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Yellowstone River from Atop the Lower Falls in August 2011

Courtesy of Dr. A. Michael Henrie

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Independent Research - Case Study

SCAPULOCOSTAL BURSITIS - A CONDITION OFTEN OVERLOOKED: A CASE STUDY

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ABSTRACT

Purpose: To help the clinician recognize and successfully treat this condition. Scapulocostal bursitis is often overlooked or misdiagnosed. History, examination and treatment of this condition will be outlined. Properly treated, this condition responds favorably to conservative chiropractic management.

Anatomy: An understanding of the anatomy and physiology of the scapulothoracic articulation is required to understand the pathogenesis of scapulothoracic disorders. The scapula is a triangular-shaped bone articulating with the posterior thorax. It is attached to the axial skeleton by only the acromioclavicular joint, and therefore its stability is dependent on surrounding musculature.

The periscapular musculature creates stability of the scapulothoracic articulation. The levator scapulae and rhomboids attach to the medial border of the scapula, whereas the subscapularis is on its anterior surface. (1) The serratus anterior originates on the ribs and inserts on the medial scapular anterior surface. A cushion between the scapula and the thoracic wall is created by the serratus anterior and the subscapularis. Two spaces, the subscapularis space and the serratus anterior space, are created by the musculature of the joint. The serratus anterior space is located between the chest wall, serratus anterior, and rhomboids. The subscapularis space is bounded by the serratus anterior, subscapularis, and axilla. Three muscles of the rotator cuff originate at the scapula: the supraspinatus and the infraspinatus on the posterior surface of the scapula and the subscapularis on the anterior surface. (4) Seventeen muscles have their origin or insertion on the scapula making it the command center for coordinated upper extremity activity. A number of muscles secure the scapula to the thorax, including the rhomboids major and minor, the levator scapula, serratus anterior, trapezius, omohyoid and pectoralis minor. (3, 9)

There are several important neurovascular structures surrounding the scapula. The accessory nerve goes through the levator scapulae muscle near the superomedial angle of the scapula and runs along the medial scapular border deep to the trapezius muscle. The transverse cervical artery branches into the dorsal scapular artery (deep branch) and a superficial branch that travels with the accessory nerve. The dorsal scapular artery travels with the dorsal scapular nerve 1 cm medial to the medial border of the scapula. They pierce the scalenus medius and travel deep to the rhomboid major and minor. The nerve innervates both of these structures. The long thoracic nerve is located on the surface of the serratus anterior. The suprascapular nerve and artery pass toward the suprascapular notch on the superior scapular border medial to the base of the coracoid

Several scapular bursae have been implicated in the development of scapular bursitis, which can lead to pain and snapping. Bursae are located in areas of friction and are potential spaces lined by a synovial membrane. Two major bursae are found consistently in patients: the infraserratus bursa located between the serratus anterior and the chest wall and the supraserratus bursa located between the subscapularis and serratus anterior. (3, 4, 9) Scapulothoracic movements are of a gliding nature and occur at an interface between the ventral surface of the scapula and the rib cage. The contacting surfaces involve the subscapularis and bare areas of the scapula with the serratus anterior overlying the second through seventh ribs. Normally the scapula is set obliquely on the thorax at an angle of 30°. (6)

Methods: A 36-year-old Caucasian female presented for care and treatment of chronic upper back pain and a burning sensation in the area. It had been getting more intense and frequent over the past two years. She had increasing upper posterior arm pain that did not radiate below the elbow and an ache just below the clavicle and adjacent to the humeral head. Imaging studies read by a chiropractic radiologist of the neck and thoracic spine revealed spondylosis at C5-C6 and C6-C7 with moderate disc space narrowing at C5-C6. She had a slight right dorsal scoliosis. The upper lung field on the left was negative.

The patient was treated with active chiropractic manipulative therapy (CMT) at C6 and T5. Ultrasound and EMS was applied to the upper back and scapulocostal bursa. Elastikon tape was placed over the left scapula. Cryotherapy was outlined for self-care. Exercises were given to the patient when the acute phase subsided. Visual analog scale (VAS) was used to measure her response.

Results: The patient presented with classic scapulocostal bursitis. She responded to the treatment and was pain-free for the first time in two years. She received active CMT to the lower neck and upper back. In addition, ultrasound was applied to the bursa area and EMS to the lower cervical and mid-thoracic paraspinals. Elastikon tape was applied twice during the treatment. Cryotherapy was used for the first 48 hours and then discontinued. Upper thoracic stretches and lower cervical exercises were given after the second visit. She was treated a total of four sessions.

The presenting complaints were treated for numerous diagnostic presentations prior to admittance to this treatment facility. This included the following: "pinched nerve", muscle spasms, subluxations and muscle strain. Obtaining a thorough history and understanding her job requirements were instrumental in arriving at the causation of her complaints.

Background

This condition is not well understood and should be considered in any presentation of lower neck, upper back pain, paresthesias medial to the scapula, anterior and posterior shoulder discomfort without range of motion restriction and upper extremity pain that does not radiate below the elbow (2).

Case Presentation

A 36-year-old Caucasian female presented to the office with a two year history of increasing upper back and lower neck discomfort. Pain was present into the posterior aspect of the left upper arm and chest region. She described an ache and burning in the upper back next to the spine on the left. She works as a Registered Nurse (RN) with primarily administrative duties. Her symptoms are worse when she has the telephone trapped between her neck and top of the left shoulder while working with her computer entering data. She has pain and stiffness arising in the morning and does not sleep well because of her pain. She has tried various pain and anxiety medications, physical therapy and adjustments with a hand held (mechanical) device to the spine. She had a motor vehicle accident four years prior and her left shoulder hit the door frame. She had a brief course of medical care and she reported that her symptoms resolved.

Her pain severity scale was 7/10 on the day of the initial examination. Her Oswestry back index was 17/50 or 34%. Her height was 66 inches and her weight was 115 lbs. She was afebrile with a pulse rate of 66 per minute and a respiration rate of 14 per minute. The blood pressure on the left was 110/68, and on the right was 102/64 at 1400 hours. LMP: Two months ago, she recently had an ablation. She demonstrated a flattening of the cervical lordosis. A review of the HEENT was unremarkable. Optic disc margins were sharp and clear. Point tenderness was +1 (0/+4 scale) at C5/C6 on the left. Cervical distraction was negative. Cervical flexion (90) 60°, extension (70) 50°, left lateral bending (45) 30°, right lateral 35°, left rotation (90) 60° and right rotation

55°. Muscle stretch reflexes were +2/+2. No motor or sensory changes were noted. Triad of Dejerine was negative. Her left shoulder range of motion was unencumbered.

