Differential Diagnosis of Local Cervical Syndrome versus Cervical Brachial Syndrome

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Cervical pain is a common affliction experienced by most individuals at one time or another in life. This commonality inspired Bland to suggest that "Pain in the neck is such an everyday event that it is often used to describe a situation, certain people, an unpleasant job to be done, or an institution."[1] This commonality presents a great challenge for clinicians, whose task of diagnosis and treatment is complicated by the structural and functional complexities found in the cervical region. Not only does the cervical spine function as a very sensitive sensory organ, but it also demonstrates prevalent three-dimensional coupling behaviors. These physiological and mechanical intricacies lend to clinical consequences that could easily mislead the clinician in diagnosis and treatment of cervical disorders.

Authors have attempted to classify cervical disorders from structural and functional perspectives. Winkel et al clinically categorized cervical disorders in the following fashion, based on the location of the symptoms related to the disorder: (1) Local Cervical Syndrome (LCS); (2) Cervico-Brachial Syndrome (CBS); (3) Cervico-Cephalic Syndrome; and (4) Cervico-Medullary Syndrome.[2] Within each of these categories, various etiologies were observed that might explain the pattern and behavior of the related symptoms.

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One can describe LCS as a disorder that presents with local neck complaints, due to either 1° or 2° disc-related conditions. In a 1° disc-related LCS, the patient suffers due to pain generation from the disc itself, as the disc is a structure with the capacity for generating both proprioceptive and nociceptive afferent information. While the cervical disc segments can produce observable radiographic changes associated with disc protrusion, a LCS can also emerge from an internal disc disruption without any findings on standard MRI or CT, making the diagnosis more challenging for the clinician.

LCS can also be related to 2° disc-related changes. Local cervical symptoms can stem from long-standing changes in the articular structures of the cervical spine that were triggered by previous degradation in the intervertebral disc. Similar to the lumbar spine, pain can emerge from the zygapophyseal joints (ZAJ), as these joints experience significant proportional load bearing that is associated with their more horizontal orientation (coursing approximately 45° ventral cranial—dorsal caudal).[3] These loads are accentuated by the postural muscle tone that is aimed at supporting the head, whose center of mass and line of gravity course anterior through the trunk and produce a perpetual tendency for the head to fall forward. Thus, joint orientation that is coupled with increased compressive loading can lend to ZAJ irritation and pain generation. This tendency can be further compounded by macrotrauma, as witnessed after a whiplash injury. Macrotrauma produces greater involvement of the ZAJ in the cervical spine versus the other levels, due to the same orientation.[4]

Unlike the lumbar spine, symptoms associated with LCS can also stem from irritation to the uncovertebral joints (UVJ), which are only found in the cervical disc segments of the spine (C2C3 to C6C7). These unique joints, which develop more completely in childhood and early adolescence, can become significant pain generators. The UVJ demonstrate significant influence on the control of sidebending and can lead to local cervical symptoms that are especially aggravated through sidebending behaviors.[5]

CBS produces complaints in both the local cervical region and one or both upper extremities. The upper extremity pain indicates there is nerve root irritation, which can stem from either a tension event associated with a protruded or prolapsed intervertebral disc (1° disc-related disorder), or a compression event associated with a 2° disc related disorder.[6] The compression event, or Nerve Root Compression Syndrome (NRCS), develops as result of the segment adapting to degenerative disc changes. Here, the affliction is not so much due to instability (as seen in the lumbar spine), but rather to architectural changes in ZAJ/UVJ. These changes lead to anterior-posterior narrowing of the intervertebral foramen.

Cervico-Cephalic Syndrome produces complaints both locally in the cervical region and the head (including headache, dizziness, and tinnitus). These symptoms, if mechanically activated, can arise from afflictions to the upper cervical segments, including limitation and instability. Patients suffering from this category of affliction can also suffer from Vertebro-Basilar Insufficiency (VBI). This disorder can be purely *mechanical*, but is more commonly related to sympathetic nervous system dysfunction and, therefore, is considered to be *functional* in nature. On the other hand, Cervico-Medullary Syndrome may produce local cervical complaints, but is mainly characterized by spinal cord symptoms associated with cord compression at the cervical spine.

Classification of the Cervical Segments

A clinician's clear understanding of the pathoanatomy and mechanics of the cervical spine can serve as foundation for clarity in differential diagnosis. The first through seventh cervical segments have been classified anatomically, biomechanically, and functionally. Anatomically, the cervical spine has been divided into the disc (C2C3-C6C7) and nondisc (C0C1-C1C2) segments. Biomechanically, C0C1 and C1C2 are collectively labeled as the upper cervical spine, due to their tendency to synkinetically sidebend opposite to kinetic rotation. Conversely, C2C3–C6C7 are labeled as the lower cervical spine, as they synkinetically sidebend in the same direction as the kinetic rotation. Additionally, this lower cervical coupling behavior can also be observed from C7T1 to T3T4, allowing those segments to clinically affect the mechanical behaviors of the segments above.

Functionally, the cervical spine demonstrates many unique features. First, rotational movement of the head produces segmental movement caudally to T3T4. This is also observed with upper extremity elevation, which produces movement of the thoracic segments from T1T2 to T5T6. Second, motion at C2C3 is critical to the entire function of the cervical spine. Motion limits at C2C3 can restrain movement at C0C1 and C1C2, due to the powerful influence of the alar ligaments. Additionally, any motion limits at C2C3 will negatively influence the sensory function of the upper cervical spine, as these functions are directly linked to motion and position of the head.[4]

Osteology

The vertebral bodies of the cervical disc segments are short and wide. There is a ridge, particularly posterolateral, where the UVJ can be found. While the pedicles are very small and run dorsolateral, the laminae are long, extensive, and can be easily palpated.[7] The spinous processes from C3-C6 are bifid, typically off-center in their orientation, and difficult to palpate. Thus, any clinical or radiological interpretation of segmental malpositioning is confounded by this asymmetrical tropism. The spinous process of C2 is particularly large, as it serves as an insertion point for many cervico-occipital muscles. The transverse processes demonstrate 2 tuberculi (anterior and posterior) that serve as levers for muscles inserting upon them. These processes form gutters (or sulci) through which the nerve roots pass. In the lower cervical disc segments the nerve roots are anchored by connective tissue within these gutters, complicating the use of dural testing for the detection of a primary disc affliction (Figure 1).[2]

One witnesses the intervertebral foramen just proximal to the sulcus. The spinal nerves course through neural grooves and then enter the foramen anterior-medial to the articular processes and lateral to the uncinate processes. Because of the unique orientation, the foramena are best observed on an anterior oblique x-ray. As result of the bony walls of the canal, patients rarely present with a pure posterolateral disc protrusion imposing tension load on the spinal nerve (which would result in only arm pain). Rather, arm pain related to a disc lesion will almost always be accompanied by neck pain, by virtue of an irritated posterior longitudinal ligament. When

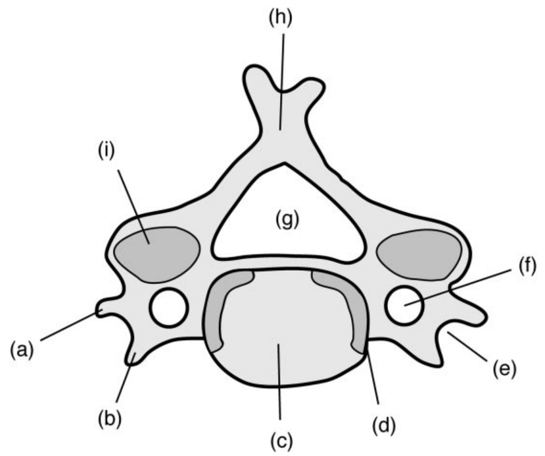

Figure 1. Cervical Bony Segment, Transverse View. (a) Posterior transverse tubercle; (b) Anterior transverse tubercle; (c) Vertebral body; (d) Uncinate process; (e) Transverse process; (f) Transverse foramen; (g) Spinal foramen; (h) Spinous process; (i) Articular facet.

