0749-0712/96 $0.00 + .20

ANTERIOR INTRAMUSCULAR TRANSPOSITION OF THE ULNAR NERVE

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Entrapment neuropathy of the ulnar nerve at the elbow has been recognized for 100 years.[37] Conservative therapy has been advocated for the treatment of ulnar nerve neuropathy[14, 15, 24] and the mainstay of treatment prior to the advent of surgical intervention. Several surgical modalities have been proposed for the treatment of ulnar nerve neuropathy at the elbow refractory to conservative management. Curtis[10] first published his case report in 1898 of surgical management for ulnar nerve neuropathy at the elbow. The operation described an anterior transposition of the ulnar nerve to a subcutaneous bed.

Adson,[1] in 1918, first described the technique of anterior transposition of the ulnar nerve into a flexor–pronator intramuscular bed. This technique now is one of five standard procedures for the surgical treatment of entrapment neuropathy of the ulnar nerve at the elbow. These five surgical techniques include in situ decompression, decompression in conjunction with medial epicondylectomy, and decompression with anterior transposition of the ulnar nerve to a subcutaneous, submuscular, or intramuscular bed. Other interventions for the treatment of cubital tunnel syndrome such as supracondylar osteotomy, deepening of the ulnar groove, or resection of neuroma[19] are only of historical significance.

Osborne,[15] in 1957, described ulnar nerve constriction at the aponeurosis bridging the two heads of the flexor carpi ulnaris muscle, now known as the *arcuate ligament of Osborne*. The term *cubital tunnel syndrome* first was proposed a year later by Feindel and Stratford,[16, 17] to describe ulnar nerve constriction at that ligament. The applied definition of the term has broadened to encompass ulnar nerve entrapment and compromise to intraneural microcirculation at the medial elbow.

Some authors have considered anterior transposition of the ulnar nerve to an intramuscular bed to be the optimum choice among surgical solutions for cubital tunnel syndrome.[1, 6, 19, 22, 23, 24] Transposition of the ulnar nerve to a new course anterior to the medial epicondyle of the humerus to a position within the substance of the flexor–pronator muscle mass places the nerve in a less vulnerable, more protected environment than deep only to the subcutaneous layer. Complications of the submuscular transposition, such as extensive scar formation and a potentially difficult revision ulnar neuroplasty, also are avoided.[22]

Anterior transposition of the nerve from its anatomic location in the retrocondylar groove and cubital tunnel to a new course decreases or eliminates nerve tension and avoids further irritation of the nerve. The new course of the nerve in the intramuscular bed is situated anterior to the flexion–extension axis of the elbow. The postural effect of elbow flexion, causing an ulnar nerve stretch, leading to a traction neurapraxia is eliminated because the retrocondylar groove no longer serves as a tensioning pulley for the nerve once the nerve has been transposed anteriorly.

Gay and Love,[19] in 1947, reported on the treatment of tardy ulnar nerve paralysis and published a series of 100 patients using Adson's procedure

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without the fascial sleeve. Their study considered cases performed over a 25-year period and they reported 70% satisfactory results. Their report of anterior intramuscular transposition and others,[6,7] however, all neglected to quantify their results and compare that technique to the other methods of anterior transposition.

Kleinman and Bishop,[24] in 1989, reported on 47 consecutive cases of ulnar nerve symptoms treated with intramuscular transposition performed over a 5-year period. The average follow-up period was 28 months. The findings included 87% excellent or good results, with return of normal grip strength and two-point discrimination. No patient in this series of 47 required reoperation because of cicatricial scarring. Since the initial report, the senior author has performed over 200 additional consecutive cases using the intramuscular technique following decompression.

