Failed Surgery for Ulnar Nerve Compression at the Elbow

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The elbow is the most common area for entrapment of the ulnar nerve. Although techniques available for revision decompression of the ulnar nerve at the elbow are similar to those used in the primary setting, results after repeat surgical intervention are less predictable \cite{1–5}. As ulnar neuropathy at the elbow is the second most common focal compression neuropathy in the upper extremity \cite{6}, familiarity with nonoperative, as well as surgical options, is paramount to optimize patient outcomes following failure of initial decompressive procedures.

Five potential sites for ulnar nerve compression in the elbow begin proximally at the arcade of Struthers and end where the nerve leaves the flexor carpi ulnaris (FCU) muscle \cite{7–9}. The most proximal and most distal sites rarely are involved. The second site, just proximal to the medial epicondyle is also rarely involved unless a valgus deformity of the elbow secondary to an old epiphyseal injury to the lateral condyle or a malunited supracondylar fracture is present. The third site, the epicondylar groove, and the fourth site, where the ulnar nerve passes under the fibrous arch connecting the two muscle heads of the FCU muscle, are the most common sites for compression. The causes of compression at the epicondylar groove are the most numerous of any site. They include lesions within the epicondylar groove, such as fracture fragments or arthritic spurs, and lesions that predispose the nerve to displace from the groove, such as developmental laxity of the fibroponeurotic covering of the groove or a posttraumatic deformity of the medial epicondyle.

Generally, surgical decompression of the ulnar nerve is necessary for patients with muscle weakness or persistent symptoms lasting 3 to 6 months despite nonoperative measures. The measures include avoiding resting on the elbow, particularly in a flexed position, and extension splinting of the elbow for 1 month. Earlier surgery is indicated when profound muscle weakness is accompanied by atrophy, which indicates that the problem probably has been present for years. The presence of muscle weakness is important and is the main indication for surgery, even when sensory complaints are mild. In the absence of muscle weakness, indications for surgery depend on the severity of pain and sensory complaints and the degree of disability. Only the patients can determine the severity of their disabilities, and when symptoms interfere with work or leisure time activities, surgery is recommended.

Five operative procedures are performed and are divided into two categories: (1) decompression of the nerve without transposition, which includes decompression in situ and medial epicondylectomy, and (2) decompression with transposition, where the nerve is positioned subcutaneously, intramuscularly (within the flexor-pronator muscle group), or submuscularly (deep to that muscle group). Each

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operative procedure is the subject of other articles in this issue, except for intramuscular transposition of the nerve. Most surgeons avoid that procedure because the positioning of the nerve within the muscle subjects it to traction forces that often result in increased pain, numbness, and muscle weakness. Outcomes and failure rates for the other operative procedures are well documented in the medical literature [10,11]; however, few prospective, randomized clinical studies are available for level I evidence-based guidelines [12–16].

Although the results of surgery generally are favorable, failures do occur [17]. A meta-analysis of 30 clinical studies suggests that success after any surgical procedure is partly dependent on the preoperative stage of compressive ulnar neuropathy [18]. A number of factors, some unrelated to the neuropathy itself, have been correlated with poorer prognoses. They include age older than 50 years; endocrine comorbidities, especially diabetes mellitus; neuropathies involving multiple peripheral nerves; and chronic neuropathy accompanied by muscle atrophy and poor sensibility [6].

Although opinions might differ regarding a precise definition of failed surgery for ulnar nerve compression at the elbow, few would disagree that failure includes subjective and objective worsening of the condition. Symptoms of numbness, paresthesias, and/or pain can be persistent, increased, or recurrent and can be related to inadequate initial decompression, formation of new sites of compression (ie, nerve kinking), or traction neuropathy (ie, nerve trunk adhesions to surrounding cicatrix) [19]. Clinical findings might include increased weakness of extrinsic and/or intrinsic muscles; persistent tenderness over the surgical scar; resubluxation of the nerve back into the epicondylar groove after transposition or subluxation of the nerve after decompression without transposition; medial instability of the elbow; and severe flexion or extension contracture of the elbow joint.