Internal and external rotation was adequately accomplished. Auscultation of the thorax was unremarkable. Point tenderness was +1 on the left side T4-5. She had +2 pain at the superior medial aspect of the left scapula. She exhibits the classic "jump sign" when the bursa area was palpated. She commented that it felt "like the pain I get". There was +1 spasm in the rhomboid muscle on the left. Myofascial trigger points were was present in the rhomboid and subscapularis muscles on the left. The approximation test was negative for upper thoracic nerve root problems. (2) The shoulder examination was unremarkable. A 3 view cervical and a two view thoracic imaging studies were ordered. No prior imaging studies of any kind were taken of this area. With the failure of treatment, arm pain and difficulty sleeping these were appropriate studies. The films were read by a chiropractic radiologist. He reported that they were unremarkable for the exception of moderate degenerative changes at C5-C6 and to a lesser extent C6-C7. There was a very slight right dorsal scoliosis.

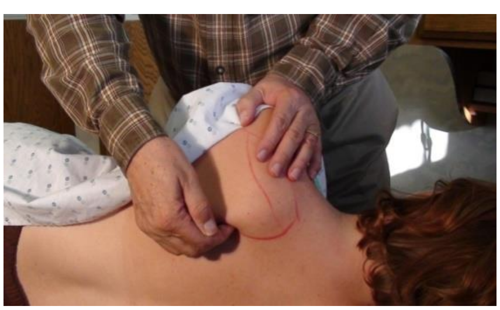
Treatment consisted of active CMT to C6 and T5. Diversified technique was used to the two areas of biomechanical dysfunction. Manual myofascial treatment to the trigger points, and Ultrasound was applied to the scapulocostal bursa *(Figure 1).* The left arm needs to be placed on the chest with the hand on the right shoulder. This exposes the bursa as it moves the scapula so the bursa could be treated.

Figure 1 *- Ultrasound being applied to the superior scapulocostal bursa. The left hand is placed on the opposite shoulder to open the space between the scapula and the rib cage. The ultrasound head is directed at an angle towards the bursa and not flat against the back.*

Low volt EMS was applied to the rhomboid muscles for 10 minutes using the intermittent cycle. Adjustment to the scapula was also performed *(Figure 2).*

Figure 2 *- Scapular Adjustment*

a) The scapula is moved by gently rocking it back and forth several times. Be careful as aggressiveness may work against your goals for treatment.



b) The second part of the adjustment of the scapula is done by taking the medial boarder of the scapula and gently pulling it away from the rib cage. All treatment to the scapula is predicated on the comfort of the patient.

Elastikon tape was applied *(Figure 3)* to the left scapula.

Figure 3 - *Progression of applying the support to the posterior shoulder.*

a) In this example, the left hand is placed on the front of the right shoulder for taping of the left scapula. Anchor straps are used to prevent or limit peeling of the Elastikon strips.

b) Proceed from the medial to lateral aspect to complete the procedure.

The following demonstrates taping of a bilateral scapulocostal bursitis *(Figure 4)*:

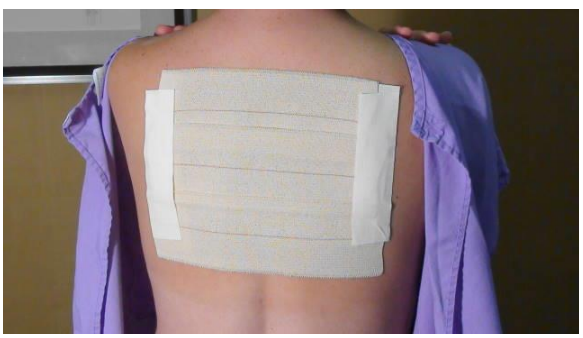


Figure 4 *- Bilateral Scapulocostal problems. The arms are crossed to tape the bilateral condition. The Elastikon should be applied from bottom to top of the scapula.*

The tape was to be left on until the next day's appointment. She was 50% better the next day and the same treatment was provided. Discussion was held about her pillow and sleep surface. She and her husband had been contemplating getting a new mattress. Time was spent discussing the various types of sleep surfaces. The third visit her pain was no more than 1/10 on the VAS scale. Elastikon tape was not applied and she was given standard range of motion (ROM) and isometric exercises for the lower neck and upper back. Additional exercises were outlined for the mid-back to include the rhomboids and subscapularis. The ergonomics of her work were also discussed and she was able to get a head set and a wrist support for her mouse pad.

A discussion about good posture while sitting to include obtaining the proper chair height resulting in the "90°/90°" sitting posture. This posture includes feet flat on the floor, knees, hips and elbows at 90° when at the computer. With a short person, a phone book may have to be placed under the feet. Her last appointment was 10 days later and she did not have a return of her symptoms. Her pain severity scale was 0-1/10. She reported that she had only a slight stiffness and was feeling no pain. She and her husband elected to get a sleep set with an air controlled firmness. She chose a pillow that is designed for side sleeping. This was the first time in two years she had been pain free in the upper back. There was a marked positive attitudinal change in her demeanor.

Discussion

The scapulocostal syndrome is a clinical syndrome characterized by pain and paresthesias over the medial border of the scapula that radiates into the neck, upper triceps, chest wall and the distal upper extremity. (1) The condition has also been called "snapping scapula", "washboard", scapulothoracic syndrome and scapulothoracic dissociation. (3, 4, 5, 9) Scapular winging has been identified in 50% of patients with a scapula without bony abnormalities. (2) There have been many factors that contribute to this syndrome as described in *Table 1*. (2, 3, 4, 5, 7, 10)

Table 1 - *Causes That Contribute to Scapulocostal Bursitis or Snapping Scapula*

Sports:
- Swimming, weight lifting, throwing and gymnastics

Posture:
- Kyphosis
- Scoliosis
- Posture of daily living to include work and sleeping

Bone:
- Rib or scapula osteochondroma
- Poor union of a rib fracture
- von Luschka's tubercle (superior medial angle)
- Skeletal exostosis
- Surgical resection 1st rib, cervical rib, breast implants

Work-related:
- Computer use
- Telephone cradled between neck and shoulder
- Repetitive activity above shoulder height (construction and factory)
- Chronic strain

Infectious and soft tissue:
- Lyme disease
- Tuberculosis
- Syphilis
- Infectious arthritis
- Villonodular synovitis
- Rheumatoid arthritis

