Upper Extremity Focal Neuropathies

Dianna Quan, MD
V. Vedanarrayan, MD
A. Arturo Leis, MD
Tanya Oswald, MD
Please be aware that some of the medical devices or pharmaceuticals discussed in this handout may not be cleared by the FDA or cleared by the FDA for the specific use described by the authors and are “off-label” (i.e. use not described on the product’s label). “Off-label” devices or pharmaceuticals may be used if, in the judgment of the treating physician, such use is medically indicated to treat a patient’s condition. Information regarding the FDA clearance status of a particular device or pharmaceutical may be obtained by reading the product’s package labeling, by contacting a sales representative or legal counsel of the manufacturer of the device or pharmaceutical, or by contacting the FDA at 1-800-638-2041.
Upper Extremity Focal Neuropathies

Table of Contents

Program Committee & Course Objectives 4
Faculty 5
Median Nerve Entrapment Syndromes  Dianna Quan, MD 7
Radial Nerve Focal Neuropathies  V. Vedanarayanan, MD 11
Ulnar Nerve Focal Neuropathies  A. Arturo Leis, MD 15
Surgical Treatment of Upper Limb Neuropathies  Tanya Oswald, MD 19
CME Questions 23

No one involved in the planning of this CME activity had any relevant financial relationships to disclose.
Authors/faculty have nothing to disclose

Chair: A. Arturo Leis, MD

The ideas and opinions expressed in this publication are solely those of the specific authors and do not necessarily represent those of the AANEM.
Objectives

Objectives - Participants will acquire skills to (1) Explain the anatomy of the major nerves in the upper limb, (2) perform motor and sensory NCS in median, ulnar, and radial nerves, (3) discuss the common focal neuropathies and differential diagnoses affecting upper limb nerves, including CTS, ulnar neuropathy at the elbow, and Saturday night palsy, (4) understand the EDX strategy in focal neuropathies to arrive at a proper diagnosis, and (5) recognize the indications and types of surgery for focal neuropathies of the upper extremity.

Target Audience:
- Neurologists, physical medicine and rehabilitation and other physicians interested in neuromuscular and electrodiagnostic medicine
- Health care professionals involved in the diagnosis and management of patients with neuromuscular diseases
- Researchers who are actively involved in the neuromuscular and/or electrodiagnostic research

Accreditation Statement - The AANEM is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education (CME) for physicians.

CME Credit - The AANEM designates this live activity for a maximum of put in 3.25 AMA PRA Category 1 Credits™. If purchased, the AANEM designates this enduring material for a maximum of 5.75 AMA PRA Category 1 Credits™. This educational event is approved as an Accredited Group Learning Activity under Section 1 of the Framework of Continuing Professional Development (CPD) options for the Maintenance of Certification Program of the Royal College of Physicians and Surgeons of Canada. Physicians should claim on the credit commensurate with the extent of their participation in the activity. CME for this course is available 10/2013 – 10/2016.

CEUs Credit - The AANEM has designated this live activity for a maximum of 3.25 AANEM CEU’s. If purchased, the AANEM designates this enduring material for a maximum of 5.75 CEU’s.

2012-2013 Program Committee

Vincent Tranchitella, MD, Chair
York, PA

Robert W. Irwin, MD
Miami, FL

David B. Shuster, MD
Dayton, OH

Thomas Bohr, MD, FRCPC
Loma Linda, CA

Shawn Jorgensen, MD
Queensbury, NY

Zachary Simmons, MD
Hershey, PA

Jasvinder P. Chawla, MBBS, MD, MBA
Atlanta, GA

A. Atruro Leis, MD
Jackson, MS

Jeffrey A. Strommen, MD
Rochester, MN

Maxim Moradian, MD
New Orleans, LA

T. Darrell Thomas, MD
Knoxville, TN

2012-2013 AANEM President

Peter A. Grant, MD
Medford, OR
Upper Extremity Focal Neuropathies

Faculty

Dianna Quan, MD  
Professor of Neurology, Department of Neurology  
Program Director, Neuromuscular Medicine Fellowship  
Director, Electromyography Laboratory  
University of Colorado Denver School of Medicine  
Aurora, CO

Dr. Quan received her undergraduate degree from the University of Chicago and her medical degree from Columbia University College of Physicians and Surgeons. She completed her neurology residency and fellowship training in neuromuscular disorders and electrodiagnosis at the Hospital of the University of Pennsylvania. She is currently professor of neurology at the University of Colorado Denver School of Medicine located in Aurora, where she is program director of the neuromuscular medicine fellowship and director of the electromyography (EMG) laboratory. She has been an active member of the American Academy of Neurology and the American Association of Neuromuscular & Electrodiagnostic Medicine, and serves as a medical editor for the online e-Medicine Textbook of Neurology and a frequent ad hoc reviewer for Muscle & Nerve, among other journals. Her interests include peripheral neuropathy, amyotrophic lateral sclerosis, and postherpetic neuralgia. Dr. Quan is an American Board of Electrodiagnostic Medicine Diplomate.

A. Arturo Leis, MD  
Professor of Neurology, University of Mississippi Medical Center  
Electrodiagnostic Consultant, Methodist Rehabilitation Center  
Jackson, MS

Dr. Leis received his medical degree from the University of Arizona, and completed his residency at the University of Texas Health Science Center, and a fellowship at the University of Iowa Hospitals and Clinics. He is a clinical professor of neurology at the University of Mississippi Medical Center and an electrodiagnostic consultant at the Methodist Rehabilitation Center. He has published more than 100 papers, letters, and chapters in peer-reviewed journals and is the author of Atlas of Electromyography, © 2000, and Atlas of Nerve Conduction Studies and Electromyography, © 2012 by Oxford University Press. Dr. Leis’ major research focus is neuromuscular disorders, including West Nile virus infection and silent period studies.

Veda V. Vedanarayanan, MD, FRCPC  
Professor of Neurology, Pediatrics and Pathology  
Director, Division of Neuromuscular Medicine  
University of Mississippi Medical Center  
Jackson, MS

Dr. Vedanarayanan received his medical degree from the University of Madras, and completed residency training in neurology and child neurology from Duke University. He also trained in neuromuscular medicine at Johns Hopkins Medical Center. Dr. Vedanarayanan is a professor of neurology, pediatrics and pathology, and is the director of the Division of Neuromuscular Medicine at the University of Mississippi Medical Center. He is board certified in neurology, electrodiagnostic medicine, clinical neurophysiology, autonomic medicine and neuromuscular medicine.