Opponents to the technique of anterior intramuscular transposition cite the nerve's vulnerability to tensile stresses[4] or cicatricial scarring within the intramuscular bed.[20] Dellon et al[14] in 1986 demonstrated the in vivo (primate model) paucity of scarring when a nerve is transposed into a living muscle sleeve. The complication of dense scar formation was not reported in the study done by Gay and Love,[20] nor encounted in the study by Kleinman and Bishop.[25]

ETIOLOGY/CLASSIFICATION

Cubital tunnel syndrome caused by compression, friction, or traction at the five known sites along the course of the ulnar nerve at the medial elbow is well recognized. These sites include (1) the medial intermuscular septum, (2) the arcade of Struthers, (3) the retrocondylar groove, (4) the arcuate ligament of Osborne, and (5) the deep aponeurosis of the flexor–pronator mass. The various causes of cubital syndrome include osteoarthritis secondary to old fractures, occupational trauma, tumors, osteophytes, ganglia, or anterior dislocation or subluxation of the ulnar nerve. Rheumatoid arthritis is another potential cause of the cubital tunnel syndrome, but patients suffering from that disease often are omitted from study.[31]

Presenting symptoms typically include sensory dysesthesias in the hand along the distribution of the ulnar nerve. Weakness and clumsiness of the hand are more frequent symptoms than pain.[9] Postural nerve compression and irritation often occur with repetitive elbow flexion and produce the first intermittent paresthesias.

A differential diagnosis that includes other sites of ulnar nerve compression neuropathies must be entertained. The surgeon must rule out other causes of dysesthesias and weakness occurring along the cervical root C8–T1 distribution.[23] Thoracic outlet syndrome as well as ulnar nerve impingement at Guyon's canal at the wrist must be considered in the differential diagnosis because both can be responsible for ulnar nerve paresthesias and hypesthesias. Clinical examination will distinguish the level of the nerve lesion.

A motor nerve deficit may be noted first by the patient as a loss of dexterity or diminished pinch and grip strength. In advanced cases, the clinician may observe ulnar-innervated intrinsic muscle weakness and inability to abduct the small finger (Wartenberg's sign).[43] Muscle atrophy and clinical muscle weakness usually are not primary diagnostic indicators and may not be observed until months or even years after the onset of initial symptoms.[42] Because of internal topography of individual ulnar nerve fibers at the elbow, fibers innervating the intrinsic hand muscles course superficially, whereas fibers innervating the flexor digitorum profundus and flexor carpi ulnaris run more deeply in the nerve.[42] For this reason, a motor deficit in the flexor digitorum profundus or flexor carpi ulnaris rarely is elicited in cubital tunnel syndrome.

McGowan,[33] in 1950, developed a grading system based on the severity of lesions to assess the benefit derived from operative intervention: Grade I: Minimal lesions—symptoms include paresthesias in the ulnar nerve distribution and a feeling of clumsiness in the affected hand. No wasting or weakness of the ulnar intrinsic muscles noted. Grade II: Intermediate lesions—the interossei are weak; muscle wasting, typically obvious; some voluntary motor strength. Interossei are the most affected muscle group. Grade III: Severe lesions—the interossei are paralyzed; there is marked weakness of the hand. The cause typically is post-traumatic, arthritic, or congenital. The predictability of success with operative intervention coincides with the severity of McGowan classification.

In 1989, Kleinman and Bishop reported the results of 47 consecutive anterior intramuscular ulnar nerve transpositions.[25] The grading system they devised is seen in Table 1, and 87% of their patients had an excellent or good result.

ANATOMY

The course of the ulnar nerve at the medial aspect of the elbow renders it particularly vulnerable to compression, entrapment, and external trauma. The nerve passes in the arm from anterior to posterior through the medial intermuscular septum and then courses subcutaneously from the extensor mass of the arm to the flexor mass of the forearm via the medial epicondylar groove of the distal humerus. As the nerve exits the groove, it courses between the two heads of the flexor carpi ulnaris. A fibrous tissue band, the arcuate ligament of Osborne, lies directly over the nerve and spans the two muscle heads. This ligament originates at the medial epicondyle and has a mobile insertion at the olecranon.[35] The band tightens with elbow flexion and may compress the ulnar nerve. The

