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Neurovascular Compression in the Thoracic Outlet

Changing Management Over 50 Years

Harold C. Urschel, Jr., MD and Maruf A. Razzuk, MD

From the University of Texas Southwestern Medical School, Baylor University Medical Center, Dallas, Texas

Summary Background Data

During the past five decades, significant improvements have been made in the diagnosis and treatment of thoracic outlet syndrome (TOS) secondary to sports activities, breast implants, or median sternotomy.

Methods, Results, and Conclusions

Of more than 15,000 patients evaluated for TOS, 3914 underwent primary neurovascular decompression procedures and 1221 underwent second surgical procedures for recurrent symptoms. Of 2210 consecutive patients, 250 had symptoms of upper plexus compression only (median nerve), 1508 had symptoms of lower plexus compression only (ulnar nerve), and 452 patients had symptoms of both. Ulnar and median nerve conduction velocities confirmed the clinical diagnosis. Transaxillary first rib removal alone for neurovascular decompression relieved both upper and lower plexus symptoms (without a combined transaxillary and supraclavicular approach). There are two reasons for this: most upper compression mechanisms attach to the first rib, and the median nerve is also supplied by C8 and T1 as well as C5, C6, and C7 nerve roots.

Axillary subclavian artery aneurysm or occlusion was treated successfully in 240 patients. Dorsal sympathectomy was performed concomitantly in 71 patients for occlusion or embolectomy. It was combined with first rib resection in 1974 patients for sympathetic maintained pain syndrome and causalgia that did not improve with conservative therapy.

Of 264 patients with effort thrombosis (Paget-Schroetter syndrome), 211 were treated by urokinase thrombolysis and prompt first rib resection with excellent long-term results. Recurrent TOS symptoms required a second procedure using the posterior approach in 1221 patients with brachial plexus neurolysis and dorsal sympathectomy. The use of hyaluronic acid significantly reduced recurrent scarring.

Thoracic outlet syndrome (TOS) refers to compression of one or more of the neurovascular structures traversing the superior aperture of the chest. Previously, the name was designated according to the etiologies of compression, such as scalenus anticus, costoclavicular, hyperabduction, cervical rib, or first rib syndromes. Peet[1] coined the term "thoracic outlet syndrome" in 1956 to designate compression of the neurovascular bundle at the thoracic outlet.

Most compressive factors operate against the first rib and produce a variety of symptoms, depending on which neurovascular structures are compressed. The functional anatomy and pathophysiology of compression in the thoracic outlet and the symptomatology of each of the specific structures compressed is summarized in Figure 1.

During the past five decades, many changes in the recognition and management of TOS have evolved. The purpose of this paper is to elucidate these changes and improvements in the diagnosis and management of TOS. From 1947 to 1996, in our group practice of nine surgeons and three physiatrists, more than 15,000 patients were evaluated for TOS. Of these, 3914 underwent neurovascular decompression procedures and 1221 had second procedures for recurrent symptoms (these patients were primarily from other centers). Evaluation of these patients provides the basis for this report.

In the late 1950s and early 1960s, the surgical procedure of choice in our practice was the supraclavicular scalenotomy, partial scalenectomy with neurolysis of the brachial

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Address reprint requests to Harold C. Urschel Jr., MD, 3600 Gaston Ave., Dallas, TX 75246.
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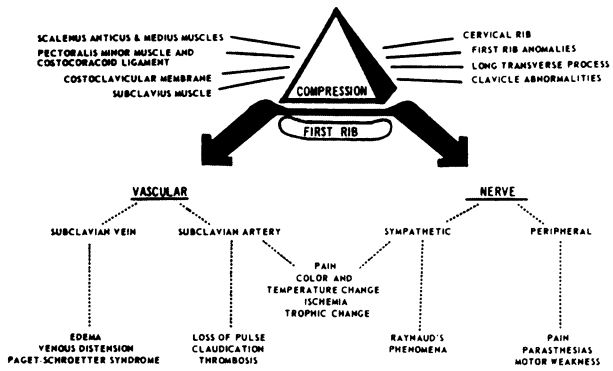

Figure 1. Compression factors in the thoracic outlet with potential signs and symptoms produced.

