John R. Fowler *Editor*

Cubital Tunnel Syndrome

Diagnosis, Management and Rehabilitation

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I dedicate this book to my wife and best friend, Amy. Without her unending love and support, I would not be the person I am today.

Preface

The diagnosis and treatment of cubital tunnel syndrome remains a challenge for therapists, physicians, and surgeons. Despite years of research, there remains little consensus on the ideal diagnostic criteria, the benefits of nonoperative treatment, and the optimal technique for surgical release. In addition, the results of surgical treatment are more variable and often less successful than those of carpal tunnel release. The purpose of this text is to bring together experts in the fields of hand surgery and hand therapy to review the current state of the art in the diagnosis, nonsurgical management, and surgical management of cubital tunnel syndrome. I am confident that this textbook will improve patient care and outcomes.

Pittsburgh, PA, USA — John R. Fowler

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Part I
Diagnosis

Chapter 1
Anatomy of the Ulnar Nerve and Cubital Tunnel

Cassandra Lawrence and Richard J. Tosti

Introduction

Compression neuropathy of the fascicles comprising ulnar nerve can occur at several locations along its course from the central nervous system to the peripheral end organ (Table 1.1). The aim of the following chapter is to describe the anatomy of the ulnar nerve from the nerve roots that exit the spine and through the brachial plexus and the upper extremity while focusing on potential sites of compression. Additionally, this description will detail the internal anatomy of the nerve as it relates to management of acute injury and/or reconstruction of function through nerve transfers.

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Table 1.1 Common sites of compression of the ulnar nerve

Location	Common etiologies	Clinical manifestation
Spine	Trauma, tumors, intervertebral disk herniation, osteophyte	Cervical radiculopathy
Anterior and middle scalene	Tumors, spasm, cervical rib	Thoracic outlet syndrome
Intermuscular septum, arcade of Struthers	Triceps hypertrophy, iatrogenic following anterior transposition	Cubital tunnel syndrome
Osborne's ligament or anconeus epitrochlearis	Idiopathic, elbow flexion contracture, trauma	Cubital tunnel syndrome
Ulnar and humeral heads of FCU	Muscle hypertrophy, iatrogenic following anterior transposition	Cubital tunnel syndrome
Guyon's canal (ulnar tunnel)	Tumor, ulnar artery thrombosis, hook of hamate fracture	Ulnar tunnel syndrome

Central Nervous System

Volitional movement of the upper extremity is initiated in the motor cortex neurons in the dorsal portion of the frontal lobe. These neurons relay to the thalamus, decussate in the brain stem, descend in the lateral corticospinal tract, and synapse in the anterior horn. The lower motor neuron cell bodies originate in the anterior horn, exit via the ventral root, and blend with the dorsal root to become a peripheral nerve that exits the spine through the neural foramina. The lower neurons could potentially become compressed at this level by a herniated intervertebral disk, fracture, or an osteophyte. After exiting the spine, the peripheral nerve root divides to form dorsal or ventral rami. Ventral rami from C5 to T1 coalesce to form the brachial plexus.

Sensibility of the upper extremity follows an afferent pathway from the periphery to the central nervous system. The first neuron cell body is located in the dorsal root ganglion of the spinal nerve. The dorsal root ganglion cells will relay with neurons in the brainstem or spinal cord. Neurons sensing touch and pain are relayed to the thalamus and then ultimately to the postcentral gyrus of the parietal lobe.

Nerve Roots and Brachial Plexus

The brachial plexus is a network of nerves beginning at the lower cervical and upper thoracic spine, which extend to the axilla (Fig. 1.1). This collection of nerves originates from the ventral rami of the inferior four cervical nerves and first thoracic nerve (C5, C6, C7, C8, T1). These five spinal nerve roots of the brachial plexus course along with the subclavian artery and pass between a potential compression site at interval between the anterior and middle scalene muscles (i.e., thoracic outlet syndrome). The nerve roots of the plexus combine to form three trunks. C5 and C6 unite to form the

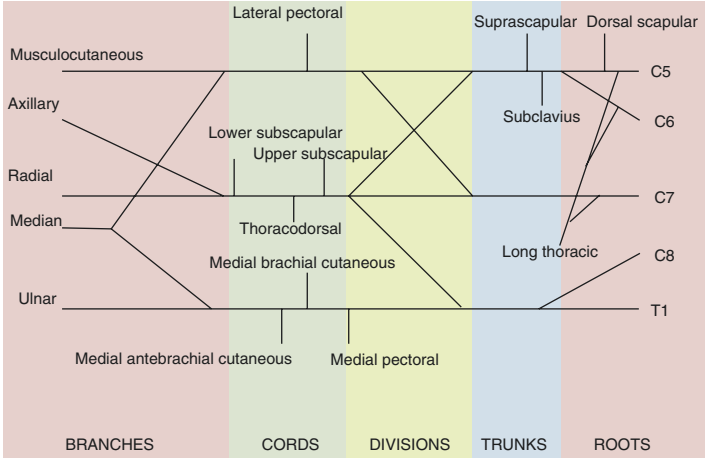

Figure 1.1 Schematic of the brachial plexus

superior trunk. The continuation of the C7 nerve root constitutes the middle trunk. The C8 and T1 nerve roots unite to form the inferior trunk. Next, the trunks pass through the cervico-axillary canal. This space lies posterior to the clavicle and is bound by the first rib and superior scapula. At this point, the superior, middle, and inferior trunks each divide into anterior and posterior divisions. Derivatives of the anterior division innervate the flexors of the arm, while derivatives of the posterior division innervate extensors of the arm. The anterior divisions of the superior and middle trunks combine to form the lateral cord. The anterior division of the inferior trunk constitutes the medial cord. The posterior divisions of all three trunks (superior, middle, and inferior) constitute the posterior cord. The peripheral nerve branches coming from the brachial plexus can be split broadly into two categories, supraclavicular and infraclavicular. The supraclavicular branches originate from the anterior rami of C5–7 and superior trunk. The infraclavicular branches originate from the lateral, medial, and posterior cords. While the brachial plexus gives rise to several peripheral nerve branches, this text will focus on the ulnar nerve. The ulnar nerve is the terminal branch of the medial cord and receives fibers from C8, T1, and sometimes C7.

Upper Arm to Cubital Tunnel

After exiting the medial cord of the brachial plexus, the ulnar nerve passes anterior to the insertion of teres major and the long head of the triceps. It runs medially in the anterior compartment of the upper arm and remains posteromedial to the brachial artery. Approximately 8 cm proximal to the medial epicondyle (or 2/3 of the distance distally in the arm), the nerve pierces the intermuscular septum and enters the posterior compartment of the arm along with the superior ulnar collateral artery. These structures descend between the intermuscular septum and the medial head of the triceps. In many individuals, the ulnar nerve passes underneath the arcade of

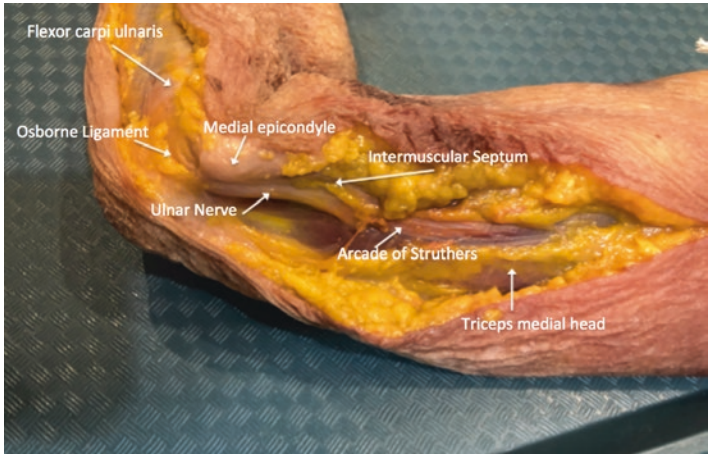

FIGURE 1.2 Anatomy of the ulnar nerve at the elbow in a cadaver

Struthers. The arcade is a thin band of connective tissue extending from the medial intermuscular septum to the medial head of the triceps approximately 8 cm proximal to the medial epicondyle of the humerus and is one potential site of ulnar nerve entrapment [1] (Fig. 1.2). After descending along the medial head of the triceps, the ulnar nerve then travels posterior to the medial epicondyle of the humerus and medial to the olecranon. The ulnar nerve, medial epicondyle, and olecranon are all palpable structures. The ulnar nerve has no branches in the arm proximal to the elbow, but it does supply articular branches to the elbow joint.

Cubital Tunnel

Distal to the medial epicondyle, the ulnar nerve passes through the cubital tunnel. The roof of the cubital tunnel is formed by Osborne's ligament. This extends from the medial epicondyle and the humeral head of the flexor carpi ulnaris (FCU) muscle to the olecranon and ulnar head of the FCU muscle [2, 3]. The ligament is typically approximately 2.2 cm

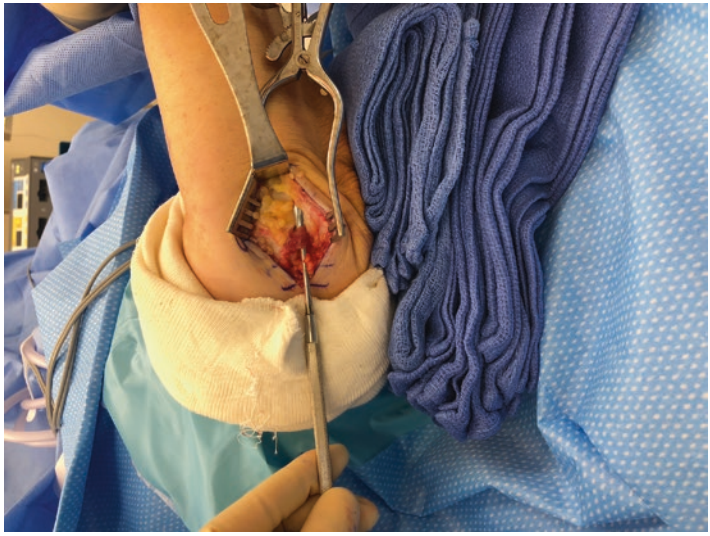

Figure 1.3 Anconeus epitrochlearis muscle above the freer elevator

in length and 4 mm in width [4]. In up to 30% of the population, the anconeus epitrochlearis muscle follows a similar course to that of the Osborne's ligament, which can also compress the nerve when present [5] (Fig. 1.3). The floor of the cubital tunnel is composed of the medial collateral ligament of the elbow, the elbow joint capsule, and the olecranon.

Forearm

After passing through the cubital tunnel, the ulnar nerve courses through the deep flexor pronator aponeurosis and between the ulnar and humeral heads of the FCU muscle. The deep flexor pronator aponeurosis in the forearm represents another potential site of compression of the ulnar nerve. The nerve then travels along the ulna superficial to the flexor digitorum profundus (FDP) muscle and deep to the FCU muscle (Fig. 1.4). It courses medial to the ulnar artery. The ulnar nerve gives off two motor branches in the anterior fore-

arm to the FCU and medial half of the FDP muscles. Contraction of the FCU produces flexion and ulnar deviation at the wrist, while the ulnar innervated portion of the FDP muscle flexes the interphalangeal joints of the fourth and fifth digits. The ulnar nerve also gives off the palmar cutaneous branch and dorsal cutaneous branch in the forearm, which provide sensory innervation for the medial half of the palm and the dorsal medial 1½ digits and associated dorsal hand region, respectively [6, 7].

Guyon's Canal, Wrist, and Hand

At the level of the wrist, the ulnar nerve passes superficial to the flexor retinaculum and enters the hand through Guyon's canal or the "ulnar tunnel," which is the most distal site of compression (Fig. 1.4). The canal spans the proximal end of the pisiform to the hook of the hamate, the roof of which is formed by the volar carpal ligament and pisohamate ligament. The canal contains the ulnar nerve and artery (radial to

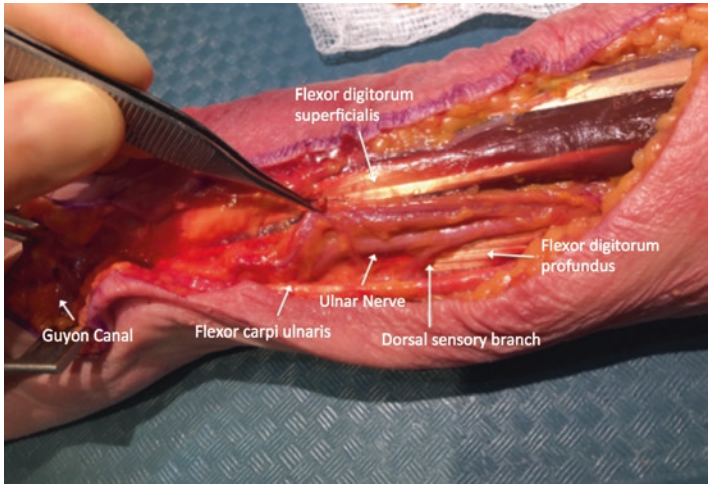

Figure 1.4 Anatomy of the ulnar nerve at the forearm in a cadaver

Table 1.2 Ulnar tunnel zones

Zone	Location	Symptoms
1	Proximal to bifurcation of deep and superficial branches	Mixed motor and sensory
2	Deep motor branch	Motor only
3	Superficial sensory branch	Motor only

the nerve). The ulnar nerve gives off superficial and deep branches within the canal. The superficial branch travels on the ulnar side, while the deep branch travels on the radial side. The superficial branch provides sensory innervation to the palmar surface of the medial 1½ digits. The deep branch innervates the hypothenar muscles and then courses radially beneath the hook of the hamate with the ulnar artery to innervate the majority of the intrinsic hand muscles, including the ulnar two lumbricals, adductor pollicis, interosseous muscles, deep head of the flexor pollicis brevis, and palmaris brevis. Occasionally the ulnar tunnel is described in zones (Table 1.2).

Internal Anatomy of the Ulnar Nerve

In 1945, Sunderland studied the intraneural topography of peripheral nerves in the upper extremity and determined the distance over which individual peripheral nerves innervating muscular and cutaneous structures maintained their discrete identities [8]. His work is clinically significant as it provides the applied anatomy for nerve transfers in the upper extremity.

When treating upper extremity peripheral nerve injuries, distal median to ulnar nerve transfers can be performed to restore motor and sensory function of the ulnar nerve. Knowledge of ulnar nerve topographic anatomy is of particular importance when performing these procedures. Typically, three discrete fascicles of the ulnar nerve are identified 9 cm proximal to the radial styloid [9, 10]. At this level, the ulnar

nerve topographic pattern is sensory-motor-sensory. From ulnar to radial, the fascicles are arranged as follows: dorsal cutaneous branch, ulnar motor branch, and superficial sensory branch. The motor fascicular group is smaller, constituting approximately 40% of the main ulnar nerve bundle, while the sensory fascicular group comprises approximately 60% of the ulnar nerve bundle [11]. Intraoperatively, microforceps can be used to apply gentle pressure to distinguish the natural cleavage lines between the motor and sensory groups.

Another clinical application of the topographical anatomy of the ulnar nerve is restoration of elbow flexion after brachial plexus injury. In 1994, Oberlin et al. described a technique for transferring one or more fascicles of the intact ulnar nerve to the nerve to the biceps after C5–6–7 brachial plexus root injury, also recognized as the Oberlin transfer [12]. An anteromedial incision is made in the arm, and the ulnar nerve is identified in the medial mid-brachium adjacent to the brachial artery. An epineural incision is made, and the ulnar nerve fascicles are identified with electrical stimulation. Typically, visual inspection of the fascicles is performed to match the appropriate size of the donor nerve to the recipient. From comparison of the cross-sectional areas of the ulnar nerve and the musculocutaneous nerve, the authors determined that 10% of the ulnar nerve would be required to innervate the biceps muscle at the same level. This percentage typically translates to 1–3 individual fascicles of the ulnar nerve [12]. Fascicles supplying the flexor carpi ulnaris muscle can be distinguished from intrinsic muscles of the hand with electrical stimulation and are selected for transfer. Often the FCU fascicle is anterior and medial within the nerve.

Conclusion

Various potential sites of compression exist as the ulnar nerve courses through the upper extremity. Knowledge of the detailed anatomy of the ulnar nerve is critical for the diagnosis and treatment of cubital tunnel syndrome among other peripheral nerve injuries.

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Chapter 2
Cubital Tunnel: History and Physical Examination

Joseph F. Styron

Cubital tunnel syndrome is the second most common compressive neuropathy within the upper extremity [1]. The cubital tunnel is the most common location for compression of the ulnar nerve, although there are multiple other potential compression sites along its course from the neck to the hand. Patients rarely present with pain as their primary complaint. The most common presentation is paresthesias within the ulnar nerve distribution. Weakness of the intrinsic muscles within the hand is also a common symptom at presentation which may manifest as subtly as subjective clumsiness, or since the ulnar nerve is a primary driver of grip strength, its compromise often results in weakening of grip strength [2]. Multiple other etiologies can similarly produce paresthesias, weakness, and pain though, including C8/T1 radiculopathy, thoracic outlet syndrome, and ulnar nerve compression within Guyon's canal at the wrist. An astute clinician must

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distinguish these other potential sources of ulnar nerve impairment from cubital tunnel syndrome.

History

The following are critical aspects of the patient's history that should be obtained:

1. Duration of symptoms.
2. Consistency of symptoms.
3. Subjective sense of numbness or pain.
4. Location of numbness (radial/ulnar side of hand and volar/dorsal).
5. Grip or pinch strength weakness.
6. Positional or temporal patterns of symptoms.
7. Aggravating/alleviating factors/positions.
8. Previously attempted interventions and their efficacy.
9. Comorbidities and prior elbow injuries.

It is important to ascertain the duration of symptoms. An acute onset may be secondary to an injury. Recent elbow trauma may produce swelling within the cubital tunnel causing acute compression of the ulnar nerve. Patients typically cannot recall any specific insult but report a more insidious onset of numbness over the previous weeks, months, or even years.

The consistency of symptoms may be the most important information to glean. While the symptoms are still intermittent with periods of normal nerve function, the likelihood for a complete recovery is still a reasonable expectation. However, once the patient develops constant symptoms with intermittent exacerbations, the goal of intervention shifts toward an effort to prevent the symptoms from worsening, because a restoration of their prior "normal" baseline nerve function may be unattainable and is certainly unreliable.

Pain is an uncommon complaint of patients presenting with cubital tunnel syndrome [3]. Patients often will endorse their "entire" hand having episodes of paresthesias; however, when probed and asked to monitor their symptoms more closely, they will admit the numbness is predominantly in

their small finger and the ulnar half of the ring finger. As compression of a sensory nerve progresses, it occurs in a predictable manner. The first threshold change is loss of vibratory perception; then with progressive conduction block or degeneration, there is loss of innervation density manifested as decreased two-point discrimination [4]. If the patient has a sensory disturbance on the dorsum of the hand, it confirms that the compression of the ulnar nerve is proximal to Guyon's canal based on the origin of the dorsal cutaneous branch of the ulnar nerve in the distal forearm. If the patient does complain of pain, other etiologies must be excluded such as flexor carpi ulnaris tendinitis, medial epicondylitis, and ulnohumeral osteoarthritis. Cubital tunnel syndrome may be associated with pain at the cubital tunnel region, medial epicondyle, and into the forearm.

Weakened grip strength is not uncommon with compression of the ulnar nerve at the cubital tunnel. Fine motor control of the hand may be impaired as these are primarily performed by the ulnar-innervated intrinsic muscles. This may be manifested as difficulty buttoning a shirt, clipping nails, or easy fatiguability. Less chronic compression may result in a subjective sense of clumsiness but not result in muscular atrophy. Intrinsic muscle atrophy is evidence of long-standing compression of the ulnar nerve. In extreme cases, ulnar clawing is possible due to weakness of the third and fourth lumbricals, resulting in hyperextension at the metacarpophalangeal joints and flexion of the interphalangeal joints in the small and ring fingers. Several classic physical exam findings are based on the weakness of ulnar-innervated muscles [5]. The Wartenberg sign is a manifestation of interossei weakness, Froment sign represents weakness in the adductor pollicis, and as mentioned, the claw hand deformity is due to weakness in the ulnar lumbrical muscles. Patients with cubital tunnel syndrome are four times more likely to present with muscle atrophy than are patients with carpal tunnel syndrome [6].

Positional variation in the symptoms is common. Most often, patients will complain of increased numbness in the ulnar nerve distribution with repetitive or prolonged elbow

flexion. Gelberman et al. demonstrated that progressive elbow flexion to 130° resulted in increased intraneural pressure in the ulnar nerve as it courses behind the medial epicondyle in the cubital tunnel [7]. In addition, the nerve itself undergoes significant strain and elongates 4.7 mm with elbow flexion. This further increases to 8 mm with the addition of shoulder abduction and external rotation [8]. There are multiple sites of potential compression for the ulnar nerve around the elbow. Therefore, identifying if a particular movement elicits the symptoms can help distinguish the location of compression. The ulnar nerve travels through the arcade of Struthers, posterior to the medial intermuscular septum, then posterior to the medial epicondyle, into the cubital tunnel, and then through the deep flexor pronator aponeurosis, all of which are potential locations of compression and potential sites for intervention at the time of a surgical release. Unfortunately, it is due to these variable sites of potential compression and the dynamic nature of the compression that lead to false-negative results in electrodiagnostic studies.

Aggravating or alleviating factors are always important to take into consideration as they may help guide non-operative management early in the course of treatment. Frequently patients may experience spontaneous resolution of their mild intermittent symptoms with avoidance of provocative causes. Such alleviating factors will be discussed in greater detail later but may include activity modification (e.g., arm position while driving or using the telephone) and nocturnal splints to prevent maximum or repetitive elbow flexion.

Identifying which treatment modalities the patient may have already attempted can help guide the diagnosis and treatment of cubital tunnel syndrome. For example, wearing nocturnal wrist splints may be effective for the treatment of mild carpal tunnel syndrome, but splinting to prevent elbow flexion is more efficacious in treating cubital tunnel syndrome.

Certain comorbidities are associated with increased risk for the development of cubital tunnel syndrome, including diabetes, thyroid disease, hemophilia, or general peripheral neuropathies [1]. In addition, the practitioner should determine if the patient may have compression due to other struc-

tures such as osteophytes from degenerative arthritis of the elbow, ganglion cysts (Fig. 2.1), tumors, anomalous bands of fibrous tissue, or an anconeus epitrochlearis muscle such that proper excision of these structures can eliminate compression of the ulnar nerve. A distal humerus fracture resulting in cubi-

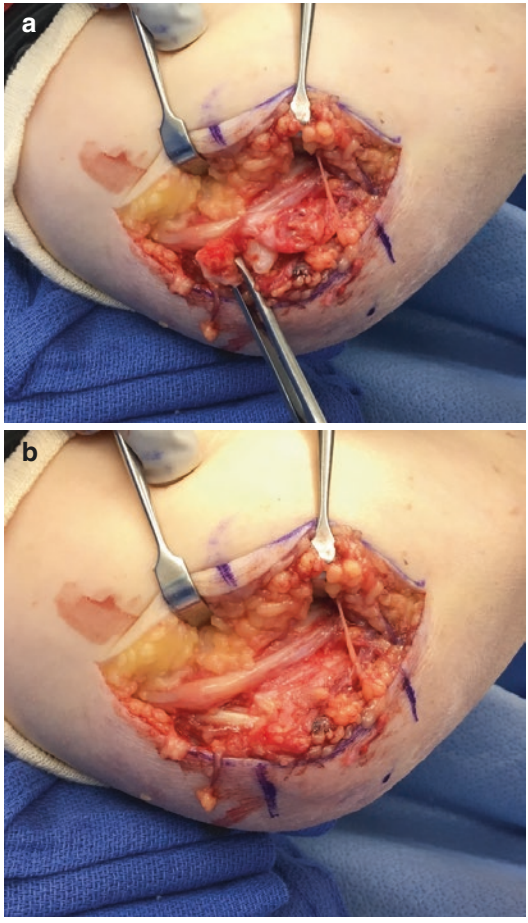

FIGURE 2.1 (**a**) Ganglion cyst compressing the ulnar nerve in the cubital tunnel; (**b**) the ganglion cyst has been excised and you can see the hourglass compression shape of the ulnar nerve

tus valgus and a tardy ulnar nerve palsy may be a more obvious post-traumatic etiology for ulnar nerve compression at the elbow. More commonly and less obvious though, would be a fracture around the elbow (olecranon or distal humerus) resulting in swelling and compression of the ulnar nerve. In addition, postoperative cubital tunnel syndrome has been reported following cardiac, spine, or shoulder surgery, often due to patient positioning, use of a tourniquet, or even a blood pressure cuff on that arm.

Multiple classification systems for cubital tunnel syndrome have been proposed, but they are infrequently utilized. The classification systems are often helpful in describing the severity of neuropathy, but not in determining the best treatment for the patient [9]. McGowan first proposed a three-grade classification system in 1950, and this was later modified independently in 1989 by both Goldberg and Dellon (Tables 2.1 and 2.2) [4, 10, 11]. The modified McGowan system classifies intermittent paresthesias and subjective weakness as mild nerve dysfunction. Moderate dysfunction is characterized as intermittent paresthesias but with measurable weakness, while severe dysfunction is seen in patients with persistent paresthesias and measurable weakness. Dellon's classification system maintains the three grades but also adds additional physical exam findings, including finger crossing.

Table 2.1 Modified McGowan classification for ulnar neuropathy [11]

Grade	Sensory examination	Motor examination
I	Mild paresthesias or sensory loss	No measurable weakness
IIA	Moderate sensory loss	No intrinsic atrophy, mild weakness
IIB	Moderate sensory loss	3/5 intrinsic strength, moderate weakness
III	Severe sensory loss or paresthesias	Severe intrinsic atrophy and weakness

Table 2.2 Dellon staging of ulnar nerve compression at the elbow [4]

Grade	Sensory examination	Motor examination	Exam findings
I (mild)	Paresthesias intermittent; vibratory perception increased	Subjective weakness, clumsiness or loss of coordination	Elbow flexion test and/or Tinel's sign may be positive
II (moderate)	Paresthesias intermittent; vibratory perception normal or decreased	Measurable weakness in pinch and/or grip strength	Elbow flexion test and/or Tinel's sign are positive; finger crossing may be abnormal
III (severe)	Paresthesias are persistent; vibratory perception decreased; abnormal 2-point discrimination (static ≥6 mm, moving ≥4 mm)	Measurable weakness in pinch and grip plus muscle atrophy	Positive elbow flexion test and/or positive Tinel's sign may be present; finger crossing usually abnormal

Physical Examination

The physical examination of a patient with suspected cubital tunnel syndrome should always start with observation. Cubital tunnel patients are four times more likely to present with muscle atrophy than patients with carpal tunnel syndrome (Fig. 2.2) [6]. In particular, the first dorsal interosseous muscle is readily visualized and palpated to assess for atro-

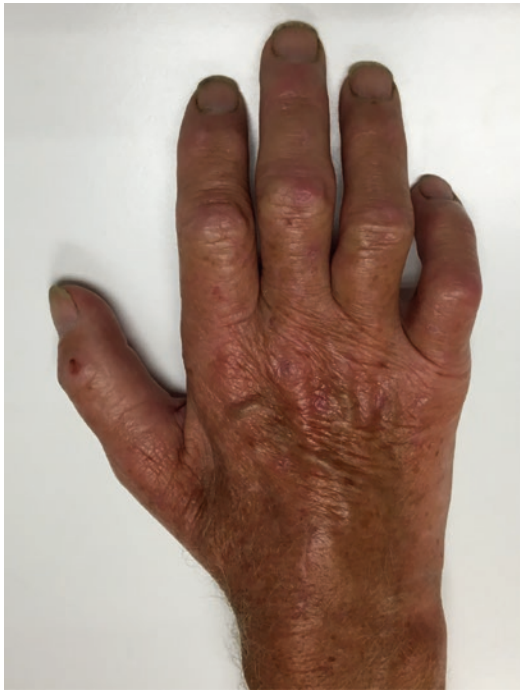

FIGURE 2.2 Atrophy of the first dorsal interosseous muscle in the right hand compared to the left

phy, especially in unilateral cases. The patient should be asked to cross his/her fingers which would demonstrate moderate or severe dysfunction of the ulnar-innervated intrinsic muscles if this proves difficult or impossible (Fig. 2.3). The advanced degree of atrophy and muscle weakness often seen in advanced ulnar compressive neuropathy can be identified by several physical exam findings [12]:

1. Wartenberg's sign: This is noted as the inability to actively adduct the small finger due to weakness in the ulnar-innervated third palmar interosseous muscle. As a result, the small finger is abducted due to the ulnar insertion of the extensor digiti quinti (Fig. 2.4).

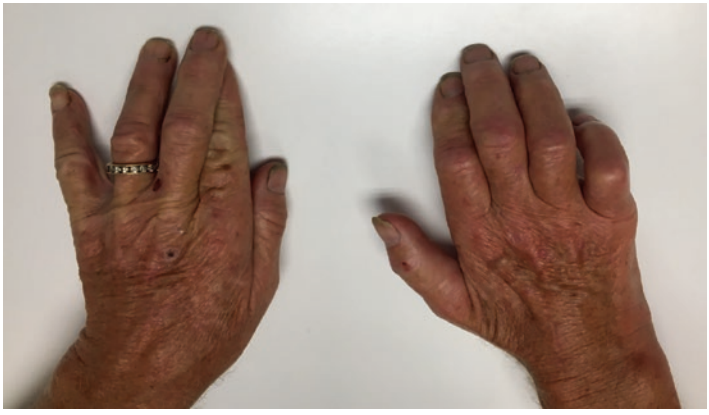

FIGURE 2.3 Note the inability of the patient to cross his fingers with his right hand compared to his left

FIGURE 2.4 Wartenberg sign: note the inability to adduct the small finger on the right hand

2. Froment sign: While attempting to perform a key pinch, the thumb interphalangeal joint flexes. This happens because the flexor pollicis longus attempts to compensate for the weak adductor pollicis.

3. Jeanne sign: This also occurs while having the patient perform a key pinch. In this sign, the thumb metacarpophalangeal joint will hyperextend as the extensor pollicis longus attempts to adduct the thumb again to compensate for the weakened adductor pollicis.
4. Claw-hand deformity (Duchenne's sign): This is visualized as hyperextension at the metacarpophalangeal joints of the ring and small fingers with flexion at the interphalangeal joints. This is due to the loss of the lumbrical and interosseous muscles which are ulnar-innervated and the over-powering effect of the intact extrinsic finger flexors.
5. Masse's sign: This is described as flattening of the dorsal transverse metacarpal arch causing the hand to appear flattened. This is caused by paralysis of the hypothenar musculature which eliminates the normal flexion and supination of the fifth metacarpal (Fig. 2.5).

In addition, several provocative maneuvers may be performed in the physical examination to provide support to the diagnosis of cubital tunnel syndrome.

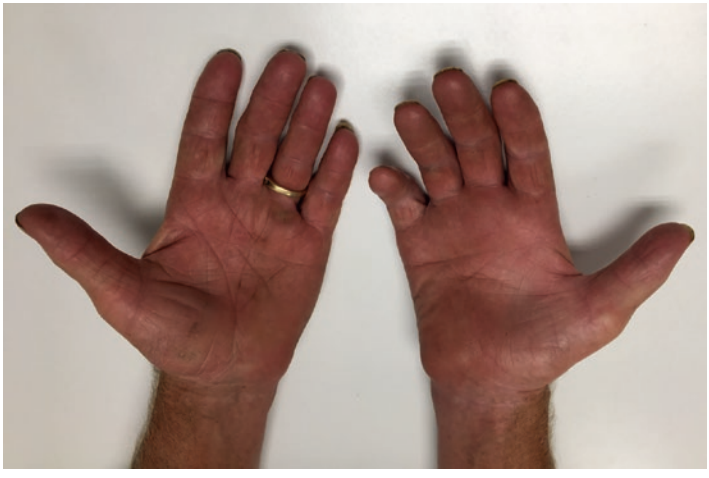

Figure 2.5 Masse's sign: note the flattened appearance of the hand due to the paralysis of the hypothenar eminence

Percussion of the ulnar nerve (Tinel's test) at the retrocondylar groove may reproduce the patient's paresthesia and pain (positive test with reproduction). The flexion-compression test is performed by flexing the elbow while simultaneously applying manual compression over the ulnar nerve posterior to the medial epicondyle [13].

The scratch-collapse test has been described as being diagnostic for both carpal and cubital tunnel syndromes [14]. The test is performed by the examiner first having the patient actively perform external rotation of the arm at the shoulder with the elbow flexed to 90°. The examiner then lightly scratches the patient's skin overlying the ulnar nerve posterior to the medial epicondyle, while the patient sustains resisted external rotation. The light scratching over the compressed ulnar nerve produces an allodynia believed to impart a brief loss of muscle resistance, thus causing the patient's efforts at external rotation to collapse under the resistance applied by the examiner. According to the initial paper, the scratch-collapse test had higher sensitivity than either Tinel's or flexion-compression testing for cubital tunnel syndrome. The favorable results for the scratch-collapse reported by the original authors were not reproduced by others who have found increased variability among examiners and less sensitivity [15]. Therefore, the scratch-collapse test may be one tool with which to diagnose cubital tunnel syndrome, but a compliment of provocative maneuvers is frequently performed rather than any single test in isolation. The sensitivity and specificity of some of the provocative maneuvers are summarized in Table 2.3.

An important physical exam maneuver for preoperative planning, more than diagnosing cubital tunnel syndrome, is assessing the stability of the ulnar nerve. The stability of the nerve can be assessed by placing the examiner's finger posterior to the medial epicondyle while taking the elbow through an arc of motion to determine whether the nerve remains stable, perches on the epicondyle, or subluxates out of its retrocondylar groove. Ulnar nerve hypermobility is present in 37% of elbows and is not associated with symp-

Table 2.3 Provocative testing for cubital tunnel syndrome [14, 16]

	Tinel's [16]	**Flexion-compression** [16] **(for 30/60 seconds)**	**Scratch collapse** [14]
Sensitivity (%)	70	91/98	69
Specificity (%)	98	97/95	99
Positive predictive value (%)	94	93/91	99
Negative predictive value (%)	87	96/99	86

tomatic cubital tunnel syndrome [17]. Identifying an unstable nerve in clinic though may cause the surgeon to consider transposing the ulnar nerve rather than performing an in situ decompression.

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Chapter 3
Cubital Tunnel Syndrome: Evaluation and Diagnosis

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History and Physical Exam

History

Numbness and altered sensibility of the ulnar aspect of the hand and forearm typically are the most common presenting complaints, but motor symptoms can also precede sensory complaints in an ulnar neuropathy. Hand intrinsic or grip weakness may be reported, along with muscle atrophy, clawing of the hand, or complaints of the fifth digit getting "stuck" outside of a pocket when a patient tries to reach in with the affected hand (due to interossei weakness causing fifth digit

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abduction). Pain or paresthesias may be present, and a lesion at the elbow would be expected to cause subjective symptoms that involve the dorsal ulnar cutaneous distribution as well as the fifth digit and the ulnar side of the fourth digit, though anatomical variants in terms of digital involvement exist. This condition will often develop insidiously, and a history should include questions about precipitating activity, repetitive elbow flexion or pressure, acute injury, and prior traumatic injury to the elbow. Medial elbow pain and sensitivity may also be a symptom [1].

Physical Examination

Ulnar neuropathy can have a number of physical examination findings including muscle weakness and atrophy, altered range of motion, deformity of the elbow, and changes to the hand. We recommend performing a physical examination for ulnar neuropathy methodically including inspection, palpation, motor testing, and sensory examination (Tables 3.1 and 3.2).

Table 3.1 Ulnar-innervated muscles

Forearm
Flexor digitorum profundus (medial heads)
Flexor carpi ulnaris
Hand
Hypothenar
Opponens digiti minimi
Abductor digiti minimi
Flexor digiti minimi
Thenar
Adductor pollicis
Other intrinsics
Palmar interossei
Dorsal interossei
Two medial lumbricals

Table 3.2 Ulnar nerve elbow entrapment sites

Above the elbow (approximately 8 cm proximal to the medial epicondyle):
Arcade of Struthers (tough fascial plane)
At the elbow:
In the groove or the area of the medial epicondyle secondary to bony changes
Below the elbow:
The aponeurosis formed by the two heads of the flexor carpi ulnaris (this is the "cubital tunnel")

Proximally, inspection of the elbow should include identifying any elbow deformity, arthritic changes, and/or altered range of motion or stability. Elbow joints with arthritic changes can apply pressure from the underlying joint synovitis and/or joint thickening resulting in pressure to the ulnar nerve through its floor (Fig. 3.1). Flexion contracture of the elbow joint can cause both traction and pressure on the ulnar nerve due to prolonged flexed posturing and from the thickened elbow joint capsule, respectively. Elbow joint instability, more common in younger patients and often in throwers, can also cause dynamic ulnar traction. Lastly, past history of an elbow fracture, most commonly supracondylar and/or lateral condyle fractures, can result in a residual valgus malunion of the elbow resulting in chronic traction on the ulnar nerve or the so-called tardy ulnar palsy [2].

