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Cubital tunnel syndrome

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- Cubital tunnel syndrome (CuTS) is the second most common compression neuropathy of the upper limb, presenting with disturbance of ulnar nerve sensory and motor function.
- The ulnar nerve may be dynamically compressed during movement, statically compressed due to reduction in tunnel volume or compliance, and tension forces may cause ischaemia or render the nerve susceptible to subluxation, further causing local swelling, compression inflammation and fibrosis.
- Superiority of one surgical technique for the management of CuTS has not been demonstrated. Different techniques are selected for different clinical situations with simple decompression being the most common procedure due to its efficacy and low complication rate.
- Adjunctive distal nerve transfer for denervated muscles using an expendable motor nerve to restore the axon population in the distal nerve is in its infancy but may provide a solution for severe intrinsic weakness or paralysis.

Keywords: cubital tunnel syndrome; neuropathy; ulnar nerve

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Introduction

Cubital tunnel syndrome (CuTS) describes dysfunction of the ulnar nerve (UN) in the region of the elbow. It is the second most common compression neuropathy in the upper extremity, with 25 male and 19 female new cases per 100,000 people in the United Kingdom every year.[1] Sensory symptoms are typical and paraesthesia, dysaesthesia and numbness affect predominantly the ulnar side digits in the hand. Motor symptoms result in loss of strength in power grip due to weakness in the flexor carpi ulnaris (FCU) and the ulnar flexor digitorum profundi (FDP). Intrinsic motor weakness affects delicate finger control and key pinch grip. Pain is a common feature and may be exacerbated by elbow flexion. Diagnosis is clinical and may be confirmed with neurophysiological studies. The role of imaging is poorly defined. Dynamic ultrasound may demonstrate abnormal subluxation of the nerve around the medial epicondyle at the elbow.

Non-operative management is recommended in mild cases of limited duration with no motor weakness. Surgery is recommended for persistent symptoms, severe symptoms, motor weakness and when non-operative measures have failed. There is no consensus on the optimum method of surgical management. Simple decompression (SD) is reliable in the majority of patients with a low complication rate. Adjunctive procedures including anterior transposition (AT) and medial epicondylectomy (ME) may be performed in the setting of nerve instability identified at surgery or following decompression.

This aim of this review is to describe the current theory on the pathophysiology of nerve compression and the defining characteristics for a diagnosis of CuTS, to outline severity classification, and to explore the evidence to support different surgical strategies and outcome assessment.

Anatomy

The UN arises from the medial cord of the brachial plexus and contains nerve fibres from the C8 and T1 spinal nerve roots. It lies medially in the upper arm, passing obliquely through the intermuscular (IM) septum. The arcade of Struthers is an aponeurotic band between the medial intermuscular septum and the medial head of the triceps approximately 8 cm proximal to the medial epicondyle. At the elbow, the ulnar nerve passes posterior to the medial epicondyle in the retrocondylar groove where it enters the cubital tunnel. The tunnel is bordered medially by the medial epicondyle and laterally by the olecranon process. The floor is formed by the posterior and transverse bands of the medial collateral ligament (MCL) of the elbow joint capsule. The roof is formed by Osborne's fascia with a thickened proximal edge termed Osborne's band. Distally

the fascial roof merges with the aponeurosis between the humeral and ulnar heads of the FCU.

The UN provides a proximal posterior branch to the ulnar head of the FCU before the nerve enters the cubital tunnel and a distal anterior branch to the humeral head of the FCU within the tunnel. It enters the forearm between the two heads of the FCU, then lies between the FCU and the FDP, which is supplied on its ulnar half by the UN approximately 4 cm distal to the medial epicondyle. In the distal third of the forearm, a dorsal sensory branch arises from the UN to supply the dorsal ulnar hand and digits. Within Guyon's canal the superficial ulnar nerve provides a branch to innervate the hypothenar skin and then divides to the ulnar digital nerve (DN) to the small finger and the fourth webspace common digital nerve, supplying innervation to the radial DN to the small and the ulnar DN of the ring fingers. The deep branch of the UN passes around the hook of the hamate and then provides motor innervation to the hypothenar muscles. The flexor pollicis brevis may have dual innervation from both the deep branch of the UN and the motor branch of the median nerve.

Posner described five potential sites of ulnar nerve compromise at the level of the elbow. These are the intermuscular septum, the area of the medial epicondyle, the epicondylar groove, the cubital tunnel, and the exit of the UN from the FCU.[2]

Pathophysiology of ulnar neuropathy at the elbow

The UN is subjected to compression, traction and frictional forces at the elbow during normal flexion and extension cycling.[3] The nerve may be dynamically compressed during movement, statically compressed due to reduction in tunnel volume or compliance, and tension forces may cause ischaemia or render the nerve susceptible to subluxation, further causing local swelling, compression inflammation and fibrosis.

