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CERVICOGENIC HEADACHE:ASSESSMENT
(INCLUDES GENERAL ASSESSMENT OF HEADACHES)

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On Using This Document

This care pathway is intended to be a guide to identify, diagnose and manage a specific group of headaches that originate from the joints or muscles of the cervical spine, more specifically cervicogenic and myofascial pain headaches. In addition, the care pathway outlines a general approach to assessing headaches, ruling out serious causes, and differentiating various unilateral headaches from one another.

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Search Strategy

The literature search was done using CINHAL, Cochrane Database of Systematic Reviews, DARE, Index to Chiropractic Literature, Medline, Ovid and PubMed spanning the years 2002-2012. *Search terms were cervicogenic, headache, chiropractic, manual therapy, physical therapy, chiropractic, manipulative therapy, manipulation,* and *mobilization.* The searches were performed by Betsy Mitchell, DC and Janet Tapper, MLS, university librarian of UWS. The relevant citations were reviewed by the primary authors and John Muench, MD, Oregon Health Sciences University. Additionally, recent textbooks and citations from articles were also consulted.

Codes for Common Headaches

Condition/Diagnosis	ICD-9-CM Code	Comments
Headache	**784.0**	This is a symptom code. It is nonspecific. This code can also be used for a nonspecific vascular head pain and facial pain; in other words, a nonspecific migraine or TMJ. This code is applicable to sinus head pain.
Tension type headache, unspecified	**339.10**	
Chronic tension-type headache	**339.12**	Daily headache, hatband distribution
Episodic tension-type headache	**339.11**	Occasional headache, hatband distribution
Migraine	**346.0**	Excludes: headache: NOS (784.0), syndromes (339.00-339.89)
Migraine with aura	**346.0**	The code for classical migraines that are preceded by a neurological sign or symptom. Basilar migraine, classic migraine, migraine preceded or accompanied by transient focal neurological phenomena, migraine triggered seizures, migraine with acute-onset aura, migraine with aura without headache (migraine equivalents), migraine with prolonged aura, migraine with typical aura, retinal migraine. Excludes: persistent migraine aura (346.5, 346.6)
Migraine without aura	**346.1**	The code for common migraines
Migraine, unspecified	**346.9**	The least specific code for migraine headaches
Cervicogenic Headache	**784.0**	Coded as headache excluding atypical face pain, migraine, tension-type headache
MFTP causing headache	**339**	Code as a tension-type headache

Codes for Less Common Headaches

Condition/Diagnosis	ICD-9-CM Code	Comments
Acute post-traumatic headache	339.21	Associated with whiplash and similar conditions
Chronic post-traumatic headache	339.22	Associated with whiplash and similar conditions
Cluster headache syndrome, unspecified	339.00	Ciliary neuralgia, cluster headache NOS, histamine cephalgia, lower half migraine, migrainous neuralgia
Post-traumatic headache, unspecified	339.20	Associated with whiplash and similar conditions
Postconcussion syndrome	310.2	Postcontusion syndrome or encephalopathy; post-traumatic brain syndrome, nonpsychotic. Status post commotio cerebri Use additional code to identify associated post-traumatic headache, if applicable (339.20-339.22). Excludes: any organic psychotic conditions following head injury (293.0-294.0), frontal lobe syndrome (310.0), postencephalitic syndrome (310.8).
Primary thunderclap headache	339.43	

Table of Contents

Tables

Appendices

BACKGROUND

Cervicogenic headache (CGH) belongs to the group of chronic benign headaches along with migraine and tension-type*. It is considered a secondary headache – one arising from or attributed to a disorder known to be a cause of headache. In this case, it is referred pain from the cervical spine to different areas of the head. This can include bony or soft tissue structures, specifically those innervated by cervical nerves C1-C3. (Bogduk 2009) (See Table 3.) This referral is the result of the convergence of sensory input from the cervical spine in the spinal trigeminal nucleus. This convergence is discussed in detail in the upcoming pathophysiology section.

The referral of pain to the head particularly from myofascial trigger points is not completely explained with the convergence idea. This myogenic referral generally comes from the muscles of the upper back and cervical region, and their referral patterns are well mapped. (See Appendix X) These referral and myofascial trigger points have been implicated in contributing to or co-existing with CGH. The myofascial *trigger point* relationship is different than myofascial *tender spots*, which are associated with tension-type headaches.

Causes or perpetuating factors include macro and micro-trauma, degenerative joint disease, and postural strains. (Jull 1997) Cervicogenic headache has a strong correlation to a history of trauma but may also occur in its absence. Onset of headaches originating from the cervical spine can occur at any age.

Historically, the first mention of headache arising from a structure in the neck was around 1860 by Hilton (Haldeman 2001). Since then, various terms have been used to describe this phenomenon such as cervical headache, headache of cervical origin, vertebrogenic headache, spondylitic headache and cervical migraine. In 1983, Sjaastad coined the term "cervicogenic headache." Fredriksen offered a description of the clinical presentation of patients diagnosed with cervicogenic headache in 1987. (Haldeman 2001)

* Note that a tension-type headache is different from a tension headache which has more of an emphasis on psychological stress as a cause.

COMPETING CRITERIA

Currently there are two main sets of criteria for diagnosing CGH. One is the International Headache Society's (IHS) criteria and the other Cervicogenic Headache International Study Group criteria (CHISG).

In 1990 Sjaastad introduced diagnostic criteria for CGH which he later revised in 1998 and published on behalf of CHISG. The IHS's International Classification of Headache Disorders first edition included CGH as a distinct disorder and included diagnostic criteria in their 1995 guidelines and in the revised 2004 guidelines. These revised IHS guidelines are internationally accepted as the standard for headache diagnosis.

There are distinct differences between the CHISG's and IHS's cervicogenic headache diagnostic criteria (Tables I and 2).

International Headache Guidelines (IHS)

The current IHS guidelines (2004) have removed the specific clinical signs initially presented in the 1995 edition. Those signs included pain precipitated or aggravated by special neck movements or sustained neck postures; resistance to or limitation of passive neck movements; changes in neck muscle contour, texture, or tone; response to active and passive stretching and contraction; and abnormal tenderness of neck muscles. IHS states that these signs have limited to no validity or reliability and are not acceptable. According to the IHS, the issue that arises with use of the "clinical features such as neck pain, focal neck tenderness, history of neck trauma, mechanical exacerbation of pain, unilaterality, coexisting shoulder pain, reduced range of motion in the neck, nuchal onset, nausea, vomiting, photophobia etc." is that "they are not unique to cervicogenic headache…. These may be features of cervicogenic headache, but they do not define the relationship between the disorder and the source of the headache."

Paradoxically, however, the IHS still allows for making the diagnosis on clinical assessment alone (as opposed to requiring laboratory or imaging), but they do so without stating *what* specific clinical signs should be used to "implicate a source of the neck pain." Without the ability to use these clinical features, the practitioner would have to rely on controlled diagnostic nerve blocks to establish a diagnosis. The use of blocks is neither practical in most clinical settings nor a cost-effective method of selecting a trial of conservative care. (See Table 1: IHS Criteria for Cervicogenic Headache Cervicogenic Headache Checklist.)

Cervicogenic Headache International Study Group Criteria (CHISG)

CHISG's diagnostic criteria, on the other hand, encompass more of the clinical signs useful to manual therapists and, despite their limitations, are the criteria used most consistently in studies on manual therapy for cervicogenic headache. They include producing or aggravating the patient's familiar headache by any of the following: 1) neck movement, 2) sustained awkward head positioning or 3) by a combination of external pressure over the upper cervical/ occipital region on the symptomatic side *associated with* restriction of neck range of motion. Additional findings include ipsilateral neck, shoulder, or arm pain of a vague non-radicular nature (occasionally, arm pain of a radicular nature). (See Table 2: CHISG Cervicogenic Headache Checklist.)

As demonstrated by the foregoing discussion, there is a significant lack of agreement in the definition of the diagnosis of cervicogenic headache. (Becker 2010, Bogduk 2009, Antonaci 2011) The IHS diagnostic criteria rely, at least in part, on a patient's response to diagnostic facet injection blocks. Controlled diagnostic blocks into the cervical facet joints are invasive, expensive and not readily available, and so they cannot be considered as useful in most practitioners' offices. Despite questions of diagnostic accuracy, the clinical features of CGH from the CHISG checklist provide a readily available, safe and inexpensive approach to diagnosis that is particularly useful in manual therapy.

There is considerable overlap in the clinical presentations of cervicogenic, migraine and tension-type headaches, implying that many of these signs and symptoms are not unique to any particular headache type. Because of these issues, according to Hall (2008) "incorrect headache diagnosis may occur in more than 50% of cases." Therefore, it is important that the practitioner carefully keep in mind competing diagnoses and monitor response to treatment.

A carefully directed history and physical exam focusing on the cervical spine and utilizing both the 1998 CHISG and the 2004 IHS guidelines should enable a practitioner to arrive at a reasonable working diagnosis of CGH adequate to drive a therapeutic trial.

TABLE 1: IHS Criteria for Cervicogenic Headache
2004 International Headache Society's Diagnostic

A. Pain, referred from a source in the neck and perceived in one or more regions of the head and/or face, fulfilling criteria C and D
B. Clinical, laboratory and/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 that the pain can be attributed to the neck disorder or lesion based on ≥1 of the following:
    1. demonstration of clinical signs that implicate a source of pain in the neck
    2. abolition of headache following diagnostic blockade of a cervical structure or its nerve supply using placebo or other adequate controls
D. Pain resolves within 3 mo. after successful treatment of the causative disorder or lesion

Notes

Cervical spondylosis and osteochondritis are NOT accepted as valid causes fulfilling criterion B.

When myofascial tender spots are the cause, the headache should be coded under tension-type headache (subform associated with pericranial tenderness).

Abolition of headache means complete relief of headache, indicated by a score of zero on a visual analogue scale (VAS). Nevertheless, acceptable as fulfilling criterion C2 is ≥90% reduction in pain to a level of <5 on a 100-point VAS.

From International Headache Society: The international classification of headache disorders, 2nd edition. Cephalalgia 24(Suppl 7):114-116,2004.

TABLE 2: CHISG Cervicogenic Headache Checklist

MAJOR CRITERIA (must have at least one; presence of all 3 increases confidence)

■ **Symptoms and signs of neck involvement**
- ☐ Precipitation of familiar head pain
    - — by neck movement and/or sustained awkward head positioning
    - — or by external pressure over the upper cervical or occipital region on the symptomatic side
- ☐ Restriction of the range of motion (ROM) in the neck combined with ipsilateral neck, shoulder, or arm pain of a vague non-radicular nature or, occasionally, arm pain of a radicular nature

■ **Confirmatory evidence by diagnostic anesthetic blockades** (*obligatory in scientific works)*

■ **Unilateral head pain without sideshift**

HEAD PAIN CHARACTERISTICS *(None of the following points is obligatory)*
- ☐ Moderate to severe, nonthrobbing, and nonlancinating pain, usually starting in the neck
- ☐ Episodes of varying duration, or fluctuating continuous pain

OTHER CHARACTERISTICS OF SOME IMPORTANCE *(None of the following points is obligatory)*
- ☐ Only marginal effect or lack of effect of indomethacin
- ☐ Only marginal effect or lack of effect of ergotamine and sumatriptan succinate
- ☐ Female sex
- ☐ Not infrequent occurrence of head or indirect neck trauma by history, usually of more than only medium severity

OTHER FEATURES OF LESSER IMPORTANCE
- ☐ Various attack-related phenomena, only occasionally present:
    - — nausea
    - — phonophobia and photophobia
    - — dizziness
    - — ipsilateral "blurred vision"
    - — difficulties on swallowing
    - — ipsilateral edema, mostly in the periocular area

From Sjaastad O, Fredriksen TA, Pfaffenrath V: Cervicogenic headache: Diagnostic criteria. Headache 38:442-445,1998.

PATHOPHYSIOLOGY

The mechanisms of cervicogenic headache pain referral are well documented. Two related neurological mechanisms appear to contribute to the headache symptoms: convergence of neurological pathways and sensitization of the nervous system.

TABLE 3: Afferents from C1-C3 Spinal Nerves	
C1-C3 Ventral rami • Atlanto-occipital joint • Lateral atlanto-occipital joint • Longus capitis • Longus cervicis • Rectus capitis anterior • Rectus capitis lateralis • Trapezius • Sternocleidomastoid • Dura mater of posterior fossa • Vertebral artery	**C1-C3 Dorsal rami** • C 2/3, 3/4 zygapophyseal joints • Semispinalis capitis, cervicis • Multifidis • Longissimus • Splenius capitis **C1-C3 Sinuvertebral nerves** • Median atlanto-axial joint • Transverse ligaments • Alar ligaments • Dura mater of spinal cord • Dura mater of clivus • C2/3 intervertebral disk

1. CONVERGENCE

Bogduk has extensively described the convergence of trigeminal and cervical afferents in the trigeminocervical nucleus. (Bogduk 2009) The nociceptive afferents arise from a wide variety of spinal tissue innervated by spinal nerves C1-C3. (See Table 3 above).

While the exact neurophysiology is still being investigated, the following is a current model potentially explaining the biological process.

The nociceptive afferents of these C1-3 nerves converge with the afferents from the trigeminal nerve on the same pool of second order neurons in the spinal cord.

Figure 1

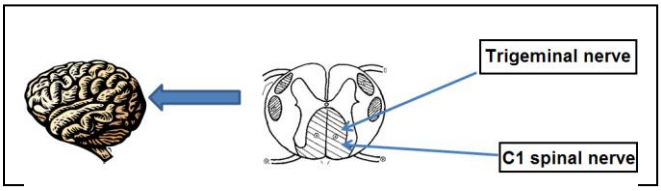
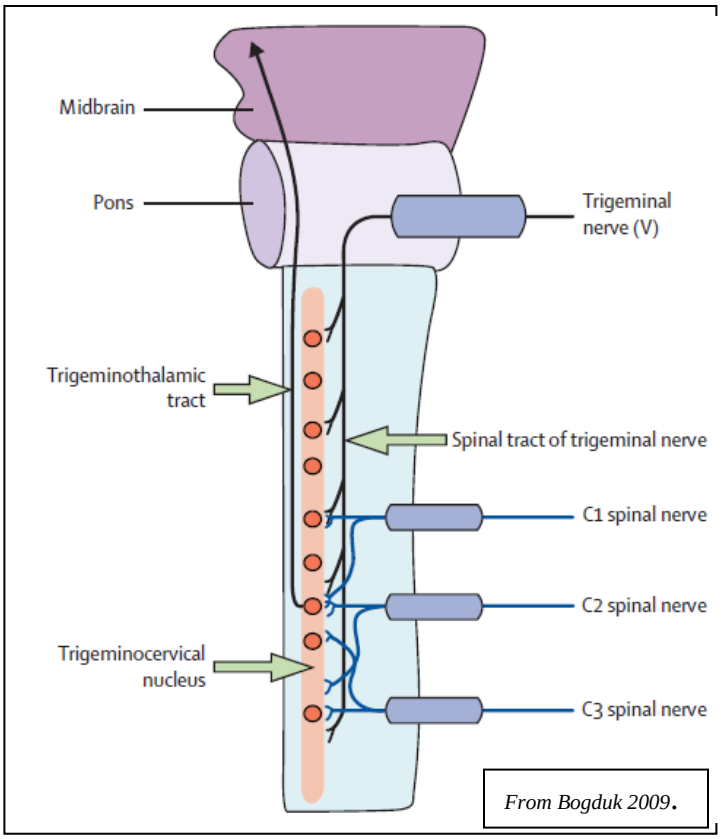


Figure 1 above depicts afferent input from both spinal tissue and receptors in the trigeminal nerve territory as they converge on secondary neurons located in the spinal trigeminal nucleus caudalis in the upper cervical segments of the spinal cord and lower brainstem. Subsequent projections ascend though the pain pathway to higher cortical centers. (Note: this diagram is purposely simplistic; there are actually thousands of neurons with multiple, complex synaptic connections).

The second order neurons do not differentiate which afferents activated them, those from the local spinal tissue or those from trigeminal nerve territories. As a result the patient may feel pain in a location that is different from where the actual pain generating lesion is found. Headache pain referral is a direct result of this convergence. Consequently, pain from the upper cervical spine can be referred to those regions of the head innervated by cervical nerves 1-3 (around the ear and occipital region) and areas innervated by the trigeminal nerve (around the eyes, forehead and parietal region). (Bogduk 2009) See *figure 2 on next page*.

Figure 2



Since most spinal tissue is pain sensitive, as a result, a large variety of injuries, dysfunctions, or causes of tissue irritation can refer pain to the head. (See Table 4: Possible anatomic sources of CGHs.) Furthermore, this neurological convergence is believed to be bi-directional. The referral headache pain can come from cervical structures and cervical pain can be referred from structures in the head. This may, in part, account for the neck pain reported in some migraine patients who appear to have no discernable musculoskeletal dysfunction present. Finally, it has been shown that the nociceptive stimuli may converge from further afield. Pain signals from the larynx, pharynx and viscera of the thorax and abdomen are transmitted by the vagus nerve and converge with the upper cervical afferents as well. (Foreman 2000) This increased afferent activity may "spill over" affecting the cervical spinal motor neurons and contribute to the palpable increase in tissue tension in the upper cervical region. This would be an example of a viscerosomatic reflex.

TABLE 4: Possible Anatomic Sources of CGHs
(Adapted from Haldeman 2001)

Structure	Pathology	Mechanism
Facet joint	Irritation, RA	Trauma or immobility stimulates the C1-C3 nerves
Cervical muscles	Myofascial trigger points, myospasm*	Referred symptoms from muscles innervated by C1-C3
Intervertebral disk	Trauma, Herniation,	Irritates the dura, Stimulates sinuvertebral nerve
Nerve roots	Compression/ irritation	Disk herniation, spondylosis,
Vertebral artery	Compress	Apophyseal exostoses impacting vertebral artery blood flow
Uncovertebral joints	Mechanical irritation	Nerve roots producing sternocleidomastoid and trapezius muscle spasm
Ponticus posterior	Articular lock, instability	Tension on the dura or vertebrobasilar artery compression
Rectus capitis muscle	Connective tissue bridge with the dura	Tension on the dura
Ligamentum nuchae	Attaches to the dura	Tension on the dura

* The IHS guidelines suggest that MFTPS essentially be classified as tension-type headaches with tender points—these guidelines have no classification for myofascial pain syndromes causing headaches.

2. Central and peripheral sensitization

The second mechanism driving cervicogenic headaches is thought to be sensitization (Alix 1999), a process which involves neuronal/synaptic plasticity and dynamic changes in neuron responses (Boal 2004). In this case, sensitization results in increased nociceptive response. Severe or chronic pain stimulus has been associated with central sensitization (need a ref), which is an alteration of the central nervous system's neuronal processing ability resulting in a hyper-response (Sandkuhler 2007) of the converging pathways previously described. As part of this process, hyper-excitable second order neurons and other upstream central/brain neurons may generate pain signals *with minimal stimulus from the peripheral tissue and even continue to spontaneously discharge after the injured peripheral tissues have healed*. In a similar manner, peripheral sensitization can occur when the nociceptors in the paraspinal tissues themselves become hyper-excitable (Alix 1999). Both central and peripheral sensitization are products of neuroplasticity.

Epidemiology

Most people experience headaches at some time in their lives. By one report, the lifetime prevalence of headache, including migraines and tension-type headaches, for men and women 25-64 years old is 96%. (Rasmussen 1991) According to the World Health Organization's 2011 Atlas of Headache Disorders, it is estimated that half to three quarters of the world's population aged 18-65 have had a headache in the past year.

General population studies of headache sufferers suggest that 42% report episodic tension-type headaches, over 10% have migraine, and 2-4% suffer chronic headaches (i.e., those with symptoms present greater than two weeks per month). (Stovner 2007) Other sources suggest an overall rate for tension-type headaches (including both episodic and chronic) as high as 78% and a relatively similar migraine rate of 16% (Godwin 2001).

