NEUROGENIC THORACIC OUTLET SYNDROME: AN INDEPTH REVIEW

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1. LITERATURE REVIEW

1.1 HISTORY OF THORACIC OUTLET SYNDROME

Thoracic Outlet Syndrome (TOS) is currently one of the most controversial syndromes in medicine in terms of definition, anatomy, aetiology and treatment. Although the symptomatology of TOS was described over 150 years ago, it was only in 1956 that the term 'Thoracic Outlet Syndrome' was suggested in an attempt to include a number of entities with similar symptoms.[1, 2]

Compression of the subclavian artery was the first event documented as a complication of TOS – initially known as Scalenus Anticus Syndrome, but it was thereafter realised that the subclavian vein and brachial plexus could also undergo compression and that the pressure could be from structures other than the anterior scalenus muscle.[2]

Sir Paget described axillary vein thrombosis in 1875 and the entity was more clearly defined by Von Schroetter 9 years later. In 1945 Hughes described venous TOS as effort-related thrombosis and named it: 'Paget-Schroetter Syndrome'. [2,3]

Adson described his test for arterial occlusion in 1927.[2]

Conservative management including physical therapy and operative treatment for recurrent symptoms have all been promoted as the best practice by different authors.[1,4,5]

In 1742 Hunauld described the importance of a cervical rib as a cause of symptoms. More than a century later cervical rib resection was performed.[6]

Adventitious ligaments and the costoclavicular ligament were noted to also be culprits of the pathology and Rosati and Lord added claviculectomy to the standard anterior scalenectomy and cervical rib resection.[6]

By 1903 the first rib was recognised as a potential cause of symptoms and resection thereof was incorporated into the operative treatment.[6]

A posterior thoracoplasty incision, an anterior and a transaxillary approach were all described for resection of the first rib and favoured by different authors.[6]

Whilst it took many years to define the syndrome and develop surgical techniques it still has controversies making the syndrome a continued area of interest and research.

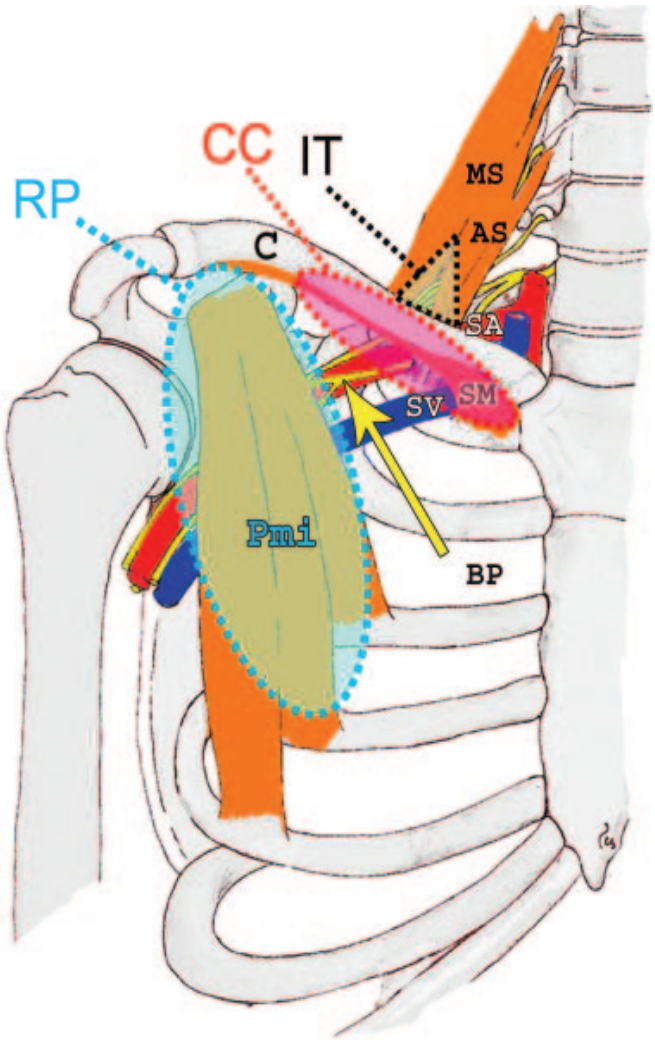
1.2 DEFINITION OF TOS

The definition of TOS is generally accepted as: " Upper extremity symptoms due to compression of the neurovascular bundle in the area of the neck just above the first rib".[1]

1.3 ANATOMY OF TOS

Normal anatomy

The thoracic outlet, also known as the cervicothoracobrachial junction, consists of three important compartments through which vital structures such as nerves and blood vessels course. These compartments are the ***interscalene space*** (the most commonly involved compartment in TOS), the ***costoclavicular space*** and the ***retropectoralis minor*** space [4] and can be seen in figure 1.



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Figures 1, 2. **(1)** Diagram shows the three compartments of the thoracic outlet and their components. *AS* = anterior scalene muscle, *BP* = brachial plexus, *C* = clavicle, *CC* = costoclavicular space, *IT* = interscalene triangle, *MS* = middle and posterior scalene muscles, *Pmi* = pectoralis minor muscle, *RP* = retropectoralis minor space, *SA* = subclavian artery, *SM* = subclavius muscle, *SV* = subclavian vein. **(2)** Anatomic sections show the compartments of the thoracic outlet. **(a)** Section obtained after removal of the pectoralis major muscle shows the costoclavicular space (red oval) and retropectoralis minor space (yellow oval). *Pmi* = pectoralis minor muscle. **(b)** Section obtained after removal of the pectoralis minor muscle shows the neurovascular bundle. *C* = clavicle, straight black arrow = axillary artery, curved black arrow = axillary vein, white arrow = brachial plexus.

Figure 1: Diagram depicting the three compartments involved in Thoracic Outlet Syndrome.

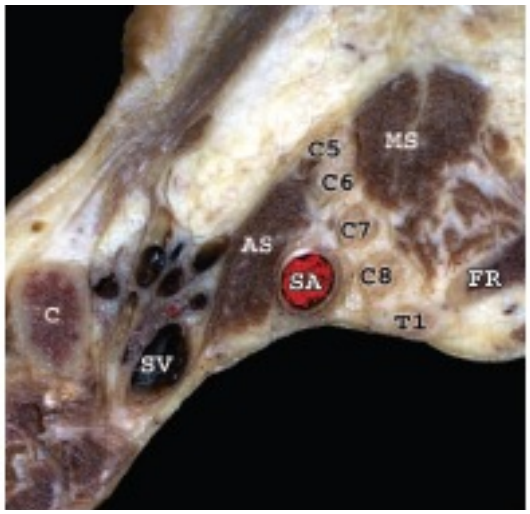
(With permission from Xavier Demondion, MD et al, Imaging assessment of Thoracic Outlet Syndrome. Radiographics 2006; 26: 1735-1750)

The **interscalene space** is triangular in shape and lies medially. It is bounded by the anterior scalenus muscle anteriorly and the medial and posterior scalenus muscles posteriorly with the first rib forming the base. [4]

The anterior scalene muscle, arising from C3-C6 transverse processes, inserts onto the scalene tubercle of the first rib. The middle scalene muscle arises from C2-C7 transverse processes and inserts onto the first rib posterior to the scalene tubercle. The posterior scalene muscle, arising from C4-C6 transverse processes, inserts inferiorly onto the 2nd rib.[4]

The structures within the triangle include the subclavian artery, which lies within the subclavian groove on the 1st rib, and the three trunks of the brachial plexus. The lower trunk (C8/T1) lies behind the artery while the upper and middle trunks lie above the artery in the triangle.[4]

The interscalene triangle as well as all the structures that traverse this space can be seen below, in figure 2.



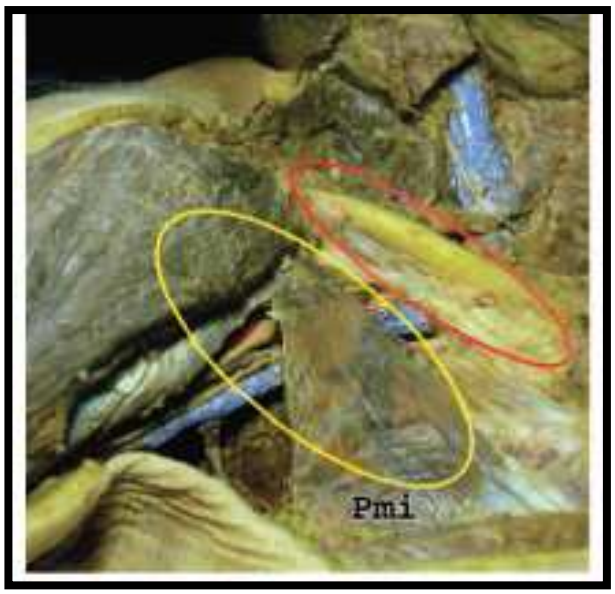
Interscalene triangle
C5-T1 = nerve roots
MS = middle & posterior scalene muscles
AS = anterior scalene muscle
C = clavicle
FR = first rib
SA = subclavian artery
SV = subclavian vein

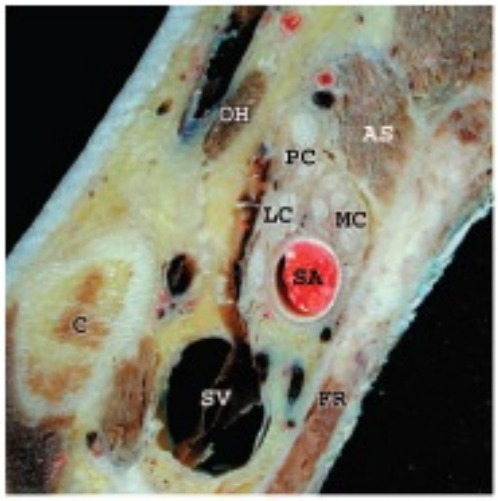
Figure 2: The interscalene space

(With permission from Xavier Demondion, MD et al, Imaging assessment of Thoracic Outlet Syndrome. Radiographics 2006; 26: 1735-1750)

The **costoclavicular space** (see figure 3 below) is also triangular in nature with the clavicle forming the roof, the subclavius muscle anteriorly and the first rib and anterior scalenus muscle together comprising the posterior portion.[4]

Structures running through this space include the subclavian artery, subclavian vein and the lateral, medial and posterior cords of the brachial plexus.[4]



