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Current Clinical Concepts: Rehabilitation of Thoracic Outlet Syndrome

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Thoracic outlet syndrome (TOS) involves inconsistent symptoms, presenting a challenge for medical providers to diagnose and treat. Thoracic outlet syndrome is defined as a compression injury to the brachial plexus, subclavian artery or vein, or axillary artery or vein occurring between the cervical spine and upper extremity. Three common subcategories are now used for clinical diagnosis: neurogenic, arterial, and venous. Postural position and repetitive motions such as throwing, weightlifting, and manual labor can lead to symptoms. Generally, TOS is considered a diagnosis of exclusion for athletes due to the poor accuracy of clinical testing, including sensitivity and specificity. Thus, determining a definitive diagnosis and reporting injury is difficult. Current literature suggests there is not a gold standard diagnostic test. Rehabilitation has been shown to be a vital component in the recovery process for neurogenic TOS and for arterial TOS and venous TOS in postoperative situations.

Key Words: TOS, neurogenic, arterial

Thoracic outlet syndrome (TOS) involves inconsistent symptoms, presenting a challenge for medical providers to diagnose and treat. It is defined as a compression injury to the brachial plexus, subclavian artery or vein, or axillary artery or vein occurring between the cervical spine and upper extremity.[1,2] The 3 common subcategories that are now used for clinical diagnosis include neurogenic, arterial, and venous TOS. Common areas of compression are the scalene triangle, clavicle, and first rib, which previously were used to assign a clinical diagnosis.[3] Bony and soft tissue abnormalities such as an accessory rib, congenital abnormality of the clavicle, and accessory scalene muscles, while rare, can lead to TOS symptoms.[4–6] The incidence of first rib abnormalities has been reported to be approximately 0.25%.[5] Postural position and repetitive motions of the upper extremity and neck, such as throwing, weightlifting, and manual labor, can lead to symptoms.

The prevalence of TOS is challenging to estimate because of the inconsistent presentation and lack of understanding of the diagnostic process.[1–3] Overhead athletes, such as pitchers, weightlifters, and swimmers, typically have a higher incidence of TOS because of the repetitive nature of their sports.[1,7] With repetitive strain, these individuals typically exhibit hypertrophic scalene and pectoralis minor muscles and a depressed shoulder complex that results in compression of and tension on the brachial plexus and associated vascular structures.

Generally, TOS is considered a diagnosis of exclusion for athletes because of the poor accuracy of clinical testing, including sensitivity and specificity. Thus, determining a definitive diagnosis and reporting injury is difficult.[5] Current literature suggests that no criterion standard diagnostic test exists. Alternatively, imaging, such as ultrasound, electromyography, magnetic resonance imaging, and computed tomography scans, and botulinum toxin injections (scalene and pectoralis minor muscles) can be used to identify the location of compression. Clinical tests have been shown to have poor diagnostic accuracy when used individually. Proposed best practice for clinical tests is to use a cluster of tests.[4] A combination of the Roos and Adson test yields 82% specificity. Thoracic outlet syndrome is widely underdiagnosed for the reasons stated previously.[8]

Treatment for TOS is ill defined. Typical treatment involves evaluation by a physician (sports medicine physician or team physician) who will refer athletes to specialists based on the type of TOS suspected and to rehabilitation. Rehabilitation has been shown to be a vital component in the recovery process for neurogenic TOS and for arterial and venous TOS after surgery.[5,8]

Athletes are likely to present to the athletic trainer with symptoms consistent with TOS. Identifying optimal treatment pathways is imperative for the best outcome. This clinical commentary covers the epidemiology, presentation, examination, and treatment for TOS. Where applicable, strength of recommendation (SOR) taxonomy is presented to assist in grading the evidence (A, B, or C; Figure 1).[9]

EPIDEMIOLOGY

The prevalence of TOS in the general population remains unclear. Among industry and service workers including hairdressers, assembly line workers, and cash

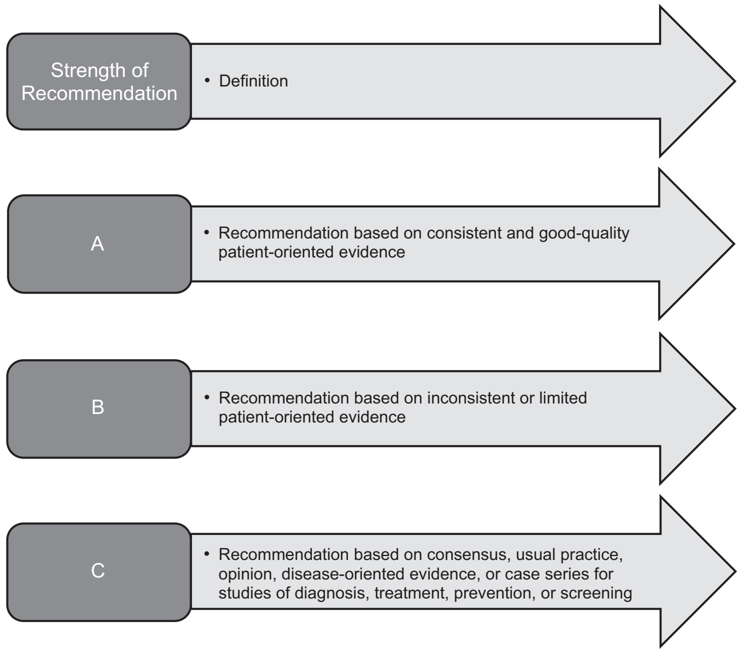

Figure 1. Strength of recommendation taxonomy.[9]

register operators, the prevalence has been reported to be 18%, with an even higher prevalence of 70% reported in computer users and musicians.[1] Secondary to challenges with diagnosing TOS, a wide range of incidence has been reported in the general population. The incidence has been reported as between 30 and 5000 per 1 million people and upward of 28 cases of neurogenic TOS and 8 cases of venous TOS per 1 million people per year.[2,10,11] Several case reports regarding all types of TOS in athletes have been published, but few researchers have studied the incidence and prevalence specifically in overhead athletes.[7,10,12–15] Otoshi et al found a prevalence of 32.8% in high school baseball players, whereas

