Techniques for Nerve Compression

INTRODUCTION

A neuropathy may be defined as any disorder that results in abnormal nerve function. Neuropathies are frequently associated with a number of systemic disorders, such as diabetes, hypothyroidism, alcoholism, vitamin B₁₂ deficiency, and lead intoxication. The neuropathy may be focal or restricted to a specific anatomical location. Many of these focal neuropathies arise as a result of nerve compression, although traction injuries can play a significant role.

Nerve anatomy and physiology

The connective tissue of nerves includes the outer epineurial layer, which contains loosely woven collagen fibers and elastin. The epineurium protects the nerve from compression and stretching and therefore tends to be thicker in places that experience repetitive shear force, such as the cubital tunnel at the elbow. The perineurium separates groups of fascicles and constitutes a diffusion barrier, which protects the axons from infection and chemical insult. The perineurium is relatively unyielding, and this property can lead to a mini-compartment syndrome when the endoneurial pressure increases. The individual axons are surrounded by the endoneurium, which provides support and a framework for regeneration of nerve fibers after injury.

Nerve compression

In early nerve compression the symptoms are caused by vascular impairment, and the initial changes occur at the blood–nerve barrier. Fluid shifts that occur with limb position result in endoneurial edema. There is no lymphatic drainage of the endoneurial space, and so endoneurial edema clears slowly. The edema cuts off the blood supply by compressing the arterioles that course through the perineurium obliquely. This impairs the Na⁺/K⁺ exchange pump that is ATP dependent. This ultimately results in an irreversible metabolic conduction block, leading to paresthesia.

The dramatic relief of symptoms that sometimes occurs after surgical decompression also suggests an ischemic etiology to compression neuropathies. The mechanical source of compression may obstruct venous return, resulting in segmental anoxia, capillary vasodilatation, and edema. The edema compounds the compressive effects and leads to abnormal axonal and cellular exchange. Surgical release of compressed nerves at this early stage generally yields good results. Prolonged compression, however, results in intra-neural fibrosis, after which nerve recovery is less likely to occur.

Nerves are viscoelastic and can change their length in order to accommodate the myriad combination of joint positions. Nerves have a longitudinal blood supply that is reinforced periodically by segmental perforators. They are surrounded by a vascularized gliding layer to facilitate the nerve gliding that accompanies joint movement. Chronic compression leads to inflammation and secondary fibrosis to disrupt this gliding layer, ultimately leading to nerve tethering. Any compressive neuropathy therefore frequently has a component of traction neuropathy as well. Traction alone can cause conduction block. Nerves can only elongate by 8% until there is a disruption of the blood supply. Traction therefore leads to nerve ischemia with secondary impairment of the Na⁺/K⁺ pump to cause a conduction block. This is clinically manifested as numbness or paresthesia. Many of the provocative tests for nerve compression induces relative nerve ischemia in the already susceptible nerves through manual pressure, awkward joint positioning and/or traction to elicit numbness or paresthesia in the distribution of that specific nerve. A knowledge of the normal nerve course and topographic anatomy is thus essential.
ELECTRODIAGNOSTIC STUDIES
Ancillary testing cannot replace a detailed history and thorough examination of the upper limb, but they can provide a means for staging the degree of neuropathy and to rule out more generalized disorders that may masquerade as a focal neuropathy.

Nerve fibers show varying susceptibility to compression. The large fibers are more vulnerable to compression and ischemia. The neurophysiology of electrical recording is such that the recording electrode will detect activity in the largest myelinated fibers first, because these fibers conduct at the fastest rates and have a lower depolarization threshold than the small unmyelinated nerves. Latency and conduction velocity depend on the time that transpires from stimulation of the nerve to the first recording. If only a fraction of the large thick myelinated fibers remain and transmit impulses, the recorded latency and conduction velocity remain normal because the recording electrode mostly detects the fastest fibers. The electrical conduction in smaller, less myelinated or non-myelinated nerves is much slower, and hence not usually detected in a routine nerve conduction study (NCS). Large myelinated and small unmyelinated fibers can be affected differently.

Connective tissue changes follow after focal nerve fiber changes. The large myelinated nerves undergo segmental demyelination, whereas the small unmyelinated nerves undergo degeneration and regeneration. Normal fascicles are adjacent to abnormal fascicles. The nerve conduction study only tests the faster-conducting fibers. This explains the seeming paradox of the patient who has established carpal tunnel syndrome but yet normal electrodiagnostic studies. It is the worst fascicles that produce symptoms, but it is the best fascicles that account for the normal nerve conduction studies.

With early compression the symptoms are intermittent, and the edema is reversible. When there are constant symptoms, there is usually myelin damage and/or chronic endoneurial edema. This demyelination is responsible for the slowing of nerve conduction. If the compression continues, some of the axons will die. If there are fewer nerve fibers, the size of the electrical charge will be smaller, leading to smaller amplitudes. When there is sensory or motor loss, there is usually degeneration of nerve fibers. Despite the restoration of neural blood flow following nerve decompression, remyelination of the axon is often incomplete, which accounts for persistently abnormal nerve conduction even though the patient may be without symptoms.

Quantitative sensory testing (QST)
Quantitative sensory testing is reportedly more sensitive than the NCS because only 25% of the large myelinated nerve fibers need to be conducting normally in order to yield a normal nerve conduction test. Nerve density is defined as the number of nerve fibers per mm². The nerve threshold is the minimum amount of force necessary to cause the touch receptors to fire. With nerve degeneration it is more difficult to distinguish two points from one point. Static and moving two-point discrimination typically tests the density of innervation. Threshold tests would include vibrometry and Semmes–Weinstein monofilament testing (SWT). Vibrometry is relatively insensitive to early changes and is not commonly used. Semmes–Weinstein testing involves placing nylon filaments of varying thickness on the skin until the patient can detect the touch of specific filament thickness. The test is repeated with varying diameter filaments until the threshold is determined. Abnormal SWT is consistent with nerve demyelination.

MEDIAN NEUROPATHIES
DISTAL MEDIAN NEUROPATHY
Carpal tunnel syndrome
Anatomy
The carpal tunnel is open-ended proximally and distally, but behaves like a closed compartment physiologically and maintains its own distinct tissue fluid pressure levels. It is a bony osseous canal that is bounded by the concave arch of the carpal bones dorsally and the flexor retinaculum palmarly. The hook of the hamate, trapezium, and pisiform form the ulnar border; the radial border consists of the scaphoid, trapezium, and the fascial septum overlying the FCR. The flexor retinaculum consists of three zones: a proximal zone that is continuous with the deep forearm fascia, a central zone that is composed of the transverse carpal ligament (TCL), and a third zone that consists of the aponeurosis between the thenar and hypothenar muscles. The median nerve at the wrist has approximately 30 fascicles. The motor recurrent branch often consists of two fascicles that are situated in a volar position, with the various sensory groups in the radial, ulnar and dorsal positions of the main trunk. The motor branch can be separated from the main trunk without harm for up to 100 mm proximal to the thenar muscles. The sensory fibers travel within the common digital nerves to the thumb, index and middle, as well as the communicating branch to the third web space.