Table 1. ANTERIOR INTRAMUSCULAR TRANSPOSITION. KLEINMAN/BISHOP RATING SYSTEM

	Number of Points
Satisfaction	
Satisfied	2
Satisfied with reservation	1
Dissatisfied	0
Improvement	
Better	2
Unchanged	1
Worse	0
Severity of residual sympstoms (pain, paresthesia/ dyesthesia, weakness, clumsiness)	
Symptomatic	3
Mild-occasional	2
Moderate	1
Severe	0
Work status	
Working or able to work at previous job	1
Not working secondary to ulnar neruopathy	0
Leisure activity	
Limited	0
Unlimited	2
Strength	
Both grasp and pinch strength (opposition) ≥80%, compared with other hand	2
Either grasp or pinch (but not both) <80%	1
Both grasp and pinch less than 80%	0
Sensibility (static two-point discrimination)	
Normal (≤5 mm)	0
Abnormal (>5 mm)	0
	12

Evaluation of Results

Excellent	10–12 points
Good	7–9 points
Fair	4–6 points
Poor	1–3 points

structure thus created, the cubital tunnel, is an elliptical fibro-osseous canal bounded radially by the elbow joint capsule and the olecranon process and ulnarly by the aponeurosis of the flexor carpi ulnaris.[42]

Elbow flexion diminishes the cubital tunnel cross-sectional area and tightens the aponeurotic band on the ulnar nerve, resulting in cubital tunnel syndrome. Ulnar nerve entrapment, however, can occur both proximally and distally to this tunnel. Proximally, compression may occur at the medial intermuscular septum—i.e., a fascial thickness over the medial head of the triceps known as the arcade of Struthers,[23] or subsequent to an osseous irregularity at the medial epicondylar groove. Distally, compression may occur beneath the arcuate ligament of Osborne or by the deep aponeurosis of the flexor–pronator mass as it compresses the nerve beneath the muscle belly of the flexor carpi ulnaris.[2] Other sites of compression may include the ligament of Spinner described below.

The medial intermuscular septum of the arm must be released as the ulnar nerve passes from anterior to posterior at the junction of the middle and distal thirds of the humerus. The nerve passes from the brachial neurovascular bundle anterior to the septum and then beneath the arcade of Struthers posterior to the septum. If the septum is not released, anterior transposition may cause kinking of the nerve proximally.

The arcade of Struthers, known to be present in 70% of anatomical dissections, is a fascial thickening located 8 cm proximal to the medial epicondylar groove. When present, this structure must be released prior to anterior transposition of the ulnar nerve to avoid a potential source of recurrent nerve irritation.

The ligament of Struthers, found in no more than 1% of the anatomical specimens, lies anterior to the medial intermuscular septum.[23] Like the arcade of Struthers, resection of this structure is recommended when present if the ulnar nerve is to be transposed into a scar free, well vascularized bed.

The ligament of Spinner,[20] described in 1986, is the aponeurosis between the flexor digitorum superficialis of the ring finger and the humeral head of the flexor carpi ulnaris. This septum is independent of other aponeuroses and attaches directly to the medial surface of the coronoid process of the ulna. Failure to release the ligament of Spinner prior to anterior transposition may result in kinking of the nerve[22] or recurrence of symptoms.

PHYSICAL EXAMINATION

The ulnar nerve may be swollen or tender to palpation at the retrocondylar groove. Elbow flexion alone may elicit paresthesias. A positive Tinel's sign typically is present, although its findings do not have high sensitivity and specificity. Direct pressure on the nerve at the arcuate ligament also may precipitate paresthesias in the sensory distribution of the ulnar nerve.

Successful treatment of cubital tunnel syndrome treated nonoperatively has been reported.[9, 14, 15, 33] Craven[9] found that patients demonstrating intermittent symptoms in the absence of muscle atrophy did well with conservative therapy when sustained elbow flexion was avoided. Conservative treatment includes prolonged long-arm splinting at 40 degrees of elbow flexion, neutral forearm rotation, and 15 degrees of wrist extension; modification of activities at work and home; and oral nonsteroidal anti-inflammatory medication. Cases detected early—i.e., a lower McGowan grade—respond more favorably to conservative measures.[14] Nevertheless, no known conservative form of treatment has proven to be of long-term benefit to the patient.[14] The goals of conservative treatment are to reduce the inflammatory state of

the perineural tissues of the ulnar nerve, eliminate compromise to the normal hemodynamics of the intraneural microcirculation, and allow physiologic axonal transport.[23] Failure to meet these goals within a reasonable treatment period connotes chronic symptoms refractory to conservative therapy and the need for surgical intervention. These patients are potential candidates for anterior intramuscular transposition.