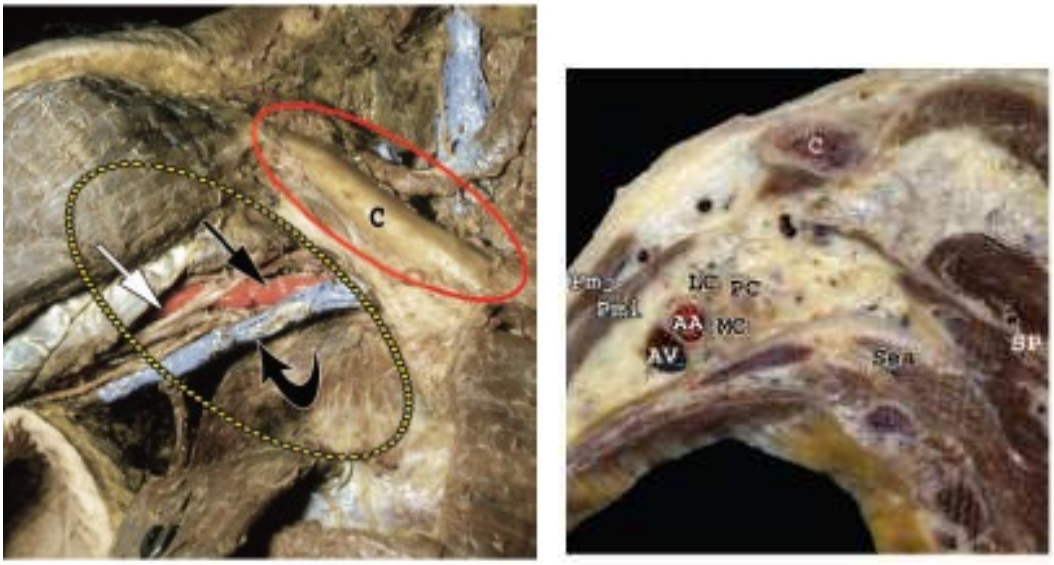


Costoclavicular space
AS = anterior scalene muscle
C = clavicle
FR = first rib
LC = lateral nerve cord
MC = medial nerve cord
OH = omohyoid muscle
PC = posterior nerve cord
SA = subclavian artery
SM = subclavius muscle
SV = subclavian vein

Figure 3: Costoclavicular space

(With permission from Xavier Demondion, MD et al, Imaging assessment of Thoracic Outlet Syndrome. Radiographics 2006; 26: 1735-1750)

The most lateral space in the cervicothoracobrachial junction is the **retropectoralis minor space** (see figure 4 below). This space is bordered by the pectoralis minor muscle anteriorly, the subscapularis superiorly and posteriorly and the anterior chest wall forming the posterior and inferior border. The cords of the brachial plexus travel through this space dividing into their five terminal branches after exiting the space. Once again, the subclavian artery and vein traverse this susceptible region.[4]



Retropectoralis minor space

AA = axillary artery
AV = axillary vein
C = clavicle
LC = lateral nerve cord
MC = medial nerve cord
PC = posterior nerve cord
Pmi = pectoralis minor muscle
Pmj = pectoralis major muscle
Sea = serratus anterior muscle
SP = scapula

Figure 4: Retropectoralis minor space

(With permission from Xavier Demondion, MD et al, Imaging assessment of Thoracic Outlet Syndrome. Radiographics 2006; 26: 1735-1750)

1.4 PATHOGENISIS

Compression of the neurovascular structures in any of these spaces may result in features of TOS.

Neurogenic TOS, resulting from nerve compression, is the commonest pathology accounting for over 95% of TOS cases. This is followed by venous compression identified in 2-3% of cases and lastly arterial entrapment making up only 1% of TOS symptoms.[1]

The majority of cases result from anatomical distortion at the interscalene triangle.[4]

The lower roots of the brachial plexus are at risk of compression as they rise to traverse the first rib (or cross over an anomalous cervical rib) and are sandwiched between the anterior and middle scalene muscles. The upper roots may be compressed between the scalene muscles whilst exiting the neck, and are more accurately labelled Cervical Outlet Syndrome.[5]

The brachial plexus roots can be identified in figure 5 below as they exit the interscalene space and are susceptible to compression at the inferior angle of the interscalene triangle.

For various reasons the described anatomical spaces transform and evolve into 'entrapment spaces'. The aetiology of this is largely unclear but it is thought that changes may be congenital or acquired and that these alterations involve either the bony structures or the soft tissues.[1,4]

Of note, is that a congenital abnormality alone may not produce symptoms but when combined with an acquired change the syndrome may develop.[1,5]

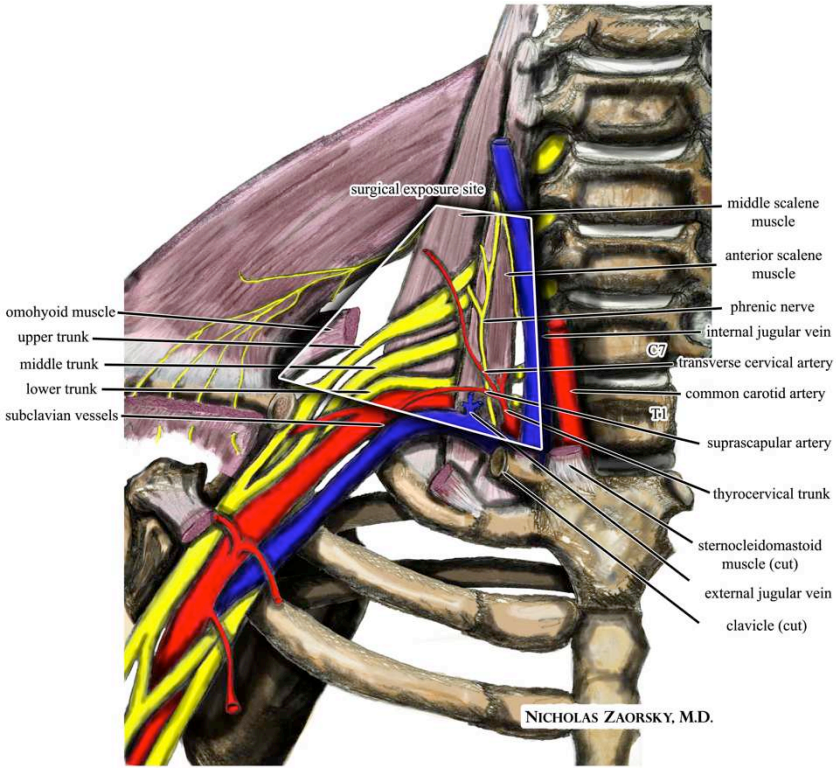


Figure 5: Interscalene space – site of nerve entrapment

(From: http://en.wikipedia.org/wiki/File:Wikipedia_medical_illustration_thoracic_outlet_syndrome_brachial_plexus_anatomy_with_labels.jpg)

Bony abnormalities include:

1. Cervical Rib

A cervical rib is defined as a supernumery rib arising from the 7th cervical vertebra and may be complete or incomplete. A complete rib will fuse with the scalene tubercle forcing the subclavian artery to lie anteriorly in a narrow space between the cervical rib and the anterior scalene muscle, which also inserts into the scalene tubercle. An incomplete cervical rib often has a fibrous band that inserts onto the tubercle, resulting in the same compression as a complete rib, but is not ossified and therefore not detected on x-ray imaging.[4]

Approximately 0.5%-1.0% of the population have cervical ribs. Of these only 10% will develop TOS.[7]

Of patients with TOS, 3-30% have a cervical rib. Thus it can be concluded that a cervical rib predisposes one to TOS but other factors are required for the development of the true pathology. In addition, a cervical rib is not a prerequisite for the development of TOS. [7]

2. Elongated transverse process of C7

Elongation of C7 is diagnosed if the tip of the transverse process extends beyond the tip of the inferior T1 transverse process. It may produce neurogenic symptoms in a similar manner to a cervical rib.[4]

3. Abnormal first rib or clavicle[4,5]

The first rib plays a role in neurological pathology of the interscalene space as it tents the lower trunk of the brachial plexus and the subclavian artery.

It also plays a part, along with the clavicle, in forming the costoclavicular space.

A fused first and second rib may result in a bony mass that narrows the interscalene space with resultant neurogenic pathology.

Trauma in the form of fracture with callus formation or posterior subluxation of the acromioclavicular joint and disease processes such as exostosis may alter these bony structures distorting the outlet compartments with resultant TOS.

Soft Tissue Abnormalities

1. Scalene Muscle Abnormalities

A large variety of scalene muscle abnormalities have been described.

These include hypertrophy of the anterior scalene muscle, a common origin of the anterior and middle scalene muscles dividing into two separate muscles more distally, an enlarged middle scalene muscle inserting more anteriorly than usual, amalgamation of the anterior and middle scalene muscles and supernumerary muscles including the presence of a scalenus minimus muscle.

These additional muscles originate from C6-C7 and insert onto the first rib behind the scalene tubercle or to the cupola of the lung.[4]

The brachial plexus may also run through the anterior scalene muscle rather than between it and the middle scalene muscle.[4]

2. Anomalous Fibrous Bands

Numerous variations of fibrous bands have been described.