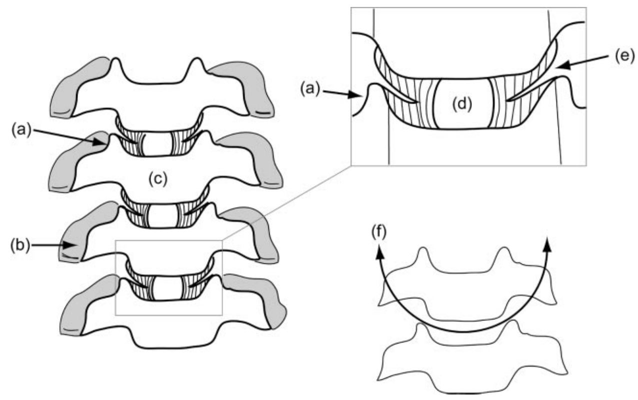

Figure 2. Cervical Disc Segments, Frontal View. (a) Uncinate process; (b) Sulcus within the transverse process; (c) Vertebral body; (d) Nucleus, intervertebral disc; (e) Uncovertebral joint; (f) Bony segments: Cranial convex segment on caudal concave segment.

a patient presents with isolated arm pain, this is not typically associated with a primary disc affliction, but rather with degenerative changes in the uncinate and or zygapophyseal articular processes. These degenerative changes result in narrowing of the intervertebral foramen, which is especially observed in a dorsal-ventral direction versus the cranial-caudal direction that is frequently witnessed in the lumbar spine.[8] The consequences of compression can be clinically reduced by dorsoventral mobilization to the caudal bony segment, as this increases the a-p diameter of the canal.[2]

Ebraheim et al reported that the antero-posterior diameter of the intervertebral foramina was smaller for the C4, C5, and C6 root levels compared with that associated with the C3 or C7 levels. Additionally, the length of nerve root between the lateral border of the dural tube and the medial border of the vertebral artery gradually increased from C3 (3.3 +/− 1.1 mm) to C7 (8.1 +/− 2.1 mm). These investigators reported that a combination of higher uncinate process, smaller antero-posterior diameter of intervertebral foramina, and longer course of nerve roots in close proximity of the UVJ at the C4 to C6 root levels may explain the predilection of nerve root compression by uncovertebral osteophytes at these levels. Whereas foraminotomy may decrease root compression, it may also compromise the stability of the C-spine, due to uncinate process loss.[7]

Found in the lateral zone of the cervical disc segment, the UVJ are comprised of the uncinate process observed on the caudal vertebra and an indentation on the caudal aspect of the cranial vertebra (see Figure 2). This joint, which maintains the synovial compartment, is intimate with the intervertebral disc as it forms the disc's posterior-lateral border.[9] The uncinate processes are not present at birth, but instead develop within the first 12 years as result of weightbearing imposed by the head. The uncinate processes can first be appreciated as they begin to develop at approximately 7 years of age. By 33-years-old, individuals present with well-developed uncinate processes and accompanying UVJ.[10]

The uncinate process, which is curved in shape when observed from above, demonstrates 3 regions: anterior, posterior, and foraminal. This architecture allows the UVJ to enhance the stability of the cervical spine.[11,12] The uncinate processes act as a "rail" to guide translatory behavior of the cranial vertebra on the caudal vertebra of a motion segment. As a result, this feature contributes to the regulation of extension and lateral bending in the cervical disc segments. Penning suggests that the most prominent (tallest) uncinate process is found at C2C3 (see Figure 3), by virtue of the increased translatory movement demonstrated at this segment during flexion/extension.[13] This stabilizing feature develops and changes with age adjacent to changes in the uncinate processes.[14–16]

The uncinate processes contribute to the reduced incidence of isolated arm pain associated with a primary disc lesion. The uncinate processes act as a barrier to posterior lateral disc protrusion or prolapse, reducing the disc's access to the root. Any disc affliction associated with arm pain will typically access the root medial to the uncinate process, irritate the posterior longitudinal ligament, and create accompanying cervical pain. Furthermore, if the patient presents with isolated arm pain that is referred in nature, the clinician must suspect a huge prolapse extending over the uncinate process, a neurinoma, Thoracic Outlet Syndrome (TOS), foraminal stenosis, or serious pathology.

The ZAJ are formed by the articular processes of two adjacent cervical segments. These processes are located

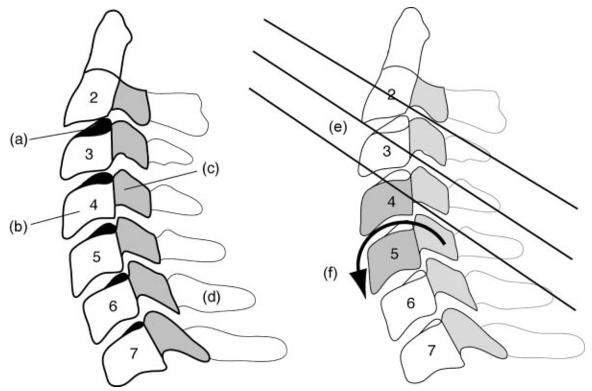

Figure 3. Cervical Disc Segments, Sagittal View. (a) Uncinate process, C3; (b) Vertebral body, C4; (c) Articular pillar, C4; (d) Spinous process, C6; (e) Orientation of zygapophyseal joints, pointing towards the orbit; (f) Bony segments: Cranial concabe segment on caudal convex segment.

just posterior to the transverse processes and are oriented from ventral-cranial to dorsal-caudal at 45° in relation to the vertebral end plate. In general, the articular surfaces are oriented towards the orbita with the neck in the zero position (see Figure 3). The presence of the UVJ and the frontally-inclined position of the ZAJ facets are among the more important factors causing a strong coupling between kinetic (voluntary, active) and synkinetic (involuntary, passive) motions.[12]

The Intervertebral Disc

The intervertebral disc presents a disc to vertebral body height ratio of 1:4 compared to a 1:3 ratio observed in the lumbar spine. Whereas the lumbar disc determines the mechanics of those spinal motion segments, the cervical disc simply interacts with the influences of both the UVJ and ZAJ. The cervical disc can develop both mechanical and chemical phenomena similar to Internal Disc Disruption (IDD) found in the lumbar spine, thus, rendering it as a potent pain generator. However, the form and function of the adult cervical intervertebral disc are considerably different than that observed in the lumbar spine.[17,18]

The annulus fibrosus has a series of anular lamella sheets with collagen fibers oriented at 65° in relation to the vertical axis in each layer.[19,20] The cervical disc nucleus is closer to the center of the disc, and, therefore, the annulus does not demonstrate the same structural asymmetry as in the lumbar spine. The key distinctive feature of the cervical disc is the lateral clefts, or "fissures," associated with the UVJ (see Figure 2). The annulus extends beyond the vertebral body and up the slope of the uncinate process. Along this slope the fissures form over time and ultimately communicate with the cavity of the UVJ, becoming a component of the UVJ as they fill with synovial fluid. The fissures develop in the anular lamellae, and ultimately the adjacent collagenous fibers reorient to course parallel with the fissure.[15,21,22] Along with the uncinate processes, these clefts are most prominent from C2C3 to C4C5 and less prominent from C5C6 to C7T1. This prominence is a function of greater translation in the upper cervical disc segments due to more caudal location of the instantaneous axis of rotation for flexion/extension. Greater translation merits more prominent uncinate processes for stability. More prominent uncinate processes induce a greater deformation in the outer anulus, leading to clefts in the disc. Additionally, greater uncinate processes reduce pure axial rotation in the cervical disc segments, requiring coupled synkinetic sidebending to achieve adequate osteokinematic rotation.

The cervical disc segments ultimately transform into sellar joints.[14,15,25] This transformation is a function of several factors. First, the functional architecture of the cervical motion segment contributes to this transformation. From a sagittal view, the cranial segment is concave on a convex caudal segment (see Figure 3). From a frontal view, however, the cranial vertebral body is convex on the concave caudal body (especially in the posterior aspect of the intervertebral segment due to uncinate processes, see Figure 2). Secondly, the fissures in the disc associated with the UVJ ultimately communicate, reducing the constraining contribution from the annulus and rendering the disc to sliding and gliding. The splitting of the disc is greater from C2C3 through C4C5, due to the greater translatory motion in those segments. The important feature to remember is that these changes are physiological and not pathological in nature.

