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Compression and entrapment neuropathies of the upper extremity

William W. Eversmann, Jr., M.D.*

Entrapment neuropathies are nerve injuries that occur in predictable areas; they are evaluated in similar fashion and probably have a common pathophysiology. Ionic, mechanical, and vascular lesions are involved in the pathophysiologic mechanism of entrapment neuropathies.[1] The initiating mechanism in the neurologic disturbance of a compression neuropathy is often uncertain in any particular patient; in one patient the ionic lesion may be most prominent as the initiating factor, while in another a vascular or mechanical lesion may produce the initial symptoms. The vascular lesions seem to be most understandable from the standpoint of pathophysiologic mechanisms.[2] Study of the vascular anatomy of nerves over the last 300 years has revealed a segmental vascular supply to the nerve trunk that is carried in a mesoneurium, permitting motion and allowing changes of position and tension of the nerve with motion of the joints of the extremity.[3] The arcades of the mesoneurium vary with location. The median nerve at the wrist is supplied by a mesoneurium from the anterior medial side of the nerve proximal to the transverse carpal ligament as well as by a series of vessels from the superficial palmar arch distal to the transverse carpal ligament.[3, 4] The smaller vessels enter the epineurium of the nerve and immediately divide into ascending and descending epineural branches. The epineurial network of vessels, after forming an anastomotic network in the subepineurium of the nerve, further subdivides to a vascular plexus at the perineurium. The capillary bed within the nerve itself is contained within the fascicles of the nerve so that from the perineurial level of vascular plexus, small end arteries and capillary beds form the remainder of the vascular network within the nerve trunk.[3]

Obstruction of venous return from the nerve initially causes venous congestion in the epineurial and perineurial vascular plexuses and a generalized slowing of circulation within the nerve trunk. Anoxia of a nerve segment results. This anoxia leads to dilatation of the small vessels and capillaries within the nerve, and endoneural edema of the tissue results.[5] The swelling of the nerve attendant to this edema increases the effect of the original compression, with further slowing of venous return; if this is allowed to persist for prolonged periods, fibroblasts proliferate within the nerve. The proliferation of fibroblasts, of course, will result in permanent scarring within the nerve, further rendering segments of the nerve anoxic because of a barrier of fibroblasts that inhibits circulation within the nerve and the exchange of vital nutrients between the vascular system and the nerve fibers.

The axoplasmic transport system within the neuron transports protein molecules synthesized in the endoplasmic reticulum of the cell body to a location in the axon where these molecules are active, either along the wall of the axon or at the terminal endings.[6] Accordingly, protein polypeptides, glycolipids, glycoproteins, catecholamines, and acetylcholinesterase are transported from the cell body where they are synthesized along the axon by some mechanism of axoplasmic transport, such as transport filaments, microtubules, or neurofilaments, or even by simple diffusion. Axoplasmic transport uses as its energy source the segmental axonal mitochondria, which, through oxidative phosphorylation, generates high-energy phosphate to maintain the system of transport, as do the sodium pump and cell membrane. When a segment of the axon is rendered ischemic by reduced blood flow, possibly as small a reduction as 30% to 50% of normal, the resultant loss of oxidative phosphorylation and high-energy phosphate will lead to some decreased efficiency of the sodium pump and the axoplasmic transport system and some reduction of maintenance in the cell membrane; this in turn will lead to a loss of conduction and transmission[2] by the axon. The segment of axon rendered ischemic or relatively ischemic, through either a change of position, local anatomy, or internal pressure, not only will react through a series of vascular mechanisms, but by so doing, will alter its own ionic relationship to its environment and further aggravate the normal internal pressure of the nerve trunk, resulting in a deterioration of its normal function. The swift reversibility of intraoperative motor conduction latencies associated with carpal tunnel syndrome suggests that the vascular lesion may be paramount in the production of a compression neuropathy.[2]

Reprint requests: William W. Eversmann, Jr., M.D., 411 Tenth St. S.E., Cedar Rapids, IA 52403.

