CLINICAL REVIEW

The Thoracic Outlet Syndrome in Athletes

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Background: **The array of symptoms that characterize thoracic outlet syndrome (TOS) often lead to a failure or delay in diagnosing this condition in persons who are physically active.**

Methods: **Using the key words and phrases "thoracic outlet syndrome," "sport," "exercise," and "athlete," the MEDLINE files from 1991 to April 1996 were searched. Articles dating before 1991 were accessed by cross-referencing the more recent articles.**

Results **and** ***Conclusions:*** **TOS results from compression of the neural or vascular structures of the upper extremity at the thoracic outlet. Clinical manifestations can include upper extremity pain, paresthesias, numbness, weakness, fatigability, swelling, discoloration, and Raynaud phenomenon. Four symptom patterns have been described: upper plexus, lower plexus, vascular, and mixed. The lower brachial plexus pattern is the most common. Specific causes of outlet compression include injury to the scalene or scapular suspensory muscles, anomalous fibromuscular bands, cervical ribs, clavicular deformity, and pectoralis minor tendon hypertrophy. The diagnosis of TOS is established on the results of the history and physical examination. Ancillary studies are most helpful to rule out other conditions rather than confirm the diagnosis of TOS. In most cases the initial treatment is nonoperative with an emphasis on rehabilitative exercises for the neck and shoulder girdle. Surgery is indicated for acute vascular insufficiency, progressive neurologic dysfunction, and refractory pain that fails conservative treatment. The surgical technique involves the release or removal of the structures that cause compression and can involve scalene muscle release, first rib resection, cervical rib excision, and resection of fibromuscular bands. (J Am Board Fam Pract 1996;9:346-55.)**

An increasing number of people are physically active and are continuing to be active into their middle and older ages. Consequently, more patients are experiencing physical conditions that can be associated with athletic activity. The array of symptoms that characterize thoracic outlet syndrome (TOS) often lead to a failure or delay in diagnosing the condition. This review details the clinical manifestations, causes, diagnosis, and management of TOS in physically active persons.

Methods

The MEDLINE files from 1991 to April 1996 were searched using the following key words and phrases: "thoracic outlet syndrome," "sport," "exercise," and "athlete." Articles dating before 1991 were accessed by cross-referencing the more recent articles.

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Thoracic Outlet Syndrome

TOS is a clinical syndrome characterized by symptoms attributable to compression of the neural or vascular anatomic structures that pass through the thoracic outlet. Epithets for TOS describe numerous potential sources of compression, and include cervical rib syndrome, scalenus anticus syndrome, hyperabduction syndrome, costoclavicular syndrome, pectoralis minor syndrome, and first thoracic rib syndrome. TOS affects women twice as frequently as men, and the mean age of those who require surgical treatment is the fourth decade.[1-4] The absence of a definitive diagnostic test that confirms TOS has led some authors to question its very existence.[5] The incidence of TOS varies markedly between countries. It is recognized with great frequency in the United States, but only rarely in Australia and Great Britain.[6]

During the normal growth of children and

adolescents, the scapulae gradually descend upon the posterior thorax. The distance of descent is slightly greater in women than in men. Strain injury to the scapular suspensory muscles, which lengthen in conjunction with scapular descent during normal development, is known to be associated with TOS. These facts help to explain the rarity of symptomatic TOS until after puberty, and the increased prevalence in women.[7]

TOS is an uncommon athletic injury, although epidemiologic data are lacking.[7,8] Several authors have reported TOS vascular symptoms in overhand-throwing athletes such as baseball pitchers.[8-10] It has also been described in a football player after trauma, an oarsman, and a former US national champion tennis player who had dominant "shoulder dependency."[8,10-12]

Historical Perspective

In 1743, Hunald associated the cervical rib with the development of TOS, and Cooper, in 1818, was the first to discuss the medical management of TOS.[8] Coote showed that cervical rib excision could successfully relieve pressure on the axillary nerves and vessels in 1861, and Stopford and Telford implicated the first thoracic rib as a cause of neurovascular compression in 1919.[8] During the next decade, Adson[13] stressed the role of the scalene muscles in neurovascular compromise and thus popularized surgical scalenectomy as a means of therapy. Wright[8] showed that shoulder hyperabduction could produce thoracic outlet obstruction in 1945. Roos[14] emphasized the importance of the first rib and its muscular and ligamentous attachments in causing thoracic outlet obstruction during his work of the early 1960s.

