Compressive Ulnar Neuropathies at the Elbow: I. Etiology and Diagnosis

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Abstract

Ulnar nerve compression at the elbow can occur at any of five sites that begin proximally at the arcade of Struthers and end distally where the nerve exits the flexor carpi ulnaris muscle in the forearm. Compression occurs most commonly at two sites—the epicondylar groove and the point where the nerve passes between the two heads of the flexor carpi ulnaris muscle (i.e., the true cubital tunnel). The differential diagnosis of ulnar neuropathies at the elbow includes lesions that cause additional proximal or distal nerve compression and systemic metabolic disorders. A complete history and a thorough physical examination are essential first steps in establishing a correct diagnosis. Electrodiagnostic studies may be useful, especially when the site of compression cannot be determined by physical examination, when compression may be at multiple levels, and when there are systemic and metabolic problems.

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Ulnar nerve compression at the elbow is commonly accepted as the second most frequently encountered nerve entrapment in the upper extremity, exceeded in prevalence only by carpal tunnel syndrome. The incidence of ulnar nerve compression is probably greater if one includes those individuals who experience transient numbness and paresthesias when they lean on the flexed elbow or when the elbow is flexed for a prolonged period.

Anatomy and Etiology

The boundaries for potential ulnar nerve compression begin approximately 10 cm proximal to the elbow and end about 5 cm distal to the joint. The ulnar nerve can be compressed anywhere along this pathway at one or more of five sites (Fig. 1).

In the middle third of the arm, the ulnar nerve pierces the medial intermuscular septum and descends along the medial head of the triceps muscle. The first area of potential compression, which is the widest, begins proximally at the arcade of Struthers and ends distally near the medial epicondyle. The arcade of Struthers is a musculofascial band, 1.5 to 2.0 cm in width, which is located an average of 8 cm proximal to the medial epicondyle. In an anatomic study of cadaver extremities, it was present in 70% of specimens.[1] The arcade, which runs oblique and superficial to the ulnar nerve, is composed of the deep investing fascia of the arm, superficial muscle fibers from the medial head of the triceps (its most obvious component), and the "internal brachial ligament," which arises from the coracobrachialis tendon. The anterior border of the arcade is the medial intermuscular septum. The lateral border is formed by deep fibers from the medial head of the triceps.

The arcade of Struthers should not be confused with the far less commonly encountered ligament of Struthers. The ligament of Struthers is associated with compression of the median nerve. Although the ligament itself has not been implicated in compression of the ulnar nerve, compression by the supracondylar process has been reported.[2]

In the absence of an arcade of Struthers, the medial intermuscular septum can cause compression as the nerve passes over its edge, which is thicker distally than proximally. This can occur after anterior dislocation of the nerve or as a postoperative complication of ulnar nerve transposition when the septum has not been excised. The medial head of the triceps muscle can also compress the nerve in this