Compression

Compression of the UN by Osborne's fascia against the medial epicondyle is implicated in the development of CuTS.[4] The cubital tunnel is round in cross section in extension, becoming ovoid in flexion. Compression is maximal in full flexion at 135 degrees with a 55% decrease in the volume of the tunnel.[5,6] Flexion also results in a decrease of the canal height of approximately 2.5 mm,[7] a reduction in sagittal curvature, increasing distance between the medial epicondyle and the olecranon by 5 mm for every 45 degrees of flexion with a consequent 40% increase in the length of Osborne's band.[8] The pathological changes from compression are related to the force and duration.

Dynamic forces will leave the nerve susceptible to intermittent ischaemia. Sustained compression increases hydrostatic pressure in the fluid columns in the endoneurial tubes. Damage to the blood–nerve barrier follows with extravasation of fluid and intravascular proteins leading to oedema, and subsequently intraneural fibrosis. The fluid distorts the Schwann cells at the nodes of Ranvier, blocking conduction. Schwann cell death may follow severe and sustained compression. The fibrosis increases the nerve modulus of elasticity, further increasing strain and impeding interfascicular gliding. The loss of the protective myelin sheath increases the susceptibility of the axons to further compression damage and eventually axon death follows.[9]

Given the nerve sits in a bony groove with very little overlying soft tissue padding at the elbow, it may be vulnerable to external compression, for example prolonged pressure at an office desk.

Strain

The UN passes posterior to the rotational axis of the elbow. Tension in the UN as it passes posterior to the medial epicondyle is increased during elbow flexion.[10] The resultant strain renders the nerve vulnerable to ischaemia, compression against the bone and to subluxation. Sciatic nerves from rat specimens subjected to elongation forces demonstrated that 8% strain elongation correlated to a 50% reduction in blood flow and 80% reduction at 15% strain.[11] Elbow flexion to 90 degrees with shoulder abduction to 90 degrees results in a 14% strain.[12] The UN structure is locally adapted to areas of high strain. Surgical techniques to address focal compression points may modify the strain distribution along the nerve, and postoperative scar may limit excursion, further contributing to areas of high strain.[13] AT, ME and/or circumferential neurolysis may result in less redistributive strain.

Inflammation

Buzzard made the association between excessive limb use and ulnar neuritis at the elbow.[14] In some cases there may be additional anterior subluxation of the UN at the medial epicondyle. The result is a segmental inflammation with thickening of the nerve and increased susceptibility to further compression. Chronic inflammation results in increasing nerve stiffness, intraneural scar and potentially extraneural scar tether. Multiple factors may account for this subluxation including congenital or acquired arcuate ligament laxity, a shallow retrocondylar groove or medial triceps hypertrophy.[15] In 1975, Childress classified UN subluxation at the elbow.[16] In Type A, the nerve subluxes to the tip of the medial epicondyle when the elbow is flexed to 90 degrees or beyond. Here, the nerve is vulnerable to trauma and the stretch

forces are greater. In Type B, the nerve has greater excursion and subluxes past the medial epicondyle when the elbow is completely flexed.

Trauma

The UN is vulnerable to contusion injury from blunt trauma. Severe trauma may disrupt the axons, resulting in axonotmesis and, rarely, a neuroma in continuity can result. This should be distinguished from an inflammatory neuritis, often the result of repeated forceful elbow flexion, minor direct trauma or nerve instability. Distal humeral fractures are associated with UN irritation, and swelling can precipitate CuTS in a previously asymptomatic individual. During surgical management of a distal humerus fracture, direct trauma to the nerve at the elbow is rare; however, retraction can result in axonotmesis and postoperative scar tissue may tether the nerve and produce symptoms suggestive of an ulnar compression neuropathy.[17] Ulnar compression neuropathy may be precipitated by direct pressure in the anaesthetized patient in theatre or the ventilated patient in critical care. Careful elbow positioning, avoiding excessive flexion and padding behind the elbow are all measures that can reduce the incidence of this event. There is a strong association of compression neuropathy of the UN in wheelchair users when the flexed elbow is rested on the arm of the chair for long periods.[18]

Clinical presentation and investigations

The term 'cubital tunnel syndrome' was first coined by Feindel and Stratford in 1958.[19] Patients usually present with altered sensation in the UN territory with pain. The sensory symptoms may be described as tingling, pins and needles, electric shocks or burning sensations, usually into the small and ring fingers and also affecting the dorso-ulnar hand. Numbness of these digits is a common feature in moderate and severe CuTS. Motor weakness affecting the extrinsic UN innervated FDP to the small and ring fingers, plus weakness of the UN innervated hand intrinsic muscles is a feature of moderate CuTS, and wasting or paralysis is seen in severe CuTS. A postural deformity of the ulnar digits with hyperextension of the metacarpophalangeal joints and flexion of the interphalangeal joints is uncommon; however, it may become more apparent following decompression surgery when there is good functional recovery in the proximally innervated FDPs with persistent intrinsic weakness or paralysis.