Causes of failed surgery

When surgery fails, it is important for the examining physician to determine the reason(s) for the failure. Numerous factors can be categorized as preoperative, intraoperative, and postoperative (Box 1).

Preoperative factors

Preoperative factors begin with the possibility that the patient’s original complaints and clinical findings were not caused by a problem involving the ulnar nerve at the elbow but rather by compression of another nerve (or other nerves) more proximally in the cervical roots, thoracic outlet, or brachial plexus or more distally in the forearm, wrist, or hand, including compression of the ulnar nerve in the canal of Guyon. Frequently, the original diagnosis is partially correct in that the patient has had compressive neuropathy of the ulnar nerve at the elbow that was properly treated; however, the patient might have had additional nerve compression(s) or other problems for which surgery might have been required. In some cases, the original problem was not neurogenic but rather a condition that resulted in ulnar nerve irritation and was misdiagnosed as a compressive neuropathy. The patient might have had an injury to the FCU muscle, causing some inflammation near its origin, or might have had medial epicondyliitis. Subluxation or snapping of the medial head of the triceps over the medial epicondyle with elbow flexion is another problem that can be misdiagnosed as subluxation of the ulnar nerve [20–23]. Although this phenomenon might eventually cause sufficient ulnar nerve irritation to require decompression, early treatment should address the abnormal medial head of the triceps with lateral transposition to the central tendon or resection [21].

The most common preoperative factor for failure is that the expectations for improvement after surgery were unrealistic. Failure to fully relieve numbness or improve muscle strength after decompression can be anticipated in the patient

Box 1. Factors contributing to previous surgical failure

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<th>Preoperative</th>
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with severe and chronic ulnar neuropathy. In that setting, the nerve often is so severely and permanently damaged with irreversible axonal changes that little chance exists for any significant improvement in sensibility or muscle strength independent of the surgical procedure. However, surgery might still be indicated as the only treatment with the potential to improve other complaints, such as pain and tenderness at the site of compression and dysesthesias in the hand. Obviously, patients with severe and chronic neuropathies should be apprised of the limited objectives of surgery, regardless of the operative technique recommended to decompress the nerve. If the objectives are not fully addressed preoperatively, patients who experience pain relief but not changes in sensibility and weakness after surgery might incorrectly regard the operation as a failure.

Intraoperative factors

Intraoperative factors usually comprise indirect errors of omission that result in inadequate surgical decompression or omissions in technique that are likely to lead to later problems. Less common are direct errors of commission where iatrogenic injury occurs to an important regional anatomic structure. Indirect surgical errors predominate.

Inadequate decompression of the regional anatomy

Failure to completely decompress the nerve can occur at any of the five potential sites for compression and occasionally occurs at more than one site [4,5,24]. In the two largest published series of revision ulnar nerve decompression at the elbow (30 and 20 patients, respectively), at least two levels, on average, were found to be compressed during reexploration [4,5]. The most common site is the origin of the FCU, where a fibrous arch rises from the medial epicondyle and olecranon that connects the humeral and ulnar heads of the muscle, respectively. The arch is commonly referred to as the ligament of Osborne, and the passage beneath the ligament is the cubital tunnel. Although ulnar nerve compression at the elbow often is referred to as cubital tunnel syndrome, the term actually refers to compression specifically at the cubital tunnel. The cubital tunnel is only one of five potential sites for compression. Therefore, in our discussion, we prefer to use the terminology failed surgery for ulnar nerve compression at the elbow rather than failed surgery for cubital tunnel syndrome. It is important with any of the ulnar nerve decompression operative procedures, with or without transposition of the nerve, that Osborne’s ligament is sectioned in its entirety together with the fascia distally over the FCU. The muscle heads can be safely separated for a distance of 3 to 4 cm.