Distally, inspection of the forearm and hand can yield important findings including atrophy or wasting of the medial forearm and intrinsic muscles of the hand, radial deviation of the wrist, claw deformity of the hand, and a Wartenberg's sign. A detailed motor examination should be performed to identify weak or atrophied muscles. Other, non-ulnar-innervated muscles should by comparison be normal, and the location of an ulnar lesion will determine which muscles, if any, are affected. For example, a lesion at the elbow would be expected to have muscle weakness in the hand intrinsics such

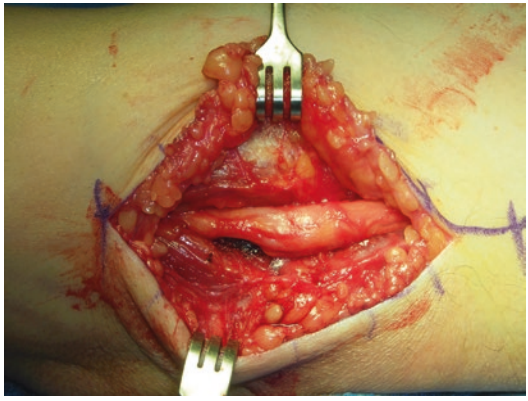

Figure 3.1 Finding of an ulnar nerve in the case of cubital tunnel syndrome with underlying elbow arthritis resulting in significant constriction on the ulnar nerve within the cubital tunnel causing it to take on an "hourglass" configuration

as the first dorsal interossei of the hand, the abductor digiti minimi, and the adductor pollicis (Fig. 3.2), as well as more proximal muscle involvement with weakness of the flexor carpi ulnaris and the flexor digitorum profundus to the ulnar two digits and wrist radial deviation with a weakened flexor carpi ulnaris [1], whereas a lesion at the wrist might only involve the distal hand intrinsics.

Clawing of the ulnar hand is manifested by weakness of all of the interossei and the ulnar-innervated lumbricals which results in unopposed hyperextension of the metacarpophalangeal joints and flexion of the fourth and fifth digit interphalangeal joints (ulnar claw deformity) by the finger "extrinsics" (Fig. 3.3). Typically, this passively correctable and active composite flexion is maintained, albeit with weaker grip strength. However, with time chronic clawing can result in contracture into a clawed position composite flexion prohibiting active or passive composite flexion.

A Wartenberg's sign is described as a passive abduction of the fifth digit that also occurs due to weakness of the interossei (Fig. 3.4). The patient will be unable to actively adduct the

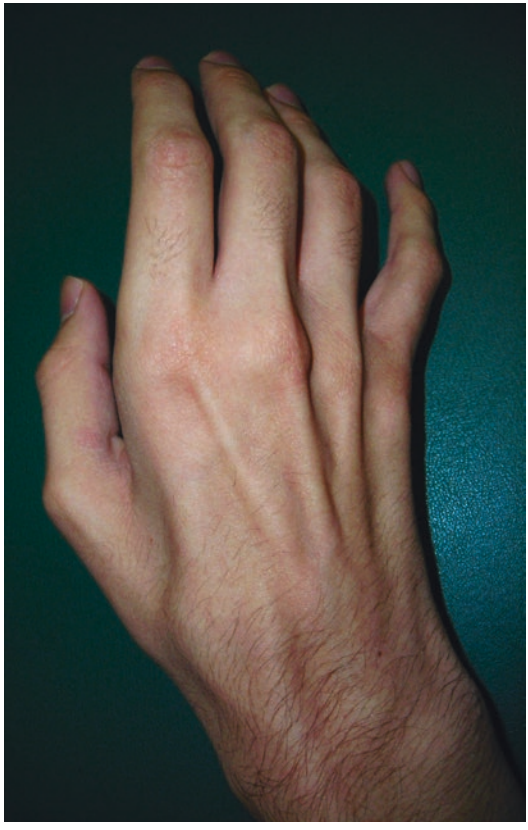

FIGURE 3.2 Intrinsic atrophy of the hand. Note how the atrophy is most evident within the first webspace due to loss of the adductor pollicis muscle

fifth digit. The patient may also complain of the finger getting stuck outside pant and jacket pockets because of the inability to adduct the fifth digit actively.

Froment's sign is seen in ulnar hand intrinsic weakness, when the patient is asked to hold a piece of paper between the thumb and the index finger against resistance. Patients with normal intrinsic function can perform this activity with the strength of the adductor pollicis generating most of the

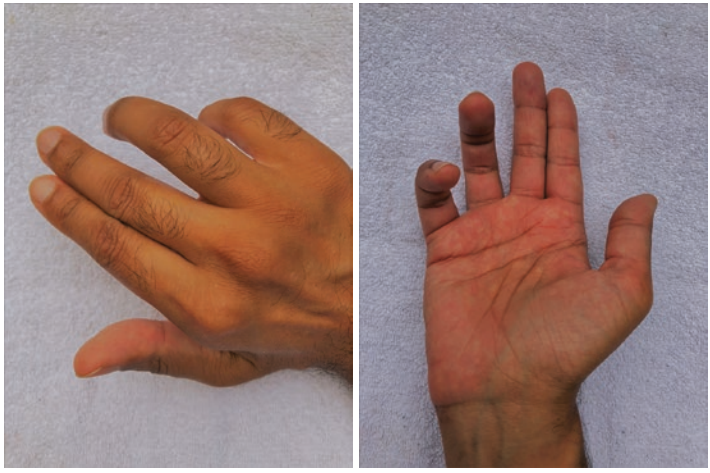

FIGURE 3.3 Clawing of the ulnar two digits of the hand due to advanced cubital tunnel syndrome resulting in unopposed pull of the "extrinsic" flexor and extensor tendons due to the loss of the interossei and lumbrical muscles (the "intrinsics") to these two digits. Note that the radial two digits do not claw and that is because their lumbricals are innervated by the median nerve and are still functioning

force necessary without the need for the first digit interphalangeal joint to flex. In the presence of adductor pollicis weakness, the patient will compensate by using the flexor pollicis longus (spared and innervated by the median nerve) to hold the paper between the thumb and index finger. This visible interphalangeal joint flexion is referred to as a "Froment's sign" (Fig. 3.5).

Palpation of the ulnar nerve at the elbow is useful to assess for baseline nerve instability, sensitivity, and response to provocative testing. Although a subluxing ulnar nerve is present in 15% of asymptomatic patients, identification of a subluxing ulnar nerve during the physical examination may be important for operative decision-making. Moreover, increased subluxation or instability of the ulnar nerve from side to side may indicate a pathologic amount of nerve instability. Palpation and Tinel's testing of the ulnar nerve may also help identify

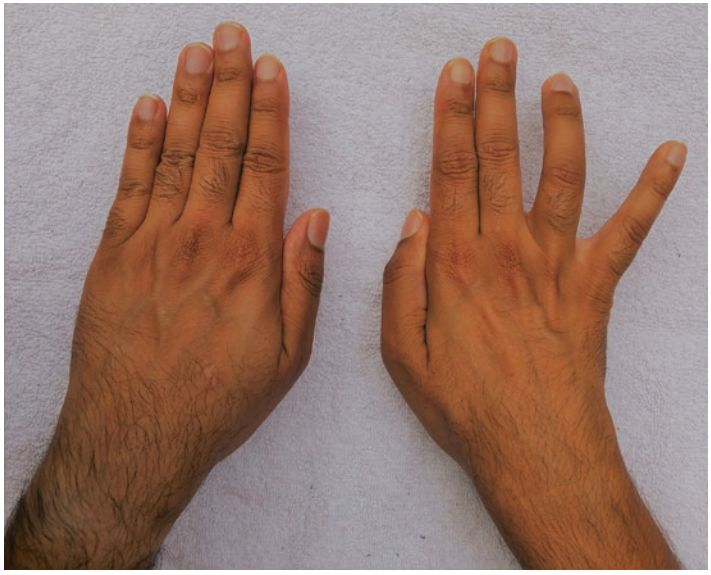

FIGURE 3.4 Wartenberg's sign is an observational examination finding where the fifth digit sits in an abducted position and cannot be adducted actively

the site of greatest nerve sensitivity and subsequently the site of greatest compression. The "elbow flexion" test is a simple provocative maneuver where the elbow is held in a hyperflexed position and is deemed positive when the patient begins experiencing ulnar nerve paresthesias within 30–60 seconds. Although controversial, the scratch-collapse maneuver may also suggest an ulnar nerve lesion at the elbow [3, 4].

A thorough sensory exam of the upper extremity is also warranted when assessing ulnar neuropathy. Most commonly, the patient will complain of altered sensation and/or numbness in the ulnar aspect of their affected hand. However, careful sensory examination is warranted as both light touch and two-point discrimination may be altered in various distributions aiding in making the most accurate diagnosis. After crossing the elbow, the two sensory

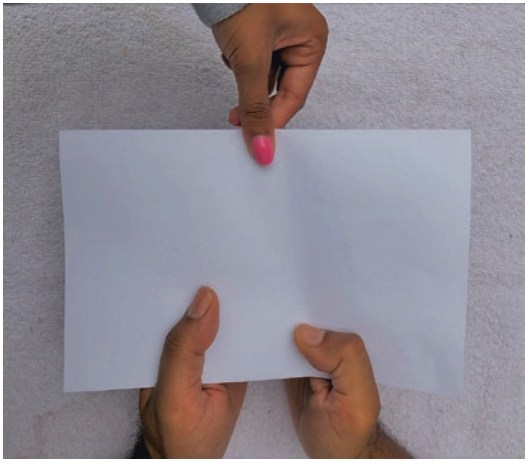

Figure 3.5 Froment's sign is a provocative examination finding due to the inability to hold a paper pinched between the thumb and index finger due to weakness or atrophy of the adductor pollicis muscle requiring the flexor pollicis longus muscle to compensate by flexing the thumb interphalangeal joint and hold the paper in position

branches of the ulnar nerve are the dorsal and palmar ulnar cutaneous branches. The palmar ulnar cutaneous branch provides sensory innervation to the volar aspect of the fifth digit and the volar aspect of the medial half of the fourth digit in most patients. The dorsal ulnar cutaneous nerve supplies sensation of the dorsal medial half of the fourth digit and the dorsum of the fifth digit, along with the medial and dorsal side of the hand. Sensory testing would be expected to show abnormalities of both sensory nerves with cubital tunnel entrapment at the elbow, whereas a wrist lesion would spare the dorsal ulnar cutaneous nerve. As with motor testing, evaluation of other sensory distributions and dermatomes is necessary to evaluate for other more proximal causes of numbness such as a brachial plexopathy, cervical radiculopathy (likely at C8-T1), or central nervous system disease.

Diagnostic Testing: Electrodiagnostics

Nerve Conduction Studies

Electrodiagnostic testing with nerve conduction studies (NCS) and electromyography (EMG) remains the only way to study nerve function along its entire pathway. Each study begins with and becomes an extension of the history and physical exam. Taking into account the patient's symptoms helps to guide the examiner in setting up each test for each specific patient. Data from NCS and EMG testing can help to determine the degree of nerve injury, the timing of injury, and the amount of recovery that may take place. Changes in nerve conduction studies may be present immediately, whereas changes on the EMG portion of the test may develop weeks later (Tables 3.3, 3.4 and 3.5) [5].

Table 3.3 Routine nerve studies for evaluation of ulnar neuropathy at the elbow

1. Ulnar motor NCS recorded at the ADM, with stimulation at the wrist, below the elbow, and above the elbow
2. Median motor NCS recorded at the APB and stimulated at the wrist and antecubital fossa
3. Ulnar sensory NCS stimulated at the wrist and recorded at the fifth digit
4. Median sensory NCS stimulated at the wrist and recorded at digit 2 or 3
5. Radial sensory NCS stimulated at lateral wrist recorded at the thumb
6. Additional sensory studies may be performed if the lesion is unable to localized; these may include medial antebrachial cutaneous and dorsal ulnar cutaneous NCS
7. Additional motor studies that may be considered include recording at the FDI and performing segmental inching studies across the elbow
8. Based on initial studies, a side-to-side comparison may be warranted

Table 3.4 Motor NCS criteria for ulnar neuropathy at the elbow (from AANEM practice parameters)

The most important criterion is that with the more abnormalities that are noted, it becomes more likely that the findings represent ulnar neuropathy as opposed to artifact
(a) Abnormal motor nerve conduction velocity across elbow (<50 m/s)
(b) Nerve conduction velocity across elbow segment is more than 10 m/s slower than the forearm segment
(c) CMAP amplitude decreases more than 20% from below elbow to above elbow (conduction blow)
(d) Significant change in CMAP configuration/shape at the above elbow site compared to the below elbow site
(e) If inconclusive, consider other motor studies and segmental studies

Table 3.5 Electromyography approach to ulnar nerve entrapment

Evaluate ulnar-innervated muscles
Distal hand intrinsics
FDI[a], ADM
Include forearm musculature to look for evidence for/against an elbow vs. wrist lesion
FCU, medial heads of the FDP
If ulnar muscles are abnormal –
At least two non-ulnar C8-T1 muscles
Abductor pollicis brevis Extensor pollicis brevis Extensor pollicis longus
C8-T1 paraspinals (not necessarily diagnostic)
Additional muscles can be tested for suspicion of a brachial plexopathy if needed

[a]The FDI of the hand is most commonly abnormal in cubital tunnel syndrome

Ulnar Motor Study Technique

A routine ulnar motor nerve conduction study is set up with a recording electrode placed over the center of the abductor digiti minimi (ADM) muscle. Reference and ground electrodes are attached, and a stimulus is applied 8 centimeters proximal to the recording electrode. After obtaining an adequate compound muscle action potential (CMAP), a second stimulus is administered approximately 4 centimeters distal to the medial epicondyle. The examiner then measures 10 centimeters proximally across and above the elbow to deliver a third stimulus [6]. During this study it is important that a flexed elbow position of 90 degrees is maintained to retain tension on the nerve. If not held in this position, the nerve will have slack and redundancy to it. This gives a false reading of nerve length resulting in an error of calculation of nerve conduction velocity [7]. Once all three sites have been stimulated with optimal responses, obtained measurements are done between them to calculate nerve conduction velocity.

If a patient has ulnar neuropathy symptoms and ADM motor studies were normal, additional motor nerve testing is indicated. Some studies suggest that a first dorsal interossei (FDI) recording study may be more sensitive than ADM recording and may demonstrate findings of cubital tunnel earlier than the ADM [8]. This may be a result of the anatomical arrangement of the fascicles of the ulnar nerve at the elbow where those that supply the FDI lie closest to area of compression [8, 9]. The study involves stimulating at the wrist, below the elbow, and above the elbow while recording over the FDI muscle and again calculating conduction velocities.

Segmental Studies

A technique known as “inching” or short segmental nerve conduction studies has been described to look for an abnor-

mality across a short distance such as the elbow segment. It has been found that these studies increase detection rate as well as help to confirm the location of the lesion when routine ulnar studies were normal [10]. Initially the examiner will map out the ulnar nerve using a series of submaximal stimuli above and below the elbow. Generally 1 centimeter increments are marked off above and below the elbow. The examiner then stimulates between the points of these smaller segments of measurement to determine the exact area of velocity slowing or conduction block across the elbow [11]. Conduction block is defined as a reduction of proximal CMAP amplitude of at least 20% compared to distal CMAP amplitude [12].

Upper Extremity Anomaly Studies

Generally a median motor nerve study would be part of routine nerve conduction study so as to evaluate for an additional median neuropathy and rule out other diseases such as a peripheral neuropathy. The Martin-Gruber anastomosis between the ulnar and median nerves is the most commonly encountered upper extremity nerve anomaly [13]. The most common type involves the anastomosing of the proximal median and ulnar motor fibers that supply the FDI [14]. However, since nerve conduction studies are not as commonly done on the FDI, one may not ending up detecting this anomaly. Findings suggestive of a Martin-Gruber anastomosis on median motor studies include increased median motor study amplitudes with proximal versus distal stimulation, a small initial positive deflection on the waveform, as well as a spuriously fast conduction velocity [15]. In ulnar motor studies, it is important to be wary of this especially when ulnar motor studies demonstrate a conduction block in between the forearm and below elbow stimuli sites [15]. Though rare, there are cases of a median neuropathy with a Martin-Gruber that can mimic ulnar neuropathy at the elbow [16].

F-Wave Studies

The ulnar F-wave is a late response that is obtained similar to a motor NCS; however the stimulus is directed to the anterior

horn cell on the spinal cord as opposed to the ADM directly [17]. Having the signal travel proximally to the anterior horn and then back down the fibers to the ulnar nerve and assessing the F-wave that occurs much later than the CMAP allows one to infer the status of the entire nerve by sampling a longer segment. Thus, the F-waves in general are useful more in evaluation of a diffuse process such as a polyneuropathy as opposed to detecting a specific mononeuropathy [18].

Data Analysis

Reference values may vary by laboratory, but commonly a normal *distal motor latency* for the ulnar nerve is considered <3.7 milliseconds (ms) [19]. This can be altered by nerve compression or other dysmyelination as well as loss of axons. The compound muscle action potential (CMAP) has an *amplitude* that is measured from baseline to the negative peak [20]. Theoretically this is a measure of the proportion of functioning nerve fibers [21]. Changes of amplitude can indicate axonal damage, but a significant amplitude drop across a specific site can indicate a conduction block due to myelin injury [20]. A normal ulnar motor nerve amplitude can be between 6 and 8 milliamps [19]. Normally there should be minimal change in amplitude in above and below elbow stimulation sites [22]. A decrease in CMAP from below elbow to above elbow greater than a twenty percent drop is suggestive of ulnar neuropathy at level of the elbow [22]. *Conduction velocity* is calculated in meters/second as the speed of the fastest conducting axons between two stimulus points with a single recording point. An acceptable lower range for upper extremity studies is 50–52 m/s [19]. Across the elbow segment, velocity that is greater than 10–11 m/s slower than the forearm segment can also be suggestive of cubital tunnel syndrome. In normal patients no significant conduction velocity slowing across the elbow is expected [23].

Ulnar Sensory Studies

Ulnar sensory nerve conduction studies are performed using ring-shaped active and reference electrodes on the fifth digit

with a stimulus applied 14 centimeters proximally. The test is conducted by testing the nerve from proximal to distal, which is the opposite of the normal physiologic function of the nerve [24]. Onset latency, peak latency, amplitude, and conduction velocity are then measured. Unlike motor studies, the sensory studies are performed only over short distances and are not valuable over longer distances due to a phenomenon known as phase cancellation. This term describes the decrease in sensory amplitude as a result of increasing the distance from the recording site [25]. Limited data suggest a value to performing sensory studies above and below the elbow; however this is not a typical part of routine ulnar sensory studies [26].

Data Analysis

Onset latency represents the time when the fastest and largest sensory fibers conduct a signal to the recording site from the stimulation site [27]. An upper limit of normal of 3.1 ms has been described [19]. *Peak latency* is the time between onset of stimulation and the peak of sensory nerve action potential (SNAP), hence the name peak latency. The peak latency is more effectively reproduced and reflects a greater variety of axon sizes [28]. A reasonable upper limit for peak latency in ulnar sensory studies is <4.0 ms [19]. The SNAP *amplitude* is representative of the cumulative sum of all sensory nerve fiber depolarization. It is typically measured in microvolts (uv) with a lower limit of normal being 13 uV [19]. Unlike motor studies the *conduction velocity* of sensory studies is calculated using a single stimulation distance measurement. Typically, this is noted to be greater than 50 m/s [19].

Other Sensory Studies

Additional ulnar sensory studies can be performed, such as recording over the dorsal ulnar cutaneous nerve distribution. The takeoff for this portion of the ulnar nerve is proximal to the wrist [29]. If an abnormality is noted, it suggests a nerve injury proximal to the wrist though not necessarily at the

elbow. It can still be normal in an ulnar neuropathy at the elbow due to the fascicular arrangement of the nerve [30]. Typically, other sensory studies are done in addition to the ulnar sensory ones, with the median nerve being routinely evaluated. Studying other nerves allows one to look at possible concomitant peripheral median nerve entrapment or a peripheral polyneuropathy. If ulnar sensory studies demonstrate no abnormalities and there is suggestion via history or physical examination of a brachial plexus injury, a medial antebrachial cutaneous study can be performed [31]. Sensory studies are generally normal in a cervical radiculopathy [32].

Axonal Versus Demyelinating Lesions

In most cases the earliest electrodiagnostic findings are secondary to demyelination. Demyelinating changes result in prolongation of internodal conduction time, slowing the conduction velocity as well as possible prolongation of distal latency [33]. Conduction block may be evident. Acute lesions in ulnar neuropathy across the elbow are demyelinating 60% of the time [34]. It follows that an earlier diagnosis may improve chances of recovery before any axonal damage develops. From a recovery standpoint, a nerve that has suffered demyelination has a greater possibility of remyelinating and recovering in a shorter time frame than an axonal lesion. Secondary to the slow growth rate of axons and the need to regrow and regenerate distal to an axonal lesion, the chance of full recovery may be limited in pure axonal lesions [35].

Abnormal ulnar sensory studies alone (even for both the fifth digit and the dorsal ulnar cutaneous distribution) are not adequate for a diagnosis of ulnar neuropathy at the elbow [8]. When present, amplitude abnormalities can provide information in regard to severity of axonal damage of ulnar neuropathy at the elbow [24]. In axonal loss one would expect both DUC and digit five SNAP amplitudes to be decreased. In a pure demyelinating lesion, both distal amplitudes would be expected to be normal [28]. If only the digit five recordings are abnormal this supports a lesion at the level of the wrist,

but does not rule out an elbow lesion, as the DUC has been shown to sometimes be normal in lesions at the level of the elbow as noted above [30, 36]. It follows that an abnormality of the DUC suggests a lesion proximal to the wrist though not necessarily at the level of the elbow.

In patients who have undergone ulnar nerve surgery, studies have verified the value of repositioning the ulnar nerve with subsequent improvement in nerve conduction studies [37]. Patients with more severe nerve conduction values (specifically with those with more severe motor findings) preoperatively also show poorer recovery postoperatively [38].

Electromyography

Electromyography (EMG) involves the use of a recording electrode to evaluate electrical activity in skeletal muscle, particularly at the level of the motor unit. The motor unit is defined as the motor neuron or anterior horn cell, its axons, and the muscle fibers innervated by those axons. Thus, electromyography cannot evaluate the central nervous system or lesions "proximal" to the motor unit. Electromyography of a suspected neuromuscular condition is often performed with a needle electrode and should be done in association with nerve conduction studies [8].

This evaluation is generally safe with minimal risk. The goal of the needle EMG study is to evaluate for changes in skeletal muscle indicative of abnormalities to localize a lesion and possibly lend information about severity and prognosis. Axonal damage can be evident on EMG evaluation of a muscle in the form of abnormal spontaneous activity, abnormal motor unit potentials or diminished activation, or recruitment being present. Motor unit action potentials (MUAPs) can be evaluated with needle EMG and may demonstrate changes suggesting an acute lesion or an older lesion with signs of reinnervation. Motor unit recruitment can be diminished in the setting of clinical weakness due to neurapraxia and conduction block without having axonal damage

being present. Similarly, a patient may present with clinical signs and symptoms of cubital tunnel syndrome with a normal EMG study if a focal axonopathy or dysmyelination has not developed. Some MUAP abnormalities may be minimal and not detectable on routine EMG studies [32, 39, 40].

It is noteworthy that on the needle EMG portion of an EMG/NCS study, the EMG only evaluates motor fibers, and the majority of abnormal findings will occur in lesions with axonal loss [32]. A "sensory-only" neuropathy could therefore have a normal EMG evaluation.

Spontaneous potentials such as fibrillations and positive sharp waves will be seen in muscles of a specific nerve root or peripheral nerve distribution with absence in muscles supplied by other, unaffected nerves. These findings are the most sensitive indications of recent motor axon loss.

In an acute lesion, even with axonal loss or denervation, spontaneous activity may not be immediately present. Fibrillation potentials develop in a proximal to distal sequence in recent axonopathy. It can take several weeks to see changes in muscles. Conversely, reduced recruitment changes can develop rapidly. This can be the earliest finding but is rarely seen in isolation [32, 41, 42].

Acute lesions with axonal loss are more likely to cause membrane instability with fibrillation potentials and positive sharp waves appreciated on EMG. More mild disorders may not produce any axonal loss. Motor unit action potentials can develop polyphasicity over time if collateral sprouting occurs. This may stabilize over time in a static lesion or decrease due to reinnervation. Motor unit action potential polyphasicity alone is not enough to diagnose an active lesion but does provide evidence of prior axonal loss. Over time, fibrillation potential amplitude will decrease, and smaller amplitude fibrillation potentials can suggest an older lesion [43].

Testing Approach

As with the NCS portion of the study, a preliminary history and physical exam should be used to guide the approach to

testing. A differential diagnosis should be considered before and during testing, with a proximal to distal sample differential including central nervous system pathology, C8/T1 radiculopathy, brachial plexopathy, or ulnar neuropathy. The focus of course, with an elbow ulnar nerve entrapment, should be on ulnar-innervated muscles in the forearm and hand [8, 44, 45] (Table 3.5). Routinely tested muscles include the first dorsal interosseous of the hand (most commonly abnormal in a cubital tunnel syndrome [8]) and the abductor digiti minimi. These muscles are easily tested with needle EMG. If abnormalities are appreciated in these muscles, evaluation of the ulnar-supplied forearm musculature must also be performed. EMG abnormalities of the ulnar-innervated heads of the flexor digitorum profundus or flexor carpi ulnaris would provide evidence of a lesion proximal to the wrist [8, 44, 45].

There has been discussion about expected abnormalities of the FCU on EMG testing with ulnar nerve entrapment at the elbow. Clinical and electrodiagnostic "sparing" of the FCU muscle has been identified [46] in ulnar nerve entrapment at the elbow, even though it is rare for the ulnar nerve branch to the FCU to come off the ulnar nerve before the site of elbow entrapment. Fascicular architecture and topography of the ulnar nerve may be a reason why the FCU is more likely to be spared than other muscles with ulnar nerve entrapment. FCU fibers tend to be more medial within the ulnar nerve and possibly less susceptible to trauma. Also the relatively shorter length of these fibers from the site of entrapment to the target muscle may render them less vulnerable to a compressive lesion.

Additional testing of other C8-T1 innervated muscles is appropriate to look for an isolated or concomitant cervical root lesion [45]. Other medial cord and lower trunk muscles should be evaluated to exclude a brachial plexus lesion [8]. Overlap of a C8-T1 radiculopathy is a common concern among treating providers. If a radiculopathy is highly suspected, an evaluation of six to seven upper limb muscles including the paraspinals is optimal. Nerve conduction studies and physical exam will help to identify a concomitant

median nerve entrapment at the wrist. EMG of the abductor pollicis brevis and other distal limb muscles can be helpful in looking at a coexisting carpal tunnel syndrome or peripheral polyneuropathy [47, 48].

It is noteworthy that the presence of denervation potentials is not enough to suggest a poor prognosis. Similarly, though electromyographers generally "quantify" the amount of fibrillation potentials or positive sharp waves seen (often reported as 1+, 2+, 3+, or 4+), these data do not help in determining prognosis. A more important factor is the presence vs. absence of any voluntary motor unit action potentials during recruitment, as a complete absence of voluntary MUAPs can be considered a negative prognostic finding. Collateral sprouting where an intact axon can reinnervate other motor fibers can occur with roughly a four-to-one compensatory effect. If the surviving or unaffected axons have to compensate for more than 80% loss of other axons, they will not be able to sustain function long-term. Again, this is a more meaningful prognostic factor for a longer-term lesion. Also, the duration of symptoms should be correlated with electrodiagnostic findings, and symptoms or findings suggesting a longer duration can also portend a poorer prognosis. The distance from the site of entrapment becomes important as well. A nerve can regenerate axons from proximal to distal if an insult has been removed, but the time it takes an axon to regenerate across a longer distance can result in the collapse of neural tubule structures with scar formation before reinnervation occurs [32, 40, 49].

Reporting Data

Electrodiagnostic data should be presented, along with an interpretation stating what diagnosis is supported electrically and what diagnosis is not. Normal reference values and details of testing conditions along with the appropriate history and physical examination should be included. Generally speaking, electrodiagnostic testing does not give any informa-

tion about causality, though a clinical correlation should be presented in the context of a known or suspected trauma or injury. When available the data should be compared with prior studies. An impression or interpretation should be formulated identifying the diagnosis and specific site of nerve entrapment if available and characterizing the type of lesion as dysmyelination, axonopathy, or both. It is paramount to consider findings in the context of symptoms. For example, mild slowing of nerve conduction velocity across the elbow with normal amplitude and no evidence of conduction block has little to no meaning in a patient without symptoms. Conversely, if the patient has significant symptoms and clinical findings, the electrodiagnostic data may still be normal, particularly early in the course of a neuropathy. It is important that multiple diagnoses be listed and identified clearly in the report if they are found during testing.

Repeating the Electrodiagnostic Testing Study

Repeat testing is not necessary in patients who respond to treatment and are improving symptomatically. However, a repeat study may be helpful in patients with persistent or, in particular, worsening symptoms. Similarly, new symptoms or a change in neurologic status warrants repeat evaluation. If patients have ongoing or progressive symptoms, repeating the study may be helpful to ensure that the initial diagnosis was correct and to rule out development of a new problem such as a cervical radiculopathy. In the postoperative patient, repeating NCS and EMG can be helpful to rule out a "kinked" nerve as well as evaluate for a proximal brachial plexus tourniquet lesion. The study can also help to identify the location of the ulnar nerve after surgical relocation. If new neurological symptoms develop, changes may not be fully realized on EMG until several weeks after onset of symptoms. Nonetheless there may be some value in NCS early testing across the site of potential nerve injury, and EMG may demonstrate recruitment changes earlier than

other changes. If a patient fails to improve but is not having increasing symptoms, a repeat study may demonstrate a static lesion, and a lack of progression of neurologic dysfunction may be reassuring. Data should always be compared with results obtained from prior testing.

Neurologic recovery may occur rapidly after surgical decompression of a nerve entrapment in a purely demyelinating lesion [32, 40, 49]. If there is preoperative evidence of axonal injury, it may take time for regeneration and regrowth of surviving axons from proximal to distal. Repeat testing may help to delineate if there are any surviving axons, as some neural continuity is regarded as a positive prognostic sign. Time course becomes important, and some of the data from an EMG and NCS evaluation can be helpful in addressing chronicity (see above). In addition, it is important to remember that the ulnar nerve provides innervation to the medial heads of the FDP muscle. In the setting of a proximal ulnar neuropathy that results in FDP weakness, the patient may present initially with a less pronounced "claw" deformity due to weakness of the finger flexors allowing the digits to extend somewhat in their relaxed state. As the nerve recovers from proximal to distal, the FDP will recover function and muscle tone prior to the hand intrinsics, and the patient will actually develop more of a "claw" deformity until the nerve reinnervates the distal ulnar muscles. This phenomenon is known as the "ulnar paradox" and should not be confused with progression of neurologic injury.

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Chapter 4
Diagnostic Testing: Alternative Modalities

John R. Fowler and Brian Chenoweth

Introduction

Patients presenting with cubital tunnel syndrome exhibit a wide variety of clinical signs and symptoms. Provocative tests such as a Tinel sign, pressure provocation, and elbow flexion aid in the diagnosis, but their sensitivity is variable [1]. In an effort to improve accuracy and standardization of the diagnosis of cubital tunnel syndrome, attention has been focused on diagnostic studies. The most common studies obtained are electrodiagnostic studies. This typically includes both nerve conduction studies (NCS) and electromyography (EMG). Due to criticisms such as cost and patient discomfort, additional testing modalities such as MRI and ultrasound are being studied.

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Electrodiagnostic Studies

Of the two types of electrodiagnostic studies, NCS are particularly useful in chronic compressive neuropathy. In NCS, two electrodes are placed along the path of a nerve. The first electrode provides an electrical stimulus, and the characteristics of the subsequent nerve firing are measured by the second electrode. This measurement may be performed for pure sensory or motor nerves, with resulting sensory/motor nerve action potentials (SNAPs or MNAPs, respectively), or for combined motor and sensory nerves, with resulting compound nerve action potentials (CNAPs). Values obtained include the amplitude of the response as well as the proximal and distal latencies. The distance between the electrodes along with the proximal and distal latencies are used to calculate the nerve conduction velocity (NCV). The NCV is affected by a number of different variables including age, height, weight, among others. These variables must be considered when reporting and interpreting the results of a NCS [2].

Large myelinated nerve fibers are best detected by the electrical stimulation and recording of the NCS because they demonstrate a lower threshold stimulus and transmit the action potential faster. It is those large myelinated fibers that are most effected in chronic compressive neuropathies such as cubital tunnel syndrome [3]. In states of chronic compression, the myelin layer is damaged in nerve fibers, causing the electrical current to be leaked into surrounding areas. The resulting findings obtained by NCS include increased latency across the affected area as well as slowed conduction velocity [4]. Initially, the findings outside the area of compression are reported within the normal range because the nerve's myelin remains intact. As the compression continues, additional damage occurs to the nerve axon itself causing altered values outside the area of compression. Sensory fibers are more susceptible to compression, and as a result, the SNAP values usually decrease before the MNAPs/CNAPs [5].

In contrast to NCS, EMG specifically measures the integrity and characteristics of muscle contraction. EMG may be

performed with either surface or intramuscular electrodes. The advantage of surface electrodes is that there is reduced pain and discomfort during the exam, but this comes at the cost of reduced muscle specificity between adjacent muscles, and the recordings are also influenced by the thickness of subcutaneous tissue. Based on these factors, surface EMG is used predominantly in physical therapy settings and when muscle specificity is less important for diagnostics, such as muscle fatigue syndromes. Needle EMG involves the insertion of a needle recording electrode directly into the muscle. The electrode records activity within a 1.0–2.5 mm radius, so precision during needle tip placement is necessary. Normal muscle exhibits brief activity when stimulated by needle movement. This is measured as insertional activity and is evaluated by placing the needle into multiple locations in the muscle. The resting activity of the muscle is also observed. Finally, voluntary muscle contraction is performed by the patient, and the characteristics of the corresponding muscle unit action potential (MUAP) are recorded [6].

Normal muscle tissue demonstrates electrical silence at rest unless the electrode is close to the endplate. When the needle tip is near the endplate, miniature endplate potentials (MEPPs) may be observed. These waveforms are non-propagating and irregular and thought to be caused by mechanical irritation of the endplate. Endplate spikes (EPS) may also be observed as short biphasic waves caused by sub-threshold endplate activation of single muscle fibers. Lack of spontaneous activity, including MEPP and EPS, is indicative of denervation. MUAPs are also recorded in normal muscle and may be distinguished from spontaneous activity by more regular and slower waveforms [7, 8]. Abnormal signals include fasciculation potentials as well as positive sharp waves. When a muscle is denervated, its membrane becomes unstable leading to spontaneous depolarization. This results in cyclical activations of individual muscle fibers [9]. These activations occur at rest but may also be triggered by needle movement and are termed fasciculations. Sharp waves are monophonic waveforms of larger amplitude and are the result of the same

membrane instability that causes fasciculations. By observing the different types of waveforms and their frequency, the EMG evaluates muscle disorders in addition to peripheral and central nerve disorders.

