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Original Articles

Myodural Cephalgia

Is It Time to Re-Classify Headaches?
The Migraine Myth

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Abstract

In 1996 three scientists at the University of Maryland at Baltimore discovered a web of tissue directly connecting muscle tissue to the dura mater. Traditional treatment and understanding of headaches and particularly migraine headaches as a vascular phenomenon from studies performed in the 1930s-50s has been questioned. Migraine headache is now recognized as a neurological process. The cerebral vascular changes are an epiphenomenon to the alterations of the central nervous system. Single photon emission computed tomography scans have clearly linked abnormalities in the meninges as the epicenter of migraine pain. The International Headache Society discusses the trigeminal nerve as providing a biological explanation of referred pain. The new anatomical discoveries afforded us the structure for the completion of the neuromusculoskeletal connections. This will lead to an entire new field of research and development in all branches of medicine.

Key Words:

1. Headache
2. Migraines
3. Trigeminal nerve
4. Occipital neuralgia
5. Web of tissue
6. Cervical nerves

NEW ANATOMICAL DISCOVERIES

In 1996 three scientists at the University of Maryland at Baltimore discovered a web of tissue directly connecting the Rectus Capitis Posterior Minor Muscle (which extends from the skull to C1, by way of the space between occiput and C1) to the dura mater(1). This noble, prize-worthy discovery had never been previously described in medical anatomical texts. This discovery is expanding the understanding of an old malady, migraine headache.

The work of these three scientists, Gary D. Hack, D.D.S., Gwendolyn Dunn, D.D.S. and Mi Young Toh, M.S., M.A., has resulted in providing the impetus for discoveries of additional connections to the central nervous system, capable of producing traction of the dura mater. Some researchers have reported a thickening of the spinal dura mater in the region of attachment(1). This discovery helps to complete the missing pieces of the puzzle of muscle hypertonus, headache and mechanically mediated neurological symptoms produced via the Trigeminal and the Occipital (C1 spinal nerve) nerves, as well as the Cervical Plexus.

Subsequent to the “Hack-Dunn-Toh web of tissue” discovery from the Rectus Capitis Posterior Minor, other muscle dura connections were found between C1 and C2 from the Rectus Capitis Posterior Major and the Obliquus Capitis Inferior.

In 1998 a team of anatomists at the Anglo-European College of Chiropractic reported another important connection. The Ligamentum Nuchae is one of the major stabilizing ligaments of the Posterior Cervical Spine. The chiropractic team found a branch of the Ligamentum Nuchae that passes between the first two cervical vertebrae attaching to the dura and the lateral part of the occipital bone (2).

Some researchers believe that headache pain is primarily caused by changes in brain chemistry that result in a lowering of the threshold at which pain is perceived. An increasing number of researchers postulate that headache pain may be produced by structures located in the neck (1).

TRIDITIONAL VERSUS CURRENT CONCEPTS

The authors and current literature believe traditional treatment and understanding of migraine headaches solely as a vascular phenomenon are now inaccurate, disproven and the classification presently used is antiquated. The following research supports this statement: Single photon emission computed tomography (SPECT) scans have clearly linked abnormalities in the meninges and the epicenter of migraine pain, shifting the diagnostic focus for this enigmatic disorder away from dilated or constricted blood vessels in the back of the

head. Several things point instead to the protective tissue layers covering the brain. “For one thing, the symptoms of a bad migraine – nausea, vomiting, light and sound sensitivity, throbbing headaches – are essentially the same as in meningitis, the bacterial or viral inflammation of the meninges” (3). “In a migraine attack, the trigeminal nerve, which possesses pain fibers, is **somehow** (author’s emphasis) activated, enervating the sensitive area in the meninges. The neuropeptides that are released trigger inflammation and sensitize nearby pain receptors in the meninges that send the pain message” (4).

THE NEUROMUSCULAR CONNECTION

We know that the brain itself is relatively insensitive, but the dura is extremely sensitive. What we have not seen in the medical literature at this point is the research that would direct definitive neurological connection to head pain, by way of the myodural and ligamentous attachments to the dura. “Dura mater – the outermost, toughest, and most fibrous of the three membranes (meninges) covering the brain and spinal cord. Dura mater – of the brain is composed of two mostly fused layers; endosteal outer layer (endocranium) adherent to the inner aspect of the cranial bones and the inner, meningeal layer. **Venous sinuses and trigeminal ganglia are located between the layers**” (author’s emphasis) (5).

THE NEUROMUSCULOSKELETAL CONNECTION

The neuromuscular mechanical component, related to the Rectus Capitis Posterior Minor and Rectus Capitis Posterior Major muscles and Ligamentum Nuchae branched ligament are in effect producing a mechanical tug on the dura resulting in irritation of the trigeminal ganglia that is located between the two dural layers. In the author’s opinion this is the completion of the puzzle, the missing link, connecting the mechanical traction of the dura mater to the trigeminal, occipital or cervical plexus neurological aberrations.

ANATOMICAL REVIEW

To review the origin, insertion and nerve supply of the three muscles of interest and keeping the web of tissue and the attachments in mind, it is easy to see the correlation.

“The Rectus Capitis Posterior Minor muscle arises by a narrow pointed tendon from the tubercle on the posterior arch of C1 and inserts into the medial inferior nuchal line of the occiput and the foramen magnum. Its function is to extend the head and its nerve supply in a branch of the dorsal primary division of the occipital nerve” (6).

“The Rectus Capitis Posterior Major arises from a pointed tendon from the spinous of C2 which inserts into the lateral inferior nuchal line of the occiput. It extends and rotates the head to the same side. Its nerve supply is a branch of the posterior ramus of the occipital nerve” (6).

“The Obliquus Capitis Inferior arises from the spinous apex of the axis and inserts into the posterior transverse process of the atlas. It rotates C1 and turns the face to the same side. Its nerve supply is a branch of the dorsal primary division of the suboccipital nerve” (6).

Note all three muscles are supplied by the Greater and Lesser Occipital Nerves that are branches of the First Cervical Nerve. These nerves are located in the deep groove on the upper surface of the posterior arch of the atlas along with the vertebral artery. According to the 39th edition of Gray’s Anatomy, “The innervation of the cranial dura mater is derived mainly from the three divisions of the trigeminal nerve, the first three cervical spinal nerves and the cervical sympathetic trunk. Less well-established meningeal branches have been described arising from the vagus and the hypoglossal nerves and possibly from the facial and glossopharyngeal nerves” (7). “The dura in the posterior cranial fossa is innervated by ascending meningeal branches of the upper cervical nerves, which enter through the anterior part of the foramen magnum (second and third cervical nerves) and through the hypoglossal canal and jugular foramen (first and second cervical nerves). Meningeal branches of both the vagus and hypoglossal nerves have been described” (7).

The trigeminal nerve is the largest of the Cranial Nerves and is the Great Cutaneous Sensory Nerve of the face, the sensory nerve to the mucous membranes and other internal structures of the head (sinuses, lacrimal

gland, mucous membranes of the paranasal sinuses) and the motor nerve to the muscles of mastication (masseter, pterygoid, etc.)

THE ALL IMPORTANT TRIGEMINAL NERVE

The following brief synopsis explains how extensive and the importance of the Trigeminal Nerve and its three branches are to head pain and other encephalopathies. The Ophthalmic Branch Of The Trigeminal Nerve “is a sensory nerve supplying the bulb of the eye, conjunctiva, lacrimal gland, part of the mucous membrane of the nose and paranasal sinuses, and the skin of the forehead, eyelids and nose” (8). The Maxillary Branch Of The Trigeminal Nerve “supplies the skin of the middle portion of the face, lower eyelid, side of the nose, and upper lip, the mucous membrane of the nasopharynx, maxillary sinus, soft palate, tonsil and roof of the mouth, the upper gums and teeth” (8). The Mandibular Branch Of The Trigeminal Nerve are “sensory fibers [that] supply the skin of the temporal region, auricula, external meatus, cheek, lower lip, and lower part of the face; the mucous membrane of the cheek, tongue, and mastoid air cells; the lower teeth and gums; the mandible and temporomandibular joint; and part of the dura mater and skull. The motor fibers supply the muscles of mastication (Masseter, Temporalis, and Pterygoidei), the Mylohyoideus and anterior belly of the Digastricus and the Tensor tympani and veli palatine” (8).

