THE THORACIC OUTLET SYNDROMES: Part 2. The Arterial, Venous, Neurovascular, and Disputed Thoracic Outlet Syndromes

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Abbreviations: AHC, anterior horn cell; AP, action potential; APR, anterior primary rami; A-TOS, arterial thoracic outlet syndrome; CT, computerized tomography; CTS, carpal tunnel syndrome; DRG, dorsal root ganglion; EDX, electrodiagnostic; EMG, electromyography; FTR, first thoracic rib; LABC, lateral antebrachial cutaneous; MABC, medial antebrachial cutaneous; Med-D1, median sensory response recording from the thumb; Med-D2, median sensory response recording from the index finger; MR, magnetic resonance; MRI, magnetic resonance imaging; MRN, magnetic resonance neurography; MUAP, motor unit action potential; NCS, nerve conduction study; PNS, peripheral nervous system; SR-DH, superficial radial sensory response recording dorsum of hand; TN-TOS, true neurogenic thoracic outlet syndrome; TOS, thoracic

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outlet syndrome; Uln-D5, ulnar sensory response recording from the little finger; V-TOS, venous thoracic outlet syndrome.

Key Words: cervical rib, fibrous band, rib and band syndrome, thoracic outlet, thoracic outlet syndrome, brachial plexus, lower trunk, subclavian artery, subclavian vein

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ABSTRACT: The thoracic outlet syndromes (TOSs) are a group of etiologically and clinically distinct disorders with 1 feature in common: compression of 1 or more neurovascular elements as they traverse the thoracic outlet. The medical literature reflects 5 TOSs: arterial, venous, traumatic neurovascular, true neurogenic, and disputed. Of these, the first 4 demonstrate all of the features expected of a syndrome, whereas disputed TOS does not, causing many experts doubt its existence altogether. Thus, some categorize disputed TOS as cervicoscapular pain syndrome rather than as a type of TOS. To better understand these disorders, their distinctions, and the reasoning underlying the categorical change of disputed TOS from a form of TOS to a cervicoscapular pain syndrome, a thorough understanding of the pertinent anatomy, pathology, pathophysiology, and the electrodiagnostic manifestations of these pathophysiologies is required. This review of the TOSs is provided in two parts. The first part covered general information pertinent to all 5 TOSs and reviewed true neurogenic TOS in detail.[1] Part 2 reviews the arterial, venous, traumatic neurovascular, and disputed forms of TOS.

INTRODUCTION

The thoracic outlet syndromes (TOSs) are a diverse group of etiologically and clinically distinct disorders with 1 feature in common, namely, the compression of 1 or more neurovascular elements as they traverse the thoracic outlet.[1] The current medical literature considers 5 entities as TOSs: arterial TOS (A-TOS), venous TOS (V-TOS), traumatic neurovascular TOS, true neurogenic TOS (TN-TOS), and disputed TOS.[2] Of these 5 disorders, the first 4 demonstrate all of the features expected of a syndrome, including: (1) an anatomical abnormality, (2) a pathogenesis related to that anatomical abnormality, (3) clinical features consistent with that anatomical abnormality, (4) a diagnostic test that identifies that anatomical abnormality, and (5) a treatment that addresses that underlying anatomic abnormality. Conversely, disputed TOS lacks a consistent anatomical abnormality, a recognized pathogenesis, consistent clinical features, a reliable method of testing, and an agreed upon treatment. Consequently, many experts doubt its existence altogether. In 1984, Wilbourn introduced the term "disputed TOS" to identify this form of TOS.[3] As will be proposed in this manuscript, disputed TOS is better considered a cervicoscapular pain syndrome rather than a type of TOS.

To better understand these disorders and their distinctions, a thorough understanding of the pertinent anatomic, pathologic, pathophysiologic, and electrodiagnostic (EDX) manifestations of these diverse disorders is required. Thoracic outlet anatomy and the pathologic, pathophysiologic, and EDX manifestations of nerve fiber injury were discussed in part 1 of this review, which also covered true neurogenic TOS (TN-TOS)[1]. Part 2 focuses on the arterial, venous, traumatic neurovascular, and disputed forms of TOS.

ARTERIAL TOS

A-TOS is a rare disorder that involves subclavian artery compression and affects individuals of all ages and both genders. Like TN-TOS, it is almost always unilateral and more commonly involves young adults.[1,2,4,5] Responsible etiologies include a large bony anomaly, a deformed first thoracic rib (FTR), another osseous process, anterior or middle scalene muscle hypertrophy, or intramuscular fibrous bands.[6-9] Of these, a fully formed cervical rib is the most common cause.[10] Vessel compression may result in intimal damage, turbulent blood flow, thrombus formation, distal embolization, aneurysm formation, poststenotic dilatation, or effort-related symptoms of claudication.

