
TUTORIAL

Diagnosis and Management of Cervicogenic Headache

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■ **Abstract:** Upper cervical pain and/or headaches originating from the C0 to C3 segments are pain-states that are commonly encountered in the clinic. The upper cervical spine anatomically and biomechanically differs from the lower cervical spine. Patients with upper cervical disorders fall into two clinical groups: (1) local cervical syndrome; and (2) cervicocephalic syndrome. Symptoms associated with various forms of both disorders often overlap, making diagnosis a great challenge. The recognition and categorization of specific provocation and limitation patterns lend to effective and accurate diagnosis of local cervical and cervicocephalic conditions. ■

Key Words: cephalgia, cervical, cranial pains, headache disorders

INTRODUCTION

Upper cervical pain and/or cervicogenic headaches are disorders that are frequently encountered in the general public. Approximately 70% to 90% of adults report a

minimum of one headache annually, with only 5% seeking medical attention,¹ and a portion of these headaches originate from the cervical spine (approximately 17.8%).² Sjaastad et al. suggested that the cervicogenic headache group comprises one of the three large groups of headache sufferers, accompanied by tension type and migraine. This is of no surprise, considering the numerous pain generators located in the C0 to C3 region of the spine.³

In 1990 the International Headache Society established criteria for diagnosing cervicogenic headache. These included: (1) pain localized to neck and occipital region, which may project to forehead, orbital region, temples, vertex, or ears; and (2) pain that is precipitated or aggravated by specific neck movements or sustained neck posture. In addition, the criteria suggested that at least one of the following occurs: (1) resistance to or limitation of passive neck movements; (2) changes in neck muscle contour, texture, tone, or response to active and passive stretching and contraction; and (3) abnormal tenderness of neck muscles. Radiological examination of cervicogenic headache reveals at least one of the following: (1) movement abnormalities in flexion/extension; (2) abnormal posture; and (3) fractures, congenital abnormalities, bone tumors, rheumatoid arthritis, or other distinct pathology (not spondylosis or osteochondrosis).⁴

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The complex architecture of the upper cervical articular system affords considerable mobility.⁵ This, in concert with the considerable weight of the cranium and its contents, renders the muscles, ligaments, tendons, and joints to injury predilection. Mechanical cervical spine pathology produces painful and limited cervical movement, as a consequence of dysfunction in the muscles and C0 to C2 articulations. Additionally, affliction in the C2C3 intervertebral disc, zygapophyseal joints (ZAJ), and/or uncovertebral joints (UVJ)⁶ can contribute to the clinical presentation, complicated by the hypomobile adaptation,⁷⁻⁹ and/or hypermobility/instability⁷ of the upper cervical spine in the context of aging. Finally, the clinician is challenged with understanding the role of systemic disease¹⁰⁻¹³ or trauma¹⁴⁻²⁰ in the patient's upper cervical clinical picture.

PATHOANATOMY

The upper cervical spinal motion segments are distinctively different on several accounts from segments in the lower cervical spine and each architectural component of the upper cervical spine contributes to the structural distinctions observed in this spine region. The atlas (C1; Figure 1) is typified by an absent vertebral body, where

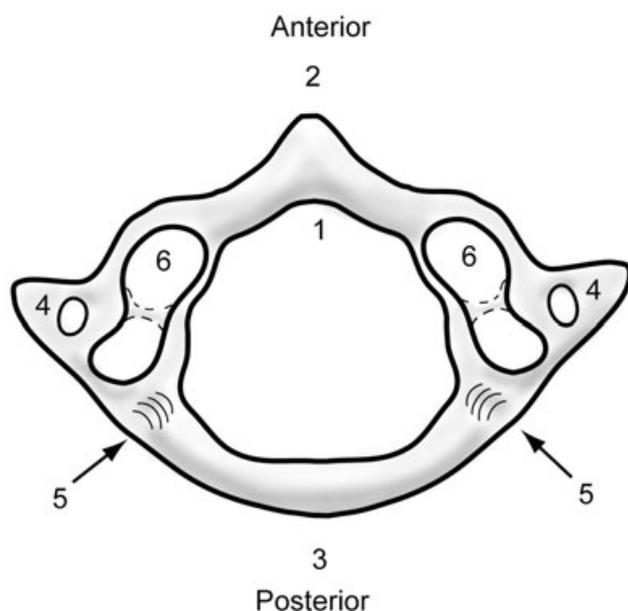


Figure 1. The atlas (C1). 1 Facet for the dens; 2 anterior tubercle; 3 posterior tubercle; 4 transverse foramen; 5 groove for the vertebral artery; 6 cartilage over superior articular facet. (Reprinted from Racz GB, Anderson SR, Sizer PS, Phelps V. Atlantooccipital and atlantoaxial injections in the treatment of headache and neck pain. In: Waldman S, ed. *Interventional Pain Management*. 2nd edn. pp. 295–305, 2000, with permission from Elsevier.)

its bony ring serves as an intercalated “relay center” between the occiput and C2. The C1 lateral masses, in accompaniment with the anterior and posterior arches, border the triangular spinal foramen that accommodates the brainstem. The posterior arch, found caudal to the occiput, lacks a spinous process and is difficult to palpate, because of its depth beneath the dorsal skin of the neck. The long C1 transverse processes have transverse foramina that accommodate the vertebral arteries, which continue on to course through grooves on the posterior lateral masses. These grooves, or occasional tunnels, accommodate the vertebral arteries as they loop for a second time in the upper cervical region. Bony adaptations can be witnessed in this region that can produce vertebrobasilar insufficiency (VBI) as a consequence of vertebral artery function compromise.²¹ This condition is especially observed in females, who exhibit architectural differences in the previously described grooves.²²

The cranial articular surfaces of C1 are large and concave in both the frontal and parasagittal planes. This shape complements the kidney-bean-shaped articular surfaces of the occiput that are oriented ventromedial to dorsolateral, 50° to 60° from the frontal plane. These ellipsoid C0C1 joints permit the considerable flexion/extension, as well as small amount of sidebending that are required of the head in function.

The most prevalent feature of the axis (or C2) is the dens, which is the asparagus-shaped projection that emerges from the front of the C2 bony segment. This dens, or odontoid process, serves as a pivot around which C1 and the head turn (Figure 2)²³ and is highly prone to deformity, an architectural feature with which clinicians must be aware before incorporating manual techniques to the upper cervical spine. The dens may vary in its orientation in the cervical space, as well as the depth and orientation of the tip and dorsal notch, which serve as a barrier to vertical disarticulation of the C1 vertebra. Axial separation is prevented between C1 and C2, because of the strong constraint by the transverse ligament positioned behind a normally configured dens. Any deformity to the dens (such as that found in Downs syndrome)²⁴ could potentially reduce upper cervical stability, produce excessive separation, and merit avoiding any therapeutic cervical traction maneuvers. In addition, a gap, which can be appreciated on sagittal view X-ray between the dens and the anterior arch of C1, is full of cartilage and should not exceed 3 mm, even during cervical flexion or extension. Otherwise, one may suspect atlantodental dislocation or lesions of the ligaments, articular capsules, or facet joints.

