Thoracic Outlet Syndromes

Review of the Controversies and a Report of 17 Instrumental Musicians

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Few diagnostic entities in medicine provoke as much disagreement and dialogue as the thoracic outlet syndrome. A detailed historical survey and literature review are well beyond the scope of this paper, but the interested reader may consult one of several recent accounts.[1-4] I will review the major areas of controversy surrounding this diagnosis and offer my own observations on 17 instrumental musicians who have this entity. A preliminary account of this view has been presented previously.[5]

Terminology

Over the years, a number of clinical syndromes attributable to neural or vascular compression between the neck and the axilla have been described. Terms used for these disorders have primarily referred to the structures compressing the neurovascular bundle, such as anterior scalene, cervical rib, costoclavicular, and pectoralis minor syndromes, although others refer primarily to the mechanism of compression, such as the hyperabduction syndrome. In 1956, Peet et al.[6] attempted to combine these, using the collective term "thoracic outlet syndrome." Rob and Standeven[7] modified that to "thoracic outlet compression syndrome," although the former term has become generally accepted. I will follow this common usage, with the single modification suggested by Gilliatt,[3] to use thoracic outlet syndromes (TOS) as a means of emphasizing the existence of several forms.

One may readily identify three general subcategories of TOS, the arterial, venous, and neurologic (or neurogenic) forms, depending on the specific or predominant structure compressed. It is generally acknowledged that neurologic symptoms are most common.[8,9] Within this category, there is a relatively rare form, variously called "classical," "motor," or "true neurogenic" TOS, with recognizable weakness, atrophy, and sensory loss, consistent electrodiagnostic changes, and a cervical rib or elongated transverse process on the seventh cervical vertebra.[10] A second, and much larger, group consists of patients in whom sensory symptoms are prominent but actual motor impairment and sensory loss are minimal or absent. This category has recently been called the "disputed" neurogenic type,[4] a term that has a pejorative rather than the "neutral" connotation suggested by the author[4] and fails to convey the major clinical features. At the risk of further confusing the issue, I will refer to this as the "symptomatic" type, a concept utilized by others such as Lascelles et al.[11]

TOS is a relatively common cause of upper extremity symptoms among instrumental musicians.

While it may be viewed as a pedantic and unimportant criticism, the term "thoracic outlet syndrome" itself is anatomically incorrect. The area in question, which lies above the apex of the lung, bounded in front by the clavicle (collar bone), behind by the scapula (shoulder blade), and on either side by the neck and shoulder, should be called the thoracic inlet or superior thoracic aperture. In fact, the thoracic outlet is actually that area covered by the diaphragm. However, the anatomically inaccurate term is generally accepted and will likely persist.

Diagnosis

A second and more important area of controversy revolves around the diagnosis itself. All seem to agree that the basic clinical symptoms are those of pain and paresthesias or spontaneous sensations that may be described by the patient as numbness, pins and needles, prickling, tingling, coldness, itching, or swelling. The difficulty lies in trying to separate TOS from the many clinical entities presenting with neck and arm pain, including cervical radiculopathy with spondylosis or disc herniation; shoulder disorders such as acromiohumeral impingement, rotator cuff injuries, bursitis, and tendinitis; various entrapment mononeuropathies involving the upper extremity, most notably median neuropathy at the wrist (carpal tunnel syndrome) and ulnar entrapment at the elbow; and a large but poorly delineated group of muscle pain syndromes that may include myofascial pain, fibrositis, nonarticular rheumatism, and the "overuse" syndromes (muscle-tendon overuse, repetition strain injury, cervicobrachial occupational syndromes, etc.). Weakness is often described in this symptomatic form of TOS but objective weakness is generally poorly documented. Other features may include skin changes such as

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blanching, pallor, cyanosis, or coldness, suggesting a vascular component to the process. By including still other symptoms such as headache,[12] chest pain,[13] vertigo, blurred vision, memory difficulty, unsteady gait, and drop attacks[14] among those associated with TOS, one risks stretching credibility, particularly if these symptoms are seen as predominant. I will arbitrarily exclude these from consideration.