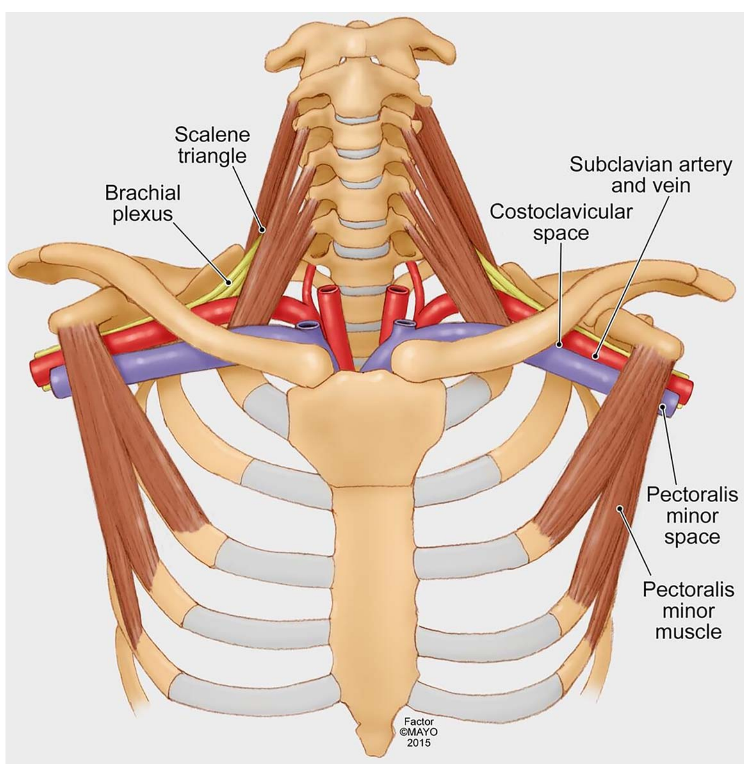

Figure 2. Anatomy of the thoracic outlet.[18,19] From Illig KA, Donahue D, Duncan A, et al. Reporting standards of the Society for Vascular Surgery for thoracic outlet syndrome. *J Vasc Surg.* 2016;64(3):e23–e35, used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.

Table 1. Risk Factors Summary

Congenital	Traumatic	Physical
• First rib anomalies • Cervical ribs • Cervical fibrocartilaginous bands • Muscle anomalies: abnormal origins or insertions, overlapping scalenes, fused muscles, asymmetries, hypertrophy • Hypercoagulability	• Hyperextension-hyperflexion injury to the neck: car accidents, whiplash injuries • First rib or clavicle bony fractures • Falls • History of trauma	• Repetitive neck or arm movements from work or sport • Hyperabduction of shoulder • Shoulder girdle instability • Adaptive muscle shortening • Alterations in joint biomechanics • Pregnancy • Obesity

van de Pol et al found a prevalence ranging from 11% to 27% in elite volleyball players.[12,13]

The prevalence of each subcategory has been better established: around 95% for neurogenic TOS, 3% for venous TOS, and 1% for arterial TOS.[16] Neurogenic and venous TOS are most common in athletes because the repetitive overhead stresses create mechanical compression due to hyperabduction and extension of the upper extremity.[7] Neurogenic TOS in the general population is common in 20- to 50-year-old women.[14] Although the reason is not completely understood, researchers have speculated that it is due to the higher incidence of cervical ribs in women.[17] Venous TOS in the general population is more common for those in their 20s to 30s and more typically affects men and the dominant arm. Arterial TOS is usually due to congenital or anatomic abnormalities, making it less common in the general and athletic populations. Arterial TOS, like neurogenic TOS, is more common in women because of the greater occurrence of cervical ribs.[11]

ANATOMY, PATHOPHYSIOLOGY, AND RISK FACTORS

The type of TOS refers to which structure in the thoracic outlet is affected: brachial plexus, subclavian artery, or subclavian vein. The 3 potential sites of compression include the interscalene triangle, costoclavicular space, and subcoracoid space (Figure 2).[18] These locations contain at least 2 neurovascular structures, so neurogenic, venous, or arterial compression can occur at these locations. The interscalene triangle comprises the anterior and middle scalene muscles and the first rib. Both the anterior and middle scalene muscles attach on the first rib, with the first rib forming the base of the interscalene triangle. The costoclavicular space comprises the subclavius muscle, the clavicle, and the first rib or anterior scalene muscle and is most easily visualized as the space between the first rib and the clavicle. This is where the subclavian vein is most vulnerable. Lastly, the brachial plexus and the axillary artery are most implicated in the subcoracoid or retropectoralis space. This space comprises the pectoralis minor muscle, ribs 2 to 4, and the coracoid process, with the pectoralis minor muscle and chest wall forming the anterior and posterior borders, respectively.[14,19,20]

Thoracic outlet syndrome involves compression and irritation of the brachial plexus as it runs through any of the aforementioned locations. Compression and irritation are usually due to repetitive injury that results in scarring and hypertrophy of the muscles surrounding the nerves, leading to scar tissue deposition on the nerves themselves. In the presence of preexisting anatomic variations, this nerve disruption can be exacerbated.[7] Presentation varies because of the potential sites of compression combined with the location of the brachial plexus that is involved (ie, root, trunk, division, cord, branch).

Venous TOS is a chronic disease process in which repetitive insult to the subclavian vein during upper extremity elevation causes scar tissue development. The term is often used interchangeably with Paget-Schroetter syndrome (PSS).[16] Although this process may be

Table 2. Differential Diagnosis of Thoracic Outlet Syndrome (TOS)[22]

Condition	Common Symptoms With TOS	Differing Symptoms From TOS
Carpal tunnel syndrome	Paresthesias in the hand; night pain; hand pain that increases with use	Loss of wrist extension
deQuervain tenosynovitis	Pain over the lateral wrist, anatomic snuffbox, and thumb	Local swelling; pain with resisted thumb extension; pain with Finkelstein test
Lateral and medial epicondylalgia	Pain in the medial and lateral forearm	Pain localized over the medial and lateral epicondyle; pain with resisted wrist extension and flexion
Complex regional pain syndrome	Burning upper extremity pain	Skin color change; temperature change over the skin
Cervical disc disease	Neck pain with arm pain; possible reports of paraesthesias affecting the arm	Loss of cervical ROM; decreased reflexes; myotomal weakness; symptoms worsened with neck movement
Paget-Schroetter syndrome	Pain and heaviness reported throughout the upper arm	Increased tissue temperature and swelling in the upper arm; painful and limited shoulder ROM
Rotator cuff pathology	Painful shoulder ROM	Positive rotator cuff stress testing
Glenohumeral instability	History of overuse involving the upper extremity	Joint dislocations; joint subluxations

Abbreviation: ROM, range of motion.