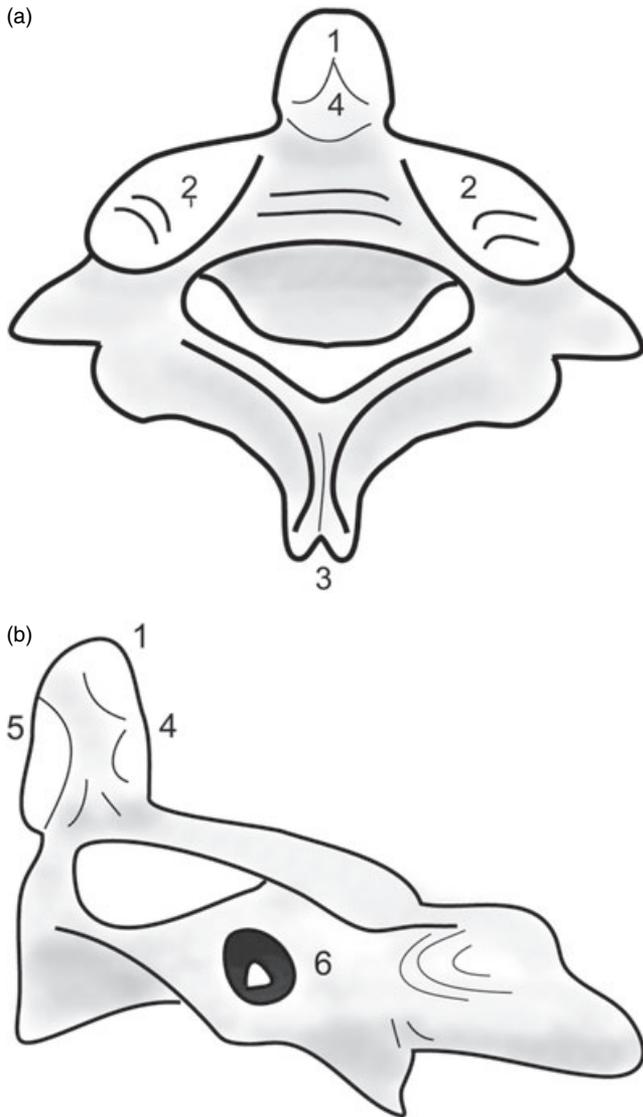


Figure 2. The axis (C2): (a) cranial view; (b) sagittal view. 1 Dens, or odontoid process; 2 superior articular facet; 3 spinous process; 4 groove for transverse ligament of atlas (TLA); 5 facet for anterior arch of the atlas; 6 transverse foramen. (Reprinted from Racz GB, Anderson SR, Sizer PS, Phelps V. Atlantooccipital and atlantoaxial injections in the treatment of headache and neck pain. In: Waldman S, ed. *Interventional Pain Management*. 2nd edn. pp. 295–305, 2000, with permission from Elsevier.)

The upper cervical spinal motion segments (C0C1 and C1C2; atlanto-occipital and atlantoaxial segments, respectively) are distinctively different on several accounts from segments in the lower cervical spine. Both motion segments lack the intervertebral discs and unciniate processes found in lower cervical segments. Rather, they rely on distinctive central and lateral articular systems to create stability, while affording considerable mobility to the head and neck. Additionally, the

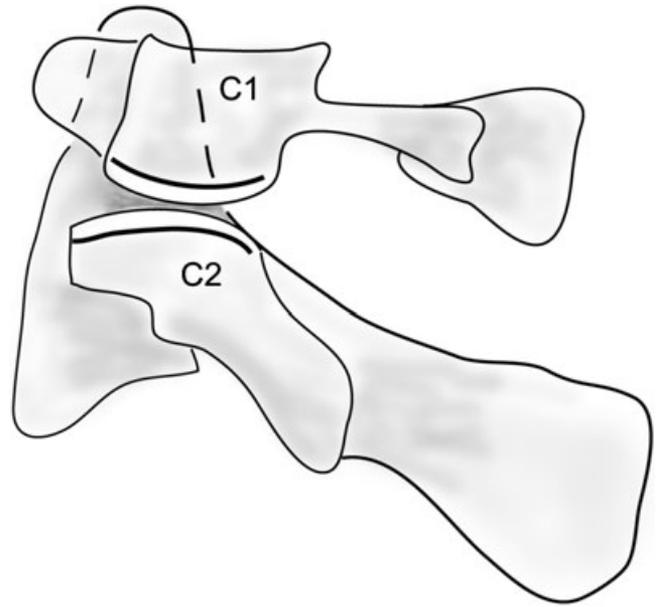


Figure 3. Lateral atlantoaxial joint (C1C2). Biconvexity in the sagittal plane. (Reprinted from Racz GB, Anderson SR, Sizer PS, Phelps V. Atlantooccipital and atlantoaxial injections in the treatment of headache and neck pain. In: Waldman S, ed. *Interventional Pain Management*. 2nd edn. pp. 295–305, 2000, with permission from Elsevier.)

orientation of these articular processes differs from lower cervical zygapophyseal surfaces in each anatomical plane. The convex occipital condyles sit in the superior articular facets of C1, which form deep concave sockets on each side of the vertebral structure. On the other hand, four synovial articular systems can be observed between C1 and C2: two central joints and two lateral joints. The anterior atlantoaxial joint, or atlantodental joint, is observed between the dens and the anterior arch of the atlas. Posterior to the dens lies a synovial compartment between the dens and transverse ligament of atlas (TLA) (lower component of the cruciform ligament), also called the atlantodental bursa.²³

From a sagittal view, the left and right biconvex lateral atlantoaxial joints (Figure 3) are characterized by thick articular cartilage (1.4 to 3.2 mm) and surrounded by intra-articular menisci that emerge from flaccid, roomy joint capsules. These menisci fill in the incongruent joint spaces and are subject to degradation, producing sharp, local catching pain that results from interposition with rotation between C1 and C2.²³ These four joint systems allow for flexion, extension, and rotation, with very little sidebending afforded.

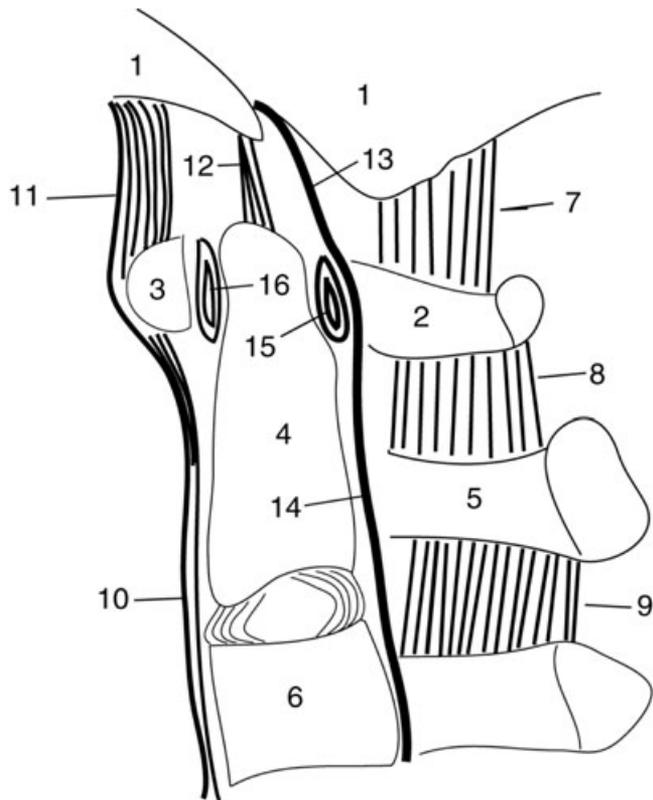


Figure 4. The ligaments of the upper cervical spine. 1 Occiput; 2 posterior arch of the atlas; 3 anterior arch of the atlas; 4 dens of the axis; 5 posterior arch of C2; 6 vertebral body of C1; 7 posterior atlanto-occipital membrane; 8 ligamentum flavum, C1C2; 9 ligamentum flavum, C2C3; 10 anterior longitudinal ligament; 11 anterior atlanto-occipital membrane; 12 apical ligament of the atlas; 13 tectorial membrane; 14 posterior longitudinal ligament; 15 transverse ligament of atlas (TLA); 16 synovial compartment between the dens and the anterior arch of the atlas. (Reprinted from Racz GB, Anderson SR, Sizer PS, Phelps V. Atlantooccipital and atlantoaxial injections in the treatment of headache and neck pain. In: Waldman S, ed. *Interventional Pain Management*. 2nd edn. pp. 295–305, 2000, with permission from Elsevier.)