Pathophysiology
There are two potential sites of compression anatomically. The first is at the proximal edge of the TCL, where compression may be produced by acute wrist flexion. This accounts for the positive Phalen’s test (wrist flexion test) in CTS. The second is adjacent to the hook of the hamate, where an hourglass deformity of the median nerve may be seen. Patients with compression in this area will have a positive median nerve compression (Durkan’s) test but a negative Phalen’s test. Compression within the carpal tunnel may also result from any lesion that takes up space within the canal, such as flexor tenosynovitis, hematoma, palmar carpal dislocation, distal radius fractures, tumors and ganglia. Although many cases have been attributed to a non-specific synovitis, synovial biopsies typically fail to show evidence of inflammation. They do reveal edema and vascular sclerosis, which may be secondary to compression rather than the primary event.
Techniques for Nerve Compression Syndromes

History
The patient typically complains of numbness and paresthesia in the median nerve distribution. Initially the symptoms occur at night, owing to a combination of wrist flexion during sleep and fluid shift that occur with the horizontal position, which increases the carpal canal pressure. In this early stage of nerve compression the symptoms are of a vascular nature, which culminate in endoneurial edema. With early compression the symptoms are intermittent and the edema is reversible. As the symptoms progress, they become more frequent during the day and are precipitated by gripping and pinching activities as well as those tasks requiring repetitive wrist flexion. When there are constant symptoms, there is usually myelin damage and/or chronic endoneurial edema.16

Physical examination
CTS represents a constellation of signs and symptoms in which no one test absolutely confirms its diagnosis. A positive Tinel’s sign may be present over the median nerve at the wrist, and produces paresthesia in the thumb and radial 2½ digits. Phalen’s test consists of passive wrist flexion for 1 minute, which when positive produces subjective paresthesia in a median nerve pattern. This is best performed with the elbows extended because simultaneous wrist and elbow flexion may reproduce ulnar nerve symptoms as well. Direct compression of the nerve or the Durkan’s test is thought to be more sensitive. Szabo et al.15 found that if a patient had an abnormal hand diagram, abnormal sensitivity by SWT testing, a positive Durkan’s test, and night pain, the probability of having CTS was 0.86. If all four of these conditions were normal, the probability of having CTS was 0.0068.

Electrodiagnostic studies
The nerve conduction study can yield useful information, but the severity of the preoperative conduction deficit does not provide significant data for prediction of the final outcome or return to work after carpal tunnel release. There are some caveats for nerve conduction studies in CTS. First, sensory abnormalities usually occur before motor abnormalities. In other words, the distal sensory latencies are often slow before the distal motor latency. This is not surprising, because 94% of the axons in the median nerve at the wrist level are sensory.17 The sensory nerve axons are larger than the motor axons and hence more susceptible to compression. If the distal motor latency (DML) is abnormal in the presence of normal sensory nerve action potentials (SNAPs), extra care must be taken to rule out anterior horn cell disease or a C8 radiculopathy, although isolated recurrent motor branch compression has been reported.18 Second, the nerve conduction studies may not return to normal after decompression owing to retrograde fiber degeneration or incomplete remyelination, even in the presence of a full clinical recovery.

Non-operative management
Non-operative therapy includes splinting the wrist in a neutral position, steroid injections, and management of any underlying systemic diseases. Steroid injection offers transient relief to 80% of patients, but only 20% will be symptom free 12 months later. Those most likely to benefit from conservative management have had symptoms for less than 1 year, only intermittent numbness, normal two-point discrimination, <1–2 ms prolongation of distal motor and sensory latencies, and no motor findings; 40% of this group will remain symptom free for longer than 12 months.21

Surgical indications
- Mild CTS: Failed trial of conservative treatment with splints, NSAIDs, and activity modification for at least 1 month. No sensory or motor loss, nocturnal symptoms and/or transient paresthesia only with prolonged gripping or pinching. No thenar wasting, no change in 2PD. May have a prolonged 2PS. NCS may be normal, increased CS1 or slowing of distal median SNAPs but with a normal DML. Normal EMG.
- Moderate CTS: Failed trial of splinting and/or cortisone injections. Frequent daytime symptoms even without gripping. May have abnormal 2PS and 1PS, 2PD <15 mm. No wasting of ABP but may have weak abduction. NCS may show slowing of distal SNAPs. DML slowing of <1–2 ms, but no drop in amplitude. Normal EMG.
- Severe CTS: No indication for conservative treatment. Frequent to constant symptoms. Abnormal 2PS, 1PS, 2PD >15 mm. Slowed to absent SNAPs, prolonged DML with amplitude loss. EMG shows membrane instability, decreased recruitment and fibrillation potentials/positive sharp waves.
- Acute CTS secondary to distal radius fracture, bleeding disorder, burn, or other cause of massive swelling.

Contraindications
- Untreated hypothyroidism, diabetes or other metabolic neuropathy.

Surgical technique
The procedure is performed under tourniquet control. A 3–5 cm incision is made in the palm parallel to the thenar crease and in line with the ring finger axis in order to protect the palmar cutaneous branch of the median and ulnar nerves. Tenotomy scissors are used to spread down to the palmar aponeurosis; this is divided to expose the transverse carpal ligament (TCL). The TCL is divided from distal to proximal. A hemostat may be used to protect the median nerve. The skin is retracted and the deep flexor retinaculum is divided under direct vision for an additional 2 cm. The nerve and tendons are retracted to the radial side and the floor of the canal is inspected for any masses. The recurrent motor branch is inspected and decompressed separately if necessary. The same considerations apply when using a mini-incision technique (Fig. 19.1A–E). The tourniquet is released and hemostasis obtained. The wound is closed with 4/0 nylon mattress sutures after injection of local anesthetic.
FIGURE 19.1 Carpal tunnel release. A The standard carpal tunnel incision is parallel to the thenar crease along the ring finger axis to avoid injury to the palmar cutaneous branches of the median and ulnar nerves. B The palmar aponeurosis is identified by the longitudinally oriented bers. C The bers of the transverse carpal ligament (TCL) are transversely oriented. Arrows are pointing to a partial incision in the ligament.
Postoperative care
Finger movement begins immediately, which also aids in median nerve excursion. A below-elbow splint is applied for comfort for the first week, followed by desensitization and progressive strengthening.

Ancillary procedures
In advanced cases of nerve compression, internal neurolysis and epineurotomy have been described. No significant differences were found between the outcomes of these two ancillary procedures, hence they are no longer recommended. Numbness of the small finger due to coexistent Guyon’s canal compression often improves after carpal tunnel release alone, and MRI studies have demonstrated an increase in the volume of Guyon’s canal after a CTR. Routine tenosynovectomy does not provide better results than carpal tunnel release alone, and is mostly recommended with associated proliferative tenosynovitis from some other cause, such as rheumatoid arthritis or granulomatous infection.

Complications
Pillar pain is an oft-cited complication after carpal tunnel surgery. The cause of this condition is unclear and the treatment debatable. Procedures such as z-plasty lengthening of the TCL to mini-incision and endoscopic techniques have been proposed. Injury to the palmar cutaneous branch of the median nerve is a cause of persistent scar tenderness and led to the plea for a more ulnarly based incision. Blind release of the TCL, which was commonplace in the 1960s, occasionally resulted in laceration to the deep motor branch. Injury to branches of the superficial palmar arch are repaired or tied off as indicated. Median nerve anomalies or altered anatomy may lead to inadvertent nerve laceration. Any recognized fascicular lacerations should be repaired under microscopic magnification immediately or as soon as it is recognized postoperatively to maximize the chances for recovery. Acute infection is treated aggressively with antibiotic treatment and/or drainage as necessary. Wound coverage problems with exposed tendons or nerve may be covered with pedicled or free flaps. Causalgia should be treated with stellate blocks and aggressive therapy, including edema control and dynamic finger splinting.

Outcomes
Open median nerve decompression leads to symptomatic relief in the majority of patients. When there is clinical evidence of demyelination, patients should be informed of the possibility of residual symptoms and delayed improvement. After open carpal tunnel release, patients typically regain their preoperative baseline grip strength within 3 months, and pinch strength within 6 weeks. Mini-incision open techniques have similar outcomes but may carry a higher risk of incomplete TCL release.

Endoscopic carpal tunnel release
Indications for surgery
The indications for endoscopic carpal tunnel release (ECTR) are the same as for an open release. The patient should have the appropriate symptoms and signs of carpal tunnel syndrome and have failed a trial of conservative treatment with splinting, NSAIDs, and activity modification.

Contraindications
Absolute contraindications include any distortion of the carpal canal due to tumor, previous surgery, carpal fracture/dislocation or malunion of the radius. A loss of wrist extension of 20–30º, either from bony fusion or from wrist contracture, would hamper this procedure by impeding the correct placement of the endoscope. Unfamiliarity with the regional anatomy is another contraindication.

Relative contraindications abound and are not universally agreed upon. Flexor tendon thickening due to synovial hyperplasia or adhesions from previous releases will complicate the procedure. Recurrent or persistent carpal tunnel syndrome following a previous release often includes a component of traction neuropathy due to scar that may thwart attempts at ECTR. If there is suspicion of separate entrapment of the recurrent motor branch an open procedure is required.