These physiological adaptations increase linearly with age. Investigators have demonstrated that 86% and 89% of discs of asymptomatic men and women over 60 years of age, respectively, will demonstrate some degree of this degeneration. However, the findings of degeneration may not always be clinically relevant.[26] This may be explained by the characteristics of the degenerative event, where selected segments of the cervical spine demonstrate degenerative patterns that are different from the other spinal levels. Whereas the lumbar spine degenerates from inside to outside, the upper cervical disc segments (C2C3 to C4C5) degenerate from outside to inside. This pattern contributes to a reduced incidence of 1° disc afflictions in those levels. On the other hand, the C5C6 to C7T1 segments degenerate from inside to outside (likened to lumbar spine). Because of this particular pattern, patients can experience increased incidence of

1° disc afflictions in these lower cervical levels. Furthermore, the anterior intervertebral disc is the last region of the disc to degenerate. This is due to the proximity of the anterior disc to the oblique IAR (Instantaneous Axis of Rotation) for SB/rotation. Here less rotation occurs, rendering this area to greater stability and reduced degeneration.[13] However, if the anterior disc indeed degenerates, then the patient may present with pain during swallowing or with palpation to thyroid/crycoid cartilage.

Capsuloligamentous Structures

One can witness synovial-lined capsular structures surrounding both the ZAJ and UVJ. Mercer and Bogduk reported the potential for intraarticular inclusions from the capsule into the cervical synovial joints.[17] The most common of these inclusions are fibro-adipose meniscoid slips. Less common are thickenings surrounding the entire joint perimeter, or capsular rims, whereas projecting fat pads are least common. These projections have the potential for obstructing movement of the cervical motion segments by becoming lodged between articular surfaces. The patients who possess these intrusions typically report variable, unpredictable catching pain that is local, unilateral, and transient.

The capsule of the ZAJ allows for a great deal of segmental motion, both in rotary and translatory directions (up to 9 mm at a given level). Most stress is imposed on the capsular structures during rotation that is performed in a three-dimensional fashion. Pure sagittal motion cannot produce enough motion to stress these capsules. On the other hand, the capsule of the UVJ is best stressed with three-dimensional sidebending motion. Thus, if the greatest pain is produced with three-dimensional rotation, the clinician should suspect ZAJ as the primary pain generator. Conversely, one should suspect UVJ involvement if the greatest pain is produced with sidebending. Finally, the clinician should suspect the disc as the primary pain generator if the greatest pain is produced with flexion and or extension.[4]

The posterior longitudinal ligament (PLL) is very prominent and more developed versus the same ligament found in the lumbar spine. Additionally, this ligament provides a greater biomechanical contribution to motion restraint in cervical spine versus lumbar spine. The PLL is attached to tectorial membrane and demonstrates a very tight connection to the disc. This ligament possesses a rich population of nociceptive endings, rendering it as a potent pain generator in the context of afflictions to the posterior and posterior-lateral intervertebral disc. Finally, the PLL is a possible site for ossification and subsequent central stenotic clinical events.[27]

The anterior longitudinal ligament (ALL) demonstrates multiple layers and configurations.[17] The ALL is connected to anterior atlanto-occipital and atlanto-axial membranes in upper cervical spine, contributing to mechanical continuity between the head and the cervical spine. This ligament is also a probable site for ossification with increased incidence accompanying diabetes.[27]

The nuchal ligament is a long connective tissue structure spanning the entire length of the dorsal cervical spine. This system demonstrates 2 primary layers: the funicular layer, coursing cranial-caudal from the tips of the spinous processes of C5, C6, and C7 up to the occiput;[28] and the lamellar layer, coursing in an anterior-posterior direction from the lamellar layer to the spinous processes of C2, C3, & C4.[29] This ligament possesses insertions from the trapezius, splenius capitus, serratus posterior superior, and rhomboid major, suggesting a dynamic function of the ligament for assisting control of head position and movement. This ligament may also contribute to a paradoxical extension of the C0C1 motion segment during full cervical flexion, explaining why individuals are capable of producing greater C0C1 flexion during retraction versus full cervical flexion.[30]

Neural Structures

When observing the neural structures of the cervical spine, one must consider both the neural networks linked to the structures of the cervical spine as well as the nerve roots exiting the cervical spine. The posterior aspect of the vertebral motion segment is innervated with 2 different layers of the nerve fibers, a superficial and deep network. These layers, especially addressing the posterior longitudinal ligament and posterior annulus, are responsible for supplying these areas with nociceptive endings.[31] The deeper layer is monosegmental, lending to more focal symptoms. Conversely, the superficial layer is polysegmentally innervated by the sinuvertebral nerve, lending to more diffused nonradicular pain patterns when irritated.[32] This may explain why deeper disc afflictions present with more localizing symptoms versus the more diffused nonradicular symptoms associated with afflictions that reach the outside of the cervical disc and posterior longitudinal ligament. The medial branch of the dorsal primary ramus innervates the ZAJ. Once again, the joints associated with each segmental level are polysegmentally innervated. This innervation pattern may contribute to the overlapping nonradicular pain reference zones associated with afflictions to the ZAJ.[33]

Ventral rami of C5 to T1 (occasionally as high as C4 and as low as T2) join to create the brachial plexus. Each root is comprised of several ventral and dorsal rootlets that emerge from the spinal cord. Each of these rootlets leaves the spinal cord and traverses to the intervertebral foramen at a different angle. The more cranial rootlets course in a more dorsal lateral oblique direction, versus the more horizontal orientation of the more caudal rootlets of a given segment.[34] This relationship is especially noted at the C5 root level, where any lateral deviation of the spinal cord may increase the tension loading in the caudal rootlets at C5. This may explain why a posterior paramedian primary disc affliction at C6C7 may result in C5 radiculopathy. As the disc prolapse shoves the spinal cord aside, the lateral movement loads the C5 root resulting in C5 radiculopathy. Thus, clinicians cannot trust the presence of a C5 radiculopathy to convince them of a C4C5 disc affliction.

The C7 root maintains several unique characteristics as well.[35] First, it is relatively larger than the other roots in the cervical spine. Second, the root canal may be smaller in diameter. Third, the root courses closer to the facet pillar than other roots. Finally, it courses more lateral in the transverse plane. These features lend the C7 root to frequent compression when the canal is compromised by degeneration associated with secondary disc-related disorders.

Detecting a primary tension sign during dural tests, including the slump test and straight leg raise supports the diagnosis of a primary disc affliction of the lumbar spine.[36] A similar testing paradigm would serve the clinician for diagnosis of cervical primary disc-related disorders. However, the roots C5, C6, and C7 are fixated on the transverse processes through inter-transverse ligamentous anchors. This relationship makes it difficult for clinicians who wish to use a neural tension test to identify 1° tension signs in those cervical levels that are prone to disc afflictions. Thus, alternative testing must be performed in order to tension load the nerve structures (to be discussed later).

One may observe anatomical variance in the position of the dorsal root ganglion within the intervertebral foramen.[37] The ganglion may be found either outside the bony root window or within it. When positioned within, the ganglion is more predisposed to bending or kinking around the pedicle during movements of the upper extremity or cervical spine. Lu and Ebraheim observed this phenomenon in 48% of C6 and 27% of C7 root levels.[38] This predisposition is enhanced when the disc segment has degenerated, as the size of the intervertebral foramen will likely be narrowed. Similar to the lumbar spine, the dorsal root ganglion is sensitive to pressure and can result in immediate referred pain. Antithetically, the upper extremity symptoms that start several hours or days after the onset of cervical pain or exacerbating circumstances are likely due to chemical irritation of the root itself, as roots are only mechano-sensitive when chemically irritated. Thus, it obligates the clinician to ask the patient about the time involved in the onset or provocation of his or her upper extremity pain, as that timing will indicate whether the root affliction is mechanical or chemical in nature. This information will guide the clinician in selecting a management strategy, as mechanical root disorders respond more effectively to mechanical treatments including posterior-anterior mobilization.[23] Conversely, the effects of mechanical therapeutic interventions may be less effective for chemical root irritations and these roots may respond better to pharmacological interventions that are followed up by root mobilization, which can prevent or reduce negative consequences associated with chemically-activated adhesions.

