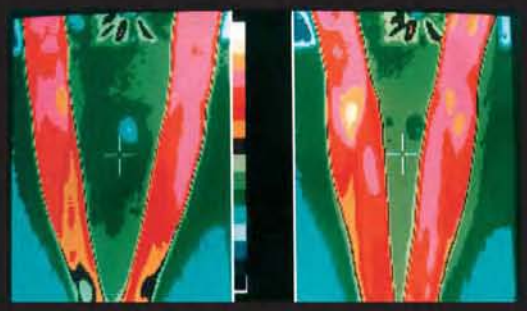

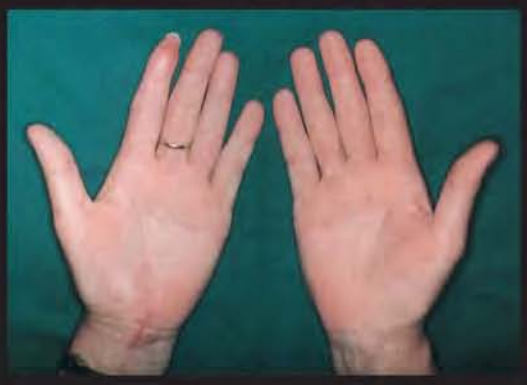

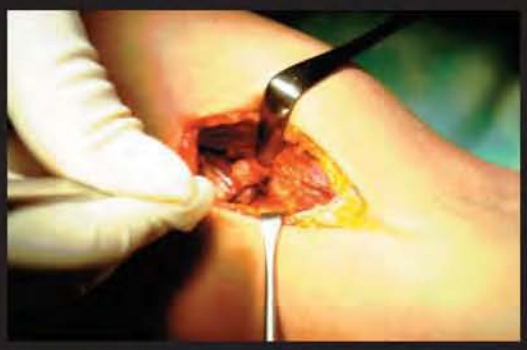

Third Edition

Tunnel Syndromes

Peripheral Nerve Compression Syndromes

Marko M. Pećina
Jelena Krmpotić-Nemanić
Andrew D. Markiewitz

CRC PRESS

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Third Edition Preface

It is with pleasure, but also a sense of responsibility, that we present, in a span of only 10 years, the third edition of our book. The implied question is, are the changes in the field that this book covers so important and frequent that so many new editions are necessary? The answer is yes. Exactly 10 years ago, when the first edition of this book, *Tunnel Syndromes*, was published, only a few of the tunnel syndromes described at that time were accorded any clinical significance. However, in recent years, tunnel syndromes or entrapment neuropathies have become increasingly recognized as a cause of pain and dysfunction in various parts of the human body. This has been accompanied with a rapidly enlarging body of literature on this subject.

What is the same, and what has changed in the third edition of our book? The authors are the same, as is the concept of the book. The third edition continues to probe the origins of these painful syndromes and to propose the possible causes that lead to them. This edition, similar to the preceding two, is intended to give a quick overview of the definition, anatomy, etiology, clinical symptoms and signs, and treatment of tunnel syndromes. Like the first two editions, this book is intended for a wide spectrum of medical students, general practitioners, and specialists from different fields of medicine, not only those whose primary concern is surgery. This is also an answer to the most common criticism of the first two editions, that treatment, particularly surgical procedures for various syndromes, was not described in enough detail.

The third edition of the book has nine new syndromes, bringing the total number described in the book to more than 50. The credit for the drawings in these new chapters goes to Milivoj Mervar from the Faculty of Civil Engineering, University of Zagreb. Professor Dr. Marija Šoštarko, a neurologist from the School of Medicine, University of Zagreb, completely rewrote the chapter Neurophysiology and Electrodiagnosis of Entrapment Neuropathies. All chapters from the second edition feature new references, as well as new insights acquired during these 10 years. Some chapters also feature new illustrations. Because of the continued support and understanding of our publisher, this edition also surpasses the preceding ones in graphic design and presentation, for which we are truly grateful, not only for ourselves, but, also we hope, for the still numerous readers of this book.

Marko M. Pećina
Zagreb

The Editors

Marko M. Pećina, M.D., Ph.D., is chairman and professor of orthopaedic surgery at the School of Medicine, University of Zagreb, Croatia.

Professor Pećina graduated from Zagreb University Medical School in 1964. From 1965 to 1970 he was assistant lecturer at the Anatomic Institute of Zagreb Medical School. He obtained his M.Sc. degree in experimental biology at Zagreb University Faculty of Natural Sciences in 1968, and defended his Ph.D. in medical science in 1970 at Zagreb University Medical School. In 1970 he became an assistant lecturer at Zagreb Medical School Orthopaedic Department, and in 1977, became senior lecturer, in 1980 associate professor and in 1984 professor in orthopaedics.

Professor Pećina furthered his professional training in many orthopaedic institutions around the world (Lyon, Bologna, Vienna, Basel, London, Milwaukee, New York, Baltimore, Los Angeles, Columbus, and others), as well as participating in numerous international symposia and congresses. He is also engaged in professional associations in Croatia and abroad. He is an active member of the Croatian Medical Association, the Croatian Orthopaedic Society (president), and the Croatian Sports Medicine Society, as well as the following international societies: International Society of Arthroscopy, Knee Surgery and Sports Medicine; Spine Society of Europe; French Society for Orthopaedic Surgery and Traumatology; European Society of Sports Traumatology, Knee Surgery and Arthroscopy; College Européen de Traumatologie du Sport; International Association of Olympic Medical Officers; Italian Club of Knee Surgery; American Academy of Orthopaedic Surgeons; European Calcified Tissue Society and International Federation of Sports Medicine. He is a national delegate of Croatia and a member of the International Committee of International Society of Orthopaedic Surgery and Traumatology (SICOT).

Professor Pećina is an honorary member of the Hellenic Orthopaedic and Traumatology Society. He is one of the founders of the European Spinal Deformities Society and was vice-president from 1989 to 1992. He was a member of Croatian Olympic Committee and Chairman of the Medical Commission. In addition, he is editor-in-chief of the *Croatian Sports Medicine Journal*, assistant editor of *International Orthopaedics*, a corresponding member of the Editorial Board of The Hip International and *Acta Chirurgiae Orthopaedicae et Traumatologie Cechoclovaka*.

Particularly interested in clinical anatomy and applied biomechanics of the musculoskeletal system, scoliosis, knee surgery and sports traumatology, Professor Pećina has published more than 300 expert and scientific papers and several books, including *Tunnel Syndromes*, CRC Press 1991; and second edition 1997; and *Overuse Injuries of the Musculoskeletal System*, CRC Press 1993.

Professor Pećina has been awarded numerous tokens of appreciation for his professional achievements, such as the Balkan Medical Union Award for Scientific Achievement, the Croatian Award of Sports Medicine, the ESSKA Award for a Scientific Poster, the "Juan Anatonio Samaranch" Award for Scientific Lecture, and the Croatian Award for Scientific Achievement, among others. He is a regular member of the Croatian Medical Academy and an associated member of the Croatian Academy of Sciences and Arts.

Jelena Krmpotić-Nemanić, M.D., Ph.D., is emeritus chairman and professor of anatomy, School of Medicine, University of Zagreb, Croatia.

Professor Jelena Krmpotić-Nemanić obtained her M.D. degree in 1944 from the School of Medicine University of Zagreb. She defended her Ph.D. in 1957 at the School of Medicine University of Zagreb, where she also specialized in otorhinolaryngology, 1960–1963. In 1942, she began work in the Department of Anatomy, School of Medicine University of Zagreb. In 1949 she became a lecturer, in 1953 associated professor and in 1963, a professor. From 1961 to 1980 she was head of the Department of Anatomy.

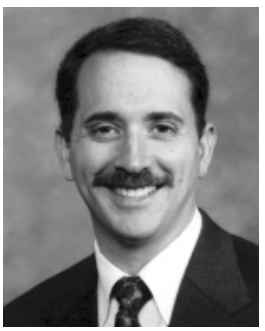
Professor Jelena Krmpotić-Nemanić is an honorary member of the Austrian Otorhinolaryngological Society, a corresponding member of the German Otorhinolaryngological Society in Weisbaden and Berlin, honorary professor at the University of Munich, and a member of the Collegium Otorhinolaryngologicum Amicitiae Sacrum. She is also the member of the Croatian Academy of Sciences and Arts. Professor Krmpotić-Nemanić is redactor of Zeitschrift fur Laryngologie, Rhinologie und Otologie; European Archives of Oto-Rhino-Laryngology, and Otorhinolaryngologia Nova.

Among the awards she has received are the Ludwig Haymann's Price Decoration, the Austrian Cross of Honor and the Laureat of the French Medical Academy.

Professor Krmpotić-Nemanić has been a researcher and collaborator on three consecutive USA joint board grants concerned with the prenatal, perinatal and postnatal development of the human frontal lobe, and has been the principal investigator on several research grants in Croatia.

Professor Krmpotić-Nemanić has presented more than 30 guest lectures in Europe and the United States. She has published more than 200 research papers, many of which are cited in *Science Citation Index*. She has authored and co-authored 20 books.

Her current interests include the development of the skull, paranasal sinuses, and interference of anomalies with the nervous system.



Andrew D. Markiewitz, M.D., is an assistant professor at the University of Arkansas for Medical Sciences in Little Rock, Arkansas, where he is also the director of the Hand and Upper Extremity Center and the director of the ACGME accredited Hand Fellowship. He remains a clinical assistant professor at the Uniformed Services University of the Health Sciences in Baltimore, Maryland.

Dr. Markiewitz also functions as the director of Hand Care at the Arkansas Children's Hospital and John L. McClellan Memorial Veterans Administration Medical Center in Little Rock.

Trained in orthopaedic surgery at the Cleveland Clinic Foundation in Cleveland, Ohio, Dr. Markiewitz pursued further training in hand surgery at the New England Medical Center and the New England Baptist Hospital. He is board certified in orthopaedic surgery as well as having received a certificate of added qualifications in hand surgery through the American Board of Orthopaedic Surgeons.

Dr. Markiewitz is an active member in the Academy of Orthopaedic Surgeons, the American Association for Hand Surgery, the American Society for Surgery of the Hand, the Orthopaedic Research Society, the Mid America Orthopaedic Association, the Society of Military Orthopaedic Surgeons and the American Medical Association.