Surgical technique (Agee method)
The use of the Agee endoscope is integral to this procedure. The patient is positioned supine with the arm abducted on an arm board. The procedure is performed under tourniquet control after limb exsanguination. The author’s preference is to use a general anesthetic because of the not infrequent difficulties with instrumentation,
including fogging of the camera lens, which can be minimized by warming the scope in saline. Regional and local anesthesia are acceptable alternatives.

A number of anatomical landmarks are identified and outlined with a marking pen to guide placement of the scope. The hamate hook, flexor carpi ulnaris (FCU) and pisiform are palpated and drawn on the skin. It is vital to keep the scope radial to the hamate hook at all times in order to prevent inadvertent penetration of Guyon’s canal. A straight line is drawn connecting the longitudinal axis of the ring finger with a transverse line across the proximal wrist crease. The palmaris longus (PL) tendon (if present) is traced. The dissection should stay ulnar to the PL, which protects the median nerve. A 2-cm transverse incision is placed in the proximal wrist crease between the PL and the pisiform. As the surgeon’s comfort level increases, this incision can be placed in the distal wrist crease, although there is more subcutaneous fat to contend with. The plane between the subcutaneous fat and the deep forearm fascia or flexor retinaculum (FR) is identified and developed. A distally based U-shaped flap of the FR is elevated by making two parallel incisions approximately 1 cm apart and 1–2 cm long through the deep fascia between the FCU and the PL/median nerve. The incisions are then connected by a proximal transverse cut. Care is taken to avoid penetrating the underlying flexor synovium to lessen unwitting tendon laceration. The distally based flap is elevated with a skin hook while developing the plane between the superficial flexor tendon synovium and the undersurface of the TCL. Establishing this plane is crucial in avoiding improper scope placement.

At this point, the wrist is hyperextended over two folded towels. A small dilator is gently inserted superficial to the flexor tendons in line with the ring finger. With proper placement the dilator will abut against the hook of the hamate by sweeping it in an ulnar direction. If there is no obstruction to the ulnar passage of the dilator, the dilator has entered ulnar to the hook of the hamate and should be removed and reinserted, otherwise there is the dire risk of injury to the ulnar nerve and artery. Two incrementally sized dilators are then gently inserted to enlarge the gliding path for the scope. The distal tip of the dilators can be easily palpated in the midpalm by levering the tip palmarwards. Multiple passes of the dilators increase the risk of a median neuropraxia, especially in small wrists. The soft tissue elevator is then inserted and used to gently scrape any synovium from the undersurface of the TCL. While the assistant maintains the wrist in hyperextension, the tip of the Agee endoscope is then gently inserted into the canal. The surface line marking the axis of the ring finger is used as a guide. The shiny transverse fibers of the undersurface of the TCL should now come into view. Often fogging of the scope, fluid from fatty tissue, or synovial remnants will block the view. In this event, the scope should be removed and defogged and the synovial elevator used once more. Because the scope is often colder than the carpal canal, keeping it in warm water until just before use may minimize fogging. The distal edge of the TCL must be completely visualized. This can be identified by a change of the transverse fibers to an ill-defined fat pad, which contains the superficial palmar arch and the common digital branch to the third web space. Ballottement of the fat pad can help identify the demarcation zone. The hook blade should not be engaged until the distal edge is well seen. It is prudent to start the blade 2–3 mm from the distal edge on the first pass. The hook blade is then main-tained upright while the scope is slowly withdrawn proximally, keeping the blade in sight at all times. The scope is then reinserted to visualize the cut. The TCL is under tension and will usually split apart, allowing identification of the muscle fibers of the overlying palmaris brevis, or the longitudinally oriented fibers of the palmar aponeurosis. A second pass may be made at this point to release the distal most fibers of the TCL. The scope is then removed (Fig. 19.2A–F).

The proximal skin flap is then retracted to allow visualization of the deep flexor retinaculum. Tenotomy scissors are used to divide the retinaculum for an additional 2 cm. The scope is then reinserted into the carpal canal and the tourniquet is deflated. Pulsatile bleeding is suggestive of an injury to the superficial palmar arch, which is then explored through an open incision. Digital nerve injury cannot be recognized by any means, but a high level of suspicion and due diligence will dictate the need for exploration. It is possible for the mesoneurial tissue surrounding the median nerve to become interposed between the scope and the undersurface of the TCL. It will not have the appearance of nerve tissue and lead to a false sense of safety, but it can be drawn in by the hook blade, which may lead to a partial or complete median nerve laceration. If there is any doubt, the scope should be removed and the fl exed cleared as described above. If the view cannot be improved, then conversion to an open procedure is necessary. The wound is infiltrated with marcaine and closed with an absorbable subcuticular suture and Steristrips.

Postoperative care
The patient is placed in a large bulky hand dressing with a volar wrist splint for comfort. Finger motion begins immediately, which also results in nerve excursion. The splint is removed at 1 week and the patient is started on a home program of range of motion exercises. The sutures are removed at 2 weeks and light gripping exercises begun.

Complications
Significant and devastating complications have been reported, often related to poor scope placement or inadequate visualization of the TCL. This includes partial and complete median nerve lacerations, common digital nerve lacerations, and injuries to the superficial palmar arch. The author has noted a 6-month neurapraxia of the third common digital nerve branch in small female patients with small carpal canals. Flexor tendon lacerations have also been reported. If there is anything less than complete visualization, the procedure should be converted to an open CTR. Placing the scope in Guyon’s canal as a result of inadequate identification of the hook of the hamate has resulted in ulnar nerve and artery lacerations.
FIGURE 19.2 Endoscopic carpal tunnel release. A Landmarks for locating the hamate hook (*). P = pisiform, M4 = fourth metacarpal line, I-P = Index pisiform line. B Skin incision at distal wrist crease (in red). C Outlining a distally based fascial flap. D Insertion of the Agee endoscope, which is aligned with the ring finger axis. E View through the endoscope of the white undersurface of the transverse carpal ligament (TCL). The appearance of the midpalmar fat pad demarcates the distal edge of the TCL(*). F After complete release of the TCL. Note the increased separation of the ligament edges (arrows) with exposure of the palmar brevis, which denotes a complete ligament release. (From Slutsky DJ. Endoscopic carpal tunnel release: the Agee method. In: Slutsky DJ, Nagle DJ, eds. Techniques in wrist and hand arthroscopy. Philadelphia: Elsevier, 2007, with permission.)
release of the TCL is commonly reported and is a major criticism of this technique.

Outcomes
Agee et al. carried out a 10-center randomized prospective study of endoscopic release using his technique. For patients in the device group with one affected hand, the median time for return to work was 21.5 days less than that for the control group. Another large multicenter prospective study of 192 cases demonstrated that, at least in the short term, the patients treated with the endoscopic method had significantly greater grip strength, pinch strength, and hand dexterity. The open technique resulted in greater scar ten-derness during the first 3 months after surgery as well as a longer time until the patients could return to work. Not everyone agrees with the advantage of the endoscopic tech-nique. In a small study of 25 patients with bilateral carpal tunnel syndrome who underwent an endoscopic release by the Agee technique on one hand and open release on the other, the investigators found no significant advantages in terms of return of grip strength, manual dexterity or sensation.

PROXIMAL MEDIAN NEUROPATHY

Pronator syndrome
Median nerve compression neuropathies in the distal arm and forearm are extremely uncommon compared to CTS. The moreproximal of the median nerve compression neuropathies is the pronator syndrome. The syndrome is classically associated with any of four potential areas of compression, including the ligament of Struthers, the lac-er tus fibrosis, the aponeurotic fascia of the superficial or deep head of the pronator teres (PT), or the flexor digitorum sublimum(FDS) arch. Confounding conditions such as carpal tunnel syndrome and anterior interosseous nerve entrapment should be excluded.