“Trigeminal nerve pain is more frequently the seat of severe neuritic or neuralgic pain than any other nerve in the body. The pain of localized irritation or infection may be confined to that area, but quite commonly that is not the case. Involvement of an internal branch is likely to set up severe distress in a related cutaneous area by referred pain. As a general rule the diffusion of pain over the branches of the nerve is confined to one of the main divisions although in severe cases it may radiate over the other main divisions” (8).

MIGRAINE HEADACHE MISCONCEPTIONS

In 2004, a paper titled “Migraine Headache Misconceptions: Barriers to Effective Care” demonstrated some of the confusion and misunderstanding related to headaches and their treatment (9).

“The pivotal Spectrum Study showed that in patients with the International Headache Society (IHS) migraine regardless of whether an individual attack was labeled as tension or migraine, the migraine-specific drug sumatriptan provided relief supporting the concept of a single pathologic process. As discussed earlier, the various innervations of the trigeminal system provide a biologic explanation of referred pain in the nasal passages and other areas. In practice, patients often endorse a variety of symptoms, such as facial pain and tenderness, congestion, lacrimation, or rhinorrhea, not listed in the IHS checklist of diagnostic criteria for migraine. This contributes significantly to under-diagnosis of migraine and over diagnosis of sinus problems. Direct to consumer advertising for numerous Over The Counter (OTC) sinus headache remedies adds to this confusion” (9).

“The IHS objectively defines sinus headache by purulent nasal discharge; pathologic sinus finding at radiography, computed tomography, or magnetic resonance imaging; simultaneous onset of headache and sinusitis; and headache localized to specific facial and cranial areas of the sinuses. Of important note, the IHS does not validate chronic sinusitis as a cause of recurrent headaches. Despite this clear definition, the mere pressure or pain in the nasal area often is the foundation of a diagnosis of sinus headache, impacting the quality of patient care” (9).

“A recent headache clinic-based study of 2524 patients with self-diagnosed or physician-diagnosed sinus headache found that 90% fulfilled IHS criteria for migraine. Notably, only four patients had evidence of active sinus infection. Two thirds of these patients were completely dissatisfied with their therapy, a not surprising statistic given these patients’ incorrect diagnosis” (9).

“The notion of recurrent, incapacitating tension or sinus headache affecting large numbers of individuals is not supported by published evidence or clinical experience. Furthermore, improper diagnosis leads to inappropriate drug therapy, increased risk of adverse effects, as well as patient and clinician frustration from failed therapy. To avoid these pitfalls, we propose pharmacists adopt a philosophy that any patient presenting with episodic, debilitating headaches be considered to have migraine until and unless appropriate evaluation proves otherwise. Simply put, ‘debilitating headache, think migraine’” (9).

In our opinion myodural headaches may be initiated by mechanical, chemical, and/or psychological irritants resulting in muscle spasm of the suboccipital muscles causing myofascial traction on the dura mater. This traction may trigger irritation of the trigeminal nucleus and/or nerves, irritation of the occipital nerves, and initiating inflammation of the dura mater, and this is the common denominator for myodural headaches.

TREATMENT AND MANAGEMENT – NEW FRONTIER FOR RESEARCH

These headaches respond to appropriate and precise mechanical manipulation of the cranio-cervical structures alleviating the sequelae of muscle spasm, nerve irritation, myodural traction and ultimately, the dural inflammation. The chemical approach utilizing pharmaceuticals specifically directed toward reducing muscle spasm and/or inflammation of the dura may be helpful. An example of this would be pain management specialist with injection of Depo-Medrol into the rectus capitus posterior minor and/or major muscles. Could this be another use for Botox (clostridium botulinum toxin)? Psychological counseling by a psychiatrist/psychologist may be helpful if directed toward relieving psychosomatic etiologies. Surgical intervention (mechanical) such as myofascial lysis and/or necrotization of the nerve(s) could be the treatment of choice after failure to respond to conservative care in patients with intractable pain.

Scientific investigation of this premise of neurological and somatic phenomenon of cause, treatment, and effect, presented above, is imperative. A new portal has been opened expanding the understanding of an old malady that causes millions of people pain and suffering that can now be observed, recorded, and information disseminated with a completely different concept and approach. The connection of this long held anatomical secret, web of tissue, discovered by Gary D. Hack, D.D.S., Gwendolyn Dunn, D.D.S. and Mi Young Toh, M.S., M.A., has opened a door of enlightenment and new thought on the old issue of severe, debilitating, life-altering headaches.

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References

- 1 Hack GD, et al. The anatomist's new tools: In: Bernstein E (ed) 1998 Medical And Health Annual, Encyclopedia Britannica, 1997.
- 2 Mitchell BS, et al. Attachments of the ligamentum nuchae to cervical posterior dura and the lateral part of the occipital bone, Journal of Manipulative and Physiological Therapeutics 1998, 21:145
- 3 Pappagallo M, *Radiology and Imaging Letter*. 1999 June; 10: 19(10):75.
- 4 Pappagallo M, *Radiology and Imaging Letter*. 1999 June; 10: 19(10):76.
- 5 Dorland's illustrated medical dictionary. 30th ed. Philadelphia: W.B Sanders; 2003. Dura mater; p.570.
- 6 Gray's anatomy. 27th ed. Philadelphia: Lea and Febiger; 1959. p. 445.
- 7 Gray's anatomy. 39th ed. Edinburgh: Ersevier; 2005. p. 282.
- 8 Gray's anatomy. 27th ed. Philadelphia: Lea and Febiger; 1959. p. 973.
- 9 Wenzel R, Dortch M, Cady R, Loffand J, Diamond S. *Migraine Headache Misconceptions: Barriers to Effective Care*. Pharmacotherapy. 2004 June [20 July 2004]. Available from: <http://www.medscape.com/viewpublications/1327>.

Reprints

Guidelines – Degenerative Spondylolisthesis

http://www.guideline.gov/summary/summary.aspx?view_id=1&doc_id=12680

Brief Summary

GUIDELINE TITLE

Diagnosis and treatment of degenerative lumbar spondylolisthesis.

BIBLIOGRAPHIC SOURCE(S)

- North American Spine Society (NASS). Diagnosis and treatment of degenerative lumbar spondylolisthesis. Burr Ridge (IL): North American Spine Society (NASS); 2008. 133 p. [191 references]

GUIDELINE STATUS

This is the current release of the guideline.

BRIEF SUMMARY CONTENT

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RECOMMENDATIONS

MAJOR RECOMMENDATIONS

The grades of recommendations (A-C, I) and levels of evidence (I-V) are defined at the end of the Major Recommendations field.

Recommendations for Diagnosis and Treatment of Degenerative Lumbar Spondylolisthesis

A. Diagnosis and Imaging

What are the most appropriate historical and physical examination findings consistent with the diagnosis of degenerative lumbar spondylolisthesis?

Obtaining an accurate history and physical examination is essential to the formulation of the appropriate clinical questions to guide the physician in developing a plan for the treatment of patients with degenerative lumbar spondylolisthesis.

In older patients presenting with radiculopathy and neurogenic intermittent claudication, with or without back pain, a diagnosis of degenerative lumbar spondylolisthesis should be considered.

Grade of Recommendation: B

Diagnosing Spondylolisthesis with Imaging

What are the most appropriate diagnostic tests for degenerative lumbar spondylolisthesis?

The most appropriate, noninvasive test for detecting degenerative lumbar spondylolisthesis is the lateral radiograph.

Grade of Recommendation: B

The most appropriate, noninvasive test for imaging the stenosis accompanying degenerative lumbar spondylolisthesis is the magnetic resonance imaging (MRI).

Plain myelography or computed tomography (CT) myelography are useful studies to assess spinal stenosis in patients with degenerative lumbar spondylolisthesis.

Grade of Recommendation: B

CT is a useful noninvasive study in patients who have a contraindication to MRI, for whom MRI findings are inconclusive or for whom there is a poor correlation between symptoms and MRI findings, and in whom CT myelogram is deemed inappropriate.