The methods employed for electrodiagnostic studies allow for multiple potential benefits including supporting the clinical diagnosis in question, assessing the severity of nerve compression, detecting additional abnormalities causing the nerve compression, and evaluating response to treatment [10]. Despite these benefits, electrodiagnostic studies have come under increasing criticism when used for diagnosis of peripheral nerve compression. Clinical exam maneuvers for cubital tunnel syndrome including the flexion/pressure test have been reported to have variable sensitivity and specificity, but those results are often comparable to the results obtained when evaluating nerve studies [11, 12]. Electrodiagnostic studies have also been reported less reliable in patients with mild symptoms due to a high false-negative rate. In early stages of nerve compression, there may be variable compression of nerve fascicles. The remaining large fibers are able to respond normally during nerve testing resulting in a normal exam [5]. This false-negative rate has resulted in controversy of what treatment recommendations should be made for patients with a clinical diagnosis of nerve compression and normal electrodiagnostic studies [13, 14]. On the other end of the spectrum, there are issues related to patients with confounding variables or systemic disease that are frequently excluded in studies. In one example, Atroshi et al. found that 23 (18%) of 125 patients with no clinical signs or symptoms of carpal tunnel syndrome had positive findings on electrodiagnostic study of carpal tunnel syndrome [15]. In this study, the authors excluded patients with systemic disease. One must consider how much higher the number of false-positive results, already almost one in five, would be if the patients with systemic disease were included. Regarding outcomes, despite reports supporting preoperative electrodiagnostic studies as prognostic, additional studies, including one meta-analysis performed by Shi and colleagues, report

no association between preoperative studies and surgical outcome [16, 17]. In the study of electrodiagnostic testing, difficulties have been encountered due to lack of standardization and population-based reference intervals causing variability in reported sensitivity and specificity [18]. In addition, the effect of bias involving electrodiagnostic testing reporting led Turkelson et al. to conclude that the results of publications on this topic are often not beneficial to clinical practice [19]. Finally, the exam itself is uncomfortable with patients reporting pain and anxiety during testing. This includes both the nerve conduction and electromyography portion of the exam [20]. Patient distress may be significant enough to alter the results of the study. Consideration of this issue in anxious patients may require additional time in clinic for discussion and, on occasion, medication prior to the exam.

Alternative Imaging Modalities

A different diagnostic method involves imaging of anatomical changes to the nerve in states of chronic compression. Multiple types of imaging modalities have been utilized to visualize peripheral nerves including CT, MRI, and ultrasound (Fig. 4.1). The use of CT and CT myelograms for nerve visualization have traditionally been limited to conditions involving bone impingement such as nerve root impingement near the spine or in brachial plexus lesions. Based on improved ability to visualize peripheral nerves in soft tissue, the two primary imaging techniques are MRI and high-resolution ultrasound [21]. The reasoning behind this type of diagnostic technique involves ulnar nerve swelling proximal to the site of compression at the cubital tunnel [22]. This was demonstrated by Yoon and colleagues in an ultrasound-based study comparing the cross-sectional area of the ulnar nerve in various places in the arm in both controls and patients with cubital tunnel. In their study, control patients maintained a 1:1 ratio of nerve size when comparing the diameter in the arm, near the epicondyle, and in the mid forearm. In contrast, the patients with cubital

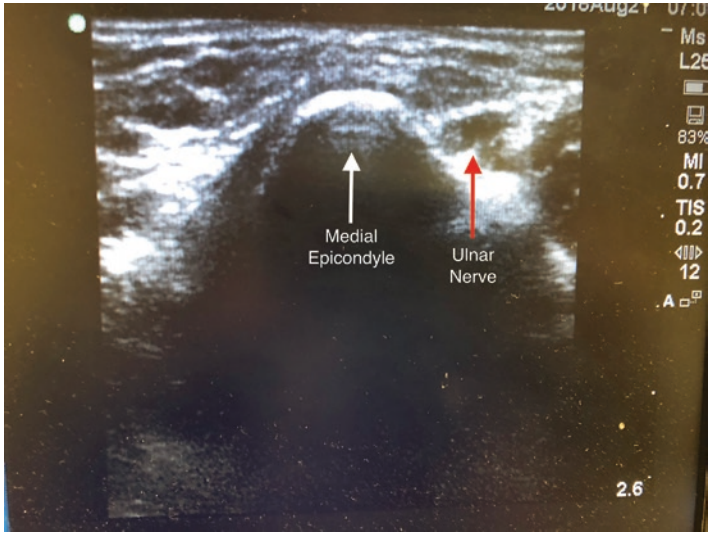

FIGURE 4.1 Cross-sectional ultrasound image demonstrating the position of the ulnar nerve relative to the medial epicondyle

tunnel demonstrated a near threefold increase in size of the ulnar nerve just proximal to the cubital tunnel compared to measurements at the arm and forearm [23]. Similar findings have also been demonstrated in the median nerve in patients with carpal tunnel syndrome [24]. The pathophysiology of the nerve swelling is likely related to damage to the protective layering of the nerve causing alterations in microvascular permeability of the blood-nerve barrier. As demonstrated in animal models utilizing Evans blue staining and compression of large nerves, the epineurium was most susceptible to compression followed by the endoneurium. As the amount of compression and length of time of compression is increased, the epineural layer is effected first causing isolated swelling. The endoneural layer is effected next causing both swelling and alterations to conduction. Additional anatomic findings that have been reported to result in swelling of the nerve include fibrotic changes of intrafascicular, endoneural, and epineural

tissue [25–27]. At this time, the exact mechanism of swelling is unclear in patients with cubital tunnel as anatomic studies have been limited to animal models and a precise model of chronic compression neuropathy has not yet been developed.

Despite its inception in the 1970s, it wasn't until the early 1990s that MRI was utilized for imaging of peripheral nerves [28]. Since that time, additional sequencing techniques and increasing use of 3 T scanners have further refined the use of MRI for pathologies such as cubital tunnel syndrome. One of the principal benefits of MRI for diagnosis of cubital tunnel syndrome is that it provides additional information about the anatomy of the elbow as well as potential etiologies for the causative diagnosis. Although the majority of cases of cubital tunnel are idiopathic, an identifiable etiologic factor may be found in up to 42% of cases [29]. MRI has been utilized in identifying underlying causes such as trauma, degenerative arthritis, overuse, masses, and mass-like lesions such as pigmented villonodular synovitis or peripheral nerve sheath tumors, scar tissue, infection, among others [30]. MRI is also useful in identifying morphological characteristics of the nerve including caliber and continuity in addition to pathologic changes within the nerve itself such as fibrosis and edema represented by altered signal intensity or enhancement. These findings have been found to have high correlation with abnormal findings on EMG and NCS [31, 32]. Abnormal signal characteristics were not found, however, to correlate with resolution of symptoms when serial MRIs are obtained [33]. Consequences of chronic nerve compression and muscle denervation, such as atrophy in corresponding muscles, may also be observed.

Based on current MRI standards, the sequences that should be obtained include axial, sagittal, and coronal planes of T1, T2 fat saturation (FS), short tau inversion recovery (STIR), proton density (PD), and a postcontrast T1 FS if contrast is utilized. The best sequences for studying anatomic characteristics including cross-sectional area of the nerve are the axial T1 and PD. The STIR and T2FS are fluid sensitive and useful for evaluating abnormal characteristics within the

nerve or surrounding tissue such as edema. If a tumor is suspected based on history or clinical exam, intravenous contrast should be administered with appropriate postcontrast sequences obtained. These standards will continue to evolve as new technology, such as capacity for three-dimensional reconstructions, emerges [30]. The cost of this newer technology is notable and significantly higher than some of the alternatives discussed here. The latest MRI scanners cost several million dollars, and individual exams are typically billed over 1–2000 dollars for a single upper extremity study [34]. This is in contrast to NCS/EMG at approximately half that cost or ultrasound at slightly less compared to nerve studies if not available in office for no cost. One other notable downside is for patients who experience claustrophobia. In order to obtain the desired sequences, patients may undergo exams that take from 45 to 60 minutes. This may be intolerable to some requiring sedative medications or scans in lower resolution open MRIs.

Similar to MRI, the use of high-definition ultrasound has dramatically expanded since its original description for examination of peripheral nerves in 1988 [35]. In addition to diagnostic utility in states of chronic nerve compression, it is utilized for evaluation of hand and upper extremity soft tissue masses, traumatic or overuse tendon and ligament pathology, foreign bodies, vascular flow, and as an adjunct for injections and aspiration of fluid collections [35]. Benefits for use in evaluation of cubital tunnel syndrome include low cost, noninvasive technique, absence of radiation exposure, as well as potential for immediate and portable access. In addition, the ultrasound is unique in that it may be performed as a dynamic assessment of nerve position evaluating for subluxation.

One of the criticism of ultrasound as a diagnostic technique is that results are operator dependent. This effect can be reduced by use of appropriate equipment in a stepwise exam. The exam is best accomplished with a high-frequency (12–18 MHZ) linear array transducer. The higher-frequency transducers demonstrate higher resolution in a shallow depth

of field in contrast to low-frequency transducers which can penetrate further but at the expense of resolution. For the exam, the patient may be placed into one of two positions. In the supine position, the patient is placed into a relaxed position on the exam bed with the arm abducted to 90 degrees in an externally rotated position with the elbow flexed to 90 degrees (Fig. 4.2). In this position the nerve may be traced proximally and distally with ease and also evaluated during elbow range of motion. Alternatively, the patient may be seated on the exam table with the shoulder extended and internally rotated. The elbow is slightly flexed, and the palm is placed flat on the exam table (Fig. 4.3). The ultrasound operator is positioned behind the patient for this exam, and although it is a comfortable and easy setup, it does not allow for dynamic assessment during range of motion as demonstrated in the supine position [36]. In either position, the probe should be placed perpendicular to the course of the ulnar nerve near the cubital tunnel at the level of the medial epicondyle for a cross-sectional view. Once the nerve is identified, it can be traced proximally into the arm and distally into the mid forearm. The site of maximal nerve swelling,

Figure 4.2 Position 1 for ultrasound allowing for a dynamic assessment of nerve size and position

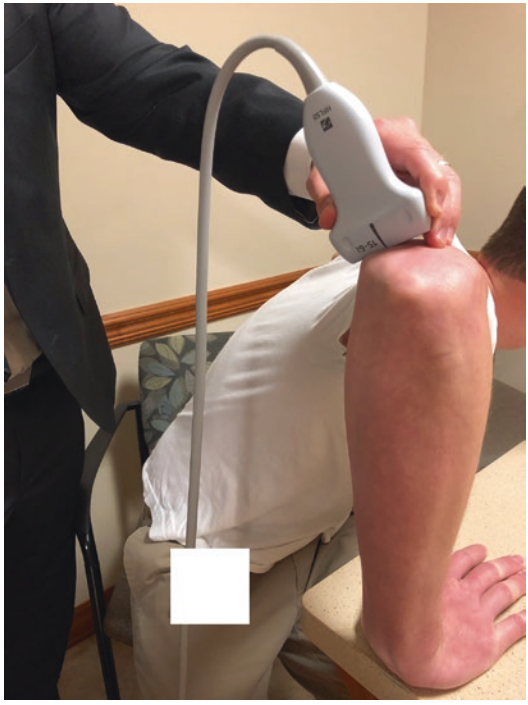

Figure 4.3 Position 2 in the seated position

typically at the level of the epicondyle or in the supracondylar region, is identified [23]. Using either the freehand drawing tool or the ellipse tool, the nerve is traced, and the cross-sectional area is obtained. This value may then be compared to reference standards, to other areas demonstrating reduced swelling proximally or distally, or to the contralateral arm. It is important to position the transducer as close to perpendicular to the nerve as possible when measurements are performed. If the transducer is angled when the nerve is measured, apparent elongation will occur, and the measurement will be artificially elevated [36]. The nerve may also be viewed in long axis for additional comparison of swelling and consideration of continuity. Finally, adjacent soft tissues may

also be evaluated for causes of nerve compression such as cysts, tumors, and aberrant muscles.

In a 1994 study by Wiesler, 15 elbows with a clinical and electrodiagnostic diagnosis of cubital tunnel were evaluated with ultrasound and compared to 60 controls. The mean cross-sectional area of the ulnar nerve was measured at 19.1 mm^2 in patients diagnosed with cubital tunnel based on exam and nerve studies and 6.5 mm^2 in controls. These findings demonstrated a correlation coefficient of 0.8 when compared to nerve conduction velocity across the elbow when a cutoff value of 10 mm^2 was used [37]. This study involved evaluating the nerve from 4 cm above to 4 cm below the elbow and measuring the point of maximal swelling. No recommendations were made to routine scanning of a specific location. Similar findings were produced in a study by Terayama. In their study of 28 patients with ulnar neuropathy, the recommendation was made for a cutoff value of 11 mm^2 with resulting sensitivity and specificity over 90% for both MRI and ultrasound when measured 1 centimeter above the medial epicondyle. Values obtained by both MRI and ultrasound in 12 patients were compared and found to demonstrate no statistical difference [38]. The amount of swelling was also noted to be significantly larger in patients with severe symptoms, defined as McGowan grade 3, compared to those with mild symptoms, defined as McGowan grade 1 or 2. Additional studies on cutoff values have ranged from 8.3 to 11 mm^2 with most evidence suggesting use of 10 or 11 mm^2 [23, 39].

As detailed in the study performed by Wiesler, diagnostic sensitivity and specificity with imaging compares favorably to that of nerve studies. In a prospective study by Ellegaard and colleagues, the sensitivity of ultrasound was found to be 80.5%, compared to 71% for nerve conduction studies [40]. Podnar et al. performed a double-blind prospective study to evaluate the relationship between ultrasound and electrodiagnostic studies. In 106 patients, the authors noted high correlation between the cross-sectional area at the retrocondylar groove and maximal slowing of the nerve conduction velocity at that location [41]. This comparison has also been performed

in patients with carpal tunnel syndrome with similar results [24]. Unfortunately, electrodiagnostic studies demonstrate inconsistent findings after nerve decompression. The response of nerve swelling following decompression, as measured on ultrasound, has been evaluated in one study by Duetzmann et al. [42]. The authors found that the cross-sectional area of the ulnar nerve decreased after surgical release in a statistically significant fashion, but it did not return to normal values. Interestingly, 12 of the 48 patients did not show any change in nerve size following decompression.

The study of imaging modalities for diagnosis of chronic nerve compression has gained significant support over recent years, but there is still a need for additional research. Additional studies comparing ultrasound/MRI with results from electrodiagnostics and clinical exam are warranted to determine if the results obtained in the studies detailed above can be replicated. Due to low cost, potential for immediate results in office, and a comfortable exam, the ultrasound examination shows promise. Given the potential for lack of provider experience with ultrasound, additional studies regarding the learning curve as well as intrarater and interrater reliability would also be helpful. As further data on imaging is obtained, it will become more clear how to use ultrasound and MRI in instances where clinical exam and electrodiagnostic results are unclear or as a complete alternative to electrodiagnostics.

Summary

The gold standard confirmatory test for cubital tunnel involves a combination of nerve conduction studies and electromyography. Additional research is being performed on imaging studies, including MRI and ultrasound, as an alternative option to electrodiagnostic studies. To date, several studies have been published showing similar diagnostic ability between electrodiagnostic studies and both MRI and ultrasound. Ultrasound, in particular, shows promise attributable to its lower cost, immediate results, and comfortable exam, but additional research is needed to confirm its diagnostic utility.

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Chapter 5
Cubital Tunnel: Nonsurgical Management

Nicole J. Jarrett and David M. Kahan

Background

Many authors have studied the roles of nonsurgical management of cubital tunnel syndrome. It is helpful to first review the basic science work that serves as the theoretical basis of the interventions.

Mackinnon describes three stages of compressive ulnar neuropathy: dynamic ischemia, demyelination, and axonal loss [1]. It is through careful history, clinical examination, and interpretation of electrodiagnostic studies that help identify where a patient is on this spectrum of disease. Dynamic ischemia is defined by compression of the ulnar nerve during specific positioning or movement of the elbow that results in transient decreases in neural perfusion. Symptoms occur during the ischemic event and resolve when blood flow is

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returned. Electrodiagnostic studies are likely not sensitive enough during this stage of disease, as conduction velocity of the fastest conducting fibers have not yet slowed [1].

Through the work of Apfelberg on the dynamic anatomy of the ulnar nerve at the elbow, it has been shown that the cubital tunnel flattens and narrows by 55% with elbow flexion [2], which renders the nerve susceptible to dynamic ischemia [3–5]. Gelberman et al. quantified this positional compression by studying extraneural and intraneural pressures in cadaveric specimens during elbow flexion and extension [6]. They showed that extraneural pressure measurements increased from 7 mm Hg to 28 mm Hg with flexion and intraneural pressure measurements increased from 8 to 41 mm Hg with flexion. The lowest mean extraneural and intraneural pressures of the ulnar nerve occurred when the elbow was flexed to 40–50°. Conversely, the highest pressures were recorded with the elbow in maximal flexion, which was approximately 130°. It should be noted that the elbow in full extension also recorded higher pressures than when the elbow was flexed between 30 and 70°.

In addition to the increases in pressure with elbow flexion, Apfelberg also showed that the nerve elongates approximately 4.7 mm with the elbow in flexion [2], and others have demonstrated an even greater elongation with abduction of the shoulder and extension of the wrist [7]. Histologic and electrodiagnostic studies have confirmed decreased blood flow and axonal transport with compression and tension on a nerve [8, 9].

Due to the subcutaneous nature of the ulnar nerve at the elbow, it is also subject to mechanical compression. This is evidenced by reports of postoperative ulnar neuropathy secondary to inadequate padding and positioning in the operating room [10].

With prolonged ischemia there is demyelination of the ulnar nerve axons. Extraneurally, fibrotic changes occur that are thought to affect the ability of the nerve to glide [5, 11, 12]. It is during this demyelinating stage that symptoms worsen and become more frequent, and electrodiagnostic

studies show decreased conduction velocity [1]. Chronic or severe compression may eventually lead to axonal loss, which presents with constant symptoms, changes in two-point discrimination, motor weakness, and muscle atrophy. Decreases in signal amplitude, abnormal insertional phase activity, and fibrillations during the resting phase on electrodiagnostic studies represent a decrease in functioning axons and demise of neuromuscular junctions.

Many of the nonsurgical treatments of cubital tunnel syndrome are therefore aimed at decreasing the above-described compressive and tensile forces on the ulnar nerve in order to decrease the frequency and/or severity of ischemic events. After extensive work on staging cubital tunnel syndrome, Dellon showed that mildly affected patients achieved better outcomes with conservative therapy, whereas those with higher scores were more likely to need surgical treatment. He reported that 50% of patients with a minimal degree of nerve compression had excellent results by nonsurgical techniques [13, 14]. Additionally, Iba studied intraoperative cubital tunnel pressures and found that those with severe neuropathy had the highest pressure measurements with elbow flexion [15]. Therefore, halting or reversing the progression from dynamic ischemia to axonal loss is the goal of nonsurgical interventions. Once there is progression to moderate or severe stages of disease, the efficacy of nonsurgical management dramatically decreases.

Activity Modification

Commonly prescribed nonsurgical measures include discontinuing triceps exercises, avoiding the application of direct pressure to the medial aspect of the elbow by avoiding resting the medial elbow on firm surfaces, maintaining a resting elbow position of 40°–50°, limiting wrist extension and shoulder abduction, and nerve gliding exercises [16, 17]. This may require the patient to modify habits and the work environment [18, 19]. Although elimination of these inciting events has been

shown to provide relief, there is no data to support that work-related activities are a causal risk factor for development or progression of cubital tunnel syndrome [20]. Similarly, there is no clinical literature demonstrating that nerve gliding exercises that cause the nerve to glide proximally and distally through coordinated arm and neck movements, as an isolated intervention, successfully treat cubital tunnel syndrome [16].

Beekman studied 74 patients who had cubital tunnel syndrome, of which 46 patients were treated conservatively and 28 were treated surgically [21]. Those treated conservatively had more mild disease: intermittent paresthesias and mild intrinsic weakness. The instructions for conservative treatment included avoiding leaning on the elbow, avoiding crossing the arms while sitting, and keeping the elbow extended as much as possible. At 6 months 35% of the conservatively treated patients achieved improvement, and 11% experienced complete remission. It was noted that those with only sensory symptoms did not progress with any worsening symptoms or any motor involvement.

Splinting

In combination with activity modification, splinting has been shown to be efficacious in the nonsurgical treatment of cubital tunnel syndrome. Splinting can hold the elbow in an optimal position, protect the ulnar nerve from mechanical compression or trauma, and serve as a reminder to the patient to be compliant with activity modification.

Dimond reported an 86% improvement of symptom severity in 73 patients who underwent splinting over an average of 8.7 months [22]. In a study of 22 patients with electrodiagnostically confirmed cubital tunnel syndrome, Seror showed that night splints that limited flexion from 15° to 60°, when worn for 6 months, resulted in improvement in symptoms in all patients [23]. Additionally, of the 17 patients who had repeat electrodiagnostic studies, 16 showed improvement. It was noted that the best responders were those who initiated splinting less than 3 weeks after the onset of symp-

toms. Nocturnal paresthesias were the first symptom to resolve. The more severely affected patients had a more prolonged recovery but did show signs of improvement with sensation and strength. In this series, three of the patients had undergone prior surgical decompressions, and all three saw improvements in symptoms. Clinical improvement of these three was reported as ranging from 60% to 95%.

Three common elbow splints were evaluated in an anatomic study by Apfel, which found that the Pil-O-Splint elbow support (IMAK Corp, IA), the Hely and Weber orthosis (Body Glove Corp, CA) (Fig. 5.1, [16]), and a folded towel wrapped around the elbow all prevented the elbow from flexing 90° [24].

Shah et al. performed a prospective evaluation of 19 patients (25 extremities) with cubital syndrome treated with activity modification and a 3-month course of rigid night splinting in 45° of elbow flexion [25]. Treatment failure was defined as progression to operative management. Twenty-four of 25 extremities had at least 2 years of follow-up. Twenty-one of 24 (88%) of extremities were successfully treated without surgery. Quick Disabilities of the Arm, Shoulder, and Hand scores and Short Form-12 physical summary scores both significantly improved. Additionally, grip strength improved, and ulnar nerve provocative testing resolved in 82% of patients.

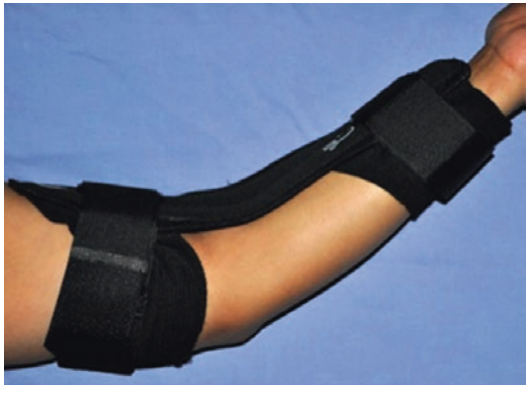

FIGURE 5.1 Hely & Weber orthosis used in this investigation. The orthosis can be adjusted to keep the elbow at 45° of flexion

Splinting with patient education, nerve gliding exercises with patient education, and patient education alone were compared in a randomized trial of 58 patients with mild to moderate cubital tunnel syndrome performed by Svernlöv [26]. No significant differences were found between the three groups. Ninety percent of patients improved at 6 months, and 10% required surgical intervention. Of note, most patients in this series had unremarkable pre-intervention electrodiagnostic testing.

Injections

Steroid injections into the cubital tunnel have not enjoyed the success rates of those performed for treatment of carpal tunnel syndrome. Beneficial response has been reported to be low in several studies [19, 27]. Hong studied 12 patients, divided into 2 groups [28]. Both groups had closely monitored nocturnal and intermittent daytime splinting with elbow padding. One group also received a steroid injection into the cubital tunnel. After 6 months, splinting alone was sufficient in treating mild symptoms, and no additional benefit was seen from the injection.

vanVeen performed a randomized, double-blind, placebo-controlled trial of corticosteroid injections for patients with ulnar neuropathy [29]. All patients had positive electrodiagnostic or positive ultrasound findings. Participants were randomized in a 1:1 ratio between a US-guided injection of 1 ml containing 40 mg methylprednisolone acetate and 10 mg lidocaine hydrochloride (Depo-Medrol; Pfizer) or a placebo injection with 1 ml of NaCl 0.9%. Additionally, all patients received education about activity modification. After 3 months, 9 of 30 participants allocated to corticosteroid injection had a favorable outcome compared with 7 of 25 participants allocated to placebo injection. There was no significant difference in outcome between the groups. Of the patients who had positive electrodiagnostic studies, repeat

electrodiagnostic examination showed improvement in 7 of 19 participants allocated to corticosteroid injection and 6 of 17 participants allocated to placebo injection. Again, there was no significant difference in outcome between groups. The mean duration of symptoms was 9 months in participants with a favorable outcome and 16 months in those with an unfavorable outcome, irrespective of the allocated treatment. Complications were seen in both groups (four in the steroid group, one in the placebo group) and included injection site pain, local swelling, hand swelling, and depigmentation.

Despite the above results, there has been much attention in the literature given to injection technique. One such study by Hamscha demonstrated a reliable ultrasound-guided perineural injection in 21 cadaveric limbs [30] (Figs. 5.2 and 5.3 [30]). Ink was successfully injected into the perineural sheath in the cubital tunnel in 21 of 21 limbs.

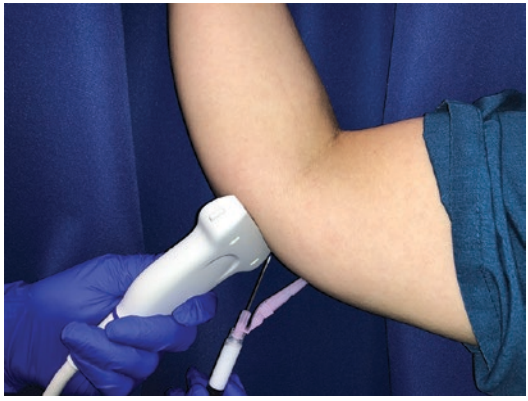

FIGURE 5.2 Schematic illustration of the injection procedure. Right arm in supination, with elbow in 90° flexion (anterior view). The injection is performed with the US transducer held in a plane aligned to the transverse axis of the elbow. The needle is advanced to the infiltration site aligned with the transducer plane

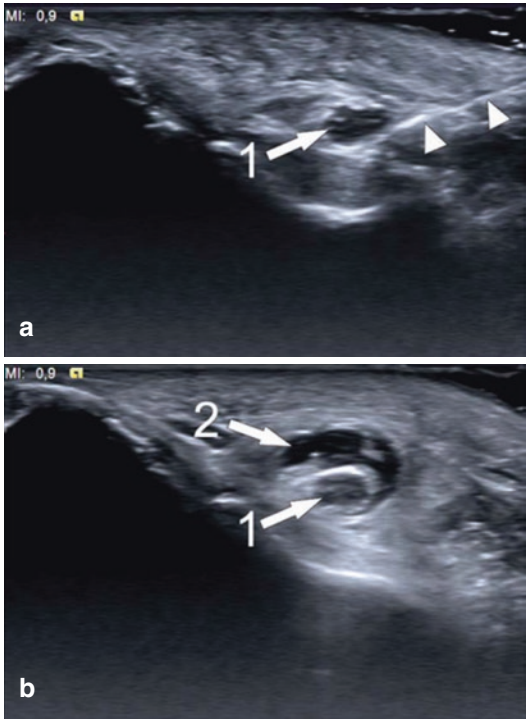

FIGURE 5.3 US-guided injection. (**a**) US image at the beginning of the injection. The needle (arrowheads) has been advanced to the perineural sheath of the ulnar nerve (1). (**b**) US image after the injection process. The ulnar nerve (1) is surrounded by hypoechoic injection fluid (2). The needle has been withdrawn. (Color figure can be viewed at wileyonlinelibrary.com)

Conclusion

As cubital tunnel syndrome is so common, successful non-operative treatment would be ideal. Mild cubital tunnel syndrome can be successfully treated with nighttime splinting and activity modification. Unfortunately, many studies into corticosteroid injection, including ultrasound-guided, have not demonstrated any significant improvement over placebo.

We therefore recommend a trial of splinting and activity modification for mild cubital tunnel syndrome and surgical intervention for persistent or advanced cases, including those with any evidence of denervation.

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Chapter 6
Nonsurgical Management: The Role of Therapy

Jodi Seftchick

When a patient presents to therapy for treatment of cubital tunnel syndrome, a detailed medical, work, home, and leisure history should be obtained. The therapist should evaluate active range of motion (AROM), passive range of motion (PROM), grip/pinch, upper body strength, coordination, sensation, pain, and functional outcome measures for thoroughness.

Patient education and activity modification are the most critical aspects for a successful therapy program and improved functional outcomes. It is important that the patient understands and attempts to eliminate aggravating conditions. A compliant motivated patient will rehab from this condition quicker and with less complications than a patient that continually exacerbates their symptoms.

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Please refer to Chap. 5 for activity modification, ergonomic adjustments, patient positioning, splinting, injections, and protecting the nerve from external pressure to alleviate symptoms; with that in consideration, there is no clear-cut protocol for the treatment of cubital tunnel. The therapist must have a solid anatomy foundation and progress of the patient while monitoring the patient's response to treatment. This chapter will highlight techniques and treatment that have been proposed to alleviate symptoms.

Treatment

If the patient has been prescribed an orthosis, they should begin weaning from it once the pain has begun to decrease and inflammation subsides. The patient should be cautioned not to dispose of their orthosis at any time. They may need to return to wearing it if symptoms return or if they are participating in an activity that may aggravate their condition.

After increased time has been spent with patient education and perhaps immobilization, the first stage of rehabilitation is to begin AROM of the involved extremity in a pain-free range. Joint mobilizations and aggressive PROM are contraindicated at this time. The patient's program should also include shoulder, wrist and digit AROM, as well as core strengthening.

Typically a patient will present with full AROM of the elbow; however, if end range elbow motion is lacking, the therapist can glide the ulna in a posterior direction relative to the humerus using the treatment plinth as a fulcrum [1]. Medial and lateral gliding of the ulna relative to the humerus is generally performed to increase elbow AROM and joint play. The 30-degree angulated alignment of the distal humerus and sigmoid notch is an important anatomical relationship for the clinician to understand to ensure proper direction of force application during mobilization [2]. Again, all movements should not increase pain; the patient's pain level will guide treatment duration, intensity, and frequency.

Modalities

Therapeutic modalities can be utilized to reduce pain and increase extensibility of structures in conjunction with therapeutic exercises.

Thermotherapy agents, such as moist heat or fluidotherapy, can be utilized pre-treatment to assist with increasing extensibility of tissues and for increased comfort with exercises. The benefits of combining heat and stretch to enhance tissue elongation cannot be overemphasized [3].

Cold therapy or ice can be utilized anytime during the healing process but is most effective for pain relief during the initial flare-up stage. Placing an ice pack on the elbow area can be tricky. Patients are encouraged to freeze water in a paper cup with a tongue depressor/popsicle stick. This ensures a quick and effective application. Patients should be made aware of the physiological effects of cold: first they will feel cold, followed by burning, pain/discomfort, tingling, and numbness [4]. In this author's experience, the patient needs to get to the numb phase for pain relief. Patients should be precautioned to avoid applying ice for too long and to avoid prolonged icing over a superficial nerve. It may take a repeated application for several days before symptoms subside [5].

Ultrasound (US) has shown to be effective with overuse conditions, owing to its nonthermal effects, which include both cavitation and microstreaming [6]. Low intensity and appropriate frequency can increase the sensory and motor conduction rate of a nerve. High-intensity US is contraindicated and has been shown to have negative effects on the nerves. Hong et al. compared US intensity for nerve compressions and found that an intensity of 0.5 W/cm^2 and a frequency of 1.0 MHz increased the recovery rate of the nerve, while an intensity of 1.0 W/cm^2 and 1.0 MHz frequency slowed the rate of nerve recovery [7].

Low-level laser therapy (LLLT) has some research to support its use with cubital tunnel syndrome. LLLT is thought to reduce inflammation, increase blood supply, stimulate nerve

function, and reduce pain. Ozkun and colleagues [8] investigated the efficacy of LLLT and US in the treatment of ulnar nerve entrapment. Thirty-two patients were randomized into two groups: US treatment (frequency of 1 MHz intensity of 1.5 W/cm^2 continuous mode) and LLLT (0.8 J/cm^2 with 905 nm wavelength), both applied five times a week for 2 weeks. At the first month, significant improvements were noted for both groups with no statistically significant difference between them. They concluded that both LLLT and US provided improvements in clinical and electrophysiological parameters and have satisfying short-term effectiveness in the treatment of ulnar nerve entrapment.

Electrotherapy devices may be used for decreasing pain and enhancing tissue healing; however, there are not any studies in the literature at this time specific to conservative cubital tunnel syndrome and effectively decreasing symptoms with electrotherapy.

All modality applications should be monitored for effectiveness. If progress is not noted after a 2- to 3-week application, then the treatment plan should be modified.

Techniques

Myofascial techniques involving a gentle sustained stretch may assist with taking pressure off of the ulnar nerve. It is critical to "unload" or place the proximal portion of the nerve on slack while gently stretching the flexor pronator muscles [9]. The patient can perform this stretch by themselves by bending their head to the affected side, elevating their scapula, supinating the forearm, extending the elbow, and grasping the hand with the opposite hand to stretch the wrist into extension. This stretch can be gradual and done in a smaller range of motion if the patient's nerve is irritable. The patient can then return the scapula and head to neutral, as the wrist and ulnar digits are placed in slight flexion.

According to Porretto-Loehrke [9], a mnemonic that can assist with the concept of neural mobilization is FLOSS (*F*ix

the adjacent joint, *L*imit ROM, *O*scillate proximal or distal (or both) to the level of compression, *S*low rhythmic motion, and *S*ymptom-free). Nerve flossing attempts to restore normal kinematics of the nerve and surrounding structures to decrease pressure. Flossing exercises should be performed carefully and gently as so to not increase nerve irritability and pain. It is not uncommon for patients to experience increase of symptoms in the initial stages of nerve flossing. An example of ulnar nerve flossing exercises includes "the mask" or "OK sign" exercise where the patient makes an "OK" sign with their thumb and index fingers and then turns their hand upside down and places their fingers flat on their face. The patient can then tilt their torso side to side to increase excursion (Fig. 6.1a, b). A more advanced flossing treatment would be to have the patient perform the "plate" exercise. The patient holds a plate with the palm flat. They then revolve the wrist under the arm toward their back, and then they press the arm up overhead while balancing the plate. Five to ten repetitions should be performed to floss the nerve but not over stress it (Fig. 6.2).

Nerve gliding exercises may limit fibroblastic activity, decrease scar formation, prevent adhesions, and decrease

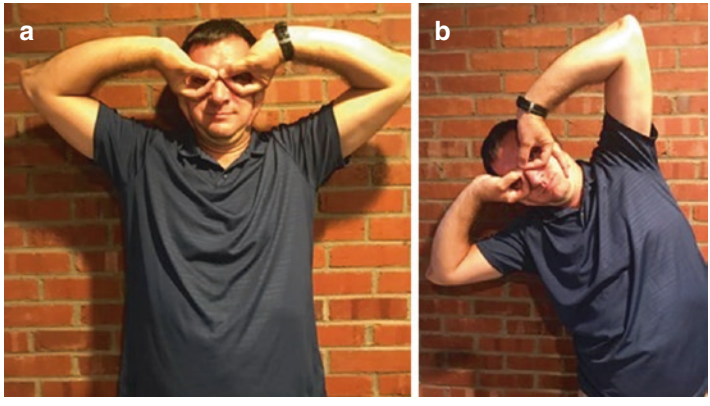

FIGURE 6.1 (**a**, **b**) Ulnar nerve conservative therapy 2018.jpg

Figure 6.2 Ulnar nerve conservative therapy 2018.pptx

fluid pressure around the nerve. Lund and Amadio [10] stated these maneuvers can help delineate where the problem lies and, if performed properly, could help although they are not confident that it is the nerve itself that is being mobilized.

The concept of nerve tension (stress and strain) and glide (excursion) plays a major role in the planning treatment [11]. These "glides" and "slides" may make symptoms better but they can also exacerbate symptoms. The slide creates strain within the nerve by pulling on both ends of the nerve simultaneously, causing the nerve to unfold [12]. The glide refers to placing tension on the nerve at one place while it is released at another. Treatment and exercises should be performed in the pain-free and tension-free range of motion. It may be necessary to begin at remote sites away from the speculated position of entrapment and advance toward the site of involvement as the patient tolerates, thus beginning at the wrist or shoulder for cubital tunnel (Figs. 6.3 and 6.4a, b).

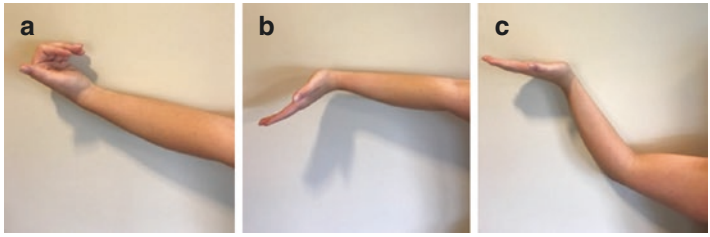

Figure 6.3 (**a**–**c**) Ulnar nerve conservative therapy 2018.jpg

A common exercise for cubital tunnel is to have the patient place the nerve on slack proximally by elevating the scapula and side bending their head toward the affected side with the forearm supinated (Fig. 6.5). The patient then performs gentle ring and small finger extension as they elevate and depress the scapula, as if they were shrugging their shoulders. If they are able to tolerate elbow flexion and extension in a short arc of motion, then that can be added for one repetition every 1–2 seconds to maximize excursion of the ulnar nerve.