Headaches in general are more prevalent among women than men, for both tension-type (5:4) and migraine (2-3:1) (WHO 2011). The prevalence of tension-type headaches and migraines peaks from 30-39 years for both sexes, and then decreases with age. (Jensen 2008).

The WHO report cited that headaches from medication overuse surpass all other secondary headaches (a headache category which includes strokes, tumors, chronic illness, sinusitis, etc.) with a world-wide prevalence of 1%, affecting women more than men. (WHO 2011)

The prevalence of CGH in the general population varies depending upon the diagnostic criteria used, study design and targeted populations. If the International Headache Society's first edition (IHS I) criteria are used, the prevalence is only 2.5%, but increases in those people with frequent headaches (Nilsson 1995). Likewise, when the Cervicogenic Headache International Study Group's (CHISG) criteria are used, the prevalence is reported to be 2.5% (95% CI 1.1-4.8) (Nilsson 1995). In one study, however, where both CHISG and IHS II criteria were used, the prevalence was only 0.17% with a female dominance (slightly less than a 2:1) in subjects aged 30-44 (Knackstedt 2010). Overall, when considering sub-populations, these estimates increase to 14-18% in chronic headache patients (Pfaffenrath 1990; Nilsson 1995) and 17.8% (95%CI 8-32%) of subjects with severe headaches (using the CHISG criteria). (Evers 2008)

Pre-test probability for cervicogenic headache may then be estimated to range from 1-2% overall to approximately 15-20% (Haldeman 2001) for those with severe or chronic headaches (presumably patients more likely to seek care). It may be, however, that the prevalence is higher yet among patients who seek care from manual therapy practitioners since these patients may be more likely to have a significant neck pain component.

Confounding all of this data is the high possibility of co-occurrence of migraine or tension-type headache with CGH. The Knackstedt study demonstrated a 42 % co-occurrence of CGH and migraine and a 50% co-occurrence of medication overuse in its study population. (Knackstedt 2010)

Headache Assessment

The Role of History

Primary headaches, like migraines and tension-type headaches, are considered to be idiopathic rather than the result of an underlying disease process or identifiable pain generator. Since there are no diagnostic tests for primary headache disorders, a thorough history is of utmost importance. In a medical setting, about 90% of patients who present with a headache have a normal physical examination and a primary headache diagnosis. The history may instead suggest that the headache is a secondary headache. Secondary headaches may be benign in nature (e.g., cervicogenic headache) or serious (e.g., subarachnoid hemorrhage). The initial goal of the history is to make sure a serious secondary cause of headache is not overlooked (Dodick 2010), especially in patients without a clear diagnosis of a primary headache.

DIFFERENTIAL DIAGNOSIS

The overall differential diagnosis of headache includes all headache subtypes and all head, neck, upper body/upper extremity pathologies.

The history, however, may elicit recognizable patterns of symptoms consistent with one of the many headache subtypes which include migraine headache, tension-type headache, cluster headache/chronic paroxysmal hemicrania and hemicrania continua, and other miscellaneous headaches. Combination headaches are also possible; for example, migraine, tension-type, and cervicogenic are reported to occur concurrently.

HEADACHE TRIGGERS

Potential headache triggers should be sought in the patient's diet, behaviors or environment. Substances such as MSG, alcohol, and nitric oxide donors (e.g., nitroglycerine, hot dogs) can trigger headaches. Headaches can also be caused by withdrawal of caffeine, medications or other substances. In the case of cervicogenic headache, the patient's pain may be provoked by sustained head posture, or particular neck movements associated with recreational, job or daily activities

OUTCOME MEASURES

Finally, the history plays an important role in providing many of the outcome markers used to measure the patient's response to treatment. See Outcome Measures on p37.

A thorough headache history requires a practitioner's time. In cases where a patient unexpectedly presents with an undocumented headache during a visit for an unrelated problem (e.g., a shoulder impingement syndrome), the history must be sufficient to rule out serious conditions and contraindications to manipulation. A more thorough history and physical can be scheduled for a subsequent visit.

For subsequent visits, an aid to practitioners is to have the patient keep a headache diary. This will provide very useful information such as patterns, potential triggers, severity and effects on daily living. A diary can be as simple as a paper notebook or as sophisticated as a HIPAA protected online access site for both patient input and practitioner review.

MEDICATION HEADACHES

Many headaches are iatrogenic. The history should screen for drug misuse, whether OTC or prescription. (MacGregor 2010) The patient's medications and supplements should be recorded and checked for possible drug interactions. Regardless of the patient's age, the following information is important to acquire:

- **New medications or change in dose:** Many medications, prescription or over the counter can cause headache as an acute adverse side effect. Practitioners should be especially vigilant with the elderly (see Appendix I: Medication Overuse headache).(IHS 2004)

- **Medication Overuse Headache (MOH) (formerly called rebound headaches):** According to the International Headache Society (IHS), a common problem is overuse of headache drugs which can eventually cause headaches themselves in the headache-prone patient. The IHS suggests

that overuse of symptomatic migraine drugs and analgesics are an important cause of headaches that mimic migraines or mimic a mixed tension-type, migraine-type headache. In the case of analgesics, expert opinion suggests that the medication must be taken ≥ 15 days per month. (IHS 2004) (For the IHS criteria for Medication Overuse Headaches, see Appendix I).

- **Chronic use**: Chronic use of medications, even when they are prescribed for long-term use can sometimes cause headaches (e.g., anabolic steroids, lithium, thyroid replacement therapy, and tetracycline). (IHS 2004)

- **Discontinuing medication:** Examples include discontinuing opioids or estrogen along with anecdotal reports of NSAIDs, corticosteroids, caffeine containing medications and anti-depressants.

> ***Clinical tip***: Relative to medications, the practitioner should elicit the following:
> 1. **NEW?** Whether the patient is taking any new medications
> 2. **DURATION?** How long the patient has been taking each medication
> 3. **CHANGE?** Recent change in dosage (and current dosage)
> 4. **WITHDRAWAL?** Recent withdrawal from a medication

The Role of Physical Examination

A problem focused physical exam is used to investigate the list of possible diagnoses derived from the patient history. It can 1) help rule out serious causes of the headache, 2) rule in or out a possible cervicogenic headache or myofascial pain syndrome, 3) assess cervicocranial structures that may be amenable to manipulation and exercise therapy, and 4) identify biomechanical factors that may be contributing or sustaining the headache symptoms (e.g., poor motor control, inefficient movement patterns, and/or muscle imbalances).

The detail level of the exam will be dictated by the history and especially by the presence of any focal neurological symptoms (suggesting an intracranial lesion).

The British Association for the Study of Headaches (MacGregor 2010) suggests to both neurologists and portal of entry physicians that "the physical examination adds to the perceived value of reassurance and, within limits, the more thorough the examination the better."

For many people with troublesome but benign headache, *reassurance* is a key goal of the patient encounter.

Summary of Physical Examination for Headaches

- ✓ Observation (e.g., alertness, orientation, antalgia, gait, color, sweating) (see p. 15)
- ✓ Blood pressure (see p. 15)
- ✓ Fundoscope exam (see p. 15)
- ✓ Neurologic exam (see p. 16)
  - Mental status
  - Cranial nerve exam (including visual acuity)
  - Sensory, motor and reflex assessment of the upper and lower extremities
  - Central nervous system screen (e.g., clonus, Babinski, Hoffmann)
- ✓ Postural analysis (see p. 33)
- ✓ TMJ exam (optional)
- ✓ Cervical and thoracic AROM (see p. 27)
- ✓ Cervical orthopedic testing (see p. 29)
- ✓ Full spine palpation with emphasis on the cervical region and upper thoracics (see p. 27)
- ✓ Soft tissue palpation especially of muscles related to the cervical spine and cranium(see p. 29)
- ✓ Jull's test (see p. 30), cervical flexion rotation test (see p. 32), and cranio-cervical flexion test (see p. 31)
- ✓ Valsalva

At the practitioner's discretion, some or all of the above procedures may be appropriate.

These examination procedures are further elucidated throughout the rest of the assessment section of the care pathway. See pp. 15-17 and 27-35.

Assessment Strategies

When working up a patient with headache, a number of diagnostic questions must be answered by the end of the history and physical. These can be organized in a series of steps based on priority.

Summary of Evaluation Strategy

Step 1: Rule out ominous headaches.
Step 2: Identify the type of headache (cervicogenic, myofascial, migraine, etc.) and pain generating tissue (pp. 19-26)
Step 3: Identify any significant triggering or contributing factors (e.g., forward head carriage, deep flexor weakness). (pp. 33-35)
Step 4: Determine the need for ancillary tests/studies. (pp. 36-37)
Step 5: Establish outcome measures and severity of the condition. (pp. 37-38)
Step 6: Determine if there are significant psychosocial factors. (p. 38)
Step 7: Establish a prognosis. (p. 39)

Step 1: Rule out ominous headaches.

Ominous headaches include tumors, strokes, and post traumatic lesions. These intracranial lesions are, fortunately, uncommon. In patients presenting with an initial onset of headache, the probability of a serious secondary headache diagnosis ranges from 0.7% to 2.49% in a primary care setting (MacGregor 2010). One published series of general practice patients with new-onset headache of apparent benign origin reported that the 1-year risk of a malignant brain tumor was only 0.045%. (MacGregor 2010). The clinician should reassure patients that most headaches are benign in origin.

TUMORS

Potentially serious headaches usually provide clues in the history. With the exception of pituitary tumors, intracranial tumors do not cause headaches until quite large and are more likely to create other associated symptoms. (MacGregor 2010) Headache as a presenting symptom of a CNS mass lesion occurs 20% to 50% of the time. (Royce 2011)

The classic brain tumor headache is severe, worse in the morning on rising, and associated with vomiting. The Valsalva test may increase headache severity. In one study, however, this classic presentation occurred only 17% of the time and mostly in individuals with concomitantly increased intracranial pressure (ICP). Furthermore, headaches with increased ICP were distinctive in that they were resistant to

treatment with common analgesics. (Royce 2011) Red flags for an intracranial lesion include epilepsy (a cardinal symptom of intracerebral space occupying lesions) and loss of consciousness. Focal neurological signs in the physical will usually be present. More problematic are slow growing tumors, especially those in parts of the frontal lobes. In some cases, subtle personality change may result in treatment for depression, with headache erroneously attributed to the psychological state. (Macgregor 2010)

STROKES AND TIAS

Headache occurs in 20% of ischemic strokes (range across 11 studies, 8%-34%) (Royce 2011). Half of these headaches are unilateral, focal, and are of mild to moderate severity. Associated symptoms include nausea (2/5 of patients), vomiting (1/4), and photophobia and sonophobia (1/4). Aggravating factors include the Valsalva maneuver and nitroglycerine. The mean duration is 3.8 days (Ferro 1995, Tenschert 2005). Headache is also associated with TIA, occurring at a frequency of 17% to 54% of the time. The mean duration of headache in TIA is 17 hours. Severe headache is the key presenting symptom in subarachnoid hemorrhages (see below).

Quick screening questions for serious pathology

1. Is the headache of recent onset (less than 6 months)?
2. Is there any progression in the frequency or severity of the headaches?
3. Was the onset sudden and severe?
4. Are there any clues suggesting hard neurologic signs associated with the headaches?
5. Are there any cognitive changes associated with the headaches (e.g., memory loss, confusion, personality changes)?

If the answer to these five questions is no, the likelihood of a serious organic disease is remote.

WARNING SIGNS

Important red flags for serious secondary headaches are (Godwin 2001):

1) Sudden onset
2) Associated neurologic signs and symptoms.
3) Age $\geq$ 50

1. Sudden Onset of Worst HA

A sudden onset headache with excruciating pain suggests a possible subarachnoid hemorrhage or vertebral artery dissection.

Subarachnoid hemorrhage. This type of headache often reaches its maximal intensity within seconds and is sometimes referred to as a "thunderclap headache." The first concern should be to consider a subarachnoid hemorrhage (SAH). In one prospective study, 70% of patients (35/49) presenting with a thunderclap headache had a SAH. (Godwin 2001)

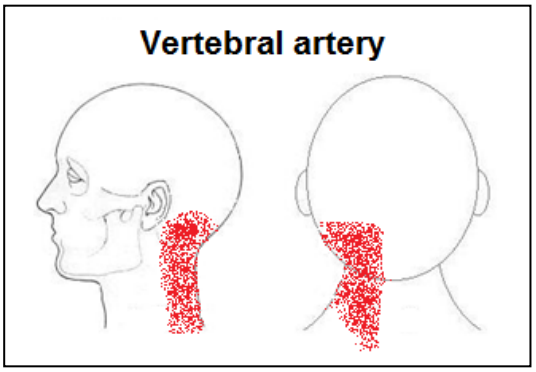
In contrast, one study found that only 17% of patients (18/107) reporting the "worst headache of their life" had a SAH (Morgenstern 1998). Mills (1986) in a prospective study reported positive head CTs in 29% of patients complaining of the worst headache of their life or severe, persistent headache.

While other studies suggest little correlation between worst or sudden headache and final diagnosis, Godwin (2001) suggests that "…each of these studies suffers from a variety of methodological flaws."

!!! Clinical warning: Because SAH is an emergent referral with potentially serious consequences, practitioners should err on the side of referral when this type of headache is strongly suspected.

Vertebral artery dissection. A sudden, extremely severe occipital headache, associated with neck pain, suggests a vertebral artery dissection. The neurological examination may be normal at the time of the initial visit. Neurologic deficits may not present for several days. About half of the patients complain of dizziness or vertigo and experience nausea or

vomiting. Some have unilateral facial numbness, drop attacks, diplopia. (Godwin 2001) Practitioners should be aware of the 5 Ds (diplopia, dizziness, drop attacks, dysarthria, and dysphagia) and the 3 Ns (nausea, numbness and nystagmus). (See CSPE protocol Vertebrobasilar Artery Insufficiency and CAD).



2.Associated neurologic signs

The presence of focal neurological lesions identified during the history or physical examination increases the concern for a possible intracranial lesion. A normal neurological exam, however, *does not completely rule out* the possibility of a serious headache. In a study of consecutive headache patients who had no neurological signs, the prevalence was intracranial pathology was 0.9. (MacGregor 2010)

3. Age

Age ≥ 50 is less of a red flag than sudden onset and abnormal neurological findings. However, a new-onset headache in older patients who have no prior history of similar pain is of some concern. One review of 468 emergency department headache patients found that age greater than 55 was a strong predictor of intracranial pathology. (Godwin 2001) Besides intracranial lesions, consider glaucoma and temporal arteritis in patients over 50. (Godwin 2001) For a review of temporal arteritis, see Appendix II.

TABLE 5: Clues from the HISTORY when considering serious causes of headaches

Finding	Consideration
Quality: Thunderclap headache	Subarachnoid hemorrhage (SAH)
Severity: Worst headache	SAH, cerebral venous thrombosis, vertebral artery dissection
Onset: Use of gas space heater	Carbon monoxide
Onset: Pregnancy	Pre-eclampsia, eclampsia, cerebral venous thrombosis
Associated symptoms: Change in vision	Glaucoma, optic neuritis, vertebral artery dissection, intracranial lesion, post-traumatic headache, temporal arteritis, CVA, idiopathic intracranial hypertension
Onset: New headache after age 50	Temporal arteritis, mass lesion, glaucoma
Onset: Younger than 10	Space occupying lesion
Onset: New headache in patient with a history of cancer	Metastasis to the brain
Onset: New headache in an HIV patient or in immunocompromised patient	Space occupying lesion
Onset: Headache that begins with exertion	Encephalitis
Onset: Headache associated with postural change	Space occupying lesion
Timing: Persistent morning headache with nausea	Space occupying lesion
Timing: Progressive, worsening over weeks or longer	Space occupying lesion
Quality/Severity: Headache dramatically different from past headaches	Space occupying lesion
Associated symptoms: Headache with atypical aura (duration>1 hour, or including motor weakness)	Stroke
Associated symptoms: First time aura in a patient using combined oral contraceptives (estrogen & progesterone)	Stroke

(modified from Godwin 2001 and MacGregor 2010)

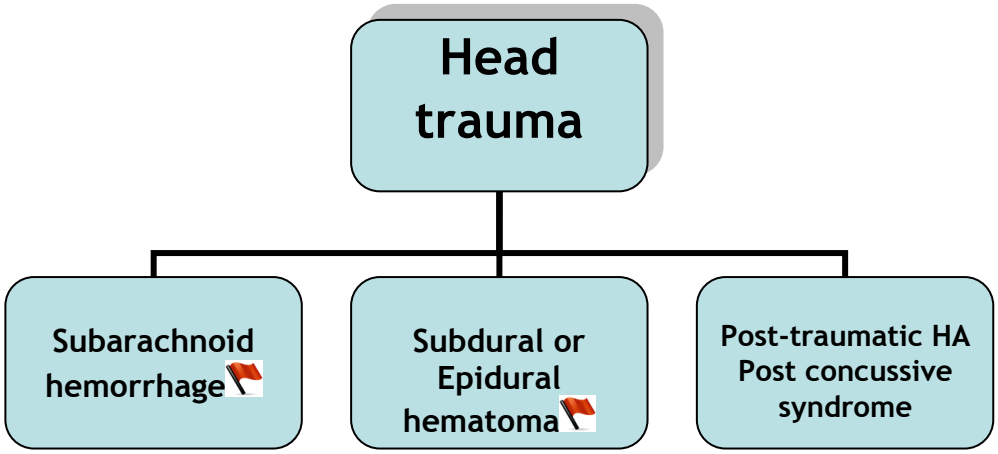
TRAUMATIC HEADACHES

Although a cervicogenic headache is an important differential diagnosis in trauma cases, more serious conditions such as subdural bleeds, epidural bleeds and subarachnoid hemorrhages must be considered. A history of headache associated with remote head trauma (may have been weeks prior) or with trauma in general (the patient may not remember whether they hit their head) introduces the possibility of chronic subdural hematoma or post-traumatic headache/post-concussive syndromes.

However, patients may not remember the traumatic event and, therefore, do not report it during the history. *Up to 20% of patients with chronic subdural hematoma have no identifiable etiology and can present with symptoms up to three months from a known traumatic event* (Black 1985). The elderly, alcoholics, epileptics, patients on dialysis or warfarin, and those with coagulopathies are at highest risk for chronic subdural hematomas. (Godwin 2001) A mild to moderate nonspecific headache is present in 80% of patients with chronic subdural hematomas.

Although focal deficits and focal head pain are not common, patients may suffer confusion or fluctuating sensorium (e.g., changes in alertness or consciousness). (Lipton 1993)

Post-traumatic headache can occur in up to 80% of patients in the first three months post head trauma. Acute posttraumatic headache (APTH) develops with 7 days. It occurs in barely half of patients and usually resolves in a few weeks. Chronic post-traumatic headaches (CPTH), lasting longer than 3 months, are reported to occur in 15-50% of patients.The symptoms fall into three groups: physical (e.g. headache, dizziness, double vision, blurred vision, headache, nausea, sensitivity to light an noise, sleep disturbance), emotional (e.g. being irritable, feeling frustrated, depressed or restless) and cognitive (e.g. forgetfulness, poor concentration, taking longer to think). When associated with mild head injury, symptoms are common and usually self-limiting. They frequently display migrainous features. (Lieba-Samal 2011, Rimel 1981)



Serious conditions to rule out after head trauma.

= potentially life threatening.

Physical Examination to Rule Out Serious Pathologies

Observation

Observation begins as soon as the physician sees the patient, noting any unsteadiness and whether the patient appears ill. Pallor, diaphoresis, or cyanosis can indicate serious illness. Decreased alertness or cognition can suggest an intracranial lesion. Petechial or purpuric rash on trunk or extremities may suggest meningitis.

