Review Article

Thoracic Outlet Syndromes**Russell W. Hardy, Jr.**

**Department of Neurological Surgery
Case Western Reserve University School of Medicine
Cleveland, Ohio, USA**

ABSTRACT

Thoracic outlet syndrome is a complex and controversial entity. We describe one form, the "true" thoracic outlet syndrome, which results from compression of the lower brachial plexus by a medial scalene compressive band. It occurs in patients with a cervical rib or prominent C7 transverse process; this syndrome is characterized by sensory symptoms and muscle atrophy in the affected limb, and may be diagnosed electrically. The indications for the results of surgery are well-defined.

Other forms of thoracic outlet syndrome have been described. These include post-traumatic and vascular forms of outlet syndrome, which are rare. The most controversial entity, "non-specific outlet syndrome" is characterized by sensory symptoms and a normal neurological exam; there are no objective tests which can unequivocally diagnose this condition. The indications for surgery are poorly defined and controversial.

Key word: thoracic outlet syndrome, brachial plexus, medial scalene band, cervical rib, scalene triangle

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THORACIC OUTLET SYNDROMES

The diagnosis and treatment of thoracic outlet syndrome is one of the most difficult and controversial areas of neurology. The reasons for this can be discovered by reviewing the anatomy of the thoracic outlet and the historical background of this condition.

ANATOMY

The thoracic outlet (or inlet, as it is sometimes called) is a somewhat complex anatomical construct [7, 9, 18]. It basically consists of two parts; the first is the scalene triangle, which is formed anteriorly by the scalenus anticus muscle and posteriorly by the scalenus medius muscle. The base of the triangle is the first rib. The brachial plexus and subclavian

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Address reprint request to : Russell W. Hardy, Jr., M.D., Professor, Department of Neurological Surgery, University Hospitals of Cleveland, 11100, Euclid Avenue, Cleveland, Ohio, 44106, U.S.A.

artery pass through this triangle on their way to the arm. These structures are subsequently joined by the subclavian vein in a second portion of the outlet, the costoclavicular space. This is bordered anteriorly by the first rib and posteriorly by the clavicle. Some writers identify a third entity, the subcoracoid space, which is beneath the pectoralis minor tendon; both neural and both vascular structures pass through this region.

Thus, in the normal individual there are several potential sites of neurovascular compression. In addition, some patients have anomalous anatomical structures[14], such as compressive cervical bands, cervical ribs or enlarged C7 transverse processes, which also may compress neurovascular structures [7].

Some authors believe that an incongruity between the skeletal structures (ribs and vertebrae) and the neural elements (nerve roots and brachial plexus), is a factor in these compressive syndromes. It is postulated that compression occurs in those patients who have either an incomplete prefixed or post-fixed brachial plexus. A prefixed plexus has a contribution only from cervical roots, with no T1 component; a complete cervical rib is also present. A post-fixed plexus is one that has a contribution from the upper two thoracic roots. An incomplete prefixed plexus has an incomplete cervical rib with some T1 contribution; an incomplete post-fixed plexus has an anomalous first thoracic rib. Symptoms may occur in either of these situations [9,19].

Finally, it should be noted that there are both neurological and vascular structures passing through the various spaces in the thoracic outlet. Compression of the brachial plexus, subclavian artery and subclavian vein might be expected to give rise to neurological and/or vascular symptoms which are sometimes distinct but in other situations may overlap, and therefore lead to confusion in diagnosis.

HISTORY

The history of this condition dates back to the mid-nineteenth century when Cooper described

symptoms of thoracic outlet syndrome and to Coote, who performed the first cervical rib resection, in a patient with neurological findings [18]. Bramwell and Dyke, Thomas and Cushing [2, 15], as well as others independently reported patients with motor symptoms which were attributed variously to first rib compression, cervical rib compression or anomalous bands. Unfortunately, these early reports were based solely on clinical findings without the advantage of electrodiagnosis or x-ray. Moreover, these papers were published long before carpal tunnel syndrome was described, so, early on, considerable confusion existed as to the true etiology of thoracic outlet syndrome. This confusion was later compounded by the description of three separate conditions: the scalenus anticus syndrome, the costoclavicular syndrome and the hyperabduction syndrome. The first was based on work by Adson and Coffey [1] and by Ochsner [8] who attributed compressive symptoms to the anterior scalene muscle. Falconer and Weddel ascribed symptoms to compression of the neurovascular elements between the clavicle and first rib [4], and Wright to compression of the neurovascular structures beneath the pectoralis muscle, when the arm was elevated [20].