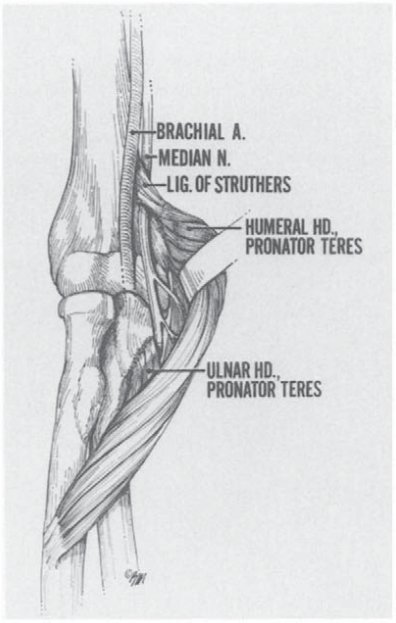

Fig. 1. The reflection of the humeral head of the pronator teres, as the ligament of Struthers passing over the median nerve can cause compression of the nerve above the elbow joint. The brachial artery may or may not be involved in the compression. (Reprinted with permission of Elizabeth Roselius, M.S., A.M.I., from Green DP, editor: Operative hand surgery. New York/London, 1982, Churchill Livingstone.)

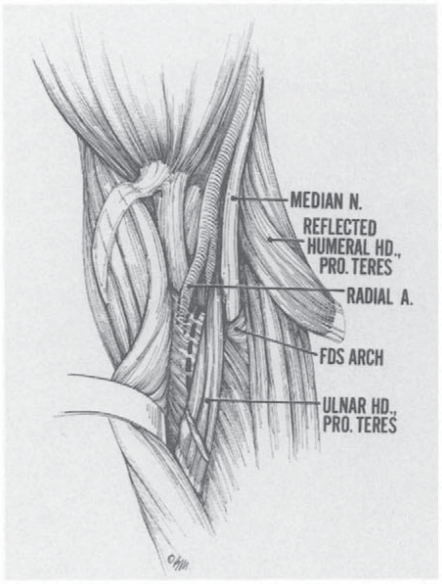

Fig. 2. The fourth component of the pronator syndrome. The arch of the flexor superficialis should be released by division of the fascial arch of the superficialis and elevation of the first 1 or 2 cm of the radial border of the superficialis as indicated by the *broken line*. (Reprinted with permission of Elizabeth Roselius, M.S., A.M.I., from Green DP, editor: Operative hand surgery. New York/London, 1982, Churchill Livingstone.)

Entrapment neuropathies of the median nerve

Although three distinct entrapment neuropathies of the median nerve have been described, their clinical presentation often is similar. Accordingly, the differentiating points in both clinical presentation and examination must be carefully sought by the surgeon to successfully eliminate one or more of the possibilities from the differential diagnosis.

Pronator syndrome. The pronator syndrome, the most proximal entrapment neuropathy of the median nerve,[7] presents with pain on the proximal volar surface of the distal arm and proximal forearm, which is generally increased with activity and may be associated with reduced sensibility or at least sensory symptoms in the radial three and a half digits of the hand. The wrist flexion test of Phalen is notably negative in this syndrome. After the usual examination of the upper extremity, the surgeon suspecting this syndrome should pay particular attention to functional muscle tests that may aggravate the symptoms of pain, weakness, and numbness associated with pronator syndrome. Accordingly, if the symptoms are aggravated by flexion of the elbow against resistance between 120° and 135° of flexion, either a Struthers ligament or lacertus fibrosus level of compression should be suspected.[8, 9] If the symptoms are increased by resistance to pronation of the forearm, usually combined with flexion of the wrist, one must be suspicious of the pronator muscle being instrumental in compression of the median nerve.[1, 10] If the symptoms of the syndrome are aggravated by resisted flexion of the flexor superficialis muscle of the middle finger, the arch of the superficialis should be carefully inspected at the time of surgery. Electrodiagnostic studies should confirm the clinical diagnosis by localization of the lesion with nerve conduction velocity and by electromyographic investigation of the specific neuromuscular pattern of the syndrome. The value of the electrodiagnostic studies will depend directly on the surgeon's ability to control a multitude of variables inherent to these determinations, not the least of which is the expertise of the electromyographer.

Surgery for pronator syndromes should consist of a detailed exploration of the median nerve. The exploration begins 5 cm above the medial epicondyle, searching for a small hook-shaped process of bone, the supercondyloid process, from which an accessory origin of the pronator teres muscle (by a ligament described by Struthers[8, 9]) may compress the median nerve and/or brachial artery (Fig. 1). If isolated, both the ligament of Struthers and the supercondylar process should be resected. The second site of compression might be found at the fascia of the lacertus fibrosus, coursing from the bicipital tendon over the flexor mass of the proximal forearm.[11] The third possible site of compression at the pronator teres muscle may be caused by reflections of muscle fascia forming fibrous bands, the sharp aponeurotic edge of the deep head of the pronator muscle, or simple hypertrophy of the muscle itself.[1, 10] The fourth site of compression in the pronator syndrome will be visualized after the superficial head of pronator teres is reflected proximally and after the flexor digitorum superficialis from the radial side is elevated with a small retractor; the radial attachment of this muscle forms a tendinous aponeurotic arch under which the median nerve passes (Fig. 2). This arch constitutes the distal extent of the exploration for entrapment of the median nerve from pronator syndrome. Operations of a more limited scope carry with them an inherent risk of failure by limiting one's ability to recognize multiple areas of compression.

