Thoracic outlet syndrome: a controversial clinical condition. Part 2: non-surgical and surgical management

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Background: Proper management of thoracic outlet syndrome (TOS) requires an understanding of the underlying causes of the disorder. A comprehensive examination process, as described in Part 1 of this review, can reveal the bony and soft tissue abnormalities and mechanical dysfunctions contributing to an individual's TOS symptoms.
Objective: Part 2 of this review focuses on management of TOS.
Conclusion: The clinician uses clinical examination results to design a rehabilitation program that focuses on correcting specific problems that were previously identified. Disputed neurogenic TOS is best managed with a trial of conservative therapy before surgical treatment options are considered. Cases that are resistant to conservative treatment may require surgical intervention. True neurogenic TOS may require surgical intervention to relieve compression of the neural structures in the thoracic outlet. Surgical management is required for cases of vascular TOS because of the potentially serious complications that may arise from venous or arterial compromise. Post-operative rehabilitation is recommended after surgical decompression to address factors that could lead to a reoccurrence of the patient's symptoms.

Keywords: Conservative management, Review, Thoracic outlet syndrome, Surgical management

Thoracic outlet syndrome (TOS) remains a challenging and often misunderstood upper extremity disorder.[1,2] Optimum management of TOS requires an understanding of the underlying cause(s) of the neurovascular compression or tension. Although over 90% of all TOS cases are of neurogenic origin,[3] the clinician must remember to rule out potential vascular sources. Part 1 of this two-part series reviewed the pathoanatomy and examination of this disorder.[4] Part 2 describes conservative treatment measures for TOS based upon the clinician's examination findings. Finally, surgical management of neurogenic and vascular TOS and post-operative rehabilitation will be briefly reviewed.

Non-surgical Management

After a thorough clinical examination is performed and the underlying cause(s) of the patient's symptoms are identified, attention can be turned toward treatment of the patient's TOS. Although controversy exits regarding the optimal treatment approach for these patients, conservative measures should be attempted for patients with disputed neurogenic TOS before surgery is considered.[5–11] A majority of patients with neurogenic TOS can be expected to improve with proper conservative treatment. A recent review of 13 studies published between 1983 and 2001 found that good or very good results were achieved in 76 to 100% of disputed neurogenic TOS patients at short-term follow-up (within a month) and 59 to 88% after at least one year.[12] Novak *et al.*[13] found that poor outcome to conservative therapy was associated with obesity, worker's compensation, and double crush pathology involving the carpal or cubital tunnels.

Many sufferers of TOS have a long history of pain and disability. Management of these complex patients requires an individualized approach to the patient and his or her particular symptoms. The major focus of early treatment efforts should be symptom reduction.[14] Attempts to correct postural or biomechanical abnormalities prior to efforts at pain

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The patient is positioned in a chair, elbows flexed to 90° and placed on toweling at a height that sufficiently produces a passive shoulder girdle elevation. The patient sits upright with spine supported and the head in neutral. The forearms and wrists are positioned in neutral. The position is held until peripheral symptoms are produced. The patient is encouraged to allow symptoms to occur as long as can be tolerated up to 30 minutes, observing for a symptom decrescendo as time passes.

Figure 1 Cyriax release maneuver.

relief could result in an increase in symptoms and should therefore be approached cautiously in the initial treatment stages.

Nonsteroidal anti-inflammatory drugs may be prescribed to reduce pain and inflammation.[9] Injection of botulinum toxin into the anterior and middle scalenes for temporary relieve of pain and spasm resulting from neurovascular compression in the thoracic outlet has also been investigated.[15,16] Jordan *et al.*[15] found that 64% of subjects had a minimum of 50% decrease in pain, numbness, and fatigue for at least one month following injection. Others[17] have found that a three-week course of mechanical cervical traction along with a hot pack and exercise program reduced complaints of numbness significantly more than hot pack and exercise alone.