Tanya Oswald, MD  
Plastic and Hand Surgeon, Joseph M. Still Burn and Reconstruction Center  
Central Mississippi Medical Center  
Jackson, MS

Dr. Oswald received her medical degree from the University of Mississippi Medical Center, where she completed her residency in general surgery and residency in plastic and reconstructive surgery. She also did fellowships in hand, peripheral nerve, and microsurgery medicine at Washington University in St. Louis. Dr. Oswald is currently a plastic and hand surgeon at the Joseph M. Still Burn and Reconstruction Center at the Central Mississippi Medical Center in Jackson, MS. She also serves as an editorial board member for Annals of Plastic Surgery. She is a member of the American College of Surgeons and the American Society for Plastic Surgeons, and has published 35 articles.
INTRODUCTION

Median nerve entrapment is a common problem encountered in neuromuscular and electrodiagnostic (EDX) clinical practice. Most commonly, entrapment occurs at the wrist, resulting in carpal tunnel syndrome (CTS). Proximal injury is less common but well described and may occur as pronator syndrome (PS), anterior interosseous nerve syndrome (AINS), or supracondylar process syndrome due to injury at the ligament of Struthers (also known as ligament of Struthers syndrome). This discussion will cover the anatomy of the median nerve, the clinical manifestations of these four disorders, and the expected EDX findings.

ANATOMY

The median nerve contains fibers originating from the C5 to T1 nerve roots and all three trunks of the brachial plexus. Fibers from the medial and lateral cords join to form the median nerve, which runs along the medial aspect of the arm, passing under the ligament of Struthers (if present), under the lacertus fibrosis or bicipital aponeurosis, between the two heads of the pronator teres muscle, and under the fibrous arch of the flexor digitorum superficialis (FDS) muscle, also known as the sublimis arch. Distal to this, the anterior interosseus nerve (AIN) arises from the radial aspect of the median nerve in most people and innervates the flexor pollicis longus (FPL), the flexor digitorum profundus (FDP) in the radial aspect of the hand, and the pronator quadratus muscles. The median nerve proper continues into the distal forearm, giving off the palmar cutaneous sensory branch to the base of the thenar eminence before traversing an enclosed tunnel at the wrist formed by fibro-osseous elements and covered by the transverse carpal ligament. The nerve is particularly susceptible to injury at several locations along its course. From proximal to distal, these include the ligament of Struthers, the proximal medial forearm around the pronator teres muscle, around the origin of the AIN, and in the wrist at the carpal tunnel.

CARPAL TUNNEL SYNDROME

CTS is the most common nerve entrapment encountered in clinical and EDX practice. Because the tunnel is an enclosed space, any process that reduces the volume of free space within may put pressure on the median nerve. Complaints typically involve a combination of pain, numbness, and paresthesias affecting the thumb, index finger, middle finger, and radial aspect of the fourth finger. While strength in the ulnar aspect of the hand normally is unaffected, patients may complain nonspecifically of weakness or clumsiness of the entire hand. Wrist flexion and extension may increase the pressure within the carpal tunnel causing more noticeable symptoms during activities such as driving, holding books or papers when reading, gripping and manipulating small electronic devices, or other similar activities. Nocturnal awakening due to pain and paresthesias is common.

Examination may reveal reduced sensation in the thumb, index, middle, and lateral aspect of the fourth fingers. Thumb opposition and abduction may be weak, and, in more severe cases, thenar atrophy may be noted. Provocative maneuvers can be used to attempt to recreate symptoms in the office, though their sensitivity and specificity vary widely in the published literature. Phalen’s sign is present when complete palmar flexion of the wrist produces paresthesias and numbness. Tinel’s sign is present when percussion along the course of the nerve at the flexor retinaculum results in median distribution paresthesias.
EDX testing in milder cases may simply demonstrate slowing of sensory conduction velocity (CV) using a standard recording technique between the proximal wrist and median-innervated fingers. Milder cases may only demonstrate CV slowing on measurement across a short segment between the palm and wrist, thereby eliminating the contribution of more normal nerve in the distal hand and fingers. With more severe involvement, larger motor fibers may also show focal slowing of CV across the wrist, visible as a prolonged distal motor latency of the compound motor action potential. With even more severe pressure the median-innervated hand muscles may have evidence of axonal damage with acute or chronic denervation on needle electromyographic (EMG) examination.

**ANTEOR INTEROSSEUS NERVE SYNDROME**

In the much less common AINS, the exact site of entrapment is probably variable. The AIN separates from the median nerve about 5-8 cm distal to the lateral epicondyle and may originate in association with arches formed by the FDS, deep head of the pronator teres, superficial head of the pronator teres, or a combination of fibrous arches from these muscles. Gantzler's muscle, an accessory component of the FPL muscle may also play a role in AIN compression. Other possible sites of injury according to Spinner include the thrombotic ulnar collateral vessels, the aberrant radial artery, the tendinous origin of palmaris longus or flexor carpi radialis brevis, and an enlarged bicipital bursa. In some cases, the problem is due not to structural impingement but rather spontaneous inflammation or ischemia.

Symptoms and findings are predominantly motor. Patients complain of weakness of the thumb and index finger with difficulty pinching items between these two fingers due to weakness of the FPL and radial portion of the FDP muscles. This can manifest as problems using eating utensils, gripping a pen, or manipulating small items with the thumb and index finger. Pain may be noted, but sensory loss should not be observed.

The characteristic findings on clinical examination are related to dysfunction of these muscles. This is most obvious during attempted pinching, which reveals an extended distal joint of the index finger and thumb and exaggerated flexion of the proximal interphalangeal joint of the index finger and metacarpophalangeal joint of the thumb. Pronator weakness is more subtle and must be tested in elbow flexion to reduce the action of the pronator teres and highlight weakness of the pronator quadrates muscle. EDX testing demonstrates denervation in the three muscles supplied by the nerve.

**PRONATOR SYNDROME**

Pronator syndrome may be a somewhat misleading name for the pain and median nerve distribution paresthesias experienced by patients with entrapment in the proximal medial forearm, since not all cases involve the pronator teres muscle. As the median nerve passes through the antebrachial fossa, potential compressive injury may occur not only between the two heads of the pronator teres, but also under the edge of the flexor digitorum sublimis arch, or occasionally under the lacertus fibrosis of the biceps tendon. Pain may be exacerbated by simultaneous forearm pronation and extension of the elbow. The distribution of paresthesias and numbness in the median distribution in the hand may suggest symptoms of the more common CTS, but tenderness or a Tinel’s sign are expected at the region of the pronator teres rather than at the wrist and decreased sensation may be identified along the thenar eminence, normally spared in CTS. Activation of the FDS of the middle finger against resistance may cause pain if the lesion is at the FDS arch. In the case of lacertus fibrosis compression, elbow flexion of the supinated forearm may reproduce or exacerbate symptoms.