Clinical Features. The clinical features of A-TOS, which were initially published by Sir Astley Cooper in 1821, are divided into vascular and neurologic.[11] Subclavian artery compression leads to features of chronic upper extremity ischemia, including pain with effort, easy fatigability, claudication, extremity coolness, pallor, decreased capillary refill, an audible bruit, and diminution or absence of distal pulsations.[3,9,10,12] Pulse diminution may not be recognizable unless the extremity is elevated into a position that compresses the artery.[13] A painless, pulsatile mass may be associated with subclavian artery aneurysms.[14] Less frequently, subclavian artery thrombosis results in downstream embolic events manifested by digital ulcerations or the abrupt onset of severe limb ischemia distal to the occlusion. In addition, cervical rib-induced subclavian artery damage may generate emboli that enter the vertebral or carotid artery circulation, causing ischemic brain infarction.[15,16] With more severe ischemia, hand pain occurs at rest.[9] Tissue necrosis from unrecognized ischemia may necessitate amputation. The neurologic features are secondary to the vascular abnormalities.

Evaluation. The diagnosis of A-TOS relies predominantly on radiologic studies, including plain radiography, Doppler ultrasonography, computerized tomography (CT) angiography, magnetic resonance (MR) angiography, and conventional angiography; the latter study is also used for surgical planning. The seated position (as opposed to supine) and the use of various upper extremity postures may be necessary to identify dynamic circulatory disturbances.[17] Plain films or CT scanning of the cervical spine may show bony abnormalities ipsilateral to the symptomatic limb, such as a cervical rib, a prominent C7 transverse process, or an abnormal FTR. Of these, the most common bony abnormality is a cervical rib (85% of patients in one series).[9] Conventional arteriography of the subclavian artery, introduced by Lang in 1962, identifies vascular compression or occlusion and other vascular abnormalities (poststenotic aneurysms or mural thrombi) and dynamic imaging of various upper extremity positions reveals the relationship between the bony abnormality and the vascular compression.[9,18] Due to its invasive nature, the diagnostic role of conventional arteriography has decreased, and less invasive procedures are typically utilized. Doppler ultrasound (duplex) may detect vessel narrowing, vessel occlusion, arterial thrombosis, or poststenotic aneurysms.[9] CT angiography generates a more detailed assessment of the vascular abnormalities.[9] When these procedures demonstrate reduced arterial blood flow, they are confirmatory. However, an indented or angulated subclavian artery without associated flow reduction is nonspecific and seen in patients with other disorders.[19]

Treatment and Prognosis. To avoid loss of all or part of the upper extremity, patients who present with acute arterial compromise must be promptly recognized and treated to restore distal arterial flow. Surgical decompression of the vessel (removal of the responsible compressive structure) was first performed by Coote in 1861[20]; additional structures may also be removed

(e.g., scalenectomy). When an aneurysm is identified, repair is required. When a thrombosed aneurysm or artery is noted, a bypass procedure to restore blood flow is best. The surgical approach is best tailored to the procedure. When subclavian artery reconstruction is required (repair or resection with either end-to-end anastomosis or bypass grafting), a supraclavicular approach gives the best visibility and provides excellent exposure.[9,14,21-25] When cervical rib and FTR resection are planned, a transaxillary allows both ribs to be resected using a single incision[14] although a dual approach may be necessary in some cases. The prognosis is typically determined by the vascular surgeon and reflects the underlying etiology, the severity of the vascular damage, and the promptness of its recognition.

VENOUS TOS

V-TOS is a relatively rare disorder, with a reported incidence of about 1-11 per 100,000,[26-28] that is associated with venous thrombosis involving the subclavian-axillary veins. In 1948, Hughes coined the eponymic moniker "Paget-Schroetter syndrome" to acknowledge its original descriptor, Sir James Paget,[29] and the theory of its pathogenesis, put forth by von Schroetter, that upper extremity muscle stretch and strain precipitate spontaneous thrombosis.[30] It is also referred to as "effort thrombosis" because of its association with repetitive upper extremity activities. V-TOS is a unilateral disorder that occurs more commonly among men and more frequently involves the dominant limb,[2,31] likely related to the preference of the dominant limb for performing acts requiring only a single limb (e.g., pitching). Accordingly, it typically occurs among young, able-bodied adults, particularly athletes, manual laborers, and overhead workers following vigorous activity.[5,9,32] Among athletes, the performance of repetitive activities that constrict the thoracic outlet (e.g., upper extremity hyperabduction and extension as occurs in

baseball players and swimmers) increases the risk.[33] The subclavian vein may be affected by space-occupying structures within the costoclavicular space (e.g., osseous exostoses, callus from prior fracture, fibrous band, anterior scalene muscle or tendon hypertrophy, subclavius or pectoralis minor muscle or tendon hypertrophy, costoclavicular or coracocostal ligament hypertrophy, or tumor).[34] Cervical ribs are infrequently responsible.[35] Venous hypertension may follow excessive physical activity in the setting of a preexisting compressive process (e.g., muscle hypertrophy). Other contributory mechanisms include perivenular inflammation leading to fibrosis and limiting subclavian vein mobility, and repetitive venous endothelial trauma from injuries related to upper extremity movement. With progressive venous damage, fibroelastic stricture formation and turbulent blood flow occur, further narrowing the venous system and predisposing to the development of a thrombosis.[31,32] Conditions that produce venous stasis or hypercoagulability may also be responsible.

