Anterior submuscular transposition of the ulnar nerves by the Learmonth technique

Thirty-eight patients with progressive posttraumatic ulnar neuropathy at the elbow underwent anterior submuscular transposition of their nerves. Multiple mechanical causes of neuropathy were identified. Fourteen patients had undergone previous surgery for ulnar neuropathy, while 24 had not. Postsurgical follow-up averaged 23.1 months. The operative technique is described and illustrated in detail. Complications attributable to surgery were minimal. No absolute prognostic factors could be identified, and even those patients with significant muscular atrophy or time delay before operation were generally benefited. If prior surgery had induced significant scarring and neural damage, the prognosis for recovery was considerably worse, as it also was for patients who had severe preoperative dysesthesia or pain.

Four patients thought to represent examples of double crush or compression syndrome were identified.

Robert D. Leffert, M.D. Boston, Mass.

Because of its anatomical location on the subcutaneous extensor surface of the elbow, the ulnar nerve is vulnerable to a variety of insults, any one or combination of which can result in progressive neuropathy. Although the functional deficits in the hand are easily appreciated by patients and recognized by their surgeons, the precise identification of the pathomechanical factors causing the neurological abnormalities may not always be possible.

A list of the potentially injurious conditions resulting in ulnar neuropathy at the elbow has been advanced by Apfelberg and Larson1 and it is quite useful, with minimal modification, when attempting to define the etiological spectrum of the lesion. The list, as follows, includes: (1) bony trauma caused by fracture of medial epicondyle or 1° nerve injury fracture of ulna; fracture of lateral epicondyle, supracondylar fracture 2° carrying angle? etiology, or tardy ulnar palsy; lateral dislocations; collateral ligament injury (short- or long-term with instability); or posttraumatic arthritis; (2) compression as seen after anesthesia; in patients bedridden over a long period; in occupational or habitual external compression; after crush injuries with loss of elbow motion; or after contusion; (3) other miscellaneous injuries or abnormalities such as ganglions or synovial cysts; congenital anomalies of the bony or soft tissue, including epitrochleocaneous muscle and Arcade of Struthers; lacerations or foreign bodies; toxic, metabolic, or nutritional neuropathy; burns; or hemorrhage; (4) habitual subluxation or dislocation; and (5) cubital tunnel compression.

In 1898 Curtis2 published the first successful report of an anterior transposition of the ulnar nerve, and for the past 82 years the literature has been replete with diverse descriptions of surgery designed to either modify the relationship of the ulnar nerve to the olecranon groove and its soft tissue environs or to remove it from its bed entirely.3–14 This paper does not attempt to directly compare all variations of these operations. A review of the numbers of cases contained in most reports and differences in the methods of evaluation and documentation makes it quite clear that this approach is difficult to employ in planning for an individual patient.

In the past 8 years I have performed 52 anterior transpositions of the ulnar nerve for neuropathy not involving nerve suture or graft. In all cases the indication for surgery has been progressive loss of ulnar nerve function, with the cause localized clinically and ulnar electrodagnostic methods to the region of the elbow. Whenever possible, I have used the method described by Learmonth in 1942,8 wherein the nerve is rerouted anteriorly beneath the flexor-pronator muscles to lie on the brachialis fascia. Patients who have had subcutaneous transpositions and those with underlying generalized peripheral neuropathy or inadequate follow-up have been eliminated from analysis, leaving 38 patients
who are the basis for this report. All have been examined by me at regular intervals until they have either returned to normal or their conditions have stabilized. Follow-up has ranged from 3 to 86 months, with an average of 23.1 months.

This report is a summary of the experiences of treating these 38 patients using the Learmonth technique of submuscular anterior transposition of the ulnar nerve.
Fig. 5. Detachment of the flexor pronator muscles from the epicondyle. The ulnar nerve is carefully retracted.

Fig. 6. The nerve has been transposed next to the median nerve. All sutures have been placed before tying.

Fig. 7. The flexor pronator musculature has been replaced. A gap has been left in the flexor carpi ulnaris next to the bone to avoid constriction of the nerve by resutured muscle.