plexus when indicated and combined with resection of a cervical rib if present. Early results in 336 patients were extremely good (310/336). However, the longer-term follow-up was not as satisfactory. Five-year improvement was present in 150 of 336 patients, but at 20 years only 31 of 336 patients were still improved (20 patients were lost to follow-up). Because of this and because of the 1962 presentation by Clagett,[2] the posterior approach for resection of the first rib was used to remove the common denominator for thoracic outlet compression forces. Subsequently, the initial procedure was usually performed through the transaxillary approach because no muscle division was required and morbidity rates were less. The supraclavicular or infraclavicular approach or the combined approach was used for arterial lesions. The posterior approach is now reserved for second procedures in patients with recurrent TOS symptoms for removal of rib remnants and regenerated fibrocartilage with neurolysis of C7, C8, and T1 nerve roots and the brachial plexus.

NERVE COMPRESSION

The symptoms of nerve compression most frequently observed are pain and paresthesias (present in approximately 95% of patients) and motor weakness (<10%). Pain and paresthesias are segmental in 75% of cases, 90% involving the ulnar nerve distribution.[3]

TOS can occur in older patients (the oldest reported has been 87 years). However, when nerve compression symptoms occur in patients older than 60, causes such as degenerative or traumatic cervical spine, cardiac, or pulmonary pathologies should be suspected.

There may be multiple points of compression of the peripheral nerves between the cervical spine and hand, in addition to the thoracic outlet. When there are multiple compression sites, less pressure is required at each site to produce symptoms. Thus, a patient may have concomitant TOS, ulnar nerve compression at the elbow, and carpal tunnel syndrome. This phenomenon has been called the multiple crush syndrome.[4]

Pseudoangina

Pain is frequently insidious in onset, commonly involving the neck, shoulder, arm, and hand. In some patients, the pain is atypical, involving the anterior chest wall and parascapular area, and is termed pseudoangina because it simulates angina pectoris. A group of patients with chest pain simulating angina pectoris, but with normal coronary angiograms, were evaluated in 1973. Because the cardiologist was married to one of our physiatrists, this group of patients was referred for diagnostic studies to confirm the presence of TOS. When either medical or surgical therapy of TOS relieved the symptoms of pseudoangina, the diagnosis was considered confirmed.[5]

To explain chest pain from TOS compression, it is important to remember there are at least two types of pain pathways in the arm: the commonly acknowledged (C5 to T1) somatic fibers, which transmit more superficial pain, and the afferent sympathetic nerve fibers, which transmit deeper painful stimuli. The cell bodies of the two types of neurons are situated in the dorsal root ganglia of the corresponding spinal segments. They synapse in the dorsal gray matter of the spinal cord, and the axons of the second-order neurons ascend in the spinal cord up to the brain. Compression of the superficial C8 to T1 cutaneous afferent fibers elicits stimuli that are transmitted to the brain and are recognized as integumentary pain or paresthesias in the ulnar nerve distribution. In contrast, compression of the predominantly deeper sensory fibers elicits impulses that are appreciated by the brain as deep pain originating in the arm or the chest wall, even if the source of the impulses is cardiac (referred pain).

Diagnostic and Objective Tests

A careful history and physical examination are critical for accurate diagnosis. The multiple physical signs of thoracic outlet compression and the classic diagnostic tests have been thoroughly reviewed.[4,6] Other causes of TOS-like symptoms, such as cardiac or pulmonary disease, must be ruled out. The electromyogram should be normal, ruling out other neuromuscular disorders.

The primary objective test for thoracic outlet peripheral nerve compression in our clinic is the nerve conduction velocity (NCV).[6–9] Reduction in NCV to less than 85 m/s of either the ulnar or median nerves across the thoracic outlet corroborates the clinical diagnosis. More than 8000 NCV studies were performed each year at our medical center for many years, with approximately 2000 per year demonstrating TOS.[3,10,11]

Management

With an NCV exceeding 60 m/sec, the patient usually improves from conservative physical therapy. Excellent principles of conservative management have been outlined

by Novak and Mackinnon[10] and Caldwell and Crane.[8] Initially, most patients, except those with vascular problems, were treated conservatively with physical therapy. The primary goals of physical therapy are to open up the space between the clavicle and first rib, improve posture, strengthen the shoulder girdle, and loosen the neck muscles. This is accomplished by pectoralis stretching, strengthening the muscles between the shoulder blades, good posture, and active neck exercises, including chin tuck, flexion, rotation, lateral bending, and circumduction.