Anterior interosseous syndrome. Entrapment of the an-

terior interosseous nerve is generally manifested by a vague pain in the proximal forearm that is increased with exercise and relieved by rest. This syndrome is characteristically without sensory signs or symptoms,[12] but on clinical examination, there is weakness or paralysis of the flexor digitorum profundus of the index finger, the flexor pollicis longus, and possibly the pronator quadratus muscle. The characteristic posture of pinch in this syndrome consists of hyperextension of the distal interphalangeal joint of the index finger and hyperextension of the interphalangeal joint of the thumb on attempts to pinch the thumb to the index finger. Confirmation of this diagnosis by electrodiagnostic studies is necessary in the evaluation of these patients. Electromyograms of the involved muscles should show denervation of these muscles.

Surgical procedures for the anterior interosseous syndrome begin, as for the pronator syndrome, with exploration of the median nerve from just proximal to the elbow joint to the anterior interosseous nerve. Reflection of the superficial head of the pronator teres, and even occasionally division of its deep head at its insertion on the radius, will provide adequate exposure of the distal portion of the median nerve at the branching of the anterior interosseous nerve. Reflection of the origin of the flexor digitorum superficialis muscle, as described by Henry in the subcutaneous transfer of the median nerve, may also be necessary. In these cases separation of the origin of the superficialis from its radial border, with reflection ulnarward, will allow visualization of the entire median nerve lying beneath the superficialis arch and within the fascia of the deep surface of the superficialis muscle (Fig. 3). Through this technique the entire median nerve can be explored and even transposed subcutaneously, placing the superficialis muscle beneath the median nerve but, of course, superficial to the anterior interosseous nerve. The usual findings on exploration of the anterior interosseous nerve syndrome are fascial bands on either the deep head of the pronator teres or the tendinous origin of the flexor superficialis, as the anterior interosseous nerve separates from the median nerve and comes to lie with a branch of the ulnar artery in the deep volar compartment of the forearm. A variety of aberrant muscles, muscle tendon units (such as the accessory head of the flexor pollicis longus or Gantser's muscle), as well as the so-called palmaris profundus or flexor carpi radialis brevis have all been identified as creating a compression neuropathy of the anterior interosseous nerve.[1, 13–16]

Carpal tunnel syndrome. The original description of carpal tunnel syndrome, the most common compression neuropathy in the upper extremity, was probably given to us by Sir James Paget in 1863. Marie and Foix[17] described the pathologic changes in the median nerve in 1913. Moersch named the syndrome in 1938; Cannon and Love[18] described the first series of patients in 1946; but Brain et al.,[19] in 1947, described six patients treated successfully by surgical release of the transverse carpal ligament. A variety of symptoms and a multitude of diseases have been associated or have presented with carpal tunnel syndrome.[20–27]

A typical case is associated with weakness or clumsiness in the use of the hands, hypesthesia or paresthesia in the distribution of the median nerve, aggravation of these symptoms with use of the hands, and awakening from sleep with numbness of the fingers or pain in the wrist or distal forearm. The proximal migration of pain from the area of the wrist toward

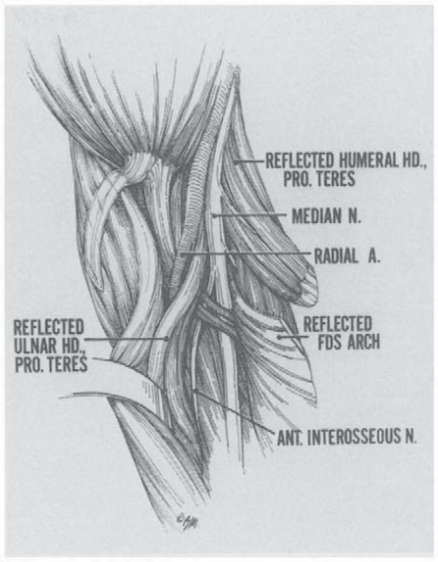

Fig. 3. Having completely reflected the flexor superficialis in the anterior interosseous syndrome, not only the deep median nerve but the anterior interosseous nerve are easily visualized. (Reprinted with permission of Elizabeth Roselius, M.S., A.M.I., from Green DP, editor: Operative hand surgery. New York/London, 1982, Churchill Livingstone.)