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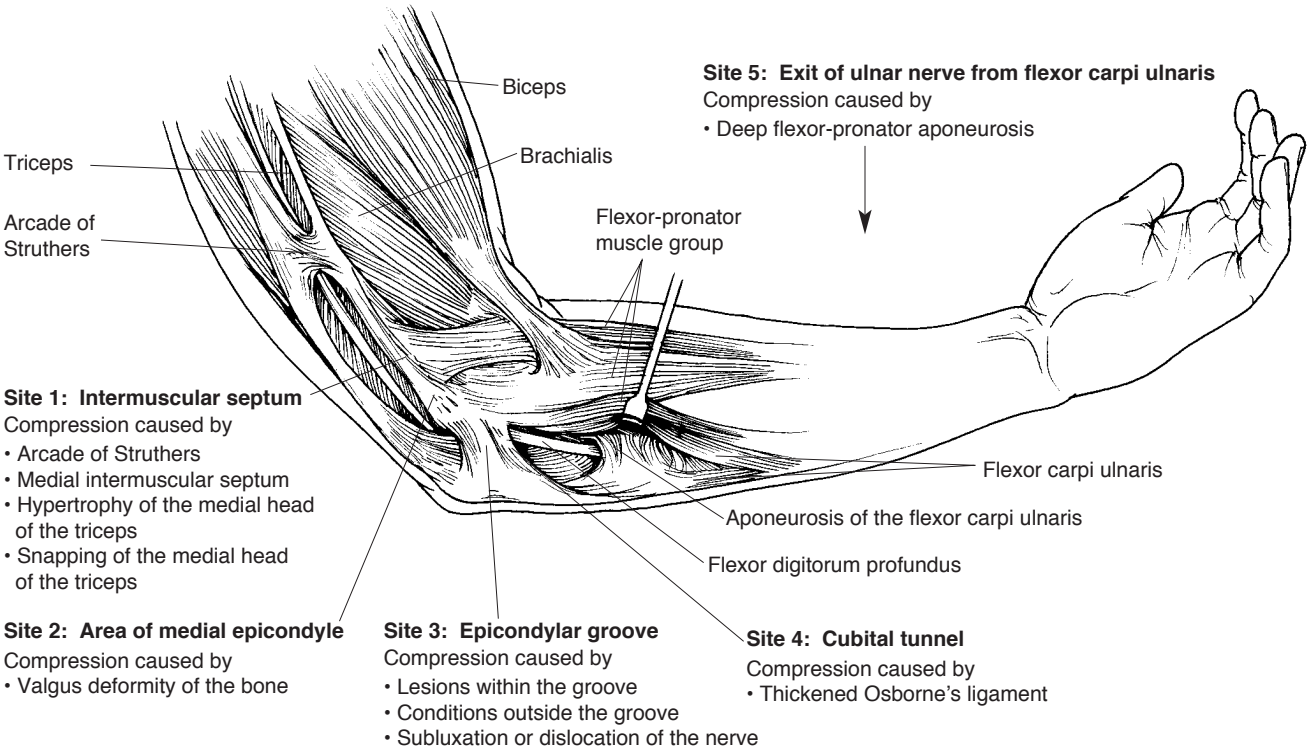

Fig. 1 The five sites for potential ulnar nerve compression and the causes of compression at each site. (Adapted with permission from Amadio PC: Anatomical basis for a technique of ulnar nerve transposition. *Surg Radiol Anat* 1986;8:155-161.)

area. The muscle head can be hypertrophied, as is commonly seen in bodybuilders, or it can snap over the medial epicondyle, causing a friction neuritis.

The second site of potential compression is the distal end of the humerus, at or just proximal to the medial epicondyle. Compression in this area develops as a consequence of a valgus deformity of the bone secondary to an old epiphyseal injury to the lateral condyle or a malunited supracondylar fracture. Ulnar neuropathy secondary to a humeral fracture was first described by Mouchet in 1914; soon thereafter it became known on the European continent as the "maladie de Mouchet." Two years later, Hunt introduced the term "tardy ulnar palsy" in the United States.

The third area of potential compression is the epicondylar or olecranon groove. This is a fibro-osseous groove, which is bounded anteriorly by the medial epicondyle and laterally by the olecranon and the ulnohumeral ligament; medially, the groove is covered by a fibro-aponeurotic band. In its passage through the groove, the ulnar nerve is accompanied by an anastomotic arterial system composed of the superior and inferior ulnar collateral arteries from above and the posterior ulnar recurrent artery from below.

Compression at this site can be caused by a wide variety of lesions and conditions, which can be grouped in three categories: lesions within the groove, conditions outside the groove, and conditions that predispose the nerve to displace from the groove. Lesions within the groove include fracture fragments and arthritic spurs arising from the epicondyle or the olecranon, hypertrophic bone, soft-tissue tumors, ganglia, osteochondromas, synovitis secondary to rheumatoid arthritis, infections (e.g., tuberculosis), and hemorrhage due to trauma or bleeding disorders, such as hemophilia.