Closer inspection of the face also can offer useful clues:

- The presence of Horner's syndrome (i.e., ptosis, meiosis, anhydrosis) in conjunction with a headache may represent a carotid dissection. (Godwin 2001)
- In trauma cases, periorbital ecchymosis (raccoon's eyes) suggests basilar skull fracture. (Godwin 2001)
- A unilateral red sclera is often seen with either glaucoma or cluster headache. In glaucoma, the cornea is usually cloudy and the pupil mid-position and unreactive. Such patients need measurement of their intraocular pressure. (Godwin 2001)

Blood Pressure

High and low blood pressure can be related to headaches or to the medications used to treat hypertension. It is important to establish a baseline with a new patient and for monitoring existing patients, especially those presenting with a new or different headache. Because chronic stage I and stage II hypertension are not thought to generally be a cause of headache, it should not be presumed to be the cause of the patient's presenting headache (IHS 2004). On the other hand, stage II hypertension (>180 systolic or >110 diastolic), especially if representing an acute change, can induce headache.

Fundoscopic Exam

A fundoscopic exam should be performed whenever there is suspicion of an intracranial lesion. When the history is benign, the decision whether or not to perform a fundoscopic exam is more controversial. Intracranial lesions are rare and when the history is devoid of red flags, the chances of an undetected tumor or bleed are somewhat remote. On the other hand, the 2010 guidelines from the British Association for the Study of Headaches recommend "The optic fundi should always be examined during the diagnostic consultation. *Fundoscopic examination is mandatory* at first presentation with headache, and it is always worthwhile to repeat it during follow-up."

The fundoscopic exam of the headache patient can reflect elevated intracranial pressure, manifesting as papilledema. Papilledema is consistent with brain tumor or other space occupying lesion, pseudotumor cerebri, brain hemorrhage secondary to trauma (subdural and epidural bleeds), and hemorrhagic strokes. Papilledema is a rare condition, affecting less than 200,000 people in the US population (Office of Rare Diseases of the National Institutes of Health). Early detection, however, can be lifesaving and it should be identified by all practicing physicians. (Mangione 2000)

> 👍 ***Clinical tip***: Godwin (2001) writes "the sophisticated emergency physician will train him-or herself to look for spontaneous venous pulsations (SVPs). This is a subtle throbbing of the central retinal vein (the fattest, darkest vessel in the retina) just where it emerges from the disc. No patient with SVPs had increased intracranial pressure. While the presence of SVP essentially rules out intracranial hypertension, pulsations may be absent in about 12% of normal patients."

Another rare cause of headache should be kept in mind, especially in young obese women. Idiopathic intracranial hypertension (IIH) (formerly termed benign intracranial hypertension or pseudotumor cerebri) is associated with papilledema and can lead to blindness (MacGregor 2010)

A normal fundoscopic exam, however, would be anticipated in the large majority of headache patients unless they had some other underlying pathology.

NEUROLOGICAL EXAM

Sample neurological exam

- Gait, orientation, responsiveness
- Cranial nerves II-XII
- Light touch or sharp/dull (upper and lower extremities)
- Muscle testing (minimally including a mix of proximal and distal muscles in all four extremities, may include pronator drift)
- Deep tendon stretch reflexes (biceps, triceps, patella, Achilles)
- Pathological reflexes (Babinski/ foot tapping and Hoffmann and/or clonus at the wrists and ankles)
- The jolt maneuver

CRANIAL NERVES

The neurological exam should include a cranial nerve exam, focusing on the pupils (II, III), visual fields (II), eye movements (II, IV, VI), facial sensation (V) and expression (VII), soft palate (IX, X) and tongue movement (XII). Focal deficits can signal an intracranial lesion (e.g., cranial nerve VI palsy suggests increased intracranial pressure). Cranial nerve I is not commonly tested unless the patient reports a loss of sense of smell.

CNS ASSESSMENT

A baseline assessment of muscle tone, strength, reflexes and coordination in all four limbs including plantar responses (i.e. Babinski) or clonus as well as an assessment of gait can be used as a central nervous system screen. (Scottish Intercollegiate Guideline Network, 2008)

While a sensory exam is often suggested, there is little evidence to show that it is helpful. (Godwin 2001)

Rapid foot tapping has been suggested as perhaps a more accurate test for a CNS lesion than eliciting a positive Babinski reflex. The supine patient is asked to tap their feet rapidly against the examiner's hands. A positive finding is evidence of poor speed and coordination. In one small study foot tapping demonstrated better inter-examiner reliability (kappa .73 vs. .30), sensitivity (86 vs. 30%) and specificity (84 vs. 77%) than the Babinski test. (Miller 2005)

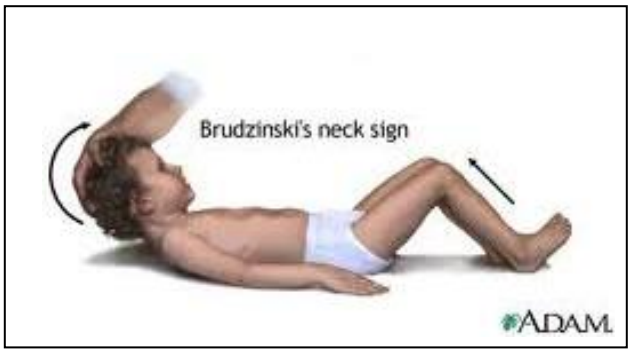
PRONATOR DRIFT

Pronator drift is one of the most commonly employed tests for motor deficit in suspected strokes (Godwin 2001). The patient, eyes closed, holds his/her upper extremities out, palms up for 20-30 seconds. Pronation and slow inferior drift of the arm suggests regional weakness perhaps secondary to a stroke or intra-cranial lesion. Variations include the examiner gently pushing down on the extended arms to trigger the drift or asking the patient to slowly shake his/her head "no" to tease out milder positive findings.

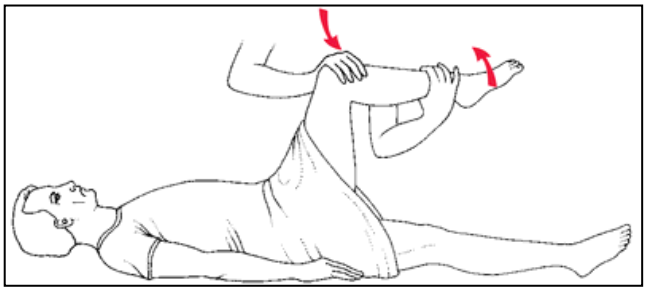
MENINGES TESTS

A headache accompanied by neck stiffness (meningismus) and fever suggests possible meningitis (Quattromani 2008). On the other hand, the absence of fever, neck stiffness and altered mental status effectively eliminates meningitis (99-100% sensitivity) (Attia 1999). Neck stiffness can be ascertained by active or passive range of motion, especially in flexion. Nuchal rigidity has traditionally been signaled by the presence of Brudzinski or Kernig's signs. Altered mental status with fever, even in the absence of headache or neck stiffness, can still be consistent with bacterial meningitis. The addition of a rash (petechial, purpuric or even maculopapular) is an alarming sign.

Brudzinski and Kernig's signs: Nuchal rigidity may be suggested by the presence of Brudzinski sign (rapid flexion of the neck resulting in involuntary hip and knee flexion) or Kernig's sign (inability to extend the leg from a flexed hip position due to pain and patient resistance).



Brudzinski sign



Kernig's sign

Although these tests are rated as having high specificity (95%), they have such low sensitivity (5%) that **they add little to no value to clinical decision making (LR=1.0)** (Thomas 2002). One study reported that 13% of acute-care patients and 35% of geriatric patients had nuchal rigidity despite the absence of meningitis, likely owing to presence of cervical arthritis and spondylosis among older patients. (Puxty 1983)

The jolt test: A promising test is the jolt test. The jolt accentuation test has been recommended to help rule out meningitis in a low-risk, nontoxic patient with headache and fever. (Attia 1999, Godwin 2001) To perform this test, ask the patient to rapidly shake his or her head from side to side (2-3 times per second). A positive test is the exacerbation of an existing headache. The test has a sensitivity of 97% and specificity of 60% for the presence of CSF pleocytosis, signaling CSF infection. A negative test essentially can exclude meningitis in patients with fever and headache. A positive result aids in the decision to proceed with lumbar puncture even in patients who do not otherwise have evidence of meningismus (Uchihara 1991).

TABLE 6: Clues from the PHYSICAL when considering serious causes of headaches
(modified from Godwin 2001 and MacGregor 2010)

Finding	Consideration
Altered mental status	Intracranial lesion (e.g., stroke, tumor)
Meningeal sign	Meningitis, stroke
Positive "jolt" test	Meningitis
Focal neurologic signs	Intracranial lesion (e.g., stroke, tumor)
Rash	Lyme disease, Rocky Mountain spotted fever, meningococcemia
Change in vision	Glaucoma, optic neuritis, vertebral artery dissection, intracranial lesion, post-traumatic headache, temporal arteritis, CVA, idiopathic intracranial hypertension
Fever	Infection (CNS vs. systemic)
Double vision	Intracranial mass, idiopathic intracranial hypertension, post- traumatic headache, dissecting aneurysm
Ptosis, miosis	Carotid artery dissection
Horner's syndrome	Space occupying lesion
Papilledema	Mass lesion, optic neuritis, pseudotumor
Dilated pupil	Aneurysm compressing third cranial nerve

A final word on serious headaches

The mnemonic SNOOP4 can be helpful in establishing a more complete list of headaches secondary to serious conditions. The red flags fall into the general categories of **S**ystemic symptoms, **N**ervous system findings, **O**nset being sudden and rapidly reaching a peak, **O**nset in patients over 50, and a set of four changes in headache **P**atterns (progressive, precipitated by a Valsalva maneuver, aggravated by head position) or the presence of papilledema.

TABLE 7: SNOOP4 Mnemonic for Secondary Headache Disorders†		
Clues	**Clinical Presentation**	**Important Headaches to Rule Out**
Systemic	• Unexplained fever, chills, weight loss • New onset headache in patient with malignancy, immunosuppression or HIV	Primary or metastatic tumors, meningitis, brain abscess, temporal arteritis
Neurologic	• Motor weakness, sensory loss, diplopia or ataxia • Abnormal neuro exam	Malignant, inflammatory and vascular disorders of the brain
Onset sudden	• Headache reaches peak intensity in <1 minute	Vascular events: subarachnoid hemorrhage (most common),dissecting vertebral aneurysm, CVA, carotid dissection, cerebral vasoconstriction syndromes, dural venous thrombosis
Onset >50	• New headache after age 50	Neoplastic, inflammatory disorders, & temporal arteritis
Pattern change	• **P1**: Progressive evolution to daily headache • **P2:** Precipitated by Valsalva • **P3:** Postural/positional aggravation • **P4**:Papilledema	Malignant, inflammatory, and vascular disorders of the brain Chiari malformation, primary and metastatic lesions of brain, hydrocephalus Low pressure headache syndromes, intracranial hypertension, postural orthostatic tachycardia syndrome Malignant and inflammatory disorders of brain, idiopathic intracranial hypertension, dural venous thrombosis

†Adapted and revised from Dodick D. *Semin Neurol*. 2010;30:74-81.

Step 1: Rule out ominous headaches.
Step 2: Identify the type of headache (migraine, tension-type/myofascial, cervicogenic, myofascial, etc.) and pain generating tissue.
Step 3: Identify any significant triggering or contributing factors (e.g., forward head carriage, deep flexor weakness).
Step 4: Determine the need for ancillary tests/studies.
Step 5: Establish outcome measures and severity of the condition.
Step 6: Determine if there are significant psychosocial factors.
Step 7: Establish a prognosis.

Step 2: Identify the general type of headache (cervicogenic, tension-type/myofascial, migraine, etc.) and pain generating tissue.

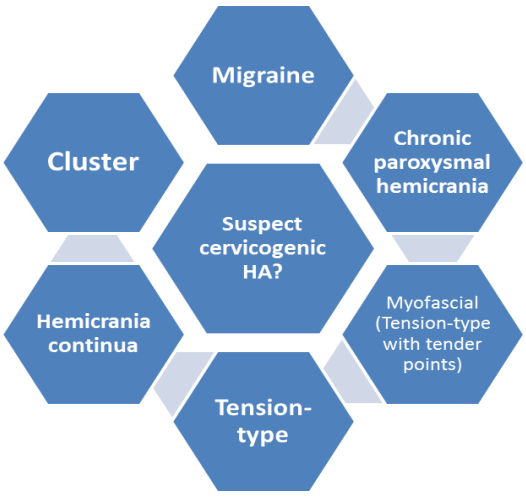
The most common headaches to be seen in a chiropractic care setting are likely to be tension-type, migraine, and cervicogenic. The chiropractic physician must decide which general category of headache the patient has as well as whether there is a musculoskeletal component amenable to manual therapy and exercise. Jull (2008) reports that, in general, there is evidence of moderate agreement in practitioners' ability to identify headache types.

Clinical tip: Determine whether the headache presentation is unilateral or bilateral. Tension-type headaches are usually bilateral (although migraine and, to a lesser degree, cervicogenic headaches are still possible). Unilateral headaches suggest migraine, cervicogenic headache, and a number of less common possibilities.

Generally, when cervicogenic headache is suspected (see p12), it must be differentiated from the following 6 causes of unilateral headache. Note: most of these are considered primary headaches without clear etiology.

- Migraine without aura
- Cluster headache
- Hemicrania continua
- Chronic paroxysmal hemicranias (CPH)
- Myofascial pain syndrome causing a headache (may be classified as tension-type with tender points)*
- Tension-type headache (unilateral uncommon)

UNILATERAL HEADACHES



* The referred head pain associated with myofascial pain syndromes are categorized as "tension-type headaches with tender points" in the international Headache Society system — paradoxically, a type of primary headache *without* known etiology. From a manual therapist perspective, the etiology *is* the MFTP itself. In UWS clinics, we recommend that the headache be charted as the more familiar myofascial pain headache, but coded as tension-type headache with tender points ICD 339-11 or 339.12.

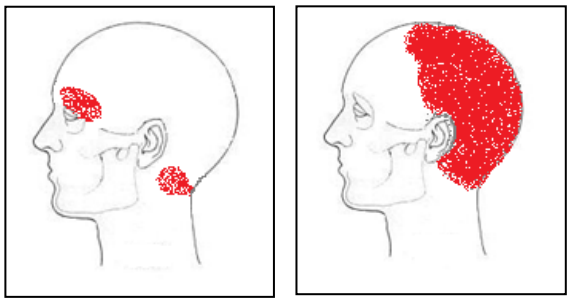
1. Cervicogenic Headache

The following key findings for diagnosing cervicogenic headache are the result of merging the International Headache Society IHS and the Cervicogenic Headache International Study Group (CHISG) criteria. They are further augmented by The Vaga headache study (Sjaastad 2008) which was a 2-year population investigation of 1838 members of the rural population of Vaga, Norway. In this population, 2.2% of the participants had CGH based on CHSIG criteria. Finally, a number of newer physical examination procedures which show promise in aiding in the diagnosis of CGH have been added.

Clues from the History

- **Onset:** Frequently associated with a history of trauma. In one study, the initiating trauma averaged 5.8 years prior (Dumas 2001). When diagnostic blocks have been used, they have *only* been positive in patients with a history of trauma (Bogduk 2009).

- **Location & severity:** Patients complain of a unilateral headache of moderate intensity located over the forehead and temporal regions. The location is "fixed" (i.e., it does not shift sides during a single episode or from episode to episode). The headache is generally described as nagging and non-throbbing in nature. It is less severe than a migraine, but more severe than a typical tension-type headache (Antonaci 2011). When a patient experiences a more severe episode, the headache may become bilateral; in those cases the usual symptomatic side may continue to present as the worst side. (Antonaci 2011) There also can be an associated vague non-radicular type pain in the shoulder or upper extremity.



Cervicogenic Headache

- **Chronology & timing:** The pattern of the attacks tends to be episodic with an unpredictable duration (hours to days) and can evolve into a chronic fluctuating headache. (van Suijlekom 2010).

- **Onset triggers:** Headaches may be triggered by neck movement or awkward head positioning (e.g., sustained postures at work).

- **Associated symptoms:** Autonomic symptoms are infrequent and less severe than in migraines, but may include nausea, vomiting and ipsilateral periocular edema or flushing. Other possible symptoms include dizziness (see CSPE protocol: Dizziness/Vertigo Immediate Care for Sudden Onset), phonophobia or photophobia*, ipsilateral vision blurring, and difficulty swallowing. (Sjaastad 1991. Antonaci 2006)

The most common symptom features are pain that originates in the neck (97% of cases in the Vaga population study) and then spreads to the forehead and temple region; vague non-radicular type pain in the shoulder or upper extremity; and reproduction of neck pain with neck movement (van Suijlekom 1999, 2000; Sjaastad 2008).

> ***Clinical tip***: Suspect CGH in patients with unilateral headache that starts in the neck and spreads to the forehead or frontotemporal regions.

Many of the above reported symptoms are not unique to cervicogenic headaches. They can be present in patients with other types of headaches and so other competing diagnoses (e.g., migraine, tension-type headache) must be ruled out.

*According to the IHS method of categorizing headaches, cervicogenic headache may present with either phonophobia or photophobia, but not both; when both are present the symptoms are more suggestive of migraine headache.

Clinical Tip **The following information from the history can be useful:**	
Does your headache shift from side to side, either during the headache or from episode to episode?	**If yes**, cervicogenic headache is less likely and migraine more likely.
Where does your headache start?	**If HA starts in the neck *first*,** cervicogenic headache or a myofascial pain syndrome is more likely.
If the headache is bilateral, does one side consistently hurt more than the other?	If one side is dominant, cervicogenic headache remains in the differential.

A pragmatic approach to making this diagnosis is to consider the presence of unilateral headache without side-shift associated with pain starting in the neck and spreading to the oculo-fronto-temporal areas as a <u>possible</u> CGH diagnosis. If all of the following findings are also present, a <u>probable</u> CGH working diagnosis can be assigned:

- Symptoms and signs of neck involvement: pain triggered by neck movement or sustained awkward posture and/or external pressure on the posterior neck or occipital region; ipsilateral neck, shoulder, and arm pain; reduced range of motion.
- Pain episodes of varying duration or fluctuating continuous pain
- Moderate, non-excruciating pain, usually of a non-throbbing nature

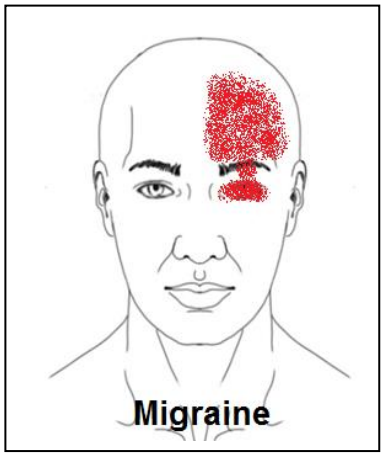
Clues from the Physical Examination

Key physical exam findings for cervicogenic headache include the following:

- **Painful, hypomobile, upper cervical spine segments (C0-3).** Palpation over the occipital area and upper cervical spine may reproduce head and neck pain.
- **Restricted global/active cervical range of motion (CROM).** Although restricted CROM is one of the diagnostic criteria, it is commonly restricted in other headache conditions, although usually not to the same degree.
- **Impaired deep neck flexors.** A number of procedures including the Jull test or the cervicocranial flexion test may be able to detect weakness or poor motor control.
- **The cervical flexion rotation test (CFR).** A positive cervical flexion rotation test, on the other hand, is more strongly linked with probable cervicogenic headache than with migraine or tension headaches.

2.Migraine Headaches without Aura

Cervicogenic headaches must be differentiated from migraines without aura. The *ID Migraine*™ screen is a quick way to do an initial screen for migraine headaches (including those with aura).



ID Migraine Quick Screen (Lipton 2003)

Step One: Patients must report two or more headaches in the previous three months.

Step Two: The symptoms must be severe enough that the headache limits their ability to work, study or enjoy life or at least the patient presents with a desire to have their headache assessed. (Lipton 2003)

Step Three: The patient must respond YES to at least <u>two</u> of the following three questions:

- Has a headache limited your activities for a day or more in the last three months?
- Are you nauseated or sick to your stomach when you have a headache?
- Does light bother you when you have a headache?