When discussing clinically significant neurological structures about the cervical spine, one must also consider selected cranial nerves. The spinal accessory nerve (SAN) is the only cranial nerve without nuclei in the brain. Rather, the nuclei are located in the brainstem and spinal cord coursing from C1 to C4 (occasionally, as far caudal as C6).[39, 40] The SAN is the only cranial nerve that lacks a sensory component and is exclusively motoric in function. These nuclei innervate the trapezius muscle, which frequently demonstrates increased tonicity in patients with chronic cervical symptoms. Although this increased muscle activity has been historically interpreted as a primary muscle lesion associated with a cervical condition, it may be more related to interneuronal pool firing in the cervical spine. Incoming nocisensoric impulses from C2 to C6 can be relayed ventrally to the motor neurons associated with the SAN via interneurons. This increase of interneuronal activity may escalate the motor output of the SAN, elevating the resting tone in the trapezius. Thus, pain in the midcervical region can trigger increased muscle tone of the trapezius muscles. Furthermore, clinicians must be careful with the use of trapezius stretching for patients with neck problems, as this may not address the source of increased trapezial activity, but rather simply aggravate the underlying cervical condition.[4]

Branches of trigeminal nerve (opthalmic, maxillary, and mandibular) are entirely sensory in nature. As with the SAN, the trigeminal nerve possesses nuclei in the spinal cord from C1 to C4 (possibly C5). In instances of chronic pain in cervical spine, different adaptive processes begin to take place in the spinal cord. Chemical substances are released in the spinal cord and new interneuronal connections are constructed as the interneurons reorganize. Increased interneuron activity can lead to cervicotrigeminal relay, where a patient experiences chronic headache and/or facial pain that is associated with various different afferent inputs from local cervical afflictions at mid cervical segments. Whereas cervical headache is very common (ie, Cervico-Cephalic Syndrome), it may be due not only to compression of the greater occipital nerve, but also to irritation of the trigeminal nerve nuclei in the upper spinal cord.[31,41,42] Thus, the clinician must consider involvement of the cervical disc segments when evaluating a patient's Cervico-Cephalic Syndrome.

Differential Diagnosis of Afflictions in Cervical Disc Segments

Cervical Postural Syndrome. Cervical Postural Syndrome (CPS), although not belonging to any other category of syndromes, is closely related to the onset of primary disc-related disorders. Onset of CPS is linked to imbalance of muscle activity within the cervico-thoracic spine. The muscles of the cervical spine encounter overload when the head is positioned too far forward, increasing the flexion movement at the lower cervical segments and compensatory muscular response in the dorsal musculature. This "tired neck" is frequently experienced by individuals involved in activities requiring static forward bent posturing such as drafting technicians, computer operators, and students. This condition is seen more frequently in women vs. men (3:1) and is perpetuated by bifocal use and prolonged driving. Clinically, individuals (from 6 to 40 years of age) will complain of a tired feeling in their necks, stiffness, and potential symptoms in the TMJ. They may present with tenderness, but frequently present with a negative functional examination, where movement and force do not provoke the symptoms. These patients are best managed through postural reeducation, isometrics for strengthening, and local infiltration to muscle trigger-point regions.[2]

Local Cervical Syndrome. One can describe LCS as a disorder that presents with complaints that are local and referred in a nonradicular distribution. Pain related to LCS is typically aching in nature and extends from C2C3 down to midthoracic levels. Dwyer et al demonstrated that pain associated with each segmental level overlaps, potentially extending far below the level of origin (see Figure 4).[33] Due to these nonradicular referral patterns, a clinician must consider a cervical origin for patient's pain that extends into the interscapular region.

For LCS that is 1° disc-related, the patient suffers due to pain generation from the disc itself, as the disc is a structure with the capacity for generating both proprioceptive and nociceptive afferent information. The most frequent disc levels to present as LCS are C5C6 and C6C7, due to the previously mentioned inadequate uncinate processes, as well as the characteristic internal degenerative changes. Patients may experience pain with swallowing (due to instantaneous cervical flexion during a swallow) and motions that impose either a tension or compression load on the disc. Pain associated with this local cervical disorder, however, is not limited to the cervical spine and cervico-thoracic junction. Shellhas et al were able to provoke symptoms in seemingly dubious locations around the head, neck, and shoulder girdle regions (see Table 1).[43] Thus, the cervical intervertebral disc can be responsible for pain in otherwise unsuspected areas, such as the cranium, jaw region, throat, anterior chest, and scapula.

LCS associated with a 1° disc affliction can be classified into 1 of 2 different subcategories: (1) acute torticollis or (2) disc protrusion. Acute torticollis is seen most frequently in children and young adults as result of sustained poor posturing (eg, during sleeping) in a sidebent and rotated position. While individuals normally change position frequently at night, sleeping pills, alcohol, or extreme fatigue will lead a person to maintain a single position throughout the entire sleep cycle. During this time, the disc's nuclear material can migrate into the lateral fissure of the UVJ on the contralateral side to the sidebent position. After waking, patients develop extreme stiffness and local pain in the cervical spine. They present with a head laterally shifted away from the pain. Motion is limited in a noncapsular pattern of extension, ipsilateral sidebending, and ipsilateral rotation. This

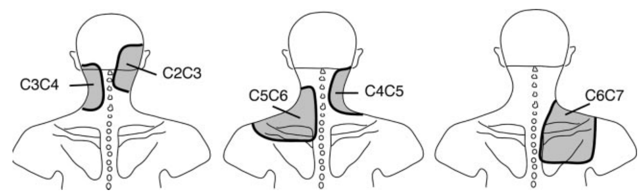

Figure 4. Nonradicular pain reference zones for the cervical zygapophyseal joints. Adapted from Dwyer A, Aprill C, Bogduk N. Cervical zygapophyseal joint pain patterns I: A study in normal volunteers. Spine. 1990; 15:453-461.

Table 1. Discogenic pain reference patterns of the cervical disc segments.

	Involved Disc Level			
Region of Pain	C3C4	C4C5	C5C6	C6C7
Mastoid	*	*		
Temple	*			
Jaw	*			
TMJ	*			
Parietal Cranium	*	*		
Occipital Cranium	*	*	*	
Craniovertebral Junction	*	*	*	
Neck	*	*	*	*
Throat	*	*	*	
Upper Back	*		*	*
Trapezius	*	*	*	*
Top of Shoulder	*	*	*	*
Upper Extremity	*	*	*	*
Anterior Chest		*	*	*
Scapula				*

Adapted from Shellhas KP, Smith MD, Gundry CR, Pollei SR. Cervical discogenic pain. Prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers/. Spine. 1996;21:300–312.

commonly experienced "crick in the neck" can be interpreted as a disc affliction as evidenced by difficulty in sidebending towards the side of pain. This affliction is regularly self-limiting, but recovery can be encouraged through soft tissue mobilization, axial separation in the deviated position with correction in rotation and sidebending, and short-term use of a firm cervical collar.[2]

Patients with a LCS that is associated with protrusion present with a characteristic profile. These patients are more commonly between the ages of 30 and 45 years with episodic histories of acute torticollis.[2] Pain is again distributed in previously mentioned nonradicular areas and is exacerbated with forces that stress the disc's annulus with either compression or tension loads. Examination of the MRI may or may not reveal the protrusive event. Patients typically demonstrate the most pain during movements in the sagittal plane (flexion and or extension), due to increased mechanical loading[5] and intradiscal pressure[44] that can be especially demonstrated in the lower cervical disc segments. Additionally, the pain is also provoked during sidebending and or rotation towards the painful side. Because the nonradicular pain can arise from the posterior annulus, posterior longitudinal ligament, and dura of the root, tension loading in the root may provoke the symptoms. Motor, sensory, and reflexive tests are typically negative. While a standard neural tension test may not provoke these symptoms (due to the anchoring of the C5, C6, and C7 roots in the sulcus of the transverse processes), a clinician can nevertheless increase root tension loading. This accomplished by first flexing the neck forward, followed by retraction of both scapulae (Disc Test 1, see Appendix A). Here, the retraction event can increase the tension loading of the T1 root level, thus, loading the cervical spinal dura and more cranial roots by virtue of the short distances any rootlet courses from the spinal cord to the root anchors. This increased tension will provoke the patient's local cervical symptoms, whereas the same symptoms would not be aggravated in the case of ZAJ or UVJ involvement. This "back door" technique may be sensitive and specific for the detection of primary disc afflictions that would otherwise be confusing and vague.[4]

LCS can also be related to 2° disc-related disorders. Local cervical symptoms can stem from long-standing changes in the disc and articular structures of the cervical spine that were triggered by previous intradiscal degradation. This family of disorders can include: (1) annular tears with IDD; (2) UVJ afflictions; and/or (3) ZAJ afflictions. Patients with LCS that is associated with internal disc disruption present with vague, and often confusing, symptoms. The condition is regularly associated with chronic irritation to the disc and is related to a chemical cascade that was initiated earlier in the disc with apparently innocuous trauma. With trauma to the nuclear envelope of the disc, chemical factors enter the disc, triggering the production of Metallo-Matrix Protease Inhibitors (MMPI's) that initiate deterioration of the internal environment of the disc anulus.[45] This process is accentuated by collagenase release and is accompanied by autoimmune activities, including the release of bradykinin and serotonin, the aggregation of T-lymphocytes, and the production of phospholipase A2 (PLA2), which facilitates production of prostaglandins and leukotrienes.[45,46] These responses sensitize silent nociceptors in the outer sanctum of the disc, leading to a painful response even under loading and movement conditions that were previously considered normal.

