CUBITAL TUNNEL SYNDROME

I. Background

The ulnar nerve originates from the C8 and T1 spinal nerve roots and is the terminal branch of the medial cord of the brachial plexus. The ulnar nerve travels posterior to the medial epicondyle of the humerus at the elbow and enters the cubital tunnel. After exiting the cubital tunnel, the ulnar nerve passes between the humeral and ulnar heads of the flexor carpi ulnaris muscle and continues distally to innervate the intrinsic hand musculature.

Ulnar nerve compression occurs most commonly at the elbow. At the elbow, ulnar nerve compression has been reported at five sites: the arcade of Struthers, medial intermuscular septum, medial epicondyle/post-condylar groove, cubital tunnel and deep flexor pronator aponeurosis. The most common site of entrapment is at the cubital tunnel.

Ulnar nerve compression at the elbow may have multiple causes, including:

A. chronic compression
B. local edema or inflammation
C. space-occupying lesion such as a tumor or bone spur
D. repetitive elbow flexion and extension
E. prolonged flexion of the elbow, as a habitual sleeping in the fetal position
F. in association with a metabolic disorder including diabetes mellitus.

Ulnar neuropathy at the elbow can occur at any demographic but is generally seen between 25 and 45 years of age and occurs slightly more often in women than in men.

II. Diagnostic Criteria

A. Pertinent History and Physical Findings

Patients often present with intermittent paresthesias, numbness and/or tingling in the small finger and ulnar half of the ring finger (i.e. ulnar nerve distribution). These symptoms may be more prominent after prolonged periods of elbow flexion, such as sleeping in the “fetal position”, sleeping with the arm tucked under the pillow or head, or with repetitive elbow flexion-extension activities. Subjects may progress to develop atrophy or weakness of the intrinsic hand musculature manifested as hand weakness or impaired dexterity.

Several provocative exam techniques have been validated to aid in the diagnosis of these patients. The elbow flexion test, in which the elbow is held in maximal flexion for one minute, may reproduce symptoms. Tinel’s test, in which the post-condylar groove is tapped by the examiner, may also reproduce symptoms. Patients may develop weak finger abduction secondary to interosseus muscle atrophy; weak small finger adduction may be noted (Wartenberg sign) and some patients may note that the small finger gets caught when placing the hand inside of a pocket. Patients may also be unable to grasp with a lateral pinch grip and instead compensate with a fingertip grip (Froment sign). Severe clawing of the ring and small fingers (i.e. flexion of the interphalangeal joints with extension of the metacarpophalangeal joints) may be noted secondary to interosseus and lumbrical muscle atrophy.
Other potential causes of medial hand numbness or weakness include nerve root compression at the cervical spine, brachial plexopathy, thoracic outlet syndrome and/or ulnar nerve compression at the wrist (Shea neuropathy, including entrapment of the ulnar nerve at Guyon’s canal).

**B. Appropriate Diagnostic Tests and Examinations**

1. Electromyographic and nerve conduction studies
2. Radiographs of the elbow
3. Magnetic resonance imaging of the elbow
4. Clinical laboratory tests to assess for potential causes of peripheral neuropathy

**C. Supporting Evidence**

1. Electromyographic and nerve conduction studies are particularly helpful in localizing the site of nerve compression, quantifying the degree of demyelination, evaluating patients with atypical symptoms, and/or assessing for alternative diagnoses. These studies may also aid in determining the prognosis for nerve and muscle recovery. Performing these studies with the elbow in flexion may increase sensitivity. Elbow radiographs may be helpful to identify osteophytes or bone fragments in patients with arthritis or prior trauma. MRI may be helpful if a space-occupying lesion is suspected, but otherwise is not routinely used. Clinical laboratory tests may help assess for potential causes of peripheral neuropathy including such as diabetes, pernicious anemia, chronic alcoholism, or hypothyroidism.

**III. Treatment**

**A. Outpatient Treatment**

1. **Conservative Management**
   
   i. **Indications**
   
   1. In the absence of intrinsic muscle atrophy, four to eight weeks of conservative treatment should be attempted.

   ii. **Treatment options**
   
   1. Activity modification to avoid elbow flexion and/or reduce cubital tunnel compression, such as use of an elbow extension splint, adjusting posture to reduce elbow flexion, using a hands-free headset for the phone and/or padding the elbow.
   
   2. Non-steroidal anti-inflammatory drugs may be used for analgesia.

   iii. **Rehabilitation**
   
   Exercise therapy may be utilized to improve strength, dexterity, and hand function.

   iv. **Supporting Evidence**
   
   Most cases of mild or moderate cubital tunnel syndrome will improve and/or resolve with conservative management.

2. **Ambulatory Surgery**
i. **Indications**
   1. Failure to respond to conservative treatment
   2. Intrinsic muscle atrophy or weakness
   3. Severe, persistent symptoms

ii. **Treatment Options** – requiring referral to an orthopedic surgeon, neurosurgeon, or hand surgeon
   1. Ulnar nerve release at the cubital tunnel (i.e. in situ decompression)
   2. Ulnar nerve release at the cubital tunnel with subcutaneous, intramuscular or submuscular transposition of the ulnar nerve
   3. Ulnar nerve release at the cubital tunnel with medial epicondylectomy.
   4. Endoscopic ulnar nerve release at the cubital tunnel

**Ulnar nerve recovery after revision cubital tunnel surgery is less consistent than that after primary cubital tunnel surgery.**

iii. **Rehabilitation**
   Post-operative rehabilitation is often directed by the surgeon.

iv. **Supporting Evidence**
   Given the similarity in outcomes reported between the surgical treatments for cubital tunnel syndrome, the choice of procedure is based largely on surgeon experience, as well as underlying etiology. Studies have demonstrated similar outcomes between in situ decompression and anterior transposition (subcutaneous, intramuscular or submuscular) of the ulnar nerve with a 65% to 96% patient satisfaction rate. Patients with recurrent disease following in situ decompression may benefit from subsequent anterior transposition of the ulnar nerve. However, revision surgery outcomes are often disappointing.

**B. Estimated Duration of Care**

1. Non-operative treatment – maximum medical improvement should be achieved by eight weeks after diagnosis.
2. Operative treatment – eight to twelve weeks post-operatively.

**PROTOCOL HISTORY:**
Passed: 6/18/1996
Amended: 3/22/2011
Amended: 9/27/2022