ELSEVIER

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Ulnar Neuropathy About the Elbow

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Ulnar neuropathy about the elbow has been described and debated for years, though there is no consensus on the best surgical treatment. Current meta-analyses show equivalent outcomes after in situ decompression and anterior transposition. Recent trends have been toward in situ decompression through limited incisions or with endoscopic techniques. Further research is needed to determine the strength of prognostic factors, such as duration of symptoms and preoperative severity of disease, which may change our threshold for surgical intervention. The ulnar nerve should also be addressed in cases of distal humerus fracture, generally with in situ decompression, as transposition does not appear to decrease rates of ulnar neuropathy. In elbow contractures, prophylactic ulnar nerve decompression should be liberally used, especially in cases of heterotopic ossification, scarring about the nerve, and preexisting symptoms, to prevent postoperative and delayed-onset ulnar neuropathy.
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Background

Although a seemingly simple, anatomical problem, the pathophysiology and optimal treatment for ulnar neuropathy around the elbow remain topics of debate and dissension. Treatment trends tend to be cyclical. In 1922, Buzzard[1] was already debating anterior transposition vs in situ decompression when he remarked, "I am inclined to think that perhaps a better result might have been obtained if in addition to releasing the nerve from constriction…it had been placed in front of the elbow." Recently, the trend has been in situ decompression, though the matter is far from settled.

Anatomy

After originating from the C8 and T1 nerve roots, the ulnar nerve arises as the larger terminal branch of the medial cord of the brachial plexus. In the upper arm, it runs inferior and medial to the brachial artery. Approximately 8 cm proximal to the medial epicondyle, the nerve passes under the arcade of Struthers, from the anterior to the posterior compartment of the arm, to lie on the medial head of the triceps. The arcade of Struthers is a deep fascial band between the medial intermuscular septum and the triceps. The nerve then becomes more superficial as it runs along the ulnar groove, which begins 3.5 cm proximal to the medial epicondyle.[2] The nerve wraps posteriorly around the medial epicondyle before passing through the cubital tunnel. The olecranon, elbow capsule, and the posterior oblique and transverse ligaments of the medial collateral ligament (MCL) of the elbow[3] lie at the floor of the cubital tunnel; Osborne's ligament, running between the humeral and ulnar heads of the flexor carpi ulnaris (FCU), forms the roof (Fig. 1). The nerve exits the cubital tunnel, passing between the heads of the FCU. A common flexor aponeurosis has also been described, which lies anterolateral to the nerve, stretching between the FCU and flexor digitorum superficialis. The common flexor aponeurosis ends at an average distance of 3.7 cm distal to the medial epicondyle.[4] The medial antebrachial cutaneous (MABC) nerve innervates the skin in this region, and its branches are at risk with any surgical intervention. Most patients have 2 major cutaneous branches (range: 1-4 branches) off the posterior branch of the MABC nerve, one crossing an average distance of 3.1 cm distal to the medial epicondyle and the other crossing 1.8 cm proximal to it.[5]

The ulnar nerve is typically described to have 5 potential sites of compression about the elbow: the medial intermuscular

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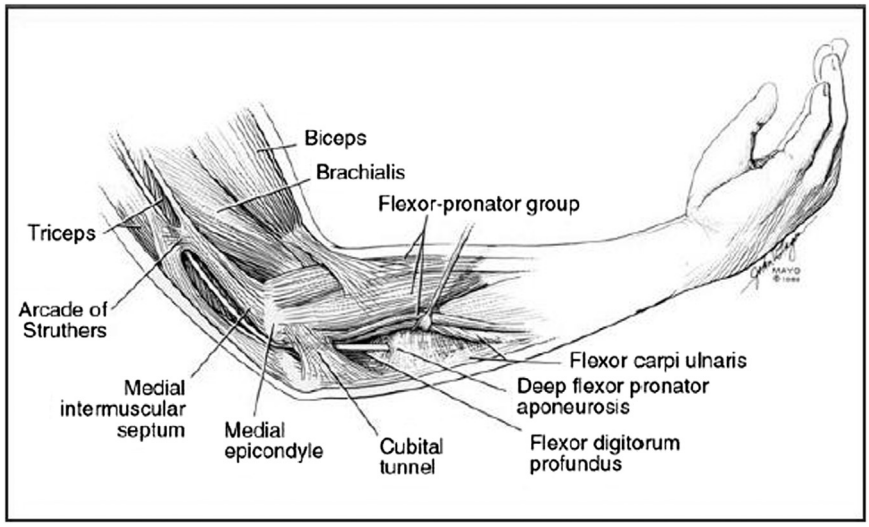