The diagnosis of cervical IDD is difficult at best. Patients present with local and nonradicular symptoms that are most easily provoked by movements in the sagittal plane (flexion and or extension) that produce tension and or compression in the discal structures. Patients report stiffness in the early morning and increased symptoms by the end of the day. Pain is located in the midline and either unilateral or bilateral paramedian regions of the cervical spine. The pain is aching in nature and is typically long-standing. Patients may or may not present with motion limits and the previously mentioned dural tension test will be negative, along with negative motor and or sensory findings. Conclusively, IDD may appear to be very similar to LCS associated with the ZAJ or UVJ. Thus, the IDD diagnosis should be made in exclusion of joint involvement from the local examination findings that will be discussed next.

While the cervical disc segments can produce observable radiographic changes associated with disc protrusion, internal disc disruption can be absent of any findings on standard MRI or CT, making the diagnosis more challenging for the clinician. However, patients suffering from IDD frequently present with a loss of cervical lordosis, as witnessed on sagittal imaging. Reduced lordosis is associated with accelerated discosis, whereby the disc height diminishes, the cranial vertebral body of a motion segment descends and contacts the posterior curve of the uncinate process, and further disc height loss forces the cranial bony segment to "tip" forward (see Figure 5). This angulation is typically accompanied by a posterior shift of the disc's nucleus, which progressively disintegrates with further changes.[23] These changes can be useful to the diagnostician, as a reduced cervical lordosis on the imaging can suggest IDD and discosis. These patients will present with a non-capsular pattern demonstrating a disproportional loss of extension due to pain, while rotation and sidebending are often found to be within normal limits. Symptoms and the loss of lordosis can be reduced through posterior-anterior mobilization and specific home exercises.[23]

Unlike the lumbar spine, symptoms associated with LCS can also stem from irritation to the UVJ, which are only found in the cervical disc segments of the spine (C2C3 to C6C7). These unique joints, which develop more completely in childhood and early adolescence, can become significant pain generators. The UVJ demonstrate significant influence on the control of sidebending and can lead to local cervical symptoms that are especially aggravated through sidebending behaviors.[5,11] Conversely, sidebending is typically not the greatest pain-producing motion in a cervical IDD, due to an actual decrease in intradiscal pressure at end range of the motion.[44] Neither is this motion most provocative with ZAJ afflictions, due to the apparent laxity in the capsule. Onan et al observed 28° of isolated segmental sidebending allowed by the ZAJ capsule, which again far exceeds the physiological motion demonstrated by any cervical segments.[47] However, sidebending can induce increased pressure and irritation to the posterior and foraminal portions of the uncinate process on the side ipsilateral to the direction of sidebending, as well as increased tension on the UVJ capsule on the contralateral side.[11]

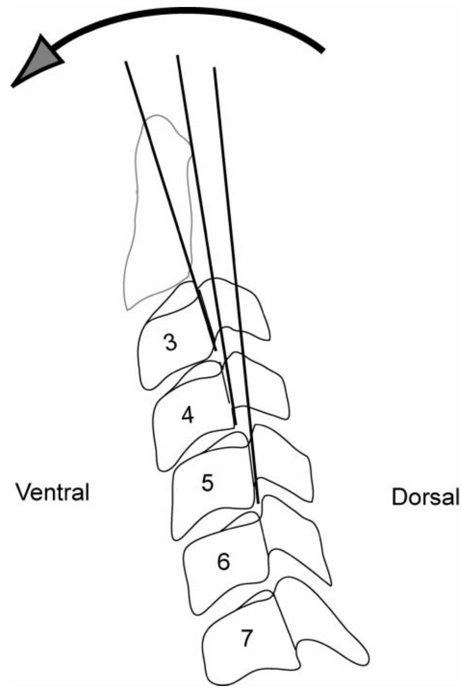

Figure 5. Anterior kinking of cervical disc segments associated with accelerated cervical disc degradation.

Similar to the lumbar spine, pain can emerge from the ZAJ, as these joints experience significant proportional load bearing that is associated with their more horizontal orientation (coursing approximately 45° ventral cranial—dorsal caudal).[3] These loads are accentuated by the postural muscle tone that is aimed at supporting the head, whose center of mass and line of gravity course anterior through the trunk and produce a perpetual tendency for the head to fall forward. Thus, joint orientation that is coupled with increased compressive loading can lend to ZAJ irritation and pain generation. This tendency can be further compounded by macrotrauma, as witnessed after a whiplash injury. Macrotrauma produces greater involvement of the ZAJ in the cervical spine versus the other levels due to the same orientation issues.

LCS associated with ZAJ irritation must be differentially diagnosed from internal disc disruption and/or UVJ afflictions. Unlike a cervical disc affliction, the greatest pain provocation associated with ZAJ synovial irritation is not witnessed during flexion and or extension. This is related to the apparent laxity of the joint capsule during those movements. Onan et al observed that isolated facet joints allowed up to 19° of segmental flexion and 14° of segmental extension, which far exceeds the movements that physiologically occur in the sagittal plane at any give cervical motion segment.[47] This suggests that flexion and or extension are not sufficient to tension load the capsule of the joints. Rather, rotation appears to maximally tension load the ZAJ capsule, suggesting the primary role of the ZAJ in controlling rotation. Furthermore, while extension does increase the loading on the ZAJ articular surfaces, the same symptoms can be provoked with contralateral rotation and ipsilateral sidebending (by virtue of the loading in the contralateral ZAJ with these motions). This behavior allows for differential diagnosis from a cervical IDD, which will also provoke greatest pain during extension but not during the previously mentioned three-dimensional motion.[5]

Therefore, a clinician must differentiate between 5 different local conditions associated with 2° disc-related disorders: (1) internal disc disruption and discosis; (2) synovitis of the UVJ; (3) arthropathy of the UVJ; (4) synovitis of the ZAJ; and (5) arthropathy of the ZAJ. Although this differential diagnosis is not simple at face value, it is necessary to guide the clinician to a more expedient management strategy. The differential diagnosis can be determined through a brief, but specific, three-dimensional provocative examination (see Appendix B). The examination is divided into 5 different test categories: (1) active Sagittal motions (Flexion/Extension); (2) active & passive rotation; (3) active & passive sidebending; (4) active & passive three-dimensional rotation; and (5) active & passive three-dimensional sidebending. Passive tests are only conducted if the patient's hesitance does not allow the clinician to provoke the symptoms through active testing, and many times the active tests are sufficient for effective differential provocation.

The first motions performed in the examination are flexion and extension. If the patient suffers from disc involvement, these sagittal motions will induce the greatest provocation, versus planar and or three-dimensional movements in the directions of sidebending or rotation (Figure 6). One can note that this provocation can be greatest with flexion, extension or retraction. These provocation patterns are related to tension and or compression events in the disc, addressing specific structures in an age-specific fashion. The second set of motions in the exam (frontal plane motions) involves sidebending. While the clinician can begin to suspect uncovertebral involvement when planar sidebending is most provocative, this diagnosis can be strengthened with three-dimensional movements. The three-dimensional sidebending movements produce either a coupled or combined motion in the cervical disc segments. Coupled motion is one where a synkinetic motion predictably occurs in response to a kinetic motion, allowing maximal motion in the kinetic