the forearm or elbow is relatively common. The syndrome is seen more frequently in female rather than male subjects, and at least 50% of cases occur in the age group of 40 to 60 years. Thenar atrophy, sensitivity, and distal lancinating paresthesias with percussion of the median nerve at the wrist, as well as reproduction of symptoms with the wrist flexion test described by Phalen,[28] are typical of the carpal tunnel syndrome. Relief of symptoms by splinting the wrist in a neutral position, taking care that the splint does not lie over the median nerve on the volar surface of the wrist and forearm, is typical of the syndrome. Decreased sensibility in the distribution of the median nerve and thenar atrophy are advanced signs of this entrapment neuropathy. Motor or sensory conduction latencies across the carpal tunnel are commonly used to confirm the diagnosis of this syndrome, but the electromyogram is most useful to differentiate this syndrome from the thoracic outlet syndromes or the compression syndromes of the lower cervical roots.[29–31] Although many anatomic variations of muscles and tendons have been reported as factors in carpal tunnel syndrome, there has always been some question whether such aberrant muscles as the palmaris profundus variations in lumbrical muscles[32] and flexor superficialis[33] or palmaris longus[34] can be factors in this syndrome. There is little doubt, however, that the median artery as it becomes enlarged or thrombosed can be a factor in neuritic symptoms in the median nerve.[35] Carpal tunnel syndrome has been reported in children, presenting as an atrophy of the index finger.[36]

Except in those cases where the symptoms can be predicted to be limited, such as in pregnancy, the surgical indications might be summarized as a failure to respond to conservative therapy, such as splinting the wrist in a neutral position. A program of splinting of the patient's wrist for 3 to 4 weeks, followed by weaning from the splint over an additional 4 weeks without recurrence of symptoms, has been effective

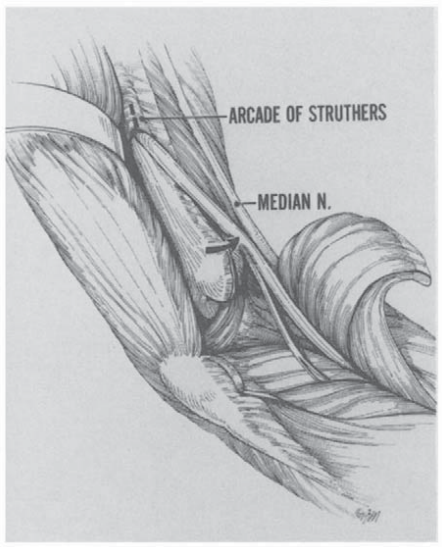

Fig. 4. At the time of submuscular transposition of the ulnar nerve, care must be taken to release the arcade of Struthers, excise the medial intermuscular septum, and carefully replace the flexor muscle mass on the medial epicondyle without creating a new compression of the ulnar nerve. (Reprinted with permission of Elizabeth Roselius, M.S., A.M.I., from Green DP, editor: Operative hand surgery. New York/London, 1982, Churchill Livingstone.)

conservative therapy in my patients. Recurrence of symptoms after complete removal of splints has been an indication for operative intervention.

Because of the numerous variations of the motor branch of the median nerve reported by a multitude of authors, and more recently classified by Lanz,[37] for carpal tunnel syndrome I have preferred a rather extensive exploration of the median nerve at the time of release of the transverse carpal ligament. It has seemed prudent to design an incision for this operation that, on one hand, could protect the variations of the motor branch of the median nerve and yet, at the same time, adequately divide the transverse carpal ligament. Taleisnik[38] has pointed out the critical position of the palmar cutaneous branch of the median nerve and its relationship to incisions for release of the transverse carpal ligament. Accordingly, I have generally used an incision that begins distally at the distal border of the transverse carpal ligament, follows the longitudinal crease of the palm, and crosses the base of the palm in a zigzag fashion ulnar to the long axis of the ring finger. The incision is continued above the proximal wrist crease for approximately 3 cm in order to perform a superficial fasciotomy of the forearm. By remaining on the ulnar side of the median nerve and using a hemostat to protect the deep structures, one can identify, explore, and divide the transverse carpal ligament, taking care throughout to protect the normal and anomalous branches of the median nerve. After completion of the release of the transverse carpal ligament, the motor branch of the median nerve should be identified and protected and, if necessary, the ulnar nerve in Guyon's canal explored by release of the canal.