One patient dated the onset of symptoms to a surgical procedure with general anesthesia for an unrelated problem and one developed ulnar palsy after hemorrhage on anticoagulants. Fourteen patients had undergone previous surgery on their ulnar nerves elsewhere including four simple decompressions or explorations, nine subcutaneous anterior transpositions, and two intramuscular anterior transpositions.

The total of reoperations, fifteen, exceeds the number of patients by one because one patient had two previous operations on the ulnar nerve. In all cases the neurological deficit for which the surgery had been done continued to get worse until the time of reoperation. The findings of two patients at surgery have already been reported in a previous communication, but will be included in the section on results.

The time of onset of symptoms prior to submuscular anterior transposition of the ulnar nerve in the 38 patients ranged from 3 weeks to 10 years (Table I).

Manual muscle testing usually disclosed some degree of weakness and atrophy of the interossei and hypothenar muscles, with progressively less impairment of the flexor digitorum profundus of the small and ring fingers and least of the flexor carpi ulnaris. Sensory examination tested appreciation of light touch, pinprick, and two-point discrimination, which was usually impaired over the small and ulnar half of the ring finger as well as the corresponding area of the palm and dorsum of the hand. All patients were appropriately screened for the possibility of generalized peripheral neuropathy, mononeuritis multiplex, or more proximal compression lesions. Because their data were felt to be germane, only the last category of patients was retained in this study; they will be identified and their clinical courses will be discussed subsequently.

Twenty-eight patients had electrodiagnostic studies that consisted of either needle electromyography and/or ulnar nerve conduction velocity determination. For the latter study, a velocity below 45 m/seconds at the elbow was considered pathological. Normal conduction velocity for the nerve segment from above the elbow to the wrist is usually 61.4 ± 6.5 m/sec. Twenty-two patients had abnormal nerve conduction velocity determinations: eleven had abnormal EMGs. There were four patients with normal nerve conduction studies and evidence of denervation on EMGs of ulnar innervated muscles. No patient was operated on if normal EMGs and nerve conduction velocity determinations were obtained. F responses were not routinely determined.

The range of motion of the elbow was measured in all patients pre- and postoperatively. Six patients had limitation before surgery; none of them had flexion
contractures greater than 30°. Collateral stability of the elbow was also assessed and in six patients there was significantly increased mediolateral laxity when compared to the other side. Carrying angle on the side of the lesion was found to exceed that of the normal elbow by 15° in eight patients.

Subluxation of the ulnar nerve with elbow flexion was demonstrable in nine patients. Combinations of these abnormalities existed in several patients.

Radiographs of the elbow were taken in all cases and 12 showed abnormalities. Four patients had significant degenerative joint disease, one had a radial head resection 20 years previously, two had deformed radial heads, and one a nonunion of the lateral condyle. One patient had a malunited medial condylar fracture and two had healed olecranon fractures.

Calcification of the soft tissue surrounding the ulnar nerve was seen in radiographs of one patient who had had "decompression" with unrelenting progression of neurological loss prior to reoperation.

To try to relate the data to a comprehensive matrix, we can apply patient data that includes definite etiological or pathological information to the classification described earlier.

Additionally, although the classification of the severity of the neuropathy advanced by McGowan is imperfect, it has the virtue of simplicity. She graded patients clinically as follows: grade I, minimal lesions with no detectable motor weakness of the hand; grade II, intermediate lesions; and grade III, severe lesions with paralysis of one or more ulnar intrinsic muscles. Thus, of the 38 patients in this series, two would be classified grade I, 30 would be grade II, and six would be grade III according to McGowan.

**Indications and technique**

All patients were evaluated as described and if there was any question regarding the progressive nature of the nerve deficit, 1 to 2 months of observation with repeat examination were required before surgery was recommended. During this time several patients, especially if their neuropathy had followed an acute episode and was recent, were treated by means of muscle immobilization and oral nonsteroidal anti-inflammatory agents for 3 to 4 weeks; none showed significant improvement. Only occasionally have these conservative measures proved successful in reversing a progressive ulnar neuropathy to produce a complete resolution of the problem.