Origins of these bands include cervical ribs, first ribs, elongated C7 transverse processes and the anterior and middle scalene muscles. Insertion is onto the first rib or cupola of the lung.[4]

It is interesting to note that some bands may be fibromuscular in nature and exhibit variations similar to the scalene muscle abnormalities.[4]

Fibromuscular ligaments have been demonstrated in 33% of the normal population (cadaveric dissection) whilst patients with TOS have a 98% incidence of a fibromuscular band.[7]

This indicates that there may be an underlying congenital aetiology for TOS and symptoms develop if the patient has additional anatomical alterations from trauma or disease.[1,7]

3. Acquired soft tissue abnormalities.

These acquired pathologies are further classified into patients having received direct trauma to anatomically vulnerable areas and those who have experienced repetitive microtrauma.[4,5]

Flexion – extension neck injuries, typically whiplash, accounts for the injury in the former group.[4]

Patients whose jobs or activities entail repeated elevation of the upper limb form the latter group.[4]

It is postulated that this results in fibrosis and spasm of the scalene muscles elevating the first rib and thus distorting the interscalene space.[4]

Alternatively, microtrauma may induce perineural fibrosis.[4]

Poor posture has also been shown to produce symptoms of TOS. Drooping shoulders, hanging the head forward and lowering the anterior chest wall will narrow the thoracic outlet space. Such a patient is usually thin, not having experienced muscle trauma and in the absence of muscle hypertrophy. Clearly, correction of the posture is curative in this instance.[5,7]

1.5 PATHOLOGY

Scalene muscle specimens of patients undergoing surgery for TOS have been sent for histological examination in only 2 reported studies. The findings were non-specific demonstrating muscle fibrosis only. TOS typically occurs after neck trauma involving hyperextension. The patient then experiences neck pain during the first few days due to interscalene haemorrhage and swelling. With time, scar tissue develops, compressing the brachial plexus with resultant arm and hand symptoms after several weeks.[1]

Arterial TOS almost always has a definite underlying anatomical abnormality causing compression of the artery. Resultant post-stenotic dilatation ensues and clot forms in the aneurysmal sac. Embolisation of this clot is the major cause of symptoms.[1]

The anatomical abnormality is typically a cervical rib or anomalous first rib and thus a thoracic inlet x-ray makes for a good screening test.[1,4]

Symptoms are usually spontaneous with no precipitating cause.[2]

Adolescents with vascular TOS are more symptomatic than adults.[8]

Venous TOS may be thrombotic or non-thrombotic. The thrombotic form of venous TOS, also known as Paget-Schroetter Syndrome, Primary Subclavian Thrombosis or

Upper Limb Effort Thrombosis, is caused by a combination of predisposing and precipitating factors.[1]

The predisposing cause is an anatomical narrowing of the subclavian vein at the junction of the internal jugular vein and subclavian vein. The precipitating cause is usually overuse of the arm, although a coagulopathy may precipitate the condition.[1]

Thrombosis may be idiopathic with no demonstrable anatomical abnormality. Coagulopathies must be sought in such patients.[1]

Typically females tend to have idiopathic upper limb thrombosis whilst males experience effort related thrombosis.[3]

The non-thrombotic form of TOS is caused by intermittent obstruction from the subclavius tendon and anterior scalene muscle.[3]

1.6 CLINICAL FEATURES

The clinical features of TOS are controversial and debatable as the differential is wide and the diagnosis is usually established by exclusion.[4]

Incidence

TOS affects 8% of the population, being exceptionally rare in children.[7]

TOS typically occurs in female patients, being four times more common than in males, between the ages of 20-40 years. Vascular TOS has a higher incidence in adolescents than in children.[8]

Neurogenic TOS accounts for approximately 95% of cases, arterial for 1% and venous TOS the remainder.[2]

Classification

Thoracic Outlet Syndrome has been classified in numerous ways. The most common and sensible description is that of the anatomical structure involved – neurogenic, arterial or venous TOS.

Wilbourn combined anatomical and aetiological classifications and described 5 types: true neurogenic, disputed neurogenic, venous, arterial and traumatic.[1] In this classification true neurogenic is evidenced by objective findings, such as a cervical rib and only accounts for 1% of all neurogenic TOS.[1] Traumatic TOS refers to TOS caused by clavicular fractures. This is slightly inaccurate as trauma plays a large role in development of the syndrome as discussed above. Neurogenic TOS may produce more vague symptoms that are varied and do not exhibit a structural abnormality. In these situations they are labelled as disputed TOS rather than true neurogenic TOS.[1]

Other classifications describe true TOS with regard to arterial TOS, venous TOS or neurogenic symptoms caused by a structural abnormality. This is because vascular TOS usually has evidence of a structural abnormality causing the symptoms, which are generally quite obvious and typical. Symptomatic TOS encompasses the class of patients with neurogenic TOS in the absence of an anatomical abnormality analogous to disputed TOS described above.[7]

1.7 DIAGNOSIS

Diagnosing arterial and venous TOS is usually straightforward but the diagnosis for neurogenic TOS is not always clear-cut and a wide differential exists for these symptoms.

The neurovascular bundle may be compressed in any or all three of the spaces of the thoracic outlet. Three separate syndromes exist – neurogenic, arterial or venous, but

these may overlap and present with combined pathologies. The symptoms of TOS can usually be reproduced on elevation of the arm.[4]

Clinical diagnosis

Clinical diagnosis is based on a detailed history and examination of the neck, shoulder, arm and hands. The symptoms obviously depend on which structures are involved.[5]

Neurogenic TOS may produce sensory or motor symptoms depending on the nerves undergoing compression. The majority of patients with neurogenic TOS have a history of trauma.[1]

Compression of the upper or lower nerve roots cause slightly differing neurogenic symptoms. The commonest general symptom is paraesthesia followed by neck pain, arm pain and lastly shoulder pain.[1]

Lower plexus compression is the commonest site of brachial plexus irritation as it rises above the first rib and is at risk of being tented. The nerve roots involved are C8 and T1 with resultant pain in the shoulder region that radiates down the posterior axilla, the medial side of the arm and elbow and then affects sensation in the ulnar distribution which is experienced as paraesthesia in the 4^{th} and 5^{th} digits.[4,6] Patients commonly report parasthesia in all 5 digits but with the 4^{th} and 5^{th} digits more severely affected.[1]

Motor involvement may include weakness in the rhomboids and scapular muscles proximally, in the wrist flexors and, most notably, in the intrinsic hand muscles.[5]

Upper plexus compression refers to symptoms elicited by involvement of the upper and middle trunks of the brachial plexus, namely C5 & C6 and C7 nerve root involvement respectively.[4,5]

Symptoms from this compression are experienced in the lateral neck radiating towards the ear and occiput. Patients may complain of occipital headaches, earache and pain in the face and mandible. Less commonly pain is experienced in the rhomboids

posteriorly, across the clavicle anteriorly to the pectoralis region, laterally through the deltoid and trapezius and then down the lateral and outer aspect of the arm including the arm extensors.[4,5]

Sensory changes, again usually in the form of paraesthesia, are felt in the thumb, index and middle fingers indicating median nerve involvement.

Rarely, dizziness, blurred vision and vertigo occur from upper plexus lesions.[6]

The autonomic nervous system is also at risk of involvement and vasomotor disturbances may be expressed.[2]

It is of interest to note that, although it is a more typical feature of arterial TOS, Raynauds phenomenon may occur in patients with neurogenic TOS.[15] The aetiology of Raynauds phenomenon in the two types of TOS is different. Sympathetic nerve fibres travel with C8/T1 nerve roots and compression or irritation of these fibres cause constriction of the arterioles with resultant cold hands and colour changes. This differs from arterial TOS where compression of the subclavian artery causes the phenomenon.[2]

Rarely, a Horner's Syndrome may be evident from compression of the stellate ganglion. A sinister cause always needs to be excluded before attributing this sign to TOS.[5]

The actual clinical examination in a patient suspected of suffering from neurogenic TOS starts with a basic neurological examination.

Inspection of the arm may reveal loss of the thenar eminence with intrinsic muscle wasting particularly on the ulnar side of the hand[5], loss of fine movements and uncommonly contracture formation of the fourth and fifth digits.

Certain clinical tests are suggestive of the syndrome although no one test is diagnostic or exclusive - positive tests indicate nerve compression in an area that may or may not be the thoracic outlet. The tests essentially aim to reproduce symptoms.[1]

Firstly palpation of the neck – the scalene muscles, trapezius and anterior chest wall – may reproduce similar pain as nerve roots are compressed.[5] A postive Tinel sign over the brachial plexus may be elicited and decreased sensation over the affected fingers may be experienced with very light touch.[1]

Basic tests that have been designed to elicit symptoms and aid in diagnosis include:

- The Upper Limb Tension Test of Elvey (ULTT) or modified ULTT creates tension on the brachial plexus reproducing symptoms.

  Firstly both arms are abducted to 90 degrees with the forearms flexed to 90 degrees. The elbows are then straightened. The wrists are then dorsiflexed and lastly the head is tilted to one side and then the other. The test may be performed passively or actively.

  The first three steps mimic symptoms on the ipsilateral side whilst the head tilt will recreate symptoms on the contralateral side.

  Symptoms experienced in the initial manoeuvres depict more severe disease.[2]

- Elevated Arm Stress Test (EAST) also known as The Ross stress test comprises externally rotating the arm and abducting the arm 90 degrees. For this to be confirmed positive, the symptoms should be reproduced in less than 60 seconds.[1,2]
- The Wright Test: the shoulder is hyperabducted to 180 degrees with the elbows flexed. Once again, reproduction of symptoms indicates a positive test result.[7]
- The Costoclavicular Compression test describes depression of the patients shoulder by the examiner and assessment of symptom recurrence.[5]
- Head Tilt – ear to shoulder head tilt will reproduce pain on the opposite side, should a neurogenic TOS be present.[2]
- The Adson test – The examiner holds the patient's arm down and palpates the radial pulse whilst the patient takes a deep breath in and, keeping the head extended, turns it to toward the symptomatic side. The pulse will decrease or disappear or the blood pressure will alter in a positive test. It is surprising to note that turning the head to the contralateral side will obliterate the pulse in a greater proportion of patients: 63% vs 22%. In addition 11-53% of the normal population will have a positive test and it is thus unreliable.[2,4]
- The Halstead or Costoclavicular Test – The military position is assumed with the shoulders backwards and downwards and disappearance of the radial pulse constitutes a positive test result.[7]

The EAST, considered the most accurate test, was found to be positive in 100% of patients tested with neurogenic TOS and the ULTT positive in 98% of patients. Change in sensation to light touch was positive in 68% whilst the remainder of the tests were positive in 90-94% of patients.[1,2]

Arterial TOS presents with slightly different clinical symptoms from neurogenic TOS and is due to arterial insufficiency.

When compression is longstanding a post-stenotic arterial aneurysm develops that may contain thrombus, and patients usually present with symptoms related to peripheral embolisation. Weakness of the forearm and hand, described by some patients, may be attributed to arterial ischaemia resulting from poor perfusion of these regions.[4]

Most patients will only experience symptoms once embolisation has occurred with the arterial stenosis and aneurysm being initially asymptomatic.[1]

Parasthesia, claudication, coldness and colour changes are all expressions of arterial TOS. Pain becomes a feature if the patient develops ischaemic neuritis of the brachial plexus. The parasthesia is also caused by ischaemia of the sensory nerves.[5] These features, although similar in presentation, should not be labelled Raynauds phenomenon as the aetiology is arterial ischaemia.[2]

Most symptoms arising from arterial TOS are in the hand and forearm with the neck and shoulder girdle relatively spared.