Internal neurolysis, sometimes called endoneurolysis, has been described as an adjunct to the treatment of carpal tunnel syndrome.[39] Internal neurolysis should not be confused with epineurotomy. The incision of the epineurium, compared by some authors to a "fasciotomy" of a peripheral nerve, is a much simpler procedure and can be performed with appropriate care at any level of a peripheral nerve. Conversely, internal neurolysis must be reserved for those areas of the peripheral nerve with minimal interfascicular plexuses and to those patients who exhibit the specific indications for internal neurolysis. The indications for internal neurolysis[39] include atrophy of the thenar muscles, constant loss of sensibility in the distribution of the median nerve, deterioration of light touch or two-point discrimination sensibility, severe causalgia confined to the distribution of the median nerve, and true neuroma in continuity of the nerve at the time of exploration for carpal tunnel syndrome. The procedure of internal neurolysis must be performed with adequate magnification (usually greater than 3½×) to visualize the interfascicular plexus and should be confined to such an area of the peripheral nerve that only those fascicles contributing to the indication for internal neurolysis are relieved by microsurgical technique. If, for instance, the indication for internal neurolysis is atrophy of the thenar muscles, the surgeon should begin the procedure by isolating the motor branch of the median nerve distally and tracing those fascicles that contribute to the motor branch through the carpal tunnel. Only those fascicles that contribute to the motor branch would then undergo internal neurolysis. Care should be taken throughout the procedure to preserve the interfascicular plexus of nerves and the blood supply, particularly the subepineural plexus of arteries and veins within the epineurium.

Palmar cutaneous nerve. The palmar cutaneous branch of the median nerve, which arises from the radial side of the median nerve usually in the distal third of the forearm and passes distally, paralleling the course of the median nerve, may be compressed as it pierces the antebrachial fascia or the volar carpal ligament at or near the proximal wrist crease prior to dividing into the medial and lateral branches at the base of the palm. Compression of this nerve, manifested most often by paresthesias and dysesthesias over the thenar eminence, has been described by Stellbrink[40] and may require surgical decompression.

Entrapment neuropathy of the ulnar nerve

Entrapment neuropathies of the ulnar nerve may occur at two areas in the upper extremity. They are usually easily differentiated by physical findings and clinical presentation unless, of course, symptoms are minimal and the physical examination or electrodiagnostic studies inconclusive.

Ulnar nerve entrapment at the elbow. Pain of either a lancinating or aching quality at the medial side of the proximal forearm, which may radiate proximally or distally and may be accompanied by paresthesias, dysesthesias, or anesthesia in the ulnar one and a half fingers, is the hallmark of proximal ulnar nerve entrapment, the so-called cubital tunnel syndrome. Muscle wasting of the ulnarly innervated intrinsic muscles of the hand is not uncommon. Important localizing physical findings on examination are positive percussion test over the ulnar nerve at the elbow, abnormal mobility of the ulnar nerve over the medial epicondyle of the humerus,[41] or a positive elbow flexion test that increases the numbness or paresthesia in the ulnar one and a half fingers. Electrodiagnostic studies of conduction velocities across the elbow[42] that are less than 25% reduced from the velocities above or below the elbow are insignificant. Nerve conduction velocities across the elbow that are reduced by more than 33%

from those above or below the elbow are always significant and suggestive of a cubital tunnel syndrome. As with other entrapment neuropathies, use of a conduction velocity as the sole indication for surgery is not wise.

Entrapment neuropathy of the ulnar nerve of the elbow has been associated with compression by the arcade of Struthers,[1, 9] the anconeus epitrochlearis[1]; the medial head of the triceps; the aponeurosis of the flexor carpi ulnaris[43]; osteophytes, ganglia, or lipomas associated with elbow joint[44]; and subluxation of the ulnar nerve across the medial epicondyle of the humerus.[41]

The surgical treatment of the cubital tunnel syndrome may include decompression of the cubital tunnel from the medial epicondyle of the humerus distally by division of the aponeurosis of the flexor carpi ulnaris[43] or by anterior transposition of the ulnar nerve[45, 46] or even by medial epicondylectomy of the humerus,[47, 48] which in recent years is regaining popularity after losing same for more than 20 years. After the decompression procedure it is important to be sure that a subluxation of the ulnar nerve has not been created, since this could further injure the nerve.