Clinical Features. The clinical features of V-TOS were first published by Paget.[29] Most commonly, the presentation is sudden in onset and involves young, healthy individuals following prolonged upper extremity exertion (hence, the term "effort thrombosis"). With incomplete occlusion, patients may be asymptomatic, oligosymptomatic, or only become symptomatic with upper extremity orientations that worsen venous flow.[36,37] With acute occlusion, the clinical manifestations are readily apparent, including diffuse upper extremity swelling, palpable clotted axillary veins, variable degrees of cyanosis, and pain. With chronic thrombosis and venous collateral formation, dilated veins appear in the neck, upper chest, and shoulder areas.[38-40] Like A-TOS, the neurologic features reflect the vascular changes induced by the primary lesion rather than direct neural injury. Among individuals who perform repetitive activities that constrict the thoracic outlet (e.g., baseball players and swimmers) and have no identifiable structural

abnormalities to account for extrinsic compression, an underlying thrombotic disorder must be considered.

Evaluation. The diagnosis of V-TOS is primarily established by a combination of clinical and imaging features. Early diagnosis and treatment are associated with better outcomes[32,41] Because none of the clinical features are specific, imaging studies are mandatory.[32] Because of its high sensitivity and specificity, venous ultrasonography (with color duplex imaging) is the initial study of choice. Depending on the historical features, a dynamic study utilizing the limb position initially precipitating the symptoms may be necessary to identify the occlusion. Acutely, when the clot is echolucent, the key finding is lack of venous compressibility.[42] CT and CT venography are useful when ultrasonography is unrevealing, but they are associated with radiation exposure and bone-related image degradation. Therefore, although expensive and time consuming, the lack of radiation and bone-related image degradation, as well as its multiplanar imaging and soft tissue differentiating abilities and its noninvasiveness make contrast-enhanced magnetic resonance imaging (MRI) with MR venography the most helpful radiologic study for defining the extent and chronicity of the thrombosis, the site and cause of the compression, and the presence of venous collaterals.[43] The diagnostic utility of contrast venography includes its ability to define the vascular anatomy, extent of the thrombosis, and the presence of collaterals. In addition, by defining the degree of compression associated with different limb positions, positional venography (as well as postural CT and MR venography) may more accurately reflect the site, severity, and hemodynamic effects of the underlying compressive lesion.[31, 44, 45] Ultrasonography also assesses the vascular consequences of various limb postures. However, subclavian vein compression occurs among normal individuals when the limb is positioned in extremes of abduction and external rotation.[34] In addition to its diagnostic utility, contrast

venography has therapeutic utility (thrombolysis and angioplasty, discussed below). Using the cephalic vein for contrast injection may produce falsely negative studies.[32] Because of the higher incidence of prothrombotic disorders among individuals with spontaneous upper extremity deep venous thrombosis, patients with V-TOS should be screened for prothrombotic disorders,[32] especially those individuals without an identifiable structural abnormality or with recurrent disease.[33]

Treatment and Prognosis. The goals of treatment include symptom resolution (bed rest with limb elevation, warm compresses, and analgesics), blood flow restoration (thrombolysis; stent placement), and recurrence prevention (address underlying cause). The optimal treatment is unclear because the rarity of V-TOS precludes randomized, controlled trials. Hence, the therapeutic regimen must be individualized. Typically, anticoagulation is started once the disorder is suspected and, following confirmatory testing, thrombolysis is provided. Anticoagulation alone is generally not recommended because of its association with lower recanalization rates; higher rates of residual thrombosis, pulmonary embolism, and recurrent thrombosis; and poorer outcomes.[32,35] Rather, following thrombus identification, catheter-directed intravascular thrombolysis is emergently initiated to reestablish venous patency. This also lowers the complication rate (e.g., pulmonary embolism), thereby reducing patient morbidity and mortality. Thrombolysis is most successful when started within 1 week of presentation[46] and is usually unsuccessful when started after 14 days, although it can still be attempted.[33] Although adjunctive mechanical thrombectomy may be performed, because thrombus formation almost invariably recurs, it is considered ineffective. Decompressive surgery is usually recommended in the setting of residual thrombosis or when venous compression is due to an extrinsic structural process. Although some authors prefer early surgical intervention,[47]

others advocate a delay to optimize patient selection and avoid unnecessary surgery.[45,47] Because of the unyielding nature of most of the compressive abnormalities, stenting is ineffective and associated with recurrent thrombosis.[33,48] Surgical intervention is followed by long-term anticoagulation (3-12 months).[9,32,33,49-53] Although serial ultrasonography is often used to monitor the lesion site, identified changes typically do not alter management in an asymptomatic individual.[33] Following thrombolysis, percutaneous venoplasty may be required.[54]

