



Masterclass

Thoracic outlet syndrome part 1: Clinical manifestations, differentiation and treatment pathways

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ABSTRACT

Thoracic outlet syndrome (TOS) is a challenging condition to diagnose correctly and manage appropriately. This is the result of a number of factors including the multifaceted contribution to the syndrome, the limitations of current clinical diagnostic tests, the insufficient recognition of the sub-types of TOS and the dearth of research into the optimal treatment approach. This masterclass identifies the subtypes of TOS, highlights the possible factors that contribute to the condition and outlines the clinical examination required to diagnose the presence of TOS.

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1. Introduction

Opinions in the literature about thoracic outlet syndrome (TOS) vary in the extreme, swaying from the belief that it is the most underrated, overlooked and misdiagnosed peripheral nerve compression in the upper extremity (Shukla and Carlton, 1996; Sheth and Belzberg, 2001) to questioning whether it exists (Wilbourn, 1990). These varying beliefs highlight the need for the clinician to be rigorous in their clinical assessment so that patients are not misdiagnosed and are appropriately managed. Unfortunately the diagnosis of TOS remains essentially clinical and is often one of exclusion with no investigation being a specific predictor. This may be attributed, in part, to the fact that TOS is considered to be a collection of quite diverse syndromes rather than a single entity (Yanaka et al., 2004). Consequently, this also results in TOS being one of the most difficult upper limb conditions to manage.

The aim of this paper (Part 1) is to clarify the nomenclature, classification, varying clinical presentations and assessment techniques so that the reader may attempt to assess and differentially diagnose patients presenting with TOS. The second paper (Part 2) will outline specific rehabilitation approaches used by the authors to treat one sub-type of TOS.

2. Definition

A broad definition of TOS is a symptom complex characterized by pain, paresthesia, weakness and discomfort in the upper limb which is aggravated by elevation of the arms or by exaggerated movements of the head and neck (Lindgren and Oksala, 1995).

3. Anatomical considerations

The pain and discomfort of TOS are generally attributed to the compression of the subclavian vein, subclavian artery and the lower trunk of the brachial plexus as they pass through the thoracic outlet (Cooke, 2003; Samarasam et al., 2004; Barkhordarian, 2007).

Three sites of compression of the vessels and nerves are possible (Fig. 1). The lower roots of the brachial plexus may be compressed as they exit from the thoracic cavity and rise up over the first rib (or a cervical rib or band when present) and pass between the anterior and middle scalene muscles (or even sometimes through the anterior scalene muscle). The upper roots of the brachial plexus can also be compressed between the scalene muscles but actually exit the cervical spine not the thorax, and should technically be referred to as cervical outlet syndrome (Ranney, 1996). The second potential site of entrapment is beneath the clavicle in the costoclavicular space, where the neural elements are already outside the thorax. The third potential site is more distal in the sub-coracoid tunnel (beneath the tendon of the pectoralis minor) where the plexus may be stretched by shoulder abduction (Ranney, 1996; Rayan, 1998; Demondion et al., 2003; Wright and Jennings, 2005).

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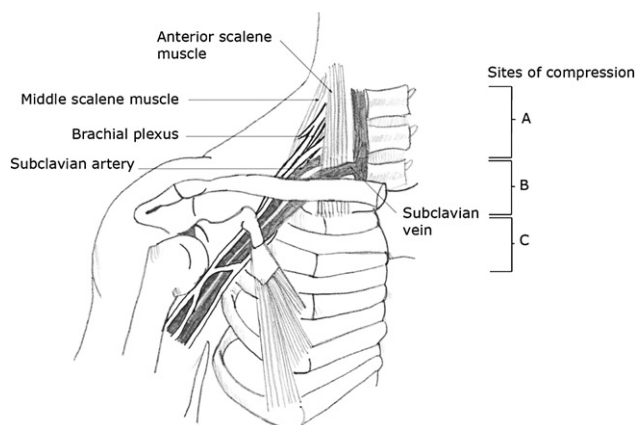


Fig. 1. Thoracic outlet anatomy. Three possible site of compression and structures compressed; A: Subclavian artery and lower roots of the brachial plexus may be compressed as they exit from the thoracic cavity and rise up over the first rib and pass between the anterior and middle scalene muscles. B: Subclavian artery and vein and/or lower trunk of the brachial plexus beneath the clavicle in the costoclavicular space. C: The axillary artery and/or vein and/or one of the cords of the brachial plexus in the sub-coracoid tunnel.

Very rarely is this clarified in the literature and the reader should be aware that many authors utilize the global term of TOS with little attempt to differentiate which sub-type of TOS they are treating. This may well account for the enormous variation in treatment outcomes described. We believe it is essential that the clinician carefully consider and at least attempt to clinically differentiate, where possible, exactly which component of the neurovascular complex is being affected and precisely where it is being compressed. This will direct not only what further investigations are required, but may well impact on what is the most appropriate treatment strategy. In reality this is often easier said than done.

