Ulnar Nerve Tendon Transfers for Pinch

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KEYWORDS

• Key pinch • Lateral pinch • Tip pinch • Power pinch • Ulnar nerve • Tendon transfers • Pinchplasty

KEY POINTS

- In ulnar nerve paralysis, power pinch is significantly affected and can lead to difficulty with performing daily tasks.
- When nonoperative treatment fails, tendon transfers may be used.
- The primary muscle for power pinch is the adductor pollicis (AP), and secondary muscles are the first dorsal interosseous (DI) and the deep head of the flexor pollicis brevis (FBP).
- Preferred tendon transfers are extensor carpi radialis brevis (ECRB) to AP with z-lengthening for AP restoration, abductor pollicis longus (APL) to first DI tendon transfer using tendon graft for first DI restoration and splin flexor pollicis longus (FPL) to extensor pollicis longus (EPL) transfer thumb stabilization.
- Combinations of transfers should be evaluated on an individual basis.

INTRODUCTION

Anatomy

The adductor pollicis (AP) has the largest mass and cross-sectional diameter of any hand intrinsic muscle.[1] It is also able to produce the most force and work capacity of all the intrinsics.[2] It is composed of transverse and oblique heads, which are innervated by the deep branch of the ulnar nerve. The transverse head originates from the third metacarpal and inserts on the ulnar aspect of the thumb proximal phalanx and continues into the lateral bands. The oblique head shares its insertion with the transverse head by a conjoint tendon that attaches to the ulnar sesamoid bone of the thumb but originates from multiple locations. This includes the palmar aspects of the second and third metacarpals, the capitate, the intercarpal ligaments, and the flexor carpi radialis sheath. Together, the transverse and oblique heads contribute to thumb carpometacarpal (CMC) flexion and adduction, metacarpal phalangeal (MP) flexion, interphalangeal (IP) extension, and thumb supination. The deep head of the flexor pollicis brevis (FPB), also innervated by the deep branch of the ulnar nerve, runs almost parallel to the AP and mimics its function. Between the 2 heads of the AP, the deep motor branch of the ulnar nerve runs along with the radial artery, which course from dorsal to palmar to become the deep arch.[3]

The first dorsal interosseous (DI) muscle is the second largest intrinsic muscle of the hand in terms of mass and cross-sectional area.[1] It is a bipennate muscle, nearly always innervated by the ulnar nerve that originates from the radial side of the second metacarpal and the proximal ulnar aspect of the first metacarpal. The insertion is on the radial aspect of the base of the index finger proximal phalanx and into the radial lateral band.[3] Its primary action is to abduct the index finger, but it also assists the lumbrical in MP joint flexion and

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IP joint extension. Similar to the AP, the low fiber length/muscle length (FL/ML) ratio of DI makes it suitable to provide a large amount of force to allow for power pinch.[1] Together, the AP and DI contribute 75% of the adduction force of the thumb.

The FPB is a weak contributor to power pinch, as it has only one-third the work capacity, force, and mass of the AP.[1,2] The superficial (lateral) head, innervated by the recurrent motor branch of the median nerve, originates from the trapezium and the distal edge of the transverse carpal ligament and inserts on the radial aspect of the base of the thumb proximal phalanx and radial sesamoid. The deep (medial) head, innervated by the ulnar nerve, inserts on the ulnar aspect of the thumb proximal phalanx base along with the AP. The FBP assists in flexion at the MP joint of the thumb along with CMC flexion and IP extension.[3] Innervation can be variable with both heads being supplied by either the median or ulnar nerve or have dual innervation.[4]

Etiology of Ulnar Nerve Neuropathy

Etiology of ulnar neuropathy can be multifactorial and a broad differential must be considered by the surgeon. Ulnar neuropathy may be due to mechanical cause or systemic pathology. Metabolic derangements, such as diabetes and hypothyroidism, are common causes of generalized neuropathy and should be evaluated. In addition, hereditary neuropathies, such as Charcot-Marie-Tooth, commonly affect the ulnar nerve.[5] Mechanical causes of ulnar neuropathy are often compressive in nature and vary based on anatomic location. Ulnar nerve palsy is generally classified as high or low. Low ulnar nerve palsy is due to an injury at the wrist, whereas high ulnar nerve palsy injury is at the level of the elbow or above, affecting flexor carpi ulnaris (FCU) and flexor digitorum profundus (FDP) to the ring and small finger. Some mechanical etiologies of ulnar nerve palsies include the following:

1. At or near the elbow
   a. Compressive neuropathy (eg, cubital tunnel syndrome)
   b. Penetrating or blunt trauma
   c. Deformities (ie, cubital valgus)
   d. Rheumatoid arthritis
   e. Hemophilia leading to hematomas
   f. Subdermal contraceptive implants
   g. Compression during general anesthesia
   h. Previous elbow surgery (both open and arthroscopic)
2. Near the wrist or distal
   a. Compressive neuropathy
   b. Ganglion cysts
   c. Tumors
   d. Penetrating or blunt trauma
   e. Ulnar artery aneurysm

Clinicians should also consider local clinical conditions at the thumb and index finger as causes for alterations in a patient's pinch that is not due to ulnar nerve dysfunction. This includes congenital anomalies, previous trauma, inflammatory or osteoarthritis, and intrinsic/extrinsic muscle/tendon abnormalities. Last, more proximal nerve compression, such as lower trunk brachial plexopathy, cervical radiculopathy, or a Pancoast tumor, should be considered in the differential diagnosis.

Pathoanatomy

In patients with ulnar nerve paralysis, the main complaint is loss of effective power pinch and coordination between the thumb and index finger.[6] This can be exaggerated or minimized based on the location of the lesion and anatomic variations of innervation of the hand musculature (ie, Martin-Gruber or Riche-Cannieu cross-innervation patterns). In addition, dysfunction of the ulnar nerve can also lead to dyskinetic finger flexion and deformity such as claw hand, loss of active digital abduction/adduction including persistent little finger abduction, thumb interphalangeal joint hyperflexion, metacarpophalangeal joint hyperextension and severe interossei muscle wasting (**Fig. 1**). In complete high or low ulnar nerve palsy the only remaining adductors of the thumb are the extensor pollicis longus (EPL), extensor pollicis brevis (EPB), and the flexor pollicis longus (FPL), all which create a weak adduction force. Thus, lesions of both the ulnar and median nerve lead to

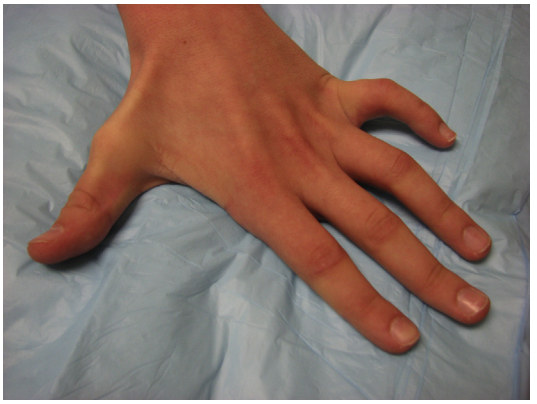

Fig. 1. A clinical photograph of a patient with an ulnar nerve palsy. Atrophy of the first DI and intrinsics is noted with abduction of the small finger and clawing.