Because the complication rates of decompressive surgery such as major bleeding and brachial plexus injury are high, nonoperative approaches continue to be investigated. A recent retrospective study reported that 23 of 27 patients (85%) followed for a mean time of 53.4 months were asymptomatic following nonoperative intervention by catheter-directed thrombolysis followed by long-term anticoagulation).[33] In that study, factors suggesting a poor outcome included identifiable structural abnormalities, a long duration of symptoms prior to diagnosis, symptom persistence, and symptom recurrence. Therefore, some patients with V-TOS such as those with hypercoagulable disorders without a demonstrable compressive lesion may not need decompressive surgery, whereas those with the previously-mentioned risk factors likely will. If patients present in the subacute or chronic time period and decompressive surgery (with or without venoplasty) is planned, thrombolysis may not be necessary.[55] Following decompressive surgery, long-term anticoagulation is started. The prognosis is typically determined by the vascular surgeon and reflects the severity of the vascular damage, the underlying etiology, and the promptness of its recognition.

TRAUMATIC NEUROVASCULAR TOS

Traumatic neurovascular TOS is a rare disorder that follows clavicular trauma, usually a remote midshaft fracture. Therefore, it is usually unilateral and more common among adult men.[56-58] Unlike the other TOSs, which involve either neural or vascular elements, traumatic neurovascular TOS usually involves both. When only one system is involved, it is usually the neural system. The presentation may be delayed with isolated neural involvement.[4]

In general, with traumatic neurovascular TOS, the clavicular fracture is primary and the neural and vascular features are secondary. With severe enough trauma, however, the clavicular, neural, and vascular injuries may all be primary, in which case the mode of brachial plexus injury is usually supraclavicular traction.[59,60] Less commonly, the clavicular fracture secondarily causes the neural or vascular injuries. The mechanisms of secondary neurovascular injury in the acute setting are: (1) neurovascular compression or laceration by displaced fracture fragments, (2) vascular laceration with secondary nerve compression related to an expanding hematoma or an aneurysm (pseudoaneurysm), and (3) iatrogenic injury (during initial fracture manipulation or from a figure-of-eight bandage that is secured too tightly). Chronic mechanisms include clavicular motion related to nonunion and delayed neurovascular injury related to excessive callus formation.[61]

Clinical Features. The clinical features reflect the neurovascular elements most susceptible to trauma with clavicular fractures, namely the cords of the brachial plexus (especially the medial cord) and the proximal portions of the axillary vessels. Among the cord elements, the medial cord is more susceptible because it crosses the FTR directly behind the middle segment of the clavicle.[56,62,63] Acutely, the most common clinical feature is pain at the trauma site, frequently radiating into the upper extremity. Other clinical features at the trauma site include focal

tenderness, swelling, bruising, excessive clavicular mobility (with complete clavicular fracture, the lateral portion tends to move posteriorly and inferiorly with respect to the medial fragment), a palpable mass (hematoma), and an audible bruit (pseudoaneurysm formation). The vascular features observed with A-TOS and V-TOS, such as upper extremity edema (with venous compromise) or diminution or loss of arterial pulsation (with arterial compromise), may also be present. With more chronic presentations, a palpable callus may be appreciable.

The neural features are straightforward and primarily reflect the cutaneous and muscle domains of the medial cord. Thus, because the medial cord contains sensory nerve fibers destined to enter the medial brachial, medial antebrachial, and ulnar nerves, loss of sensation involves the medial aspects of the arm, forearm, and hand, as well as the medial ring and little fingers. The lateral aspect of the ring finger and, less frequently, the medial aspect of the middle finger, may be involved when the medial cord contains sensory fibers innervating these digits.[64] Because the medial cord contains motor fibers derived from the C8 and T1 spinal cord levels that ultimately contribute the median and ulnar nerves, weakness may involve any ulnar nerve-innervated muscle and any median nerve-innervated muscle except those receiving their innervation via the lateral cord (pronator teres; flexor carpi radialis). Because the C8-derived radial motor fibers traverse the posterior cord, they are spared with medial cord lesions.[64] When the etiology also affects the lateral cord or posterior cord, the distribution of the sensory and motor abnormalities expands to include their sensorimotor domains.[13,57,60,62,64]