Normal median CV at the wrist further distinguishes the problem from CTS. Focal slowing more proximally in the forearm may be noted, though more detailed examination with inching studies may be needed to identify the specific area of slowing. If axonal damage has occurred, needle EMG demonstrates denervation in median forearm and hand muscles but relative sparing of the pronator teres, depending on the exact site of compression.

**SUPRACONDYULAR PROCESS SYNDROME**

Entrapment at the ligament of Struthers (a ligament which connects the medial epicondyle to an anomalous bony spur in the distal humerus) is rare among median nerve syndromes. This ligament is present in less than 3% of individuals and even when present typically causes symptoms mainly in the setting of superimposed trauma. Extension of the elbow, wrist, or fingers or forearm supination with the elbow extended may cause or exacerbate pain. Examination may demonstrate a palpable bony anomaly and weakness in the median nerve distribution distal to the entrapment, including the pronator teres. Slowing of median CV across the site of entrapment may be observed with associated denervation of distal median-innervated muscles in more severe or longstanding cases.

**SUMMARY**

Median entrapment syndromes in the upper extremity are common, especially CTS, though more proximal injury is well described particularly around the origin of the AIN, proximal medial forearm around the pronator teres, and at the ligament of Struthers. The clinical history provides important clues regarding the lesion site, and physical examination findings including provocative maneuvers provide further localizing information. EDX testing is a key component of confirming the diagnosis and ensuring appropriate treatment.
REFERENCES

Radial Nerve Focal Neuropathies

Vettaikorumakankav Vedanarayanan, MD
Professor of Neurology, Pediatrics and Pathology
Director, Division of Neuromuscular Medicine
University of Mississippi Medical Center
Jackson, MS

INTRODUCTION

Radial nerve entrapment syndromes are common, particularly radial mononeuropathies at other sites also occur. The etiology, general comments, clinical features, and electrodiagnostic (EDX) strategy for these entrapment neuropathies will be reviewed.

ANATOMY

The radial nerve is the largest branch of the brachial plexus, arising from the posterior cord and the fifth, sixth, seventh, and eighth cervical roots, with occasional contribution from the first thoracic root. From its origin on the posterior axillary wall, it descends behind the axillary artery to reach the angle between the medial aspect of the arm and the posterior wall of the axilla (known as the brachio–axillary angle). Branches to the heads of the triceps and anconeus arise in the axilla and the brachio–axillary angle. The radial nerve inclines downward between the long and medial heads of the triceps, after which it passes obliquely along the back of the humerus in the spiral groove (also known as the radial groove). Here it is in direct contact with bone. The nerve then descends in the furrow between the brachialis medially and brachioradialis laterally. The nerve gives off branches to the brachialis (this muscle is supplied primarily by the musculocutaneous nerve) and brachioradialis. The nerve gives off branches to the brachialis (this muscle is supplied primarily by the musculocutaneous nerve) and brachioradialis. The nerve is overlapped, in turn, by the extensor carpi radialis (ECR) longus and ECR brevis. The radial nerve supplies both muscles, although the ECR brevis may receive its nerve supply from the posterior interosseous nerve (PIN). Anterior to the lateral epicondyle, the radial nerve divides into its two terminal branches: the PIN and the superficial radial nerve (SRN). The former is a pure muscular nerve that innervates the supinator before passing through the arcade formed by the superficial and deep heads of this muscle (i.e., arcade of Frohse). On the dorsum of the forearm, the PIN divides in a variable manner to innervate the extensor carpi ulnaris (ECU), extensor digitorum communis, extensor digiti minimi, abductor pollicis longus, extensor pollicis longus and brevis, and extensor indicis. The superficial radial branch is primarily a sensory nerve. It provides cutaneous innervation to the dorsum of the hand lateral to the ring finger, the dorsum of the thumb, the radial aspect of the thenar eminence, and the dorsum of the index, middle, and radial half of the ring fingers as far distally as the middle phalanx.

Although the radial nerve, or its branches, may be involved in penetrating injuries at any level, there are certain sites where the nerve is more prone to injury. In the brachio–axillary angle, compression may result from a misused crutch or from fractures of the upper third of the humerus. In the region of the spiral groove and lateral intermuscular septum, compression neuropathies of the radial nerve are common and widely recognized. The nerve is most commonly damaged by fractures of the humerus or during deep intoxication with the arm draped over the edge of a bed, chair, or bench (known as “Saturday night palsy”). In its course through the supinator (i.e., arcade of Frohse), the PIN is prone to damage from fractures of the upper third of the radius and less commonly from entrapment. Injury to the superficial radial branch may result from tight handcuffs (known as handcuff neuropathy) or carelessly administered intravenous infusions.
RADIAL NERVE LESION IN THE ARM

In the region of the spiral groove, the nerve lies on bone and is commonly injured by a fracture of the humerus. It is also commonly injured by external pressure applied to the region of the spiral groove (e.g., Saturday night palsy).

Etiology

Compression of the nerve in the spiral groove (i.e., radial groove) of the humerus can cause a radial nerve lesion in the arm. As described above, Saturday night palsy occurs when sedatives or alcohol produces deep sleep in someone and an arm is draped over the edge of a bed, chair, or bench. “Honeymooner’s palsy” occurs when compression results from the weight of a partner’s head lying on the arm. A radial nerve lesion can occur secondary to a fracture of the humerus, prolonged or excessive muscle contraction (e.g., experienced by masons, carpenters, or athletes in throwing sports), anatomical anomalies of the triceps, and deep intramuscular injections.

General Comments

A radial nerve lesion in the arm is a common compression neuropathy. Predisposing factors include diabetes, malnutrition, alcohol or sedative use, and hereditary susceptibility to pressure palsies.¹ The differential diagnosis includes lead poisoning, porphyria, diabetes, and periarthritis nodosa.

Clinical Features

Wrist drop occurs with a radial nerve lesion in the arm. There is normal function of the triceps (e.g., extension of forearm at elbow). There is weakness of the brachioradialis and all other muscles supplied by the radial nerve beyond the spiral groove. Pain is not a typical feature, but it may be perceived in the area of the lateral epicondyle, radial styloid, or dorsum of hand. Numbness can occur in the distribution of the SRN and occasionally in the territory of the posterior cutaneous nerve of the forearm. Most cases of Saturday night palsy fully resolve in days to a few weeks, which is consistent with a demyelinating lesion. Delayed or incomplete recovery implies a greater degree of axonal loss.

