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REVIEW ARTICLE

Ulnar Nerve Entrapment at Elbow: Current Debates

Chetan Patel[1], Praveen Bhardwaj[2], Shyam Chandrasekar[3]

Abstract

Cubital Tunnel Syndrome (CTS) results from ulnar nerve entrapment (UNE); an anatomical combination of tension and compression to the fibres of the ulnar nerve as they pass around the elbow through the distal arm into the proximal forearm. The acute on chronic nature of the syndrome has been elegantly outlined in a wide variety of studies. Demographics that relate to peripheral nerve compressions in general have contributed to an increase in the incidence of Cubital Tunnel Syndrome. Clinical examination supported by neurophysiological confirmation forms the basis of obtaining a diagnosis; more recently radiologic corroboration of anatomical changes and specific site localization has improved and added to pre-operative findings. Whilst primary surgical management of cubital tunnel syndrome has been on the increase with no clear preference for a procedure in randomized controlled trials, a trend toward minimal nerve handling/dissection has emerged in primary cases; whilst conservative measures used continue to provide relief in the majority of patients with mild disease. Transposition of the ulnar nerve still remains the procedure of choice for recurrent symptoms or revision surgery.

Introduction

Much has been written with respect to cubital tunnel syndrome and ulnar nerve entrapment in the recent literature. Basic science, diagnostics and therapeutic options have been researched, with intricate details of anatomy, morphology, and physiology better understood. Although the incidence of cubital tunnel syndrome would seem to be on the increase, a definitive algorithm for management still remains elusive and the outcomes of meta-analysis and randomized controlled trials comparing various surgical treatments are divergent to case series with long term follow up. Prognostic factors have certainly come to the fore in recent studies with certain identifiable pre-operative Grades, patient demographics and anatomical considerations being better suited to more favorable outcomes. As recognition of cubital tunnel symptoms tends to be later than carpal tunnel, latency in seeking treatment has increased, resulting in permanent muscle weakness and sensory changes in the ulnar nerve distribution. Despite the differences in opinion, the majority of patients treated for ulnar nerve entrapment do fairly well.

Anatomy

Whilst the basic macro-anatomy of the ulnar nerve has been well described with multiple points of potential compression about the elbow joint; the retinaculum overlying the ulnar groove spanning olecranon to the medial epicondyle and the fascia that links the two heads of the flexor carpi ulnaris muscle (Osborne's band) proximally are the two primary sites of compression with other secondary sites of compression being the arcade of Struthers (about 8cm proximal to the medial epicondyle), volar antebrachial fascia proximal to the wrist crease and the hypothenar musculature over the deep branch of the ulnar nerve in Guyon's canal [1] (Fig. 1). This has clinical implications of pre-operatively determining a level of compression using a "hierachical" Scratch Collapse Test as described by Mackinnon[1]. Entrapment suggests that nerve dysfunction is due solely to compression by surrounding structures, however nerve injury is also induced by local friction and traction[2]. Conservative treatment attests to this fact and is substantiated by anatomical morphological studies. The cubital tunnel was shown to narrow by 55% in full flexion when compared to extension[3]. Patel showed a change in the shape of the cubital tunnel from round to triangular

✉ Praveen Bhardwaj
+91 9944562422
drpb23@gmail.com

1 MBBCh, FCS PlastSurg (SA), MMedPlastSurg (Wits)
Plastic and Reconstructive Surgeon, SandtonMediclinic,
Cnr Peter Place and Main Road, Johannesburg, South Africa

2 MS (Ortho); DNB (Ortho); FNB (Hand & Microsurgery)
European Diploma Hand Surgery
Consultant- Hand and Reconstructive Microsurgery
Ganga Hospital, Coimbatore, India

3 Third year undergraduate - B.S. in Physiology and
Neuroscience, University of California, San Diego.

when flexed and demonstrated a height decrease of 2.5mm[4]. Blood supply and axonal transport are affected by these compressive and traction forces[5]. Gelberman and colleagues summed it up well in their cadaveric study, demonstrating that the morphological changes in the tunnel translated to differential changes in intra and extraneural pressures, reasoning that traction on the nerve played as significant role in diminished axonal transport. Their study also demonstrated a greater than 50% decrease in the area of the tunnel. With the elbow flexed 135 degrees, the cross-sectional areas were significantly smaller than the areas at all other positions of the elbow ($p < 0.05$)[6]. Interestingly, the work showed that the angle range of least pressure was between 40-50 degrees of flexion, a measurement of practical value in conservative splinting[6].