Dr. Markiewitz remains active in both basic and clinical research into upper extremity problems from trauma and nerve compression. Fellows, residents, and medical students participate with him and the Orthopaedic Research Section in the investigations at the cellular and biomechanical level.

Contents

Section I

Overview

1 Significance of Tunnel Syndromes

As they pass through bony, fibrous, osteofibrous, and fibromuscular tunnels, nerves, from their origin in the spinal cord to their effector organ, risk compression, damage, and impairment of their end function. Virtually all nerves carry afferent and efferent impulses along a combination of motor, sensory, and autonomic fibers. However, patients present with signs and symptoms usually associated with the motor or sensory function of the involved nerve. Careful linking of these signs and symptoms can indicate a specific compressive or painful pathology commonly known as a tunnel or canalicular syndrome.

The expressions canalicular, canal, channel, and tunnel have been used interchangeably for these syndromes. *Webster's* illustrates this dilemma in semantics; canal and canaliculus imply an enclosed passage — in either bone or soft tissue — whereas channel is defined as a "bed where a material body may run" and tunnel represents a "bodily channel." In German literature, the expressions Engpass-Syndrome, Einklemmungsneuropathis, and Tunnel syndrome compare with English literature's channel syndrome, entrapment compression neuropathy, and tunnel syndrome; or French literature's canal syndrome or loge syndrome. All of these terms are appropriate, because they seek to describe the damage to neurovascular structures running in a common course through a small area (whether intrinsic or extrinsic in source). To simplify the discussion, this book will use the general term "tunnel syndromes" but will note the common names currently ascribed to each clinical picture.

Multiple approaches have been used to categorize the tunnel syndromes.[1] Any syndrome can, therefore, bear the descriptive name, which could originate from any one of the following sources:

- The compressed nerve (e.g., the ilioinguinal syndrome)
- The anatomical area affected (e.g., metatarsalgia)
- The anatomical tunnel (e.g., carpal tunnel syndrome)
- The motion producing the compression (e.g., hyperabduction syndrome)
- The names of the describing authors (e.g., Kiloh-Nevin's syndrome)

While the names may vary, these syndromes all originate from a lesion to neurovascular elements in a narrow anatomical space. The damage may be caused by tumor compression (intraneural or extraneural); trauma (blunt, sharp, or secondary to repetitive action); infection (inflammation or actual bacterial invasion); metabolic, toxic, iatrogenic, idiopathic, vascular (ischemic,[2] aneurysmal, or tumor in nature), or muscular compression; or anatomical variations; or specific sports activities.[32,33]

When a patient presents with neurovascular symptoms, a careful history and physical exam must be done prior to ordering tests, scans, or studies.[3] Pain of a radicular nature could be a sign, not only of a tunnel syndrome, but also a herniated disc or tumor (piriform muscle syndrome vs. herniated nucleus pulposus herniation vs. ependymoma). Raynaud's phenomenon in the hand could be the result of carpal tunnel syndrome or autonomic dysfunction secondary to autonomic nerve compression. Vascular disease can lead to isolated nerve ischemia, in turn producing symptomatology characteristic of a tunnel syndrome or a combination of syndromes as in thoracic outlet syndrome, carpal tunnel syndrome, and Guyon's tunnel canal syndromes. Symptoms and signs

depend on the type of nerve compressed in the tunnel: motor, sensory, or mixed. While most nerves carry afferent and efferent impulses in addition to autonomic nerve fibers, this book will follow a classic didactic approach and describe symptoms as sensory or motor. Understanding the signs and symptoms as well as the dynamic anatomy of the tunnel allows the practicing physician to identify the syndrome and promptly remove the affecting agent before irreversible damage occurs.

ETIOLOGY AND PATHOGENESIS

Narrowing of the osteofibrous or the fibromuscular neurovascular tunnel represents one of the major factors in the pathogenesis of tunnel syndromes. Narrowing can be caused by changes intrinsic or extrinsic to the tunnel, as detailed in Table 1.1. These changes include tumors, cysts, inflammatory processes (rheumatic, tubercular), trauma (blunt-hematoma formation, sharp fractures), or anatomic variations. Operative exploration frequently will locate and relieve these compressive factors; however, tunnel syndromes secondary to vascular insufficiency, endocrine disease, or metabolic disturbances may not be relieved by surgical intervention. Additionally, tunnels can be compressed by anatomical variations that are significant only with motion. Idiopathic etiologies remain for several tunnel syndromes where extensive investigation fails to yield a cause.

Tunnel compromise does not require major changes in space to dramatically alter function.[4] Inflammatory changes resulting in slight connective tissue thickening of tendon or nerve sheaths can compress a nerve or its vascular supply. Ischemic events initially affect sensory nerve fibers.[5] If the ischemia continues, motor fibers begin to be damaged. Edema secondary to the hormonal changes associated with pregnancy, birth control pills, menopause, and hypothyroidism has been

TABLE 1.1
Several Causes Leading to Alteration of Nerve Function

General Categories	Compressive Causes
Idiopathic/spontaneous	Fibrositis
External (to tunnel)	
Acquired	Spondylosis, arthritis, spinal stenosis, herniated nucleus pulposus
Congenital	Cervical rib
Trauma	Fracture callus, shoulder dislocations
Vascular	Aneurysms, ischemia
Inflammation/autoimmune	Viruses (measles, chicken pox, polio), bacterias, diphtheria, tetanus, leprosy, tuberculosis rheumatic disease
Metabolic	Diabetes, beriberi, pellagra, hypothyroidism, pernicious anemia, drugs (ETOH), metals/chemicals (mercury, arsenic, lead, silver)
Hormonal	Pregnancy
Iatrogenic	Casting
Tumor/neoplasm	Apical lung tumors, ganglions
Internal (to tunnel)	
Acquired	Occupation, dynamic
Congenital	Narrow suprascapular notch, anomalous musculature
Trauma	Hematoma, crush injuries, lacerations
Vascular	Aneurysm, ischemia, arteritis
Inflammation/autoimmune	Rheumatic diseases, tuberculosis
Metabolic	Lead, hypothyroidism, nutrition
Hormonal	Pregnancy
Iatrogenic	Surgical trauma
Tumor	Extrinsic (lymphomas, multiple myelomas), intrinsic (schwannoma, hemangioma)

TABLE 1.2

CHARACTERISTICS OF SEVERAL NERVE LESIONS

Nerve Lesions	Definition
Neuropraxia	Temporary loss of function (not necessarily complete); no neural disruption
Axonotmesis	Axonal and sheath disruption with connective tissue sheath preserved; recovery dependent on distance from lesion to insertion
Neurotmesis	Complete anatomical interruption of the nerve; complete loss of function; no spontaneous recovery; usually secondary trauma

thought to cause tunnel compression. Dynamic changes of a tunnel during daily activity can create traction or compression of a nerve if slight anatomical variations exist. The variations become important because the nerve has restricted mobility between its origin and its course through the tunnel. Nerve damage ranges from temporary and reversible to complete loss of function with or without the chance of regeneration.[34]

Seddon[6] indicated that a tunnel syndrome could produce a neuropraxis or eventually an axonotmesis; however, complete nerve interruptions do not occur in tunnel syndrome. Recognition of symptoms may allow early intervention to relieve the compression leading to neuropraxia (see Table 1.2).

CLINICAL SYMPTOMS AND SIGNS

Patients present to their physician with symptoms that can range from vague complaints of diffuse pain or numbness to specific complaints of muscle weakness or of sensory changes over localized skin areas. Precise assessment of a patient's symptoms yields a better picture of the nerve or nerve types affected, as shown in Table 1.3.

Pain represents the most common symptom. Sharp, burning pain accompanied by paresthesia may be limited to a specific dermatome caused by compression or incipient ischemia of sensory fibers. Compressed sensory fibers lead to a constellation of symptoms such as hyperesthesia, hypesthesia, hypalgesia, hyperalgesia, loss of two-point discrimination, or loss of vibratory sense. Compressed motor nerves create a diffuse, deep pain that can best be localized to a muscle group or joint; however, nerve compression of any type can present with symptoms proximal and distal to the actual area involved. While the pain from tendinitis intensifies with motion and decreases with rest, the pain from a tunnel syndrome may actually be present at all times, worsen with motion, and wake one from sleep.

TABLE 1.3
Examples of Syndromes Associated with Compression of Different Nerve Types

Nerve Type	General Symptoms
Sensory	Loss of discrimination; sharp burning, vibratory sense, paresthesia, hypesthesias, hypalgesia, hyperesthesias, hyperalgesia, pain
Motor	Vague pain, blunt pain, on appropriate muscle group pressure and use; night pain
Mixed	Combined and varying effects
Weakness	Muscle atrophy
Automatic	Vegetative disturbances; less autonomic sweating

For example, because their sensory and motor fields overlap, carpal tunnel syndrome can masquerade as cervical spine or brachial plexus disease and vice versa, therefore, physicians must be cautious in their approach to any neurological complaint. Motor nerve involvement can lead to weakness and atrophy secondary to denervation or disease due to pain. Assessing which muscle or muscle groups are affected helps differentiate which nerves are involved.

Since many nerves are mixed in nature, nerve compression varies in its presentation depending on whether sensory or motor nerve damage dominates. Vegetative symptoms can develop as autonomic fibers become involved. Disturbed sweating, one of the more noticeable symptoms, can be tested in the ninhidrinic test. Decreased sweating is observed in the affected area of innervation, verifying the physical exam.

Tunnel syndromes require specific testing to determine the level of compression as well as the accuracy of the patient's presentation. Sensory symptoms and signs appear before motor signs; basing diagnosis and treatment on the appearance of motor signs would place many patients in an unacceptable position. The length of the wait without any treatment might limit many conservative therapeutic options, since time under compression greatly decreases the chances for maximal nerve recovery. With prolonged nerve compression, impairment of motor strength and function become manifest. Typically, motor strength ranges from 0 to 5, as shown in Table 1.4. Understanding muscle-group innervation allows the physician to use the physical examination to delineate where compression occurs between the brain, the spinal cord, and the destination of the peripheral nerves. This understanding also allows the physician to find those patients with a true pathologic basis among groups of malingerers. Asymmetrical reflexes or a change in reflex strength indicates nerve root disease, as shown in Table 1.4.