B. Outcome Measures for Medical/Interventional and Surgical Treatment

What are the appropriate outcome measures for the treatment of degenerative lumbar spondylolisthesis?

The Zurich Claudication Questionnaire (ZCQ)/Swiss Spinal Stenosis Questionnaire (SSS), Oswestry Disability Index (ODI), Likert Five-Point Pain Scale and 36-Item Short Form Health Survey (SF-36) are appropriate measures for assessing treatment of degenerative lumbar spondylolisthesis.

Grade of Recommendation: A

Note: The Zurich Claudication Questionnaire (ZCQ) represents an evolution of Swiss Spinal Stenosis Questionnaire (SSS). Conclusions made about either questionnaire have a high likelihood of being applicable to the other.

The Japanese Orthopedic Association (JOA) Score and the calculated Recovery Rate may be useful in assessing outcome in degenerative lumbar spondylolisthesis.

Grade of Recommendation: B

The Shuttle Walking Test (SWT), Oxford Claudication Score (OCS), Low Back Pain Bothersome Index and Stenosis Bothersome Index are potential outcome measures in studying degenerative lumbar spondylolisthesis.

Grade of Recommendation: I (Insufficient Evidence)

C. Medical and Interventional Treatment

Medical/interventional treatment for degenerative lumbar spondylolisthesis when the radicular symptoms of stenosis predominate, most logically should be similar to treatment for symptomatic degenerative lumbar spinal stenosis.

D. Surgical Treatment

Do surgical treatments improve outcomes in the treatment of degenerative lumbar spondylolisthesis compared to the natural history of the disease?

Surgery is recommended for treatment of patients with symptomatic spinal stenosis associated with low grade degenerative spondylolisthesis whose symptoms have been recalcitrant to a trial of medical/interventional treatment.

Grade of Recommendation: B

Does surgical decompression alone improve surgical outcomes in the treatment of degenerative lumbar spondylolisthesis compared to medical/interventional treatment alone or the natural history of the disease?

Direct surgical decompression is recommended for treatment of patients with symptomatic spinal stenosis associated with low grade degenerative lumbar spondylolisthesis whose symptoms have been recalcitrant to a trial of medical/interventional treatment.

Grade of Recommendation: I (Insufficient Evidence)

Indirect surgical decompression is recommended for treatment of patients with symptomatic spinal stenosis associated with low grade degenerative lumbar spondylolisthesis whose symptoms have been recalcitrant to a trial of medical/interventional treatment.

Grade of Recommendation: I (Insufficient Evidence)

Does the addition of lumbar fusion, with or without instrumentation, to surgical decompression improve surgical outcomes in the treatment of degenerative lumbar spondylolisthesis compared to treatment by decompression alone?

Surgical decompression with fusion is recommended for the treatment of patients with symptomatic spinal stenosis and degenerative lumbar spondylolisthesis to improve clinical outcomes compared with decompression alone.

Grade of Recommendation: B

Does the addition of instrumentation to decompression and fusion for degenerative lumbar spondylolisthesis improve surgical outcomes compared with decompression and fusion alone?

The addition of instrumentation is recommended to improve fusion rates in patients with symptomatic spinal stenosis and degenerative lumbar spondylolisthesis.

Grade of Recommendation: B

The addition of instrumentation is not recommended to improve clinical outcomes for the treatment of patients with symptomatic spinal stenosis and degenerative lumbar spondylolisthesis.

Grade of Recommendation: B

How do outcomes of decompression with posterolateral fusion compare with those for 360° fusion in the treatment of degenerative lumbar spondylolisthesis?

Because of the paucity of literature addressing this question, the work group was unable to generate a recommendation to answer this question.

What is the role of reduction (deliberate attempt to reduce via surgical technique) with fusion in the treatment of degenerative lumbar spondylolisthesis?

Reduction with fusion and internal fixation of patients with low grade degenerative lumbar spondylolisthesis is not recommended to improve clinical outcomes.

Grade of Recommendation: I (Insufficient Evidence)

What is the long-term result (four+ years) of surgical management of degenerative lumbar spondylolisthesis?

Decompression and fusion is recommended as a means to provide satisfactory long-term results for the treatment of patients with symptomatic spinal stenosis and degenerative lumbar spondylolisthesis.

Grade of Recommendation: C

Definitions:

Grades of Recommendation for Summaries or Reviews of Studies

- A.** Good evidence (Level I Studies with consistent finding) for or against recommending intervention.
- B.** Fair evidence (Level II or III Studies with consistent findings) for or against recommending intervention.
- C.** Poor quality evidence (Level IV or V Studies) for or against recommending intervention.
- I.** Insufficient or conflicting evidence not allowing a recommendation for or against intervention.

Levels of Evidence for Primary Research Question¹

	Types of Studies			
	Therapeutic Studies – Investigating the results of treatment	Prognostic Studies – Investigating the effect of a patient characteristic on the outcome of disease	Diagnostic Studies – Investigating a diagnostic test	Economic and Decision Analyses – Developing an economic or decision model
Level I	- High quality randomized trial with statistically significant difference or no statistically significant difference but narrow confidence intervals - Systematic review² of Level I RCTs (and study	- High quality prospective study⁴ (all patients were enrolled at the same point in their disease with $\geq 80\%$ follow-up of enrolled patients) - Systematic review² of Level I studies	- Testing of previously developed diagnostic criteria on consecutive patients (with universally applied reference "gold" standard) - Systematic review² of Level	- Sensible costs and alternatives; values obtained from many studies; with multiway sensitivity analyses - Systematic review² of Level I studies

	Types of Studies			
	results were homogenous ³⁾		I studies	
Level II	• Lesser quality RCT (e.g., <80% follow-up, no blinding, or improper randomization) • Prospective⁴ comparative study⁵ • Systematic review² of Level II studies or Level I studies with inconsistent results	• Retrospective⁶ study • Untreated controls from an RCT • Lesser quality prospective study (e.g., patients enrolled at different points in their disease or <80% follow-up) • Systematic review² of Level II studies	• Development of diagnostic criteria on consecutive patients (with universally applied reference "gold" standard) • Systematic review² of Level II studies	• Sensible costs and alternatives; values obtained from limited studies; with multiway sensitivity analyses • Systematic review² of Level II studies
Level III	• Case control study⁷ • Retrospective⁶ comparative study⁵ • Systematic review² of Level III studies	• Case control study⁷	• Study of nonconsecutive patients; without consistently applied reference "gold" standard • Systematic review² of Level III studies	• Analyses based on limited alternatives and costs; and poor estimates • Systematic review² of Level III studies
Level IV	Case Series ⁸	Case Series	• Case-control study • Poor reference standard	• Analyses with no sensitivity analyses
Level V	Expert Opinion	Expert Opinion	Expert Opinion	Expert Opinion

RCT = randomized controlled trial

¹ A complete assessment of quality of individual studies requires critical appraisal of all aspects of the study design.

² A combination of results from two or more prior studies.

³ Studies provided consistent results.

⁴ Study was started before the first patient enrolled.

⁵ Patients treated one way (e.g., cemented hip arthroplasty) compared with a group of patients treated in another way (e.g., uncemented hip arthroplasty) at the same institution.

⁶ The study was started after the first patient enrolled.

⁷ Patients identified for the study based on their outcome, called "cases" (e.g., failed total arthroplasty) are compared to those who did not have outcome, called "controls" (e.g., successful total hip arthroplasty).

⁸ Patients treated one way with no comparison group of patients treated in another way.

CLINICAL ALGORITHM(S)

None provided

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EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of supporting evidence is identified and graded for most of the recommendations (see "Major Recommendations" field).

[Top^](#)

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

- North American Spine Society (NASS). Diagnosis and treatment of degenerative lumbar spondylolisthesis. Burr Ridge (IL): North American Spine Society (NASS); 2008. 133 p. [191 references]

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GUIDELINE AVAILABILITY

Electronic copies: Available in Portable Document Format (PDF) from the [North American Spine Society \(NASS\) Web site](#).

