Outcomes for Peripheral Nerve Entrapment Syndromes

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Peripheral nerve entrapment lesions are common clinical disorders. Despite this fact, many neurosurgeons do not have vast training or experience in the diagnosis and management of patients with peripheral nerve entrapments or do not treat these patients because of lack of interest or referral patterns. There are many other reasons to develop a niche for this area of neurosurgery and to incorporate this into one's training and practice.

Peripheral nerve surgery is more than a time honored part of neurosurgical heritage. It is a dynamic subspecialty encompassing a wide range of pathology: entrapments (the most frequently treated), traumatic lesions, tumors, and the like. The management of patients who are successfully diagnosed with entrapments can be especially rewarding. It is a discipline centered on anatomic and pathoanatomic principles. Recent advances in diagnostic tools, treatments, and experimental studies have served as catalysts to fuel the excitement. Currently, conditions that are difficult to diagnose or those that are undiagnosed are still ripe for investigation in the future.

Although compression neuropathies typically affect certain nerves at specific sites (e.g., fibroosseous tunnels), any nerve may be compressed anywhere along its course. Entrapment neuropathies share a common pathophysiology (namely, localized ischemia from mechanical pressure) and pathogenesis. A set of well accepted tunnel syndromes has been described, including the most common carpal tunnel and cubital tunnel syndromes. Patients present with a predictable pattern of neurological symptoms (pain, sensory, motor complaints) specific to the affected nerve. The diagnosis can often be made by pattern recognition from a detailed clinical history and physical examination, and confirmed with electrical studies and/or imaging studies. The diagnosis of these entities is relatively easy when the following are present: a classic history, a characteristic physical examination (supplemented with certain provocative tests), and electrodiagnostic studies that are positive or imaging modalities that are revealing.

Typically, nonoperative therapy is recommended for at least 3 months and consists of a trial of anti-inflammatory or pain medication, splinting, avoidance of exacerbating activities or positions, physical therapy, and local steroid injections. If worsening symptoms (despite nonoperative treatment), severe symptoms, or advanced findings (i.e., significant atrophy) are observed at the time of initial presentation, operative intervention should be considered. Surgery consists of decompression of the nerve, combined, at times, with other procedures to provide a better path of or bed for the nerve (e.g., transposition).

This chapter will review the diagnosis, management, operative intervention, and outcomes in a spectrum of common and controversial entrapment lesions. To remain somewhat of a purist, I have decided to exclude other types of compressions, most notably mass lesions from the discussion. As the reader will see, the common and the controversial will converge in answering the questions of, for example, how to best diagnose or treat the common entrapments; whether or not certain groups of patients with neurologic conditions should be operated, some with a predictable favorable outcome (i.e., inflammatory conditions) and others with a progressively deteriorating course (metabolic); whether or not certain groups of patients with unexplained pain have entrapments (in fact, whether or not certain entrapments exist at all); and, finally, what are the outcomes really and how do we interpret them? In the end, one must consider exactly what and whom he or she is treating.

COMMON ENTRAPMENTS

Carpal Tunnel Syndrome

Carpal tunnel syndrome is by far the most common of the entrapment syndromes, and, as such, we will use it as a prototype in this chapter. The median nerve may become compressed as it passes along with nine flexor tendons beneath the transverse carpal ligament at the level of the wrist. Any condition that decreases the space within the carpal tunnel or increases the volume of its contents can result in the clinical manifestations of carpal tunnel syndrome (*Fig. 39.1*). A long list of causes has been put forth, including the most prevalent, idiopathic/developmental (e.g., tight flexor retinaculum or stenotic canal), inflammatory (nonspecific or inflammatory tenosynovial proliferation), metabolic/hormonal (e.g., diabetes, thyroid disease, acromegaly, pregnancy, etc.), or related to direct trauma (fractures) or indirect microtrauma (i.e., repetitive, cumulative activities).

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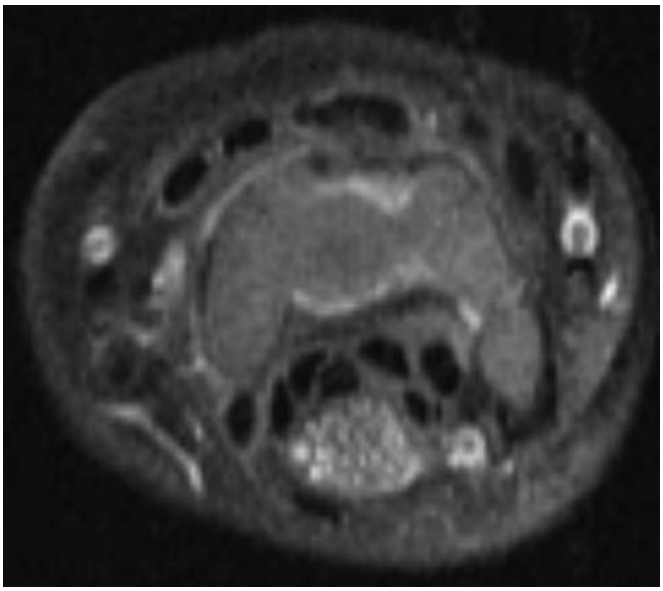

FIGURE 32.1. This toddler presented with macrodactyly of the middle finger and was found to have lipomatosis of the median nerve (fibrolipomatous hamartoma) within the carpal tunnel. This entity is known to produce median nerve symptoms and findings, without considering the overgrowth, that may mimic idiopathic carpal tunnel syndrome. T2-weighted fast spin echo image with fat saturation shows an enlarged hyperintense median nerve at the level of the carpal tunnel with fascicles displaced by interdigitating fat. These radiographic features are pathognomonic for lipomatosis of nerve.