TABLE 1.4
Currently Used Muscle and Reflex Grading Systems

Grade	Motor	Reflex
0	Paralysis	Flaccid paralysis
1	Fasciculations	Hyporeflexia
2	Muscle contraction, no motion	Normal
3	Muscle motion with gravity eliminated	Hyperreflexia
4	Muscle action, weak	Hyperreflexia with clonous/spasticity
5	Maximal muscle action	

While electrodiagnostic testing can indicate or confirm a diagnosis, the reliability and accuracy of such testing depends on the tester as well as on the nerve tested;[7,8] therefore, the availability of these tests does not eliminate the need for a detailed history and physical exam. The contribution of electrodiagnostic testing is based on its ability to stimulate specific nerves in a constant and known fashion with an expected result. The failure of the nerve to respond in a timely and appropriate fashion is defined as pathologic. Patterns of response can indicate compression, division, degeneration, or regeneration at specific levels of the nerve's course. Evaluation of muscle response to direct nonneurogenic stimulation (galvanic, faradic steam) differentiates muscle from nerve pathology. Electromyography and nerve-conduction velocity studies help analyze the muscle's response to neural stimulation.

Electromyography records the muscle response to various neural stimuli. The muscle's intrinsic response can be judged from a resting state to a maximally stimulated state. While resting, few, if any, fasciculations should be detected. As nerve stimulation increases, more motor units should be recruited. Their recruitment comes in waves and will become superimposed during maximal stimulation. The patterns for voluntary action, denervation, compression, reinnervation, myopathy,

and lower motor neuron disease are characteristic when fully developed; therefore, testing may need to be repeated to clarify some situations. Nerve-conduction velocity studies use maximal stimulation to assess the time between the stimulus and the motor response. The duration of the action potential and its amplitude also can be evaluated. By varying the stimulated point in the nerve's course, the site of the lesion can be identified. Motor and cutaneous afferent nerves can be tested in this manner. Each nerve has a characteristic conduction time and configuration; however, as noted above, one should not fully rely on these tests, since they require specialists. Furthermore, a normal or borderline-normal electrodiagnostic battery does not exclude the existence of a tunnel syndrome.[9,10]

DIAGNOSIS

Diagnosis begins with a thorough history and physical exam. One should use technological tools to verify any hypothesis, but the modern physical sometimes neglects the clinical examination and relies solely on laboratory tests, radiologic examinations, and electromyographic studies for diagnosis. This approach places the cart before the horse. Wartenberg[11] wrote that if the laboratory examinations do not agree with the clinical findings, one should make the diagnosis based on the clinical findings. Thus, physicians should always reevaluate their hypotheses in the light of changes in their examinations or lab results and redirect their inquiries appropriately.

Understanding the etiology of a tunnel syndrome and the anatomy of the tunnel allows the physician to develop a hypothesis from the patient's history (Figures 1.1A and B). The history should direct one to an appropriate physical exam. If questions remain, then special tests could be ordered to differentiate among the possible disorders.[12–15] The history should elicit the patient's chief complaint, whether it be pain, sensory disorders, or disturbances of mobility. Since descriptions can be confusing, one must explicitly determine what the patient means to say. Special attention must be paid to the following:

- When did the symptoms develop?
- Were they preceded by trauma, hospitalization, surgery, or immobilization?
- What is the actual complex of symptoms? (Care should be taken to differentiate pain from paresthesia and weakness, and temporary from permanent.)
- What areas are involved?
- Does anything relieve or aggravate the patient's symptoms?
- Has the patient been treated previously? How, and was treatment successful?
- What is the patient's profession and what physical tasks and mental stresses does it entail?
- What is the patient's dominant arm?
- What is the patient's general state of health (metabolic or hormonal disorders, developmental deformities, chronic contagious disease)?
- What is the patient's surgical and medical history?
- What medications does the patient take?

This approach should become routine to avoid missing any telltale signs.

Analysis of the history allows one to direct the patient's physical examination. This examination, in itself, should be methodological. First, the skin and nails should be inspected for dystrophic or atrophic changes. As dystrophic or infiltrative changes occur, the skin loses elasticity, splits, and becomes hyperkeratotic. This glossy skin proceeds to loss, blistering, ulcers, and pigment changes in atrophoderma neuriticum. Hypertrichosis or hypotrichosis can also be a consequence of nerve injury. Nails may become thinner, break more easily, and have uneven surfaces; as in Alfod's syndrome, they may have white lines and nail-root elevation. These changes in skin and nails are rarely manifested to large degrees in tunnel syndromes, because partial innervation is usually maintained.

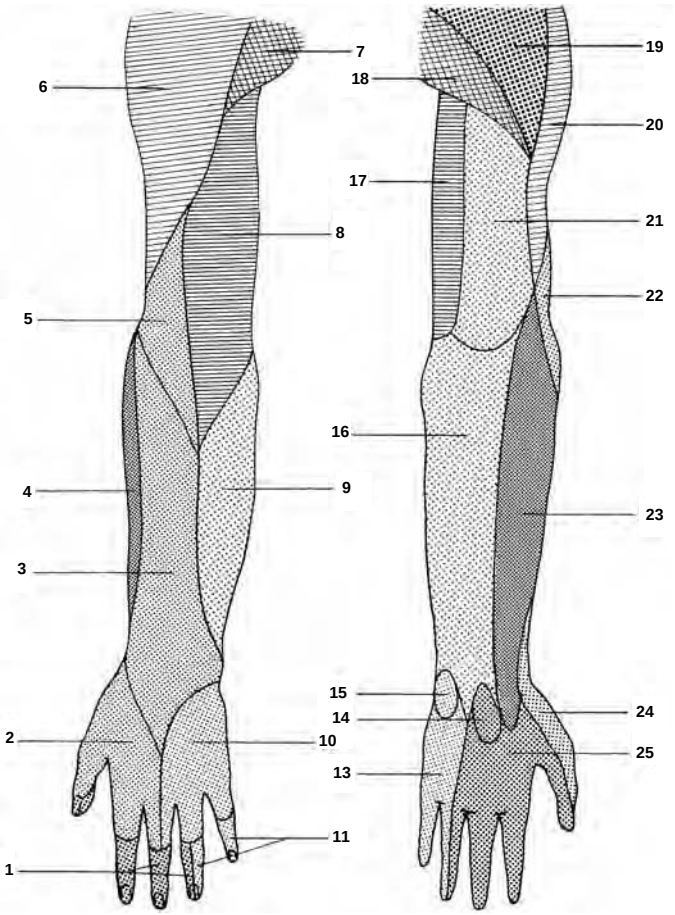


FIGURE 1.1A Dermatomes of the upper extremity. Having a working understanding of these dermatomes allows the physician to delineate the involved nerves — 1: proper palmar digital nerves, median nerve; 2: superficial branch of the radial nerve; 3: posterior cutaneous nerve of the forearm, radial nerve: 4: lateral cutaneous nerve of the forearm, musculocutaneous nerve; 5: dorsal cutaneous nerve of the arm, radial nerve; 6: axillary nerve, cutaneous branch; 7: cutaneous branches of the intercostal nerves; 8: medial cutaneous nerve of the arm; 9: medial cutaneous nerve of the forearm; 10: dorsal branch of the hand, ulnar nerve; 11: proper digital palmar nerves, ulnar nerve; 13: superficial branch of the ulnar nerve; 14: palmar branch of the median nerve; 15: palmar branch of the ulnar nerve; 16: medial cutaneous nerve of the forearm, palmar branch; 17: medial cutaneous nerve of the arm; 18: cutaneous branches of the intercostal nerves; 19: supraclavicular nerves; 20: axillary nerve, cutaneous branch; 21: medial cutaneous nerve of the arm; 22: lower lateral cutaneous nerve of the arm, radial nerve; 23: lateral cutaneous nerve of the forearm, musculocutaneous nerve; 24: superficial radial nerve; 25: palmar digital nerves, median nerve.

Nerve compression in the late stages leads to weakness and atrophy of the involved muscle groups. Inspection of a patient's overall symmetry, posture, scars, swelling, and static deformities can raise or answer questions about his or her disease (Table 1.5). Paralysis of the radial or ulnar nerve will leave the upper extremities in characteristic positions, just as the patient with ilioinguinal nerve oppression can remind the physician of a person with appendicitis.

History and inspection target potential areas for palpation. Smooth skin that allows the hand to glide, decreased sweating, pain, and temperature differences found along the dermatome, when compared to the patient's other side, may be due to the involved nerve. To differentiate tunnel syndromes from ischemic diseases, peripheral pulses should be evaluated. While palpation can