Table 3. Special Testing for Thoracic Outlet Syndrome[22,a]

Test	Description of Test	Positive Findings	Sensitivity	Specificity	Positive Likelihood Ratio	Negative Likelihood Ratio
Elevated arm stress test	While seated with arms at 90° of abduction, the patient fully externally rotates the arms. The clinician instructs the patient to open and close the hands (into a fist) for up to 3 min.	Pain; paresthesia; reduction of symptoms with lowering of the arms	0.52–0.84	0.30–1.00	1.2–5.4	0.4–0.53
Supraclavicular pressure	With the patient seated with the arms at rest by the sides, the clinician places his or her fingers on the upper trapezius and thumbs on the anterior scalene muscle near the first rib then squeezes his or her fingers together for 30 s.	Pain; paresthesia	NR	0.85–0.98	NR	NR
Costoclavicular maneuver	With the patient seated with the arms at the sides at rest, the clinician palpates the radial pulse. The clinician instructs the patient to retract and depress the shoulder (ie, military posture) and hold this position for 1 min.	Pain; paresthesia; decrease or change in radial pulse	NR	0.53–1.00	NR	NR
Adson test	With the patient positioned in sitting with the arms at the sides, the clinician palpates the radial pulse. The patient extends and rotates the neck toward the tested side.	Pain; paresthesia; decrease or change in radial pulse	0.79	0.74–1.00	3.29	0.28
Wright test	With patient positioned sitting with arms at the sides, the clinician palpates the radial pulse. The clinician passively moves the shoulder into abduction above the patient's head and holds this position for 1–2 min.	Pain; paresthesia; decrease or change in radial pulse	0.70–0.90	0.29–0.53	1.27–1.49	0.34–0.57
Cyriax release	The patient stands or sits. The clinician stands behind the patient, grasps under the forearms, and flexes the elbows to 80°. The clinician leans the patient posteriorly and elevates the shoulder girdle. The clinician holds this position for up to 3 min.	Pain; symptom reproduction; increased numbness (described as "release")	NR	0.77–0.97	NR	NR
Upper limb tension test	With the patient supine, the clinician depresses the shoulder girdle on the tested side, abducts the arm to 110° in neutral extension while flexing the elbow to 90°, and externally rotates the shoulder. The clinician then systematically supinates the wrist, extends the fingers, and extends the elbow. The neck can be side-bent to the contralateral side to test for neural sensitivity.	Symptoms with distal segment movement or neck movement	0.90	0.38	1.5	0.3
Cervical rotation lateral flexion	With the patient sitting with arms at the side, the clinician passively rotates the head away from the tested side and side bends the patient's ear toward the sternum.	Decreased forward flexion with hard end feel	1.00	NR	NR	NR

Abbreviation: NR, not reported.

[a] Diagnostic criteria developed by Balderman et al recommended the use of supraclavicular pressure, upper limb tension test, and elevated arm stress test along with other subjective patient concerns.[25]

Table 4. **Strength of Recommendations (SORs) for Examination and Rehabilitation**[9]

Rehabilitation Considerations	SOR	Summary of Recommendation
Examination technique		
Use of outcome measures	B	To measure patient outcomes, the Cervical-Brachial Symptom Questionnaire demonstrates high reliability and consistency; the Disabilities of the Arm, Shoulder and Hand may also be used to better assess improvements in arm function.
Palpation and observation	C	Postural evaluation should include assessment of head, scapulae, and shoulder position; observation should also detect any upper limb edema or muscle atrophy.
Range-of-motion assessment	B	Range-of-motion assessment should include measures of the cervical spine, shoulder, elbow, and wrist and hand; care should be taken to also observe for the presence of scapular dyskinesia during shoulder range of motion.
Muscular assessment	C	Pectoralis major and minor, scalene, and upper trapezius muscles should be assessed for flexibility as they are often shortened; muscle force production should assess the rotator cuff, pectoralis, and accessory scapular muscles.
Neurologic assessment	C	Neurologic examination should include assessment of reflexes, sensation, and vibration sense.
Orthopaedic special testing	B	Special tests should be clustered to aid in differential diagnosis; inclusion of the elevated arm stress test, upper limb tension test, and Adson tests can help provide insight into presence of TOS.
Diagnostic imaging	B	Plain film radiographs may be used to assess for fracture and identification of bony abnormalities (ie, cervical rib); magnetic resonance imaging may be used to identify soft tissue structures contributing to compression; duplex ultrasound may be used for detection of venous and arterial TOS.
Treatment technique		
Soft tissue mobilization or manual therapy	C	In the case of postural abnormalities associated with soft tissue contracture, soft tissue mobilization can be an effective tool to improve static posture.
Joint mobilizations	C	First rib mobilization, thoracic spine mobilization, cervicothoracic junction mobilization and lateral cervical glides can be effective techniques to improve joint mobility.
Stretching	C	Caution with stretching as TOS is often associated with a traction-based injury and depending on stretching position could irritate symptoms.
Therapeutic exercises	A	Emphasis on middle and lower trapezius combined with serratus anterior to improve scapular control. Starting with short lever arm and a set/repetition scheme, athletes can complete without compensatory patterns.
Blood-flow restriction	C	Blood-flow restriction is not recommended secondary to arterial and venous TOS and potential to progress to upper extremity deep venous thrombosis.
Neural mobilizations	C	Primarily targeting median and ulnar nerves; completed in pain-free manner and when no tensile sensitivity is identified.
Breathing and core stability	C	Intervention to improve diaphragmatic breathing can help decrease hypertrophy of accessory breathing muscles. Progressing strategies to sport-specific activities.
Taping and external support	C	This support can provide short-term management for acute and highly irritable TOS. Helps to facilitate scapular elevation and upward rotation.
Anesthetic injections	B	These injections are used as a short-term symptom relief; often a predictor of who will be successful with a decompression surgery versus as an adjunct to rehabilitation approaches.
Botulinum toxin injections	B	These injections can serve as an adjunct to rehabilitation approaches with short-term results in symptom relief lasting up to 3 months.