Anatomy
The median nerve arises from the medial and lateral cords of the brachial plexus. It contains the nerve root fi bers from C6 to T1. It lies lateral to the axillary artery but then crosses medial to it at the level of the coracobrachialis. At the elbow, it travels behind the bicipital aponeurosis but in front of the brachialis. At the distal part of the cubital fossa the motor branches of the median nerve consistently divide into three fascicular groups. There is an anterior group (to the PT and FCR), a middle group (motor to the FDS and hand intrinsics, sensory to the thumb, index and middle fi ngers), and a posterior group (to the AIN branch). These branch groups can be traced proximally without harm, within the main trunk of the median nerve for 2.5–10 cm. The nerve and artery pass through the antecubital fossa underneath the lacertus fi brosis and give off branches to the palmaris longus (PL), flexor carpi radialis (FCR), flexor digitorum superficialis (FDS), and rarely the flexor digitorum profundus (FDP). The nerve then dives between the deep and superficial heads of the PT, to which it four branches. The flexor arch of the PL lies 3–7.5 cm below the humeral epicondylar line. The fi bros arch of the superficial arch lies 6.5 cm below the humeral epicondylar line. The median nerve enters the forearm deep to the flexor arch of the FDS and emerges beneath the radial side of the muscle belly of the middle finger superficial, where it is quite superficial and near the palmaris longus tendon.

Pathophysiology
The nerve may be compressed by the ligament of Struthers, which may or may not be associated with a supracondylar process (1% of cases). This ligament spans the supracondylar process and medial epicondyle and creates an arcade that contains the median nerve and brachial artery. The ligament of Struthers has also been described in the absence of an associated supracondylar process. In the forearm, the median nerve can be compressed by the PT, the FDS arch or the bicipital aponeurosis. Rare causes of compression include a persistent median artery, Gantzer’s muscle (accessory head of the FPL) and the palmaris profundus.

History
Clinical symptoms of pronator syndrome include forearm pain as well as paresthesia and hypoesthesia in the median nerve distribution. Symptoms are typically precipitated by activity, especially repetitive elbow flexion/extension and forearm pronation/supination. Nocturnal paresthesias are not common, unlike with CTS. Numbness and tingling may also involve the thenar area supplied by the palmar cutaneous branch, which takes off proximal to the carpal transverse ligament and is not compressed in CTS.

Physical examination
Phalen’s and Durkan’s tests are negative unless there is coexistent CTS. There may be a positive Tinel’s sign over the proximal nerve as well as a firm and tender PT, and a tenderness along the median nerve in the proximal forearm. Manual compression of the median nerve over the PT for 30 seconds (i.e. pronator compression test) may elicit paraesthesia in the median nerve distribution. Pain or paresthesia produced by resisted forearm supination combined with resisted elbow flexion beyond 120° suggests compression at the bicipital aponeurosis. Paresthesia resulting from resisted forearm pronation while the elbow is slowly extended from full flexion is indicative of compression between the two heads of the PT. Resisted proximal interphalangeal joint flexion of the middle fi nger by producing paresthesia in the radial three digits and/or pain over the FDS arch is consistent with entrapment under the fi bros origin of the FDS. These tests are, however, neither specifi c nor sensitive.

Electrodiagnostic studies
NCS are rarely diagnostic for pronator syndrome. There may be prolongation of the distal median sensory latencies as well as a reduced amplitude of the compound motor action potential to the abductor pollicis brevis. There may be slowing of forearm conduction, but this can be mislead-
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ing because a reduced forearm NCV is seen up to 20–30% of the time when there is a markedly delayed distal median motor latency due to severe CTS. This slowing was initially thought to be due to retrograde nerve demyelination but is currently attributed to test artifact. Sequential needle stimulation of the median nerve in 1-cm increments while recording from the APB may identify an area of focal conduction block near the PT muscle. Nerve conduction studies during provocative maneuvers such as elbow flexion, forearm pronation and middle finger flexion against resistance do not increase the yield. The EMG, however, may be of some value because membrane instability may be seen in the median-innervated muscles, including the PT, flexor carpi radialis and dFDS.

Quantitative sensory testing
As with CTS, testing of the index pulp may initially show an abnormal 2PS pressure threshold. With progression one sees a widening of the 2PS distance, followed by an abnormal 1PS threshold. In this syndrome, PSSD testing of the thenar eminence is especially useful and may help to differentiate pronator syndrome from CTS. Because the palmar cutaneous branch of the median nerve (PCBMN) takes off proximal to the TCL, one would expect an abnormal 2PS and possibly 1PS in pronator syndrome, whereas this should be normal in isolated CTS. In the author’s experience, however, a coexisting C6 radiculopathy can complicate the picture, and can lead to abnormal 2PS and 1PS over both the thenar eminence and the dorsum of the first web space. This can mimic compression of both the PCBMN and superficial radial nerve, hence clinical correlation is prudent.

Non-operative management
The hallmark of treatment consists of activity modification and NSAIDs. Steroid injections are of no benefit. Immobilization in an above elbow splint with the elbow flexed 90° with slight forearm pronation and wrist flexion for 4–6 weeks may relieve pressure and traction on the median nerve. The majority of patients will respond to non-operative therapy.

Surgical indications
The diagnosis of this condition is based mainly on the exclusion of other causes. Many patients will have already undergone a carpal tunnel release. The main indication is failure to improve to conservative treatment combined with reproducible physical findings compatible with this diagnosis. Other straightforward indications are a positive EMG demonstrating denervation of the proximal forearm muscles or MRI evidence of nerve compression due to an underlying tumor.

Contraindications
A coexisting polyneuropathy, C6–7 radiculopathy or a brachial plexitis (Parsonage–Turner syndrome) may mimic the physical findings and should be ruled out, because these conditions will not benefit from a median nerve decompression.

Surgical technique
The procedure is performed under tourniquet control. An anteromedial incision is made crossing the antecubital fossa, starting 5 cm above the elbow flexion crease. The medial antebrachial cutaneous nerve is isolated as it runs with the basilic vein. The median nerve is identified proximal to the elbow, next to the brachial artery, and it is traced distally, resecting the ligament of Struthers if present. The nerve is followed through the two heads of the PT where branches from the anterior bundle (FCR, PT fascicles) are given off. Both the superficial head of the PT and the FDS can be divided and tagged for later repair if better exposure is needed. The posterior (AIN branch) and the middle (intrinsic, sensory) bundles pass deep to the superficial arch, which is also incised. Epineurotomy or internal neurolysis are of no benefit. Subcutaneous transposition of the median nerve is also not recommended.

Postoperative management
Postoperatively, the arm is placed in an above elbow splint for comfort with the elbow flexed 90°, the forearm in mid-pronation and the wrist in slight flexion. Gentle elbow range-of-motion exercises are begun after the first week unless it is necessary to protect the reattachment of the PT and/or FDS insertions. Resistive activities are avoided until 6–8 weeks after surgery.

Complications
One main complication is failure to improve due to misdiagnosis. Vascular injury is a significant risk and meticulous hemostasis is necessary. Injury to the proximal muscular branches ranging from neuropraxia to laceration can be minimized by careful nerve handling.

Outcomes
Surgical decompression generally yields good results. Olehnik et al. reported improvement in 77% of their patients, with 33 of 36 returning to work. A clinical series by Johnson et al. and Hartz et al. reported improvement in 92% of patients. Patients with normal NCS seem to fare better than patients with abnormal studies, which may be related to the severity of the compression.

Anterior interosseous nerve syndrome
Isolated anterior interosseous nerve (AIN) palsy is a rare occurrence, accounting for <1% of all upper extremity peripheral neuropathies. It is still uncertain whether this syndrome is due to a neuritis and/or a compressive neuropathy.

Anatomy
The AIN is the largest muscular branch to emerge from the median nerve. It innervates the FDP to the index and middle fingers, the FPL, and the pronator quadratus (PQ). The terminal portion also provides sensory innervation to the carpal joints. The AIN arises from the median nerve on the dorsoradial surface about 5–8 cm distal to the medial
epicondyle. The AIN travels between the FDS and FDP, and then passes dorsally in the interval between the FPL and FDP, giving off two to six branches to each of these muscles. The nerve reaches the anterior surface of the interosseous membrane and travels with the anterior interosseous artery, where it passes deep to the PQ, which it also innervates. It ends by sending sensory afferent branches to the intercarpal, radiocarpal, and distal radioulnar joints.