Anatomy

The thoracic outlet is the anatomic portal through which the great vessels and nerves of the upper extremity pass (Figure 1). Bony boundaries of the outlet include the clavicle, first rib, and scapula. The outlet passage is further defined by the interscalene interval, a triangle with its apex directed superiorly, bordered anteriorly by the anterior scalene muscle, posteriorly by the middle scalene muscle, and inferiorly by the first rib. The brachial plexus comprises the anterior rami of nerve roots C5 through T1, which exit through the intervertebral formina and form trunks that pass through the interscalene triangle before dividing behind the clavicle. Divisions of the neural trunks reunite to form cords that surround the axillary artery as it passes deep to the pectoralis minor tendon.

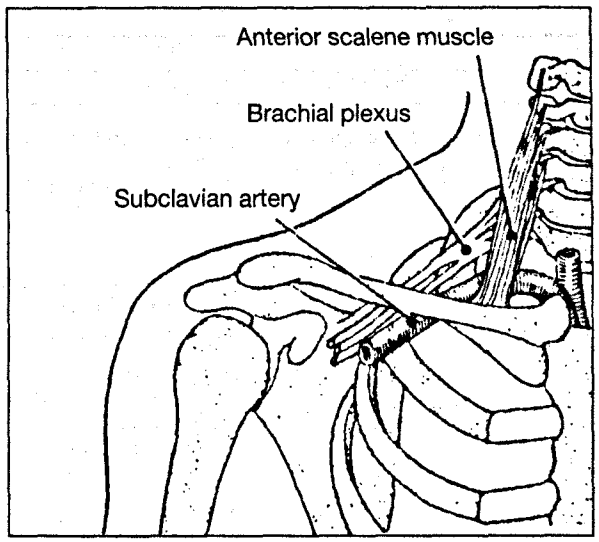

Figure 1. Anatomy of the thoracic outlet.
Reprinted with permission from Priest JD, Nagel DA. Tennis shoulder. Am J Sports Med 1976;4:28-42. Figure 13. "Normal anatomy thoracic outlet region."

The motor and sensory branches of the brachial plexus typically divide distal to the pectoralis minor tendon. The lowest trunk of the plexus, made up of rami from C8 and T1 nerve roots, lies above the first rib and behind the subclavian artery. It provides sensation to the fourth and fifth fingers of the hand and motor innervation to the hand intrinsic muscles. The lower trunk is the most commonly compressed neural structure in TOS. The subclavian arteries exit the thorax bilaterally behind the sternoclavicular joints, pass over the first ribs between the anterior and middle scalene muscle insertions, course laterally behind the clavicles where they become the axillary arteries, and continue posterior to the tendons of the pectoralis minor muscles, where they change to the brachial arteries. The axillary veins pass behind the costocoracoid ligaments and pectoralis minor tendons and advance over the first ribs anterior to the anterior scalene muscle to join the jugular vein at the base of the neck.

Clinical Manifestations

TOS can cause a myriad of symptoms reflecting neurovascular compression. Potential symptoms include pain localized in the neck, face, head, upper extremity, chest, shoulder, or axilla; and upper

Table 1. Symptom Patterns in Thoracic Outlet Syndrome.

Symptom Pattern	Occurrence	Symptoms
Lower trunk	Most common	Neck and shoulder pain and paresthesias Radiation into medial arm, forearm, 4th-5th fingers Grasp weakness
Upper trunk	Uncommon	Neck, shoulder, and face pain and paresthesias Radiation into lateral arm
Vascular	Rare	Venous: arm edema, bluish discoloration, collateralization of vessels Arterial: arm coolness, numbness, exertional fatigue
Mixed	Common	Combination of neurologic and vascular

Reprinted with permission from Karas.[16]

extremity paresthesias, numbness, weakness, heaviness, fatigability, swelling, discoloration, ulceration, or Raynaud phenomenon. Neural compression symptoms occur more commonly than vascular symptoms, accounting for up to 90 to 95 percent of symptoms.[15]