Abbreviation: TOS, thoracic outlet syndrome.

asymptomatic for years because of compensatory blood routes, it can eventually create an acute thrombus in the subclavian vein that can impede circulation. Sudden swelling, cyanosis, heaviness, pain, and early fatigue in the upper extremity often result. With PSS, the subclavian vein is compressed between the clavicle and the first rib; whereas PSS is far less common than neurogenic TOS, the incidence is higher in younger competitive athletes. Chronic overuse of the upper extremity can cause an initial injury to the subclavian vein, which initiates a cascade of fibrosis within and around the wall of the subclavian vein. This repetitive cycle leads to the fibrosis and narrowing of the subclavian vein. Like venous TOS, it is usually asymptomatic until a clot forms.[15]

Arterial TOS results from prolonged and sustained compression of the subclavian artery, commonly seen in the presence of a bony abnormality such as cervical rib or hypoplastic first rib. This compression leads to degeneration in the arterial wall and subsequent thrombus formation.

Table 5. **Rehabilitation Therapeutic Exercise Considerations for Phase 1: Scapular Position at Rest**

Goal	Exercises	Criteria to Progress
Develop scapular control with depression and retraction in neutral positioning	Scapula setting in standing or sitting (Figure 3A) Scapula setting in prone (Figure 3B) Scapula retraction with external rotation (Figure 3C)	3 sets of 20 repetitions completed without compensatory patterns noted

Table 6. Rehabilitation Therapeutic Exercise Considerations for Phase 2: Scapular Control in <30° of Abduction

Goal	Exercises	Criteria to Progress
Develop scapular control in 0°–30° of abduction	Bilateral prone extension (Figure 4A) Unilateral prone or bent-over extension (Figure 4B) Unilateral prone or bent-over row (Figure 4C) Elastic-band W or robbers (Figure 4D)	3 sets of 20 repetitions completed with 1–3 lb (0.45–1.35 kg) and without compensatory patterns noted

Congenital, traumatic, and a variety of functional factors such as postural positioning and repetitive stresses can contribute to the onset of TOS. Congenital causes include cervical ribs and fibrous bands, but first rib anomalies and cervical muscle variations have also been reported.[1,12,20] Cervical ribs are more commonly seen in individuals with symptomatic TOS than in asymptomatic individuals.[21] Muscle anomalies including variable insertion sites and overlapping, extra, fused, or hypertrophied muscles are risk factors for compressing neurovascular contents.[20] Traumatic causes include whiplash injuries in which a rapid hyperextension-flexion moment to the neck, causing a chronic inflammatory response and compressing neurovascular structures, occurs. Other traumatic causes include first rib or clavicular fractures, falls, repetitive neck movements, and repetitive arm movements with work or sports (ie, repetitive overhead motion).[19] These repetitive injury mechanisms can lead to any of the 3 types of TOS.

Specific to overhead athletes, the inherent high-velocity, repetitive overhead movements performed for their respective sports put them at risk for developing TOS. The hyperabduction and overhead positions in sports such as baseball, softball, swimming, and volleyball can disrupt the neurovascular structures. These motions can cause microtrauma in the muscle fibers, leading to hemorrhage and microscopic scar tissue within the scalene muscles and ultimately leading to muscle fibrosis. Muscle hypertrophy, which may result from or cause shoulder girdle instability, muscle imbalances, adaptive muscle shortening, or alterations in joint biomechanics, can lead to the onset of TOS. Over time, this neurovascular stress can impair blood flow, causing inflammation and fibrotic changes that can further decrease nerve compliance in an already narrowed thoracic outlet.[14,15] If overhead athletes have any control, strength, or joint restriction as the force is transferred from the lower body up the kinetic chain, the energy could dissipate, and the thorax, shoulder, and arm must make up for the lost energy.[10] This can lead to instability in the cervicothoracic and scapulothoracic regions, stiffness in the scalene muscles, and other positional changes of the scapula on the thorax. Without adequate serratus anterior muscle counterforces, maladaptive first rib elevation and a depressed and downwardly rotated scapula can compress the neurovascular structures running through the thoracic outlet.[10] A summary of risk factors is provided in Table 1.

EXAMINATION

Clinical Presentation

Symptoms of neurogenic TOS include neck, shoulder, and arm pain at rest; paresthesias; night pain; weakness; and occipital headaches.[1,3,7,22] Pain and paresthesias are worsened with overhead movements.[1,3] Paresthesias often affect the arm and hand and do not follow peripheral or nerve root distributions, depending on the location of compression. Individuals may report a loss of grip strength or finger dexterity.[3,22] In overhead athletes, clinicians should suspect neurogenic TOS when athletes report loss of velocity and accuracy and heaviness in the arm after throwing.[7] *SOR: A*

Symptoms of venous TOS include diffuse shoulder, neck, and arm pain accompanied by edema throughout the arm, cyanosis, and a subjective report of a feeling of heaviness.[3,22,23] On visual examination, the patient may have dilated collateral veins of the shoulder, chest wall, and arm.[23] Reports of nondermatomal paresthesias throughout the fingers and hand are common.[22] *SOR: A*

Symptoms of arterial TOS include paresthesias in the fingers and hands; however, the most common concerns are coldness and cold intolerance.[3,22] Overhead movements typically worsen symptoms and can lead to pallor, pulselessness, or both in the hand.[3,22] If clinicians suspect arterial TOS, they should refer patients to a vascular surgeon for optimal management.[3,23] *SOR: A*

Various conditions present with symptoms like those of the TOS types referenced above; however, a hallmark of TOS is the presence of unilateral, upper extremity symptoms. A list of common differential diagnoses and their associated symptoms is provided in Table 2.