The operative technique is essentially that described by Learmonth in 1942 (Figs. 1 to 7). Several points deserve emphasis since their neglect results in avoidable complications.

The skin incision should be centered 1.5 cm posterior to the medial humeral epicondyle and as longitudinal as possible to avoid injury to the medial antebrachial cutaneous nerve. Such an injury would produce a locally tender neuroma and a variable area of sensory loss over the proximal-medial forearm (Fig. 1). Because of a tendency to local variation in the path of this cutaneous nerve, it should be searched for and protected since it may cross the skin incision. Location of the ulnar nerve is facilitated, especially in cases where there has been scarring or previous surgery, if it is sought proximally in relation to the medial triceps and distally in unscarred tissue. It should be retracted only by means of soft rubber or plastic strips. As many of the accompanying vessels as possible should be allowed to remain with the nerve as it is mobilized and only a bipolar microcoagulator or small ligatures should be used to ensure hemostasis. The medial intermuscular septum must actually be excised for 5 to 8 cm proximal to its attachment to the epicondyle to ensure that the transposed nerve will not rest against its edge and cause a new site of compression (Fig. 2). In this way, compression against an Arcade of Struthers is also prevented. Care must be taken to avoid the numerous blood vessels that underlie the intermuscular septum and which are part of the extensive collateral circulation at the elbow.

As the nerve is mobilized distally in the olecranon groove, the first (articular) branch to be encountered enters the joint, and it may be sacrificed with impunity (Fig. 3). That it is not a muscular branch can be verified by means of a nerve stimulator, as can the first significant branch below that (to the flexor carpi ulnaris). When the fascia overlying the nerve is split and the humeral and ulnar heads of origin are separated, the flexor carpi ulnaris branch will be apparent and dissection need go no further unless the transposed nerve appears to take a sharp bend. It will be necessary to lyse the epineurium around the first muscular branch up proximally into the substance of the nerve for about 2 cm, which can be done safely under magnification (Fig. 4). Without this releasing maneuver the mobilization of the nerve will end abruptly and cause a kink at the level of the branch.

The detachment of the flexor pronator muscle mass is generally done without difficulty unless there is a congenitally high origin of the muscle. This was the case in one patient in whom it rose to the level of a prominent supratrochlear process. To avoid trauma to the muscle,
or burying the nerve partially in muscle, a subcutaneous transposition was done. When there was significant scarring or distortion of the elbow joint capsule due to a malunited fracture, submuscular transposition was considered advisable because of the possibility of secondary involvement of the transposed nerve. The same contraindication resulted in subcutaneous transpositions in patients undergoing excisional arthroplasty of the elbow. Finally, very obese or sedentary patients were treated by subcutaneous rather than submuscular transposition. I have not done the procedure in a professional athlete. In uncomplicated situations, the median nerve as well as its first branch to the pronator muscle and the brachial artery were then identified and protected, and the bed for the ulnar nerve was prepared in a position immediately medial to them.

While in a few cases I have detached a flake of bone of the medial epicondyloge with an osteotome along with the flexor pronator muscles, the vast majority of the muscles have been detached by cutting through 1 cm from the bone, leaving sufficiently strong soft tissue to serve for reattachment (Fig. 5). The plane of dissection is readily apparent, although a few fibers of the flexor pronator muscles may require blunt dissection with a periosteal elevator. Both the ulnar and median nerves have to be carefully protected during this maneuver, at the end of which it should be possible to transpose the ulnar nerve forward of the epicondyle in a gentle curve that is 2 cm in advance of where the suture line for muscular reattachment will lie (Fig. 6). I have no personal experience with epicondylectomy alone as treatment for ulnar neuropathy.