Ligament integrity is essential between the bony structures of the upper cervical spine, because of the close proximity of the brainstem and spinal cord (Figure 4). The TLA courses between lateral masses of C2 posterior to the dens, prohibiting any separation between C1 and C2. Although the ligament is primarily type I collagen, it exhibits the aggrecan, link proteins, and type II collagen that are characteristic of fibrocartilage. These substances, found primarily in the proximity of the ligament's contact with the dens, demonstrate the consistent load on the ligament throughout various movements of the head and C1.²⁵ This ligament prevents posterior tipping of the dens into the brainstem and spinal cord during movements of the head and neck,

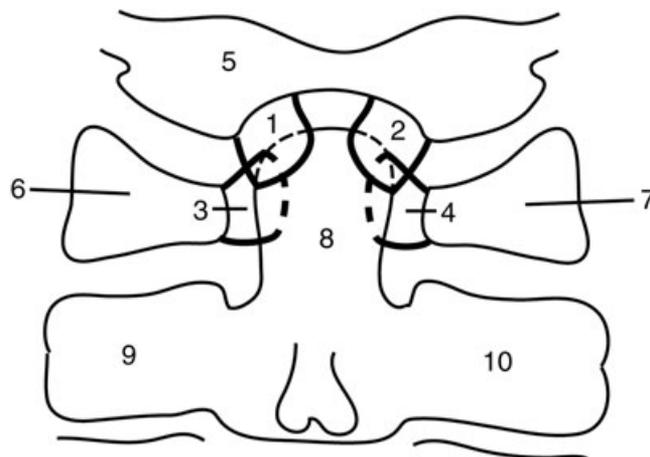


Figure 5. The alar ligaments (dorsal view). 1 Left occipital alar ligament; 2 right occipital alar ligament; 3 left atlantal alar ligament; 4 right atlantal alar ligament; 5 occiput; 6 left C1; 7 right C1; 8 dens; 9 left C2; 10 right C2. (Reprinted from Racz GB, Anderson SR, Sizer PS, Phelps V. Atlantooccipital and atlantoaxial injections in the treatment of headache and neck pain. In: Waldman S, ed. *Interventional Pain Management*. 2nd edn. pp. 295–305, 2000, with permission from Elsevier.)

especially with cervical flexion. Without this support during such a maneuver, an individual could experience brainstem/cord compression and a clinical “drop attack,” seen in context with any ligament compromise sustained during a whiplash injury.

The TLA is assisted by the alar ligaments in maintaining a centralized dens position (Figure 5). Eleven- to thirteen-millimeter-long right and left occipital alar ligament branches course in all individuals from the posterior tip of the dens to the occiput. These Type I collagen branches are very stiff, producing only a maximum of 5 to 6% length deformation. Right and left atlantal branches course 3 to 4 mm, from the anterior dens to the posterior internal surface of the anterior arch of C1 in select individuals. The alar ligament system is tension loaded during cervical extension, sidebending, and ipsilateral rotation, thus lending the upper cervical spine to very powerful and easily observable coupling behaviors. Any kinetic rotational motion is accompanied by significant contralateral synkinetic sidebending activity and visa versa, especially when the patient is sitting (because of the gravity loading the articular facets).^{26,27} A compromise to these coupling behaviors emerges in response to an alar ligament disruption, whereby cervical movements increase and coupling is distorted in the upper cervical spine.²⁸ With the same compromise the vertebral artery is placed at greater risk for stretching injuries and/or sympathetic plexus irritation.²⁹

The anterior atlantoaxial and atlanto-occipital membranes are an upper cervical continuation of the anterior longitudinal ligament from the lower cervical spine. Similarly, the flaval ligament continues cranially as the posterior atlantoaxial and atlanto-occipital membranes. While serving a similar stabilizing role, these stiff and inelastic membranes are not as flexible as their lower cervical counterparts, especially in the case of the elastic flaval ligament. The nuchal ligament contributes to constraining this region of the spine, as a continuation of the interspinous and supraspinous ligaments. This system demonstrates both a funicular component continuing cranially from the supraspinous ligament and a lamellar component coursing ventrally from the funicular component to spinous processes.³⁰ This ligament clearly constrains upper cervical movements, especially during full cervical flexion and retraction. As a consequence, the C0C1 segment paradoxically extends during full cervical flexion.³¹ Thus, testing upper cervical flexion with full cervical retraction and upper cervical extension with full cervical protraction can offer the clinician a view of the relative role the ligament is playing in a patient's upper cervical limitations.³²

Of historical concern for clinicians has been the course and configuration of the greater and lesser occipital nerves, considering the relative importance they have been assigned as pain generators in selected cervicocephalic disorders. The greater occipital nerve emerges to course subcutaneously over the cranium at approximately 0.5 to 2.8 cm lateral to the midline along the intermastoid line. Similarly, the lesser occipital nerve emerges at the mid-part of the posterior border of the sternocleidomastoid muscle, approximately 3 to 5 cm lateral to the midline on the same intermastoid line. However, considerable variance has been observed in the locations of these nerves relative to one another, as well as in relation to the occipital artery.^{33,34}

The dorsal root ganglia (DRG) of C2 spinal nerves, along with their bony and soft tissue containers, vary in shape and size, appearing as oval, spherical, or spindle-like.³⁵ These ganglia appear to be particularly vulnerable to compressive and/or tension loading events. The spinal DRG is found to emerge anterior to the epistrophic ligament (or posterior atlantoaxial membrane) and in the close proximity of the dorsal C1C2 lateral joint capsule and a rich venous plexus.^{36,37} The DRG can be compressed by any of these structures. However, neural deformity between the dorsolateral bony arches of C1 and C2 appears controversial.^{35,36} Moreover, the ventral ramus of C2 can be stretched along its course

behind the articular processes associated with the C1C2 lateral joint, especially in response to certain rotatory cervical movements.³⁸

Preganglionic sympathetic neurons coursing to the head, upper cervical spine, and throat begin at cervicothoracic levels from C8 to T4, whereas the sympathetic supply to the mid- and lower cervical spine, upper back, and arm begins in a region of the spinal cord from T4 to T9. Those neurons exit the ventral rami through the white communicating rami and enter the ascending paravertebral ganglion chain and then synapse with postganglionic fibers in the upper, middle, and lower cervical ganglia. These arrangements play a role in managing the sympathetic contributions to chronic headache and neck pain.

Noteworthy are the accessory nerve nuclei located in the spinal cord between C1 and C4.³⁹ Chronic upper cervical conditions may produce an increased sensation of tightness without increased muscle tone⁴⁰ in the patient's upper trapezius muscles, by virtue of sensitization and reorganization of interneurons at those same cervical cord levels. A similar neuroadaptive response can be observed originating from the cervical trigeminal nuclei found in the same levels of the spinal cord.³⁹ Chronic cervical afference (especially pain) can sensitize these cranial nerve nuclei, resulting in chronic pain and headache in the cutaneous trigeminal distribution ("cervicotrigenal relay").⁴¹ This explains the retro-orbital pain so often encountered in patients with cervicogenic headache.

BIOMECHANICS

The principle motions allowed at the C0C1 motion segment are designed primarily for head movement in the sagittal plane about an axis that courses through both external auditory meati of the ears. The upper cervical spine demonstrates a total maximum range of approximately 30° to 35° from a fully flexed to a fully extended position, with isolated flexion approaching 10° and isolated extension measuring approximately 25°. To achieve this movement, the convex occipital condyles arthrokinematically roll in the same direction of motion and slide in the opposite direction, exhibiting a greater rolling vs. sliding component during the movement. This segment exhibits sidebending axis coursing through the nose, where right sidebending produces occipital condylar rolling to the right and sliding to the left. At the same time the atlas (C1) exhibits relative right translation, which can be easily palpated between the lateral edge of the transverse process and the mas-

toid process on the ipsilateral side. Each of these motions can be impeded when any of these sliding motions are lost, suggesting the utility of therapeutic manual techniques aimed at restoring the translatory behaviors.^{42,43}

The greatest range of motion observed in any neck motion segment is witnessed at C1C2 (the atlantoaxial segment). Sagittal motion at C1C2 is limited to anterior and posterior rocking (20° total),^{32,42} whereas 40° to 45° axial rotation around a vertical axis through the dens is allowed to each side.^{5,42,44} With right rotation, the right C1 inferior articular facet slides posterior to the right C2 superior articular facet and the left C1 facet anterior to left C2. Additionally, the convex-on-convex relationships of the C1C2 ZAJ allow the entire C1 segment to exhibit caudal translation on C1 in a limited fashion, thus allowing further rotation without premature movement constraint associated with tension loading of capsuloligamentous structures.