The intermuscular septum frequently is cited as an area where nerve decompression is inadequate or incomplete [1–3,5]. The septum is a factor only when decompression of the nerve is combined with transposition. When the ulnar nerve is transposed, regardless of the technique used, its new course in relationship to the axis of ulnohumeral motion changes from posterior to anterior. Consequently, the nerve traverses the medial intermuscular septum where it can become kinked or compressed, resulting in a new problem. This complication can be avoided by excising the septum, particularly at its distal portion where it is thicker and wider than it is proximally.

Ulnar nerve instability

Other indirect surgical errors can result in postoperative instability of the nerve. The nerve can subluxate anterior to the medial epicondyle after decompression without transposition. Antoniadis and Richter [24] identified an anteriorly subluxated ulnar nerve in 50% of patients undergoing revision ulnar nerve surgery after failed decompression in situ. Alternatively, the nerve can shift back into the epicondylar groove after subcutaneous transposition if an adequate fascial sling is not constructed [1,2].

Direct surgical errors result from inadvertent injury to important anatomic structures, such as branches of the medial antebrachial cutaneous nerve, a motor fascicle(s) of the ulnar nerve to the FCU, and the medial collateral ligament of the elbow.

Injury to the medial antebrachial cutaneous nerve

Considerable variability exists in the anatomy of the medial antebrachial cutaneous nerve, particularly its posterior or olecranon branches. The branches are almost always encountered in any surgical decompression of the ulnar nerve. They vary in number from one to three and can be found anywhere from approximately 2 cm proximal to the medial epicondyle to 3 cm distal to the epicondyle [25]. Iatrogenic injury to one or more branches can result in neuroma formation that causes hypesthesias and hyperalgesia in the area of the olecranon. The symptoms can be so annoying that the patient compromises the beneficial results of the surgery for ulnar nerve compression [1,26,27]. The patient’s attention might be focused...
on the new elbow discomfort rather than on any improvement in sensibility and muscle strength. When the neuroma is directly over the ulnar nerve, a Tinel sign at that site can be misinterpreted as tenderness over the ulnar nerve itself, leading to a misdiagnosis of persistent compression or compression at a new site. Injection of 1 to 2 mL of a local anesthetic is a good diagnostic test. When the problem is neuroma of a sensory branch, treatment initially is nonoperative, consisting of local massage and the use of desensitization modalities. When symptoms persist (generally, at least 4 months is a reasonable time to wait), resection of the neuroma is warranted. The nerve branch is cut proximally away from the surgical area. Burying the cut end of the nerve in the triceps has been recommended [26,28]. Identification and protection of the medial antebrachial cutaneous nerve and its branches are therefore important during the primary operative procedure for ulnar nerve compression.

Injury to the ulnar nerve or its motor fascicles

Although iatrogenic injury to the main trunk of the ulnar nerve is rare, injury to a motor fascicle(s) to the FCU is more likely, particularly when decompression and transposition of the nerve are performed. All anterior transposition techniques require the nerve to be sufficiently mobilized to permit it to be shifted volar to the medial epicondyle. It is important to identify and protect branches of the nerve in the epicondylar groove and not assume that they are all articular branches that can be sacrificed. Motor fascicles to the FCU need to be preserved by interfascicular dissection performed with the use of loupe magnification [29].

Elbow instability

Iatrogenic injury to the MCL of the elbow can occur during medial epicondylectomy and possibly during submuscular transposition of the nerve, because the ligament is deep to the origin of the flexor-pronator muscle group. With a medial epicondylectomy, the anterior and posterior surfaces of the epicondyle are exposed by subperiosteal dissection without detaching the flexor-pronator muscle origin. Scoring the epicondyle before excision will reduce the risk of propagating the ostectomy site into the ulnohumeral joint or origin of the MCL. Care must be taken when performing an epicondylectomy because the anterior bundle of the ligament will be damaged if more than 20% of the epicondyle in the coronal plane is removed [30]. During submuscular transpositions, differentiating the MCL from the flexor-pronator muscle is best accomplished by color rather than by determining the direction of fibers. The red muscle mass is sharply elevated from the white ligament that extends from the anterior margin of the epicondyle to the ulna.