4. Classification and pathophysiology

TOS is often categorized into two specific clinical entities: Vascular TOS (vTOS) and Neurological TOS (nTOS) (Atasoy, 1996; Rayan, 1998; Sharp et al., 2001). vTOS can be divided into arterial and venous TOS syndromes due to compression or angulation of either the subclavian or axillary artery or vein (Rayan, 1998; Davidovic et al., 2003). Usually it is caused by a structural lesion, either a cervical rib or another bony anomaly (Rayan, 1998). Arterial involvement is more common than venous involvement (Davidovic et al., 2003; Singh, 2006) and vTOS is generally easier to define, diagnose and treat than nTOS (Sharp et al., 2001). The subset of patients with bony abnormalities such as cervical ribs, are generally accepted as “true cases” of TOS and this commonly occurs in vTOS and true neurological TOS (tnTOS) (Roos, 1982; Samarasam et al., 2004). tnTOS is caused by irritation, compression or traction of the brachial plexus.

The remaining larger group of patients with no radiological or electro-physical abnormalities are usually labeled as “disputed TOS” (Cherington, 1989) or “non-specific nTOS” (Sobey et al., 1993) or “symptomatic TOS” sTOS (Rayan, 1998). sTOS remains the most controversial form of TOS. There has been some suggestion that this may be an early or mild form of vTOS or nTOS and hence may mimic the symptoms with no definitive evidence for either (Rayan, 1998; Seror, 2005; Lee et al., 2006). In some cases patients may present with “combined TOS,” the simultaneous compression of vascular and neurological structures. This may be mixture of arterial and venous or arterial/venous and neurological or all three.

The classifications for vTOS, tnTOS, and sTOS can be seen in Table 1.

5. Incidence

The incidence of TOS is reported to be approximately 8% of the population (Davidovic et al., 2003), is extremely rare in children (Cagli et al., 2006), and affects females more than males (between 4:1 and 2:1 ratios) (Gockel et al., 1994; Davidovic et al., 2003; Demondion et al., 2003; Degeorges et al., 2004). In particular, tnTOS is typically found in young women (van Es, 2001).

According to Davidovic et al. (2003), 98% of all patients with TOS fall into the nTOS category and only 2% have vTOS. However this figure is clouded by the fact there is no distinction between tnTOS and sTOS (Urschel et al., 1994; Urschel and Razzuk, 1997; Goff et al., 1998). While neurological symptoms appear more prominently, the majority of these will fall into the sTOS classification (Wilbourn, 1990; Rayan, 1998; Davidovic et al., 2003).

6. Etiology

Bony pathology or soft tissue alterations are commonly attributed to the etiology of TOS. Numerous causes have been cited in the literature ranging from congenital abnormalities (anomalies of the transverse process of seventh cervical vertebra, cervical rib, first rib, enlarged scalene tubercle, scalene muscles, costoclavicular ligaments, subclavius or pectoralis minor) to traumatic in origin (such as a motor vehicle accident or sporting incident) (Gruber, 1952; Makhoul and Machleder, 1992; Rockwood et al., 1997; Athanassiadi et al., 2001; Jain et al., 2002; Barkhordarian, 2007). Cervical ribs and other anatomic variations are not prerequisites for the diagnosis of TOS but may be implicated in some cases.

Traumatic bony lesions include bone remodeling after fractures of the clavicle or first rib or posterior subluxation of the acromioclavicular joint. Soft tissue pathologies such as anterior scalene muscle hypertrophy, muscle fibre type adaptive transformation, spasm and excessive contraction particularly post cervical trauma have all been implicated in TOS (Roos, 1982; Machleder et al., 1986; Mackinnon, 1994; Schwartzman and Maleki, 1999; Kai et al., 2001; Pascarelli and Hsu, 2001; Davidovic et al., 2003). Less commonly, upper lung tumors have been implicated in the etiology (Machleder et al., 1986; Makhoul and Machleder, 1992; Barkhordarian, 2007).

Postural or occupational stressors with repetitive overuse and associated soft tissue adaptations such as hypertrophy in some muscles and atrophy in others, have been implicated in all forms of TOS. Poor posture, especially in patients with large amounts of breast tissue or swelling due to trauma in the area, may predispose to TOS. Compression occurs when the size and the shape of the thoracic outlet is altered. This is commonly caused by poor posture, such as lowering the anterior chest wall with drooping shoulders and holding the head in a forward position (Aligne and Barral, 1992; Novak et al., 1995; Ranney, 1996; Rayan, 1998; Skandalakis and Mirilas, 2001; Barkhordarian, 2007).

7. Diagnosis

Diagnosis of TOS is clinical and based on a detailed history, subjective and objective examination of neurovascular and musculoskeletal systems of the neck, shoulder, arm and hands (Roos, 1982; Novak et al., 1995). Frequently a multitude of further investigations are required, many of which in the case of sTOS may indeed prove to be negative (Barkhordarian, 2007). The literature laments that there is no one test or investigation that consistently proves the diagnosis of TOS. Given that TOS really is a “collection” of symptom complexes, often multifaceted, it is unreasonable to

Table 1
Classifications, pathophysiology and investigations.