Pathophysiology
The AIN may be compressed by fibrous bands from the deep (most common) or superficial head of the PT, the fibrous arcade of the FDS, enlarged bursae or tumors, aberrant or thrombosed vessels, an accessory bicipital aponeurosis, and fractures of the forearm and distal humerus.

History
AIN syndrome (AINS) must be differentiated from a brachial plexus neuritis, i.e. Parsonage–Turner syndrome. In a brachial neuritis there is usually no history of trauma. The pain symptoms may be unrelated to the motor findings, and can appear spontaneously or following a viral illness or vaccination. By contrast, the patient with AINS will usually have a history of trauma or repetitive injury. They typically complain of vague, aching pain in the proximal forearm, which occurs at rest and is exacerbated by activity. The patient can have difficulty with writing and pinching activities, and may have sensory abnormalities in the median nerve distribution.

Physical examination
AINS is characterized by loss of function of the FPL and FDP to the index and sometimes the long finger and the PQ. Sensibility is unaffected. The patient cannot make an ‘O’ sign (see Fig. 19.1A). During attempted tip pinch, the index finger extends at the distal interphalangeal joint with compensatory increased flexion at the proximal interphalangeal joint. The thumb hyperextends at the interphalangeal joint with increased flexion of the metacarpophalangeal joint. The PQ can be tested by resisted pronation with the elbow flexed to relax the pronator teres.

AINS can be incomplete, with either weakness or absence of the FPL or FDP of the index finger alone and normal PQ function. All of the profundus tendons may be AIN-innervated, with subsequent weakness of all the fingers. The hand intrinsic muscles may be affected if there is a coexisting Martin–Gruber connection. This is where C8–T1 motor fibers destined for the hand intrinsic muscles travel in the median nerve. They then cross over to the ulnar nerve, usually through connections in the AIN (91%). If the FDS is also innervated by the AIN (30%), patients may present with weakness in this muscle as well.

One must differentiate between AINS and rupture of the FDP or FPL, which may occur from rheumatoid arthritis, Keinböck’s disease, and scaphoid non-union. The integrity of these tendons can be assessed by observing the tenodesis effect of the intact FPL and the index FDP, which causes passive thumb IP and index DIP flexion with passive wrist extension.

Electrodiagnostic studies
Shoulder girdle and upper limb EMG should be performed to rule out a brachial plexitis. In this case there will be membrane instability in the proximal limb muscles. The most appropriate technique involves recording the compound motor action potential (CMAP) from the pronator quadratus. The active surface electrode is placed in the midline dorsally, 3 cm proximal to the ulnar styloid. The median nerve is stimulated at the cubital fossa. The mean onset latency for the PQ is 3.6 ± 0.4 ms, with a side-to-side difference of 0.4 ms. Normal amplitudes range from 2.0 to 5.5 mV. Side-to-side comparative latencies are helpful in establishing the diagnosis. EMG testing of the PQ and FPL muscles should demonstrate signs of membrane instability (Fig. 19.1B).

Quantitative sensory testing
This should be entirely normal because the condition only affects a motor branch. Any abnormalities are due to unrelated causes and conditions.

Surgical indications
If there is no improvement either clinically or by EMG studies 4–6 months after onset, surgical exploration is indicated. Even though return of function has been reported up to 18 months after onset of symptoms, expectant treatment is less predictable than surgical intervention.

Surgical technique
The surgical approach for AIN palsy is essentially the same as for pronator syndrome. The median nerve is followed through the two heads of the PT where branches from the anterior bundle (FCR, PT fascicles) are given off. Both the superficial head of the PT and the FDS can be divided and tagged for later repair if better exposure is needed. The posterior (AIN branch) and the middle (intrinsic, sensory) bundles pass deep to the superficial arch, which is incised (Fig. 19.3A–E). A microscopic interfascicular neurolysis of the AIN up to the elbow should be considered in cases in which there is no obvious compression site, because hourglass-like fascicular constrictions that are not discernible through the intact epineurium have been reported.

Postoperative management
The arm is placed in a bulky, above-elbow plaster splint that maintains the elbow in 90° of flexion, the forearm in 45° of pronation, and the wrist in slight flexion for comfort.

Complications
The anatomy and hence the complications are virtually identical to those of pronator syndrome.

Outcomes
Clinical improvement may be seen for up to 1 year following surgery, but recovery is often incomplete (see Fig. 19.1C). Non-surgical management is recommended for brachial plexus neuritis-induced AIN palsy.
FIGURE 19.3 Anterior interosseous nerve compression. A Inability to perform the “O” sign. B Needle placement during EMG of the pronator quadratus. C Anteromedial approach to the median nerve. Note the brous border of the sublimus muscle. PT = pronator teres, AIN = anterior interosseous nerve. D Following release of the sublimus arch (FDS). E Clinical photo at 8 months showing active extension of the distal phalanx of the index but no active thumb interphalangeal joint extension. (From Slutsky DJ. Electrodagnostic studies of the upper extremity. In: Slutsky DJ, Hentz VR, eds. Peripheral nerve surgery: practical applications in the upper extremity. Philadelphia: Elsevier,
ULNAR NEUROPATHIES

Proximal ulnar neuropathy

Ulnar nerve compression at the elbow is the second most common neuropathy in the upper limb after carpal tunnel syndrome.

Anatomy

The ulnar nerve arises from the medial cord of the brachial plexus and contains the nerve root fibers from C8 to T1. It provides the motor supply to the hypothenar muscles, the ulnar two lumbricals, the interosseous muscles, the adductor pollicis, the FCU and the profundus muscles to the ring and small fingers. Its sensory distribution includes the palmar surface of the small and the ulnar half of the ring finger, as well as the dorsoulnar hand. It lies medial to the axillary artery and continues distally to the mid-arm. It then passes through a bony canal (erroneously labeled the arcade of Struthers) from the anterior to the posterior compartments of the arm. The canal is approximately 6 cm long, with the proximal extent located approximately 10 cm from the medial epicondyle. The proximal opening of the canal is a V-shaped window made up of the medial intermuscular septum, which divides the anterior and the posterior compartments of the arm and the internal brachial ligament. The internal brachial ligament supports the origin from the inferior border of the teres major to its fusion back to the septum. This layer appears as an oblique muscular curtain covering the medial aspect of the ulnar nerve. In one anatomical study, the internal brachial ligament was present in 22/30 specimens. The septum makes up the anterior part of the canal; the rest is made up of deep investing fascia and epimysium of the triceps muscle. The nerve is often accompanied by the superior ulnar collateral artery. At the elbow, it lies between the medial epicondyle and the olecranon, where it is covered by Osborne’s ligament. It enters the forearm between the two heads of the FCU and is covered by a fibrous aponeurosis (the cubital tunnel). It runs deep to the FCU as far as the distal forearm.

Pathophysiology

The ulnar nerve may be compressed anywhere from 10 cm proximal to approximately 5 cm distal to the medial epicondyle. Five potential sites for compression have been described. The most proximal site is the point at which the ulnar nerve takes a direction change as it passes through the V-shaped canal from the anterior to the posterior compartments of the arm. Hourglass indentations of the nerve were found at this point in 11 of 11 cadaver arms. Although this has been labeled the arcade of Struthers, recent investigators have shown this to be historically incorrect. They note that Struthers did not describe an arcade over the ulnar nerve, but did describe the internal brachial ligament. Other investigators, however, have noted a musculo-tendinous arcade in eight of 60 cadaver arms that could form a secondary compression site after anterior transposition of the ulnar nerve. The second site is at the medial epicondyle, where a cubitus valgus deformity is present. The third site is the olecranon groove, which is a fibro-osseous tunnel bounded anteriorly by the medial epicondyle, laterally by the olecranon and ulnohumeral ligament, and covered by a fibroaponeurotic band. Compression at this site can be caused by lesions within the groove, such as tumors, conditions outside the groove such as external compression by anomalous muscles, and conditions that predispose the nerve to displace from the groove. The fourth site is where the nerve passes between the two heads of the flexor carpi ulnaris (FCU) muscle. The floor of the passageway through FCU is the medial collateral ligament of the elbow and the roof is a fibrous band (Osborne’s ligament or the arcuate ligament), which is a continuation of the fibroaponeurotic covering of the cubital groove.