Biomechanical:
- Abnormal scapular motion
- Myofascial trigger points of the shoulder and upper thoracic spine
- Injury to the long thoracic nerve
- Rotator cuff injury
- Direct trauma
- Osteoarthritis

Non-Neuromusculoskeletal Disorders:
- Pancoast tumor, Ischemic chest pain,
- Vertebral artery dissection
- Pneumonia, Peptic ulcer
- Dental Pain

The clinician should examine the scapula as a contributing factor to lower neck and upper back pain. Correct diagnosis and treatment of this condition may make the difference between failure of care and successful results. This current case is an example - multiple examinations and treatment without benefit. This condition is most often confused with cervical radiculopathy. The differential diagnosis is made easier as scapulocostal bursitis does not exhibit nerve root signs such as weakness and numbness. (2, 4)

Conservative chiropractic case management can be effective and dramatic in some cases of scapulocostal bursitis. The clinician must determine if there could be other contributing factors causing the complaints that need to be managed. (2)

Treatment of this patient included active diversified adjustments to C6 and T5 based upon the examination and imaging studies. Supportive modalities were used to the bursa and muscles. Myofascial trigger points were also treated manually and with modalities. Supportive Elastikon tape, exercises, ergonomic changes at work and the addition of an air support sleep set with the appropriate pillow have been successful in resolving the presenting complaints. Successful treatment must reduce and work to eliminate the cause. It has been the authors experience with this condition that it has the tendency to return if ergonomic changes are not made and patient compliance is lacking.

If the patient does not respond to conservative management, additional investigation or referral needs to be considered. *Table 1* outlines several other factors that could contribute to similar complaints. Medical management varies from oral pharmacology, steroid injections, physical therapy, rehabilitation and surgery. (2, 3, 4) These treatment protocols are outside the intent of this article, but it is the chiropractic clinician's responsibility to work with the care and treatment of their patients that do not respond to care and be able to discuss alternatives in treatment and co-manage problems if necessary.

Conclusion

With a good history and examination, this condition can be treated successfully and economically with chiropractic care. It is the application of the proper treatment plan and procedures that makes a dramatic and successful conclusion to either an acute or chronic manifestation of this condition. Understanding the differential diagnosis with this presentation is essential to minimize patient suffering and expense. If the patient does not respond to conservative management, additional investigation or referral needs to be considered. *Table 1* outlines several other factors that could contribute to similar complaints.

Competing Interests

The author has no competing interests.

Acknowledgments

The author has obtained consent from the patient for publication of this case report as well as the photographs.

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Abstracts & Literature Review

Characterization of a First Thoracic Rib Ligament - Anatomy and Possible Clinical Relevance

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Authors' Abstract:

Study Design: Cadaver dissection and measurement.

Objective: To describe a previously undocumented intracostal ligament that limits the potential space through which the T1 ventral ramus passes before joining the C8 ventral ramus.

Summary of Background Data: Preclavicular entrapment of the T1 ventral ramus can lead to radiculopathy, neurogenic thoracic outlet syndrome, or both, the so called "double crush" phenomenon. The usual sites of entrapment include the neural foramen, the interscalene interval, an aberrant cervical rib, the first rib itself, or an apical thoracic mass.

Methods: A total of 42 shoulders from 21 embalmed cadavers (13 male, 8 female) were dissected. The presence of the ligament was noted and its anatomic characteristics were measured with digital calipers by 3 independent investigators. Means, ranges, and standard deviations were calculated.

Results: The average ligament length was 31.0 mm (SD, 4.3). The ligament was trapezoidal in shape, and wider anteriorly. The mean anterior width was 7.1 mm (SD, 3.8), midsubstance width 3.6 mm (SD, 1.5), and posterior width 3.5 mm (SD, 1.3). The mean thickness was 0.5 mm (SD, 0.3), and the maximal opening through which the T1 nerve passed between the first rib and the ligament was 6.3 mm (SD, 1.6). The ligament was present on at least one side in 81% of individuals (67% of shoulders): 52% bilateral and 29% unilateral.

Conclusion: This previously undescribed ligament is a robust structure, present on at least one side in over 80% of the individuals studied. When present, the ligament creates a narrow interval between the ligament and the first rib that the T1 ventral ramus traverses before crossing the first rib superiorly and contributing to the inferior trunk of the brachial plexus. Although the actual clinical significance has not been demonstrated, this ligament may represent another entrapment site for the T1 ventral ramus.

Background

Thoracic outlet syndrome (TOS) is a compressive neuropathy that is commonly diagnosed and treated by chiropractors. TOS can be caused by the presence of a cervical rib, clavicular fracture, narrowing of the anterior/middle scalene space, as well as, other etiologies. The authors of this study are adding to the etiology list by suggesting that a first thoracic rib ligament (FTRL) can also cause TOS. During gross anatomic instruction of the thoracic region, the authors found an aberrant ligament (which, they called a FTRL) that proximally attached to the posteromedial aspect of the first rib and distally attached to the anterolateral aspect of the same rib opposite the scalene tubercle. The objective of this study, therefore, was to report the incidence and size of this ligament in a series of cadavers.

Methods

The sample included 21 embalmed cadavers (13 males and 8 females). A methodical dissection of both shoulders in each cadaver was undertaken, and the relationship of the FTRL to the C8 and T1 nerve roots was noted. The ligament was measured in several places using digital calipers and the data were analyzed with descriptive statistics.

Results

The FTRL was found on at least one side in 17 of 21 cadavers (81%). The ligament was present in 11 of 13 male cadavers (85%) and 6 of 8 female cadavers (75%). Mean length was 31 mm and mean thickness was 0.5 mm. The mean anterior, midsubstance, and posterior width was 7.1 mm, 3.6 mm, and 3.5 mm, respectively. The mean size of the opening through which the T1 ventral ramus traversed was 6.3 mm.

Conclusions

The incidence of the FTRL in this sample was over 80% and may have a role in the genesis of TOS. However, the claim by the authors that this ligament is “previously undocumented” is erroneous as this ligament has been previously described in the literature (for example, see Brantigan CO, Roos DB. Etiology of neurogenic thoracic outlet syndrome. Hand Clin. 2004, 20: 17-22).

Clinical Relevance

This study highlights another possible anatomic structure that may compress the T1 ventral ramus and cause TOS. Because clinicians may not be aware of the FTRL, this study has value in reminding us to think of all the anatomic possibilities that can cause TOS.