Print copies: Available from the North American Spine Society (NASS), 7075 Veterans Boulevard, Burr Ridge, IL 60527; Toll-free: (866) 960-6277 . An order form is available from the [North American Spine Society Web site](#).

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

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Abstracts & Literature Review

T4 Syndrome

Topic Summary of T4 Syndrome

By The CCGPP Thoracic Spine Team

Submitted by Jeffrey R. Cates, DC, MS, FACO, DABCC Team Lead

T4 Syndrome is not addressed in the higher quality literature, however, clinical observation and reports have been made in the medical, chiropractic, and physical therapy literature. (1-9) While quality information regarding T4 syndrome is lacking, the clinician and researcher might benefit by a short summary of the lower level literature.

Evans opines that the term "upper thoracic disorder" might be a more accurate name for the condition since the condition generally includes T1 to T7. (1) None-the-less it is generally referred to as T4 or sometimes T3 syndrome. Matthisjs et al. attribute the first description of the disorder to Maitland and Burnell in 1957 and report that an English MD and Dutch physiotherapists have described a similar condition. (4)

The condition is reported to present with a unique constellation of signs and symptoms. The history is generally unremarkable and a non-traumatic onset is common. The reported symptoms include paresthesia in a glove like pattern, an altered sensation of the hands feeling hot, cold, heavy or swollen. Interscapular pain, upper quadrant pain, and suboccipital headache are commonly reported. The condition may display elements mimicking complex regional pain syndrome, thoracic outlet syndrome, and even chest or cardiac pain. Unlike cardiac pain the timing of T4 syndrome pain is reportedly different. Whereas cardiac pain generally comes on with exercise and improves with rest, T4 syndrome pain is thought to be aggravated with resting postures but not by exercise.

Examination: Examination is generally unremarkable with no hard neurological symptoms to speak of. Radiographs are generally noncontributory. Stiffness in the upper thoracic spine and costovertebral/costotransverse joints is commonly reported. There have been reports of hand discoloration, weakness, and thermosensory loss.

The etiology of the condition is unknown. A possible pathophysiological mechanism put forth involves autonomic nerve dysfunction of the sympathetics to head and neck and or upper trunk and limb. (1, 6) The dysfunction could be related to irritation and dysfunction of afferent sympathetic spinal nerve fibers, or sympathetic nerve entrapment or ischemic events from rib or osteophytic involvement. Vascular changes might be related to sympathetic motor control changes. The lack of literature and understanding does not allow for conclusive conclusions.

The condition has been reported to respond well to manual therapies including mobilization and manipulative treatment to the upper thoracic spine, lower cervical spine and ribs. Relief has also been reported with intramuscular injections of bupivacaine at the T4 level. Breathing, ergonomic, and postural instruction may also help. Additional research is needed to determine whether-or-not T4 syndrome actually is a diagnostic entity and, if so, how it responds to manual medicine and other treatments.

1. Evans P. The T4 syndrome. Some basis science aspects. *Physiotherapy* 1997;83(4):186-189.
2. DeFranca G, Levine L. The T4 syndrome. *J Manipulative Physiol Ther* 1995;18(1):34-7.
3. Fraser D. T-3 Revisited. *Journal of Orthopaedic Medicine*. 1993;15(1):3-4.
4. Matthijs O, Phelps V. T-4 Syndrome. In: *The AAOM Quarterly: International Academy of Orthopedic Medicine*; 1997.
5. McGuckin N. The T4 Syndrome. In: Grieve GP, editor. *Modern manual therapy of the vertebral column*. Edinburgh ; New York: Churchill Livingstone; 1986. p. 370-376.
6. Mellick G, Mellick L. Clinical presentation, quantitative sensory testing, and therapy of 2 patients with fourth thoracic syndrome. *J Manipulative Physiol Ther* 2006;29(5):403-8.
7. Maitland GD, Edwards BC. *Vertebral manipulation*. 5th ed. London ; Boston: Butterworths; 1986.
8. Menck J, Requejo S, Kulig K. Thoracic spine dysfunction in upper extremity complex regional pain syndrome type I. *J Orthop Sports Phys Ther* 2000;30(7):401-9.
9. Grieve GP. *Common vertebral joint problems*. 2nd ed. Edinburgh ; New York: Churchill Livingstone; 1988.

Book Review

Functional Soft-Tissue Examination and Treatment by Manual Methods, by Warren Hammer, 3rd Edition, 2007, Jones and Bartlett Publishers, 775 pages.

A Book Review by Jonice (Joni) Owen, DC, FACO, MAppSc, DACRB

Our extensive, post-doctoral education in non-surgical orthopedics has offered training, which we wish to continue; this book will serve as a guidepost, idea book, research document *and* how-to-manual. Dr. Hammer's book, a "must have" for chiropractic orthopedists, covers just what the title spells out.

This is the sort of book that will stimulate you to want to read more, know more, examine more, and get your "hands on" the varied, common and rare patient presentations that walk into your office. The tone of the book is informative, well researched, and practical. The author speaks to the reader with respect. He was able to deliver an excellent basic review, which will never wasted on you, the advanced reader.

As chiropractic orthopedists we teach patients, staff, team members and other health care professionals. This book may be utilized in training programs to assist the chiropractic doctor in explaining basic contacts. It would also serve as a great text for those enrolled in post-graduate programs engaged in learning the basic concepts of Functional Soft-Tissue approaches, as well as, reviewing the basic anatomy. Extensive yet concise background material on biological, physiological and biomechanical lend invaluable tools, as well as, offer a guaranteed anchor for a very complex field of study. Just be prepared to take off with advanced concepts, which involve simple applications in the hands of the trained.

As a chiropractic orthopedist, I enjoyed reviewing tests and signs. For example: The Rowe sign (p. 139); Testing for deltoid extension lag sign (p. 144); Doormat sign, p. 288; and Functional tests are include throughout the book.

This book helped me "think outside the box" Elbow loose body manipulation, p. 187 Figure 4-23, A, B, and C. portray a long-axis traction with the elbow flexed 90 degrees for 10 conditions with more steps to follow. I was very happy to find Figure 11-50; a photograph of a portable drop-adjusting device made by the Thuli Corporation, Dodgeville, Wisconsin. This page suggests possibilities, and thankfully, it doesn't advertise. I located the item with a Google search of "Thuli adjusting device".

Here are further highlights:

- ✓ Innovative neurological information Figure 2-4: Flow chart of several processes involved in the neural dynamics of immediate tissue plasticity in myofascial manipulation
- ✓ Detailed anatomical charts
- ✓ Decent photographs demonstrating doctor/patient positioning for exam and treatment.

- ✓ Introduction to the Human Anatomy from a variety of sources, including photographs of a live human with overlay drawing of the bones under discussion.
- ✓ Clear photos of “home stretching and home exercises” which may be shown to our patients to clarify our instructions
- ✓ Solid contributions from P. Michael Leahy (Ch: 22, Active Release Techniques: Long Tract Nerve Release), David Seaman (Nutritional Considerations in the Treatment of Soft Tissue Injuries), Peter Gale (Ch. 11 Joint Mobilization), and Gary Ierna (Ch 12 Muscle Dysfunction and Muscle Energy Techniques), Marc Heller (Ch. 16 The Lower Cervical Spine: An Integrated Approach to Joints, Fascia, and Muscles), Stuart McGill (Ch 10 Lumbar Spine Instability: Assessment and Exercise Based Restabilization), to name a few
- ✓ Functional Diagnosis Charts
- ✓ Treatment sections are included for each discussed description
- ✓ Suggestions for splints
- ✓ Unusual conditions with examination suggestions, differential diagnosing, and treatment recommendations for conditions, both in the spine and extremities. For example: Slap lesions, Radial tunnel syndrome, Anterior shoulder dislocation, Common pelvic entrapments.
- ✓ Brief Kinetic Chain Description
- ✓ Extensive documentation at the conclusion of each chapter will enliven your research

Functional Soft-Tissue Examination and Treatment by Manual Methods brings an invaluable tool and an exceptional key to your professional toolkit, and it offers very stimulating reading!

