obstructive sleep apnea were diagnosed with CGH, although no explanation was offered as to the mechanism of action.

Analysis of patient descriptive data (age, gender, etc.) from studies where such information was given reveals that patients with CGH appear to form a fairly homogeneous population, with a mean age of 42.9 years, a gender distribution that is 79.1% female and 20.9% male, and a mean duration of symptoms of 6.8 years. More detailed demographic data were found in a study by Shah and Nafee [36] in India, where patients with CGH were described as 43% urban and 57% rural, with 55.7% employed as handicraft workers, 28.3% as laborers, 10.0% as clerks, 4.9% as business executives, and 1.6% as doctors. Shah and Nafee speculated that the poor ergonomics associated with the handi-

Table 4
Prevalence estimates for cervicogenic headache

Population	Criteria	Prevalence (%)	Reference
Migraine patients	Sjaastad	0.0	[29]
Headache center patients	IHS	0.4	[88]
Headache center patients	Sjaastad	0.7	[89]
General population	IHS	0.4	[31]
	Sjaastad (6 of 6)	1.0	
	Sjaastad (5 of 6)	4.6	
Headache center patients	Not specified	1.5	[90]
Headache patients	IHS	2.08	[36]
General population 20–59 years old	IHS	2.5	[32]
Chiropractic patients	Not specified	3.33	[72]
Whiplash patients	Not specified	8% after 8 weeks 5% after 6 months 3% after 1 year	[31]
Obstructive sleep apnea patients	Not specified	10	[35]
Chiropractic patients ages 12–24	Not specified	13.3	[73]
Headache center patients	Not specified	13.8	[40]
Headache center patients	Not specified	15	[91]
Headache patients	Not specified	15.8	[33]
Idiopathic headache patients	Own criteria	36.2	[34]
Frequent headache patients (>5/month) ages 20–59 years	IHS	17.8	[32]
Recurrent benign headache patients	IHS	15–20	[92]
Chiropractic patients	IHS	22.5	[48]
Patients with degenerative cervical spine disease	Not specified	39.1	[38]
Unilateral headaches without sideshift starting in neck and spreading to frontal area	Sjaastad	47	[39]
Whiplash patients	Sjaastad	54.3	[93]
Side-locked unilateral headaches or headaches starting in the neck	Five or more Sjaastad criteria	79	[37]
Headache patients	Not specified	80	[30]

IHS = International Headache Society.

craft occupations may account for the higher prevalence in that group. They also reported that the population in his study had a mean age of onset of 62.5 years for CGH, which is considerably older than the mean age of typical patients with CGH reported above.

Another factor influencing prevalence rates in headache centers is the apparent overlap between the diagnosis of CGH, tension-type headache (TTH), and common migraine headache. Bono et al. [37] report that 75% of patients fulfilling IHS criteria for MH also meet most of the criteria for CGH. Furthermore, the CGH diagnostic criteria by Sjaastad et al. [16] include many of the systemic symptoms, such as nausea, vomiting, phonophobia, and photophobia commonly seen in MH. CGH also appears to frequently co-exist with primary headache disorders such as MH and TTH. One study of headache center patients reported that whereas only 16.1% were diagnosed with CGH, an additional 20.1% were diagnosed with both MH and CGH, for a total prevalence of 36.2% [34]. A recent study of patients with neck injuries reported that 34.3% had CGH, whereas an additional 11.4% had both CGH and MH, and an additional 8.6% had CGH in combination with headaches associated with the neck, for a total prevalence of 54.3% [38]. The reason for distinguishing CGH from headaches associated with the neck was unclear. When narrowing the field among patients with headache to those with unilateral pain without sideshift and pain starting from the neck and spreading to the oculofrontal area, Bono et al. [39] diagnosed 47% of such patients with CGH, including 15% where there was overlap between MH and headaches associated with the neck, although again the distinction of the latter from CGH was unclear. Another study reported that 56.4% of CGH diagnoses occur in combination with other headaches, including MH, TTH, and drug-induced headache [40]. Although Sjaastad has tended to disagree that CGH symptoms are commonly found in patients with MH, he and Bovim [41] reported on four patients where both MH and CGH co-existed. These patients were able to distinguish between episodes of each headache, reporting improvement of MH but not CGH with sumatriptan and ergotamine, and relief of CGH but not MH with greater occipital nerve anesthetic blockade.