Differential Diagnosis. The differential diagnosis of traumatic neurovascular TOS depends on the clinical features present at the trauma site, the neural and vascular elements involved, and the timing of the presentation. When the clavicular trauma is recognized, the sensory and motor

abnormalities are more easily localized to the brachial plexus. When unrecognized, the neural abnormalities may be mislocalized proximally as radiculopathies or distally as neuropathies. The most common sites of mislocalization are the C8 root, T1 root, and ulnar nerve with medial cord involvement; the C7 root and radial nerve with posterior cord involvement; and the C5 root, C6 root, and median nerve with lateral cord involvement. Localization to the thoracic outlet is more obvious when both neural and vascular elements are simultaneously affected. When the inciting trauma also involves the spinal cord, mislocalization solely to the brachial plexus may result in mismanagement.

Evaluation. The evaluation of traumatic neurovascular TOS is straightforward. Plain films of the chest and clavicle identify the clavicular damage (fracture; exuberant callus) and vascular imaging procedures (arteriography; venography) delineate the vascular abnormalities. Axial views provide a more accurate view of the clavicle than anteroposterior views.[57] Thrombosed pseudoaneurysms are detectable by CT scanning and MRI.[13] By localizing and characterizing the lesion, EDX testing contributes to both clinical management and prognostication.[57,64] It is best performed after day 21, although the NCSs can be performed earlier, when required. The motor NCSs, which best estimate the severity of the lesion, should be performed after day 6 and the sensory NCSs, which best localize the lesion, should be performed after day 10. Although ulnar somatosensory evoked responses may be abnormal, this technique is much less sensitive to focal axon loss than routine EDX testing and, moreover, neither localizes nor characterizes the lesion.[65]

Treatment and Prognosis. The appropriate management, although dictated by a number of factors, primarily reflects lesion severity and the specific vascular, clavicular, and neural injuries.

The vascular injuries typically dictate the immediate management. Surgical intervention is usually required, with the specific vascular injury dictating the procedure performed. When vascular injuries are not present, the primary clavicular injury and the secondary damage to adjacent structures, including disrupted neural structures, dictate the immediate management, which may be surgical or nonsurgical. With neural disruption, the primary consideration is the completeness of the lesion. With incomplete nerve injuries, conservative treatment is usually advocated, although some surgeons perform external neurolysis.[57,61] Analgesics, including neuropathic pain medications, and braces are helpful for associated pain. Once the pain is controlled, physical therapy (range of motion and strengthening exercises) is employed. Synergistic muscle strategies combat the loss of a specific muscle function. The prognosis for vascular system recovery reflects the severity of the vascular damage, the underlying etiology, and the promptness of its recognition and is typically determined by the vascular surgeon. The prognosis for the neural recovery reflects the likelihood of reinnervation (see Part 1)[1].

DISPUTED TOS

Epidemiology. Disputed TOS (also known as nonspecific TOS, assumed TOS, and symptomatic TOS) has an adult onset, is more common among women, and, unlike the 4 nondisputed forms of TOS, is frequently bilateral. Among the proponents of disputed TOS, most believe that it involves the brachial plexus and, therefore, consider it to be a type of neurogenic TOS.[4] Consequently, they often lump it with TN-TOS as "neurogenic TOS." Because of the high prevalence of disputed TOS, this approach renders neurogenic TOS much more prevalent than V-TOS, which it is not.

Pathogenesis and Pathophysiology. Despite its introduction into the literature several decades ago, the specific pathogenesis of disputed TOS remains uncertain. Most proponents believe it represents either a compression injury or a traction injury of the brachial plexus within the thoracic outlet.[4] Four explanations for this have been proposed, including: (1) an underlying congenital anomaly (cervical ribs; congenital fibromuscular bands; FTR or scalene muscle abnormalities), (2) trauma, (3) poor posture or a particular body habitus (long neck with droopy shoulders), and (4) some combination of these. [4,61,66-69] Some authors consider trauma to be the most common cause and, in one series, it was the underlying etiology in 91%.[70] The trauma occurs in 1 of 2 ways: a single episode (most commonly whiplash related to a motor vehicle accident) or a series of minor injuries producing cumulative trauma.[70] These injuries, in turn, cause: (1) scalene muscle fibrosis, (2) spasm of congenital musculotendinous ligaments, (3) traction-induced scarring in and around the brachial plexus,[4,61] or (4) muscle imbalance.[58,61] Regarding muscle imbalance, it is thought that joint position changes related to muscle atrophy or hypertrophy cause the affected muscles to function at less than their ideal lengths. Over time, the inappropriately shortened muscles undergo adaptive shortening and tightening. When movement stretches these shortened muscles, the resultant pain causes the patient to modify the joint position even more inappropriately.[71,72] A small group of proponents classify disputed TOS as a neurovascular disorder due to combined brachial plexus and subclavian artery compression based on "arterial insufficiency" precipitated by specific ancillary maneuvers.[61,73-75] Thus, this group considers disputed TOS to be a mixed (neurovascular) form of TOS, similar to traumatic neurovascular TOS.