JACO Editorial Summary:

- The paper was written by physicians from the Department of Orthopaedic Surgery and Sports Medicine, Temple University School of Medicine, Philadelphia, PA.
- The purpose of the study was to describe and characterize what the authors called the FTRL.
- This ligament is actually called a Roos type 3 band and it has been previously described in the literature. In fact, there are 10 types of fibrous bands that have been well described by Roos (see Roos DB, Annest SJ, Brantigan CO. Historical and anatomic perspectives on thoracic outlet syndrome. Chest Surg Clin N Am. 1999, 9: 713-23).
- The FTRL (Roos type 3 band) has potential clinical relevance and the data provided in the paper are useful.

Summary

I commend the authors of this paper for contributing to the anatomic literature and highlighting another anatomic structure that can, in theory, cause TOS. The paper is well written and would be a good read for clinicians in practice. The dissections were well done and the illustration (Figure 1) is clear.

However, there are several methodologic issues that readers should be aware of when critically appraising this paper:

- The FTRL is not a novel structure as suggested by the authors. In fact, the FTRL is called a Roos type 3 band (see right-side of Figure 1B in the paper by Brantigan CO, Roos DB. Etiology of neurogenic thoracic outlet syndrome. Hand Clin. 2004, 20: 17-22). There are many variant bands in this region as described by Roos. Although the authors of the current paper did a nice job reviewing the clinical literature related to TOS in their discussion, they failed to adequately review the anatomic literature. Since this was an anatomic study, citations from peer-reviewed anatomy journals (e.g., Clinical Anatomy) should have been included in the references.
- If the authors believed that the FTRL was novel, then they should have performed a histologic analysis of the FTRL to provide evidence that it is a ligament.
- There is a typographic error in the caption of Figure 1 (page 2031): C7 ventral ramus is incorrect and should read C8. Fortunately, the text is correct in this regard.
- The cadaveric dissections depicted in Figures 2 through 5 should have superimposed rulers at the top or bottom of the figures so that readers can get a sense of the sizes of structures. Mentioning that the width of the probe tip is 2 mm is not adequate.

Abstracts & Literature Review

Thoracic Outlet Syndrome

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Authors' Abstract:

Purpose of review: Thoracic outlet syndrome continues to be a difficult clinical entity to diagnose and treat. This review aims to discuss the diagnosis, treatment, and clinical outcomes of thoracic outlet syndrome in the recent literature.

Recent findings: Clinical history, physical examination and diagnostic studies are important in the diagnosis of thoracic outlet syndrome. Recent reports question the utility of electrodiagnostic modalities (somatosensory evoked potentials and motor evoked potentials) in assisting with the diagnosis. Surgical decompression remains the definitive treatment, with good clinical outcomes ranging from 50–95%. Success rates of surgery are decreased in patients with symptoms present for longer than two years before surgery and in patients who have previously undergone surgical treatment of thoracic outlet syndrome.

Summary: Recognizing the signs and symptoms of thoracic outlet syndrome facilitates prompt diagnosis and treatment. Surgical decompression consisting of first rib resection and release of the scalene musculature can provide relief of the pain, weakness, and paraesthesias that define this complex syndrome. Postoperative range-of-motion exercises prevent scar formation and recurrence of symptoms.

Keywords: Brachial plexus compression, effort thrombosis, Paget-Schroetter syndrome, subclavian vein thrombosis, thoracic outlet syndrome.

Introduction

The complex of signs and symptoms caused by compression of the brachial plexus and subclavian vessels in the cervicoaxillary region is known as thoracic outlet syndrome (TOS). The diagnosis of TOS remains a challenge for the treating physician. Many different clinical entities can encompass the presenting symptoms of pain and paraesthesias in the affected extremity and a number of specialists may be called upon during the treatment of this complex entity. Koknel Talu (1) recommends that the diagnosis and treatment of TOS involve neurologists, physiatrists, family physicians, orthopedic surgeons, vascular surgeons, thoracic surgeons, neurosurgeons, and psychiatrists.

All should be familiar with the presenting signs and symptoms of TOS, and include the syndrome in their differential diagnosis. TOS typically presents with aching-type pain radiating from the scapula down the upper extremity, with associated numbness or tingling. It is common in women between 20 and 50 years of age (1). TOS is usually divided into three groups: vascular TOS, in those with compression of the subclavian vessels; neurogenic TOS, those with compression of the brachial plexus; and disputed neurogenic TOS. The diagnosis of TOS can be made by history, clinical examination, provocative tests, ultrasound, vascular studies, radiological evaluation, and electrodiagnostic evaluation. Initially, conservative treatment is offered to most patients. Definitive treatment involves surgical decompression of the related structures.

Risk Factors/Etiology

Anatomical variants are believed to play an important role in the cause of TOS. Bony factors such as a cervical rib, a long transverse process of the seventh cervical vertebrae, an anomalous first rib, or a clavicle fracture can compress the vital neurovascular structures near the cervical spine or in the axilla. Soft tissues such as congenital bands, ligaments, and tight scalene muscles can cause similar compression. Additional potential causes of TOS have also been described. Chon et al. (2) present an uncommon case of a 34-year-old woman with TOS of the right upper extremity secondary to a calcifying fibrous pseudotumor occurring in her thoracic outlet.

Ucerler et al. (3) described a muscular arch, which is most likely a congenital anomaly, in a male cadaver that could result in axillary pathology such as TOS. TOS may be exacerbated by certain positions of the arm. Fiorentini et al. (4) presented a case report of a hard-working 43-year-old racecourse farrier with primary subclavian vein thrombosis, also known as ‘effort thrombosis’ or Paget–Schroetter syndrome. The patient spent 75% of his time at work changing horseshoes, with his back bent greater than 70 degrees and his right shoulder flexed and abducted. This position is hypothesized to increase the pressure on the subclavian vein. The patient underwent successful surgical decompression with first rib resection and relief of his symptoms. Primary subclavian vein thrombosis is generally not recognized as a work-related disorder, but this proved to be the first report of “effort thrombosis” likely occurring secondary to occupational activities.