Postoperative factors

Postoperative causes for failed surgery include scarring around the ulnar nerve and elbow stiffness that usually presents as a flexion contracture.

Perineural fibrosis

Perineural scarring is difficult to quantify because it is observed only at the time of revision surgery, and, when significant, revision decompression is technically challenging. Perineural scarring can be patient specific and not the result of any surgical error; however, severe perineural scarring can occur because of inadequate decompression of the nerve or poor placement of the nerve after transposition. As previously noted, that is most likely to occur when the nerve is placed in an intramuscular groove within the flexor-pronator muscle [1,3]. The muscle fibers are at almost a right angle to the nerve, and the nerve is therefore subjected to repeated traction forces (Fig. 1). A similar problem can occur after submuscular transposition [5,31] when only a portion of the muscle is elevated: the entire muscle must be detached. Prominent perineural scarring has also been reported in the area of the medial epicondyle in patients who had undergone unsuccessful subcutaneous transposition of the ulnar nerve [2,4,32]. Scarring within the cubital tunnel after decompression without transposition and medial epicondylectomy can cause the nerve to adhere to the ostectomy site, preventing normal nerve excursion during ulnohumeral motion [1,32].

Elbow stiffness and flexion contracture

Postoperative elbow stiffness usually is the result of prolonged immobilization and is most likely to occur after submuscular transposition, which necessitates extended elbow immobilization to optimize healing of the flexor-pronator mass. In contrast, elbow immobilization after other techniques is usually for no more than 7 to 10 days. Elbow stiffness after submuscular transposition was encountered when the elbow was immobilized in 90° of flexion for 4 weeks or longer. In our experience, elbow stiffness has not
been a problem when postoperative immobilization is limited to 3 weeks with the elbow flexed no more than $30^\circ$ to $35^\circ$ [33]. Reattachment of the flexor-pronator muscle group to the medial epicondyle must therefore be secure to permit early active elbow motion. A secure method of muscle reattachment includes suturing the flexor-pronator muscle origin directly to the epicondyle through drill holes in the bone. Generally, four holes are drilled into the epicondyle in a sequential fashion beginning just proximal to the origin of the MCL and extending further proximally. In a patient with a large epicondyle, six holes are drilled. The four holes accommodate two mattress sutures (three mattress sutures when six holes are drilled) of a 0-grade braided synthetic material. The sutures are placed in the fibrous origin of the muscle, rather than in the thinner fibroaponeurotic covering of the epicondylar groove, and they are tied with the knots posterior to the epicondyle. In that fashion, the knots cannot be palpated beneath the skin in patients with thin layers of subcutaneous tissue. The fascia over the FCU and the fascial covering of the epicondylar groove are then closed with 3-0 nylon horizontal mattress sutures. This method of fixation is so secure that the elbow can be immediately and completely extended at surgery without any risk of disruption to the muscle reattachment.

**Diagnostic work-up**

**Examination**

The evaluation of patients after failed surgery begins with obtaining a thorough history that includes previous medical treatments. It is important to determine whether any change in
character, intensity, or periodicity of symptoms has occurred. Numbness and/or dysesthesias might remain unchanged postoperatively or might have decreased for a period of time only to have increased after several weeks or months. Patients might describe new symptoms in anatomic areas different from the site of surgery, indicating a new and different problem. Symptoms that have worsened postoperatively and are becoming progressively more severe are an obvious cause for concern.