Classification	Sub-type	Pathology	Signs & Symptoms
Vascular TOS (vTOS)	1. Arterial TOS (aTOS)	Compression of the subclavian artery that produces any combination of stenosis, post-stenotic dilatation, intimal injury, formation of aneurysms and mural thrombosis.	Upper limb ischaemia Multiple upper limb arterial embolization Acute hand ischaemia Claudication Vasomotor phenomena Digital gangrene Absent or decreased arterial pulse Swelling, feeling of stiffness/heaviness, fatigability, coldness, pain of muscle cramp in the upper limb or hand Paresthesia (due to ischaemia)
	2. Venous TOS (venTOS)	Unilateral arm swelling without thrombosis, when not caused by lymphatic obstruction may be due to subclavian vein compression.	Asymmetrical upper extremity oedema (bilateral oedema can occur) Pain, cyanosis, fatigability and a feeling of stiffness or heaviness of the upper extremity Venous engorgement with collateralization of peripheral vessels Axillary or subclavian vein thrombosis Pulmonary embolism Paresthesia
Neurological TOS (nTOS)	1. True Neurological TOS (tnTOS)	Irritation, compression or traction of the brachial plexus creating compromised nerve function. Compression usually occurs via a bony or soft tissue anomaly present congenitally, created by either repetitive or significant trauma and often influenced by postural, occupational or sporting factors.	<i>Upper plexus syndrome – C5/6/7 pattern:</i> Sensory changes in the first three fingers +/- numbness in cheek, earlobe, back of shoulder, or lateral arm Weakness in deltoid, biceps, triceps, scapula muscles and forearm extensors Pain in anterior neck, chest, supraclavicular region, triceps, deltoids, parascapular muscle, outer arm to the extensor muscles of the forearm +/- pain in the neck, pectoral region (pseudoangina), face, mandible, temple and ear with occipital headaches +/- dizziness, vertigo and blurred vision <i>Lower plexus syndrome – C7/8/T1 pattern:</i> Sensory changes in the fourth and fifth fingers, sensory loss above medial elbow Pain and paresthesia over the medial aspect of the arm, forearm, ulnar 1½ digits Hand weakness, loss of dexterity and wasting (lateral thenar muscles, profundus of the little and ring fingers, the ulna intrinsics and the hypothenar muscles and extend into the forearm) Predominantly neurological, intermittent and transient in nature Paresthesia in digits (variable distribution) on awakening Distal symptoms range from pain, aching “spasm” to tingling, numbness and tightness Feelings of weakness and fatigue either in the hand or entire upper limb (especially when it is used overhead) Feeling of tenderness, swelling or loss of motor control Pain in forearm, hands and wrist +/- Pain in lower neck and shoulder, elbow and upper back, especially over pectoralis minor, lateral humerus, suprascapular and medial scapula regions +/- Concurrent cervical pain and headache Pain aggravated by repetitive, suspensory, or sustained overhead forward elevation of the shoulder and activities that depress the shoulder girdle Pain at rest and night pain
	2. Symptomatic TOS (sTOS)	Usually no bony or soft tissue anomaly can be demonstrated. Intermittent compression of the neurovascular complex due to repetitive postural, occupational or sporting forces that create temporary compression at varying sites in the cervical or thoracic outlet	

assume that any one test or one investigation can always accurately examine the whole spectrum of pathology.

Diagnosis of sTOS is dependent on a systematic, comprehensive upper-body examination and several authors highlight that postural exacerbation of symptoms is an essential component of the diagnosis (Roos and Owens, 1966; Novak et al., 1995). Lindgren (1997) first tried to systemize the diagnosis of sTOS by describing a clinical index (Fig. 2). While this index is a good initial guideline, there are other criteria that need to be added to ensure that the sTOS diagnosis is not missed in patients.

7.1. Subjective examination

A detailed global body chart must be completed looking for total distribution of pain, neurological and vascular symptoms not only in the upper limb but in the head, neck, chest and the other side. In particular the type, nature and intensity of symptoms should be

monitored as well as any changes in skin temperature, color, texture, blotching, hair growth, swelling, stiffness or loss of motor control.

Less commonly seen are symptoms of tachycardia or pseudoangina, occipital headache, vertigo, dizziness, and tinnitus (Malas and Ozcarar, 2006). Behaviour of the symptoms should be noted including, morning and/or night pain and any specific

Patients should have at least three of the following four symptoms or signs.

1. a history of aggravation of symptoms with the arm in an elevated position
2. a history of paraesthesia originating from the spinal segments C8/T1.
3. supraclavicular tenderness over the brachial plexus
4. a positive hands up abduction/external rotation or stress test.

Fig. 2. Clinical index for diagnosis of sTOS (Lindgren, 1997).

Table 2
Differential diagnosis.