The ID migraine screen has been tested in a variety of clinical settings (primary, secondary and tertiary care). A 2011 systematic review that pooled 5,688 patients from 13 studies evaluated the validity and found the following characteristics (Cousins 2011): sensitivity 84% (95% CI 75%-90%), specificity 76% (95% CI 69%-83%), +LR 3.6 (95% CI 2.8-4.6), -LR 0.21 (95% CI 0.14-0.32).

These characteristics suggest that the ID Migraine screen is very helpful for ruling out migraine headache and mildly to moderately useful for confirming the diagnosis.

Additional symptoms include unilateral weakness, paresthesia or numbness. Aphasia or speech difficulty is also possible during the headache episode. All of these symptoms are reversible. (Godwin 2001)

The IHS requires five previous attacks to make a firm diagnosis. (IHS 2004)

MIGRAINE VS. CERVICOGENIC HEADACHE

Migraine must be differentiated from a cervicogenic headache. These two types of headaches in their most classic presentations are relatively easy to differentiate. In many cases, however, there is considerable overlap making diagnosis less certain. Similarities include the fact that both types of headaches can be unilateral, are more common in women seeking care, and may present with nausea or vomiting. The duration of CGH is generally longer lasting than for a typical migraine (Antonaci 2006). Additional clues that would suggest a migraine headache over a cervicogenic headache would include a prodrome, an aura, or a clear non-musculoskeletal trigger (e.g., red wine, menses, etc.)

According to the IHS classification system, prodromes include symptoms that precede the migraine headache by hours up to a couple of days. They can include photophobia, phonophobia, nausea, blurred vision as well as more general symptoms such as trouble concentrating, neck stiffness, yawning and pallor. The prodrome phase is different from an aura and can occur with migraines classified as with or without aura.

The aura is a complex of neurological symptoms that occur just before or at the actual onset of the headache and consist most frequently of visual symptoms (e.g., flickering lights/spots/lines, loss of vision), followed by unilateral sensory symptoms (e.g., paresthesia or numbness which may include the extremities) and, least commonly, dysphasia. There is usually no motor component and all of the symptoms are fully reversible, usually within an hour although the headache lasts longer. Cervicogenic headaches and other headaches, including migraine, may co-exist in the same patient. Studies have reported combination headaches ranging from 17% (Antonaci 2001) to 56% (Pfaffenrath 1990). Carefully questioning can reveal that the patient is very aware of experiencing two distinctly different headaches. (Sjaastad 1999) For key differentiating features, see Table 8 below.

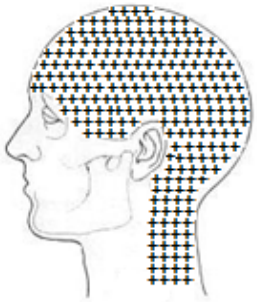
TABLE 8: DDX CGH and Migraine Headaches

Finding	CGH	Migraine no aura
History		
Unilateral HA that shifts from side to side (during or between episodes)		+
Pulsatile headache		+
Pain typically begins in neck	+	
Pain typically begins in head		+
Associated vague non-radicular pain in the shoulder or upper extremity	+	
Severe/dominating nausea, vomiting, photophobia, phonophobia	Mild, if present	+
HA responds to ergotamine or sumatriptan		+
History suggests the HA improves or goes away during pregnancy		+
Prodrome		+
Finding	**CGH**	**Migraine no aura**
Physical		
Decreased cervical active ROM	++	+
Reduced cervical rotation with the neck in flexion	+	
Palpatory pain and loss of joint play in upper cervical joints	++	+
HA provoked by manual pressure on the upper cervical spine (sympto-matic side), or with continuous neck extension	+	
Poor endurance and control of the deep neck flexors	+	
Combination: Reduced AROM, upper cervical joint dysfunction, and positive craniocervical flexion tests	+	
Favorable response to diagnostic nerve/joint block	+	

One plus sign (+) and two plus signs (++) represent relative degrees of response. (van Suijlekom 2010, Jull 2008, Antonaci 2011)

3.Tension-Type Headaches

Tension-type headaches are thought to be the most common form of headache, with one year prevalence ranging from 31-74% (Rasmussen 1991, Scher 1999). Unilateral presentations, however, are uncommon. Tension-type headaches should not be confused with tension headaches which are considered to be primarily psychological in origin. The cause of tension-type headaches is less clear although it can be linked with pericranial tenderness (IHS 2004).



Composite pain pattern of tension type headache

A tension-type headache diagnosis is based on patients having 10 or more episodes fulfilling <u>all</u> of the following criteria (IHS 2004):

- **Duration:** Headaches lasts from 30 minutes to 7 days
- **<u>≥</u> 2 of the following characteristics**:
  - **Quality** of pain is pressing/tightening, but not pulsating
  - **Severity** is mild to moderate (inhibiting, but not prohibiting activities)
  - **Location** is bilateral (although unilateral forms occur that must be differentiating from other unilateral headaches)
  - **Aggravating factors**—*no* aggravation with walking stairs or similar activities
- **<u>Both</u> of the following pertinent negatives**
  - *No* nausea or vomiting
  - Photophobia and phonophobia are not *both* present (but one *or* the other may be present)
- **The headache is *not* attributable to another type of headache** (e.g., probable migraine without aura, cervicogenic headache or medication overuse headache)

If the patient's headache fulfills all but one of the above criteria and does <u>not</u> fulfill the criteria for migraine without aura, a *probable* tension-type headache diagnosis can be made. (IHS 2004)

Once a tension-type headache diagnosis is made, it can be further characterized as *infrequent episodic* (<1 attack per month), *frequent episodic* (1-14 attacks per month), and *chronic* (>15 attacks per month). The headache can be further classified as associated *with pericranial tenderness* and *without pericranial tenderness*. When pericranial tenderness is present it may be exacerbated during headache attacks.

An example of a tension-type headache diagnosis would be

> *"Chronic tension-type headache without pericranial tenderness."*

Differentiating cervicogenic, tension-type and migraine headaches

Van Suijlekom (2010) suggests that unilateral CGH is easy to differentiate from a tension-type headache, although in the bilateral form this is more difficult. The presentation of these two conditions overlap and a patient may have a mix of both types of headaches. The key differentiating features suggesting CGH are provocation of the headache symptoms by mechanical pressure and/or continuous backward tilting of the head, limitation in movement of the neck, and a non-radicular, ipsilateral diffuse shoulder/arm pain.

In one sample population of patients with headaches, the breakdown in clinical features comparing tension-type headaches, cervicogenic headaches and migraines without aura can be found in the Table 9: Comparison of Clinical Traits of Cervicogenic, Tension-Type and Migraine without Aura.

TABLE 9: Comparison of Clinical Traits of Cervicogenic, Tension-type and Migraine without Aura Based on Vaga Population Study			
Clinical trait	**CGH**	**T-TH**	**Migraine (no aura)**
Unilaterality, %	**100**	**8**	**52**
Mechanical precipitation, %	**100**	**4**	**4**
Posterior onset, attacks, %[a]	**97**	**30**	**22**
Throbbing pain quality, %	**20**	**22**	**81**
Chronicity of pain[b]	**+**	**-/+**	**-**
Diffuse arm discomfort, %	**100**	**7**	**8**
Restriction, ROM, %[c]	**93**	**17**	**16**
Photophobia, %	**19**	**15**	**68**
Cervicogenic factor score (0-5)	**2.37**	**0.72**	**0.93**

[a]CGH versus migraine without aura: *P<0.001* (chi-squared test); [b]*Plus sign* (+) indicates invariably/close to invariably present; *minus/plus sign* (-/+) indicates may or may not be present; *Minus sign* (-) indicates generally not present; [c]Reduction of rotation: ≥15 (Data from Sjaastad and Bakketeig)

THE CERVICOGENIC FACTOR (CF)

The cervicogenic factor (CF) is a score based on the sum of five components (Antonaci 2011):

1) ROM deficit;
2) positive skin-roll test in the shoulder area;
3) precipitation of head pain with 3 to 4 kg of external pressure against the occipital tendon insertions;
4) tenderness of the splenius muscles/upper trapezius area;
5) cervical facet joint tenderness.

In the Vågå study, the mean CF values were < 1.0 in headache-free inhabitants (0.42),for T-TH (0.72); for migraine without aura (0.93). Patients with CGH averaged a score of 2.37. (Antonaci 2011)

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4. Hemicrania continua

The prevalence of this headache is unknown but considered to be relatively rare (Cittadini 2011). A unilateral primary headache (i.e., without known etiology), it presents on a chronic daily basis. The severity may change throughout the day. Unlike a cervicogenic headache, however, it can present with conjunctival injection, lacrimation and ptosis, and it responds dramatically to indomethacin. (van Suijlekom 2010, Lipton 2003)

5. Cluster Headaches

Characteristic findings include severe, strictly unilateral pain located about the eye. The ratio of male vs. female varies from 7.2:1 when age of onset is 30-49 to 2.3:1 when age of onset is over 50 (Ekbom 2002). This is an uncommon headache with reported incidence of 2.5 per 100,000/year (95% CI 1.14-4.75) and prevalence 56 per 100,000 (95% CI 31.3-92.4) (Tonon 2002).

Its brief, episodic nature and intense pain (the patient cannot sit still) help to differentiate it from CGH (van Suijlekom 2010). The pain may last anywhere from 15 minutes to 3 hours, repeating 1-8 times throughout the same day. The headache swarm occurs over a matter of days or weeks, followed by long headache-free periods. Important differentiating symptoms include conjunctival injection, lacrimation, nasal congestion, rhinorrhea, forehead and facial sweating, miosis, and eyelid edema. As many as 30% of such patients may display ptosis (Godwin 2001).

6. Chronic paroxysmal hemicranias (CPH)

CPH, like cervicogenic headaches, is a unilateral headache that may be rapidly precipitated by palpation or mechanical stimulation of posterior cervical structures, especially the transverse processes of C4 or C5 and along the groove behind the mastoid process (see table on next page). Unlike cervicogenic headache, episodes are generally very severe, frequent (periods with > 5 episodes per day), and of short duration (3 to 30 minutes). Like cluster headaches, they are often accompanied by autonomic symptoms such as unilateral conjunctival injection, lacrimination, rhinorrhea, eyelid edema, and meiosis or ptosis. CPH is much less common than cluster headache, often of shorter duration (2-30 minutes), more frequent (often ≥ 5 per day) and more common in females (Lipton 2003). A key differentiating factor is that indomethacin completely blocks episodes of CPH (similar to hemicrania continua). This not the case for cervicogenic and cluster headaches (van Suijlekom 2010). (IHS 2004).

TABLE 10: Hypersensitive areas of chronic paroxysmal hemicranias (CPH) and cervicogenic headache

Location	CPH	Cervicogenic
Groove behind mastoid process	++	++
Greater/minor occipital nerves	+	+
Transverse processes, C4/C5	++	(+)
Tendon insertions, along bony ridge: external occipital protuberance, mastoid process	+	++
Upper part sternocleidomastoid muscle	?[a]	++

This version represents the current practice, after some mostly minor adjustments from the original version

One plus sign and two plus signs represent two clearly different degrees of responses; with (+) there may or may not be a weak response

[a]The hypersensitive area may correspond to the area where the minor occipital nerve crosses over the dorsal margin of the muscle

Adapted from Antonaci (2011)

Note: Sensitivity over the occipital nerve also suggests occipital neuralgia (See Appendix VI). It must be differentiated from CPH and cervicogenic headache. Both greater occipital nerve and C2 root allodynia have been reported in cervicogenic headache. (Sjaastad 1998, Jull 2008)

7. Myofascial Pain Syndrome causing Headaches

The IHS does not recognize myofascial pain syndrome as a separate headache category. The closest description IHS has is tension-type headaches with tender points (IHS 2004). The tender points associated with tension-type headaches are peri-cranial, whereas myofascial trigger points most often are cervical or temporomandibular in origin. Manual therapists and many other types of practitioners recognize headaches of myofascial origin. To further complicate the issue, both tension-type headache patients and migraine patients have been reported to harbor myofascial trigger points (Dommerholt 2011).

MFTPs that have been reported to refer pain into the head include those in the upper trapezius, SCM, temporalis, splenius capitis, splenius cervicis and semispinalis (Dommerholt 2011). For diagrams of individual referral patterns, see Appendix III.

Three systematic reviews of the literature (Lucas 2009, Myburgh 2008, Tough 2008) found that there remains a lack of consistent agreement on the diagnostic criteria, a lack of standardization in exam procedures, and a lack of methodological quality in clinical studies of MFTPs.

According to Fernandez-de-lasPenas (2010), the most likely part of the examination to be positive is palpation for a hypersensitive spot, followed by the patient recognizing the elicited pain as familiar, finding a palpable taut band and finally, referred pain. Those tender points that reproduce local pain only have a higher interexaminer reliability than those causing referred pain.

In contrast, based on two high quality studies, Myburgh (2008) reports considerably lower reliability ratings with the best *kappa* values related to local tenderness of the trapezius

muscle (*k* .15-.62) and pain referral of the gluteus medius (*k* range .29-.48). Relative to MFTPs in those muscles capable of producing headaches, McEvoy (2011) offered a "best evidence synthesis" suggesting that acceptable interrater reliability had been established for tenderness in the upper trapezius, taut band in the upper trapezius, and presence or absence of active trigger points in the SCM and upper trapezius (based on a combination of tenderness and a palpated taut band reproducing the patient's familiar pain).

The minimal acceptable criteria for trigger point diagnosis proposed by Simons are: 1) palpation of a tender point in a taut band within skeletal muscle, 2) the patient recognizes the elicited pain from palpation as a typical symptom (Dommerholt 2011). Gerwin (1997) reports that a muscle harboring the trigger point may also be weak. One systematic review (Lucas 2009) suggested that a more practical approach may be to narrow the MFTP criteria to include only local tenderness and local pain reproduction (the two features where an adequate level of agreement may be achievable).

☝ ***Clinical tip***: Based on location of the headache, the practitioner should consider a variety of potential MFTPs. (See Appendix IV)

OTHER CAUSES OF HEADACHE

There are many other types of headaches which may be detected in the history but are not included in this care pathway. They include secondary headaches (i.e., attributable to a known cause) such as unilateral headaches related to dental pain and TMD as well as headaches that are more commonly bilateral such as extra-cranial viral infection, hypoglycemia, allergies, eye strain and refractive errors, and dehydration and sinusitis (see CSPE Sinus Pain care pathway).

A Word on Temporomandibular disorder (TMD)

Temporomandibular joint dysfunction (TMD) should be considered as a possible contributing factor or even a primary cause in patients with headaches. If indicated, a thorough exam of the temporomandibular joint (TMJ) and surrounding structures should be performed in addition to the headache specific exam.

Several studies have revealed shared signs and symptoms between TMD and headaches, specifically with tension-type headaches. (Armijo-Olivo 2012) About 10% of people in the United States suffer from TMJ problems at any given time. One retrospective study of 426 consecutive patients with TMD reported a headache prevalence of 17.1% (Ungari 2012).

The most commonly reported headache locations associated with TMD are the suboccipital and frontotemporal (Tallents 1994) or headaches around and behind the eyes. Some patients describe a headache that comes up from the neck onto the skull (Steigerwald 1996).

A suspicion of TMJ involvement may be triggered by overt clues such as a report of accompanying jaw pain or clues derived from a brief jaw range of motion exam and palpation screen. More circumstantial clues would include evidence of malocclusion, patient habits or behaviors (e.g., nail biting, pen chewing, jaw clenching, leaning face or chin on hands or mouth breathing) or work activities (e.g. holding the phone with a cheek, playing violin or a wind instrument). However, it should be noted, that patients in some cases can present with headache *without* any symptoms in the TMJ. Consequently, before concluding that a headache is essentially a primary headache without known etiology, the TMJ should be assessed.

The International Headache Society suggests that a diagnosis of TMD-related headache can be made based on the clinical exam and ancillary studies. The patient must report recurrent pain in one or more regions of the head and/or face associated with at least one of the following: 1) pain is precipitated by jaw movements and/or chewing of hard or tough food, 2) reduced range of or irregular jaw opening, 3) noise from one or both TMJs during jaw movements, or 4) tenderness of the joint capsule(s) of one or both TMJs. In addition, there should be x-ray, MRI or bone scintigraphy evidence of TMD and the headache should resolve within 3 months, without recurrence, after treatment of the TMJ and/or related muscles. (IHS 2004)
For specific recommendations on how to assess the TMJ, see CSPE protocol: TMJ: A Clinical Assessment.

Physical Examination Procedures Useful for Identifying Cervicogenic Headache and other Musculoskeletal Pain Generators

Summary of Key Examination Procedures to Identify Cervical Pain Generators

- ✓ Cervical AROM
- ✓ Cervical orthopedic testing
- ✓ Palpation of the cervical region and upper thoracic spine
- ✓ Soft tissue palpation especially of muscles related to the cervical spine and cranium
- ✓ Jull test, cervical flexion rotation test (CFR), and cranio-cervical flexion test

The physical examination is important not only to help rule out serious causes of headaches (see (pp.17-18), but it also plays a role in identifying headaches amenable to manual therapy. A reported 70%-87% of patients complaining of headaches report associated neck pain (Jull 2008). Key physical impairments may be useful in differentiating cervicogenic headaches from other types of headaches. (Amiri 2007). In addition, positive exam results may be used to identify other diagnoses such as tension-type headaches (e.g., cranial muscle tenderness) or myofascial pain syndromes (e.g., palpable and tender trigger point in the SCM).

1. Active Range of Motion

Global active range of motion (AROM) evaluation of the cervicothoracic spine should be part of establishing abnormal movement patterns for the patient. Active range of motion is commonly restricted. Zito (2006) reports that while CGH subjects demonstrate changes in AROM, they are only statistically significant for decreased flexion and extension. Antonaci (2011), on the other hand, suggests that cervical rotation is usually reduced by 10˚or more in one direction. Likewise, in the large Vaga series, rotation was also reduced (by 15˚or more) in 93% of the cases. Jull (2007) found AROM to be more consistently limited in CGH patients in both extension and rotation compared to patients with migraines, tension-type headaches or healthy controls. These findings, however, have not been universally reported; two other studies did not find limitations in AROM (Dumas 2001, Hall 2004). Consequently, normal AROM does not strictly rule out a headache of cervical origin.

STANDARDIZING THE PROCEDURE

While the seated patient should first be observed without prompts, assessment of AROM should be done with the patient sitting up straight. The patient should explicitly be encouraged to move *as far as possible* into each direction. If the range is estimated by simple observation, it can be recorded as degrees or as a percentage change either from normal (e.g., "cervical extension is decreased by 50%") or compared to the other side ("right rotation 50% < left"). It should be understood that these visual estimates are not very accurate. Another option is to measure ranges with an inclinometer. Using an inclinometer is particularly recommended in personal injury cases, worker's compensation cases or other medico legal situations where precise measurement may be more relevant.

Interexaminer reliability of measurements using an inclinometer or goniometer generally ranges from moderate agreement (ICC > .60) to substantial agreement (ICC >.80). (Cleland 2011) Besides recording the range of motion, it is important to note if the patient's headache is actually reproduced.

2. Cervical Orthopedic Tests

As a general screen for significant cervical joint lesions, routine orthopedic tests such as cervical compression (including maximum), distraction and shoulder depression may be performed. It is important to note symptom reproduction.

3. Palpation of Cervical and Thoracic Joints

Both static and motion palpation procedures are routinely employed in the assessment of CGH, especially by manual therapists (Hall 2010, Vavrek 2009, Jull 2007, Zito 2006, Dumas 2001, Schoensee). Segmental motion assessment should include occiput on atlas as well as thorough segmental evaluation of the rest of the cervical spine in various vectors (e.g., flexion,

extension, rotation, lateral bending). Palpation of the involved upper cervical structures may reproduce headache or neck pain and provides some of the more useful findings in planning conservative treatment for cervicogenic headaches. Palpation may also reveal loss of joint play, restriction, or altered end feel. Palpatory evidence of cervical joint dysfunction, specifically upper cervical levels C0-C3, seems to be a common finding in CGH.