From a micro-anatomic perspective acute compression is dependent on axonal injury whereas chronic nerve ischemia results in a process of de-myelination and re-myelination with axonal injury occurring late in the process[7]. The Schwann cell proliferation is macrophage independent in chronic injuries and more related to mechanical factors. This correlates well with the early symptoms not being accurately diagnosed by electrodiagnostic testing.

Further anatomical work applies to the surgical approaches. The vascular supply of the ulnar nerve around the elbow consists of 3 branches, superior and inferior ulnar collaterals arising from the brachial artery and posterior ulnar recurrent artery arising from the ulnar artery[8]. The most appropriate distance a nerve could be moved while retaining its blood supply was 1.8 cm based on the tension created by retaining the vascular supply. Subcutaneous anterior transposition hence would not be appropriate if vascular supply is to be preserved and one should resort to submuscular transposition. Nakamura et al studied the effect of vascular pedicle reservation on blood flow and clinical outcome following ulnar nerve transposition[9]. They found that preserving the vascular pedicles can prevent compromise of the blood flow to the ulnar nerve immediately after nerve transposition. However, preservation of the vascular pedicle had no correlation with the improved recovery of ulnar nerve function after surgery[9]. So, preservation of the vascular pedicle while performing anterior transposition of the ulnar nerve is still debatable. The preserved vascular pedicle does retain better blood flow to the nerve. However, it adds to the complexity of the surgery and its clinical results are still not proved. This could be because of the fact that the intra-neural blood supply of the nerves is good enough to preserve adequate nerve vascularity after transposition. Although not conclusive these anatomical treatises do point to minimal disturbance of the nerve in surgical decision making in primary surgeries.

Other significant anatomical studies found that the most common location for crossing branches of the medial antebrachial cutaneous nerve was 2cm distal to the medial epicondyle[10]; and a high degree of variability was found in the distal flexor aponeurosis, the cutaneous nerves and the proximal fascial bands of Struthers (12/26 specimens)[11]. Knowing all these anatomical facts does help a surgeon to effectively decompress the nerve while preserving the cutaneous nerves.

The predominance and early onset of sensory symptoms is explained by the fact that the sensory fascicles are superficial at the level of the compression and are the first to be affected. Deeper location of the fascicles for the flexor carpi ulnaris may result in sparing of this muscle till severe compression stages (Fig. 2).

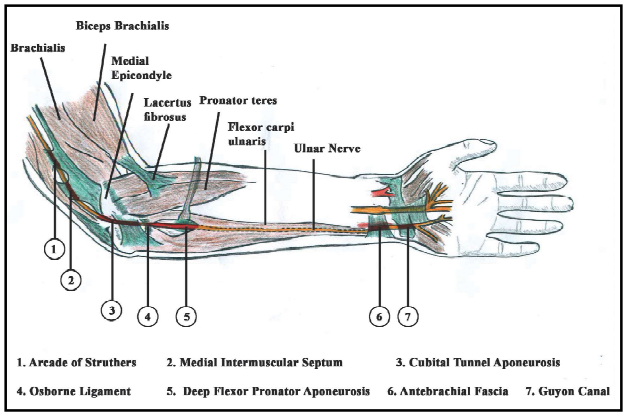

Fig. 1 Anatomical sites of compression of ulnar nerve.

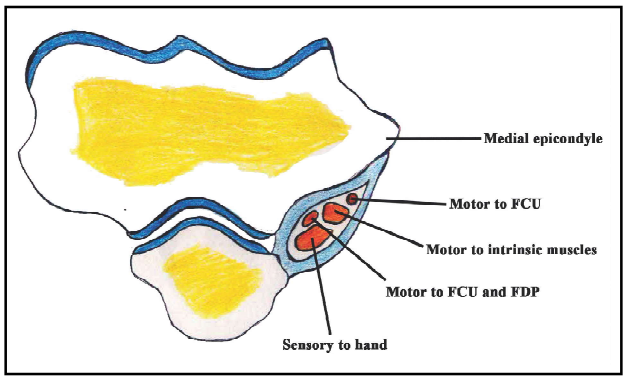

Fig. 2 Fascicular arrangement of ulnar nerve at the elbow

Epidemiology

Musculoskeletal disorders have increased dramatically in recent years[12], consequently resulting in concomitant increases seen in the demand for elective hand surgery conditions such as cubital tunnel syndrome and others[13,14]. Obesity is a well-known risk factor for which there is a good correlation to an increase in peripheral nerve pathology[14]. Diabetic patients are more likely to have both carpal and cubital tunnel procedures simultaneously and should be evaluated for multiple compression nerve pathologies[15]. No conclusive data points toward work related activities being causal factors but numerous case series indicate that repetitive motions of flexion and extension such as in musician and tennis players may play a role in presenting cubital tunnel symptoms[16,17]. Altered anatomy such as in wheelchair athletes has allowed further study into the causal relationship between chronic postural abnormalities and the occurrence of nerve compression disorders[18].