Outcome Measures

Given the complexity and systems affected by TOS, clinicians may include the Short-Form McGill Pain Questionnaire instead of a visual analog scale to assess pain.[14] This questionnaire has patients localize their pain, describe their pain and its intensity, and identify the pattern of pain. Strand et al reported that the minimal clinically important difference (MCID) was a change greater than 5 points.[24] *SOR: B*

The Cervical Brachial Symptom Questionnaire is used to assess outcomes in the nonoperative and operative

Table 7. Rehabilitation Therapeutic Exercise Considerations for Phase 3: Scapular Control in 45°–90° of Abduction

Goal	Exercises	Criteria to Progress
Develop scapular control in 45°–90° of abduction	Bilateral prone W (Figure 5A) Bilateral prone T: horizontal abduction (Figure 5B) Unilateral prone T: horizontal abduction (Figure 5C) Unilateral prone row with external rotation (Figure 5D)	3 sets of 20 repetitions completed with 1–3 lb (0.45–1.35 kg) and without compensatory patterns noted

Table 8. Rehabilitation Therapeutic Exercise Considerations for Phase 4: Scapular Control in Flexion

Goal	Exercises	Criteria to Progress
Develop scapular control in the sagittal plane	High to low row (Figure 6A) Elastic-band extension (Figure 6B) Scaption lift to 90° (Figure 6C) Quadruped serratus protraction (Figure 6D) Serratus press in supine or standing (Figure 6E)	3 sets of 20 repetitions completed with 3–30 lb (1.35–13.5 kg) and without compensatory patterns noted

management of patients diagnosed with TOS.[25] The instrument has demonstrated high reliability ($r = 0.87$) and an internal consistency of 0.93 (Cronbach α); however, the MCID is unknown.[26] *SOR: B*

The Disabilities of the Arm, Shoulder and Hand has excellent test-retest reliability ($r = 0.81–0.91$) for patients with musculoskeletal upper extremity problems.[27] This instrument has been found to be responsive and is frequently used to track patient outcomes, with an MCID of 12.6 points.[27] *SOR: B*

Physical Examination

Palpation and Observation. Postural observation may reveal rounded shoulders and scapulae that are downwardly rotated, depressed, or both.[22] In advanced cases of TOS, atrophy may be detectable in the thenar and hypothenar compartments.[3] Clinicians should observe the upper limb for edema, cyanosis, or pallor, which may indicate vascular TOS.[22] Palpation of the scalene triangle, subcoracoid space, or both may result in pain or reproduction of paresthesias.[3,7] *SOR: C*

Range of Motion Assessment. Range of motion (ROM) of the neck and upper quarter should be assessed.[7,22] Scapular dyskinesis is commonly seen in TOS and should be evaluated. Baseball players with neurogenic TOS have been reported to have reduced external rotation and total ROM in the throwing arm compared with healthy controls.[28] *SOR: B*

Muscular Assessment. Pectoralis major and minor, scalene, and upper trapezius muscles are commonly shortened and require evaluation.[14] Force-production assessment should include examination of the scalene, pectoralis major and minor, rotator cuff, and accessory scapular muscles.[3] We recommend measuring strength via a handgrip dynamometer or manual muscle test. Clinicians should consider using a handgrip dynamometer to assess for deficits in grip strength, as hand gripping can result in pain or parasthesias.[3,7] *SOR: C*

Neurologic Assessment. A neurologic examination should include reflex, sensation, and vibratory testing, which may be diminished.[3,7] *SOR: C*

Orthopaedic Special Testing. Provocative testing can aid in the diagnosis of TOS. A full list of tests and their psychometric properties is provided in Table 3.[22] Balderman et al recently designed diagnostic criteria to assist in the diagnosis of neurogenic TOS.[29] They advocated for using the upper limb tension test (ULTT); 3-minute elevated arm stress test (EAST); and assessment involving principal symptoms, symptom characteristics, clinical history, and physical examination.[7,29] The detection of upper limb swelling and cyanosis along with positive EAST, ULTT, and Adson tests increases the likelihood of venous TOS.[22,30] The detection of upper limb ischemia, bruits, and blood pressure differential greater than 20 mm Hg along with positive EAST, ULTT, and Adson tests increases the likelihood of arterial TOS.[22,30] *SOR: B*

Diagnostic Imaging. Thoracic outlet syndrome is a diagnosis of exclusion and determined with a thorough clinical examination; however, imaging can play an important role in characterizing the extent of possible compression.[30,31] Radiographs of the chest and cervical spine are used to detect bony abnormalities including cervical ribs.[30–33] Magnetic resonance imaging is used to characterize soft tissue structures contributing to compression. Results should be interpreted with caution, as the rate of venous compression is high in patients without symptoms of TOS.[30,34] Duplex ultrasound, computed tomography, or magnetic resonance imaging venography may be ordered to rule in venous TOS.[30,34] Duplex ultrasound, contrast arteriography, or finger plethysmography may be ordered to rule in arterial TOS.[30] The clinician may also order nerve conduction studies or needle electromyography to assist with differential diagnosis.[30,31] *SOR: B*

REHABILITATION CONSIDERATIONS

The primary rehabilitation goal is to limit tensile loads, compressive loads, or both across the thoracic outlet region when athletes return to sport (RTS) activities.[35] A multimodal treatment program is recommended for athletes with TOS. Summarized recommendations are provided in Table 4.

Table 9. Rehabilitation Therapeutic Exercise Considerations for Phase 5: Scapular Control in >90° of Elevation

Goal	Exercises	Criteria to Progress
Develop scapular control in overhead strengthening progressions	Unilateral prone Y-scapular–plane elevation (Figure 7A) Elastic-band external rotation at 90° (Figure 7B) Serratus wall slide (Figure 7C) Landmine press (Figure 7D)	3 sets of 20 repetitions completed without compensatory patterns; loading varies from 2–55 lb (0.9–24.75 kg) depending on exercise

Table 10. Rehabilitation Therapeutic Exercise Considerations for Phase 6: Scapular Control in Sport-Specific Movements

Goal	Exercises	Criteria to Progress
Returning to sport maintaining scapular control	Dependent on the functional demands of the athlete—individualize program	Completion of return-to-sport testing