Carpal tunnel syndrome often affects middle-aged women who present with symptoms developing gradually over a period lasting several months. The classic presentation of paresthesias in the radial 3½ digits, nocturnal symptoms (improved with shaking), and positional symptoms with wrist hyperflexion or extension (such as driving a car) are easily recognizable when they are all present. Pain frequently radiates proximally. These combination of symptoms are not always present. For example, nocturnal symptoms were found in only 71% of patients.[40,41]

Physical examination is characteristic when there is decreased sensation in the radial 3½ digits and weak thumb abduction or opposition, although motor and sensory testing is often normal, especially in those patients with early or mild compression. Vibratory threshold and monofilament testing are the most sensitve measures of sensibility testing. Motor weakness and atrophy are usually late findings. Provocative testing is positive in approximately 75% of patients. Various tests, including percussion over the median nerve (Tinel's test), Phalen's tests, compression test, and tourniquet tests, are helpful and are widely used. Phalen's test has been described as the most sensitive and Tinel's the most specific of these tests.[27]

To most people, electrodiagnostic testing remains the "gold standard" with sensitivities and specificities greater than 90% in most series. It is more sensitive than these methods of sensory testing. The author performs electrodiagnostic testing in all cases of carpal tunnel syndrome to support the diagnosis, quantify the severity, exclude other entities, and provide a baseline for those who may experience persistent symptoms. Abnormal electromyelograms (EMG) may be needed for approval of decompressive surgery. Normal studies, however, do not preclude the clinical diagnosis of carpal tunnel syndrome.[11,49] In contrast, abnormal studies should be taken in context and be correlated with the history and examination as they may be seen in patients without appropriate clinical symptomatology. Magnetic resonance imaging (MRI) scans are also a sensitive diagnostic modality. Features of compression include bowing of flexor retinaculum, increased signal within the median nerve, abnormal nerve configuration, increased distance between flexor tendons, and, in cases with denervation, increased signal in the thenar muscles.[12] I order MRI scans in patients in whom carpal tunnel syndrome is suspected, but in whom electrodiagnostic studies are normal, and in cases of failed carpal tunnel release.

Nonoperative treatments are routinely used as first-line agents in the management of most patients with mild or moderate compression. Splinting (in neutral position) and steroid injection frequently provide short-term relief in many patients.[28] Long-term relief is achieved in a relatively small percentage of patients when compared with operative intervention, which offers better long-term success. A single steroid injection can be valuable. Between 25 and 50% of patients are symptom-free 12 months after injection.[16] Improvement after nonoperative therapy often correlates with favorable surgical outcome. There has been no benefit of vitamin B6 (pyridoxine) in several recent controlled studies.

Various procedures are commonly used to treat carpal tunnel syndrome. Extended open, standard open, limited open (mini-open) with release performed under direct or indirect vision, and endoscopic (one- and two-portal), and even percutaneous techniques all have been shown to be extremely effective with success rates greater than 90%. Over the years, routine use of the extended open technique has fallen out of favor and more limited incisions have become more popular. Several prospective, controlled randomized (+/− blinded) studies have compared standard open or mini-open techniques with endosocpic methods. The major advantages of the more limited techniques seem to decrease with time and final results seem to be similar to open techniques. Advantages of endoscopic release include a quicker return to work (14–21 days shorter) in some studies, decreased scar tenderness, quicker return of grip and pinch strength, and, in some series, cost effectiveness. These potential benefits diminish after 3 months. To many, the disadvantages are that the

endoscopic or blinded mini-open techniques have steep learning curves and limited visualization. Despite the fact that the risk of neurovascular injury in most comparative studies is not higher using the endoscopic tecnique, it was borne out in a recent meta-analysis.[44] Complications leading to neurovascular and tendon injury have been reported with all techniques. The risk of incomplete ligament release is higher with endoscopic or limited open techniques. Routine neurolysis or tenosynovectomy is not supported by recent studies.

Failed carpal tunnel release, although relatively rare, still occurs. Incorrect diagnosis remains the most prevalent mangement error. With this in mind, surgeons should be especially vigilant when the history is not classic, the physical examination not chararacteristic, and the electrodiagnostic studies do not correlate with the history and physical examination (*Fig. 32.2*). The differential diagnosis is broad and proximal median nerve lesions, cervical radiculopathies, and wrist disorders should always be considered amongst other conditions.