At this point the sutures for reattachment of the flexor pronator muscles should be placed with the ulnar nerve under direct vision at all times, lest it become caught up by one of them. This potential complication is avoided by not trying any of the sutures until all have been placed. Nonabsorbable braided 2-0- or 0-gauge suture has been used in most cases (Fig. 7). Before the sutures are tied, however, the tourniquet is removed and hemostasis is assured. The forearm is then pronated 45° past neutral and the elbow is flexed to 90° to assist in approximation of the muscle. It is unnecessary to fully pronate the forearm to achieve sufficient relaxation of the reattached muscle, and in two cases where the procedure was done in elderly patients, they complained of transient pain in the distal radio-ulnar joint during the postoperative period. The position of the elbow and forearm must be maintained by an assistant until the wound is closed and a posterior splint has been applied. The wrist is immobilized in 20° of flexion and

free motion of the fingers is encouraged. The technique for those patients without previous surgery on the ulnar nerve or its environs was, invariably, as described above. For the 14 patients who had undergone previous operative procedures it was necessary to modify the technique as dictated by the findings. These will be considered for the two groups separately.

**Findings**

**Patients without previous surgery.** Patients without prior surgery had a surprising variety of injury that ranged from no visible lesions of the nerve to marked scarring. In seven out of 24 uncomplicated cases there was significant hyperemia, thickening, and fixation of nerve (Fig. 8). One patient with marked osteoarthritis at the elbow had a large osteophyte around which the ulnar nerve was kinked every time the elbow was flexed, and distal to this point the nerve was severely constricted. One patient definitely dated the onset of his ulnar neuropathy to general anesthesia for a surgical procedure. He had an essentially normal appearing nerve. At the time of the operation it was not always possible to be certain of the etiology and pathogenesis of the neuropathy. In the eight patients with subluxating nerves there was no uniformity of local pathology in the nerve—some patients had no visible change, but only a palpable increase in resistance on palpation along the nerve, which changed to normal consistency both proximally and distally. It was also difficult to accurately assess the degree of compromise of the cross-sectioned area of the cubital tunnel in those patients with no other obvious lesions involving the nerve. One 40-year-old patient with a 2-year history of numbness and clawing of the small finger after a closed fracture of the adjacent fifth metacarpal proved to have an ulnar neuropathy due to an anomalous muscle slip.
extending from the triceps to the epicondyle and compressing the nerve.\textsuperscript{17}

**Patients with previous surgery.** The 14 patients who had previously undergone surgery for ulnar neuropathy at the elbow at other institutions all had scarring of the nerve and a variety of lesions that could often be related to the technique with which the surgery had been done. All of the six patients who had subcutaneous transposition without resection of the medial intermuscular septum had marked tenderness and a Tinel’s sign localized to the area just above the epicondyle preoperatively. At operation the nerve was always found to be kinked across the septum where it had been brought forward. In one patient whose nerve had also been buried in the flexor pronator muscles the resulting scarring was so extensive that a 2-hour external neurolysis was required (Fig. 9). Another patient, who also had the nerve buried in muscle, required extensive external lysis and epineurectomy even though the septum had been removed. Where fascial slings had been constructed a significant amount of scar was encountered and, in a few cases, additional scarring due to large nonabsorbable suture material was seen. The two patients who only had the nerve explored at the time of fixation of fractures were found to have the nerve adherent to the healed fracture sites. One patient whose operation was limited to a decompression at the elbow had a strikingly hypermobile but scarred nerve with actual calcification of surrounding tissues that was visible on the x-ray films (Fig. 10). One patient, whose lesion followed an anticoagulant hemorrhage, had an extraordinary degree of scarring in all compartments of the limb and a poor clinical course after excellent initial improvement lasting 14 months.

In all cases where there was significant scarring or opacity of the epineurium, an external neurolysis and epineurectomy was carried out. In no case was an internal neurolysis done.

Postoperative management was identical in all cases, with immobilization of the elbow, forearm, and wrist continued for 3 weeks. Shoulder exercises were encouraged, especially in elderly patients, although a sling was usually worn for the first week or two.

**Results**

Twenty-four patients had had no prior operations for their ulnar neuropathy. Their preoperative rating, according to McGowan, was as follows: grade I, 1 patient; grade II, 20 patients; and grade III, 3 patients.