Pathogenesis

One of the most controversial areas within the CGH literature is the discussion of its cause. Almost every structure and pathology within the cervical spine has been implicated as a cause of these headaches. Table 5 summarizes the structures suggested as the origin of CGH and the types of pathology associated with these headaches. The rationale for most of the theories is the observation, usually in a small number of cases, of either a reproducible finding on clinical examination, a response to stimulation of the structure, or relief of symptoms after treatment directed at the structure. Examples include the response of patients to surgery for disk disease [42], injections of posterior facets with anesthesia [43], and injections of cervical muscles with botulinum toxin [47].

One theory of CGH etiology comes from anatomical studies showing an attachment of the suboccipital tissues to the dura mater at the cervical–cranial junction, and the observation that mechanical traction on these tissues can cause movement of the dura [48–51]. The rectus capitus posterior minor muscle [51] and ligamentum nuchae [50] have been shown to have direct connections to the suboccipital dura on very delicate dissection in a small number of cadavers. This suggests a role for the dura as a nociceptive structure in CGH.

Structures implicated in the genesis of CGH all have their sensory innervations through the upper cervical and

Table 5
Theories of pathogenesis for cervicogenic headache

Structure	Pathology	Mechanism	Reference
Zygapophysial or facet joint	Irritation	Trauma or immobility stimulates the C1–C3 nerves	[104]
	Rheumatoid arthritis		[52]
			[11]
Cervical muscles	Myofacial trigger points, myospasm	Restrict joint motion. Referred symptoms from muscles innervated by C1–C3	[105]
			[47]
			[58]
Intervertebral disk	Trauma	Irritates the dura	[52]
	Herniation	Stimulates sinuvertebral nerve	[106]
Nerve roots	Compression	Disk herniation, spondylosis, or scar tissue	[19]
	Irritation		[107]
Vertebral artery	Compress	Apophyseal exostoses impacting vertebral artery blood flow	[8]
Uncovertebral joints	Mechanical irritation	Nerve roots producing sternocleidomastoid and trapezius muscle spasm	[108]
Ponticus posterior	Articular lock, instability	Tension on the dura or vertebrobasilar artery compression	[48]
Rectus capitis muscle	Connective tissue bridge with the dura	Tension on the dura	[49]
			[51]
Ligamentum nuchae	Attaches to the dura	Tension on the dura	[50]