Diagnosis

Clinical presentation usually starts with numbness and tingling (pins and needles) in the little and ulnar half of the ring finger. These symptoms present at early on at night when the likelihood of the elbow being flexed, resulting in increased tension and ischemia to the nerve, is higher. As the disease progresses the symptoms can persist during the day. With early ischemia, activities or occupations that aggravate the compression may worsen symptoms leading to later loss of fine motor co-ordination. Any pre-existing condition such as epicondylitis, valgus deformity or elbow instability from previous trauma may worsen the symptoms. Cervical radiculopathy, chronic postural abnormalities, thoracic outlet syndrome and more rarely spinal cord pathologies may present with ulnar nerve symptoms. Less frequently, distal to the cubital tunnel similar symptoms can occur by compression and pathology in Guyon's canal, wrist arthritis or flexor carpi ulnaris tendonitis. The sum of a good history; documenting onset, duration and degree of symptoms with a symptom diary in those patients in whom the diagnosis is not certain; assists greatly in further clinical evaluation and choosing investigations. In a cost conscious health care setting, the judicious use of investigations will be increasingly important.

Clinical examination will show loss of sensation in the ulnar digits, which can be quantified and serially repeated using both monofilament testing and two-point discrimination. The location of sensory loss in the volar versus dorsal distribution can differentiate compression in Guyon's canal as opposed to the cubital tunnel [Fig 3]. Motor weakness although intuitively a late sign and poor prognostic indicator can be detected early by testing those ulnar innervated muscles distal to the compression site. Jepsen et al. showed good inter-rater reproducibility of neurological patterns on examination of various nerve conditions[19]. This lends support to an examination that follows a set pattern for every nerve compression disorder beginning proximally and advancing distally testing the various muscle groups looking for subtle changes in muscle strength and comparing with the contralateral limb. Late signs of muscle wasting and weakness and consequent changes in the hand are as those described for ulnar nerve palsy. Provocative testing with compression at the level of the medial epicondyle with the arm flexed, forearm supinated and wrist in extension for 3 minutes is most reliable currently (91% sensitive[20]). Classifications of ulnar nerve entrapment were introduced by McGowan and later modified by Dellon[21] (Table 1). Accordingly, mild, moderate and severe types are based on sensory symptoms, loss of two-point sensibility and degrees of motor weakness. It is a useful classification stratifying patients for non-surgical versus surgical treatments.

Electrodiagnostics remain the standard of diagnosis for most upper limb compression neuropathies[7]. Anatomical correlation of electrical changes is reliably explained[7]; with an absolute decrease in nerve conduction velocity < 50m/s or relative drop of >=10m/s across the elbow being diagnostic. Errors do occur due to soft tissue bulk over elbow, skin temperature and variable elbow positioning. Ultrasound and MRI have been evaluated as adjunctive tests. In a recent meta-analysis ultrasound detection with nerve cross sectional area threshold set at $10mm^2$ has found to be diagnostic of a compression of the ulnar at the medial epicondyle when compared to asymptomatic individuals[22]. Long axis diameter and cross sectional area has been shown to be discriminatory between asymptomatic and symptomatic ulnar nerve entrapment factoring in confounding variable such as age, weight, body mass index[23]. Investigations inform us further of a clinical diagnosis of ulnar nerve entrapment and do add confidence (and objectiveness) to the clinical diagnosis of these complex presentations.