Although controversial, nerve conduction studies often can be useful to indicate the need for operative intervention. Kleinman and Bishop[24] reported that the severity of clinical signs and symptoms did not correlate well with objective electrodiagnostic changes. Dawson et al[11] reviewed multiple clinical series of cubital tunnel syndrome and found that reported abnormalities in nerve conduction velocities varied in incidence between 27% and 93% of cases among the different studies. Craven[9] reported routinely testing nerve conduction velocities and considers normal values found on testing to be a contraindication for surgery.

Kleinman and Bishop[24] noted that age, duration of symptoms, and conduction velocity were of little prognostic value. MacNicol,[31] in his retrospective study of 110 cases in 100 patients, however, concluded that the presence of symptoms for more than 1 year prior to surgical intervention significantly decreases the likelihood of satisfactory nerve recovery. These authors stressed the impor-

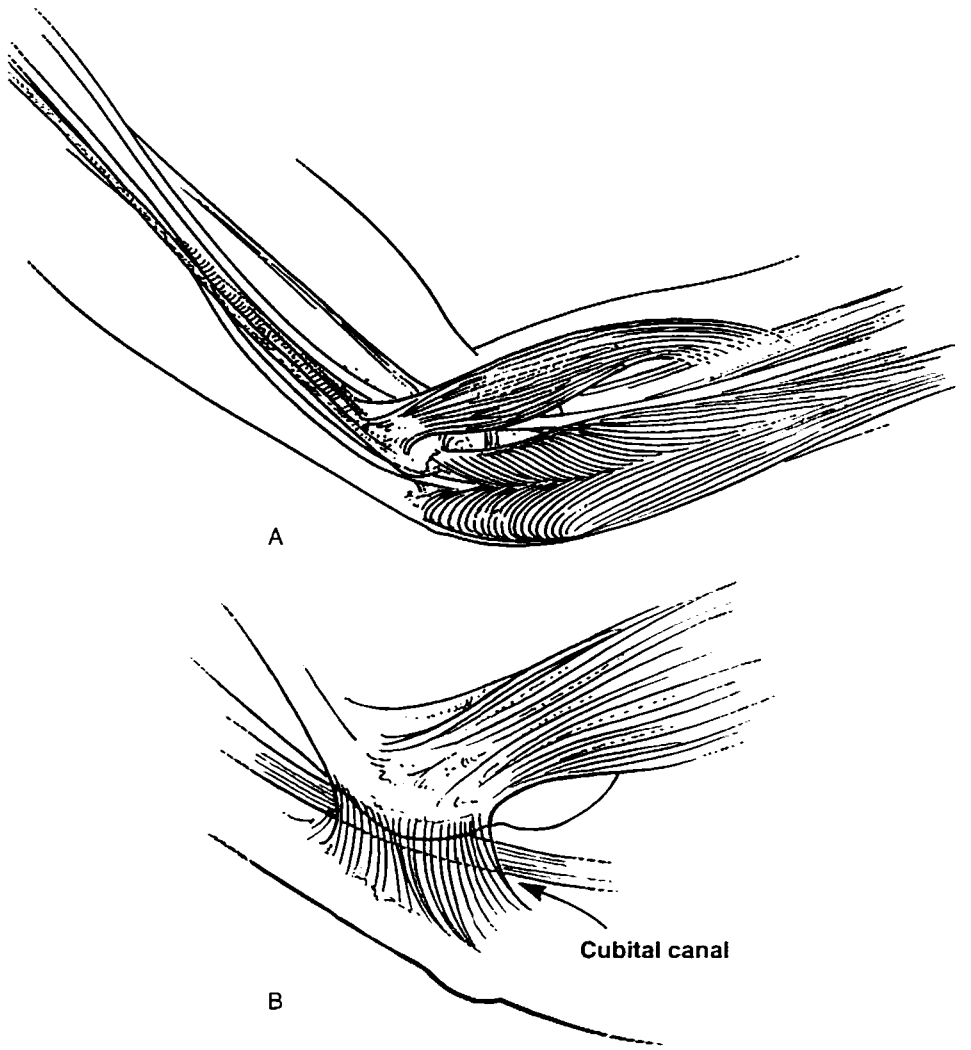

Figure 1. A, The pathway of the ulnar nerve at the elbow in relation to the median nerve and the medial intermuscular septum. In the retrocondylar groove, the nerve is covered by the arcuate ligament, proximal to its course between the humeral and olecranon heads of the flexor carpi ulnaris, deep to the enveloping fascia and septa of this muscle *(B)*. Courtesy of Gary Schnitz, The Indiana Hand Center.

tance of early diagnosis and implementation of conservative measures, to allow prompt evaluation of their efficacy. Should signs and symptoms prove refractory to conservative therapy, the authors felt postponing surgery carries little benefit.

SURGICAL TECHNIQUES

The surgical approach for neurolysis and anterior intramuscular ulnar nerve transposition is along a line equidistant between the medial epicondyle and medial border of the olecranon. The incision parallels the medial intermuscular septum approximately 10 cm proximal to the medial epicondyle. Distally, the incision runs approximately 10 cm from the medial epicondyle between the two heads of the flexor carpi ulnaris (Fig. 1*A*, *B*). The posterior division of the medial antebrachial cutaneous nerve must be identified and protected (Fig. 2) upon cutting the skin. Care must be taken throughout the length of the incision because the anatomic location of the posterior division of the median antebrachial cutaneous nerve varies widely. It may consist of up to three branches that may cross the skin incision anywhere from 6 cm proximal to 6 cm distal to the medial epicondyle.[13]