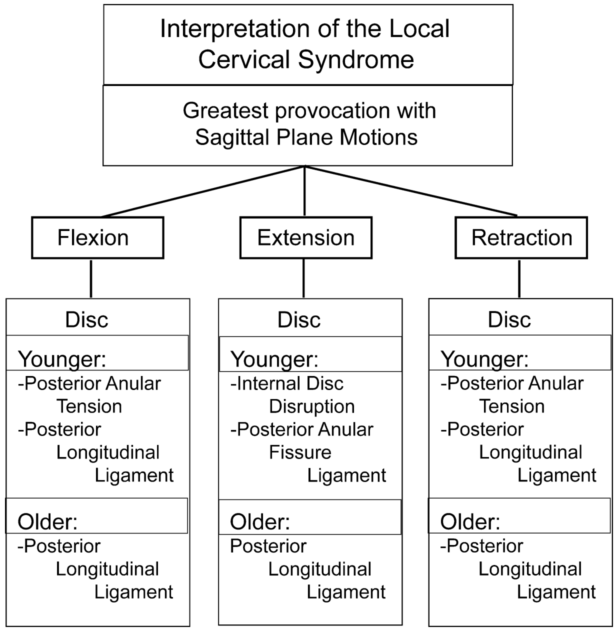

Figure 6. Interpretation of Local Cervical Syndrome: Greatest Pain Provocation in Sagittal Plane Motions.

direction and the capsular structures to reach their maximum tension load. The coupled motions of the cervical spine are sidebending and ipsilateral rotation. Combined motions, on the other hand, limit the kinetic motion by adding a second motion that contradicts the normal synkinetic behavior of the segment. This reflects the articular surfaces getting in the way of the motion and locking the segment. For the cervical spine, combined motions are sidebending and contralateral rotation.[2]

The first three-dimensional movement is performed by initiating sidebending, accompanied by rotation in 2 different directions. After the sidebending is initiated, the patient then rotates to the ipsilateral side, producing a coupled motion. Here, the capsule and synovium of the UVJ are maximally stressed on both the ipsilateral and contralateral sides to the direction of sidebending. If these tests maximize provocation, then the clinician should suspect synovitis of the UVJ (Figure 7). Next, the sidebending is accompanied by contralateral rotation, where the articular surfaces of the UVJ are compressed on the side ipsilateral to the direction of sidebending. If this test is most provocative, then one may strongly suspect an UVJ arthropathy as the principal pain generator (Figure 8). Conversely, if the same test provokes most pain on the contralateral side of sidebending, then the clinician can be more suspicious of an IDD, as the UVJ on that side is unloaded.

If the patient suffers from ZAJ involvement, the pain produced during planar and or three-dimensional rota-

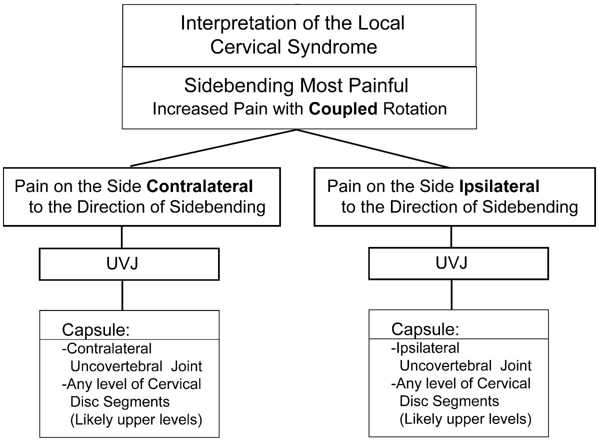

Figure 7. Interpretation of Local Cervical Syndrome: Greatest Pain Provocation in Sidebending and Coupled Rotation Movements.

tion will be greatest, as the ZAJ are most responsible for controlling rotation of the cervical disc segments. Thus, the third set of motions in the exam (transverse plane motions) involves rotation. First, the patient rotates and then ipsilaterally sidebends to produce a coupled motion. Flexion and extension are each superimposed onto this coupled motion. These motions are added because rotation produces osteokinematic extension from C0C1 to C3C4 and extension from C4C5 to C7T1. Additionally, rotation produces arthrokinematic extension on the ipsilateral ZAJ and flexion on the contralateral joints.

Diagnostically, if a combination of rotation, ipsilateral sidebending, and extension is the most provocative movement in the upper cervical disc segments on the same side as rotation, then the clinician should suspect ZAJ synovitis at C2C3 or C3C4. This is because this movement maximally extends the upper ZAJ and maximally stresses the capsule, along with the synovial lining. However, if pain is produced those same segments on the opposite side to rotation, then the clinician should suspect IDD. Conversely, if rotation, ipsilateral sidebending, and flexion produce the most pain in the lower cervical disc segments on the opposite side to rotation, then the clinician should again suspect ZAJ synovitis in that region. This is because the contralateral lower ZAJ is maximally flexed and the capsule is maximally stressed on that side. Furthermore, if pain is produced in those same segments on the same side as rotation, then the clinician should suspect IDD at those levels (Figure 9). Finally, the patient performs a combined motion of rotation and contralaterally sidebending. If the greatest pain is produced during this movement on the same side as the sidebending, then the clinician should suspect ZAJ arthropathy, due to the compression of the joint surfaces during combined motion (Figure 10).

After the suspected structure of involvement has been identified (Disc, UVJ or ZAJ), the clinician can use a mobility test to propose the segmental level of involvement. This test is performed by positioning the patient in cervical sidebending to the corresponding segmental level (determined by palpating the closure of the ZAJ) and providing overpressure in a direction contralateral to the sidebending. The segment that produces the greatest local pain with brief overpressure would be the segment that the clinician could suspect as the pain generator.[2] Although this test outcome may be clouded by

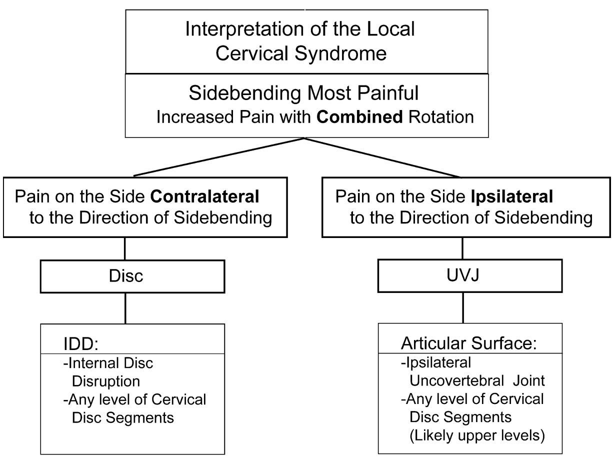

Figure 8. Interpretation of Local Cervical Syndrome: Greatest Pain Provocation in Sidebending and Combined Rotation Movements.

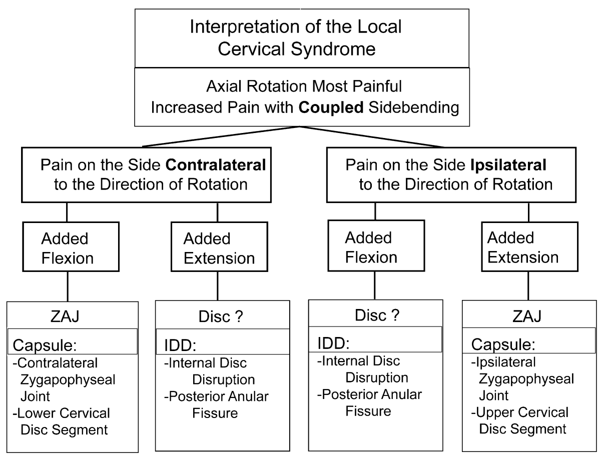

Figure 9. Interpretation of Local Cervical Syndrome: Greatest Pain Provocation in Rotation and Coupled Sidebending Movements.

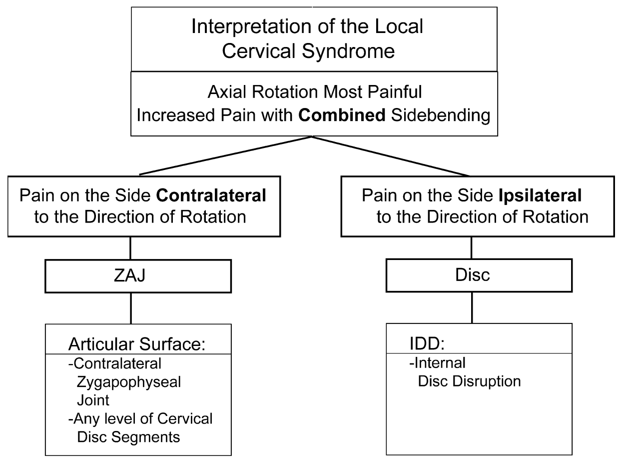

Figure 10. Interpretation of Local Cervical Syndrome: Greatest Pain Provocation in Rotation and Combined Sidebending Movements.

referred tenderness, it provides a starting point for the clinician in administering appropriate treatments.