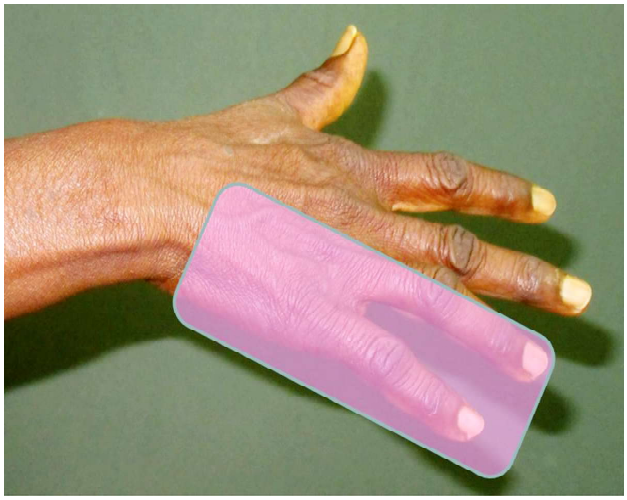

Fig. 3 Pattern of sensory loss helps to localize the level of compression. Sensory loss over the dorsal aspect of the ulnar one and half digits indicates compression proximal to the origin of the dorsal sensory branch of the ulnar neve which arises in the distal third of the forearm.

Table 1: Staging of Ulnar nerve compression at the elbow[21]

Mild	
Sensory	Paresthesias come and go Vibratory perception increased
Motor	Subjective weakness, clumsiness or loss of coordination
Tests	Elbow flexion test and/or Tinel's sign may be positive
Moderate	
Sensory	Paresthesias come and go Vibratory perception normal or decreased
Motor	Measurable weakness in pinch and/or grip strength
Tests	Elbow flexion test and/or Tinel's sign are positive Finger crossing may be abnormal
Severe	
Sensory	Paresthesias are persistent Vibratory perception decreased Abnormal two- point discrimination (static>/=6mm, moving>/=4mm)
Motor	Measurable weakness in pinch and grip strength plus muscle atrophy
Tests	Positive elbow flexion test and/or positive Tinel's sign may be present Finger crossing usually abnormal

Treatment

Non-Surgical treatment

Mild and moderate symptoms warrant conservative strategies with follow-up assessments for a period of 6 months. Night splinting (rigid or soft), limiting elbow flexion, patient education regarding avoidance of flexed positions, limiting direct pressure over the medial epicondyle and in active patients, avoidance of triceps exercises all assisted in improving symptoms. Svernlöv[2] found in a relatively small population that 90% of patients with mild and moderate symptoms did well with conservative management and only 10% went onto surgical treatment at 6 months. Dellon[24] had a 21% surgical conversion with mild symptom patients, 33% with moderate symptoms and a two thirds turnover rate with severe symptoms over an 8-year review period. They recommended only patients with mild symptoms be treated conservatively. Education alone showed as much improvement as addition of splinting techniques and is a good first line therapy for entrapment[2,25].

Surgical Treatment

Despite a plethora of literature there is no definitive consensus between in-situ decompression (endoscopic, open or mini-open technique) and anterior transposition surgery (subcutaneous, submuscular or intramuscular) as being the treatment of choice. A Cochrane review[26] looked at 3 trials (Bartels[27], Gervasio[28] and Biggs[29]) providing moderate evidence that simple decompression is as effective as decompression with transposition but that transpositions presented with more wound infections. A recent survey[30] of American hand surgeons found that two thirds of surgeons would choose in-situ decompression as their treatment for positive symptoms (including 2-point discrimination) and electrodiagnostic studies showing compression. However, almost 80% responded with a change in plan if the nerve was subluxing. Bartels[27] moreover calculated the cost of these two treatments, concluding that decompression alone was more cost effective as a treatment, when follow up and time off work were taken into account for idiopathic ulnar nerve entrapment.

Systematic reviews and meta-analyses[31-32] highlight the lack of controlled trials and preponderance of observational studies with high risk of selection bias in the surgical treatment of ulnar nerve entrapment. Eberlin[33] proposes that the change in presentation of ulnar nerve entrapment from primary elbow trauma and valgus deformities to an idiopathic type of

presentation has lead surgeons to increase the use of minimal surgical approaches such as in-situ decompression or endoscopic treatments, reserving anterior transpositions (subcutaneous, submuscular or intramuscular [Fig. 4 and 5]) to treat selected patients in whom diagnostics and examination reveal complicated, recurrent pathology or for revision surgeries. This view is supported by the ambulatory surgery database trends[13] that indicate a decrease in the number of transposition surgeries being performed.