Electrodiagnostic Strategy

Nerve conduction studies (NCSs) are used to confirm a focal lesion (i.e., conduction block, slowing of conduction velocity) at the spiral groove and to obtain information on the severity and prognosis. Cases characterized by conduction block resolve quickly; those with axonal loss and Wallerian degeneration show a reduction of motor or sensory amplitudes and resolve slowly or incompletely. In cases of axonal loss, needle electromyography (EMG) may show neurogenic changes (i.e., spontaneous activity, abnormal motor unit potentials, and abnormal recruitment) in the brachioradialis and other muscles supplied by the radial nerve distal to the spiral groove. Needle EMG is normal in cases of demyelination and in the early stages of axonal loss in which Wallerian degeneration has not yet occurred. Needle EMG of the triceps and anconeus is normal. The superficial radial sensory response is reduced in amplitude or absent in cases of axonal loss (normal in cases of demyelination and in the early stages of axonal loss). Needle EMG of non-radial–innervated C7 muscles may be necessary to exclude C7 radiculopathy.

RADIAL NERVE LESION IN THE AXILLA

Etiology

Compression of the nerve in the axilla by high-riding crutches can cause the condition called “crutch palsy” or “crutch neuropathy.” Most cases of crutch neuropathy involve the posterior cord of the brachial plexus,⁴,⁵ although other cords and individual nerves may also be affected. A radial nerve lesion can occur secondary to shoulder trauma, humerus fractures, tumor, or anatomical anomalies of the coracobrachialis or triceps.

Clinical Features

Wrist drop occurs with a radial nerve lesion in the axilla. Weakness occurs in all muscles supplied by the radial nerve, including the triceps. The triceps reflex is absent. Numbness occurs in the distribution of the SRN and occasionally in the territories of the posterior cutaneous nerves of the forearm or arm and inferior lateral cutaneous nerve of the arm. Most cases fully resolve in days to a few weeks, which is consistent primarily with a demyelinating lesion. Delayed or incomplete recovery implies a greater degree of axonal loss.

Electrodiagnostic Strategy

An EDX evaluation can obtain information on the severity and prognosis. Cases associated with axonal loss and Wallerian degeneration show a reduction of motor or sensory amplitudes and resolve slowly or incompletely. In cases of axonal loss, needle EMG may show neurogenic changes in the triceps and all other muscles supplied by the radial nerve. Needle EMG is normal in cases of demyelination and in the early stages of axonal loss in which Wallerian degeneration has not yet occurred. The superficial radial sensory response is reduced in amplitude or absent in cases of axonal loss (normal in cases of demyelination and in the early stages of axonal loss). Needle EMG of non-radial–innervated C7 muscles may be necessary to exclude C7 radiculopathy.

POSTERIOR INTEROSSEOUS NERVE SYNDROME

Etiology

Compression of the nerve as it passes between the two layers of the supinator muscle in the arcade of Frohse can cause posterior interosseous nerve syndrome (PINS). PINS can occur secondary to radial subluxation, a fracture of the proximal radius, prolonged or repeated pronation–supination movements, tumors (lipoma), neuralgic amyotrophy, and compression by the ECR brevis.
General Comments

PINS is also known as the “supinator syndrome.” Injury to the PIN usually is associated with trauma (e.g., fractures, gunshot wounds, lacerations) rather than with true entrapment.

Clinical Features

There is normal supination of the forearm and radial wrist extension (i.e., the supinator and ECR are normal). Weakness occurs in the ECU; attempted wrist extension results in characteristic radial deviation of the wrist. Weakness occurs during finger extension and thumb extension. Thumb abduction may be normal because the abductor pollicis brevis (median nerve) is unaffected. There is pain in the lateral upper forearm (usually 5-8 cm distal to the lateral epicondyle). There is no sensory impairment because the SRN arises above the arcade of Frohse.

Electrodiagnostic Strategy

The superficial radial sensory response is normal. Motor NCSs may reveal a focal lesion (i.e., conduction block, slowing of conduction velocity) across the entrapment.2 Cases characterized by conduction block resolve quickly; those associated with Wallerian degeneration show a reduction of motor amplitude and resolve slowly or incompletely. Needle EMG may show neurogenic changes (i.e., spontaneous activity, abnormal motor unit potentials, and abnormal recruitment) in all muscles supplied by the PIN below the supinator. Needle EMG of radial-innervated muscles (i.e., triceps, anconeus, brachioradialis, and ECR longus) is normal.

SUPERFICIAL RADIAL NERVE LESION

Etiology

Compression of the sensory branch of the radial nerve at the wrist or distal forearm can cause a SRN lesion. The condition is called “cheiralgia paresthetica” when tight-fitting watches, bracelets, or bands compress the SRN. The condition is also referred to as “handcuff neuropathy” when tight handcuffs compress the SRN. A SRN lesion can occur secondary to a fracture of the distal radius because the nerve lies adjacent to the bone.

General Comments

A SRN lesion at the wrist or distal forearm is a common cutaneous neuropathy.

Clinical Features

Sensory disturbance occurs in the radial dorsum of the hand. In most cases, sensation is blunted but not lost, and it is limited to a portion of the radial dorsum of the hand, usually maximal in the interspace between thumb and index finger.3 In some cases, profound sensory deficits may occur. In cases of anomalous SRN innervation to the ulnar dorsum of the hand, SRN injury may result in more extensive sensory deficits over the entire dorsum of the hand.3 Occasionally, pain or dysesthesias in the radial dorsum of hand is the chief complaint. There is no wrist drop or muscle weakness, since the SRN is a pure sensory branch.

Electrodiagnostic Strategy

In a SRN lesion characterized by neurapraxia (i.e., demyelination) the superficial radial sensory response will be preserved. These patients typically recover quickly. The superficial radial sensory response will be reduced or absent in more severe cases associated with axonal loss (Wallerian degeneration). These cases recover slowly or incompletely. Radial motor studies are normal. Needle EMG is normal in all muscles supplied by PIN and the radial nerves.

REFERENCES

INTRODUCTION

Ulnar nerve entrapment syndromes are common, particularly ulnar neuropathy at the elbow (i.e., the retrocondylar groove and the cubital tunnel), although ulnar neuropathy at the wrist (i.e., Guyon’s canal) also occurs. The etiology, general comments, clinical features, and electrodiagnostic (EDX) strategy for these entrapment neuropathies will be reviewed.

ANATOMY

The ulnar nerve is the main continuation of the medial cord of the brachial plexus. Its fibers are usually derived from the eighth cervical and first thoracic roots, although occasionally the seventh cervical root makes a contribution via the lateral cord. In the axilla, the nerve runs between the axillary artery and vein. In the arm, it stays between the brachial artery and vein, sharing the neurovascular bundle with the median nerve. At the midarm, it leaves the neurovascular bundle and passes posteriorly through the medial intermuscular septum to descend on the medial aspect of the medial head of the triceps. The nerve is superficial throughout this course and innervates no muscles in the arm.