The ulnar nerve is identified proximally, posterior to the medial intermuscular septum and distally deep to the fascia overlying the two heads of the flexor carpi ulnaris. The arcade of Struthers is identified and divided and the nerve, with its accompanying blood vessels, is mobilized in a proximal-to-distal direction (Fig. 3*A*, *B*).[22, 23, 27] The arcuate ligament of Osborne is divided and the small articular branches of the nerve are sacrificed. All short motor branches to the ulnar head of the flexor carpi ulnaris should be spared by extraperiosteal mobilization of the muscle belly from the ulnar. The nerve is followed distally beneath the flexor carpi ulnaris to ensure its free mobility. Compression at this level has been observed.[7] Failure to release the fibrous deep flexor–pronator aponeurosis between the ring finger superficialis muscle belly and the flexor carpi ulnaris may result in ulnar nerve compression in its transposed position.[20]

In preparation for the nerve's transposition, the ligament of Struthers, when present, is resected.[22, 23, 27] The proximal border of the pronator teres fascia is excised, as is the medial intermuscular septum from mid-humerus to elbow. It now is possible to transpose the ulnar nerve anteriorly (Fig. 4*A*, *B*). The position of the nerve on the underlying flexor–pronator muscles is noted (Fig. 5*B*) and the nerve is replaced temporarily behind the medial epicondyle. A 5-mm-deep trough is fashioned in the flexor–pronator musculature in the line of the ulnar nerve in its anterior position. The fibrous septae separating the flexor–pronator muscles must be excised further to provide a soft, well vascularized intramuscular bed for the ulnar nerve (Fig. 4*C*, *D*). Then, with the forearm fully pronated and the elbow flexed 90 degrees, the flexor–pronator fascia is repaired over the nerve (Fig. 4*E*). When properly transposed, free excursion of the nerve within its intramuscular tunnel can be demonstrated (Fig. 4*F*). Closure of the skin is completed in routine fashion.

