Nerve Compression: Ulnar Nerve of the Elbow

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Keywords

Ulnar nerve • Cubital tunnel • Compression neuropathy at elbow • Weakness in hand • Clumsiness in hand • Numbness or tingling in ring and small fingers • Elbow anatomy • Elbow examination • Treatment • Complications • Revision surgery

Introduction

Ulnar nerve compression neuropathy was first described by Panas in 1878 [1]. Feindel and Stratford initially described the significance of the 'cubital tunnel' and coined the term in 1958 [2]. Entrapment of the ulnar nerve is regarded as one of the most common compression neuropathies of the upper extremity, second only to carpal tunnel syndrome [2–4]. Although the nerve can be compressed at any location along its course, the most common location is at the elbow; and the most common site is at the cubital tunnel [5]. Diagnosis is made from a combination of history, examination findings, and provocative tests. Some authors still advocate the use of electrodiagnostic studies to confirm findings and localize the compression. Still, no standard exists for the surgical treatment of cubital tunnel that is refractory to conservative measures [6]. The available evidence at this point is insufficient to identify the best treatment technique [7]. Understanding the anatomy and pathology are critical steps towards successfully diagnosing and managing ulnar nerve compression regardless of the technique used.

Anatomy

The ulnar nerve is the terminal branch of the medial cord of the brachial plexus . It contains fibers derived from the ventral rami of C8 and T1 with occasional contribution from C7 (5–10 % of patients have a flexor carpi ulnaris motor branch from C7). The ulnar nerve enters the arm medial to the axillary artery and courses along the medial head of triceps and brachialis muscle eventually lying posteromedial to the brachial artery. It traverses the medial intermuscular septum posteriorly and passes through the Arcade of Struthers, a thickened fascia between the medial head of

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triceps and intermuscular septum located approximately 8 cm proximal to the medial epicondyle. The nerve enters the ulnar sulcus 3.5 cm proximal to the medial epicondyle.

At the elbow, the ulnar nerve continues posterior to the medial epicondyle and enters the cubital tunnel proper. The retrocondylar groove is the most common site of compression according to intraoperative electrical studies [8]. The roof of the cubital tunnel consists of a fibrous aponeurosis that thickens to form the cubital tunnel retinaculum also known as the 'arcuate ligament' or 'Osborne's Ligament [9].' Khoo et al. described the retinaculum as 4 mm wide and extending from the medial epicondyle to the olecranon [10]. The fibers are oriented in a transverse fashion and tension is dependent on elbow position (fibers are tightest in flexion). The retinaculum serves as the proximal-most roof of the tunnel and prevents anterior subluxation of the nerve with elbow flexion. The deep layer of the aponeurosis of the two heads of the flexor carpi ulnaris (FCU) muscle, also termed 'Osborne's fascia,' forms the distal roof of the tunnel. The floor of the cubital tunnel consists of the elbow capsule and the posterior and transverse components of the medial collateral ligament. The walls of the tunnel are the medial epicondyle and olecranon respectively.

Just before entering the cubital tunnel, the ulnar nerve gives off its first branch, which is thought by some to provide articular proprioception [11]. However, anatomic studies by Jabaley et al. and Watchmaker et al. have contested that articular branches, if present, are rarely found [12, 13]. Within the cubital tunnel, the ulnar nerve gives off multiple motor branches to the FCU and the ulnar half of the flexor digitorum profundus (FDP). An average of 3.4 motor branches to the FCU has been documented, with the majority branching on the ulnar side of the nerve [14].

After exiting the cubital tunnel, the ulnar nerve passes between the humeral and ulnar heads of the FCU. The ulnar nerve pierces the flexor pronator aponeurosis 3 cm distal to the cubital tunnel and travels deep to the FCU on the surface of the FDP. The flexor pronator aponeurosis may compress the nerve up to 5 cm distal to the medial epicondyle [15]. The branches of the ulnar nerve continue into the hand through Guyon's canal. Terminal branches of the nerve provide motor innervation to the hypothenar muscles, all the interossei, third and fourth lumbricals, adductor pollicis, medial head of the flexor pollicis brevis, and articular branches to the adjacent carpal joints. Terminal branches provide sensory innervation to the ulnar aspect of the palm, dorsal ulnar hand, small finger, and half of the ring finger.

Ulnar Nerve Blood Supply

The blood supply to the ulnar nerve was initially described in work by Sunderland [16]. Three main extrinsic arteries have been identified: the superior ulnar collateral artery, inferior ulnar collateral artery and posterior ulnar recurrent artery. Yamaguchi et al. demonstrated a consistent but segmental extraneural and intraneural vascular supply from these vessels [17]. Some authors, however, have questioned the clinical significance of the extrinsic blood supply to the ulnar nerve [18, 19]. Accurate characterization has implications for safe transposition. Lundborg and Sunderland showed that the integrity of the intrinsic microcirculation of peripheral nerves is critical for oxygen supply to individual nerve fascicles [20, 21]. Preservation of the proximal and distal intrinsic blood supply may allow its safe transposition but the clinical evidence awaits future study [22].

Medial Antebrachial Cutaneous Nerve Anatomy

Clear understanding of the position and anatomy of the medial antebrachial cutaneous nerve (MABCN) is crucial to avoid injury during surgery . Leffert was the first to demonstrate the importance of avoiding injury to MABCN during ulnar nerve decompression [23]. The MABCN is a direct branch of the medial cord (C8-T1) and descends in the arm anterior and medial to the brachial artery; it emerges from under the brachial

fascia adjacent to the medial epicondyle and olecranon to innervate the skin over the anterior and medial surface of the forearm [24]. Dellon and MacKinnon reported that a common cause of medial elbow pain following cubital tunnel surgery was injury to posterior branch of the MABCN. In their study, 23 of 25 patients with persistent symptoms following surgery for ulnar nerve decompression had evidence of injury to the cutaneous nerve [25]. Sarris et al. found similar findings in their cohort of 20 patients with recurrent symptoms following surgery [26]. Lowe evaluated 97 patients undergoing primary surgery for cubital tunnel to identify the proximity of MABCN branches. The authors found that branches lie on average 1.8 cm proximal to the medial epicondyle and 3.1 cm distal to the medial epicondyle [27]. They emphasized that knowledge of the anatomy can prevent iatrogenic injury to the MABCN and neuroma formation.

Avoiding injury to the MABCN can definitely improve the chance for clinical success in treatment regardless of surgical technique chosen [27, 28]. However, if injury is identified, persistent neuromas should be resected, electrocauterized and transposed deep into muscle [29–31]. An alternative treatment is to suture the nerve stump, end to side, into a functioning nerve. This may permit the nerve sprouts to incorporate into an intact nerve as opposed to forming a recurrent neuroma [32].

Clinical Pearl

Always look for, and preserve, branches of the Medial Antebrachial Cutaneous Nerve following skin incision

Sites of Potential Compression

The areas of compression in cubital tunnel syndrome have been well described in the literature. Sites of compression lie 10 cm proximal to and 5 cm distal to the elbow so it is imperative that the clinician understands the anatomy in this region. There are five potential sites of compression, including the Arcade of Struthers, the intermuscular septum, the area of the medial epicondyle, Osborne's ligament and Osborne's fascia.

The Arcade of Struthers and the intermuscular septum only appear to cause nerve compression following anterior transposition or with a nerve that subluxates anteriorly over the medial epicondyle during elbow flexion [33]. The septum and arcade must be released in these circumstances. The ulnar nerve can be compressed along the length of the Arcade which averages 5.7 cm. Hypertrophy of the medial head of the triceps and snapping of the triceps head are two more well-reported causes of compression in this region [33, 34].

The area of the medial epicondyle and epicondylar groove is thought by some authors to be the most common site of compression [8]. The nerve may be compressed by bone spurs in an individual with osteoarthritis [35]. Compression from a post-traumatic valgus deformity at the elbow can be another cause of cubital tunnel syndrome (Fig. 14.1).

Space occupying lesions such as ganglia, soft tissue tumors and hypertrophic synovium have the potential of impinging the nerve in this location [36, 37] (Fig. 14.2). The anconeus epitrochlearis is an anomalous muscle found outside the groove that is present in 3–28 % of cadavers [31, 38]. When present, the muscle is divided in conjunction with ulnar nerve decompression [39] (Fig. 14.3). Habitual subluxation or dislocation of the nerve from the groove makes the nerve more susceptible to injury as the nerve can become inflamed from repetitive friction over the medial epicondyle. Childress found a 16 % incidence of subluxation in his cohort of asymptomatic individuals. Patients with nerves that subluxed to the tip of the medial epicondyle were termed 'Type A,' while those with a nerve that subluxed beyond the medial epicondyle were 'Type B' [40]. The authors surmised that although asymptomatic, the position and hypermobility make the nerve vulnerable to harm.

The cubital tunnel proper was initially described by Osborne [9]. Its limited dimensions and volume make it a common cause of compression. The dynamic changes that occur

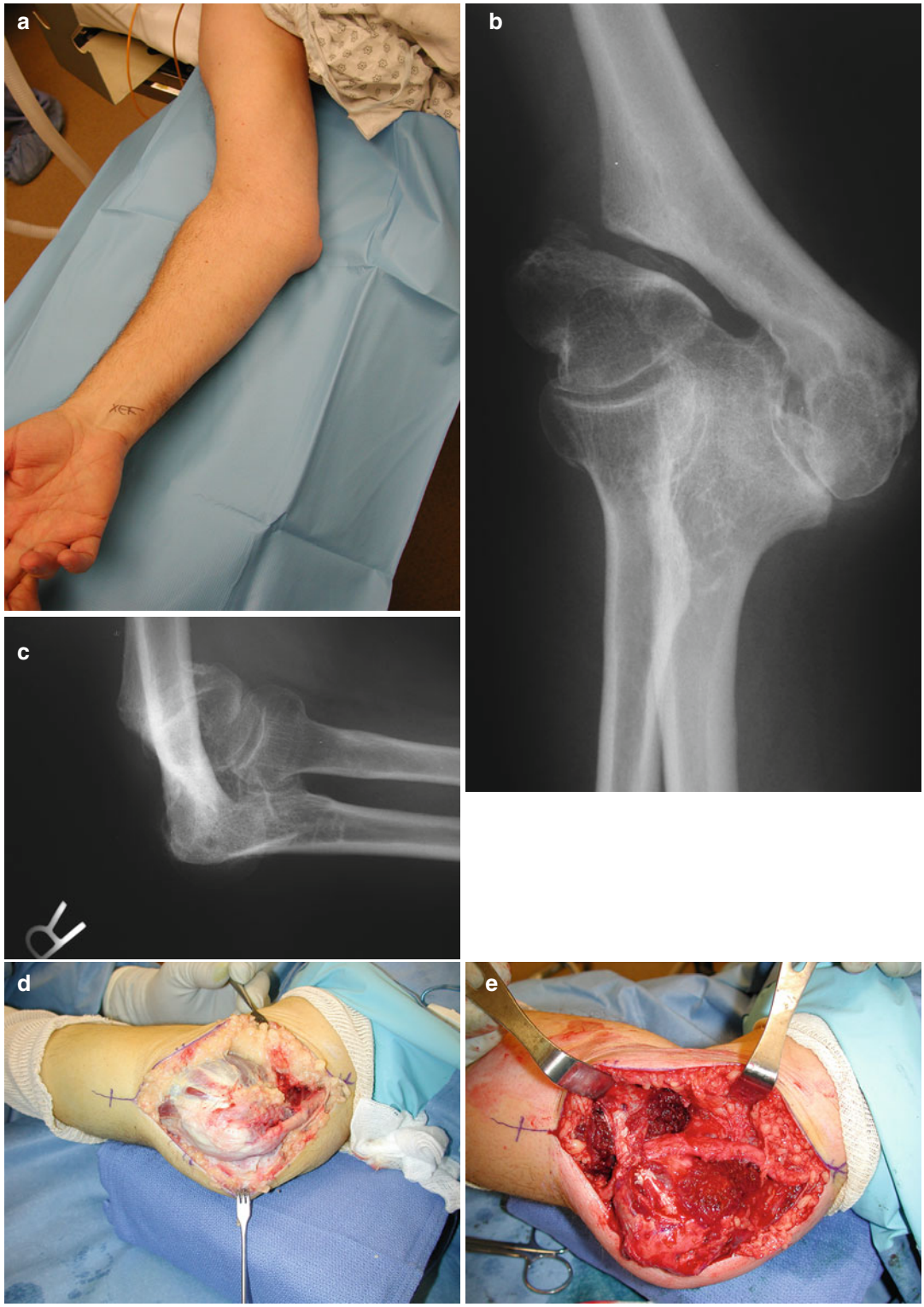


Fig. 14.1 Picture of a patient with a cubitus valgus deformity (**a**). AP (**b**) and lateral (**c**) radiographic images show a persistent traumatic valgus non-union. Intraoperative images before (**d**) and after ulnar nerve decompression and anterior transposition (**e**)

