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Epineurectomy for thoracic outlet syndrome

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Treatment of nerve compression syndromes varies a great deal from physician to physician. Internists believe strongly in expectant treatment. The belief that "time heals" is so strong that hand surgeons are always complaining about "late referrals." This long-lasting struggle has been perpetuated by engrained beliefs and isolation, rather than factual basis and communication across specialties.

As a result, treatments have spanned the spectrum of doing nothing, rest, immobilization, therapy, anti-inflammatory medication, and steroid injections. Surgical treatments vary no less and usually are aimed at decompression of the affected nerve. This is accomplished by dividing neighboring structures, transposing the nerve to a contiguous location, and neurolysis.

Neurolysis carries a variety of meanings. One surgeon may consider neurolysis the removal of contiguous structures, such as a synovectomy in conjunction with carpal tunnel release or the freeing of the nerve from surrounding structures. So releasing adhesions from one side of a nerve is a neurolysis. Releasing the nerve circumferentially is also a neurolysis, and the length of nerve segment involved is usually not taken into consideration. This vague definition of terms leads to inadequacy of most studies on the subject and prevents one from comparing the results of the various studies.

Epineurotomy involves the incision of the epineurium; the concept is that this limited procedure releases any constricting scar tissue and restores normal nerve turgor.

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Epineurectomy carries the concept of neurolysis and epineurotomy further. Strictly defined, it is the excision of epineurium from a nerve or nerve bundle. It therefore involves circumferential dissection of the nerve and removal of the outer fascial envelope of the nerve, together with all interfering extrinsic structures.

Surgical technique

The authors' approach to thoracic outlet surgery has been consistent for more than 20 years, yielding satisfying results. The surgical approach is initiated by a vascular surgeon, exclusively through a supraclavicular incision. The involved extremity is prepped and draped free to allow for mobilization of the limb during dissection. After an anterior scalenectomy is completed, the procedure is taken over by the hand surgeon who carries out an epineurectomy and a partial scalene medius myofasciectomy.

The length of the dissection is limited proximally by the intervertebral foramina and distally by the clavicle. Simply put, any structure impinging on the brachial plexus is removed. The dissection begins at the anterior ramus of C5 and progresses from there distally and caudally. Each nerve group is dissected out circumferentially and the epineurium is excised. Any vessel impinging on the plexus is ligated and divided. Any muscle, fascia, or fibrous band impinging on the plexus also is excised. This usually does not require a complete excision of the scalene medius.

The lower trunk of the plexus is usually difficult to visualize. It is often easier to find it somewhat distally (at the level of the divisions, where it connects to the middle trunk). C8 is first identified and dissected out. The lower trunk then

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is followed medially until the bifurcation of C8 and T1 is observed. At that point, T1 ramus can be easily dissected out also.

There is a great deal of variability in the anatomy of the brachial plexus. The most common variation seems to be the length of the various components. For example, the trunks can be short, bringing the divisions close to the rami. The relative diameter of the various fascicle groups varies a great deal also; C7 and C8 are usually the largest. The interconnections in the plexus also vary in location, pattern, and size. None of these variations, however, changes the plan of surgical decompression to any degree. All structures are systematically dissected out and decompressed, and an epineurectomy is performed.

The patient is given an intravenous bolus of dexamethasone (8 mg) and the wound is bathed with 5 ml of betamethasone solution (30 mg). The surgical closure is then performed by the vascular surgeon. A Jackson Pratt drain is left in the wound. After surgery the patient is admitted overnight (23-hour admission). The patient is discharged the next day and stops by the Hand Therapy Department on the way home.

Results

Fifty-two procedures were included in this study, with a minimum follow-up of 12 months (mean, 34 months; range, 12 months to 22 years). These were performed on 49 patients (three patients had bilateral thoracic outlet syndrome [TOS] release performed); mean age was 40 years (range, 23-66 years). Female to male ratio was nearly 3 to 1, with 73% female and 27% male. Litigation was ongoing in two thirds (62%) of cases.

These patients had various aspects of the syndrome (Table 1). Severe pain in the involved extremity, headache, and paresthesias were the most common symptoms. Physical findings included supraclavicular tenderness in most patients (75%); supraclavicular Tinel's sign was uncommon (13%) and a positive Roos test was clearly positive in only a minority of patients (19%) (Table 2). Roos test was equivocal in one third of patients (35%), for whom it elicited sensations of fatigue or heaviness in the involved extremity, rather than paresthesias.

Electromyography was positive in 90% of these patients. The incidence of bilateral TOS was 15%, although only three patients had sufficient follow-up for both sides to be included in this study. Three patients with previously failed surgeries performed elsewhere also were included. Multiple peripheral nerve entrapments were found in 86% of cases (Table 3).

On the first postoperative day, 40% of cases had complete resolution of all symptoms. All patients without exception stated at last follow-up that, considering what they went through, they would go through that surgery again with no regrets. Of patients with severe symptoms, only one (2%) still had severe pain postoperatively, two (4%) still had severe headaches, and two (4%) still had significant paresthesias.

Operative complications occurred in six cases (12%). Three of these were minor: a wound infection, a wound dehiscence, and a seroma; these resolved within a couple of weeks. There were three more serious complications, including two Horner syndrome and one pneumothorax; these complications did not require any treatment and resolved spontaneously.

Recurrence was seen in five cases (10%). Each one was related to a new car accident and pending litigation. A transient flare-up occurred in nine cases (17%) 5-18 months after surgery; all these were related to overuse and resolved with a brief course of therapy.