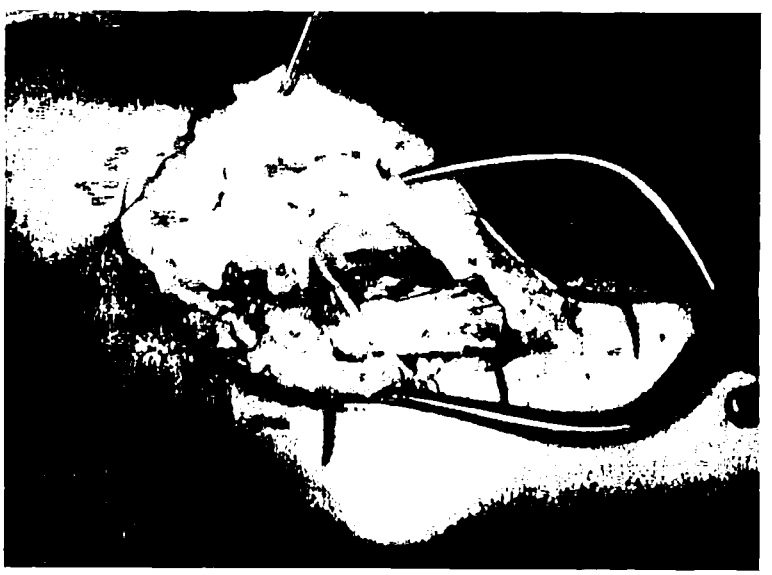

Figure 2. Surgical exposure of the medial aspect of the elbow (*left:* proximal; *right:* distal) demonstrates the position of the posterior branch of the medial antebrachial cutaneous nerve. Critical attention must be paid to the structure avoiding injury during the initial exposure of the ulnar nerve. Courtesy of Kevin D. Plancher, MD.