The physical examination begins with the neck. Proximal neurological pathological conditions involving cervical nerve roots, components of the brachial plexus, or compression in the thoracic outlet must be excluded. Neuropathies involving other peripheral nerves also need to be considered. The ulnar nerve should be palpated along its course beginning in the upper arm, and its position in reference to the medial epicondyle with flexion and extension of the elbow should be determined. In cases in which the nerve had previously been transposed subcutaneously, it should remain anterior to the epicondyle and not course over the epicondyle (Fig. 2). In those cases where previous surgery consisted of decompression without transposition, the nerve should remain within the epicondylic groove with elbow flexion and should not shift anterior to the epicondyle or even onto the tip of the epicondyle. If the nerve does shift with elbow flexion, it is probably the reason for the failed surgery. In addition to determining the course of the ulnar nerve, it is important to determine whether any site(s) of tenderness is present over the nerve. Percussion at that site(s) usually produces Tinel sign with radiation of paresthesias distally and sometimes proximally. Paresthesia often radiates distally into the ring and little fingers, but sometimes the radiation of symptoms is confined to the forearm. Provocative testing can be helpful, and the sensitivity of simultaneous elbow flexion and direct digital pressure over the ulnar nerve (pressure-flexion test) has been established [34]. In some cases, tenderness might be localized to the surgical scar rather than to the ulnar nerve, indicating a possible neuroma of an olecranon branch of the medial antebrachial cutaneous nerve.

Sensibility is then evaluated. It is helpful to have the patient differentiate sensibility between the median and ulnar nerve distributions in the affected limb and to compare sensibility in those distributions with the uninvolved limb. Proximal sensory deficits in the forearm and upper arm should also be sought and elicited. Although evaluating sensibility is subjective and dependent on a patient’s responses to stimuli, an attempt is made to quantify those responses. Initial changes with nerve compression compromise thresholds for vibratory perception and light touch discrimination measured with Semmes-Weinstein monofilaments. Two-point discrimination reflects innervation density and is affected much later, when nerve compression has resulted in axonal degeneration. Therefore, with early nerve compression, threshold is abnormal whereas two-point discrimination usually remains intact. Thus, impaired two-point discrimination usually is a poor prognostic sign for recovery of normal sensibility. When it is absent, permanent numbness, to some degree, can be predicted. The prognosis for recovery is even worse when

![Fig. 2. (A) Previous operation was subcutaneous transposition. Examination showed that the ulnar nerve had shifted behind the medial epicondyle. Both the nerve and epicondyle were marked on the patient’s skin. (B) At surgery, the nerve was in the epicondylic groove and no indication that a fascial sling had been constructed to maintain the nerve after it was transposed was noted.](image-url)
two-point discrimination has deteriorated after the initial operative procedure.

Strength of both the extrinsic and intrinsic muscle groups is tested. This part of the examination is not totally objective because it depends on patients making maximal effort to contract the muscles being tested. For ulnar neuropathies at the elbow, the flexor digitorum profundus to the little finger most commonly is affected. The flexor digitorum profundus to the ring finger usually is not as weak, and can even be normal because of dual innervation of the flexor profundus muscle in the forearm by the median nerve. The FCU rarely is weak. In the hand, it is important to evaluate the first dorsal interosseous muscle, the last intrinsic muscle supplied by the ulnar nerve. The adductor pollicis commonly is weak, resulting in a positive Froment’s sign. Any muscle atrophy should be noted and is best documented by measuring the circumferences of the forearm and hand and comparing the measurements with the unaffected limb.

Electrodiagnostic studies

Electrodiagnostic studies almost always are obtained after failed surgery, and are an important part of the diagnostic work-up, especially when symptoms and muscle weakness have worsened. The studies are not nearly as useful for patients who report that their conditions have persisted but not deteriorated. Even with clinically successful surgery, electrodiagnostic studies might not show any postoperative improvement on the basis of permanent axonal damage secondary to long-standing nerve compression. With failed surgery, it is therefore common for electrodiagnostic studies to show persistent slowing of motor and sensory conduction and persistent electromyographic deficits; however, the studies should not show significant worsening of the parameters. Obviously, electrodiagnostic studies are most helpful when they can be compared with preoperative studies. Even in the absence of previous studies, electrodiagnostic studies can provide useful information. When additional surgery is contemplated, the site of nerve damage frequently can be localized by using the “inching technique,” especially when the site of electrodiagnostically evident compression corresponds to the site of clinical tenderness [35]. Electrodiagnostic studies also help to determine the presence of concomitant compressive peripheral neuropathy, cervical radiculopathy, or brachial plexopathy.