Case History

Lung cancer metastasis to the scapula and spine: a case report

Originally published at: <http://www.chiroandosteo.com/content/16/1/8>

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Abstract

Background

The objective of this case report is to describe the clinical presentation of a patient who complained of shoulder pain and was diagnosed with carcinoma of the scapula and spine that metastasized from the lung.

Case presentation

A 76-year-old man without a history of cancer sought chiropractic care for right shoulder pain. Careful evaluation, radiographs, and subsequent imaging revealed primary and metastatic lung cancer. The patient was referred to his primary care physician for immediate medical care. Diagnostic images are included in this case to provide a comprehensive depiction of the scope of the patient's disease.

Conclusion

Musculoskeletal symptoms are commonly encountered in chiropractic practice. It is important to recognize that primary lung cancer may be unidentified, and musculoskeletal symptoms may reflect the first sign of primary or metastatic pulmonary disease. Thoughtful evaluative procedure and clinical decision making, combined with the use of appropriate diagnostic tests may allow timely identification of primary or metastatic disease.

Background

In the USA, more people die from lung cancer than any other type of cancer [1]. This is true for both men and women. In 2004, lung cancer accounted for more deaths than breast cancer, prostate cancer, and colon cancer combined [2].

Lung cancer can metastasize to virtually any bone, although the axial skeleton and proximal long bones are most commonly involved [3]. The primary symptom resulting from bone involvement is pain, which may have a pleuritic component when the ribs are involved. Bone pain is present in up to 25% of all patients at presentation [3].

Patients commonly seek chiropractic care with musculoskeletal complaints [4,5]. Through history and examination, chiropractic physicians have an opportunity to assess patients and determine whether serious conditions are present that may necessitate medical referrals.

Patients with previously identified or yet to be identified cancer may seek care with chiropractic physicians. This case report demonstrates previously undiagnosed lung cancer with widespread metastatic foci.

Case presentation

Case report

A 76-year-old male sought chiropractic care for complaints of right shoulder pain and mild right arm weakness. The onset of pain was insidious and of one week's duration. Pain was rated 8/10 on a visual analogue scale (0=no pain, 10=the worst pain of one's life). The pain was described as severe and worsened with movement. Additional symptoms included mild shortness of breath and posterior thoracic pain on respiration.

The patient's past medical history included headache, degenerative joint disease affecting the cervical spine, and a benign thyroid nodule. The patient reportedly smoked tobacco products for 50 years. He was a retired electrician.

The patient was afebrile. Vital signs were normal. Respirations were 18 cycles per minute. The lungs were clear to auscultation. The patient reported upper thoracic pain on inspiration.

A non-tender, mild decrease in active range of motion of the cervical spine was noted in all planes. No tenderness was elicited on palpation of the cervical spine. Cervical compression and Soto-Hall tests were negative. Valsalva maneuver was negative. Neurologic examination revealed no focal deficits.

Examination of the right shoulder revealed exquisite tenderness on palpation of the lateral border of the scapula with muscle spasm affecting the ipsilateral infraspinatus, teres major, and teres minor muscles. Active ranges of shoulder motion were restricted and painful in abduction, internal, and external rotation.

Plain film radiographs of the right shoulder (AP with internal and external rotation views) and thoracic spine (AP and lateral views) were performed. Disruption of the cortical margin of the lateral border of the right scapula was noted as evidenced by an indistinct lucency (see Figure 1). In addition, a suspicious mass was noted in the hilar region of the right lung. Complete loss of the right hilar vascular detail secondary to the tumor mass effect were noted with visualized subsegmental infiltrate densities. No evidence of pleural effusion was noted.



Figure 1. AP radiograph of the right scapula reveals a focal indistinct lucency and lytic destruction of the lateral scapular cortical margin.

The initial diagnostic impression included: suspicious right lung pathology and apparent lytic process affecting the scapula of an unknown origin. The patient was referred for imaging evaluations that included chest x-ray (CXR) and computed tomographic (CT) evaluation of the chest. He was referred to his primary care medical physician.

The CXR and CT examination of the chest, abdomen and pelvis revealed:

1. A large mass in the right upper lobe of the lung with associated mediastinal and hilar adenopathy (see Figures 2 and 3).
2. Metastatic disease of the scapula (see Figure 4).
3. Metastatic liver disease.



Figure 2. PA chest radiograph reveals a right hilar mass.

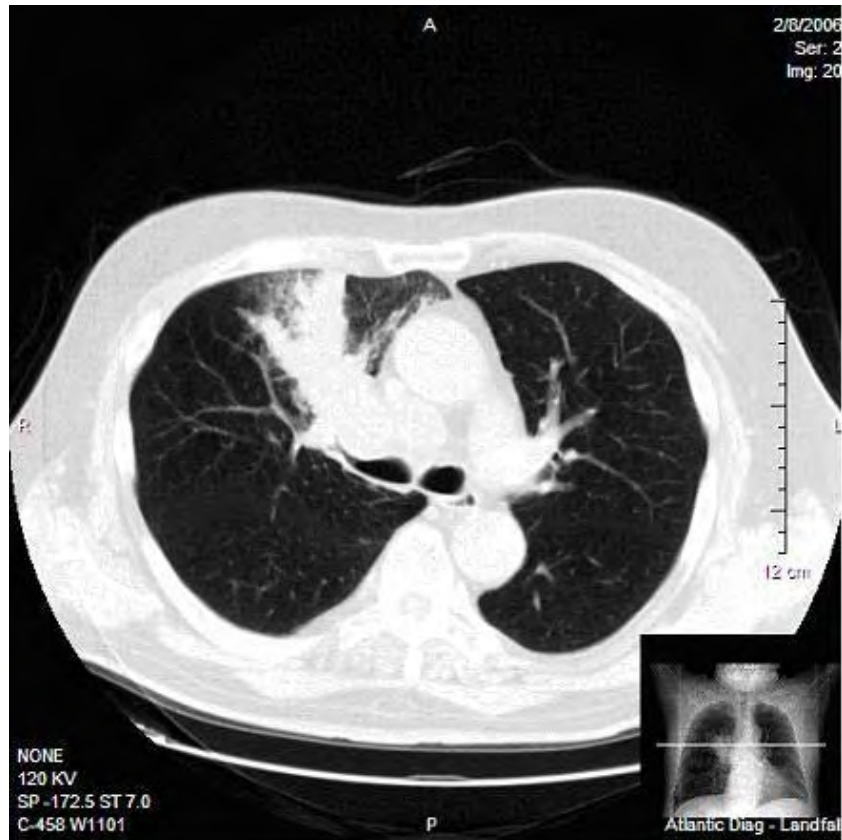


Figure 3. CT of the chest reveals a large mass in the right upper lobe of the lung with associated mediastinal and hilar adenopathy.



Figure 4. CT of the chest reveals cortical lucency, expansile destruction, and medullary invasion due to metastatic lung carcinoma affecting the right scapula.

Subsequent bone scintigraphy revealed abnormal increased accumulation of radiopharmaceutical along the lateral aspect of the right scapula (see Figure 5). MRI evaluation revealed additional metastatic foci including the cervical, thoracic and lumbar spinal regions as evidenced by multiple regions of decreased signal intensity are visualized on T1 weighted images (see Figures 6 and 7). Biopsy confirmed a primary lung carcinoma origin. Unfortunately, the patient succumbed to the disease within 3 months of its diagnosis.