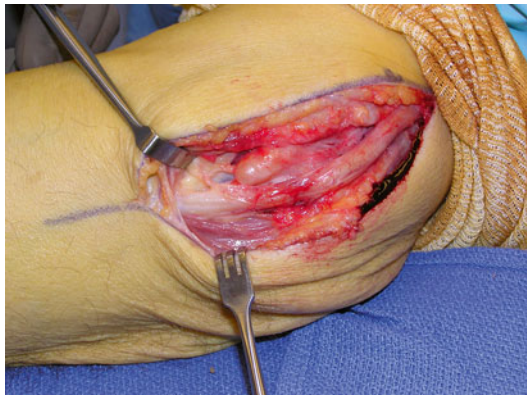


Fig. 14.2 Cyst located within the cubital tunnel is one example of a space occupying lesions (ganglia, osteochondritis, soft tissue tumors, hypertrophic synovium) that has the potential of impinging the ulnar nerve within the cubital tunnel

during motion can mechanically stretch the nerve and alter pressures within the tunnel, leading to ischemia and irritation of the ulnar nerve. Appelberg et al. described the dynamic anatomy of 15 cadaveric elbows in flexion and extension [4]. Flexion pushed the ulnar nerve anterior and medial secondary to the bulge of the medial head of the triceps during this motion. The nerve immediately adjacent to the epicondyle was found to stretch and elongate 4.7 mm during flexion. The roof of the tunnel plays a vital role in entrapment in the cubital tunnel proper. Thickening of Osborne's ligament and Osborne's fascia can cause direct nerve entrapment and are another critical structure to be released during surgical decompression.

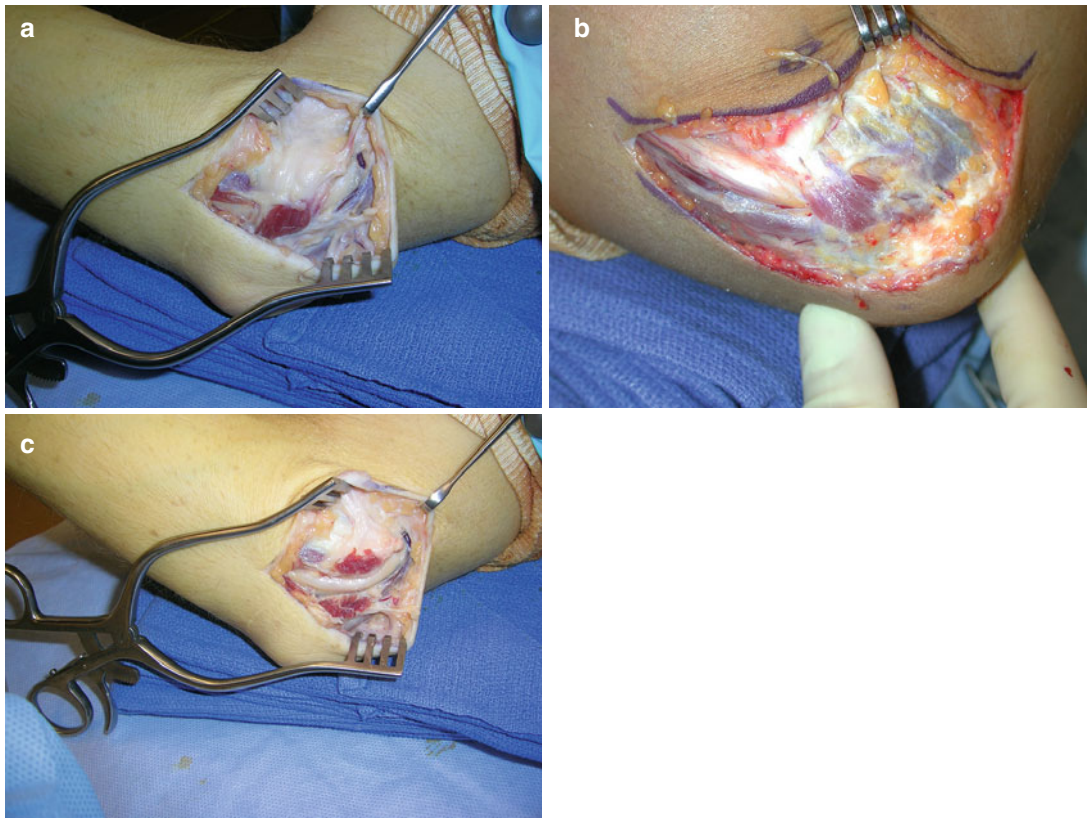


Fig. 14.3 The anconeus epitrochlearis is an anomalous muscle that is present in 3–28 % of cadavers. The clinician must be familiar with its appearance and aware of its compressive potential. (**a**, **b**) The anconeus epitrochlearis arises from the medial border of the olecranon and triceps and inserts onto the medial epicondyle. (**c**) Ulnar nerve following complete release of the anomalous muscle

After exiting the cubital tunnel, the ulnar nerve passes between the humeral and ulnar heads of the FCU. The flexor pronator aponeurosis may compress the nerve up to 5 cm distal to the medial epicondyle as mentioned above [15]. This area must be incised at the time of ulnar nerve decompression to ensure that the nerve is free at its most distal extent in cubital tunnel syndrome.

Karatsa et al. noted that the regional anatomic structures of the elbow show variability in the number and location of fibrous bands that have the potential to compress the ulnar nerve [41]. It is essential that during surgical release the nerve be released at all sites of compression from its proximal to distal end.

Clinical Pearl: Potential Sites of Nerve Compression

Arcade of Struthers between medial intermuscular septum and medial head of triceps

Cubital tunnel itself – Osborne's ligament

Osborne's fascia between heads of FCU

Pathology

The ulnar nerve can be subjected to traction and compression within the confines of the cubital tunnel with dynamic motion. Mechanical irritation and ischemia are thought to be factors associated with this disease process [4, 36, 42].

With elbow motion, associated changes occur to the shape and space of the tunnel. Bulging of the medial collateral ligament, tightening of the arcuate ligament or Osborne's ligament and firing of the FCU and medial head of the triceps are just a few of the changes observed. MRI studies showed that the tunnel is circular in shape and most spacious in extension. With flexion, the tunnel adopts a wider and flatter configuration. The tunnel becomes triangular or ellipsoid in flexion with a measurable height decrease of 2.5 mm. The cubital tunnel has been shown to narrow by 39–55 % with elbow flexion which places the nerve at increased risk for ischemia [4, 43].

Pechan and Julius found that the ulnar nerve is on maximal stretch with the shoulder abducted, elbow flexed and wrist extended [42]. In this setting, the ulnar nerve was observed to elongate 4.7 mm [4]. The authors found that the intraneural pressure of the cubital tunnel increases 600 % in this position. Wierich and Gelberman used MR and ultrasonographic imaging to measure the intraneural and extraneural pressure of the cubital tunnel in twenty cadaveric arms and found that intraneural pressure was significantly higher than extraneural pressure with the elbow flexed greater than 90° [43]. The tunnel pressure is on average 9 mmHg in extension. In flexion, the pressure increases to 63 mmHg; representing a seven-fold increase in pressure on the ulnar nerve.

Clinical Pearl

Ulnar nerve intraneuaral pressure increases sevenfold in elbow flexion

Perfusion studies in animals have shown that blood flow and axonal transport are affected by compression and stretch. Nerve damage is related to strain; lengthening of only 8 % has been shown to decrease neural blood flow in the sciatic nerve of rats and rabbits [44, 45].

There has been no scientific data to support particular vocational or avocational activities as causal risk factors for developing cubital tunnel syndrome. There are specific occupations that have been speculated to be associated with the diagnosis. Painters, carpenters, musicians, basketball players and tennis players all perform activities that involve repetitive elbow flexion. Other suggested risk factors include prior trauma, fracture, habitual subluxation and systemic disease [46].

Diagnosis

Diagnosis is typically based on history and examination. Sunderland studied the intraneural topography of the ulnar nerve as it courses within

the elbow and forearm [21]. He observed that the motor fibers that supply the intrinsics have a more superficial course compared with those that supply the FCU and FDP. He believed that this explained why intrinsic weakness of the hand is a common finding on presentation. Similarly, the sensory fibers of the ulnar nerve have a superficial location as the nerve traverses the elbow potentially explaining why paresthesias in the small and ring finger are another common finding.

The earliest sign of cubital tunnel syndrome is typically numbness and tingling of the ring and small finger. Some patients have difficulty localizing their symptoms. Patients may complain of medial elbow pain with radiation into the forearm [47]. Subjective motor loss, usually described as grip weakness, is a frequent complaint; this is especially true when torque is applied to a tool. Additionally, pinch weakness and difficulty with grasp can be involved. These complaints are usually related to intrinsic weakness. Early on, the same patients may exhibit clumsiness and difficulty with fine motor coordination. Sensory loss over the ulnar dorsal portion of hand helps differentiate between cubital tunnel syndrome versus compression of the ulnar nerve at Guyon's canal. The onset of symptoms and whether they are intermittent or constant can provide information about the chronicity and severity of disease. It can be helpful to determine if there is symptomatic worsening with the elbow in a flexed position, if the patient experiences night pain, and what relieves their symptoms.

Clinical Pearl

Sensory loss over the ulnar dorsal portion of hand helps differentiate between cubital tunnel syndrome versus compression of the ulnar nerve at Guyon's canal

Forced elbow flexion may increase symptoms with cubital tunnel compression

The presentation of cubital tunnel is sometimes indistinguishable from other disease processes. A careful history of comorbidities should be performed for thyroid disease, diabetes mellitus, haemophilia, acromegaly (Fig. 14.4), and peripheral neuropathy. One must have a high index of suspicion to rule out cervical radiculopathy, thoracic outlet, and compression at guyon's canal which can each be easily confused with cubital tunnel syndrome. Cervical root compression can present with neck pain, worsened with neck extension and ipsilateral rotation and improved with shoulder abduction. Patients with thoracic outlet syndrome complain of a vague shoulder ache with numbness along the medial forearm. Symptoms tend to worsen with overhead activities. Ulnar tunnel syndrome presents with wrist pain and, when associated with ulnar artery thrombosis, digital ischemia. Patients with ulnar artery thrombosis and ulnar nerve compression at the wrist have a history of repetitive wrist trauma and a positive Allen's test. A key distinguishing feature in patients with ulnar nerve compression at the wrist is the lack of dorsal hand numbness.