Electrodiagnostic studies should always be interpreted in the context of the clinical picture.

Grading system

Currently, no generally accepted grading system is available to evaluate results after ulnar nerve decompression. A system proposed by McGowan [36] in 1950 often is cited in the literature, but it focuses only on preoperative ulnar nerve function. The system consists of three grades: Grade I, mild neuropathies characterized by paresthesias and numbness but no weakness; Grade II, intermediate neuropathies with numbness and intrinsic muscle weakness; and Grade III, severe neuropathies with numbness and complete intrinsic muscle paralysis. The system essentially grades preoperative intrinsic muscle function. Despite its shortcomings, the system has been used to report outcomes after revision ulnar nerve decompression surgery at the elbow [1].

A more useful grading system is one that evaluates sensory and muscle functions both pre- and postoperatively. Although muscle function can be evaluated with a reasonable degree of objectivity, it is difficult to grade subjective symptoms. Symptoms that one patient might characterize as “mild,” another might consider to be “severe.” To minimize the problem, a system was developed at our institution that simply acknowledges the presence of a complaint but makes no effort to grade the severity of that complaint [37]. The three most common symptoms of ulnar nerve compression at the elbow are evaluated as: (1) local pain, including tenderness over the nerve; (2) numbness in any part of the ulnar nerve distribution of the hand; and (3) paresthesias in that same distribution. When only one of the three symptoms is present, regardless of its severity, it is assigned a grade of S-1; two symptoms are graded S-2; and all three symptoms, S-3. For muscle strength, extrinsic and intrinsic muscles are evaluated separately and each group is graded M-0 to M-3. M-0 represents no weakness; M-1, mild weakness; M-2, moderate weakness; and M-3, severe weakness with intrinsic atrophy that often includes clawing of the ring and/or little fingers. Postoperatively, the same grading systems are used. For symptoms, a grade is reduced to zero if the patient notes an improvement in that symptom. For example, a patient with a preoperative symptom grade of S-3 who reports that pain and tenderness at the elbow
and paresthesias in the hand have improved but that numbness persists would be graded S-1 postoperatively. Reduction in a symptom grade indicates a significant improvement in the symptom that only the patient can determine; it does not necessarily indicate complete elimination of the symptom. For muscle strength, the same system is used postoperatively, and again, extrinsic and intrinsic muscles are evaluated separately (Box 2).

**Indications for additional surgery**

An algorithm for the treatment of failed ulnar nerve decompression at the elbow has not been established. Revision surgery has been recommended based on motor conduction velocities that are less than 20 m/s [38]. Although slow conduction velocities are significant, especially if they have deteriorated since the preoperative study, we prefer to base the decision for additional surgery on a clinical picture of increasing deterioration of sensory complaints and motor function.

**Statistics of failed operations**

In published series of failed ulnar nerve decompressions, subcutaneous transpositions predominate as the most commonly performed primary procedure and account for 60% to 80% of failures [2–5,18]. The two largest series of revision ulnar nerve surgery were composed of 30 and 20 patients, respectively [4,5]. In a much larger series of 400 submuscular transpositions for ulnar nerve compression at the elbow performed at our institution in 374 patients (26 with bilateral disease), previous surgery had failed for 37 patients. In that revision group, 27 had previous subcutaneous transpositions, 7 intramuscular transpositions, 2 decompressions in situ, and only 1 submuscular transposition.

