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Thoracic Outlet Syndrome

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Soft tissue cervical injuries are among the most common posttrauma problems faced by general practitioners, orthopedists and neurosurgeons. The development of the thoracic outlet syndrome (TOS) following such injuries is more frequent than is generally recognized. Conservative treatment with physiotherapy, muscle relaxants, anti-inflammatory drugs and percutaneous electrostimulation usually proves effective in relieving TOS. In this series of 1,958 soft tissue cervical injuries, 459 patients (23 percent) were diagnosed as having TOS. Of these patients, 185 (41 percent) failed to respond to all conservative measures and consequently surgical operation in the form of a supraclavicular neurovascular decompression became necessary to relieve not only the symptoms of the neck-shoulder-arm syndrome but also to relieve a frequently unrecognized (72 percent) vertebrobasilar artery syndrome. The latter is most definitely an integral part of TOS. The most common misdiagnoses were cervical disc syndrome (42 percent) and cervical strain (42 percent). A long-term follow-up (average of seven years) of 90 patients showed that the results were judged good in 75 percent, fair in 11 percent and poor in 14 percent.

DURING THE DECADE from 1965 through 1975 the author has had the opportunity, in his private neurosurgical practice, to examine 1,958 soft tissue neck injuries. In 459 (23 percent) of these patients a concomitant thoracic outlet syndrome (TOS) developed. Such a high incidence has not been reported previously. A complete bibliography may be obtained from articles by Woods,[1] Lord and Rosati,[2] Kremer[3] and Tyson.[4]

Definition

The term thoracic outlet syndrome is used synonymously with scalenus anticus (Naffziger) syndrome and cervicobrachial neurovascular compression syndrome. TOS can be defined most simply as a syndrome in which there is sufficient pressure on the neurovascular bundle in the supraclavicular fossa or the costoclavicular space, or both, to cause local and distal disturbances of nerve and vascular function. Such pressure may be caused by tightening, fibrosis or hypertrophy of the anterior scalene muscle, the pressure of a cervical rib, narrowing of the costoclavicular space, deformity of the clavicle or first rib, an aneurysm or a tumor, or any other space occupying lesion. Symptoms result from dysfunction of the brachial plexus, phrenic nerve, subclavian artery, subclavian vein, or vertebral artery.

This study of the thoracic outlet syndrome (TOS) arose out of the opportunity this author has had to personally examine and diagnose a large series of soft tissue neck injuries and, therefore, be able to analyze statistically the occur-

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TABLE 1—*General Analysis of Cases Reviewed*

Soft Tissue Neck Injuries Studied (1965-75)	1,958
Incidence of Thoracic Outlet Syndrome (TOS)	459 (23%)
Sex Distribution	
Male	29%
Female	71%
Age	
Distribution (years)	10-84
Mean (years)	37
Average Time Lapse Between Injury and Examination	14 months

rence of TOS, its causes, symptomatology and treatment, and to confirm an earlier statement[1] that all too often TOS was not diagnosed before examination.

Table 1 shows an unexpectedly high incidence of TOS (23 percent) following neck injury and confirms the fact that females are far more vulnerable (by a ratio of 2.5 to 1) than males.

By far the most common cause of TOS was a neck injury suffered in an auto accident (61 percent). The ubiquitous rear-end collision, resulting in a "whiplash injury," accounted for 44 percent of the cases. Inevitably these patients became not only therapeutic but medical-legal problems. Accurate diagnosis, including psychological evaluation of the patient and his complaints, was of prime importance because of the intensive legal scrutiny each case underwent. Accuracy of prognosis was of equal importance.

Other causes include industrial accidents, 23 percent; miscellaneous trauma, 8 percent, and unknown, 8 percent.

The diagnosis of TOS would appear to be simple. A patient suffers a musculoligamentous cervical strain (only 8 percent of the patients in this series had no history of neck trauma). The patient is usually not rendered unconscious but states he was "dazed." Neck pain and stiffness increase, and, if the patient is one of the 23 percent in whom TOS develops, he eventually has a combination of the 20 symptoms and signs listed in the order of their frequency in Table 2.