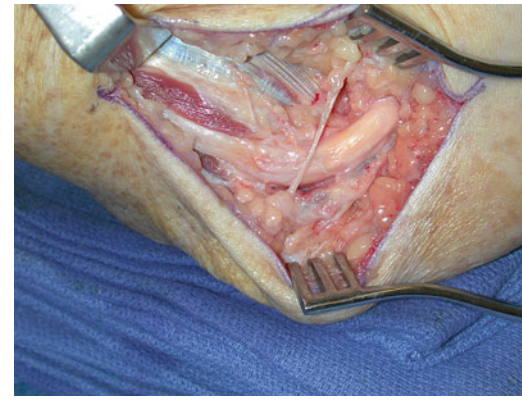


Fig. 14.4 Intra-operative image of an acromegalic ulnar nerve. Note the markedly enlarged ulnar nerve. Peripheral nerve enlargement seems to be an intrinisic part of the disease

The Double Crush phenomenon was initially described by Upton and McComas in 1973 [48]. It was hypothesized that impairment of axoplasmic flow at more than one site along a nerve can cause neuropathy. Moreover, compression at one site renders other sites more sensitive to compression. It is certainly possible that multiple sites within the cubital tunnel can be involved or

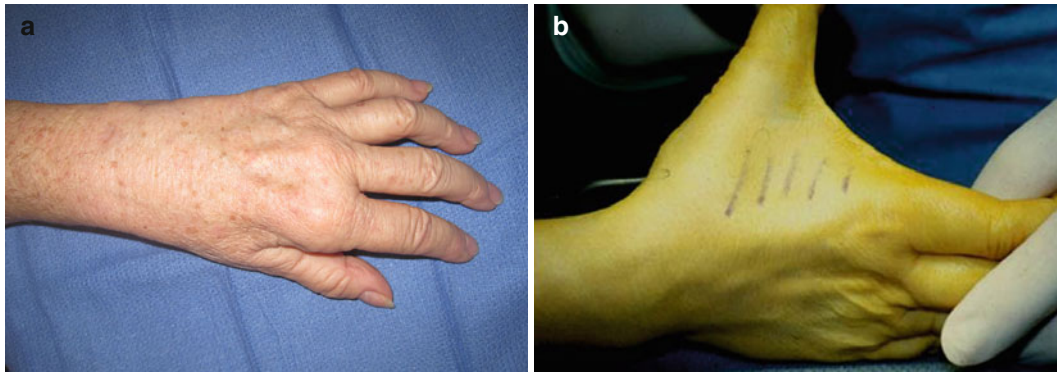


Fig. 14.5 Atrophy in patients with long-standing cubital tunnel is most evident at the dorsal first web space (**a**, **b**). This 'sunken in' region represents muscle loss of the first interosseous muscle

that local compression can be superimposed on proximal disease (cervical compression, thoracic outlet, etc.). A high index of suspicion is necessary to rule out other causes of neuropathy and physical examination should evaluate all potential sites of compression.

Sensory hypoesthesia within the ulnar nerve distribution can be objectively evaluated by Semmes-Weinstein or two-point discrimination tests. Motor symptoms present as either intrinisic or extrinsic weakness. Atrophy and clinical weakness is usually not seen for months to years following onset and subsequently can be an indicator of chronicity of symptoms. Atrophy is most evident at the dorsal first web space, representing loss of muscle volume in the 1st interossei muscle (Fig. 14.5). Mallette et al., however, noted that atrophy on presentation was four times more common in cubital tunnel than carpal tunnel [49]. As compression advances, loss of thumb adduction can present as Froment's sign, compensatory flexion at the IP joint of the thumb to aid in pinch against the index finger. Froment's sign confirms weakness of the adductor pollicis. Loss of adduction of the index and middle finger can be elicited with the 'crossed fingers test' representing weakness of the first volar and second dorsal interosseous muscles (Fig. 14.6). Wartenberg's sign reveals an inability to adduct the small finger, presenting as an ulnar deviated digit (Fig. 14.7). Wartenberg's confirms weakness of the third palmar interossei. A weak grip may be the result of denervation of the ulnar nerve innervated FDP to the small finger and ring finger. Advanced signs

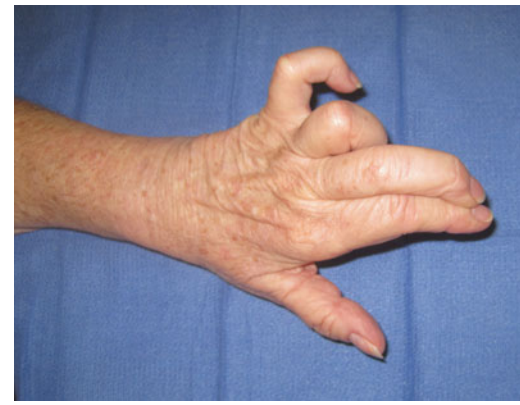


Fig. 14.6 Demonstration of loss of adduction of the index and middle finger secondary to ulnar nerve neuropathy. A positive 'crossed fingers test' represents weakness of the first volar and second dorsal interosseous muscles. There is also apparent 'clawing' of the ring and small finger

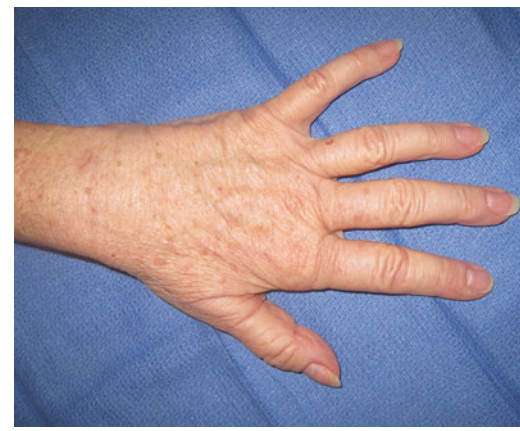


Fig. 14.7 Wartenberg's sign reveals an inability to adduct the small finger, presenting as an ulnar deviated digit. Wartenberg's sign confirms weakness of the third palmar interossei

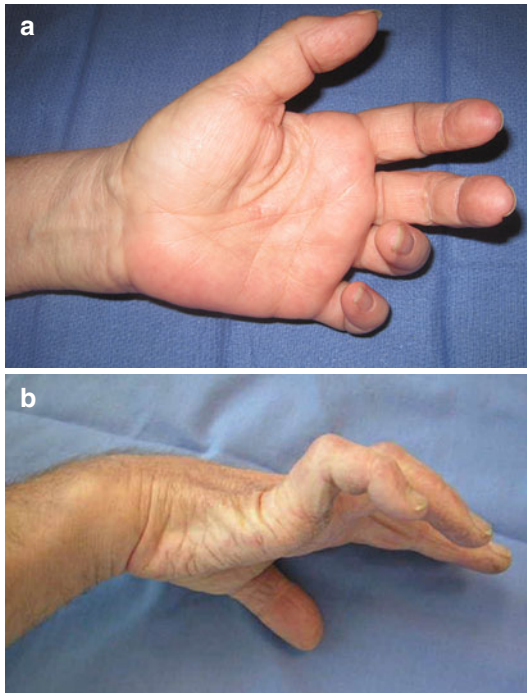


Fig. 14.8 Intrinsic minus posture, or ulnar clawing, of the ring and small finger secondary to weakness of the intrinsics (third and fourth lumbricals and interossei) (**a**, **b**)

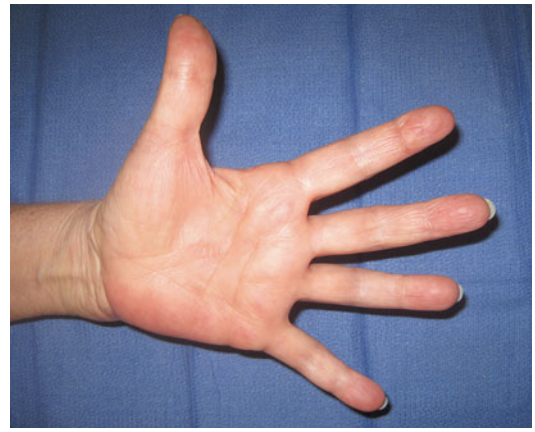


Fig. 14.9 Masse's sign is visible flattening of the hand secondary to loss of the dorsal transverse metacarpal arch combined with hypothenar muscle atrophy

of ulnar nerve compression include clawing (hyperextension of the metacarpophalangeal joint and flexion of the interphalangeal joint) of the ring and small finger secondary to weakness of the third and fourth lumbricals (Fig. 14.8). The claw hand is typically seen in conjunction with a low ulnar nerve lesion where the intrinsics are denervated and unable to flex the MP joints. With intact radial nerve innervated extensors the MP joints hyperextend. Since the FDP to the ring and small finger retain their innervation from the ulnar nerve in the forearm there is flexion of the PIP and DIP joints. Masse's sign is flattening of the hand secondary to loss of the dorsal transverse metacarpal arch and hypothenar atrophy (Fig. 14.9).

On examination the nerve may be palpable and tender at the retrocondylar groove of the elbow [50] Tinel's test involves gentle percussion of the nerve along its course in the retrocondylar groove. Percussion causes paresthesia along the ulnar nerve distribution. The test, however, produces a high number of false positives. Rayan et al. found a positive result in 23.5 % of normal volunteers [51]. Novak et al. described the sensitivities and specificities of a series of tests to diagnose cubital tunnel. A positive percussion test (Tinel's test) had 70 % sensitivity, 98 % specificity, 94 % PPV, and 87 % NPV [52]. The elbow flexion test is performed by flexing the elbow maximally with the forearm supinated and the wrist extended for 1–3 min. This maneuver is analogous to Phalen's test for carpal tunnel syndrome. A positive test reproduces symptoms in the ulnar nerve distribution. After 1 min of compression, the test was found to have 32 % sensitivity, 99 % specificity, 93 % PPV, and 74 % NPV. However, after 3 min of compression, the test was found to have a 75 % sensitivity, 99 % specificity, 97 % PPV, 89 % NPV [52]. Combining the elbow flexion test and pressure at the retrocondylar groove revealed a 98 % sensitivity, 95 % specificity, 91 % PPV, 99 % NPV after 1 min of compression. The scratch collapse test is more recently described by Cheng et al. [53]. The elbow is flexed at the patient's side and the examiner resists external rotation of the shoulder while the area overlying the ulnar nerve is scratched. A temporary loss of resistance is a positive test. Cheng et al. reported 69 % sensitivity, 99 % specificity, 99 % PPV, 86 % NPV.

Imaging is not a frequent part of the work-up for ulnar nerve compression neuropathy, but in some instances it can be useful. Plain radiographs

can identify post-traumatic malunion, heterotopic bone and arthritis. St John and Palmaz found that radiographs displayed abnormalities in 20–29 % of patients with cubital tunnel (versus 6 % in the control group) [54]. Imaging studies should be ordered on an individual basis for patients that display risk factors identified by history and physical examination.

Electrodiagnostic studies can be performed to confirm diagnosis, localize compression, and rule out other disease processes (cervical compression, upper motor neuron disease, thoracic outlet, peripheral neuropathy, etc.). EMG studies evaluate the function of larger myelinated nerve fibers that are vulnerable to compression. Fibrillations and sharp waves reveal whether axonal degeneration has occurred. The first dorsal interosseous muscle is the most commonly affected. The abductor pollicis brevis (T1) should be examined to exclude a C8-T1 nerve root or inferior brachial plexus lesion. Concerns about electrodiagnostic studies exist because just a few normally functioning nerve fibers can lead to an artificially normal result. Greenwald et al. and others believe electrodiagnostic testing is unnecessary in predicting surgical outcomes [55–58].

Absolute slowing of nerve motor conduction velocity at the elbow of <50 m/s supports the diagnosis of cubital tunnel syndrome [59]. Decreased conduction velocity of more than 10 m/s from regions above and below the elbow, decreased amplitude of more than 20 % (Green's), and absence of sensory responses or evidence of muscle atrophy are highly suggestive of cubital tunnel disease [60]. Double crush phenomenon may be detected with F-wave indicating cervical compression or thoracic outlet syndrome [61].