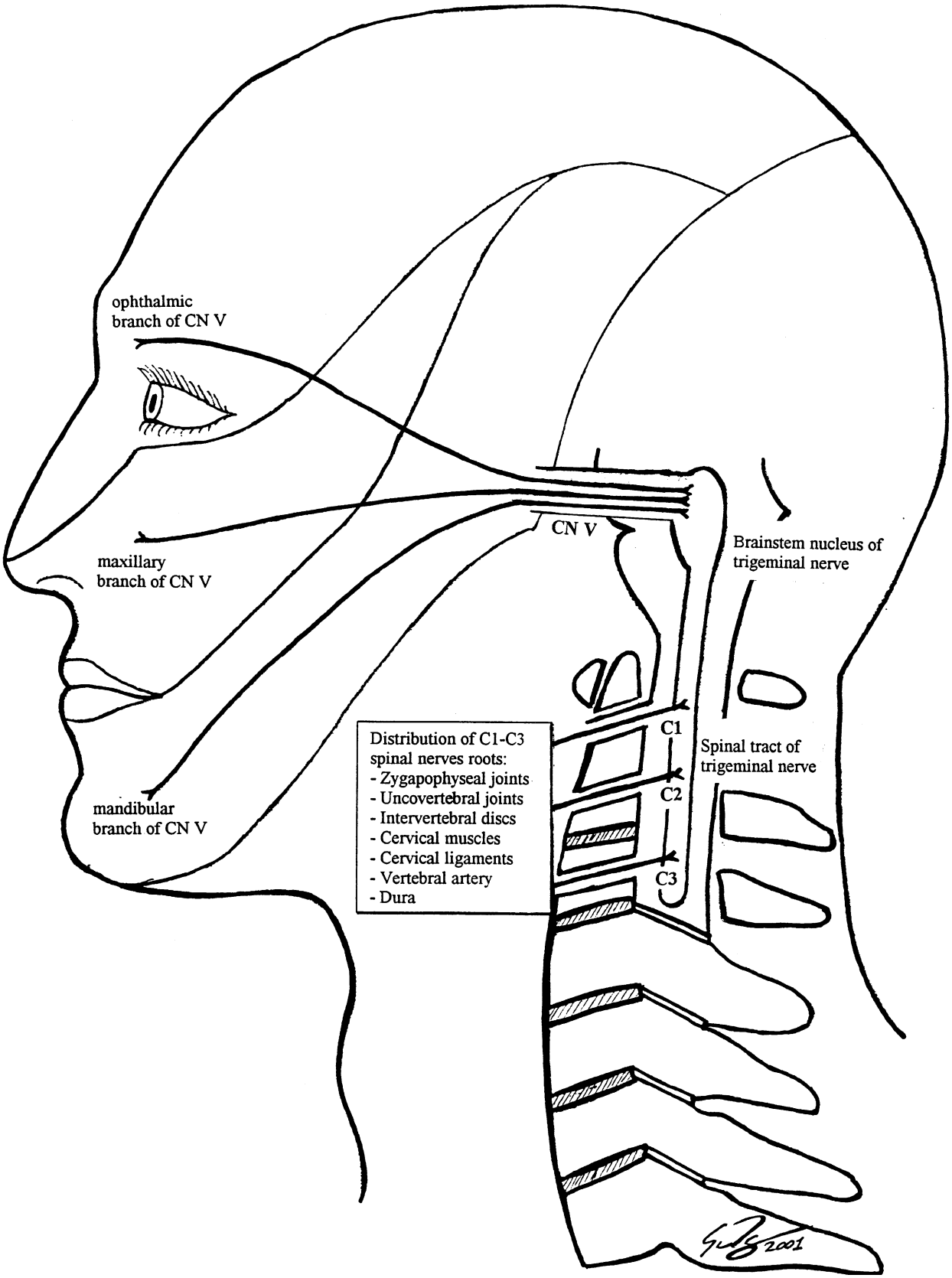

Fig. 1. Convergence of sensory input from the upper cervical nerve roots into the trigeminal nucleus.

midcervical nerve roots, which lead to the cervical cord and converge within the spinal tract of the trigeminal nucleus [49] (Fig. 1). This allows nociceptive input from cervical structures to be perceived as head pain, including pain to the temporal, frontal, and orbital regions. This convergence may also help to explain the systemic and sympathetic nervous system features accompanying CGH. Studies showing relief of headache after lower nerve root blocks have cast a doubt on whether only the upper nerve roots are significant in CGH [43,44].

Martelletti has reported increased levels of pro-inflammatory cytokines interleukin-I β and tumor necrosis factor-α during mechanically induced attacks of CGH; these were significantly higher than in patients with MH [45,46]. He postulated that this could represent a specific signal from the immune system to activate such pain-producing agents as substance P and calcitonin-gene–related peptide. This may help define CGH as an inflammatory consequence of cervical trauma, explaining the wide variety of pathological processes in different structures that can cause similar headaches.

The inability to find a definitive structure or pathology as the cause of CGH has lead some to believe that CGH does not represent a single pathological entity but rather a pain syndrome resulting from the nociceptive stimulation of almost any structure in the cervical spine [52].

Differential diagnosis

The differential diagnosis stressed in most of the literature on this topic is between CGH, MH, and TTH. It is generally assumed that intracranial pathology from infection, neoplasm, trauma, and so forth has been ruled out. Headaches associated with sinusitis, temporomandibular joint syndrome, visual or auditory disturbances, and cluster headache are rarely confused with CGH, because each possesses unique distinguishing characteristics. To aid the task of differential diagnosis, several studies have reported the results of various radiographic, neurologic, and physiologic testing in patients with CGH.

The significance of radiological findings in CGH has been difficult to establish (Table 6). Only one of these studies used a control group [53], and most had a small number of subjects. These shortcomings make it difficult to draw conclusions regarding the relationship of radiological findings and CGH. Although degenerative changes have been found in patients with CGH on plain film radiography and magnetic resonance imaging scans of the cervical spine, these changes cannot be considered specific and unique to CGH [27,36,54,55]. A study by Jansen [54] in 1998 found that 100% of patients with CGH had radiographic evidence of retrospondylosis and osteochondrosis. With multiple spinal levels involved (42.9% at C5–C6, 22.7% at C4–C5, 21.4% at C6–C7, 11.0% at C3–C4, 0.6% at C7–T1, and only 1.3% at C2–C3), it is difficult to assess the importance of this finding. The description of Chiari type 1 malformations and spinal cord compression in small numbers of patients with characteristics of CGH may simply represent pathology that causes headaches by stimulating cervical structures rather than a finding specific to CGH.