Soft Tissue Mobilization or Manual Therapy

Postural abnormalities associated with TOS are due to strength and recruitment difficulties or soft tissue contracture. With soft tissue contracture, soft tissue mobilization can be used to improve static posture.[36] Techniques used include direct pressure, parallel deformation, or perpendicular strumming to the muscle or muscle-tendon unit.[36] Instrument-assisted soft tissue mobilization can be used as an adjunct to manual therapy to reduce tissue viscosity, provide myofascial release, decrease pain, and improve flexibility.[37] Dry needling is a form of trigger-point release used to help decrease muscle hypertonicity. The medical provider must be competent with dry-needling technique for the scalene muscles to avoid injuring nearby neurovascular structures. Dry needling as an adjunct treatment for targeting postural mobility of the latissimus dorsi and pectoralis muscles is a consideration for athletes who are not making adequate progress with standard-of-care techniques. *SOR: C*

Joint Mobilizations

Joint mobilizations can be used depending on examination findings. Common mobilization techniques include the first rib, thoracic spine, cervicothoracic junction, and lateral cervical glides.[35,38] Inferior first rib mobilizations can be used to increase the costoclavicular space and decrease compression on the neurovascular structures.[36] Glenohumeral joint mobilizations can be used if ROM deficits exist, as mobilizations can compress the costoclavicular space.[39] Posterior glenohumeral joint mobilizations are used to improve posterior capsule mobility and improve function in overhead athletes.[35] Thoracic spine mobilizations including posterior-anterior mobilizations can be used to decrease thoracic kyphosis and improve static posture. Secondary to cervical musculature hypertonicity associated with TOS, decreased cervical and thoracic facet-joint mobility is a common examination finding. Cervical mobilizations include lateral glides to improve facet-joint mobility. *SOR: C*

Stretching

Pectoralis major and minor, scalene, upper trapezius, sternocleidomastoid, and latissimus dorsi muscles are all commonly shortened with TOS.[14] A thorough examination is important to identify any tensile stress associated with symptoms. If tensile stress is present, lengthening shortened tissues can recreate symptoms.[35] An example would be pectoralis stretching completed in a door frame or over a foam roller recreating the EAST position and bringing the clavicle into the first rib, recreating symptoms.[35] A supine position with the arm supported is recommended to help mitigate symptoms.[35,36] Scalene muscles attach on the first rib and contribute to first rib elevation, which can compress the thoracic outlet. These muscles can be stretched using an inferior first rib mobilization with cervical spine ipsilateral rotation and contralateral side bend.[39] Other cervical musculature that can be stretched includes suboccipital, upper trapezius, and levator scapulae muscles secondary to their effects on posture. Other techniques such as contract-relax or other proprioceptive neuromuscular facilitation techniques can be used to help improve soft tissue mobility.[36] Clinical decision making based on the objective examination findings and monitoring patient response are important considerations for dosing stretching interventions. *SOR: C*

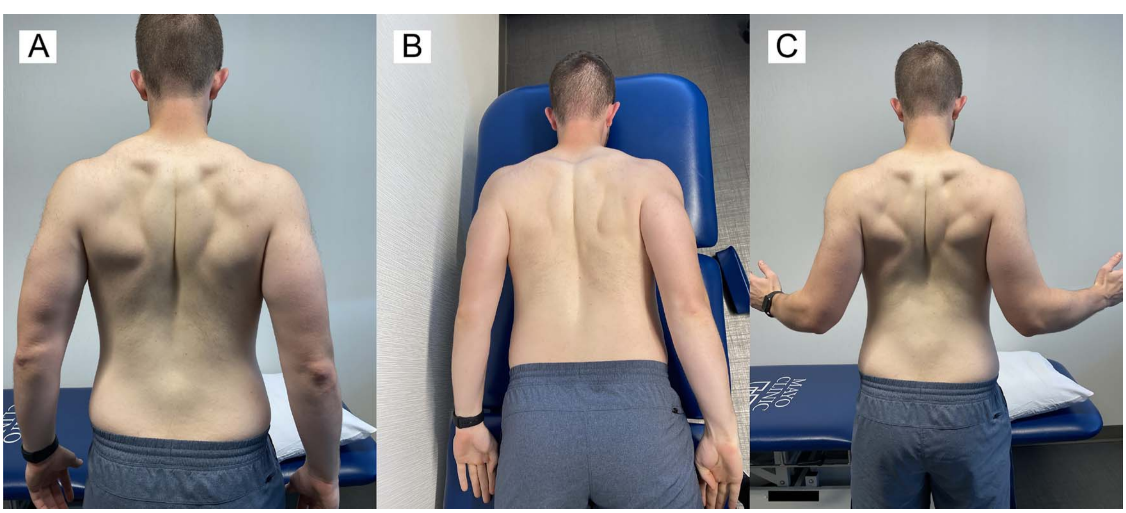

Figure 3. Exercises for rehabilitation phase 1: scapular position at rest. A, Scapula setting in standing or sitting. B, Scapula setting in prone. Scapula retraction with external rotation.

Figure 4. Exercises for rehabilitation phase 2: scapula control <30° of abduction. A, Bilateral prone extension. B, Unilateral prone or bent over extension. C, Unilateral prone or bent over row. D, Elastic-band W or robbers.

Therapeutic Exercises

A cornerstone of TOS rehabilitation consists of normalizing scapular muscle recruitment. Muscles primarily requiring facilitation to improve scapular control include the middle and lower trapezius and the serratus anterior.[40] The emphasis with strengthening progressions should be placed on neutral positioning of the scapula and maintaining control against the thorax with sufficient posterior tilt of the scapula to keep the medial border stabilized.[40] Individuals with TOS typically develop fatigue earlier than healthier individuals; thus, endurance and isometric strength should be prioritized early in the rehabilitation process.[41]

A gradual progression of forces when working on scapular control is a critical rehabilitation consideration. Starting with a set/repetition scheme to challenge the athletes without use of compensatory patterns is important. A combination of isometric or light-weight, high-set/repetition schemes is recommended, gradually progressing to an isotonic and hypertrophy-oriented set/repetition scheme as scapular control progresses. Humeral-abduction angles increase the lever arm and can further challenge activation and recruitment. In previously published outlines, authors described a rehabilitation progression focusing on scapular control in ranges of abduction.[40,42] Phase 1 starts with shoulder movements below 30° of abduction, and each subsequent phase progresses shoulder movements to 90° of abduction and overhead activities.[40,42] An example of scapula-neutral strengthening progressions is outlined in Tables 5 through 10, with exercises illustrated in Figures 3 through 7. In late-stage rehabilitation, working through a sport-specific progression with scapular control to minimize symptoms is an important consideration before returning to sports. *SOR: A*