It is postulated that compression induces endoneurial oedema, demyelination and remyelination, inflammation, fibrosis, distal axonal degeneration, growth of new axons, and thickening of the perineurium and epineurium [62]. Ultrasound is a relatively new study that has been used to identify these changes. Wiesler evaluated 15 elbows with cubital tunnel confirmed by clinical exam and NCS and compared them with a control group of 60 elbows from normal volunteers [63]. There was a strong correlation with cubital tunnel and an increase in the cross-sectional area of the nerve. The average cross sectional area was 0.065 cm^2 in controls vs 0.19 cm^2 in the ulnar cubital tunnel group. Pearson coefficient between motor nerve conduction velocity of the ulnar nerve and cross sectional area was 0.80. A cut-off point of cross sectional area of 0.10 cm^2 or higher yielded a sensitivity of 93 % (14/15 elbows) and a specificity of 98 % (59/60 elbows) with a PPV 93 %, and NPV 98 %. The authors caution that greater standardisation is required [63]. Ultrasound can also be useful to detect compression due to an anconeus epitrochlearis [64] ganglion [65], or nerve subluxation [66].

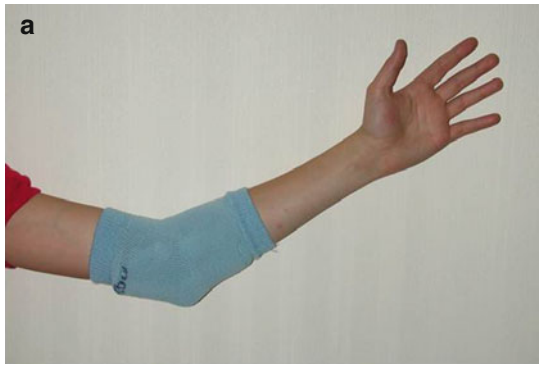
Staging

In 1950, McGowan developed a classification system for ulnar nerve compression based on the severity of motor deficit [67]. Lesions classified as 'grade I' displayed no muscle weakness where 'grade II' lesions exhibited partial weakness and 'grade III' lesions exhibited severe weakness and atrophy. Sensory findings were later introduced into the staging classification in 1988.

Dellon and Amadio created a sophisticated rating system to assess nerve function using a scale from 0 to 10, with 10 representing severe disease with evidence of muscle atrophy [68]. Kleinman and Bishop formulated a 12-point grading scale incorporating objective data and patient reported outcomes [69]. Unfortunately, the complexity of these classification systems limits their clinical utility. Most clinicians utilise a system of involved motor and sensory manifestations to determine severity of nerve compression.

Non-operative Treatment

Non-operative treatment is appropriate for mild to moderate symptoms and includes activity modification and patient education. The patient is taught to avoid activities that result in sustained increased stretch and pressure on the



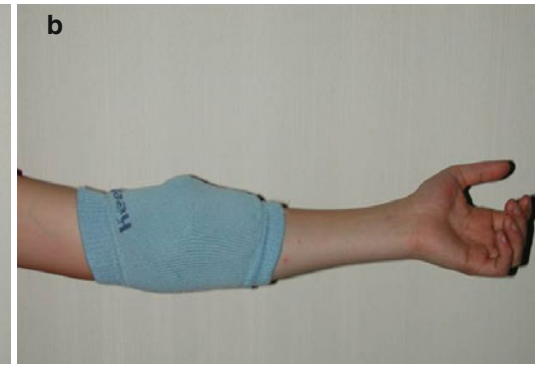


Fig. 14.10 Soft elbow pads (**a**, **b**) are a staple part of non-operative treatment for cubital tunnel and are worn during the day and night to limit flexion to 45–70°. Activity modification, patient education and stretching of the flexor carpi ulnaris muscle are other commonly applied techniques of conservative care

nerve. The patient should be counseled to consider avoiding actions that require the elbow to be fully bent for long periods of time and to avoid resting the elbow on hard surfaces. Soft elbow pads are worn during the day and night splints are provided to limit flexion to 45–70° (Fig. 14.10). Patients are instructed in exercises that stretch the FCU and to avoid sleeping with a flexed elbow. Dimond and Lister reported an 86 % improvement of symptom severity in 73 patients who underwent splinting during an average 8.7 months [70].

Svernlov et al. followed patients with mild to moderate cubital tunnel syndrome who were treated non-operatively [71]. Patients were divided into three groups based on the method of treatment offered (night splints, nerve gliding exercises, or education and activity modification). The authors observed that 89.5 % of patients improved regardless of group. Dellon et al. prospectively studied 121 patients treated non-operatively for a minimum of 3–6 months [72]. The authors evaluated the patients that went on to need surgical treatment. Only 21 % of patients with mild symptoms went on to require surgery within 6 years. Thirty-three percent of the patients with moderate symptoms required surgery within 3 years, while 62 % with severe symptoms required surgery over the same time frame. The authors found that symptom severity was highly correlated with surgical intervention.

Operative Treatment

Operative treatment is chosen for those who failed non-operative treatment and present with weakness, atrophy, and significant denervation on electrodiagnostic studies. Tomaino et al. suggested that subjective symptoms of cubital tunnel syndrome alone warrant operative intervention [56]. The authors argued that electrodiagnostic studies and objective symptoms (atrophy, weakness, loss of two-point discrimination, etc.) reflect a more advanced state of ulnar neuropathy. Advanced disease is associated with potentially irreversible intraneural changes raising the risk for incomplete recovery. In their prospective study, surgical treatment (in situ release and medial epicondylectomy) was offered to patients with McGowan I electrodiagnostic-negative cubital tunnel syndrome who did not benefit from initial implementation of non-operative treatment. Sixteen patients (18 elbows) were enrolled in the study. All patients had complete relief of symptoms following in situ release and medial epicondylectomy and 17 of 18 elbows had return of normal range of motion and grip strength. The authors found that surgery has favorable outcomes and low morbidity in this patient population.

Principles of surgical decompression include release of all sites of potential entrapment, protection of the MABCN, creating a straight path for the nerve to follow if transposed, haemostasis,

and early elbow motion. Treatment options involve simple decompression, decompression with anterior transposition (subcutaneous, intermuscular, submuscular), partial and complete medial epicondylectomy, and endoscopic release. There is controversy in the current literature over which procedure is optimal. The choice depends on surgeon preference as meta-analyses and review of the literature has consistently shown little difference between procedures [6, 73–75]. Absence of consensus on nerve-specific outcome measures limits the ability to design convincing randomised trials for comparison [76].

Macadam et al. performed a recent meta-analysis of ten studies [75]. Analysis was limited to randomised controlled trials and comparative observational studies. The authors compared outcomes of in situ decompression versus decompression and anterior transposition. Over 449 simple decompressions were compared with 457 transpositions for cubital tunnel syndrome. Odds of improvement with simple decompression versus anterior transposition were 0.751, 95 % confidence interval (0.542, 1.040). No difference in clinical outcomes was found between the techniques. Subanalyses on the basis of transposition technique (submuscular versus subcutaneous) showed no statistical differences.

Surgical Technique

In Situ Decompression

In situ decompression was first described by Osborne in 1957 [40]. The technique is performed with a 3–4 cm curvilinear incision centered over the course of the ulnar nerve between the medial epicondyle and the olecranon (Fig. 14.11). Supple skin allows easy subcutaneous dissection. The ulnar nerve is identified proximal to the ligament of Osborne. Passage is created above the nerve proximally and distally. With a deep retractor, the nerve is released approximately 6 cm proximal (Fig. 14.12) and

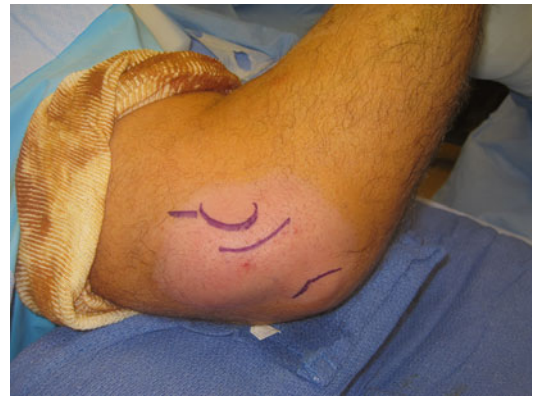
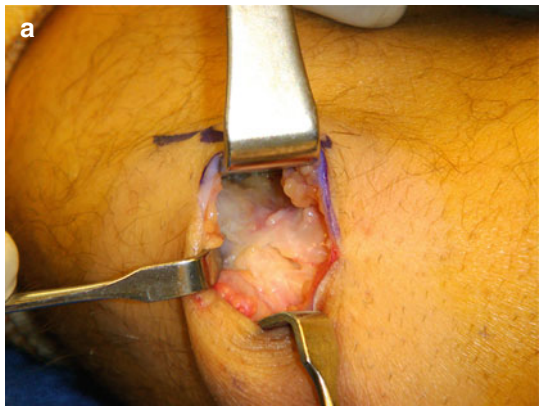


Fig. 14.11 In situ decompression utilizes a 3–4 cm curvilinear incision centered over the course of the ulnar nerve between the medial epicondyle and the olecranon. Supple skin allows easy subcutaneous dissection proximal and distal



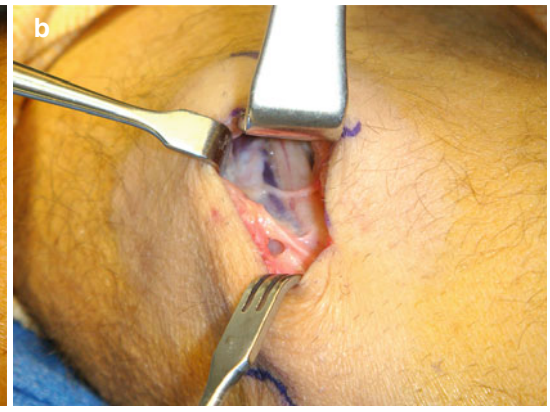


Fig. 14.12 In-situ ulnar nerve decompression preserves the bed of the nerve proximally (shown) and distally. Dissection through the fascia proximal to the medial epicondyle (**a**) is performed to protect branches of the Medial Antebrachial Cutaneous Nerve (MABCN). Complete release of the ulnar nerve proximally with preservation of branch of the MABCN (**b**). Circumferential dissection is not performed as part of this technique

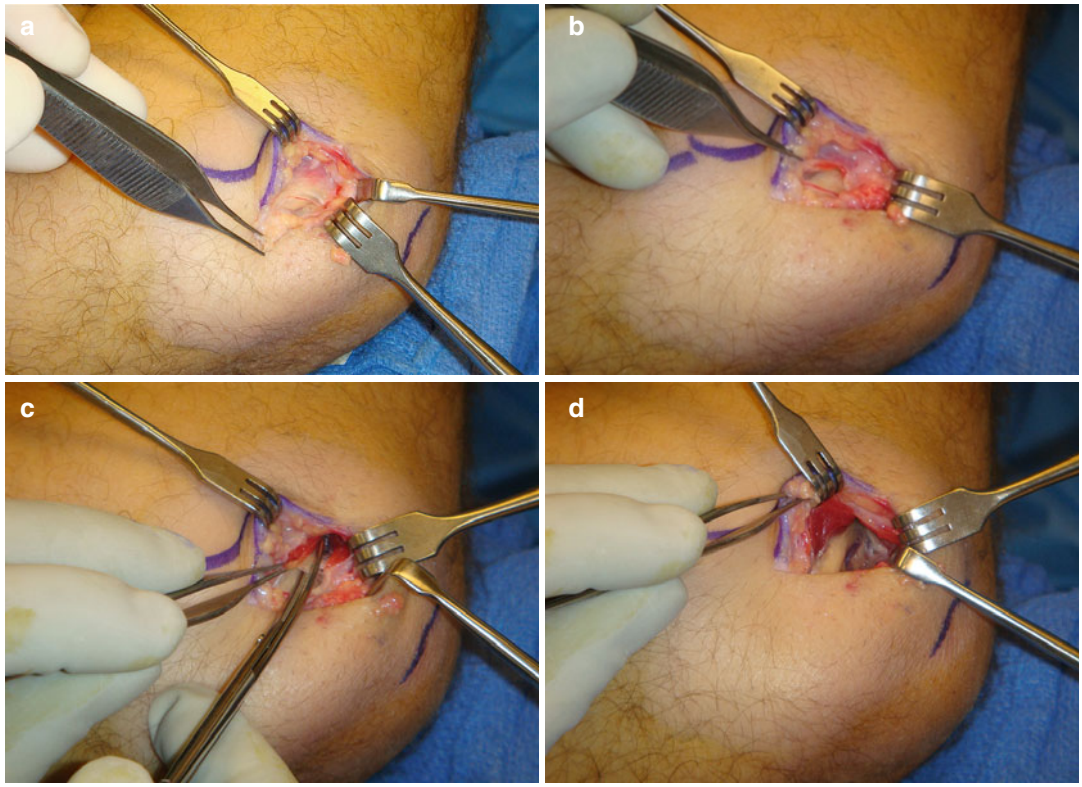


Fig. 14.13 The ulnar nerve distally is identified entering the cubital tunnel beneath the ligament of Osborne (**a**). The ligament is released and dissection is carried up to the two heads of the flexor carpi ulnaris (FCU) (**b**). The deep layer of the aponeurosis between the ulnar and humeral heads of the FCU, termed 'Osborne's fascia', is incised next (**c**). Complete distal release of the ulnar nerve following in situ decompression is shown (**d**)

distal (Fig. 14.13) to the epicondyle until the nerve is surrounded by healthy appearing fat. The approach, like all the methods to be described, takes care to protect branches of the MABCN. Circumferential dissection is not performed as part of this technique [77]. After decompression, the ulnar nerve should be observed through a full range of motion. The nerve should be lax or even redundant in full extension and should remain within the cubital tunnel during elbow flexion (Fig. 14.14). If the nerve subluxates when the elbow is flexed, it should be transposed anteriorly.