The thoracic spine has not been directly linked to cervicogenic headaches but should also be evaluated. A T4 syndrome, which is more of a pattern of signs and symptoms than a diagnosis, has been anecdotally reported to include a generalized headache around the top of the head. It may be unilateral or bilateral and is associated with painful and restricted thoracic joint dysfunction in the region of T4 as well as paresthesia in a glove like distribution in the hands. (Boyling 1994, Conroy 2004, DeFranca 1995)

Rationale and test performance

A number of small studies have suggested that the presence of painful, hypomobile joints detected by motion palpation can help differentiate CGH, migraine and no headache populations (Zito 2006, Hall 2010, Jull 2007, Dumas 2004).

The C1-C2 segment has been the most commonly involved segment based on motion palpation (Hall 2010, Hall 2004, Zito 2006). C1-C2 is also commonly identified as a cause of CGH pain using diagnostic blocks although C2-3 is more common, reported in 70% of cases (Bogduk 2009).

Good quality evidence on test validity specifically for CGH is scarce. A 2007 study (King 2007) using double nerve/facet blocks (short acting and long acting) reported a poor +LR 1.7 (95% CI 1.2-2.5), but a potentially useful –LR (0.2) pooled results for the cervical spine. The results for the C2-3 level were slightly poorer at +LR 1.4 (95% CI 0.87-2.40) and –LR 0.3. Patients with negative palpation results, however, were not subjected to diagnostic blocks which could have led to an underestimation of test specificity, which in turn, would make the likelihood ratios poorer. In addition, positive palpation results were correlated only with blocks that affect the posterior elements of the spine and not the intervertebral disc. Jull (2007) reported a +LR 16.6 (sensitivity 100%, 94% specificity) for the combination of palpably painful upper cervical joints, decreased AROM and a positive craniocervical flexion test. Unfortunately, this study as well as most other studies looking at the validity and reliability of physical examination procedures for CGH have not employed diagnostic blocks as a gold standard and are limited to using questionnaires or questionnaires plus physical examination as reference standards.

With these limitations in mind, Schoensee reported excellent inter-examiner reliability for passive accessory joint play in 5 CGH headache patients (k=0.81, p =.0001). Hall (2010) reported moderate interexaminer reliability for painful hypomobility in the C0-C4 region (k=0.68), with the following breakdown C0-1 (k=0.40), C/1-2 (k=0.70), C2-3 (k=0.71). Note that these numbers are considerably better than those reported for cervical palpation on the whole in two large systematic reviews, although most of studies in the reviews were done on asymptomatic subjects or patients with neck pain (Stochkendal 2006, Schoensee 1995).

In the studies cited above specifically on CGH patients, palpation targeted unilateral passive accessory intervertebral motion (PAIM) and passive physiological intervertebral motion (PPIM) (using techniques described by Maitland 2001). In PAIM testing the subject is prone with the neck in a neutral position. The examiner applies unilateral P-A pressure over the articular pillars of the cervical vertebrae. A positive test result is reproduction of the patient's pain, especially their exact symptoms. In PPIM testing (which is similar to standard motion palpation) the subject is supine. The examiner palpates the segments through all planes of motion. A positive test result occurs when the examiner finds hypomobility along with symptom reproduction with PAIM testing. (For more information, see Appendix V: Evidence Table.)

4. Palpation of Cranial, Cervical and Thoracic Soft Tissues

Palpation should be directed toward finding possible soft tissue dysfunction (e.g., changes in texture, tone, sensitivity). Length testing (i.e., diagnostic stretching) key muscles for tightness is also useful.

Several studies have shown muscular involvement with cervicogenic headaches. A common finding is muscle tightness associated with cervical dysfunction. The most commonly identified muscles are the upper trapezius, splenius, scalenes, levator scapulae, suboccipitals and pectoralis major and minor (Zito 2006, Jull 1999). Zito (2006) reported a statistically significant difference between the incidence of tightness in the cervicogenic headache group compared to the migraine and control groups for the upper trapezius ($p=0.003$), levator scapulae ($p=0.001$), scalenes ($p= 0.001$) and the suboccipital muscles ($p= 0.035$) but not for the pectoralis muscles. Overall the incidence of muscle tightness was significantly higher in the cervicogenic headache group, 34.9% of all muscle length tests, than the migraine with aura 16.7% or controls groups 16.3% (Zito 2006).

Pectoralis minor, if shortened, can contribute to an internally rotated and forward shoulder, either unilaterally or bilaterally. It can also contribute to rib cage dysfunction through its anterior attachments. According to Cleland (2011), one small study reported the interrater reliability of length assessment of the pectoralis muscle is *k* 0.90 (0.72-1.0) for the right but only 0.50 (0.01-1.0) for the left side.

Shortened scalene muscles can contribute to an elevated first rib and to cervical joint dysfunction. According to Cleland (2011), based on a small study of subjects with neck pain, the interrater reliability of length assessment of the scalene muscle is *k* 0.81 (0.57-1.0) for the right and 0.62 (0.29-0.96) for the left side.

Palpatory tenderness may be associated with other diagnoses as well. For example, tender points over the cranium are consistent with tension-type headaches. Head pain may be referred from various myofascial trigger points in the neck. (See Appendices III and IV.)

5. Palpation of Special Pain Sensitive Points Associated with Cervicogenic Headache

Van Suijlekom (2010) recommends palpating specific "pressure points" with about 3-4 kg of pressure which may then provoke both local and spreading pain, and, unlike in healthy individuals, will continue to last seconds after the stimulus is withdrawn. An algometer can also be used to standardize the testing pressure and can then be used as an outcome marker (Vavrek 2010). Approximately, doubling of the pressure may provoke the familiar headache presentation. Many of these sensitive points are at tendinous insertions (Antonaci 2011).

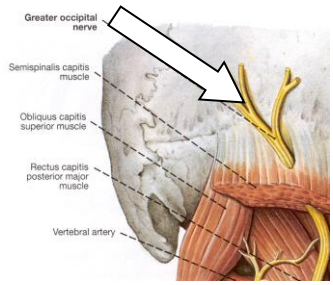
The presence and significance of these sensitive points are based on observation and expert opinion.

- Anterior, posterior, and on the ventral upper trapezius border (Sjaastad 2003)
- C4, C5 transverse processes
- The groove behind the mastoid process (Antonaci 2011)
- Tendon insertions along the ridge of the EOP and mastoid process

Less commonly:

- Lesser occipital nerve (at the attachment of the SCM to the skull)
- Greater occipital nerve (occipital-temporal part of the skull). Note: tenderness of this structure can also be associated with chronic paroxysmal hemicrania. Sharp pain when palpating the greater occipital nerve as an isolated finding is consistent with C2 neuralgia or greater occipital neuritis. (See Appendix VI.)

Palpating the greater occipital nerve



Palpate approximately 2 cm down and 2 cm lateral to the external occipital protuberance. This can be approximated by placing the thumb along side of the EOP (pointing super ward) and using the base of the nail bed to identify an area to palpate. (Loukas 2006)

6. Special In-Office Tests

The number of special physical examination procedures may also be useful in the assessment of a suspected cervicogenic headache.

SUMMARY

1. Deep flexor endurance and motor control tests.
   - Neck flexion movement pattern
   - Jull/cervical instability test
   - Craniocervical flexion test
2. Cervical flexion-rotation test (CFR)

1. DEEP FLEXOR ENDURANCE AND COORDINATION TESTS

Inhibition or poor endurance of the deep flexors has ben associated with cervicogenic headaches (see below). There are a several methods to assess the function of the deep neck flexors (i.e., longus colli and longus capitis). Poor endurance, motor control, and contraction speed of the deep cervical flexor muscles can be assessed by performing the neck flexion movement pattern, Jull test with variations, and the cranio-cervical flexion test.

1a. Neck flexion movement pattern

To establish a baseline movement pattern the practitioner should observe the supine patient's active neck flexion. Without further instruction, the patient is asked to slowly raise their head from the table. If the chin pokes forward at the beginning of the movement, generally the first 10 degrees, suspect weakness or inhibition of the deep neck flexors and overactive SCMs. Slight fingertip resistance on the forehead may be used to emphasize the abnormal pattern (Murphy 2000).

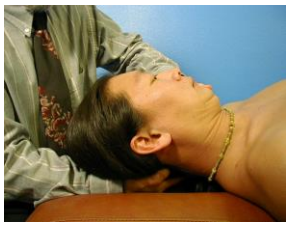
Rationale and test performance

This movement pattern has not been investigated in clinical trials and is based on clinical experience, expert opinion (Janda 2002, Murphy 2000, Liebenson 2007) and biomechanical plausibility. The protrusion of the chin is thought to signal deep flexor weakness which is compensated by early and inappropriate activity of the SCM (which produces upper cervical extension and lower cervical flexion resulting in chin poking).

1b. Jull test/cervical instability test/deep flexor endurance test

The Jull test is a test of deep cervical flexor muscle endurance (e.g., longus coli, longus capitis) and is thought to reflect functional stability.

With the patient supine, the examiner retracts the patient's chin, raises their head slightly off the table (about ½ inch), and then slowly releases.



A common threshold is for patients to hold this position for 10 seconds without chin poking, excessive head shaking, or global flexion or extension (Murphy 2000). When used purely as an endurance test (shaking allowed), a 2007 (Peolsson) study of 116 asymptomatic subjects (age 25-64) suggested cut points of 56 seconds for men and 23 seconds for women (median times were 150 seconds and 30 seconds respectively but with a wide range).

Failure indicates overall poor functional stability of the cervical spine, inhibited deep flexors, and perhaps overactive SCMs (Murphy 2000, Liebensen 2007).

Clinical tip: An optional method to standardize the starting position for the Jull test is for the practitioner to rest hands on the table, cradling the patient's head. The patient is then instructed to elevate his/her head just until there is no contact with the practitioner's fingers.

A variation of this test can be used to qualitatively evaluate the speed of contraction and reaction of the deep neck flexors.
The practitioner raises and positions the patient's head as described in the cervical stability test above. In this variation, the patient is warned that their head will be suddenly released in the next few moments, but that meanwhile they should allow the neck to remain relaxed and supported by the practitioner. The head is then released suddenly. The practitioner observes how quickly the patient can recover and how accurately they can return to the starting head position. Excessive overshooting, slow response, or inability to return to roughly the same starting point indicates poor control and speed of contraction and perhaps poor kinesthetic awareness.

***!!! Clinical warning*:** This test is only performed when the flexors demonstrate good strength, endurance and any acute injury has had time to heal.

Rationale and test performance

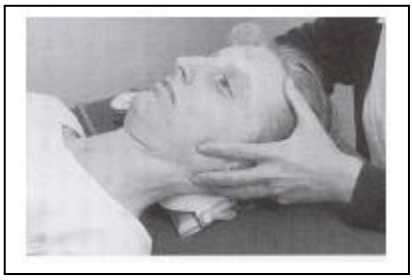
A 2008 systematic review suggested that the Jull test had adequate test reliability for the endurance component. Most studies contained in the review calculated the intra-observer reliability to be above an ICC 0.85 (deKoning 2008). Test results correlates well with patient's symptoms as measured by the Neck Disability Index (NDI). Validity of this test is unknown. The test variation which assesses speed of contraction has not been studied. (See Appendix VII: Evidence Table.)

1c. Craniocervical flexion test (using pressure sensors)

The craniocervical flexion test is a noninvasive, low load test to evaluate the holding capacity of the deep neck flexors.

With the neck in a neutral position, an inflatable air-filled pressure sensor (Stabilizer, Chatta-nooga South Pacific) is placed suboccipitally behind the neck and inflated to 20 mmHg

Watching the pressure gauge, the patient very slowly flexes the upper cervical spine with a gentle head nodding motion to increase the pressure in 2 mmHg increments and holds each new position steady for 10 seconds. This should occur with minimal activity in the superficial muscles. An ideal response is that the patient can increase pressure by 10mmHg in 2mmHg intervals for a total of 5 levels of increased pressure. Most neck pain patients fail after the first two intervals and demonstrate an inability to hold the position steady (Jull 1997). For specific details on performing this test, see Appendix VIII.



Rationale and test performance

Several studies have shown neuromuscular impairment during this test in patients with chronic neck pain and CGH patients but not with tension-type headaches or migraine (Amiri 2007, Dumas 2001, Jull 2007, Zito 2006, Zwart 1997).

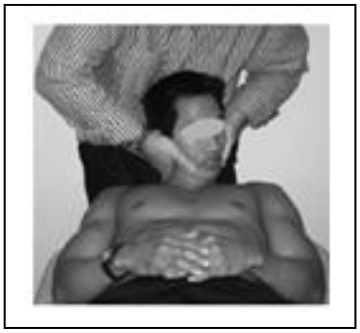
***Clinical Tip*:** In one study, test failure had 100% sensitivity and 94% specificity (+LR 16) for differentiating CGH from tension-type headache and migraine when in combination with palpably painful upper cervical joint dysfunction and restricted global range of cervical extension. (Amiri and Jull 2007a).

A 2008 systematic review questioned the test's reliability (de Koning 2008). The review reported acceptable intra-examiner ratings (ICC scores 0.65 to 0.93) from four studies, but three of these suffered from design flaws and small cohort sizes. (Chiu 2005, Jull 1999, Jull 2000). One study judged to have a "satisfactory" design reported "fair" inter-examiner reliability (ICC values of 0.51) but below the criterion of 0.70. (Hudswell 2005) A subsequent 2010 study (James 2010) found intra-examiner reliability to be excellent (0.983; standard error of the mean = 8.94) although this test, too, had issues with blinding. A 2011 study (Arumugam 2011) on asymptomatic subjects reported excellent inter-examiner reliability with an ICC of 0.91 (95%CI 0.83-0.96). Other studies have related an altered electromyographic amplitude of the deep and superficial neck flexors to changes found on the pressure gauge during the craniocervical flexion test. (Jull 2004, Falla 2004) Although

electromyography of the superficial neck muscles has been shown to be reproducible, (Falla 2002,Falla 2003, Oksanen 2007) evidence for the reproducibility of measuring deep cervical flexor muscles with electromyography is lacking (Falla 2004). Therefore, whether the craniocervical flexion test truly reflects deep flexor activity is still in question (deKoning 2008). (See Appendix IX: Evidence Table.)

2. CERVICAL FLEXION-ROTATION TEST (CFR)

The cervical flexion-rotation test is performed on a supine subject. First, the cervical spine is passively fully flexed. The head is then rotated to the left and the right until firm resistance is encountered or pain is evoked.



Positive findings include limited range of rotation before the expected end range (less than 32 degrees of rotation as measured with a goniometer or at least a 10 degree loss from the expected range by visual estimation), reproduction of familiar headache pain, or reproduction of familiar neck pain.

Rationale and test performance

Most clinical research on the validity of physical examination procedures for making a CGH diagnosis are limited by the fact that facet blocks were not used as the gold standard. Instead for the following studies, the reference standard for cervicogenic headaches was based purely on history and patient interview using the Cervicogenic Headache International Study Group criteria and Antonaci's criteria for "probable" CGH which may further limit the true test validity (see p. 5).

Keeping in mind those caveats, there is evidence that the CFR test may be useful to help differentiate headache from non-headache patients (Hall 2010b), CGH from migraine headache (Ogince 2007), and identifying neck pain originating from the upper cervical spine (Hall 2010c). In addition, the test appears to have good reliability (Hall 2008) and may be useful in tracking progress in cervicogenic headache patients (Hall 2010d, Hall 2007, Ogince 2007)

Relationship to C1-C2. The CFR test is presumed to assess the rotation ROM at C1-2. Rotation in this position normally ranges from 40-44 degrees (Hall 2004, Amiri 2003) and has been reported to be significantly reduced in patients with C1-C2 joint dysfunction. (Hall 2004, Ogince 2007) When the cervical spine is placed in end-range flexion prior to head rotation, rotation is blocked at the levels below C2. This presumed effect was confirmed in a 2011 study with 19 asymptomatic female volunteers (Takasaki 2011). In vivo MRI observation of the CRF test demonstrated a 61%-77% reduction in segmental rotation from C2-3 through C6-7 and only a 16% reduction at C1-2. (Takasaki 2011). In addition, pain originating from the lower cervical joints does not appear to substantially influence the test. (Hall 2010)

Differentiating CGH from non-headache patients. Compared to patients without headache complaints, the range of motion is reduced to reported averages of 22 degrees (Hall 2010) and 26 degrees (Hall 2008)—well below the normative range of 40-44 degrees. When interpreted as a simple positive or negative test, it has a +LR of 5.18 and –LR of 0.26 for CGH when compared to normal controls (based on clinical criteria as a reference standard). These findings are present even on patients who are not experiencing a headache at the time of the testing. The degree of motion reduction appears to correspond to the patient's average headache intensity and can be further reduced (an average of 6 degrees) if tested while a headache is in progress (Hall 2010d). It is interesting to note that traditional cervical range of motion measurements while reduced in migraine headache patients in general, is not further reduced while experiencing a migraine (Bevilaqua-Grossi 2009).

Differentiating CGH from migraine. In a 2007 single blind comparative study, Ogince et. al. evaluated the validity and interexaminer reliability of the CFR test. The sensitivity was 91% and the specificity was 90% for C1/2 involvement in patients with cervicogenic headache. They demonstrated that the cervical flexion-rotation test was useful in differentiating cervicogenic headaches from migraines.

The test has a reported +LR 2.33 (95% CI 1.34-4.06) and –LR 0.43 (95% CI 0.21-0.85) in predicting the presence of CGH vs a cohort of patients with either migraine or mixed headaches. (Hall 2010e).

Reliability. Inter-examiner reliability of the CFR test was reported to be excellent with an ICC* of 0.93 (CI 95%, 0.87-0.96). (Hall 2008) It is worth noting that this level of agreement was based on the therapists' estimates of a firm end feel with at least a 10° reduction from the expected range (independent of ROM measuring instruments). This method of test interpretation has been shown to be valid and reliable when compared with goniometry. (Hall 2008)

Excellent intra-rater test-retest reliability (k = 0.92 (95% CI: 0.77-1.00; p<.001) has also been demonstrated when the test is followed by an immediate retest for both asymptomatic subjects and subjects with CGH (Hall 2007, Ogince 2007). A minimal detectable change (MDC) of 7 degrees has been reported (Hall 2007), well within the estimated 10 degree change necessary to constitute positive test. (See Appendix X: Evidence Table.)

ASSESSMENT STRATEGY

Step 1: Rule out serious ominous headaches.
Step 2: Identify the type of headache (cervicogenic, myofascial, migraine, etc.) and pain generating tissue.
Step 3: Identify any significant triggering or contributing factors (e.g. forward head carriage, deep flexor weakness).
Step 4: Determine the need for ancillary tests/studies.
Step 5: Establish outcome measures and severity of the condition.
Step 6: Determine if there are significant psychosocial factors.
Step 7: Establish a prognosis.

Step 3: Identify any significant triggering or contributing factors (e.g., forward head carriage, upper cross syndrome).

In addition to identifying the type of headache, the practitioner should assess the patient for the presence of potential contributing factors. These are factors which, while they themselves are not pain generators, may create alterations in the overall function of the spinal kinetic chain or promote mechanical loads which result in tissue irritation—contributing to or sustaining the patient's headache presentation. This section will focus on forward head carriage and muscle imbalances. For the most part, these entities as they relate to headache have not been extensively studied and so the possible relationship is based primarily on biomechanical plausibility and expert opinion.

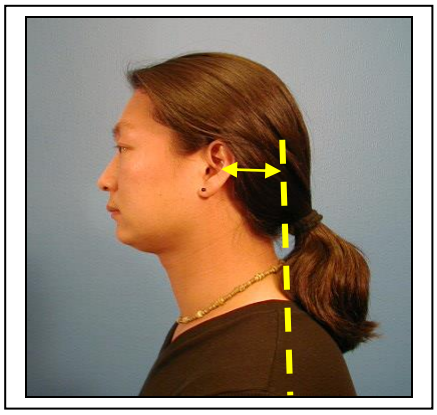
1. Postural analysis

Forward head posture has been implicated in neck pain and certain types of headaches, such as tension-type headaches associated with MFTPs. Fernandez-de-las-Penas 2006) The relationship between forward head posture and cervicogenic headaches, however, has not been clearly established (Zito 2006). Anterior head carriage causes stress on the structures of the cervical spine that theoretically could contribute to joint dysfunction or myofascial pain syndromes other than cervicogenic headache specifically.