Disturbed sleep patterns are common in many people suffering from TOS, often as a result of either sleeping with the arms in an abducted, overhead position, or the consequence of the 'release phenomenon'.[18] If position dependent, then patients who cannot avoid the provocative position during the night may benefit from pinning the sleeve of the pajama arm to the pajama leg. The patient should sleep on the uninvolved side and avoid lying prone. Pillows may be placed under each arm when lying supine or between the body and the involved upper extremity when on lying on the side.[19]

The presence of a 'release phenomenon,' with paresthesias, numbness, or pain that wakes the patient during the night, may be confirmed with the Cyriax release test.[20] These patients are instructed in the Cyriax release technique (Fig. 1).[21] The goal of this technique is to fully unload the neurovascular structures in the thoracic outlet prior to going to sleep at night, which will allow the patient to sleep through the night without waking. Before going to bed, the patient sits in a chair with adequate arm rests to place the shoulder girdle in a passively elevated position. As the load on the brachial plexus is released, the person's symptoms begin and then gradually increase. Subsequently, as nerve function is normalized, the paresthesias begin to wane and eventually disappear. The clinician must explain to the patient that symptoms can rapidly decrease, but it may take up to two to three hours for the symptoms to sufficiently resolve. It must be emphasized that the patient should remain in this position as long as can be tolerated (preferably until the symptoms are appreciably resolved) in order to completely unload the thoracic outlet. Symptoms may not decrease during the first few sessions of this technique, but repeating the technique over time can cause the symptoms to gradually resolve more quickly. As a result of performing this technique, the patient will sleep longer into the night without waking. After one to two weeks of consistent use of this technique, it may be possible for the patient to sleep through the night without waking.

Patient education is an important component of any management strategy. Informing patients about the disease process and their potential prognosis can help lessen anxiety and encourage compliance with a home exercise program and recommendations for activity modifications. Compliance to an exercise program is an important factor in determining the outcome of conservative therapy.[22] Clinicians should question patients regarding postures or activities that increase their symptoms and suggest methods to modify them. Overhead activities can increase compression in the thoracic outlet and commonly result in increased symptoms. Workers whose occupation requires high levels of overhead use should modify their activities as necessary. Patients should avoid carrying heavy objects with the affected extremity, which would further decrease the size of thoracic outlet and increase the load on neurovascular structures in the thoracic outlet container.

Once the individual's symptoms are reduced, the clinician can begin to address the dysfunctions in the neural container that are responsible for causing the patient's symptoms. Management of container dysfunction is aimed at restoring the normal arthrokinematics of surrounding joints, correcting related muscle weaknesses and imbalances, and improving nerve mobility in order to decrease tension or compression of the brachial plexus in the thoracic outlet container. It is possible that failed attempts at conservative management are related to improper or

A B

(A) Costovertebral (CVJ) mobilization: the patient is positioned with the head mildly elevated and the cervical spine rotated away from the treatment side. The clinician uses the (R) radial hand at the second MCP to direct the mobilization force to the cranial surface of the first rib in a caudal and contralateral direction (towards the opposite hip). (B) Costotransversal (CTJ) mobilization: the patient is positioned with the head mildly elevated and the cervical spine rotated towards the treatment side. The clinician uses the (R) radial hand at the second MCP to direct the mobilization force in a ventral and ipsilateral direction. The (L) hand is used to stabilize the (R) shoulder. Oscillatory grade III and IV mobilizations or a grade V high velocity thrust maneuver can be performed in both cases.[24]

Figure 2 First rib mobilization.

incomplete diagnosis of the underlying dysfunctions causing the patient's symptoms. Thus, a thorough clinical examination allows the clinician to identify the specific dysfunctions within the thoracic outlet gates involved and direct treatment to the necessary structures.