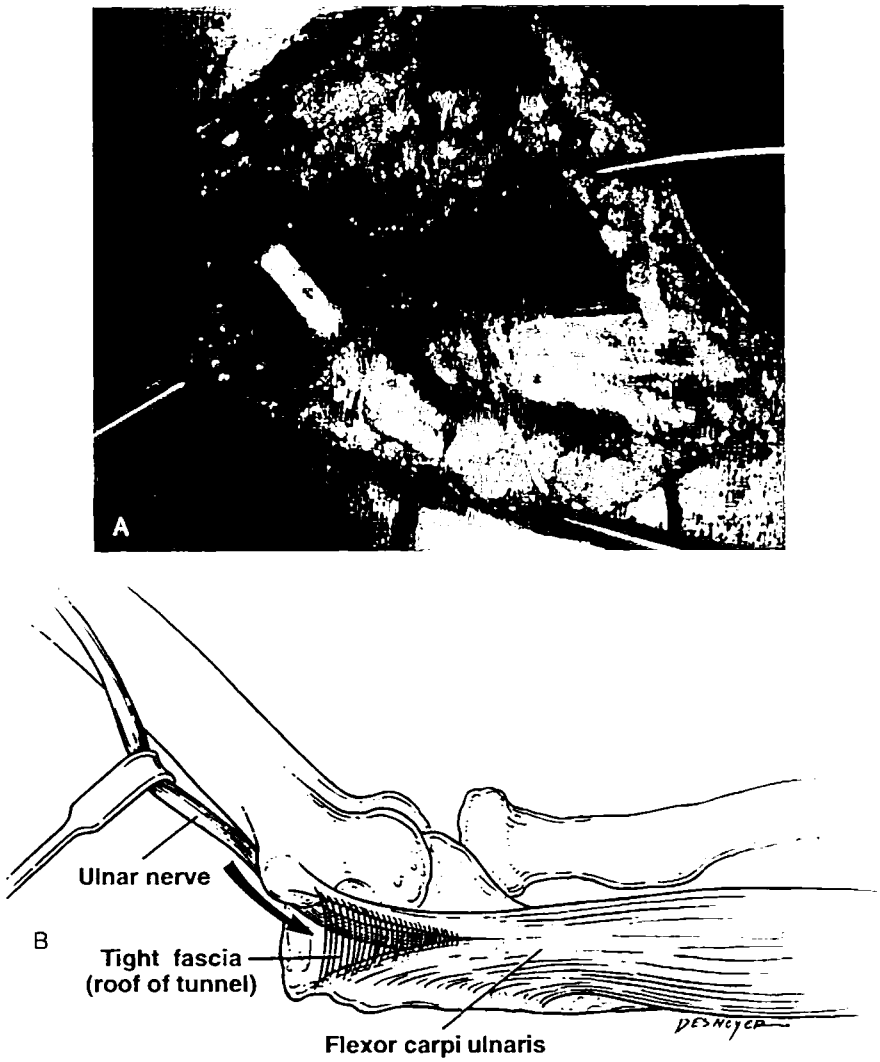

Figure 3. *A* and *B*, Medial aspect of the elbow (*left:* proximal; *right:* distal) with a Ragnell on the ulnar nerve after release of the medial intermuscular septum. The arcade of Struthers originates from the posterior aspect of the medial intermuscular septum in the distal third of the humerus, passing superficial to the ulnar nerve to insert on the deep fascia of the medial head of the triceps. Courtesy of Kevin D. Plancher, MD *(A)* and Desnoyer, The Indiana Hand Center *(B)*.

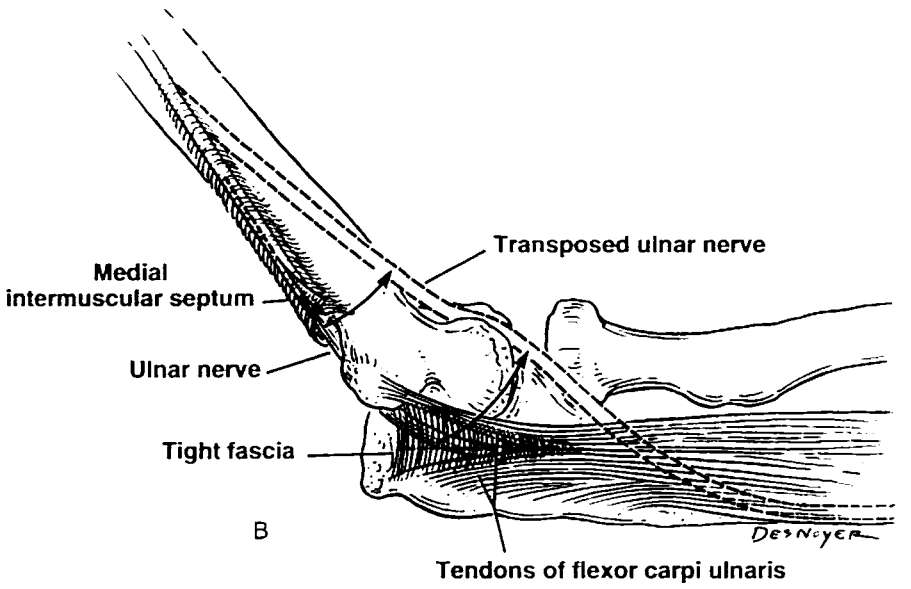

Figure 4. *A*, Once neurolysis of the ulnar nerve has been completed proximal and distal to the retrocondylar groove (including the medial intermuscular septum and fascia), the nerve is ready for anterior transposition. *B*, Adequate release of the ulnar nerve allows transposition without constrictions or acute bends; the ulnar nerve parallels the median nerve.