On inspection of such a patient, one may identify colour changes, ischaemic fingertips and even digital gangrene.[5]

Palpation of the supraclavicular area may reveal a tender lump, bony prominence or pulsation of an aneurysmal artery.[1]

An infraclavicular or supraclavicular bruit may be audible on auscultation.[7]

The radial pulse may be absent due to emboli in the antecubital space.[2]

Clinical tests to confirm arterial TOS are seldom necessary as the diagnosis is usually obvious. One may abduct the arm to prove obliteration of the ipsilateral pulse. Other tests performed for neurogenic TOS are likely to be negative due to the differing pathology.[2]

Venous TOS has classical symptoms of an obstructed vein with congestion – a painful, swollen and cyanotic arm occasionally with associated parasthesia. The parasthesia results from the hand oedema and not nerve compression in the neck. Uncommonly a patient may present with a pulmonary embolus.[5]

Clinical examination will reveal a swollen, discoloured arm with visible subcutaneous veins over the shoulder and chest wall.[1]

Radiographic diagnosis

Investigations for TOS are of more value in excluding other aetiologies of patients' symptoms rather than confirming a diagnosis.[7] They will however identify bony anatomical abnormalities and have an important role in planning surgery.

X-ray:

A chest x-ray (CXR) or thoracic inlet x-ray is performed in all patients with symptoms suggestive of TOS or in patients with cervical and shoulder pain.

Bony abnormalities, most commonly a complete cervical rib, are easily detected by this simple investigation.[4]

The diagnosis of TOS may be excluded by the presence of other radiological findings such as degenerative cervical spine disease.[4]

The value of x-rays is limited in that the majority of patients will have normal x-rays and even if a positive finding is seen, it may not be the cause of symptoms

– most patients with cervical ribs are asymptomatic[7] and degenerative cervical spine disease is apparent in older patients.

A normal x-ray will essentially exclude arterial TOS as there is almost always a radiologically identifiable bony abnormality in symptomatic patients. No further investigations are necessary for arterial TOS.[2]

Arteriography and Venography:

These studies will depict external compression of the vessel.

Arteriography is only indicated in arterial TOS for planning surgery and not for diagnosis, which is essentially clinical.[1] An abnormal x-ray will encourage one to undertake imaging of the artery. Some clinicians will perform duplex doppler ultrasound initially and only if a stenosis or aneurysm is visualised will an arteriogram be considered.[2]

With the use of arteriography, the compression can usually be detected in the neutral position and dynamic testing is not necessary.[2]

It is noteworthy that if arteriography is performed for neurogenic TOS it will reveal subclavian artery compression in dynamic positioning. This compression is not diagnostic of arterial or neurogenic TOS.[2] One must therefore not request this invasive study unnecessarily.

Venography should only be performed for suspected venous TOS. Occlusion of the subclavian vein in the neutral position is diagnostic. However, if the vein is patent the arm should be abducted to 90 degrees and 180 degrees and the venography repeated, looking for occlusion in these dynamic positions.[1]

CT Angiography

This test is relatively invasive, expensive and not routinely used.

It is nonetheless a useful investigation as it outlines the compression of neurovascular structures. It will also reveal the impinging anatomy.

The CT Angiography (CTA) in this instance is fairly unique in that it is first performed with the arms alongside the body and then repeated with the arms elevated reproducing the symptoms and possibly capturing the anatomical pathology.[4]

CTA boasts its best results with imaging for arterial compression. It will show a reduction in arterial diameter on comparison of the films, thus not only confirming the diagnosis but also locating the site of compression and identifying bony abnormalities.[4]

Venous compression, on the contrary, is often inaccurate and difficult to assess with CTA. If normal subjects undergo CTA with arm elevation, the subclavian vein will be compressed in all three compartments of the thoracic outlet.[4]

Thrombosis of the subclavian vein as well as the compensatory collateral circulation is readily identified with CTA. [4]

CTA falls short as an imaging modality for neurogenic TOS, which is the commonest presentation - it cannot portray a detailed analysis of the brachial plexus.[4]

With the arm in abduction, for dynamic imaging, the investigation is limited by the inadequate space in the CT machine, as the patient can only elevate the arm to less than 130 degrees.[4] The patient has to be supine for the investigation and this position has shown up to 32% of false negative results.[4]

Electrodiagnostic Studies

Nerve Conduction Studies

Nerve conduction tests are utilised by many institutions in aiding diagnosis and hence appropriate management of TOS. Electrodiagnostic studies include sensory nerve action potentials (SNAP), compound motor action potential (CMAP), nerve conduction velocity (NCV) and F-wave latency.[1]

The most useful tests in TOS are the nerve conduction velocity (NCV) and the F-wave latency test that detects compression in the region of the TOS.[1] Pressure or prolonged injury to a nerve will slow the conducting velocity within a nerve. Velocities < 85m/s are considered abnormal. Values between 60-85m/s are usually managed conservatively whilst those below 60 m/s will benefit from surgical intervention.[5]

Median nerve conduction velocity deterioration indicates upper plexus compression whereas ulnar conduction abnormalities reflect lower plexus involvement.[6]

NCV is not diagnostic and patients with TOS may have a normal nerve conduction study. [1]

The reason NCV as well as SNAP and CMAP may be normal in the presence of TOS is that the sampled area is distal to the lesion.[1]

F-wave studies, on the other hand, will detect abnormalities from the plexus proximally to the anterior horn cells. False negatives do occur as compressed nerves have a few functioning fibres which conduct normally and reflect a normal test result. F-wave will also not locate the area of compression and is non-specific as to the aetiology of the compression.[1]

Nerve conduction studies aid in differentiating between TOS, carpal tunnel syndrome and other causes of neck, shoulder and arm pain.[7]

Electromyography (EMG)

This test essentially shows denervation of nerves. It serves a larger role in patients with severe disease and is more sensitive in detecting motor nerve function.[1]

Direct cervical nerve root stimulation of C8

Results are similar to NCV, which is favoured, due to it being a less invasive test.[1]

Somatosensory evoked potentials

Although found useful by some clinicians, somatosensory evoked potentials are not specific, cannot locate the lesion and are not recommended.[1]

Electrodiagnostic testing is considered useful for excluding the diagnosis of TOS, although results may still be normal in mild disease. They are more beneficial for excluding: carpal tunnel syndrome, cubital tunnel syndrome, polyneuropathy, motor neuron disease and radiculopathy.[1]

Medial Antebrachial Cutaneous (MAC) Nerve Conduction

Recently, interest has been ignited in MAC as it may detect subtle changes in sensory nerve conduction in patients with normal NCV's and EMG's.
This test may hold promise for the future.[1]

Blocks

Scalene Muscle block

This test is a subjective procedure that involves the injection of local anaesthetic into the anterior scalene muscle. Relief of pain implies a possible TOS that will likely be successfully treated by surgery.[1]

1.8 DIFFERENTIAL DIAGNOSIS

Numerous conditions exist in the neck, shoulder and arm that may cause pain and need to be differentiated from TOS. Unfortunately no one test achieves this aim of differentiating.

It is equally important to bear in mind the well described "Double Crush Syndrome" described by Upton and McComas in 1973, hypothesising that a proximal compression of a nerve subjects it to compression more distally. The accumulative effects of minor compressions cause the painful symptoms.[6] More than two sites may be affected and the combined symptoms are appropriately called "Multiple Crush Syndrome".[6]

Typically ulnar nerve compression at the elbow or carpal tunnel syndrome co-exists with TOS. (All three equate to the "Multiple Crush Syndrome".) [5]

More recently TOS associated with cervical spine disease has been described.[5]

The differential diagnoses include: [5]

- Carpal Tunnel Syndrome
- Radial/Cuboid/Pronator tunnel syndrome
- DeQuervains tenosynovitis
- Lateral epicondylitis
- Medial epicondylitis
- Complex Regional Pain Syndrome
- Raynauds Disease
- Cervical disease (commonly cervical disc disease)
- Systemic disorders: Inflammatory disease, oesophageal pathology, cardiac disease
- Rotator cuff pathology
- Tendon tears

Pectoralis Minor Syndrome (PMS)

Although part of the thoracic outlet and known to cause symptoms, PMS has been described as its own entity. It is also known as the Hyperabduction Syndrome and is characterised by pain in the anterior chest wall, trapezius muscle and scapula region. Patients may concomitantly describe arm pain and hand parasthesia.[1]

Left sided PMS may mimic angina.[1]

Few articles have been published on PMS and although it veers away from the classical description, it is still essentially part of the Thoracic Outlet syndrome .[1]

1.9 TREATMENT OPTIONS

The management of TOS is, unsurprisingly, not well defined.

Generally, neurogenic TOS with mild to moderate symptoms should undergo a trial of conservative management. Severe symptoms may proceed directly to surgery. Severe symptoms are determined by muscle wasting, interruption of daily activities and sleep disturbance. Failed conservative management after a 3-month trial will also require surgery.[1]

Arterial TOS requires surgery as the first line of treatment as there is almost always an anatomical abnormality that requires correction.[5]

The majority of physicians treat venous TOS surgically but some may still attempt a conservative trial involving thrombolytic therapy, as a demonstrable anatomical abnormality is not usually appreciated.[5,7]

Conservative Treatment

This is predominantly advocated for neurogenic TOS.[1]

Physical therapy is the mainstay of conservative management and attempts to open up the thoracic outlet space. Treatment is directed at relaxation of the scalene muscles

and aims to increase the strength of the postural muscles. Patients are given relaxation and stretching home exercises (Feldenkrais method) and are counselled on behaviour modification.[1]

Massage, nerve glides and hydrotherapy are useful adjuncts.[1,4]

This current therapy focuses on gentle treatment that has evolved from more painful modalities. Certain physiotherapy treatments may aggravate symptoms of TOS and should be avoided. These include: resistance exercises, neck traction, therabands, heavy weights and strengthening exercises.[1]

Biofeedback mechanisms and trigger point injections have also been described as conservative treatment modalities.[1]

Medication including simple analgesics, non-steroidal anti-inflammatory drugs and muscle relaxants are given as part of the non-operative approach.[1]

It is thought that the majority of patients will improve without surgery and an adequate trial of conservative management should be given for at least 3 months.[1]

Operative Treatment

Definitive Surgery

The aim of surgery for all forms of TOS is to open up the thoracic outlet and relieve pressure on the affected structure.