Upper cervical sidebending directly influences movement at C2C3. Right sidebending at C0C1 tension loads the right occipital and left atlantal branches of the alar ligament system, thus producing right rotation of C2 on C3 as a consequence of the connection between these ligaments and the dens (Figures 6, 7). This resultant

C2C3 right rotation creates a relative C1C2 left rotation, creating the expected upper cervical contralateral coupling.^{27,45} Additionally, C0 will not sidebend when C2 cannot rotate on C3, thus generating an apparent upper cervical motion loss. Therefore, one should view the C2C3 motion segment as a “keystone” to upper cervical motion, as normal C2C3 movement is essential for upper cervical function.

Additional upper cervical motions are required to keep the eyes level during cervical axial rotation. The alar ligaments force the occiput to sidebend opposite the direction of rotation, producing upper cervical contralateral coupling.^{27,45} At the same time, the lower cervical spine sidebends in the same direction as the rotation so to counter the sidebending in the upper cervical spine.⁴⁶ Instantaneously, the cervical spine extends above and

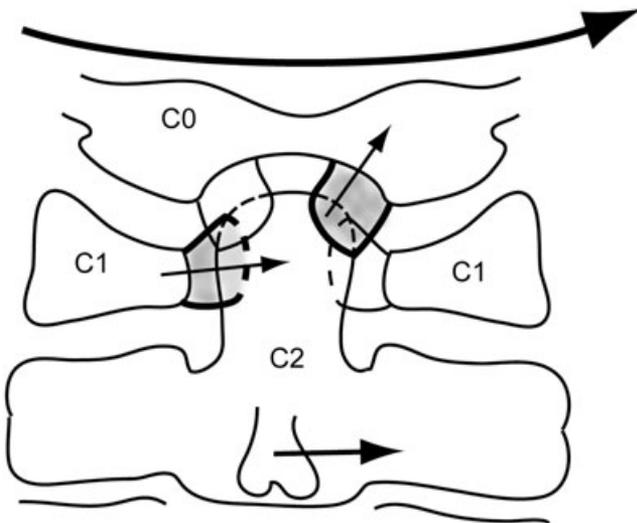


Figure 6. Influence of the alar ligaments on coupled motion in the upper cervical spine (C0 to C3; dorsal view). Left occipital sidebending is accompanied by right occipital translation. Right translation tension loads right occipital and left atlantal alar ligaments. (Reprinted from Racz GB, Anderson SR, Sizer PS, Phelps V. Atlantooccipital and atlantoaxial injections in the treatment of headache and neck pain. In: Waldman S, ed. *Interventional Pain Management*. 2nd edn. pp. 295–305, 2000, with permission from Elsevier.)

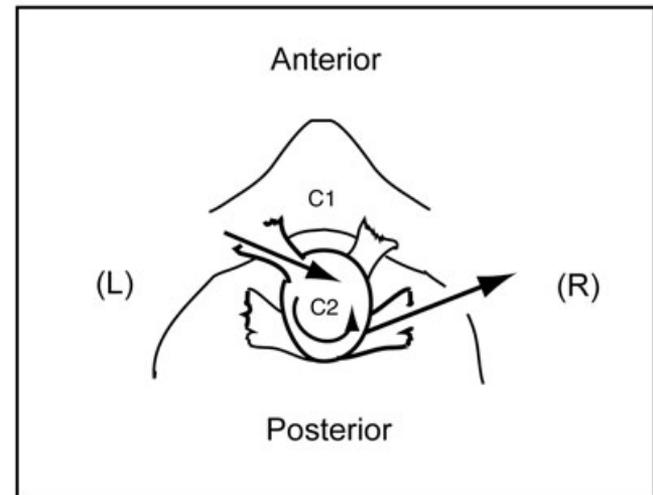


Figure 7. Influence of the alar ligaments on coupled motion in the upper cervical spine (C0 to C3; cranial view). Left occipital sidebending is accompanied by right occipital translation. Right translation tension loads right occipital and left atlantal alar ligaments. This tension behavior induces left rotation of C2 on C3, and subsequent right rotation of C1 on C2. (Reprinted from Racz GB, Anderson SR, Sizer PS, Phelps V. Atlantooccipital and atlantoaxial injections in the treatment of headache and neck pain. In: Waldman S, ed. *Interventional Pain Management*. 2nd edn. pp. 295–305, 2000, with permission from Elsevier.)

flexes below C3C4. Otherwise the individual will look down when attempting cervical axial rotation.

A “chin tuck” motion produces flexion at C0C1 and considerable load on the TLA. Clinicians often use this “chin tuck” motion therapeutically for treating lower cervical conditions or when attempting to correct postural deviations; however, headaches may result because of these imposed stresses and tension loading on the posterior atlantoaxial membrane, resulting in C2 segmental nerve compression (especially when accompanied by cervical rotation). Otherwise, solitary segmental movements are impossible in the upper cervical spine, in response to the powerful influence that the TLA and alar ligaments impose on kinematic behaviors. During kinetic sidebending, the upper cervical spine is obligated to rotate contralaterally. Additionally, sidebending may require the upper cervical spine to flex or extend, depending on each person’s unique kinematic behaviors.^{27,47–49}

EXAMINATION

History

A patient’s gender, occupation, and age may lend to the prevalence of cervicocephalic symptoms, or cervicogenic headache. For example, managerial and professional occupations appear to be more related to cervical pain and headache vs. clerical or blue-collar jobs, especially in women.⁵⁰ A similar female prevalence appears in osteoarthritis in the upper cervical joints.⁹ Local cervical syndrome (LCS) most commonly occurs with individuals between the ages of 20 and 45 years. Primary disc-related disorders usually occur in the younger ages of this group, while the latter end of this same group marks the beginning of secondary disc-related disorders, such as chronic internal disc disruption, or joint arthropathies.⁵¹

Occipital numbness and tingling suggest lesions of the C1 to C3 roots. Upper cervical pain occurring with upper cervical movement or specific head positions suggests pathology between the levels C0C1 and C2C3. The local or referred pain produced from these levels can be intermittent or constant in nature,^{9,52} possibly accompanied by dizziness, nausea, vomiting, blurred vision, photophobia, and phonophobia.^{53–57}

The location of the patient’s symptoms may suggest the general location of the pain generator, but specific localization is complicated by overlapping zones of pain reference. The ZAJ pain referral pattern from C1C2 is local and unilateral in the suboccipital region, whereas

C0C1 produces more diffuse unilateral suboccipital and occipital pain.⁵⁸ Further diagnostic difficulty arises when considering that suboccipital and occipital pain could arise from internal disc irritation at C2C3, C3C4, C4C5, and C5C6.⁵⁹ Internal disc irritation of the various cervical disc levels can produce referred pain in a myriad of locations that overlap in the neck, head, face, and ear regions. Because of this overlapping symptomatology, specific historical interpretation and/or systematic testing should be performed to differentiate between afflictions at each of these sites.⁶⁰

The initiation and natural progression of a patient’s symptoms are worthy considerations. Although cervicocephalic symptoms can occur spontaneously, they are often precipitated by an awkward position of the neck for an extended position, such as awakening with a stiff neck and headache, turning the head to back up the car, and turning toward someone when sitting beside them in a conversation.^{54,61} The duration of cervicocephalic symptoms can be variable. Fredriksen et al. documented a 13-year mean duration for occipital pain and upper cervical symptoms in a relatively small group. During periods of exacerbation, their subjects’ symptoms continued for a period of 3 hours to 3 weeks, while intervals between these flare-ups ranged from 2 days to 2 months.⁶¹

Structural and functional complexities in the upper cervical spine frequently contribute to local cervical pain and cervicogenic headaches.^{6,62} Symptoms arising from these regions are complicated by afflictions involving the dura mater, vertebrobasilar arterial system, and the autonomic nervous system.^{63–65} The vertebral arteries and upper cervical dura are innervated by the first three cervical nerves, making them capable of produce similar headache symptoms.^{6,66} Moreover, hemorrhage, tumors, arteriovenous malformations, and systemic diseases (such as temporal arteritis, systemic arthritides, hypertension, migraine, infections) must be differentially ruled out when examining a patient with headache.