Errors in surgical treatment also occur, most commonly from incomplete release of the transverse carpal ligament. Patients with failed carpal tunnel syndrome can be subdivided into those with persistent symptoms, recurrent symptoms, or new or increased symptoms. These classifications may help devise a management strategy: those with persistent symptoms may have had an incomplete decompression or an incorrect diagnosis; those with recurrent symptoms may have scarring around the nerve or reformation of the ligament; and those with new or increased symptoms may have had iatrogenic injury. No statistical difference has beeen shown between the type of primary procedure and outcome after revision surgery. Cobb and Amadio[14] showed that the majority of patients undergoing revision surgery have some degree of residual morbidity and only one-fourth are completely satisfied. Poor outcomes are seen more frequently in those patients with worker's compensation or ongoing litigation, who had multiple previous operations, and those with symptoms beyond the median nerve distribution and/or normal electrodiagnostic studies. "Success" rates range from 50 to 65%. Pain improvement should be the focus of most revision operations.

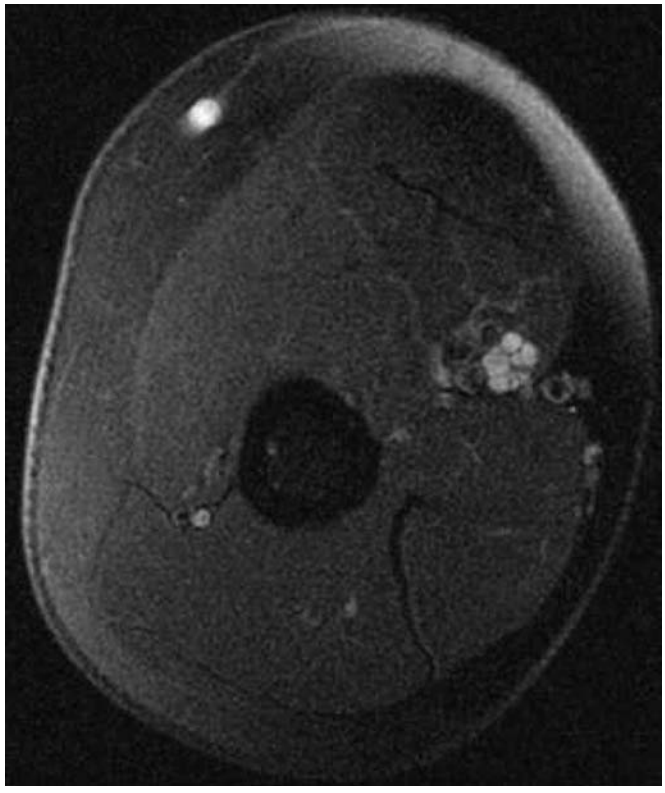

FIGURE 32.2. Failed carpal tunnel syndrome. A 55-year-old man presented with progressive numbness in the dominant left thumb, index, and middle fingers over 5 years. He did not benefit at all from left endoscopic carpal tunnel release performed 3 years earlier by his surgeon. He did not relate any paresethesisas or weakness, nocturnal, or positional symptoms. Motor examination was entirely normal. Two-point discrimination was greater than 15 mm in the radial 3 ½ digits versus 4 mm elsewhere. He had no percussion tenderness at the wrist. EMG performed at his outside facility showed only a non-localizable median neuropathy. On more careful testing, he had decreased palmar sensation, suggesting a more proximal lesion. Percussion tenderness over the course of the median nerve was positive at the level of the antecubital fossa and in the distal arm. Although no bony spur was felt on examination, the possibility of a supracondylar spur and ligament of Struthers compression was suspected, but elbow x-rays did not reveal any bony abnormalities. T2-weighted fast spin echo image with fat saturation shows enlarged hyperintense fascicles within the median nerve extending for over 10 cm in the distal arm. Fascicular biopsy proved it to be a perineurioma. The neurological examination and the MRI appearance has been stable for several years.

Cubital Tunnel Syndrome

Ulnar nerve entrapment at elbow is the second most common peripheral nerve compression syndrome. It occurs at the point where the ulnar nerve passes through the cubital tunnel. Despite its well known clinical presentation and findings, the diagnosis of ulnar nerve entrapment at the elbow is not always easy to establish and is, as a rule, more difficult to make than for carpal tunnel syndrome. Careful history and physical examination with elbow flexion test should be performed in all cases. Electrodiagnostic studies with inching studies across the cubital tunnel are helpful in establishing the diagnosis. In one study, electrodiagnostic studies could confirm a diagnosis of ulnar neuropathy in 75% of patients (68% could be localized to the elbow). In contrast, 97% were diagnosed with elbow MRI scans.[13] In this study, MRI scans proved to be more sensitive than EMG, though these may be limited by technical aspects or interpretative skills. Other

causes of medial elbow pain, ulnar sided hand paresthesias, or hand weakness should be considered in all of these patients.

Nonoperative techniques can yield excellent outcomes in 50% of those with mild symptoms and should be attempted.[20] Splinting should be performed with the elbow flexed approximately 60 degrees. Various surgical options are currently available to treat ulnar nerve entrapment at the elbow. The most commonly performed techniques include in situ decompression (either with simply unroofing the tunnel or with circumferential mobilization) and subcutaneous and submuscular transposition; intramuscular transposition and medial epicondylectomy are less frequently performed by neurosurgeons. Newer endoscopic techniques are being introduced.[47]

These techniques have success rates between 65 and 90% in many series.[21] Meta-analyses are available which summarize the large volume of disparate data.[7,18] Several recent prospective randomized, controlled studies have shown similar findings comparing simple decompression, and subcutaneous and submuscular transposition.[8,10,29] There has been no statistical difference between clinical and electrophysiological outcomes among groups treated with these techniques. Each procedure has advantages and disadvantages and each surgeon is an advocate of his or her own procedure of choice. Based on these recent studies, simple decompression may well gain further favor. Simple decompression is easier and quicker to perform and is less expensive.[9] It also has had a lower complication rate than the transposition groups. Still, these studies have been performed in a relatively small patient cohort. In the end, surgeons should perform the procedure they are most comfortable and experienced with which.