Figure 1 Sites of compression of the ulnar nerve around the elbow. (Adapted with permission from the Mayo Foundation for Medical Education and Research. All rights reserved.)

septum, arcade of Struthers, medial epicondyle, cubital tunnel proper (Osborne's ligament), and the deep flexor-pronator aponeurosis.[6] Anomalous structures and aberrant anatomy can also cause compression. Cysts,[7,8] elbow joint osteophytes, anconeus epitrochlearis muscles, cubitus valgus deformity, and medial epicondyle nonunions have all been identified as causes of cubital tunnel syndrome.

A Simple Compression Neuropathy?

Evidence from cadaver studies indicates that cubital tunnel syndrome is not solely a compression neuropathy; elbow flexion causes dynamic changes in the ulnar nerve cross-sectional area,[9] cubital tunnel volume,[9,10] intraneural pressures,[9] and nerve length.[11] Gelberman et al[9] demonstrated significantly greater intraneural pressure within the cubital tunnel compared with extraneural pressure with elbow flexion beyond 90°. The increase in intraneural pressure correlated with decreased cross-sectional area of the cubital tunnel and ulnar nerve with elbow flexion.

With elbow flexion, there is a clear increase in distance that the ulnar nerve must travel. However, there is disagreement whether this increase in distance comes from intrinsic slack in the nerve[12] or from nerve stretching.[11] If a site of compression or scarring develops, the nerve may become tethered, causing pathologic stretch of the nerve around the elbow during flexion. This was confirmed when redistribution of the nerve lengthening was observed after in situ decompression; the majority of additional length in flexion came from the area of the nerve proximal to the cubital tunnel, while there was a decrease in nerve stretch within and distal to the cubital tunnel.[13] Changes in nerve length may not be eliminated by anterior transposition. After anterior transposition, the nerve experiences a 23% increase in length with elbow extension, which is equivalent to the stretch it experiences in situ during flexion.[13]

Presentation and Examination

Patients generally present around the age of 40-70 years[7] and are more often men than women. Cubital tunnel syndrome is uncommon in children and is usually associated with trauma or with ulnar nerve subluxation.[14] The initial complaint is usually numbness, especially at night, in the small finger, ulnar-half of the ring finger, and ulnar side of the hand. There may also be elbow and forearm aching or pain. Patients with more advanced disease report weakness of grip, especially when attempting application of torque, such as while opening a jar.[15] As the lesion progresses, the hand becomes weak and clumsy. In severe cases, intrinsic muscle atrophy becomes apparent, and claw hand deformity may develop.[16]

The diagnosis is made clinically. The elbow is examined for any structural causes, such as cubitus valgus or space-occupying lesions that may compress the ulnar nerve. The ulnar nerve is palpated through a range of motion (ROM) to evaluate for subluxation over the medial epicondyle. The hand should be examined for intrinsic muscle wasting and a Wartenberg sign (inability to adduct the little finger). Sensation is also assessed. Semmes-Weinstein monofilament and vibration testing may detect earlier stages of sensory impairment, whereas static 2-point discrimination and moving 2-point discrimination are altered in more advanced cases.[15] Motor testing is also performed, including evaluation for a Froment sign. The patient is asked to pinch a piece of paper between the thumb and radial side of the index finger (key pinch) as the examiner attempts to pull the paper away. In individuals with ulnar nerve palsy (weak adductor pollicis), the thumb flexes at the interphalangeal joint to compensate (anterior interosseous nerve innervated flexor pollicis longus), producing a positive Froment sign.

Provocative maneuvers have high sensitivity and specificity: combined pressure over the cubital tunnel with the elbow in flexion for 60 seconds has a sensitivity of 0.98 and specificity of 0.95.[17] A Tinel sign over the ulnar nerve at the elbow was found to have a sensitivity of 0.70 and specificity of 0.98.[17]