It should be noted that the original papers described patients with objective neurological findings (muscle wasting) even though there was doubt as to the exact site of compression. However, as time went by, patients with subjective symptoms such as pain, cold and numbness were diagnosed with thoracic outlet compression even in the absence of objective signs and symptoms.

Thus, what had originally been a fairly narrowly defined condition, namely muscle wasting associated with an anomalous cervical rib, evolved into a very broad and poorly defined clinical syndrome. What had originally been regarded as an unusual ailment became one which was probably over-diagnosed and over-treated. A large number of individuals underwent a variety of operations and predictably, a number of failures of surgical therapy. In North America and Europe some surgeons continue to operate on many patients, while others believe the

Fig. 1: Diagram showing the site of the medical scalene band which compresses the lower portion of the brachial plexus. Arrow points to location of band.

Fig. 2: AP radiograph showing bilateral prominent C7 transverse processes.

syndrome exists rarely if at all. As with most things, the truth lies somewhere between these extremes. There clearly is a syndrome of sensory symptoms associated with objective neurological findings in patients with anomalous first ribs and there appears to be a larger group of patients without neurological findings or skeletal anomalies [6]. Patients in the first category can be helped by an operation and some of the patients in the second may also benefit from surgery. The problem with the second group is establishing the diagnosis preoperatively.

CLINICAL FEATURES

We will discuss the clinical features of the various forms of this condition using a schema devised by Wilborn [15]. He has divided thoracic outlet syndrome into three categories: those with a vascular

etiology, those with a clear or possible neurologic etiology, and those which are combined, including those which may be traumatic in origin. He further divides the neurological compressive syndromes into those where there is a demonstrable deficit, and those with non-specific sensory symptoms but no objective neurologic deficit.

< Classic (True) Thoracic Syndrome >

This form of thoracic outlet syndrome is the one originally described and the least controversial. It results from compression of the C8/T1 roots of the brachial plexus by a fibrous band running between a cervical rib (or prominent C7 transverse) process and the first rib; this band is continuous with the medial edge of the scalenus medius. (Fig 1). Some authors have described various anatomical variations on this

basic anomaly [7]. In all cases, however, neurological signs and symptoms result from compression of the lowest portion of the brachial plexus.

Patients typically present with atrophy beginning in the abductor pollicis brevis and eventually involving other thenar and hyperthenar muscles; in advanced cases, the forearm flexors may be involved as well. Most, but not all, have some sensory signs or symptoms in the medial portion of the arm. The diagnosis is made by the finding of cervical ribs or prominent transverse C7 processes on plain x-ray (Fig 2), combined with characteristic electromyographic/nerve conduction findings [5]. These consist of slowing of the ulnar sensory nerve action potential in the face of a normal median sensory potential. The median and ulnar motor potentials are also reduced, the median more than the ulnar. Needle examination will show motor unit potential loss and neurogenic changes in a lower trunk distribution (Fig 3).

In a patient with characteristic clinical features, x-ray and electrical findings should establish the diagnosis. The differential diagnosis includes syringomyelia, tumors of the lower brachial plexus, carpal tunnel syndrome and ulnar neuropathy. The latter two can be excluded by EMG/NCT testing. If any doubt exists regarding an intraspinal lesion or a nerve tumor these can be excluded by an MRI; usually, however, this is not required.

Once the diagnosis has been confirmed, we treat these patients by supraclavicular section of the medial scalene band. It is not necessary to remove the cervical rib. Results of this operation have generally been gratifying, although we caution patients that pre-existing motor deficits will not disappear [6].

< Vascular Thoracic Outlet Syndromes >

Syndromes of both arterial and venous compression have been described [7, 8]. The arterial form presents with symptoms of distal limb ischemia, including Raynaud's phenomenon and ulcerations. The vascular changes are due to thrombosis or emboli which may result from occlusive disease at the site of

compression, or from post-stenotic dilatation. In these rare patients the diagnosis may be confirmed by photoplethysmography, phonoangiography and Doppler flow studies, although standard arteriography is the most definitive to establish the diagnosis.