Persistent or recurrent symptoms are more commonly seen in patients with ulnar nerve entrapment than those who underwent carpal tunnel release. Failed ulnar nerve surgery at the elbow also has diagnostic and treatment errors. The most common diagnostic errors are due to misdiagnoses of radiculopathy, ulnar nerve compression at Guyon's canal, or thoracic outlet syndrome. Treatment errors relate most frequently to incomplete decompression, secondary compression, neuroma formation (i.e., medial antebrachial cutaneous nerve), and symptomatic ulnar nerve dislocation.

Other Peripheral Nerve Entrapment Sites/Tunnel Syndromes

Other peripheral nerve entrapment sites and tunnel syndromes have been described, well characterized, and validated. They share good success rates. The most common may include the ulnar nerve at the wrist (Guyon's canal) (*Fig. 32.3*), superficial radial nerve in the distal forearm, peroneal nerve entrapment at the fibular neck, and the lateral femoral cutaneous nerve at the inguinal ligament (meralgia paresthetica).

CONTROVERSIAL ENTRAPMENTS

"In these matters the only certainty is that nothing is certain." –Pliny the Elder

CONTROVERSIAL ENTRAPMENTS IN THE FACE OF NEUROLOGICAL DEFICIT

Certain mononeuropathies exist at locations where entrapments have been described, but where Parsonage-Turner Syndrome also has a predilection. Many patients note the onset of pain and/or neurological symptoms after an activity, which may suggest overuse or misuse. Deciphering whether or not there is a causal or a temporal relationship is important, but is not always done. Differentiating between these two distinct conditions may not be easy. Surgeons operating on patients with an inflammatory condition, rather than an entrapment, may be taking credit for the often favorable natural history of a medical condition that can be treated nonoperatively.

Physicians must inquire specifically about the typical presentation of the onset of pain approximately 10 days before a deficit ensues (typically, the pain then resolves); any possible relationship to a virus or immunization, pregnancy, or operation; previous unspecified neurological attacks that the patient or family members have had. They must examine for other neurological findings that may be occult, suggesting more widespread disease that may be diagnostic of Parsonage-Turner Syndrome. Electrophysiologists should also sample muscles predisposed to this process (spinati, deltoid, rhomboids, serratus anterior, anterior interosseous nerve-innervated muscles, etc.), specifically looking to support such a diagnosis– even looking bilaterally for subclinical evidence.

Although I do think that entrapment at these various sites does occur, I also acknowledge that an increasing number of my patients referred with entrapments are determined to have an inflammatory or immune-mediated process. I have treated several patients who experienced dramatic improvements immediately after surgical decompression. Still, in others, I did not find any evidence of compression and readily admit that patients likely improved, in spite of my surgery, due to spontaneous resolution of the underlying inflammation. In these cases, I generally feel that surgery should be performed 6 months after the onset of symptoms if there is no evidence of clinical or electrical improvement. This is in contrast to my recommendation that surgery be performed earlier after well defined trauma or in the presence of a mass lesion. Reliable tendon transfers should be considered in cases of permament deficit. In many situations, they can improve or restore useful function and sometimes diminish pain that is determined to be of a musculoskeletal nature.

Suprascapular Nerve Entrapment

A well-described entrapment site for the suprascapaular nerve is the transverse scapular ligament.[3,34] Patients with