At the elbow, the nerve lies in a groove formed by the medial epicondyle of the humerus and the olecranon process of the ulna (known as the retrocondylar groove). It enters the forearm through an aponeurotic arcade (known as the cubital tunnel) joining the two heads of the flexor carpi ulnaris (FCU), which it innervates. The arcade typically lies about 1.5 cm distal to the medial epicondyle. The nerve travels through the belly of the FCU and then exits by piercing the aponeurosis on the undersurface of the muscle. It then lies in the plane between the FCU and the flexor digitorum profundus (FDP) (to the ring and little fingers), which it innervates.

The nerve is joined by the ulnar artery in the upper forearm to form a neurovascular bundle. About the middle of the forearm, the ulnar nerve gives rise to the palmar cutaneous branch, which descends to provide sensory innervation to the medial aspect of the proximal palm. About 7 cm proximal to the wrist it also gives off a dorsal cutaneous branch, which provides innervation to the medial aspect of the dorsum of the hand and the dorsoproximal aspect of the little and medial ring fingers. The ulnar nerve provides no sensory innervation above the wrist. Hence, sensory loss in the forearm or arm is not a feature of an ulnar nerve lesion. At the wrist, the ulnar nerve and artery lie in a canal formed by the pisiform medially and the hook of the hamate laterally (known as Guyon’s canal). In this region, the nerve divides into superficial and deep branches. Although the superficial branch generally is considered a sensory branch, it supplies the palmaris brevis, a thin muscle beneath the skin of the proximal medial palm, which cannot be studied using needle electromyography (EMG). It then provides sensory innervation to the distal palm and terminates in two digital branches that are distributed to the ulnar side of the little finger and the adjoining sides of the ring and little fingers. The deep muscular branch gives off a hypothenar branch to innervate the abductor, opponens, and flexor digit minimi. It then follows the course of the deep palmar arch across the hand. As it crosses, it supplies the dorsal and palmar interossei and the third and fourth lumbricals. At its termination between the thumb and index fingers, it supplies the flexor pollicis brevis (deep head) and adductor pollicis.
Etiology

Ulnar neuropathy at the elbow can be caused by compression at the retrocondylar groove due to repeated trauma (e.g., habitual leaning on the elbows, sustained hyperflexion at the elbows), traumatic joint deformity, distal humerus fractures, elbow dislocations, callus formation, rheumatic and degenerative joint disease, congenital anomalies of the medial epicondyle, an epicondylo–olecranon ligament, valgus deformity, and immobilization during surgery.

General Comments

Originally, the term “tardy ulnar palsy” referred to antecedent traumatic joint deformity or recurrent subluxation of the nerve out of the retrocondylar groove. However, it is not clear that recurrent subluxation is a risk factor for ulnar neuropathy (the author’s personal observation) and many clinicians now use the term for any entrapment of the ulnar nerve at the elbow. The appearance of ulnar mononeuropathy may herald the onset of a more generalized neuropathy. Ulnar neuropathy at the retrocondylar groove should be distinguished electrodiagnostically from cubital tunnel syndrome. The distinction can be important in surgical management; the former generally requires surgical transposition of the nerve, whereas the latter may warrant simple decompression in the tunnel, without transposition.

Clinical Features

Paresthesia, pain, or numbness occurs in the sensory distribution of the ulnar nerve, including the dorsum of the hand. There is pain or tenderness at or slightly distal to the elbow. In severe cases, weakness and wasting of the FDI and other ulnar-innervated hand muscles may occur. Clinical evidence of weakness may preferentially involve the FDI. Weakness of the FCU and FDP (to the ring and little fingers) may be variable. Radiographic studies are usually normal.

Electrodiagnostic Strategy

Use routine motor nerve conduction studies (NCSs) to demonstrate slowing of conduction velocity or conduction block at the elbow. Use routine sensory NCSs to demonstrate an absent or reduced superficial sensory response (from the little finger) and dorsal ulnar cutaneous response. Use special NCSs (e.g., “inching technique”) to precisely localize the conduction abnormality (e.g., focal slowing, conduction block) to the retrocondylar region. Demonstrate neurogenic needle EMG abnormalities in the FDI and other ulnar-innervated hand muscles. Needle EMG abnormalities in the FCU and FDP (superficial head) localize the lesion to the elbow. In many patients, however, these muscles may be normal, particularly in mild ulnar neuropathy.

ULNAR NEUROPATHY AT THE ELBOW (CUBITAL TUNNEL SYNDROME)

Etiology

Entrapment of the ulnar nerve occurs in the tunnel formed by the tendinous arch connecting the humeral and ulnar heads of the FCU 1-2 cm distal to the medial epicondyle.

General Comments

When the elbow is flexed the cubital tunnel narrows and the ulnar nerve is pulled tightly across the retrocondylar groove. Sustained hyperflexion at the elbow (e.g., using the hand as a pillow, prolonged talking on a cell phone, placing the elbow on a firm surface) is important in the development of cubital tunnel syndrome and ulnar neuropathy at the retrocondylar groove. There may be no predisposing joint deformity or prior trauma to the elbow. Bilateral ulnar neuropathy occurs commonly. Ulnar neuropathy at the retrocondylar groove should be distinguished by EDX examination from cubital tunnel syndrome. The former generally requires surgical transposition of the nerve, whereas the latter may warrant simple decompression in the tunnel, without transposition.

Clinical Features

Paresthesia, pain, or numbness occurs in the sensory distribution of the ulnar nerve, including the dorsum of the hand. There is pain or tenderness at or slightly distal to the elbow. In severe cases, weakness and wasting of the FDI and other ulnar-innervated hand muscles may occur. Clinical evidence of weakness may preferentially involve the FDI. Weakness of the FCU and FDP (to the ring and little fingers) may be variable. Radiographic studies are usually normal.

Electrodiagnostic Strategy

Use routine motor NCSs to demonstrate slowing of conduction velocity or conduction block at the elbow. Use routine sensory NCSs to demonstrate an absent or reduced superficial sensory response (from the little finger) and dorsal ulnar cutaneous response. Use special NCSs (e.g., “inching technique”) to precisely localize the conduction abnormality (e.g., focal slowing, conduction block) to the cubital tunnel. Demonstrate neurogenic
needle EMG abnormalities in the FDI and other ulnar-innervated hand muscles. Needle EMG abnormalities in the FCU and FDP (superficial head) localize the lesion to the elbow. These muscles may be normal, however, particularly in mild ulnar neuropathy.2,4,6

ULNAR NEUROPATHY AT THE WRIST (GUYON’S CANAL)

Etiology

Entrapment of the ulnar nerve occurs in the tunnel formed by the pisiform bone medially and the hook of the hamate laterally. The firm floor consists of the thick transverse carpal ligament and subjacent bone. The distal roof is rigidly bound by the pisohamate ligament. Entrapment may be associated with a lipoma, ganglion cyst, aneurysm, other mass lesion, or chronic compression to the hypothenar region (e.g., bicycle bars, crutches, or occupation).