For severe *neurogenic TOS* or a failed conservative trial of treatment for TOS operative intervention is necessary.[1]

The surgical procedure entails a routine anterior scalenectomy. Previously a rib resection would be done routinely but it is now undertaken more selectively, often only reserved for vascular TOS. A first rib resection will require resection of the anterior and middle scalene muscles.[1] Neurolysis is subsequently performed.[9]

Disabling neurogenic TOS may benefit from cervical sympathectomy[9] but this is certainly not routinely done.

The operative procedure may be done via a supraclavicular or transaxillary approach with most favouring the former. [9]

The choice of approach (transaxillary versus supraclavicular) and procedure (scalenectomy with or without first rib resection) have all been shown to be equally successful and the choice is based on individual preference.[1]

Arterial TOS, demonstrating stenosis or aneurysmal dilatation, should proceed directly to surgery for revascularisation and reversal of ischaemia. The principles of surgery include:

- subclavian artery decompression
- repair of arterial lesions (removing the source of the embolus)
- management of the ischaemic hand (restoring distal circulation)[9]

Typically these patients have an obvious anatomical abnormality, most commonly a cervical rib, causing the subclavian artery compression and it is this structure that needs to be resected to relieve the arterial compression.[5,9]

Removal of a cervical rib, the prominence on the first rib and an anterior scalenectomy is considered curative by many surgeons. Occasionally removal of the pectoralis minor tendon is necessary. The scalene muscle must be excised (not merely divided) as it may reattach and cause recurrent symptoms. First rib resection, once thought to be imperative in cure, is no longer considered necessary and not undertaken by most vascular surgeons.[9]

Most surgeons prefer the supraclavicular approach when operating for arterial TOS as a cervical rib is easily identified. Good exposure of the subclavian artery and surrounding structures is achieved. The axillary approach may be more useful for resection of the first rib but this is no longer routinely used. The first rib may be accessed through an infraclavicular incision which also allows exposure of the distal subclavian and axillary arteries.[9]

First rib excision must be undertaken as close to the T1 transverse process articulation as possible as a longer stump has been shown to have adverse outcomes.[10] Alternatively, the rib may be disarticulated.[7]

A combined supraclavicular and infraclavicular approach may prove useful when access is difficult. Claviculectomy will increase exposure but is avoided by most surgeons because of its morbidity.[11]

Repair of arterial lesions is necessary for aneurysms but not for simple post stenotic dilatation as the latter will return to normal following decompression of the artery.[6,9]

Aneurysmal development, with or without thrombus formation, requires resection and reconstruction.[6,9]

Thrombus and emboli should be treated with thrombectomy and embolectomy respectively and then followed by vessel reconstruction.[6]

Thromboendarterectomy is a lesser alternative to aneurysm excision and reconstruction.

Should the axillary or brachial arteries be occluded, embolectomy, while feasible, may prove to be difficult if it is chronic and direct exploration with a subsequent bypass of the vessels may be necessary.[9]

Cervical sympathectomy is used selectively for patients with autonomic disturbances of the arm, severe symptoms or failure to achieve arterial patency below the level of the elbow. Sympathectomy is a debatable adjunct that is typically performed through a supraclavicular approach.[6, 11]

Management *of venous TOS* is traditionally staged and depends on the chronicity of the thrombosis. A patient may present with intermittent obstruction with or without thrombosis, acute thrombosis or chronic thrombosis.[3]

The management of a patient with intermittent venous obstruction with vein stenosis without thrombosis is initial surgery with the purpose of correcting the underlying aeitiology followed by anticoagulation for 2 weeks. At 2 weeks the venogram should be repeated. Should there be evidence of a stenosis the patient should undergo a

venoplasty and anticoagulation should be continued for another 4 weeks. If no stenosis is detected then no further treatment is required. [3]

If the patient has intermittent venous obstruction without vein stenosis and thrombosis then surgery is the sole intervention required. [3]

In the patient with venous obstruction with acute thrombosis the management should be thrombolysis to address the acute thrombosis, surgery to correct the cause followed by 4 weeks of anticoagulation. [3]

If the presenting problem is Venous Obstruction with chronic thrombosis then surgery is indicated followed by anticoagulation for 6 months. [3]

The first stage of interventional treatment begins with catheter directed thrombolytic therapy for patients with thrombus formation addressing the acute symptoms. Maintenance treatment with heparin and warfarin follow.[3]

Operative intervention is performed approximately 3 months later. A scalenectomy, subclavius tendon release, brachial plexus neurolysis, rib resection of the first rib and venolysis need to be performed. It is essential to perform a scalenectomy and detach the subclavius tendon from the costosternal junction to prevent scar tissue causing recurrent vein occlusion. If the above treatment fails, one would need to undertake venous reconstruction. This is seldom necessary.[3, 4]

After surgery, the patient is anticoagulated.[3]

A study comparing early treatment – that is immediate surgery within 2 weeks of thrombolysis – to the standard staged approach shows results of early surgery to be excellent with the only drawback being a longer post-operative stay than the staged management. This delay was attributed to anticoagulation post-operatively and found to be acceptable.[3]

If a patient is known to have vein stenosis or suspected occlusion post-operatively, a venogram should be performed 2 weeks after surgery and a confirmed stenosis may be dilated followed by anticoagulation.[3]

Stenting has shown generally shown poor outcomes likely due to an inflammatory reaction by the vein and is not favoured.[3] They are being used successfully by some groups. However, this is only after decompression; they do not advocate the use of endovascular treatment prior to decompression.[9]

1.10 RESULTS / OUTCOME

Mortality from the management of the TOS is virtually non-existent. Post-operative complications are uncommon with the major morbidity being recurrence from a rib remnant.[6] Opening of the pleura facilitates drainage of fluid and reduces recurrence rates.[6] Bleeding and infection are additional complications. Major arterial injury is rare.[6] Pneumothorax has been described and opening of the pleura should be treated with intercostal drainage or pleural suture.[7]

Nerve and brachial plexus injuries have been described and are sometimes attributed to forceful retraction.[9] Phrenic, long thoracic and recurrent laryngeal nerve injuries have all been documented. Injury to the stellate ganglion is identified by Horner's Syndrome. [1,6]

Operative success rates vary between 82%-96% with venous TOS usually above 90%.[3,7] Accuracy of these figures may be questioned as documentation of follow-up has included patients reviewed for less than 12 months. This is of importance as 60% of recurrences occur within 12 months and 80% within 24 months.[1]

In terms of success rates for the operative approach used, results are similar.

Success is documented in terms of surgery[1] – this is applicable for vascular TOS, where surgical treatment of a causative anomaly is usually curative, therefore demonstrating high success rates but neurogenic TOS is incompletely understood with a variety of treatment options and surgical success rates range widely. Some authors believe the

majority of patients with neurogenic TOS will improve with proper conservative treatment.[1,12,13]

Surgery for neurogenic TOS is considered successful with good outcomes varying between 45%-86%.[1]

Worse outcomes are seen in patients with a combination of a cervical rib and fibrous bands, females and a delay between symptoms and surgery.[9]

The undertaking of this study was based on the observations of a single surgeon operating on patients with neurogenic thoracic outlet syndrome over a ten-year period. Most of the epidemiology and investigations mirrored the literature. However it was noted that, with careful inspection, almost all patients had some form of anatomical abnormality. The anomaly was typically with respect to the brachial plexus but abnormalities of the soft tissue and bony structures were also noted.
Based on these findings, a more detailed record of surgical findings was deemed necessary in order to properly identify and classify these anatomical abnormalities.
This was done for the last 63 patients (67 procedures) and a retrospective review of a prospectively maintained database was performed which is presented in this research.

2. AIMS

Type of Study: Observational

a. To categorise the neurological presentation of Thoracic Outlet Syndrome and correlate these features with intra-operative findings.

b. Assess the outcome of surgery for neurogenic Thoracic Outlet Syndrome.

c. To attempt to clearly define anatomical anomalies causing Thoracic Outlet Syndrome.

3. HYPOTHESIS

The aeitiology of neurogenic Thoracic Outlet Syndrome is multifactorial with bony tissue abnormalities and soft tissue abnormalities described as definite contributors to the syndrome. These abnormalities contribute to the syndrome by altering the space within which the brachial plexus trunks run. Brachial plexus anomalies, however, have not classically been described as a direct association with the syndrome. Recent interest in brachial plexus anomalies suggest them as a cause for various pathologies including Thoracic Outlet Syndrome.[14]

We hypothesise that brachial plexus anomalies – alone or in conjunction with additional pathology – are almost always associated with the neurogenic Thoracic Outlet Syndrome.

4. METHODS

Summary of the proposed research

The object of the study is to evaluate Thoracic Outlet Syndrome in patients who present with neurological manifestations of the condition and to correlate these with the intra-operative findings.

A retrospective study was performed over a 10-year period (1999-2009) and it was noted from these observations that a detailed inspection of these patients' anatomy needed to be undertaken and over the following four years the surgeon meticulously recorded the anatomical findings. Thus, the records from a prospectively maintained computer database of all patients in the last four (2005-2009) years with this pathology, presenting to a single vascular surgeon, was reviewed.

In addition the outcomes of surgery for the syndrome will be assessed and a possible classification of the syndrome suggested.

Keywords

Thoracic outlet syndrome, neuropathy, cervical rib

5. DESIGN OF INVESTIGATION

A prospectively maintained computer database from a single vascular surgical practice was used for the review.

From the computer database, the records of all patients with Thoracic Outlet Syndrome were scrutinized and information regarding their pre-operative symptoms, investigations, operative surgery and clinical outcomes documented.

The neurological presentation of pain, paraesthesia, weakness, wasting of the hand muscles and the segmental distribution in conjunction with the clinical investigations (including the Elevated Arm Stress Test (EAST)), investigations (a chest radiograph, a MRI and nerve conduction studies) and the pathology (cervical rib, fused rib) have been correlated with the surgical procedures of neurolysis with cervical or first rib resection.

All data were captured into an excel spreadsheet and subjected to multivariate analysis.

A total of 219 patients were analysed with regard to epidemiology, surgical intervention and outcome. It became apparent that there appeared to be a high incidence of brachial plexus anomalies. Over the last 4 years a more detailed assessment and evaluation of the anomalies was made, and that subset of patients is reported separately.