There are several types of elastic tape on the market that can be used to stretch the fascia around the cubital tunnel and allow increased space for the ulnar nerve (Fig. 6.6). When placing the tape, the patient's arm should be placed in end range flexion, and the path of the ulnar nerve should be followed from the middle of the humerus to the wrist. Maximal stretch of the tape is placed over the cubital tunnel area to prevent end range flexion and assist with reminding the patient to avoid the flexed position. Tape can be left on the patient for as long as it sticks and feels beneficial to the patient.

Strengthening

Nirschl [13] recommended that the patient should be able to perform a firm handshake without increased pain prior to the initiation of strengthening exercises. Very low or no weight is recommended to start. Low weight with high repetitions of 20–40 per set will improve blood flow to the area and assist with increasing muscular endurance.

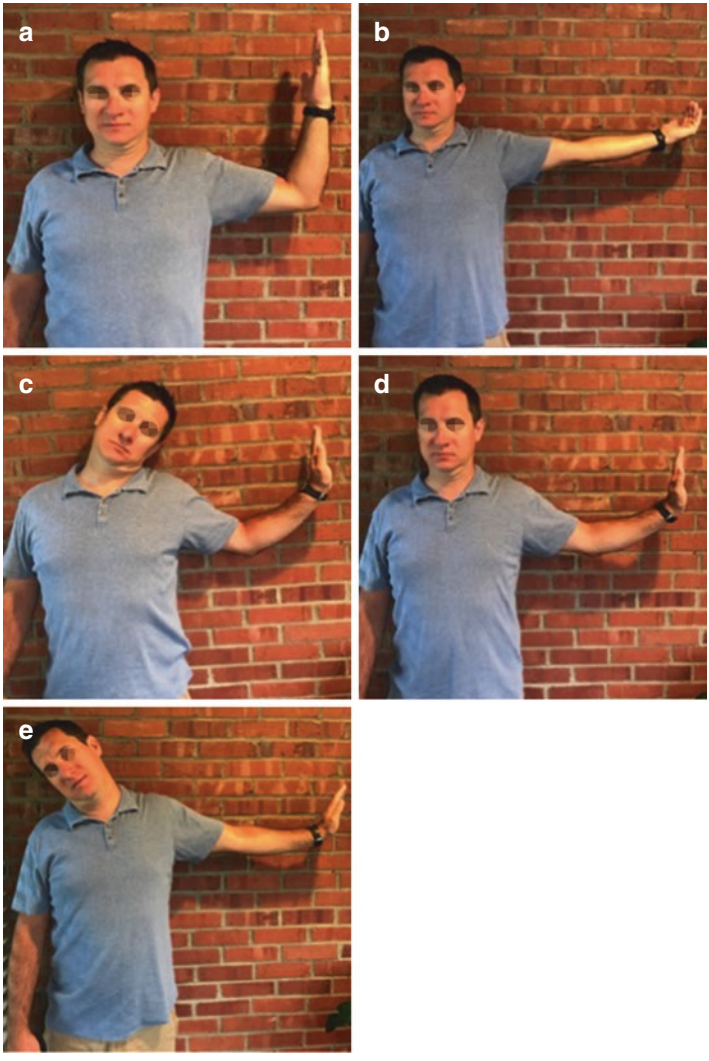

Figure 6.4 (**a**–**e**) Ulnar nerve conservative therapy 2018.jpg

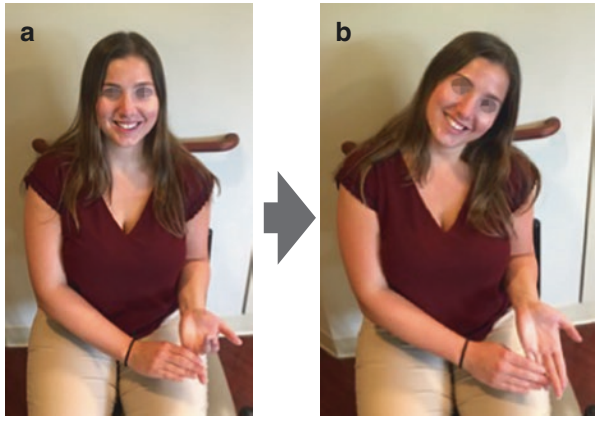

FIGURE 6.5 (**a**, **b**) Ulnar nerve conservative therapy 2018.jpg

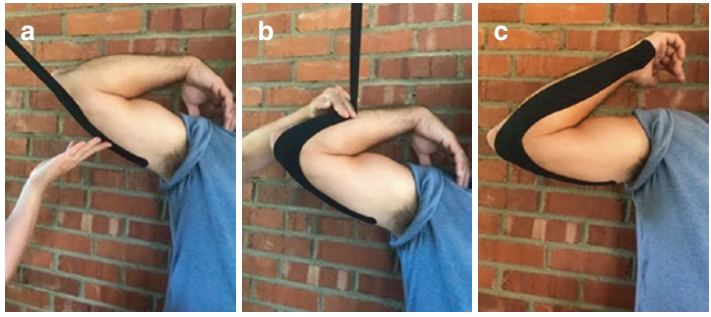

FIGURE 6.6 (**a**–**c**) Ulnar nerve conservative therapy 2018.jpg

Submaximal strengthening should begin with no resistance and then progress to isometric, isotonic, and finally isokinetic exercises. Additional exercises to strengthening the proximal muscles and core will assist with enhancing the overall rehab process. Rotator cuff strengthening and abdominal core exercises will promote muscular strength, as well as endurance for activities of daily living. Exercises can begin with closed chain and progress to plyometric exercise as needed. Plyometric

exercises should be incorporated for athletes in the last phase of rehabilitation and only if pain has been eliminated.

If there was weakness or atrophy of the intrinsic hand muscles that was noted, then dexterity and coordination exercises, as well as compensatory strategies, should be instructed for optimal hand function.

Return to Sport

If the patient desires to return to a throwing sport, then a specific throwing program should be established and monitored by a knowledgeable therapist. An interval-throwing program is designed so that each level will gradually return motion, strength, and confidence in the patient's throwing arm before the next level is initiated. Proper throwing techniques must be employed or the athlete risks reinjury and lingering pain.

Outcome Studies

Dellon and colleagues reported conservative treatment of ulnar nerve symptoms was beneficial in approximately 90% of their patients with mild symptoms; additionally, they concluded that 38% of their patients with moderate symptoms were also treated effectively nonoperatively [14].

Oskay et al. stated, although cubital tunnel is a common entrapment neuropathy and a well-defined clinical entity, the conservative treatment is not well documented and treatments are unclear [15]. The objective of their case series was to describe the effect of nerve mobilization techniques in the standard conservative management of cubital tunnel.

Their treatment plan consisted of 25% pulsed US at an intensity of 1.0 W/cm^2 for 5 minutes to the ulnar nerve traces for 10 sessions 3 times a week at the start of treatment. They then incorporated nerve mobilization of sliding techniques, which consists of gliding the nerve by alternating movements of at least two joints in which one movement loads the periph-

eral nervous system while the other movement simultaneously unloads the nervous system. It is speculated that by doing the mobilization, it would not place increased tension on the nerve. The nerve will slide around the surrounding tissue.

The patients were placed supine with the therapist placing their upper extremity into variable positions. Each position was applied five times in all sessions. Patients were to perform home exercises 10 times per day. Ice was applied posttreatment for 15 minutes to decrease pain and inflammation effects. Patients attended treatment sessions three times a week for 8 weeks. After 2 weeks, gentle upper extremity exercises were introduced. By the eighth week, Swiss ball exercises were added as closed chain exercises. At the end of 8 weeks, the patients were instructed to continue the planned exercises for 1 year. Ergonomic modifications and postural adaptations were instituted. They included seven patients and concluded that conservative treatment may be beneficial for selected patients with mild to moderate symptoms. All patients demonstrated improvement by the end of the treatment sessions.

Coppieters and colleagues [16] reported a case study of a 17-year-old female who had a traumatic onset of cubital tunnel. They reported using nerve gliding techniques and segmental joint manipulations. Their treatment plan incorporated a sliding technique that alternated combined movements of (at least) two joints in which one movement loads the peripheral nervous system while the other movement simultaneously unloads the nervous system. This technique was thought to glide the nerve without aggravating the nervous system. They also perform a tensioning technique, which alternately loaded and unloaded part of the peripheral nervous system. Passive manual joint mobilizations and manipulations were utilized for the articular dysfunctions to the elbow and thorax. Two weeks into treatment, the patient's home exercise program was upgraded to include the use of light free weights for the upper body to optimize tissue healing, to restore the tissue health of the target tissues of the ulnar nerve, and to reintegrate neuromotor control.

The patient reported no symptoms after participating in six treatments over a 1-month period. This improvement was

maintained after 6-week and 10-month follow-ups. The authors assumed that the sliding of the nerve against the surrounding structures and intraneural sliding of the nerve against its surrounding structures have a similar physiological effect and that endoneurial circulation is enhanced during neurodynamic mobilizations. Based on this principle, they preferred sliding techniques over tensioning techniques because they hypothesized that tensioning techniques induce more tension and elongation of the nerve segment, which is known to compromise intraneural circulation [17]. They also concluded that movement-based management may be more beneficial in the conservative management of cubital tunnel syndrome than the traditional recommendation of partial immobilization.

There was one case report in the literature by Kearns [18] that showed positive and rapid resolution of elbow symptoms using thrust manipulation of the elbow and carpals that had a positive impact on the patient's symptoms and functional abilities. The authors, however, cautioned limited reliability and validity of the examination and treatment methods and the nature of a case report. One must be cautious not to infer a causal relationship between thrust manipulation and resolution of symptoms.

Svernolov et al. [19] evaluated three methods of conservative treatment: avoidance of provoking symptoms, splints, and nerve mobilizations for 57 patients over a 6-month period. Fifty-one patients (89.5%) were improved at follow-up with no significant differences between the groups for any of the variables. They reported that night splints and nerve gliding exercises did not help the patients and patients with mild or moderate symptoms have a good prognosis if they are informed of the causes of the condition and how to avoid provocation.

Summary

There is not a definite consensus as to the length of conservative treatment once symptoms have decreased. It has been reported anywhere from 90 days up to 1 year [20, 21]. If symp-

toms return the patient should be instructed to reinstate their home exercise program.

Nonsurgical management may be used as long as the patient desires; however 3–6 months seems sensible based on the literature. If despite all conservative treatment, symptoms persist, then surgical management should be considered.

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Part II
Surgical Treatment

Chapter 7
Simple Decompression (In Situ and Endoscopic)

Mark Baratz, Jennifer D'Auria, and Cassidy Costello

Historical Background

The earliest reported surgical treatment of ulnar nerve compression at the elbow occurred in 1816 [1]. At that time, Henry Earle excised a segment of the ulnar nerve proximal to the cubital tunnel in a 14-year-old girl with ulnar nerve paresthesias [1]. While this addressed her symptoms, this procedure left her with significant motor and sensory deficits. The first report of decompression came in 1878 by Emile-Paul Fèvre [1]. He described the procedure as liberation and elongation of the nerve; however, the technique was criticized and largely abandoned due to recurring symptoms [1]. As many of the earlier documented cases of ulnar neuritis were secondary to trauma or severe arthritic deformities, excessive tension on or friction of the ulnar nerve was identified as the underlying source of neuropathy. Thus, for nearly 80 years,

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the predominant treatments were not simple decompression but anterior transposition or medial epicondylectomy.

In the 1950s, three surgeons—Geoffrey Vaughan Osborne, William Feindel, and Joseph Stratford—encountered patients with ulnar nerve symptoms that did not have the typical bony deformities seen in the past. It was their contributions that laid the foundation for simple decompression as a reliable surgical option. In 1957, Osborne identified a fibrous band of tissue between the two heads of the flexor carpi ulnaris (FCU) as a source of compression of the ulnar nerve particularly with the elbow flexed [2]. He likened this tissue, now known as Osborne's ligament, to the transverse carpal ligament noting that at the area of tension, the nerve was flattened and proximally it was enlarged [2]. He concluded that in cases of clinically and radiographically normal elbow joints, this fibrous band and not excessive traction or frictional trauma on the nerve was the source of idiopathic ulnar neuritis [2]. Osborne argued that for early and mild cases of neuritis, simple decompression was all that was needed and provided a brief description of the technique [2]. He placed a 3-inch incision over the ulnar nerve at the elbow and dissected to expose the swollen nerve. The aponeurosis of and fibrous band of tissue between FCU was divided to expose the flattened nerve. Subcutaneous fat was sutured on top of the nerve for protection. The skin was closed, and the arm was immobilized in extension for 10 days.

While Osborne has received lasting acknowledgment with his eponymous ligament, Feindel and Stratford even more clearly described and coined the terms cubital tunnel and cubital tunnel syndrome in 1958 [3]. Like Osbourne, they identified "focal constriction and retrograde edema" of the ulnar nerve at a restricted opening distal to the ulnar groove [3]. They called this point the cubital tunnel where the ulnar nerve transitions from a superficial to a deeper submuscular course [3]. They described the floor of the tunnel as the medial ligaments of the elbow joint and the roof as the aponeurotic arch between the medial epicondyle and olecranon where the heads of the FCU are attached [3]. In addition to but deep to

the aponeurosis, they discovered a thicker fibrous band that accentuated the compression [3]. They theorized that compression at the cubital tunnel is exacerbated with activities of flexion as the roof becomes tauter and the floor of the tunnel balloons out [3]. They also noted that the FCU was largely spared as its motor contributions from the ulnar nerve branch proximal to the cubital tunnel [3]. They went on to define cubital tunnel syndrome as a tardy nerve palsy where there is a focal constriction of the ulnar nerve that spares the FCU and is relieved with simple decompression [3].

Relevant Surgical Anatomy

While cubital tunnel syndrome was more narrowly defined by Feindel and Stratford, it has become the colloquial term given to ulnar neuropathy at the elbow (UNE). To understand the sites of compression and the structures that need to be released during decompression, the anatomic course of the ulnar nerve must be understood. In the upper arm, the ulnar nerve lies in the anterior compartment posteromedial to the brachial artery. It travels from the anterior to the posterior compartment through the medial intermuscular septum at the level of distal insertion of the coracobrachialis, which is approximately 10 cm proximal to the medial epicondyle of the humerus [4]. While lying on the anterior aspect of the medial head of the triceps, the ulnar nerve travels through the arcade of Struthers, which is a fibrous canal that extends from the intermuscular septum to the medial head of the triceps approximately 8 cm proximal to the medial epicondyle of the humerus [4]. At the elbow, the ulnar nerve continues its course posterior to the medial epicondyle and then travels through the cubital tunnel. After entering the forearm between the humeral and ulnar heads of the FCU, the ulnar nerve continues its course in the anterior compartment of the forearm between the flexor digitorum profundus and FCU.

From proximal to distal, potential sites of compression of the ulnar nerve include the medial intermuscular septum,

arcade of Struthers, osteophytes from medial epicondyle, cubital tunnel retinaculum (Osborne's ligament), aponeurosis of the two heads of FCU, and the deep flexor/pronator aponeurosis [4]. The most common site of compression at the elbow is at the cubital tunnel [5]. While Feindel and Stratford based their original description of the cubital tunnel on only three patient dissections, O'Driscoll et al. provided a more detailed picture in a cadaveric study of 27 elbows [3, 6]. The roof of the cubital tunnel includes the aponeurosis between the two heads of the FCU and variations of the cubital tunnel retinaculum [6]. Type 0 had an absent retinaculum which resulted dislocation of the ulnar nerve from its typical location behind the medial epicondyle. Type Ia had a normal retinaculum with its fibers oriented transversely and perpendicular to the FCU aponeurosis. With flexion, the retinaculum became taut but did not compress the nerve. Type Ib had a thick retinaculum that compressed the nerve. Type 2 had the retinaculum replaced by the anconeus epitrochlearis. A variable incidence of this muscle has been reported to be 4–34% [7]. The floor of the cubital tunnel was comprised of posterior and transverse medial collateral ligament and the elbow joint capsule [6].

Similar to the cubital tunnel retinaculum, the arcade of Struthers has variability. Cadaveric studies have characterized the arcade of Struthers less as a localized band-like structure and more as a canal. In a cadaveric study of 11 specimens, von Schroeder and Scheker determined the average length of this canal structure to be 5.7 cm with its superior end 9.6 cm and distal end 3.9 cm proximal to the medial epicondyle [8]. The narrowest portion of the canal was at the proximal end in all specimens [8]. It is our bias that the arcade of Struthers is not typically a site of compression except in the case of anterior transposition of the ulnar nerve where it may act as pivot point of compression if not released [8]. In a larger study of 25 specimens, the arcade of Struthers was found in only 68% of specimens [9].

Similarly, it is our opinion that the medial intermuscular septum has been incorrectly characterized as a site of compression. In individuals with cubital tunnel syndrome, it does

not compress the ulnar nerve. Only when the nerve has been transposed will this structure, if left intact, apply focal pressure to the ulnar nerve.

Another structure of importance, particularly to avoid postoperative complications, is the medial antebrachial cutaneous nerve (MABC). There are two discrete layers of subcutaneous tissue on the medial aspect of the forearm separated by a thin fascial layer. The MABC resides in the deep layer. Branches of the MABC are generally thought to be crossing the ulnar nerve 6 cm proximal and distal to the medial epicondyle [10]. Cadaveric studies provide conflicting reports on the precise location of branching and where the majority of these branches cross the ulnar nerve [11–13]. A more recent cadaveric study based on 40 specimens showed that there was an average of 2.95 branches of the MABC that crossed the ulnar nerve [13]. Seventy-seven percent of branches were found distal the medial epicondyle at an average distance of 2.87 cm [13]. Overall, the variability and superficiality of the MABC make it prone to injury if careful dissection is not undertaken.

Indications

In general, surgical treatment is recommended for patients who present with significant disease and who have failed conservative treatment [14, 15]. As a first-line treatment, surgical intervention is advised in patients with objective evidence of sensory or motor impairment such as increased two-point discrimination or intrinsic atrophy [15]. Failure of conservative treatment is also an indication for surgical treatment [14]. Dellon et al. calculated the probability of patients needing surgical treatment based upon disease severity. For patients with mild disease, those having subjective symptoms without objective sensorimotor deficits, there was 21% probability of requiring surgical management [15]. For patients with moderate disease, those with weakness or atrophy, there was a 33% probability [15]. For patients with severe disease, those with

significant atrophy or sensory abnormalities, there was a 66% probability of requiring surgical management [15].

The ultimate goal of surgery for ulnar nerve compression is to relieve areas of constriction and tension. While many treatment options exist, the procedure must match to the clinical findings and diagnosis for it to be successful. Decompression can release soft tissues causing constriction but does not address a subluxating or dislocating nerve, osteophytes, post-traumatic bony deformities, or the presence of masses or ganglions.

In Situ Decompression

Osborne's original description of simple decompression, where only the roof of the cubital tunnel was divided, is close to what is generally performed during a modern version of this technique. The senior author's preferred technique begins with a 3–4 cm curved skin incision between the medial epicondyle and olecranon (Fig. 7.1). Subcutaneous dissection defines the two layers of the subcutaneous fat, and branches of the MABC are sought out in the deep layer

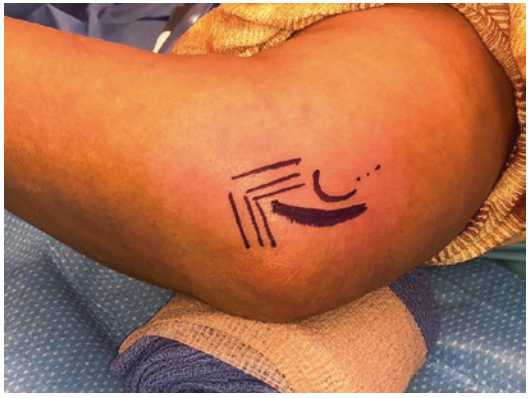

Figure 7.1 Curved incision placed posterior to the medial epicondyle and between the two heads of the FCU

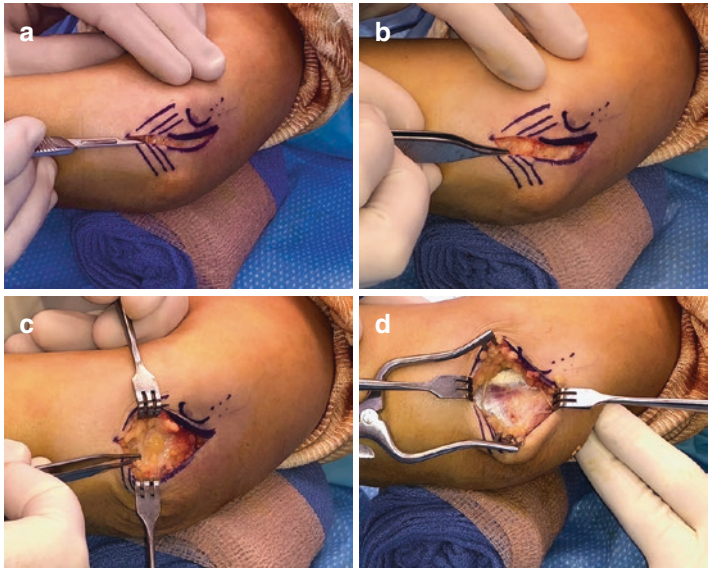

FIGURE 7.2 Subcutaneous dissection. Superficial layer of fat (**a**) contains veins (**b**). Deep layer of fat (**c**) contains branches of the medial antebrachial cutaneous nerve (**d**)

(Fig. 7.2a–d). We define the two heads of the FCU. The ulnar nerve can be initially identified posterior to medial head of the triceps, where the nerve enters the cubital tunnel, or slightly distally between the two heads of the FCU. There is value to having a familiarity with several approaches to the ulnar nerve, particularly in the setting of fracture and revision surgery. The ulnar nerve is decompressed, including both the superficial and deep fascia from the cubital tunnel distally until it is surrounded by healthy perineural fat (Figs. 7.3 and 7.4). This is typically 5–6 cm from the epicondyle. The nerve is decompressed proximal to the epicondyle until it is surrounded by loose areolar tissue (Fig. 7.5). Again, this is approximately 5–6 cm from the epicondyle. A circumferential neurolysis is not performed in the absence of prominent osteophytes or extrinsic masses on the lateral aspect of the nerve [5]. The elbow is taken through a range

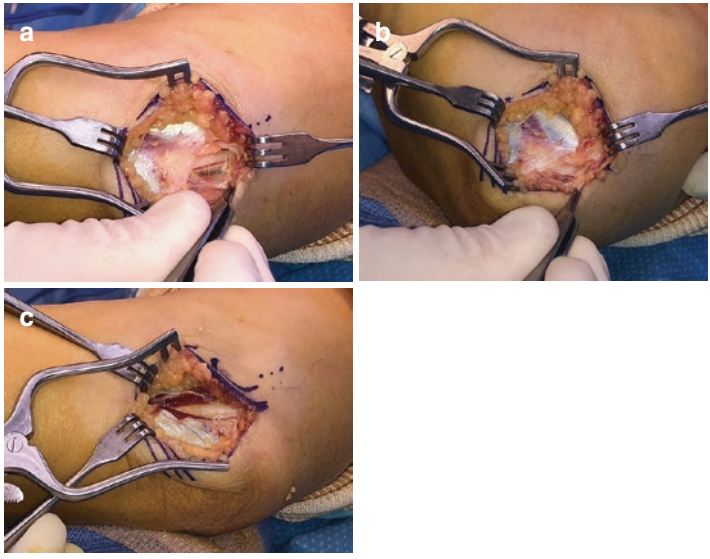

FIGURE 7.3 Ulnar nerve coursing under Osborne's ligament (**a**) that becomes more taught with elbow flexion (**b**). Release of Osborne's ligament and fascia over the heads of FCU (**c**)

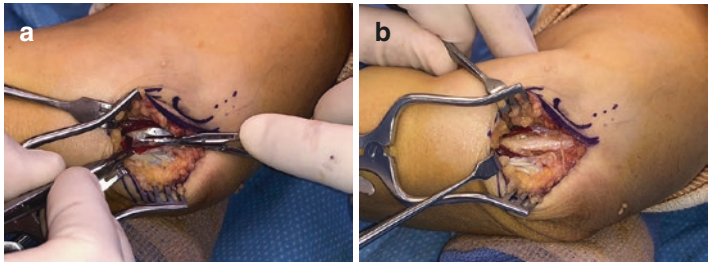

FIGURE 7.4 Distal release of deep fascia over the ulnar nerve (**a**) to achieve decompression until healthy fat surrounds nerve (**b**)

of motion to assess for subluxation or dislocation of the nerve (Fig. 7.6). The definition of subluxation is subjective and somewhat controversial. If the nerve crests the medial edge of the medial epicondyle, we typically chose to convert to a subcutaneous transposition. In combination with

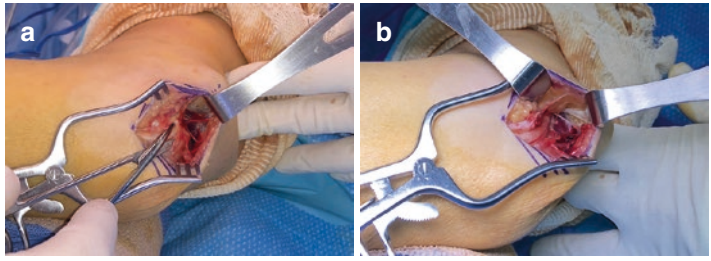

FIGURE 7.5 Proximal exposure of the ulnar nerve (**a**) with release until it is surrounded by loose areolar tissue (**b**)

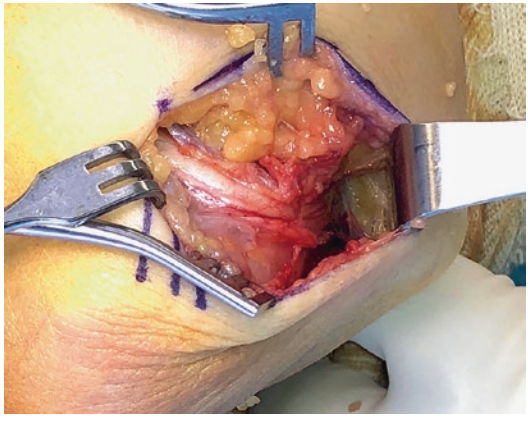

FIGURE 7.6 The elbow is flexed intraoperatively to evaluate for ulnar nerve subluxation or dislocation which is not seen in this case

decompression, there are series of partial medial epicondylectomy to manage subluxation, as well as fascial slings to prevent anterior translation of the nerve [16–19].

In situ decompression provides improvement in symptoms. Broken down by specific symptom, Goldfarb et al. reported outcomes in 69 limbs treated with in situ decompression [20]. Immediately after surgery, 94% had improved elbow or forearm pain, 91% had improved hand pain, 89% had improved sensation, and 85% had improved weakness [20]. At final follow-up at a mean of 4 years, these percentages remained

high at 86%, 85%, 82%, and 85%, respectively [20]. Only 7% had persistent symptoms requiring an additional procedure, in their series, anterior submuscular transposition [20].

As anterior transposition and not simple decompression was the mainstay of treatment prior to the 1960s, it took time for the outcomes of simple decompression to find a place in the treatment of cubital tunnel syndrome. In 1979, Clark reviewed 135 decompressions [21]. Sensory recovery was reported as nearly complete and on the timescale of weeks; however, motor recovery was slower with improvements seen up to 5 months and more consistent in patients <35 years old [21]. In 1995, another retrospective series reported favorable results in 164 decompressions [22]. Within 6 months postoperatively, 89% had excellent or good relief of symptoms [22]. This percentage decreased to 79% at an average follow-up of more than 4 years later [22]. Average return to work was 20 days [22]. Many other studies with smaller study groups have also reported favorable results [23–27].

In 2005, several separate prospective randomized studies compared decompression and transposition. Nabhan et al. reported on 66 patients with clinical and electrodiagnostic evidence of cubital tunnel syndrome [28]. There were no significant differences at 3 and 9 months postoperatively with regard to pain, motor and sensory deficits, and motor conduction velocities [28]. Gervasio et al. found no differences in 70 patients with severe cubital tunnel syndrome when comparing simple decompression and anterior submuscular transposition [29]. At 6 months, 80% of simple decompressions and 83% of transpositions had excellent or good outcomes when looking at a number of factors: residual symptoms, subjective improvement, work status, grip strength, and two-point discrimination [29]. Bartels et al. published the largest comparative series: 152 patients treated with either simple decompression or anterior subcutaneous transposition [30]. Interestingly, subluxation or dislocation of the ulnar nerve was not part of the exclusion criteria, which was found in 27% of the decompressions and 29% of the transpositions [30]. At 1 year, excellent or good results were achieved in

65% of decompressions and 70% of transpositions. This difference was not statistically different [30]. There were no significant differences when further subdivided either by preoperative grade or between ulnar nerve subluxation and dislocation [30]. However, an important finding in this larger cohort was that decompressions had a lower complication rate: 9.6% vs. 31.1%, risk ratio 0.32, CI 0.14–0.69 [30].

Other factors outside of patient outcome make simple decompression an attractive option. First, simple decompression takes less operating room time. A prospective randomized study found a significant difference in operative times: 13.7 minutes for simple decompression and 31.3 minutes for transposition [31]. This finding is also reflected in an analysis of the National Survey of Ambulatory Surgery that reported that transposition was on average 11 minutes longer than simple decompression [32]. Second, simple decompression allows patients to return to work sooner. Patients who underwent simple decompression returned to work in roughly 9 days compared to 20 days for transpositions [31].

Trends in procedures performed reflect the growing acceptance of simple decompression as a valuable treatment of UNE. Based on analysis of the National Survey of Ambulatory Surgery from 1996 to 2006, the percentage of transposition decreased from 49% to 38% [32]. Based on a Florida ambulatory surgery database from 2005 to 2012, simple decompression represented 70% of procedures performed in 2005, and this proportion increased by 27% in 2012 [33]. Transposition had the opposite trend: it constituted only 27% cases in 2005 and this proportion decreased by 67% in 2012 [33].

Considering that transposition requires more operating room time and results in longer return to work without proven benefit in outcome, simple decompression may appear to be the logical choice for UNE secondary to compression. However, a Cochrane review from 2016 concluded that overall there was insufficient evidence to provide one recommended treatment for UNE [34]. This review simply concluded that simple decompression and transposition were equally effective in improving clinical and neurophysiologic markers

and that transposition had more wound infections (RR 0.32, CI 0.12–0.85) [34].

Endoscopic Decompression

Tsai et al. were the first to publish the technique on endoscopic decompression in 1994 [35]. Subsequent descriptions with greater detail on technique were published in 1995 and 1999 [36, 37]. Tsai's approach is an inside-out approach with custom glass tubes inserted into the tunnel surrounding the ulnar nerve. Since Tsai et al. published their technique of endoscopic release, multiple different techniques with various specialized instrumentation have been described; however, no study has compared the techniques directly. Other inside-out techniques include Mirza et al. with the Stratos Endoscopic Release System (A.M. Surgical, Smithtown, NY), Cobb et al. with the EndoRelease Endoscopic Cubital Tunnel Release System (Integra LifeSciences, Plainsboro, NJ), and Bain et al. with the Agee device (3M, Orthopedic Products, St. Paul, MN) [38–41].

Hoffman et al. developed an outside-in approach in that instrumentation is not inserted within the tunnel surrounding the ulnar nerve [42, 43]. It uses a specialized instrument set that includes illuminated speculums, an endoscope with dissector tip, and long blunt-tipped scissors (Hoffman Cubital Tunnel Set, Karl Storz, Tuttlingen, Germany) [42, 43]. A 1.5–2 cm incision is placed over the retrocondylar groove. Dissection is carried down to the ulnar nerve with clear identification of the epifascial structures. Both distally and proximally to the incision, spreading with blunt instruments creates a tunnel superficial to the nerve tunnel. Distal dissection is first carried out by direct visualization with illuminated speculums. Then, the speculum is removed, and an endoscope with a dissector tip is used for the remainder of the procedure. Long blunt-tipped dissection scissors are used to release structures (Fig. 7.7a). Hoffman et al. comment that an advantage of their technique is that it achieves a longer length of

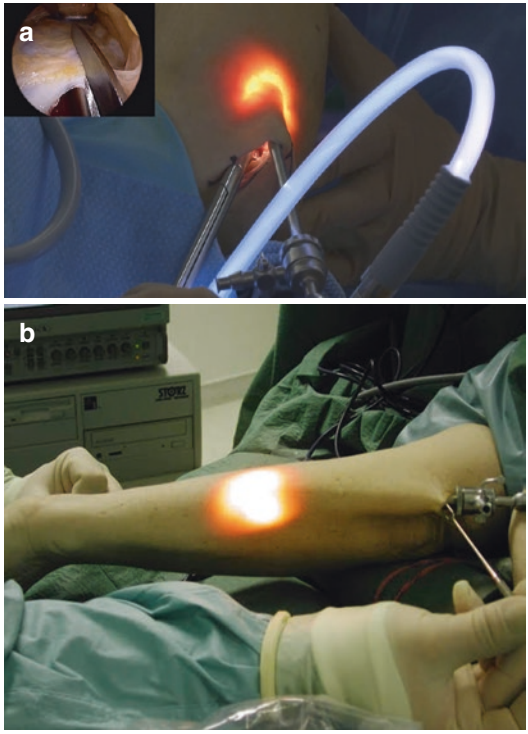

FIGURE 7.7 (**a**) An endoscope fitted with a dissector tip creates a space superficial to the cubital tunnel to allow for full visualization to release structures with long blunt-tipped dissection scissors. (**b**) Light on endoscope highlights the length of decompression possible with the Hoffman endoscopic technique. (Photos courtesy of Dr. John Lubahn)

decompression of up to 10 cm proximally and distally to Osborne's ligament (Fig. 7.7b) [42].

Initial results of the endoscopic techniques were promising. Excellent or good Bishop scores were high—Tsai et al. 87% (n = 85 elbows), Cobb et al. 94% (104 elbows), and Hoffman et al. 94% (n = 76 elbows) [37, 43, 44]. Proposed advantages of endoscopic decompression include decreased incision tenderness and reduced time to return to work and

activities of daily living [35]. With an endoscopic approach, 76.4% patients returned to full activity only 2–7 days after surgery compared to 18.6% patients with simple decompressions [45]. This difference in return to work is even more magnified when comparing endoscopic decompression at 8 days and anterior transposition at 71 days [46].

More recent literature has aimed to directly compare simple and endoscopic decompression. A systematic review found 80% excellent or good outcomes, 12% complication rate, and 3% reoperation rate for 425 simple decompressions and 72% excellent or good outcomes, 9% complication rate, and 2% reoperation rate for 556 endoscopic decompressions [47]. A further meta-analysis of 190 patients found a significantly lower complication rate for endoscopic decompressions with subgroup analysis showing significantly higher incision tenderness and elbow pain for simple decompressions [47]. Analyzing 417 simple and 691 endoscopic decompressions, another review confirmed this difference in complication rates showing that endoscopic technique has a reduced odds of complications (OR 0.280, 95% CI, 0.125–0.625) [48].

However, a prospective randomized study comparing simple versus endoscopic decompression provides a different perspective. At both early (16 weeks) and long-term (17 months) follow-ups, there were no significant differences between the two techniques in pain or in Bishop score; however, there was a significantly higher postoperative hematoma in the endoscopic group (24% vs. 4%) [49]. Endoscopic surgery also took significantly longer by 25 minutes [49].

Complications

Injury to the MABC, particularly the posterior branches, has been reported to be one of, if not, the most common complications of cubital tunnel surgery causing scar tenderness, painful neuromas, and paresthesia [10]. In our experience, it rarely is a complication of our patients but is often the reason for referral due to failed ulnar nerve decompression at the elbow.