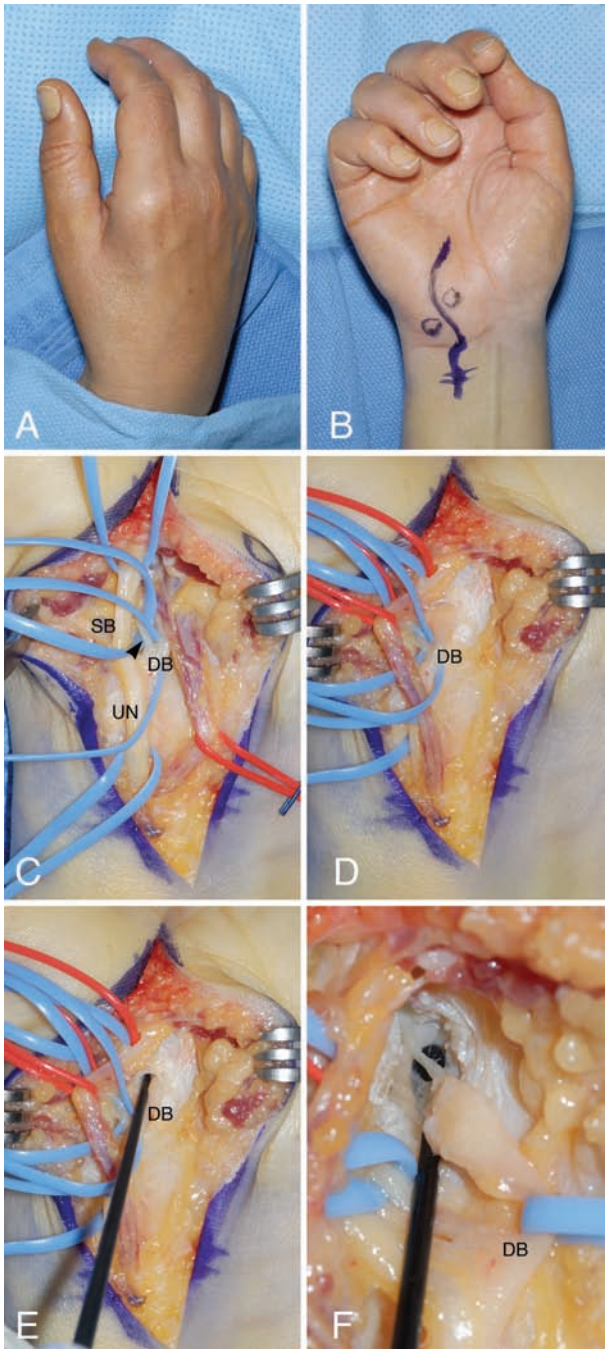

FIGURE 32.3. Deep ulnar nerve paralysis in the distal ulnar tunnel. A, this 52-year-old woman presented with progressive atrophy of the first dorsal web space and hand weakness due to ulnar innervated intrinsic paralysis over an 18-month period of time. She had preserved abductor digiti minimi function. Sensation in the hand was normal. Electrodiagnostic studies confirmed a lesion of the ulnar nerve isolated to the deep branch of the ulnar nerve. MRI scans did not reveal a mass (joint-related ganglion may produce this presentation). Entrapment of the deep branch (distal to the abductor digiti minimi branch) by the hypothenar fibrotic arch is a relatively rare, but well known, entrapment. One should always consider compression of this nerve at this proximal site typically present with posterior shoulder pain and weakness in abduction and external rotation. They may be found to weakness and atrophy of supraspinatus and infraspinatus function and have tenderness over the transverse scapular ligament. Electrodiagnostic studies show fibrillations in the supraspinatus and infraspinatus muscles and delayed conduction from Erb's point compared with normals and the patient's contralateral side. MRI scans are useful to rule out rotator cuff pathology, which is in the differential diagnosis, and to identify ganglia, which may be the compressive agent. Other conditions that need to be excluded, besides Parsonage-Turner syndrome, include C5 radiculopathy and distal suprascapular entrapment at the spinoglenid notch (patients present with isolated, painless weakness or atrophy of the infraspinatus muscle). Surgery to decompress the suprascapular nerve as it passes underneath the transverse scapular ligament often provides excellent pain relief, good improvement in abduction, and moderate improvement in external rotation. I prefer to perform the release through a posterior approach with the patient in a prone position using the intraoperative microscope. A 6-cm incision is made 1 cm superior to the scapular spine and centered on the coracoid process. The trapezial fibers are split and the supraspinatus is reflected inferiorly. The notch is defined, the ligament released, and the nerve decompressed.

Posterior Interosseous Nerve Syndrome

The arcade of Frohse has been described as the most common site for entrapment of the posterior interosseous nerve in the proximal forearm. Patients with posterior interosseous nerve syndrome characteristically present with posterior forearm pain and finger drop affecting some or all of the digits without sensory abnormality. Examination reveals paresis or paralysis of the finger extensors at the metacarpophalangeal joints. Wrist extension is in a radial deviation due to preservation of the extensor carpi radialis longus and loss of the extensor carpi ulnaris. The nerve may be compressed by mass lesions, such as elbow joint-related ganglia or lipo-

FIGURE 32.3 Continued other lesions in the face of such pure motor involvement, especially amyotrophic lateral sclerosis. B, surgical exploration was centered on the pisiform and hamate (in circles). C, the ulnar nerve (UN) was identified just proximal to the wrist crease radial to the flexor carpi ulnaris tendon. The ulnar vessels were mobilized. The superficial (SB) and deep branches (DB) were identified. The abductor digiti minimi branch (arrowhead) can be seen arising from the deep branch (DB). D, the deep branch (DB) has been traced to the fibrotic arch, the suspected site of entrapment. E, a probe was placed above the deep branch (DB) as it passed beneath the leading edge of the fibrotic arch. F, after division of the arch, there was marked compression of the deep branch. A large neuroma can be seen with attenuation of the distal nerves. The deep branch was decompressed to the midpalmar space. Despite the marked compression seen, the patient made good recovery over the next 6 months.

mas, which may be "sandwiched" between the nerve and the arcade. MRI scans are helpful in excluding mass lesions. Decompression of the nerve can be performed through a volar or dorsal approach to the proximal forearm.

Anterior Interosseous Nerve Syndrome

Patients may present with spontaneous volar forearm pain or weakness in the hand affecting pinch (e.g., writing or picking up small objects). Examination may reveal weakness in the flexor pollicis longus and flexor digitorum profundi to the index and/or middle fingers. Patients may be unable to make an "O." Instead of flexing their terminal phalanges of the thumb and index finger, they keep them extended and perform a "square" pinch. Electromyography may reveal fibrillation potentials in the pronator quadratus muscle as well. This particular anterior interosseous innervated muscle is more difficult to test in isolation and distinguish from the stronger pronator teres, which is functional. Fibrous bands have been described most commonly as the cause for entrapment. Decompression of the median nerve and the anterior interosseous branch in the proximal forearm can be done through an anterior approach using an intermuscular interval.