Blood-Flow Restriction

Blood-flow–restriction (BFR) training is an intervention used in rehabilitation for a wide variety of injuries. However, secondary to arterial TOS and venous TOS, BFR is not a recommended treatment for TOS.[43] From a mechanistic standpoint, BFR creates an ischemic and hypoxic muscle environment, causing high levels of metabolic stress in combination with exercise.[43] In a case study, Noto et al described a patient with TOS who progressed to PSS secondary to KAATSU training, a form of BFR.[44] No other literature exploring BFR as a treatment consideration for TOS exists. *SOR: C*

Neural Mobilization

Neural glides in athletes with neurogenic TOS can be incorporated when neural mobility is decreased on clinical assessment. Neural mobilizations with TOS primarily target

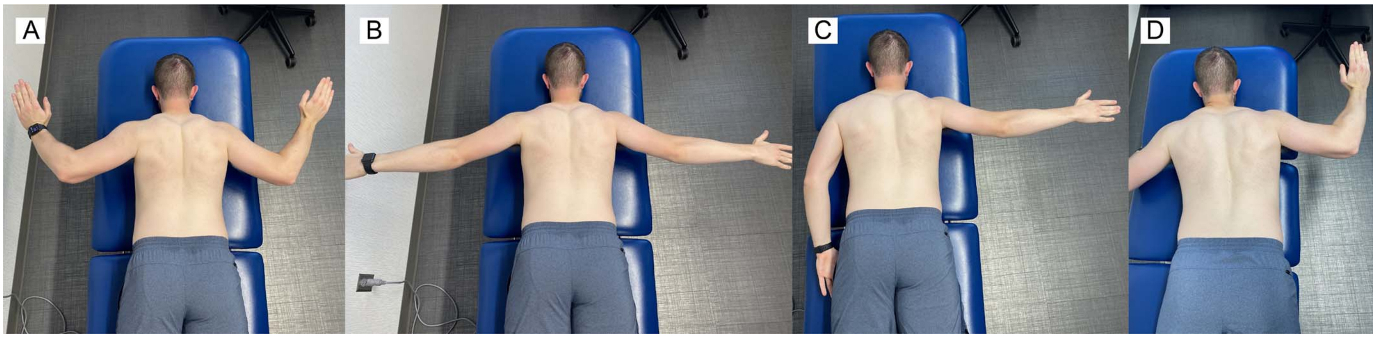

Figure 5. Exercises for rehabilitation phase 3: scapula control 45°–90° of abduction. A, Bilateral prone W. B, Bilateral prone T: horizontal abduction. C, Unilateral prone T: horizontal abduction. D, Unilateral prone row with external rotation.

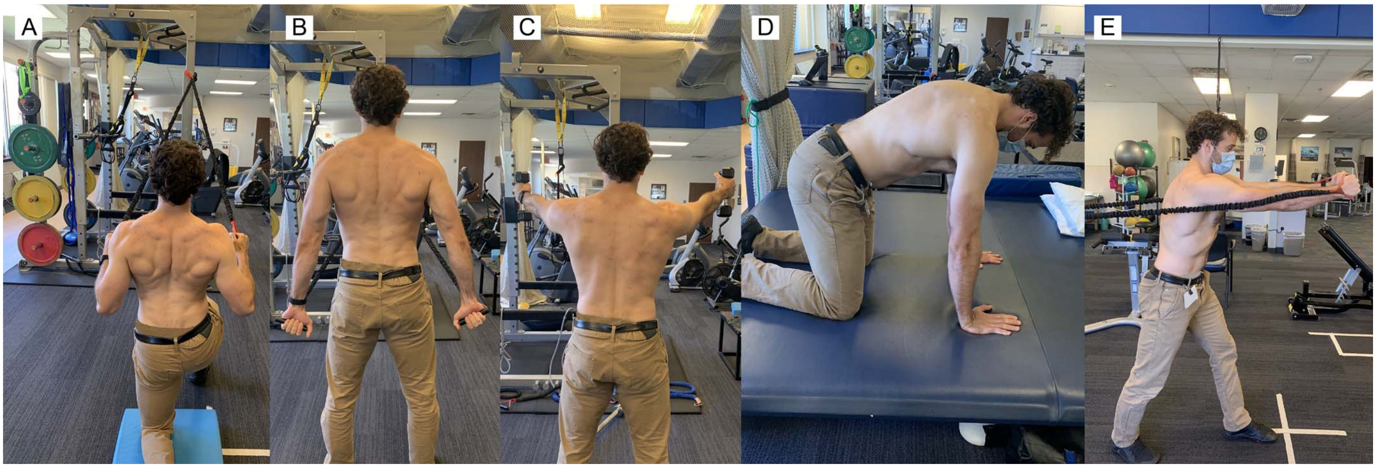

Figure 6. Exercises for rehabilitation phase 4: scapula control in flexion. A, High to low row. B, Elastic-band extension. C, Scaption lift to 90°. D, Quadruped serratus protraction. E, Serratus press in supine or standing.

the median and ulnar nerves.[39] Caution should be exercised when using neural mobilizations to ensure they are completed in a pain-free manner and only when no tensile sensitivity is identified. *SOR: C*

Breathing and Core Stability

Breathing is an important consideration in decreasing thoracic outlet compression. Accessory breathing strategies can contribute to hypertrophy of scalene, sternocleidomastoid, and trapezius muscles.[45] Athletes with neurogenic TOS often demonstrate accessory breathing strategies versus diaphragmatic breathing, potentially contributing to their symptoms.[45] Using interventions to improve diaphragmatic breathing can help to reduce thoracic outlet compression. Core stability training combined with diaphragmatic breathing can help improve carry-over of breathing techniques to sport-specific activities. *SOR: C*