Differential	Signs in common with TOS	Differing signs	Investigations/Tests
Carpal tunnel syndrome	Paresthesia of the hand (can be the entire hand) Proximal pain Night pain Hand pain aggravated by use	Loss of wrist range of motion (predominantly extension)	Wrist range of motion Tinell's sign Phalen's and reverse Phalen's Tethered median nerve stress test (Pascarlel) EMG and nerve conduction
deQuervain's tenosynovitis	Pain over lateral wrist and thumb	Local tenderness and swelling Pain – resisted thumb extension Pain – passive thumb flexion	Finkelstein's test
Lateral epicondylitis	Pain in lateral forearm	Pain and point tenderness lateral epicondyle Pain – resisted wrist extension, gripping and morning stiffness	Ultrasound scan
Medial epicondylitis	Pain in medial forearm	Pain and point tenderness medial epicondyle Pain – resisted wrist flexion and wringing activities	Ultrasound scan
Complex regional pain syndrome (CRPS I or II).	“Burning” pain in the upper limb, Motor disability	Changes in the color and temperature of the skin over the affected limb, skin sensitivity, sweating, swelling and changes in nail and hair growth.	Investigation autonomic nervous system
Horner's Syndrome	Can co-exist with TOS due to compression affecting nerves as well as stellate ganglion	Ptosis of the eye and a constricted pupil	Radiological, autonomic and neurological investigation to differentiate.
Raynaud's disease	Vasospastic disorder mimic TOS Discolouration fingers and cold sensitivity	Discolouration also of toes (occasionally other extremities) in a characteristic pattern in time: white, blue and red	May need to be excluded from vTOS by an angiogram Allen's test
Cervical disease (especially disc)	May present with pain in cervical spine, radiating in to the upper limb and medial scapula	Symptoms aggravated by cervical movements rather than arm motion. Ease factor may be elevation of the arm whilst this is an aggravating position in TOS	Cervical range of motion Neurological examination (decreased reflex in severe disc pathology) Cervical compression and distraction tests Spurling's maneuver MRI
Brachial plexus trauma	Varying from a neuropraxia to a neurotmesis		Brachial Plexus Traction test
Systemic disorders: inflammatory disease, esophageal or cardiac disease	Upper limb pain +/- chest pain		Nerve conduction tests Blood tests (inflammation) Stress electrocardiography (cardiac disease)
Upper extremity deep venous thrombosis (UEDVT), Paget–Schroetter syndrome	Tightness or “heaviness” in affected biceps muscle, shoulder, neck, upper back and axilla Provocation tests are positive	Hand, upper arm, posterolateral shoulder can be swollen and red with increased tissue temperature over the shoulder Painful limitation of internal and external rotation active motion may be present as well as positive rotator cuff tests Ecchymosis and non-edematous swelling of the shoulder, arm and hand, functional impairment, discolouration and mottled skin and distention of the cutaneous veins of the involved upper extremity Positive rotator cuff testing	This condition can cause a potentially dangerous or even fatal complication.
Rotator cuff pathology	Restricted and painful shoulder range of motion Weakness in shoulder muscles		Clinical tests: - Neer and Hawkins impingement tests - Jobe (supraspinatus) test - Speed's test (biceps) - External rotation test (infraspinatus) - Lift off & press belly test (subscapularis).
Glenohumeral joint instability	History of repeated overuse in the overhead position or trauma “Dead arm” symptoms or transient neurological symptoms	Positive glenohumeral instability testing	Clinical tests: - Apprehension test, - Anterior and posterior draw tests in the adducted & abducted shoulder - The sulcus test - Dynamic anterior & posterior stability tests

aggravating factors especially; sustained shoulder elevation, suspensory holding activities, lying on the arm, carrying a back pack, carrying articles by the side, prolonged postures (especially sitting), repetitive use of the upper limb and hand dexterity.

A detailed history should include the past history of prior traumatic insult to the surrounding neck, shoulder and arm areas that may indicate co-existence of cervical, glenohumeral (especially instability), acromioclavicular or sternoclavicular joint pathology that may confound, confuse or contribute to the clinical presentation (Barkhordarian, 2007). Loss or gain of weight or muscle mass (especially scalenes or pectoralis minor region) should be noted as should a history of use of growth hormone or steroids (Simovitch et al., 2006).

7.2. Physical examination

The physical examination of TOS is frequently long and complex as the clinician needs to examine the entire upper limb and cervical spine. Not only is a neurological examination required, but frequently peripheral nerve entrapment tests also need to be performed.

7.2.1. Postural alignment

Postural malalignment should be examined. The physique of classic TOS patients is that of a long neck with sloping shoulders (Kai et al., 2001). Many other variations of scapula malpositioning or “poor posture” may also occur in TOS (Pascarelli and Hsu, 2001). If sTOS is suspected then specific attention should be made to scapula position both at rest, motion and on loading (Refer to Part 2).

7.2.1.1. Palpation. Upper limb pain or symptom reproduction after digital palpation and palpation tenderness (mechanical allodynia) (Schwartzman and Maleki, 1999), especially in the supra and infraclavicular fossae, are considered to be useful in the diagnosis of nTOS. Morley test or the brachial plexus compression test (compression of the brachial plexus in the supraclavicular region) is considered “positive” if there is reproduction of an aching

sensation and typical localized paresthesia and not just mere tenderness of the area (Hasan and Romeo, 2001). This test is reported to be positive in up to 68% of patients with nTOS (Seror, 2005). In some cases fullness or even a palpable hard mass may be present in the supraclavicular region (Cagli et al., 2006). This may be an indicator of a true structural lesion potentially creating either vTOS or tnTOS but the mass itself must also be examined (chest x-ray and ultrasound) to make sure it is not of a more significant nature (Ozguclu and Ozcakar, 2006). Palpation distally may also be required if local joint pathology or peripheral nerve entrapment needs to be excluded.

7.2.1.2. Active/passive motion. Active and passive motions of the cervical spine, cervicothoracic junction, shoulder, elbow, wrist and hands should be performed looking for; joint hyperlaxity, limitation of motion, dyskinesia or abnormal compensatory motions or symptom reproduction (Pascarelli and Hsu, 2001). At a minimum, cervical and shoulder range of motion should be objectively documented using a goniometer or inclinometer at initial assessment. Restriction of glenohumeral joint range of motion has been noted by several authors in sTOS (Sucher, 1990; Aligne and Barral, 1992; Rayan, 1998). This restriction may be due to the increased anterior tilt of the scapula.

Any shoulder, scapula, elbow, wrist or hand muscle weakness should also be objectively assessed preferably using an objective assessment device (such as a dynamometer) or at the very least by using the standard 0–5 classification (Kendall et al., 1971).