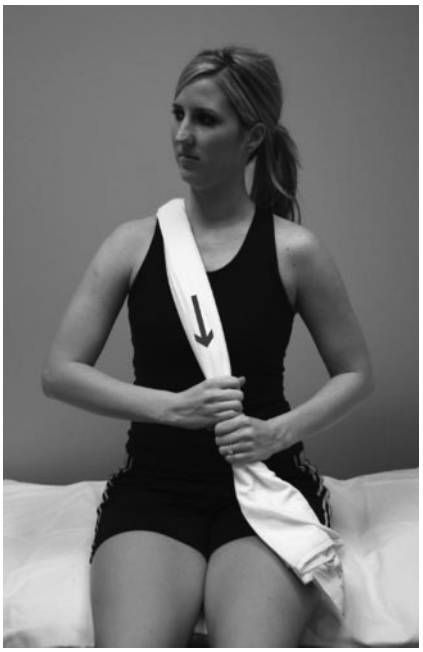
Costoclavicular space

Restoring mobility to the first rib can increase the costoclavicular space and reduce the imposed load on the neurovascular structures in the thoracic outlet container. Investigators have reported decreased TOS symptoms by restoring the mobility of the first rib through manual therapeutic procedures.[23] Other authors have recommended mobilizations or manipulative treatment to the first rib costotransverse and costovertebral joints in order to restore first rib mobility and open the costoclavicular gate (Fig. 2).[24–26] As a follow-up, patients may be taught a self-mobilization technique for the first rib to be performed as a home exercise program (Fig. 3).[24]

It is possible that these mobilization techniques may reproduce the patient's symptoms, particularly upper extremity paresthesias. Selected authors discourage the use of first rib mobilization in these patients for this reason.[11,23] However, if the symptoms are the result of a 'release phenomenon', patients should be encouraged to continue with the measures, as the symptoms may be occurring due to a normalization of nerve function associated with unloading of the brachial plexus.

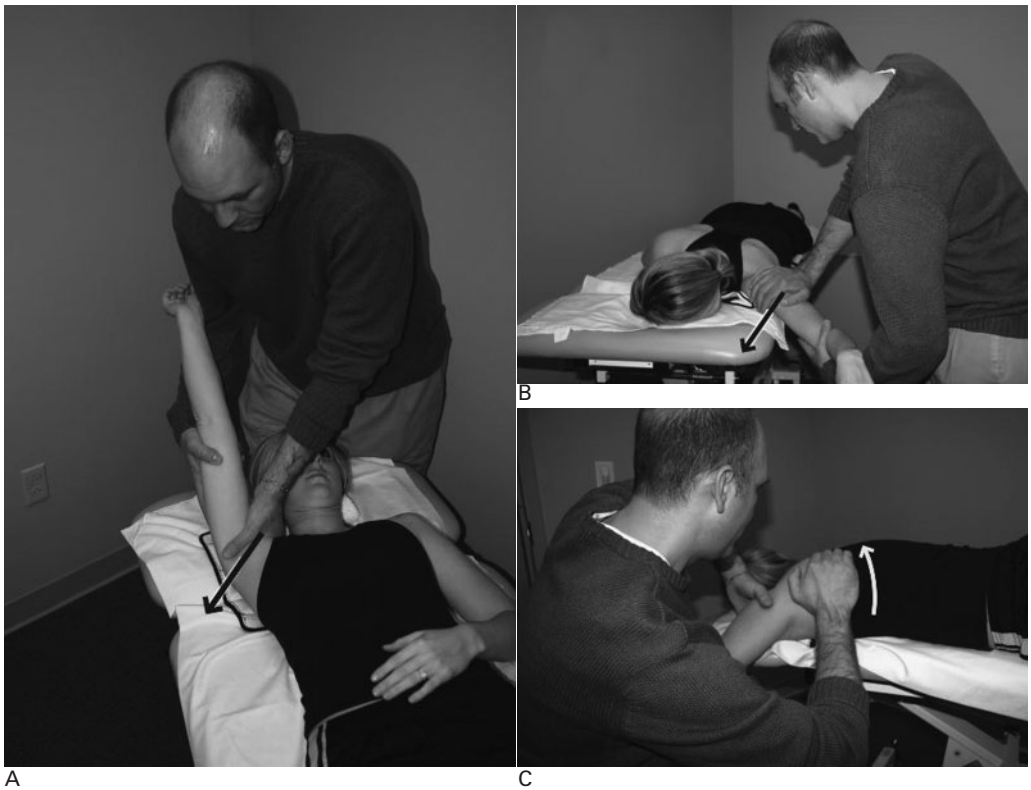
Overuse of the scalenes and other accessory respiratory muscles may result in an elevation of the first rib and rib cage, reducing the costoclavicular space. Encouraging diaphragmatic breathing helps reduce the activity of these muscles, increasing the costoclavicular space.[7] Vigorous aerobic activities may increase scalene activity and elevation of the first rib, so careful use of aerobic activities may help reduce symptoms, especially early in the rehabilitation process.[14,26]