**Treatment of failed surgery**

Although nonoperative management, including activity modification, the use of an elbow pad, and extension splinting, can be attempted for failed ulnar nerve decompression, the results are not nearly as effective as when used for the primary treatment of the condition. Additional surgery is most likely necessary, and the same procedures recommended for primary surgery have also been recommended for revision surgery [1–5,33,38–43].

Decompression with transposition has been recommended when the previous operation was decompression without transposition. Pain relief and neurological improvement have been reported with relocation of a previously transposed ulnar nerve back into the epicondylar groove when reoperation shows that the nerve is compressed at the medial intermuscular septum [24,32]. Such surgery for the latter situation seems counterintuitive and is likely avoided by most surgeons. Almost all revision operations involve transposition of the nerve, and advocates for any of the three techniques can find support for their decision in the literature [1–5,33,38–43].

**Submuscular transposition for revision surgery**

Although subcutaneous transpositions have been recommended even when the same technique previously has failed [5] and intramuscular transpositions have also been advocated [38–41], most surgeons prefer submuscular transpositions when revision surgery is necessary [1–4,33,42,43]. At our institution, it is the procedure of choice for failed previous surgery and is also the most frequently performed procedure for primary surgical decompression of the ulnar nerve at the elbow. Submuscular transposition is a more difficult operation when performed as a revision procedure rather than as a primary procedure. A more extensive operative incision is required both proximally in the upper arm and distally in the forearm. An attempt is made to identify and protect branches of the medial antebrachial cutaneous nerve, which

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**Box 2. Ulnar nerve grading system**

**S-0 to S-3**

0, 1, 2, and 3 representing “present” or “absent”; ie, S-2 = 2 of 3 below present)

1. Local pain/nerve tenderness
2. Numbness in ulnar nerve distribution
3. Paresthesias in ulnar nerve distribution

**M-0 to M-3**

- M-0, No weakness
- M-1, Mild weakness
- M-2, Moderate weakness
- M-3, Severe weakness with intrinsic atrophy and clawing
usually are scarred in the subcutaneous tissues. The integrity of the medial cutaneous nerves of the elbow has been shown to impact clinical outcomes after revision surgery [27]. Neuromas of the medial cutaneous nerve branches are excised, and the nerves are cut back proximally and buried in muscle. Because extensive perineural scarring of the ulnar nerve is almost always present, particularly when the previous operation was intramuscular transposition, the nerve is first identified proximal to the previous operative area. Mobilizing the nerve can be technically challenging and is facilitated by using loupe magnification. It is important to detach the entire flexor-pronator muscle group. In the one patient in our series who had undergone previous submuscular transposition, only a portion of the muscle had been detached and the nerve was placed beneath it. The operation was, in effect, intramuscular transposition rather than submuscular transposition (Fig. 3).

When detaching the flexor-pronator muscle, its proximal margin should first be identified and the fascia along that margin incised, facilitating identification of the underlying brachialis, an important landmark in submuscular transpositions. Identifying the brachialis before detaching the flexor-pronator muscle group from the medial epicondyle helps to avoid the risk of inadvertently dissecting in an incorrect plane deep to the brachialis. To ensure that the ulnar nerve is not kinked or compressed distally, the origin of the FCU should be released from the ulna for a distance of approximately 2 cm distal to the insertion of the MCL. The surgical objective is to create a near-linear course for the ulnar nerve from the upper arm into the forearm. The ulnar nerve frequently is enlarged and its epineurium thickened at the site(s) of compression. The white glistening appearance and longitudinal striations of the fascicles of a normal nerve often are absent, especially when previous surgery included silicone sheathing of the nerve (Fig. 4). Epineurolysis of the nerve at that site should be performed with the use of loupe magnification. Reattachment of the

Fig. 3. (A) Although the previous operation was listed as “submuscular transposition,” only a portion of the flexor-pronator muscle was detached from the medial epicondyle. The ulnar nerve had then been inserted into the split in the muscle, entrapping it. (B) Entire flexor-pronator muscle was detached, and bone (at probe) was noted encircling the ulnar nerve. (C) A bone cutter was required to remove the bone. (D) When released, the ulnar nerve was compressed over a distance of several centimeters. Postoperatively, elbow pain and dysesthesias in the hand were relieved but numbness and muscle weakness persisted.
Flexor-pronator muscle is the same as for primary submuscular transposition, as are the method and duration of postoperative immobilization.