Karas[16] described four symptom patterns of TOS, characterized by the primary structures compressed, which are outlined in Table 1. The lower trunk pattern reflects lower plexus compression and manifests with neck and shoulder pain, paresthesias that often radiate into the medial arm, forearm, and fourth and fifth fingers, and weakness of grasp. The upper trunk pattern results from upper plexus compression and is distinguished by pain in the neck, shoulder and face, and paresthesias that radiate into the lateral arm and simulate C5-6 nerve root compression.[14,16,17] Figure 2 displays the pain distributions of the upper and lower trunk patterns. The vascular pattern results from venous or arterial compression at the thoracic outlet. Studies are conflicting as to which type occurs more frequently.[1,4] Subclavian venous compression causes arm edema, bluish discoloration, and collateralization of vessels across the chest and shoulder. Arterial compression produces arm coolness, numbness, and exertional fatigue.[16] Finally, the mixed pattern consists of a combination of vascular and neurologic symptoms.[16]

Etiology

Neurovascular compression can occur at any of three sites on the thoracic outlet: the interscalene triangle, the costoclavicular interval, and the pectoralis minor tendon insertion on the coracoid process of the scapula. Specific causes of thoracic outlet obstruction are listed in Table 2.

Interscalene triangle compression can result from injury of the scalene or scapular suspensory

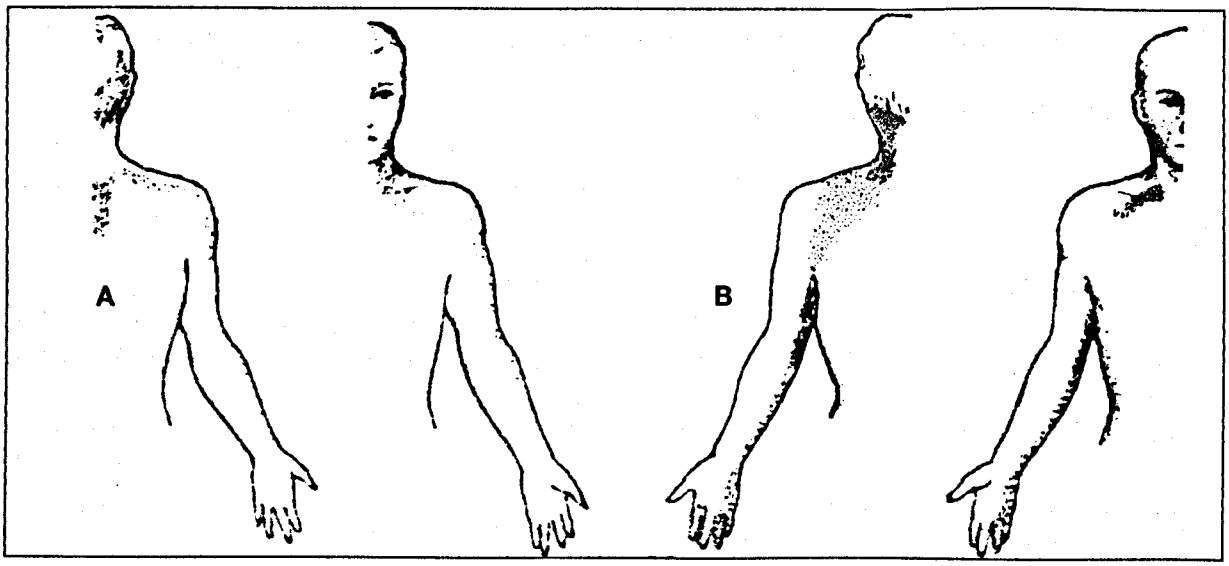

Figure 2. Distribution of pain caused by the (A) upper plexus type and (B) the lower plexus type of thoracic outlet syndrome.

Reprinted with permission from Roos DB. The place for scalenectomy and first-rib resection in thoracic outlet syndrome. Surgery 1982;92:1077-85.

muscles. The scalene muscles are often associated with anomalous fibromuscular bands, which can compress the neurovascular structures of the outlet in patients with the TOS. Histochemical analysis of the scalene muscles in affected individuals has confirmed excess amounts of fibrous tissue. Nine anatomic variations of anomalous fibrous bands have been described. The most common type (type 3), stretches from the neck of the first rib and passes anteriorly across the outlet to lie between the T1 nerve root and the subclavian artery (Figure 3).[4] Fibrous bands can also originate from long transverse processes of lower cervical vertebrae or from cervical ribs.