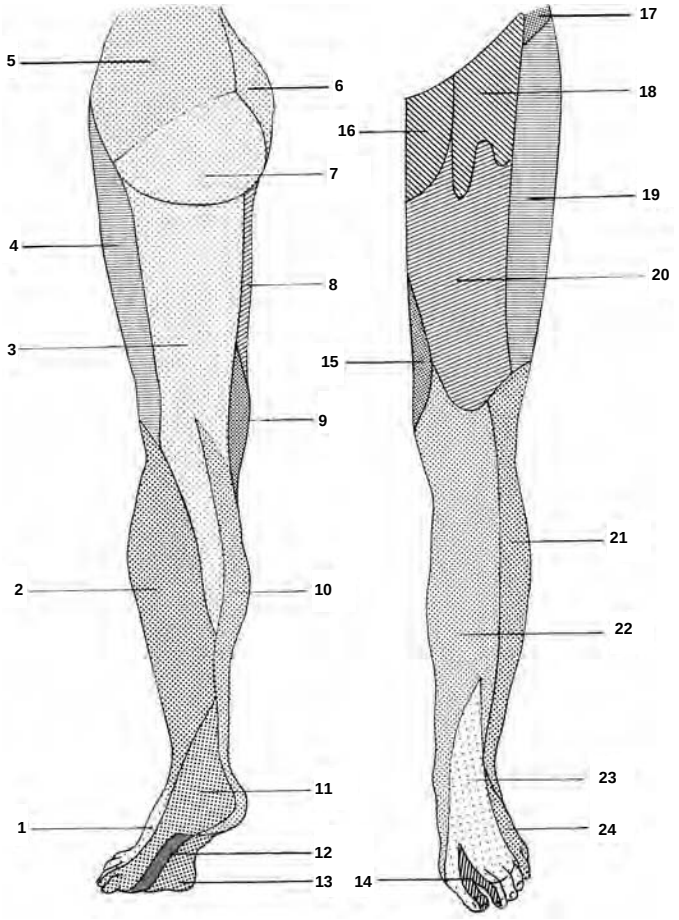


FIGURE 1.1B Dermatome pattern of the lower extremity — 1: superficial peroneal nerve; 2: lateral cutaneous nerve of the calf from the common peroneal nerve; 3: posterior femoral cutaneous nerve; 4: lateral femoral cutaneous nerve; 5: superior cluneal nerves; 6: medial cluneal nerves; 7: inferior cluneal nerves; 8: medial cutaneous nerve of the thigh (from the femoral nerve); 9: obturator nerve, cutaneous branches; 10: saphenous nerve, medial cutaneous branches from the femoral nerve; 11: sural nerve; 12: lateral plantar nerve; 13: medial plantar nerve; 14: deep peroneal nerve; 15: obturator nerve, cutaneous branches; 16: combination of the ilioinguinal and genitofemoral nerves; 17: iliohypogastric nerve, lateral cutaneous branches; 18: genitofemoral nerve, femoral branch (also known as the lumboinguinal nerve); 19: lateral femoral cutaneous nerve; 20: intermediate and medial cutaneous nerves of the thigh (also known as anterior femoral nerve, cutaneous branches); 21: lateral cutaneous nerve of the calf; 22: saphenous nerve, cutaneous branch; 23: superficial peroneal nerve; 24: sural nerve.

indicate painful or weak areas, the physician needs a detailed neurologic examination with objective tests.

Evaluation of pain requires a twofold approach — objective and subjective. Objective examination requires a cooperative patient and repetitive exams, not only to follow the disease, but also to show consistency. One examines light touch, appreciation of temperature, two-point discrimination, vibrational sense, proprioception, pain, and graphesthesia. The standard neurological tests are not appropriate in children, mentally retarded persons, or people in extreme pain. Because patients may exaggerate their problems, both the physician and the patient must, with great patience, experience thorough and repetitive exams, with accurate documentation every time the patient responds.

TABLE 1.5
Physical Signs

Physical Area	Signs
Skin	Temperature
	Scars
	Dystrophic Hyperkeratotic
	Atrophic Glossy, red hairless
	Sweating ability
Nails	Thickness
Muscles	Tone
	Bulk
	Strength
Skeleton	Deformities
	Anomalies
	Old fracture callus

Objective evaluation of pain is based on sweating in response to stimulation. Many investigators have developed tests, as listed in Table 1.6.[16–19] All require a reaction between sweat and indicators such as starch, chimisaum, and ninhidrin. One investigator has introduced the simple method of immersion of the affected area in 40ºC water for 30 minutes.[20] While normal skin would wrinkle after prolonged exposure, skin in the dermatome of a compressed nerve remains smooth.

TABLE 1.6
Tests for Eliciting Nerve Compromise

Tested Response	Test
Sensations	
Touch	Brush
Temperature	Hot/cold water in tubes
Pain	Needle, Tinel's
Vibration	Tuning fork
Graphesthesia	Coins, keys, writing
Two-point	Calipers
Sweating (sense of heat)	Minor (1928): Starch iodine on skin, then stress; potato-starch sweat caused a color change in the starch[17]
	Guttman (1931): Chinisarin on the skin, then rice-starch with a red color developing with sweat[16]
	Moberg (1958): Ninhidrin-soaked paper reacts with amino acid in sweat; when placed in an incubator becomes violet (amino acids: asparagine, gultaine, thiamine, valine, serence, methionine).[18]

The aforementioned tests are clinically important as long as disturbances of sense do not coincide with disturbances of sweating. The autonomic nervous system controls the body's vegetative functions, such as sweating. The hypothalamus processes differences between the environmental temperature and the body's set point and reacts to maintain equilibrium by vasodilatation and sweating to cool or by vasoconstriction to prevent heat loss. Peripheral control of sweating can be influenced by both pilocarpine and heat. Today, computerized telethermography is available to diagnose tunnel syndromes.[21]

Radiographic studies have limited uses, since soft-tissue variations are the compressive agents in many tunnel syndromes. In the ambiguous cases, computed tomography (CT), ultrasonography, and magnetic resonance imaging (MRI) will, with an increase in resolution and a refinement in application, be of use prior to surgical exploration.[22] Plain films can reveal only exostosis, callus, and anatomical anomalies. Special techniques such as angiography can be used occasionally to assess the vascular contribution to a neurovascular compressive syndrome.

TREATMENT

Tunnel syndrome treatment, whether conservative or surgical, must address the etiology causing nerve compression. While conservative measures of splinting and rest may relieve compression caused by repetitive actions, these measures will be ineffective if the compression is caused by fracture callus, soft-tissue compression, exostosis, or anatomical anomalies. Systemic or hormonal disease or changes can initiate or aggravate tunnel syndromes. Appropriate response to these causes might decrease the compression syndromes. Alleviating nerve compression becomes paramount, because time increases the risk of irreversible nerve injury.[35–37]

Where appropriate, conservative measures consisting of immobilization, rest, exercise, ultrasound, heat, massage, and anti-inflammatory medications can be tried. These trials must be monitored to assess the patient's response. If symptoms worsen, corticosteroid injections can be tried as the last conservative option prior to surgery. Steroid injections typically consist of a water-soluble, depot preparation introduced with a thin needle into the tunnel. The injection actually compounds the patient's complaints because it decreases the space remaining in the tunnel. The pain and stiffness decrease over 2 days as the inflammatory portion of the compression decreases. Injections can be tried several times, but caution must be exercised, as repetitive steroid injections can damage tendons and joint surfaces. The physician and the patient must approach each conservative trial with a time limit in mind. If definitive therapy is postponed, the prognosis for nerve recovery worsens.

Surgical decompression (open or endoscopic) remains the last resort when conservative therapy fails.[23–29] Semple and Cargil[30] demonstrated a 97% surgical success if decompression was performed within 6 months of the onset of symptoms. Bilić and Pećina[31] decided that treatment depended on the status of the impaired nerve as assessed by clinical exam and specific tests. Surgery allows direct visualization of the tunnel and the surrounding tissue. Failing that, some tunnel syndromes are treated by transposing the nerve. Neurolysis, tenosynovectomy, arthrodesis, or osteotomy might be required to increase the space within the tunnel.

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2 Neurophysiology and Electrodiagnosis of Compression Syndromes*

INTRODUCTION

Electrodiagnostic studies should be used to confirm or clarify clinical findings of weakness or amyotrophy caused by myogenic or neurogenic etiologies. Electromyography (EMG) confirms axonal lesions of lower motor neurons, helps to define the lesion location and is specifically helpful in tunnel syndromes. Additionally, it can provide information on the level of damage and the degree of recovery, if present. EMGs also help detect physiological variations of the peripheral nerve innervations and test the neuromuscular junction. Nerve conduction velocity (NCV) tests provide information about the state of a peripheral nerve, including its motor and sensory nerve fibers. The obtained results give information about the kind and intensity of the nerve injury, as well as its location. Nerve conduction velocity tests provide information about the state of motor and sensory nerves' sheath. Indirectly, the finding of a slow nerve conduction velocity helps to define the location of the nerve compression. This chapter will review both normal and abnormal findings of electrodiagnostic tests so the clinician can better understand the results and apply them to improve patient care.

BASIC ELECTROMYOGRAPHY ANALYSIS

Normal Activity

The EMG records muscle activity. Electrodes are used to pick up muscle action potentials. Several types of needles are available: concentric needle electrodes consist of a wire within a pointed outer cannula, usually 0.1 and 0.3 mm in size, respectively. Size may vary, depending on area sampled. Special care must be taken to avoid disease transmission if using reusable needles. Surface electrodes are also used in some analyses and especially in NCV studies. Nerves are stimulated and their transmission monitored. The electrical signals are amplified and visualized on an oscilloscope and transformed to auditory signals heard through a loudspeaker. Normal muscle activity includes spontaneous and voluntary activities.[1]

Electrode insertion mechanically stimulates the muscle and produces a brief burst of **insertional activity** (Figure 2.1). Its duration in normal muscle should be short, less than 300 msec. The resting muscle should then return to its electrically silent baseline. When a patient feels a deep, burning, unpleasant sensation, the needle electrode is close to neuromuscular end plate, usually in the belly of the muscle. If it is in that position, **end-plate noise** can occur. This is a normal spontaneous activity. The end-plate noise consists of monophasic negative potentials of high frequency with amplitudes up to 50μV. **End plate spikes** may also appear. Their firing is irregular, with a frequency of up to 50 Hz. They are biphasic and usually present with end-plate noise.[2]

* Contributed by Maria Šoštarko.

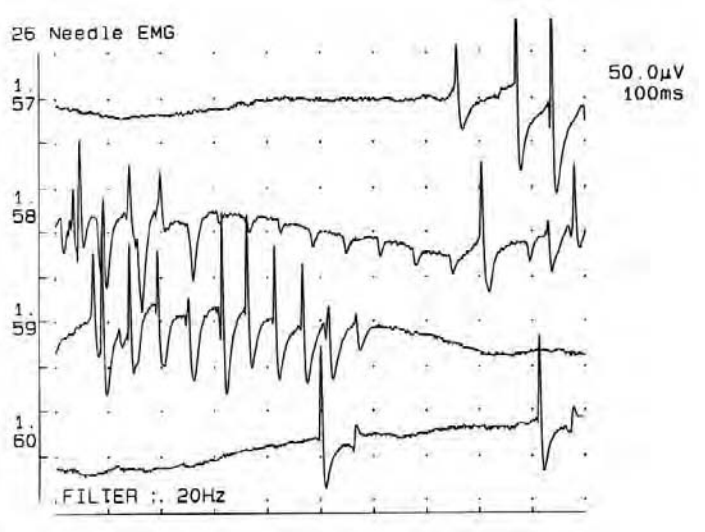

FIGURE 2.1 Insertion activity.