Clinical Features. The clinical features associated with disputed TOS are unclear and debated among its proponents. Whereas some argue that the clinical features are specific and refuse to

make the diagnosis in their absence, others diagnose disputed TOS based solely on vague symptoms (pain; subjective sensory symptoms; limb fatigue or heaviness; weakness unrelated to a particular muscle group). Regarding its sensory features, some authors report a lack of sensory abnormalities as typical, whereas others describe sensory loss in a lower trunk distribution as typical. Regarding its motor features, some authors report weakness, whereas other cite that weakness is inconsistent with the diagnosis.[4,61,69,70,76-79]

Overall, sensory complaints (pain; paresthesias) are the predominant manifestation and are present in over 90% of patients.[70] Two pain patterns have been described: a lower plexus type and an upper plexus type.[75,78] With the lower plexus type, the pain occurs in the neck or supraclavicular region and radiates along the medial aspects of the arm, forearm, and hand; the associated sensorimotor abnormalities have a C8 or T1 distribution. With the upper plexus type, the pain is located in the shoulder region and radiates into the ipsilateral aspects of the head and neck, the anterior and posterior aspects of the upper portion of the thorax, and the proximal aspect of the arm (well beyond the upper plexus distribution); the associated sensorimotor abnormalities have a C5 or C6 distribution.[61,72] Motor complaints are commonly reported.[76] Typically, when intrinsic hand muscle atrophy has been reported, it has been in association with a cervical rib, suggesting TN-TOS rather than disputed TOS Other reported features include symptom worsening with effort (especially activities requiring abduction or an overhead position), occipital or orbital headaches, facial pain, facial numbness, and anterior chest wall pain.[4,70,72]

Another group of proponents diagnose disputed TOS whenever a patient with a “typical” history has a positive provocative maneuver, even when the neurological examination is normal.

As expected, with this approach, disputed TOS is often identified bilaterally. Several provocative maneuvers have been utilized in this regard, including the Adson, Wright maneuver, costoclavicular, and EAST (elevated arm stress test) maneuvers. With the Adson maneuver (also called the "scalene test" because it was believed to identify scalenus anticus syndrome), the patient is seated with the hands resting on the thighs, the head rotated toward the symptomatic side, and the neck hyperextended. In this position, the patient deeply inspires and holds the inspiration while the examiner palpates the radial pulse.[72,81] Radial pulse obliteration and symptom reproduction indicate a positive test. With the Wright maneuver, the symptomatic limb is maintained in the surrender position (abducted to 180 degrees with the elbow flexed and the arm externally rotated) for 60 seconds, while the examiner palpates the radial pulse.[82] Symptom reproduction or a diminished or absent pulse indicates a positive test. Some keep the elbow extended (to avoid positivity related to cubital tunnel syndrome) and the wrist neutral (to avoid positivity related to CTS).[72]

With the costoclavicular maneuver (Halsted test; military brace maneuver), which theoretically narrows the costoclavicular space and was previously used to identify costoclavicular syndrome, the radial pulse is palpated while the patient is in a modified at-attention posture, with the shoulders depressed and retracted.[75] Radial pulse obliteration and symptom reproduction indicate a positive test.[72] The most popular provocative maneuver is the EAST maneuver (the Roos test).[83] With this maneuver, the upper extremities are maintained in the surrender position and the hands are opened and closed every 2 seconds for 3 minutes.[11,83] Symptom reproduction, hand paresthesias, inability to maintain the position, or radial pulse diminution indicates a positive test.[4] Two prospective studies assessing the EAST maneuver in normal individuals reported high false-positive rates.[61,84,85] In another study of healthy adult

volunteers, this maneuver precipitated paresthesias in 36% and pulse alterations in 62%.[86] Because patients with CTS frequently develop hand paresthesias in response to upper extremity elevation, the EAST procedure is falsely positive in most CTS patients. This maneuver also purportedly identifies A-TOS and V-TOS. With A-TOS, the upper extremity becomes ischemic and claudicates, whereas with V-TOS, it becomes cyanotic and the forearm veins become distended.[11] In summary, when these maneuvers are utilized, many normal individuals and individuals with CTS test positive. To avoid misdiagnosis, unless the maneuver reproduces the patient's symptoms exactly, it should be considered negative.[61,70,74,75,87,88] Even with exact symptom reproduction, diagnostic caution is advised.