Nerve compression secondary to conditions outside the groove is common among individuals who lean on the flexed elbow for prolonged periods of time, such as truck drivers who rest their elbows on the lower edge of the window frame while driving and patients confined to bed. External compression can also occur during surgery due to improper positioning of the arm. Many patients in whom symptoms develop after surgery are found to have had preoperative subclinical nerve compressions that were simply aggravated, but not caused, by the operation.[3] Another condition outside the groove that

can cause ulnar nerve compression is the presence of an anomalous anconeus epitrochlearis muscle that arises from the medial border of the olecranon and inserts into the medial epicondyle. In humans, the muscle is probably atavistic and is replaced by a band passing in the same direction as the muscle, called the epitrochleoanconeus ligament.[4]

The third category of neuropathy develops as a consequence of the nerve shifting out of the epicondylar groove with elbow flexion and returning to its normal position with elbow extension. The nerve can either subluxate onto the tip of the epicondyle or dislocate anterior to the epicondyle. Either situation can occur as a consequence of congenital laxity of the fibroaponeurotic covering over the epicondylar groove or a traumatic tear in the covering. It can also result from congenital hypoplasia of the trochlea or posttraumatic deformity of the medial epicondyle. Subluxation or dislocation of the ulnar nerve, both pathologic conditions, should not be confused with asymptomatic hypermobility of the nerve, which is usually bilateral and is found in approximately 20% of the population.[5] However, hypermobile nerves are predisposed to become inflamed by constant friction over the medial epicondyle. They are also at risk to be compressed, when the elbow is flexed, by external forces such as tight casts or splints applied for conditions unrelated to the ulnar nerve. A hypermobile nerve can also be inadvertently injured by an injection administered to treat medial epicondylitis.[6]

The fourth site of potential compression is where the nerve passes through a tunnel between the humeral and ulnar heads of the flexor carpi ulnaris muscle. This site and the epicondylar groove are the most common sites for ulnar nerve compression. The floor of the tunnel is the medial collateral ligament of the elbow. Its roof is a fibrous band that is a continuation of the fibroaponeurotic covering of the epicondylar groove. The fibrous band has been referred to as Osborne's ligament, the triangular ligament, the arcuate ligament, and the humeroulnar arch. In 1958, Feindel and Stratford named this area the "cubital tunnel." Although the term "cubital tunnel syndrome" is often used to describe compression of the ulnar nerve anywhere in the elbow, it more accurately refers to a neuropathy at this specific anatomic location.

The nerve is vulnerable to compression within the cubital tunnel during elbow flexion, because the tunnel normally narrows as Osborne's ligament stretches and becomes taut, and the medial collateral ligament relaxes and bulges medially (Fig. 2). Osborne's ligament stretches 5 mm for every 45 degrees of elbow flexion; from full extension to full flexion, it elongates 40%.[7] The cross-sectional contour of the tunnel changes from an oval in elbow extension to a flattened ellipse in elbow flexion.[8] Pressure within the tunnel increases 7-fold with elbow flexion and more than 20-fold when contraction of the flexor carpi ulnaris muscle is added.[9] These increases in pressure cause mechanical deformation of the nerve and, more important, compromise its intraneural circulation.

Animal studies have demonstrated the vascular effects of pressure. At a pressure of 20 to 30 mm Hg, there is impairment in flow in the epineurial venules and slowing of intracellular axonal support. However, capillary flow in the endoneurium and arteriolar flow in the epineurium and perineurium remain unchanged. As pressure increases, its effects become more profound. At 60 to 80 mm Hg, circulation ceases in the venules, arterioles, and capillaries, and the nerve becomes ischemic. If pressure is relieved within 2 hours, intraneural circulation is rapidly restored, although the nerve remains edematous for hours due to increased permeability of the epineurial vessels. Prolonged compression, which mimics many clinical situations, leads to permanent nerve damage.

The fifth site of potential compression is where the ulnar nerve leaves the flexor carpi ulnaris. Normally, the nerve enters the muscle at the cubital tunnel, remains intramuscular for a distance of approximately 5 cm, and then penetrates a fascial layer to lie between the flexor digitorum superficialis and flexor digitorum profundus muscles. The nerve can be constricted by this fascia, which