Venous compression also may result in chronic or acute symptoms. Paget-Schroetter syndrome is a form of acute subclavian vein thrombosis which presents with sudden arm edema, cyanosis, deep arm pain and paresthesias. This condition can be established by venography. Intermittent venous obstruction has also been described. The current treatment includes first rib resection and treatment with thrombolytic agents.

We have personally seen one case of venous obstruction related to a non-union of a clavicular fracture; in this instance, resection of the fracture callous was followed by a pulmonary embolus from the affected arm.

< Non-Specific Thoracic Outlet Syndrome >

This is the largest single category of thoracic outlet syndrome, and unfortunately, the most controversial. The syndrome consists of pain and paresthesiae involving the arm, but sometimes the neck and suprascapular region. These symptoms are attributed to compression of the lower portion of the plexus although an upper plexus variation has also been described. Symptoms tend to be increased by activity or elevation of the arm. Autonomic symptoms, weakness, fatigue and headache have also been attributed to this condition. The neurologic exam is typically normal although some investigators describe "weakness" of a rather non-specific type. Various maneuvers have reported to bring on the symptoms, including Adson's maneuver, the hyperabduction test and bracing of the shoulders [11]. Adson's maneuver consists of the demonstration of a reduced radial pulse which is obtained when the neck is extended and the head rotated toward the affected side [1]. Roos has described reproduction of symptoms with the arm abducted and in external rotation [12]. Unfortunately, the accuracy of both the

Fig. 3: Diagram summarizing the electrical changes seen in "true" thoracic outlet syndrome.

Adson's and the elevated arm stress test have been called into question.

Similarly, other objective tests, including Doppler flow studies and angiography may give misleading or inaccurate data. NCT/EMG studies are typically normal in this condition. It has been suggested that measuring nerve conduction across Erb's point may be a way of diagnosing this condition, but the accuracy of such measurements have been vigorously challenged by a number of observers [16, 18]. Finally, attempts have been made to measure somatosensory evoked potentials, but the results of these have also been ambiguous.

In view of the vagueness of the symptoms and physical findings, and the lack of objective tests to establish the diagnosis, it is difficult to decide which, if any, patients should undergo surgery for this condition. Some enthusiastic surgeons treat large numbers of patients with either transaxillary or other types of first rib resection [13, 16]. Our own view is that this condition is rarely encountered, but that there are some patients who will respond to an operation. We will recommend surgery for a patient with non-specific pain and fatigue which is increased by an elevated position of the arm or by activity. Normally

we also insist that other diagnoses, as well as functional or legal factors be excluded and that the patient undergo an unsuccessful program of physical therapy designed to strengthen the shoulder-girdled musculature [10].

< Traumatic Thoracic Outlet Syndromes >

As noted above, we have seen one case where a clavicular fracture produced a thoracic outlet syndrome by virtue of venous compression. Other similar cases have been described, producing a brachial plexopathy or vascular injury [18]. This may occur acutely with a fracture or may develop as a result of a hyperthrophic callous forming at the fracture site. In such cases the diagnosis may be made by a combination of history, x-ray examination of the clavicle and electrodiagnostic studies, and arteriogram and venogram as indicated.

TREATMENT

We believe that "true" thoracic outlet syndrome should be treated surgically. The operation which we have used is a supraclavicular exploration of the

Fig. 4:

A. Location of supraclavicular incision required for exposure of medial scalene band.

B. Arrow demonstrates resection of band which can be performed above or below subclavian artery.

brachial plexus with sectioning of the medial scalene band [6]. The plexus is exposed through a standard supraclavicular incision; the lower plexus and subclavian artery are identified and then traced medially beneath the anterior scalene. This muscle may be partially sectioned (care being taken to preserve the phrenic nerve) or in some cases retracted. Once this is done it is possible to palpate the sharp knife-like edge of the medial scalene band. With further blunt dissection one may expose the lower plexus and observe the neural elements arched over the band. The band may be sectioned between the subclavian artery and the lower plexus or, in some cases, in which it is difficult to visualize the neural elements as they are stretched over the band, just below the subclavian artery. Once the band has been divided, the surgeon will be able to appreciate the immediate decompression of the neural elements by direct visualization and by palpation (Fig. 4, A and B). It is not necessary to resect the remnant of the cervical rib in order to obtain a good result. In 27 patients thus treated, we have had good results in 25. In all cases sensory symptoms (pain, paresthesias and

cold sensations) were relieved. We have not seen any substantial recovery of motor function in any of these patients, presumably because of the prolonged effects of axonal compression. Two patients were failures. One stated she had never had good relief of pain but in her case there were worker compensation issues which clouded the result. One other patient had good relief for one year but ultimately required first rib resection when symptoms returned.