Table 7 summarizes the results of various diagnostic tests on patients with CGH. Although many of these studies report statistically significant findings, the number of subjects is too small to reach any conclusion. Many of the physiological tests, such as sweating patterns [56] and electronystagmography [57], are so esoteric that it is difficult to determine their significance or relevance in clinical practice. The finding of various forms of muscle dysfunction, such as myofascial trigger points [58], responses of differ-

Table 6
Diagnostic imaging studies for CGH

Evaluation	% Abnormal	Abnormalities	Number of patients	Reference
CS radiography	100	Retrospondylosis Osteochondrosis	87	[54]
CS radiography	100	Not specified	61	[36]
CS radiography	55	Uncovertebral arthrosis Osteophytic protrusion	11	[27]
Flexion/extension radiography	$P>.05$	Significant ↓ C0–C5 mobility Significant ↑ C6–C7 mobility	15	[53]
Skull/sinus/mandible radiography	0	—	11	[27]
Cervical myelography	50	Disk protrusion Uncovertebral spondylosis	6	[27]
Cerebral angiography	0	—	6	[27]
Cerebral CT	9	Chiari type I malformation Arachnoidal cyst	11	[27]
Cervical CT	9	Reduced cord diameter	11	[27]
MRI	100	Retrospondylosis Disk protrusion Ventral dura compression Narrowed subarachnoid space	8	[55]
MRI	100	Spondylosis	6	[36]
Orbital phlebography	0	—	12	[97]

CS = cervical spine; CT = computed tomography; MRI = magnetic resonance imaging.

Table 7
Neurological and other diagnostic studies for CGH

Evaluation	% Abnormal	Abnormalities	Number of patients	Reference
Perception of verticality	10	Deviation to left	14	[57]
Posturography	8	Romberg quotient 3.1 for anterior posterior sway	14	[57]
Pupillometric examination	0	—	11	[108]
Pilocarpine stimulation	0	—	11	[56]
Heat-induced sweating	$P<.05$	Significant difference in CGH	11	[56]
Forehead sweating	$P<.05$	Low during spontaneous attacks of CGH	11	[56]
Trigeminocervical reflex	$P<.05$	Significant ↓ amplitude on painful side	13	[109]
Greater occipital nerve electrical stimulation	$P<.05$	Abnormal in CGH but not controls	13	[109]
Masseter activity suppression	0	—	13	[109]
Muscle dysfunction	$P<.05$	Significant ↑ upper trapezius passive stretching response vs. controls	30	[104]
Myofascial trigger points	$P<05$	Significant more on symptomatic (70) vs. asymptomatic (22) side	11	[58]
C2–C4 instantaneous axis of rotation	$P>.05$	No relation between abnormality and headache	54	[110]
Electronystagmography	31	Spontaneous nystagmus Asymmetry in rotatory nystagmus Congenital cranial nerve VI palsy Caloric hyporesponsiveness	14	[57]
Response to mental load on shoulder, neck, and facial muscles	$P<.05$	Significant ↓ shoulder maximum conduction velocity in CGH Significant ↑ pretest activity in frontalis on symptomatic side Significant ↑ electromyography response in trapezius on symptomatic side	17	[59]
Head posture	$P<.05$	↑ Forward carriage vs. controls	60	[111]
Cervical flexor endurance	$P<.05$	↓ Endurance vs. controls		
Cervical flexor strength	$P<.05$	↓ Strength vs. controls		
Response to:			27	[61]
Nitroglycerine	$P<.05$	Significant ↓ response in CGH for all medications vs. cluster headache		
Oxygen				
Ergotamine				
Morphine				

CGH = cervicogenic headache.

ent muscle groups to mental stress [59], and cervical muscle strength and endurance [60], seems to confirm the involvement of cervical and paraspinal muscles in CGH. The lack of response to common vasoactive medications used in MH [61] argues against arterial involvement in CGH and can be one of the clues that a patient may have a CGH.