7.2.1.3. Rotator cuff tests and glenohumeral joint instability tests. Rotator cuff tests are examined for pain, weakness, and symptom reproduction to assess rotator cuff pathology (Table 2). If there is a history of repeated overuse in the overhead position (throwing athlete) or trauma, then the glenohumeral joint should be examined for instability (Table 2).

7.2.1.4. Neurological examination. A thorough neurological examination of the upper extremities, including motor, sensory and deep tendon reflexes is essential. Sensation can be measured to light

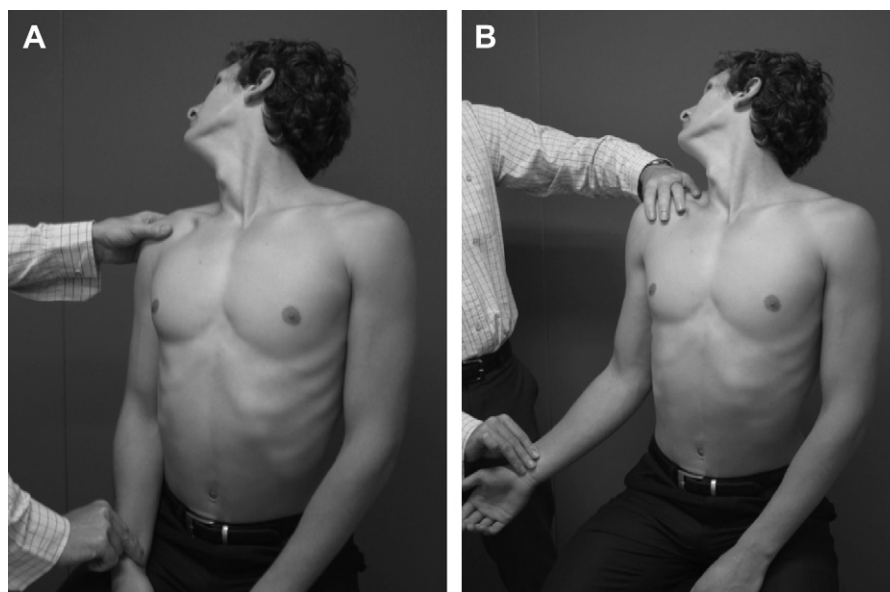


Fig. 3. Adson's maneuver. A. Patient seated upright. Arms remain supported in patient's lap and the patient performs cervical spine rotation and extension to the tested side. This is followed by a deep inspirational breath, which is held for up to 30 s, as the examiner palpates for any changes in the radial pulse. B. Modification – perform in 15° shoulder abduction and maintain the head in the tested position for 1 min while the subject breathes normally.

touch and pin prick at a minimum but if possible by Semmes–Weinstein monofilament testing (Gillenson et al., 1998). Attention should be given to; skin temperature, presence of tremor, atrophy and swelling (Pascarelli and Hsu, 2001).

A decrease in sensation and strength in the absence of any aberrance of the deep tendon reflexes may indicate tnTOS. Any change in deep tendon reflexes (regardless of any change in sensation and motor control) may indicate a more proximal or central neurological pathology and needs to be referred on for further investigation.

Muscle weakness will be manifested in either C5,6 muscle groups (upper plexus) or C8, T1 muscle groups (lower plexus) reflected by poor grip strength. This is not common and largely presents in tnTOS (Athanassiadi et al., 2001).

7.2.1.5. Peripheral nerve tests. There are many peripheral examination tests that can be performed. Carpal tunnel syndrome (CTS) is the most commonly cited peripheral nerve entrapment that may be

confused with TOS and therefore should be assessed as part of the standard physical examination (Seror, 2004, 2005). Further tests may be required as part of differential diagnosis (Table 2).

7.2.1.6. Cervical spine. In addition to active/passive range of motion, scalene muscle tightness should be examined. Restriction of cervical range and scalene muscle tightness is more likely associated with upper plexus entrapment (Skandalakis and Mirilas, 2001). Cervical nerve root compression caused by cervical disc disease should be excluded (Table 2) using a test such as Spurling's test (Spurling and Bradford, 1939; Bradford and Spurling, 1942). Thoracic spine kyphosis or scoliosis should be noted and any compensatory lordosis (Sucher, 1990).

7.2.1.7. Provocation testing. The provocation tests most commonly described in the literature to diagnose TOS are presented in Figs. 3–6. These tests are purported to help delineate the possible level of compression of the neurovascular structures in either the