Fifteen of these patients ultimately achieved complete resolution of their motor and sensory deficits and had essentially normal limbs with no symptoms of ulnar neuropathy. This group included two of the three grade III patients, one of whom had had symptoms for 10 years. The third patient with grade III neuropathy and severe pain was the one who had severe scarring due to hemorrhage while on anticoagulants. He made excellent recovery both objectively (in terms of resolution of his neurological signs) and subjectively, with relief of pain until 14 months after surgery when he began to regress: he ultimately reverted to his preoperative status. One grade II patient achieved normal motor and sensory status but had occasional paresthesias.

Two patients, both grade II, ultimately achieved normal motor and sensory status but persisted in slight clawing of the small finger. One had had the neurological defect for 15 months preoperatively, the other for 24 months.

Two grade II patients, despite normal neurological examination at final evaluation, alleged significant pain and disability. Both were worker’s-compensation cases and one related that his attorney had advised him to stay out of work for at least a year.

One grade II patient, although neurologically normal at 6 months, developed pain in the operative site without any positive findings and was ultimately thought to have cancerophobia.

A grade II patient who had sustained blunt trauma to his elbow at work had mild restriction of motion. Loose
bodies had been removed from this joint 7 months previously and he had a subluxating nerve. Electrodiagnostic studies indicated the cubital tunnel as the locus of neuropathy and at operation the nerve was found to be quite hyperemic. A technically uneventful submuscular transposition resulted in rapid resolution of the motor deficit but also in the prompt emergence of an occult thoracic outlet syndrome with positive Wright’s maneuver and hypesthesia over the C8-T1 dermatomes. Noninvasive vascular studies confirmed the diagnosis, but the patient elected to change occupations; since he no longer does heavy lifting he is asymptomatic and pleased with the result. Possibly the abducted arm position at operation combined with 3 weeks lack of use was sufficient to unmask a subclinical thoracic outlet syndrome.

An elderly grade II patient, who had significant weakness of profundi of the small and ring fingers as well as intrinsicis, had achieved complete recovery of long flexors and significant improvement in the interossei but died of other causes 7 months after the operation.

Of the 14 patients who had undergone a previous operation on their ulnar nerves, none were rated grade I before submuscular transposition, 11 were rated grade II, and three were grade III. The last group was of interest in that two of the three patients had had their nerves explored and left in situ during open reduction and internal fixation for fractured olecranonos. The ulnar neuropathy present since injury became progressively more severe after the open reduction, despite healing of the adjacent fracture which compressed the nerve with callus. However, both nerves showed improvement after submuscular transposition, leaving one patient (a 14-year-old girl) with a normal hand, and the other (a 40-year-old iron worker) with slight intrinsic weakness and slightly impaired sensibilities. The last patient in this group, a 73-year-old woman with a 27-month history of ulnar neuropathy and a subcutaneous transposition 13 months prior to submuscular transposition, achieved full recovery by 9 months and was totally asymptomatic with a full range of elbow motion when last seen.

Of the 11 grade II patients with prior ulnar nerve surgery, seven ultimately achieved full motor and sensory recovery and were asymptomatic. One of these patients, reported by Broudy et al. as “improved” after 30 months, ultimately lost all symptoms only to develop documented ipsilateral thoracic outlet syndrome. The patient responded to first rib resection 6 months later and is now totally asymptomatic 5 years following that procedure.

The four remaining patients in the grade II group who had had prior surgery and who presented with pain and dysesthesia as well as weakness before reexploration and transposition all had severe circumferential scarring and fixation of their nerves demonstrated at operation. Two of these patients had had intramuscular transposition and in the third the attempted subcutaneous transposition had failed and the nerve had slipped posteriorly. The fourth patient had severe scarring that extended distally from a retained intermuscular septum to well within the substance of the flexor carpi ulnaris. All these patients experienced some improvement in their motor power, although none returned to normal. All had little or no improvement in their sensory deficit or pain.