Physical Signs

A major problem in rendering the diagnosis of TOS is that neurologic signs, despite the symptoms, are either nonexistent or meager. Indeed, this is the reason for describing this form as "symptomatic." Muscle weakness and wasting are constant features of the true neurogenic form of TOS. In the much more common symptomatic form (which does not appear to be simply an early stage in the evolution of true neurogenic TOS[4]), weakness may be more apparent than real and can often be attributed to pain or fear of producing pain. One of the distressing features of this diagnosis is that neurologists, who should be the most skilled at recognizing subtle manifestations of weakness as well as sensory loss when compared to other specialists, generally seem the least successful in identifying such changes, whereas vascular or thoracic surgeons, who have generally reported the largest series of such patients, seem to have much less difficulty finding objective weakness and sensory change. This in no small measure adds to the controversy. Sensory loss is sometimes described by the patient, usually in the little and ring fingers and along the ulnar (medial) aspect of the hand and forearm, but again, this can be very difficult to document on examination and tends to be inconsistent. Unilateral decrease or absence of tendon reflexes, even the triceps and finger flexor jerks, is rarely identified despite alleged involvement of the appropriate nerve pathway. The absence of physical signs, however, cannot be taken as evidence that the syndrome does not exist, since intermittent nerve compression could cause transient symptoms without demonstrable nerve injury.

Maneuvers and Ancillary Tests

Critical to the diagnosis and equally controversial is the use of one or more provocative maneuvers or ancillary tests. Various authors have emphasized the usefulness of placing the affected limb in one or another position (hyperabduction,[15] Adson maneuver,[16] costoclavicular maneuver[17]) as an aid in diagnosis. This may provoke the symptoms or produce specific physical signs such as pulse obliteration. As examples of the difficulties encountered one may point to the demonstration by Wright[15] that obliteration of the radial pulse with the hyperabduction maneuver can be seen in the large majority (over 80%) of normal individuals and the demonstration by Costigan and Wilbourn[18] that the "stick-up" position with hand exercise (the elevated arm stress test of Roos[19]) seems particularly sensitive in patients who have electrically-documented carpal tunnel syndrome.

Even more important, because of the additional factors of expense and potential risk, is the utilization of various procedures, including nerve conduction studies, somatosensory evoked potentials and measurement of arterial flow, either "noninvasively" or by arteriography. Since it has been reasonably well established that the symptoms are largely neurologic in the symptomatic form, and since reduction in arterial flow may be so commonly provoked in normal individuals or in the asymptomatic limb of a patient diagnosed as having unilateral thoracic outlet syndrome, the relevance of demonstrating arterial compression is questioned. Many clinicians who see and treat large numbers of such patients have abandoned these procedures in all but a small minority.[4,8,9] It has been correctly pointed out that arterial compression does not necessarily imply brachial plexus compression[8] and a procedure to detect more specifically the latter phenomenon should be sought.

The usefulness of nerve conduction studies and needle electromyography has been another subject of intense debate. The ulnar nerve conduction velocity, popularized by Urschel and his colleagues,[20,21] has been touted as the most effective means of confirming the diagnosis and of determining suitability for surgical intervention,[21] and has been criticized as being unnecessary and ineffective.[8] Several electrodiagnostic laboratories have been unable to confirm abnormalities in patients with clinically diagnosed thoracic outlet syndrome[22–24] and, indeed, the credibility of the procedure has recently been challenged when the only published example of an ulnar nerve conduction study purporting to show this abnormality[20] was found to be simulated.[25] More recently, somatosensory evoked potentials (SEPs) have been suggested as being more accurate and sensitive,[26,27] but this experience has not been universal.[28] We have been unable to reproduce consistent changes in SEPs, even with positions producing clinical symptoms.

Incidence

How common is this entity? Accurate data are simply not available. As has been pointed out above, any clinician can attest to the fact that neck and arm pain are extremely common complaints among patients seeking medical help. This is no less true and indeed seems particularly notable in performing artists. Mention was also made previously of the many possible causes of such symptoms and, for reasons that should already have become apparent, I suspect that many of the reported series of patients with thoracic outlet syndrome are contaminated with other diagnoses.

Gilliatt[3] has estimated that the true neurogenic or classical form may have an incidence of one per one million population. The symptomatic form appears to be far more common than that. At the other end of the spectrum, Urschel et al.[21] reported that, in one year, his service evaluated 8400 patients with relevant symptoms and found 720 with some degree of neurovascular compression at the thoracic outlet, an average of almost three per working day! If this were indeed an indication of true frequency, the number of new cases occurring annually in this country alone would be truly astounding, even if one takes into account selective referral of patients suspected to have TOS to this and similar centers. The very fact that the diagnosis is made with such frequency in some centers and so rarely in others certainly contributes to the virtual impossibility of determining incidence or prevalence figures at the present time.