Relative contraindications for simple decompression include severe cubitus valgus [6, 78, 79], a subluxing ulnar nerve, advanced compression and recurrence following a previous surgery [6, 68, 80]. There is, however, some controversy over the best treatment for advanced compression. Some have suggested that simple decompression for patients with severe symptoms can yield good to excellent results [7, 81, 82].

The post-operative management involves little or no immobilization. Sling or bulky dressing is provided for the first few days to allow the wound to settle. Immediate return to activities of daily living is allowed as tolerated by the patient, however, heavy lifting is restricted until 4–6 weeks postoperatively.

In situ decompression appears to equal results obtained after anterior transposition [83–85]. Proponents of simple decompression favour its relative simplicity. Another advantage is that it does not influence blood supply [85, 86]. The reported success rate is between 80 and 92 % [8, 87–89].

Bartels prospectively compared simple decompression with anterior subcutaneous

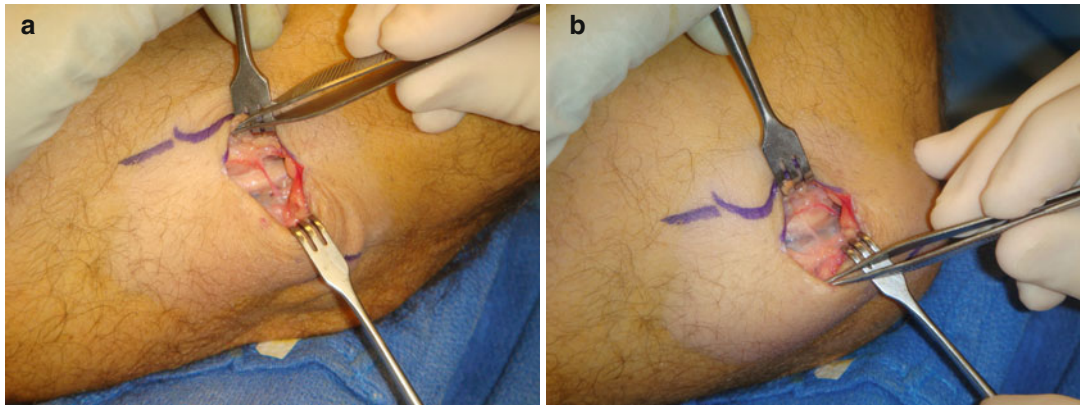


Fig. 14.14 After decompression, the ulnar nerve should be observed through a full range of motion. The nerve should be lax or even redundant in full extension and should remain within the cubital tunnel during elbow flexion. The intraoperative pictures show the position of the ulnar nerve with the elbow in extension (**a**) and flexion (**b**). There is no anterior subluxation with the elbow in a flexed position following decompression

transposition [90]. 100 and 52 patients were followed for an average of 1 year. Outcomes were equivalent. A higher complication rate was observed within the transposition group. A total of 30 complications were observed. Twenty-three complications were found in the anterior transposition group and only seven were found in the simple decompression group. The most common complication was loss of sensation around the surgical scar. Cost analysis found simple decompression to be less expensive. Nabhan followed the results of 66 patients who were randomised to treatment by simple decompression or subcutaneous anterior transposition [85]. No statistical significant difference was found in outcomes (pain, motor, sensory, NCV). The authors recommended simple decompression, describing it as a less invasive procedure.

Anterior Subcutaneous Transposition

The first ulnar nerve transposition was reported by Curtis in 1898 [91]. The goal of transposition is to move the nerve anterior to the elbow axis of flexion and thus create a straight path for the nerve, which decreases tension and presumably enhances neural blood flow. Anterior transposition removes the nerve from the volume limited cubital tunnel and away from sites of mechanical irritation. The basic principles of decompression are the same regardless of technique.

Transposition of the ulnar nerve requires a longer skin incision than simple decompression. The medial intermuscular septum is identified, dissected free and resected. The plexus of veins associated with the posterior surface of the septum must be coagulated. Approximately 4 cm of the septum is excised, beginning at the medial condylar ridge and extending proximally. Particular attention is paid to the proximal and distal ends of the nerve after transposition and care is taken to avoid sharp bends (especially at the FCU). A fascial flap based in proximity to the medial epicondyle can be created. The ulnar nerve is lifted and transposed anterior to the condyle and is held by the fascial sling. Motor branches to the FCU and FDP are preserved. The senior author prefers to create a trough in the distal 25 % of the flexor pronator mass to ease the bend. This eliminates the need for a fascial flap. Additionally, incision of the posterior septum of the humeral head of the FCU allows the nerve to follow a more direct path (Fig. 14.15).

Rehabilitation is similar to in situ decompression. The arm is protected in a bulky dressing and sling for 2 days followed by return to activities of daily living. Weirich studied 36 patients with cubital tunnel syndrome that underwent anterior subcutaneous transposition [42]. Twenty patients

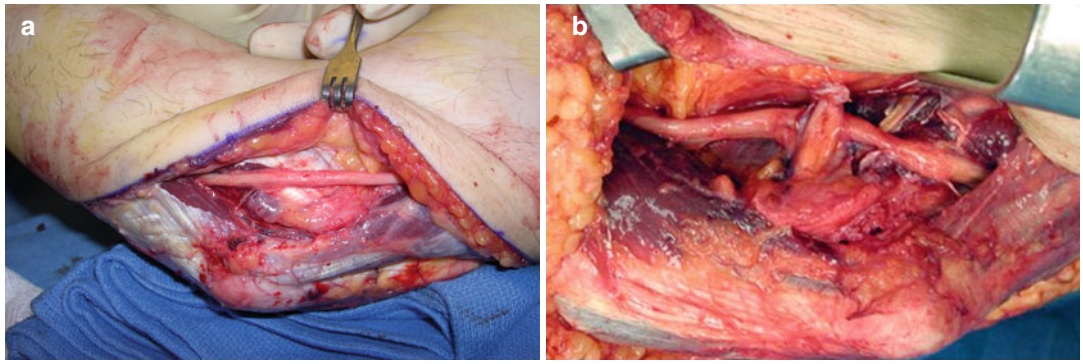


Fig. 14.15 Anterior subcutaneous transposition creates a straight path for the nerve (**a**), which decreases tension and enhances neural blood flow. Particular attention is paid to the proximal and distal ends of the nerve after transposition and care is taken to avoid sharp bends. The ulnar nerve is lifted and transposed anterior to the condyle and is held by the fascial sling (**b**)

underwent immediate post-operative mobilisation and began active range of motion exercises on the day of surgery. Sixteen patients were immobilised (well-padded plaster splints with elbows flexed at 90°) for an average of 14.4 days before beginning active and active-assisted range of motion exercises. Quantitative outcomes were not significantly different between groups (grip strength, pinch, two-point discrimination) at final follow-up. Both groups had improvement in first dorsal interosseous and adductor pollicis muscle strength. However, the immobilised group returned to work later than the mobilised group (2.75 months versus 1 month).

Those that are critical of transposition have concerns about its new superficial location and the extensive dissection required. Circumferential dissection has the potential to decrease blood supply to the nerve [92]. Placing the nerve in a subcutaneous location potentially exposes it to trauma and injury. Because of these concerns, we try to avoid anterior subcutaneous transposition in slender patients with little subcutaneous fat about the elbow.

Richmond followed 18 patients after subcutaneous transposition and observed good to excellent results in 83 % of their patients at mean follow-up of 23 months [93]. Rettig reported good results in athletes that underwent anterior subcutaneous transposition [94]. In his retrospective review of 20 athletes, all returned to full activity and reported minimal to no symptoms following surgery. Similar results were found in other studies [95–97].

Intramuscular Transposition

Intramuscular transposition was initially described by Adson in 1918 [98]. The technique for decompression of the nerve is the same as with other forms of transposition with special attention to the proximal and distal ends of the transfer. Once the nerve is mobilised over the top of the flexor-pronator, a 5–10 mm trough is made in the flexor pronator mass along the course of the nerve in its anterior location [99]. The nerve is placed in the trough. The fascia is repaired with the elbow flexed and the forearm fully pronated. Care is taken to not allow the repaired muscle to compress the nerve. Alternatively the fascia can be left unrepaired. Anecdotally, we have seen no adverse consequences of placing the nerve in a shallow trough in the flexor pronator mass and not repairing the flexor pronator fascia. Following transposition, the nerve should glide freely within the new intramuscular tunnel.

The advantage of intramuscular transposition is that it requires less dissection than submuscular transposition and allows the nerve to follow a straight path. It potentially removes the nerve from a vulnerable subcutaneous position as with subcutaneous transposition. Kleinman and Bishop found this procedure to be simple and

reliable with 87 % good or excellent results in their cohort of 47 patients followed for an average of 28 months [69]. Concerns exist regarding formation of cicatricial scar, although less so than potential scarring following submuscular transposition [23, 100]. Some authors feel the nerve may be vulnerable to traction forces when the flexor pronator muscles contract and have expressed concern that muscle division can lead to post-operative haematoma [32].

Kleinman recommended immobilizing the elbow in a bulky long arm dressing for 3 weeks in mid-pronation and 90° of flexion [101]. Dellon argued that earlier mobilisation can avoid the complication of fibrosis and scar [102].

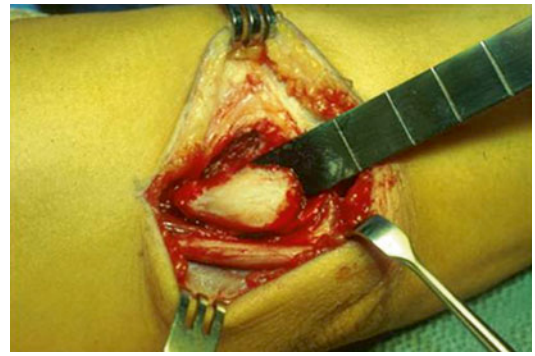


Fig. 14.16 Medial epicondylectomy is performed along the posterior third to half, leaving behind a ridge of bone to protect the attachment of the ulnar collateral ligament. After the osteotomy is made, the flexor pronator mass is repaired

Submuscular Transposition

Submuscular transposition was initially described by Learmonth in 1942 [103]. After the ulnar nerve is decompressed, the entire flexor-pronator mass is detached. A cuff of tissue is left behind for repair to its original position. Dellon offered a modification of the original technique in his description of a Z-lengthening of the flexor pronator fascia [104]. This technique eliminates pressure from submuscular placement of the large diameter nerve. Once again, complete excision of the intramuscular septum proximally and dissection distally to free the ulnar nerve is imperative to prevent potential sites of "kinking" or compression. The ulnar nerve is placed completely beneath the flexor pronator mass and repair of muscle is performed over the transposed nerve [77].