The operation of anterior transposition (Fig. 4) is an extremely demanding one since during this procedure the surgeon must be careful not to create a second site of compression or entrapment of the ulnar nerve at either the arcade of Struthers, some 8 cm proximal to the medial epicondyle, or at the medial intermuscular septum, which should be excised during the procedure, or finally if a submuscular transposition is performed, beneath the flexor muscle mass by the conjoined tendons of the flexor carpi ulnaris, flexor carpi radialis, and palmaris longus as they arise from the medial epicondyle. Resuturing the flexor muscles to the medial epicondyle after submuscular transposition can create a new site of compression on the transposed ulnar nerve. Before undertaking either subcutaneous or submuscular transposition, the surgeon should consult a standard text on this procedure[49] in addition to reviewing Learmont's[46] classic article on anterior transposition of the ulnar nerve.

Ulnar nerve entrapment in Guyon's canal. Entrapment neuropathy of the ulnar nerve at the wrist (the ulnar tunnel syndrome) presents with a variety of signs and symptoms[50, 51] depending on whether the ulnar nerve itself or one of its major branches has been compressed. Compression of the nerve by a deep carpal ganglion may produce bizarre patterns of motor neuropathy affecting only part of the ulnar innervated intrinsic muscles.[50] Occupational neuritis secondary to repeated blunt trauma of the hypothenar eminence of the palm should be sought in the history since discontinuance of the blunt trauma will likely lead to resolution of the neuropathic symptoms.[52] Preoperative examination of the patient with ulnar entrapment neuropathy at the wrist should always include an Allen test for confirmation of ulnar collateral circulation to both the superficial and deep palmar arches. Thrombosis or aneurysm of the ulnar artery or one of its branches can cause ulnar neuropathy, which may be predicted if the Allen test reveals slowed circulation from that artery.

As in the carpal tunnel syndrome, ulnar neuropathy at the wrist is associated with occupational causes predisposing the nerve to repeated blunt trauma,[53–55] occult tumorous conditions,[50, 52, 54] fractures of the hamate or triangular bone,[56] or even fractures of the bases of the ring and little metacarpals.[55] Compression of the ulnar nerve in Guyon's canal has been associated with traversal of the canal by accessory or aberrant muscles, usually arising from the deep fascia of the forearm, which blend with the fibers of the abductor digiti quinti.[51] Anatomic variations in the arrangement of the branches of the ulnar nerve have been described but have not been consistently associated with motor or sensory signs.[57] Although recent work has again called our attention to the presence of the palmar cutaneous branch of the ulnar nerve,[58] injury to this nerve is likely to be avoided if the incision to decompress Guyon's canal is not carried directly over the hypothenar eminence.

The presence of a neuropathy of the ulnar nerve with evidence of localization at the level of Guyon's canal, particularly if the neuropathy is progressive, is sufficient indication for a careful surgical exploration of the ulnar nerve through Guyon's canal. If the neuropathy is related to an occupationally induced trauma, conservative therapy to relieve the trauma is indicated prior to exploration.

At the time of surgical exploration the surgeon should begin in the distal third of the forearm to divide the fascia of the distal forearm on the radial side of the flexor carpi ulnaris tendon. On exploration through Guyon's canal and into the palm, one should observe both the superficial and deep branches of the ulnar nerve as they course as far distally as the distal palmar crease or the hiatus of the adductor pollicis.

Compression neuropathies of the radial nerve

As the radial nerve passes from the posterior to the anterior compartment at the distal third of the humerus, fixation of the nerve by the lateral intermuscular septum can cause a compression neuropathy.[59, 60] Recorded cases of compression neuropathy relieved surgically at the lateral intermuscular septum[60] are sparse except in those instances where major trauma to the humerus, particularly fracture,[61] causes displacement of the humerus and resultant neuropathy of the radial nerve.

The radial tunnel syndrome,[62, 63] a compression neuropathy of the radial nerve between the radial head and the supinator muscle, is the most common entrapment neuropathy of the radial nerve. Since the radial tunnel syndrome has been associated with four anatomic lesions, which may have differing physical findings, these will be considered separately. The first lesion of the radial tunnel syndrome is a series of fibrous bands lying anterior to the radial head at the entrance of the radial tunnel. These may be demonstrated as a cause of the radial tunnel syndrome when the symptoms of radial compression are aggravated by full flexion of the elbow with the forearm in supination and the wrist in neutral position. The fan-shaped leash of vessels across the radial nerve, which supply the brachioradialis and extensor carpi radialis longus muscles, may compromise a second anatomic lesion, contributing to entrapment of the radial nerve. There are apparently no specific physical findings for these vessels. The third anatomic lesion in the radial tunnel consists of a tendinous margin of the extensor carpi radialis brevis. The fourth lesion is the arcade of Frohse, which forms a ligamentous band over the deep radial nerve as the nerve enters the supinator muscle. Both the tendinous margin of the extensor carpi radialis brevis and the arcade of Frohse will increase symptoms of radial tunnel syndrome when the forearm is held in full pronation and full flexion of the wrist, since in this position each of these fibrous bands will tighten across the