Table 2. Causes of Thoracic Outlet Syndrome by Anatomic Site of Compression.

Site	Cause
Interscalene triangle	Scalene muscle fibromuscular bands
	Scapular suspensory muscle weakness
	Cervical ribs
	Long transverse process of C7
	"tennis shoulder"[12]
	Tumor[18]
	Fibrous dysplasia of the first rib[19]
Costoclavicular interval	Hypertrophic callus from clavicular fractures
	Cervicothoracic scoliosis[20]
	First costovertebral joint osteoarthritis[21]
	Congenital clavicular pseudoarthrosis[22]
Pectoralis minor tendon coracoid insertion	Repetitive hyperabduction or overhead activity
	Pectoralis minor hypertrophy

Cervical ribs, which are present in 0.2 percent of the population and occur bilaterally in 80 percent of those affected, are an additional source of interscalene triangle obstruction.[14] Fewer than 10 percent of individuals with cervical ribs ever experience TOS, however, and only 7.5 to 9 percent of surgically treated cases involve cervical ribs.[14] As cervical ribs lengthen to at least 5.5 cm, they tend to lift up and kink the subclavian artery and stretch the seventh cervical nerve root.[11] Tennis shoulder, as described by Priest and Nagel,[12,23] has also been associated with interscalene triangle compressive symptoms of TOS. Tennis shoulder affects the relatively muscular, dominant upper extremity and is characterized by a downward displacement of the upper extremity and shoulder girdle as a result of its relative weight, which causes stretching of the scapular suspensory muscles.

Compression at the costoclavicular interval, the space that lies between the rib cage and the posterior aspect of the clavicle, can be exacerbated by repetitive shoulder abduction or a clavicular deformity. Shoulder abduction causes the S-shaped clavicle to move backward, which compromises the costoclavicular space. Previous clavicular fracture with a hypertrophic callus deformity can further predispose to costoclavicular interval compression.

Pectoralis minor tendon compression is associated with shoulder hyperabduction. During hyperabduction, the tendon insertion and the coracoid act as a fulcrum about which the neurovascular structures are forced to change direction. Hypertrophy of the pectoralis minor tendon has also been noted as a cause of outlet compression.[8]

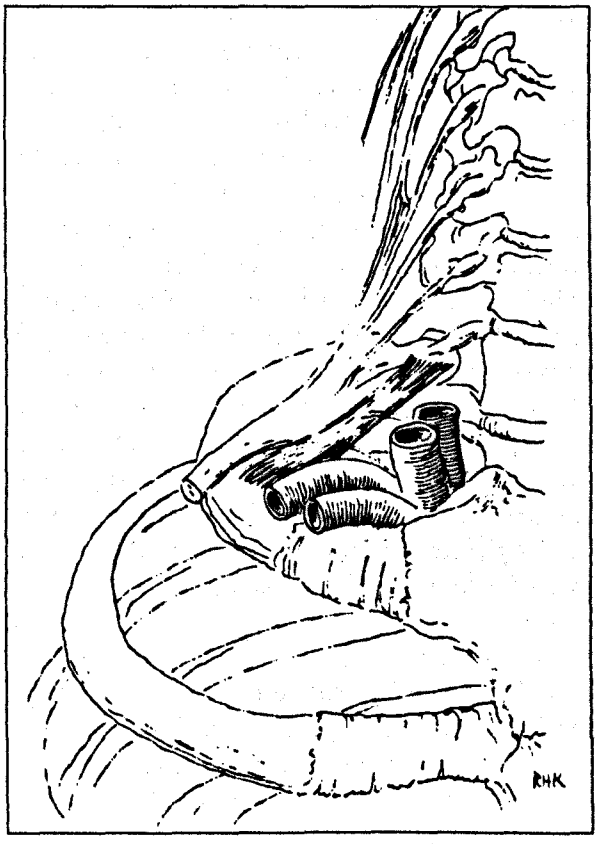

Figure 3. The most frequent anatomic type of anomalous band encountered.