Counter arguments to the above assertions are made by Mackinnon's group[34] and supported more recently by Lauretti[35]. The arguments are that in-situ decompression surgery has an up-to 20% revision rate, most of which are undertaken in the first 2 years after the index surgery[36]. So despite having a lower initial morbidity and quicker recovery, the revision surgical outcome following an in-situ release is poor. However, no direct comparison between outcomes of revisions for failed decompressions versus failed transpositions has been undertaken. Nakamura[9] also showed that anterior transposition with vascular preservation versus with vessel ligation showed no difference in outcomes at 1 year, contradicting the earlier evidence that decreased blood supply lead to worse outcomes because of scarring.

The advent of endoscopic cubital tunnel surgery and minimal incision surgery has heralded yet another twist in the tale. Two systematic reviews and meta-analyses by Aldekhayel[37]and Toirac[38] a year apart, utilized different statistical methods. Whilst one group[37] concluded that endoscopic and open in-situ decompression had similar complication, re-operation rates and outcomes, the latter group[38] asserted that endoscopic release resulted in superior outcomes and fewer complications. This is different to previous literature in which endoscopic surgery was seen to have higher hematoma rates. These could be explained by a more mature learning curve and greater experience with the endoscopic technique. The rationale of minimal surgery is to have the least possible surgical impact on the nerve by releasing under vision all of the potential compression sites in the course of the ulnar nerve about the elbow. These minimal access procedures should be reserved for idiopathic cases with moderate to severe symptoms in which there are no co-existing surgical or pre-operative factors (subluxing nerve, elbow trauma, arthritis or epicondylitis).

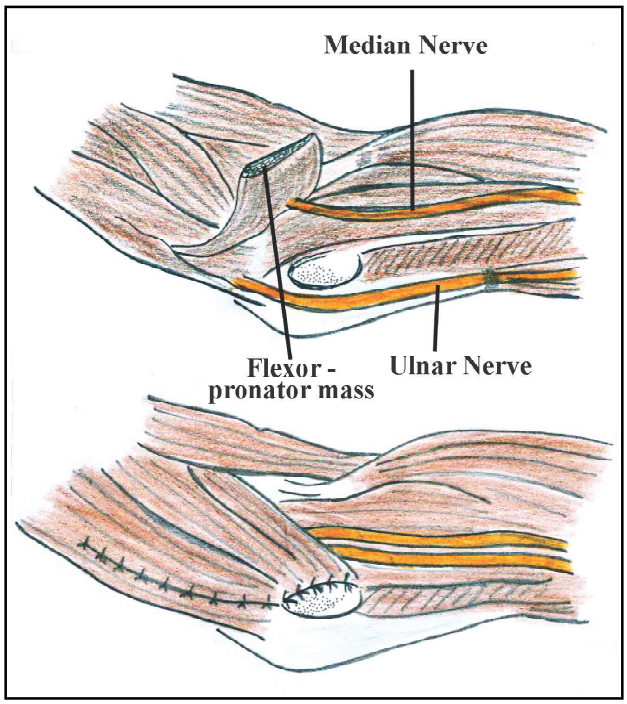

Fig. 4 Illustration of the technique for submuscular transposition of the ulnar nerve

Revision Surgery

Retained intermuscular septum and perineural scarring are echoed themes in the few small case series published on this subject[39,40]. The method of treatment is often decided upon at the time of surgery based on the findings. Pre-operative counseling is crucial in explaining potential for improvement in outcomes as we know that revision surgery outcomes are not as satisfactory as those of primary surgery. Factors predictive of poor outcome were elbow trauma and patient requesting surgery for mild clinically graded disease[36]. Patient factors namely age, obesity, smoking, diabetes were not predictive[36]. No one particular technique has been shown to be superior but the principals of wide exposure with attention to freeing all adhesions and prior compressive areas with due respect to medial antebrachial cutaneous nerves; and based on nerve dynamics transposing or re-siting the nerve back into the canal[40] should be respected.

Authors' Current Practice

The diagnosis of the cubital tunnel syndrome is mainly clinical. The typical distribution of the paresthesia and numbness over the ulnar nerve distribution is the commonest presentation. Association of night symptoms and positive provocative tests further confirms our clinical diagnosis. Nerve conduction