Differential Diagnosis

Tension headaches, which are frequently triggered by central sensitization and cervical trigeminal dysfunction, are nonpulsatile and more common in women.^{67–70} Tension headaches produce band-like pain that bilaterally radiates from the forehead to the occiput and possibly the neck musculature. This type of headache lacks the features commonly associated with migraine headaches (unilateral, throbbing pain, nausea, photophobia).

Patients frequently describe this headache as a tightness, pressure, and/or dull ache. Interestingly, tension headaches are not accompanied by changes in electromyography of cervical or facial musculature.⁷¹

Cluster headaches are severe headaches that are apparently triggered by melatonin and cortisol secretion abnormalities and subsequent circadian rhythm disturbances in the inferior hypothalamus.⁷²⁻⁷⁵ Cluster headaches are more common in males, relatively rare, episodic, frequented at night, and characterized by trigeminal nerve-mediated pain. They are unilateral, retro-orbital, and possibly accompanied by ptosis and lacrimation.^{76,77} However, they are not typically accompanied by nausea, vomiting, or seizures. Cluster headaches can be precipitated by alcohol consumption and relieved by physical activity. Painful cluster headache attacks can persist for several weeks (or even years) when chronic, followed by variable periods of remission.^{78,79}

Migraine headaches are common, severe, and debilitating. However, investigators have debated the etiology of migraine, suggesting either a vascular or neurological origin.⁸⁰ More recent evidence suggests that migraine headaches may emerge from vascular changes in the rostral brainstem (including midbrain, pons, and periaqueductal gray matter) in response to a trigeminoparasympathetic reflex trigger.^{79,81-84} Migraine headaches can clinically present in a fashion similar to cervicogenic headaches, in that they can be unilateral, moderate to severe, and lasting from 4 to 72 hours. They are more often in females, throbbing and accompanied by nausea, vomiting, and photo- and/or phonophobia. Both cervicogenic headaches and migraines can produce paresthesias in the face, arm, tongue, and palate, complicating the diagnostic pictures of both conditions.

Investigators have attempted to differentiate migraine symptoms from those produced during cervicogenic headache. Sjaastad et al. reported that 90% of migraine patients experience initial pain that begins in the forehead and temporal regions. In contrast, 73% of cervicogenic headache patients report initial pain extending from the neck into the head. However, differences in these conditions may be indistinct during a full-blown attack of either a cervicogenic headache or migraine.⁸⁵ Unlike cervicogenic headache and the cluster headache, the migraine can alternate sides (also called "side shift"). Unique to migraine headaches are visual auras and/or halos around light sources. While physical activity or positional changes frequently trigger cervicogenic headaches, migraines are less likely to be affected by those changes.⁸⁶

Exertion headache is a bilateral throbbing headache that is related to Valsalva-like behaviors associated with exertion, resulting in an increase in intracranial pressure. This form of headache, triggered by heavy exertion or exhaustion, is slow in onset, slightly more frequent in females, and lasts from a few minutes to a full day.⁸⁷ Jab/Jolt syndrome, also called idiopathic stabbing headache, produces brief paroxysmal episodes of headache pain that are sharp, piercing, brief, irregular, penetrating, and nonpulsatile. The pain is unilateral in the temporal or fronto-orbital regions and is typically mild to moderate in nature. While Jab/Jolt syndrome can immediately precede the onset of a migraine, they are not necessarily related to migraine incidence.⁸⁸

Vertebrobasilar insufficiency is capable of producing headache symptoms that are accompanied by vertigo and triggered by a compromise to the vertebral artery on its cranial course to join the basilar artery.⁸⁹ The compromise is related to tension that results in arterial compromise or sympathetic plexus irritation (known as "Functional VBI").^{29,39} This compromise activates a local inflammation, angiospasm, vascular dysregulation, and possible latent fibrosis in the arterial wall. The arterial vulnerability can be located high in the proximity of C1 and C2 transverse processes, at the dorsomedial margin of the atlantal lateral mass, and at the junction of both vertebral arteries with the basilar artery.

Other causes of headache have been identified. Headaches can emerge after head trauma, mediated by central and peripheral neural processes. Investigators have implicated the sphenopalantine ganglion as a pain generator in these headaches.⁹⁰ Hypertension, hypotension, ischemic or hemorrhagic stroke, vascular dissection, hematoma, and vasculitis can produce headache symptoms. These frequently demonstrate rapid onset and are accompanied by nausea, vomiting, and possible seizures. Neuroimaging is indicated for the triage of these headaches, especially when the symptoms are the first and severe, a new onset after age 50 years, or one that is progressive worsening after a subacute headache. These headaches can be accompanied by decreased consciousness, abnormal neurological examination outcomes, and associated symptoms of meningismus and fever.⁹¹

Inspection

Before initiating the physical examination, the clinician should inspect the patient for postural abnormalities. These initial observations should include an appraisal

of patient's cervical lordosis, which can be confirmed by radiographs. Decreased cervical lordosis suggests a kyphotic kink related to internal disc disruption.^{92,93} On the other hand, increased cervical lordosis in elder patients reflects advanced degenerative disc disease.⁹⁴ While observed cervical lordosis alterations can be clinically noteworthy, they do not necessarily indicate segmental abnormalities.⁹⁵

Postural deviations can accompany selected upper cervical conditions. Increased forward head posture, resulting in upper cervical extension and a head position outside its base of support at the shoulder girdle, can be observed in patients suffering from a cervical postural syndrome. This position potentially produces posterior capsular adaptation in the upper cervical spine and exposes those segments to abnormal and excessive stresses.²³ These deviations are likely related to disturbed sensory motor control, along with gradual segmental adaptation and dysfunction, making them difficult for patients to self-correct. For example, post-whiplash patients may be unable to reproduce a "neutral" head position during recovery. At rest, they demonstrate a tendency to position the head in slight axial rotation or sidebending, which they perceive to be straight. When there is injury or dysfunction of the sensory receptors innervating or surrounding the cervical structures (such as post-whiplash), impairment of these structures leads to altered proprioception and persistent postural deviations.⁹⁶

Specific upper cervical conditions can produce torticollis in the cervical spine. Upper cervical (C1C2) rotatory subluxation can produce cervical postural distortions, as one might observe after trauma to the upper cervical ligaments. Another cause of a dissociation of the dens from the C1 is Grisel's syndrome, which is a nontraumatic atlantoaxial rotatory subluxation.^{97,98} This infrequent condition can be congenital (dens malformation or C1C2 ligament laxity) or can be the consequence of rheumatoid arthritis, ankylosing spondylitis, psoriasis, infectious processes of the throat and upper respiratory tract.^{23,97,99-101} This condition presents with a flexion-rotation-deviated head posture that is the effect of C1C2 dissociation,²³ potentially producing profound consequences that include spinal cord injury.¹⁰²

Clinical Examination

A clinical examination should help to confirm or rule out various cervical pathologies that relate to the patient's history. This examination is implemented for the diagnosis of cervicocephalic conditions that can be

provoked during the examination. The examination includes cervical movements in the sagittal (flexion, extension, protraction, and retraction), transverse (rotation), and frontal (sidebending and sidenodding) planes. The clinician notes movement disturbances and provocation patterns that can reflect the location of the pain generator and associated cervical dysfunctions.

The upper cervical spine movement should complement lower cervical segmental behaviors in keeping the eyes horizontally oriented in space. If the upper cervical segments are hypomobile, then this compensatory relationship could be disturbed, presenting itself in a number of "deviated" patterns when the patient performs active cervical movements.⁶⁰ For example, this may be witnessed during full cervical rotation, where the patient's upper cervical sidenodding limitations force the patient to ipsilateral sidebend, whereas upper cervical extension limitations require that the patient looks down during the rotation. Additionally, when upper cervical sidenodding is limited, full cervical sidebending will be performed primarily at the lower cervical spine and will appear as if the patient is laying the head on the shoulder.