Taping and External Support

Short-term management of acute and highly irritable TOS can include external support with bracing or taping. Taping can provide additional support for the shoulder girdle and is recommended for athletes who have improved symptoms with scapula elevation.[40] Watson et al described an axillary-sling technique to help create scapular elevation and upward rotation (Figure 8).[40] Other external support or bracing includes using a pillow or towel roll under the arm to assist with lifting the shoulder girdle.[35] *SOR: C*

Injections

Anesthetics. Local anesthetic blocks of the anterior scalene muscles provide both analgesic and muscle relaxation for symptom relief.[46] Anesthetics are used as a predictor of which athletes would benefit from decompression surgery.[46] Given the short-term relief provided by local anesthetics, they are more useful as a predictor of success with decompression than as an adjunct to rehabilitation approaches.[47] *SOR: B*

Botulinum Toxins. Botulinum toxins are neurotoxins injected to help treat focal muscle hyperactivity.[47] Researchers have hypothesized that they can help patients with neurogenic TOS secondary to their analgesic and muscle-relaxant properties.[47] Literature support has been inconsistent regarding its efficacy in patients

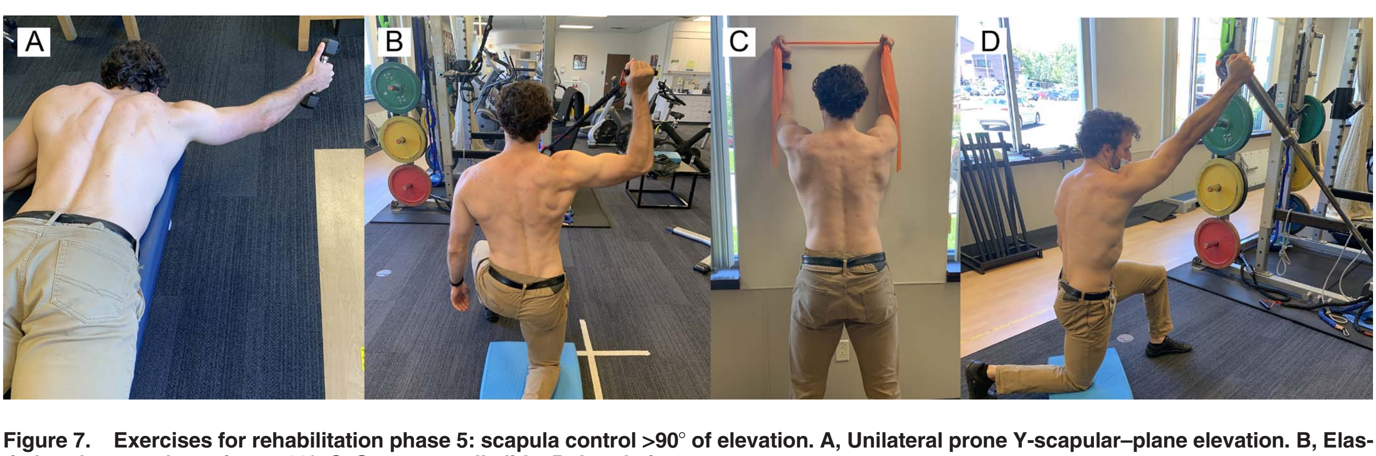

Figure 7. Exercises for rehabilitation phase 5: scapula control >90° of elevation. A, Unilateral prone Y-scapular–plane elevation. B, Elastic-band external rotation at 90°. C, Serratus wall slide. D, Landmine press.

Figure 8. Axillary taping technique.[40]

with neurogenic TOS.[16] Authors of observational studies have demonstrated varying levels of long-term success with botulinum toxins.[47] However, botulinum toxins can help athletes progress in supervised rehabilitation, with short-term results lasting up to 3 months.[48] *SOR: B*

EXPECTED OUTCOMES

Neurogenic TOS

Approximately 60% to 70% of athletes with neurogenic TOS have success with nonoperative management consisting of supervised rehabilitation and activity modification.[48] A 4- to 6-month trial of nonoperative management of neurogenic TOS should be completed before surgery is considered.[16] Operative outcomes for neurogenic TOS have been well established, with 85% to 90% of the general population reporting symptom improvement and similar outcomes reported in the overhead athlete population.[7,15]

Venous and Arterial TOS

Often, for venous or arterial TOS, the initial line of treatment is surgical intervention. Excellent outcomes have been shown after surgical decompression for venous TOS, with most athletes returning to high levels of functional performance within an average of 3.5 months of treatment.[7] With arterial TOS, postoperative RTS is expected by 4.7 months postintervention.[49]

RETURN TO SPORT

Timeline

Timelines for RTS readiness vary greatly depending on symptom severity and cause. With neurogenic TOS managed nonoperatively, gradual RTS can be integrated within 4 to 6 weeks depending on rehabilitation progressions.[50] Postoperative management of neurogenic TOS has a slightly prolonged RTS timeframe. Four to 6 months postoperatively has been recommended for RTS, with athletes continuing to note improvements in strength and function for 9 months to 1 year postoperatively.[50]

Table 11. Return to Sport (RTS) Testing Considerations[51]

Testing Consideration	Criteria
Pain	Pain free with RTS and interval programs
Active and passive range of motion	Equal to preinjury or uninjured contralateral side
Strength: peak isometric force symmetry	Minimum 90% limb symmetry index
Physical performance tests	Closed Kinetic Chain Upper Extremity Test Unilateral seated shotput test Posterior shoulder endurance test Lower extremity and kinetic chain tests
Other	Sport-specific, position-specific, or both physical performance tests, interval programs, etc

Criteria

No functional testing algorithms have been previously established to determine RTS readiness in athletes with TOS. Using previously established upper extremity functional testing algorithms can help objectively determine RTS readiness.[51,52] Schwank et al discussed the importance of individualizing the RTS testing based on sport-specific demands of the individual athlete.[49,52] Table 11 provides minimum RTS considerations.[51]

SUMMARY

Thoracic outlet syndrome is a complex constellation of symptoms and is a diagnosis of exclusion. Multidisciplinary, multimodal management is recommended to ensure optimal patient outcomes. Understanding the pathophysiology of TOS is necessary to perform a comprehensive examination. Rehabilitation is often the first line of management for patients with TOS, focusing on various manual therapies, stretching, and neuromuscular coordination exercises. The individualized physical impairments that may be causing or worsening the patient's symptoms must be addressed.

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