5.1 Statistical Analysis

The study design is a retrospective review from a prospectively maintained computer database of patients presenting with Thoracic Outlet Syndrome over a ten-year period and statistical analysis is not applicable here.

5.2 Source

The source of the information for the observational study was patient records.

Inclusion Criteria:
All patients diagnosed with Thoracic outlet syndrome that underwent a surgical procedure as part of the treatment management.
Exclusion criteria:
Patients with TOS that did not undergo operative intervention.

The patient population was 219 patients in total.

5.3 Limitations of the study

This is a retrospective study and therefore subject to limitations of incomplete data in some instance, inadequate follow up and occasional inaccurate documentation. There is no information on patients who were not submitted to surgery. It is possible that patients who did not improve after the surgical procedure may have an alternative diagnosis to TOS.

5.4 Ethical considerations

As this is a retrospective study with details taken from patient records, no further clinical contact is necessary. Confidentiality will be maintained.
Ethics Approval has been obtained from the Biomedical Research Ethics Committee.

6. RESULTS

6.1 Epidemiology

There were a total of 219 patients with the 160 (73%) being female. The ages ranged from 5 to 63 years with the average age being 31 years.

6.2 Presenting complaint

All but one patient had pain or parasthesia as the presenting complaint, with pain being the dominant symptom.

Seventeen percent of patients experienced weakness and 8% had muscle wasting. Less common symptoms included: headache, swelling on exertion, colour change and temperature change. These are recorded in table 1.

Table 1: Presenting Symptoms

SYMPTOM	Total Patients n=219	Total Patients %
PAIN & PARASTHESIA	217	99.5%
WEAKNESS	38	17%
HEADACHE	4	2%
SWELLING ON EXERTION	2	1%
COLOUR & TEMPERATURE CHANGE	1	0.5%
MUSCLE WASTING	17	8%

Percentages to the nearest whole number.

6.3 Distribution

In terms of neurogenic TOS, the perceived nerve roots affected on presentation were as follows (n=225). (This includes 6 patients that had bilateral symptoms).
Table 2 demonstrates the neurological segmental distribution of symptoms.

Table 2: Segmental Distribution of Symptoms

Segmental Distribution	N=225 Total number of patients: 219
C5-C7	29 (13%)
C8-T1	162 (72%)
C5-T1	34 (15%)

Percentages to the nearest whole number. Six bilateral symptoms recorded.

6.4 Investigations

Results of the EAST (Elevated Arm Stress Test) were performed on all patients on the affected side. One hundred and sixty-five patients (73%) had positive findings.

In terms of investigations, all patients had a thoracic inlet or chest radiograph. This was found to be abnormal in 77 (35%) of patients. The abnormalities included: cervical ribs, high insertion of the first rib and a residual stump of the first rib. The latter patient had undergone a previous rib resection. Mild spondolysis was noted on some of the imaging but was not thought to contribute to the presenting symptoms.

Nerve Conduction Studies (NCS) were not routinely performed. Fifty-five patients had abnormal NCS, but these results were very difficult to interpret and the results did not contribute to management.

Magnetic Resonance Imaging (MRI) was performed in 45 (21%) of patients by referring doctors. The majority were normal and the few that reported an abnormality were all minor and not considered as the primary pathology causing symptoms. These included mild spondylitic changes, encroachment and minor disc herniation.

6.5 Surgical Approach

The standard surgical approach is an incision 1 cm above clavicle, extending from the sternoclavicular joint approximately 8-10 cm laterally. The platysma muscle and the superficial cervical fascia are incised and the external jugular vein ligated. The clavicular head of sternocleidomastoid muscle is divided and the middle cervical fascia is incised. The scalene fat pad is identified anterior to scalenus anterior and reflected superiorly. The phrenic nerve must be identified anterior to scalenus anterior, running form lateral to medial on the muscle. The scalenus anterior muscle is divided – a scalenectomy should be performed and not merely a scalenotomy as the muscle may reattach, once again, narrowing the interscalene space. The subclavian artery must be routinely identified and all trunks of the brachial plexus dissected out and isolated. In

order to perform a first rib resection, scalenus medius usually requires division and the resection is performed from the neck of the rib posteriorly down to and including the scalene tubercle.
Neurolysis is performed of the nerve trunks and one can assess whether the space has been adequately opened up and the nerves are lying free within the space at the end of the procedure.

All patients in this series had a supraclavicular approach. Of these, 14 had an additional infraclavicular incision and 8 had an additional transaxillary incision. The reason for these combined procedures was the need for increased surgical access in obese patients for the first rib resection. The supraclavicular approach was chosen routinely, so as to clearly define the pathology.

6.6 Complications

Twenty-five patients (11%) had complications. These included an empyema, severe pain (which required re-operation and sternocleidomastoid repair), haemothorax, pneumothoraces, neuropraxias and parasthesias. See table 3 below for the complications.

The patient who complicated with the haemothorax eventually required lung decortication.

The neuropraxias and neurological complications all resolved within 3 months.

Table 3: Post Operative Complications

Complications	Total Number of Operations n=225	Total Number of Operations %
Empyema	1	0.004%
Haemothorax	1	0.004%
Pneumothorax	3	0.01%
Pain	1	0.004%
Phrenic neuropraxia	3	0.01%
Parasthesia/weakness	16	7%
TOTAL	**25**	**11%**

Percentages to the nearest whole number.

6.7 Follow up:

Recorded follow-up ranged from 2 weeks to 15 years.

6.8 Outcomes: (n=219)

Fourteen (6%) patients did not have resolution of the presenting symptoms and one had recurrence of symptoms one year later. See table 4 for the initial outcomes of the operations.

Seven (3%) patients underwent re-operation and of these 6 improved. Overall, 9 (4%) patients were displeased with their outcome. The actual reasons for these unsatisfactory outcomes remains undefined despite further investigations. Possibly these patients were not well selected.

Generally, patients with residual symptoms seemed to present late with chronic symptoms that had often already also led to muscle wasting by the time of intervention. This delayed intervention may also be the reason for persistence of symptoms.

Table 4: Initial Outcomes of Operations

OUTCOMES	**Total Patients n=219**	**Total Patients %**
RESOLUTION	204	93%
RESIDUAL SYMPTOMS	14	6%
RECURRENCE	1	1%

Percentages to the nearest whole number.

7. OPERATIVE DETAILS OF 63 PATIENTS OVER 4 YEARS

7.1 Background and Investigations

Over the initial six-year period of surgery, it became apparent to the primary surgeon that an anatomical abnormality was almost always present, and thus the surgeon began to carefully record the intra-operative findings over the last four years. This included the last 63 patients of this series. Sixty-seven operations were performed in 63 patients. (There were 4 patients with bilateral neurogenic TOS).

Background of these 63 patients:

A total of 17 patients (27%) had previous trauma to the neck.
Eleven patients had undergone previous surgery to the neck or shoulder region – mostly anterior spinal fusions. Six patients (10%) experienced previous trauma. See table 5 below.

The trauma included a shoulder dislocation, motor vehicle accidents (with presumed whiplash) and clavicle fractures.

Table 5: Predisposing Injury

Previous Injurious Event	**Total Patients n = 63**	**Total Patients %**
Previous surgery (cervical spine/shoulder)	11	17%
Previous trauma	6	10%
TOTAL	17	27%

Percentages to the nearest whole number.

All patients had chest radiographs performed of which 14 (22%) were abnormal. See table 6.

Table 6: Abnormalities seen on chest xray (CXR)

CXR Abnormalities	**Total Patients n=63**	**Total Patients %**
Cervical ribs - complete - incomplete - bilateral	**11** - 4 - 1 - 6	**17%** - **6%** - **2%** - **9%**
High insertion of first rib	**1**	**2%**
Residual stump of first rib	**1**	**2%**
Mild spondolysis	**1**	**2%**

Percentages to the nearest whole number.

7.2 Surgical Approach (n=67)

Sixty-seven operations were performed on the 63 patients; 4 patients had bilateral disease that was treated with staged operations. The operative procedure is as described earlier. All patients underwent anterior scalenectomy.

7.3 Surgical Operations (n=67)

Fourty-nine patients (73%) had first rib resections and 14 (21%) had cervical rib resections. One patient had a combined cervical and first rib excision (2%). This was done as the thoracic outlet space still seemed confined after the cervical rib was excised. Three patients (4%) had division of fibrous bands alone, without rib resection. This was performed in isolation as the thoracic outlet space appeared to be sufficiently opened up intra-operatively. See table 7 for details of the surgical procedure.

Table 7: Details of Surgical Procedure

PROCEDURE	NUMBER
1st Rib resection	**49 (73%)**
Cervical rib resection	**14 (21%)**
Division of bands only	**3 (4%)**
Combined cervical & rib resection	**1 (2%)**

Percentages to the nearest whole number.

7.4 Pathological Findings at Surgery

Operative findings at surgery that were considered to be contributory to symptomatology are described in table 9. These include bony abnormalities, abnormal muscle configurations and abnormal brachial plexus anomalies. The majority of patients had combinations of these abnormal findings.

Table 9: Anatomical Abnormalities Identified at Surgery

ANATOMICAL ABNORMALITY	**n = 67 (%)**
BONY ABNORMALITY	**17 (25)**
Cervical Rib	15 (22)
Fused 1st & 2nd rib	2 (3)
SOFT TISSUE ABNORMALITY	**39 (58)**
Bands	10 (15)
Abnormal muscle mass	29 (43)
BRACHIAL PLEXUS ABNORMALITY	**66 (99)**
Post-fixed brachial plexus	41 (61)
Pre-fixed brachial plexus	14 (21)
Other brachial plexus abnormalities	11 (17)
OTHER ABNORMALITIES	**2 (3)**
Subclavian artery compressed lower trunk	1 (1.5)
Fibrosis alone	1 (1.5)
GRAND TOTAL	**124**

Percentages to the nearest whole number.

Seventeen patients had bony abnormalities of which 15 were cervical ribs (complete or incomplete). Two patients had fused first and second ribs, which formed a bony mass and confined the thoracic outlet space.

Over half of the patients were found to have a soft tissue abnormality (58%). These included bands in 10 patients, which are considered along the spectrum of vestigial cervical ribs extending from C7 to the scalene tubercle. Often incomplete cervical ribs have a band completing their insertion into the scalene tubercle.