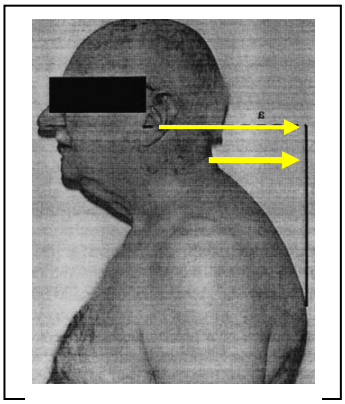
* An interclass co-efficient is the measure of agreement when scales with continuous data are used (such as range of motion). An ICC above .75 is considered excellent.

It may be useful to ascertain whether the patient can correct the forward head posture on their own. If not, more significant structural causes need to be evaluated. The patient should be observed standing, seated, and while transitioning from seated to standing posture.

Anterior head carriage is indicated when the external auditory meatus is drawn forward past an imaginary plumb line that runs from the lateral maleolus perpendicular up through the glenohumeral joint (see picture below)



If anterior head carriage is present, it can also be quantified by measuring the distance of the external meatus to the wall or the distance of the deepest portion of the lordosis to the wall (Simons 1999).



"Fixed." Anterior head carriage that is present both seated and standing is likely "fixed." This type of posture may be associated with inhibited deep flexor muscles of the cervical spine, an upper cross syndrome (see below) and/or hyperkyphosis of the thoracic spine. If the anterior head carriage is present while standing, but not when seated, the forward head may be the result of a forward lean originating at the lumbar spine or even at the ankle. Part of this postural shift could be associated with a lower cross syndrome. In such cases, treatment will need to be directed to these causes. (For more information, see Appendix XI: "Fixed" Head Position.)

> ***Clinical tip*:** In patients with forward head carriage, palpate for hypertonic cervical extensor muscles in the standing patient. If the muscles seem to relax when sitting, the tension may be related less to a local cervical problem and more to a problem in lumbars, pelvis or lower extremity (Murphy 2000)

Behavioral. For some patients, inefficient posture may be more of just a habit, present only in certain situations. Sitting may be one such situation and can be observed while the patient is in the waiting room or sitting during the history. In other patients, the problem may occasionally be a "rest" position adapted after exercising. Patients sit and lean forward, resting their arms and upper body on their thighs. Finally, it may be the result of ergonomic problems at the work place or at home.

Jull (2008) postulates that whereas the evidence linking cervicogenic headache with a "fixed" posture is weak, sustained cervical extension loading in a seated posture might present a more promising connection.

Dynamic. Occasionally "dynamic" anterior head carriage may be detected only when the patient is moving from a seated to standing position (leading with the chin while getting out of the chair), with physical stress (e.g., lifting, performing exercises such as sit ups or flies) or in some pathological states (e.g.,COPD). (Murphy 2000).

(For more information on postural analysis, see Appendix XII.)

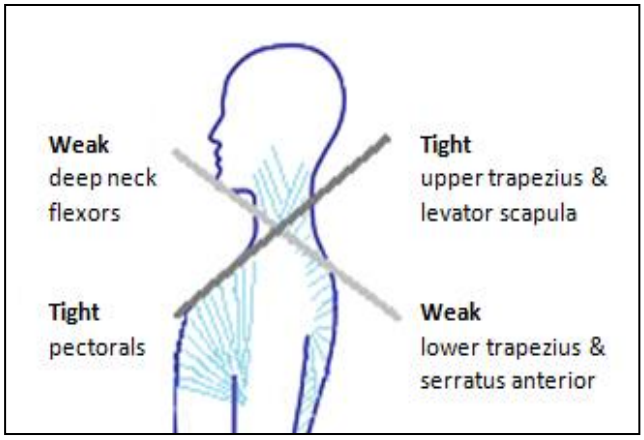
2. Muscle imbalances

Muscles imbalances, by altering biomechanical loads on joints and muscles, are speculated to contribute to neck pain and headaches. (Murphy 2000, Jull 2007)

Inhibition of lower trapezius and serratus anterior muscles is thought to be a common clinical finding (Janda 1994; Jull 1994; White & Sahrmann 1994) although there is little research evidence supporting this contention (Beeton & Jull 1994, Grant 1998). Lower trapezius activity and endurance is evaluated by isometric testing of the scapula in a position of adduction and depression. To restrict the action to the lower trapezius as much as possible, load is removed from the test by leaving the arm by the side. The patient is prone. The practitioner places the scapula in the test position with the patient's arm by his/her side. Recruitment of latissimus dorsi, levator scapulae or upper trapezius is noted. (For more information, see Appendix XIII.)

Deep neck flexor inhibition may be a part of a larger muscle imbalance referred to by Janda as an upper cross (or proximal cross) syndrome

(Janda 1994.) The patient may have all or part of the aberrant pattern. The upper half of the pattern includes an imbalance in the cervical spine comprised of short tight extensor muscles (suboccipitals, upper trapezius and levator scapulae) and inhibited deep flexors (longus colli and longus capitis). An associated finding can be overactive sternocleidomastoid muscles.



The lower imbalance is primarily comprised of short tight pectoralis muscles and inhibited middle/lower trapezius, sometimes associated with an inhibited serratus anterior. This pattern is seen in association with chronic recurrent neck pain, headaches and shoulder problems.

ASSESSMENT STRATEGY

Step 1: Rule out ominous headaches.
Step 2: Identify the type of headache (cervicogenic, myofascial, migraine, etc.) and pain generating tissue.
Step 3: Identify any significant triggering or contributing factors (e.g., forward head carriage, deep flexor weakness).
Step 4: Determine the need for ancillary tests/studies.
Step 5: Establish outcome measures and severity of the condition.
Step 6: Determine if there are significant psychosocial factors.
Step 7: Establish a prognosis.

Step 4: Determine the need for ancillary studies.

Ancillary studies, including imaging, diagnostic blocks, and blood work are rarely indicated in assessing headache patients. In special circumstances, however, advanced testing procedures can be critically important.

TABLE 11: Types of ancillary studies

Condition	Ancillary studies
Suspected instability (secondary to trauma or inflammatory disease (e.g., RA, AS)	Dynamic cervical films, cervical MRI
Suspected intracranial lesion (e.g., tumor)	MRI or CT (head)
Suspected subarachnoid hemorrhage or subdural hematoma	MRI or CT (head), spinal tap
Suspected meningeal infection	MRI or CT (head), spinal tap
Temporal arteritis	Biopsy
Refractory CGH	Facet block

IMAGING

Unless traumatic in origin, diagnostic imaging is not usually necessary in the assessment of headaches outside an emergency department arena.

Cervicogenic headaches
While imaging may demonstrate other potential pathologic causes of headache (see table 12), there are no diagnostic radiographic findings that will either rule in or rule out cervicogenic headache (van Suijlekom 2010, Bogduk 2009). Although osteoarthritis of the spine is thought to generate headaches, even in this case the presence of radiographic degenerative changes is not diagnostic. Some practitioners, however, may choose to alter their approach to manipulation based on the degree of degeneration seen on a radiograph. MRI scans of patients with the clinical features of CGH demonstrate no unique features to differentiate them from controls (Hoppenfeld 2010).

Migraine headaches
For migraine patients in a primary care setting, neuroimaging (e.g., CT/MRI) is not usually warranted unless accompanied by an abnormal neurological examination. (Miller 2006, Grayson 2005)

Intracranial lesions and other serious headaches
The following information is based primarily on emergency department recommendations

TABLE 12: Indications for imaging

Indication	Imaging
"Thunderclap" HA with abnormal neuro exam	Emergent MRI
Isolated thunderclap HA	Consider referral for CT; abrupt onset HA has +LR 2.5 for intracranial lesion (based on one validating cohort study)
New onset if high risk for intracranial disease (e.g., HIV positive, prior dx of cancer)	Consider MRI or CT
HA with abnormal neuro exam (e.g., papilledema, unilateral loss of sensation, weakness, hyper-reflexia)	Consider MRI or CT, +LR 4.21 for intracranial lesion (based on one validating cohort study)
HA with fever or nuchal rigidity	MRI or CT
Progressively worsening HA	MRI or CT
Change in character of the HA	Consider MRI, +LR 2.0 for intracranial lesion (based on validating cohort study)
Persistence despite analgesics/treatment	X-ray, MRI or CT

(adapted from Grayson 2005, Miller 2006)

DIAGNOSTIC BLOCKS

Diagnostic blocks can confirm the presence of cervicogenic headache. They are rarely necessary in routine practice, but can be used when patients' response to conservative care is not satisfactory. To improve their accuracy, blocks should be placebo-controlled and performed under fluoroscopic guidance (Jull 2008, Lord 1995). Although not usually necessary, they are indicated when therapeutic facet blocks or more aggressive treatments such as neurotomies are being considered. (Govind 2003, van Suijlekom 1998, Jull 2008)

Step 5: Establish outcome measures and severity of the condition.

ASSESSMENT STRATEGY

Step 1: Rule out serious organic headache.
Step 2: Identify the type of headache (cervicogenic, myofascial, migraine, etc.) and pain generating tissue.
Step 3: Identify any significant triggering or contributing factors (e.g. forward head carriage, deep flexor weakness).
Step 4: Determine the need for ancillary tests/studies.
Step 5: Establish outcome measures and severity of the condition.
Step 6: Determine if there are significant psychosocial factors.
Step 7: Establish a prognosis.

A number of outcome measures can be used. Many of these will establish baseline severity and provide a means to track and measure patients' response to care.

FROM THE HISTORY

- **Symptoms.** Headache frequency, duration and severity, measured on an oral pain scale (OPS) or visual analogue scale (VAS), should all be routinely monitored. Pain referral from the neck that centralizes to the cervical spine in response to specific loads (e.g., chin retraction) or treatment may also be monitored.

- **Effects on work performance and daily activities.** These disabilities should be recorded and monitored for improvement. The patient specific functional scale (see CSPE protocol) and/or a specific disability questionnaire such as the Headache Impact Test (HIT /HIT 6) [www.headachetest.com] or the HDI should be used.

  The HIT-6 is distilled from 4 validated headache questionnaires: the Headache Disability Index (HDI), the Headache Impact Questionnaire (HlmQ), the Migraine Disability Assessment Questionnaire (MIDAS) and the Migraine-Specific Quality of Life Questionnaire (MSQ).

- **Analgesics.** Baseline severity and patient response can also be measured in part by evaluating analgesic use.

FROM THE PHYSICAL EXAM

Indirect measures of patient improvement (surrogate markers) such as active range of motion, the CFR, cervical deep flexors, and palpatory findings such as tenderness and joint restriction may also be used in addition to more patient-centered outcomes. Reproduction of headache pain with exam procedures should be noted in every instance

- **Cervical ROM and algometry:** Vavrek et al (2010) reported the following positive correlations: early in the course of treatment reduction in global cervical range of motion was correlated with the number of headaches a patient suffered ($p < .001$) and ROM-elicited pain most closely reflected the severity of the patient's neck pain and disability ($p < .001$ to .035). By the end of the 12 week treatment regime, however, the previous measures no longer paralleled the patient's subjective complaints. Instead pain pressure thresholds measured by an algometer correlated with neck pain and both headache and neck pain related disability days ($p < .001$ to .048).

***Clinical tip*:** Painful global range of motion may be a useful outcome to monitor in the initial phases of care, but pressure sensitivity using an algometer, comparing improvement to baseline, may be a more responsive measurement later. (See CSPE protocol on the Algometer).

- **Cervical Flexion Rotation Test (CFR):** The instrument-based (as opposed to observation) minimal detectable change (MDC_{90}) has been reported to be 4.7° for right rotation and 7.0° for left rotation in subjects with CGH (Hall 2010). This indicates that a change in CFR range of motion of at least 7° is required to be 90% confident that a change has occurred rather than measurement error. It has previously been reported that a reduction in range of motion greater than 10° identifies a positive CFR. Hence, the minimum clinically important change that is important to a patient might be 10°. Furthermore, Hall et al (Hall 2004) reported a 15° change in CFR range of motion after 1 treatment aimed at improving impairment identified by the CFR. Note that if a baseline measurement is taken while a headache is in progress, the range of motion may be reduced an average of 6 degrees.

  Consequently, slight improvement on subsequent pain free visits may erroneously be interpreted as a positive treatment response.

- **Cervical motion palpation:** There is evidence that cervical motion palpation for end feel improvement appears to be a responsive[2] post-manipulation assessment tool for determining whether perceived motion restrictions found before treatment improved after manipulation. Results showed that the sensitivity was excellent (93%) and the specificity was adequate (67%) for achieving this particular outcome. This reported degree of responsiveness was detected in symptomatic participants but not in asymptomatic participants. (Lakhani 2009)

- **Deep cervical flexors:** Tests directed at the integrity of the deep flexors may be useful in directing exercise therapy and can provide end points for these interventions (e.g., when a patient can hold the Jull test position for 10 seconds with good form and without shaking, deep flexor endurance exercises may no longer be required).

- Change in pain reproduction with exam procedures should be recorded using the tenderness scale (refer to CSPE Protocol "Tenderness Grading, Soft Tissue").

[1] Tests used to assess a patient do not always normalize at the same rate and to the same degree that the patient's overall condition improves. A *responsive* test is a test that does detect change over time and parallels the patient's overall symptomatic improvement.

ASSESSMENT STRATEGY

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Step 6: Determine if there are significant psychosocial factors.

Headaches can be associated with a variety of mood disorders, including anxiety and depression. This relationship is especially true with migraine. Merikangas (1994) found that the 1-year prevalence for dysthymia (mild depression) was 6.6% (OR 1.8 compared to controls) and for major depression 14.7% (OR 2.2). Lifetime prevalence was 3-4 times higher than controls. Likewise migraine headaches carry an increased risk for panic disorders (OR 2.37; 95% CI 1.42-3.99) and generalized anxiety disorders (OR 3.13 95% CI 1.56-6.30) (McWilliams 2004). The IHS (2004) suggests that psychosocial stress, anxiety and depression are factors that can lead to tension-type headaches, but whether these are causes or simply associated factors remains speculative. The presence of these disorders can also have a negative effect on the prognosis for both migraine and tension –type headaches (Guidetti 1998). Prevalence rates specifically for cervicogenic are not known.

If a mood disorder is suspected, consider referral for psychological evaluation and possible co-management by a behavioral/cognitive therapist.

ASSESSMENT STRATEGY

Step 1: Rule out ominous headaches
Step 2: Identify the type of headache (cervicogenic, myofascial, migraine, etc.) and pain generating tissue
Step 3: Identify any significant triggering or contributing factors (e.g., forward head carriage, deep flexor weakness).
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Step 7: Establish a prognosis.

Step 7: Establish a prognosis.

Prognosis, of course, starts with the type of headache. In the case of cervicogenic headache, specific evidence is generally lacking, but is thought to be good overall and responsive to conservative therapy. For example, there is some evidence that manual therapy or manual therapy combined with exercise has attained positive clinical results within 3-6 weeks (Nilsson 1997, Jull 2002). IHS definitions (2004) suggest that if the patient's headache does not respond within 3 months of appropriate care, the diagnosis of cervicogenic headache should be questioned.

Factors that may affect prognosis include the chronicity of the patient's symptoms and the patient's desire to improve and participate in their care and rehabilitation. In the case of migraine and tension-type headaches, co-morbid factors such as anxiety and depression can have a negative effect (Guidetti 1998).

The following factors also may have a significant influence on a patient's recovery:

- general health
- work and social influences/stressors
- response to past treatment
- duration of the problem (e.g. acuity, chronicity)
- patient's attitudes and beliefs
- degree and severity of traumatic mechanism of injury.

Practitioners should be realistic in communicating their expectations about the results of treatment. The patient should understand that the treatment is expected to yield significant, measureable, lasting improvement within two weeks to a month of therapy introduction. Details of each patient's history and examination should be incorporated into a clear and positive message which includes specific outcomes. The patient's response to care and treatment goals should be reviewed at each visit.

***Clinical tip*:** The practitioner might tell the patient, Example: "If the treatment is successful, you can expect to return to your normal, full-time work schedule, decrease your medication use to less than once per week and you will be able to turn your head all the way to the left to look over your shoulder."

A plan for re-evaluation within an explicit time interval (or within a certain number of treatments) should be given as well as a plan for referral for a second opinion or further testing in case of a lack of improvement.

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Appendix I: Medication Overuse Headache (MOH)

According to the IHS, a medication overuse headache (MOH) is a chronic headache resulting from the regular use of headache medication. The headache resolves or reverts to its previous more episodic pattern within 2 months after medication withdrawal.

Without having to wait for improvement after medication withdrawal, a provisional diagnosis can be made based on the following combination: a headache occurring ≥ 15 days per month, a history ≥ 3 months of regular medication use, and a report of the headache seeming to have worsened over time corresponding to the medication overuse.

MOH is estimated to be the third most frequent type of headache seen in clinical practice [3]. One-third of people with chronic daily headache[4] can be classified with MOH.

ICHD Classification of medication overuse and medication overuse headache
1. Headache present > 15 days per month
2. Regular overuse for > 3 months of one or more acute/symptomatic treatment drugs defined as follows: a. Ergotamine, or triptans (any formulation), or opioid, or combination analgesic medication intake > 10 days per month on a regular basis for > 3 months b. Simple analgesics or any combination of ergotamine, triptans, analgesics, opioids >15 days per month on a regular basis for >3months, without overuse of a single class alone
3. Headache has developed or markedly worsened during medication overuse.
Must Have All Three

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Appendix II: Temporal Arteritis

Suspect temporal arteritis (AKA giant cell arteritis) in patients over 50-55 years old who complain of a new primary complaint of headache accompanied by jaw claudication, vision complaints, symptoms of polymyalgia rheumatica (PMR), or constitutional features such as fever or fatigue.

The condition is rarely seen in patients under the age 50, with the mean age being 72 years, and is more common in women (3:1). (Smentana 2002) In one study spanning 42-years, no person younger than 50 years old was diagnosed with temporal arteritis. The overall prevalence is estimated to be low in patients 50 and above, about .003% (Waits 2010), rising to .01% in patients 85 and above (Smetana 2002). It remains an important headache in the differential diagnosis in older patients because of its association with sudden irreversible blindness.

!!! Warning: Suspected temporal arteritis warrants an urgent referral for further assessment and biopsy.

History

Location: The headache may be diffuse or localized. It is bilateral about half the time and is usually temporal. It may also radiate to the neck, jaws, face, and tongue. The headache occasionally may be occipital.

Onset: A new headache, recent onset, or one that is different from previous headaches.

Chronology: The duration of the headache is commonly 2 to 3 months before seeking care.

Quality: Typically throbbing, but sometimes sharp, dull or burning.

Associated symptoms:

- **Scalp tenderness** (e.g., aggravated by combing the hair or putting on a hat).
- **Jaw claudication** near the TMJ after a brief period of chewing, especially firm foods such as steak or a bagel (reported in 30% to 40% of cases); a positive LR of 4.2 (95% CI 2.8-6.2). (Smentana 2002, Lipton 1993) The DDX includes TMD (pain more immediate with chewing) or poorly fitted dentures.
- **Vision complaints** commonly include diplopia (+LR 3.4, 95 CI 1.3-8.6), sudden transient monocular blindness (seen in 10% of individuals), amaurosis fugax (unilateral vision loss lasting seconds to minutes), or a visual field cut.
- **PMR symptoms** (in about ½ of the cases) which include abrupt onset of morning stiffness involving the neck and shoulder girdle or low back and pelvic girdle with myalgia and significant tenderness in the proximal arms or thighs.
- **Constitutional symptoms** such as unexplained weight loss or fever (reported in 48% of cases).