The diagnosis of TOS is often not made because the patient only has complaint of neck and arm pain. He is reticent to reveal other symptoms which he cannot relate to a mere "neck strain." Common among these hidden symptoms are postural vertigo (48 percent), blurred vision (44 percent), difficulty with memory and concentration (31 percent), unsteady gait (18 percent) and syncopal attacks (11 percent). These

TABLE 2.—*Frequency of the Symptoms and Signs of Thoracic Outlet Syndrome*

	Percent
Exact reproduction with anterior scalene pressure	100
Numbness and tingling of hand	99
Neck pain and stiffness	96
Arm pain	94
Weakness of grip	89
Occipital headache	85
Radial pulse signs:	
Adson	7
Reverse Adson	51
Retro-orbital pain	54
Coldness of hand	52
Scapular pain	52
Postural vertigo	48
Blurred vision	44
Difficulty with memory and concentration	31
Reflex diminution	24
Color change of hand	21
Anterior chest pain	21
Unsteady gait	18
Nondermatomal hypalgesia:	
Hand and arm	15
Hand, arm, face and neck	15
Hand alone	10
Hand, arm and neck	5
Syncope ("drop attacks")	11
Tinnitus	6

particular "subjective symptoms," arising from vertebral artery compression, must be elicited from the patient by an experienced examiner. All too often when the patient volunteers such information he is dismissed as having a "functional overlay," or even as a malingerer. The role of vertebrobasilar artery insufficiency, most often brought on by sudden neck rotation or change of posture, is all too frequently overlooked in the patient with TOS.

An accurate history is the sine qua non leading to the diagnosis of TOS. The frequent lack of objective findings on examination was the primary factor for the amazingly high percentage of failure to diagnose TOS in this series (72 percent), in spite of the fact that the time lapse between the original injury and the author's first examination averaged 14 months.

The incomplete clinical diagnosis of "cervical strain" was made in 42 percent of the reports previously sent to referring physicians, insurance companies or attorneys. Of great significance is the fact that another 42 percent of the patients entered for examination with the erroneous diagnosis of "cervical disc syndrome." In 15 patients cervical discectomies had actually been done elsewhere previously. In none of the patients had symptoms been relieved. Following supraclavicu-

lar cervicobrachial decompression there was good relief of symptoms in all 15 of these patients.

The direct or implied previous diagnoses of psychoneurosis, "functional overlay" (5 percent), and malingering (2 percent) were encountered far more frequently in TOS patients than in any other symptom complex encountered in this author's neurosurgical practice. Again, the cause of this discrepancy was failure on the part of the examiners to question the patients, when originally seen, as to the presence or absence of the symptoms listed in Table 2.

Other previous misdiagnoses included carpal tunnel syndrome (2 percent) and others such as "angina" or "stroke" (2 percent).

Another cause for the frequent failure to diagnose TOS is that there is no purely objective diagnostic procedure.

Seventy patients were tested with electromyography and only in 14 percent of these was lower motor neuron dysfuntion reported.

Myelography is, of course, not indicated. It was, however, carried out previously in 49 patients.

Cervical x-ray films were obtained in all of the patients in the series. The only truly diagnostic finding was that of bilateral cervical ribs in eight patients (2 percent). Osteoarthrosis was reported in 36 percent and diminished cervical lordosis in 14 percent. Findings in 48 percent showed no abnormalities.

Electronystagmography (ENG) has a definite value by providing a positive "objective" finding of abnormality in those patients whose postural vertigo, occasional nystagmus and "drop attacks" result from vertebrobasilar artery insufficiency of the medullary vestibular nuclei. This has been reported in detail previously.[5] Electronystagmograms were carried out in 28 patients, with positive findings in 93 percent.

Nerve conduction studies across the thoracic outlet in ten patients failed to show any significant abnormalities.

Subclavian-vertebral arteriograms have proved of little value except in those rare cases where there is a supraclavicular bruit.

Treatment

All patients were treated conservatively with medication—muscle relaxants such as diazepam (Valium®), and anti-inflammatory drugs such as indomethacin (Indocin®), ibuprofen (Motrin®) or naproxen (Naprosyn®). Treatment with physiotherapy was done in 109 patients. This conservative treatment proved to be effective in 50 percent of the cases. The average length of treatment was nine months.

TABLE 3.—*Results of Operative Treatment*

	185 Patients Immediate Follow-up. (Average 4 mos.) (Percent)	*90 Patients Long-term Follow-up. (Average 7 Yrs.) (Percent)*
Good result	81	75
Fair result	16	11
Poor result	3	14

Temporary relief of painful symptoms was obtained by transcutaneous electrostimulation in 75 percent of the 40 patients in which this modality was used. Very little relief was obtained with acupuncture. Most patients were made worse temporarily by cervical traction.