General Comments

Within the canal, the nerve divides into superficial (sensory), deep (muscular), and hypothenar (muscular) branches.

Clinical Features

Compression within Guyon’s canal may predominantly affect certain fascicles or branches of the ulnar nerve to produce distinctive patterns of symptoms and signs.9 Pattern 1 is a lesion primarily involving the deep branch that will produce weakness in the interossei and lumbricals but not the hypothenar muscles; there are no sensory deficits. Pattern 2 is a lesion involving the deep branch and the hypothenar motor branch that will produce weakness in the interossei, lumbricals, and hypothenar muscles; there are no sensory deficits. Pattern 3 is a lesion at or proximal to the bifurcation into the deep and superficial branches that will produce weakness in the interossei, lumbricals, and hypothenar muscles and sensory deficits in the distal palm, the little finger, and the ulnar side of the ring finger. Pattern 4 is a lesion primarily involving the superficial sensory branch that will produce only sensory deficits in the distal palm, the little finger, and the ulnar side of the ring finger.

Lesions that compress the deep and hypothenar motor branches (patterns 1 and 2) are the most common and may be confused with focal onset of amyotrophic lateral sclerosis, particularly in the all-ulnar hand (Riches–Cannieu anastomosis). Lesions that compress the superficial branch (patterns 3 and 4) do not produce loss of sensation over the ulnar dorsal surface of the hand (dorsal ulnar cutaneous distribution).

Electrodiagnostic Strategy

Use NCSs to localize the conduction abnormality (e.g., prolonged distal latencies in motor or sensory responses, reduced amplitudes) to the wrist. Motor NCSs should be recorded over the FDI as well as hypothenar muscles. This allows detection of distal deep branch lesions. Elicit normal sensory NCSs of the ulnar dorsal cutaneous branch. Demonstrate neurogenic needle EMG abnormalities in the FDI and other ulnar-innervated hand muscles. Perform needle EMG in additional muscles to exclude anterior horn cell disease and C8 and T1 radiculopathy.
INTRODUCTION

Surgical treatment of upper extremity focal neuropathies occurs commonly. Yet, too few physicians who refer patients for surgical intervention have a clear understanding of the indications or of the surgery that awaits their patients. This discussion will review the indications and highlight the various surgeries available for treatment of common focal neuropathies of the upper extremity. It will also provide a brief overview of more recent surgical techniques that can be utilized to promote recovery of nerve function after severe nerve injury.

Patients with focal neuropathies involving the following nerves are often referred for surgical evaluation.

MEDIAN NERVE

The median nerve, or its branches, may be affected by penetrating injuries at any level, although there are certain sites where the nerve is prone to injury. In the upper arm, the nerve is closely bound to the axillary artery and then to the brachial artery as far as the cubital fossa. This close relationship explains why combined nerve–arterial injury is common in this region and why the nerve is subject to compression from aneurysms. In the lower arm, a spur of bone may rarely project from the anteromedial aspect of the supracondylar surface of the humerus and be joined to the medial epicondyle by a strong ligament (called the Struthers’ ligament). This ligament may compress the median nerve proximal to the innervation to the pronator teres, giving rise to the ligament of Struthers syndrome (also known as the supracondylar process syndrome). In the pronator teres syndrome, the median nerve is injured in the upper forearm due to trauma, fracture, or, under exceptional circumstances, compression between the two heads of the pronator teres or a fibrous band as it emerges from this muscle. More distally in the forearm, the anterior interosseous branch may be injured by trauma or as a consequence of neuralgic amyotrophy. Entrapment of the anterior interosseous nerve may also result from fibrous bands or anomalous muscles, giving rise to the anterior interosseous nerve syndrome. At the wrist, damage to the median nerve is commonly due to compression of the nerve in the carpal tunnel. This results in carpal tunnel syndrome, the most common entrapment neuropathy in humans.

ULNAR NERVE

The ulnar nerve, or its branches, may be involved by penetrating injuries at any level. However, there are certain sites where the nerve is prone to injury. Compression neuropathies of the ulnar nerve at the elbow are common and widely recognized. In the retrocondylar groove, the nerve lies on bone covered only by a thin layer of skin and is subject to chronic compression from multiple etiologies. At 1-2 cm distally, the nerve may be entrapped at the cubital tunnel. Compression at either site may result in the clinical presentation known as ulnar neuropathy at the elbow. Rarely, the nerve may be entrapped in the proximal forearm as it pierces the deep aponeurosis investing the undersurface of the flexor carpi ulnaris, or in the distal forearm by a fibrovascular band or hypertrophied flexor carpi ulnaris tendon. Entrapment at the wrist (Guyon’s canal) may present with different patterns of sensorimotor deficits, depending on the degree of involvement of the superficial (sensory), deep (motor), or hypothenar (motor) branches.
RADIAL NERVE

The radial nerve, or its branches, may be involved in penetrating injuries at any level, but there are certain sites where the nerve is more prone to injury. In the brachio–axillary angle, compression may result from a misused crutch (known as crutch palsy or crutch neuropathy) or from fractures of the upper third of the humerus. In the region of the spiral groove and lateral intermuscular septum, compression neuropathies of the radial nerve are common and widely recognized. The nerve most commonly is damaged by fractures of the humerus or during deep intoxication with the arm draped over the edge of a bed, chair, or bench (known as “Saturday night palsy”). In its course through the supinator (arcade of Frohse), the posterior interosseous nerve is prone to damage from fractures of the upper third of the radius and less commonly from entrapment. Injury to the posterior interosseous nerve gives rise to posterior interosseous nerve syndrome. Injury to the superficial radial branch may result from tight handcuffs (known as handcuff neuropathy) or carelessly administered intravenous infusions. A superficial radial nerve lesion at the wrist or distal forearm is a common cutaneous neuropathy.

AXILLARY NERVE

The axillary nerve arises in the axilla as the smaller of the two terminal divisions of the posterior cord. The fibers are derived from the fifth and sixth cervical ventral rami. The nerve descends posterior to the axillary artery and anterior to the subscapularis muscle. At the lower border of the subscapularis, it curves posteriorly through the quadrangular (quadrilateral) space with the capsule of the shoulder joint above (i.e., humeroscapular articular capsule), the surgical neck of the humerus laterally, the long head of the triceps medially, and the teres major below. In this part of its course it is accompanied by the posterior circumflex artery.