Cervical retraction and protraction maximize upper cervical spine motion in the sagittal plane. Active and passive retraction, where a chin tuck (upper cervical flexion) is combined with lower cervical extension, produces greater upper cervical flexion than does simple cervical flexion. Conversely, protraction of the cervical spine best tests maximal extension of the upper cervical segments.³² While these can be provocative in lower cervical disc pathology, they may suggest upper cervical pathology when they produce cervicocephalic symptoms. If sagittal plane motions are the most provocative for cervicocephalic symptoms, then the clinician can differentiate between involvements of the upper cervical joints vs. the disc in the cervical disc segments. If the clinician provokes the patient's upper cervical symptoms during protraction and/or retraction, the clinician can begin to suspect involvement of structures found in either the C0C1 or C1C2 motion segments. Involvement of these segments is best represented by greatest provocation during rotation or sidenodding in a protracted and/or retracted position. If sidenodding in either protraction or retraction produces the greatest pain in the examination, then the clinician should suspect a pain generator in the C0C1 joint system. Similarly, when the greatest symptoms are provoked through rotation in a protracted or retracted position, then the C1C2 segment should be considered. If the previous selective provoca-

tion tests do not produce or increase the patient's symptoms, then greatest cervicocephalic pain during sagittal plane motions suggests cervical disc pathology.

Zygapophyseal joint afflictions produce the greatest pain during three-dimensional rotation, in concert with their role in constraining three-dimensional movements.^{103,104} When the patient reports the greatest cervicocephalic symptoms during active or passive cervical axial rotation in the transverse plane, then the clinician should suspect ZAJ involvement the upper cervical disc segments. When the pain generator is located in the C2C3 ZAJ capsular synovium, then the patient may experience greatest pain provocation with cervical rotation and sidebending toward the side of pain, along with extension at the end range. On the other hand, when the patient experiences the greatest pain with rotation away from the side of pain accompanied by sidebending toward, then the examiner should suspect involvement of the C2C3 ZAJ articular cartilage.

A sidenod movement can be performed to emphasize upper cervical motion in the frontal plane. This test allows the clinician to assess motion in the upper cervical segments (C0C1 through C2C3), as sidenodding is primarily constrained by structures in this region.^{45,47,105} If the greatest cervicocephalic symptoms are produced with sidebending, then 3-dimensional testing for the C2C3 UVJ should be implemented.¹⁰⁶ After sidebending, the patient should then ipsilaterally rotate, producing a motion that maximally stresses the UVJ capsule and synovium on both the ipsilateral and contralateral side to the direction of sidebending.¹⁰⁷ Contralateral rotation can added to the sidebending so to compress the C2C3 UVJ articular surfaces on the side ipsilateral to the direction of sidebending, thus provoking a UVJ arthropathy.¹⁰⁸

Mobility can be further evaluated through quick tests and segment-specific testing. Movement C0 to C4 can be screened by fully rotating the patient's cervical spine to one direction, followed by cervical flexion. A flexion limitation at the C0 to C4 segments is suggested when the patient is unable to flex. Similarly, if the individual is unable to achieve full rotation to one direction, and in this position is unable to extend the head, there is likely to be an upper cervical extension limitation (because full upper cervical extension is needed to achieve full cervical rotation).

A quick test for C1C2 rotation can be performed by asking the patient to sidebend fully, and then from this position rotated in an ipsilateral direction. If the patient performs the sidebend and is unable to maintain the



Figure 8. Sidebending (lateral flexion) end-feel test to right (R) C2 on C3. While palpating the C2C3 segment in a segmental sidebent position, the therapist uses the same finger to produce a springing overpressure movement in a medial and slightly ventral direction. (Reprinted from Sizer P, Phelps V, Brismee JM. Diagnosis and management of cervicogenic headache and local cervical syndrome with multiple pain generators. *J Man Manip Ther.* 2002;10:136–152. with permission from Journal of Manual and Manipulative Therapy.)

nose in a forward facing position (ie, the head also rotates toward the sidebending side) there is likely to be a contralateral rotation limitation at C1C2. If the patient is able to fully sidebend, then they should be able to ipsilaterally rotate a total of approximately 80° to 90° from this position. If not, there is likely an ipsilateral rotation limitation at C1C2.

Segmental mobility can be tested at each cervical disc segment using a sidebending end-feel test (Figure 8).²³ The lateral aspect of a particular ZAJ is palpated along the articular pillar with the index finger pointing toward the orbit.¹⁰⁸ Once palpated, the cervical spine is passively sidebent until an extension (closing) movement is detected in the articular space. This new position is maintained while the palpating finger produces a springing overpressure movement in a medial-ventral-cranial



Figure 9. Left alar ligament test: standing to the side of the patient, with stability hand contacting C2 (thumb to left lamina and spinous process and index finger to right lamina); the mobility hand contacts the patient's right lateral occiput as pictured. The clinician provides mild distraction to the occiput and then sidenods the head to the right while stabilizing C2, preventing C2 rotation to the right. Normally, no sidenod motion is permitted. The test is positive for ligament compromise if the sidenodding is permitted. (Reprinted from Hay A, Azevedo E, Phelps V, Sizer P. Upper cervical disorders part I: history, inspection and clinical examination. *MD Consult* 2004; Available at <http://home.mdconsult.com/das/stat/view/34714741-2/pers.>)

direction toward the orbit.⁶⁰ Additionally, the clinician can assess lateral translation testing of the C0C1 segment, as well as segmental rotation of C1 on a stabilized C2.

Special tests can be added to the upper cervical examination. The integrity of the ligaments and TLA should be tested in a patient who has symptoms of upper cervical pain, dizziness, and nausea. For the alar ligament test the clinician stabilizes the seated patient's C2 segment and attempts to sidenod the head after providing minimal traction to the cranium (see Figure 9).⁶⁰ An alar ligament lesion allows sidenodding of C0 to C2 in spite of C2 vertebral stabilization, whereas no motion is allowed with a healthy alar ligament.^{17,45}



Figure 10. Dorsal transverse ligament of atlas (TLA) test. With the stability hand contacting dorsal C2 (thumb to left and index finger to right dorsal C2 transverse process) and the mobilizing hand contacting the patient's right and dorsal occiput and C1, the clinician provides ventral-caudal stabilization to C2 accompanied by gradual ventral translation of the occiput and C1. Movement of the occiput takes C1 along ventrally. If the TLA is intact, C1 movement is arrested by the TLA. The test is positive for ligament compromise if symptoms are produced. (Reprinted from Hay A, Azevedo E, Phelps V, Sizer P. Upper cervical disorders part I: history, inspection and clinical examination. *MD Consult* 2004; Available at <http://home.mdconsult.com/das/stat/view/34714741-2/pers.>)

The TLA can be tested with the patient in a seated position (see Figure 10). For testing in the sagittal plane, the clinician posteriorly stabilizes C2 by pushing the inferior articular processes caudal direction onto C3. Then the clinician exerts a ventral translatory pull against the posterior arch of C1 and the cranium, so to pull C0 and C1 forward and allow the dens to move relatively backward into the direction of the spinal canal. The TLA can be laterally tested by stabilizing C1 at the lateral transverse process while attempting to translate the head in a pure lateral direction. An intact TLA will resist any C2 posterior subluxation, whereas a compromised TLA allows C2 to translate posteriorly into the brain stem region of the spinal cord. This excessive translation can provoke cord symptoms, including nausea, dizziness, and/or deep agonizing pain.

In addition to the above ligament tests, patients with selected cervicocephalic symptoms (especially dizziness) should be evaluated for VBI. This condition can be detected through a series of movement tests that attempt to compromise the vertebral arteries as they course through the transverse foramina to join the basilar artery in the subarachnoid space, also known as the

modified DeKleyn and Nieuwenhuys tests.²³ While functional VBI can be triggered by positions of flexion or extension with rotation,¹⁰⁹ these positions do not necessarily identify the patient at risk of experiencing vertebral artery dissection during spinal manipulation or trauma,^{110,111} suggesting limitations to the utility of the tests.