Reprinted with permission from Wood VE, Twito R, Verska JM. Thoracic outlet syndrome. The results of first rib resection in 100 patients. Orthop Clin North Am 1988;19:131-46.

Twenty-one to 75 percent of TOS patients have an antecedent history of trauma.[4,6,17] Most

Table 3. Differential Diagnosis of Thoracic Outlet Syndrome.

Adhesive capsulitis
Brachial plexus neuritis
Carpal tunnel syndrome
Cervical disc herniation
Cervical spondylosis
Glenohumeral or acromioclavicular degenerative joint disease
Instability syndromes
Reflex sympathetic dystrophy
Rotator cuff tendinitis, bursitis, or impingement
Tumor- or space-occupying lesion
Ulnar neuropathy at wrist or elbow

Adapted from Tucker.[25]

cases of trauma involve motor vehicle accidents in which injury to the shoulder girdle causes local hemorrhage and altered shoulder mechanics, both of which can lead to neurovascular compression. Additionally, strain injury of the scapular-stabilizing muscles—the trapezius, levator scapulae, and rhomboids—predisposes to outlet obstruction. Histologic muscle fiber examination of individuals with posttraumatic TOS show type II muscle fiber atrophy, increased number of type I fibers, and high amounts of connective tissue.[24] Many case reports of TOS in athletes fail to describe preceding macrotrauma; instead, the manifestations are often vascular and seem related to cumulative microtrauma from repetitive overhead activities.[9,10] Tucker[25] recently reported the case of a football player with TOS following a motor vehicle accident.

Vascular Outlet Obstruction

Durham et al[26] retrospectively reviewed 50 cases of thoracic outlet subclavian artery compression injury and discovered that most are associated with a bony anomaly. A cervical rib or anomalous first thoracic rib can compress the subclavian artery, resulting in aneurysm formation caused by poststenotic dilatation. Similarly, the bony structures can compress the axillary artery and its humeral circumflex branches against the humeral head during shoulder abduction.[16] Peripheral embolization is a potential complication.[9] McCarthy et al[9] published a series of 11 cases of athletes with compressive syndromes of the subclavian or axillary arteries, most of whom were involved in overhead sports. Treatments were tailored to the individual athlete and included resection of the pectoralis minor or scalene muscles, embolectomy, or saphenous vein grafting.

Axillary vein thrombosis occurs by two mechanisms: (1) venous compression between the clavicle and the first rib while the arm is overhead; and (2) repeated rubbing of the vein against the clavicle, which initiates the thrombotic mechanism.[27] Pulmonary embolus, a potential complication of axillary vein thrombosis, occurs uncommonly in those with effort-induced symptoms but much more frequently in individuals with underlying medical diseases, such as malignancy, drug abuse, and central venous catheterization.[28] Treatment options for axillary vein thrombosis include anticoagulation, relative rest, elevation of the upper extremity, surgical thrombectomy, and local thrombolytic therapy.[28] Axillary vein obstruction that can occur from extravascular compression of the vein by bone or soft tissue structures, with or without venous thrombosis, is known as the Paget-Schroetter syndrome. A postulated case of the syndrome was reported in a competitive swimmer who developed acute subscapularis intramuscular edema after an impromptu, intense upper body weight-lifting session.[29] Venography provides the best diagnostic means of differentiating between extravascular and intravascular causes of axillary vein obstruction.

Physical Examination

TOS is a clinical diagnosis, made almost entirely on the basis of the history and physical examination. As previously mentioned, there is no single diagnostic test available to confirm the diagnosis. To make the diagnosis, however, it is often necessary to rule out other conditions that can mimic TOS. A differential diagnosis of TOS appears in Table 3.

The physical examination should include a careful inspection of the spine, thorax, shoulder girdles, and upper extremities for postural abnormalities, shoulder asymmetry, muscle atrophy, excessively large breasts, and drooping of the shoulder girdle.[16] The supraclavicular fossa should be palpated for fibromuscular bands, percussed for brachial plexus irritability, and auscultated for vascular bruits that appear by placing the upper extremity in the position of vascular compression. The neck and shoulder girdle should be assessed for active and passive ranges of motion, areas of

tenderness, or other signs of intrinsic disease. A thorough neurologic examination of the upper extremity should include a search for sensory and motor deficits and abnormalities of deep tendon reflexes.