Results of submuscular transposition for revision surgery

The literature on outcomes after submuscular transposition as a revision technique is limited, but our review suggests that submuscular ulnar nerve transposition more reliably yields improvement in pain symptoms than it does in sensibility and motor function [1–4,33,42,43]. In our series of 37 patients whose previous surgery had been unsuccessful, revision submuscular transposition was beneficial for all patients. Pain and paresthesias decreased, although numbness persisted to some degree. Muscle strength, primarily involving the extrinsic muscles in the forearm, improved in 25 patients.

Adjunctive techniques to prevent recurrent epineural fibrosis

Various techniques have been used in an attempt to reduce perineural scarring after
revision surgery. The use of synthetic materials, such as silicone and a silicone-polymer material, to sheath the nerve have yielded poor results, as shown in our illustrated case [33,44]. Greater success has been achieved with autologous vein grafts [45–50]. Several groups have shown histologically that autologous femoral vein graft barrier-wrapping of the sciatic nerve results in significantly less epineural scarring and almost no adherence between the epineural layer of the nerve and the intimal layer of the vein in a rat model [51–53]. In addition, autologous vein grafting is associated with significantly less inflammatory response when compared with allograft vein wrapping [51]. The technique of autologous saphenous vein grafting has been successful in cases of recurrent compressive upper extremity neuropathies after previous surgical decompression [46–50]. The vein might provide a circumferential insulation-like effect on the nerve, inhibiting scar formation, and might improve nerve gliding and excursion with elbow motions. Although the results of autologous vein wrapping seem promising, larger studies with long-term follow-up are required before the technique can be recommended for all cases. Donor site morbidity for the vein graft remains a concern. Local interposition techniques using a triceps muscle flap and a pedicle fat flap also have been described [2,54].

Pain management

Rarely is additional surgery indicated for the patient who has undergone two or more decompressions that failed. Such a patient might be better served with a comprehensive pain management program that includes the use of pharmacological agents and a peripheral nerve stimulator. Stimulators often are effective in recalcitrant cases of ulnar nerve neuropathic pain not amenable to further surgical intervention [55]. The devices deliver continuous, high-frequency electrical stimulation to the involved peripheral nerve and are thought to ameliorate pain symptoms by modulating the gate control mechanism of peripheral nerve physiology.

Summary

Unsatisfactory clinical and subjective patient results after primary ulnar nerve decompression at the elbow remain challenging problems for the upper extremity surgeon. We have found it useful to distinguish potential causative factors contributing to failed decompression into preoperative, intraoperative, and postoperative categories. A thorough history and physical examination combined with electrodiagnostic studies help to identify the cause(s) of failure and location of pathological abnormality before revision surgery. When revision decompression is indicated, patients must be informed preoperatively that both patient-based subjective outcomes and objective postoperative improvements in sensory and motor functions might be more variable than results anticipated in the primary setting. A frank discussion with the patient regarding the goals of surgery and realistic expectations is imperative.

The literature on outcomes after revision surgery for compressive ulnar neuropathy at the elbow remains limited to relatively small retrospective cohorts. Only five series reporting results after revision surgery have been published [1–5]: four [1–4] support the use of submuscular transposition in the revision setting, whereas one series [5] recommends subcutaneous transposition. Although our results support decompression with submuscular transposition as both an index and revision technique, prospective, randomized studies with long-term follow-up are needed to ascertain which technique best optimizes outcomes in the revision setting.

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References