Attempts to differentiate CGH from MH and TTH on the basis of some clinical or experimental measure are presented in Tables 8 and 9. It does not appear that any specific test or clinical finding can be used to define patients with CGH. A detailed clinical history is therefore imperative in order to diagnose CGH. The difficulties of diagnosing CGH were stressed by Sjaastad and Bovim in 1991 [41], when they compared CGH with MH and reported that CGH fulfilled seven of eight common MH criteria (the exception being aggravation by physical activity). Nausea and/or vomiting was reported in 55% of patients with CGH versus 70% to 85% for MH. Photophobia was reported in 45% of patients with CGH versus 88% of patients with MH. Patients with MH, however, did not fulfill the most important criteria for CGH, that is, precipitation of headaches with neck movements and/or external pressure on the neck. A second study by Sjaastad et al. [63] in 1992 compared pain patterns in CGH and MH and reported that whereas typical MH attacks were unilateral without side shift (the typical CGH pattern) in only 16% of patients, 75% of nontypical MH at-

Table 8
Comparison of CGH with tension-type headache

Evaluation	Findings	Number of patients	Reference
Skin roll test	Pain values ↑ in CGH	73	[93]
Response to greater occipital nerve blockade	Pain reduction 54.5% CGH vs. 14% TTH	52	[96]
Response to supraorbital nerve blockade	No significant difference	52	[96]
Pressure-pain threshold	Significant ↓ in CGH	95	[95]
R1/R2 blink reflex latency	No significant difference	41	[112]
Photophobia, phonophobia	Both groups had lower thresholds than controls	166	[113]
Temporalis muscle activity	No significant difference	41	[114]
Neck mobility	Significant ↓ flexion/extension and rotation in CGH	139	[114]

CGH = cervicogenic headache.

Table 9
Comparison of CGH with migraine headache

Evaluation	Findings	Number of patients	Reference
Skin roll test	Pain values ↑ in CGH	73	[93]
Thermal sensitivity	Significant ↑ thermal thresholds on both sides	103	[94]
Pressure-pain threshold	Significant ↓ in CGH	95	[95]
Response to supraorbital nerve blockade	No significant difference	52	[96]
Response to greater occipital nerve blockade	Significant ↑ pain reduction in CGH (54.5%) vs. MH (6%)	52	[96]
R1/R2 blink reflex latency	No significant difference	41	[112]
Temporalis muscle activity	No significant difference	41	[114]
Neck mobility	Significant ↓ flexion/extension and rotation in CGH	139	[114]
Localization of the initial pain	88% of MH have initial pain anteriorly 79% of CGH have initial pain posteriorly	61	[100]
Localization of the initial pain	77% of MH have initial pain in temples 73% of CGH have initial pain posteriorly	61	[115]

CGH = cervicogenic headache; MH = migraine headache.

tacks presented in this fashion. Their group of patients with CGH fulfilled only 3.79 of 7 IHS criteria for common MH criteria, compared with 6.78 for patients with MH.

A study by Vincent and Luna in 1999 [62] examined the number of patients with CGH, TTH, and MH who could fulfill Sjaastad's criteria for CGH. Patients with CGH met 10.51 of 18 criteria versus 3.85 for patients with MH and 4.89 for patients with TTH, a statistically significant difference. One third (33.3%) of patients with CGH met the IHS criteria for MH, whereas only 3.3% of patients with CGH met IHS criteria for TTH. In other words, 63.4% of patients with CGH could not be classified according to IHS criteria as having either MH or TTH. D'Amico et al. [64] similarly examined the characteristics of patients with headache and reported that in patients with long-lasting, side-locked, unilateral pain, the diagnosis was MH in 85.1%, TTH in 10.8%, and CGH in 4.1%. The percentage of patients with headache in whom the pain was localized in the occipitonuchal region was 100% in CGH, 12.5% in MH, and 20.0% in TTH. Conversely, the percentage of patients in whom the initial pain was nonoccipital was 0% in CGH, 76.6% in MH, and 30.0% in TTH. CGH may then be differentiated from

Table 10
Surgical treatment of cervicogenic headache

						Relief		
Reference	Procedure	Structure(s)	Study type	Follow-up	Number of patients	Complete (%)	Partial (%)	None (%)
[65]	Radiofrequency neurotomy	C2–C4 sinuvertebral nerves and roots	Case series	2–6 months	24	38	45	17
[66]	Radiofrequency neurotomy	C2 medial rami	Case series	Not specified	100	43	36	21
[67]	Radiofrequency neurotomy	Planum nachae	Case series	4.5 years	7	43	29	29
[68]	Radiofrequency neurotomy	Medial branch of C3–C6 dorsal ramus	Case series	2 months	15	7	73	20
[68]	Radiofrequency neurotomy	Medial branch of C3–C6 dorsal ramus	Case series	12–22 months	15	7	73	20
[55]	Ventral decompression	CS	Case series	Postoperative	51	85	15	0
[70]	Ventral decompression	CS	Case series	14 months	56	86	14	0
[70]	Dorsal decompression	C2–C7	Case series	5 months	8	88	12	0
[55]	Dorsal decompression	CS	Case series	Postoperative	8	75	25	—
				2–3 months	8	38	38	13
[98]	Decompression	C2 nerve root	Case series	21 months	35	36	54	10
[70]	Ganglionectomy	C2	Case series	44 months	38	68	16	16
[116]	Neurolysis	Greater occipital nerve	Case series	Postoperative	58	43	36	18
[118]	Release	RCPM attachment to dura	Case report	5 months	1	100	—	—
[42]	Robinson-Smith stabilization operation	C5–C6 disk	Case report	8 years	1	100	—	—