As the nerve emerges from the quadrangular space, it divides into anterior and posterior terminal divisions. The posterior branch deviates medially and supplies the teres minor and the posterior fibers of the deltoid, which take origin from the spine of the scapula. It then gives off a branch, the upper lateral cutaneous nerve of the arm, to reach and ramify over the skin superficial to the deltoid. The anterior branch turns laterally around the surgical neck of the humerus, accompanied by the posterior circumflex artery. It then supplies the middle and anterior fibers of the deltoid, which take origin from the acromion and clavicle, respectively.

An axillary nerve lesion can occur secondary to inferior dislocation of the humerus at the shoulder joint (the nerve is stretched across the head of the humerus) or secondary to fractures of the surgical neck of the humerus. Indeed, injuries about the shoulder joint are the most common cause of axillary neuropathy. In athletes playing contact sports, a direct blow to anterior or middle deltoid muscle fibers can cause an isolated axillary nerve lesion. Deep penetrating wounds and improperly placed injections can also injure the axillary nerve. Upward pressure in the axilla from misused crutches can compress the axillary nerve. Most cases of crutch neuropathy also involve the radial nerve. The axillary nerve can be entrapped within the quadrangular space by fibrous bands, muscle hypertrophy, or repetitive trauma. Neuralgic amyotrophy (also known as idiopathic brachial plexopathy) can be causative.

Most axillary nerve lesions resolve spontaneously. However, if axonal loss injury has occurred, prognosis for recovery of function will be less favorable and will depend on reinnervation and regeneration, which may be slow and incomplete. In the latter cases, surgical intervention may be needed.

MUSCULOCUTANEOUS NERVE

Fibers to the musculocutaneous nerve are derived primarily from the fifth and sixth cervical roots, with an occasional contribution from the seventh cervical root. The nerve trunk arises from the lateral cord of the brachial plexus near the lower border of the pectoralis minor muscle. It proceeds downward to pierce the coracobrachialis muscle, which it innervates. The nerve passes through this muscle to reach the interval between the brachialis and biceps muscles. The nerve supplies both of these muscles as it descends between them. Just below the elbow it pierces the deep fascia lateral to the tendon of the biceps, continuing as the lateral cutaneous nerve of the forearm (also known as the lateral antebrachial cutaneous nerve). This branch provides cutaneous innervation to the lateral aspect of the forearm, as far as the midline anteriorly and posteriorly, and as distal as the base of the thenar eminence and dorsolateral aspect of the wrist.

Trauma and deep penetrating wounds can cause a musculocutaneous lesion.

Neuralgic amyotrophy also can be causative. Rarely, vigorous upper extremity exercise can cause entrapment of the musculocutaneous nerve as it passes through the coracobrachialis muscle. Isolated injury to the lateral cutaneous nerve of the forearm also can occur. This pure sensory nerve may be entrapped between the biceps aponeurosis and tendon and the brachialis muscle. Other causes of isolated injury to the lateral cutaneous nerve of the forearm include hyperextension sports injuries to the elbow and antecubital phlebotomy. Trauma almost always results in combined lesions of the musculocutaneous nerve and the lateral cord of the brachial plexus.

SUPRASCAPULAR NERVE

Fibers to the suprascapular nerve are derived from the fifth and sixth cervical roots. Occasionally, the nerve is derived solely from the fifth or from the fifth and fourth cervical roots. The nerve arises from the upper trunk of the brachial plexus and passes obliquely outward beneath the trapezius and omohyoid muscles to reach the suprascapular notch of the scapula. This notch is bridged by the superior transverse scapular ligament to form an osseofibrous foramen through which the suprascapular nerve passes to enter the supraspinous fossa. In the fossa, the nerve lies beneath the supraspinatus muscle, which it innervates.

The nerve then continues around the curved free lateral border of the spine of the scapula to reach the spinoglenoid notch. This notch is covered by the inferior transverse scapular (or spinoglenoid) ligament, which may also form an osseofibrous foramen through which the suprascapular nerve passes to enter the infraspinous fossa. In this fossa, the nerve supplies the infraspinatus muscle. One of the most important uses of the supraspinatus and infraspinatus muscles is the protection they
afford to the shoulder joint; the supraspinatus supports it above and prevents displacement of the head of the humerus upward, while the infraspinatus (and teres minor) protect it posteriorly and prevent dislocation backward.

Trauma to the shoulder, fractures of the scapula or humerus, and penetrating wounds can cause direct nerve injury.\textsuperscript{14} Mass lesions such as ganglion cysts or neoplasms can also cause suprascapular nerve injury. Entrapment at the suprascapular notch of the scapula, or rarely at the spinoglenoid notch,\textsuperscript{15,16} can cause a suprascapular nerve lesion.

Neuralgic amyotrophy also is causative. Trauma often results in combined lesions of the suprascapular nerve and the upper trunk of the brachial plexus.\textsuperscript{1} Clinically, the shoulder pain is usually described as a deep, dull ache located posterolaterally in the shoulder. There is wasting of the supraspinatus and infraspinatus muscles. Initiating of abduction of the arm may be difficult, although the deltoid usually compensates for the loss of the supraspinatus. Weakness of external rotation of the humerus due to infraspinatus weakness is the major clinical manifestation of a suprascapular nerve lesion. Teres minor only partially compensates for the loss of the infraspinatus. In a patient with shoulder pain and weakness of external rotation of the arm, a normal needle electromyography (EMG) examination of the infraspinatus suggests a rotator cuff tear. In a suprascapular nerve lesion, there is no sensory loss.

REFERENCES

Upper Extremity Focal Neuropathies

CME Questions:

1. Which of the following nerves is a branch off the median nerve?
   A. Posterior interosseus nerve.
   B. Palmar cutaneous sensory nerve.
   C. Supracondylar nerve.
   D. Medial antebrachial cutaneous nerve.

2. Which of the following muscles is innervated by fibers from the median nerve?
   A. Adductor pollicis.
   B. Supinator.
   C. Flexor carpi radialis.
   D. Extensor pollicis longus.

3. During an EMG, denervation is observed in the following muscles: abductor pollicis brevis, flexor digitorum profundus to the index finger, and flexor carpi radialis. Which of the following is the most likely site of compression:
   A. The lacertus fibrosis of the biceps tendon.
   B. Guyon’s canal.
   C. Pronator quadratus.
   D. Transverse carpal ligament.