Cervicomedullary conditions can accompany cervicocephalic syndrome. When assessing reflexes for the upper cervical spine, Babinski is only positive in about one-third of the cases of neural compression at the levels of C0C1 and C1C2. However, the scapulothoracic reflex has been found to be a more accurate test for myelopathy at C0 to C4 segments.¹¹²

INTERPRETATION AND MANAGEMENT

Successful management of a cervicocephalic disorder is more likely when the specific etiology is considered and diagnosis-specific management strategies are implemented. For example, blockade measures can reduce symptoms associated with cervicogenic headache, while migraine symptoms remain relatively untouched by similar blockade procedures. In contrast, the vasoactive medications that are frequently effective in managing migraines are less useful for treating cervicogenic headache. While lithium and/or melatonin can reduce cluster headache frequency, these interventions are not effective with cervicogenic or migraine headaches. Finally, while surgical release can ablate selected conditions that contribute to cervicogenic headache, similar procedures do not address symptoms of migraine or cluster headache.¹¹³ Therefore, the clinician is encouraged to engage in diagnosis-specific measures when treating various headache conditions.

Cervical Postural Syndrome

Cervical postural syndrome (CPS) can generate cervicocephalic symptoms and precede primary disc-related disorders. This syndrome is related to cervicothoracic muscle imbalance and resulting aberrant posture. It is often seen in those who exhibit a persistent forward head posture and tired neck, such as students, drafting technicians, and computer operators, especially when female. Sustained positional activities, such as prolonged driving and bifocal use, may contribute to its onset. While patients frequently report fatigue and temporal mandibular joint symptoms they rarely demonstrate a positive functional examination for limits and/or symptom provocation.²³

The important conservative management strategies for these patients include postural re-education. Postural control is the result of motor programming that is developed through extensive repetition. Thus, these patients are best treated with postural training that uses simple postural cues, such as tape adhered lengthwise along the thoracic spine that can serve as a cue to the patient for maintaining upright postural positioning. In addition, extraocular muscle activation is incorporated for insuring cervical stabilization and postural control,¹¹⁴⁻¹¹⁶ while low-load isometrics exercises can address the diverse neuromuscular re-education needs associated with this syndrome.¹¹⁷ A patient's cervical range of motion can be increased and pain decreased with cervical resistance training.^{118,119} Finally, local infiltrations to muscle trigger-point regions can help ameliorate muscle guarding and symptoms.^{23,120}

Local Cervical Syndrome

The pain associated with LCS can be localized to the upper cervical spine. In the context of upper cervical afflictions, the discussion will be limited to the C2C3 segment. While each segment can produce its own region of pain, these zones overlap and can potentially extend above and below the level from which the pain originated (as previously described).^{108,121}

An LCS can produce suboccipital local cervical symptoms resulting from primary and/or secondary disc-related disorders, including: (1) internal disc disruption; (2) synovitis of the UVJ; (3) chondropathy of the UVJ; (4) synovitis of the ZAJ; and (5) chondropathy of the ZAJ. The differential diagnosis of these conditions has been previously discussed.¹⁰⁸

Diagnosis-specific management strategies can include several different conservative and invasive measures for the treatment of LCS. Manual therapy treatments can include soft tissue mobilization, joint-specific mobilization, and neuromuscular re-education. Internal disc disruption is best treated with dorsal ventral mobilization to the caudal vertebral body of the pain-generating motion segment.^{60,92} Pain-relieving and motion-improving manual therapy techniques are both necessary for the management of cervical ZAJ disorders. Oscillatory joint-specific gliding can be applied to the pain-generating segment, while hypomobilities in adjacent segments may be treated with manual therapy measures that are aimed at improving segmental extension and/or flexion mobility. Segmental sidebending can be best treated with UVJ mobilization, where the cranial vertebral body is glided laterally while sidebending the

segment in the opposite direction to the gliding (“Segmental Scooping”).⁶⁰ Considering the neural and histochemical complexities of the cervical intervertebral disc^{122,123} and ZAJ,¹²⁴ invasive procedures can be utilized to address pain from local cervical syndrome. Disc pathology can be treated with epidural steroid injections,¹²⁵ while ZAJ pathology can be diagnosed and treated with medial branch blocks and radiofrequency ablation, respectively.^{126–128}

Cervicocephalic Syndrome

Cervicocephalic syndrome, or cervicogenic headaches are typically unilateral in location (but can present bilaterally), moderate to severe in nature, and last 4 to 72 hours. They are commonly aggravated by physical activity, such as cervical movement or prolonged posturing. However, these headaches can demonstrate unprecipitated attacks that could increase in frequency over time and finally merge into a pattern of chronic, fluctuating persistent headache symptoms. They can be dull and diffuse, as well as throbbing in nature. They can be accompanied by photo- and phonophobia, as well as nausea and vomiting. They are often accompanied by pain in the interscapular and upper trapezial regions, as well as cervical motion limitations.^{56,86,113}

Cervicocephalic syndrome can be the result of several of the same pain generators as LCS. However, the distinguishing factor for cervicocephalic syndrome is the location of pain and the symptoms produced. Cervicocephalic syndrome includes symptoms produced in the head and face. Cervicocephalic symptoms can emerge from: (1) disc pathology at C2C3 to C5C6;^{59,129} (2) C2C3 ZAJ synovitis; (3) C2C3 ZAJ chondropathy; (4) epidural irritation and/or adhesion;⁴ (5) C0C1 and C1C2 joint arthropathy; (6) upper cervical nerve root lesions (C0 to C3);^{4,36,38} (7) upper cervical DRG impingement;^{36,37} and/or (8) cervicotrigeminal relay (C0 to C4).¹³⁰

The differential diagnosis and treatment of cervical disc and ZAJ conditions have been previously discussed.¹⁰⁸ Cervical dural irritation/adhesion can result in diffuse-referred cervicocephalic pain, because of its polysegmental innervation. The cervical dura has a direct connection with the nuchal ligament and rectus capitus posterior minor. The connective tissue link spans the dorsal intervertebral spaces of C0C1 and C1C2.¹³¹ Consequently, any cervical movements that tension load the dura and/or roots, such as flexion, could provoke symptoms. One’s suspicion of epidural adhesions can be substantiated through fluoroscopically guided, diagnos-

tic radiolucent injection and, while difficult to address with conservative measures, this condition may be effectively treated with fluoroscopically guided epidural steroid injection series.^{125,132}

Upper cervical joint arthropathy presents with unique symptoms and can be diagnosed through joint-specific provocation tests. Mild symptoms may be produced with sagittal, frontal, and transverse plane movements. However, selected provocation tests will likely be the most painful. Joint arthropathy (synovitis or chondropathy) of a joint at C0C1 presents with unilateral vague pain over the suboccipital region and over the back of the ipsilateral occiput. Trauma may precede symptom onset and the affliction could be accompanied by C1C2 hypermobility. While absent of numbness or pain with Valsalva, this condition is most provoked with protraction (upper cervical extension) or retraction (upper cervical flexion) accompanied by sidenodding. The patient may demonstrate joint mobility limits at C0C1. Investigators have demonstrated that joint-specific manual treatments and exercise are effective in reducing symptoms associated with cervicogenic headache.¹³³ Thus, joint-specific mobilizations, limitation-specific active movements, and home exercise are indicated for patients with C0C1 limitations.

Joint arthropathy at C1C2 will present as unilateral paramedian pain and will be most painful with rotation in a protracted or retracted position. Once again, the condition will not be accompanied by numbness, hyposthesia, or symptoms with Valsalva. A trauma history is possible but not required to initiate this condition, which can present with hypo- or hypermobility at C1C2. Mobility alterations can be detected during the C1C2 joint-specific testing and hypermobility can be accompanied by a positive alar ligament laxity, suggesting upper cervical instability. Hypomobilities at this level are effectively treated with joint-specific mobilizations, repetitive movement, and home exercise. In addition to joint mobilizations, postural re-education and neuromuscular re-education should be performed to the cervical spine. However, if the segments are too sensitized to treat manually or are not responding appropriately to manual therapy, injections can be performed at the C0C1 and C1C2 joints.⁴ Hypermobilities are best treated with a progressive cervical stabilization program. This program emphasizes eye movements to activate the cervico-ocular reflex system and activation of the deep anterior muscles of the cervical spine (such as longus colli),^{134,135} whose control can be altered during functional movements of the neck and upper extremi-

ties.^{134,136} Radiologically appreciable upper cervical instability may merit surgical stabilization, especially when the transverse ligament of atlas is involved in the compromise.