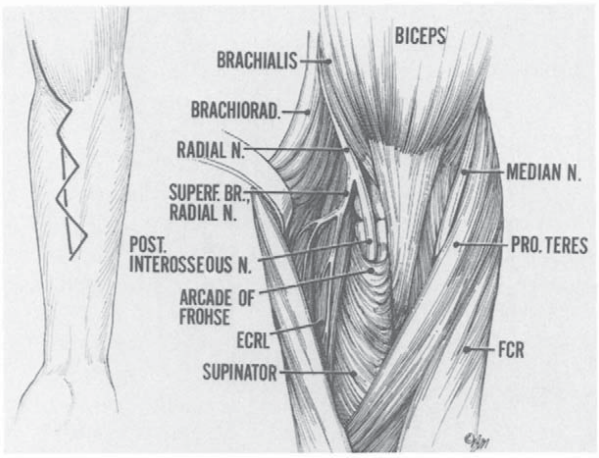

Fig. 5. The exploration of the radial tunnel syndrome begins at the level of the radial head and should extend through the superficial head of the supinator muscle. (Reprinted with permission of Elizabeth Roselius, M.S., A.M.I., from Green DP, editor: Operative hand surgery. New York/London, 1982, Churchill Livingstone.)

radial nerve. Electrodiagnostic studies, both nerve conduction velocities and electromyograms, are helpful in the diagnosis of radial tunnel syndrome. I have also used diagnostic blocks with small amounts of lidocaine administered at various points along the radial nerve in an attempt to confirm the diagnosis of radial tunnel syndrome.

Except in those patients where the entrapment neuropathy of the radial nerve can be dependably localized to the area of the arcade of Frohse, which in my experience is rare, a generalized approach to the radial tunnel, in which the dissection is carried from the anterior surface just above the elbow joint, tracing the radial nerve distally into and through the radial tunnel with care taken to divide all of the superficial fibers of the supinator muscle and protect the radial nerve in the process, will be necessary to effectively and dependably relieve the patient of radial tunnel compression (Fig. 5). Only by positioning the forearm in marked pronation with wrist flexion will the compression of the extensor carpi radialis brevis be visualized across the radial nerve. Since the operation is performed with the forearm supinated, the surgeon must position the arm in the desired position of pronation with wrist flexion to observe this portion of the compression neuropathy. I would emphasize once again that the superficial fibers of the supinator muscle should be completely divided from the arcade of Frohse to the distal extent of the supinator muscle where the deep fibers of the radial nerve arborize and divide into the several extensor muscles. Only with this complete dissection will the fibers of the deep branch of the radial nerve be completely decompressed.

Radial nerve compression of the wrist. An isolated neuritis of the superficial radial nerve (cheiralgia paresthetica) was described by Wartenberg[64] in 1932. In five patients the syndrome was characterized by persistent pain on the radial dorsal surface of the distal third of the forearm radiating to the dorsum of the hand, thumb, index finger, and middle finger. Ordinarily percussion sensitivity over the radial nerve as it arises from beneath the edge of the brachioradialis muscle is a cardinal physical finding of this syndrome. As frequently as not, this syndrome seems to be associated with the wearing of elastic or expansion bands on jewelry and watches at the wrist. Removal of these items generally relieves the syndrome; in those few cases where conservative therapy fails, exploration of the superficial branch of the radial nerve will often reveal a fibrous band crossing the nerve at or near its exit from beneath the brachioradialis muscle. The division of this fibrous band and/or a portion of the brachioradialis muscle is usually sufficient to relieve this syndrome.

Thoracic outlet syndrome

The thoracic outlet syndrome may present with a variety of vague, ill-defined, and inconsistent symptoms including pain in the shoulder or supraclavicular fossa that radiates down the arm in a radicular pattern and often involves the medial border of the brachium and antebrachium. The symptoms may be provoked or increased by a particular activity such as working overhead, reaching down and behind repetitively, lying in particular positions, especially with the hands over the head or behind the head, or lying on one's side, allowing the symptomatic shoulder to fall away on the upward side.