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Chapter 8
Anterior Transposition in Cubital Tunnel Syndrome

Joseph Said and Asif M. Ilyas

Introduction

Ulnar nerve transposition is a technique first described by Curtis in 1898 [1, 2]. He described subcutaneous transposition of the ulnar nerve with good results. While the procedure gained acceptance, concerns grew regarding the superficial location of the transposed nerve and its vulnerability to trauma. It was thought that recurrent neuritis may also develop if the nerve inadvertently relocated back to its retrocondylar position. Two modifications addressed these concerns. In 1917, Klauser described an "intramuscular" modification by moving the nerve into the anterior flexor muscle bellies [3], with the theory being that the surrounding

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muscle prevented recurrent neuritis as well as restraint from relocation. In 1942, Learmonth described another modification in which the ulnar nerve was transposed in a submuscular location for the theoretical advantage of greater stability and greater protection against neuritis [4]. It was not until 1957 that Osborne described the in situ release alone of the ligament that bears his name as an alternative method of treatment from cubital tunnel syndrome [5]. He reported on 13 cases with "results comparable with those after anterior transposition."

Advocates for anterior transposition of the ulnar nerve believe that in situ decompression alone does not address the dynamic tension and traction on the ulnar nerve during elbow flexion. For example, Gelberman et al. measured intraneural pressure of the ulnar nerve at the cubital tunnel in human cadavers at varying angles of elbow flexion [6]. With progressive elbow flexion, intraneural pressure significantly increased up to 45% more than the extraneural pressure at 135 degrees of flexion. The pressure measurements did not significantly change after simple decompression. Checkles performed conduction velocity measurements on healthy nerves. With elbow flexion, the nerve experiences a decrease in conduction velocity with normal elbow flexion beyond 90 degrees [7]. For these reasons, Kleinman argued that anterior transposition was superior to simple decompression because it relieves natural and pathologic stretch that reduces oxygen tension to the nerve [8].

Proponents of in situ decompression cite more limited dissection and manipulation of the nerve, thereby protecting the perineural blood supply. It also reduces the risk of associated nerve injury to the ulnar nerve and its branches and reduces operative time and incision length. These features have reduced the risk of complications with in situ decompression relative to anterior transposition [9–12].

Today, both anterior transposition and in situ decompression are commonly performed. Despite greater complications reported in some studies, the clinical benefit of one type of procedure over another has not been convincingly shown [9, 12].

Rather, current trends indicate the selective use of transposition in certain situations. Specifically, anterior transposition is generally performed for preoperative or perioperative nerve instability, recurrent or persistent neuropathy after prior in situ decompression, neuropathy associated with existing hardware or lesions within the cubital tunnel, and posttraumatic neuropathy. This practice pattern is reflected in recent surveys [13, 14]. Novak and Mackinnon surveyed the American Society for Surgery of the Hand (ASSH) members on choice of procedure and found that 86% of respondents use more than one procedure for cubital tunnel syndrome [13]. In another survey of ASSH members, Yahya et al. found that surgeons most commonly performed open in situ decompression for cubital tunnel syndrome [14]. However, 79% of respondents stated their choice would differ if nerve subluxation was present. The following is a review of the literature comparing these procedures in detail.

Anterior Transposition Versus In Situ Decompression

Although several studies have compared transposition vs in situ decompression of the ulnar nerve in cubital tunnel syndrome, the best approach remains unclear. Bartels et al. performed a review of the literature in 1998 and found that irrespective of preoperative nerve dysfunction, patients undergoing transposition performed worse than patients undergoing in situ decompression only [15]. However, when sub-analyzed by severity, patients with greater severity (McGowan Grade 3) had better outcomes with transposition procedures. Of note, the authors reported no randomized studies in the analysis and only three prospective studies.

Mowlavi et al. performed a meta-analysis of 30 studies from 1945 to 1995 including 903 patients treated nonoperatively and operatively [16]. They used an outcome status determination algorithm applied to each study's patients and found that for mild-staged cases of cubital tunnel syndrome,

all forms of management including nonoperative were effective. For moderate-staged cases of cubital tunnel syndrome, submuscular transposition produced the highest percentage of total relief and satisfaction and highest percentage of normal strength. For severe-staged cases of cubital tunnel syndrome, results were inconsistent with transposition cases having a greater percentage of pain relief but simple decompression providing a greater percentage strength recovery.

Macadam et al. performed a meta-analysis and identified 10 studies with 449 in situ decompressions and 457 anterior transposition procedures [9]. Because of variable reporting of outcomes, they used an odds ratio of improvement vs no improvement as their metameter. The analysis detected no significant difference between procedures. Although a trend toward improvement with transposition was found, the meta-analysis was insufficiently powered. The data was also limited by an abundance of retrospective reviews, concerning for potential selection bias.

In an attempt to counter the potential selection bias, Zlowodzki et al. performed another meta-analysis of only prospective randomized studies [12]. The results demonstrated no significant difference between the two procedures. However, the analysis was limited to only four studies in which three did not describe whether surgeons were blinded to each patient's procedure allocation.

Although clinical improvement may be comparable, some studies have demonstrated increased complications with transposition. Zhang et al. reviewed short-term complications of in situ decompression vs transposition procedures and found more complications with transposition procedures [11]. In a prospective comparison study, Staples et al. found greater short-term complications and greater narcotic use in the transposition group [10]. Bartels et al. found increased infection (9.3% vs 2.5%) and less sensation (18.6% vs 2.5%) in the transposition group vs in situ decompression group [17]. Considering this, judicious use of anterior transposition is warranted. The following is a review of the indications favoring anterior transposition.

Indications

Traditional indications for anterior transposition include preoperative or perioperative nerve instability, recurrent or persistent neuropathy after prior in situ decompression, neuropathy associated with existing hardware or lesions within the cubital tunnel, and posttraumatic neuropathy.

Pre- or intraoperative ulnar nerve instability or subluxation is considered an indication from transposition (Fig. 8.1). Ulnar nerve instability is highly variable between patients, with ulnar nerve hypermobility noted in about 37% of patients preoperatively [18]. A thorough examination of patients with cubital tunnel syndrome should include palpation of the nerve during elbow hyperflexion. Ulnar nerves may be further destabilized and become hypermobile after simple decompression. Therefore, it is imperative to assess the ulnar nerve after decompression intraoperatively by passively flexing the elbow and directly visualizing the nerve for subluxation and/or dislocation of the ulnar nerve out of the intercondylar groove. Matzon et al. identified 12% of cases of simple decompression that required transposition for instability after in situ decompression [19]. Of particular risk for iatrogenic intraoperative nerve instability are young males, perhaps due to more muscular triceps pushing the nerve out of the groove [19].

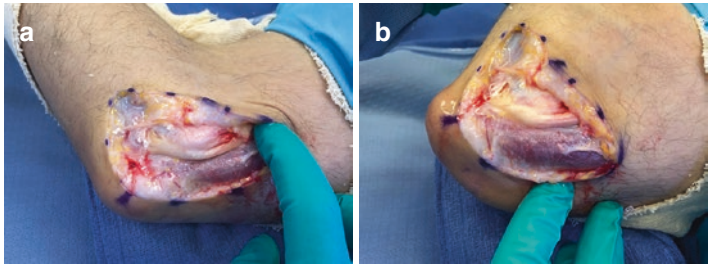

Figure 8.1 Intraoperative nerve instability evident with passive (**a**) extension and (**b**) flexion of the elbow prior to decompression. Note how the ulnar nerve rolls onto the epicondyle with flexion

When in situ simple decompression is unsuccessful, anterior transposition may be indicated. In recent studies of risk factors for revision surgery after in situ decompression, age and a history of trauma correlated with a higher risk of revision ulnar nerve decompression surgery [20, 21]. Moreover, factors traditionally considered to predispose to ulnar nerve simple decompression ultimately requiring revision surgery for ongoing or recurrent symptoms including high body mass index, tobacco use, diabetes, and higher McGowan grade have not been borne out in the literature.

Prominent hardware or lesions within the cubital tunnel, including arthritic changes and masses, may irritate the ulnar nerve [20, 22–24]. In the setting of acute distal humerus fractures with preoperative neuropathic symptoms, the nerve may be transposed. In a systematic review of distal humerus fractures, Nauth et al. found fair evidence to support anterior transposition in patients with preoperative ulnar nerve symptoms [23]. These findings were primarily based on a prospective, randomized study of patients with preoperative nerve symptoms. The results of that study showed improved outcomes in the transposition group vs in situ decompression group [24]. However, there was insufficient evidence for transposition in patients without preoperative nerve symptoms.

Posttraumatic deformities predispose the ulnar nerve to traction. Tardy ulnar nerve palsy can arise from cubitus valgus deformities or chronic degenerative arthritis changes after elbow trauma. In these situations, simple decompression may not resolve neuropathy generated from bony deformities.

Anterior Subcutaneous Transposition

Curtis described the first use of ulnar nerve anterior subcutaneous transposition [2]. The goal of the procedure is to transpose the nerve anterior to the flexion axis of the elbow and thereby decreasing tension during elbow flexion. By reducing tension, nerve traction injury is avoided and the epineural

vasculature to the nerve maximized. An advantage of subcutaneous transposition over submuscular or intramuscular transposition is that there is no flexor muscle dissection required. A common criticism of this procedure is the vulnerability of the nerve in its new subcutaneous location and the potential risk of the ulnar nerve relocating back into its retrocondylar position if not restrained. For this reason, some surgeons choose to fashion a loose fascial sling to restrain the nerve and/or close the cubital tunnel (our preferred technique). Regardless, maintaining ulnar nerve vascularity, meticulous surgical field hemostasis, avoiding iatrogenic injury to the nerve or medial antebrachial nerve, and complete decompression are paramount to successful anterior transposition.

Technique, Tips, and Pitfalls

The patient is positioned supine on the operating table under general anesthesia. The operative arm is extended across a hand table. With a sterile tourniquet applied to the upper arm, the limb is exsanguinated and the tourniquet inflated to 250 mmHg. The elbow is flexed and shoulder externally rotated and abducted to expose the medial epicondyle. A stack of sterile towels is placed under the elbow to improve access to the posteromedial elbow. An 8–12 cm incision is placed across the posteromedial elbow centered just posterior to the medial epicondyle, the subcutaneous fat is exposed, and dissection is carried down to the level of the medial epicondyle. The branches of the medial antebrachial cutaneous nerves are typically located about 3 cm distal to the medial epicondyle and should be identified and carefully protected [25]. These nerves typically travel in an anterior-proximal to posterior-distal direction.

The ulnar nerve is found directly posterior to the medial epicondyle (Fig. 8.2). Using sharp dissection, the cubital tunnel is approached, and Osborne's ligament is identified and completely released parallel to the ulnar nerve. In line with

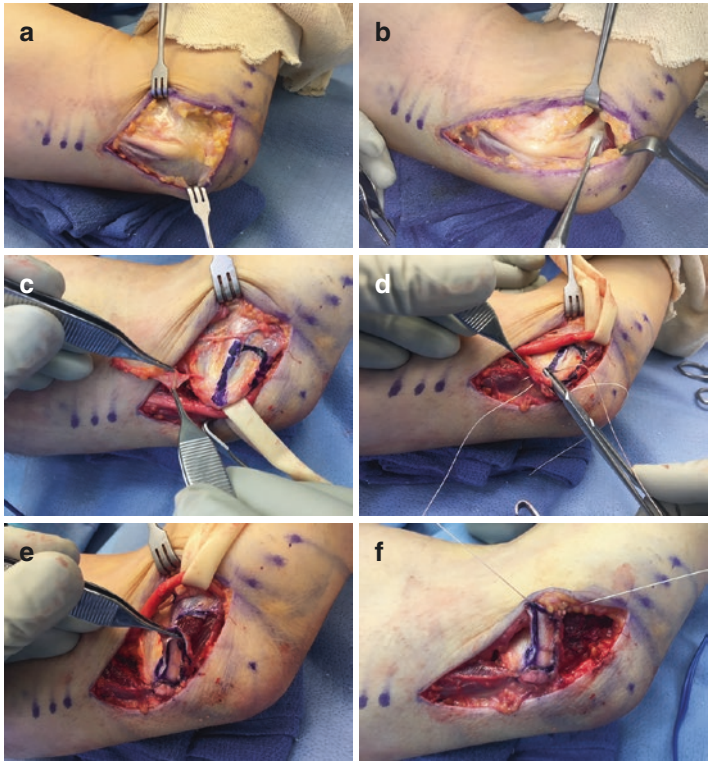

FIGURE 8.2 Case example of a subcutaneous anterior transposition: (**a**) Superficial dissection down to the level of the cubital tunnel with release of the ulnar proximally prior to its entry into the retrocondylar groove. (**b**) Complete release of the ulnar nerve across the retrocondylar groove and through the two heads of FCU. Note, the deep investing fascia (identified with a freer elevator) also warranting release. (**c**) Release and excision of the distal 2–3 cm of the intermuscular septum off of the supracondylar ridge. (**d**) Closure of the cubital tunnel in order to avoid redislocation within the retrocondylar groove. (**e**) Elevation of a fascial sling off of the flexor pronator mass. (**f**) Repair of the fascial flap to the anterior subcutaneous flap in order to keep the ulnar nerve anteriorly but without tension

the release of the cubital tunnel, the overlying fascia is carefully elevated and sharply released proximally at least 8 cm across the arcade of Struthers and distally at least 6 cm between the two heads of the flexor carpi ulnaris (FCU). The perineural vasculature can be seen around the ulnar nerve and should be carefully maintained alongside the nerve whenever possible. Similarly, an attempt is made to preserve the small articular branches and FCU motor branches of the ulnar nerve, but often the proximal-most branches may need to be cauterized to ensure a tension-free transposition. Once released proximally and distally, the ulnar nerve is mobilized circumferentially and retracted with a broad Penrose drain in order to facilitate safe manipulation and mobilization of the nerve, rather than with surgical instruments. Similarly, the ends of the drain are not clamped together to avoid accidental traction on the nerve. Attention is then turned to the medial intermuscular septum as it inserts into the supracondylar notch of the medial epicondyle. This septum can both compress the nerve and form a sharp corner when the nerve is transposed. Here, large vessels lie hidden under the fascia, and a bipolar electrocautery is used to coagulate them as the distal 1–2 cm of the septum is excised. Note, the collateral ulnar inferior artery is located in this region proximal to the medial epicondyle and should be preserved. Distally too there may be new sites of compression, including the superficial fascia of the flexor pronator mass and the deep investing fascia between the two heads of FCU, which may require release to ensure a compression-free position of the ulnar nerve one transposed anteriorly.

Once fully mobilized, the ulnar nerve can be moved anterior to the medial epicondyle to rest on the flexor mass fascia. Again, the nerve must be confirmed to be completely free of both restrictive bands and compression proximally and distally. To secure the position of the ulnar nerve anteriorly and avoid nerve instability or the relocation of the nerve back into the cubital tunnel, a number of surgical options exist

including (1) creating a fascial sling, (2) repairing the superficial subcutaneous tissue to the deep posterior fascia of the flexor pronator origin, and/or (3) closing the cubital tunnel. However, whichever technique is chosen, care must be taken to avoid any new iatrogenic compression of the ulnar nerve.

Prior to initiating closure, the elbow is taken through a range of motion to confirm that the ulnar nerve is not subject to any compression. Once transposed, the greatest compression of the nerve can be expected in elbow extension and that position in particular should be scrutinized for any sites of new ulnar nerve compression following transposition. Once satisfied, the tourniquet is released and hemostasis is attained with bipolar cautery. The wound is irrigated and closed. The authors prefer absorbable sutures in an interrupted subdermal fashion, followed by a running subcutaneous monofilament absorbable suture, and lastly skin glue.

The wound is dressed in a bulky sterile dressing. Cast padding followed by loose elastic bandage is applied. No splint is applied as the bulky dressing provides adequate restriction to extremes of motion. Normal activities of daily living are allowed. Formalized physical therapy is rarely required.

Outcomes

Most recent studies support the notion that in situ decompression is comparable to subcutaneous transposition in clinical improvement [9, 12, 17, 26, 27]. Black et al. reviewed 51 anterior transpositions at a minimum of 2-year follow-up and found 92% patient satisfaction with 91% good or excellent results using the Bishop rating [28]. Nabhan et al. performed a randomized, prospective study of 66 patients comparing outcomes of subcutaneous transposition vs in situ decompression [27]. There were no differences in muscle strength, pain, and nerve conduction velocity at 9-month follow-up between the two groups. In another prospective randomized study comparing in situ decompression vs subcutaneous transposition, Bartels et al. again found no significant differ-

ence in outcomes at 1 year. However, more complications were seen in the transposition group (31.1% vs 9.6%) [17]. Sensation loss and infection were the most common complications. Sousa et al. performed a retrospective review of 97 in situ decompression and subcutaneous transposition procedures and found no significant difference in Wilson and Knout scores between groups [29].

Intramuscular and Submuscular Transposition

Klauser described the first anterior intramuscular transposition in 1917 [3] (Fig. 8.3). Later, Learmonth described the submuscular transposition technique in 1942 [4]. Historically, because of muscle dissection and transplant of the ulnar nerve within the muscle fibers of the flexor mass, patients were immobilized to avoid muscle contractions and allow healing time for the flexor mass fascia. As a consequence of immobilization, elbow stiffness and/or increased ulnar nerve scarring was found to be a potential problem. To address this issue, Dellon in 1988 described a modification of the Learmonth procedure in which the myofascia of the flexor muscles is Z-lengthened [30]. The nerve is then transferred underneath the flexor muscle bellies, and the lengthened myofascia is closed loosely over the nerve. By lengthening

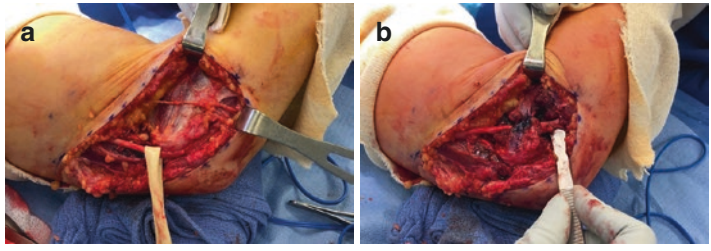

Figure 8.3 Note a case of anterior transposition followed by intramuscular transposition. Note the subfascial position without Z-plasty lengthening

the flexor muscles, immobilization was not needed. However, the criticism of all of these procedures is the extensive muscle dissection, increased bleeding risk, increased operative time, and potentially increased postoperative pain, strength, and mobilization time compared to subcutaneous transposition alone. Moreover, the increased muscular dissection also lends to concern for greater scar tissue formation around the nerve potentially facilitating recurrent nerve compression (Fig. 8.4).

Technique, Tips, and Pitfalls

The initial positioning, approach, and dissection for intramuscular and submuscular transposition are similar to subcutaneous transposition as described above. Turning to the flexor muscle fascia, a Z-cut is made directly anterior to the medial epicondyle [30]. This is best done by outlining the planned incision with a marking pen directly on the fascia. The proximal flap is kept attached to the medial epicondyle, and the distal flap is elevated from posterior to anterior. Particular care is taken to avoid injury to the median nerve located more anteriorly. There is a vertical component to the fascia deep to the muscle belly. These fibers may be left intact if they do not abrade the transposed nerve. The authors prefer to dissect down to the periosteum using a bipolar cautery so that the nerve will rest completely under muscle and no medial epicondylectomy is performed. The medial antebrachial and brachial septae located distal and proximal to the medial epicondyle, respectively, are excised. The nerve is then transferred under the raised fascial flaps. At this point, the nerve is reassessed for any remaining constrictions or sharp turns. The two flaps are sutured together, effectively lengthening the fascia. Care is taken to avoid over-constriction of the transposed nerve. If a finger can be placed under the sling, then adequate space should be available for the nerve. The tourniquet is released and hemostasis is attained. The wound is irrigated and closed with an absorbable, monofilament suture in a manner previously described.

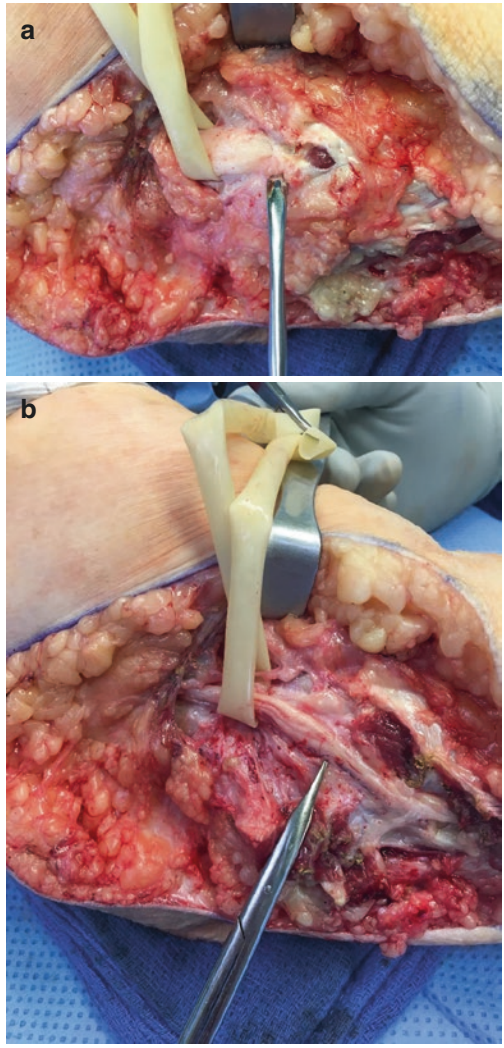

FIGURE 8.4 Case of a recurrent ulnar neuropathy of the elbow following prior anterior transposition in an intramuscular fashion. (**a**) Note the compression on the nerve at the proximal aspect of the fascia of the flexor pronator mass with the instrument applied to the medial epicondyle. (**b**) Note the status of the ulnar nerve followed by its decompression in its anteriorly transposed position, with the instrument pointing at the site of maximal compression

The wound is dressed in a bulky sterile dressing with abdominal pads over the wound and olecranon prominence for comfort. Cast padding followed by loose elastic bandage is applied. No splint is applied as the bulky dressing provides adequate restriction to extremes of motion. Normal activities of daily living are allowed. The dressing is maintained until follow-up within 2 weeks. Formalized physical therapy is rarely required.

Outcomes

Dellon and Coert reviewed their results of submuscular Z-lengthening in 2003 and found 88% good or excellent results [31]. Zimmerman et al. performed a 6-year follow-up of submuscular transposition in 82 elbows [32]. They found pain, sensation, motor strength, and Dellon scores improved. They reported 89% good or excellent results on the Bishop scale and 94% patient satisfaction. Keiner et al. compared submuscular vs in situ transposition and found no complications or revision surgeries in 33 patients followed up for at least 3 years. Nine of the 16 patients in the transposition group were satisfied, and 11 of 17 patients in in situ decompression group were satisfied at final follow-up [33]. In a prospective, randomized study comparing Z-lengthening submuscular transposition vs in situ transposition, Gervasio et al. found no significant difference between in electrophysiology and Bishop scores [34].

Summary

The most appropriate surgical approach to cubital tunnel syndrome is unclear in the literature but points toward in situ cubital tunnel release in cases of primary uncomplicated cubital tunnel syndrome without preoperative nerve instability. Traditional indications for anterior transposition include preoperative or perioperative nerve instability, recurrent or persistent neuropathy after decompression, neuropathy with

existing hardware or lesions within the cubital tunnel, and posttraumatic neuropathy. More important than the choice of procedure is meticulous surgical technique to ensure complete nerve decompression, avoiding iatrogenic nerve injury, avoiding devascularization of the ulnar nerve, and preventing wound complications.

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Chapter 9
Minimal Medial Epicondylectomy

Loukia K. Papatheodorou and Dean G. Sotereanos

Introduction

Medial epicondylectomy combined with ulnar nerve decompression is one of the various accepted surgical treatment options in the management of cubital tunnel syndrome. A primary advantage of medial epicondylectomy is the preservation of the ulnar nerve's intraneural and extraneural blood supply as compared to other ulnar nerve transposition techniques [1]. However, medial elbow instability, elbow flexion contracture, and weakness related to detachment of the flexor pronator origin have been reported after conventional medial epicondylectomy [2–4].

To avoid these potential complications, further modifications have been described of partial medial epicondylectomy with excision of approximately 40% of total width of medial epicondyle in the coronal plane [5–8]. Although good results have been reported with this modified technique, valgus instability of the elbow may occur [7].

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O'Driscoll et al. in a cadaveric study on the anatomy of the medial ulnar collateral ligament observed that only 19% of the width of the medial epicondyle in the coronal plane can be resected without potentially injuring the anterior band of the medial collateral ligament [9]. Subsequently, authors have described modifications of the minimal medial epicondylectomy with excision less than 20% of medial epicondyle in the coronal plane to preserve the medial collateral ligament minimizing the potential disadvantage of elbow instability [10–14].

Indications

The minimal medial epicondylectomy combined with in situ ulnar nerve decompression is indicated for surgical treatment of primary cubital tunnel syndrome. This technique is also useful in cases of concomitant ulnar nerve subluxation or recurrent cubital tunnel syndrome. In addition, it is particularly useful for cases of failed anterior, submuscular or subcutaneous, ulnar nerve transposition where the ulnar nerve is often tethered anteriorly by the medial epicondyle. The posterior aspect of the ulnar nerve is compressed against the anterior aspect of the medial epicondyle resulting in a Z-deformity of the ulnar nerve [15, 16]. A minimal medial epicondylectomy can eliminate the anterior tether and enable smooth gliding of the ulnar nerve throughout the elbow range of motion.

Surgical Technique

The procedure can be performed under general or regional anesthesia, tourniquet control, and loupe magnification. The patient is positioned supine with the affected arm placed on an arm table. After prepping and sterile draping, the extremity is exsanguinated with a sterile bandage, and the tourniquet is inflated at 250 mmHg.

A medial incision 5 cm proximal and 5 cm distally to the medial epicondyle is made (Fig. 9.1), and dissection is carried

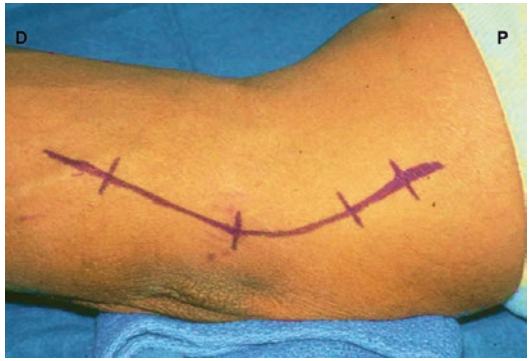

FIGURE 9.1 Skin incision for minimal medial epicondylectomy centered over medial epicondyle. *D* distal, *P* proximal

down through the skin and subcutaneous tissue. Care is taken to identify and protect the medial antebrachial cutaneous nerve. The medial intermuscular septum is released and the ulnar nerve is identified. The medial intermuscular septum is resected to avoid impingement on the nerve. The arcade of Struthers is released proximally. Then the ulnar nerve is released through Osborne's ligament and the cubital tunnel. The Osborne's ligament is released as posterior as possible to avoid subluxation of the ulnar nerve. The aponeurosis and deep fascia of the flexor carpi ulnaris are released distally. During the ulnar nerve decompression, care is taken to preserve the perineural blood supply.

After completion of the ulnar nerve decompression (Fig. 9.2), the flexor pronator origin is incised over the medial epicondyle, and a sharp subperiosteal dissection is performed preserving good flaps anteriorly and posteriorly to facilitate closure (Fig. 9.3). The medial epicondyle is exposed and the medial collateral ligament is visualized. A minimal bony resection (less than 20%) of the medial epicondyle is performed with the use of a small 12 mm osteotome while protecting the anterior band of the medial collateral ligament (Figs. 9.4 and 9.5). The osteotomy is performed from distal to proximal removing more bone

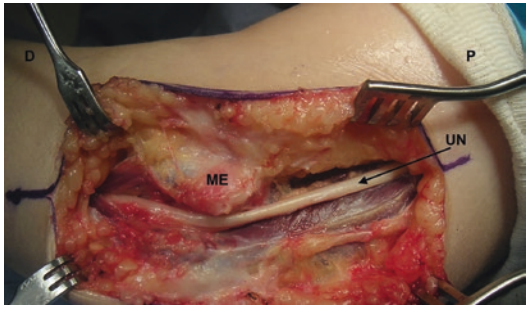

Figure 9.2 Intraoperative photograph demonstrates decompression of the ulna nerve. *UN* ulnar nerve, *ME* medial epicondyle, *D* distal, *P* proximal

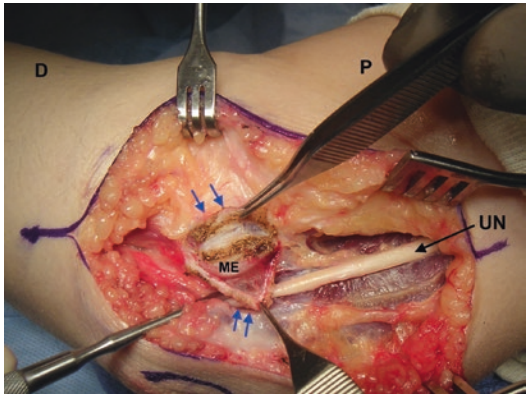

Figure 9.3 Intraoperative photograph demonstrates the exposure of the medial epicondyle with subperiosteal dissection preserving good flaps anteriorly and posteriorly (blue arrows). *UN* ulnar nerve, *ME* medial epicondyle, *D* distal, *P* proximal

posteriorly than anteriorly (Fig. 9.4). A rongeur is used to smooth all sharp edges. The elbow is flexed and extended to ensure that the nerve is gliding over a smooth surface with elbow motion. Bone wax is applied at the osteotomy site (Fig. 9.6), and subperiosteal flap closure is performed with sutures buried (Fig. 9.7). A dynamic flexion test of the elbow is then performed to ensure that the nerve is no lon-

FIGURE 9.4 Intraoperative photograph demonstrates the use of a small osteotome to perform a minimal medial epicondylectomy form distal to proximal. *UN* ulnar nerve, *ME* medial epicondyle, *D* distal, *P* proximal

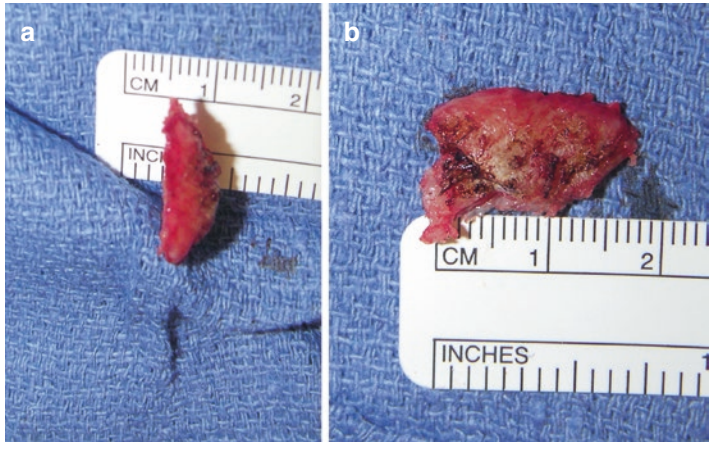

FIGURE 9.5 (**a**, **b**) Measurement of the size of the osteotomy fragment, less than 20% of the medial epicondyle was resected

ger compressed and is not subluxated anteriorly over the medial epicondyle with flexion. The tourniquet is released and proper hemostasis is obtained. The wound is irrigated and the incision is closed in layers with 3–0 Vicryl for subcutaneous tissues and 4–0 nylon for the skin. Sterile dressing is applied. At the conclusion of the procedure, the arm is placed in a bulky soft dressing.

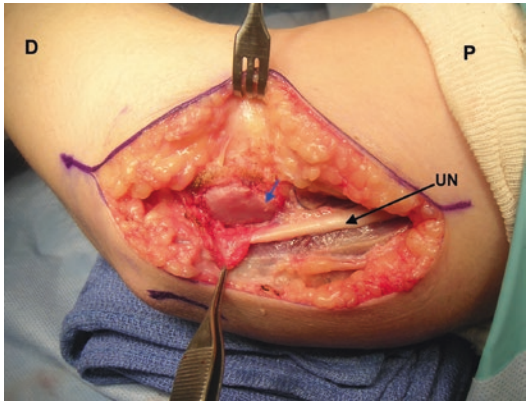

Figure 9.6 Intraoperative photograph demonstrates the application of bone wax at the osteotomy site (blue arrow). *UN* ulnar nerve, *D* distal, *P* proximal

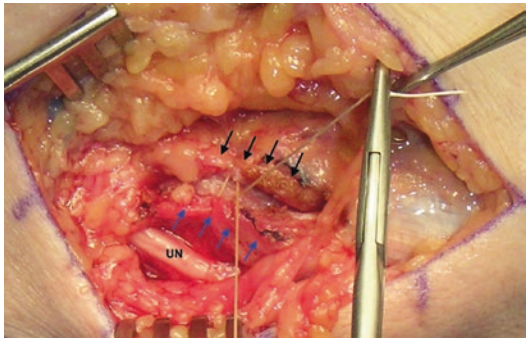

Figure 9.7 Intraoperative photograph demonstrates subperiosteal flap closure (black arrows, anterior flap, and blue arrows, posterior flap) with sutures buried. *UN* ulnar nerve

Complications

Great attention must be paid to the size of the osteotomy to avoid elbow instability after medial epicondylectomy [2–4]. The risk of damage to the anterior band of the medial collateral ligament can be minimized by removing less than 20% of the width of the medial epicondyle in the coronal plane.

Ulnar nerve subluxation over the remaining medial epicondyle may occur after medial epicondylectomy [3, 5, 15]. This potential complication can be avoided by using correct surgical technique creating smooth surface to allow the ulnar nerve to freely glide anteriorly and posteriorly with elbow motion. Weakness related to detachment of the flexor pronator origin and injury to the medial antebrachial cutaneous nerve are potential complications and can be avoided with careful dissection [2–4]. After minimal medial epicondylectomy, transient medial elbow pain at the site of osteotomy may occur up to 6–12 months [2–7, 10–13].

Outcomes

Good to excellent outcomes have been reported in 79–94% of patients with cubital tunnel syndrome based on the Wilson and Krout criteria [5–8, 10–13]. These clinical outcomes compare favorably to those of the other surgical techniques for cubital tunnel syndrome. However, comparison between surgical techniques is difficult because of heterogeneous populations and lack of prospective randomized studies.

According to the Wilson and Krout grading system [17], patients with minimal sensory and motor deficit and no tenderness at the incision site were graded as excellent; patients with a mild deficit but occasional ache or tenderness at the incision or osteotomy site were graded good; patients with an improved but persistent deficit were graded fair; and those with no improvement or a worsened condition were graded poor.

In a series by Gobel et al., 64 patients (66 elbows) with cubital tunnel syndrome were evaluated after minimal medial epicondylectomy [10]. The authors reported excellent results in 44%, good in 35%, fair in 10%, and poor in 6% of patients. No clinical evidence of ulnar nerve palsy, ulnar nerve subluxation, or elbow instability was noted postoperatively [10]. Similarly, Kim et al. reviewed 25 patients treated with minimal medial epicondylectomy for cubital tunnel syndrome [11]. They noted excellent outcomes in 64%, good in 20%, fair in 8%, and poor in 8% of patients [11]. There were no clinical signs of ulnar nerve subluxation or medial elbow

instability during the follow-up period [11]. Osei et al. achieved good to excellent results in 25 of 27 patients (93%) using a modified oblique minimal medial epicondylectomy [12]. No patient had symptomatic ulnar nerve subluxation or elbow instability with valgus stress testing [12].

Beak et al. compared the outcomes between minimal medial epicondylectomy and the anterior subcutaneous transposition for the surgical treatment of cubital tunnel syndrome in 56 patients [18]. In this retrospective study, 22 patients were treated with minimal medial epicondylectomy, while 34 patients were treated with anterior subcutaneous transposition [18]. The authors found no significant difference between the two surgical techniques [18].

Since the original clinical study [10], the senior author (D.G.S.) has noted consistently good to excellent results with the minimal medial epicondylectomy technique in more than 300 patients with primary or recurrent cubital tunnel syndome.

In summary, the minimal medial epicondylectomy is an effective alternative technique for the surgical treatment of primary or recurrent cubital tunnel syndrome. This technique can address the compressive and tensile forces on the ulnar nerve while minimizing injury to the blood supply to the ulnar nerve.