Cervical Brachial Syndrome

One can describe Cervical Brachial Syndrome (CBS) as a disorder that presents with complaints in both the local neck and upper extremity, due to either 1° or 2° disc-related conditions. The neck pain associated with CBS is typically aching in nature and extends from C2C3 down to midthoracic levels, as observed with LCS. These symptoms in the neck are related to the same processes previously described and can be typically provoked with similar testing. The arm pain associated with CBS is related to either irritation of the dorsal root ganglion or the root itself, and is related, for the most part, to the size of the spinal canal, intervertebral foramen, and or sulcus within the transverse process.[2] The arm pain associated with dorsal root ganglion irritation is sharp and immediate upon mechanical stimulus, as a dorsal root ganglion is mechano-sensitive.[48] The pain associated with root compression demonstrates a slow onset and is aching in nature. This root irritation is the consequence of vascular compromise and an inflammatory cascade that sensitizes the nerve root. Once chemically sensitized, the cervical root then becomes mechano-sensitive, as described for the lumbar spine.[45]

Upper extremity pain that is associated with a cervical disorder is typically accompanied by cervical pain. When the patient presents with isolated upper extremity pain, the clinician should suspect other pathologies, such as disorders of the shoulder, TOS, neurinoma, or pancoast tumor.[2,6] Individuals with TOS commonly present with distal upper extremity pain in a nonradicular distribution associated with the lower trunk and medial cord of the brachial plexus (C8-T1). Their symptoms are provoked with upper extremity elevation and use versus a release phenomenon causing sleep disturbances in the early hours of the morning. Neurinoma produces pain and sensory changes that begin distal and radiate proximally. Symptoms associated with a pancoast tumor can be elusive, but many times can be accompanied by other organic signs such as malaise, lethargia, dry cough, and weight loss. Additionally, the upper extremity symptoms are not easily provoked through physical examination and may be more profound at night.

A clinician should differentiate a cervical patient's upper extremity pain as related to 1° versus 2° disc-related changes. Upper extremity symptoms associated with a 1° disc-related disorder accompany the local cervical symptoms previously discussed, due in part to the irritation of a well-developed posterior longitudinal ligament. Primary disc-related CBS is typically observed in patients younger than 45 years of age, who commonly report numerous previous episodes of LCS. The root tension event is associated with disc protrusion or prolapse, especially at C5C6 or C6C7. This soft disc lesion is typically preceded by direct or indirect trauma that has occurred recently or in the past. These lesions produce radicular pain that can be provoked with coughing, sneezing, or straining. The pain is worse in the morning, due to the increased water content of the disc, and can be accompanied by parasthesias associated with vasoneurvorum irritation producing a relative ischemic event. More extensive disc lesions may produce motor deficits and numbness, depending on the size of the spinal canal. As previously mentioned, radicular symptoms are frequently observed at the C5 root level, due to the configuration of the rootlets. Symptoms are also commonly observed in the C6 and C7 distribution. Serious pathology must be ruled out if patients in this age group present with radicular symptoms at more cranial root levels, due to the low frequency of 1° disc lesions at higher levels.[6,49]

Physical examination of these patients produces the limitation patterns that are similar to the 1° disc-related LCS. The patient may experience increased neck and upper extremity pain when the shoulder girdles are retracted with the neck in a forward bent position (as previously discussed). In addition, patients may report increased arm symptoms associated with cervical sidebending in a direction contralateral to the side of upper extremity pain, due to increased neural tension around the disc prolapse (Disc Test 2, see Appendix A). When this pain is reduced by passively abducting the patient's painful shoulder while the neck is sidebent in this fashion, then the 1° disc-related diagnosis is confirmed. This condition is best treated with physical agents, gentle soft tissue techniques, cervical traction, home exercise, and possible anaesthetic procedures when the symptoms persist.

Upper extremity symptoms associated with a 2° disc-related disorder are associated with a NRCS. Although the symptoms can be isolated to the upper extremity, they can also be accompanied by local cervical symptoms previously discussed, due in part to the irritation of the UVJ and or ZAJ. Nerve NRCS is more frequently associated with bony changes versus forminal narrowing from instability, as witnessed in the lumbar spine. The degenerative changes associated with UVJ and ZAJ commonly leads to exostosis, which can account for the front-back narrowing of the intervertebral foramen and sulcus. In addition, the intervertebral foramen can narrow in an up-down direction, due to settling of the cranial vertebral body in response to disc degeneration. The area of the intervertebral foramen can decrease as much as 40% to 45% with a mere 3 mm settling of the cranial body.[38]

This NRCS is more frequently seen with individuals older than 45 years of age, who typically have a long-standing history of local cervical problems. The pain demonstrates a gradual onset, is provoked towards the end of the day and can be accompanied by parasthesias, sensory loss, and possibly motor disturbances. These patients present with capsular pattern motion limitations that include reduced extension, bilateral sidebending and rotation. These limitations are associated with long-standing degenerative changes and may or may not be painful or clinically relevant. Using a modified Spurling test can most easily provoke the upper extremity symptoms. Here, the clinician places the patient's head and neck in extension plus ipsilateral rotation and sidebending. While stabilizing the contralateral shoulder, the clinician then provides sustained axial compression to the head and neck from above. If the patient suffers from a NRCS, then the symptoms are provoked in the upper extremity. Conservative management of NRCS can include neural flossing, ventral dorsal mobilization to the caudal bony segment, and anaesthetic procedures. Symptoms that persist can be managed by surgical intervention including dorsal foramenotomy, anterior discectomy and fusion, or anterior foramenotomy.[29]

CONCLUSIONS

Afflictions of the cervical spine can be associated with either a 1° or 2° disc-related disorder. These conditions can present as a LCS, which produces cervical pain that is local and referred in a nonradicular distribution. A 1° disc-related LCS can be classified as either an acute torticollis or a disc protrusion, while a 2° disc-related LCS is associated with either chronic disc irritation or arthropathy of the UVJ and or ZAJ. Patients may also develop upper extremity symptoms associated with a 1° or 2° disc-related disorder. Tension on the dorsal root ganglion and nerve root can be caused by a disc protrusion or prolapse, leading to a 1° disc-related CBS. NRCS can be caused by 2° disc-related changes that produce front-back and or up-down compromise to the intervertebral foramen. A clinician's careful examination and differential diagnosis of these disorders can serve as a pathway to expedient and effective management.

REFERENCES

1. Bland JH. Epidemiology and demographics: phylogenesis and clinical implications. In: JH Bland, ed. *Disorders of the Cervical Spine. Diagnosis and Medical Management*. 2nd ed. Philadelphia, Pa: WB Saunders Co; 1994:3–10.
2. Winkel D, Aufdemkampe G, Matthijs O, Meijer OG, Phelps V. *Diagnosis and Treatment of the Spine: Nonoperative Orthopaedic Medicine and Manual Therapy*. Gaithersburg: Aspen Publishers, Inc.; 1996.
3. White AA III, Panjabi MM. *Clinical Biomechanics of the Spine*. 2nd ed. Philadelphia, Pa: JB Lippincott; 1990.
4. Matthijs O. Clinical Patho Anatomy. Proceedings from conference on the Nonoperative Orthopaedic Medicine and Manual Therapy of the Lower Cervical Spine. Minneapolis, Minn: International Academy of Orthopaedic Medicine; 1999.
5. Goel VK, Clausen JD. Prediction of load sharing among spinal components of a C5-C6 motion segment using the finite-element approach. *Spine* 1998;23:684–91.
6. Manifold SG, McCann PD. Cervical radiculitis and shoulder disorders. *Clin Orth Rel Res* 1999;368:105–13.
7. Ebraheim NA, Lu J, Biyani A, Brown JA, Yeasting RA. Anatomic considerations for uncovertebral involvement in cervical spondylosis. *Clin Orthop Rel Res* 1997;334:200–06.
8. Ebraheim NA, An HS, Xu RM, Ahmad M, Yeasting RA. The Quantitative Anatomy of the Cervical Nerve Root Groove and the Intervertebral Foramen. *Spine* 1996;21:1619–23.
9. Stahl C, Huth F. [Morphological evidence of synovial spaces in the uncovertebral region of cervical discs]. *Z Orthop* 1980;118:721–28.
10. Tillman B, Tondury G, Ziles K. [Human Anatomy: Locomotor System]. Stuttgart: Thieme; 1987:219.
11. Kotani Y, McNutty PS, Abumi K, Cunningham BW, Kaneda K, McAfee PC. The role of the anteromedial foraminotomy and the uncovertebral joint in the stability of the cervical spine. *Spine* 1998;23:1559–65.
12. Clausen JD, Goel VK, Traynelis VC, Scifert J. Uncinate

processes and luschka joints influence the biomechanics of the cervical spine-Quantification using a finite-element model of the C5-C6 segment. *J Orthop Res* 1997;15:342–47.