The usual indications for surgery are failure of appropriate conservative therapy in a patient with a significantly reduced NCV (<60 m/s; normal is 85 m/s) and the elimination of other possible etiologies for the symptoms.

Initial surgical therapy involves complete first rib resection, anterior scalenectomy, resection of the costoclavicular ligament, and neurolysis of C7, C8, and T1 nerve roots and the brachial plexus through a transaxillary approach.[12] The first rib with the compressive elements may also be removed using the supraclavicular approach.[13] This approach, however, requires working through and retracting the brachial plexus and produces a visible scar in women (the preponderant gender with TOS). The posterior thoracoplasty approach for first rib resection may be used for initial therapy, but it should be reserved for a second procedure and neurolysis of the brachial plexus.[14,15] Cervical ribs may be removed using any of the approaches described. Dorsal sympathectomy may also be performed with neurovascular decompression through any of the above incisions for sympathetic maintained pain syndrome (SMPS), reflex sympathetic dystrophy, causalgia, and Raynaud's phenomenon and disease.[15,16]

Upper Plexus Versus *Lower Plexus* Management

Most cases of neurologic TOS requiring surgery have been successfully managed with transaxillary resection of the first rib. However, for upper plexus (median nerve) compression, Roos and Stoney[16] wrote that transaxillary rib resection alone was not enough, and that it should be combined with the supraclavicular approach to achieve best results.

Upper plexus compression was initially described by Swank and Simeone[13] with symptoms secondary to C5, C6, and C7 nerve root compression. Sensory changes occurs primarily in the first three fingers, and muscle weakness or pain occurs in the anterior chest, triceps, deltoids, and parascapular muscle areas, as well as down the outer arm to the extensor muscles of the forearm. In contrast, lower plexus irritation involves C8 and T1 nerve root compression and includes sensory changes primarily in the fourth and fifth fingers, with muscle weakness or pain from the rhomboid and scapular muscles to the posterior axilla, down the ulnar distribution to the forearm, involving the elbow, flexors of the wrist, and intrinsic muscles of the hand. Roos,[16] Urschel and Razzuk,[17] Wood and Ellison[18] expanded the upper plexus symptoms to involve pain in the neck, face, mandible, and ear with occipital headaches. Wood and Ellison also noted dizziness, vertigo, and blurred vision in some patients with upper plexus lesions. In addition to these clinical symptoms and signs, median nerve conduction slowing indicates upper plexus compression, whereas ulnar nerve conduction slowing suggests lower plexus compression.

The rationale for why relief of upper plexus symptoms results from transaxillary first rib resection alone is based on several factors. Anatomic observations show that the median nerve, usually incriminated in upper plexus compression of C5, C6, and C7 nerve roots, also receives significant fibers from C8 and T1 nerve roots. In addition, most muscles and ligaments that compress the upper plexus attach to the first rib. Thus, removing the first rib, with release of all the muscles and ligaments involved in compression, theoretically should relieve upper plexus compression.

To assess the optimal management of upper plexus TOS, we reviewed 2210 primary procedures for TOS in 1988 patients, 222 undergoing bilateral transaxillary resections, over a period of 30 years.[17] Two hundred fifty procedures were performed for symptoms, signs, and NCVs showing median nerve or upper plexus compression only. Four hundred fifty-two were performed for compression of both the median and ulnar nerves or the combination of the upper plexus and lower plexus by symptoms, signs, and NCVs. One thousand fifty-eight procedures were performed for symptoms, signs, and NCVs demonstrating predominately ulnar nerve or lower plexus compression. This study showed that transaxillary first rib resection with anterior scalenectomy relieved symptoms of upper plexus (96%) and combined upper and lower plexus (95%) compression as well as it did for lower plexus compression (95%). Patients were followed at 3 weeks and 3 months, and yearly contacts were made by phone. Wood and Ellison[18] and Sanders[16] independently corroborated these findings.