Voluntary muscle contraction produces a variable recording depending on the degree of contraction elicited. The recording should start with an electrically quiet muscle before increasing in noise as individual motor units are recruited and activity initiated.

Single motor unit potentials might be discerned early in stimulation. As more units are recruited, the pattern becomes denser, preventing differentiation. A full **interference pattern** is developed with maximal contractions (Figure 2.2). Motor units of healthy muscle produce biphasic or triphasic action potentials. If they have more phases, they are called polyphasic. Normal muscle might have up to 12% polyphasic action potentials. The **amplitudes** of normal action potentials range from 1 to 4mV, depending on the size of muscle fibers and motor units involved in contraction. Amplitude is measured as the distance between the negative and positive peaks. If the electrode is far from the muscle being studied, the amplitude and area of action potentials will be decreased, therefore, accurate electrode placement is necessary. **Action potentials** range from 3 to 16 msec in duration,

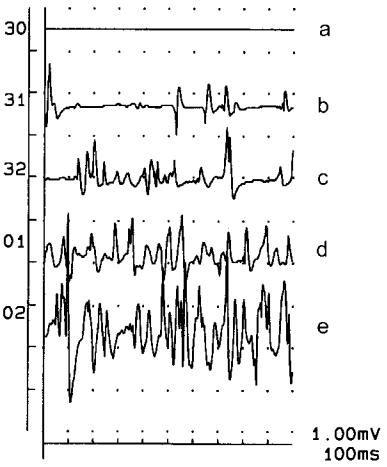

FIGURE 2.2 (a) Electrical silence, (b) single motor unit pattern, (c) partial intermediate muscle pattern, (d) intermediate muscle pattern, and (e) partial interference muscle pattern.

TABLE 2.1
Electrical Activity in Various States[6]

Activity state	Normal	Neurogenic lesion	Myogenic lesion
Rest	Quiet	Fibrillations, fasciculations, positive sharp waves	Quiet, crescendo–decrescendo trains
Mild effort	Individual unit potentials	Large, long-duration potentials	Small, polyphasic, short duration
Maximum effort	Full interference pattern	More large, long-duration potentials but not full pattern	Large number of small units easily recruited with weak contraction

with a frequency of 5 to 20 per second. The parameters of action potentials are slightly affected by voluntary muscle contraction to form a smooth curve. The innervated muscle pattern is positive in proportion to the degree of voluntary muscle contraction[1,3–5] (Table 2.1).

Abnormal Activity

A normal muscle is electrically quiet at rest, but denervating muscle will show fibrillations, positive sharp *w*aves and fasciculations or spontaneous firings of individual motor units (Figure 2.3). **Fibrillation potentials** occur in denervated muscle and are the result of a denervated fiber firing (Figure 2.3). They usually appear 2 to 3 weeks following denervation and slowly disappear as the lesion matures or the muscle is reinnervated. Their amplitude is 10–100μV, with duration of 1 to 5 msec and frequency of 1 to 30 per second. Occurring regularly, fibrillations are biphasic or triphasic and can be heard as a sharp clicking sound with no visible muscle contraction.[2–4]

Positive sharp waves also indicate denervated muscle cells; the cells, seeking nerve stimulation, alter their sarcoplasmatic reticulum, making them more sensitive. Their peaks are directed below the baseline (negative would be a peak above baseline, according to the definition in electromyography). Positive sharp waves are monophasic with high frequency. When compared with parameters of fibrillation potentials, their amplitudes are the same or higher and the duration is longer than fibrillations (Figure 2.3).

Complex repetitive discharges result from the depolarization of a single muscle fiber. They are generated by spontaneous activity of a single muscle fiber that activates one or more adjacent fibers. Frequency is 20-150 Hz, with abrupt onset and termination and can last up to 5 minutes in duration. They usually occur in trains of spikes and can be recorded in muscles with long lasting

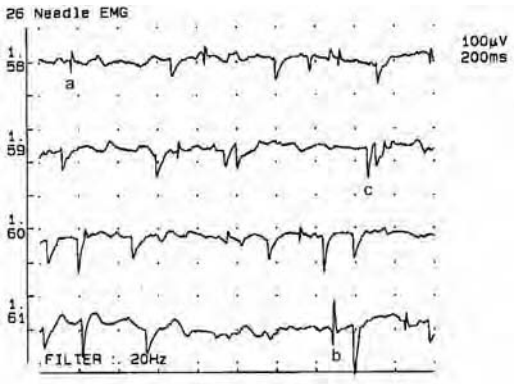

FIGURE 2.3 Spontaneous muscle activity: a) fibrillation, b) fasciculation, and c) positive sharp wave.

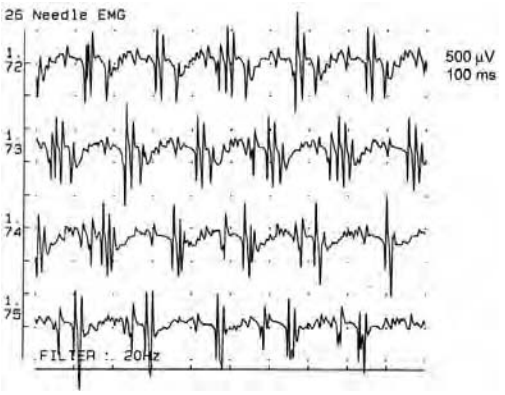

FIGURE 2.4 Complex repetitive discharges.

neurogenic atrophy or in myopathy, especially polymyositis (Figure 2.4). Patients may be unable to produce a full interference pattern.[2]

Fasciculations are involuntary firings of groups of muscle fibers or motor units. They can produce a visible movement in the form of brief irregular twitches. Amplitude varies from 200–800 µV with duration approximately 5–10 msec and frequency 0.1–10 Hz. Fasciculations have morphology of motor unit potential. Two types of fasciculations can be differentiated. **"Benign" fasciculations** are limited and there are no clinical signs or symptoms of lower motor neuron disorders. **"Malignant" fasciculations** are related to different disorders of lower motor neurons. They may rarely be present in some forms of myopathies. Their firings can produce visible muscle contraction[1,2] (Figure 2.3).

Myokimia is a clinical spontaneous phenomenon. It appears as an involuntary, irregular rippling contraction of a muscle segment, especially seen in fascial or periorbital muscles. EMG traces show bursts consisting of multiple spikes that originate from motor units induced by an abnormal excitation of a motor nerve fiber. Bursts last up to 1 minute in duration with the frequency of 3 to 73 Hz. Their firings can produce visible muscle contraction.[2,5]

A **myotonic discharge** is seen as a prolonged contraction on EMG, whether voluntarily or mechanically induced (Figure 2.5). The trains of spikes and positive waves wax and wane in frequency and amplitude, producing a thunderstorm sound over the loudspeaker during needle EMG. Typically seen in congenital and dystrophic myotonia, a myotonic discharge represents a high-frequency discharge that represents rapid firing of single motor fibers. The frequency may be as high as 150 per second. Electrical silence may exist after discharge. Blockade of motor nerve fibers may abolish the discharge.[7]

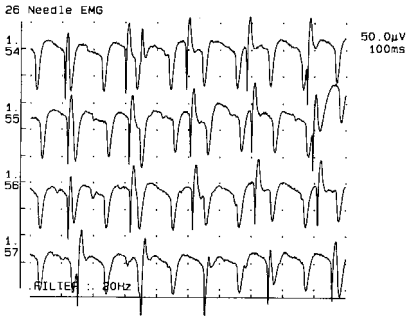

FIGURE 2.5 Spontaneous discharges in dystrophic myotonia.

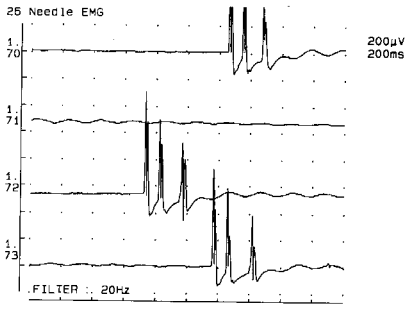

FIGURE 2.6 Spontaneous discharges in neuromyotonia.

Discharges in **neuromyotonia** are continuous activation of motor units sustained for variable periods. This muscle activity, present during sleep or barbiturate sedation, may produce a clinical finding similar to myokimia, but may also be so intense that it produces a fixed posture (Figure 2.6). The EMG pattern reveals motor unit potentials with high frequency 150–250 Hz. Discharges in neuromyotonia produce pinging sounds.[2,7]

Voluntary muscle patterns are equally affected and the cause can be neurogenic or myogenic. There is no voluntary muscle activity if a muscle is completely denervated. If it is partially denervated, the individual will be unable to produce a maximal contraction. Motor unit recruitment will be limited, and those that do fire will soon reach maximum frequency. If the nerve damage is severe, spontaneous activity will include fibrillations, fasciculations, positive sharp waves and complex repetitive discharges, which can be seen in long-lasting peripheral motor neurons injury. If the denervation is severe but not complete, EMG analysis will show single motor unit potentials with abortive potentials. **Reinnervation** produces classic low-amplitude, prolonged-duration, and polyphasic wave forms (Figure 2.7).[1,9,10]

Giant motor unit potentials develop as remaining healthy motor nerve fibers sprout to innervate a larger group of muscle fibers. This produces a larger motor unit called **space compensation** (Figure 2.8). As they are not tightly knit, the potential changes, becoming longer in duration, polyphasic, of higher frequency, and of a variable amplitude from 5 to 15 mV or even higher in chronic conditions and lower in acute situations.[5] These giant motor units require time to develop, and are not present in acute progressive motor neuron disorders. Their frequency is higher than normal and it is known as **temporary compensation**.[2,7] (Table 2.1)

Myopathic muscle produces a different EMG pattern (Figure 2.9). It is usually quiet at rest, but several fibrillations or fasciculation may be present. In its chronic stage, complex repetitive discharges

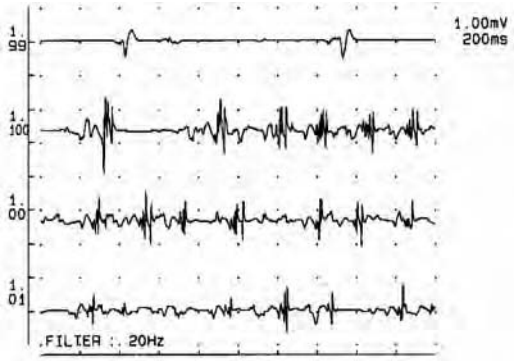

FIGURE 2.7 Reinnervation, muscle action potentials with low amplitude and increased polyphasis.