Diagnostic testing

The diagnosis of TOS is not always straightforward. Many disease processes may mimic the presenting symptoms of pain and paraesthesias in the upper extremity and few objective measurements may be available to make the clinical diagnosis. Furthermore, causes of weakness and fatigue in patients with TOS can be due to either neurogenic or vascular etiologies. A study by Ozcakar et al. (5) investigated differences in isokinetic strength measurements in 23 patients with TOS and 15 age-matched healthy controls. Patients were confirmed to have TOS if they exhibited weakness and fatigue with provocative maneuvers. Isokinetic measurements were used to assess the peak torque and fatigue ratio in the ulnar and median nerve distributions of the upper extremities. There was no difference between peak torque values in the TOS and asymptomatic controls or between the symptomatic and asymptomatic extremities of the same individuals. Fatigue ratios were found to be higher in the symptomatic sides at all velocities, but the differences reached statistical significance at 608/s and 1808/s. The authors concluded that TOS patients were found to have muscular performance similar to controls but the upper extremities developed fatigue more easily.

Seror (6) performed a prospective conduction study of the ulnar and medial antebrachial cutaneous nerves to evaluate the function of the lower brachial plexus in 100 women with known carpal tunnel syndrome (CTS). Amplitude ratios were measured and compared with 70 normal controls. Only one of the study subjects was found to meet the electrophysiologic criteria for TOS. The authors concluded that there is no appreciable link between CTS and TOS.

Seror (7) also evaluated the frequency of signs and symptoms suggestive of TOS in 100 upper limbs of women aged 60 years or less with unambiguous CTS. The author found no major signs or symptoms suggestive of TOS or neurogenic TOS in any limb studied. On the contrary, mild clinical signs and symptoms of disputed neurogenic TOS were frequently found despite the absence of a lower brachial plexus lesion on electrodiagnostic studies. The author concluded that CTS can easily be misdiagnosed as neurogenic TOS, and recommends electrodiagnostic conduction studies of the median, ulnar, and medial antebrachial cutaneous nerves to confirm the diagnosis before initiating treatment protocols.

Haghighi et al. (8) studied the utility of mixed nerve somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs) in two patients with neurogenic and vascular TOS. They found the cortical and cervical (C7) ulnar SSEPs demonstrated no change in latency of major peaks at rest and after abduction of the shoulder. The MEPs demonstrated significant decreases in amplitude after dynamic position of the arm in both cases, signifying neurovascular compression during elevation of the affected arm. No change in MEP latency was noted. The authors concluded that evoked potential studies are helpful in the diagnosis of neurogenic or vascular TOS. They found reduced amplitude of MEPs after elevation of the affected arm above the head, and return to baseline after normal positioning of the arm.

Rousseff et al. (9) also examined the utility of electrodiagnostic modalities in the diagnosis of TOS. They presented their results of electromyography, electroneurography, and somatosensory evoked potentials after ulnar nerve stimulation in 20 patients with surgically verified neurovascular compression at the thoracic outlet by a cervical rib or fibrous band. All complained of pain and paraesthesias in the hand, but only two had neurologic signs in a ‘pseudoulnar’ distribution. Anterior scalenectomy was performed with a successful outcome in 15 cases. Symptoms remained unchanged in four patients and worsened in one patient. Electrodiagnostic tests were normal in all 18 patients without neurologic signs. The authors concluded that electrodiagnosis is useless in confirming the presence of TOS, but it is very useful to exclude painful conditions requiring different treatment, such as CTS, cubital tunnel syndrome, and cervical neuropathies. The objective diagnosis of TOS remains a challenge.

Treatment Strategies

The definitive treatment for symptomatic TOS is surgical decompression. Clinical outcomes following surgery, however, have varied. Altobelli et al. (10) presented a retrospective cohort of patients evaluating the pattern of clinical results in patients with neurogenic TOS after operative decompression. The authors reported on 254 affected extremities in 185 patients with neurogenic TOS who underwent surgical decompression at the same institution from 1994–2002. Diagnosis of neurogenic thoracic outlet syndrome was confirmed by clinical evaluation, provocative clinical tests (Tinel’s sign, Adson’s sign, abduction and external rotation test, elevated arm stress test), electrophysiologic tests (SSEPs across the brachial plexus), anatomic studies, and provocative stimulation with anesthetic blocks of the anterior scalene muscle.

The patients were all treated by the same surgical protocol: transaxillary first rib resection and a lower scalenectomy (25%) for the primary procedure; with or without the subsequent upper scalenectomy (75%) through a supraclavicular approach for patients with persistent or recurrent symptoms. Evaluations included primary success, defined as uninterrupted success with no additional procedures performed on the operative side, and secondary success, defined as success maintained by an operation after failure of the primary procedure. Successful outcome was defined as 50% or greater improvement in subjective pain as determined by a 10-point scale, successful return to preoperative work status without the need for an additional procedure, or both. These results were analyzed using a standard life table analysis to evaluate the freedom from recurrent symptoms over time. Follow-up averaged 2–76 months (average 25 months).

Overall, the primary and secondary success rates were 46.5 and 64.2%, respectively. Eighty extremities underwent a secondary operation for the primary clinical failure. Of the 136 primary failures, 111 (81.6%) failed within the first 12 months, and 122 (89.7%) failed within 18 months. After 2 years, the failure rate plateaued. Thirteen complications were reported, including seven pneumothoraxes, three minor injuries to the subclavian vein, one minor injury to the long thoracic nerve, one case of the internal mammary artery being severed, and one suture granuloma that required an additional operation.

The outcomes were not significantly different between patients with unilateral and bilateral decompression operations. Three risk factors were used to divide the patients into three subgroups: workers' compensation, duration of symptoms greater than or equal to 2 years, and at least one previous surgery. Patients with a history of symptoms present for greater than 2 years had significantly reduced success rates following the primary procedure. A history of at least one previous surgery had significantly decreased success rates following secondary surgical procedures.

The authors admitted that some patients had favorable outcomes for 2–6 months following surgery, only to fail with recurrent symptoms at a later date. The initial success rate for primary procedures was 87% at 2 months, 53% at 12 months, 45% at 24 months, and 38% at 36 months. Long-term follow-up is therefore crucial, as recurrent symptoms may not occur until as a long as 18 months following surgical decompression. Short-term follow-up may mask the overall success rate of the procedures performed. Recurrences were attributed to scar tissue formation and the presence of a long posterior first rib stump. The authors recommended early active range-of-motion exercises and indefinite physical therapy to minimize the harmful effects of scar tissue formation.

Divi et al. (11) presented their results in 71 patients diagnosed with vascular TOS, 29 of whom also suffered from neurogenic TOS. The patients underwent a total of 73 operative procedures. A diagnosis of subclavian vein compression was made by contrast venography, duplex ultrasound, or other modalities. TOS decompression was performed via a supraclavicular approach. TOS decompression consisted of anterior scalenectomy, division of the middle scalene, and partial excision of the first rib from the vicinity of the vein followed by venolysis of the scar tissue. Patients with neurogenic symptoms also underwent brachial plexus neurolysis.

