CURRENT CONCEPTS

The Management of Persistent and Recurrent Cubital Tunnel Syndrome

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Cubital tunnel syndrome (CuTS) is the second most common compressive neuropathy in the upper extremity. There are considerable diagnostic and therapeutic challenges associated with treating patients after a failed primary procedure for CuTS. Distinguishing cases of recurrence versus persistence and identifying concomitant pathology can guide treatment. Conditions that mimic CuTS must be carefully ruled out and coexisting dysfunction of the medial antebrachial cutaneous nerve needs to be addressed. Results of revision procedures are not as reliable as primary procedures for CuTS; however, improvements in pain and paresthesias are noted in approximately 75% of patients. Nerve wraps represent a promising adjuvant treatment option, but long-term outcome data are lacking. External neurolysis and anterior transposition after failed CuTS procedures are supported by case series; multicenter, prospective randomized trials are needed to guide treatment further and improve outcomes. *(J Hand Surg Am. 2018;■(■):■−■. Copyright © 2018 by the American Society for Surgery of the Hand. All rights reserved.)*

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CUBITAL TUNNEL SYNDROME (CUTS) IS the second most common compressive neuropathy in the upper extremity.[1] Although primary ulnar nerve (UN) decompression with or without transposition can result in improved outcomes, reoperation rates have been variable.[2–4] It is estimated that 25% of patients treated for CuTS will have recurrence.[5] There are major diagnostic and therapeutic challenges associated with treating patients with persistent or recurrent CuTS after primary decompression. Atrophy and weakness from chronic UN compression can add additional morbidity and are unlikely to improve after revision procedures.

Symptoms of CuTS are multifactorial and can be related to external compression, decreased neural excursion from perineural scarring, and longitudinal tension in the nerve. Ruling out conditions that mimic CuTS and identifying associated pathology are particularly important in patients who have failed a primary procedure. Revision procedures result in pain relief and paresthesia reduction in 75% of patients, but results are inferior compared with primary procedures.[6,7] This article reviews the pertinent anatomy and current management options for patients with recurrent or persistent CuTS.

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ANATOMY

The UN originates from the C7 to T1 nerve roots, coursing posterior and medial to the brachial artery in the arm. Approximately 8 cm proximal to the medial epicondyle (ME), the nerve enters the arcade of Struthers and travels posterior to the ME through the

cubital tunnel. The Osborne ligament and ulnar collateral ligament serve as the roof and floor, respectively, of the cubital tunnel. The nerve then enters the leading edge of the flexor-pronator aponeurosis (arcuate ligament) between the 2 heads of the flexor carpi ulnaris (FCU) before traveling down the forearm between the FCU and the flexor digitorum profundus.

There is substantial anatomic variability in both the articular and FCU motor branches of the UN at the elbow. As many as 3 sensory articular branches can be present, originating as far as 7 mm proximal to the ME.[8] On average, 3 motor branches to the FCU are present, and whereas they commonly originate distal to the ME, more proximal FCU motor branches have been described.[8] In the case of prior surgery for CuTS, particularly anterior transposition, these branches may have been sacrificed. Distal to the ME, motor branches to the ulnar half of the flexor digitorum profundus are found. Riche−Cannieu and Martin−Gruber anastomoses may confound an already complex neurological examination.

From proximal to distal, points of compression in CuTS include the arcade of Struthers, the medial intermuscular septum, the Osborne ligament, the ME, and the deep flexor-pronator aponeurosis. Perineural scarring, the medial intermuscular septum, and flexor-pronator aponeurosis were found to be the most common points of residual compression at the time of revision surgery.[7,9,10] Less common points of compression have been described, including anomalous muscles on the medial side of the elbow, fascial bands within the FCU, the flexor digitorum superficialis aponeurosis, and the medial head of the triceps muscle (Video 1, available on the *Journal's* Web site at www.jhandsurg.org).[11] Osteophytes, heterotopic ossification, and masses (ganglion cyst) in the cubital tunnel also may cause UN compression.

The medial antebrachial cutaneous nerve (MABCN) is the terminal sensory branch of the medial cord of the brachial plexus, where it forms an anterior and posterior branch. Although the anterior branch can typically be found 2 to 3 cm anterior to the ME, it can be located proximal, at or distal to the ME, which makes it susceptible to injury during surgery. Multiple terminal branches may be in the surgical field of a large revision exposure. A neuroma of the MABCN is found in as many as 73% of revision cases.[11] Patients with local hypersensitivity anterior and numbness posterior to the prior incision likely have a neuroma present.

Ulnar nerve stability relative to the cubital tunnel can contribute to intraoperative decision making and postoperative outcome. With dynamic testing and elbow flexion, the UN can be described as stable (posterior to the ME), perched (rests on the ME), or dislocated (remains anterior the ME).[12] We believe that increased longitudinal tension is also present in many cases of CuTS and repetitive motion can cause deformation of the nerve against the epicondyle in flexion as well as intraneural ischemia and subsequent fibrosis.[13] Although no reproducibly objective measure exists, we believe that nerves are pathologically tight if a finger cannot easily be permitted between the nerve and the posterior aspect of the ME at 90° flexion. Pathologic longitudinal tension also needs to be addressed at revision surgery by either a posterior partial medial epicondylectomy or anterior transposition of the ulnar nerve.

ETIOLOGY

Cubital tunnel syndrome is multifactorial, resulting from increased pressure on the UN, decreased neural excursion, and subsequent pathologic changes. Elbow flexion decreases cubital tunnel volume, increasing pressure on the UN. Pressure increases can be further exacerbated by perineural fibrosis and scarring from prior injury or surgical procedures, because the UN is less able to elongate with elbow flexion. Animal models have demonstrated that after decompression and anterior transposition, nerve ultimate strain, ultimate strength, and modulus are notably reduced compared with controls and nerve amplitudes begin to change with as little as 6% strain.[14]

It is helpful to categorize patients who have failed a primary CuTS procedure into 3 groups: those with persistent symptoms, recurrent symptoms, or new symptoms.[15] Patients with persistent symptoms, who experience either no relief or incomplete relief after a primary procedure, may have had an incomplete decompression of the UN, an inaccurate diagnosis, or irreversible intraneural pathology. Recurrent symptoms may result from scar and perineural fibrosis after surgery. New symptoms can result from iatrogenic creation of a secondary compression point (proximally at the intermuscular septum, at the point of transposition, or distally in the flexor-pronator mass) or from an iatrogenic nerve injury (typically an MABCN neuroma).

Authors recently identified risk factors for failure after a primary CuTS procedure. Young patients