Physical Examination

Key procedures

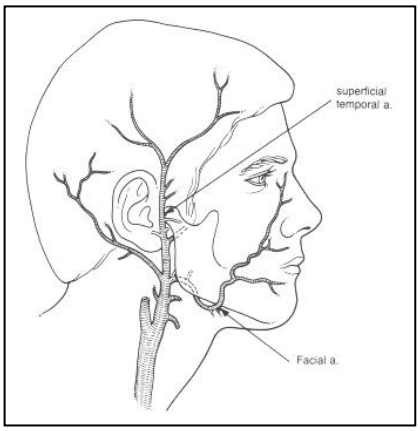
- Temperature
- Visual inspection of the scalp
- Palpation of the scalp
- Opthalmoscopic examination
- Visual field testing
- Upper and lower extremity joint assessment

The physical examination is frequently unremarkable, but certain key abnormalities increase suspicion of disease.

- **Temperature:** The patient may run a low grade fever.

- **Visual inspection of the scalp:** Look for nodules and redness along the temporal artery. The scalp and tongue should be inspected for ischemic or necrotic skin changes.

- **Palpation of the scalp:** Lightly palpate, just anterior to the tragus of the ear and follow the temporal pulse superior and anteriorly along the temples while comparing side to side. Positive findings include tenderness, reduced or absent pulsation, nodularity (reported in 35% of cases), or swelling. A string of nodules (i.e., beading) has a +LR of 4.6 (95% CI 1.1-18.4). In a systematic review, "prominent" nodules had a +LR of 4.3 (95% CI 2.1-8.9), hard nodules were present in 51% of cases, and tenderness in 55% (+LR 2.6, 95% CI 1.9-3.7). A decreased pulse was reported in 51% of cases.



- **Ophthalmoscopic examination**: This may reveal a pale or swollen disc (evidence of ischemic optic neuropathy) or retinal artery occlusion. A retinal occulsion is indicated by visible embolism/occlusion in a vessel or, in the case of a major occlusion, a cherry red macula with pale, milky edematous retina. (Smetana 2002, Lipton 1993)

- **Vision field testing:** Look for a loss of visual field.

- **Upper and lower extremity joint assessment:** If PMR accompanies the headache, range of motion in the shoulder or hip may be decreased because of pain or more distal synovitis, particularly of the wrist. The patient's muscle may be significantly tender.

Ancillary Studies

Erythrocyte sedimentation rate (ESR): An ESR of 50 mm/hr or greater is one of the American College of Rheumatology's criteria for the classification of giant cell arteritis (temporal arteritis) and it may exceed 100 (Hunder 1990). Because 10% to 20% of patients can have a normal ESR (-LR 0.2, 95% CI 0.08-0.51), a normal ESR cannot be used to completely rule out temporal arteritis in cases where there is a strong clinical picture. For example, a new headache in a 72 year old patient with a normal ESR is associated with a risk of disease of 12 %, but the probability of temporal arteritis increases dramatically to 78% when jaw claudication and scalp tenderness occur together.

C-reactive protein: CRP may also be elevated. Unfortunately, this acute-phase reactant is also not perfect at ruling out temporal arteritis (or PMR). (Waits 2010)

Thrombocytes: The platelet count may be increased. In one study the LR+ was 6.3 (confidence interval [CI], 2.4-17) for platelet count greater than 400 x 103/µL.

CBC: Normocytic anemia is present in 44% of patients.

Temporal artery biopsy is mandatory to establish the diagnosis (sensitivity of 85%, specificity 100%).

Test combinations

Certain combinations of findings may increase the probability of a positive biopsy. In a study by Younge, the combination of jaw claudication and decreased vision was associated with +LR of 44.

Smentana et al derived useful combinations from a large sample of 1113 patients undergoing temporal artery biopsy, all of whom were older than 50 years.

Combinations of Findings[a]:

	Posttest Probability
Headache +	
jaw claudication + scalp tenderness at age 60 y	65%
jaw claudication + scalp tenderness at age 80 y	74%
jaw claudication + scalp tenderness at age 60 y, ESR = 50 mm/h	84%
jaw claudication + scalp tenderness at age 80 y, ESR = 50 mm/h	88%
No headache +	
no jaw claudication + no scalp tenderness at age 60 y, ESR = 50 mm/h	7%
no jaw claudication + no scalp tenderness at age 80 y, ESR = 50 mm/h	10%

[a]These are examples of various combinations of findings for patients with 3 of 3 symptoms vs. 0 of 3 symptoms present at various ages.

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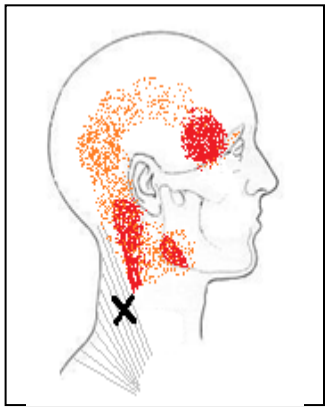
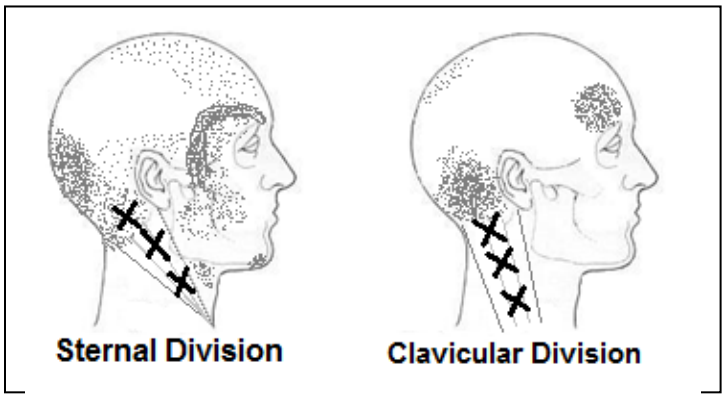
Appendix III: Myofascial Pain Referral of Individual Muscles

Note myofascial pain patterns charts are based on a relatively low number of cases and individual presentations may vary. These drawings based on Fernandez-de-las-Penas C, et al. Myofascial trigger points in the suboccipital muscles in episodic tension-type headache *Manual Therapy*. August 2006; Vol. 11, Iss. 3, pp. 225-230 and Dommerholt J, Huijbregts Myofascial Trigger Points: Pathophysiology and Evidenced-Informed Diagnosis and Treatment, Jones and Barlett, Boston, 2011.



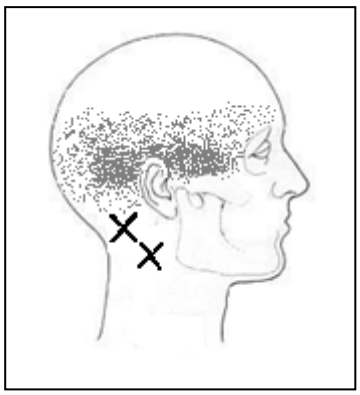
Upper trapezius muscle

Pain refers to behind the ear and to the temporal region.



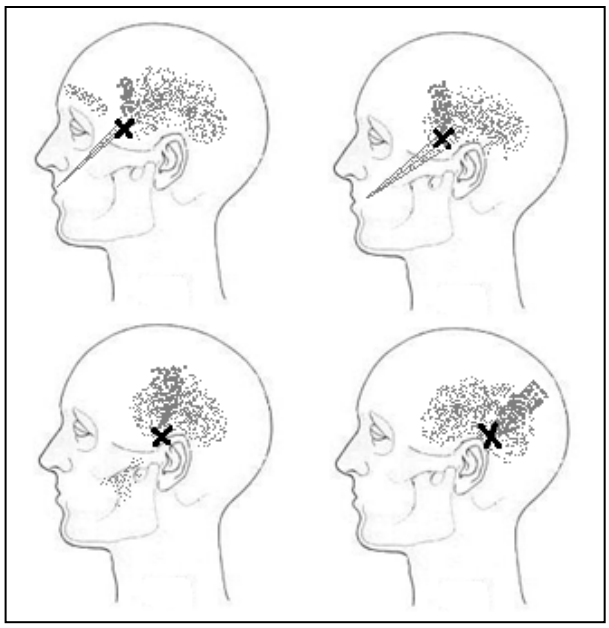
Sternocleidomastoid muscle

Pain refers to the occiput, the frontotemporal area, behind the ear, the forehead and the cheek.



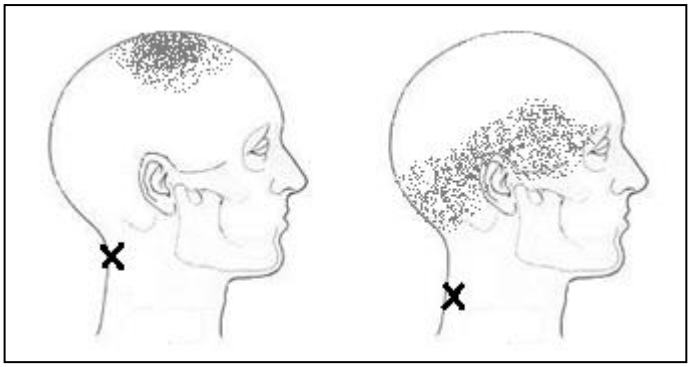
Suboccipital muscles

Suboccipital MFTPs refer over the occipital and temporal bones and is usually felt bilaterally.



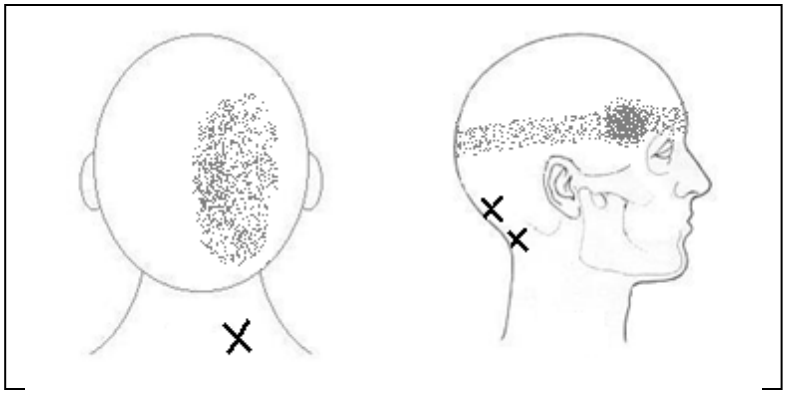
Temporalis muscle

Pain refers to the temporal and parietal areas and can be felt inside the head.



Splenius capitis and cervicis muscle

Splenius capitis refers to the vertex. Splenius cervicis is felt on the side of the head and behind the eye.

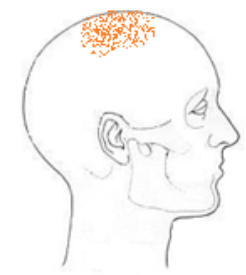
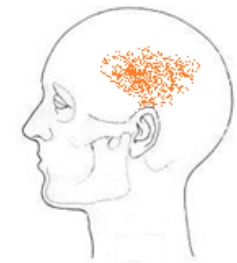
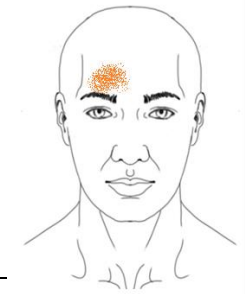
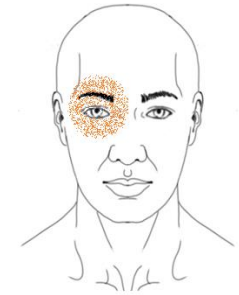


Semispinalis cervicis muscle

Pain refers to occiput, temporal region and behind eyes.

Appendix IV: Myofascial Trigger Point Pain Referral Areas

The following table offers suggestions of which muscles to palpate for myofascial trigger points based on headache location.

Pain Location	Examine the Following Muscles
Vertex of the head 	Sternocleidomastoid (sternal portion) Splenius capitis
Occipital headache 	Trapezius Semispinalis cervicis Splenius cervicis Suboccipital muscles Occipitalis Digastric Temporalis
Temporal headache 	Trapezius Sternocleidomastoid (sternal and clavicular portions) Temporalis Splenius cervicis Suboccipital muscles Semispinalis capitis
Frontal headache 	Sternocleidomastoid (sternal and clavicular portions) Semispinalis capitis Frontalis Zygomaticus major
Eye and eyebrow pain 	Sternocleidomastoid (sternal portion) Temporalis Splenius cervicis Masseter Suboccipital muscles Occipitalis Orbicularis oculi Trapezius

Based on Dommerhaolt J, Huijbregts. Myofascial Trigger Points. Jones and Bartlett, Boston, 2011

Appendix V: Palpation Evidence Table

Author, date	Patient group	Study type	Reliability/Validity	Reference standard	Study characteristics
Hall 2010	N=60 with CGH; 20 with no hx of HA	Cross sectional	Positive correlation between positive manual examination and presence of upper cervical joint dysfunction in CGH vs. controls. Inter-rater reliability for painful hypomotility in the upper cervical spine (C0-C4) was substantial (k=0.68); agreement per segment was 0.40 for C0/1, 0.70 for C1/2, and 0.71 for C2/3)	Clinical criteria: side dominant HA with no side shift, precipitated /aggravated by neck movement or posture	Convenience sample, CGH clinical criteria 2 examiners blinded to each other's findings as well as patient status
King 2007	N=173	Cross sectional	Pooled results for all levels: +LR 1.7 (95% CI 1.2-2.5); sensitivity 0.89 (95% CI 0.82-0.96); specificity 0.47 (95% CI 0.37-0.57) -LR 0.2 C2-C3 level -LR 0.3 and +LR 1.4 (95% CI 0.87-2.40); sensitivity 0.88 (95% CI 0.79-0.97); specificity 0.39 (95% CI 0.22-0.56)	Double nerve block as gold standard.	Consecutive cases; gold standard not applied to all cases which could lead to an underestimate of test specificity. Gold standard based on 100% obliteration of pain (may be a high threshold). No disc block to cover negative facet blocks.
Jull 2007	N=130: CGH (18), migraine (22), tension-type (33), controls (57)	Cross sectional	The combination of palpably painful upper cervical joints, decreased AROM (primarily in extension) and + CCFT had a 100% sensitivity, 94% specificity for CGH	Questionnaires based on IHS and Cervicogenic Headache International Study Group criteria	Blinding of outcome assessors, QUADAS score 10/14, but lack of facet or nerve block as gold standard. Unclear whether validity scores are dependent on EMG readings during CCFT or the standard use of the test
Zito 2006	N=27 CGH, 25 migraine with aura, 25 controls	Cross sectional	Manual examination for painful/hypomobile joints could discriminate CGH from the other categories with 80% sensitivity ($p<.05$)	Questionnaires based on IHS and Cervicogenic Headache International Study Group criteria	Single blinded assessor. QUADAS score 10/14, but lack of facet or nerve block as gold standard. Unclear whether validity scores are dependent on EMG readings during CCFT or the standard use of the test. Sensitivity but no specificity rating was reported.
Stochkendal 2006	48 studies	Systematic review	Strong evidence that pain provocation with osseus palpation had excellent intra-tester reliability (k = 0.91) and acceptable inter-examiner reliability (k=.53 ; CI 95% 0.32-0.74); global assessment of joint dysfunction had acceptable intra-tester reliability (k=0.44), but not inter-tester reliability; motion palpation for joint restriction had acceptable intra-tester reliability 0.44 (0.14-0.73) if the SI joint was not included.		Systematic review was well done and focused on high quality studies. However, the numbers reflect the whole spine, not just the cervical spine.

Seffinger 2004	53 studies	Systematic review	Overall palpation to provoke pain in the cervical spine had low to medium inter-examiner reliability (e.g., k=0.31-0.52). Motion palpation to detect altered motion scored generally lower; most studies showing poor inter-examiner reliability, with only a few studies showing medium ranges.		Systematic review was well done, thorough search, graded quality. Few of the 53 studies were on the cervical spine.
Dumas 2004	N=77: CGH post traumatic (20), CGH non-traumatic (24); migraine(16), control (17)	Cross sectional	Slight to severe loss of accessory mobility was more common in CHG (approximately 90% post-traumatic, 8-% nontraumatic) vs. 60% migraine, and 35% controls)	Based on patient interview using IHS and Cervicogenic Headache International Study Group criteria. 27/33 of the CGH patients had 50% response to block of greater occipital nerve	Double facet block, considered the gold standard was not used. The assessors were blinded to only 75% of the patients' diagnoses.
Jull 1997	N=40: non headache (20), headache (20)	Cross sectional	Manual palpation (any technique); 3 pairs of examiners in different clinics; inter-examiner reliability *K* values ranged from 0.78-1.00 for agreeing there was joint dysfunction anywhere in the upper cervical spine. Agreement percent was high for identifying the level with the greatest degree of joint dysfunction, but k values ranged from poor to excellent.		Assessors were blinded.
Schoensee 1995	N=15 : CGH (5), asympto-matic (10)	Inter-examiner reliability data nested within this treatment study.	Passive accessary joint play inter-tester reliability was fair, K 0.72 () in asymptomatics but higher (k=.79) in the CGH patients. ; Intra-tester reliability was excellent, k=.81. p values were <.0001		No report of blinding.
Sandmark 1995	N=75 subjects with neck pain (22), subjects with no history of neck for 1 year (53)	Cross-sectional	Static palpation of facet joints had a +LR 3.9 (sensitivity 82%, specificity 79%) and –LR of 0.23 for identifying patients with neck pain.	Patient questionnaire	Single blinding of assessor; may or may not be not be generalizable to headache patients
Jull 1988	N=20: neck pain and headache (14), neck and arm pain (3), neck pain only (3)	Cross over	Sensitivity and specificity was reported to be 100%.	Single block (medial nerve or facet joint)	Single blocks are no longer considered the gold standard. See King 2007 for an update of this study.

Double joint blocks are generally considered the gold standard for CGH studies and for cervical palpation validity studies. However, the appropriateness of blocks as a gold standard has its. See Humphrey's 2004.

References:

Dumas JP, Arsenault AB, Boudreau G, et al. Physical impairments in cervicogenic headache: Traumatic vs. non-traumatic onset Cephalalgia 2001;21:884–893.

Hall T, Biffra K, Hopper D, Robinson K. Reliability of manual examination and frequency of symptomatic cervical motion segment dysfunction in cervicogenic headache. *Man Ther*, 2010. 15(6): p. 542–6.

Humphreys BK, Delahaye M, Peterson CK. An investigation into the validity of cervical spine motion palpation using subjects with congenital block vertebrae as a 'gold standard'. BMC Musculoskelet Disord. 2004;5:19.

Jaeger B. Are cervicogenic headaches due to myofascial pain and cervical spine dysfunction? Cephalgia 1989;9:157-64.

Jensen O, Nielsen F, Vosmar L. An open study comparing manual therapy with the use of cold packs in the treatment of post concessional headache. Cephalagia 1990;10:241-9.

Jull G, Amiri M, Bullock-Saxon J, Darnell R, Lander C. Cervical musculoskeletal impairment in frequent intermittent headache. Part 1: Subjects with single headaches. Cephalalgia, 2007. 27(7): p. 793–802.

Jull G, Zito G, Trott P, Shirley D, Richardson C. Inter-examiner reliability to detect painful upper cervical joint dysfunction. Aust J. Physiother 1997; 43:125-9.

Jull G, Bogduk N, Marsland A. The accuracy of manual diagnosis for cervical zygapophyseal joint pain syndromes. Medical Journal of Australia 1988;148:233-6.

King W, Lau P, Lees R, Bogduk N. The validity of manual examination in assessing patients with neck pain. Spine J 2007;7:22-26.

Sandmark H, Nisell R. Validity of five common manual neck pain provoking tests. Scand J Rehabil Med. 1995;27:131-6.

Seffinger MA, Najm WI, Mishra SI, Adams A, Dickerson VM, Murphy LS, Reinsch S. Reliability of spinal palpation for diagnosis of back and neck pain: a systematic review of the literature. Spine (Phila Pa 1976) 2004;29(19):413-25.