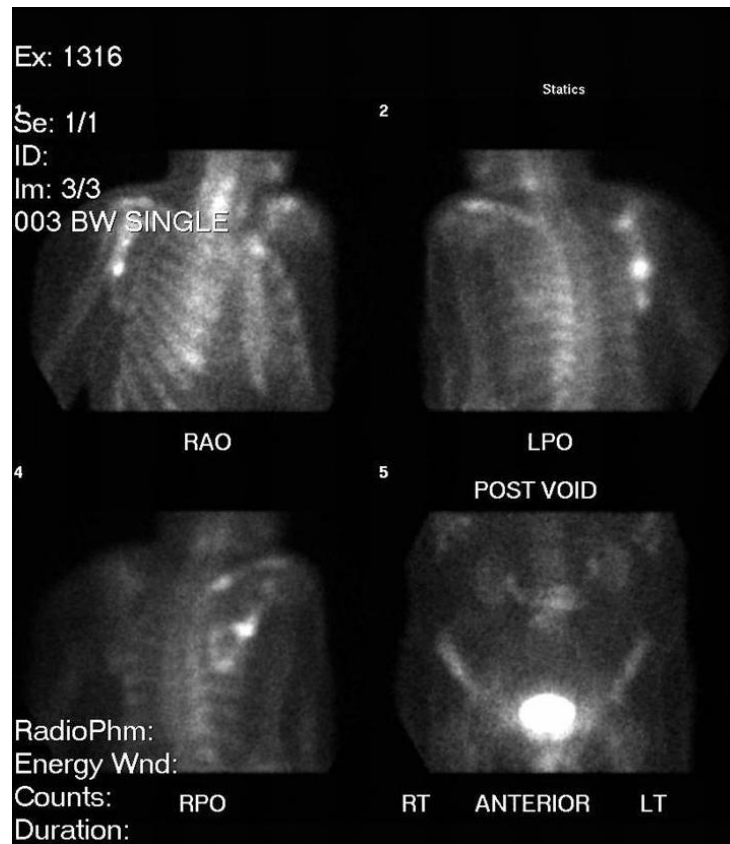


Figure 5. Bone scintigraphy of the right scapula reveals increased uptake where metastatic lung carcinoma is present.



Figure 6. MRI sagittal T1WI reveals scattered foci of decreased signal intensity reflective of metastatic disease affecting the cervical and thoracic spine regions.



Figure 7. MRI sagittal T1WI reveals scattered foci of decreased signal intensity reflective of metastatic disease affecting the thoraco-lumbar spine.

Discussion

Chiropractic considerations

The identification of primary or secondary metastatic cancer requires careful consideration with regard to history and physical examination. A key objective for the chiropractic physician is to identify “red flags” as quickly as possible. This is especially true for any disease process that may weaken bone.

The application of directed force into spinal or osseous structures inherent to the chiropractic adjustment mandate careful evaluative procedure. Janse defined the adjustment as a specific form of articular manipulation using long or short lever techniques with specific contacts and is characterized by a dynamic thrust of controlled velocity, amplitude and direction [6].

While chiropractic physicians are challenged with the responsibility of attempting to identify relative and absolute contraindications to spinal adjustments, sometimes early onset, insidious and seemingly innocuous symptoms may delay early identification [7,8].

Clinical considerations

When primary cancer is not yet identified, metastatic extension to skeletal structures can at times be difficult to detect [7,8]. As was illustrated in this case, clinical considerations that may assist or delay the identification of metastatic bone disease include:

1. Early in the course of the disease progression, important red flag identifiers may not initially be present and can delay early identification.
2. Initial pain presentations may be suggestive of common clinical conditions that are less aggressive.
3. Patients may or not be aware of, or report, the existence of a primary cancer.
4. Pain can be initially mild to severe and is often progressive in nature and unremitting despite therapeutic interventions.
5. It is sometimes extremely difficult to positively identify metastatic disease due to complex clinical factors [7,8].

Red flag indicators for metastatic bone disease include: age over 50 or under 20 years, a history of cancer, constitutional symptoms including unexplained weight loss, pain worse at night or in atypical areas, no significant improvement after >1 month of conservative (non-invasive) care, pain that has no mechanical exacerbating or remitting factors, and severe disabling pain affecting a child or adolescent [9].

Diagnostic imaging considerations

Humphrey reported that about 25% of people with lung cancer do not have symptoms from advanced cancer when their lung cancer is found [10]. Maghfoor reported that 7-10% of patients with lung cancer are asymptomatic and their cancers are diagnosed incidentally after a CXR was performed for other reasons [11]. Numerous studies have shown that the chest radiograph lacks sensitivity in detecting mediastinal lymph node metastases and in detecting chest wall and mediastinal invasion [12].

CT has become the major imaging modality of choice in the evaluation of patients with bronchogenic carcinoma [13]. Traditionally, chest CT for staging of lung cancer is extended into the abdomen to include the adrenal glands. Whether this requires intravenous contrast material is debatable [13]. Patz et al. [14] concluded that contrast-enhanced CT extended to include the liver rarely adds to routine nonenhanced CT through the adrenal glands and does not influence management decisions.

The evaluation of the mediastinum with magnetic resonance imaging (MRI) is approximately equal to that of CT with regard to the staging of bronchogenic carcinoma and MRI is significantly more accurate for detecting direct mediastinal invasion [15]. Other studies have confirmed the usefulness of MRI, particularly in the evaluation of chest wall invasion and the local staging of superior sulcus tumors [16,17]. The general conclusion of these studies is that MRI has advantages in the assessment of both chest wall and mediastinal invasion [13].

Indications for the use of whole body positron emission tomography imaging in lung cancer using 18-fluorodeoxyglucose (FDG-PET) in patients with non-small cell lung cancer include high clinical index of suspicion of high grade malignancy and radiographic evidence of nodal enlargement [13]. In addition, PET scans may be helpful in centers where mediastinoscopy is not readily available and in patients with significant comorbid conditions who are borderline candidates for surgery, with locally advanced disease, solitary brain metastasis, and cases of local recurrence that might qualify for reoperation [18,19].

Bone scintigraphy in the detection of metastatic disease has significant limitations. Although it has high sensitivity, it is noted for having very low specificity that ranges from 50%-60% [13]. Bone scintigraphy should probably be limited to cases in which patients have specified clinical indicators of bone metastasis [20].

When evaluating suspected pulmonary metastasis, CXR and CT of the chest are rated by the American College of Radiology (ACR) scale as: “9 - most appropriate” (Rating Scale: 1-Least appropriate, 9-Most appropriate)

[21]. It is generally accepted that chest radiography, with posteroanterior (PA) and lateral views, should be the initial imaging test in patients without known or suspected thoracic metastatic disease [22-24]. Compared with chest radiography, CT is much more sensitive for detecting pulmonary nodules, because of its lack of superimposition and its high contrast resolution [22-24].

Conclusion

Lung cancer is a significant and aggressive primary cancer with a predilection for skeletal metastasis. When primary lung cancer is not previously identified, metastatic disease to skeletal structures may initially manifest as musculoskeletal complaints. Careful diagnostic evaluation and decision making may allow for earlier diagnosis.

Competing interests

The authors declare no competing interests.

Authors' contributions

JD conceived the study and drafted the manuscript. GJD participated in the care of the patient and provided data related to the case. Both authors read and approved the final manuscript.

Acknowledgements

Written informed consent was obtained from the decedent's wife for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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References

1. American Cancer Society: *Cancer Facts and Figures*. Atlanta; 2007.
2. U.S. Cancer Statistics Working Group. *United States Cancer Statistics: 2004 Incidence and Mortality*. Atlanta (GA): Department of Health and Human Services, Centers for Disease Control and Prevention, and National Cancer Institute; 2007.
3. Beckles MA, Spiro SG, Colice GL, Rudd RM: **Initial evaluation of the patient with lung cancer - symptoms, signs, laboratory tests, and paraneoplastic syndromes**. *Chest* 2003, **123**:97S-104S.
4. Manga P: **Economic case for the integration of chiropractic services into the health care system**. *Journal of Manipulative & Physiological Therapeutics* 2000, **23**(2):118-22.
5. Dagenais S, Haldeman S: **Chiropractic**. *Clinics in Office Practice* 2002, **29**(2):419-37.
6. Haldeman S: **Appendix B – Chiropractic glossary of commonly used terms**. In *Principles and Practice of Chiropractic*. 2nd edition. Edited by Haldeman S. Connecticut: Appleton & Lange; 1992:621-628.
7. Demetrious J: **Metastatic testicular seminoma of the cervicothoracic spine**. *Journal of Manipulative and Physiologic Therapeutics* 1992, **15**(8):525-528.
8. Demetrious J: **Renal cell carcinoma of the sternum and spine that mimicked costochondritis**. *Journal of the Neuromusculoskeletal System* 1995, **3**(1): 16-19.