ARTERIAL COMPRESSION

The diagnosis is suspected by the history, physical examination, and Doppler studies and is confirmed with arteriography.[19] Therapy for arterial compression depends on its degree of involvement:

- An asymptomatic patient with cervical or first rib arterial compression producing poststenotic dilatation of the axillary subclavian artery should undergo rib resection, preferably using the transaxillary approach, removing the ribs, both first and cervical, without resecting the artery. The dilatation usually returns toward normal after removal of compression.
- Patients with compression from the first or cervical rib producing aneurysm with or without thrombus should undergo rib resection and aneurysm excision with graft using the supraclavicular and infraclavicular combined

approach.

- Thrombosis of the axillary subclavian artery or distal emboli secondary to TOS compression should be treated with first rib resection, thrombectomy, embolectomy, arterial repair or replacement, and dorsal sympathectomy.

We successfully treated 151 patients with axillary subclavian artery aneurysm and 62 patients with occlusion. Dorsal sympathectomy was performed when indicated. The 62 bypass grafts were successful, with the exception of 3 that occluded, requiring a second surgical procedure, and 1 that could not prevent amputation because of the delay before the patient presented for therapy. No cerebrovascular accidents occurred.

SYMPATHETIC COMPRESSION

Compression of the sympathetic nerves in the thoracic outlet may occur alone or in combination with peripheral nerve and blood vessels. The sympathetics are intimately attached to the artery as well as adjacent to the bone. They may be compressed or irritated in primary or recurrent TOS.

Atypical chest pain (pseudoangina) simulates cardiac pain. Many arterial compressions result in more severe symptoms because of the additive or synergistic sympathetic stimulation. Trauma frequently is associated with SMPS or reflex sympathetic dystrophy.

For uncomplicated, nontraumatic TOS symptoms, usually first rib resection alone with neurovascular decompression relieves the sympathetic symptoms; dorsal sympathectomy is not required. However, if trauma is significant in the etiology, causalgia or SMPS is often present and concomitant dorsal sympathectomy is routinely required to ameliorate the symptoms. Also, if surgery is required for recurrent TOS symptoms, the relief of accompanying causalgia usually requires dorsal sympathectomy. Initially, dorsal sympathectomies were performed at an interval after procedures for traumatic or recurrent TOS, if necessary. However, because they were necessary in so many cases and because of the inconvenience of a second procedure, dorsal sympathectomy is now routinely combined with the initial TOS procedure for either trauma or recurrence cases.[20,21]

Major indications for dorsal sympathectomy include hyperhidrosis, Raynaud's phenomenon or disease, causalgia, SMPS, reflex sympathetic dystrophy, and vascular insufficiency of the upper extremity. Except for hyperhidrosis, most indications for sympathectomy require the usual diagnostic techniques, including cervical sympathetic block, to assess the relief of symptoms with temporary sympathetic blockade. When Raynaud's phenomenon of a minor to moderate degree is associated with TOS, the simple removal of the first rib with any cervical rib, in addition to stripping the axillary subclavian artery (neurectomy), generally relieves most symptoms after the initial procedure.[11] It is rarely necessary to perform a sympathectomy unless Raynaud's phenomenon is severe, in which case a dorsal sympathectomy is performed with first rib resection. The only contraindication to dorsal sympathectomy is venous obstruction (Paget-Schroetter syndrome [PSS]; effort thrombosis of the axillary subclavian vein).

Surgical Approaches for Dorsal Sympathectomy

Historically, the anterior cervical approach to the cervical sympathetic chain has been used.[22] The stellate ganglion lies on the transverse process of C6, and this approach is used primarily by neurosurgeons and vascular surgeons. For hypertension, Smithwick and Urschel[23] popularized the posterior approach, with a longitudinal paraspinal incision with the patient in the prone position. Small pieces of the second ribs are removed, and the sympathetic chain is identified in the usual position. This approach has the advantage of allowing bilateral procedures to be performed at the same time without changing the patient's position. The most common current approach is the transaxillary, transthoracic approach, which is performed through the second interspace with a transverse subhairline incision.[24] This is more painful than the other approaches, but with video-assisted thoracoscopy it can be performed with minimal discomfort.[21]

The approach most frequently used for TOS and dorsal sympathectomy is the transaxillary approach, for first rib resection.[20,25] This causes minimal pain and combines two procedures with a low morbidity rate. Video assistance is also used frequently with this approach.