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Chapter 10
Anterior Interosseous Nerve to Ulnar Nerve Transfer

Aaron B. Mull

Introduction/Background

High ulnar nerve injuries can be devastating given the vital importance of the ulnar nerve in grip strength and fine dexterity. The long distance from the site of injury to the motor end plates usually precludes the ability of the nerve to regenerate in a timely fashion to recover intrinsic hand function. Historically, restoration has depended on distant tendon transfers, mostly static or dynamic anticlaw procedures in addition to transfers to restore thumb adduction and prevent small finger abduction [1–3]. More recently, in those individuals who present early after injury, distal nerve transfer is a viable option, specifically an end-to-end (ETE) anterior interosseous nerve (AIN) to ulnar motor branch transfer. This procedure has the potential to allow intrinsic muscle recovery without much donor morbidity [4–7]. However, this procedure may not be beneficial in those patients with mid-

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level injuries (i.e., at, and just below, the elbow) and in those patients with Sunderland grades 2–4 or 6 [8]. This would include those patients with severe ulnar nerve compression at the elbow. In these patients, there is a reasonable chance of proximal nerve regeneration, and/or these patients already have partial nerve function [9, 10]. It is important to not downgrade a patient's function or disrupt potential recovery.

Popularized within the last decade, a second option for preservation of the distal motor end plates involves transferring the distal end of the AIN to the side of the ulnar motor branch [11, 12]. This is often referred to as a reverse end-to-side or supercharge end-to-side (SETS). The concept involves using a more distal nerve transfer to help maintain, or "baby-sit," motor end plates until more proximal nerve recovery takes place. Described as far back as 1988 by Julia Terzis in her work with cross-facial nerve transfers, it has been used in ulnar nerve injuries since 2011 [13].

There have been several laboratory studies in animal models looking at the ability for a SETS transfer to contribute to nerve regeneration. In 2005, Isaacs et al. in Sprague-Dawley rats compared direct peroneal nerve repair (Group A), SETS transfer of the distal end of the tibial nerve to the side of an initially undamaged peroneal nerve (Group B), and SETS transfer as before but with proximal neurotomy of the peroneal nerve (Group C) [11]. There were nine rats in each group, and their outcome measure was contractile force of the extensor digitorum longus muscle. Just prior to electrical stimulation, the authors transected the peroneal nerve proximal to the SETS transfer in Group B. They found that in the initially uninjured peroneal nerve group (Group B), no measurable contractile force was seen. However, there was no statistically significant difference between direct repair (Group A) and the group with a SETS transfer with proximal neurotomy (Group C). This study helps confirm that a proximal nerve injury is required for collateral sprouting of a motor nerve. Additionally, and importantly, it shows in a rat model that a SETS transfer can regenerate function. This is consistent with subsequent work by others [14, 15].

Isaacs et al. followed up this study in 2008 comparing a SETS with a proximal nerve injury in various additional scenarios and equally concluded that a SETS transfer will produce contractile force in a rat model [16]. They showed that both the tibial and peroneal nerves contributed to the reinnervation of the distal muscle. Kale et al. additionally proved that the SETS transfer will contribute axons into a recipient nerve based on fluorescent labeling [12]. Farber et al. later quantified the motor axon contributions from a SETS transfer [17]. They showed a significant increase in motor nerve counts, myelinated fiber counts, and muscle force in those rats with a SETS transfer in an incomplete nerve injury rat model compared to the incomplete nerve injury rat model alone.

Technique

With the patient in a supine position and an upper arm tourniquet, a longitudinal incision is made over the ulnar aspect of the wrist 5–6 cm proximal to the wrist crease (Fig. 10.1). The ulnar nerve is identified, and the dorsal cutaneous

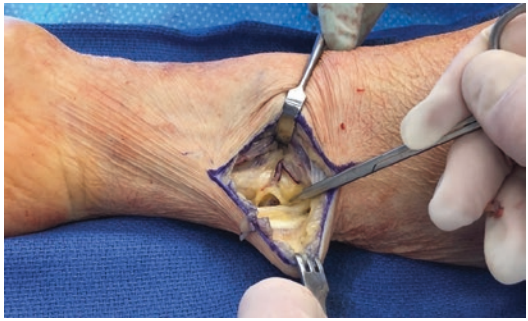

Figure 10.1 An incision is made over the ulnar aspect of the wrist 5–6 cm proximal to the wrist crease. The dorsal cutaneous branch will be the most ulnar and deep component of the ulnar nerve at this level. Note: In this photograph, the dorsal cutaneous branch appears to be radial as the view is of the undersurface of the ulnar nerve. For reference the hand is to the left

branch of the ulnar nerve is visualized. Next, the flexor digitorum profundi are retracted radially to expose the pronator quadratus. At the proximal end of the pronator quadratus, the AIN is identified and dissected proximally for several centimeters in order to prevent any kinking on nearby vessels or fascia. The dissection then continues distally through the fibers of the pronator quadratus for approximately half the length of the muscle (Fig. 10.2). Care is taken not to inadvertently damage an intramuscular nerve branch. At the midportion of the pronator quadratus, the AIN begins to significantly branch. These branches are then transected and brought proximally over to the ulnar nerve (Fig. 10.3).

The ulnar nerve topography at this part of the forearm runs sensory-motor-sensory from ulnar to radial. The dorsal cutaneous branch of the ulnar nerve, which represents the most ulnar component of the ulnar nerve at this level, is neurolyzed proximally to a point proximal to the area where the transferred AIN crosses. The motor branch to the ulnar nerve is identified in a natural cleavage plane from the main palmar ulnar sensory branch (Fig. 10.4). If this is not readily apparent, the incision should be extended distally into Guyon's

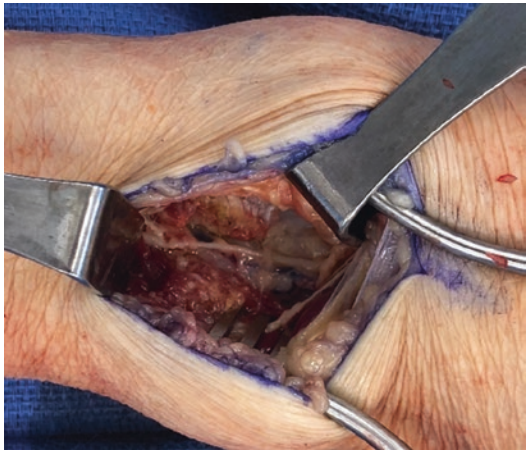

Figure 10.2 The anterior interosseous nerve is dissected underneath the pronator quadratus. For reference the hand is to the left

canal to confirm the deep motor fascicles. Once the deep motor branch is identified, it should be neurolyzed proximally to the area of nerve transfer. Once identified, instead of transecting the ulnar motor nerve, an epineurotomy is made

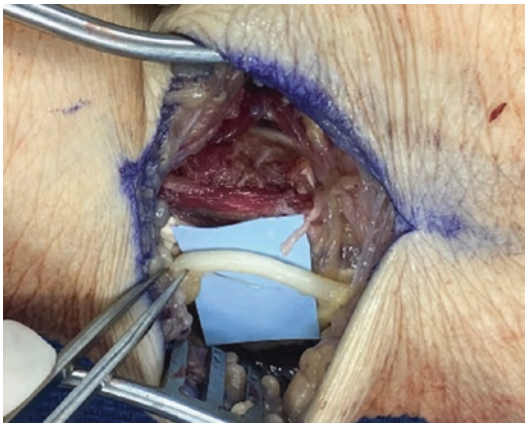

FIGURE 10.3 The anterior interosseous nerve is then moved over toward the ulnar nerve in a tension-free manner. For reference the hand is to the left

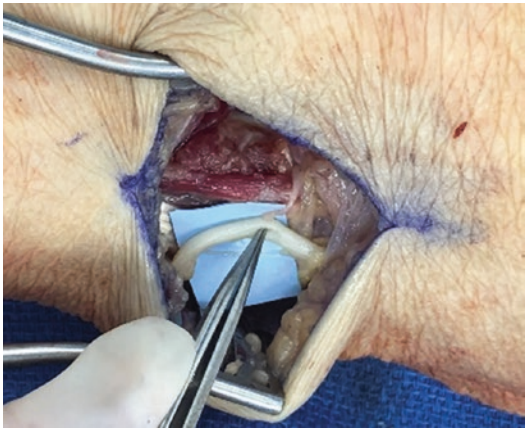

FIGURE 10.4 A cleavage is identified in the ulnar nerve to separate the ulnar motor fascicles from the main sensory fascicles. For reference the hand is to the left

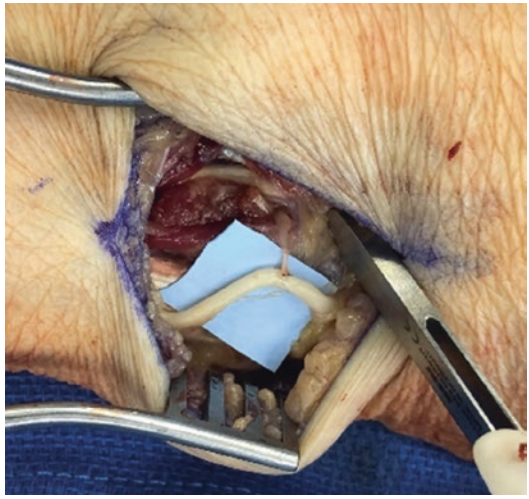

Figure 10.5 A neurorrhaphy is performed with 9–0 nylon under a microscope or loupe magnification. For reference the hand is to the left

to expose the underlying motor fascicles. The end of the AIN is then coapted to the side of the motor branch with 9–0 nylon (Fig. 10.5). This is usually performed 8–9 cm proximal to the wrist crease. There should be no tension on the coaptation. The tourniquet is lowered in order to obtain hemostasis deep in the wrist, and the incision is closed.

Outcomes

Since the original clinical case report in 2011, there have been scarce reports on clinical outcomes of SETS transfers [12]. Davidge et al., in a retrospective review of 55 patients who had undergone SETS AIN to ulnar motor nerve transfer, showed an improvement in pinch strength from 7.5 lb. ± 5.1 lb. to 9.7 lb. ± 4.5 lb. ($p = 0.012$) [18]. Likewise, the grip strength (35.7 lb. ± 23.8 lb. to 46.3 lb. ± 20.2 lb. ($p < 0.001$)), DASH score (48.2 ± 20.4 to 38.3 ± 19.1 ($p = 0.002$)), and British Medical Research Council grade ($p < 0.0001$) all improved. However

there were only outcomes for 71% of the patients, and only 15 patients (27%) were in the cubital tunnel cohort as the cause of nerve dysfunction. Outcomes specifically looking at those patients with severe cubital tunnel were not analyzed. Most patients (60%) had an anterior ulnar nerve transposition (either primary or revision) at the time of the SETS transfer. In order to tease out the difference between recovery from ulnar nerve transposition and the SETS transfer, the authors recorded the time interval of maximum recovery. Fifty percent of the patients had improvement in intrinsic function at the 3–12-month range and the greatest improvement in pinch and grip strength at the 1–3-month and 3–6-month interval. This may suggest recovery from axonal regeneration from the SETS transfer. It would be assumed that regeneration from the elbow would take longer than 12 months. Importantly, this study shows that those patients who had injury to the AIN or also have a diagnosis of peripheral neuropathy have worse outcomes with this nerve transfer.

In a smaller study, the same success with a SETS transfer was not found when specifically looking at compression neuropathies. Baltzer et al. matched 13 patients who had conventional ulnar nerve treatment (primary repair, nerve grafting, or decompression) in addition to a SETS AIN to ulnar motor nerve transfer to patients who only underwent conventional treatment [19]. Patients were matched based on age, type of injury, and level of injury. Patients were included if they had return of intrinsic nerve function or had 1 year of follow-up. Six of the 13 patients were treated for nerve-in-continuity/compression lesions. When all patients were compared, those who had a SETS transfer with conventional ulnar nerve surgery had a much higher rate of intrinsic muscle function (84% vs 38%, $p < 0.05$). However, when subgroup analysis was performed of nerve-in-continuity lesions, this difference went away (67% vs 67%, $p > 0.05$). It is important to note that the average follow-up in the conventional group was much longer than those in the SETS transfer group (39 months vs 13.5 months, $p = 0.02$) and the authors did not document the duration of symptoms. Additionally, the choice to perform a

SETS was at the discretion of the surgeon. Although all patients showed evidence of axonotmesis, it is unknown whether these patients had fibrillations or compound muscle action potentials suggestive of viable motor end plates.

There have not been any prospective or randomized studies comparing decompression/transposition alone versus decompression/transposition with a SETS AIN to ulnar motor transfer in the English literature. None of the current studies showing good outcomes for SETS transfer, either for compression or high ulnar nerve injury, can demonstrate how much recovery is attributed to the SETS transfer or proximal regeneration. This may be accomplished with postoperative electrodiagnostic studies.

When to Add a SETS Transfer for Severe Cubital Tunnel Syndrome?

In patients with high ulnar nerve injuries, an ETE AIN to ulnar motor transfer results in improved motor strength and grip [6, 7, 20–22]. Additionally, in those patients with Sunderland grade 5 (neurotmesis), injuries around the elbow may benefit from a SETS AIN to ulnar motor transfer [18, 19]. What is not specifically known is whether patients would benefit from a SETS AIN to ulnar motor for severe compression at the elbow. There are at best mixed results in the literature. The ideal candidates are those patients who have severe ulnar nerve compression at the elbow, have fibrillations and low amplitude compound muscle action potentials on electrodiagnostic studies, have an uninjured AIN, and do not have underlying peripheral neuropathy [23].

Conclusions

Supercharge end-to-side AIN to ulnar motor nerve transfer may be a good adjunctive procedure with conventional surgeries for patients with severe ulnar nerve compression at the

elbow; however, more studies are needed in order to pinpoint those patients who would benefit the most from the additional operation.

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Chapter 11
Management of the "Failed" Cubital Tunnel Release

Rikesh A. Gandhi, Matthew Winterton, and Stephen Y. Liu

Introduction

Cubital tunnel surgery is considered a failure if patients have no improvement in symptoms or if symptoms recur. Reoperation rates after primary cubital tunnel release have been variable. It is estimated that up to 25% of patients treated for cubital tunnel syndrome will have persistent or recurrent symptoms [1]. Goldfarb and colleagues [2] reported a 7% failure rate after primary in situ decompression at 4-year follow-up, and failure rates of 8–10% have been reported for anterior submuscular transposition and partial medial epicondylectomy [3, 4]. The secondary surgery rate after a primary cubital tunnel surgery has been reported to be 5.7% overall, 2.5% for in situ release, and 11.1% for transposition [5]. Patients who are under 50 years old, have a history of elbow trauma, and underwent primary transposition are at

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increased risk of requiring revision surgery [5, 6]. Those with severe disease may experience less improvement secondary to chronic nerve changes [7, 8].

Causes of Failure

Failure of the index cubital tunnel surgery can be due to any combination of diagnostic, technical, or biologic factors. It may be the result of inadequate decompression, ulnar nerve subluxation, iatrogenic creation of a new site of compression, devascularization, perineural fibrosis or cicatrix formation, or intraoperative nerve injury [9]. Categorization of patients who have failed a primary cubital tunnel syndrome procedure into those with persistent, recurrent, or new and distinct symptoms may guide the appropriate treatment.

Patients with persistent symptoms experience either no relief or incomplete relief after a primary procedure may have residual sites of compression after index procedure, a new site of compression after transposition (Z-deformity), irreversible nerve pathology, or inaccurate diagnosis. The medial intermuscular septum and flexor-pronator aponeurosis were found to be the most common sites of residual compression at the time of revision surgery [10–12]. Less common sites of residual compression include anomalous muscles on the medial side of the elbow such as the anconeus epitrochlearis, fascial bands within the flexor carpi ulnaris, the flexor digitorum superficialis, and the medial head of the triceps [13]. Osteophytes, heterotopic ossification, and masses such as ganglion cysts in the cubital tunnel may also cause residual ulnar nerve compression. Ruling out conditions that mimic cubital tunnel syndrome and identifying associated pathology are particularly important. Conditions that that can mimic cubital tunnel syndrome include cervical radiculopathy, thoracic outlet syndrome, ulnar tunnel syndrome, brachial neuritis, systemic neuropathy (diabetes mellitus, chronic alcoholism with vitamin B12 deficiency), multiple sclerosis, and peripheral nerve tumors.

Recurrent symptoms are those that return after a period of symptomatic improvement and usually result from cicatrix formation and perineural fibrosis after surgery [14]. Perineural fibrosis is found to compress the ulnar nerve at an average of 2.2 sites during revision decompression [15]. Care must be taken to minimize soft tissue injury during the index procedure to minimize scar formation. The stability of the ulnar nerve, if not transposed during the index procedure, may also be the etiology of persistent or recurrent symptoms, although a meta-analysis of four randomized controlled trials showed no difference between in situ and anterior transpositions regardless of nerve stability [16, 17]. Stability is assessed intraoperatively after in situ decompression by evaluating the nerve for anterior translation during passive flexion of the elbow. The nerve is classified as stable (posterior to medial epicondyle), perched (rests on the medial epicondyle), or subluxated (remains anterior to medial epicondyle) [18]. Longitudinal tension may be present in cases of cubital tunnel syndrome that are not adequately addressed by in situ decompression alone [19].

New symptoms can result from direct damage to the ulnar nerve and the medial antebrachial cutaneous nerve (MABCN) or creation of an iatrogenic site of compression. We consider new symptoms complications rather than a "failed" release, although they are not mutually exclusive. Iatrogenic creation of a secondary compression point after transposition most commonly occurs at the medial intermuscular septum, the point of transposition, or the flexor-pronator aponeurosis. The MABCN is the terminal sensory branch of the medial cord of the brachial plexus, where it bifurcates into an anterior and posterior branch, and may be injured during exposure. The anatomy of the anterior branch of the MABCN is variable, and although it can typically be found 2–3 cm anterior the medial epicondyle, it can also be located proximal or distal to the medial epicondyle making it prone to injury [20]. MacKinnon found a neuroma of the MABCN in 73% of revision cases [13].

Evaluation of the Failed Cubital Tunnel Release

The evaluation begins by fully understanding the patient's symptoms prior to their index procedure. Postoperative complications, such as a hematoma or infection, should be noted. Changes in symptom frequency, severity, and character will offer critical information about whether the patient is experiencing persistent or recurrent symptoms. Other etiologies that present with similar symptoms, such as cervical spine pathology or thoracic outlet syndrome, should be ruled out.

The physical examination begins at the cervical spine to assess for evidence of radicular symptoms which can mimic or contribute to cubital tunnel syndrome causing a double crush injury. A positive Spurling's maneuver has a high specificity for cervical radiculopathy, and Lhermitte's sign may indicate cervical canal stenosis. The supraclavicular region is assessed to rule out a more proximal compression as seen in thoracic outlet syndrome. The Roos stress test or "elevated arm" test may reproduce symptoms that involve the entire extremity and helps identify a proximal site of compression. The Adson test with a marked decrease or disappearance of a radial pulse suggests a vascular thoracic outlet syndrome.

Elbow deformity, specifically cubitus valgus, and elbow range of motion should be noted. A small incision used to perform an anterior transposition could lead to a new site of compression. Patients with local hypersensitivity anterior to the incision and numbness posterior to the incision should raise concern for the presence of a MABCN neuroma. Palpation of the nerve should be performed along with Tinel's percussion test. A positive Tinel's sign remains useful in localizing potential sites of nerve compression. A distally traveling Tinel's at serial clinical visits may indicate nerve recovery. Assessment for traction neuropathy should be performed by asking the patient to flex and extend the elbow repeatedly (traction Tinel's sign) to see if symptoms worsen. The elbow flexion test with the elbow flexed and wrist extended may also point to a traction-related etiology for

ulnar neuropathy at the elbow. An enlarged medial head of the triceps can compress the nerve against the medial epicondyle with the elbow flexed. Radiographic imaging may demonstrate arthritic changes, orthopedic hardware, or malalignment of the elbow joint.

Atrophy of the first webspace, intrinsics, and hypothenar eminence along with weakened grip and pinch strength suggest chronic and severe ulnar neuropathy. Sensation is preserved over the dorsal ulnar hand in ulnar tunnel syndrome, as the dorsal cutaneous branch of the ulnar nerve bifurcates proximal to the site of compression. When asking the patient to composite a fist, there may be asynchronous movement as the interphalangeal joints flex before the MP joint due to intrinsic malfunction. The Wartenberg sign and Froment test indicate digital and thumb adductor weakness, respectively. Long-standing cases may demonstrate clawing of the fourth and fifth digit.

Prior operative reports should be obtained to understand the location of the ulnar nerve, although these may not always be accurate. Even in cases of failed in situ decompression, the nerve may not be located posterior to the medial epicondyle. Nerve conduction studies and electromyography (EMG) studies should be obtained and compared to preoperative electrodiagnostic studies, if available. Unchanged electrodiagnostic studies should be interpreted with caution, since even with complete surgical release and symptom improvement, the studies often show no improvement. If electrodiagnostic studies show worsening signs of ulnar neuropathy, then revision decompression is indicated, particularly if a new site of compression or ulnar nerve injury is suspected. In the event that electrodiagnostic studies localize findings to the cervical spine or Guyon's canal, consideration may be given to magnetic resonance imaging (MRI) of the affected area.

In certain circumstances, musculoskeletal ultrasound may be utilized to understand the dynamic changes of the ulnar nerve as the elbow moves from extension to flexion. Ultrasound can be useful to confirm nerve location, the presence of an ulnohumeral joint ganglion, changes in nerve

diameter, perineural scarring, and the presence of MABCN neuromas. 3-Tesla magnetic resonance neurography (MRN) has been shown to correctly localize the site of abnormality and may be useful for preoperative planning [21].

Indications for Revision Cubital Tunnel Release

There is currently no consensus on the ideal treatment for failed cubital tunnel release [22]. The literature demonstrates that outcomes for revision surgery are not as good as primary surgery, which makes it difficult to advise a patient if and when he or she requires revision cubital tunnel surgery. If there is certainty a complete release was performed, observation for up to 6 months is rational, especially if preoperative electrodiagnostic studies demonstrated severe nerve compression. Beware of the patient who experiences slight improvement, but plateaus, as this may be a result of irreversible nerve damage. If the patient reports no change in symptoms, but a nerve study shows improvement, then there is support to continue to observe the symptoms with the understanding that an incomplete release could theoretically improve electrodiagnostic findings. In patients where there is worsening sensory or motor deficits or electrodiagnostic findings, it is reasonable to pursue a repeat surgical management of their symptoms.

Treatment Options for Revision Cubital Tunnel Release

It is important to have a high level of certainty when deciding whether to observe or proceed with surgical intervention. Continued observation with persistent compression may lead to worsening and potentially irreversible nerve damage. Conversely, revision surgery is technically challenging and can lead to iatrogenic nerve injury to an otherwise recovering

nerve. Conservative therapy with night splinting and nerve-gliding exercises has not been evaluated in the revision setting. Such measures cause no harm and permit the patient to play an active role in their recovery.

The surgical options for revision cubital tunnel are varied with no clearly proven superior technique, similar to the situation for primary cubital tunnel surgery. The goals of revision surgery include removing any offending sites of external compression on the nerve, placing the nerve in a stable location without tension, and minimizing perineural scar formation.

The surgeon undertaking revision cubital tunnel syndrome should be familiar with the five potential sites of compression typically encountered during the primary procedure: the arcade of Struthers, medial intermuscular septum, medial epicondyle, Osborne's ligament, and flexor-pronator aponeurosis. It is of paramount importance to perform a thorough external neurolysis of the nerve, which requires finding the nerve outside the zone of the original surgery and dissecting toward the scar. In the event circumferential perineural scarring is encountered that is difficult to separate from the nerve, it is advisable to leave a cuff of scar to minimize iatrogenic injury.

Once the nerve has been completely freed, options include medial epicondylectomy, subcutaneous transposition, intramuscular transposition, submuscular transposition, and/or nerve wrapping. In the event that an obvious site of compression is found, some may not proceed with a transposition, although in our opinion any revision decompression should be performed with a transposition to release tension on the nerve and place it in a stable bed of vascularized tissue.

Anterior submuscular transposition remains the most commonly performed procedure for revision surgery [2, 12, 15, 23]. The flexor-pronator mass is completely released, and the ulnar nerve is transposed underneath the muscle belly before the overlying fascia is repaired. This places the nerve in a well-vascularized bed of tissue but at the cost of an extensive soft tissue dissection, increased scar formation, and potential external compression as the nerve is now located

beneath a contractile structure [22]. In cases of concomitant medial epicondylitis, additional consideration may be given to this technique as it can treat both pathologies simultaneously. This technique may provide additional protection to external compression when compared to subcutaneous transposition in thin patients.

Other options for revision cubital tunnel syndrome include simple external neurolysis [24], subcutaneous transposition [11], and intramuscular transposition [25]. Simple external neurolysis should be reserved for cases where recurrent symptoms arise following a period of symptom relief as the cause is more likely from perineural fibrosis and cicatrix formation rather than residual compression or traction on the nerve. External neurolysis alone has also been shown to have good outcomes for recurrent symptoms after failed anterior submuscular transposition [24].

Subcutaneous transposition has also shown promising results, independent of index procedure [11]. The nerve is moved between the flexor-pronator mass and the subcutaneous fat and tethered in place by multiple methods. This approach limits dissection through the muscle belly, through a relatively avascular plan, which may result in less scarring. Osterman described a technique where the subcutaneous fat is divided through its natural superficial and deep layers to form a long adipose sling that minimizes acute changes in sagittal positioning of the nerve. Intramuscular transposition involves step-cut lengthening of the flexor-pronator fascia and creating a tunnel within the muscular substance that stabilizes and protects the nerve along its anterior course, although this has not been evaluated in the revision setting.

The lack of evidence clearly favoring one treatment for revision cubital tunnel syndrome over others suggests that revision outcomes may have more to do with the quality of decompression and preventing scar formation around the nerve than where the nerve is ultimately placed. Various methods of nerve wrapping to minimize cicatrix formation have been described. Silicone elastomer bands [26], allograft biomatrix scaffolds [27], amniotic membrane wrapping [28], autologous vein wrapping [29], and porcine extracellular

matrix [30] have been proposed. Allograft sources obviate donor site morbidity. When placing a nerve wrap, it should not compress the nerve and be of adequate length to cover 1–2 cm proximal and distal to the area of concern.

Outcomes of Revision Cubital Tunnel Release

The available data on outcomes after revision cubital tunnel release is limited to level IV evidence with one retrospective case control [31]. There is very little consensus on the ideal revision procedure for cubital tunnel syndrome, which is demonstrated in the heterogeneity of data regarding failed primary surgery and the choice of revision procedure performed. In addition to this, outcome measures assessed are widely variable, limiting the ability to make direct comparisons between procedures.

Revision cubital tunnel release is generally a successful procedure with a majority of patients experiencing improvement in their preoperative symptoms but to a lesser degree than primary ulnar nerve decompression at the elbow. This may be attributed to the technical demands of revision surgery, the time elapsed after the index procedure, and nerve devascularization as a result of repeated insult to the nerve millieu [32]. More severe disease, as defined by McGowan grade 3, or evidence of denervation on EMG, limits the potential for spontaneous nerve recovery [12, 15]. Other factors associated with poor recovery include number of previous operations, age >50 years, and previous submuscular transposition [11, 15].

Overall, approximately 75% of patients will have symptomatic improvement, but few will have complete recovery. Submuscular transposition of the ulnar nerve is the most commonly performed procedure in the revision setting [10, 12, 14, 15, 31]. Following this procedure, satisfaction can be expected in 73–79% of cases, although only 20% of patients will be symptom-free based upon physician assessment and even fewer when reported by patients themselves [5]. Patient satisfaction appears to be greater than scoring reports may indicate [10] and greatly exceeds the proportion of patients

who have objective improvement in McGowan grading after surgery [31]. Improvement after previous submuscular transposition may be limited due to the extensive soft tissue dissection, postoperative immobilization, and more extensive postoperative perineural scarring.

In situ decompression is generally reserved for first-line surgical management of cubital tunnel syndrome; however, 89% of patients experienced good or fair results with normal or slightly diminished two-point discrimination and modest improvements in grip strength when performed for failed submuscular transposition [24]. The use of anterior subcutaneous transposition was evaluated independent of the index procedure, and it was found that patients consistently had relief of their paresthesias with 75% achieving good to excellent results, comparable to submuscular transposition [11].

Nerve wraps are gaining popularity, but the long-term results are lacking. Autologous vein wrapping after a minimum of two prior failed cubital tunnel surgeries demonstrated improvement in pain, grip strength, and two-point discrimination in all patients, although still diminished from accepted normal values. Increased velocity of motor and sensory nerve conduction may be seen on repeat electrodiagnostic studies, which is of variable clinical significance [29]. The main complication was swelling at the harvest site. In a similar cohort, the use of human amniotic membrane demonstrated promising short-term results with improvements in pain, QuickDASH, grip, and key pinch strength [28]. Similar results without graft-related morbidity can be expected using porcine extracellular matrix, with patients generally experiencing 50% of their overall improvement within the first 4 months postoperatively [30].

Treatment Algorithms for the "Failed" Cubital Tunnel Release

It is important to counsel patients that although they are likely to gain improvement in their symptoms and func-

tion, residual deficits are commonly experienced. Expectation management prior to surgery may help patients cope with the often protracted course of nerve recovery over the following months, even years. Understanding the common sites of compression, the etiology of failure in persistent and recurrent cubital tunnel syndrome, and basic fundamentals of treatment will help guide the decision-making process.

We believe that the external neurolysis is the most important portion of the procedure. The nerve is identified both proximally and distally to the previous surgical site and traced from known to unknown. The branches of the MABCN are identified, and if a neuroma is present, it is resected back to healthy nerve. The end is cauterized with bipolar electrocautery, and the nerve end is buried within the muscle that is outside of the area of scar. In cases where perineural scarring is densely adherent to the ulnar nerve, a small cuff of tissue is left attached to minimize iatrogenic injury. This is maximally invasive surgery and is performed in all revision cases.

In the case of persistent symptoms, we generally do not utilize nerve wraps although they are available, as the cause is most likely incomplete decompression or creation of a new site of compression in a transition zone. In the event of failed in situ decompression or medial epicondylectomy, we transpose the nerve subcutaneously, unless the patient is very thin, as this can be performed with less soft tissue dissection. If the previous procedure was a transposition, we move the nerve to a plane of tissue that is different from the index procedure if possible, which may not be available in multiply revised cases. The algorithm is depicted in Fig. 11.1.

When revising a cubital tunnel release for recurrent symptoms, we follow the same algorithm in patients with recurrent symptoms with the addition of an allograft nerve wrap made of porcine extracellular matrix (Fig. 11.2), as described by Papatheodorou [30]. The main difference is that we deflate the tourniquet before application of the allograft wrap, so swelling of the perineural vasculature does not lead to secondary compression.

Treatment Algorithms

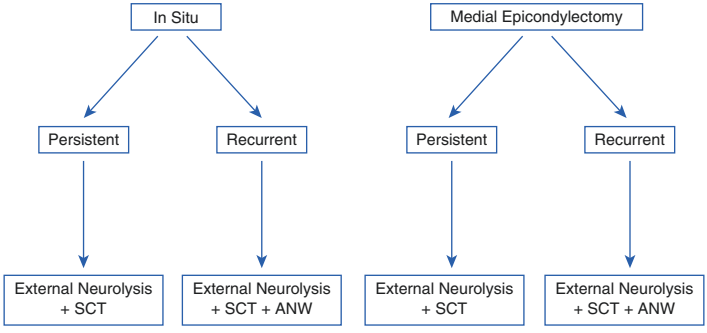

FIGURE 11.1 Revision cubital tunnel release algorithm for persistent and recurrent symptoms after in situ decompression or medial epicondylectomy. *SCT* subcutaneous transposition, *ANW* allograft nerve wrap

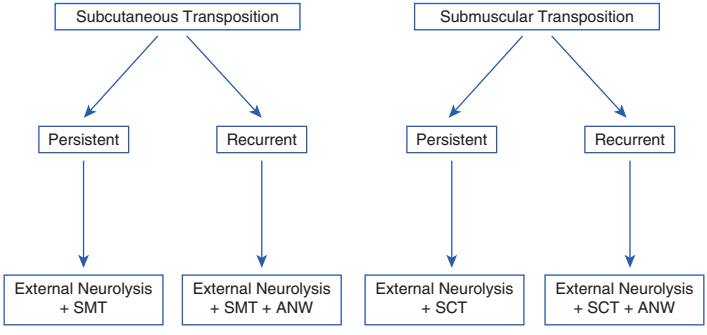

FIGURE 11.2 Revision cubital tunnel release algorithm for persistent and recurrent symptoms after subcutaneous or submuscular transposition. *SCT* subcutaneous transposition, *SMT* submuscular transposition, *ANW* allograft nerve wrap

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Part III
Postoperative Care

Chapter 12
Cubital Tunnel Rehabilitation

Marie Pace

Evaluation

Once it is determined that a cubital tunnel surgery is required to relieve symptoms of numbness and tingling in the ring and small fingers, there has been significant impact on the ulnar nerve on which conservative treatment was ineffective. Postoperative pain, edema, limited motion, impaired sensation, and weakness can be anticipated as a result of the surgical intervention [1, 2]. Rehabilitation begins with assessment of the client's postoperative condition.

Observations and documentation of the postures of the head and neck allow identification of potential proximal structures putting pressure on the ulnar nerve. Forward head posture with step off at the C7/T1 vertebra puts pressure on the C8 nerve root [3]. Tightness of the pectoralis minor or the scalene muscles can put added pressure on the brachial

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plexus. More distally, there may be wasting of the intrinsic muscles of the hand if the motor bundles of the ulnar nerve were compressed or stressed at one or more proximal locations. In some severe cases, there may be a claw hand deformity developing even at an early stage postoperatively.

Two-point discrimination of the ulnar nerve distribution is tested in the hand [4, 5]. Testing can be repeated with the Semmes-Weinstein monofilaments around the incision, in the forearm, and in the ulnar distribution of the hand. It should be noted that the sensation in the medial ulnar forearm should not be affected by compression of the ulnar nerve in the cubital tunnel as the medial antebrachial cutaneous nerve supplies this area.

Muscle strength is tested with manual muscle tests of each of the muscle in the forearm innervated by the ulnar nerve (Table 12.1) and with gross function tests of grip, tripod pinch, and lateral pinch. Strength testing of the elbow and forearm should be delayed until the 6-week postoperative visit in order not to irritate the healing structures.

The incision should be observed for size, healing, and scar formation. Most of the time, normal healing does not require intervention. However, conditions such as dehiscence, adhesions, and hypertrophic scar should be identified and treated promptly [6].

Measurements of range of motion (ROM) in the shoulder, elbow, forearm, wrist, and each joint in the hand should be completed. Any limitations in active ROM would be followed by measurements in passive ROM [5]. Because the ulnar nerve affects finger abduction and adduction, a special measurement of abduction can be taken by placing the hand on a piece of paper and tracing around the fingers in maximum abduction. Measurements can then be taken in centimeters from the middle finger to the index, middle to ring, and middle to small finger. This gross measurement can give an idea of progress in active finger abduction after cubital tunnel intervention.

Edema measurements can be taken at the level of the elbow crease. The tape measure is placed around the proximal forearm with the elbow flexed about 30° and the tape touching the elbow crease. Similar measurements can be

Table 12.1 Order of ulnar nerve muscular innervation, proximal to distal

Muscles innervated by the ulnar nerve distal to the elbow
Flexor carpi ulnaris
Abductor digiti minimi
Flexor digiti minimi
Opponens digiti minimi
Dorsal interossei
Palmar interossei
Lumbricals to the ring and small fingers
Adductor pollicis
Flexor pollicis brevis (deep head)

taken at the distal wrist crease just distal to the ulna head. Circumferential measurement of the digits at the proximal phalanx could be taken if edema is observed.

Measurement of fine motor coordination using the traditional 9-hole peg test may be useful. Results are objective and make following progress simple. Other more subjective measures of coordination and function such as the Disabilities of the Arm, Shoulder, and Hand (DASH) or Canadian Occupational Performance Measure (COPM) could be useful in goal setting and opening conversations about client-specific areas of difficulty in activities of daily living (ADL).