Discussion

The goal of surgery was defined simply by Adson in 1947: to liberate thoroughly the brachial plexus [1]. The authors' approach is similar. We

Table 1
TOS symptoms before and after surgery (percentage of patients with each complaint)

	Pain		Headache		Paresthesias	
	Preop	Postop	Preop	Postop	Preop	Postop
None	0	52	38	81	2	58
Mild-moderate	17	46	12	15	29	38
Severe	83	2	50	4	69	4

Table 2
Preoperative signs of TOS

	SC tenderness	SC Tinel's sign	Roos test
Positive	75%	13%	19%
Negative	25%	87%	46%
Equivocal	0%	0%	35%

Table 3
Incidence of ipsilateral nerve entrapment in patients with TOS

Carpal tunnel	73%
Cubital tunnel	46%
Radial tunnel	15%
None	14%

dissect out and remove any structures impinging on the brachial plexus. An understanding of the various anomalies is helpful, but intraoperatively the dissection revolves around the nerves of the plexus, not the anomalies encountered. Any anomaly that does not impinge on the brachial plexus (in any position of the head, neck, and chest) therefore is of no relevance.

Adson also stated at that time that scalenotomy yielded a success rate of 54% [1]. More recently Atasoy reports good to excellent results in 60% of patients following a combined axillary-supraclavicular approach (elsewhere in this issue) [2]. Sanders reports a 5-year success rate of 70% with scalenectomy and neurolysis with or without rib resection (elsewhere in this issue) [3]. A recent review article cites a success rate of 90%–95% in the first year and 70% in 5–10 years [4].

Success rate is difficult to define in such a complex disorder. The ultimate success may be patient satisfaction, but even that is difficult to measure. All the authors' patients declared that they would go through the surgery again if they had to; we could translate that into a success rate of 100%! Unfortunately, patient despair is not a reliable criterion by which to measure surgical success.

Severe symptom alleviation is another factor one might consider. In that respect the authors' success rate would be more than 90%. Under more stringent criteria, we might consider cure as complete resolution of *all* symptoms; by these criteria, the authors' success rate would be only 48%. This, however, excludes patients with only occasional mild symptoms who are happy with the outcome of their surgeries.

Most patients with TOS also have other disorders, some of which may mimic TOS, which may cloud the issue further. Although the differential diagnosis of TOS is not within the scope of this article, one should keep in mind that the differential diagnosis remains valid for any residual symptoms that a patient might have.

A theoretic objection to the authors' technique is that one might devascularize a nerve by performing an epineurectomy. First, the segment dissected is small, usually 5-7 cm in length, which can be supplied easily by intrinsic vascularity. Next, the success of this procedure in this patient population suggests that this is not an important issue. Further investigation would be helpful to clarify this point further.

Multiple nerve entrapments

Patients with TOS usually have multiple nerve entrapments at various locations, such as the carpal tunnel, cubital tunnel, and radial tunnel, and less frequently the ulnar tunnel or pronator tunnel. The frequency of associated neuropathies has led some investigators to state that TOS is nothing more than the referred symptoms of more distal nerve entrapments [5]. This underlines the importance of electrodiagnostic studies in the work-up of these patients.

Upton and McComas introduced the concept of "double crush" in 1973. That concept suggests that a proximal compression makes a nerve more susceptible to a distal compression [6]. Lundborg further suggested the opposite: a distal compression makes the nerve more prone to a proximal compression [7]. Both theories can be explained by alterations in axonal transport, but neither theory has been proven unequivocally.

Treatment of these neuropathies varies a great deal as to timing of surgery and technique used. Mild entrapments usually are treated conservatively. Patient education is important to avoid further nerve irritation. An exercise program can optimize nerve gliding. Medication or therapy modalities can be used to decrease local inflammation, edema, and scarring. And finally, muscle strengthening is necessary to restore muscle balance.

Surgery is reserved for the more significant neuropathies. The importance of symptoms, physical findings, and electrodiagnostic studies weigh differently for different surgeons. Some do not intervene until there are significant changes, whereas others act simply because of a symptom complex. Often peripheral neuropathies are treated before TOS just because these surgeries are easier to perform and are less risky [4].

The authors' approach has been to treat the most severe compressions first. This strategy helps move the patient faster on the way to recovery. It provides early relief of some of the symptoms and possibly prevents the more affected nerves from sustaining permanent damage, which could be the risk of prolonged severe compression. By the same token, this approach potentially restores axonal flow at the level at which it is most impaired.

The authors do not favor multiple nerve decompressions at one sitting, because this is often overwhelming for a patient to handle. Rehabilitation often requires opposing goals for various nerves, and a patient rarely can cope with the amount of time required to rehabilitate multiple nerve entrapments simultaneously.

The severity of the compression is determined by a combination of factors, including symptoms, physical findings, and electromyography. All three must be taken into account, because a single finding could indicate only a partial entrapment. For instance, muscle atrophy without any symptom could indicate that only the motor fibers of a nerve are affected. The reverse also could hold true: a sensory disturbance without a motor deficit in a mixed nerve does not mean that that nerve is intact. One also needs to consider that muscle recovery beyond 18 months of denervation is not likely; these patients need earlier intervention.

Summary

The authors' approach to patients with TOS starts with a thorough evaluation, including a detailed history, physical examination, electromyography, and radiographs. Once the work-up is complete a treatment plan is formulated with the patient. All diagnoses are prioritized, together with various options for treatment of each. Therapy is initiated when appropriate and a surgical plan addresses the worst nerve entrapments sequentially.

The authors' favored procedure for TOS release is an anterior scalenectomy with epineurectomy, with decompression of any structures impinging on the brachial plexus. The authors encourage patients to attend therapy for approximately 3 months after surgery, and to follow a home program of rehabilitation to maximize their gains and decrease the chance of recurrence.

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