We have performed dorsal sympathectomy in 2214 extremities; 1974 of these were associated with neurologic TOS, causalgia, and SMPS. Two hundred forty procedures were associated with arterial complications of TOS. In the patients with neurologic TOS, 1221 dorsal sympathectomies were related to recurrent disease and the need for a second procedure.

If symptoms recur, they happen, on average, in 3 years (range 6 months to 25 years). In 46 patients, symptoms of sympathetic activity were apparent in less than 6 months. This is most likely from sprouting, or failure to strip the artery of its sympathetic nerves. This complication seems to occur less often if the bed of the sympathetic chain is cauterized after dorsal sympathectomy. It can also be explained by high circulating concentrations of catecholamines. The postsympathetic syndrome was observed in 39 extremities. This complication involves excessive postsurgical pain (as long as 6 months) in several nerve root distributions of the involved extremity and may be the result of injury to the somatic nerve. Unexpected Horner's syndrome was noted in 27 cases; 21 of these were transient and gradually resolved.

VENOUS COMPRESSION

Effort thrombosis of the axillary subclavian vein (PSS) is usually secondary to unusual, repetitive use of the arm, in addition to the presence of one or more compressive elements in the thoracic outlet. Sir James Paget[26] in 1875 in London and Von Schroetter[27] in 1884 in Vienna independently described this syndrome of thrombosis of the axillary subclavian vein. The word "effort" was added to thrombosis because of the frequent association with exertion producing either direct or indirect compression of the vein. The thrombosis is caused by trauma or repetitive muscular activity (*e.g.*, occupations such as professional athletes, linotype operators, painters, and beauticians). Cold and traumatic factors, such as carrying skis over the shoulder, tend to increase the proclivity for thrombosis. Elements of increased thrombogenicity also increase the incidence of this problem and exacerbate its symptoms on a long-term basis.[28,29] The diagnosis is suspected by a careful history and physical examination and Doppler studies, and confirmed with venography.

Intermittent or partial obstruction should be treated by first rib removal through the transaxillary approach, with resection of the costoclavicular ligament medially, the first rib inferiorly, and the scalenus anticus muscle laterally. The clavicle is left in place. The vein is decompressed and all the bands and adhesions are removed.

For many years, complete thrombosis (PSS) was treated by elevation of the arm and the use of anticoagulants, with subsequent return to work. If symptoms recurred, a first rib resection, with or without thrombectomy, was considered, as well as resection of the scalenus anticus muscle and removal of any other compressive element in the thoracic outlet, such as the costoclavicular ligament, cervical rib, or abnormal bands.[19] We treated 36 patients by this approach; only 10 had a good to excellent result.

The availability of thrombolytic agents, combined with prompt surgical decompression of the neurovascular compressive elements in the thoracic outlet, has reduced the morbidity rate and the need for thrombectomy and has produced substantially improved clinical results, including the ability to return to work.[29–31] Through an antecubital indwelling catheter, venography is performed and thrombolytic therapy is initiated. After lysis of the clot, prompt first rib resection with removal of compressive elements is performed. We have used this technique in 240 patients with PSS. Thrombectomy was necessary in only four extremities, and the long-term results indicate that 205 extremities had good results (the patient returned to work without symptoms). Twenty-four patients had fair results (intermittent swelling but able to work) and 11 patients had poor results (chronic swelling). Seven of the poor results occurred in the 35 patients seen initially more than 3 months after the thrombotic episode. No patient had phlegmasia cerulea dolens. There were no deaths. These results were in marked contrast to those of 35 patients treated with only anticoagulants: 10 good results, 16 fair results, and 9 poor results.[29] Delay in initiating thrombolytic therapy or the prolonged use of coumadin or heparin is contraindicated. There were no obvious complications of thrombolytic therapy. With a prolonged interval between venous occlusion and diagnosis (>3 months), the same management produced less favorable results. Attempts to open the occluded vein mechanically with the use of laser or bypass grafts have uniformly been unsatisfactory.