At the elbow, the ulnar nerve contains about 20 fascicles, including the motor branches to the forearm muscles. The fascicles within a nerve are not uniformly affected by compression. Those on the periphery of a nerve sustain greater injury than centrally placed fascicles. The motor fascicles to the FCU and the intrinsic muscles are centrally located, whereas the sensory fibers are superficially located. The usual sites of compression in cubital tunnel syndrome are superficial to the nerve (Osborne’s ligament, arcade of Struthers). The internal topography of the ulnar nerve at the elbow explains the relative sparing of the flexor carpi ulnaris and flexor digitorum profundus because their motor fibers lay deep within the nerve. The intrinsic muscles are often uninvolved until the late stages of compression for similar reasons, whereas the superficially located sensory fibers are more susceptible to early compression.

FIGURE 19.4  Anconeus epitrochlearis. Note how this anomalous muscle attaches to the fibroaponeurotic bands (forceps) which arc over the ulnar nerve (*) as it traverses the olecranon groove.
History
The patient presents with complaints of numbness and/or tingling in the small and/or ring finger. Symptoms can range from mild numbness in the ring and little fingers to severe pain on the medial aspect of the elbow, with dysesthesias radiating distally into the hand. In chronic cases patients may complain of a loss of dexterity with fine manipulation tasks. Symptoms are provoked by repetitive or sustained elbow flexion activities.

Physical examination
The ulnar nerve is palpated for enlargement or subluxation during elbow flexion. There may be a positive Tinel’s sign at the epicondylar groove or over the proximal FCU. An elbow flexion test is performed by placing the elbow in full flexion with a hyperextended wrist, while manual pressure is applied to the nerve for 1 minute. The test is considered positive when paresthesia and/or numbness occur in the ulnar nerve distribution of the hand. False positive results occur in approximately 10% of patients.

There may be a sensory deficit involving all or part of the ulnar nerve distribution, including the dorsoulnar wrist.

When intrinsic muscle weakness is severe, there is often clawing of the ring and little fingers. One may see a positive Froment’s sign (flexion of the interphalangeal joint of the thumb with side pinch), a positive Jeanne’s sign (hyperextension of the metacarpophalangeal joint of the thumb) and paradoxical abduction of the small finger due to a paralyzed third palmar interosseous (positive Wartenberg sign). Extrinsic weakness may involve the flexor digitorum profundus to the little finger and ring finger. Weakness of the flexor carpi ulnaris rarely occurs.

Non-operative treatment
Most acute or subacute cases are treated with activity modification to avoid activities that require repetitive elbow flexion and to reduce external pressure from leaning on their medial elbow or forearm. Alterations can sometimes be made in the workplace, such as positioning a computer keyboard so that the operator’s elbows are not acutely flexed. When symptoms are severe and have persisted for weeks, temporarily immobilizing the elbow in approximately 35° of flexion and the wrist in neutral may provide relief. Patients are instructed to wear the splint day and night for 3–4 weeks. Non-steroidal anti-inflammatory drugs can be helpful, but corticosteroid injections around the nerve are ineffective.

Indications for surgery
Sensory loss in the ulnar nerve distribution with nerve studies and clinical evidence of intrinsic muscle denervation are major indications. In these cases, conservative treatment is not justified. Across elbow motor conduction <45m/s with a >20% drop in amplitude is another strong indication. Relative drops of >10 m/s are a softer indication that should be approached cautiously because technical error can produce this quite easily. Focal slowing on segmental stimulation is another appropriate indication.

Contraindications
Ulnar nerve symptoms due to brachial plexus compression from a Pancoast tumor or a C8–T1 radiculopathy are infrequent contraindications. Some authors maintain that normal electrodiagnostic studies are another contraindication to surgery. Patients with normal electrodiagnostic studies but appropriate signs and symptoms (McGowan Stage I: paresthesia and numbness but no weakness) can still benefit from surgery and are appropriate candidates if conservative measures over a 3–6 month period have failed.

Surgical technique
Surgery can be divided into two groups of procedure: decompression without transposition of the ulnar nerve, and decompression with transposition of the nerve. The procedures are performed under axillary block or general anesthesia using a tourniquet.

Subcutaneous anterior transposition
A 5 cm posteromedial incision is made centered on the medial epicondyle. Branches of the medial antebrachial cutaneous nerve are identified and protected. The deep fascia overlying the proximal ulnar nerve is divided, as well as any associated ligament of Struthers. The fascia over the nerve is released sequentially, first in the upper arm, followed by the brachial antebrachial covering of the epicondylar groove, then Osborne’s ligament at the cubital tunnel, and finally the fascia where the nerve passes between the two heads of the FCU. A 1 cm segment of the medial intersubscapular septum is resected where it attaches to the epicondyle to prevent a secondary site of compression, taking care to prevent injury to fragile veins medial to the septum. Articular branches to the elbow are divided, but motor branches to the FCU should be preserved. A vessel loop is placed around the nerve, which is then gently transposed anterior to the medial epicondyle along with the superior ulnar collateral ligament, exor–pronator mass then repaired with 2/0 non-absorbable sutures (Fig. 19.6A–C). Postoperatively the elbow is immobilized at 90° for 4 weeks, allowing elbow flexion but no extension. The elbow is then gradually extended over the ensuing 2–4 weeks.

Submuscular transposition
A slightly larger incision is made and the nerve is decompressed in a similar fashion. A z-plasty lengthening of the common flexor origin can be turned down and sutured to the proximal skin edge to prevent posterior subluxation of the nerve. The patient uses a sling for comfort postoperatively and is started on progressive elbow extension as tolerated. These same principles can be applied to a mini-incision technique (Fig. 19.5).
Complications
The most dire complication is ulnar nerve laceration. More commonly, injury to the medial antebrachial cutaneous nerve results in a tender scar and hypoesthesia along the medial forearm. Injury to the superior ulnar collateral artery or its branches is a cause of postoperative hematoma. The median nerve lies on the brachialis just behind the medial intermuscular septum, and is at risk during resection of the distal septum or with aggressive retraction. Large intramuscular veins medial to the septum can also be injured during this step and are very difficult to ligate. Secondary entrapment due to creation of a fascial sling can occur with subcutaneous transpositions. Postoperative ulnar nerve subluxation due to a failure of this sling can result in painful paresthesia with elbow flexion. Secondary impingement may also occur following a submuscular transposition due to a failure to release the deep fibers of the sublimus origin on the medial epicondyle. The ulnar collateral ligament can also be injured during this step, resulting in postoperative elbow instability.

Outcomes
As in CTS, the patient’s clinical findings and response to conservative measures should be a major determinant in the surgical decision making. In one study, 92% of patients recovered grade 3 motor strength or better.\(^6\) The results are largely determined by the preoperative degree of compression. When there are constant symptoms and demyelination, recovery may take 6–8 months. Residual sensory complaints are common. Even though intrinsic wasting rarely recovers in an adult, prevention of further denervation is crucial.

Distal ulnar neuropathy
Anatomy
The ulnar tunnel or Guyon’s canal is approximately 4 cm long; it begins at the proximal edge of the carpal transverse...
Techniques for Nerve Compression Syndromes

Arch. It is bounded medially by the pisiform, and radially by the volar carpal ligament and the roof consists of the continuation of the deep forearm fascia, the volar carpal ligament. The ulnar nerve has 15–25 fascicles at the wrist, and can be clearly divided into a volar sensory component and a dorsal motor component. At the wrist, the ulnar nerve passes over the TCL, medial to the ulnar artery, through Guyon’s canal. The deep motor branch is given off at the pisiform and passes underneath a fibrous arch to lie on the palmar surface of the interossei. It crosses the palm deep to the flexor tendons, to terminate in the adductor pollicis and the ulnar head of the flexor pollicis brevis.

Pathophysiology
The standard teaching divides the sites of compression in Guyon’s canal into three zones. In zone I, nerve compression leads to mixed motor and sensory symptoms. In zone II symptoms are purely sensory, and in zone III symptoms are purely motor and restricted to muscles innervated by the deep ulnar motor branch. Two sites of entrapment distal to the abductor digiti minimi (ADM) have also been described. In these cases the ADM will be preserved, but there is weakness and wasting of the intrinsic muscles.