Long Thoracic Nerve

Some think that a compression site at the nerve's entry to the middle scalene is responsible for spontaneous causes of isolated long thoracic nerve paralysis,[23] whereas others have described locations more distally along the chest wall. Most think that the vast majority of isolated long thoracic nerve palsies represent a variant form of brachial plexitis (Parsonage-Turner syndrome). Patients present with shoulder pain and weakness with abduction and are found to have scapular winging (*Fig. 32.4*). This is best demonstrated with the patient forward flexing his or her arm and attempting to push against a wall. Other causes of scapular winging from rhomboid or trapezius dysfunction should also be distinguished. Nonoperative therapy consisting of a tincture of time, physical therapy, and sometimes bracing is advocated. Hemipectoralis major tendon transfer to the inferior scapula can be performed successfully for residual winging. Some degree of winging is not an uncommon residua of Parsonage-Turner Syndrome.

CONTROVERSIAL SUPERIMPOSED ENTRAPMENTS IN THE FACE OF UNDERLYING METABOLIC NEUROPATHY

Diabetic Neuropathy

Diabetes mellitus is becoming increasingly prevalent in the United States. Recent data from the National Health and Nutrition Examination Survey demonstrate a prevalence of 6.5% of the population; an additional 2.8% go undiagnosed.[15] Neuropathy affects 60% of these people. Loss of sensation may lead to ulcers and amputations.

Lee Dellon[17] has challenged the concept that diabetic neuropathy is a progressive, irreversible condition without a surgical intervention. He popularized the concept that symptoms of neuropathy are due to multiple nerve entrapments, which help fit the well known stocking glove distribution. Hyperglycemia may make the nerve more susceptible to compression at normal sites of narrowing such as the carpal or tarsal tunnels.[39] First, using the upper limb as an example, he returned sensibility to patients and improved pain with nerve decompression at more widely known sites of entrapment. He then applied the same approach to nerves in the lower limb, typically decompressing the peroneal nerve and its branches at the fibular neck, the tibial nerve and its

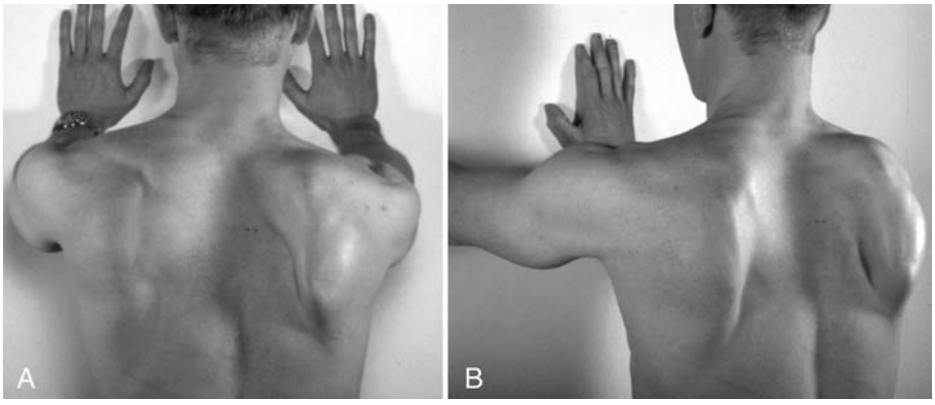

FIGURE 32.4. Long thoracic nerve palsy with winging of the scapula. This 32-year-old man developed severe shoulder pain soon after receiving a tetanus shot. The pain lasted 2 weeks. He then noted difficulty lifting his arm followed by winging of the scapula (A and B). No other neurological deficits could be found on physical examination. EMG, however, revealed fibrillations in the rhomboid and the serratus anterior. He declined surgical decompression of the long thoracic nerve that was offered by another surgeon. He made steady, but incomplete, spontaneous improvement over the next 18 months. He has mild residual winging of the scapula, but no difficulties with overhead lifting at work.

branches at the tarsal and plantar tunnels, and the deep peroneal nerve on the dorsum of the foot. In patients with more severe cases, he performs surgical decompression of multiple peripheral nerves.

These techniques have also been applied by other surgeons. There are more than 12 studies on lower limb decompression surgery for diabetic neuropathy available in the literature.[4,33] Approximately 80% of patients experience improvement in pain and in sensibility. The presence of Tinel's sign increases success rates.[30] These surgeries have been reported to decrease ulcers, amputations, and falls, as well as promoting cost savings.[5,19] Complication rates have been acceptably low.

Dellon and his supporters wonder whether, if this surgical approach works for diabetic neuropathies, will it also work for other types of symptomatic neuropathies?[22] Increasing reports are being published to support this hypothesis. Skeptics think that this approach is an oversimplistic one to treat the complexities of varied neuropathies, especially the different types of diabetic neuropathy. Long-term data are being collected.