Schoensee H, Jensen G, Gossman M, Kahtoli . The effect of mobilization on cervical headaches. Journal of Orthopaedic and Sports Physical Therapy 1995;21"181-196.

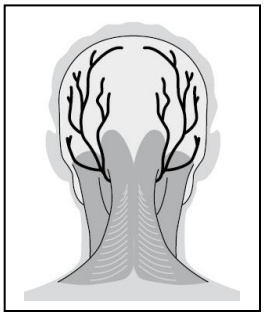
Stochkendahl M, Christensen H, Hartvigsen J, et al. Manual examination of the spine: A systematic review of reproducibility. J Manipulative & Physiol Ther 2006;29:475–485.

Watson D, Trott P. Cervical headache. An investigation of natural head posture and upper cervical flexor muscle performance. Cephalagia 1993;13:272-84.

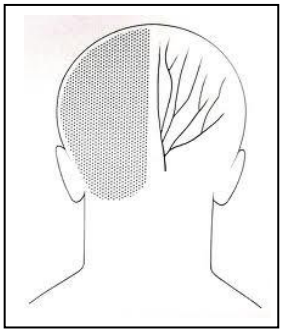
Whittingham W, Ellis W, Molyneux T. The effect of manipulation (toggle recoil techniques) for headache with upper cervical joint dysfunction: a pilot study. JMPT 1994;17:369-75.

Appendix VI: Occipital Neuralgia

Although an IHS headache classification, occipital neuralgia is controversial diagnosis. Traditionally, it is a disorder caused by irritation of the greater for lesser occipital nerve or the third occipital nerve. Bogduk (2009), however, considers this more likely a C2 neuralgia secondary to inflammatory or disorders or injuries of the lateral atlanto-axial joint. Others even consider it a sub-type of cervicogenic headache (Hoppenfeld 2010).



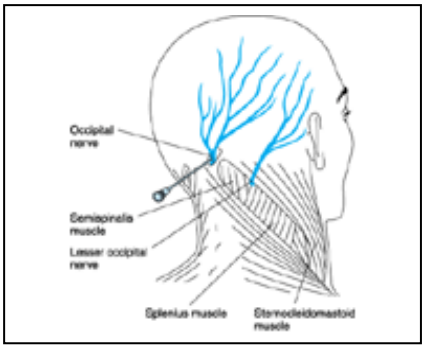
It is characterized by paroxysmal jabbing pain (e.g., burning, "shock-like") along the territories of the occipital nerves. Consequently, the pain, like cervicogenic headache pain, can begin in the neck.



A persistent ache may occur between the bouts of stabbing pain. There is palpable tenderness over the nerve. (IHS 2004)

This condition may be difficult to distinguish from myofascial trigger point pain referral or from cervicogenic headache, especially since tenderness over this nerve has been reported as an occasionally accompanying feature of cervicogenic headache (Antonaci 2011).

If the usual care for a cervicogenic headache with tenderness over the occipital nerve fails, referral for a local anesthetic nerve block may clarify the diagnosis and also offer at least temporary relief. The anesthetic block is usually combined with a corticosteroid. It has been reported that 94% of patients obtain relief, but the duration ranges from only 10-77 days. (Hoppenfeld 2010)



Appendix VII: Jull test Evidence Table
(muscle endurance of the deep neck flexors)

Author, date	Patient group	Study type, level of evidence	Reliability/Validity	Reference standard	STADT score	Study weakness
De Koning 2008	Mix of healthy patients and patients with neck pain or HA	Systematic review, 9 studies	Most studies found Intra-rater reliability ICCs above 0.85 (3 studies on healthy individuals found ICCs 0.76-0.79). Inter-rater ICCs raged form 0.57 to 1.0). Construct validity was assessed and found to have a positive correlation with the NDI.	NDI		Reliability studies, on average were rated as having "positive" quality. However, the scoring system used was not transparent.

Appendix VIII: Craniocervical Flexion Test (CCFT)

This is a test of neuromotor coordination and the holding capacity of the deep neck flexors.

The set up

The patient lies supine with knees and hips bent, feet flat on the table. The head is in a neutral position (the neck is not flexed or extended, i.e., the longitudinal plane of the neck is parallel to the table). An inflatable air-filled pressure sensor (Stabilizer, Chattanooga South Pacific) is placed suboccipitally behind the neck. The edge of the bladder should be against the occiput and inflated to 20 mmHg. The patient looks at the pressure gauge. The patient should gently press his/her tongue against the roof of the mouth just behind the teeth with teeth apart and lips closed (this is reported to help prevent recruitment of jaw muscles or muscles attached to the hyoid).

The test is conducted in two stages.

Stage 1: Motor control

Watching the pressure gauge, the patient very slowly flexes the upper cervical spine with a gentle head nodding action and holds the position steady at 22 mmHg for 2-3 seconds. The practitioner observes or palpates the superficial neck flexors (SCM, anterior scalene and hyoid muscles). There should be minimal activity.

The patient is told to relax and return to the starting point and then to nod the head and attempt to hold the needle steady at 24 mmHg for 2-3 seconds. If the patient can do this without recruiting, this process continues at 2 mm intervals up to 30 mmHg.

Failing

- ⇒ the patient substitutes neck extension or chin retraction for the head rotation (i.e., "nodding") action
- ⇒ the patient lifts the head and cannot attain or maintain the target pressure
- ⇒ the movement cannot be performed slowly (the patient picks up speed)
- ⇒ there is palpable superficial muscle activity in the first 3 stages of the test (22-26 mmHg)
- ⇒ at each step when the patient returns to the starting position, s/he cannot maintain the pressure at 20 mmHg (unable to fully relax the muscles)

The baseline assessment is the pressure grade that the patient can successfully perform. Most neck pain patients' initial performance is an increase of only 2-4 mmHg and they demonstrate an inability to hold the position steady. An ideal result is that a patient can increase the pressure 10 mmHg by 2 mm intervals up to 30.

Stage 2: Endurance

The patient is now asked to perform an endurance test by holding for 3 repetitions of 10 seconds at each of the pressure steps *that s/he was able to do correctly*, starting at 22 mmHg.

Failing:

- ⇒ the patient cannot hold for 10 seconds
- ⇒ the patient loses form
- ⇒ there is significant recruiting of superficial flexors
- ⇒ the pressure level is maintained but with a jerky movement suggesting the substitution of more phasic muscles.

The baseline assessment is the highest level of pressure that the patient can correctly perform for 3 repetitions for 10 seconds.

Reference:

Jull GA, O'Leary SP, Falla DL. Clinical assessment of the deep cervical flexors muscles: the craniocervical flexion test JMPT 2008;31:525-633.

Appendix IX: Craniocervical Flexion Test (CCFT) Evidence Table
(using inflatable pressure biofeedback unit)

Author, date	Patient group	Study type	Reliability/Validity	Reference standard	Study characteristics
Arumugam 2011	N=30 asymptomatics	Cross-section, repeated measures	Inter-rater reliability ICC 0.91 (95% CI 0.83-0.96)	N/A	Convenience sample, compared novice to experienced rater (blinded, simultaneous measures)
James 2010	N=19 asymptomatic subjects	Test-retest	Intra-examiner reliability ICC 0.98	N/A	Convenience sample, examiner not blinded to the "immediate results" but re-tested at a 7 day interval. It is not clear whether there was an attempt at blinding on the retest.
De Koning 2008	Mix of healthy patients and patients with neck pain or HA	Systematic review, 4 studies	Most studies found Intra-rater reliability ICCs ranged from 0.65-0.93. (Chiu 2005, Hudswell 2005, Jull 2000, Jull 1999). Inter-rater ICCs were reported at 0.54 and 0.57. (Huswell 2005)		The quality of the studies was rated as poor due to reporting flaws (i.e.blinding) and small cohort size. The weakness of the review itself was lack of transparency regarding key details of the quality assessment the authors performed.
Jull 2007	N=130: CGH (18), migraine (22), Tension-type (33), controls (57)	Cross sectional	The combination of palpably painful upper cervical joints, decreased AROM (primarily in extension) and + CCFT had a 100% sensitivity, 94% specificity for CGH	Questionnaires based on IHS and Cervicogenic Headache International Study Group criteria	Blinding of outcome assessors, QUADAS score 10/14, but lack of facet or nerve block as gold standard. Unclear whether validity scores are dependent on EMG readings during CCFT or the standard use of the test
Zito 2006	N=77: CGH (27), migraine with aura (25), controls (25)	Cross sectional	CGH patients performed more poorly compared to migraine and controls, but the trends did not reach statistical significance.	Questionnaires based on IHS and Cervicogenic Headache International Study Group criteria	Single blinded assessor. QUADAS score 10/14, but lack of facet or nerve block as gold standard. Unclear whether validity scores are dependent on EMG readings during CCFT or the standard use of the test.
Jull 2004	N=75: control (25), whiplash (25), chronic neck pain (25)	Cross sectional	Impairment of flexor muscle activity more strongly associated with whiplash and chronic pain patients than in controls as measured by the CCFT ($p<0.05$).	EMG measure of superficial flexors	Convenience sample, no blinding.
Falla 2004	N=20: chronic neck pain (including headache) (10) and controls (10)	Cross sectional	Physiological study. Neck pain patients more likely to have impaired function of deep flexors detected by CCFT ($p=0.002$).	Esophageal placement of EMG	Whether esophageal EMG placement measures deep flexors has not been validated.

References:

Arumugam A, Mani R, Raja K. Interrater reliability of the craniocervical flexion test in asymptomatic individuals—a cross sectional study. J Manipulative Physiol Ther 2011;34:247-253.

Chiu TT, Law EY, Chiu TH. Performance of the craniocervical flexion test in subjects with and without chronic neck pain. J Orthop Sports Phys Ther. 2005;35:567-71.

de Koning C, Heuval S, STaal J, Smits-Engelsman B, Hendriks E. Clinimetric evaluation of methods to measure muscle functioning in patients with non-specific neck pain: a systematic review BMC Musculoskeletal Dis 2008;9:142-153.

Falla DL, Jull GA, Hodges PW. Patients with neck pain demonstrate reduced electromyographic activity of the deep cervical flexor muscles during performance of the craniocervical flexion test. Spine 2004; 29(19):2108-2114.

James G, Doe T. The craniocervical flexion test: intra-tester reliability in asymptomatic subjects. Physiother Res Int. 2010 Sep;15(3):144-9.

Hudswell SMvM, Lucas N. The cranio-cervical flexion test using pressure biofeedback: A useful measure of cervical dysfunction in a clinical setting? Int J Osteop Med 2005;(8):98-105.

Jull G, Amiri M, Bullock-Saxon J, Darnell R, Lander C. Cervical musculoskeletal impairment in frequent intermittent headache. Part 1: Subjects with single headaches. *Cephalalgia*, 2007. 27(7): p. 793–802.

Jull G, Kristjansson E, Dall'Alba P, Impairment in the cervical flexors: a comparison of whiplash and insidious onset neck pain patients. Man Ther 2004: 9(2):89-94

Jull G, Barrett C, Magee R, Ho P. Further clarification of muscle dysfunction in cervical headache. Cephalalgia 1999;19:179–85.

Jull GA. Deep cervical flexor muscle dysfunction in whiplash. J Musculoskel Pain 2000; 8:143-153.

Zito G, Jull G, Story I. Clinical tests of musculoskeletal dysfunction in the diagnosis of cervicogenic headache. Man Ther 2006;11(2):118-29.

Appendix X: Flexion Rotation Test (FRT) Evidence Table

Author, date	Patient group	Study type	Reliability/Validity	Reference standard	Study characteristics
Hall 2010b JMPT	N=92 (72 with CGH and 20 with no hx of HA) 15 tested while having HA	Observational	Positive correlation between severity of CGH HA and reduced FRT; Testing during HA affected range by 6 degrees, but not enough to change positive vs. negative test. Compared to normal controls, +LR 5.18, -LR 0.26.	"probable" CGH based on clinical criteria (i.e., Antonaci criteria of fulfilling 5/7 characteristics derived from the original Sjaastad criteria and consistent with the IHS criteria)	Single blinded examiner, convenience sample, no use of diagnostic gold standard, reference standard based on history only
Hall 2010c Journal of Manual and Manipulative Therapy	Patients with CGH compared to those with lower cervical joint pain.	Singe blind comparative group design	Ability to identify clinically diagnosed CGH from patients with lower cervical facet pain had a +LR of 9.38 and -LR of 0.27.	"probable" CGH based on clinical criteria (i.e., Antonaci criteria of fulfilling 5/7 characteristics derived from the original Sjaastad criteria and consistent with the IHS criteria)	Convenience sample; random ordering, single anesthetic block (double block is considered the gold standard) not applied to both groups
Hall 2010d JOSPT	N=15 with CGH; N=10 without HA; tested at 2, 4, & 14 days	Observational	Excellent test-retest reliability: ICC right 0.95 (95%CI: 0.90-0.98); left 0.97 (95% CI: 0.94-0.99) Excellent intra-rater reliability for +/- result; *k* = 0.92 (95%CI: 0.77-1.00), p<.001 Minimal detectable change: 7 degrees with 90% confidence	IHS & Sjaastad clinical criteria	Single blinded assessor on non HA days; convenience sample. No use of diagnostic gold standard, reference standard based on history only
Hall 2010e J Headache Pain	N=60: migraine (20), CGH (20), mixed HA (20)	Single blind comparative group design	+LR 2.33 (95% CI 1.34-4.06) and -LR 0.43 (95% CI 0.21-0.85) in predicting the presence of CGH vs. a cohort of patients with either migraine or mixed headaches.	Migraine dx based on IHS criteria; CGH based on clinical criteria without a block (based on Antonaci criteria, 5/7 fulfilled = probable CGH and excluded if fulfilled any other HIS criteria).	Convenience sample, FRT tester was blinded ; no use of diagnostic gold standard, reference standard based on history only, QUADAS score 9 /14
Hall 2008 JMPT	N=40: CGH with C1/2 joint dysfunction (20), CGH without evidence of C1/2 joint dysfunction, aymptomatics (10)	Single blind comparative measures	Inter-rater reliability ICC 0.93 (95% CI 0.87-0.96); p=.001 *K* for judging the test as positive or negative = 0.85 (excellent) The likelihood for FRT to correctly identify headache patients with palpatory upper cervical joint dysfunction was +LR 9 (95% CI 3.3-24.2), -LR 0.11 (0-0.3)	CGH dx was based on history only, (i.e., unilateral HA without side shift, neck stiffness/pain)	Single blind, convenience sample, issues with reference standard selected
Ogince 2007 Manual Therapy	N=58: CGH (23), control (23), migraine with aura (12)	Single blind comparative group design	91% sensitivity, 90% specificity for identifying with CGH patients with motion palpation evidence of upper cervical joint dysfunction vs. controls and migraine patients.	CGH classified by history alone (unilateral HA without side shift, associated with neck pain/stiffness related to HA , HA precipitated or aggravated by neck movements or sustained posture) who also had palpatory evidence of upper cervical joint dysfunction	Convenience sample, blinded assessors. Migrainers with cervical involvement were excluded so findings may not be as generalizable.
Hall 2004 Man Ther	N=56: CGH (28), gender matched controls (28)	Single blind comparative measures	1000%agreement between examiners in subjects with palpatory upper C1/2 joint dysfunction.	CGH dx was based on history only, (i.e., unilateral HA without side shift, neck stiffness/pain, neck pain correlated with HA); correlation was made with manual palpation of C1-C2 joint dysfunction.	Single blind, convenience sample, issues with reference standard selected

Appendix XI: "Fixed" Head position

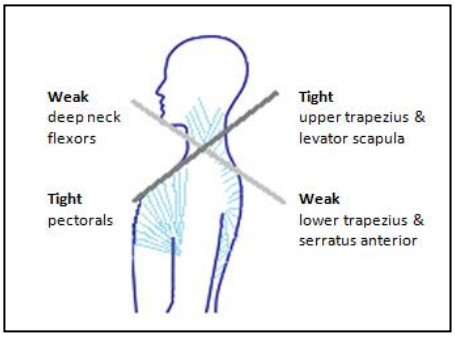
Site	Cause	Management
The head is forward in an otherwise upright body.	Possible imbalance between DEEP cervical flexors and extensors.	Treatment requires activating the deep flexors, relaxing overactive SCMs, and relaxing the extensor muscles; chin retraction exercises for postural training may be useful.
The head is drawn forward because of anterior rounded shoulders.	Tight pectoralis muscles and inhibited middle/ lower trapezius; there may be the full expression of the upper cross syndrome.	Special attention is paid to stretching the pectoralis muscles and training the lower trapezius; postural exercises such as Bruegger's rest position are indicated
The head is drawn forward due to hyperkyphosis	The thoracic hyperkyphosis may be secondary to an upper cross syndrome, thoracic joint dysfunction, or thoracic disease (e.g., AS).	In addition to the treatment cited above, include manipulating the thoracic spine and supine extension exercises over a ball or cylinder (i.e., foam roll)
The head is drawn forward because of flexion from the waist	Tight psoas as part of a lower cross syndrome or tight rectus abdominus or lumbosacral instability may be the cause.	Rehabilitation exercises will have to also target the low back and pelvis.
The whole body is leaning forward from the ankle.	Poor proprioception and balance issues may be involved. Tight calf muscles may result in over compensation in forward lean.	Consider stretching tight calf muscles and giving balance work teaching patients to distribute their weight more evenly over the foot.

Appendix XII: Postural Assessment

Assessment tool	Finding	Significance
Observation	Forward head carriage	May be associated with part or all of the upper cross syndrome; postural re-training may be necessary.
Observation	Rounded/anterior shoulders	May be associated with tight pectoralis and inhibited middle/lower trapezius.
Observation	Gothic shoulders (angular rather than curved contour of side of the neck)	Overactive upper trapezius and/or levator scapula.
History	Work or recreational postures that induce extended periods of holding the neck in a rigid flexed position (as can occur with some reading postures or computer workstations)	Behavioral or ergonomic changes should be made.
Palpation and observation for diaphragmatic breathing	Vertical chest breathing with little movement of the belly or paradoxical breathing (i.e., the abdomen retracts with inspiration and expands with expiration)	Abdominal breathing is taught as an exercise and as an adjunct to all other exercises the patient is given.

Appendix XIII: Assessment for Upper Cross Syndrome

This pattern of muscle imbalance primarily consists of inhibited deep cervical flexors, short tight cervical extensors, short tight pectoralis muscles, and inhibited middle and lower trapezius. Sometimes associated with pattern are short tight SCMs and inhibited serratus anterior.



Assessment tool	Finding	Significance
Observation	Forward head carriage	May be associated with upper cross syndrome; postural re-training may be necessary
Observation	Rounded/anterior shoulders	May be associated with tight pectoralis and inhibited middle/lower trapezius
Observation	Gothic shoulders (angular rather than curved contour of side of the neck)	Overactive upper trapezius and/or levator scapulae
Cervical instability test (Jull test)	Inability to hold 10 seconds without chin poking, shaking, or neck movement	Inhibited or weak deep neck flexors
Craniocervical flexion test	Inability to hold head nod steady at 2 mmhg intervals up to at least 26 mmHg for 10 repetitions of 10 second holds	Inhibited or weak deep flexors, poor motor control, perhaps poor kinesthetic awareness.
Supine active neck flexion (Janda test)	Early chin poking	Inhibited deep flexors and/or overactive SCM
Active shoulder abduction	1) shoulder Hiking in the first 60 degrees of abduction; 2) asym-metrical lateral movement of scapula compared to the contralateral side; 3) winging of inferior tip of scapula	1) inhibited lower trapezius/overactive upper trapezius/levator; 2) inhibited middle trapezius 3) inhibited serratus anterior
Push up	Winging of inferior tip of scapula or adduction of scapula toward midline	Inhibited serratus anterior
Length testing of pectoralis, upper trapezius, and levator scapula.	Hard end feel, poor joint excursion	Short, tight or overactive muscles
Lower and middle trapezius (static isometric hold of the scapula retracted and drawn down)	Inability to hold position for 10 seconds without shaking, recruitment or significant asymmetry in endurance compared to the contralateral side	Inhibited or weak muscles