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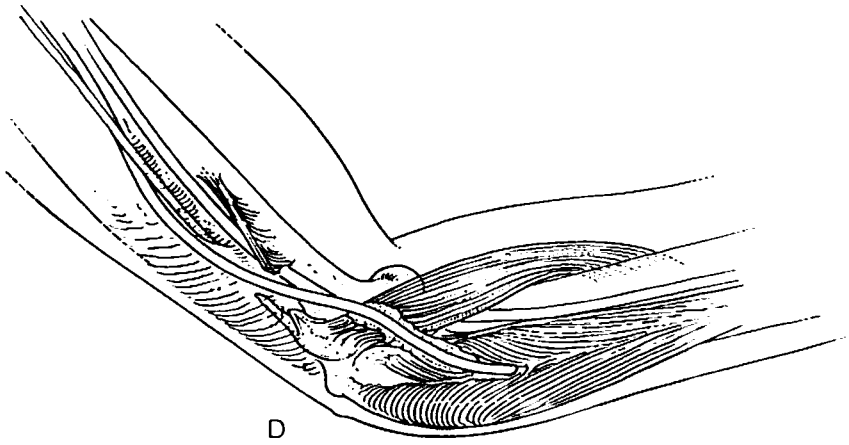

Figure 4 *Continued C, D,* A trough 5 mm deep made in the flexor pronator mass 4 cm distal to the epicondyle in line of the ulnar nerve; the ulnar nerve is then transposed anteriorly. Care is taken to sharply divide all fiber septa between the bellies of the flexor-pronator mass at this level. *E,* The fascia of the flexor pronator mass is then repaired anatomically with the elbow slightly flexed and the forearm pronated 45°. In this position, the ulnar nerve can easily move within the trough without constriction. *F,* Two Ragnells placed in the transposed ulnar nerve demonstrating a mobile nerve in its new transposed position. Courtesy of Kevin D. Plancher, MD *(A,C,E,F),* Gary Schnitz, The Indiana Hand Center *(D),* and Desnoyer, The Indiana Hand Center *(B).*

POSTOPERATIVE MANAGEMENT

The elbow is immobilized postoperatively in a bulky, compressive long-arm splint for the first 3 weeks. A long posterodorsal slab of plaster of Paris maintains the elbow in 90 degrees of flexion, the forearm in 45 degrees of pronation, and the wrist supported in 15 degrees of extension. After the third postoperative week, exercises consisting of active range-of-motion to elbow, forearm, and wrist are started. A long-arm splint assuring a comfortable resting posture for the elbow and forearm is kept in place between exercise periods and at night. After the sixth postoperative week, if needed, passive range-of-motion exercises are started, along with graduated arm-strengthening exercises. After the eighth postoperative week, most patients previously employed at manual labor jobs are able to return to work. After the 10th postoperative week, unrestricted activity of the affected elbow is permitted.

SUMMARY

The surgical management of cubital tunnel syndrome is well documented in the literature.[1, 6, 13, 15–18, 22, 23, 27] Anterior intramuscular transposition of the ulnar nerve is indicated for chronic cubital tunnel syndrome with symptoms refractory to conservative therapy. Prompt diagnosis is essential to yield excellent results.

Extreme care must be exercised in the performance of anterior intramuscular transposition. The surgeon must know the details of medial epicondylar anatomy and pathophysiology, as well as all possible sites of potential nerve compression. The placement of the transposed nerve in an intramuscular bed requires that all fibrous septae are resected from the shallow trough created for the nerve to avoid scar formation.

Postoperatively, the arm is immobilized for 3 weeks, after which range-of-motion exercises are begun. By the eighth postoperative week, most patients are able to resume their regular activities, including manual labor.

Recurrence or persistence of symptoms postoperatively typically is traced to an inadequate decompression of the nerve.[23] Common sites of persistent ulnar nerve compression include (1) the medial intermuscular septum, (2) the arcade of Struthers, (3) fibrous bands immediately proximal or distal to the cubital tunnel, (4) persistence or kinking at the arcuate ligament of Osborne, (5) Spinner's ligament or other fascial slings, and (6) incomplete anterior transposition.[23]

Anterior intramuscular transposition of the ulnar nerve is attractive for its relative ease of dissection, simplicity, reliability, and low morbidity.[13, 18] Transposition of the nerve into a shallow muscular trough deep only to the flexor–pronator fascia is a logical, effective, and consistently reliable method of treating cubital tunnel syndrome refractory to conservative management.

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