CONTROVERSIAL ENTRAPMENT SYNDROMES FOR PAINFUL CONDITIONS

Surgeons have been trying for decades to identify peripheral nerve entrapment sites to characterize and to treat various pain syndromes. Quite simply, these conditions are either underdiagnosed[26,37] or overdiagnosed.[42,48] In extremes, there are zealots and nihilists.[1] The true prevalence is probably somewhere between the two vociferous groups.

Historically, and not uncommonly, there tends to be a pattern with some of these disorders. A single report provides an anatomic explanation for a new entity with encouraging (often optimistic) results. This is followed by a flurry of publications supporting the diagnosis and introducing some provocative maneuvers. Thereafter, sentiment and enthusiasm tends to wane, but not dissipate.

These entrapment syndromes offer potential anatomic sources that account for pain that is thought to be due to incomplete (partial) nerve compression. The rationale is that pain fibers are preferentially compressed, rather than sensory or motor nerve fibers. Skeptics wonder why, unlike other conditions, the neurological picture does not worsen with chronic compression, leading to deficits and objective findings. These disorders are further unified by the difficulty in establishing their clinical diagnosis. For the most part, patients have subjective symptoms without objective criteria despite extensive evaluation. Unlike the other nerve compression syndromes discussed in which electrodiagnostic studies are extremely helpful in localizing the lesion and confirming a diagnosis, in these cases, electrodiagnostic studies are typically non-confirmatory. New high resolution MRI is being used increasingly and offers great promise in providing new levels of anatomic and pathoanatomic detail.[24]

Treatment should be centered on nonoperative measures, typically combining a trial of anti-inflammatory agents, avoidance of repetitive or exacerbating postures and positions, as well as splints, when applicable. Surgery should, in general, be considered in carefully selected patients when other methods have been exhausted.

Outcomes for all of these syndromes have been reported with ranges from 50 to 90%, with 70% at long-term. For me personally, I find it difficult to interpret many of these scientific reports when there is so much ongoing difficulty diagnosing the specific condition.

Thoracic Outlet Syndrome

Best known of these pain syndromes is disputed thoracic outlet syndrome. It represents a group of conditions with neurovascular compromise dealing with the brachial plexus (usually the lower trunk) or the subclavian artery or vein. The roots of the brachial plexus along with the subclavian artery pass within the interscalene triangle (anterior scalene, middle scalene, first rib). The subclavian vein typically passes anteriorly. Altered relationships within this fixed space account for the neurovascular compression. Compression may be exacerbated by postural changes and influenced by anatomic variations or pathology (e.g., cervical ribs, elongated cervical transverse processes, abnormalities of first rib, fibrous bands, variant muscles or muscular insertions, etc.) and trauma.

The diagnosis of disputed thoracic outlet syndrome remains a clinical one. Patients have subjective symptoms. Neurological examination and electrodiagnostic studies are normal. Provocative tests are difficult to interpret because they may be found in more than one-third of patients in the normal population. Radiographic variations may be identified, but are not specific. Thus, the diagnosis remains one of exclusion. Despite improved efforts with diagnostic testing and imaging studies, a "gold standard" for the reproducible diagnosis of disputed thoracic outlet syndrome is still lacking. Magnetic resonance neurography is being used increasingly and will hopefully clarify the diagnosis in the future.[24] Disputed thoracic outlet syndrome must be distinguished from true neurogenic thoracic outlet syndrome[43] or vascular forms of thoracic outlet syndrome, which have confirmatory testing.

Patients present with a complex of symptoms of neural (predominantly lower trunk), vascular or neurovascular compression. Pain may be present in the shoulder, arm, forearm, hand, and fingers. Paresthesias and numbness occur predominantly in the ulnar sided fingers. Patients may also describe weakness, fatigue, or claudication in the upper limb, and symptoms are often worse with overhead activities. Patients should be examined for sensory deficit (usually C8–T1), muscle weakness (similar to ulnar nerve but may also includ-

ing median nerve territories), and pulse obliteration or positive provocative maneuvers (e.g., Adson's test, Roos test, costoclavicular maneuver, Tinel's test). Radiographs may reveal cervical ribs or elongated transverse processes. The differential diagnosis includes cervical radiculopathy and ulnar nerve compression, as well as other conditions, such as apical lung tumors (Pancoast tumors).

Various surgical approaches have been described, including supraclavicular exposure (scalenotomy, scalenectomy, brachial plexus decompression, first rib resection), transaxillary exposure for first rib resection, combined supraclavicular and transaxillary procedures, and a posterior subscapular approach. Outcomes have, in general, been similar using the various approaches. However, a recent randomized trial significantly favored the transaxillary approach over a supraclavicular one in this group of patients with 75% versus 48% achieving good or excellent outcomes.[38]

My own bias is that the vast majority of patients with symptomatic thoracic outlet syndrome can be treated conservatively.[32] For most patients with disputed thoracic outlet syndrome, I offer and perform surgery cautiously, and reluctantly, in those patients who fail all other options. The presence of a radiographic abnormality (such as a cervical rib) together with positive provocative maneuvers are positive factors in my mind, and I will explore these patients with slightly more enthusiasm. I favor a supraclavicular approach with neurolysis, scalenectomy, and removal of cervical rib as necessary, typically, without removal of the first rib in a primary case. Surgery should address the proximal portion of the plexus, especially spinal nerves C8 and T1. Intraoperative recordings by David Kline have shown slowing at a proximal level at junction of spinal nerves to lower trunk.[43] Surgery is indicated in those patients with neurological and vascular deficits.