Treatment of traumatic thoracic outlet syndromes may be non-operative with physical therapy and local heat and analgesics. More often, surgical intervention is required to reduce a displaced fracture or resect a portion of the callous resulting from clavicular fracture. If a pseudo-aneurysm is present, it also may require direct vascular surgery. Our one patient with this type of injury responded well to partial resection of the clavicle.

Non-specific thoracic outlet syndrome is initially treated non-operatively. Peet and his co-workers described a series of strengthening exercises for the shoulder-girdled muscles and some writers consider these effective [10]. Weight loss, treatment of

depression and reduction mammoplasty if large pendulous breasts are present has also been recommended. We have found relatively few candidates for surgical treatment of this condition, and when we do we employ a posterior approach with resection of the cervical rib (if present) and also the first rib [3]. This operation will necessarily also result in resection of the insertion of the scalenus medius and scalenus anticus muscles. In the highly selected patient group thus treated, we have seen fairly good results. Many surgeons in North America employ the transaxillary approach described by Roos; [13] unfortunately we have seen many complications of the operation when done by inexperienced surgeons.

A supraclavicular rib resection has also been used, but also carries the risk of significant neurological complications.

Evaluation of surgical results in the non-specific form of outlet syndrome is difficult because of variability in selection and outcome criteria. Success rates reported range from 37% to 90% good results [18]. Somewhat more disturbing are reports of a large number of individuals who require a second operation following failure of the initial procedure. Whether this is due to poor patient selection or problems with surgical technique is debatable [17].

SUMMARY

Thoracic outlet syndrome remains a controversial, and possibly over-treated condition. True thoracic outlet syndrome, characterized by lower brachial plexus compression by medial scalene band, is rare, but may be readily diagnosed and responds well to operative intervention. Traumatic outlet syndrome is highly unusual but maybe treated by appropriate surgical intervention. Non-specific thoracic outlet syndrome is very controversial with widely different opinions regarding its prevalence and the place for surgical intervention. No definitive tests are available for diagnosing the condition preoperatively. We believe that surgical intervention is indicated in some patients, but we are also convinced that in many

instances this entity is over-diagnosed and surgically over-treated.

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Editor-in-Chief's comment :

Hiroshi Abe

University of Hokkaido School of Medicine

Sapporo, Japan

I appreciate Dr. Russell W. Hardy, Jr. for his contribution to submit this article to "Spinal Surgery". This is an outstanding review article to understand and recognize thoracic outlet syndrome which is a still complex and controversial entity. As the author describe in this article, we have to diagnose thoracic outlet syndrome accurately and classify to four types as "true", vascular, traumatic, or "non-specific". To diagnose thoracic outlet syndrome correctly, we should have a through knowledge of anatomy, pathophysiology, and electromyographic nerve conduction findings of thoracic outlet syndrome.

Co-Editor's comment :

Hiroshi Nakagawa

Aichi Medical University

Aichi, Japan

This is an excellent review article of so-called thoracic outlet syndrome which really remains complex and controversial both in diagnosis and treatment. As the author clearly pointed out, thoracic outlet syndromes included true, vascular, traumatic and non-specific thoracic outlet syndromes.

We, spinal neurosurgeons, may have an opportunity to see these patients at our clinics as well as orthopedic surgeons and vascular surgeons, therefore it is very important for us to have better understanding of this syndrome to differentiate its real causes. It is also essential to differentiate it from amyotrophic spondylotic myelopathy, flexion myelopathy, peripheral nerve entrapment syndrome, and ossification of the posterior longitudinal ligament by using dynamic X-ray, CT, MRI and EMG/NCT.