CS = cervical spine; RCPM = rectus capitis posterior minor.

Table 11
Manual treatment for cervicogenic headache

Reference	Treatment	Study type	Follow-up	Number of patients	Results
[119]	Spinal manipulation	Case series	4 weeks	36	Significant ↓ drug consumption index Significant ↓ total pain index
[74]	1. Spinal manipulation	RCT	1 week	38	Significant ↓ headache hours/day (group 1, 59%; group 2, 48%)
	2. Massage and laser light				Significant ↓ VAS, group 1 only (36%) Significant ↓ NSAIDs/day, group 1 only (47%)
[75]	1. Spinal manipulation	RCT	1 week	53	Significant difference in ↓ headache hours/day in favor of group 1
	2. Massage and laser light				Significant difference for analgesics/day between 2 groups
[120]	Spinal manipulation	Case series	2 weeks	26	Significant ↓ headache severity (59%) Significant ↓ headache frequency (62%) Significant ↓ headache duration (77%)
[120]	Spinal manipulation under anesthesia	Case report	3 months	1	↓ in neck and upper back pain (90%) ↓ in headaches (95%)
[121]	Upper cervical spine mobilization	Cohort	5 weeks	10	Significant ↓ headache frequency (66%) Significant ↓ headache duration (43%) Significant ↓ headache intensity (53%)
[85]	Spinal manipulation	Case report	2 months	1	Complete relief of headaches Restoration of full CS ROM
[72]	Spinal manipulation	Retrospective case series	Not specified	332	80% had >75% reduction in symptoms 10% had 50–75% reduction in symptoms 3% had 50% reduction in symptoms 2% had no change 5% had aggravation in symptoms
[123]	Manipulation, muscle re-education	Case report	6 weeks	1	↓ headache intensity (36%) ↑ CS ROM ↑ neck muscle strength and endurance
[124]	1. Manual therapy vs. 2. Maitland concept vs. 3. No-treatment control	Case series	Not specified	29	Manual therapy is better than Maitland concept Both are better than no-treatment control
[118]	Mobilization	Case report	7 months	1	Patient was headache free for 6–7 months until involved in a motor vehicle accident
[76]	1. Spinal manipulation + NSAID 2. NSAID only	RCT	3 weeks	27	A single manipulation added to NSAIDs was superior to NSAIDs only immediately after treatment but not at 3 weeks. Not statistically significant
[20]	1. Spinal manipulation 2. Mobilization 3. Wait list	RCT	3 weeks	30	Manipulation was more effective than mobilization and wait list but without statistical significance between groups

CS ROM = cervical spine range of motion; NSAID = nonsteroidal anti-inflammatory drug; RCT = randomized controlled trial; VAS = visual analog scale.

MH and TTH by a pattern of unilateral pain without sideshift, with the initial pain located in the occipital area, and failure to be classified by diagnostic criteria for other headaches.

Treatment

The type of treatment recommended to patients with CGH appears more dependent on the specialty of the treating physician than the science or research supporting it. The four treatment options generally recommended are surgery for a number of pathological entities; cervical spine manipulation; injections of various cervical structures with a variety of agents; and medication. The published literature in support of surgical intervention for CGH is listed in Table 10. The main criticisms of this literature are the small sample sizes, the marked variation in the surgical procedures used, the difference in the structures being operated on, the poor documentation of the criteria used for diagnosing CGH, and the lack of standardized outcome measures in the majority of these studies. For example, a number of papers on radiofrequency neurotomy report some improvement of symptoms in 71% to 83% of patients and complete relief in 7% to 43% of patients [65–69]. The authors, however, reported the ablation of different nerves in these studies, making it difficult to reach conclusions or compare the results. A number of papers on various decompression procedures report relief of headaches in a substantial number of patients but again have not used a standardized protocol [55,70]. The remaining papers on surgery consist mainly of isolated case reports. There are no controlled studies to support the use of any surgical procedure for the management of CGH, and current justification for surgery appears to be based solely on the anecdotal experience of the surgeon.