Piriformis Syndrome

This syndrome was predicted to exist by Yeomans in 1928 to explain sciatica before lumbar disc herniation was introduced by Mixter and Barr in 1934. Thereafter, Freiberg coined the term piriformis syndrome.[46] The syndrome was introduced based on the knowledge of known anatomic variations involving the relationship of the sciatic nerve to the piriformis muscle and the parallels between these and those used to explain thoracic outlet syndrome (scalenus anticus syndrome) at the time.[46] Currently, the term piriformis syndrome is commonly used synonymously to explain all cases of sciatica that are not of disc origin, as opposed to its usage in a pure sense, entrapment of the sciatic nerve by the piriformis muscle itself. This nomenclature is part of the problem. Whether or not the muscle itself is the causative factor is the bigger question. However, no one argues that buttock-level sciatic nerve compression or irritation does exist. We all agree that it is important to look diligently and carefully for other lesions that may affect the sciatic nerve in the buttock or the lumbosacral plexus in the pelvis. Meanwhile, in the diagnosis of "disputed" piriformis syndrome, so-called piriformis provocative maneuvers are not pathognomonic and EMGs are typically normal. Routine imaging studies may reveal atrophic or asymmetricly enlarged piriformis muscles, but the sciatic nerves are typically normal in appearance. In a large series of patients with sciatic of nondisc origin, magnetic resonance neurography has been reported to demonstrate focal abnormalities in the sciatic nerve and the piriformis muscle.[25] Note, however, that not all cases of hyperintensity of nerve are due to entrapment. Image-guided trials with steroid injections into the region are used with varied successes. They may have diagnostic and therapeutic implications. Surgical decompression through a limited muscle-splitting approach has been used with good or excellent results in 80% of patients.[25] Thus, the debate continues.[45]

Pudendal Nerve Entrapment

Anatomic explanations for chronic pelvic (perineal) pain, with or without paresthesias, have been put forth.[2,35,36] Patients with this syndrome typically describe unilateral or bilateral pain that becomes worse with sitting. Infectious etiologies must be ruled out. Potential compression sites have been described: between the "claw" by the sacrospinous and sacrotuberous ligaments and in the pudendal canal (Alcock's). Electrodiagnostic studies have been reported as being helpful by several groups, but are not widely performed and have not been validated in many laboratories. Patients typically undergo image-guided injections, which often produce temporary and, on occasion, lasting relief. Good or better results with surgical decompression of the pudendal nerve and its branches have been achieved in 65 to 70% of cases, although medical interventions may still be necessary to provide additional benefit.

Radial Tunnel Syndrome

Radial tunnel syndrome is a condition describing patients with proximal forearm pain and is attributed to compression of the posterior interosseous nerve at the same sites of "true" (neurogenic) posterior interosseous nerve syndrome.[6,31] Some patients may experience symptoms suggestive of, or overlapping with, refractory lateral epicondylitis. The pain is often aggravated by repetitive movements. Neurological examinations are typically normal. Tenderness is often centered on the mobile wad and over the supinator muscle. Provocative maneuvers have been described, including the middle finger extension test. Electrodiagnostic studies are usually normal. Diagnostic blocks are used by many physicians; patients who transiently and appropriately improve with blocks may be deemed surgical candidates. Release of the radial tunnel and nerve decompression has

generally been reported to be effective in 70% of patients, but has been as high as 95%[31]), although long-term results may be unpredicatable.[6]

Pronator Syndrome

Pronator syndrome is an entrapment syndrome characterized by patients with proximal volar forearm pain. Similar to patients with carpal tunnel syndrome, patients with pronator syndrome may describe paresthesias in the radial sided digits; they typically do not have nocturnal symptoms. Weakness as a rule is not found, but give way weakness may be present due to pain. Pronator syndrome is attributed to median nerve compression at the same anatomic sites that may yield median nerve compression, with some degree of deficit, in the proximal forearm and elbow region (e.g., beneath the bicipital aponeurosis, pronator teres, or the flexor digitorum superficialis). Provocative maneuvers have been put forth as a means to diagnose and localize the condition, including resistance to elbow flexion, pronation and middle finger flexion. Electrodiagnostic studies typically are not diagnostic. The major differential diagnosis in these patients is proximal radiation of pain due to median nerve compression at the wrist from carpal tunnel syndrome. Surgery to decompress the median nerve typically is only entertained after failure of conservative measures and a prolonged course of stretching exercises; after carpal tunnel release if symptoms persist[1]; or if EMGs or nerve conduction studies confirm a more proximal localization. Nerve decompression, if performed, should be accomplished at all potential sites in the region, not just to a single site, as suggested by a positive single provocative maneuver.

CONCLUSION

A spectrum of entrapment syndromes exists with an increasing number of "tunnel" syndromes being described. As surgeons, we are in a position to improve outcomes in many patients with peripheral nerve disorders. In the future, with more interest, expertise and experience in the field, our contributions will be even larger. More emphasis on well-designed clinical trials is necessary and will help answer many of these controversial questions raised in this chapter.

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