Mobilization of the sternoclavicular and acromioclavicular joints is necessary to restore normal end-range arthrokinematics of the clavicle during elevation activities.[26] Additionally, end-range limitations of glenohumeral motion can lead to compromise of the costoclavicular space (Fig. 4).These limits can be addressed with mobilizations in the elevated arm position. The humerus can be glided in an



The patient is positioned in sitting with spine supported. The cervical spine is retracted, laterally flexed away and rotated towards the treatment side. A thin sheet strap is positioned to contact the first rib on the cranial surface 1 inch lateral to the transverse process of T1. The patient uses her own hands to pull on the sheet loop and produce a mobilization force directed caudal and contralateral (towards the opposite hip). The pictured head rotation emphasizes scalene stretch. Rotating head to opposite side emphasizes rib mobilization.

Figure 3 First rib self-mobilization.



(A) Posterior glide: the patient is supine with a sandbag posterior to the scapula on the treatment side. The mobilization hand contacts the proximal humerus while avoiding contact with the coracoid process. The force is directed along a helper's line connecting the anterior tip of the coracoid process and the posterior angle of the acromion. (B) Anterior glide: the patient is prone with a sandbag under the coracoid on the treatment side. The mobilization hand contacts the proximal humerus while avoiding contact with the acromion process. The force is directed along a helper's line connecting the posterior angle of the acromion with the anterior tip of the coracoid process. (C) Inferior glide: the patient is prone. The stabilization hand contacts the humerus distal to the lateral acromion process. The mobilization hand contacts the axillary border of the scapula and mobilizes the scapula around the rib cage in a cranial–medial direction.

Figure 4 Glenohumeral mobilizations in end-range elevation with the elbow supported in extension.

anterior, posterior and inferior direction, respecting the orientation of the glenoid.

Posterior scalene triangle

The posterior scalene triangle can be widened by (1) mobilizing the first rib in the direction of expiration and (2) stretching the scalene muscles. First rib mobilizations are performed as previously described. Once mobility of the first rib has been restored, increased activity of the scalene muscles may be addressed. Patients with hypertrophy or spasm of the scalenes will benefit from diaphragmatic breathing and a reduced reliance on the accessory respiratory muscles. These muscles can be stretched through a caudal mobilization of the first rib with the head rotated towards and laterally flexed away from the side being treated (Fig. 5).[24] The first rib self-mobilization technique may be modified to emphasize a stretch of the scalenes (Fig. 3).

Thoraco-coraco-pectoral space

Narrowing of the thoraco-coraco-pectoral space can result from shortening of the pectoralis minor or pectoralis major muscles. The pectoralis minor is stretched in a supine position with the patient's shoulder over the edge of the table. The patient is brought into 70 degrees of glenohumeral flexion with internal rotation and slight adduction. The clinician's contralateral hand is placed over the coracoid process, and the shoulder is stretched in a cranial and dorsal direction. The pectoralis major can be stretched with a corner stretch with the shoulders abducted 90 degrees and 125 degrees to stretch the

The patient is positioned supine with the chin retracted and the cervical spine laterally flexed away and rotated towards the treatment side. The clinician uses the (R) radial hand at the second MCP to direct the mobilization force in a caudal and contralateral direction (towards the opposite hip). The (L) hand is used to maintain a chin tuck.

Figure 5 Manual scalene stretch.