Surgical Treatment

The final major area of controversy relates to treatment of thoracic outlet syndromes. Consideration of the technical aspects of treatment is beyond the scope of this review.

TABLE 1. Clinical Details of Thoracic Outlet Syndromes in 17 Instrumental Musicians

Case No.	*Sex*	*Age*	*Primary Instrument*	*Arm affected*	*Symptom duration at elevation (yrs)*	*Treatment*	*Results*
1	M	40	piano	R>L	1	P.T.	+
2	F	21	piano	R	1.2	P.T.	+
3	M	35	piano	R	1.3	surgery	+
4	F	25	piano	L	6	P.T.	+
5	F	33	piano	R=L	0.9	P.T.	+
6	M	27	violin	L	0.6	P.T.	+
7	F	38	auto harp	L	0.3	surgery	+
8	F	19	cello	L	6	P.T.	+
9	M	19	violin	R	2.5	P.T.	+
10	F	18	violin	L	2	P.T.	−
11	F	20	violin	L	2	P.T.	+
12	F	18	violin	L>R	0.3	P.T.	equivocal
13	F	19	violin	L>R	0.2	P.T.	+
14	F	26	viola	L	1.2	P.T.	+
15	F	15	flute	R	1	P.T.	+
16	F	22	flute	L	2	P.T.	−
17	M	24	saxophone	R	1.2	P.T.	−

*Lost to follow-up at 3 months.
P.T. = physical therapy.

Nonetheless, the choice of treatment engenders a controversy that cannot be ignored. The most experienced surgeons in this country report truly staggering numbers of patients operated on for TOS.[21,29] Others of equal renown in the same specialty, even in centers that might be expected to attract large numbers of patients, may report far smaller numbers.[30] Clearly this reflects differing views on diagnosis and treatment. All agree that nonsurgical therapy, primarily some form of exercise, is appropriate for at least some patients.[5,8,9,20] Despite offering "conservative" treatment for those with mild to moderate symptoms, Roos,[31] whose referrals presumably are skewed toward more difficult or intractable cases, has collected a series of almost 2000 operations for "moderate or severe symptoms." One suspects that other surgeons have different thresholds for surgical approaches to TOS. Dale for example, has suggested that surgery be carried out only as a last resort.[9] The exercise regimens proposed generally are geared to strengthening the elevator muscles of the shoulder and improving upper trunk posture.[6] Some forms of TOS may not be amenable to either exercise or surgical intervention, such as the type associated with the droopy shoulder configuration.[32] I should add that this has not been my experience.

There remains controversy about the type of surgical procedure that should be utilized when operative management is deemed necessary. This must continue to be an individual decision based upon the surgeon's experience and analysis of causative mechanisms. The usual choices include removal of the first rib, either by the popular transaxillary[29] or the more difficult posterior approach,[33] or section of one or more muscles or fibrous bands (e.g., scalenotomy[16] or scalenectomy[29]). There appears to be no consensus, even among the most experienced surgeons.[9]

Complications of Surgery

Reports of surgical complications are disturbing, regardless of the type of surgical approach. Two recent papers have emphasized the potentially crippling nature of brachial plexus injuries associated with surgical intervention.[34,35] The numbers here are small and one might argue that this is in fact quite uncommon. Nonetheless, Dale[9] surveyed a large number of cardiovascular surgeons and about one-half of the group, who accounted for approximately 1300 operations annually, were aware of 102 instances of complete arm paralysis postoperatively and an additional 171 cases of partial neurologic deficit. This suggested a considerably higher complication rate than has been generally acknowledged. It is information of this type that led Cherington to propose a moratorium on surgery for thoracic outlet syndrome.[36]

The Author's Approach

Given all of this, one might well be tempted to escape the controversy and simply avoid making the diagnosis of TOS in all but the true neurogenic and purely vascular forms. I believe this is a mistake because there are substantial numbers of patients for whom this represents the most reasonable diagnosis and who, most importantly, may benefit by treatment designed for the syndrome.