Evaluation. The appropriate evaluation for disputed TOS varies among the proponents of this disorder. At one extreme are physicians who believe that the clinical features associated with disputed TOS are so characteristic that supplemental testing (laboratory, imaging, and EDX) is unnecessary.[61] Indeed, some have argued that EDX testing should be avoided because it is uncomfortable, expensive, and of no diagnostic value for TOS or any of the differential diagnostic considerations.[61,78] This belief appears to be held by a minority of disputed TOS proponents. Most proponents believe that EDX testing is required to identify other entities.[74,76,89,90]

Given the ongoing controversy surrounding disputed TOS, a diagnostic evaluation is mandatory. EDX testing is indicated because it has high sensitivity for TN-TOS and for the neuromuscular disorders in the differential diagnosis. The presentation dictates the required imaging studies (cervical spine and shoulder radiographs and MRI; chest CT). When vascular features suggest A-TOS, V-TOS, or traumatic neurovascular TOS, vascular imaging studies are

necessary. Applying this approach, the majority (64-75%) of disputed TOS patients have an alternative disorder.[74,91] All patients should undergo EDX testing prior to surgical intervention.[92] In 2010, in Washington State, because of poor outcome among the majority of workers' compensation patients undergoing surgery for "neurogenic TOS", a guideline was created requiring objective EDX evidence of brachial plexus involvement.[93]

An important EDX issue warranting focused discussion is the belief that disputed TOS can be identified by the demonstration of focal conduction slowing of ulnar motor fibers as they traverse the thoracic outlet. This technique, first reported in 1972, was claimed to be highly sensitive and capable of differentiating patients best treated surgically from those best treated conservatively.[94] These authors also introduced a similar study for assessing the median motor fibers.[61] In both of these techniques, the motor nerve conduction velocity value was calculated and considered abnormal when it was below 85 m/s. When below 60 m/s, surgical intervention was required; conservative treatment was recommended for a value of 60-85 m/s.[90] Using these techniques, the authors reported that approximately 2000 of 8000 annual NCSs demonstrated disputed TOS (roughly 25% of all patients undergoing EDX testing).[90]

In order to appreciate the shortcomings of this approach, a few comments are necessary. First, to accurately calculate the motor nerve conduction velocity value, the distance between the stimulation and recording sites must be accurately determined. Although the distance between the supraclavicular and axillary or arm stimulation sites can be accurately measured, the value underestimates the actual length of the ulnar or median axons between these sites, which falsely lowers the calculated value.[4] Second, to identify focal demyelination by NCS, the stimulating and recording electrodes must straddle the lesion so that current traverses it.[95] With

supraclavicular fossa stimulation, however, the mid-region of the lower trunk is stimulated.[64,95] Thus, the stimulating and recording electrodes are both distal to the putative compressive lesion site and, accordingly, cannot assess it. Third, because the motor nerve fibers composing the lower plexus are located deeply below the skin, a significant amount of current is required for supramaximal stimulation. Because it is unlikely that the ulnar and median motor fibers are actually being stimulated directly below the cathode, the calculated conduction velocity is inaccurate.[96] Fourth, focal demyelinating conduction slowing reflects nonuniform focal demyelination, which, clinically, is either asymptomatic or associated with episodic tingling. Negative clinical features (weakness and numbness) are associated with demyelinating conduction block and axon degeneration because only these 2 pathophysiologies prevent action potentials from traversing the lesion (see Part 1)[1].

Based on the above discussion, it is not surprising that a number of investigators could not reproduce the conduction velocity or standard deviation values reported.[97-102] In 1976, Cherington reported that the normal conduction velocity values provided by the authors of this technique were too high and that the technique was insensitive.[103] In 1977, Di Benedetto concluded that the technique was insensitive and nonspecific.[104] Finally, in 1984, the photograph included in the 1972 article[94] describing this technique was shown to be a fabrication[105]: the supraclavicular response was actually a mid-arm response that was collected using a different sweep speed. Once this technique was discredited, it was, for the most part, abandoned.[106] The original authors added a second technique to identify the upper plexus type of disputed TOS, which involved the placement of the recording electrodes over the muscles of the thenar eminence.[61] However, these muscles are innervated by the C8 and T1 roots and, thus, assess the lower plexus.

A final EDX issue concerns the use of F waves and somatosensory evoked potentials in the diagnosis of disputed TOS. Because F waves and somatosensory evoked responses are latency measurements, they only indicate the propagation speeds of the fastest conducting fibers. Therefore, they are notoriously insensitive to focal axon loss and are far less sensitive than the standard EDX studies (see Part 1)[1]. Thus, most investigators have found these techniques to be normal among this patient population.[4,61,107] Although some investigators have reported enhanced sensitivity when somatosensory evoked responses are recorded with the upper limb maintained in a particular position,[108,109] this modification may introduce abnormalities related to technical issues,[110,111] and any identified abnormalities are nonspecific.