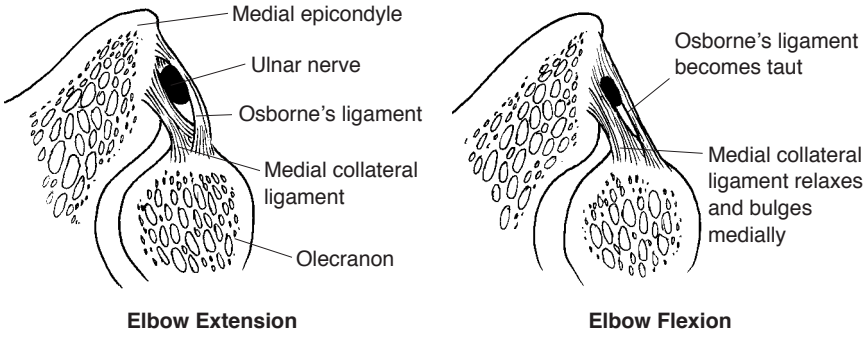

Fig. 2 Anatomy of the cubital tunnel in elbow extension and flexion. (Adapted with permission from Adelaar RS, Foster WC, McDowell C: The treatment of the cubital tunnel syndrome. *J Hand Surg [Am]* 1984;9:90-95.)

has been referred to as the "flexor pronator aponeurosis."[10]

Scarring anywhere along the course of the nerve can restrict its excursion and result in a traction injury. Normal excursion of the nerve with elbow motion is as high as 10 mm proximal to the medial epicondyle and 6 mm distal to the epicondyle.[11] The nerve itself stretches as much as 4.7 mm with elbow flexion, and additional stretching occurs with abduction and external rotation of the shoulder and extension of the wrist.

Diagnosis

Clinical Findings

A complete history, including assessment of work or leisure-time activities that aggravate the condition, and a physical examination are essential first steps in arriving at a correct diagnosis. Symptoms can vary from mild numbness and paresthesias in the ring and little fingers to severe pain on the medial aspect of the elbow and dysesthesias radiating distally into the hand and sometimes proximally to the shoulder and neck. The occurrence of mild paresthesias as an isolated symptom is not necessarily cause for concern, as it commonly occurs in individuals who keep their elbows flexed for prolonged periods of time during the day or at night while sleeping. Patients with early stages of nerve compression may not complain of any actual weakness, although they may be aware of some deterioration in hand function. They may report difficulty in carrying out certain tasks, such as opening bottles and jars, or may simply state that their hands fatigue quickly with repetitive activities.

The physical examination should always start at the neck. Any limitation of motion, particularly when accompanied by pain, may indicate cervical disk disease or arthritis. Axial compression of the spine may reproduce radicular pain. When compression in the brachial plexus is suspected, the presence of tenderness or a Tinel sign with percussion in the supraclavicular and infraclavicular areas should be checked.

Compression can also be due to thoracic outlet syndrome. There are a number of provocative tests for this condition, which are aimed primarily at obliterating the radial pulse. These tests include Adson's maneuver, Wright's maneuver, and Roos's test (also referred to as the overhead exercise test). There is also the costoclavicular maneuver, which involves scapular retraction into a military brace posture. All these tests are frequently positive in normal individuals; they are therefore nonspecific in the patient whose complaints are predominantly neurogenic. For a positive test to be considered relevant, it should reproduce the patient's symptoms and not simply obliterate the radial pulse.

The elbow is then inspected for deformity, and the normal carrying angle and active ranges of joint motion are measured. The ulnar nerve is palpated along its course for any enlargement or mass and in the epicondylar groove during elbow flexion for any subluxation or dislocation. Local tenderness anywhere along the course of the nerve aids in identifying sites of compression. A provocative test analogous to Phalen's test for carpal tunnel syndrome is the elbow flexion test, which involves maintaining the elbow in full flexion with the wrist in full extension for 1 minute (up to 3 minutes is considered by some to be a more appropriate duration). The test is considered positive if paresthesias or numbness occurs in the ulnar nerve distribution. As with Phalen's test, the elbow flexion test is more sensitive than specific, and false-positive results have been reported in 10% of normal individuals.[12]