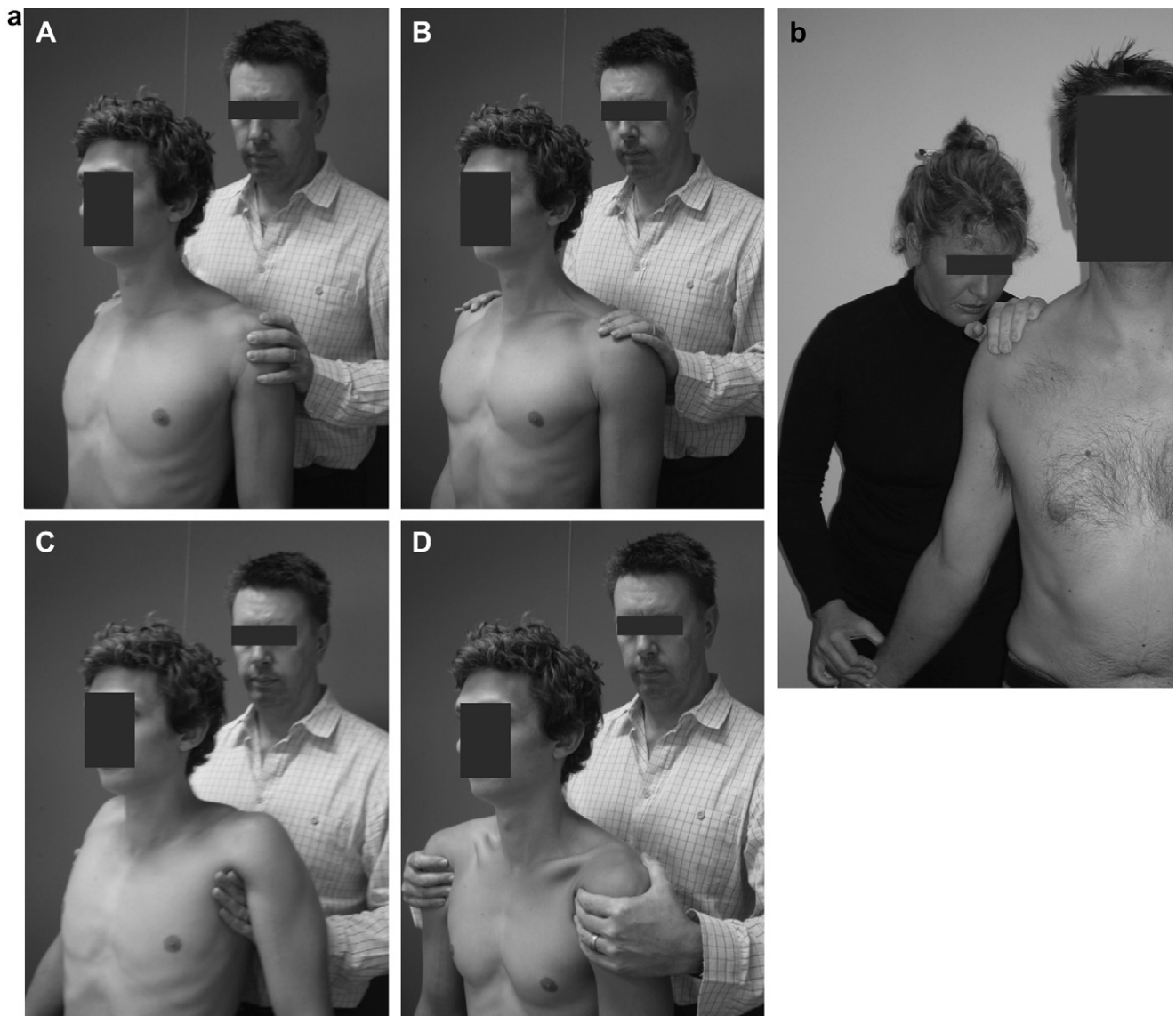


Fig. 4. a – Costoclavicular maneuver. Patient sitting, therapist assists the patient in performing scapula retraction (A), depression (B), elevation (C) and protraction (D), holding each position for up to 30 s. Subject rests his or her forearms on thighs while the examiner simultaneously monitors a change in pulse and symptom onset, note which positions exacerbates/eases symptoms. Recommended modification: performed with both arms by the side, holding each position for 1 min. b – Modified to the military brace position – exaggerates backward and downward bracing of the shoulders. This movement obliterates the pulses most readily.



Fig. 5. Wright's test – Hyperabduction maneuver. The test is performed in two steps. The patient sits in a comfortable position, head forward, while the arm is passively brought into abduction and external rotation to 90° without tilting the head. The elbow is flexed no more than 45°. The arm is then held for 1 min (Rayan and Jensen, 1995). The tester monitors the patient's symptom onset and the quality of the radial pulse. The test is repeated with extremity in hyperabduction (end range of abduction).

scalene, costoclavicular or axillary (sub-coracoid) intervals. Given the numerous possible causes and symptoms associated with TOS, no single test can unequivocally establish the presence or absence of the condition, particularly where sTOS is concerned (Roos, 1982; Lindgren, 1997). The classic provocation tests have been reported to be unreliable and frequently positive (up to 90%) for pulse obliteration in healthy patients (Hachulla et al., 1990; Urschel et al., 1994; Rayan and Jensen, 1995; Nannapaneni and Marks, 2003). No study to date has analyzed the specificity, sensitivity and predictability of the provocation tests in relationship to the separate categories of TOS.

7.2.1.7.1. Adson's maneuver. This test is considered positive if there is an obliteration or diminution of the radial pulse and/or a precipitation of patient's symptoms (Fig. 3) (Adson and Coffey,



Fig. 6. Roos stress test – EAST test (elevated arm stress test) The patient sits with the head in the neutral position, the arms abducted and externally rotated to 90° and the elbows flexed to 90°. The patient is then requested to flex and extend the fingers for up to 3 min. The examiner watches for any dropping of the extremity during this time, which could indicate fatigue or arterial compromise. The therapist should also observe the color of the distal extremity, comparing left with right and monitor symptoms onset.

1927; Leffert and Perlmutter, 1999). Distribution of pain +/- paresthesia should be noted and graded as mild, moderate, severe (Rayan and Jensen, 1995). The importance of the obliteration of the pulse has been questioned, especially in nTOS (Gergoudis and Barnes, 1980). This test is thought to stress the scalene triangle but may also stress the contralateral scalene triangle, indirectly bringing on symptoms (Walsh, 1994).