Patient follow-up consisting of an interview and physical examination varied from 1 to 60 months. Eighty-five percent of patients had pain improvement and 94% had improvement of paraesthesias following surgery. There was a significant difference in outcomes when comparing patients with venous TOS alone to patients with combined venous and neurogenic TOS. Patients with venous TOS showed a 93% return to full activity while patients with combined venous and neurogenic TOS returned to full activity 67% of the time. When subclavian vein compression or thrombosis is present, the authors recommended prompt initial treatment with lytic agents and anticoagulation followed by surgical decompression after a 3–6-week healing period. The supraclavicular surgical approach allows both complete resection of the anterior scalene muscle and satisfactory release by venolysis and rib resection.

Paget-Schroetter syndrome is a primary axillo-subclavian venous thrombosis that typically affects young male laborers. It is most often the consequence of a chronic compression of the subclavian vein at the level of the thoracic outlet. Presentation can be confirmed with a duplex Doppler study, and early diagnosis offers the opportunity for rapid venous recanalization with anticoagulation treatment or thrombolytic therapy (12). Once the thrombus has resorbed, surgical decompression of the thoracic outlet can be performed secondarily using techniques such as first rib resection.

Jakubietz et al. (13) presented a case report of a 54-year old woman who underwent staged bilateral first rib resections in treatment of constant numbness, tingling, and weakness exacerbated by sporting activity. Symptoms in both upper extremities improved following first rib resection. Postoperatively the patient sustained atraumatic fractures of the second rib, the first of which occurred at 3 weeks following surgery and the second at 2 months following surgery. The patient had a successful outcome with non-operative management of both fractures. The authors concluded that the second rib can experience increased stress that may predispose to fracture following first rib resection. Fracture of the second rib should be considered in patients developing chest pain following first rib resection.

Surgeons continue to explore new operative techniques for treatment of symptomatic TOS. Malliet et al. (14) described their experience of a combined endovascular and surgical approach for arterial TOS complicated by an aneurysm of the subclavian artery. They performed a retrospective review of three patients suffering from this clinical entity by the use of an endovascular stent-graft and decompression of the costoclavicular space by open first rib resection. All patients were free of symptoms without late complications at a mean follow-up of 37 months. The authors suggested that this treatment protocol is an attractive alternative to the conventional surgical approach for arterial TOS.

Conclusion

Thoracic outlet syndrome remains a difficult clinical entity to diagnose and treat. It may result from vascular or neurogenic etiologies. Diagnosis must be made by a combination of history, clinical evaluation, provocative maneuvers, radiographs, and vascular and electrodiagnostic studies. The diagnosis must be in the differential of any patient with activity-related arm pain, weakness, and paraesthesias. SSEPs and MEPs may assist with the diagnosis, or may rule out other potential causes. They may not, however, be as reliable in the diagnosis as once believed. Prompt diagnosis and surgical treatment provide patients with the highest probability of complete recovery. Thrombolysis should be followed by decompression with first rib resection. Postoperative range-of-motion exercises and commitment to a structured physical therapy protocol offer the best chance for successful outcome.

Clinical Relevance

This article is of value in understanding the surgical approach to treatment of TOS. Unfortunately it does a poor job of addressing the investigative approaches to TOS and fails to address and conservative care. To date, the surgical approach is still considered a last approach when all else fails.

Reviewer’s JACO Editorial Summary:

- This review article was written by medical doctors from the University of Maryland, Medical Centre in Baltimore, MD. Dr. James C. Dreese (corresponding author) is an Orthopedic Surgeon from Baltimore, MD who graduated 15 years ago.
- The purpose of the article was to discuss the diagnosis, treatment and clinical outcomes of TOS in the recent literature.
- From a surgical perspective we can see the approaches used for invasive treatment.
- Unfortunately this paper fails to address the appropriate investigations including orthopedic testing and appropriate electrodiagnostics.
- There is no relevant or practical discussion to alternative treatments which most Orthopedists understand. Conservative approaches with manual therapies are effective and have been studied.
- The 3 basic types of TOS are neurogenic, venous and arterial.
- The National Institute of Neurological Disorders and Stroke recommends as follows: “Treatment begins with exercise programs and physical therapy to strengthen chest muscles, restore normal posture, and relieve compression by increasing the space of the area the nerve passes through. Doctors will often prescribe non-steroidal anti-inflammatory drugs (such as naproxen or ibuprofen) for pain. Other medicines include thromobolytics to break up blood clots and anticoagulants to prevent clots. If this

does not relieve pain, a doctor may recommend thoracic outlet decompression surgery to release or remove the structures causing compression of the nerve or artery."
- The majority of individuals with TOS will improve with exercise and physical therapy.
- From a surgical perspective this is an interesting article. From a musculoskeletal perspective, this paper places partisanship to invasive procedures and fails to inform readers as to other approaches.

Reviewer's Commentary:

1. This abstract alludes to surgical intervention as the definitive treatment for TOS. This in itself provides a biased approach to treatment. There is no mention of appropriate orthopedic testing with sensitivity and specificity.
2. The introduction omits the role of manual therapists in the treatment of TOS.
3. There is an emphasis on a single case report versus a more global approach to TOS in the etiology segment.
4. It is interesting to note that the authors found electrodiagnosis as useless in confirming TOS yet do not state why.
5. The opinion rendered in this paper is that the definitive treatment for TOS is surgical decompression. The literature does not bear this out. There is no mention as to the specificity and sensitivity of the orthopedic tests such as Tinel's, Adson's, etc.
6. The conclusion states that diagnosis of TOS is difficult - agreed.

Summary

This is an informative review on surgical procedures for TOS. Unfortunately, it fails to appropriately address investigative and treatment procedures that are non-surgical yet effective. The literature to date bears this out.

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Abstracts & Literature Review

Provocative F Wave in the Diagnosis of Nonspecific Neurogenic – Type Thoracic Outlet Syndrome

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Authors' Abstract:

Objective: Thoracic outlet syndrome (TOS) is defined as a constellation of clinical symptoms caused by the entrapment of neurovascular structures (subclavian vessels and the brachial plexus) en route to the upper limb via the superior thoracic outlet. Nonspecific neurogenic TOS is not easy to diagnose because there is no investigational technique that has proven to be the diagnostic gold standard.