Orthoses

A nighttime orthosis may be recommended to prevent elbow flexion which would stress and stretch the ulnar nerve at the elbow [7, 8]. The orthosis should be volar to adequately limit elbow flexion (Fig. 12.1). The orthosis does not need to cross the wrist. The angle can be between 30° and 60° for comfort and sleep positioning. It is often difficult for clients to adjust to sleeping with the orthosis, but if they are instructed to position in supine or side-lying with supportive pillows, they will

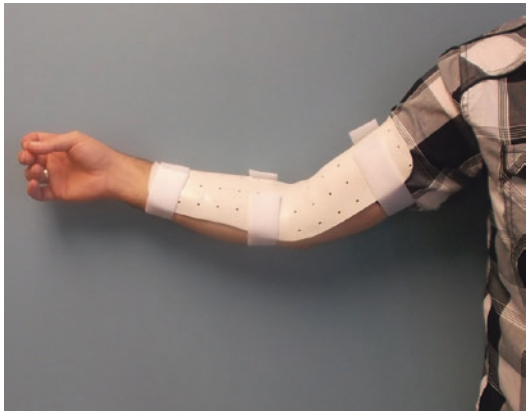

Figure 12.1 Volar elbow orthosis to limit flexion while sleeping

soon get used to the orthosis. It is generally not recommended to sleep prone when there is a problem with cubital tunnel syndrome because the position of the neck can exacerbate the ulnar nerve. Sleeping prone often makes it more difficult to find a place for the upper extremity (UE) with the orthosis in place. An elbow pad may be used during the day to prevent mechanical pressure to the medial elbow and to serve as a reminder of behaviorally limiting elbow flexed postures.

If claw hand deformity develops, figure-of-eight orthosis may be custom fabricated to substitute for failing lumbrical function (Fig. 12.2). The orthosis holds the metacarpophalangeal (MCP) joints in flexion to allow the muscles innervated by the radial nerve to extend the interphalangeal (IP) joints during daily activity or prescribed exercises [9]. The purpose is to increase the usefulness of the hand and prevent extension contractures of the MCP joints and flexion contractures of the IP joints.

Acute Postsurgical Treatment

Following the removal of the postsurgical dressing at day 10–14, a light compressive stockinette is applied to the

FIGURE 12.2 Figure-of-eight orthosis is designed to prevent IP flexion contractures by using during regular daily activity

length of the arm (axilla to distal wrist crease). If there is edema in the hand, then a compression glove on the hand should be applied. Light massage of the scar with lotion or oil may begin 3–5 days after the sutures are removed [6]. The patient is instructed to begin elbow, forearm, and wrist active ROM (AROM) exercises, for 10–20 repetitions 2 or 3 times per day [2, 10].

Overcoming stiffness in the hand can be accomplished by means of passive ROM (PROM) exercises with an emphasis in MP flexion and IP extension. Normal composite PROM exercises of each digit, intrinsic plus fisting, and digit abduction and adduction can be done as soon as there is recovery from surgery without risk to the surgical site. If the joints can remain supple postsurgery, then joint stiffness is avoided as a complication [4]. If there is muscular weakness, hand pain, and hand edema after the surgery, maintaining supple joints can be more difficult.

Desensitization around the scar and into the ulnar nerve distribution of the hand may be necessary if there is sensitivity to light touch [11, 12]. Use of brushing with a makeup brush or with a soft washcloth for 2 minutes twice a day should be tolerable and decrease sensitivity to light touch. Additionally, the client can dig lightly into a container of dry beans, rice, or corn for 5 minutes once a day. A daily regimen of small object manipulation or pickup with cotton balls, marbles, or pennies will assist in decreasing the sensitivity of the hand and improving the client's sense of the hand's capabilities. If there is diminished sensation in the hand revealed in initial testing, education in protection of the skin of the ring and small fingers is essential. Injuries from hot surfaces, food, or liquid, friction blisters or cuts are not uncommon when sensation is impaired, even temporarily.

Discussion with the client about accommodation for ADL difficulty should be made at an early visit postoperatively [13]. Use of an elbow pad for protection of the dorsal elbow in daily activity may be helpful if the nerve was decompressed and is still dorsal. Avoidance of heavy lifting tasks or repetitive or prolonged positions at end range of flexion (e.g., holding the phone to the ear, styling hair, brushing teeth) and in sleep postures and positions will facilitate healing with less pain.

Recovery of Function

At this point, the wound should be closed and healing. The soft tissues are no longer at risk from normal movement of the arm. Resolution of any ROM issues should be made at this time. Use of a figure-of-eight orthosis during daily activity can decrease any PIP flexion contractures. By holding the MCPs in flexion, the IP joints of the ring and small finger will extend with extensor digitorum communis (EDC) muscle power. In therapy, activities are provided to encourage extension so that the patient can see the difference in how the hand moves with and without the orthosis. Once the client understands how the hand can be stretched during

daily activity, orthosis wear is recommended, and exercise can be limited to a brief check of PROM one time a day (Table 12.2).

This treatment avoids the onset of MP and IP stiffness that sometimes follows ulnar nerve impairment.

Sensory reeducation activities are designed to address the deficiencies of light touch, coordination, and hypersensitivity [14]. Of primary importance is safety and protection of the skin, if there is any impairment in sensation. Since the skin is more susceptible to burns or damage with heat or cold, heat or cold extremes that could be experienced in daily activity (e.g., hot food or water or cold weather) are risky, and the client should be instructed to take precautions. Also, if using electrical or thermal modalities in therapy, the therapist should take extra precaution or avoid their use altogether. Daily stimulation with materials, brushing with a makeup brush, digging in a container of rice or dry beans, and brushing the skin with a towel are all common methods of stimulating the skin in a non-harmful way to decrease hypersensitivity.

Training in fine motor coordination activity can improve the hand's ability to do daily tasks with ease. For example, these could include manipulation of finger fitness spheres (Fig. 12.3), picking up various texture objects with alternating fingers, and wrapping bundles of pencils with rubber bands. Each of these activities utilizes finger abduction and adduction and precise positions of finger flexion and extension. Any rehab program of recovering nerve function should include these kinds of tasks.

Table 12.2 Use of the figure-of-eight orthosis decreases the amount of rote exercise that must be done to avoid contracture

Pre-learning of figure-of-eight orthosis use	**Post-learning orthosis use in activity**
PROM of MP and IP three times per day	PROM one time per day
10 repetitions with 5-second hold	5 repetitions with 5-second hold

FIGURE 12.3 Rotating the finger fitness spheres around one another in a clockwise direction and a counterclockwise direction increases coordination and strength in the muscles effected by ulnar nerve dysfunction

Strength and endurance training of the effected upper extremity should be appropriate for the level of activity to which the client expects to return [15]. For shoulder and elbow motion, the overhead pulley is a good way to encourage full motion with less than full gravity resistance. Supine wand exercises through shoulder flexion, abduction, and external rotation can be graded to increase resistance and challenge. Challenge is increased by adding weight to the wand or increasing the head angle of the bench. Hand weights can be used for biceps and triceps exercises. Forearm bars (like a hammer with circular weights on the end of a rod) are used for training of pronation and supination and radial and ulnar deviation. By increasing the weights or the length of the bar, challenge can be increased. Hand weights can be used for wrist flexion and extension training. Alternatively, using the technique of rolling a strap around a bar with a weight on the end of the strap is a good activity for strengthening the forearms (Fig. 12.4).

For strengthening of the extrinsic finger flexors, daily putty squeezing for 5 minutes a session can be effective. Acceptable alternatives include squeezing a rubber band gripper, a rolled

FIGURE 12.4 A weight is attached to a dowel from the end of the strap. To strengthen wrist flexion or extension, the strap is rolled around the dowel until the weight touches the bar

hand towel, or a guitar-player gripper. Resistance on any of these tasks should induce fatigue without pain. To strengthen the intrinsic muscles requires more creativity (Table 12.3). Using activities that use the finger abductor and adductor and lumbrical muscles with increasing resistance will make the intrinsic muscles stronger.

Nerve Glides

The use of a nerve glide after a cubital tunnel release procedure allows the nerve to glide freely from scar tissue and gradually increase the nerve's tolerance for tension [3, 16, 17]. In the normal motion of the arm, the nerve will stretch and glide. When there is stress or trauma to the nerve, it will swell and lose some ability to move normally. By putting tension on the nerve proximally and giving slack distally, the nerve can be gently glided proximally. In reverse, applying tension distally and slack proximally, the nerve will be glided distally.

To understand a nerve *glide*, it is helpful to first understand nerve *tension*. Tension in an ulnar nerve that has not been moved from its anatomical position: the shoulder is in

Table 12.3 Ideas for activities which build intrinsic muscle strength in the hand

Activity	Making the task more difficult
Opening a rubber band with one hand	Use larger rubber bands
Doing finger spelling	Larger motions with longer holds
Picking up cotton balls between fingers	Pick up smooth stones or small candies
Manipulating finger fitness spheres or golf balls	Use larger and heavier spheres
Picking up books with an intrinsic plus grip	Use larger and heavier books

90° abduction, the elbow fully flexed, the forearm pronated, and the wrist and fingers extended. The palmar side of the fingers is placed against the side of the face and the thumb and index fingers circled around the eye. This position is sometimes referred to as the junior birdman position. In the tension position, even a healthy nerve can be felt to burn slightly in the small finger. When a nerve has been stressed, only part of the tension position should be performed to glide it. For example, the shoulder is in 45° of abduction, the forearm in pronation, elbow in extension, and the wrist in extension. The elbow is then flexed and extended in a symptom-free range for five slow repetitions. In another example, the shoulder is in 90° of abduction, the forearm in supination, elbow in extension, and the wrist in neutral. The elbow is then flexed and extended in a symptom-free range for five slow repetitions. If these two glides are performed three times a day during the period from 3 weeks post-op to 8 weeks post-op, the nerve should glide well in its path.

If the nerve was transposed in the cubital tunnel surgery to the volar side of the forearm, the *tension* will be achieved with the same shoulder, forearm, and wrist positions but with the elbow in extension. A proximal nerve *glide* would then be performed with the shoulder in 90° abduction, wrist flexion, and supination, but the elbow would start in flexion and

move to extension. The distal glide would start with the shoulder at 45° of abduction, forearm pronation, and wrist extension. The elbow would start in flexion and move to extension to the point of tightness. Postsurgery the motion should only be taken to the point of stretch but not discomfort.

Role of Electrical Stimulation and Thermal Modalities

Electrical stimulation can be used to enhance efforts for muscle activation if there is loss of muscle function and to minimize pain after a nerve decompression or transposition [18]. Neuromuscular electrical stimulation (NMES) over motor points of weak muscles helps by rhythmically cueing flexion and relaxation. Muscles innervated by the ulnar nerve in the hand, such as abductor digiti minimi or flexor digiti minimi, are examples of muscles that can benefit from stimulation. When there has been nerve compression and some compromise to the nerve, the stimulation might not be able to spontaneously cause muscle contraction without the voluntary effort of the client. The stimulation can still be useful as a cue if sensation is intact for cooperation of the client with the stimulation.

Use of heat should be reserved for stiffness of joints, usually seen in the MCP or PIP of the ring and small finger. Care must be taken with thermal modalities to prevent damage to the skin if sensation is not normal. Conduction heat, such as a heating pad, is the riskiest as it heats the skin more quickly than deeper structures. A convection heat, such as hydrotherapy or fluidotherapy, allows motion during treatment and has a safe upper limit of heat, making burns unlikely.

Ultrasound treatment uses sound waves to treat a defined small area based on the size of the sound head [15, 19]. Usually in treatment of cubital tunnel syndrome, a useful location of ultrasound treatment would be over an adherent scar. To treat an adherent scar, the ultrasound setting should

maximize the mechanical effects without heat. Alternately, the ultrasound could be used over the ulnar nerve just distal to the medial epicondyle. To treat an inflamed nerve, the ultrasound setting will vary depending on preference and experience, but which should maximize physiological effects.

Summary

Treatment following a cubital tunnel surgery will focus on restoring normal function of the effected upper extremity. Joint mobility, sensory function, and strength should be addressed in assisting the client to return to normal levels of use of the upper extremity appropriate to the stage of recovery. Use of modality, activity with or without orthoses, and exercises can aid in rapid recovery.

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Chapter 13
Postoperative Outcomes of Cubital Tunnel Release

Matthew R. Walker and Anne Argenta

Critical review of surgical outcomes is vital to optimizing patient care. Multiple studies have ventured to address outcomes after cubital tunnel surgery. Due to variation in surgical technique, limited objective data, and inconsistency in measured outcome variables between studies, obtaining any generalized conclusions on anticipated postoperative outcomes remains somewhat challenging. This chapter reviews the available literature on outcomes after cubital tunnel release.

Established Grading Systems

In order to best evaluate the outcomes of cubital tunnel surgery, it is important to review the most commonly used grading systems for cubital tunnel severity and outcomes. Developed in 1950, the McGowan classification is one of the

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oldest and most frequently used systems (Table 13.1) [1]. Grade I represents purely subjective symptoms and mild hypesthesia. Grade II involves sensory loss and weakness of intrinsic hand muscles, with or without slight wasting. Grade III represents severe sensorimotor deficit. In the late 1980s, the McGowan classification was modified by Goldberg to further subdivide Grade II patients without and with intrinsic atrophy as IIa and IIb, respectively [2]. Grade IIb patients have strength of M3 to M5, while those below M3 would be classified as Grade III.

The Dellon classification is analogous to the McGowan classification, except that it specifically breaks each stage into sensory and motor components [3]. For mild disease (Dellon I), paresthesias are intermittent, paresis is subjective, and provocative tests may be equivocal. For moderate disease (Dellon II), paresthesias are still intermittent, vibratory sense may be decreased or normal, paresis is measurable in pinch or grip strength, and provocative tests are positive. With severe disease (Dellon III), paresthesias are persistent, vibration perception is decreased, 2-point discrimination is abnormal (static ≥6 mm or dynamic ≥4 mm), paresis is measurable with muscle atrophy, and provocative tests are positive with abnormal finger crossing.

The Bishop classification is designed to grade the surgical result of cubital tunnel release [4]. This system uses a 12-point scale to evaluate patient satisfaction, improvement, severity of residual symptoms, work status, leisure activity, objective strength, and objective sensibility. Currently, the modified Bishop score is more frequently used (Table 13.2). This modification removed leisure activity and patient satisfaction, creating a 9-point scale, with higher numbers indicative of better outcome.

Wilson and Krout developed another common postoperative outcome tool with grades of excellent, good, fair, and poor [5]. Excellent indicates complete return of sensation and function, while good indicates return of a functional hand without discomfort but with residual weakness or diminished sensation. Fair indicates improvement in only one area of comfort, strength, or sensation. Poor is no improve-

Table 13.1 Classification systems for cubital tunnel syndrome severity

Grade	McGowan	Goldberg (modified McGowan)	Dellon
I	Subjective symptoms; hypoesthesia	Subjective symptoms; hypoesthesia	Intermittent paresthesias; subjective paresis; equivocal provocative tests
II	Sensory loss, weakness of intrinsic hand muscles, +/− slight wasting	Sensory loss, weakness of intrinsic hand muscles, +/− slight wasting	Intermittent paresthesias; decreased or normal vibratory sense; paresis measurable in pinch or grip strength; +provocative testing
IIa		Without muscle wasting	
IIb		With muscle wasting; strength of M3 to M5	
III	Severe sensorimotor deficit	Severe sensorimotor deficit with strength below M3	Persistent paresthesias; decreased vibration perception; abnormal 2-point discrimination (static ≥6 mm or dynamic ≥4 mm); measurable paresis; +muscle atrophy; +provocative tests; +abnormal finger crossing

ment or worsening. A similar system was later developed by Messina with the same categories of outcome [6]. Excellent is complete resolution of symptoms. Good is defined as general resolution with occasional incision site tenderness and mild residual sensory or motor symptoms. Fair is defined as improvement after surgery with persistent sensory changes,

Table 13.2 Modified Bishop scoring system for cubital tunnel release

Severity of postoperative symptoms	Severe	0
	Moderate	1
	Mild	2
	None	3
Subjective improvement	Worse	0
	Unchanged	1
	Better	2
Work status	Not working	0
	Needed to change job	1
	Working old job	2
Subjective strength	Unchanged	0
	Better	1
Sensation	Unchanged	0
	Better	1

Excellent = 8–9 total points; good = 6–7 points; fair = 4–5 points; poor = 2–3 points

residual motor loss, muscle wasting, or intrinsic clawing. Poor involves no improvement or worsening after surgery. Other scoring systems and outcome measures exist, but they are less commonly used or are not specific to cubital tunnel syndrome.

Outcomes of In Situ Decompression

In situ decompression includes a large variety of techniques from standard open cubital tunnel release to 2–3 cm minimally invasive cubital tunnel release, to purely endoscopic techniques. Multiple case series have examined the results of open in situ cubital tunnel decompression. One larger study out of Portland reviewed 131 patients with 164 ulnar

nerve decompressions and an average follow-up of 4.3 years [7]. In early follow-up within 6 months, good or excellent results were achieved in 89%, based on patient reports of resolution of at least 50% or 75% of paresthesias, respectively. Seventy-nine percent of patients retained good or excellent relief at long-term follow-up. In a follow-up case series, Nathan et al. reviewed 102 elbows in 74 patients, with an average follow-up of 5 years [8]. Almost two-thirds of all arms received a patient-rated improvement of greater than 90% symptom relief, with 82% showing greater than 75% improvement. Sixty-one percent of patients had increase in nerve conduction velocity, while 35% had a decrease. Interestingly, there was no correlation between improvement in EMG findings and perceived patient outcome. Average return to work was in 15–16 days. Pavelka found increased grip strength from 20.3 kg (58% of contralateral) to 28.8 kg (79.8% of contralateral) by 13 months after simple decompression [9].

In 1999, Tsai et al. described their series of endoscopic cubital tunnel releases in 76 patients and 85 elbows, with minimum follow-up of 1 year [10]. Good or excellent modified Bishop scores were achieved in 87%. Almost 40% of patients returned to work in 3–6 weeks. Two patients required subcutaneous transposition in the follow-up period. Cobb reviewed the results of 134 consecutive endoscopic releases with an average 2-year follow-up [11]. Postoperative modified Bishop scores were good or excellent in 94%. One patient had recurrence after successful endoscopic release. Two patients failed endoscopic release and required immediate open decompression.

Since the development of endoscopic cubital tunnel release, many studies have sought to define minimally invasive techniques that do not require sophisticated viewing equipment. Taniguchi et al. performed a prospective study of 17 patients treated with a 1.5–2.5 cm incision [12]. They found improved Messina scores with no nerve subluxation and one complication from hematoma. Cho et al. reviewed 15 patients treated with small incisions, with 5 having <2-cm-length inci-

sions [13]. Ninety-three percent of patients achieved a good or excellent outcome by modified Bishop score with no complications. The mean motor conduction velocity improved from 39.8 to 47.8 m/s. Jeon performed a retrospective review of 66 elbows in 62 patients treated with a 1.5–2 cm incision. The average duration of surgery was 12 minutes and the average scar was 1.9 cm. By Messina score, 80% had good or excellent outcomes and the only complications were two hematomas.

Several recent studies have compared the results of open versus endoscopic cubital tunnel release. Aldekhayel performed a systematic review and meta-analysis of open cubital tunnel and endoscopic cubital tunnel release [14]. By including data from 20 studies, this review compared 425 open and 556 endoscopic decompressions with respect to Bishop score and found no difference between groups. Open decompression was found to have excellent outcomes in 60.5% and good outcomes in 19.3%, with a 12% complication rate and 8.5% reoperation rate. Endoscopic decompression demonstrated excellent and good outcomes in 54.3% and 27.5%, respectively, with a 9% complication rate and 0.5% reoperation rate. While the decreased complication rate for endoscopic release was significant, the study concluded similar efficacy between techniques. A subsequent systematic review by Toirac focused on modified Bishop scores and complication rates [15]. Comparing Bishop scores between 344 endoscopic decompression patients and 150 open in situ decompression patients, the modified Bishop scores were good or excellent in 92.0% for endoscopic and 82.7% for open. When comparing complication rates of 691 endoscopic procedures against 417 open decompression procedures, the complication rate analysis showed an odds ratio of 0.28 ($p = 0.002$), indicating reduced odds of complications in the endoscopic group. Their results indicated a difference in favor of endoscopic decompression with respect to patient satisfaction and complications. Further results of in situ decompression versus other techniques will be discussed in the comparative outcomes section.

Outcomes of Anterior Subcutaneous and Subfascial Transposition

Retrospective studies have provided generally positive results for anterior subcutaneous transposition. Lascar reviewed 53 cases with mean of 32 months of follow-up [16]. In 44 cases (83%), patient-reported symptoms completely resolved. There were no complications or cases of worsening symptoms. Scar tenderness was present at 1 month in 25% of patients, but this had resolved at long-term follow-up. While 91% had abnormal 2-point discrimination preoperatively, only 9% still had abnormality postoperatively. Paresis dropped from 62% to 11% postoperatively. While a common concern is expected tenderness of the nerve from its more subcutaneous position, nerve tenderness was only seen in one patient. Guinet reviewed 55 patients with an average of 5.4-year follow-up [17]. The mean postoperative DASH score was 7.27/100 with a mean time to resolution of symptoms of 4.7 months. The patient satisfaction rate was 96% and all but one returned to work. Only five patients had paresthesias at final follow-up and none had paresis.

Black reviewed 2-year outcomes in 51 patients treated with a subfascial sling and either early motion or 2–3 weeks of casting [18]. Patients treated with early mobilization were found to return to work at 9 days postoperatively versus 30 days for patients with 2–3 weeks of immobilization. Overall, 92% were satisfied and 91% had good to excellent outcomes by Bishop score. Interestingly, 31% had a positive Tinel's sign, but this was mild in most cases. No patients lost elbow motion. Weirich also found improved return to work time of 1 month versus 2.75 months with early motion versus 2 weeks of immobilization, respectively [19]. With a primary focus on elbow range of motion, Liu performed a retrospective review of 115 patients with 13.5-year average follow-up [20]. Eighty percent had good to excellent results by Wilson-Krout grading, with no complications. Regardless of prior surgery or elbow trauma, transposition did not decrease postoperative elbow range of motion. Further results of subcuta-

neous transposition versus other techniques will be discussed in the comparative outcomes section.

Outcomes of Anterior Submuscular Transposition

Anterior submuscular transposition generally requires larger open incisions and more aggressive dissection than the previously discussed techniques. Due to the increased invasiveness of this procedure, there is concern for increased pain and longer return to normal function. Despite this, multiple studies have confirmed excellent results for anterior submuscular transposition.

Zimmerman reviewed 82 patients that were followed for an average of 8.3 years [21]. Patients were a mix of moderate and severe disease with 48 Dellon II and 33 Dellon III elbows. Postoperatively, average grip strength across all patients improved to match the contralateral side. Interossei strength improved to full strength by manual muscle testing in 78%. Visible atrophy decreased from 23 to 9 hands. Half of the cases with intrinsic clawing returned to normal posture. All patients decreased by 1 or 2 Dellon grades, with more residual symptoms seen in preoperative Dellon III elbows than in the Dellon II elbows.

Lancigu followed another group of 82 patients for an average 11.1 years after submuscular transposition [22]. Overall, 86% of patients considered themselves cured. Five patients (6%) had recurrence confirmed by EMG at an average of 6.3 years after surgery and seven had progressive deterioration. Upon re-exploration, compression was typically found at the fascial and tendinous arcade. Only three patients had poor results in the immediate postoperative period. Fifty patients returned to the same occupation and 11 required modifications for return.

With a focus on severe McGowan Grade III cubital tunnel syndrome, Lee et al. reviewed 36 cases, with a mean follow-up of 4.4 years [23]. Thirty-four of these cases had at least one

McGowan grade improvement and four returned to normal. Thirty (88%) achieved good or excellent modified Bishop scores. Poor outcomes were seen in the two patients who were symptomatic for over 6 years before intervention. Motor nerve conduction velocity improved from 28.3 to 46.2 m/s and sensory nerve conduction velocity improved from 21.7 to 42.1 m/s. The mean 2-point discrimination improved from 8.7 to 3.5 mm. Grip and pinch strength improved from 48.7% to 86.6% and 36.2% to 80.2% of the contralateral side, respectively.

Comparative Outcomes

Many studies have sought to compare different techniques to determine superiority. In a 2016 Cochrane review, only four prospective randomized studies comparing decompression and transposition were identified [24]. Two studies compared in situ decompression with subcutaneous transposition. Nabhan et al. compared in situ decompression with subcutaneous transposition in 66 patients [25]. All patients experienced improvement in sensory deficits, pain, graded motor strength, and nerve conduction velocity, and there were no significant differences between groups. The authors concluded in favor of in situ decompression as the less invasive, simpler procedure. In a similar comparison, Bartels et al. randomized 75 patients to in situ decompression and 77 to anterior subcutaneous transposition with 1-year follow-up [26]. Excellent or good results were noted in 65% of decompressions and 70% of transpositions with no significant differences between groups. The complication rate was significantly lower in the in situ decompression group at 9.6% versus 31.1% in transposition. The authors again favored simple decompression, due to equivalent outcomes and fewer complications.

The other two prospective randomized studies compared in situ decompression with submuscular transposition. In their series of 44 patients, Biggs et al. found equal efficacy in

improving McGowan scores and Louisiana State University Medical Center grading scores between groups [27]. Wound complications were more common in the transposition group (14% versus 0% in the in situ decompression group). The authors concluded in favor of in situ decompression as the treatment of choice. In a slightly larger group of 70 patients with only severe (Dellon III) cubital tunnel, Gervasio et al. assessed Bishop scores and EMGs at 6 months after surgery [28]. Good or excellent Bishop scores were seen in 80% of in situ decompressions and 83% of transpositions. Both groups showed significant improvement in distal motor latency, compound muscle action potential amplitude, and motor conduction velocity postoperatively, with no significant differences between groups.

Zarezadeh et al. prospectively compared anterior subcutaneous transposition with submuscular transposition in 48 patients [29]. Patients treated with submuscular decompression had significant pain reduction compared to subcutaneous decompression, using their novel pain scoring system. There were no differences between groups with respect to sensation, muscle strength, and muscle atrophy. Based on the pain score alone, the authors favored submuscular transposition.

Multiple retrospective or non-randomized studies have provided longer follow-up, larger sample sizes, or more rigorous outcome measures to compare these surgical techniques. Kamat et al. performed a retrospective review of 480 patients treated with either in situ decompression (179) or subcutaneous transposition (301) [30]. At 3-month follow-up, the transposition group had complete resolution of paresthesias in 93%, elbow pain in 72%, and paresis in 79% and a 94% patient satisfaction rate. The in situ decompression group had complete resolution of paresthesias in 93%, elbow pain in 93%, and paresis in 86%, with a 97% patient satisfaction rate. The only significant difference was in favor of in situ decompression for relief of elbow pain. Keiner examined in situ decompression versus submuscular transposition in 33 patients with a minimum of 3-year follow-up [31]. In the in

situ decompression group, pain and paresis resolved in all patients, and improvements in hypesthesia were seen in all but three patients. In the submuscular transposition group, paresis resolved in all but one, hypesthesia remained unchanged in four, and pain remained in three. None of the differences were significant, and the authors concluded in favor of in situ decompression as the less invasive option.

Charles et al. compared subcutaneous transposition with submuscular transposition in a non-randomized study of 49 patients [32]. The procedures were equally effective for motor and sensory recovery and subjective patient-rated improvement. Another study, a systematic review and meta-analysis, was performed by Liu to compare subcutaneous transposition with submuscular transposition [33]. Combining 9 studies yielded 605 patients for comparison. There was no difference between groups in terms of improvement in subjective symptoms or postoperative 2-point discrimination. However, the incidence of adverse events was lower in the subcutaneous group (RR 0.54, 95% CI 0.33–0.87, $p = 0.01$).

One non-randomized retrospective multicenter study compared outcomes between endoscopic in situ decompression, open in situ decompression, anterior subcutaneous transposition, and submuscular transposition [34]. There were 48 open in situ decompressions, 143 endoscopic in situ decompressions, 82 submuscular transpositions, and 229 subcutaneous transpositions, with mean follow-up of 7.6 years. Ninety percent of patients showed subjective improvement or resolution of symptoms, regardless of technique. Submuscular transposition was associated with higher recurrence rate. In contrast to other reports, subcutaneous transposition was not found to have higher complications.

Staples et al. performed a prospective non-randomized study to assess the morbidity of techniques in terms of pain, narcotic use, and functional scores [35]. The study included 47 in situ decompressions and 78 transpositions (35 subcutaneous and 43 submuscular). Average visual analog scales for pain were equivalent. Narcotic use was significantly higher in the transposition group at the 4–8-week time period, with the

equivalent of 36 more 5 mg hydrocodone tablets taken compared to the in situ group. This difference resolved after 2 months. Functional scores including Levine-Katz scores and PREE scores were better in the in situ decompression group during the 1–3- and 4–8-week periods.

The determination of which procedure is superior in the treatment of cubital tunnel is still highly debated. The prospective randomized studies reviewed earlier show similar outcomes with respect to nerve recovery, with either equivalent or lower complication rates in the in situ decompression group. As a result, many of the authors favor simple decompression. Despite this, there is still concern that simple decompression does not address increased pressure in the nerve from elbow flexion or ulnar nerve subluxation. There is a potential subset of patients that may respond poorly to simple decompression, but this group has not yet been explicitly defined.

Predictors of Outcome

Multiple studies have sought to identify predictors of failure or poor outcomes following various techniques of cubital tunnel release. Age, symptom duration, stage of disease, electrodiagnostics, type of surgery, provocative signs, diabetes mellitus, smoking, and cervical disease have all been implicated [26, 28, 32, 36–41]. In 2011, Shi performed a systematic review of 26 studies [42]. The most consistent variables investigated in these studies were older age, symptom duration, severity of preoperative status, preoperative electrodiagnostic studies, type of surgery, and work compensation status. Due to the variability of reporting measures, conflicting results, and small sample sizes, no conclusion could be formed regarding these variables. More recent studies have examined predictors of outcomes. Krogue performed a case-control series of 44 failed decompressions compared to 79 successful decompressions [43]. Overall, 19% of patients undergoing simple decompression in the study period required revision.

History of elbow fracture or dislocation and McGowan stage I were independent predictors for revision. With respect to McGowan Grade I patients, the authors noted that patients undergoing surgery for lesser symptoms were more likely to require revision. The average time to revision was 1 year. In this study, age, sex, body mass index (BMI), tobacco use, and diabetes mellitus were not correlated.

Kang reviewed a series of 41 patients treated with in situ decompression (30) or anterior subcutaneous transposition based on intraoperative findings of nerve instability (11) [44]. With 2-year follow-up, grip and pinch strength increased from 19.4/3.2 to 31.1/4.1 kg. Two-point discrimination improved from 6.0 to 3.2 mm. DASH scores improved from 31.0 to 14.5. Worse DASH scores were correlated to older age, weaker preoperative grip strength, and worse 2-point discrimination. Kong examined a larger series of 235 patients status post in situ decompression [45]. With an 89% rate of satisfactory outcomes, only preoperative symptom severity was correlated with unsatisfactory outcomes. Young age was associated with increased postoperative instability, but not necessarily an unsatisfactory outcome.

For severe cubital tunnel syndrome, Tong reviewed 146 patients with McGowan Grade III treated with in situ decompression or subcutaneous transposition with a minimum of 2-year follow-up [46]. The factors that were associated with poorer outcomes were older age, longer duration of disease, absent sensory conduction on electrodiagnostic testing, and shorter postoperative follow-up. With respect to short postoperative follow-up, they concluded that patients may take up to 2 years for maximal recovery and shorter follow-up does not assess the true end point of recovery.

Revision Surgery

Submuscular transposition is frequently considered for treatment of failed cubital tunnel release using other techniques. Vogel reviewed 18 patients treated with submuscular trans-

position after prior failed in situ decompression or subcutaneous transposition [47]. After an average follow-up of 34 months, pain and scar hypersensitivity generally improved. Ten patients returned to a higher level of activity, and three of four who were unemployed returned to work. Tinel's resolved in 12 of 17 patients. Postoperative average grip strength improved by about 32%. All patients were rated as fair to poor outcome preoperatively, and good or excellent outcomes were achieved in 10/18 patients. Sixteen of 18 patients were satisfied and would repeat the procedure. Wever et al. presented a larger series of 34 patients with longer follow-up of 4 years [48]. Twenty-one of 34 patients (61%) were improved by surgery, with 8 patients completely free of symptoms. Paresthesias and pain responded better to revision than strength and sensation did. Twenty-five percent of patients remained unsatisfied with surgery.

Aleem performed a case-control series of 28 revisions, mostly submuscular, and 28 matched primary cubital tunnel releases [49]. For revision cases, 75% of nerves had scarring, but no defined explanation for failure. Seventy-nine percent of revisions experienced some relief, but 50% had persistent constant symptoms, compared to 18% in primary decompressions. The revision group had significantly weaker key pinch, worse 2-point discrimination, and more frequent ulnar nerve tenderness. Additionally, an average of 10^0 of elbow extension was lost. McGowan grade improved in 25% of revision cases versus 64% of primary cases. Twenty-one percent of revision cases experienced worsening of McGowan grade. Davidge and Mackinnon reviewed a cohort of 50 patients with recurrent/persistent cubital tunnel syndrome who were revised with a submuscular decompression [50]. Patients improved significantly with respect to multiple measures of pain. Pinch strength, grip strength, and DASH scores showed slight, nonsignificant improvements. High preoperative pain and more than one prior cubital tunnel procedure were significant predictors of increased postoperative pain, while prior simple decompression alone was predictive of improved postoperative pain scores.

In general, the results of revision are less predictable and worse than primary decompression. Revision can still allow

patients to achieve significant gains, particularly with respect to pain. Appropriate preoperative counseling is mandatory for these patients to temper their expectations.

Conclusion

Outcome reporting for cubital tunnel release is limited by inconsistency in measured outcome variables and level 3 or poorer evidence. Trends in the literature show favorable post-surgical outcomes for all techniques, particularly in subjective patient-reported outcomes and McGowan scores. Less invasive techniques tend to be favored.

Older age, longer duration of symptoms, and poor preoperative scores may be associated with worse surgical outcomes, but this finding is inconsistent in the literature. Results of revision surgery are less predictable and generally worse than primary procedures but should be considered when pain is a predominant concern. Patients should be counseled that the recovery period may last months to years and that symptom improvement, not necessarily complete resolution, may be expected.

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Chapter 14
Management of Complications of Cubital Tunnel Surgery

David Turer and Rafael J. Diaz-Garcia

Cubital tunnel syndrome is one of the most common conditions evaluated by upper extremity and peripheral nerve surgeons and the second most common compressive neuropathy only to carpal tunnel syndrome. Patients with classic presentation and symptoms can at times be treated conservatively with splinting, and those who go on to surgery, for the most part, do well. However, complications can arise after surgical intervention. Although unusual, these complications can provide significant distress to patient and surgeon alike. The surgeon should take ownership of the situation and reassure the patient that they will work together to get through

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the unexpected outcome. We will discuss the incidence of complications after surgical release and our preference in management.

General Surgical Complications

As in any operation, cubital tunnel release is associated with acute surgical complications. In most cases, these tend to be relatively mild and rarely require a return to the operating room. The rate of hematoma formation after open surgery is low, with most studies reporting rates between 1% and 5% [1–5]. Endoscopic release appears to have a similar rate of hematoma formation, with most studies also reporting rates between 1% and 5% [6, 7]. However, hematoma is reported to be the most frequent complication of endoscopic cubital tunnel surgery presumably because of bleeding from vessels not visualized during the procedure. Most infections are reported to be a superficial cellulitis that can be managed with oral antibiotics. Rarely does a deep infection occur requiring operative washout. Open procedures are associated with a 0–5% rate of infection [3–5, 8, 9] and endoscopic procedures reporting similar outcomes with rates of infection around 1–5% [7, 9]. One notable difference is that anterior transpositions are associated with a higher rate of wound infection, 9–14% [10, 11]. Seromas were rarely reported and can almost always be managed with simple aspiration. In a 10-year retrospective review of VA patients undergoing open cubital tunnel release, tobacco use was found to place patients at increased risk of complications, while diabetes, perioperative antibiotic use, obesity, and time of operation did not [5].

Complex regional pain syndrome (CRPS) has been widely reported after carpal tunnel release, but reports after cubital tunnel release are limited. In fact, no studies have reported the specific incidence of CRPS after cubital tunnel release. There is a report of 93 patients with CRPS who were found to have nerve compression syndromes and were treated with 22 carpal tunnel and 5 cubital tunnel releases [12].

Nerve Injury

Nerve injuries can be extremely challenging problems for both surgeon and patient. A thorough understanding of the relevant anatomy and careful dissection may help one avoid these complications in the first place. However, despite all attempts at caution, one must be prepared deal with any iatrogenic nerve injuries that may occur after cubital tunnel release.