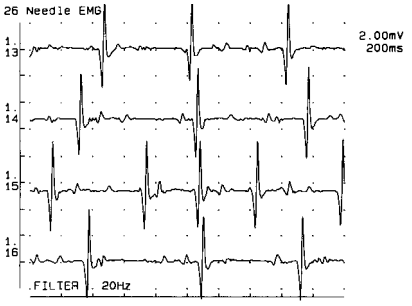

FIGURE 2.8 Lesion of lower motor neurons, giant potentials in very rarefied muscle pattern.

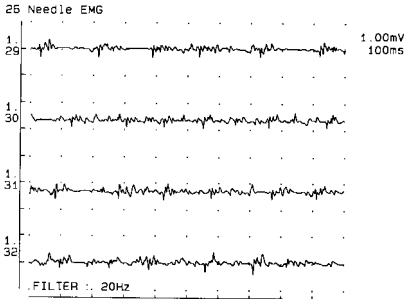

FIGURE 2.9 Myopathic muscle action potentials.

might be present as well. A high-density pattern is evident, but is the result of polyphasic potentials, not of a high number of potentials. Synchronization of action potentials is disturbed in the myopathic pattern; the result being polyphasic potentials. The myogenic potentials have low amplitude, a short duration, and a high frequency, producing a weak muscle contraction.[5,6] (Table 2.1)

NERVE CONDUCTION VELOCITY TESTING

Nerve conduction velocity (NCV) testing is a critical component of electrodiagnostic tests. Performed with surface electrodes, NCV tests are better tolerated and can be repeated to assess nerve injury and recovery. Occasionally, the recording electrode might be a needle. The test can analyze motor and sensory nerve fibers. Adult values are reached by 2 to 4 years of age, as the myelination process is complete.[5] Nerve conduction proceeds both orthodromically and antidromically and is affected by position of the stimulation and recording electrodes (Figure 2.10).[9–11]

Myelin insulates the nerve fibers and increases the conduction velocity of an individual nerve by allowing saltatory impulse conduction. This conduction allows the signal to jump between nodes rather than transverse the entire distance between nodes. This mode of transmission is much faster than conduction in unmyelinated fibers (Figure 2.11). With nerve compression, myelin is damaged and conduction slows down, as a signal cannot jump between nodes in this area. Multisegmental NCV analysis will define the area of damage (Figure 2.12).[8,12–15]

Analysis measures the **conduction time** (msec) between two known points (cm) to produce a velocity (m/sec). Time is measured as the difference of time elapsed between stimulation in two stimulation points and the appearance of the consecutive compound muscle action potential (CMAP) at the recording electrode. The parameters of the evoked potential also can be determined: amplitude, area, duration, form, and terminal latency (cm/msec). Amplitude of CMAP decreases with axonal loss. Its form is changed due to demyelination of the nerve fibers. Instead of a biphasic or tripahsic CMAP, it becomes a polyphasic CMAP (Figure 2.13).[15–18]

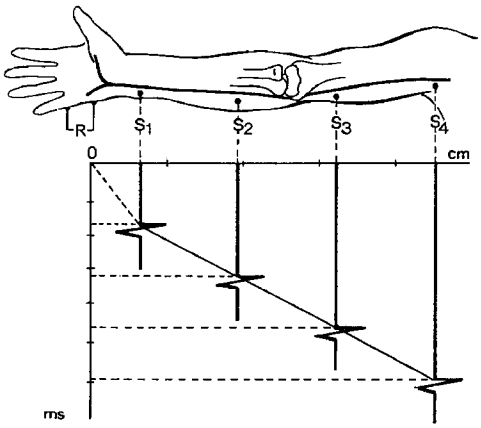

FIGURE 2.10 Scheme of the multisegmental analysis of the ulnar nerve. (R) recording electrode, (S_1, S_2, S_3, S_4) stimulation electrodes.

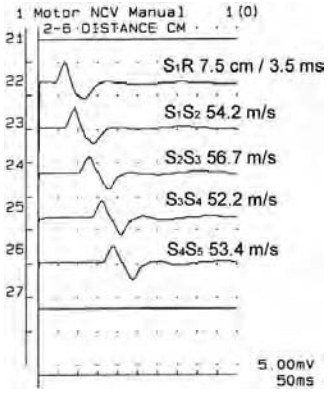

FIGURE 2.11 Normal result of multisegmental analysis of ulnar nerve motor conduction velocity.

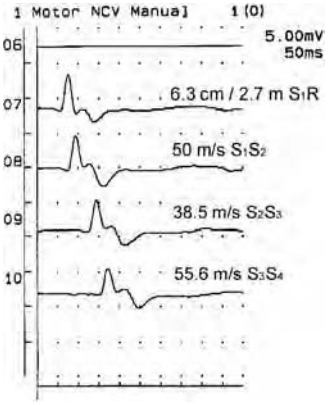

FIGURE 2.12 Ulnar groove syndrome. Decreasing of MCV in the elbow-segment where the ulnar nerve was compressed by connective tissue. Analysis of MCV performed five months after surgical decompression, improved M response.

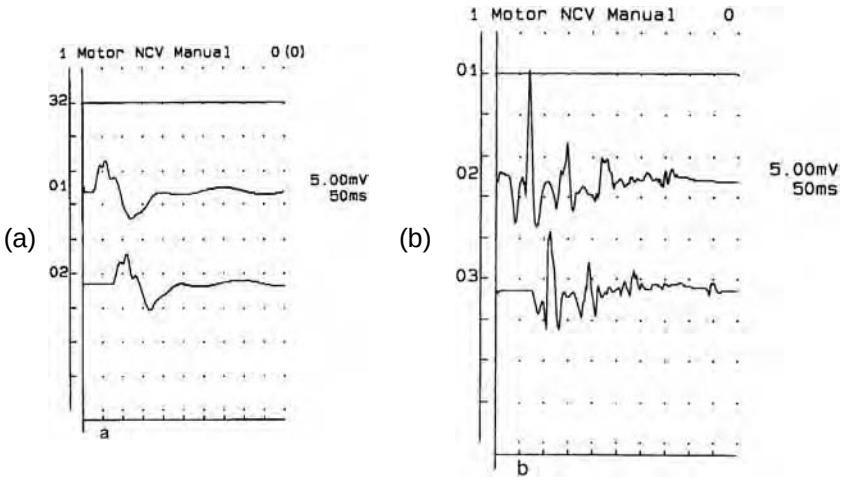

FIGURE 2.13 Compound muscle action potential (CMAP): (a) normal, (b) pathological change due to demyelination.

Typical conduction velocities are greater than 50 m/sec in the upper extremities and greater than 40 m/sec in the lower extremities (Table 2.2).[1]

The terminal latency index (TLI) is obtained by dividing the distance (cm) between the distal stimulation electrode and the recording electrode with time (msec). For example, the TLIs for median nerve and ulnar nerve are 1.9 ± 0.3 cm/msec. and 2.0 ± 0.4 cm/msec respectively. The TLI will drop below 2 cm/msec with nerve compression and start to return toward 2 cm/msec with nerve recovery. The actual measured distance is not constant, but the index normalizes the value for comparison (Table 2.3).[1] These values can be presented in different ways.[2,4,5]

The latency value and the conduction velocity of motor and sensory nerve fibers depend on nerve fiber conditions (Table 2.4).

Stimulation of demyelinated nerve fibers produces a polyphasic CMAP with reduced area and a prolonged terminal latency (Figure 2.13). Motor nerve fibers are stimulated orthodromically with the recording electrode in or over the appropriately innervated muscle. The kind and intensity of the peripheral myelinated nerve fibers dictate the value of the NCV (Table 2.4). A **neuropraxia** is a nerve injury affecting only the myelin sheath. It might be present in the early stage of entrapment syndromes. Segmental demyelination can be intersegmental or paranodal, which causes widening of Ranvier nodes. This develops after pressure on a peripheral nerve. **Conduction block**, which is reversible, is a neurophysiological manifestation of neuropraxia. Neurophysiological expression of a paranodal demyelination is conduction block. The main feature of a conduction block is a decrease of the CMAP amplitude or area obtained by proximal stimulation when compared with CMAP amplitude/area obtained by distal stimulation. There is no evidence of significant temporary dispersion. The distance between proximal and distal stimulation electrodes is recommended to be as short as possible. In the case of nuclear degeneration, as happens in motor neuron diseases, NCVs are in the normal ranges.[9,25]

There is a focal lesion of the peripheral nerve axons and myelin sheath while preserving the endoneurim, perineurium and epineurium in **axonotmesis**. Such a condition is called **Wallerian degeneration**. The axon degeneration involved axon and myelin distal to the focal injury and proximally up to the first node of Ranvier. Nerve conduction changes from saltatory to continuous nerve impulse transmission, which produces a decreased NCV (Table 2.5).[9,19]

Neurotmesis is the most severe stage of peripheral nerve injury. There is axonal loss with destruction of all supporting structures. The EMG shows signs of denervation with a severe loss of motor neurons. The compound muscle action potential (CMAP) cannot be evoked and improvement can be expected only after surgical treatment.[10,11]

TABLE 2.2
Nerve Conduction Values, Both Sensory and Motor, for Some Commonly Tested Nerves