Patients report pain in the elbow and medial arm radiating to the hand with numbness or tingling. Symptoms may present at night causing wakening from sleep, or during the day coinciding with prolonged or repeated elbow flexion. There may be reduced power grip. Patients may report a loss of control of the small finger position or clumsiness due to the intrinsic muscle dysfunction.

There may be wasting of the FCU in the proximal forearm, hypothenar and interossei wasting and loss of the muscle bulk of the adductor pollicis and first dorsal interosseous in the dorsum of the first webspace.

The UN must be examined at the elbow for instability on active and passive flexion. Elbow flexion and direct compression over the UN at the elbow for one minute may provoke the symptoms. Tapping over the course of the UN from distal to proximal may produce increased tingling symptoms at the points of maximal compression. Resisted little finger FDP muscle action is key to detecting a subtle motor deficit. The examiner's index finger should not be able to overpower the flexion of the distal interphalangeal joint in the small finger if motor function is normal.

The clinician must examine the whole upper limb to exclude other pathologies that may mimic CuTS, including cervical C8 radiculopathy, thoracic outlet syndrome, distal UN entrapment, carpal tunnel syndrome and hereditary motor and sensory neuropathies.

Imaging

High resolution ultrasonography (HRUS) may be used to assess the UN morphology, echogenicity, vascularity, mobility and the appearances of the surrounding anatomy to provide support for a diagnosis of CuTS. Static and dynamic ultrasound (US) imaging can identify underlying pathologies such as subluxation, tumours or ganglia.[20] HRUS can localize UN entrapment by measuring the cross-sectional area and the diameter of the nerve.[21,22] Beekman reported a sensitivity of more than 80% in an increased cross-sectional area or diameter of the UN at the elbow in diagnosing an ulnar neuropathy at the elbow.[23] Combining ultrasound with electrodiagnostic testing increases the diagnostic sensitivity to 98%.[22]

Magnetic resonance imaging (MRI) with specific sequences for MR neurography may demonstrate segmental pathological changes within the nerve with oedema and disruption of normal fascicular structure. US has been shown to be more sensitive than MRI (93% vs. 67%) and can better identify multifocal lesions. Both US and MRI have an equivalent specificity of 86%.[24] Comparable results were reported by studies investigating the difference between MRI and electrodiagnostic studies, with MRI being more sensitive than neurophysiologic testing.[25,26] There is no consensus in the literature on the role of imaging alone in the diagnosing of CuTS.

Table 1. McGowan grading classification

McGowan grade	Clinical features
Grade 1	Minimal lesion Paraesthesia and subjective clumsiness No motor deficit
Grade 2	Intermediate lesion Paraesthesia and sensory loss Motor weakness ± some muscle wasting
Grade 3	Severe lesion Paraesthesia and sensory loss Severe motor deficit and muscle wasting

Source: Adapted from McGowan AJ, 1950.[29]

Neurophysiological studies

The diagnosis of CuTS is usually made from clinical data combined with electrodiagnostic testing. Neurophysiology studies include nerve conduction studies (NCS) and electromyography (EMG). Nerve conduction studies demonstrate a reduction in conduction velocity across the elbow with a reduction in amplitude in the compound motor action potential (CMAP). However, there is a 10% false-negative rate as only a few functional axons are required for a 'normal' study.[27]

Higher CMAP amplitude predicts greater improvement in patients' self-reported outcome measure.[28] The current data available rely on subjective improvement postoperatively, without any objective functional outcome measures. As such, limited inference can be made about the reliability, accuracy and usability of CMAP in assessing and predicting functional recovery.

The signs of muscle denervation in moderate and severe CuTS include fibrillation potentials and positive sharp waves.