4. Which syndrome should cause no sensory loss?
   A. Supracondylar syndrome.
   B. Pronator syndrome.
   C. Anterior interosseus nerve syndrome.
   D. Carpal tunnel syndrome.

5. Which of the following muscles will be normal on needle EMG examination in a patient with severe posterior interosseous nerve lesion?
   A. Extensor carpi ulnaris.
   B. Extensor carpi radialis longus.
   C. Extensor digiti minimi.
   D. Extensor digitorum communis.

6. A 33-year-old man awakens with a complete right wrist drop. EMG and nerve conduction studies (NCS) are performed two days later to confirm acute radial neuropathy at the spiral groove. NCS show preserved radial sensory response from the dorsum of hand and motor response from extensor digiti minimi muscle. Nerve conduction studies show apparent conduction block localized to the spiral groove. EMG shows no acute denervation or voluntarily recruited motor unit potentials in forearm digital or wrist extensors. Which of the following diagnoses is correct?
   A. Prognosis for recovery of function is favorable but will depend on reinnervation, which may be slow and incomplete.
   B. Prognosis for recovery of function is excellent since this is an acute neurapraxic injury, which will resolve spontaneously.
   C. Prognosis for recovery of function is uncertain because it is too early for Wallerian degeneration to fully manifest.
   D. Prognosis for recovery of function is poor and reinnervation is unlikely to occur due to the severe nature of the injury.

7. Which of the following are risk factors for radial neuropathy at the arm?
   A. Sustained hyperflexion at the elbow.
   B. Habitual leaning on the elbows.
   C. Use of sedatives for sleep.
   D. Recurrent lateral epicondylitis (tennis elbow).

8. Clinical features of posterior interosseous neuropathy include which of the following?
   1. There is normal supination of the forearm.
   2. Radial wrist extension is preserved.
   3. There is no sensory impairment because the superficial radial nerve arises above the arcade of Frohse.
   4. Weakness occurs during finger extension and thumb extension.
      a. Only 1, 2, and 3 are correct.
      b. Only 1 and 3 are correct.
      c. Only 2 and 4 are correct.
      d. All are correct.

9. Which of the following values will typically be normal in an ulnar neuropathy at the wrist?
   A. Ulnar deep branch motor response.
   B. Superficial ulnar sensory nerve action potential.
   C. Dorsal ulnar cutaneous sensory nerve action potential.
   D. Ulnar hypothenar branch motor response.
10. EMG and nerve conduction studies (NCS) are performed for possible right ulnar neuropathy at the elbow. NCS show reduced amplitude of ulnar sensory response from little finger, dorsal ulnar cutaneous response, medial antebrachial cutaneous response, and all motor responses from intrinsic hand muscles. Which of the following diagnoses is correct?
   A. Ulnar neuropathy at the elbow (retrocondylar groove).
   B. Ulnar neuropathy at the elbow (cubital tunnel).
   C. C8 radiculopathy.
   D. Medial cord brachial plexopathy.

11. Which of the following are risk factors for ulnar neuropathy at the elbow. All of the following statements are correct EXCEPT one.
   A. Sustained hyperflexion at the elbow.
   B. Habitual leaning on the elbows.
   C. Immobilization during surgery.
   D. Recurrent lateral epicondylitis (tennis elbow).

12. Compression within Guyon’s canal may predominantly affect certain fascicles or branches of the ulnar nerve to produce distinctive patterns of symptoms and signs. Which of the following patterns are seen in this nerve entrapment?
   1. Weakness in interossei and lumbricals but not hypothenar muscles with no sensory deficits.
   2. Weakness in interossei, lumbricals and hypothenar muscles with no sensory deficits.
   3. Weakness in interossei, lumbricals and hypothenar muscles, and sensory deficits in the distal palm, fifth digit and ulnar side of fourth digit.
   4. Only sensory deficits in the distal palm, fifth digit and ulnar side of the fourth digit.
      a. Only 1, 2, and 3 are correct.
      b. Only 1 and 3 are correct.
      c. Only 2 and 4 are correct.
      d. All are correct.

13. Temperature has a profound effect on nerve conduction studies. Low temperature is common in feet and hands, and will cause which of the following changes in nerve conduction studies:
   1. Prolong distal latencies.
   2. Decrease action potential amplitudes.
   3. Slow conduction velocity.
   4. Decrease terminal segment temporal dispersion and mask an underlying mild peripheral neuropathy.
      a. Only 1, 2, and 3 are correct.
      b. Only 1 and 3 are correct.
      c. Only 2 and 4 are correct.
      d. All are correct.

14. Clinical features of severe carpal tunnel syndrome that warrant carpal tunnel release include?
   1. Persistent numbness in median nerve hand distribution.
   2. Weakness in opposition of the thumb.
   3. Thenar muscle atrophy.
   4. Failed conservative management (NSAIAs, wrist brace, steroid injections).
      a. Only 1, 2, and 3 are correct.
      b. Only 1 and 3 are correct.
      c. Only 2 and 4 are correct.
      d. All are correct.

15. EMG and nerve conduction studies (NCS) show a mild to moderate carpal tunnel syndrome. A carpal tunnel release is performed. All of the following statements are correct EXCEPT one.
   A. The majority of patients recover completely.
   B. Recurrence of carpal tunnel syndrome following carpal tunnel release is rare.
   C. Nerve conduction studies after carpal tunnel release show improvement.
   D. Simultaneous decompression of the ulnar nerve at the wrist (Guyon’s canal) results in a more favorable prognosis for recovery of function.

16. Which of the following are indications for ulnar nerve transposition at the elbow?
   1. Persistent numbness in 4th/5th digits, medial hand, and dorsum of hand.
   2. Habitual leaning on elbows.
   3. Weakness and wasting of ulnar innervated hand muscles.
   4. Recurrent medial epicondylitis (tennis elbow).
      a. Only 1, 2, and 3 are correct.
      b. Only 1 and 3 are correct.
      c. Only 2 and 4 are correct.
      d. All are correct.

17. A 50-year-old man suffers an acute humerus fracture after a fall resulting in a complete wrist drop. The indications for referring this patient to a peripheral nerve surgeon for exploration and possible nerve graft include which of the following?
   A. Complete loss of motor function in brachioradialis and all other forearm muscles supplied by the radial nerve (except anconeus) lasting >2 weeks.
   B. A drop in amplitude of the compound muscle action potential of >50% that persists for >2 months post-injury.
   C. EMG that shows persistent denervation with no voluntarily recruited motor unit potentials in radial-innervated forearm muscles at 6 months post-injury.
   D. Persistent sensory deficits in the superficial radial nerve distribution.