Nerve	Motor/Sensory	Set Point	Velocity	Investigator
Radial	MCV	S2-S1	62.0 ± 5.1	Trojaborg and Sindrup, 1969[12]
		S3-S2	70.0 ± 4.9	
	SCV	S0-S1	58.0 ± 6.0	
		S1-S2	69.0 ± 5.7	
		S2-S3	71.0 ± 5.2	
Ulnar	MCV	S4-S3	60.0 ± 5.0	Jusĭc, 1981[1]
		S3-S2	51.0 ± 6.0	
		S2-S1	56.0 ± 6.0	
	SCV	S0-S1	61.4 ± 5.5	Buchthal and Rosenfalck, 1969[21]
		S1-S2	78.3 ± 5.5	
		S2-S3	58.0 ± 4.5	
Median	MCV	S4-S3	63.8 ± 6.5	Trojaborg, 1964[20]
		S3-S2	66.2 ± 6.1	
		S2-S1	56.1 ± 0.9	
	SCV	S0-S1	63.8 ± 6.5	Ludin, 1981[4]
	SCV	S0-palma manus	74.1 ± 3.0	Buchthal and Rosefalck, 1966[21]
		S1-palma manus	70.7 ± 3.7 (middle finger)	
Femoral	MCV	S2-S1	66.7 ± 7.4	Johnson et al., 1968[22]
Ischiadicus	MCV	S2-S1	52.9 ± 3.9	Yap and Hirota, 1967[23]
Tibial	MCV	S2-S1	51.8 ± 4.0	Behse and Buchthal, 1971[24]
	SCV	S0-S1	46.1 ± 3.5	
		S1-S2	58.6 ± 3.8	
Peroneal				
Deep	MCV	S2-S1	51.0 ± 2.8	Behse and Buchthal, 1971[24]
Superficialis	SCV	S0-S1	46.1 ± 4.1	
		S1-S2	55.9 ± 3.8	
		S2-S3	55.8 ± 4.7	
Suralis	SCV	S1-S2	51.2 ± 4.5	Behse and Buchthal, 1971[24]
		S2-S3	56.5 ± 3.4	

Note: MCV = motor conduction velocity, SCV = sensory conduction velocity.

TABLE 2.3
Comparison of Terminal Latency, Amplitude and MCV for Several Common Nerves

Nerve	TL (cm/msec)	Amplitude (mV)	MCV (m/sec)	Author
Ulnar	2.7 (1.5-4-1)	10.9 (6.0)	59 (49-66)	Lambert, 1962[26]
	2.7 (1.8-3.6)	10.5 (4.3-15)	58 (49-66)	Kaeser, 1970[14]
Median	3.4 (2.4-4.6)	9.6 (6.5-12.2)	58 (49-64)	Lambert, 1962[26]
	3.4 (2.0-4.5)	9.7 (4.4-14.5)	56 (49-68)	Kaeser, 1970[14]
Peroneal	4.9 (3.4-6.8)	7.7 (2.2)	51 (45-57)	Lambert, 1962[26]
Tibial	6.0 (4.0-7.5)	5.1 (2.0-9.2)	46 (36-58)	Kaeser, 1970[14]

TABLE 2.4
Proximal Latency and Distal Latency[6]

Disease, state	Proximal latency	Distal latency	Conduction velocity
Normal	Normal	Normal	Normal
Demyelinating neuropathy	Increased	Near normal	Decreased
Axonal neuropathy	Increased	Increased	Slightly decreased
Myopathy	Normal	Normal	Normal
Neuromuscular transmission defect	Normal	Normal	Normal

Note: Latency refers to the pause between impulse and muscle-fiber response affected by the distance between site of stimulation and the muscle

TABLE 2.5
Peripheral Neuropathies and Disorders of Nerve Conduction Velocities

Low normal to mildly slow NCV	Slow NCV
Acute motor axonal neuropathy (AMAN)	Acute inflammatory demyelinating polyneuropathy (AIDP)
Acute motor and sensory axonal neuropathy (AMSAN)	Acute sensory neuropathy or ganglioneuritis
Miller-Fischer syndrome (MFS)	Chronic inflammatory demyelinating polyneuropathy (CIDP)
Acute autonomic neuropathy	Demyelinating neuropathy associated with anti-MAG antibodies
Chronic inflammatory axonal polyneuropathy	Chronic inflammatory sensory neuropathy
Brachial plexitis	Paraneoplastic sensory neuropathy
Lumbosacral plexitis	Vasculitic neuropathy non systemic
Diabetic amyotrophy or lumbosacral plexopathy	Distal symmetric diabetic polyneuropathy
Distal symmetric polyneuropathy of renal disease	Mononeuritis. Mononeuritis multiplex.
Myeloma multiplex neuropathy	AIDS neuropathy
Disulfiram neuropathy	Lyme disease
Cisplatin neuropathy	Charcot-Marie-Tooth (CMT) neuropathy type I
Alcohol polyneuropathy	Cold induced neuropathy
Deficit B_{12}	Tunnel syndromes with focal decreasing of NCV
Over dosage of vitamin B_6 and E	Critical illness polyneuropathy
postirradiation neuropathy	
Idiopathic neuropathies	

Source: Adams, R. D. and Victor, M., *Principles of Neurology*, 5th ed., McGraw-Hill, New York, 1993, 1974. With permission.

Sensory nerve fibers are more sensitive to compression. A reduced **sensory nerve conduction velocity** (SNCV) test is the first sign of peripheral sensory nerve fiber demyelination. Sensory nerve fibers are stimulated orthodromically or antidromically. The surface electrode is used to stimulate sensory skin nerves and compound **sensory nerve-evoked potential** (SNEP) is picked up proximally over the nerve in the case of orthodromical stimulation. In the antidromic stimulation the surface electrode is placed over the analyzed nerve with the recording electrode placed distally. The sensory nerve-evoked potential with higher amplitude is usually obtained by antidromic technique. It is explained by the contribution of orthodromically stimulated motor nerve fibers. The obtained SNCV with orthodromic and antidromic technique has approximately the same value. Due to better and accurate analysis, SNEP averaging technique is used because SNEP has a low

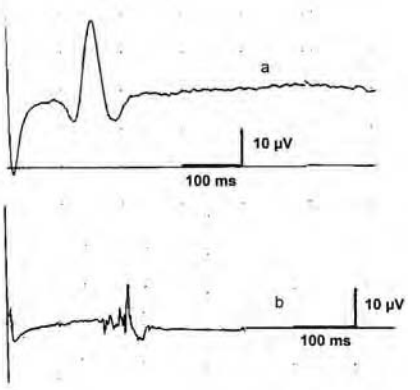

FIGURE 2.14 Sensory nerve potential: (a) normal; (b) pathological change due to demyelination.

amplitude, usually from several μV to 15 or 20μV. The sensory nerve-evoked potential is usually triphasic; and, in the case of demyelination, it becomes polyphasic (Figure 2.14).[27–30]

During the testing of sensory nerve fibers orthodromically in a sensomotor peripheral nerve, the motor nerve fibers are stimulated antidromically at the same time. The recording surface electrode is placed proximally and the stimulation electrode is placed distally over the nerve under investigation. A mixed nerve potential is evoked. The results help to differentiate entrapment syndromes from axonal degeneration. If the nerve is entrapped, the nerve potential becomes polyphasic and its amplitude is reduced (Figure 2.15). On the other hand, with axonal degeneration, the potential has normal parameters.[1,3]

Two other nerve tests help to detect proximal nerve compression near the root level. The **F wave** is recorded easily from the small muscles of the hands and feet during the motor conduction velocity study using supramaximal stimulation (Figure 2.16). It is used to measure the motor nerve conduction velocity of the proximal fibers. While there is a low incidence of F waves with peripheral compression, the presence of demyelination prolongs the F wave latency.[2,9] It may be also prolonged in carpal tunnel syndrome.[29] The **H reflex** looks at the integrity of the entire reflex arc, from the afferent fibers back to spinal cord and then back to the muscle (Figure 2.17). The H reflex can be obtained from testing the soleus muscle in the calf or the flexor muscles in the forearm. This testing allows evaluation of the S1, S2, and C7 roots.[2,9] The H reflex is also used in diagnosis of piriformis syndrome[32] and the assessment of motor neuron excitability.

If sensory disorders remain ambiguous, somatosensory testing may be necessary.[9] Peripheral nerves are stimulated, with the evoked potentials being recorded over the scalp, the cervical spine, or the lumbar spine. Averaging techniques are used to record potentials (see Color Figure 1). Demyelinated regions of the sensory pathways can be defined with this technique. If nerve injury is severe, somatosensory potentials may be absent or their latency may be prolonged. Color thermography can also be useful in diagnosis of entrapment syndrome (see Color Figure 1).[33]

PROBLEMS WITH ELECTROMYOGRAPHY INTERPRETATION

In spite of the valuable contribution of electrodiagnostic tests, errors in testing can affect their interpretation and the conclusions drawn from the tests. Thus, test results should always be compared with the clinical findings from a thorough history and physical examination. While gender has no effect, NCV can be affected by age, hand dominance,[15,25] extremity positioning,[31] lab abnormalities (as value of potassium, calcium, or magnesium) (Table 2.6), medications, drugs, environmental toxins, stimulation and recording sites selected, temperature, and cold extremities. A cold extremity or environment reduces muscle potentials and produces a rarefied pattern with

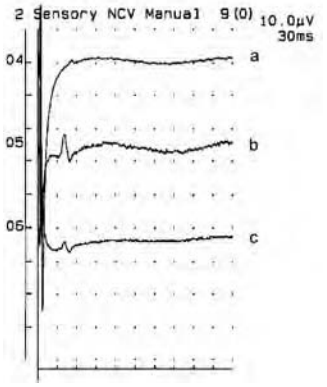

FIGURE 2.15 SNEPs in case of the right medial plantar nerve compression due to neurinoma that was found during surgery, (a) reduction of SNEP of the right medial plantar nerve with decreasing of SNCV, (b) normal SNEP of the left medial plantar nerve and SNCV was also normal, (c) normal SNEP of the right lateral plantar nerve with normal SNCV.

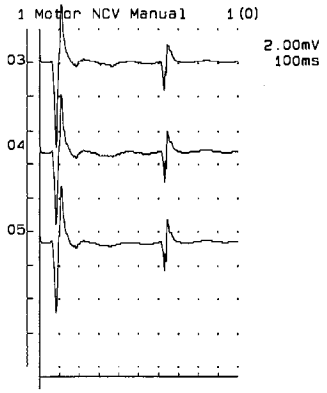

FIGURE 2.16 Normal F responses, recording extensor digitorum brevis muscle, nerve stimulation on the ankle joint level.

maximal muscle contraction. Additionally, CMAP and SNEP become more polyphasic with a reduction in area. Spontaneous electrical activity, such as fibrillations, and positive sharp waves decrease with temperature and disappear when room temperature falls below 20ºC. Aging reduces both MNCV and SNCV. Amplification also can be a source of error; its level should be recorded to allow correct interpretation of the evoked muscle activity.