7.2.1.7.2. Costoclavicular maneuver. This maneuver (Fig. 4) is thought to stress the costoclavicular interval where either the subclavian artery, vein or brachial plexus may be entrapped by structures such as subclavius or costocoracoid ligament (Falconer and Weddell, 1943). The test is positive when radial pulse changes and/or patient's symptoms are provoked.

7.2.1.7.3. Wright's test – hyperabduction maneuver. The stress hyperabduction test (Fig. 5) is thought to implicate the axillary interval (space posterior to pectoralis minor) in the etiology of TOS (Wright, 1945). The test has two components and a positive result is a decrease in the radial pulse and/or reproduction of the patient's symptoms. Distribution and severity of symptoms should be recorded (Rayan and Jensen, 1995). The first part of the maneuver could implicate the subclavian vessels and plexus as they are stretched around the coracoid process (pectoralis minor impingement). The second part places the extremity in hyperabduction. A positive test is said to implicate the costoclavicular interval (Walsh, 1994). Other authors have described adding on the effect of cervical spine motion (flexion, extension, left and right rotation) (Seror, 2005).

7.2.1.7.4. Roos stress test – EAST test (elevated arm stress test). Originally described by Roos and Owens (1966) and purported by the developers to be the most sensitive and specific test to detect nTOS (Fig. 6). This test is believed to stress all three intervals (scalene, costoclavicular, axillary) since this position places the arterial, venous and nervous systems in tension. The test is positive when the patient is unable to maintain elevation for the 3-min period or when symptoms are induced (Roos and Owens, 1966; Walsh, 1994). Accuracy of the test is reported to be best at angles of 90° or less (Hachulla et al., 1990). Despite the test being deemed the most sensitive and specific of the provocation tests (Roos and Owens, 1966), a study by Seror (2005) showed that 14% of patients could not complete the 3-min test with their symptomatic upper limb and 58% of patients with confirmed CTS also had positive stress tests. In the same study only 5% of patients with CTS had a positive Adson's test (Seror, 2005).

7.2.1.7.5. Other considerations. When using provocation tests for cases of vTOS then obliteration of the radial artery pulse, looking for distal ischaemic signs, oedema, and cyanosis of the upper extremity, measuring blood pressure and auscultation for a bruit in both upper extremities with the arms by the side and in provocation positions (Athanasassiadi et al., 2001) is required and likely to be significant if found positive (Singh, 2006). In cases of nTOS it would be logical that provocation tests should not only be performed to obliterate the radial artery pulse but also to recreate the patient's discomfort and symptoms (Konin et al., 1997). Due to the low specificity of these various tests, some authors argue that if these tests are being utilized for a diagnosis of sTOS then two or three tests should be positive in a given patient (Hachulla et al., 1990).

7.2.1.8. Postural and scapula correction. Exacerbation of symptoms during testing by alterations in posture is considered a strong argument for the diagnosis of sTOS (Hachulla et al., 1990; Rayan and Jensen, 1995). Poor posture or malalignment of the shoulder girdle (such as drooping or rounded shoulders) has been cited by many authors as being a potential cause of sTOS due to the alteration of the anatomical position of the shoulder girdle potentially

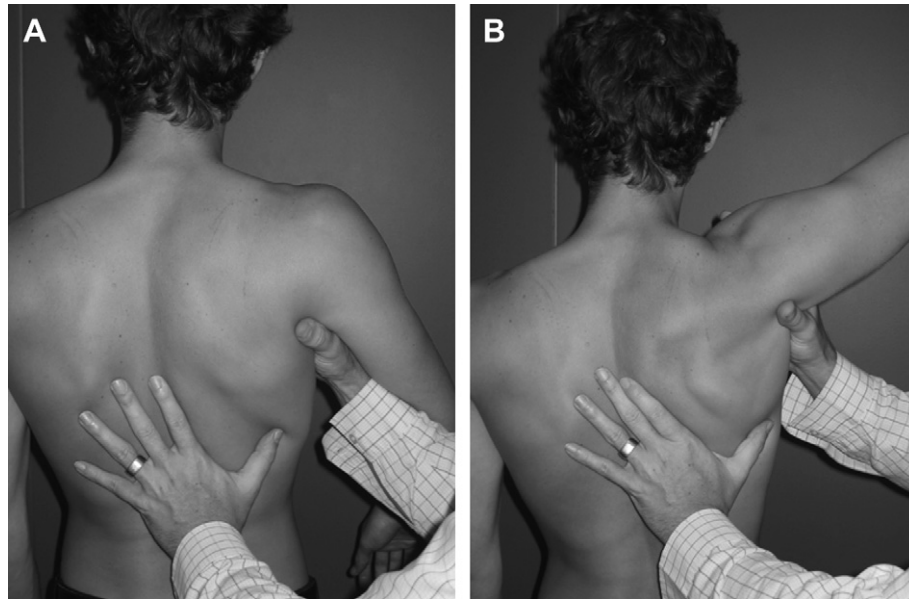


Fig. 7. Correction of scapula position – Elevation/Upward Rotation. Performed in standing with the examiner behind the patient. Patient's symptoms are provoked either by performing an active motion (such as abduction) or one of the Provocation tests. The point in range or time to onset of symptoms is objectively noted. Examiner then places his/her hand in the patient's axilla (A). The examiner's thumb in the posterior axilla, fingers anterior. The scapula is then passively elevated until it is level with the other side (if the patient has unilateral symptoms) or to a level that approximates the normal resting position of the scapula. A small component (10° – 15°) of scapula upward rotation may also be added. Correction is maintained for 1 min as this will allow tractional forces to be relieved off the brachial plexus. Any alterations in the patient's resting symptoms are noted. The patient then performs the active movement or provocation test whilst the examiner maintains the passive correction force. Any alteration in patient's symptoms – distribution, intensity, location or type (worsening or improvement) should be noted. A positive correction response is a significant reduction or absence of pain or an increased time duration in the ability to hold provocation positions or perform the stress test before symptom onset.