A B

The patient is positioned (A) with the head mildly elevated and the cervical spine laterally flexed towards the treatment side. The patient's arm is positioned at the side, shoulder girdle passively elevated and the elbow flexed with the forearm across the lap. The first rib is then manually depressed in a direction towards the opposite hip. (B) While maintaining the first rib depression, the cervical spine is gently and submaximally moved into lateral flexion away from the treatment side in a rhythmic fashion.

Figure 6 Proximal neural mobilization.

clavicular and sternal heads, respectively. Care must be taken that this position does not increase the patient's symptoms.[27]

Patients, with TOS resulting from narrowing of the thoraco-coraco-pectoral space, often present with a forward head posture and rounded, sagging shoulders. Proper posture should be emphasized throughout the treatment program with these patients. Encouraging a relative retraction of the shoulders increases the thoraco-coraco-pectoral space. A strip of hypoallergenic tape applied across the scapulae while in a comfortably retracted posture provides an effective tactile cue for the patient when the shoulders begin to fall into a protracted position.

Another factor that may cause a sagging shoulder posture is heavy breasts in females. Pressure on the neurovascular tissues may be decreased by wearing a good support bra with wide, crossed posterior straps. Reduction mammoplasty has been recommended for extreme cases.[27,28]

Compromised sensorimotor control of the posterior parascapular muscles, particularly the rhomboids, serratus anterior, and lower and middle trapezius, can alter the position of the scapula at rest and during arm elevation activities, ultimately leading to thoracic outlet narrowing. Sensorimotor control exercises for these muscles are begun once relative pain control has been achieved. Novak[7] recommends beginning these exercises in a gravity-assisted position in order to ensure proper recruitment of the lower scapular stabilizers and reduce the influence of the upper scapular elevators. All exercises should focus on muscular endurance rather than strength.[7] Because limited upward rotation, posterior tilting and retraction of the scapula during elevation activities may decrease the costoclavicular space, these exercises may be beneficial for patients with symptoms due to narrowing of that passage as well.

When a loss of neural mobility is present, neural mobilizations are incorporated in order to improve gliding of the neural tissue in relation to its surroundings and minimize tension and adhesion formation.[14,19,24,29,30] These techniques are especially important in cases of a double crush phenomenon.

Specific neural mobilization techniques can be modified to emphasize the brachial plexus proximally in the costoclavicular or thoraco-coraco-pectoral space while the clinician performs an inferior mobilization of the first rib (Fig. 6).[24] Additionally, the median and ulnar nerves more distally can be emphasized.[29,30] Neural mobilizations should be performed in a pain-free manner; therefore, any increases in symptoms with these exercises are best addressed by either decreasing the number of repetitions or altering the technique used. For a home mobilization program, patients are instructed to initially perform up to 20 repetitions and gradually increase up to 100 repetitions as tolerated. This program may be repeated one to two times daily.[29]

Surgical Management

Although a large percentage of TOS cases will improve with conservative management, a select group of these patients may require surgical intervention. Surgical management is especially indicated for the vascular forms of TOS because of the debilitating and potentially limb-threatening complications that can result from arterial or venous compromise.[5,31] Surgical management of neurogenic TOS is more controversial, and careful patient selection is required when considering surgical options.

Neurogenic TOS

Surgical decompression should be considered for those patients with true neurological symptoms, such as weakness, wasting of the hand intrinsic muscles,[27] or a nerve conduction velocity less than 60 m/second (normal 85 m/second).[5] Patients who fail a trial of conservative therapy and continue to experience significant pain that is limiting their ability to perform

activities of daily living or work tasks may additionally be candidates for surgical management.[32]

The goal of surgical management is to relieve the mechanical load on, and subsequent compromise to the neurovascular structures in the thoracic outlet. Because TOS may result from a variety of causes, the surgical techniques used should address the underlying pathology by reducing any of the bony or soft tissue structures contributing to the compression.