Claviculectomy is occasionally used for decompression, particularly if a fracture of the clavicle has occurred secondary to trauma.[22]

RECURRENT THORACIC OUTLET SYNDROME

Recurrent symptoms, primarily neurogenic, should be documented by objective NCVs. When NCVs are depressed in a patient whose symptoms are unrelieved by prolonged conservative therapy, a posterior procedure should be considered. Removal of any rib remnants or regenerated fibrocartilage and neurolysis of C7, C8, and T1 nerve roots and the brachial plexus are performed.[15] Dorsal sympathectomy is added to minimize the contribution of causalgia to symptoms. Depo-Medrol and hyaluronic acid are used to minimize recurrent scar.[32]

Two distinct groups of patients require a second procedure: those with pseudorecurrence and those with true recurrence. Pseudorecurrences were observed in 43 patients, all referred from other surgeons; these patients were never completely relieved of their symptoms after the initial procedure. They were separated into the following etiologies: mistaken resection of the second rib instead of the first (22 patients), resection of the first rib with a cervical rib left in place (11 patients), resection of a cervical rib with an abnormal first rib remaining (8 patients), and resection of a second rib with a rudimentary first rib left (2 patients). True recurrences occurred in 1221 extremities; some of these patients had relief of symptoms after the initial procedure, but the symptoms recurred 4 months to 18 years later.

The diagnosis and differential diagnosis for recurrence are similar to those for the original procedure. However, the indications for a second procedure are more stringent in that longer periods of conservative therapy are usually involved.

In this group, a substantial posterior stump (>1 cm) of the first rib remained in 1060 patients (all referred from outside physicians). Complete resection of the first rib at the initial procedure was observed in 161 patients who had recurrent symptoms associated with excessive scar formation on the brachial plexus. We performed the original procedure on 98 of these patients, for a reoperation rate of 2.5% (3914 primary procedures). Even though some of our patients did not return to us for recurrent symptoms, this rate is much less than in most series.

Few other surgeons remove the rib completely at the initial procedure for fear of injuring T1 or C8 nerve roots.

Some cover the end of the rib at the transverse process of the vertebra with scalenus medius muscle.

Results of 1221 procedures showed a moderately good early effect of a second procedure: 1092 patients had significant improvement (89%), 93 related fair improvement, and only 36 did not feel better. Late results (5-year follow-up) in 528 extremities that underwent a second procedure revealed 396 (75%) with good results and 132 (25%) with fair to poor recovery; 48 patients (3.1%) required a third surgical procedure.

The primary technical factors involved in recurrence seem to be complete extirpation of the rib during the first procedure. If a rib remnant is left (as most surgeons outside of our group do), osteocytes grow from the end of the bone and produce fibrocartilage and regenerated bone that compresses the nerves. The risk of fibrosis may be higher in patients who produce keloids, patients in whom hematomas are not drained, or patients who undergo early excessive physical therapy after the first surgical procedure. Occasionally other approaches have been used for a second procedure.[33]

Recurrent Arterial Abnormalities

Five patients referred from other physicians (two with false aneurysms, one with a mycotic aneurysm allegedly secondary to trauma at the initial procedure, and two with obstructive arterial changes at the thoracic outlet) underwent successful second procedures. We performed vascular reconstructive procedures. In each patient, a saphenous vein bypass graft from the innominate or carotid artery proximally was connected to the brachial artery distally. In the patient with the mycotic aneurysm, the graft was placed first and the vessels on each side of the aneurysm were ligated. The aneurysm was resected at an interval procedure.

MORTALITY AND MORBIDITY RATES

There were no deaths in our series of 3914 primary and 1221 repeat TOS decompressive procedures. The major complication resulted from a rib remnant left by the initial surgeon that regenerated fibrocartilage and new bone, producing a high incidence of recurrence. More retractor help (two-arm holders) and increased light improved the technique and facilitated the initial procedure. This minimized the time of anesthesia, surgery, retractor use, and arm holding.

The pleura is opened during most procedures (with the exception of pleurodesis) to provide drainage of blood and fluids, reducing recurrence. Bleeding requiring a second procedure occurred after only 3 of 5008 procedures. Significant infection requiring drainage occurred after nine procedures. There were no significant arterial injuries, and there was only one case of venous bleeding after surgery that required thoracotomy and repair. Venous injuries usually "suck" air. PSS is usually associated with severe inflammation, obliterating the vein and removing the identifying blue color. The axillary structures are usually plastered firmly to the chest wall, making the procedure technically difficult.