Concerns about submuscular transposition involve the extensive dissection and potential of creating a new site of compression. The need for immobilisation following this procedure has been questioned by some because of the potential for scaring and fibrosis. A study by Weirich compared postoperative immobilisation with early motion [42]. No impairment in outcomes was found between the two groups. However, early mobilisation may allow an earlier return to work as mentioned previously [42, 105, 106].

Biggs et al. prospectively reviewed 44 patients in a randomised series comparing in situ release versus submuscular transposition [83]. In their series, 61 % of patients improved with in situ release and 67 % improved with submuscular transposition. There was a high rate of infection in the submuscular transposition group compared with simple decompression (14 % deep infection rate for the submuscular transposition group versus no infections in the in situ group). Recent studies have confirmed that there are no differences in outcomes between in situ decompression and submuscular transposition [107]. Two recent meta-analyses of the literature determined no difference between simple decompression and anterior transposition of any kind [74, 75].

Medial Epicondylectomy

Medial epicondylectomy for ulnar nerve palsy was first described by King in 1950 [108]. Since its initial description, modifications have been made to the technique. The flexor pronator mass is incised longitudinally and the medial epicondyle is exposed subperiosteally. Partial medial epicondylectomy is performed along the posterior third to half, leaving behind a ridge of bone to protect the attachment of the ulnar collateral ligament. After the osteotomy is made, periosteum over the bone is repaired. A near-complete epicondylectomy can be performed just to the site of insertion of the medial collateral ligament (Fig. 14.16).

Rehabilitation following medial epicondylectomy is the same as all other forms of ulnar nerve

decompression. The elbow is protected in a sling and bulky dressing for 2 days followed by return to activities of daily living. The criticisms associated with medial epicondylectomy include instability from injury to the ulnar collateral ligament, medial elbow pain, and weakness from muscle detachment. When a small portion of epicondyle is preserved, instability is uncommon [109, 110]. Heithoff reported a 10 % loss of grip strength and 5 % loss of pinch strength at average follow up of 2.3 years following epicondylectomy [58]. Medial elbow pain was also noted in 10 % of the patients in his cohort. Despite these results, a recent retrospective review comparing medial epicondylectomy to anterior subcutaneous transposition revealed no differences in outcomes between the two techniques [111].

Endoscopic Release

Endoscopic cubital tunnel release was first described by Tsai in 1995 [112]. The technique offers a minimally invasive alternative to open surgical decompression. It has been suggested that the limited soft tissue dissection will result in shorter recovery time and less scarring [113]. Many variations exist, but most involve a small incision at the condylar groove [112, 114–117]. Space is made between the nerve and fascial covering and overlying subcutaneous adipose tissue. The endoscope and tenotomy scissors are used to release constricting fascial bands over the nerve.

Ahcan et al. published the findings of 36 patients with cubital tunnel that underwent endoscopic release [114]. According to the authors, decompression of 20 cm of nerve was performed through a 3.5 cm incision. Postoperatively, all patients showed improvement by electrodiagnostic testing. Excellent and good results were obtained in 33 out of 36 patients. One complication of a postoperative haematoma that subsequently resolved with no residual symptoms was reported. Hoffmann described similar findings in a cohort of 75 patients (76 endoscopic decompressions) [115]. They found that symptomatic improvement occurred in 96 % of patients and that NCV/NCS improved in all patients. Four patients developed postoperative haematomas that resolved and nine developed numbness in the MABCN distribution (8 out of the 9 improved within 3 months). Watts noted that the endoscopic technique caused less pain and provided greater satisfaction [118].

Cobb et al. examined recurrence of symptoms following 134 consecutive cases of endoscopic cubital tunnel release [113]. The authors observed a recurrence rate between 0.02 and 5.24 %. They noted that endoscopic cubital tunnel release has a recurrence rate that is not higher than open cubital tunnel release literature controls (0.02–5.24 % versus 12 % following open decompression).

Jiang et al. modified the endoscopic technique by using carbon dioxide insufflation to accomplish a subcutaneous dissection anterior to the flexor-pronator mass. In doing so they were able to perform a endoscopic-assisted subcutaneous transposition of the ulnar nerve. All 12 patients had improvement of their symptoms and 10 of 12 were rated as having an "excellent" outcome [119].

While the evidence is anecdotal, it is our opinion that the endoscopic technique carries a higher risk of injury to the ulnar nerve as it is the only technique for ulnar nerve decompression where we have seen or heard reports of ulnar nerve transection. As with any new technique there is a steep learning curve.

Clinical Pearl: Author's Recommended Surgical Approach

Simple in-situ release
Ensure branches of MABCN seen and preserved
Force flexion of elbow to ensure no subluxation
Bulky bandage
Sling for 48 h
Unrestricted use after 2–3 weeks

Failed Operative Treatment

Failure following cubital tunnel release can be defined as the inability to relieve preoperative symptoms or worsening of symptoms postoperatively

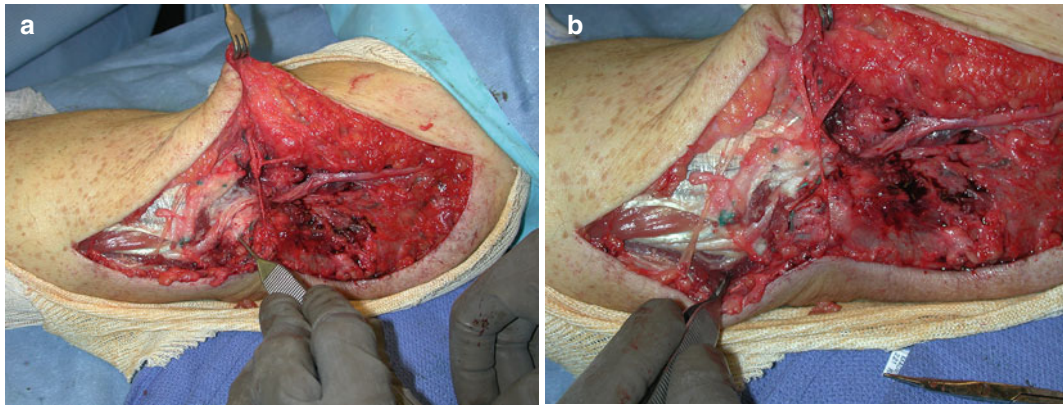


Fig. 14.17 Failed submuscular transposition. Dense cicatricial scar has developed following surgery and is serving as a new site of compression (**a**, **b**)

[28, 120]. Recurrence has been defined as return of symptoms after a 3 month period of resolution following surgery [120, 121]. Reasons for failure and recurrence are multifactorial and are best managed by determining if they have occurred as a result of preoperative, intraoperative or postoperative factors.

Preoperatively, the diagnosis of cubital tunnel can be confused with other sources of nerve compression including cervical radiculopathy, thoracic outlet, and ulnar tunnel syndrome. Patient expectations are another factor that can lead to an unsuccessful outcome. Those patients with long standing cubital tunnel, advanced compression, older patients and those with co-morbidities such as diabetes should be counseled that relief following surgery may be partial or incomplete.

Intraoperative factors that can lead to residual symptoms include inadequate decompression, failed transposition (Fig. 14.17), injury to the MABCN (Fig. 14.18), and residual ulnar nerve instability (Fig. 14.19). Rogers noted that recurrent symptoms are often attributed to incomplete decompression or scar [122]. Injury to a branch of the medial antebrachial cutaneous nerve can occur during exposure. The posterior branch of the MABCN is encountered with some frequency and injury will result in neuroma formation and pain. During in situ decompression, the ulnar nerve should be released but not destabilized causing residual ulnar nerve instability. Transposition must be carefully planned and performed to avoid placing the nerve in a location of injury and to prevent making a new site for compression.

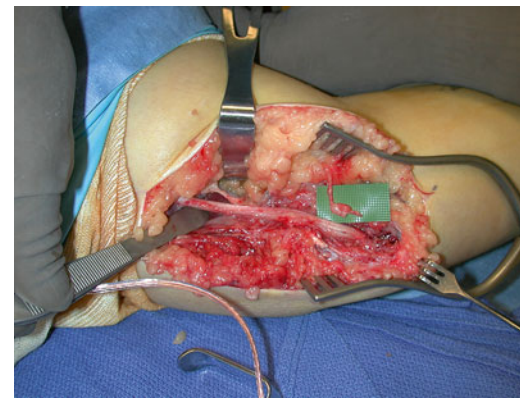


Fig. 14.18 Medial antebrachial cutaneous nerve neuroma following cubital tunnel surgery. Picture taken at time of revision surgery

Dellon and Mackinnon et al. thought the most important factor to obtain successful results after transposition was attention to releasing structures proximal and distal to the region of transfer [102]. Analysis in a primate model showed no evidence of scar after submuscular or intramuscular transposition 3 months following surgery. Broudy reviewed ten patients who had persistent or recurrent symptoms following transposition of the ulnar nerve [100]. Five patients underwent subcutaneous anterior transposition. The ulnar nerve was found surrounded by dense muscular

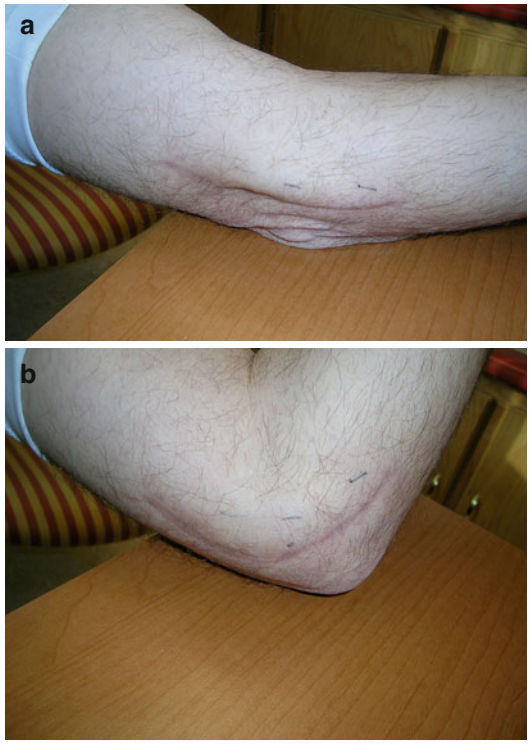


Fig. 14.19 Failed subcutaneous transposition evidenced by residual ulnar nerve instability – subluxation of the ulnar nerve over the medial epicondyle in extension (**a**) and flexion (**b**). Dark dashes on the skin represent the location of the ulnar nerve

scar in four of the five patients and two of the five were found to have a constriction in region of the subfascial sling. Three patients underwent intramuscular transposition. Two developed a dense scar and one was found embedded in muscle with kinking at the cubital tunnel. The one patient that had undergone submuscular transposition had their ulnar nerve compressed beneath the antebrachial fascia overlying the lacertus fibrosis. Only one of the transposed patients showed evidence of ulnar nerve instability; the ulnar nerve was found back in the groove behind the medial epicondyle.

In the acute postoperative period, haematoma can cause recurrent symptoms. Perineural fibrosis and scar can lead to new sites of nerve compression. Immobilisation in the postoperative period has been suggested to increase the potential for cicatricial scar and even contracture formation but studies comparing immobilisation with early mobility have failed to show a difference [42]. Snapping of the medial head of the triceps over the medial epicondyle can occur following ulnar nerve transposition. This is treated by a limited resection of the involved portion of the triceps [123].