Provocative Testing

Unfortunately, the classic provocative tests for thoracic outlet obstruction carry high false-positive and false-negative rates.[7] The greatest diagnostic utility of these tests lies in the examiner's ability to reproduce the patient's symptoms rather than to obliterate the radial pulse during the provocative maneuver. More than 50 percent of normal, asymptomatic people will exhibit obliteration of the radial pulse during classic provocative testing.[1,17]

The Adson test, or the scalene maneuver, was first described by Dr. Adson, the former chief of neurosurgery at the Mayo Clinic.[13] This test increases the tone of the anterior and middle scalene muscles and consequently provokes symptoms of interscalene triangle compression. The test is performed by having the patient extend the neck, turn the head toward the side being examined, and take a deep breath.[13]

The Wright test, or the hyperabduction maneuver, tests several points along the thoracic outlet for compression and is considered by many to be the best provocative test for thoracic outlet compression.[7] The test is performed by asking the patient to turn the head away from the side being examined and take a deep breath while the examiner passively abducts and externally rotates the patient's arm.

The costoclavicular compressive maneuver helps to single out patients with symptoms attributable to costoclavicular space compromise. During this test the shoulders are drawn back and downward in an exaggerated military position so as to reduce the volume of the costoclavicular space. As in the cases of the Adson test and the Wright test, the costoclavicular compression maneuver is considered to be positive if the radial pulse disappears or if clinical symptoms are reproduced during provocative positioning.

The overhand exercise test is useful to detect thoracic outlet arterial compression. During this test the patient elevates both arms overhead and then rapidly flexes and extends the fingers. A positive test is achieved if the patient experiences heaviness, fatigue, numbness, tingling, blanching, or discoloration of a limb within 20 seconds.[7]

Table 4. False-positive Provocative Test Results for Thoracic Outlet Syndrome in an Asymptomatic Control Group.

Test	Vascular (%)	Neurologic (%)
Adson test	13.5	2.0
Wright test	57.0	16.5
Costoclavicular compression maneuver	47.0	10.0

Reprinted with permission from Rayan and Jensen.[31]

Novak et al[30] described positive provocative test results in 94 percent of TOS patients. Rayan and Jensen[31] found that the prevalence of positive provocative tests results in a group of asymptomatic controls (false positives) without TOS and discovered that positive vascular responses (obliterated radial pulses) occurred far more commonly than neurologic symptoms. Table 4 displays the prevalence of positive vascular and neurologic provocative test results in asymptomatic controls.[31]

Riddell and Smith[1] compiled preoperative physical findings in a group of 53 patients who underwent surgery for TOS. Detectable objective findings included sensory loss in 34 percent, supraclavicular mass in 11 percent, muscle atrophy in 9 percent, and scalene tenderness in 9 percent. Vascular features consisted of a positive Adson sign in 72 percent, radial pulse diminution or absence in 17 percent, arterial insufficiency in 15 percent, and venous obstruction in 13 percent.

Ancillary Testing

As previously stated, ancillary tests are most useful to exclude conditions that mimic TOS. Perhaps the most beneficial initial test to obtain in the workup of TOS is a cervical spine radiograph series. Plain radiographs can distinguish cervical ribs, long transverse processes of the C7 vertebrae, old clavicular fracture, degenerative arthritic disease of the cervical spine, or a first rib abnormality.