As mentioned above, the prevalence of CGH ranges from 3.3% to 22.5% of chiropractic patients, indicating the frequency with which these headaches are treated with manipulation [48,72,73]. A survey of primary care physicians in Australia reported that 69% of them agreed that referral to a chiropractor was appropriate for headache provoked by head/neck postures [71]. The results of studies on cervical manipulation for CGH are listed in Table 11. The results from the case series are similar to those reported after surgery but suffer from the same shortcomings. However, there are more randomized controlled trials on manipulation than any other treatment for CGH. The studies by Nilsson et al. [74,75] have been the most rigorous and demonstrated that spinal manipulation was more effective, in the short term, than massage in reducing the frequency and severity of headaches and the amount of analgesic use by patients. A study by Howe et al. [76] indicated that the addition of one cervical manipulation to nonsteroidal anti-inflammatory drug (NSAID) therapy was superior to NSAID therapy alone immediately after treatment, but this difference was lost at 3 weeks posttreatment. Bitterli et al. [21] found an advantage for cervical manipulation compared with mobilization and controls after 3 weeks of treatment, but the differences did not reach statistical significance. In an effect size analysis of randomized controlled trials on manipulation, Bronfort [77] concluded that there is moderate evidence of efficacy of cervical manipulation in the management of CGH. Similar conclusions have been reached in qualitative analyses by Hurwitz et al. [78] and Coulter et al. [79].

Table 12
Treatment with injections for cervicogenic headache

Reference	Substance	Structure	Study type	Follow-up	Number of patients	Results
[34]	Three groups:		Case series	Not specified	180	
	1. Lignocaine + methylprednisolone	GON+LON				Group 1: 90.6% had significant relief (mean 23.5 days)
	2. Lignocaine	GON+LON Intramuscular				Group 2: 84.0% had significant relief (maximum 3 hours)
	3. Methylprednisolone					Group 3: 3.0% had relief
[102]	Bupivacaine 0.5%	GON	Case series	1 week	41	41.0% reduction in 7-day VAS score after injection (significant)
						54.4% reduction in number of days with VAS >5 (significant)
[43]	Lidocaine 1%	GON	Case series	20 minutes	14	28.6% complete relief
	Bupivacaine 0.5%	C2				50% complete relief
		C3				0% complete relief
		C4				12.5% complete relief
		C5				12.5% complete relief
		C2–3 facet				22.2% complete relief
[110]	Lignocaine 2% or Bupivacaine 0.5%	Third occipital nerve	Case series	Not specified	63	58.7% positive response 41.3% negative response
[39]	Lidocaine 0.5%	SON	Case series	30 minutes	32	69% has VAS ↓ >50%
		GON				72% had VAS ↓ >50%
		C2				69% had VAS ↓ >50%
		C3				50% had VAS ↓ >50%
		C2–3 facet				56% had VAS ↓ >50% (only 16 patients for facet)
[54]	Carbostesin 0.5%	Epidural space of affected level (C3–T1)	Case series	Not specified	87	86.2% painfree post injection 64.4% painfree for hours 13.8% painfree for days 8.0% painfree for weeks
[44]	Epidural corticosteroid injection	Epidural space of C6–T1	Case series	4 weeks	9	Significant ↓ pain numeric intensity scale Significant ↓ drug consumption index
[47]	1. Botulinum toxin A injection	Cervical trigger points	RCT	4 weeks	26	Group 1:Significant ↓ pain and ↑ range of motion compared with baseline
	2. Saline					Group 2: no change No between-group analysis
[124]	Botulinum toxin	Trapezius tender area	Case report	3 months	1	Headache frequency ↓ >50% Autonomic symptoms disappeared Full range of motion was restored in neck and shoulder
[80]	Intracutaneous sterile water and saline injections	Various tender points	Cross-over cohort	26 days	10	No significant benefit for either treatment
[80]	Sterile water Saline	Various tender points	Cohort study	2 weeks	10	No significant relief from either group

GON = greater occipital nerve; LON = lesser occipital nerve; RCT = randomized controlled trial; VAS = visual analog scale.