The first rib is a component of each gate of the thoracic outlet and appears to be a major compromising element in TOS,[33] but controversy exists regarding whether complete resection of the rib is necessary or even whether the rib needs to be resected at all. Incomplete resection may lead to a reoccurrence of symptoms due to inadequate decompression resulting from reattachment of the scalenes, scar tissue development, or bony regeneration off of the remaining tissue remnant.[5,34,35] Geven *et al.* reported that complete resection of the first rib resulted in superior outcomes versus partial removal for vascular TOS.[36] Other authors though, reported that incomplete removal of the first rib is not a major cause of TOS recurrence.[37–39]

In addition to possible resection of the first rib, partial or complete cervical ribs, when present, are removed. Anterior and middle scalenectomies can be performed to release spastic scalene muscles, which can directly compress the neurovascular structures and promote first rib elevation. Moreover, fibrous bands that may be compressing the nerves or vessels are excised. If callus formation from a previous clavicular fracture is decreasing the costoclavicular space, it can be removed as well.

Arterial TOS

The surgical management of arterial TOS depends on the type of arterial compression and whether arterial supply distal to the compression is reduced. Because of the potential for upper limb ischemia, early recognition and surgical intervention are essential. Treatment goals include decompression of the structures compressing the subclavian artery, repair of the subclavian artery and restoration of blood flow distally.[40] The artery must first be decompressed by removing any cervical ribs or fibrous bands, as well as possible first rib resection and scalene muscle revision.[31] After the decompression, the subclavian artery is inspected for arterial degeneration, dilatation, or aneurysm, which may require resection of the damaged artery and arterial reconstruction with a saphenous vein graft or synthetic prosthesis.[31] Distal thrombosis or embolization, if present, they can lead to ischemia of distal structures and must be surgically corrected. Surgical options include thrombolytic therapy or balloon thrombolectomy for acute ischemia.[27] Distal bypass grafting or formal arterial reconstruction is merited for chronic symptoms.[31]

Venous TOS

Similar to arterial TOS, venous TOS requires early recognition and treatment to avoid permanent complications. Thrombolytic therapy is the first line of treatment and is designed to dissolve an acute thrombosis.[41] It is most effective when given within one week of the onset of symptoms, but may be effective up to one month after symptoms develop.[42] In most cases, thrombolytic therapy is able to effectively dissolve the clot.[43–45]

Debate continues regarding the treatment options following thrombolytic therapy. If the vein continues to experience external compression, symptoms may reoccur.[41,43,46] A venogram is performed after thrombolysis to determine whether residual extrinsic compression occurs in either the anatomic position or during shoulder abduction.[41,43,45] Because of the risk of reoccurrence of thrombosis and symptoms, many authors recommend surgical decompression via removal of the first rib, even when the vein is completely opened with thrombolytic treatment.[41–43,47]

Once the extrinsic compression has been relieved, damaged veins may be repaired by either endovascular or open techniques. Angioplasty may be performed after the decompression to treat venous stenosis in patients with continued pain and disability.[41,48] If endovascular techniques are unsuccessful, vein patch angioplasty or venous bypass may be required to restore normal circulation.[41]

Postoperative physical therapy

Postoperative physical therapy is generally recommended after surgical decompression, but few published recommendations are available to guide this treatment.[49,50] Rehabilitation begins with shoulder and cervical range of motion exercises and gentle neural mobilization techniques. Overhead activities and lifting are avoided for 2–4 weeks.[51,52] If postural abnormalities or muscle imbalances are found, an exercise program that addresses these issues must be implemented to help prevent a reoccurrence of the patient's symptoms.[53]

Summary

The aim of this two-part series was to review the pathoanatomy relevant to TOS and to present an examination and treatment strategy focused on finding and correcting the underlying causes of a patient's signs and symptoms. Conservative therapy is the preferred option for disputed neurogenic TOS and satisfactory results can often be achieved when management is focused on correcting the dysfunctions found in the clinical examination. Treatment options should address symptom reduction, sleep adjustments, neural mobility and thoracic outlet container modification. Surgical intervention is reserved for true neurogenic TOS and disputed neurogenic TOS cases for which conservative measures have failed. The vascular forms of TOS require

surgery to relieve compression on vital vascular structures in the thoracic container. TOS is a condition surrounded by controversy and confusion; however, careful attention to the causes of TOS and appropriate management can help the clinician successfully treat this difficult population.

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