Significant nerve injuries of the brachial plexus with residual signs occurred in four patients; in none of these could prolonged stretching, inappropriate retraction, or direct surgical injury be identified. Two cases occurred in patients with diabetes and two in older patients with markedly prolonged NCVs, suggesting an increased sensitivity to "nerve pressure" similar to that observed in persons with diabetes.

Dale's[34] review of morbidity rates in 881 patients revealed significant bleeding in 11 (1.4%) and nerve injury of the brachial plexus in 13 (1.5%), the phrenic in 39 (4.9%), the long thoracic in 3 (0.1%), and the recurrent laryngeal in 6 (0.2%). In another 168 patients reported,[35] the phrenic nerve was injured in 6 (4%) and the long thoracic and recurrent laryngeal nerve in 1 (0.5%); Horner's syndrome occurred in 9 (6%). Long-term studies have been reported,[36,37] as well as other complications.[38]

CONCLUSIONS

The past 50 years have seen improved recognition and management of TOS. The most remarkable change is the use of NCV to diagnose and follow patients with nerve compression. Recognition that such procedures as breast implantation and median sternotomy may produce TOS has been revealing. Prompt thrombolysis followed by surgical venous decompression for PSS has markedly improved results compared with the conservative anticoagulation approach; thrombolysis and prompt first rib resection is the optimal treatment for most patients with PSS. Complete first rib extirpation at the initial procedure markedly reduces the incidence of recurrent neurologic symptoms or the need for a second procedure. Chest pain or pseudoangina can be caused by TOS. Dorsal sympathectomy is helpful for patients with SMPS or causalgia and patients with recurrent TOS symptoms who need a second procedure.

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Discussion

DR. SUSAN E. MACKINNON (St. Louis, Missouri): Dr. Urschel is internationally recognized as an advocate for the patient with thoracic outlet syndrome. The authors are to be congratulated on their outstanding results in a series of patients, the numbers of which exceed the cumulative experience published in the literature. Recognizing a frequent association in the typical TOS patient with distal nerve entrapments at the wrist and elbow associated with proximal muscle imbalance, our group has reserved surgical intervention for the unusual patient who cannot be treated with either distal extremity surgeries or a specific physical exercises. Management of the true neurogenic and vascular TOS symptoms is relatively straightforward.

It is the much larger group of patients with subjective complaints of numbness and pain that present the controversy, especially with respect to surgical intervention for a syndrome subjective in nature and one that lacks any reliable test to substantiate the diagnosis. In this patient group the reportedly high incidence of major neurological complications has influenced many surgeons toward conservative management. In this series, only 0.0008 percent of patients, that is 4 of approximately 5000 patients, suffered any significant neurological injury. This is a remarkable accomplishment as compared to the 6% to 10% of similar injuries published in other series and attests to the importance of fine surgical technique. The controversy with surgery for TOS relates predominantly to the ratio of benefit of relief of subjective symptoms against the potential for life-altering neurological complications. For the vast majority of patients, assessment of postoperative improvement is purely subjective. Therefore, I would like to ask Dr. Urschel for the details of his follow-up regimen and outcome measurements.

Secondly, recognizing that many surgeons in several disciplines will not evaluate and treat these patients for fear of litigation, I would like Dr. Urschel to comment on the fact that this operation is the most litigated procedure in surgery. How do you minimize postoperative patient dissatisfaction? How do you identify the potential litigious patient? And is your evaluation of a patient in litigation different from those seen for primary surgeries? Dr. Urschel, I congratulate you on your contributions to this often ignored problem.

DR. HAROLD URSCHEL, JR. (Dallas, Texas): Initially our follow-up is 3 weeks and 3 months in the office. Subsequently, yearly calls are made to the patients to find out whether their quality of life is satisfactory, whether they have been able to return to work, how much recreation can they do, and if they have had another operation. Having Physical Medicine and Pain Management physicians who care about these patients assists in making the diagnosis and helps with the management postoperatively. Heavy laborers don't do as well as housewives, who usually are relieved