Numbness in the ulnar nerve distribution of the hand is a common finding, which can vary in severity depending on the degree and duration of nerve compression. The sensory deficits usually include both sides of the little finger and the ulnar half of the ring finger, although normal variations in the sensory distribution of the ulnar nerve may extend the numbness to the middle finger or restrict it to the little finger. A sensory deficit over the dorsoulnar aspect of the hand and the dorsum of the little finger aids in differentiating a neuropathy at the elbow from one at the wrist. When nerve compression is at the wrist in the canal of Guyon (ulnar tunnel syndrome), dorsal sensibility remains intact because that area is innervated by the dorsal sensory branch of the ulnar nerve, which leaves the main body of the nerve at a more proximal level. Generally, it is 5 to 6 cm proximal to the ulnar styloid, but occasionally it is at the level of the ulnar head. Simultaneous compressive ulnar neuropathies at the elbow and wrist are common; in that instance, the Tinel sign will be positive at both locations.

Sensibility can be tested in several ways. Because the initial changes in nerve compression affect threshold, testing for vibratory perception and light touch with the use of Semmes-Weinstein monofilaments is more important than measuring static and moving two-point discrimination, which reflect innervation density. Innervation density is compromised only after there is axonal degeneration, which is more likely to occur with chronic nerve compression of at least several years' duration.

Muscle weakness generally occurs later than numbness, although occasionally inability to adduct the little finger (positive Wartenberg

sign) is an early presenting sign. Weakness affects the intrinsic muscles in the hand more commonly than the extrinsic muscles in the forearm, which can be readily explained by Sunderland's study of intraneural topography.[13] The motor fascicles to the intrinsic muscles, as well as the sensory fascicles, are situated more medial or superficial in the ulnar nerve at the elbow than the motor fascicles to the extrinsic muscles, and are therefore more vulnerable to compression (Fig. 3).

Comparing the strength of the ulnar nerve–innervated first dorsal interosseous muscle with that of the median nerve–innervated abductor pollicis brevis muscle is important. However, anomalous intrinsic muscle innervation is common, occurring in approximately 20% of the population.[14] The most common anomalous neural pathway is the Martin-Gruber communication in the proximal forearm, which carries motor fibers from the median nerve to the ulnar nerve. A similar but far less common connection between the two nerves exists in the distal forearm. In the hand, there is the Riche-Cannnieu connection between the motor branch of the ulnar nerve and the recurrent motor branch of the median nerve. These anomalous neural communications in the forearm and hand explain how the intrinsic muscles can be completely innervated by just one nerve, resulting in the so-called ulnar hand or median hand. More commonly, one or more intrinsic muscles have dual innervations.

In addition to these anomalous muscle innervations, the examining physician must also be aware of the various "trick movements" whereby intact muscles mimic movements normally provided by weakened muscles. Common examples of trick movements for the ulnar nerve–innervated intrinsic muscles are abduction of the index finger by the extensor indicis proprius, adduction of the thumb by the extensor pollicis longus, and abduction and adduction of the fingers by the extrinsic digital extensors and flexors, respectively. Trick movements are always weak movements, which can be detected by careful observation and by palpating the muscle being tested. A useful test for ulnar nerve function that is difficult to duplicate by any trick movement is the "crossed fingers" test. This test is based on the ability to cross one's middle finger over the index finger, the superstitious "good luck" gesture learned in early childhood.[15]

When intrinsic weakness is severe and associated with muscle wasting, it is indicative of chronic nerve compression of many months' or years' duration. Muscle weakness in these cases is commonly associated with clawing of the ring and little fingers and weakness of thumb pinch, characterized by a positive Froment's sign (flexion of the interphalangeal joint of the thumb) and a positive Jeanne's sign (hyperextension of the metacarpophalangeal joint of the thumb).

When extrinsic weakness occurs, it always involves the flexor digitorum profundus to the little finger. The flexor digitorum profundus to the ring finger may also be weak, but usually not to the same degree because its muscle fibers are frequently dually innervated by both the ulnar nerve and the anterior interosseous branch of the median nerve. Weakness of the flexor carpi ulnaris muscle is rarely encountered.