9. Murphy DR, Letz G, Morris CE: **Red flags for serious disease in low back syndromes.** In *Low Back Syndromes: Integrated Clinical Management*. Edited by Morris CE. United States of America: McGraw-Hill; 2006:277-286.
10. Humphrey LL, Teutsch S, Johnson MS: **Lung cancer screening with sputum cytologic examination, chest radiography, and computed tomography: an update for the US Preventive Services Task Force.** *Annals of Internal Medicine* 2004, **140**:740-753.
11. **Lung Cancer, Non-Small Cell.** Maghfoor I. [<http://www.emedicine.com/med/topic1333.htm>]. Accessed June 21, 2008.
12. Bragg DG: **The diagnosis in staging of primary lung cancer.** *Radiol Clin North Am* 1994, **32(1)**:1-14.
13. American College of Radiology: **ACR Appropriateness Criteria® screening for staging of bronchogenic carcinoma.** Virginia; 2005.
14. Patz EF Jr, Erasmus JJ, McAdams HP, Connolly JE, Marom EM, Goodman, PC, Leder RA, Keogan MT, Herndon JE: **Lung cancer staging and management: comparison of contrast-enhanced and nonenhanced helical CT of the thorax.** *Radiology* 1999, **212(1)**:56-60.
15. Webb WR, Gatsonis C, Zerhouni EA, Heelan RT, Glazer GM, Francis IR, McNeil BJ: **CT and MR imaging in staging non-small cell bronchogenic carcinoma: report of the Radiologic Diagnostic Oncology Group.** *Radiology* 1991, **178(3)**:705-713.
16. Heelan RT, Demas BE, Caravelli JF, Martini N, Bains MS, McCormack PM, Burt M, Panicek DM, Mitzner A: **Superior sulcus tumors: CT and MR imaging.** *Radiology* 1989, **170(3 Pt 1)**:637-641.
17. Padovani B, Mouroux J, Seksik L, Chanalet S, Sedat J, Rotomondo C, Richelme H, Serres JJ: **Chest wall invasion by bronchogenic carcinoma: evaluation with MR imaging.** *Radiology* 1993, **187**: 33-38.
18. Weder W, Schmid RA, Bruchhaus H, Hillinger S, von Schulthess GK, Steinert HC: **Detection of extrathoracic metastases by positron emission tomography in lung cancer.** *Ann Thorac Surg* 1998, **66(3)**:886-893.
19. Detterbeck FC, Falen S, Rivera MP, Halle JS, Socinski MA: **Seeking a home for a PET, part 2: Defining the appropriate place for positron emission tomography imaging in the staging of patients with suspected lung cancer.** *Lancet* 2004, **125(6)**:2300-2308.
20. Michel F, Soler M, Imhof E, Perruchoud AP: **Initial staging of non-small cell lung cancer: value of routine radioisotope bone scanning.** *Thorax* 1991, **46(7)**:469-473.
21. American College of Radiology: **ACR Appropriateness Criteria® Screening for Pulmonary Metastases Primary malignancy: head and neck carcinoma.** Virginia; 2006.
22. Davis SD: **CT evaluation for pulmonary metastases in patients with extrathoracic malignancy.** *Radiology* 1991, **180(1)**:1-12.
23. Snyder BJ, Pugatch RD: **Imaging characteristics of metastatic disease to the chest.** *Chest Surg Clin N Am* 1998, **8(1)**:29-48.
24. Herold CJ, Bankier AA, Fleischmann D: **Lung metastases.** *Eur Radiol* 1996, **6(5)**:596-606.

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A PUBLICATION FOR REFERRING CLINICIANS OF CENTER FOR DIAGNOSTIC IMAGING

**SPECIAL
EDITION**

INSIDE THIS ISSUE

Dr. Tom Gilbert, spine radiologist, discusses quantitative criteria for grading lumbar spinal stenosis (LSS) on CT and MRI. Dr. Gilbert reports that the reliability and reproducibility of a test can be improved by using well-defined criteria and definitions for pathologic entities. Incorporating these criteria improves inter-reader consistency among CDI's radiologists, enhancing the quality of the diagnostic reports provided to referring physicians.



Thomas J. Gilbert M.D., M.P.P.,
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Measures of Lumbar Spinal Stenosis at CDI

Thomas J. Gilbert M.D., M.P.P., Spine Radiologist

The utility of a diagnostic test in clinical practice is determined by several factors. The most commonly addressed factor is accuracy, which is expressed in terms of sensitivity and specificity. Reliability is also an important factor and reflects the reproducibility of an interpretation by the same radiologist at different times (intra-observer reliability), and the reproducibility of an interpretation by different readers (inter-observer reliability).

The reliability or reproducibility of a test can be improved by using well-defined criteria and definitions for pathologic entities. In order to improve the reproducibility of readings at Center for Diagnostic Imaging (CDI), we have incorporated quantitative criteria for the grading of lumbar spinal stenosis (LSS) on CT or MRI.

These authors proposed that a dural sac area (DSA) of 100 mm² is unequivocal evidence of central canal stenosis. Hamanishi et al. reported that a decrease in the dural sac area to below 100 mm² at two or more levels was highly associated with the presence of intermittent claudication.² Schonstrom and Hansson showed that a decrease in the dural sac area to below 75 mm² results in a significant increase in pressure on the cauda equina at L3-4.³ Based on these studies, the threshold for mild central canal stenosis was set at 100 mm² and 75 mm².

In this scheme, increasing severe grades of LSS have a greater likelihood of being clinically significant. Ogikubo et al. in a 2007 cohort study showed that the mean dural sac area at the most constricted level

(continued on page 2)

Central Canal Stenosis

The grading of lumbar central canal stenosis is based on measures of dural sac cross-sectional area. With respect to central stenosis, measures of the dural sac are more meaningful than are measures of the bony canal. Bolender et al. demonstrated that the effectiveness of CT was improved by using the AP diameter, or dural sac cross-sectional area, rather than measures of the bony canal.¹

TABLE 1

**Grading of Lumbar Central Spinal
Stenosis on MRI and CT (Figures 1,2):**
(DSA - dural sac area)

Mild	DSA	75 mm ² - 100 mm ²
Moderate	DSA	50 mm ² - 75 mm ²
Moderately Severe	DSA	25 mm ² - 50 mm ²
Severe	DSA	0 mm ² - 25 mm ²

FIGURE 1



FIGURE 2a

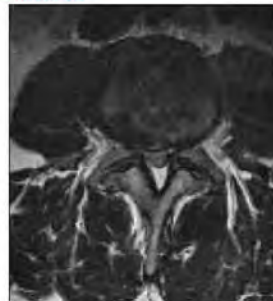


FIGURE 2b



Figure 1: Moderate central canal stenosis (DSA 73 mm²) on CT myelography. Figure 2: Moderately severe central canal stenosis with a dural sac area of 45 mm² (2a), and a dural sac area of 30 mm² (2b).

(continued from page 1)

was a strong predictor of preoperative walking ability, back and leg pain, and of the health-related quality of life in patients with central lumbar spinal stenosis. For patients with a walking ability of <100 m, the average DSA was 53 mm²; for those with a walking ability of 100-500 m, 55.8 mm²; and for those with a walking ability of >500 m, 68.8 mm².⁴

Foraminal Stenosis

Because of the large variability in the size of the normal foramina, any grading system for foraminal stenosis should measure the canal relative to the size of the neural elements. Reports of foraminal stenosis should also include some mention of perineural effacement, neural displacement and neural compression. At CDI, we utilize the criteria detailed in Table 2.

TABLE 2

Measures of Foraminal Stenosis (Figures 3,4):

Mild stenosis:	>50% narrowing of the foramen; no ganglionic impingement.
Moderate stenosis:	Area 25%-50% larger than the ganglion. May have partial effacement of perineural fat. No ganglionic compression.
Moderately severe:	Area 25-50% smaller than the ganglion. Moderate ganglionic compression.
Severe:	Marked ganglionic compression.