History
The patient may also present with complaints of numbness and tingling of the small and/or ring finger. The dorsoulnar aspect of the hand is not affected. The patient may give a history of repetitive pounding using the hypothenar eminence that may occur with autobody technicians, martial artists and Kodo drummers. Cyclists who ride for extended periods in the crouching position may entrap the deep motor branch against the hamate hook while grasping the handlebars. A history of Raynaud’s symptoms should alert one to the possibility of ulnar artery thrombosis.

Physical examination
There are no characteristic findings of ulnar tunnel entrapment per se. A Tinel’s sign may be present at the wrist, but not the elbow unless there is an associated cubital tunnel entrapment. Intrinsic muscle atrophy may also occur in chronic compression, but the FCU and the FDP are not affected. There should be a negative Tinel’s sign at the elbow and a negative elbow flexion test. If there is an associated ulnar artery aneurysm there may be a palpable thrill and an audible bruit. With ulnar artery thrombosis, the Allen’s test will be positive for ulnar artery occlusion. With an associated fracture of the hook of the hamate, there will be localized tenderness in the palm, 1 cm radial and 1 cm distal to the pisiform. Ancillary testing such as ultrasound, CT, angiography and MRI, may be used to aid in the diagnosis of these associated entities.

Electrodiagnostic studies
The usual nerve conduction studies are inadequate in assessing ulnar nerve entrapment in the palm. Short segment incremental studies (SSIS) are a sensitive and specific way to assess the deep motor branch because focal conduction abnormalities also tend to be normalized over the distance between the ADM and the FDI. The ulnar nerve is stimulated in 1 cm increments from 3–4 cm proximal and distal to the wrist crease. Abnormal values include a >0.5 ms jump or a >120% drop in amplitude. When this is combined with FDI conduction and interosseous-latency differences, the diagnostic yield increases.

Quantitative sensory testing
The 2PS to the small finger is the first to go, followed by an abnormal 1PS. The dorsoulnar wrist should remain normal unless there is an associated C8–T1 radiculopathy.

Non-operative treatment
The mainstay of treatment is activity modification. Cyclists should avoid riding in the crouching position with their hands low on the handlebars because this is a recognized precipitant of symptoms; they should change their hand position frequently. Autobody repair technicians, martial artists and Kodo drummers should avoid repetitive percussion on the ulnar border of their palm. Wrist splinting and cortisone injections have no role in this condition.

Indications for surgery
Intrinsic muscle wasting and/or sensory loss are a sine qua non for decompression. The presence of a mass occupying lesion also mandates surgical treatment. Ulnar artery thrombosis or aneurysm may be treated with ulnar artery repair or ligation.

Contraindications
Ulnar motor and/or sensory disturbances due to more proximal causes preclude a distal release. Ulnar sensory disturbances in CTS were a common indication for release in the 1980s, but have since become an infrequent indication for the release of Guyon’s canal because the majority of patients experience improvement following CTR.

Surgical technique
The ulnar nerve is identified proximal to the distal wrist crease between the FCU and the flexor tendons through a curving incision that crosses the wrist obliquely and bisects the interval between the pisiform and the hook of the hamate. The ulnar nerve is followed distally as the volar carpal ligament is released. The ulnar artery is inspected for thrombosis or aneurysm. The fibrous arch of the hypothenar muscles is incised and the floor of the canal is explored for masses, fibrous bands or anomalous muscles. With entrapment in the palm, the deep motor branch is followed distally as it traverses the palm lying on the interosseous fascia, deep to the flexor tendons and superficial palmar arch. The dissection is completed as the motor branch ends in the muscle belly of the adductor pollicis (Fig. 19.7A–D).

Complications
These are mostly related to injury to the branches of the ulnar nerve or artery. Injury to the palmar cutaneous branches of the ulnar nerve or the nerve of Henle (if present)
FIGURE 19.7 Distal ulnar neuropathy. A Dorsal preoperative photograph of attempted index finger abduction. Note the marked wasting of the first, second and third dorsal interossei. B Volar preoperative photograph of attempted index finger abduction. Note the marked wasting of the first, second and third dorsal interossei, but the normal bulk of the abductor digiti minimi (arrows).

may result in scar tenderness and hypoesthesia. Uncom-
monly injury to the pisiform ligament complex can result in
instability of the pisotriquetral joint.\textsuperscript{65}

Outcomes
Clinical recovery is seen in the majority of patients when
the ulnar nerve entrapment is due to a space-occupying
lesion.\textsuperscript{66} Motor recovery is less predictable than sensory
recovery, especially when the compression is due to a fi-
brotic hypothenar arch or is long-standing.\textsuperscript{67}

RADIAL NEUROPATHIES

Upper arm
Anatomy
The radial nerve arises from the posterior cord of the bra-
chial plexus and receives contributions from C5–C8 spinal
roots. The nerve contains approximately 16,000 myelini-
ted fibers.\textsuperscript{12} At the level of the coracobrachialis it courses
posteriorly to lie in the spiral groove of the humerus. In the
lower arm it pierces the lateral intermuscular septum to run
between the brachialis and the brachioradialis. Oppo-
site the head of the radius there are some fibrous bands from
the joint’s capsule, and immediately distal to this the
nerve is regularly crossed by several prominent veins, the
‘leash of Henry.’ It divides 2 cm distal to the elbow into a
superficial radial sensory branch (SRN) and a deep motor
branch, the posterior interosseous nerve (PIN). Before giving
off the PIN branch it gives off branches to the extensor carpi
radialis longus and brevis, brachioradialis and anconeus. The
PIN continues on between the superficial and deep head of
the supinator muscle, to exit on the dorsal forearm. After it
emerges from the distal border of the supinator, the PIN sends
branches, in descending order, to the extensor carpi ulnaris, extensor digiti quinti, extensor pollicis longus and brevis and the extensor indicis proprius, although there may be considerable variation.

History
In the arm region the radial nerve is often injured in
association with some form of unconsciousness. In a ‘Sat-
urday night palsy’ an obtunded patient sits with the arm
over a chair back or rests his/her head on the lateral
surface of the arm. Alternatively, the radial nerve can be com-
pressed in the groove between the brachialis and forearm muscles when one person rests their head on the middle third of the arm of another, for example in
‘Honeymooner’s palsy.’
Physical examination
The patient will typically present with a wrist drop and an inability to extend the fingers, thumb or wrist. In addition, the brachioradialis will be affected along with variable involvement of the triceps. There will also be diminished sensation over the dorsum of the first web space. The NCS typically demonstrates the absence of the superficial radial SNAP. Motor recordings are more difficult because no muscle is sufficiently isolated from other radially innervated muscles.

Indications for surgery
For the majority of patients, non-operative treatment is the mainstay. Failure to improve within 6 months, combined with a non-advancing Tinel’s sign, is an indication for exploration.

Contraindications
The time for reinnervation must take the distance from the injury to the motor endplate into account. As a general rule, motor endplates degrade at about 1% per week, and the nerve regenerates about 1 inch per month. By 12 months, the nerve will have grown approximately 12 inches and there will be a 50% loss of endplates, hence the maximum length that a nerve can grow to restore motor function is approximately 13–18 inches. For practical purposes, nerve decompression will be of no value with injuries that are more than 18 months old (>75% loss of endplates) and alternative methods should be explored.

Surgical technique
A 6–8 cm incision is made over the posterolateral aspect of the midhumerus. The radial nerve is identified in the spiral groove and followed distally through the intermuscular septum. Any obvious areas of nerve constriction or loss of the normal striations (bands of Fontana) should undergo epineurolysis. The use of intraoperative nerve stimulation will help differentiate a neuroma-in-continuity from non-viable nerve tissue. In the former case an internal neurolysis is justified rather than excision and grafting.
Postoperative management
Immediate elbow mobilization is instituted following nerve decompression or neurolysis. Nerve grafting may require temporary elbow splinting for 4 weeks, but it is preferable to insert a graft of sufficient length to allow early elbow extension.

Complications
The radial nerve is accompanied by the radial collateral artery in the spiral groove, which is at risk during decompression. Injury to the muscular branches may result in permanent denervation of one or more heads of the triceps. Injury to the posterior cutaneous nerve of the forearm may result in a tender scar and hypoesthesia.