Electromyography (EMG) has little specificity in the diagnosis of TOS. A prospective study found the EMG findings to be abnormal in just 1 of 50 TOS patients.[30] EMG can, however, help to

detect or rule out cervical radiculopathy, carpal tunnel syndrome, or other distal entrapment neuropathies. Passero and colleagues[32] helped to correlate the development of abnormal neurodiagnostic test findings in individuals with true neurogenic TOS. They showed that persistent outlet compression first produces alterations in the EMG, followed by changes in F-waves and somatosensory evoked potentials (SEPs), and finally by abnormalities in nerve conduction parameters. The authors concluded that neurodiagnostic testing, particularly nerve conduction studies, are of little value in making the diagnosis of TOS. Others have shown that changes in sensory thresholds elicited during provocative testing can be useful in diagnosing TOS.[30] Another group of researchers observed that abnormal SEP results (characterized by amplitude reduction and latency delays) and abnormal arterial photoplethysmography findings (defined by complete loss of arterial pulsation) are associated with better surgical outcomes in patients undergoing first rib resection for TOS.[33]

Myelography has been largely abandoned in the initial evaluation of the cervical spine for intervertebral disc herniations and nerve root compression as a result of the emergence of magnetic resonance imaging (MRI) and computerized tomography (CT). MRI has limited diagnostic utility for TOS, although attempts have been made to correlate MRI findings with abnormalities of the brachial plexus and the vasculature of the upper extremity.[34] Panegyres et al[35] showed that an MRI can detect deviation or distortion of blood vessels of the thoracic outlet with a sensitivity of 79 percent, specificity of 87.5 percent, and false-positive rate of 9.5 percent. The MRI also can detect fibromuscular bands and other causes of TOS, such as a bony callus of the first rib. The CT is less helpful in establishing the diagnosis of TOS, as only 32 percent of TOS patients had a CT-recognizable compressive anatomic abnormality in one series.[30]

Vascular testing by arteriography or venography is indicated when there are symptoms and signs of arterial insufficiency or venous obstruction. It is also indicated when a cervical rib or long C7 vertebral transverse process is found because of the high association of aneurysmal formation secondary to bony compression and poststenotic aneurysmal dilatation.[16,36]

Nonsurgical Treatment

Most cases of TOS can be successfully treated without surgical intervention. Nonoperative cure rates have been reported to range from 70 to 88 percent.[3,15] The typical patient who responds best to nonsurgical measures is middle-aged, female, obese, and has poor posture. Consequently, younger elite athletes might not respond as favorably to nonoperative treatment regimens.

The mainstay of nonsurgical treatment is rehabilitative exercise geared to correct postural abnormalities of the neck and shoulder girdle and to strengthen the scapular suspensory muscles. Kenny et al[37] prospectively evaluated a group of 8 patients whose TOS was treated with a supervised physical therapy program of graduated resisted shoulder elevation exercises. All patients showed major symptomatic improvement. This series, however, is comprised largely of middle-aged women with TOS, leaving its clinical applicability to a younger, more athletic population uncertain. In addition to rehabilitative exercises, treatment can be directed toward symptomatic relief. Examples include ice, heat, sonography, and transcutaneous nerve stimulation, nonsteroidal anti-inflammatory medications (NSAIDs), trigger point injection, weight reduction, brassiere support or breast reduction surgery, and medication or psychotherapy to treat associated depression.[7]

On occasion, TOS can be acute, causing the patient severe pain and disability. In these cases, initial treatment consists of an arm sling, analgesics, and absolute rest from the precipitating activity. As pain subsides, an exercise program should be instituted. If symptoms progress or fail to respond within 4 months, surgical intervention should be considered.[15]

Surgical Procedures

Surgical treatment of TOS is indicated in cases of acute vascular insufficiency, progressive neurologic dysfunction, and refractory pain with functional impairment that fails to improve with 4 months of conservative treatment.[15] Though several surgical procedures have been described, most involve surgical release of the anterior and middle scalene muscles, with or without first rib resection. In general, if a cervical rib is present, it also is excised. The presence of vascular abnormalities requires special surgical attention.

The transaxillary approach for first rib resec-

tion has been popularized by Roos.[14,38] Its main advantage is that the approach transects no major muscles and avoids damage to the scapular suspensory muscles.[7] It is advocated as the best operation for patients with lower trunk and vascular symptoms.[14] The procedure, which leaves an easily hidden axillary scar, involves lifting the vessels and nerves off the first rib while the arm is hyperabducted, and releasing the scalene muscles as the rib is removed. Postoperative disability tends to be minimal, though brachial plexus injuries caused by surgical positioning and excessive traction by the surgical assistant are reported. Most cases of operative brachial plexus injury resolve spontaneously, but up to 20 percent of affected patients encounter persistent deficits.[7] Pneumothorax is another potential surgical complication resulting from the proximity of the surgical dissection to the pulmonary pleura. Additionally, scapular winging develops in 15 percent of cases as a result of damage to the long thoracic nerve, which on occasion courses through the middle scalene muscle.[7] An advantage of this procedure is that all potential sites of thoracic outlet obstruction—except for the pectoralis minor tendon—are removed.