Revision Surgery

The decision to perform revision surgery should be offered to patients with persistent or recurrent symptoms who have ulnar nerve symptoms localised to the elbow without significant comorbities. We prefer to have electrodiagnostic studies on all patients considering revision surgery. In these studies we look for persistent or worsening amplitudes, conduction velocities and evidence of motor denervation.

When the primary surgery is decompression without transposition, we favor decompression with subcutaneous transposition when revision surgery is necessary. Goldfarb et al. reviewed 69 extremities in 56 patients who had an in-situ ulnar nerve decompression [124]. Five patients were found to have recurrence of symptoms. All patients with recurrence were treated with anterior submuscular transposition and had relief of their symptoms. The most commonly performed primary procedure is subcutaneous transposition and, as a result accounts, for 60–80 % of failures. Following subcutaneous transposition we will opt for submuscular transposition for the revision procedure.

Conclusion

Ulnar nerve compression neuropathy is common. Diagnosis depends on history, clinical assessment and provocative tests. Role of other modalities like ultrasound and electrodiagnostic studies are useful but await a consensus reference standard to assess their diagnostic utility [46]. If conservative treatment fails, there are many surgical options. In his review of the literature, Dellon stated that the primary factor guiding surgeon choice is "personal bias" [68]. It has been suggested that if surgical outcomes are similar, then least

invasive, least technically demanding procedure should be chosen. Simple decompression offers this advantage. Although endoscopic techniques are becoming an increasingly popular, we prefer to wait for future study to support its reproducibility and delineate its limits. With a proper understanding of the anatomy of the elbow, pathologic locations of cubital tunnel neuropathy, and adherence to surgical principles outlined, good results can be expected with all surgical options we have discussed.

References

1. Panas J. Sur une cause peu connue de paralysie du nerf cubital. Arch Generales de Med. 1878;2:5–15.
2. Feindel W, Stratford J. Cubital tunnel compression in tardy ulnar palsy. Can Med Assoc J. 1958;78(5): 351–3.
3. Adelaar RS, Foster WC, McDowell C. The treatment of cubital tunnel syndrome. J Hand Surg Am. 1984; 9A(1):90–5.
4. Apfelberg DB, Larson SJ. Dynamic anatomy of the ulnar nerve at the elbow. Plast Reconstr Surg. 1973;51(1):76.
5. Ochiai N, Honmo J, Tsujino A, et al. Electrodiagnosis in entrapment neuropathy by the arcade of struthers. Clin Orthop Relat Res. 2001;378:129–35.
6. Mowlavi A, Andrews K, Lille S, et al. The management of cubital tunnel syndrome: a meta-analysis of clinical studies. Plast Reconstr Surg. 2000;106(2): 327–34.
7. Caliandro P, La Torre G, Padua R, et al. Treatment for ulnar neuropathy at the elbow. Cochrane Database Syst Rev. 2011;16(2):CD006839.
8. Kim DH, Han K, Tiel R, et al. Surgical outcomes of 654 ulnar nerve lesions. J Neurosurg. 2003;98(5): 993–1004.
9. Osborne GV. The surgical treatment of tardy ulnar neuritis. J Bone Joint Surg Am. 1957;39B:782.
10. Khoo D, Carmichael SW, Spinner RJ. Ulnar nerve anatomy and compression. Orthop Clin North Am. 1996;27:317–38.
11. Doyle JR, Botte MJ. Surgical anatomy of the hand and upper extremity. Philadelphia: Lippincott Williams and Wilkins; 2003.
12. Jabaley ME, Wallace WH, Heckler FR. Internal topography of major nerves of the forearm and hand: a current view. J Hand Surg Am. 1980;5(1):1–18.
13. Watchmaker GP, Lee G, Mackinnon SE. Intraneural topography of the ulnar nerve in the cubital tunnel facilitates anterior transposition. J Hand Surg Am. 1994;19(6):915–22.
14. Tubbs RS, Custis JW, Salter EG, et al. Quantitation of and landmarks for the muscular branches of the ulnar nerve to the forearm for application in peripheral nerve neurotization procedures. J Neurosurg. 2006;104(5):800–3.
15. Amadio PC. Anatomical basis for a technique of ulnar nerve transposition. Surg Radiol Anat. 1986;8(3): 155–61.
16. Sunderland S. Blood supply of the sciatic nerve and its popliteal divisions in man. Arch Neurol Psychiatry. 1945;54:283–9.
17. Yagmaguchi K, Sweet FA, Bindra R, et al. The extraneural and intraneural arterial anatomy of the ulnar nerve at the elbow. J Shoulder Elbow Surg. 1999;8: 17–21.
18. Sugawara M. Experimental and clinical studies of the vascularized anterior transposition of the ulnar nerve for cubital tunnel syndrome. Nihon Seikeigeka Gakkai Zasshi. 1988;62(8):755–66.
19. Breidenbach WC, Terzis JK. The blood supply of vascularized nerve grafts. J Reconstr Microsurg. 1986;3(1):43–58.
20. Lundborg G. Structure and function of the intraneural microvessels as related to trauma, edema formation, and nerve function. J Bone Joint Surg. 1975; 57A:938–48.
21. Sunderland S. Nerves and nerve injuries. 2nd ed. Edinburgh, New York: Churchill Livingstone; 1987. p. 728–49.
22. Lowe JB, Novak CB, Mackinnon SE. Current approach to Cubital tunnel syndrome. Neurosurg Clin N Am. 2001;12(2):267–84.
23. Leffert RD. Anterior submuscular transposition of the ulnar nerves by the Learmonth technique. J Hand Surg Am. 1982;7(2):147–55.
24. Masear VR, Meyer RD, Pichora DR. Surgical anatomy of the medial of the antebrachial cutaneous nerve. J Hand Surg Am. 1989;14(2):267–71.
25. Dellon AL, Mackinnon SE. Injury to the medial antebrachial cutaneous nerve during cubital tunnel surgery. J Hand Surg Br. 1985;10(1):33–6.
26. Sarris I, Gobel F, Gainer M, et al. Medial brachial and antebrachial cutaneous nerve injuries: effect on outcome in revision cubital tunnel surgery. J Reconstr Microsurg. 2002;18(8):665–70.
27. Lowe JB, Maggi SP, MacKinnon SE. The position of crossing branches of the medial antebrachial cutaneous nerve during cubital tunnel surgery in humans. Plast Reconstr Surg. 2004;114(3):692–6.
28. Jackson LC, Hotchkiss RN. Cubital tunnel surgery: complications and treatment of failures. Hand Clin. 1996;12(2):449–56.
29. Mackinnon SE, Dellon AL, Hudson AR, et al. Chronic nerve compression: an experimental model in the rat. Ann Plast Surg. 1984;13(2):112–20.
30. Mackinnon SE, Dellon AL, Hudson AR, et al. Alteration of neuroma formation by manipulation of its microenvironment. Plast Reconstr Surg. 1985;76(3): 345–53.

31. Dellon AL, Mackinnon SE. Treatment of the painful neuroma by neuroma resection and muscle implantation. Plast Reconstr Surg. 1986;77(3):427–38.
32. Pannucci C, Myckatyn TM, Mackinnon SE, Hayashi A. End-to-side nerve repair: review of the literature. Restor Neurol Neurosci. 2007;25(1):45–63.
33. Spinner M, Kaplan EB. The relationship of the ulnar nerve to the medial intermuscular septum in the arm and its clinical significance. Hand. 1976;8(3): 239–42.
34. Von Schroeder H, Scheker LR. Redefining the "Arcade of Struthers". J Hand Surg Am. 2003; 28(6):1018–21.
35. Gay JR, Love JG. Diagnosis and treatment of tardy paralysis of the ulnar nerve based on a study of 100 cases. J Bone Joint Surg Am. 1947;29(4):1087–97.
36. Vanderpool DW, Chalmers J, Lamb DW, et al. Peripheral compression lesions of the ulnar nerve. J Bone Joint Surg Br. 1968;50(4):792–803.
37. Barber KW, Bianco AJ, Soule EH, et al. Benign extraneural soft-tissue tumors of the extremities causing compression of nerves. J Bone Joint Surg Am. 1962;44(1):98–104.
38. Wachsmuth W, Wilhelm A. The musculus epitrochleoanconaeus and its clinical significance. Monatsschr Unfallheilkd Versicher Versorg Verkehrsmed. 1968; 71(1):1–22.
39. Hirasawa Y, Sawamura H, Sakakida K. Entrapment neuropathy due to bilateral epitrochleoanconeus muscles: a case report. J Hand Surg Am. 1979;4(2): 181–4.
40. Childress HM. Recurrent ulnar-nerve dislocation at the elbow. Clin Orthop Relat Res. 1975;108: 168–73.
41. Karatsa A, Apaydin N, Uz A, et al. Regional anatomic structures of the elbow that may potentially compress the ulnar nerve. J Shoulder Elbow Surg. 2009;18:627–31.
42. Pechan J, Julis I. The pressure measurement in the ulnar nerve. A contribution to the pathophysiology of the cubital tunnel syndrome. J Biomech. 1975; 8(1):75–9.
43. Weirich SD, Gelberman RH, Best SA, et al. Rehabilitation after subcutaneous transposition of the ulnar nerve: immediate versus delayed mobilization. J Shoulder Elbow Surg. 1998;7(3):244–9.
44. Clark WL, Trumble TE, Swiontkowski MF, et al. Nerve tension and blood flow in a rate model of immediate and delayed repairs. J Hand Surg Am. 1992;17(4):677–87.
45. Lundborg G, Bordborg C, Rydevik B, Olsson Y. The effect of ischemia on the permeability of the perineurium to protein tracers in rabbit tibial nerve. Acta Neurol Scand. 1973;49(3):287–94.
46. Hutchison RL, Rayan G. Diagnosis of cubital tunnel syndrome. J Hand Surg Am. 2011;36(9):1519–21.
47. Huang JH, Samadani U, Zager EL. Ulnar nerve entrapment neuropathy at the elbow: simple decompression. Neurosurgery. 2004;55(5):1150–3.
48. Upton AR, McComas AJ. The double crush in nerve entrapment syndromes. Lancet. 1973;2(7825):359–62.
49. Mallette P, Zhao M, Zurakowski D, et al. Muscle atrophy at diagnosis of carpal and cubital tunnel syndrome. J Hand Surg Am. 2007;32(6):855–8.
50. Beekman R, Schreuder AH, Rozeman CA, et al. The diagnostic value of provocative clinical tests in ulnar neuropathy at the elbow is marginal. J Neurol Neurosurg Psychiatry. 2009;80(12):1369–74.
51. Rayan GM, Jensen C, Duke J. Elbow flexion test in the normal population. J Hand Surg Am. 1992;17(1): 86–9.
52. Novak CB, Lee GW, Mackinnon SE, et al. Provocative testing for cubital tunnel syndrome. J Hand Surg Am. 1994;19(5):817–20.
53. Cheng CJ, Mackinnon-Patterson B, Beck JL, et al. Scratch collapse test for evaluation of carpal and cubital tunnel syndrome. J Hand Surg Am. 2008; 33(9):1518–24.
54. St John JN, Palmaz JC. The cubital tunnel in ulnar entrapment neuropathy. Radiology. 1986;158(1): 119–23.
55. Greenwald D, Blum LC, Adams D, et al. Effective surgical treatment of cubital tunnel syndrome based on provocative clinical testing without electrodiagnostics. Plast Reconstr Surg. 2006;117(5):87e–91.
56. Tomaino MM, Brach MS, Vansickle DP. The rationale for and efficacy of surgical intervention for electrodiagnostic-negative cubital tunnel syndrome. J Hand Surg Am. 2001;26(6):1077–81.
57. Gelberman RH, Eaton R, Urbaniak JR. Peripheral nerve compression. J Bone Joint Surg. 1993;75A: 1854–78.
58. Heithoff SJ, Millender LH, Nalebuff EA, Petruska Jr AJ. Medial epicondylectomy for the treatment of ulnar nerve compression at the elbow. J Hand Surg Am. 1990;15A:22–9.
59. Craven PR, Green DP. Cubital tunnel syndrome. Treatment by medial epicondylectomy. J Bone Joint Surg Am. 1980;62(6):986–9.
60. Eisen A. Early diagnosis of ulnar nerve palsy. An electrophysiologic study. Neurology. 1974;24(3):256–62.
61. Eisen A, Schomer D, Melmed C. The application of F-wave measurements in the differentiation of proximal and distal limb entrapments. Neurology. 1977; 27(7):662–8.
62. Rempel D, Dahlin L, Lundborg G. Pathophysiology of nerve compression syndromes: response of peripheral nerves to loading. J Bone Joint Surg Am. 1999;81(11):1600–10.
63. Wiesler ER, Chloros GD, Cartwright MS, et al. Ultrasound in the diagnosis of the ulnar neuropathy at the cubital tunnel. J Hand Surg Am. 2006;31(7): 1088–93.
64. Okamoto M, Abe M, Shirai H, et al. Diagnostic ultrasonography of the ulnar nerve in cubital tunnel syndrome. J Hand Surg Br. 2000;25:499–502.
65. Kato H, Hirayama T, Minami A, Iwasaki N, et al. Cubital tunnel syndrome associated with medial