The operative procedure employed is carried out using endotracheal anesthesia, with the patient's head and neck turned and extended to the side opposite the TOS. An incision averaging about three inches (7.3 cm) is made paralleling the skin lines one finger's breadth above the clavicle centering over the anterior scalene muscle. The muscle is exposed and its fibers transected by crushing, and, if necessary, cutting in a transverse direction. Following this the cut ends of the muscle, which is under tension, will retract. The posterior capsule of the muscle and the confluent cervical fascia are dissected, exposing the underlying subclavian artery and brachial plexus. A periarterial stripping of the subclavian artery is done from the thyrocervical trunk to the thoracic outlet, making certain that there are no constricting fascial fibers remaining. A complete lysis of the brachial plexus is done, again making sure there are no constricting fascial fibers left *in situ*. The head is returned to a neutral position and the subcutaneous tissues and skin approximated in two layers.

In 185 patients (41 percent) there was no response to any form of conservative treatment. These patients, therefore, were considered suitable candidates for surgical intervention in the form of a supraclavicular cervicobrachial neurovascular decompression.

Immediate results of operations (see Table 3) were judged to be good in 81 percent, fair in 15 percent and poor in 3 percent. The average length of office follow-up was four months.

Since it is well known that operations for in-

tractable pain frequently offer only temporary relief, and because there has never been a long-term follow-up of patients who have undergone a "scalenotomy," an attempt was made to contact the 185 patients in this series in whom operations were carried out between 1965 and 1975. It was possible to contact 90 of the patients by means of a questionnaire. The results are presented in Table 3 and compared with the results noted at the time of the last postoperative office visit. It can be seen that although the good results dropped from 81 percent to 75 percent and the poor results rose from 3 percent to 14 percent the overall postoperative results have been most satisfactory.

In the 12 patients (14 percent) who were judged to have had a poor result following decompressive operation, a chronic localized brachial plexus neuritis almost invariably developed. In addition, these patients also had the symptoms of a widespread chronic "fibromyositis" which had arisen out of their initial trauma. Often a slightly elevated blood uric acid value was noted. Intensive treatment with colchicine and probenecid, the corticosteroids (prednisolone and the like) and other anti-inflammatory medications (Indocin, Motrin and Naprosyn) invariably failed to relieve the posttraumatic myositis or the postoperative localized neuritis. Occasional temporary relief could be obtained with the use of transcutaneous electrostimulation.

In eight patients scalenotomy had been done elsewhere and had failed to relieve TOS. Operations were carried out again in these patients via the previous supraclavicular approach. Six were found to have had inadequate lysis of the brachial plexus and decompression of the subclavian artery. Fine, tight fibers of cervical fascia were found still compressing the neurovascular bundle. The results of reoperation were judged good in all six patients. In the remaining two patients postoperative scarring was found. In these TOS was relieved by a complete decompression.

The question of which surgical approach, supraclavicular or transaxillary, is preferable for relief of TOS would seem to depend on the surgeon's training. Thoracic and vascular surgeons prefer the transaxillary technique, usually with removal of the first rib. The author, a neurosurgeon, still prefers the original supraclavicular approach because of the smaller (average 7 cm) incision, the more direct exposure of the scalenus anticus muscle and the underlying neurovascular structures, the infrequency of pulmonary complications such as pneumothorax or postoperative pleuritis, and the minimal postoperative morbidity and incisional pain. For success, both operative techniques must accomplish complete periarterial stripping and freeing of the compressed subclavian and vertebral arteries and lysis of the brachial plexus. Excision of a cervical rib can be readily accomplished by either method. Excision of the first rib (rarely necessary), and relief of the costoclavicular syndrome (also rare), can best be accomplished by the transaxillary approach.

Postoperative complications were rare. In five patients temporary dyspnea developed due to trauma to the phrenic nerve at the time of operation. Wound infection occurred in two patients. Local hemorrhage necessitating operative hemostasis occurred in one patient.

Discussion

The concept that the development of TOS following cervical trauma is due solely to pressure on the brachial plexus and subclavian artery by the overlying scalenus anticus muscle and its surrounding cervical fascia must be revised to include the symptoms resulting from vertebral artery constriction, namely postural vertigo, blurred vision, difficulty with memory and concentration, syncopal attacks, nerve deafness and tinnitus. This vertebral artery constriction is the result not only of direct mechanical compression, but most probably due also to dysfunction of the arterial autonomic nerve supply via the vertebral nerve.[1]

The frequency of occurrence, the long period of convalescence, the financial loss through disability and the inevitable medical-legal complications all make TOS one of the most prevalent and important posttraumatic problems faced by the medical profession.

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