Severity classification

Traditionally, the McGowan–Golberg and the Dellon classifications are used to describe disease severity (Table 1).[4,29,30] Both systems are based on symptoms and/or clinician opinion, without objective measures of function. Grading is hierarchical, based on sensory symptoms alone, sensory symptoms with mild weakness and sensory symptoms with severe weakness and muscle wasting or paralysis. An alternative classification is that of Akahori and Gu, who incorporate electrodiagnostic criteria in the form of conduction velocities in their classification.[31,32] A review of 3024 patients surgically treated for CuTS was published by Bartels et al in 1998.[33] SD was the most commonly performed procedure in McGowan Grade 1 mild compression, with AT and ME procedures more commonly performed in the most severely affected Grade 3 patients. The results of SD were superior to those for AT and ME, perhaps reflecting selection bias, with the more complex procedures being used in the most severe cases and those with subluxation. There is no reliable grading system that directs treatment and predicts outcome.

Management

Non-operative management

The aim of non-operative measures is symptom control and prevention of progression. Activity modification reduces the irritation of the UN. The elbow should be maintained in extension whenever possible. Repeated elbow flexion should be discouraged and pressure over the UN at the elbow should be avoided. Extension splintage of the elbow may reduce nocturnal disturbance; however, splints are poorly tolerated. There is limited evidence for the use of injected steroids in the management of CuTS. In a comparative study of splints versus splint plus steroid injections in 12 UNs, the addition of steroids showed no added improvement in sensory or motor conduction.[34]

Surgical management

Superiority of one surgical technique for the management of CuTS has not been demonstrated. Different techniques are selected for different clinical situations with SD being the most common procedure due to its good efficacy and low complication rate.[35–38]

Simple decompression

SD is performed with a short incision placed over the cubital tunnel at the posteromedial elbow. The fascial structures superficial to the nerve are released but the UN is left in its original bed. SD is generally successful with a short-term complication rate of around 3.6% and revision surgery rate of 1.8% (n = 225).[39,40] Symptom persistence may be associated with incomplete release, scar tether or UN subluxation.

Endoscopic decompression

An endoscopic approach to the cubital tunnel, first introduced in 1992, has the aim of minimizing postoperative morbidity and scarring whilst reducing the risk of injury to the medial antebrachial cutaneous nerve (MABCN) which is vulnerable during open procedures.[41] MABCN injury was noted in 82% of patients treated for failure of primary cubital tunnel surgery.[42] A small open incision risks an incomplete release of all potential compression sites; however, the superior visualization offered by the endoscope may reduce this risk.[43] A comparative study between an endoscopic technique and in situ decompression reported less pain and greater satisfaction with the endoscopic approach.[44] Objective outcomes measures were not statistically different.

Anterior transposition

Transposition involves relocating the ulnar nerve to a position anterior to the axis of rotation of the elbow. In theory this should reduce tension and prevent segmental inflammation of an unstable UN as it subluxes around the medial epicondyle during elbow flexion. Different transposition locations are described as subcutaneous, intramuscular and submuscular. Transposition was reported by Learmonth in 1942.[45] Surgeons must be aware of the risk of neo-compression sites resulting from this intervention as well as the risk of damage to the longitudinal blood supply, the motor branches to the FCU and the potential for damage to the overlying terminal branches of the MABCN.

Key components of transposition surgery involve release of both the arcade of Struthers and the insertion of the medial intermuscular septum insertion to the medial supracondylar ridge. Failure to recognize these potential points of neo-compression created by repositioning the UN may result in persistent or recurrent symptoms after surgery. More distally, the periosteal origin of the FCU has the potential to compress onto the UN if the latter is not completely dissected from the surrounding tissue and mobilized. Minimal residual nerve compression over a prolonged period may cause perineural and epineural thickening, segmental demyelination and recurrence of UN symptoms.[46,47] A recent systematic review and meta-analysis failed to identify the optimum transposition technique in the management of CuTS, concluding that the evidence was limited.[48] A concern with the subcutaneous transposition technique is that persisting instability after transposition is often resolved with creation of a fascial sling in the subcutaneous plane using the fascia over the common flexor origin to prevent dorsal relocation of the nerve. The fascia may tether the ulnar nerve resulting in symptom persistence or recurrence. The superficial placement of the UN can be sensitive when the area is touched.

Medial epicondylectomy

In situ decompression of the UN along with ME aims to decompress and detension the UN at the elbow.[49] Osteotomy and removal of the medial epicondyle allows for controlled anterior subluxation of the nerve and eliminates the abnormal sagittal plane movement during elbow flexion. The nerve lies in a similar plane to the anterior transposition described above; however, secondary compression points are avoided through release of the nerve at the arcade of Struthers, and excision of the insertion of the medial intermuscular septum. The common flexor origin is repaired over the exposed bone at the resection site. The UN lies in a more direct course without distortion as it passes deep to the FCU. This approach to CuTS transposes the nerve away from the hostile retrocondylar environment while preserving its blood supply. Care needs to be taken to prevent injury to the MABCN.