Deformation of the C2 root and DRG can produce cervicocephalic symptoms.³⁶ While chemically activated root mechanosensitivity and subsequent pain is gradual and progressive, DRG deformation produces immediate increased pressure and pain provocation.¹³⁷⁻¹⁴¹ In addition, neurophysiological after-discharges can be triggered for up to 25 minutes after DRG deformation is ceased, because of high Na⁺ channel concentration in the DRG.^{142,143} Therefore, this sharp, mechanically induced pain is differentiated from the gradual aching pain that results from chemically mediated mechanosensitivity of the root. In addition, although mechanical interventions could alter DRG-related pain, pharmacological management may be preferred for managing chemically triggered, root-related pain.¹⁴⁴

Dorsal root ganglion compression between the dorsolateral bony arches of C1 and C2 has been suspected for producing unilateral suboccipital pain that is aching in nature, along with lancinating pain up the back of the head during head and neck movement.³⁶ While, recent cadaveric analysis has questioned actual bony contact between the C2 DRG and either C1 or C2 bony arch structures,³⁵ no consideration was given for the possible alterations in the neural container with a history of trauma, which is typically reported by these patients. Patients will not complain of pain provocation during valsalva, but they can report occipital sensory loss. The patient's symptoms are most easily triggered during full cervical protraction accompanied by contralateral rotation. This affliction is best managed invasively with epidural catheterization in proximity of the C2 DRG, along with progressive upper cervical stabilization activities.³⁶

In addition to the previously described C2 DRG compressive event, the same ganglion can become entrapped under the epistrophic ligament (or posterior atlantoaxial membrane).³⁷ These patients will frequently report a trauma history and complain of the previously described occipital lancinating pain, along with constant unilateral or bilateral suboccipital pain. However, unlike the previous affliction, the patients will not demonstrate C1C2 hypermobility. These patients consistently demonstrate absolute decreased sensation over the occiput. Once again, there are no complaints of pain with Valsalva and symptoms will likely be provoked with protraction and contralateral rotation. Conclusively, this

affliction is differentiated from the previous affliction through the negative trauma history, loss of sensation, and normal mobility at C1C2. Epidural catheterization and radiofrequency lesioning can be attempted, but the patient may require partial surgical release of the epistrophic ligament. While this type of surgical intervention can ablate entrapment and related symptoms, postsurgical scarring and contracture can lead to relapse.¹¹³

SUMMARY

Upper cervical pain and/or headaches originating from the C0 to C3 segments are pain-states that are commonly encountered in the clinic. The upper cervical spine anatomically and biomechanically differs from the lower cervical spine. Patients with upper cervical disorders fall into two clinical groups: (1) local cervical syndrome; and (2) cervicocephalic syndrome. Symptoms associated with various forms of both disorders often overlap, making diagnosis a great challenge. The recognition and categorization of specific provocation and limitation patterns lend to effective and accurate diagnosis of local cervical and cervicocephalic conditions.

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Review questions

1. Uncinate processes are found on which component of the cervical vertebra?
 - a. Caudal-lateral C1 vertebral body
 - b. Cranial-lateral C2 vertebral body
 - c. Caudal-lateral C2 vertebral body
 - d. Cranial-lateral C3 vertebral body
2. All of the following cervical motion segments demonstrate four synovial joints, EXCEPT for:
 - a. C0C1
 - b. C1C2
 - c. C2C3
 - d. C3C4
3. Which upper cervical joint system demonstrates a biconvex joint surface relationship whose incongruencies are filled with meniscoid structures?
 - a. C0C1
 - b. C1C2
 - c. C2C3
 - d. C3C4
4. The upper cervical continuation of the lower cervical ligamentum flavum is the:
 - a. Anterior atlantoaxial membrane
 - b. Nuchal ligament
 - c. Posterior atlantoaxial membrane
 - d. Transverse ligament of atlas
5. The greatest range of motion observed in any cervical segment is found at
 - a. C0C1
 - b. C1C2
 - c. C2C3
 - d. C3C4
6. Occipital numbness suggests a lesion of all of the following roots, EXCEPT for:
 - a. C1
 - b. C2
 - c. C3
 - d. C4
7. Severe unilateral retro-orbital headaches that are more frequent in males and apparently triggered by melatonin and cortisol secretion abnormalities and subsequent circadian rhythm disturbances in the inferior hypothalamus are classified as:
 - a. Cluster headache
 - b. Exertional headache
 - c. Migraine headache
 - d. Tension headache
8. Unilateral, moderate to severe headaches that emerge from vascular changes in the rostral brainstem (including midbrain, pons, and periaqueductal gray matter) and more often in females, throbbing, and accompanied by nausea, vomiting, and photo- and/or phonophobia are classified as:
 - a. Cluster headache
 - b. Exertional headache
 - c. Migraine headache
 - d. Tension headache
9. If a patient looks down during full cervical rotation, one should suspect:
 - a. Lower cervical extension limitation
 - b. Lower cervical flexion limitation
 - c. Upper cervical extension limitation
 - d. Upper cervical flexion limitation
10. Which of the following movements maximizes upper cervical flexion?
 - a. Full cervical extension
 - b. Full cervical flexion
 - c. Full cervical protraction
 - d. Full cervical retraction
11. If your patient's upper cervical symptoms are most provoked with full cervical protraction and (L) sidenodding, then you would suspect which lesion?
 - a. Lesion of the ventral capsule C0C1
 - b. Lesion of the dorsal capsule C0C1
 - c. Lesion of the ventral capsule C1C2
 - d. Lesion of the dorsal capsule C1C2
12. If your patient's (R) upper cervical symptoms are most provoked with full (R) cervical rotation, (R) sidebending, and extension, then you would first suspect which lesion?
 - a. Lesion of the (L) C2C3 uncovertebral joint
 - b. Lesion of the (L) C2C3 zygaophyseal joint
 - c. Lesion of the (R) C2C3 uncovertebral joint
 - d. Lesion of the (R) C2C3 zygaophyseal joint
13. After deciding to test your patient's upper cervical ligaments, you stabilize the patient's C2 segment and attempt to sidenod the patient's head after providing minimal traction to the cranium. Which ligament are you testing?
 - a. Alar ligaments
 - b. Ligamentum flavum
 - c. Tectorial membrane
 - d. Transverse ligament of atlas
14. All of the following symptoms are considered a major component of cervicomedullary syndrome, EXCEPT or:
 - a. Cluster headache
 - b. Exertional headache
 - c. Migraine headache
 - d. Tension headache

- | | |
|---|---|
| <ul style="list-style-type: none"> a. DeKleyn and Nieuwenhuyse tests b. Diplopia c. L'Ermitte's sign d. Scapulohumeral reflex | <ul style="list-style-type: none"> c. C2 DRG entrapment under the epistrophic ligament d. C2 ventral ramus entrapment |
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| <ul style="list-style-type: none"> 15. Movement of the eyes in a caudal direction, while keeping the head directed forward, emphasizes the activity of which muscle group in the cervical spine? <ul style="list-style-type: none"> a. Longus coli b. Rectus capitus c. Semispinalis capitus d. Upper trapezius 16. Your patient complains of occipital lancinating pain and constant unilateral or bilateral suboccipital pain that is provoked with protraction and contralateral rotation. In addition, the patient reports absolute decreased sensation over the occiput and no complaints of pain with Valsalva. Which affliction do you suspect? <ul style="list-style-type: none"> a. C1C2 joint arthropathy b. C2 DRG entrapment between the C1 and C2 dorsal laminar arches | <p>Legend for Review Questions:</p> <ul style="list-style-type: none"> 1. d 2. a 3. b 4. c 5. b 6. d 7. a 8. c 9. c 10. d 11. a 12. d 13. a 14. a 15. a 16. c |
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