Another common treatment approach for CGH is therapeutic injections. The results of injection of various agents and anesthetics on CGH are listed in Table 12. There are a number of small case series on the injection of the occipital nerves where short-term improvement of was noted in 50% to 90% of patients [34,43,54]. Again, these studies suffer from the same shortcomings as those on surgery and manipulation, and many reported only immediate postinjection results with no follow-up period. One study [34] compared lignocaine, lignocaine with methylprednisolone, and methylprednisolone alone in a nonrandomized case series and found that methylprednisolone was less effective than lignocaine and did not add anything to the injection of lignocaine alone. This finding does not support Martelletti's theory about the role of inflammation in CGH [45,46]. Two relatively good cohort studies on the injection of sterile water and saline into tender points in cervical muscles failed to show any improvement of symptoms [80]. A small case series on epidural corticosteroid injection reported some degree of relief [81]. One intriguing study was a randomized controlled trial comparing botulinum toxin with saline injection into the cervical paraspinal muscles, which found a significant decrease in pain and increased cervical spine range of motion in the botulinum group [47].

Among other treatments for CGH (Table 13) we found one randomized controlled trial on the use of transcutaneous electrical nerve stimulation [82] suggesting slight temporary relief of symptoms. There were no significant studies that we could find on the use of medication for CGH. Where medications have been discussed, there has been the suggestion that CGH is relatively unresponsive to most medications commonly used to treat other forms of headache. Although other treatments, including massage, biofeedback, exercise, or nutrition, are commonly used to treat other headaches, including TTH, no studies were found on their use for CGH.

Conclusions

Despite a growing body of literature on CGH and an increasing acceptance that headaches can originate from the cervical spine, there remains considerable controversy and confusion concerning all aspects of this topic. However, a number of comments on CGH appear quite reasonable. The concept that headaches can originate from the neck is not new. The pain appears to be generated by irritation of nociceptors from structures in the cervical spine and may accompany injury and pathology in the neck. These headaches are difficult to differentiate from MH and TTH, although they possess the distinguishing characteristics of being triggered by neck movements, pain spreading to the occipital region, tenderness in the suboccipital tissues, decreased cervical range of motion, and unresponsiveness to typical headache medication. The significance of radiological findings and advanced diagnostic testing is unclear. Evidence to support treatment with surgery and injections consists mainly of case series without controls or standardized follow-up. The only treatment approach supported by a reasonable body of controlled trials is cervical manipulation, but this is by no means conclusive.

Until additional research and improved consensus on the topic of CGH becomes available, it is essential that any clinician maintain an open, cautious, and critical approach to the literature. At this point, the clinician must be wary of enthusiastic and dogmatic claims concerning CGH. As the literature on this topic grows in volume and quality, the debate will intensify and hopefully result in the clarification of the cause, diagnosis, and treatment of CGH.

Table 13
Other treatment for cervicogenic headache

Reference	Treatment	Study type	Follow-up	Number of patients	Results
[82]	TENS	Case series	2 months	60	80% had >60% improvement 20% has 40–60% improvement
[125]	TENS	Placebo-controlled trial	Not specified	20	Significant improvement in treatment group versus placebo group
[58]	Pain management	Case series	2–6 years	5	Significant ↓ in usual and highest VAS scores Significant ↓ in McGill Pain Questionnaire Significant ↓ in headache frequency (74%)
[126]	Epidural spinal cord stimulation	Case reports	3 years	2	1 female patient 60% initial improvement but deteriorated 1 male patient >90% improvement
[35]	Uvulopalatopharyngoplasty + continuous positive airway pressure	Case series	No specified	5	Minimal improvement only
[26]	Locomotor rehabilitation	Case report	None	1	Reduction in severe pain frequency

TENS = Transcutaneous electrical nerve stimulation; VAS = Visual Analog Scale.

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Spineposts

One Hundred Years Ago in Spine

In 1901 Wilhelm Conrad Röntgen received the Nobel Prize. On November 8, 1895, he had noticed that discharge from an induction coil passed through a Crookes vacuum tube caused fluorescence on a shielded paper coated with barium platinocyanide. Straightaway, he reported his new ray to the Würzburg Physico-Medical Society and submitted a paper to *Nature* [1]. He took a radiograph of his wife's hand on December 22, 1895. Robert Jones sent immediately for the apparatus and used it to take the first clinical x-ray on January 7, 1896 [2]. Sometimes things moved quickly, even without the Internet.

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