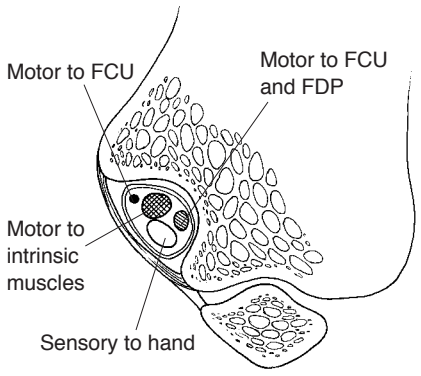

Fig. 3 The intraneural topography of the ulnar nerve in the epicondylar groove. Both sensory fascicles and motor fascicles to the intrinsic muscles are situated medially or superficially in the nerve. The motor fascicles to the extrinsic muscles, except for a small fascicle to the flexor carpi ulnaris (FCU), are situated laterally or deeper in the nerve and are therefore less vulnerable to compression. FDP = flexor digitorum profundus.

Imaging Studies

Radiographic examination of the elbow is always necessary. In addition to routine anteroposterior, oblique, and lateral views, a view profiling the epicondylar groove is useful in patients with arthritic and traumatic conditions in the elbow. Osteophytes or bone fragments from the medial trochlear lip are often seen in these patients.

The role of magnetic resonance imaging is limited. Although this modality is capable of visualizing swelling or enlargement of the ulnar nerve in the epicondylar groove as well as space-occupying lesions, its value is primarily academic. Magnetic resonance imaging is not essential for either diagnosing a neuropathy or determining appropriate treatment. Perhaps in the future, with continuing technical advancements, it will become more useful for detecting early nerve damage.

Electrodiagnostic Studies

Electrodiagnostic studies are never a substitute for a complete history and thorough physical examination. Although these studies are usually obtained when nerve compression is suspected, they are not essential when the diagnosis is obvious on clinical examination. Electrodiagnostic

studies can sometimes be misleading, and they have a false-negative rate similar to that in patients with carpal tunnel syndrome. False-negative studies occur when noncompressed nerve fibers are tested rather than the compressed fibers that are causing sensory symptoms or muscle weakness. Electrodiagnostic studies are important when clinical symptoms and findings are equivocal, when the site of nerve compression is uncertain or is thought to be at multiple levels, or when a polyneuropathy or motor neuron disease is suspected.

Electrodiagnostic studies include motor and sensory conduction velocity measurements and electromyography. Motor conduction is measured over a 10- to 12-cm segment of the ulnar nerve where it crosses the elbow. The skill and experience of the physician performing the test are important because anatomic variations can be encountered. The test should always be carried out with the elbow flexed, because conduction times are as much as 7 to 9 m/sec slower when the test is performed with the elbow in full extension.[16] The reason for this is that the true length of the ulnar nerve is frequently underestimated with the elbow in extension because the nerve is lax in that position. Slowing of motor conduction is absolute when it is less than 50 m/sec. Slowing can be relative when it is more than 10 m/sec slower across the elbow than it is farther distally in the forearm (from below the elbow to the wrist) or farther proximally in the upper arm (from the axilla to above the elbow). The age of the patient must be considered when evaluating conduction velocities because they can be as much as 10 m/sec slower than average in the elderly.

When nerve conduction is slowed, it is often accompanied by a drop in amplitude of compound muscle action potentials (CMAPs). When present, short-nerve-segment stimulation (the "inching" technique) can be used to localize the lesion.[17] This technique involves stimulating the nerve at 2-cm intervals across the elbow. When the points of maximum conduction delay and drop in amplitude are at or just proximal to the medial epicondyle, compression is probably in the epicondylar groove; when they are 2 cm distal to the epicondyle, compression is probably at the cubital tunnel.

A Martin-Gruber communication in the forearm can also lead to confusing results, as the hypothenar and first dorsal interosseous muscles are dually innervated by fibers from both nerves. Consequently, the CMAP amplitude for these intrinsic muscles will normally be greater when the ulnar nerve is stimulated at the wrist rather than at the elbow, because at the wrist the ulnar nerve also contains fibers from the median nerve. The amplitude at the elbow will normally be decreased, which may be misinterpreted as a conduction block. When ulnar nerve compression is present, weakness of the ulnar intrinsic muscles may be masked by the innervation they receive from the median nerve. Awareness of a Martin-Gruber communication is also important when planning surgery, as the point of connection is located 3 to 10 cm distal to the medial epicondyle.[18] When the connection is close to the epicondyle, there is a potential risk of damage during ulnar nerve transposition.