Complications following surgery were minimal. The two patients who experienced transient pain in the distal radioulnar joint after surgery due to immobilization in hyperpronation were treated early in the series. No further difficulty of this type has been experienced since correction of the forearm rotation in the splint. Two patients with normal elbow range of motion before surgery had persisting 15° flexion contractures follow-
ing submuscular transposition. One, a 32-year-old woman, had an arthritic elbow documented at the time of surgery. The other, an 18-year-old woman, had chronic pain at the elbow for which no organic cause could be identified and was thought to have cancerophobia. The patients with restricted range of motion before surgery did not lose more than 5° of range after the operation. There were no patients whose neurological deficit was made worse and all but one were benefited. There were no infections, wound complications, or injuries to cutaneous nerves of the forearm.

Discussion

Study of this group of patients with progressive ulnar neuropathy at the elbow indicates that single or multiple mechanical factors may be operative in producing and perpetuating the neural deficit. Anything that alters the relationship between the nerve and its bed may be causative, as has been suggested by Apfelberg and Larson.\(^1\) Even flexion of the normal elbow may significantly diminish the capacity of the cubital tunnel; an intra-articular lesion may produce the same effect. A trivial injury may cause the nerve to lose its ability to glide and stretch with flexion and extension and thus initiate a traction-friction neuritis. Alteration in carrying angle, either static or dynamic, or actual hypermobility of the nerve itself may produce further scarring. In essence, since we may not be able to identify all the potentially injurious pathomechanical factors, the method of anterior transposition that alleviates virtually all but intraneural damage has stood the test of time. The submuscular technique of Learmonth avoids many potential problems of other methods, as illustrated by the patients in this series whose treatment by other surgical methods had been unsuccessful. Of the 25 patients who had not had a previous operation for ulnar neuropathy, only one was unimproved by either objective or subjective criteria at final evaluation and he had made substantial improvement over 14 months until secondary scarring caused his regression. Sixteen patients had normal hands by all criteria, and one had occasional paresthesias with normal function; in two patients the only residual was slight clawing of the small finger. The three patients with significant subjective complaints, one with emotional problems, elbow pain, and no neurological loss and two with documented secondary gain factors, cannot be counted as successes, not because of failure of the surgery, but rather because of an error in patient selection. It is clear, however, that the poor prognosis formerly advanced for patients with lesions over 3\(^{1/1}\) or 12 months duration\(^5\) or with significant atrophy\(^9\) may not be valid unless there is severe pain, dysesthesia, and previous surgery. In fact, the patients in this series show that, in most cases, one can expect improvement in the neurological deficit although some subjective complaints may persist. No other specific preoperative prognostic factors could be identified.

The "reoperation group" serves to illustrate another facet of the problem, for here there may be a striking relationship between what was done previously and the ultimate outcome. An iatrogenic negative factor may outweigh all others. In particular, the two patients who had their transposed nerves buried in the flexor pronator muscles had an extraordinary degree of fibrosis of their nerves that produced a severe and permanent neurological defect. They were not benefited by neurolysis and replacement in a nonscarred and well-vascularized bed because their nerves had already been so severely scarred that regeneration was impossible.\(^10\) On the other hand, traumatized nerves, explored because they were at fracture sites and left in situ, went on to become compressed by fracture healing. Presumably because they were not circumferentially involved, they were greatly benefited by submuscular transposition despite the presence of significant neurological deficits at the time. The implication of this finding is that exploration per se is insufficient, and these nerves should be transposed away from the fracture site at the time of osteosynthesis if a neurological deficit already exists. Patients with severe dysesthesia or preoperative pain are likely to retain these symptoms, even in the presence of motor improvement.

Finally, the identification of four patients who in addition to ulnar neuropathy also had thoracic outlet syndrome is, I believe, more than coincidence. One patient had had a prior operation elsewhere for documented thoracic outlet syndrome. Three others still have it after anterior transposition and are being observed. The double compression or crush lesion, as described by Upton and McComes\(^19\) in 1973, suggests that proximal compression of a nerve may lessen its ability to withstand further, more distal, compression. Although none of the patients in this series had concomitant lesions of the ulnar nerve within the Canal of Guyon, their well-documented thoracic outlet compression in addition to their ulnar neuropathy at the elbow would seem to illustrate this phenomenon.

REFERENCES