The thoracic outlet syndrome represents a vascular and/or neurologic symptom complex related to the subclavian artery and/or the lower trunk of the brachial plexus as they are compressed by the scalene muscle groups, clavicle, and/or first rib. Occasionally the middle trunk of the brachial plexus, thereby involving the seventh cervical root, may be involved in this syndrome. When the subclavian artery or subclavian vein is compressed or narrowed, a relative ischemia of the upper extremity, which may be most marked during provocative muscle tests, may occur. The neurologic component of the syndrome involving a compression neuropathy of the lower trunk of the brachial plexus as it arises from the eighth cervical and first thoracic roots may be complicated by a variety of anatomic variations and fascial reflections to further aggravate the compression neuropathy. Misalignment of the shoulder, such as sagging with increasing age, fatigue, or ill health, or even poor posture, may narrow the costoclavicular space into which the neurovascular structures pass after crossing of the scalene cleft,[65] with the result being compression in the thoracic outlet. This space may also be narrowed by abnormal cervical ribs,[66] fracture calluses of the first rib or clavicle,[67] and abnormal positioning of the pectoral girdle such as with the brace syndrome[68] and will be aggravated by depression of the shoulder. The symptoms of thoracic outlet usually are increased after a long or tiring day, can be worse at night, and can be increased by sleeping on one's side, particularly if the upper shoulder is the symptomatic one. If when the patient sleeps on his side, he sleeps on the symptomatic shoulder, the compression of this shoulder generally creates a widening of the costoclavicular space and scalene cleft, thereby reducing symptoms.

One interesting facet of this syndrome is that the vasomotor changes usually affect the radial side of the hand, specifically the thumb and index finger, rather than the ulnar side of the hand and palm. These changes are often associated with a bruit or thrill in the superclavicular fossa, usually more marked with physical activity that is either at eye level or above the head. The neurologic symptoms are ordinarily located on the ulnar border of the hand in the distribution of the C8 and T1 cervical roots. Electrodiagnostic studies,[69] particularly nerve conduction velocities, are often of little help. In advanced cases electromyography that specifically examines

muscles on the C8 and T1 roots can be extremely helpful in locating this lesion. The recent use of somatosensory evoked potentials[70] seems to have considerable promise as an adjunct to the electrodiagnosis of thoracic outlet syndrome and should be considered in all patients with a possibility of this diagnosis.

Although resection of the first rib, usually by an axillary approach, is the operation most frequently performed after this syndrome has been diagnosed, it is wise to remember that scalenotomies, rib resections by other approaches, and claviectomies have all had their periods of popularity in the treatment of this disease. Some authors documented a 50% complication rate with the transaxillary resection of the first rib.[71] It seems logical that in the next few years we can expect a redefinition of the surgical approach to this syndrome. As always the value of a surgical procedure is only equal to the diagnostic procedures that localize the compression neuropathy, and the skill of the surgeon will be weighted by the preoperative evaluation as well as the intraoperative treatment in most cases. Ordinarily the surgery is performed by thoracic-cardiovascular surgeons and their consultation should be sought.

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Surgery of the spastic hand in cerebral palsy: Report of the Committee on Spastic Hand Evaluation

Eduardo A. Zancolli, M.D.,* *Chairman,* Leonard J. Goldner, M.D.,** *and* Alfred B. Swanson, M.D.***

Reprint requests: Eduardo A. Zancolli, M.D., Avenida Alvear 1535, Buenos Aires, 1014 Argentina.

*Professor of Orthopedics, University of Buenos Aires; Chief of Surgery of the National Rehabilitation Center, Buenos Aires.

**Professor and Chairman of Orthopaedic Surgery, Duke University, Durham, N.C.

***Director of Orthopaedic and Hand Surgery Training Program, Blodgett and Butterworth Hospitals, Grand Rapids, Mich.; Professor of Surgery, Michigan State University, Lansing, Mich.

Most patients with cerebral palsy involvement of the upper extremity are not candidates for surgical reconstructive procedures. However, selected deformities related to muscle imbalance secondary to spasticity may be corrected by appropriate surgical procedures. Although surgery cannot make a functionally poor limb perfect, it can greatly improve the initial condition. A reasonable degree of success can be obtained if the patients are selected carefully through precise and complete examination, testing, and clinical evaluation of impairment of daily life activities.[1, 2] Most poor surgical re-