Sensory conduction studies are similar to motor studies in that the nerve is stimulated and a distant action potential is recorded. However, unlike motor fibers, sensory fibers can be stimulated in two directions: in the physiologic direction of conduction (from distal to proximal [orthodromic]) and in the opposite direction (from proximal to distal [antidromic]). For the ulnar nerve at the elbow, antidromic responses are easier to elicit, and are recorded by a ring electrode placed around the little finger. Sensory conduction of the dorsal cutaneous nerve of the hand can also be carried out to distinguish compression at the elbow from compression at the wrist.

Electromyographic studies demonstrate the presence of axonal degeneration in muscles. Because these changes occur with chronic neuropathies, electromyography is not as useful as conduction studies for the diagnosis of early compressions. When abnormalities are noted, they are initially seen in the first dorsal interosseous muscle, followed in frequency by the muscles in the hypothenar eminence.

Differential Diagnosis

The differential diagnosis includes any lesion that affects the origins of the ulnar nerve in the cervical spine (C8-T1 nerve roots) and/or the brachial plexus (medial cord). The most common spinal lesions are those due to cervical disk disease, followed by spinal tumors and syringomyelia. In the brachial plexus, the medial cord can be compressed by thoracic outlet syndrome or a Pancoast tumor. Electromyography of median nerve– and ulnar nerve–innervated intrinsic muscles (C8-T1) is helpful in differentiating lesions in the spine and brachial plexus from distal compressive neuropathies. While ulnar nerve–innervated intrinsic muscles may be abnormal with an ulnar neuropathy, the median nerve–innervated abductor pollicis brevis should be normal.

Not infrequently, the ulnar nerve is compressed at more than one site. In 1973, Upton and McComas noted that many patients with peripheral compressive neuropathies had concomitant nerve damage at the cervi-

cal roots.[19] They observed that when neural function was compromised at one level, the axons of that nerve were more susceptible to damage at another level, probably because of impaired axoplasmic flow. They aptly termed this condition "double crush." Occasionally, the nerve can be compressed at three sites ("triple crush").

The differential diagnosis of ulnar neuropathies should also include systemic and metabolic disorders, such as diabetes mellitus, hypothyroidism, alcoholism, malignant neoplasms, and vitamin deficiencies. However, the presence of any of these problems does not exclude the possibility of a concomitant compressive neuropathy.

Classification Systems

Classification of ulnar nerve function was introduced in 1950 by McGowan, who proposed a three-grade system.[20] Grade I lesions are classified as minimal, with symptoms of paresthesias and numbness but no weakness. Grade II lesions are intermediate, with wasting of the interosseous muscles. Grade III lesions are severe, with complete intrinsic muscle paralysis. Although both grade II and III lesions are characterized by numbness, the difference between the two grades is based solely on the degree of muscle weakness. McGowan's system is, therefore, essentially a preoperative rating of intrinsic muscle function.

Currently, there is no consensus on any scoring system. Available systems either rate subjective symptoms, which are difficult to quantitate, or fail to compare preoperative and postoperative conditions.

Summary

Compressive neuropathy of the ulnar nerve at the elbow is a common problem and can result in severe disability. Considering the anatomic course of the ulnar nerve through confined spaces and posterior to the axis of elbow flexion, Lundborg[21] concluded that the ulnar nerve was "asking for trouble." Normally, the nerve is subjected to stretch and compression forces that are moderated by its ability to glide in its anatomic path around the elbow. When normal excursion is restricted, irritation ensues. This results in a cycle of perineural scarring, further loss of excursion, and progressive nerve damage. Not uncommonly, a compressive neuropathy at the elbow is associated with additional compression proximally in the neck or brachial plexus and/or distally in the canal of Guyon. Multiple sites of compression can usually be identified from the history and physical examination. While electrodiagnostic studies may be helpful, their results must be correlated with the clinical picture for proper interpretation.

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