Finally, because of the lack of a gold standard diagnostic test for disputed TOS, ancillary diagnostic procedures have been introduced such as anterior scalene muscle injections with anesthetic, steroids, or botulinum toxin, arterial Doppler procedures, and vibratory threshold measurements.[70,73,74,110,112-114] Their utility has been debated and none of them have achieved widespread acceptance.[4,70,83,115]

Treatment and Prognosis. Although both conservative and surgical therapies have been advocated, the majority of disputed TOS proponents recommend initial conservative treatment.[61] To date, the most successful conservative strategy has been (1) to identify postural abnormalities and muscle imbalances, (2) to educate the patient regarding proper postures (sitting; standing; sleeping), and (3) to initiate a stretching program targeting the pathologically shortened and tightened muscles for long-term relief.[116] When conservative approaches do not address the primary problem, they only provide short-term relief.[72] When the abnormal postures are secondary to another problem, that problem should be addressed (obesity; large breasts).

Regarding the latter, the application of a bra that crosses in the back may result in postural improvement and relief of the associated cervicoscapular pain, thereby avoiding the need for reduction mammoplasty.[76] Mental health referral is beneficial for depression. Surgical intervention is rarely required when these conservative approaches are employed.[73,89,117]

When surgery has been deemed necessary, the reasoning underlying the chosen procedure often has been unclear. Historically, Coote performed decompressive surgery (cervical rib removal) in 1861,[104] Bramwell performed FTR resection in 1903,[118] and Adson and Coffey performed scalenotomy (anterior scalene muscle division) without surgical rib resection in 1927, a procedure that Adson later modified to include total removal of the muscle (scalenectomy).[119,120] In 1962, Clagett reported that FTR resection (using a posterior approach) was useful for all forms of TOS because it removed the common denominator. In other words, regardless of the structural element responsible for the neurovascular compression, this compression was always into the FTR.[121] Because this approach was technically demanding and produced a cosmetically unappealing scar, Roos suggested a transaxillary approach, which had neither of these two drawbacks. For these reasons, this approach became more popular,[83] with posterior thoracoplasty reserved for patients with recurrent symptoms who required a second surgical intervention.[90] Because of poor visualization and a long distance between the incision site and the FTR, other approaches were also advocated (infraclavicular; transclavicular; posterior).[75,122] In addition, the list of resectable structures also grew and included muscles (anterior scalene; middle scalene; omohyoid), osseous structures (congenital anomalies), ligaments, and any potentially compressive structures (fibromuscular bands); some surgeons also performed neurolysis or dorsal sympathectomy.[61,89,123,124] Of these procedures, 2 of the more popular were first rib resection and scalenectomy (anterior and middle scalene muscles),[89] and

some surgeons recommended performing both to avoid the need for a second surgery should symptom relief be inadequate.[123]

Generally, the operation chosen reflects surgeon preference, with some performing a single technique for all patients and others tailoring the procedure to the clinical features (scalenectomy for suspected upper plexopathies; transaxillary FTR resection for suspected lower plexopathies).[61,125-127] One group reported that patient outcomes following supraclavicular scalenectomy were comparable among patients with lower plexus and upper plexus symptoms.[126] They also reported comparable results between scalenectomy alone and scalenectomy combined with FTR resection. Other groups reported satisfactory outcomes when patients with upper plexus symptoms underwent transaxillary first rib resections.[127] Thus, the outcomes reported appear comparable regardless of the plexus region involved or the surgical intervention applied. By 1995, it was clear that the earlier reports of cure rates exceeding 90% were gross overestimates.[128-132] In addition, serious complications were reported, including brachial plexus and nerve injuries, nerve transections (long thoracic; phrenic; intercostal brachial; supraclavicular cutaneous), severe and disabling postoperative pain, reflex sympathetic dystrophy, blood vessel damage requiring amputation, and death by exsanguination.[4, 115,123,124,133-136]

CONCLUSION

The term “thoracic outlet syndrome” should not be used in the singular because it reflects a group of distinct disorders with differing etiologies, clinical features, evaluations, and treatments. A-TOS follows obstruction of the subclavian artery with resultant ischemic symptoms and possible embolization. Thus, treatment is aimed at restoring arterial blood flow, followed by

surgical decompression. V-TOS follows subclavian-axillary venous occlusion with thrombus formation and, rarely, embolization. Thus, urgent thrombolysis is required, followed by anticoagulation and possible surgical decompression. The approach to traumatic neurovascular TOS reflects the underlying etiology. The treatment of TN-TOS is surgical decompression (see part 1). Disputed TOS should be considered a cervicoscapular pain syndrome that is best managed by physical therapy directed at postural and muscle imbalances. Surgical intervention is rarely, if ever, indicated because it is seldom beneficial and potentially harmful.

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