The most common complications reported are pain at the osteotomy site (15–52%), flexion contracture at the elbow (1–18%) and elbow instability (1–10%). There is a lack of consensus regarding the amount of bone excision required as flexion contractures and elbow instabilities are reported at higher rates post complete ME compared to a partial or minimal approach; however, inadequate bone excision may leave a ridge that irritates or tethers the UN.[50] The medial collateral ligaments are at risk of compromise with an osteotomy of more than 19% of the medial epicondyle,[51] but maximal nerve decompression may be achieved by adopting an oblique osteotomy instead of relying on bone mass. Other potential complications include flexor/pronator weakness, elbow stiffness and recurrence of symptoms.

Adjunctive procedures in severe cubital tunnel syndrome

Supercharging end-to-side nerve transfer (SETS)

CuTS in its most severe form has axonopathy. Owing to the distance between the cubital tunnel and the innervated motor end plates in the hand, in severe cubital tunnel, one does not anticipate much motor recovery after decompression of the cubital tunnel. Recovery of function in such cases is either through removing the compression and salvaging function in intact axons with some degree of physiological conduction block, perhaps from demyelination, or through collateral sprouting of a few intact axons at the neuromuscular junction, adopting adjacent denervated muscle fibres and creating large motor units. Axon regeneration is unlikely due to intraneural fibrosis and apoptosis of chronically compressed axons. Supercharging nerve transfer involves transferring an intact and expendable motor nerve in the vicinity of the end target to the chronically denervated distal motor nerve to restore the axon population in the distal nerve. The timing of axon loss and the uncertainty regarding the degree of residual conduction block preclude a direct end-to-end coaptation. The end-to-side technique has been suggested as a method of achieving improved intrinsic function, without damaging any retained axons at the site of nerve transfer. The consistent topography of the UN in the distal forearm allows for the targeted transfer of the terminal pronator quadratus branch of the anterior interosseous nerve to the motor component of the UN after creating an epineural window.[52] The technique is controversial, with emerging evidence to support its use; however, there is persisting uncertainty regarding the relative contributions of the decompression, collateral sprouting and neo-innervation to any functional recovery.

Tendon transfers

The motor weakness and paralysis that accompanies severe and chronic CuTS can be reconstructed using

tendon transfers. Anti-claw procedures prevent metacarpophalangeal joint (MCPJ) hyperextension and improve grasp. Reconstruction of intrinsic function in the finger for interphalangeal joint (IPJ) extension may be required if there is no active IPJ extension when the MCPJ is passively flexed. Adductorplasty and tendon transfer to the first dorsal interosseous improves key pinch grip. Tendon transfer can improve small finger ulnar deviation due to loss of the palmar interosseous function at the fourth webspace. Extrinsic FDP function rarely requires restoration other than in the most severe cases. Buddying the ulnar FDPs to the median innervated FDP middle usually provides sufficient function.

Outcome assessment

Determining the optimum method of surgical management for CuTS requires a standardized approach to outcome reporting. Co-morbidities may adversely affect the outcome from surgery.[53] Pain resolution is to be anticipated in the majority of cases. Sensory recovery is common in mild and moderate cases. Although some limited motor recovery may be expected in a select number of McGowan 3 cases, useful motor recovery is unlikely in the severe cases. Although objective measures of sensation and motor function are valuable, they may not adequately reflect patient perceptions of the outcome and a disease-specific patient-reported outcome measure is desirable.[35] The Patient Rated Ulna Nerve Evaluation (PRUNE) scoring system is a 20-item scale that measures pain, sensory and motor symptoms, and functional disability in patients with UN pathology. It has been proven to be a reliable, valid and responsive assessment tool of both symptoms and function.[54] Either total or component scores can be used in the decision-making process with a minimum change in score of 7 points required to indicate a change in symptoms and disability. Alternative scales such as the Disabilities of the Arm, Shoulder and Hand (DASH) questionnaire, 36-item Short Form Health Survey (SF-36) and the Bishop Score are either less responsive or lack reporting of validity and reliability.[55–58].

Conclusion

Cubital tunnel syndrome is common, with little consensus on the optimal surgical management approach. Simple decompression results in predictable relief of pain and treatment satisfaction in the majority of cases. Nerve subluxation and severe motor loss may favour a different approach using medial epicondylectomy or anterior transposition. Functional improvement and patient-reported outcomes are less predictable. Adjunctive distal nerve transfer remains unproven but may provide a solution for severe intrinsic weakness or paralysis.

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