Design: In this study, our aim was to investigate the role of provocative F response in the diagnosis of nonspecific neurogenic TOS. F wave analysis of median and ulnar nerves in neutral and provocative maneuvers was carried out in 21 patients with a clinical diagnosis of nonspecific neurogenic TOS and in 15 healthy volunteers.

Results: All findings were within reference range in both groups, and no statistical difference was noted among subject groups, with or without provocative maneuvers.

Conclusions: We conclude that the nonspecific neurogenic TOS is a temporary compression process that does not result in a structural damage on the nerve; therefore, significant electrophysiologic changes are not elicited.

Key Words: Electrophysiology, F Wave, Provocative Maneuvers, Thoracic Outlet Syndrome

Background

Thoracic outlet syndrome (TOS), a compression syndrome of the neurovascular structures exiting the trunk and entering the upper extremity has been classified into 2 categories: True neurogenic-type TOS and Nonspecific (or disputed) neurogenic-type TOS. The primary differentiating clinical feature includes positive neurological signs in the former vs. no neurological findings in the later. The presence of vascular losses can be assessed using arteriography, MRI or Doppler ultrasonography, which reportedly makes the differential diagnosis process between neurogenic vs. vascular TOS fairly straight forward. Electrophysiologic studies include nerve conduction velocities, F wave, and somatosensory evoked potentials are used in the diagnostic process of the TOS patient but reportedly fail to support the diagnosis in some patients.

The purpose of this study was to investigate the clinical utility and sensitivity of provocative F waves in the differentiation between two types of TOS.

Methods

A sample of 29 patients were initially consider for this study meeting the screening criteria that included at least one clinical symptom (pain, numbness, tingling, swelling, discoloration, coldness, or weakness) and at least one positive clinical test finding (Adson's, modified Adson's, Roos, Halsted, upper limb tension, and hyperabduction tests). Control group subjects were matched for age, gender, weight and height. Exclusion criteria included those with polyneuropathy, entrapment neuropathy, or cervical radiculopathy, physical examination findings of sensory or motor loss, muscle atrophy or peripheral entrapment neuropathy, polyneuropathy (EMG/NCV supported), vascular abnormalities including A-V Doppler US abnormalities, and spinal cord compression on cervical MRI.

The subjects received electrophysiologic studies in two stages (provoked and standard/non-provoked) by the same technician. The EMG/NCV median and ulnar F waves were recorded in both the standard and provoked positions. The provoked positions included Adson's test, modified Adson's, Roos, Halstead tests, upper limb tension test, and hyperabduction test each held for one minute (or less if symptomatic). Statistical significance was set at $P < 0.05$.

Results

This study included 29 patients with symptom presentations consistent with TOS. Five patients were found to have vascular TOS, one had carpal tunnel syndrome and Martin-Gruber anastomosis, and two had ulnar neuropathy and were therefore excluded from the study leaving 21 patients (18 women, age 31.3 ± 7.8 years and 3 men aged 30.2 ± 7.7 yrs). The control group was made up of 12 women and 3 men and matched in age, gender, height, weight and body mass index ($P > 0.05$). Symptom duration as 2.6 ± 2.5 yrs (r. 1-10 yrs) with the right arm involved in 12 (54.5%) of the patients. Numbness and tingling was seen in all patients and coldness in the hand was the least common symptom (n=5 patients, 23.8%).

The upper limb tension test had positive results in all patients while the Halstead, modified Adson's, Roos, hyperabduction, and Adson's tests had positive results in 81%, 81%, 76%, 71%, and 57%, of the patients, respectively. Radiography findings in the patient group included a cervical rib in 33% (n=7), cervical lordosis planation and elongated transverse process of C7 were present in 62% (n=13) and 24% (n=5), respectively. The F wave latencies in the provoked positions were reportedly "small and insignificant," except the minimal F latency in the ulnar nerve which decreased with provocation. The minimal and mean F latencies were higher in all patients vs. controls with or without provocation. This difference was reportedly statistically significant for all measurements except for minimal F latency with provocation in the ulnar nerve with the difference tending to be more accentuated when provocative tests were used.

Conclusions

Even though there were no significant changes in the ulnar and median F responses between the neutral position and provocative position in patients with nonspecific neurogenic-type TOS, a significant difference in F wave latencies was found in patients with nonspecific neurogenic TOS compared with controls. This difference was even more significant during provocative testing in some of their measurements. The authors therefore concluded these findings supports inclusion of provocative maneuvers in the electrophysiologic assessment of suspected cases of neurogenic TOS.

Clinical Relevance

This study supports the use of provocative testing during electrodiagnostic studies in patients who are being assessed for TOS. Though the likelihood of successfully showing prolonged F wave latency favors those with neurogenic-type TOS, it may also help identify some cases of nonspecific neurogenic type TOS.

JACO Editorial Summary:

- This study evaluates the benefits for combining provocative orthopedic tests during the electrodiagnostic evaluation of a patient suspected of having TOS.
- The F-wave latency is particularly useful in brachial plexus lesions because it is an action potential evoked intermittently by a muscle when a supramaximal electrical stimulus is applied to the nerve resulting in an antidromic activation of motor neurons which then follow the alpha motor fibers back to the anterior horn cells. After synapsing at the cord, it then returns back to the muscle via the alpha motor neurons. Therefore, if there is a blockage proximal to the point of stimulation, the F-wave latency will be abnormal.
- TOS can be challenging to definitively diagnose due to in part to the symptom and clinical similarities of other conditions as was noted in the "Results" section describing the subjects that were excluded from this study because of other conditions (vascular TOS, CTS, ulnar neuropathy or others confounders such as polyneuropathy, peripheral neuropathy, radiculopathy, diabetic neuropathy, as well as others).

Summary

The findings and results of this study should motivate the health care practitioner to carefully assess the patient with peripheral upper limb symptoms and in the evaluation process strongly consider ordering electrodiagnostic studies, drawing special attention

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Abstracts & Literature Review

Thoracic Outlet Syndrome: A Controversial Clinical Condition Part 1 - Anatomy, and Clinical Examination/Diagnosis

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Author's Abstract:

Background

Thoracic outlet syndrome (TOS) is a frequently overlooked peripheral nerve entrapment that creates diagnostic and management difficulties for the clinician. Because the term TOS only outlines the location of the problem, investigators have categorized the condition as vascular versus neurogenic, where vascular TOS is further subcategorized as arterial or venous and neurogenic TOS is subcategorized as either true or disputed. Current estimates reveal 90% of all TOS cases are of neurogenic origin.

Methods

Literature Review (see References).