decreasing the space available in either the scalene triangle, the thoracic outlet, costoclavicular space or sub-coracoid tunnel.

Clinically we have found that manual correction of the scapula position is an extremely useful clinical sign to help establish the diagnosis of sTOS and to determine if rehabilitation strategies that focus on strengthening of the scapula stabilizers and altering the scapula position at rest and in motion are likely to be successful (see Figs. 7 and 8). Attempts should be made, where possible, to objectively document the scapula asymmetries observed (Watson et al., 2005).

As a general rule, if correction of the scapula improves the patient's symptoms then the test is positive. If the patient's symptoms are aggravated or not changed by repositioning then the test is negative. This would indicate that either the wrong correction position for the scapula has been chosen or the patient may not have a form of TOS that will be assisted by rehabilitation strategies for the scapula. This may help establish the diagnosis of either vTOS or nTOS and may indicate greater likelihood that surgical intervention is required.

8. Differential diagnosis

The first step in the differential diagnosis of TOS is to separate it from other painful conditions of the upper extremity and neck. Other pathologies may mimic TOS or have some clinical overlap (Table 2). It should be taken into account that co-existence of pathology can occur. Upton and McComas (1973) introduced the 'double crush' hypothesis, stating that a proximal level of compression could cause more distal sites along the nerve to be more susceptible to compression (Mackinnon, 1994). This hypothesis is extremely pertinent for the patient with nTOS who may be symptomatic from a combination of multiple levels of nerve compression. Each site in and of itself may not be significant to produce symptoms but the cumulative effect of minor

compression at several sites along the nerve will result in significant symptoms. The most commonly seen clinical picture is the association of carpal and ulnar nerve compression with TOS (Nannapaneni and Marks, 2003) but a similar phenomenon has been reported with cervical spine pathology and TOS (Kai et al., 2001).

9. Treatment

Treatment strategies for TOS, particularly with regard to surgical intervention, remain highly controversial. The available literature does not provide strong support either for or against surgery or conservative management (Degeorges et al., 2004). The sub-type of TOS to some extent determines the appropriate treatment pathway. Part 2 of this article will comprehensively outline conservative management.

vTOS generally requires surgical treatment and surgery usually involves decompression of the thoracic outlet with removal of the cervical rib (if present) and/or first rib excision together with associated muscles and other soft tissue structures as indicated (Gergoudis and Barnes, 1980; Urschel and Razzuk, 1991; Bondarev et al., 1992; Atasoy, 1996; Pupka et al., 2004). The general consensus is that surgery is usually required for aTOS since there is often a structural lesion demonstrated. The decision to operate for venous symptoms is often more difficult as many patients have no bony structures that can be proven responsible for compression. Initially conservative management may be trialed (thromboembolytic therapy and monitoring) but if symptoms persist or progress then decompression may be required (Jamieson and Chinnick, 1996; Azakie et al., 1998; Sultan et al., 2001).

In nTOS there is a high association with structural anomalies (such as a cervical rib) and objective confirmatory tests are positive, potentially justifying surgical intervention. Despite this, many authors still recommend a trial of conservative treatment and only



Fig. 8. Scapula correction for posterior tilt. Performed when there is an increased anterior tilt or winging of the scapula. Examiner then places one hand anteriorly over the coracoid process and the other hand posteriorly over the blade of the scapula. The scapula is then passively tilted posteriorly and pressure maintained for 1 min. Any alterations in the patients resting symptoms are noted. The patient then reperform the active movement or provocation test whilst the examiner maintains the passive correction force. If symptoms are worsened with posterior tilt then the test should be repeated applying correction of elevation as well as posterior tilt.

perform surgery if neurological symptoms such as muscle wasting progress (Dale, 1982; Mingoli et al., 1995; Sanders and Hammond, 2002; Degeorges et al., 2004).

In STOS there is often no obvious structural cause and objective confirmatory testing may be lacking. The optimal approach for both surgical and conservative treatment remains controversial and variable (Lindgren, 1997; Sharp et al., 2001). Conservative management is almost universally accepted as the first step (Sharp et al., 2001).

10. Conclusion

To diagnose TOS is a difficult process that requires time and effort. Given that the etiology of TOS is multifactorial and the signs and symptoms so varied, it would appear logical that physical therapy can successfully be employed in the optimal management of TOS patients (both conservative and surgical).

There is a need for the development of a systemized approach to conservative management for TOS (refer to Part 2). If a better objective framework can be established this could facilitate communication between the disciplines to improve patient selection for both surgical and conservative treatment and help develop treatment algorithms that do reliably achieve consistently good or excellent objective treatment outcomes that are sustainable.

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