Anterior scalenectomy has been advocated as the procedure of choice for patients who have upper trunk symptoms.[14] This operation consists of scalene muscle exposure and resection through an anterior supraclavicular incision with its reduced risk of causing injury to the nerves and vessels. Cikrit and colleagues[2] reported fewer complications, decreased blood loss, and shorter postoperative hospitalization time with the supraclavicular approach versus the transaxillary procedure. They concluded that the transaxillary incision carries a substantial risk of damage to the subclavian vessels and brachial plexus.

Surgery should also be tailored to address specific abnormalities, such as excess clavicle callus from a previous fracture, which can be excised; a hypertrophic pectoralis minor muscle, which might require release; and fibromuscular bands in the region of the outlet, which should be resected. The posterior approach for first rib resection is generally avoided—except in cases that require vascular reconstruction—as it results in scapular suspensory muscle damage. Likewise, the posterior parascapular decompression procedure is no longer frequently performed.

Surgical Outcomes

Controversy continues about the appropriate surgical procedure for TOS. Postoperative outcome results for surgeries consisting of transaxillary first rib resections with excision of cervical rib and anomalous bands, if present, have been described in several surgical series. Most postoperative assessments are subjectively reported by the patient or surgeon, with differing durations of follow-up. Success rates are variably defined as good-to-excellent in 90 and 92 percent,[4,14] good in 80 percent,[3] complete in 79 percent,[17] successful in 43 and 52 percent,[5,39] and improvement in 60 percent.[40] Supraclavicular scalenectomy without rib resection was noted to provide substantial relief in all 93 patients who experienced upper plexus symptoms in one published series.[14]

In an attempt to better define the cause of compression symptoms of the thoracic outlet, Stallworth and Horne[41] performed hyperabduction and costoclavicular maneuvers during axillary exploration in 194 surgical procedures. The source of compression was found, and the appropriate tissue was divided or resected. The postoperative improvement rate was 96 percent. Most cases required only soft tissue muscle or accessory band division, and in only four cases was first rib excision considered necessary, leading the authors to conclude that first ribs are rarely responsible for compression symptoms. Lindgren and Oksala[5] showed that patients with preoperative nocturnal pain and neck pain had less favorable surgical outcomes after an average of 8 years.

Summary of Surgical Recommendations

The preferred procedure for patients with predominantly upper plexus symptoms, given its high reported success rate and low complication incidence, appears to be supraclavicular anterior scalenectomy with division of anomalous fibromuscular bands. If a cervical rib is found in a symptomatic patient, it additionally should be removed. Lower plexus symptoms were quite successfully treated with transaxillary first rib resection by Roos,[14] while others have reported less favorable outcomes with this procedure.[5,39,40] Yet other authors concluded that first rib resection is rarely necessary.[41] The presence of both "upper" and "lower plexus" symptoms might require extensive thoracic outlet decompression by way of synchronous supraclavicular and transaxillary ex-

posures for decompression. Hawkes[42] takes a slightly different approach, suggesting that the initial surgical procedure should be the safer supraclavicular anterior scalenectomy, including resection of any fibrous bands and release of the middle scalene muscle if the brachial plexus passes through the muscle. He advocates a second procedure to resect the first rib only if symptoms do not abate.

Broad operative exposure facilitates vascular reconstruction procedures. For subclavian venous thrombectomy, the supraclavicular approach is recommended, though peripheral thrombi could call for infraclavicular or transaxillary approaches. Subclavian arterial occlusion, which occurs secondary to bony compression, could require subclavian endarterectomy. Arterial reconstruction, when necessary, is best accomplished by a bypass from the subclavian to the axillary brachial artery using harvested vein as the preferred graft material. Synthetics should be avoided because the low flow through the prosthesis into high distal resistance could produce emboli, which predispose to early graft failure.[43]

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