studies are always done for confirmation and objective documentation. Extensive patient education about the condition and counselling regarding the treatment modalities is essential part of the management. Conservative treatment with elbow splinting and activity modification is the first line of treatment for all the idiopathic cubital tunnel syndrome patients. Analgesics and anti-inflammatory with pregabalin are generally prescribed. We continue conservative management for mild cases for 4-6 months before a decision for surgery is undertaken. For severe grade of involvement early surgical exploration is considered. Also, surgical exploration is done early for patients with pre-existing conditions which could lead to ulnar nerve compression like- cubitus valgus, elbow trauma. In situ decompression may be considered for idiopathic cubital tunnel syndrome but we have a low threshold for anterior transposition of the ulnar nerve. Anterior transposition must always be done for patients with ulnar nerve subluxing with flexion and extension of elbow intraoperatively, patients with cubitus valgus deformity, patients with arthritis with synovitis over the posterior aspect of elbow, any implants in the posteromedial aspect of elbow and in revision surgeries. There is no consensus of the best method of anterior transposition but we prefer a subcutaneous anterior transposition to make sure that the nerve is lying free of any compression under the subcutaneous fat. We assure a complete proximal (till the medial intermuscular septum) and distal (till the separation of two head of flexor carpi ulnaris) release to assure release of all the potential sites of compression and allow a straight lie of the ulnar nerve after anterior transposition [Fig 5 and 6].

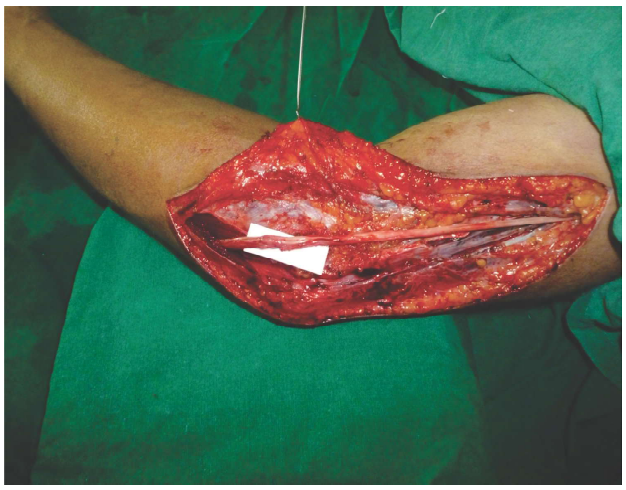

Fig. 5 Clinical picture showing the subcutaneous transposition of the ulnar nerve.

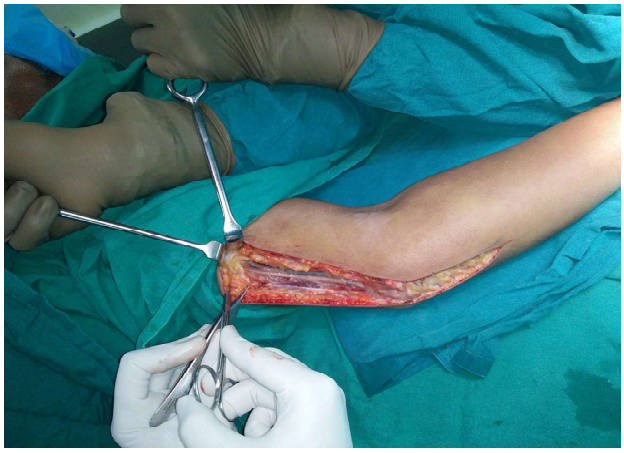

Fig. 6 Clinical picture showing the extent of the skin incision and release recommended for the complete decompression of the ulnar nerve from the medial intermuscular septum proximally and up to the penetration of the ulnar nerve between the two heads of the flexor carpi ulnaris distally.

Conclusion

Ulnar nerve entrapment is a well defined clinical entity; shortfalls lie in the data being isolated to largely mid to high socio-economic groups in developed countries with a dearth in lower socio-economic societies. The relevance relates to implementing findings from reviews in different health care models, where cost and litigation potential vary. Therefore, it would seem reasonable to approach a potential patient based on his symptom severity (moderate), and begin with an in-situ decompression procedure in an idiopathic entrapment syndrome with no obvious confounding factors without the need for special investigations. The importance of pre-operative counseling with respect to possible outcome failures and recurrent symptoms in our opinion is vital to gaining the trust and compliance of the patient. When primary procedures fail there is little doubt that thorough evaluation, exhaustive counseling regarding outcomes and complications are needed. The revision surgery has to be meticulous and more extensile to address all the potential sites of compression and avoid injury to cutaneous nerves. In spite of lack of literature consensus on various issues in cubital tunnel compression management, it is a troublesome condition for the patient and in a well selected patient the outcome of cubital tunnel release is quite satisfactory.

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