Of the 67 procedures, an abnormal muscle mass was identified in 29 (43%). This included hypertrophy of the scalenus muscle complex, amalgamation of the scalene muscles and interdigitation of the nerve roots through the muscle. This was found in addition to the other anatomic abnormalities.

Brachial Plexus Anomalies

The normal configuration of the brachial plexus can be seen in figure 6 below with the nerve roots beginning at C5 and ending at T1.

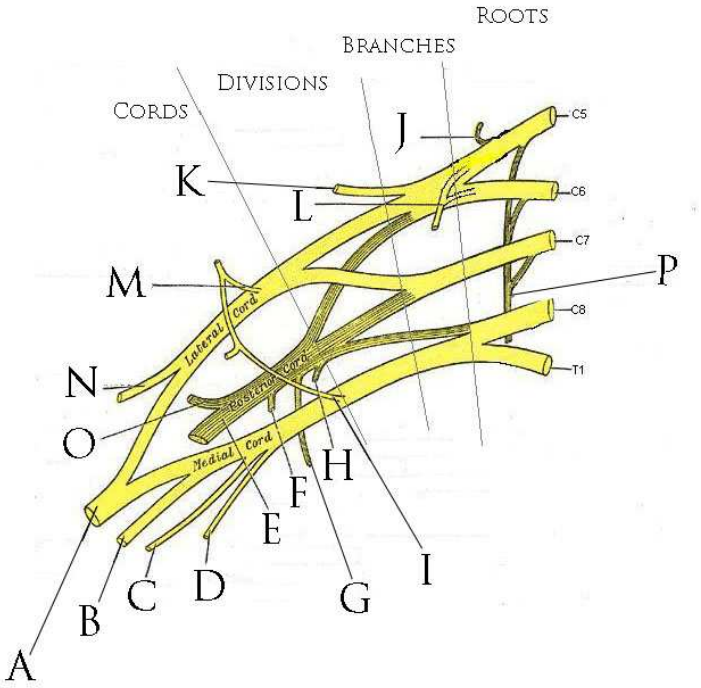


Figure 6: The brachial plexus

Website origin: http://commons.wikimedia.org/wiki/File%3AThe_Brachial_Plexus.jpg.

By Brachial_plexus.jpg:Mattopaedia at en.wikipedia. derivative work: Rafael Di Marco Barros (Brachial_plexus.jpg) [Public domain], via Wikimedia Commons from Wikimedia Commons.

Anterior view of right brachial plexus. Illustration. Modified by Mattopaedia on 02-Jan-2006 from the 1918 Edition of Gray's Anatomy. Original unmodified image sourced from http://www.bartleby.com/107.

2010-03-13 19:15 (UTC). Brachial plexus.jpg: Original uploader was Mattopaedia at en.wikipedia. Derivative work: Rafael Di Marco Barros.

The commonest variants are thought to be a pre-fixed or post-fixed brachial plexus. Although definitions may vary, a pre-fixed brachial plexus is generally thought to exist when the brachial plexus roots emerge a vertebral space higher than usual, that is, the

C4 ventral ramus forms part of the upper trunk and the root contributions to the plexus end at C8 (rather than at T1). A post-fixed brachial plexus is thought to exist when the ventral ramus of T2 contributes to the brachial plexus from below and the entire brachial plexus forms from one vertebral space lower than usual – that is from C6 – T2.

For purposes of intra-operative labelling, a pre-fixed brachial plexus refers to an enlarged upper trunk that is fused with the middle trunk. A post-fixed brachial plexus refers to an enlarged lower trunk that is fused with the middle trunk. In the dissection, the origin of the roots cannot be seen and it is assumed that these surgical descriptions of pre-fixed and post-fixed brachial plexus may have higher or lower origins respectively compared to the normal brachial configuration.

Interestingly, all but one of the patients was considered to have some form of brachial plexus anomaly.

ANOMALIES:

1. The majority of these were 'post-fixed' brachial plexus (62%).

2. The 'pre-fixed' brachial plexus anomalies followed at a distance (21%).

3. Other anomalies included abnormal position of the brachial plexus within or around the scalene muscles, abnormal configurations of the roots, trunks or branches and attenuated roots not fitting the description of a pre- or post- fixed brachial plexus. These additional anomalies are listed below:

- Nerve roots arising anterior to scalenus anterior muscle.
- Nerves interdigitating within scalenus anterior muscle.
- Nerves interdigitating with scalenus medius muscle.
- Middle & lower nerve roots amalgamated in one mass.
- Upper nerve roots coursing anterior to scalenus anterior muscle & giving rise to the phrenic nerve.
- Upper root emerging from scalenus anterior muscle.
- All nerve roots fusing at the clavicle.
- T1 nerve root running separately with a distal insertion into the lower trunk of the brachial plexus.
- Only 2 nerve trunks present.
- Nerve trunks appearing ribbon-like and attenuated.
- C6, 7, 8, T1 comprising a single nerve trunk.
- C8, T1, T2 comprising a single nerve trunk.
- C5, C6 each taking a long course and joining at the clavicle.

There was one unusual anomaly in that the subclavian artery was compressing the nerve trunk. One patient was found to have fibrosis alone and no other anatomical abnormality – the fibrosis caused confinement of the thoracic outlet space.

8. DISCUSSION

The focus of this review was on neurogenic Thoracic Outlet Syndrome.

The majority of patients were female with an average age of 31 years, which mirrors the epidemiology in the literature. Neurogenic Thoracic Outlet Syndrome is reported to occur four times more commonly in females, typically presenting between the ages of 20-40 years.[5]

Pain and paraesthesia are the main presenting symptoms of neurogenic TOS. Weakness and wasting is less common, occurring with more prolonged symptoms and seems to be associated with a poorer outcome. Again, this is in keeping with the epidemiology reported in the literature.[1] Trophic changes imply organic nerve damage, which takes some time to regenerate. These trophic changes may also predispose to chronic regional pain syndrome (CRPS) type II. [9,15] A significant proportion of patients had either previous cervical spine or shoulder surgery or trauma (including whiplash, clavicular fractures and shoulder dislocation) that may have been contributing factors to their pathology.

The predominant neurogenic distribution was C8/T1, which is in keeping with the literature as this lower nerve trunk is the one susceptible to being stretched over the first rib. [4,5,15]

All patients were examined clinically. Muscle wasting was uncommon but indicated a prolonged course of neurogenic involvement and possibly worse outcomes. A very common clinical finding in this cohort of patients was reproducible symptoms of pain or parasthesia on palpation of the supraclavicular fossa.

The Elevated Arm Stress Test (EAST), a simple clinical test, was positive in the majority of patients with a confirmed TOS but was not 100% predictive in this series although 100% accuracy has been reported in other studies.[2]

No clinical findings are confirmatory or exclusive of TOS but only suggestive.

Similarly, no one investigation confirms or excludes the diagnosis of TOS. All patients were investigated with a chest or thoracic inlet radiograph in order to detect any bony abnormality or other causes of symptoms. Even if present, however, a bony

abnormality may not be the sole contributing factor to the patients' symptoms. Chest or thoracic inlet radiographs will detect cervical ribs, anomalous 1st ribs, and long transverse processes of C7.[4,9] In conjunction with typical symptoms these findings should be addressed surgically. Chest or thoracic inlet radiographs may also highlight cervical pathology such as spondylosis. These findings need to be correlated with the clinical picture and if the findings support each other, further investigation or a trial of conservative treatment for the neck pathology is prudent prior to assuming the patient has a thoracic outlet syndrome.

MRI was usually performed prior to referral in patients suspected of having symptomatic cervical pathology. Abnormal MRI scans were assessed as to the degree of spinal pathology and the likelihood of symptoms being attributed to cervical pathology, as many silent changes do occur with age. The usefulness of MR imaging is controversial with some believing it is an 'unnecessary expense'[1], whilst some advocate its usefulness for soft tissue imaging. The brachial plexus and scalene muscles can be examined accurately with good MR imaging (especially T1 weighted imaging on the saggital plane). However, positional MR sequences need to be performed - with the arm initially by the patient's side and subsequently with the arm elevated.[4]

Insignificant radiological changes and poor clinical correlation in terms of cervical pathology led the primary surgeon to diagnose TOS in these patients. These diagnoses were seemingly accurate as the outcomes were successful. Thus the role of MRI was to exclude major cervical pathology, but once again, not confirming nor excluding neurogenic TOS.

Nerve Conduction Studies (NCS) may suggest a thoracic outlet syndrome. However, it is not considered a diagnostic investigation.[1] In this series of patients, those who had clinical features of TOS with a negative NCS still responded positively to surgical intervention. Thus, it is essentially an unhelpful and unnecessary investigation for diagnosing or excluding this pathology.

Electromyography is thought to be accurate in patients with severe disease or with motor neurological involvement as it measures malfunction of nerves.[1] However, most patients present before muscle wasting occurs and the majority of patients have sensory nerve involvement.

In terms of treatment of these patients, surgery was the mainstay of management as patients had been referred through various disciplines and conservative trials had already failed by this stage. Patients with TOS should be given a 3 month trial of conservative management before surgical intervention, unless the condition interferes with their daily quality of life (working or sleeping)[1] or they present with advanced signs (eg muscle wasting).

All operations were performed using a supraclavicular approach for the reasons described above.

All patients underwent anterior scalenectomy. If a cervical rib was present, it was resected. A 1st rib resection was performed in the majority of the patients - only 3 patients had division of fibrous bands in islolation. Routine first rib resection is controversial. The aim of surgery is to open the confined thoracic space by altering the borders of the space and to perform neurolysis, creating sufficient space that allows the nerves to lie freely. Performing an anterior scalenectomy addresses one border routinely. If the scalenectomy is adequate, the 1st rib usually does not further compress the neurological structures.[9] If a cervical rib is present it should be excised and a decision to remove the 1st rib should be made intra-operatively once the nerve roots have been assessed within the outlet space. If there is no cervical rib, it is likely a 1st rib resection will be necessary but if dense fibrosis is the cause of the symptoms this may not be consistently required – and, again, the decision should be made intra-operatively. First rib resection will, however, ensure that the thoracic outlet space is adequately opened and if fibrosis or muscle hypertrophy should occur in the future there is still sufficient space for nerve root mobility and possibly the prevention of recurrent symptoms.