Partial or complete transection of the ulnar nerve is a rare but potentially devastating complication. The incidence of this complication is hard to estimate as only case reports exist in the literature. Any patient with proximal ulnar nerve injury will likely have some residual deficit despite attempts at repair, and it is critical to identify this injury as soon as possible as motor end plates degenerate at a rate of around 1% per week. Delays in reconstruction generally result in significantly worse outcomes [13–15]. It is best to repair the nerve at the time of injury if possible, as an injury that is not immediately appreciated may result in formation of a gap as the nerve retracts. Seventy-three percent of ulnar nerve injuries recovered with direct repair compared to 56% that required cable grafting [16]. Others have demonstrated worse outcomes with around half of patients regaining meaningful motor function even after primary repair [13]. Once retraction has occurred, the nerve cannot typically be primarily repaired without undue tension, and options for reconstruction include autogenous cable grafting or the use of allograft. Decellularized nerve allograft has shown comparable outcomes to autogenous cable grafting and does not produce a donor site [17]. Gaps in large diameter nerves should generally not be repaired with conduit alone [18].

Given the long distance from the elbow to the hand intrinsic muscles, one must consider the use of a nerve transfer to prevent motor end plate degeneration while the primary nerve regenerates. If no proximal reconstruction is being performed, then an end-to-end anterior interosseous nerve (AIN) to ulnar motor nerve may be considered. However, if there is the possibility of axonal regeneration through the

native ulnar nerve, one may consider the supercharged end-to-side AIN transfer to act as a "babysitter" for the intrinsic muscles until the ulnar nerve axons regenerate [19, 20].

The posterior branch of the medial antebrachial cutaneous nerve (MACN) runs in close proximity to the cubital tunnel and may be injured during ulnar nerve release. Anatomic studies have demonstrated that branches of the MACN cross over the ulnar nerve 61% of the time proximal to the medial epicondyle and 100% of the time distally [21]. Injury to the MACN can cause painful neuromas, numbness, hyperalgesia, and painful scars [22]. MACN symptoms can be easily confused with recurrence of ulnar nerve symptoms, and careful investigation is warranted to ensure the appropriate treatment is undertaken. Diagnostic nerve blocks may be helpful in making the diagnosis of MACN injury or neuroma.

In general, patients with painful neuromas may benefit from involvement of a multidisciplinary treatment team, as a variety of pharmacologic and psychological therapies are available. However, a meta-analysis of treatment for painful neuromas demonstrated that 77% of patients who underwent surgical intervention had significant improvement of their symptoms, and surgery should be considered for any patient with a painful neuroma. In particular, patients with a long duration of symptoms (greater than two years) or previous surgery are probably best treated with resection of the neuroma and anatomic reconstruction with allograft for pain control. Other options for neuroma control include transposition into muscle or other vascularized tissue [23].

There are few reports of nerve injury in endoscopic cubital tunnel release. In one study, 12% of patients had temporary numbness in the distribution of the MACN, but almost all had resolution of their symptoms within several months [24].

Persistent Symptoms/Incomplete Release/ Recurrence, Recompression

The vast majority of patients will see improvement of their symptoms after cubital tunnel release [25, 26]. However,

some patients will have persistent or recurrent symptoms after surgery, particularly those who present at a more advanced stage.

Workup

Differentiating between an incomplete release and recurrence of nerve compression can be difficult. It may take months before patients notice the full benefit of their initial release, and one should be cautious about reoperating too quickly. Obtaining a history of the patient's symptoms is critical in determining the appropriate course of treatment. If the patient's symptoms were not improved or continued to worsen after surgery, it is possible that there was a missed concurrent diagnosis, a missed site of compression, or a severely dysfunctional nerve preoperatively. Conversely, if the patient improved after surgery and then worsened over time, it is more likely that there has been a recurrence due to recompression of the nerve.

Electrodiagnostic studies may be helpful in determining when and if to intervene on patients with persistent symptoms. If the patient has not had a previous study, this may assist in identifying additional sites of compression that may be contributing to the patient's symptoms. If there are findings of compression proximal or distal to the elbow, one may consider obtaining imaging (MRI) to investigate other sites of compression. If the postoperative study shows improvement, then a longer trial of observation may be warranted. However, it should be noted though that some patients with incomplete release may see a modest improvement in electrodiagnostic study results despite the ongoing compression. Patients with worsening results likely warrant re-exploration to ensure that all site of compression are fully released and to rule out nerve injury. One should note that even in patients with improvement in symptoms, there may not be an improvement in electrodiagnostic study results, particularly in patients with severe preoperative disease [27]. Electrodiagnostic testing should be used to assist in decision-making, as opposed to being the ultimate factor in deciding to return to the operating room.

Failure of Surgery

In patients whose symptoms did not improve or have worsened after surgery, a thorough evaluation of other causes of nerve compression should be undertaken both proximally and distally to the cubital tunnel. If no electrodiagnostic study was performed before the initial operation, one should be performed to rule out other sites of nerve compression. Even if cubital tunnel syndrome was appropriately diagnosed and released, it is possible to have multiple sites of compression resulting in "double-crush phenomena." Other etiologies to consider are listed in Table 14.1. As mentioned earlier in the chapter, MACN injury can also be confused with ulnar neuropathy.

Another possibility is that the nerve was not completely released at the initial operation. Surgeons need to be familiar with all potential sites of compression. When evaluating patients, patients with small scars or who underwent simple in situ decompression may not have had inspection of all the potential sites of compression. An iatrogenic compression can occur when the nerve is moved out of its anatomical location during anterior transposition. During transposition, the

Table 14.1 Confounding conditions

Carpal tunnel syndrome
Guyon's canal syndrome
Cervical radiculopathy
Hypothenar hammer syndrome
Pancoast tumor
Thoracic outlet syndrome
Raynaud's disease or phenomena
Flexor carpi ulnaris tendinitis
Medial epicondylitis
Brachial plexus neuritis

nerve may be compressed by the intramuscular septum or kink as it passes over the medial epicondyle and into the heads of the flexor carpi ulnaris. If another surgeon performed the operation, it may be helpful to obtain the previous operative report, but by no means should one be assured based on the operative report alone.

Patients who have had partial or complete resolution of their symptoms with subsequent worsening should be classified as having a recurrence as opposed to a failure of the initial operation. It has been demonstrated that the primary reason for recurrence is perineural fibrosis and scarring [26]. Patients with a recurrence should be managed similarly to patients who present with persistent symptoms. Other causes of their symptoms should be ruled out before undertaking operative intervention. It is often helpful to obtain a new baseline electrodiagnostic study to assist in diagnosis and prognostication. One must remember a patient with recurrent disease, and significant scarring will be predisposed to the same issue after revision, and all attempts should be made to reduce the possibility of another recurrence.

Generally, patients who present with severe disease (whether primary or secondary) have far worse outcomes than those who present with milder symptoms [14]. Setting appropriate expectations in this group of patients is extremely important, as a technically successful operation may still result in a suboptimal outcome with minimal symptom improvement. Even in patients with less severe disease, outcomes after revision are not as good when compared to primary surgery. In one study, McGowan grading improved in only 25% of revision cases compared to 64% of primary surgeries. In fact, 21% of revision patients had worsening of their McGowan grade [28].

Nerve Instability

Instability of the nerve may result in subluxation over the medial epicondyle with elbow flexion and has been seen

in some series in as many as 44% of patients with persistent symptoms [26]. One must check for subluxation during the physical exam, particularly in patients who underwent in situ decompression. If evidence of instability is detected, the patient will likely require surgical intervention with a procedure to stabilize the nerve, typically with an anterior transposition procedure. Surgeons performing endoscopic cubital tunnel release should be aware of the possibility of nerve subluxation and should check before completing the procedure. If subluxation is detected, the procedure should be converted to an open anterior transposition.

Principles of Surgical Revision

The primary goal of revisional surgery is to ensure that any potential sites of compression are released and that the nerve is placed into a stable position. The incision should be extended proximally and distally to ensure adequate exposure. The ulnar nerve should be identified proximal and distal to the previous surgical site, away from areas of dense scar. Only once the nerve has been safely identified should the dissection proceed toward the cubital tunnel. It is important to remember that the MACN will likely be encountered in this field and may be involved in dense scarring and all attempts should be made to protect it. All potential sites of compression should be explored and released. There is universal agreement that an adequate neurolysis must be performed. However, a variety of techniques may be used in revision cubital tunnel surgery, with no single technique proving superior to the others. Options for revision are listed in Table 14.2. Specifics of these procedures will be covered in another chapter.

In summary, cubital tunnel release is a safe operation that usually goes without incident. When complications occur, the surgeon may be uncomfortable with what to do next. However, a thoughtful analysis of the issue at hand can lead to ultimate success.

Table 14.2 Surgical options for management of recurrent cubital tunnel syndrome

In situ neurolysis
Subcutaneous transposition
Subfascial transposition
Submuscular transposition
Nerve wrap with autogenous vein
Nerve wrap with synthetic collagen conduit
Medial epicondylectomy

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Chapter 15
Management of Chronic Ulnar Neuropathy

Wesley N. Sivak and Robert J. Goitz

Introduction

Regardless of discipline or subspecialty, physicians treating elbow conditions must be familiar with the principles involved in the evaluation and management of ulnar nerve dysfunction. Dysfunction results from direct compression of or traumatic injury to the ulnar nerve. Unquestionably ulnar neuropathy is best treated early – prior to onset of irreversible nerve changes that result in distinct patterns of sensory loss and motor weakness [1]. Once established, chronic ulnar neuropathy leads to muscle wasting and force imbalance in the upper extremity, resulting in predictable patterns of impairment.

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Management of chronic ulnar neuropathy remains a complex problem without simple solutions. Any surgeon caring for patients suffering from chronic ulnar neuropathy must be able to recognize the impairment patterns, accurately assess the degree of nerve dysfunction, and assess the recovery potential before formulating and implementing a treatment plan. This chapter provides an overview of the management of chronic ulnar neuropathy, beginning with patient evaluation and then discussing some of the more common clinical scenarios and available treatments.

Patient Evaluation

Evaluation of the patient with chronic ulnar neuropathy begins with a thorough history focusing on duration and severity of symptoms. Numbness in the ulnar digits of the hand, weak grip/pinch, and loss of manual dexterity will be among the chief complaints. Nerve compression and nerve trauma must be differentiated, although both lead to the same debilitating condition. Unlike compression of the median nerve, compression of the ulnar nerve does not result in pain [2]. Therefore, patients are more likely to ignore or dismiss symptoms of ulnar neuropathy and present much later in the degenerative process. When the nerve is compressed, typically no recovery occurs until an intervention takes place. One must establish if any prior surgical interventions aimed at decompressing the nerve have been performed. Management of medical comorbidities such as diabetes and elimination of smoking may improve recovery potential for the nerve [3, 4].

Traumatic nerve injuries, such as a laceration or crush, require one to know not only when the insult occurred but also the degree of recovery experienced by the patient. An unrepaired nerve transection is unlikely to experience any degree of recovery, whereas a crushed nerve that remains in continuity may. Level of injury is also important when dealing with traumatic injuries. A high ulnar nerve injury in the bra-

chium is unlikely to reach the intrinsic muscles of the hand before degeneration of the motor end plates, which occurs 12–18 months after injury [5]. Nerve transfer techniques are utilized early to address high ulnar nerve injuries; they are discussed elsewhere in this text.

Regeneration in the peripheral nervous system can occur to a significant degree under favorable conditions, where human axon growth rates can reach speeds of up to 1 mm/day in the upper extremity. Age of the patient is the single most critical factor in determining recovery potential [6]. Results of nerve repair begin to decline in the second decade of life and are generally poor after the sixth decade. Mechanism of injury is also important to consider. For example, gunshot wounds are associated with neuropraxic injuries and can recover over time [7]. In such cases, consider serial nerve tests to determine if nerve recovery is occurring. However, penetrating wounds more likely result in transection and have a poorer prognosis without surgical intervention [8]. Furthermore, combination injuries where there is soft tissue damage, fracture, and nerve injury portend a grave prognosis for recovery due to the poor healing environment and scarring.

Examination begins with observation, as the posture of the hand delineates between high and low ulnar nerve lesions. Nerve injury distal to the mid-forearm results in ulnar clawing. Division of the nerve at this level results in paralysis of all intrinsic muscles of the hand except the first and second lumbricals and thenar muscles. Ulnar clawing arises due to the unopposed action of the extrinsic muscles that produce metacarpophalangeal (MPJ) hyperextension and interphalangeal joint (IPJ) flexion. If the ulnar nerve is inured above the level of the mid-forearm, clawing of the ulnar two fingers does not occur because the ulnar innervated extrinsic muscles producing IPJ flexion in the ring and small finger are also denervated. (Ulnar clawing must be differentiated from the complete claw hand, which represents a mixed nerve injury and is produced by low lesions of the median and ulnar nerve, where MPJs are extended and IPJs are flexed by the functional extrinsics.) Patients with any level of ulnar nerve injury when attempting

to pinch will compensate for loss of the adductor pollicis with the flexor pollicis longus resulting in hyperflexion of the distal thumb joint (i.e., Froment's sign). Patients will also be unable to abduct or adduct the fingers due to loss of the dorsal and palmar interossei, respectively. The extensor digiti minimi (EDM) provides an abduction moment to the small finger through an indirect insertion into the abductor tubercle on the proximal phalanx. The third palmar interosseous normally counters this effect. However, it is paralyzed in ulnar nerve palsy, leaving the EDM unopposed to produce abduction of the small finger (i.e., Wartenberg's sign). Sensation will be diminished or even absent along the ulnar border of the ring finger and the entire small finger.

Passive supple joints are an absolute necessity to ensure that any attempts to rebalance forces and supply motion across an affected joint are successful. Stiff joints and fixed contractures will limit the results of any reconstructive procedure. Joint release for fixed contractures must be performed prior to tendon transfer. Consideration must also be given to the nature and quality of the other tissues in the affected extremity – traumatic injuries are rarely isolated to the ulnar nerve. The skin envelope should be free of scar and contracture. Just as stiff joints limit the benefit of reconstructive procedures, densely scarred soft tissues will have a similar effect on the underlying joints. Locations of scar from prior procedures also need to be respected when planning incisions. The extremity also needs to be assessed for any underlying bony deformity. Non-unions and malunions must be addressed before entertaining tendon transfer procedures. Finally, function of the radial and median nerve must also be assessed. Other concurrent nerve deficits in the upper extremity severely limit options for functional tendon transfer available to the reconstructive surgeon due to lack of available donor units.

Treatment Options

Treatment of chronic ulnar neuropathy depends upon the level of injury, degree of deformity, and existing joint motion.

In low ulnar nerve lesions, the main goals of reconstruction are to improve thumb pinch, correct finger clawing, and restore a normal pattern of finger flexion. (In normal grip kinematics, the MPJs flex before the IPJs, and the object is drawn into the palm of the hand. In ulnar paralysis, the IPJs flex first followed by the MPJs, and objects can be pushed out of the palm by the fingertips.) In high ulnar nerve lesions, consideration should be given to restoration of ring and small finger DIPJ flexion. Restoration of sensation to the ring and small fingers remains possible but not often pursued as the loss of sensation in the ulnar nerve is not as devastating as loss of the median nerve [9]. Procedures have also been described to correct the small finger abduction deformity and wasted appearance of the hand due to loss of intrinsic muscle bulk.

Ulnar Claw Hand

With ulnar clawing, first assess if the finger joints have full passive range of motion. Joint stiffness is an absolute contraindication for any tendon transfer; it must be corrected with therapy and/or surgery. Next assess the ability of the extensor digitorum communis (EDC) to actively extend the PIP joint. To perform this assessment, the MPJ is blocked from hyperextending, and the patient is asked to extend the PIPJs (i.e., Bouvier's maneuver). If the PIPJs can be actively extended with MPJs blocked, static operations that address the MPJ hyperextension will correct the deformity. If the PIPJs remain flexed when the MPJ hyperextension is corrected but they can be fully extended passively, a dynamic tendon transfer is required to provide flexion at the MPJs in addition to an extension force at the PIPJs. With long-standing clawing, the extensor mechanism can stretch, and the lateral bands descend palmar to the axis of rotation of the PIPJ. The lateral bands become flexors of the PIPJ rather than extensors. The lateral bands must first be reoriented dorsal to the axis of rotation of the PIPJ. Failure to do this before performing a tendon transfer into the lateral bands only exacerbates clawing.

Static Techniques

Numerous static techniques have been described [10–14]. The ideal candidate has full motion of all the involved finger joints and positive Bouvier's maneuver. Static techniques include MPJ volar capsulodesis and numerous tenodesis procedures that prevent MPJ hyperextension while using the extrinsic flexors to flex the distal joints. The senior author (RJG) prefers the Zancolli lasso procedure (see Fig. 15.1). The flexor digitorum superficialis (FDS) tendons of the ring and small finger are cut distal at the A3 pulley, withdrawn proximal to A2, and transferred around the A1 pulley and sutured back to themselves. The tensioning is performed with the wrist in neutral position, tight enough to hold MCP joint in flexion to match resting position of adjacent digits. The Zancolli lasso procedure can be utilized in all four digits for combined nerve injuries when limited donors are available. Like all the passive correction procedures, it does not improve power to the hand but helps to correct the claw deformity and permit grasping of larger objects.

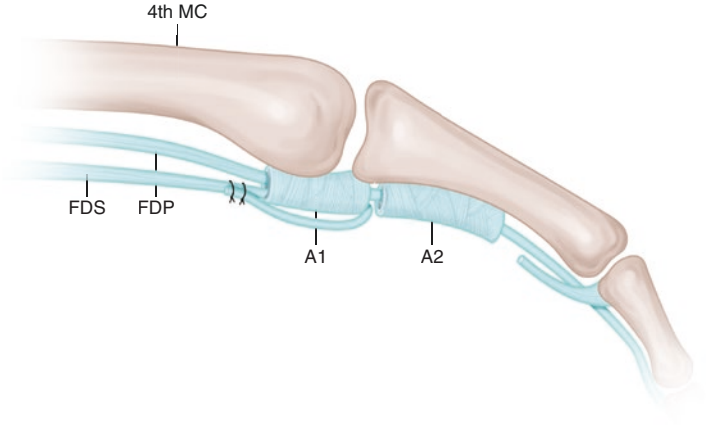

Figure 15.1 Zancolli lasso procedure

Dynamic Techniques

Dynamic tendon transfers correct finger positioning and improve power to the hand. Even if patients have a positive Bouvier's maneuver, many surgeons still perform a dynamic transfer for improved power. Numerous dynamic transfers have been described using a variety of muscles as motors [15–19]. The insertion site helps to fine-tune the function of the transfer. Insertion of the transfer onto the proximal phalanx provides pure flexion of the MPJ, improving power grip, and is ideal if patient has positive Bouvier's maneuver. Transfer to the lateral bands produces MPJ flexion as well as extension of the IPJs. In general, the muscle selected for motor transfer must pass volar to the axis of rotation of the finger MPJ (or palmar to the transverse metacarpal ligament) to achieve MPJ flexion. Tendon transfers for dynamic correction of clawing also cross the wrist joint, and their action is amplified by the tenodesis effect of wrist motion (i.e., wrist flexion tightens transfers that cross dorsally, and wrist extension tightens those that cross volarly). FDS transfers were originally described and utilized by Stiles, Bunnel, and Littler [16, 18, 20]. These transfers are long enough to allow for transfer without grafting, but are far less powerful than wrist extensors. The Brand transfer utilizes the ECRB and has become the most common dynamic tendon transfer to correct the claw hand. However, due to the need for tendon grafts, it does carry increased risk of adhesions and rupture. The senior author (RJG) will provide power transfer to all four fingers to improve overall grip strength, although most isolated ulnar nerve palsies result in clawing of only the ring and small fingers.

The senior author (RJG) prefers the Brand transfer technique as a dynamic tendon transfer to prevent MPJ hyperextension and provide active PIPJ extension (see Fig. 15.2). Dorsal transfer of the extensor carpi radialis brevis (ECRB) tendon is accomplished by identifying the tendon distally through a transverse incision over the distal radius. The tendon is withdrawn into a second transverse proximal incision

in the forearm. Palmaris longus (PL) is harvested and weaved to the distal end of the ECRB tendon. The PL graft is then passed into the original incision over the distal radius and split into four tails. Dorsal radial incisions are made at the base of the middle, ring, and little fingers, and the radial lateral band of each of these and the ulnar lateral band of the index finger are identified. Tendon-tunneling forceps are passed from the finger incisions palmar to the deep transverse metacarpal ligaments to the dorsal hand wound. The tendon grafts for each of the fingers are passed and placed under tension, suturing the index finger first and then sequentially the middle, ring, and small fingers, setting the tension so that the transfers are completely relaxed with the wrist in 45 degrees of extension, the MPJs flexed to 70 degrees, and the IPJs extended. This position is maintained for 3 weeks postoperatively. The extensor carpi radialis longus (ECRL) tendon can also be utilized to motor the transfer, preserving the ECRB, which is thought by many to be the primary wrist extensor.

Flexor route transfer of the ECRB/ECRL was also described by Brand and involves passing the transfer with PL graft through the carpal tunnel as opposed to the intermetacarpal spaces [15]. The senior author (RJG) has found this

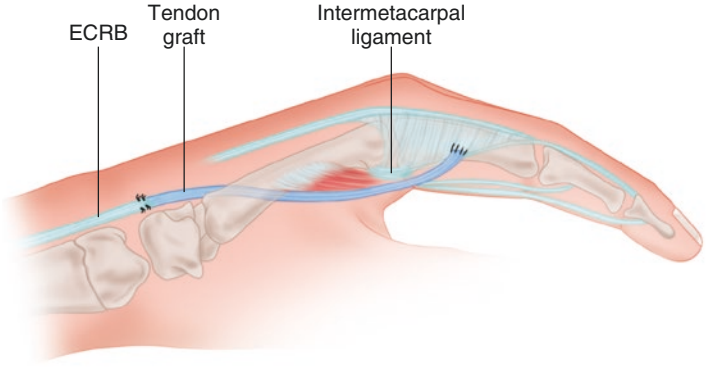

Figure 15.2 Brand transfer

transfer to be more technically demanding, as it is difficult to tension appropriately and risks injury to the median nerve.

Thumb Pinch

Up to 80% of pinch strength can be lost with ulnar nerve injury. Pinch power arises primarily from the adductor pollicis (AP) and the first dorsal interosseous (FDI) muscles, as they are responsible for thumb adduction and radial abduction of the index finger, respectively. In ulnar nerve palsy, the AP and FDI are lost. The EPL and FPL both remain functional, but only provide weak thumb adduction. Power pinch only occurs with flexion of the IPJ thru the FPL (i.e., Froment's sign). IPJ hyperflexion may also result MPJ hyperextension due to loss of flexor pollicis brevis (i.e., Jeanne's sign). The goal of reconstructive thumb surgery in ulnar nerve palsy is to restore strong adduction of the first metacarpal. The adjacent middle, ring, and small fingers can stabilize the index finger and provide an adequate post to pinch upon. Therefore, tendon transfers to restore index abduction are generally not necessary.

The most commonly used thumb adductor transfers employ the ECRB/ECRL, FDS, brachioradialis (BR), and extensor indicis propius (EIP) [21–24]. BR is least commonly used, as it requires extensive dissection for adequate mobilization. EIP is considered too weak to provide functional pinch strength. FDS and ECRB/ECRL are most often used, but depend upon the transfer used for anti-clawing. Edgerton and Brand described using the ring finger FDS to restore thumb adduction for low ulnar nerve palsy. If the patient has high ulnar nerve palsy, then the middle finger FDS is used since the FDS is the sole flexor of the ring finger due to the absence of the ulnar innervated FDP.

The senior author (RJG) prefers using the FDS of the ring (or middle in high injuries) for thumb adduction (see Fig. 15.3), leaving the ECRB for restoration of intrinsic function. The FDS to the ring finger is released distal to the A2 pulley and

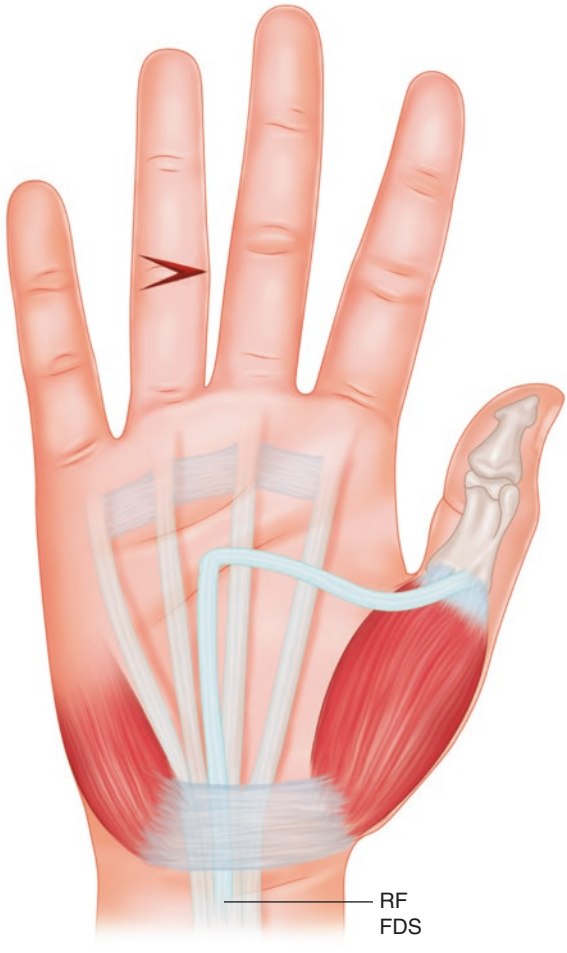

Figure 15.3 FDS transfer for thumb adduction

withdrawn back into a palmar incision. The tendon is then passed around one of the vertical septa running from the palmar fascia to the middle finger metacarpal to act as a pulley, preventing radial migration of the transfer. The FDS transfer then traverses the palm parallel to the transverse head of AP and inserts at the attachment of the adductor on the first

metacarpal. Tension is set such that the thumb abuts the index finger with the wrist in 45 degrees of extension. The patient is splinted with the wrist in neutral and thumb abducted.

If not utilized to correct clawing, the ECRB remains the most powerful dynamic procedure to restore thumb adduction. It is best utilized in patients with weak pinch and a positive Bouvier's maneuver where a static procedure corrects clawing. However, due to the need for tendon grafts, ECRB transfer does carry increased risk of adhesions and rupture. The ECRB tendon is harvested by dividing its insertion on the middle finger metacarpal base through a dorsal transverse wrist incision. The tendon is retrieved into a second dorsal transverse incision proximal to the extensor retinaculum. Between the dorsal aspect of the index and middle finger metacarpals, a third transverse incision is made over the proximal end of the second intermetacarpal space. The fascia of the dorsal interosseous is incised, and a small window is created to access the intermetacarpal space. A longitudinal incision is then made on the ulnar side of the MPJ of the thumb. A curved hemostat is tunneled from this incision, dorsal to the transverse head of the adductor pollicis (AP), through the window in the index-middle interosseous space. A PL graft is passed through this tunnel to the thumb and is sutured to the tendon of the AP. The proximal end of the graft is passed into the most proximal dorsal forearm incision, where it is sutured to the ECRB tendon. The tension is set so that the thumb lies palmar to the index finger with the wrist is in neutral. With the wrist extended, the thumb should abduct fully. With the wrist flexion, the thumb should lie against the palm. Postoperatively, the hand is immobilized with the thumb in neutral position and the wrist in 40 degrees of extension. After 3 weeks of immobilization, the patient is given a protective splint and is encouraged to perform active range of motion exercises.

Thumb Arthrodesis

In the intrinsic-minus thumb, loss of AP and FPB function results in flexion of the IPJ during pinch (i.e., Froment's sign)

and hyperextension of the MPJ (i.e., Jeanne's sign). The remaining extrinsic thumb muscles are unable to control these two joints independently, and fixed deformities may develop. Pinch function and strength are improved by fusing either the thumb MPJ or IPJ, giving the FPL or EPL better control of movement at the remaining unfused joint, respectively. Arthrodesis of the MPJ is often preferred because the retained IPJ motion better preserves pinch strength with FPL function. Preserved IPJ motion allows the patient to roll objects between the thumb and index finger. A fixed deformity of one joint is a contraindication for arthrodesis of the other.

MPJ arthrodesis is indicated when there is pain, hyperextension contracture and/or hyperextension instability during pinch. Arthrodesis of the thumb IPJ can provide patients a better pinch when there is IPJ instability. Patients again should be forewarned that they might sense a loss of dexterity due to the inability to roll objects between the thumb and index finger. The IPJ is fused in 20–30 degrees of flexion; positioning can be determined by observing the position of the contralateral thumb during pinch. There are many techniques described for IPJ arthrodesis, but if flexion is desired, then a combination of Kirschner wires and tension band is often necessary.

The split-FPL tenodesis offers an alternative to thumb IPJ fusion [25]. The FPL is exposed at the level of the proximal phalanx through a palmar longitudinal incision; the radial half of this tendon is divided at its insertion on the distal phalanx and split away from the intact ulnar half to the level of the base of the proximal phalanx. The EPL is exposed through a dorsal longitudinal incision over the proximal phalanx, and the radial slip of the FPL is passed subcutaneously around the radial border of this bone and sutured to the EPL near its insertion. The transfer promotes balanced flexion and extension forces across the IPJ, preventing hyperflexion deformity.

Correction of Little Finger Abduction Deformity

Loss of the third volar interossei results in unopposed small finger abduction (i.e., Wartenberg's sign). The most commonly uti-

lized transfer to restore adduction relies upon the ulnar slip of the extensor digiti minimi (EDM) [26]. The ulnar half of the EDM tendon is detached from the extensor hood of the little finger at the MPJ and retrieved through an incision distal to the extensor retinaculum. A palmar incision that extends obliquely from the distal palmar crease to the proximal digital crease is made to expose the deep transverse metacarpal ligament and the flexor sheath of the little finger. The EDM tendon is passed through the fourth intermetacarpal space into the palm. If the little finger is not clawed, the tendon slip is sutured onto the insertion of its MP radial collateral ligament on the proximal phalanx. If the small finger is clawed as well as abducted, the tendon slip is inserted onto a radially based flap of the flexor tendon sheath just distal to the A1 pulley. For tensioning, the wrist is held in neutral and the MPJ in 20 degrees of flexion. The ring and small finger MPJs are splinted in flexion for 4 weeks with the wrist extended, but the IPJs are left free, and motion is encouraged to prevent adhesion formation around the flexor tendons.

Restoration of Ring and Little Finger Flexion

In a high ulnar nerve palsy, the FDP muscles for the ring and small fingers are paralyzed, so these fingers have no active flexion at their DIP joints and rely on FDS function, which are often rudimentary or absent in the small finger. In order to restore flexion of the ring and small fingers, the profundus tendons of the ring and small fingers can be sutured to the profundus tendon of the middle finger in the forearm. An alternative is tenodesis of the ring and small finger DIP joints using their paralyzed FDP tendons. Transfer of the FCR to the FCU can also be performed simultaneously to restore strong flexion and ulnar deviation of the wrist.

Restoration of Sensibility

Loss of sensibility on the ulnar border of the hand and loss of proprioception in the small finger can produce functional

limitations. Repeated ulceration at the tips of the digits can lead to necrosis and shortening, despite the success of tendon transfers in correcting the claw deformity. Digital nerve transfer of the functioning median-supplied ulnar digital nerve of the middle finger to the nonfunctioning ulnar digital nerve of the small finger can be performed [27]. Studies show that 85% of patients can regain sensibility levels of S3+ or S4. This technique may be beneficial to patients who present late after ulnar nerve injuries. Tissue loss should preclude any attempt at reinnervation.

Wasting of the Intermetacarpal Spaces

Severe intermetacarpal atrophy can be quite disfiguring and may present a barrier to social reintegration in those afflicted with chronic ulnar neuropathy. Dermal grafts can mask interosseous wasting and are most successful when performed between the thumb and index metacarpals [28]. Also fat transfer to the dorsum of the hand can also be attempted [29]. Patients should be forewarned that "cosmetic" procedures such as these are not always successful in the long term, as the transplanted tissue may become atrophic and resorb over time. With fat transfer, patients should also be counseled that several sessions might be required to achieve the desired effect.

Joint Contracture

Patients with end-stage ulnar neuropathy and claw hand may develop PIPJ flexion and/or MPJ extension contractures. Certainly therapy is the first-line treatment for regaining passive mobility of the joints. If therapy is unsuccessful, then options include surgical joint contracture release and/or arthrodesis. Many times monitoring patient's tolerance of therapy or compliance may help the decision for further intervention. Patients that do not tolerate or improve with therapy may not do well with staged procedures involving joint con-

tracture release followed by tendon transfers. In these patients, salvage procedures including arthrodesis may be optimal. The senior author (RJG) has found that patients with MPJ contracture with supple PIPJs do well with operative joint contracture followed in second-stage tendon transfer. If patients are contracted at both the MPJ and PIPJ levels, they generally do not respond well to joint contracture release.

MCP Joint Contracture Release

If all four MPJs are contracted, the approach for joint contracture release can be performed through two longitudinal incisions in the second and fourth web spaces dorsally. The extensor mechanism is splint midline and separated from the dorsal capsule. The capsule is opened transversely and can be resected if grossly thickened. The collateral recess is then separated. A freer elevator is then placed between the metacarpal head and proximal phalanx base, and the volar plate is released proximally. Manipulation will then often allow for full flexion. If the MPJ is hinging and not smoothly flexing, then release of the dorsal fibers of the collateral ligaments may be necessary. The hand is splinted in full MPJ flexion for 2 weeks; then intensive therapy is initiated. When full flexion is obtained and the soft tissues have adequately healed, then tendon transfers may be considered.

PIPJ Fusion

In patients with chronic claw hand deformity and joint contracture deemed to not be a candidate for contracture release and tendon transfers, PIPJ fusion into approximately 45 degrees of flexion may be a good option to regain the ability to grasp around larger objects. This option is ideal if MPJ mobility is maintained and flexion can be harnessed from the extrinsic flexors. The senior author (RJG) has employed this technique with favorable results (see Figs. 15.4 and 15.5).

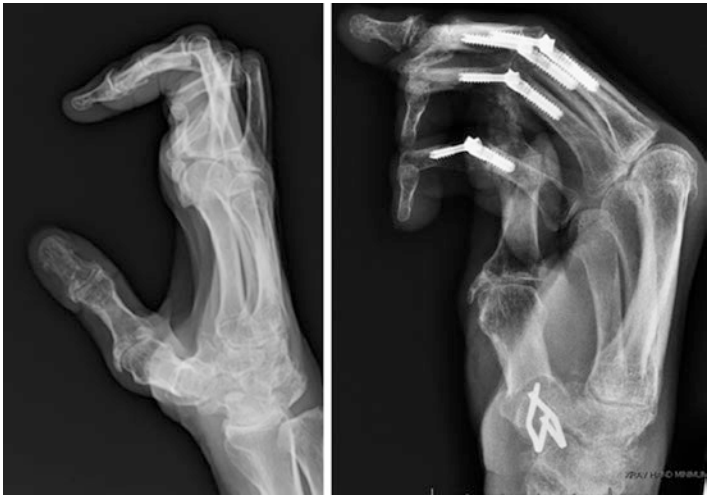

FIGURE 15.4 PIPJ fusion for chronic joint contracture: pre- and postoperative x-rays

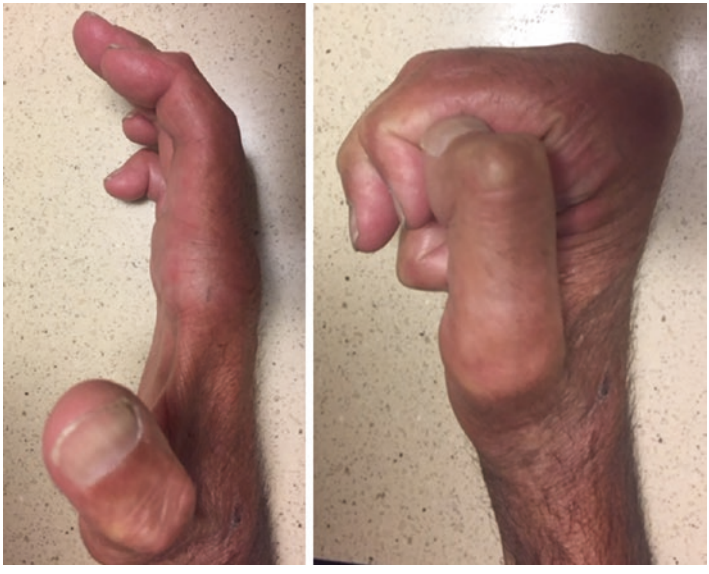

FIGURE 15.5 PIPJ fusion for chronic joint contracture: postoperative result

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