Results

Pathoanatomy: Anatomical structures that may result in neurovascular entrapment include: cervical supernumerary ribs; elongated C7 transverse process; exostosis, tumor, callus or fracture of the first rib; hypertrophy of the anterior scalene.

Epidemiology: Women are 3-4 times more likely to develop neurogenic TOS, while vascular TOS is more equal in non-athletic men and women.

History and clinical examination: Vascular TOS can develop secondary to repetitive upper limb activities that lead to claudication. Conversely, neurogenic TOS more commonly develops following macro-trauma to the neck or shoulder girdle area. Symptoms range from mild pain and sensory deficits to limb and/or life-threatening complications. Arterial TOS may present with pain, non-dermatomal numbness, coolness to touch and pale skin discoloration. Venous TOS on the other hand presents with excruciating deep chest and upper

limb pain, cyanotic discoloration and edema. Neurogenic TOS frequently presents with pain, paresthesia and/or weakness in a non-radicular or dermatomal pattern.

With the suspicion of TOS, examination of the cervical spine and shoulders is suggested to differentiate radicular versus non-radicular findings. Venous and Doppler ultrasound and angiography are typical for vascular TOS and NCV and EMG for neurogenic TOS. The clinician must also consider the possibility of a double-crush event when neural irritation or entrapment is present along the entire course of the nerve.

Clinical Relevance

With a patient presentation of neck, shoulder, arm, forearm and or hand complaints, TOS should be suspected as a differential diagnosis. A thorough history and appropriate examination can assist with a diagnosis of TOS resulting in appropriate management strategy.

JACO Clinical Summary

- The article was written by authors from the Center for Rehabilitation Research, School of Allied Health Sciences, Texas Tech University Health Science Center, Outpatient Physical Therapy Services, Northwest Texas Hospital System, Department of Rehabilitation Sciences, School of Allied Sciences, Texas Tech University Health Sciences Center, USA.
- Thoracic outlet syndrome is a frequently overlooked peripheral neurovascular compression.
- Suspicion of TOS should follow with non-dermatomal pattern presentations.
- When suspected, both vascular and neurogenic etiologies should be considered.
- The clinician is encouraged to consider double-crush event.
- Thorough history and clinical examination will assist in the proper diagnosis and management of these often overlooked conditions.

Summary

The explanations of the pathoanatomy and clinical examination/diagnosis of thoracic outlet syndrome presented in this literature review will provide a basis for more appropriate evaluation and management of patients presenting with upper extremity complaints.

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Abstracts & Literature Review

Thoracic Outlet Syndrome: A Controversial Clinical Condition Part 2 - Non-surgical and Surgical Management

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Author's Abstract:

Background

Thoracic outlet syndrome is a challenging and often misunderstood upper extremity disorder. Management of TOS requires an understanding of the various underlying etiologies of the neurovascular compression. Management may be conservative or surgical depending on the pathoanatomy and clinical presentation.

Methods

Literature review (see References).

Results

Although controversy exists regarding optimal treatment approaches for TOS, conservative measures should be attempted for patients with disputed neurogenic TOS before surgical consideration. Review of 13 studies published between 1983 and 2001 found that good or very good results were achieved in 76-100% of disputed neurogenic TOS patients at short term follow-up and 59-88% after at least one year. Poor outcome with conservative care was associated with obesity, workers comp and double-crush involving carpal or cubital tunnels.

The major focus of early management should be directed at pain/symptom reduction. These include NSAIDs, cervical mechanical traction and avoidance of arm abduction and overhead positions. Patient education is also a critical component. Once symptom reduction is accomplished, the clinician can begin to address the functional components of the disorder. These modalities may include encouraging diaphragmatic breathing, mobilization of the AC and SC joints and first rib. Pectoralis minor stretching, postural advice and taping may also prove helpful. Exercises should focus on endurance versus strength. Surgical management is typically indicated for vascular forms of TOS.

Conclusions

Conservative management is the preferred option for disputed neurogenic TOS. These management decisions should be directed initially at symptom reduction followed by correction of functional deficits.

Clinical Relevance

Based on this review it is clear that chiropractic physicians are in a position to provide treatment for patients with neurogenic TOS.

JACO Editorial Summary

- The article was written by authors from the Center for Rehabilitation Research, School of Allied Health Sciences, Texas Tech University Health Science Center, Outpatient Physical Therapy Services, Northwest Texas Hospital System, Department of Rehabilitation Sciences, School of Allied Sciences, Texas Tech University Health Sciences Center, USA.
- A thorough clinical history and examination is critical in the differential diagnosis of TOS.
- Conservative measures should be the first option with treatment of neurogenic TOS
- Initial management involves symptom reduction
- Once symptoms are controlled, care is directed at addressing various functional deficits.
- Generally, surgical management is reserved for vascular TOS

Summary

This review, along with Part 1, outlines the pathoanatomy, clinical evaluation and management options of the often overlooked thoracic outlet syndrome (TOS). Proper differential diagnosis and thoughtful conservative management of neurogenic TOS by the chiropractic physician will increase the probability of a positive treatment outcome for the patient.

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Announcements

Academy of Chiropractic Orthopedists - 2012 Diplomat Examination

This is to inform you and your departments of important changes in the Academy Diplomate examination process. These changes are the result of:

1) The Academy's compliance with NCCA Standards
2) Changing chiropractic orthopedic graduate demographics
3) Examination economic realities
4) Technological advancements in examination processes, and
5) Continuing development and refinement of the examination process.

The Academy of Chiropractic Orthopedists (ACO) has been part of the chiropractic orthopedic landscape since 1980 and has conferred Diplomate certification (DACO) since 2004. The Academy of Chiropractic Orthopedists is an active member of the Institute for Credentialing Excellence and complies with the NCCA "*Standards for the Accreditation of Certification Programs*." The Academy is currently a full voting member of the American Educational Research Association (AREA).

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Beginning in mid-2012, the Academy will begin offering an online Part I examination. Once the candidate passes Part I they are eligible to participate in the Part II, the OSCE (Objective Subjective Clinical Examination). With this format, the Academy is seeking to determine candidate knowledge-base and competency before allowing participation in the skills based clinical examination settings.

Part I examinations dates and sites will be announced in late spring 2012.

Part II OSCE examinations will be administered September 29, 2012, at NWHSU in Bloomington, MN.

If you have any questions, please do not hesitate to contact the Academy at 612-454-1472.

Sincerely,

Jerrold R. Wildenauer, DC, FACO
Executive Director - Academy of Chiropractic Orthopedists