All patients had neurolysis of the nerve roots performed and all nerve roots and trunks were individually encircled to clearly assess for any abnormalities and to ensure the nerves all lay freely in the thoracic outlet space after completion of the procedure. Each nerve root from C5 to T1 needs to be adequately dissected out and mobilised to avoid persistent symptoms.[9]

No cervical sympathectomies were performed – this is a selective procedure reserved for patients with autonomic disturbances.[9]

The anatomical findings were scrutinized at surgery and varying degrees of bony, soft tissue and brachial plexus abnormalities were found in all but one patient. The patient who had no abnormality detected had fibrosis in the interscalene space which seemed to be restricting nerve mobility. Scar tissue is known to cause compression and neurogenic irritation.[9]

The number of abnormal findings emphasises the importance of the supraclavicular approach because a transaxillary incision, while a feasible therapeutic option, will not detect these abnormalities. [9]

The findings at surgery included bony abnormalities, abnormal muscle configurations and brachial plexus anomalies. The majority of patients had combinations of these abnormal findings. It is thought that the outlet space that becomes an 'entrapment space' may be from congenital or acquired causes. Most likely it is rather a combination of both factors – slight or grossly abnormal anatomy coupled with hypertrophied muscles or a fibrotic process that encases nerve structures with resultant pain.[1,4]

Bony abnormalities have been well described as a cause or contributing factor in neurogenic TOS.[1,4] Cervical and fused 1st and 2nd ribs were both identified in this study.

Soft tissue abnormalities include scalene muscle abnormalities and fibrous bands.

Such soft tissue abnormalities have been documented in the literature, as well as varying space width between the anterior and medius scalene muscles.[1,4]

Muscle abnormalities were found of the scalenus anterior, scalenus medius and sternocleidomastoid muscles. (Listed in table 9)

Brachial plexus anomalies are not conclusively considered to be a contributing factor to neurogenic TOS. A recent paper, however, written by Megan Pellarin et al suggested that brachial plexus abnormalities are extremely common and that this anatomy may be a risk factor for certain pathologies, including TOS.[14]

The commonest abnormality was a post-fixed brachial plexus, implying a large conjoined middle and inferior trunk, susceptible to pathological stretching over the first rib or an anomalous cervical rib.

Pre-fixed brachial plexus anomalies were also fairly common in patients presenting with symptoms attributable to the higher nerve roots, or in patients with unclear symptoms.

The remainder of brachial plexus anomalies described include different combinations of nerve root formation that differ from pre- and post-fixed brachial plexus configurations.

Other brachial plexus abnormalities were of a more minor nature and were related to the abnormal position of nerve roots (eg. nerve roots arising anterior to anterior scalenus muscle) or to the course of nerve roots (eg. Nerve roots interdigitating within anterior scalenus muscle or scalenus medius muscle). The brachial plexus traversing the anterior scalenus muscle has been described which was also noted in this study.[4]

Interestingly, in one patient the subclavian artery was found to be compressing the lower nerve trunk and, as mentioned, a single patient had fibrosis alone as the apparent cause of all the symptomatology. Of note, this particular patient had no documented history of prior trauma or surgical intervention.

Acquired abnormalities contributed to a large number (27%) of patients who had either undergone previous neck or shoulder surgery or had trauma to the neck or shoulder region with resultant fibrosis, abnormal bone healing and muscle spasm. In addition, over usage caused hypertrophy of muscles in the interscalene space in a number of patients, confining the thoracic outlet space.

These acquired abnormalities are the typical causes of TOS – post traumatic scarring, post operative scarring and repetitive trauma.[1,4] It is thought, however, that a predisposing factor is necessary for neck trauma to result in TOS. The predisposing factors described include bony and muscle abnormalities both of which were noted in this study.[1]

Brachial plexus abnormalities fall under congenital anomalies, bony abnormalities may be congenital or acquired and there is a rather indistinct line between congenital and acquired soft tissue abnormalities.

The majority of patients, even those with bony abnormalities, had more than one abnormal anatomical structure. Only two patients with cervical ribs had no other abnormalities seen at surgery.

The patient who had no anatomical anomaly did have fibrosis, which was an explanation for the symptoms. This patient also responded well to surgery.

In almost all patients the classically described brachial plexus was not identified and this may well be a major contributing factor to neurogenic TOS.[14]

In keeping with the current literature there were no mortalities and complications were uncommon. Temporary neuropaxia's were the commonest complication. [6,9]

Success rates with neurogenic TOS vary between 45-96% depending on quoted series.[1,7]

Success rates were excellent in this review with 96% of patients experiencing complete or partial resolution of symptoms. This emphasises the importance of the supraclavicular approach allowing adequate neurolysis and clear identification of the underlying problem.

However, follow up averaged 6.8 weeks and as 60% of recurrences occur within a year and 80% within 2 years, this figure may not be entirely accurate.[1]

The patients who did not experience symptom resolution generally had chronic symptoms with evidence of muscle wasting. This was particularly evident in the records of the patients over the last four years. Delay in diagnosis and treatment is a known predictor of poor outcome and this may explain the results in these patients.[9]

9. RECOMMENDATIONS AND SUMMARY

As it is likely there is a combination of congenital and acquired pathology, it is best to classify patients according to the abnormality seen: bony abnormality, soft tissue abnormality and/or brachial plexus abnormality.

It is important to consider the risk the patient has for the development of a confined thoracic outlet space.

As no one test is confirmatory, a careful history of the patients' symptoms should be elicited.

Should these be suggestive of TOS simple ***clinical examination*** should include *supraclavicular palpation* (for a cervical rib as well as tenderness and symptom reproduction) and the elevated arm stress test (as although not 100% sensitive in this series, is the most accurate test available, is quick to perform and carries no cost).

A ***risk assessment*** should be performed, as these are all contributory factors.

Risk assessment notes previous neck or shoulder surgery or trauma as well as occupation and sport. The latter may be responsible for hypertrophy of the thoracic outlet muscles or muscle strain with subsequent fibrosis in this space.[1,4]

All patients should have a ***chest or thoracic inlet radiograph*** to look for a bony abnormality or cervical spine pathology.

Table 10: Making the diagnosis of Neurogenic Thoracic Outlet Syndrome

	PRESENT
CLINICAL EXAMINATION	
Supraclavicular tenderness	
EAST	
RISK ASSESSMENT	
Previous neck/shoulder surgery	
Previous trauma to neck/shoulder	
Occupation (using neck/shoulder muscles)	
Sport	
X-RAY (Chest/Thoracic inlet)	
Bony abnormality	
- Cervical rib: complete	
- Cervical rib: Partial	
- Fused 1st & 2nd ribs	
- Abnormal 1st rib	
Cervical spine pathology	

Should a bony abnormality be present, operative intervention is indicated.

If cervical changes are present and thought to be the cause of symptoms, a trial of conservative management may be attempted: physiotherapy, analgesia and bed rest. Should this fail an MRI should be performed to exclude any major pathology requiring orthopaedic or neurosurgical intervention. Should changes not be attributed to the spine, surgical intervention for TOS is feasible.

If the x-rays are normal the combination of presenting symptoms, EAST test and risk assessment needs to be considered. It would be wise to give these patients a trial of

conservative management. Should this fail, surgery should be undertaken and specific abnormalities looked for in terms of soft tissue and brachial plexus anomalies.

All patients should undergo a supraclavicular incision in preference to a transaxillary incision as this will allow one to clearly identify any anatomical abnormality from the nerve roots, muscles and tissue within the thoracic outlet space and it will allow one to accurately assess if the space has been adequately opened.

Abnormalities need to be carefully sought and recorded so that an accurate conclusion may be made from their contribution to the symptoms.

The space is bordered by the first rib, anterior scalenus muscle and scalenus medius muscle; thus, to open it up, the space needs to be altered. An anterior scalenectomy must be performed – and not merely a scalenotomy, as this may reattach and symptoms will recur.

Partial first rib excision will open up the space in the absence of any bony abnormalities, as it is one of the confining borders. It may not be routinely necessary as scalenectomy opens one of the borders of the interscalene space. The decision to remove the first rib should probably be made intra-operatively as one can assess whether the interscalene space has been adequately opened up by a scalenectomy alone.

If a cervical rib is present it should be primarily resected and the decision to subsequently remove the first rib should be based on the adequacy of the altered anatomical space.

Neurolysis should be performed routinely and as the symptoms are neurogenic, one should ensure they are lying free in a widened thoracic outlet space.

Results are generally excellent, the drawback being delay in diagnosis and operative intervention.[9] Therefore one should use the clinical examination, risk assessment and x-ray as a guide to diagnosis. Conservative treatment should probably not be prolonged more than 3 months before confirming the diagnosis and establishing a definitive plan.[1]

Neurogenic Thoracic Outlet Syndrome is a condition of neuropathic symptoms caused by congenital and acquired anatomical abnormalities (usually a combination) of the bony structures, soft tissue structures or brachial plexus that result in confinement of the nervous structures within the thoracic outlet space.

The diagnosis is difficult, being made predominantly on clinical examination, risk assessment and by exclusion. The only investigation of value is a CXR. The condition is surgically correctable by opening up the thoracic outlet space and if this is done timeously, before irreversible damage, outcomes are excellent.

10. CONCLUSIONS

We can conclude from this cohort of patients that there is always an underlying cause for the patients' symptoms that is usually due to an anatomical abnormality. The majority of these anomalies are abnormalities of the brachial plexus. Thus, all patients with neurogenic TOS should be considered as 'True neurogenic TOS' as there is some abnormality present, even if not bony.

There is often more than one abnormality, even if subtle, making the likelihood of the pathology multifactorial – for example, symptoms may develop in a person with an abnormal brachial plexus configuration which may be compressed later in life by muscle spasm or fibrosis after intense sport or a neck injury.

In addition to this, the abnormality may only be detected by careful inspection of the anatomy at surgery. A supraclavicular approach is preferred as a transaxillary approach precludes clear depiction of the neurovascular structures.

Resection of the first rib is still controversial, and the aim should be to adequately open the thoracic outlet space ensuring that the nerve trunks can lie freely within the space.

To clarify these findings, a further study is necessary recording detailed mapping of the brachial plexus findings intra-operatively. This then needs to be correlated with the specific presenting symptoms. Thirty-nine specific brachial plexus anomalies have been described in anatomy texts, emphasising that these anomalies may not be rare.

The association between brachial plexus anomalies and neurogenic TOS symptoms can never be proven, as it is impossible to conduct a trial with a control group. However, the connection may be assumed if an anomaly is detected at surgery and the patient improves symptomatically thereafter.

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