Radial tunnel syndrome
History
The presenting complaint in radial tunnel syndrome is proximal forearm pain, often coexisting with lateral epicondylitis, without sensory or motor loss. The patient often gives a history of performing repetitive pronation/supination activities, such as using a screwdriver. The symptoms of radial tunnel syndrome often coexist and overlap those of lateral epicondylitis.

Physical examination
A number of provocative tests have been described, including resisted extension of the middle finger, which tenses the ECRB and entraps the nerve, tenderness over the supinator muscle, and pain with resisted supination. None is pathognomonic for this condition. A diagnostic local anesthetic block of the PIN, which produces a temporary wrist drop, is completely relieved. Classically the NCS is normal in radial tunnel syndrome.

Non-operative management
The majority of cases will resolve with modification of activity. In the early stages above-elbow splinting with the elbow flexed to 90° and the forearm in supination will relieve the dynamic compression and allow the inflammatory response around the PIN to subside. PIN gliding exercises will help maintain nerve excursion: these consist of simultaneous elbow extension, forearm pronation, wrist flexion and ulnar deviation. In many instances the treatment that is instituted for a coexisting lateral epicondylitis will also resolve the symptoms of radial tunnel compression.

Indications for surgery
Persistent proximal forearm pain that does not resolve despite appropriate activity modification is an indication for decompression, especially if a lateral epicondylectomy has already been performed. Complete pain relief following a diagnostic PIN block is a good predictor of at least partial improvement following surgery.

Contraindications
The existence of this disorder is still questioned by some authors. All efforts should be made to rule out other causes of pain, such as radiocapitellar joint disorders, lateral elbow instability, and untreated lateral epicondylitis.

Surgical technique
The volar approach to the radial nerve is through an 8-cm anterolateral incision under tourniquet control. The muscular fascia is divided and the intermuscular interval between the brachialis and brachioradialis is developed with blunt dissection. Recurrent branches from the radial artery must be ligated to gain access to both nerve branches. The radial nerve is identified proximal to the elbow and followed distally. At the level of the radial head, the radial nerve gives off branches to the ECRB and brachioradialis. It then divides into the superficial radial nerve branch, which travels distally under the brachioradialis. The PIN continues distally and is crossed by the radial recurrent vessels, which are ligated. The proximal border of the supinator muscle (the arcade of Frohse) is divided along with the superficial head of the supinator (Fig. 19.8A,B). At this point the PIN disappears from view as it penetrates the dorsal extensor compartment. If there is a suspicion of distal entrapment, a separate dorsal approach to the PIN is necessary. This can be accomplished by extending the incision distally and dorsally, or by making a separate incision. The PIN nerve is then approached through a dorsolateral approach, developing the plane between the extensor carpi radialis brevis and the extensor digitorum communis (Fig. 19.9A–D). At this level, the PIN contains motor fibers only, hence separate fascicle identification is unnecessary. The distal border of the supinator is delineated and divided.

Outcomes
Early reports of radial tunnel decompression were generally optimistic. Beasley et al. reported 80% good or excellent results in his series of 109 decompressions. A recent review by Sotereanos et al. however, found good results in only 11 of 28 patients, with many experiencing residual symptoms. The results were worse in patients receiving worker’s compensation.

Complications
The potential complications are similar for radial tunnel syndrome and PIN decompression. The main indication to ligate the radial leash of vessels is to prevent postoperative bleeding, because they are a rare cause of compression by themselves. The superficial radial nerve is at risk of injury through laceration or retraction. Persistent radial tunnel syndrome due to unrelieved PIN compression at the distal border of the supinator may require repeat surgery.

Posterior interosseous nerve entrapment
History
In posterior interosseous nerve syndrome the presenting symptoms are weakness and/or paralysis of the extensor
Techniques for Nerve Compression Syndromes


muscles, which results in a wrist or finger drop. There may be a history of a fall onto an extended and pronated arm, although many cases are spontaneous, especially if due to an underlying lipoma, ganglion or rheumatoid nodule arising from the radiocapitellar joint.

Physical examination
The patient will present with variable weakness or paralysis of the EPL, EIP, EDC and ECU. Motor function of the ECRB/L should be preserved because they are innervated before the PIN dives between the two heads of the supinator muscle. The patient will therefore extend their wrist in radial deviation. PIN lesions do not affect the superficial radial SNAP, which should be normal. The compound motor action potential of PIN-innervated muscles may show a drop in conduction velocity or amplitude, but this is difficult to assess with surface electrodes. Needle EMG is the best technique for localization, especially with partial lesions. The management of this syndrome is identical to that for radial tunnel syndrome.

Superficial radial nerve entrapment
Anatomy
The radial sensory nerve exits from under the brachioradialis approximately 5 cm proximal to the radial styloid, and bifurcates into a major volar and a major dorsal branch at a mean distance of 4.2 cm proximal to the radial styloid. It then moves distally, where it supplies sensation to the dorsum of the thumb, the first web space and the dorsoradial aspect of the carpus, extending up to the index and middle fingers.

Pathophysiology
The superficial radial nerve (SRN) can be injured in the distal forearm or at the wrist by tight bracelets or watch bands, handcuffs, radius fractures, lacerations, venous cutdown and blunt trauma. The SRN may also be entrapped as it exits the fascia between the tendons of the BR and ECRB.

History
Pertinent history may include compressive or crushing forearm injuries, work activities requiring frequent pronation and wrist hyperextension, and associated illnesses, such as diabetes. Symptoms included altered sensibility over the dorsoradial aspect of the hand, and dorsoradial cutaneous pain with ulnar flexion of the wrist or with grip- ping and pinching.

Physical examination
Physical examination includes altered touch perception, moving 2PD >15 mm, static 2PD that is 5 mm greater than
Quantitative sensory testing
The patients will have an abnormal 2PS and possibly 1PS over the dorsal web of the first web.

Electrodiagnostic studies
The distal radial sensory latency may be normal even in the presence of abnormal forearm conduction. This commonly occurs with nerve entrapment due to segmental conduction velocity slowing. In more advanced cases slowing or a complete block of the distal SRN occurs. If the response is absent, it is difficult to localize the lesion.

Indications for surgery
Failure to improve following conservative treatment, with avoidance of repetitive wrist deviation and tight bands or jewelry, is an appropriate reason.

Contraindications
De Quervain's tenosynovitis often includes a component of superficial radial nerve irritation and should be treated before considering SRN decompression.

Surgical technique
A 2 cm incision is made approximately proximal to the radial styloid. The superficial radial nerve is identified as it exits from underneath the tendon of the brachioradialis. The overlying fascia is split, care being taken not to disturb the nerve.

Outcomes
Dellon and MacKinnon reported on a group of 51 patients with complaints related to entrapment of the superficial sensory branch of the radial nerve. Seven (37%) of 19 patients treated with non-operative modalities were improved after a mean of 28 months from the onset of symptoms or their injury. Of the 32 patients treated with surgery, there was excellent subjective improvement.
37%, good subjective improvement in 49%, and fair subjective improvement in 6%; 8% were not improved.

Complications

The complications for each procedure are similar. They include wound problems related to infection, skin healing, tender scars due to injured cutaneous nerves, hematoma and iatrogenic injury due to rough nerve handling and/or retraction. These can be minimized by meticulous hemostasis, gentle nerve handling, and precise surgical technique. Stiffness can occur and is minimized by early joint mobilization. The incidence of residual symptoms is predicated by the preoperative degree of nerve injury.

CONCLUSIONS

It is apparent that compressive neuropathies share similar features. Although each focal neuropathy has been discussed in isolation, multiple compressive neuropathies often coexist. The element common to all focal neuropathies is nerve ischemia, which leads to the sensory and/or motor abnormalities in the distribution of the affected nerve that is affected. Provocative tests exploit this feature by seeking to dynamically increase the ischemia through external pressure and/or traction in order to magnify the symptoms. Although ancillary testing can yield useful information, most hand surgeons intuitively understand that the indication for surgery still hinges on reproducible physical findings combined with the appropriate clinical symptoms rather than on a test abnormality.

REFERENCES