elbow ganglia and osteoarthritis of the elbow. J Bone Joint Surg Am. 2002;84A(8):1413–9.
66. Jacobsen J, Jebson P, Jeffers A, et al. Ulnar nerve dislocation and snapping triceps syndrome: diagnosis with dynamic sonography-report of three cases. Radiology. 2001;220:601–5.
67. McGowan AJ. The results of transposition of the ulnar nerve for traumatic ulnar neuritis. J Bone Joint Surg Br. 1950;32B(3):293–301.
68. Dellon AL. Review of treatment results for the ulnar nerve entrapment at the elbow. J Hand Surg Am. 1989;14(4):688–700.
69. Kleinman WB, Bishop AT. Anterior intramuscular transposition of the ulnar nerve. J Hand Surg Am. 1989;14(6):972–9.
70. Dimond ML, Lister GD. Cubital tunnel syndrome treated by long-arm splintage. J Hand Surg Am. 1985;10A:430.
71. Svernlov B, Larsson M, Rehn K, et al. Conservative treatment of the cubital tunnel syndrome. J Hand Surg Eur Vol. 2009;34(2):201–7.
72. Dellon AL, Hament W, Gittelshon A. Non-operative management of cubital tunnel syndrome: an 8-year prospective study. Neurology. 1993;43(9):1673–7.
73. Bartels RH. History of the surgical treatment of ulnar nerve compression at the elbow. Neurosurgery. 2001;49(2):391–9.
74. Zlowodzki M, Chan S, Bhandari M, et al. Anterior transposition compared with simple decompression for treatment of cubital tunnel syndrome: a meta-analysis of randomized, controlled trials. J Bone Joint Surg Am. 2007;89:2591–8.
75. Macadam SA, et al. Simple decompression versus anterior subcutaneous and submuscular transposition of the ulnar nerve for cubital tunnel syndrome: a meta-analysis. J Hand Surg Am. 2008;33A:1314–24.
76. Macadam SA, Bezuhly M, Lefaivre KA. Outcomes measures used to assess results after surgery for cubital tunnel syndrome: a systematic review of the literature. J Hand Surg Am. 2009;34(8):1482–91.
77. Mackinnon SE, Novak CB. Compressive neuropathies. In: Green DP, Hotchkiss RN, Pederson WC, Wolfe SW, editors. Operative hand surgery. 5th ed. New York: Churchill Livingstone; 2004.
78. Heithoff SJ. Cubital tunnel syndrome does not require transposition of the ulnar nerve. J Hand Surg Am. 1999;24A:898–905.
79. Robertson C, Saratsiotis DC. A review of compressive ulnar neuropathy at the elbow. J Manipulative Physiol Ther. 2005;28(5):345.
80. Novak CB, Mackinnon SE. Selection of operative procedures for cubital tunnel syndrome. Hand. 2008;4(1):50–4.
81. Cho YJ, Cho SM, Sheen SH, et al. Simple decompression of the ulnar nerve for cubital tunnel syndrome. J Korean Neurosurg Soc. 2007;42(5):382–7.
82. Thomsen PB. Compression neuritis of the ulnar nerve treated with simple decompression. Acta Orthop Scand. 1977;48(2):164–7.
83. Biggs M, Curtis JA. Randomized, prospective study comparing ulnar neurolysis in situ with submuscular transposition. Neurosurgery. 2006;58(2):296–304.
84. Gervasio O, Gambardella G, Zaccone C, et al. Simple decompression versus anterior submuscular transposition of the ulnar nerve in severe cubital tunnel syndrome: a prospective randomized study. Neurosurgery. 2005;56(1):108–17.
85. Nabhan A, Ahlhelm F, Kelm J, et al. Simple decompression of subcutaneous anterior transposition of the ulnar nerve for cubital tunnel syndrome. J Hand Surg Am. 2005;30(5):521–4.
86. Bednar MS, Blair SJ, Light TR. Complications of the treatment of cubital tunnel syndrome. Hand Clin. 1994;10(1):88–92.
87. Bartels RH, Menovsky T, Van Overbeeke JJ, et al. Surgical management of ulnar nerve compression at the elbow: an analysis of the literature. J Neurosurg. 1998;89(5):722–7.
88. Fillipi R, Farag R, Reisch P, et al. Cubital tunnel syndrome treatment by decompression without transposition of ulnar nerve. Minim Invasive Neurosurg. 2002;45(3):164–8.
89. LeRoux PD, Ensign TD, Burchiel KJ. Surgical decompression without transposition for ulnar neuropathy: factors determining outcome. Neurosurgery. 1990;27(5):709–14.
90. Bartels RH, Termeer E, van der Wilt GJ, et al. Simple decompression or anterior subcutaneous transposition for ulnar neuropathy at the elbow: a cost-minimization analysis – part 2. Neurosurgery. 2005; 56(3):531–6.
91. Curtis BF. Traumatic ulnar neuritis transplantation of the nerve. J Nerv Ment Dis. 1898;25:480–1.
92. Ogata K, Manske PR, Lesker PA. The effect of surgical dissection on regional blood flow to the ulnar nerve in the cubital tunnel. Clin Orthop Relat Res. 1985;193:195–8.
93. Richmond JC, Southmayd WW. Superficial anterior transposition of the ulnar nerve at the elbow for ulnar neuritis. Clin Orthop Relat Res. 1982;164:42–4.
94. Rettig AC, Ebben JR. Anterior subcutaneous transfer of the ulnar nerve in the athlete. Am J Sports Med. 1993;21(6):836–40.
95. Lugnegard H, Walheim G, Wennberg A. Operative treatment of the ulnar nerve neuropathy in the elbow region. A clinical and electrophysiological study. Acta Orthop Scand. 1977;48(2):168–76.
96. Eaton RG, Crowe JF, Parkes III JC. Anterior transportation of the ulnar with a noncompressing fasciodermal sling. J Bone Joint Surg Am. 1980;62A: 820–5.
97. Osterman AL, Davis CA. Subcutaneous transposition of the ulnar nerve for treatment of cubital tunnel syndrome. Hand Clin. 1996;12(2):421–33.
98. Adson AW. The surgical treatment of progressive ulnar paralysis. Minn Med. 1918;1:455.
99. Henry M. Modified intramuscular transposition of the ulnar nerve. J Hand Surg Am. 2006;31(9):1535–42.

100. Broudy AS, Leffert RD, Smith RJ. Technical problems with ulnar nerve transposition at the elbow: findings and results of reoperation. J Hand Surg Am. 1978;3:85.
101. Kleinman WB. Anterior intramuscular transposition. In: Gelberman RH, editor. Operative nerve repair and reconstruction. Philadelphia: Lippincott; 1991. p. 1069–76.
102. Dellon AL, Mackinnon SE, Hudson AR, et al. Effect of submuscular versus intramuscular placement of ulnar nerve: experimental model in the primate. J Hand Surg Br. 1986;11(1):117–9.
103. Learmonth JR. A technique for transplanting the ulnar nerve. Surg Gynecol Obstet. 1942;75:792–3.
104. Dellon AL. Operative technique for submuscular transposition of the ulnar nerve. Contemp Orthop. 1988;16:17–24.
105. Black BT, Barron OA, Townsend PF, et al. Stabilized subcutaneous ulnar nerve transposition with immediate range of motion Long term follow-up. J Bone Joint Surg Am. 2000;82A(11):1544–51.
106. Gasco J. Surgical options for ulnar nerve entrapment: an example of individualized decision analysis. Hand. 2009;4(4):350–6.
107. Keiner D, Gaab MR, Schroeder HW, et al. Comparison of the long-term results of anterior transposition of the ulnar nerve of simple decompression in the treatment of cubital tunnel syndrome-a prospective study. Acta Neurochir. 2009;151(4):311–5.
108. King T. The treatment of traumatic ulnar neuritis; mobilization of the ulnar nerve at the elbow by removal of the medial epicondyle and adjacent bone. Aust N Z J Surg. 1950;20(1):33–42.
109. Froimson AL, Zahrawi F. Treatment of compression neuropathy of the ulnar nerve at the elbow by epicondylectomy and neurolysis. J Hand Surg Am. 1980;5(4):391–5.
110. McPherson SA, Meals RA. Cubital tunnel syndrome. Orthop Clin North Am. 1992;23(1):111–23.
111. Baek GH, Kown BC, Chung MS. Comparative study between minimal epicondylectomy and anterior subcutaneous transposition of the ulnar nerve for cubital tunnel syndrome. J Shoulder Elbow Surg. 2006;15(5):609–13.
112. Tsai TM, Bonczar M, Tsuruta T, et al. A new operative technique: cubital tunnel decompression with endoscopic assistance. Hand Clin. 1995;11(1):71–80.
113. Cobb TK, Sterbank PT, Lemke JH. Endoscopic cubital tunnel recurrence rates. Hand. 2009;5:179–83.
114. Ahcan U, Zorman P. Endoscopic decompression of the ulnar nerve at the elbow. J Hand Surg Am. 2007;32A:1171–6.
115. Hoffman R, Sieminow M. The endoscopic management of cubital tunnel syndrome. J Hand Surg Br. 2006;31B:23–9.
116. Nakao Y, Takayama S, Toyama Y. Cubital tunnel release with lift-type endoscopic surgery. Hand Surg. 2001;6(2):199–203.
117. Tsai TM, Chen IC, Majd ME, et al. Cubital tunnel release with endoscopic assistance: results of a new technique. J Hand Surg Am. 1999;24(1):21–9.
118. Watts AC, Bain GI. Patient-rated outcome of ulnar nerve decompression: a comparison of endoscopic and open in situ decompression. J Hand Surg Am. 2009;34(8):1492–8.
119. Jiang S, Xu W, Shen Y, Xu JG, Gu YD. Endoscopy-assisted cubital tunnel release under carbon dioxide insufflation and anterior transposition. Ann Plast Surg. 2012;68:62–6.
120. Cobb TK. Endoscopic cubital tunnel release. J Hand Surg Am. 2010;35(10):1690–7.
121. Seradge H, Owen W. Cubital tunnel release with medial epicondylectomy factors influencing the outcome. J Hand Surg Am. 1998;23(3):183–91.
122. Rogers MR, Bergfield TG, Aulicino PL. The failed ulnar nerve transposition etiology and treatment. Clin Orthop Relat Res. 1991;269:193–200.
123. Spinner RJ, Gabel GT. Iatrogenic snapping of the medial head of the triceps after ulnar nerve transposition. J South Orthop Assoc. 2001;10(4):236–40.
124. Goldfarb CA, Sutter MM, Martens EJ, et al. Incidence of re-operation and subjective outcome following in situ decompression of the ulnar nerve at the cubital tunnel. J Hand Surg Eur Vol. 2009;34(3):379–83.