13. Penning L. Differences in anatomy, motion development and aging of the upper and lower cervical disc segments. *Clin Biomech* 1988; 3:37–47.

14. Ecklin U. [Age related changes of the cervical spine]. Berlin: Springer; 1980.

15. Hall MG. Luschka's Joint. Springfield: Thomas; 1960.

16. Tondury G. Entwicklungsgeschichte und fehlbildungen der halswirbelsaule. Stuttgart: Hippokrates Verlag; 1958.

17. Mercer S, Bogduk N. Intra-articular inclusions of the cervical synovial joints. *Br J Rheumatol* 1993;32:705–710.

18. Mercer S. Invited commentary. *JOSPT* 2000;30:10–12.

19. Ghosh P. The biology of the intervertebral disc. Vol. 1. Boca Raton, Fla:CRC Press Inc; 1988.

20. Pooni JS, Hukins DW, Harris PF, Hilton RC, Davies KE. Comparison of the structure of the human intervertebral disc in the cervical, thoracic and lumbar region of the spine. *Surg Radiol Anat* 1986;8:175–182.

21. Frykholm R. Lower cercial vertebrae and intervertebral disc, Surgical anatomy and pathology. *Acta Orthop Scand* 1951;101:345–59.

22. Hirsch C, Schajowicz F, Galante J. Structural changes in the cervical spine. *Acta Orth Scanf*. 1967;109(suppl):3–77.

23. Jenkner FL. Das cervikalsyndrom:Manuelle und elektrische therapie. Vienna: Springer Verlag; 1982.

24. Penning L, Wilmink JT. Rotation of the cervical spine: A CT study in normal subjects. *Spine* 1987;12:732–38.

25. Tondury G. Angewandte und Topogrphische Anatomie. Stuttgart: Thieme Verlag; 1981.

26. Matsumoto M, Fujimura Y, Suzuki N, Nishi Y, Nakamura M, Yabe Y, Shiga H. MRI os cervical intervertebral discs in asymptomatic subjects. J Bone Joint Surg 1998; 80B:19–24.

27. Shingyouchi Y, Nagahama A, Niida M. Ligamentous ossification of the cervical-spine in the late middle-aged japanese men—Its relation to body-mass index and glucose-metabolism. Spine 1996;21:2474–78.

28. Van der El. [Wervelkolom. Manuele Diagnostiek]. Rotterdam: Manthel; 1992.

29. Johnson JP, Filler AG, McBride DQ, Batzdorf U. Anterior cervical foramenotomy for unilateral radicular disease. *Spine* 2000;25:905–09.

30. Ordway NR, Seymour RJ, Donelson RG, Hojnowski LS, Edwards WT. Cervical flexion, extension, protrusion, and retraction. A radiographic segmental analysis. *Spine* 1999;24: 240–47.

31. Imai S, Konttinen YT, Tokunaga Y, et al. An ultrastructural study of calcitionin gene-related peptide-immunoreactive nerve fibers innervating the rat posterior longitudinal ligament. *Spine* 1997;22:1941–47.

32. Bogduk N. The anatomical basis for cervicogenic headache. *J Manipulative Physiol Ther* 1992;15:67–70

33. Dwyer A, Aprill C, Bogduk N. Cervical zygapophyseal joint pain patterns I: a study in normal volunteers. *Spine* 1990; 15:453–61.

34. Shinomiya K, Okawa A, Nakao K, et al. Morphology of C5 ventral nerve rootlets as part of dissociated motor loss of deltoid muscle. *Spine* 1994;19:2501–04

35. Ebraheim NA, Xu R, Stanescu S, Yeasting RA. Anatomical Relationship of the cervical nerves to the lateral masses. *Am J Orthop* 1999;28:39–42.

36. Stancovic R, Johnell O, Maly P, Willner S. Use of lumbar extension, slump test, physical and neurological examination in the evaluation of patients with suspected herniated nucleus pulposus. A prospective clinical study. *Man Ther* 1999;4:25–32.

37. Yabuki S, Kikuchi S. Positions of dorsal root ganglia in the cervical spine. An anatomic and clinical study. *Spine* 1996; 21:1513–17.

38. Lu J, Ebraheim NA, Huntoon M, Haman SP. Cervical intervertebral disc space narrowing and size of intervertebral foramina. *Clin Orthp Rel Res* 2000;370:259–64.

39. Kandel ER, Schwartz JH, Jessell TM. *Principles of Neuroscience*. 3rd ed. Norwalk, Conn: Appleton & Lange; 2000.

40. Oostendorp R. *Functional vertebrobasilar insufficiency* [doctoral thesis]. University of Nijmegen, The Netherlands: 1988.

41. Gawel MJ, Rothbart PJ. Occipital nerve block in the management of headache and cervical pain. *Cephalalgia*. 1992; 12:9–13.

42. Jansen J, Markakis E, Rama B, Hildebrandt J. Hemicranial attacks or permanent hemicrania—a sequel of upper cervical root compression. *Cephalalgia*. 1989;9:123–30.

43. Shellhas KP, Smith MD, Gundry CR, Pollei SR. Cervical discogenic pain. Prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers. *Spine* 1996;21:300–12.

44. Pospiech J, Stolke D, Wilke HJ, Claes LE. Intradiscal pressure recordings in the cervical spin. *Neurosurg* 1999;44:379–85.

45. Siddall PJ, Cousins MJ. Spine update: Spinal pain mechanisms. *Spine* 1997;22:98–104.

46. Saal JS. The role of inflammation in lumbar pain. *Spine*. 1995;20:1821–1827.

47. Onan OA, Heggeness MH, Hipp JA. A motion analysis of the cervical facet joint. *Spine* 1998;3:430–39.

48. Bandalamente MA, Dee R, Ghillani R, Chien PF, Daniels K. Mechanical stimulation of dorsal root ganglion induces increased production of substance P: A mechanism for pain following nerve root compromise? *Spine* 1987;12:552–55.

49. Kelsey JL, Githens PB, Walter SD, et al. An epidemiological study of acute prolapsed cervical intervertebral disc. *J Bone Joint Surg* 1984;66A:907–14.

Appendix A. Functional Examination of the Cervical Spine

Inspection:			
Resisted Cervical Extension			
Resisted Cervical Rotation	☐ Right	☐ Left	
Resisted Cervical Sidebending	☐ Right	☐ Left	
Resisted Cervical Flexion			
Motor Screening		Muscle Grade	Root level
Resisted Sh. Girdle Elevation			C2-C4
Resisted Sh. ABduction			C5
Resisted Sh. ADduction			C7
Resisted Sh. Internal Rotation			C5, C6
Resisted Sh. IExternal Rotation			C5, C6
Resisted Elbow Flexion			C5, C6
Resisted Elbow Extention			C7
Resisted Wrist Palmar Flexion			C7
Resisted Wrist Dorsal Extention			C6
Resisted Thumb Extention			C8
Resisted 5th digit Abduction			T1
Sensory Screening		C6 C7 C8	
Reflexes	BrachioRadialis C5	Biceps C5, C6	Triceps C7
Special Tests		Test Outcomes	
Spurling Test (Foraminal Compression)			
Disc Test 1 (Flexion + Sh. Retraction)			
Disc Test 2 (Contra SB + Sh. ABduction)			
Comments:			

Appendix B. Local Examination of the Lower Cervical Spine

Active Sagittal Motions		Provocation	Location of Sx's
Flexion			
Extention			
Retraction			
Frontal Motions	☐ Active		
	☐ Passive	Provocation	Location of Sx's
Sidebend (R)			
with (R) Rot			
with (L) Rot			
Sidebend (L)			
with (L) Rot			
with (R) Rot			
Transverse Motions	☐ Active		
	☐ Passive	Provocation	Location of Sx's
Rotation (R)			
with (R) SB, Flex			
with (R) SB, Ext			
with (L) SB			
Rotation (L)			
with (L) SB, Flex			
with (L) SB, Ext			
with (R) SB			
Comments:			