FIGURE 3



FIGURE 4

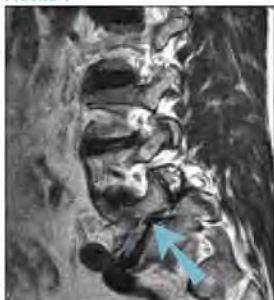


Figure 3: Moderate foraminal stenosis at L5-S1 on sagittal T2-weighted MRI (arrow). Figure 4: Severe up-down foraminal stenosis at L5-S1 with marked ganglionic compression.

Conjoined nerve roots, low take off of the nerve root, and anomalous nerve root anatomy should be highlighted as these anatomic variants can alter the position of the nerve root within the foramen, and can increase the likelihood of symptomatic foraminal stenosis. The type of stenosis (AP or up-down) and location of impingement within the foramen (medial, lateral, far lateral) should also be mentioned.

Subarticular Recess (SAR) Stenosis (Figure 5)

In many studies, the degree of subarticular recess stenosis is assessed at the tip of the superior articular process. As with foraminal stenosis, however, the size of the recess is only important relative to its impact on the exiting nerve root.

At CDI, we use a qualitative measure of subarticular recess stenosis (mild, moderate or severe) with a detailed description of nerve root position and impingement. Important variables include the take-off of the nerve root sleeve relative to subarticular recess, nerve root displacement and nerve root compression. The nerve root is more constrained within the nerve root sleeve and is more susceptible to symptomatic impingement. Symptoms are much less common when the nerve root exits the dural sac distal to the lateral recess as the nerve root is more mobile within the dural sac.

FIGURE 5

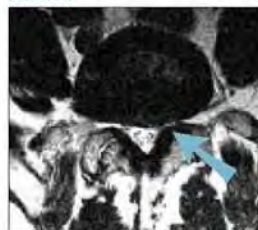


Figure 5: Moderate subarticular recess stenosis (arrow) with impingement on the traversing L5 nerve root on axial T2 MRI image.

Conclusion

The purpose of these guidelines is to improve inter-reader consistency within our group, and to improve the quality of communication with our referring physicians, not to define clinically significant disease. The diagnosis of stenosis requires or presumes the presence of radiculopathy or neurogenic intermittent claudication. The correlation of clinical symptoms with findings on MRI or CT is critical to the evaluation of a patient. On occasion, selective injections may be needed to determine the significance of findings on imaging tests in a particular patient.

References

1. Bolander, N. F., Schonstrom, N. S., et al. "Role of computed tomography and myelography in the diagnosis of central spinal stenosis." *J Bone Joint Surg Am* 67.2 (1985): 240-6.
2. Hamanishi, C., Matukura, N., et al. "Cross-sectional area of the stenotic lumbar dural tube measured from the transverse views of magnetic resonance imaging." *J Spinal Disord* 7.5 (1994): 388-93.
3. Schonstrom N., Hansson T. "Pressure changes following constriction of the cauda equina. An experimental study in situ." *Spine* 4 (1988): 385-8.
4. Ogikubo O, Forsberg L, Hansson T. "The relationship between the cross-sectional area of the cauda equina and the preoperative symptoms in central lumbar spinal stenosis." *Spine* 13 (2007): 1423-28.

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Editorial Comments

Dr Warren Jahn wrote:

Editor's (JAHN) comments:

The chiropractic orthopedist must be diligent in assessing vascular conditions that may manifest as a MSK one. Do not be lulled to sleep by the Cassidy et al paper stating "We found no evidence of excess risk of VBA stroke associated chiropractic care compared to primary care." The patient still needs to be appropriately evaluated prior to rendering manipulation. The following two studies highlight this.

Posterolateral protrusion of the vertebral artery over the posterior arch of the atlas: quantitative anatomical study using three-dimensional computed tomography angiography

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/Abbreviations used in this paper:/ CVJ = craniovertebral junction; PICA = posterior inferior cerebellar artery; VA = vertebral artery.

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Object

The vertebral artery (VA) often takes a protrusive course posterolaterally over the posterior arch of the atlas. In this study, the authors attempted to quantify this posterolateral protrusion of the VA.

Methods

Three-dimensional CT angiography images obtained for various cranial or cervical diseases in 140 patients were reviewed and evaluated. Seven patients were excluded for various reasons. To quantify the protrusive course of the VA, the diameter of the VA and 4 parameters were measured in images of the C1–VA complex obtained in the remaining 133 patients. The authors also checked for anomalies and anatomical variations.

Results

When there was no dominant side, mean distances from the most protrusive part of the VA to the posterior arch of the atlas were 6.73 ± 2.35 mm (right) and 6.8 ± 2.15 mm (left). When the left side of the VA was dominant, the distance on the left side (8.46 ± 2.00 mm) was significantly larger than that of the right side (6.64 ± 2.0 mm). When compared by age group (? 30 years, 31–60 years, and ? 61 years), there were no significant differences in the extent of the protrusion. When there was no dominant side, the mean distances from the most

protrusive part of the VA to the midline were 30.73 ± 2.51 mm (right side) and 30.79 ± 2.47 mm (left side). When the left side of the VA was dominant, the distance on the left side (32.68 ± 2.03 mm) was significantly larger than that on the right side (29.87 ± 2.53 mm). The distance from the midline to the intersection of the VA and inner cortex of the posterior arch of the atlas was ~ 12 mm, irrespective of the side of VA dominance. The distance from the midline to the intersection of the VA and outer cortex of the posterior arch was ~ 20 mm on both sides. Anatomical variations and anomalies were found as follows: bony bridge formation over the groove for the VA on the posterior arch of C-1 (9.3%), an extracranial origin of the posterior inferior cerebellar artery (8.2%), and a VA passing beneath the posterior arch of the atlas (1.8%).

Conclusions

There may be significant variation in the location and branches of the VA that may place the vessel at risk during surgical intervention. If concern is noted about the vulnerability of the VA or its branches during surgery, preoperative evaluation by CT angiography should be considered.

Vertebral Artery Anomaly With Atraumatic Dissection Causing Thromboembolic Ischemia: A Case Report.

Case Reports

Spine. 25(15):1989-1992, August 1, 2000.

/ Jackson, R. Sean MD; Wheeler, Anthony H. MD; Darden, Bruce V. II MD /

* Abstract:*

Study Design. A case report is presented.

Objectives. To illustrate a rare cause of atraumatic vertebral artery dissection resulting from anomalous entry of the vessel at the C3 transverse foramen induced by normal physiologic head and neck motion, and to review vertebral artery anatomy and mechanisms whereby it is vulnerable to pathologic compression.

Summary of Background Data. The vertebral artery usually enters the transverse foramen at C6. Rarely, the artery enters at C5 or C4. Only one prior case with entry at C3 has been reported. That patient experienced recurrent quadriplegia and locked-in syndrome caused by vertebral artery obstruction. A 27-year-old woman with a history of classic migraine experienced neurologic symptoms on three occasions related to physiologic neck and arm movements. Magnetic resonance angiogram was not diagnostic, but standard arteriography demonstrated anomalous vertebral artery entry into the C3 transverse foramen and focal dissection.

Methods. Pertinent literature and the patient's history, physical examination, and radiologic studies were reviewed.

Results. Standard cervico-cerebral arteriogram demonstrated focal dissection at C4 and thromboembolic complications in distal vertebral and basilar arteries. Initially, diagnosis by magnetic resonance angiogram was illusive. However, arteriography allowed prompt diagnosis followed by anticoagulation with resolution of neurologic symptoms.

Conclusions. Vertebral artery dissection without trauma is rare, but should be considered when neurologic symptoms accompany physiologic cervical movements. For cases in which vertebrobasilar thromboembolic ischemia is suspected, magnetic resonance angiogram may prove inadequate for demonstrating the causative vascular pathology. Therefore, standard cervico-cerebral arteriography should be performed.

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Current Events

Northwestern Health Sciences is in the process of obtaining certification of the new Masters program, Master of Science: Physical Medicine and Rehabilitation (MSc: PM&R). The letter of intent was received by the Academy in April 2008.

The Coalition of Orthopedists welcomed the Texas Council of Chiropractic Orthopedics to the Coalition group April 2008.

Academy Chiropractic Orthopedic Diplomate examination at Texas College of Chiropractic (TCC) May 2008 resulted in 5 new Diplomates of the Academy.

Attribution

Kate Hentges, FCER