I have found that TOS is a relatively common cause of upper extremity symptoms among instrumental musicians,[37] as have Hochberg et al.[38] Indeed it is my impression that this disorder occurs more frequently among musicians than other groups. Among 121 consecutive instrumental musicians with upper extremity symptoms, I have diagnosed TOS in 17 (13%). None of the patients had true neurogenic TOS; all had the symptomatic type. There were 5 pianists, 9 string instrumentalists, and 3 wind players (Table 1). Twelve were women and five men, with ages ranging from 15–40 years (mean = 25). Further clinical details are summarized in Table 1.

Symptoms consisted of pain in all 17, mostly in the forearm or hand, and paresthesias in 10 of 17, primarily along the ulnar forearm and hand and in the little or ring fingers. In every case, symptoms were aggravated by certain positions, especially playing the instrument, and could be elicited or worsened by provocative maneuvers. The most effective maneuvers in my experience have been either

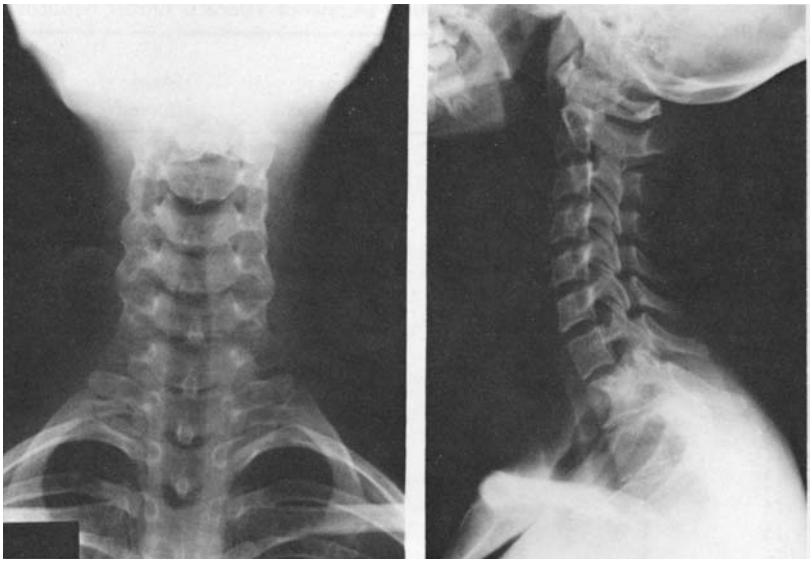

FIGURE 1. Cervical spine x-rays, case no. 13, frontal (left) and lateral (right) views, showing the droopy shoulder configuration. Note on the lateral view that the upper portion of the third thoracic vertebra is visible above the shoulders, whereas in normals the first thoracic vertebra and often the seventh (lowest) cervical vertebra are obscured by overlying structures. On the frontal view, the characteristic horizontal orientation of the clavicles is seen.

hyperabduction or downward traction on the affected arm, with simultaneous internal rotation of the shoulder. The latter was especially productive in the 9 of 17 (53%) patients with the droopy shoulder configuration. No significance was placed on obliteration of the radial pulse with these maneuvers, unless pallor or cyanosis was striking and limited to the symptomatic limb; production of a bruit was also found to be of little importance. Neurologic examination was normal in each patient. Electrodiagnostic studies, including standard nerve conduction tests and SEPs in different positions, were helpful only in excluding carpal tunnel syndrome or alternate diagnoses. Neck x-rays showed no cervical rib or elongated transverse process of C_7 in any patient, but did often show the typical changes of droopy shoulder syndrome (Fig. 1).[39]

Fifteen patients were treated with various nonsurgical measures, primarily physical therapy, and two underwent first rib resection after failure of "conservative" treatment. The two surgical patients recovered completely and have returned to full playing schedules. Of the 15 treated with physical therapy, consisting of postural training, range of motion exercise for neck and shoulders, and some strengthening of the shoulder girdle, 11 improved substantially or completely, 1 showed minimal improvement and 2 were unchanged. One of the latter is still playing (saxophone) after a number of months; the other has apparently abandoned plans for a career as a performer. One patient was lost to follow-up at 3 months but at that time had not shown any benefit from treatment. Physical therapy was effective in 11 of 17 (65%) instrumental musicians with TOS and the overall success rate in the 17, including those surgically treated, was 76%. Two of the patients who have not responded to treatment are currently considering surgery.

Conclusion

Resolution of the controversies regarding TOS, as with so many in medicine, may be difficult to achieve. Collaboration among various specialists to arrive at mutually acceptable definitions, diagnostic criteria, and treatment protocols as well as follow-up of treated patients, even if not in controlled trials, would be useful initial steps.

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