The tested muscle should be documented, because muscle size alters potential parameters (i.e., the potentials for the orbicular oculi muscle would be pathological in the quadriceps femoris muscle). Needle shape and condition can affect formation of muscle potentials. Placement of the stimulation electrode is important, as distance affects nerve or muscle response. The applied stimulus must be supramaximal to avoid altering of the evoked CMAP or SNEP.

CLINICAL USE OF ELECTRODIAGNOSTIC TESTS

Electrodiagnostic tests are objective tests of peripheral nerves, muscles, and the neuromuscular junction. However, they should be performed following clear, precise histories and physicals. Many nerve compression syndromes have histories or physical findings, which are pathogenic for the syndrome. EMG use in these cases would be indicated only in specific situations — if there were

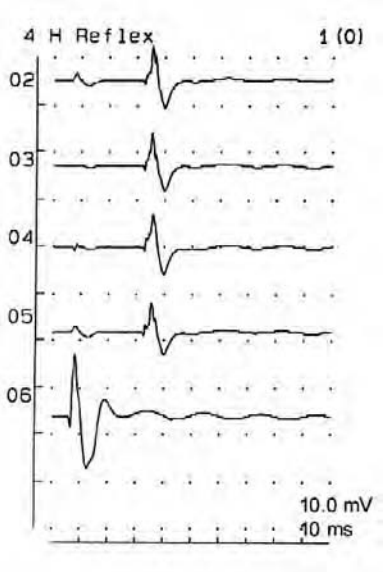

FIGURE 2.17 H reflex, recording soleus muscle, stimulating tibial nerve, with increased stimulation appearance of CMAP and H wave grows; at higher stimulation CMAP continues to grow but H wave diminishes.

TABLE 2.6
Laboratory Values and their Effect on the Electromyographic Data

Values measured	Effect
Potassium (low, high)	Weakness up to flaccid paralysis, decreased reflexes
Calcium	
Low	Increased irritability and spontaneous discharge, tetany, convulsions
High	Weakness, lethargy
Magnesium	
Low	Weakness, tremor, spasms
High	Weakness, confusion
Creatine kinase	Indicates muscle injury; check isoenzymes
Myoglobinuria	Indicates muscle injury

Source: Adams, R.D. and Victor, R.M., *Principle of Neurology*, 5th ed., McGraw-Hill, New York, 1993, 1065. With permission.

legal considerations; if patient represented a complicated Workman's Compensation case; or, if the surgery indicated was complicated, with significant risk if the diagnosis was incorrect. EMG tests in many peripheral nerve syndromes are reliable and accurate.

In the more ambiguous cases, electrodiagnostic testing should be supported by other neurophysiological and radiological tests or laboratory tests as indicated. More-proximal nerve problems may require F-wave and H-reflex testing. Ulnar and median nerve compression is reliably diagnosed by EMG and NCV testing, and EMG testing is critical in the diagnosis of deep palmar branch compression, where the differential would include initial phase amyotrophic lateral sclerosis (ALS). Saturday night palsies may be confused with a psychogenic palsy until EMG data is obtained. Anomalies in nerve supply, as in the "all ulnar hand" or "all median hand," are detectable only by EMG.

EMG and NCV tests are frequently used to define nerve injury and subsequent recovery. EMG progression can be used to define the point at which intervention becomes essential for a quality outcome. If nerve damage is severe, EMG analysis after 2 to 3 weeks will reveal signs of denervation — fibrillations, fasciculations, and positive sharp waves. If the nerve recovers, reinnervation potentials would appear during voluntary contraction (Figure 2.7), and giant action potentials can form as the nerve tries to compensate (Figure 2.8). If the nerve lesion persists, complex repetitive discharges may become obvious while the muscle is at rest (Figure 2.4). Overall, motor nerve conduction velocity will be reduced and terminal latency prolonged. The parameters will also change: amplitude will be reduced, duration will be prolonged, and polyphasic activity will be evident. If the sensory nerve fibers have been severely damaged, sensory nerve potentials may be absent, as sensory fibers are more sensitive than motor fibers.

Sensory nerve conduction velocity is especially helpful in carpal tunnel syndrome and ulnar nerve compression. Somatosensory-evoked potentials may be helpful in diagnosis of posterior and lateral femoral cutaneous nerve compression. In some cases, when a clinical neurophysiological study is not sufficient, NCV can be performed intraoperatively, with stimulation above and below the focal nerve injury. The result will define the presence of possible reinnervation and helps the surgeon to make a final decision about further treatment.

CONCLUSION

Electrodiagnostic tests are essential in diagnosing myogenic and neurogenic disorders as well as peripheral nerve compression syndromes; confirming diagnoses, finding compressive sites, clarifying etiologies, and documenting nerve recovery. However, they should not take precedence over a detailed history and physical examination. Patients tolerate nerve conduction velocity testing better than EMG analysis, so testing should be used with discrimination in the confusing cases, which do not provide clear diagnoses through standard clinical tests. Additionally, many syndromes remain ambiguous despite detailed electrodiagnostic work-up and require further observation and examination.

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Section II

Tunnel Syndromes in the Upper Extremities

3 Tunnel Syndromes in the Upper Extremities

INTRODUCTION

Media attention to nerve compression in the upper extremities has dramatically increased the number of patients presenting to physicians' offices. While multiple etiologies for tunnel syndromes exist, recent literature has emphasized occupation-induced compression. Losing considerable productivity to tunnel syndromes, employers have sought to use pre-employment screening and job assessments by physicians and occupational therapists to decrease their vulnerability to Workman's Compensation claims. Unfortunately, the long course of the nerves to the hands places them at risk in diverse locations and from various actions. This vulnerability complicates the physician's task of identifying the location and treating the compressive cause of the tunnel syndrome. Additionally, the compression may be unrelated to the patient's occupation.

To sort through the confusion, a coordinated approach to diagnosis and treatment is recommended. Office practice should consistently look for the major causes and contributors to nerve compression during the history and physical. Protocols with both occupational and physical therapists should be prearranged and then tailored to the individual case. Investigative tests should be conducted based on the information gleaned from the history and physical. Repeat examinations, including electromyographic studies or rheumatologic workups, may be helpful before proceeding to more-invasive modalities.

The list of potential etiologies for nerve compression in the upper extremity is long, and the causes may overlap. The physician must be able to isolate the primary etiology for the symptoms. Polyneuropathies and rheumatic disease can coexist, causing localized exacerbations that are amenable to direct treatment. Treatment of ulnar or medial nerve compression in the forearm without regard to possible proximal compression (i.e., cervical spine disease) could leave the patient without relief despite adequate decompression. Relief of occupation-induced nerve compression without modification of the work environment may be only a temporary solution. Functional impairment cannot be excluded, but should remain a diagnosis of exclusion.

Use of an ordered screening and testing process will help select those patients whose compressive symptoms represent true tunnel syndromes. The majority of these syndromes, when isolated, are amenable to either conservative or surgical modalities. Conservative treatment should be initiated first before considering surgical decompression; however, immediate surgical decompression may be necessary when confronted with signs and symptoms of prolonged nerve compression. Despite prompt and appropriate treatment, the outcome following nerve decompression may be varied. The following chapters address the screening, testing, and treatment of patients who present with complaints of nerve compression in the upper extremity.

4 Spinal Accessory Nerve Syndrome

The spinal accessory nerve (cranial XI nerve) can be compressed at multiple sites along its course from the base of the skull, along the lateral side of the neck, and to its termination in the region of the trapezius muscle. Nerve compression alters the function of the sternocleidomastoideus muscle and the trapezius muscle. Impaired muscle function alters scapular alignment, resulting in a more prominent inferior scapular tip. Patients present with weak shoulder elevation, scapular instability, and a decreased ability to smoothly elevate the arm.

ANATOMY

The spinal accessory nerve, a motor nerve, arises from two sources: the accessorius spinalis, whose fibers arise partly in the ganglion cells in the dorsolateral part of the ventral column of the cervical spinal cord (C1-C6), and the accessorius cranialis, whose fibers arise from the motor nucleus in the medulla oblongata. Emerging from the lateral surface of the spinal cord, the spinal roots pass between the ventral and dorsal roots of the C1-C6 and enter the cranial cavity behind the vertebral artery through the foramen magnum. They join the cranial roots, which leave the medulla oblongata inferior to the vagus nerve. The complete spinal accessory nerve leaves the cranium through the jugular foramen together with the glossopharyngeal nerve and the vagus nerve. As it passes through the foramen, the accessory nerve divides into the ramus internus (medialis) and the ramus externus. The ramus internus joins the vagus nerve. These fibers innervate muscles in the pharynx, larynx, and esophagus. Of more interest in this syndrome, the ramus externus, also called the spinal accessory nerve, crosses ventral (or occasionally dorsal) to the internal jugular vein behind the styloid muscles. As it passes along the deep surface of the sternocleidomastoideus muscle, the spinal accessory nerve branches. One branch pierces the muscle. The remainder of the nerve passes through the posterior triangle of the neck between the superficial and prevertebral fascia to reach the trapezius muscle (Figure 4.1). According to Pereira and Williams,[10] the nerve has a constant course on the deep surface of the trapezius muscle. This has clinical implications for surgery in the region of the trapezius. The ramus externus of the spinal accessory nerve communicates in a plexus with nerves from C2-C4. In the lateral triangle of the neck, the accessory nerve is adjacent to veins, branches of superficial cervical artery, and lymph nodes.

ETIOLOGY

Compression of the spinal accessory nerve at its issue from the cranial cavity may occur secondary to tumors of the skull base (a neuroma),[4] congenital anomalies of the craniovertebral junction, fractures at the base of the skull, fractures of the occipital condyle, and surgical intervention in this region such as endarterectomy.[13] In its course through the lateral triangle of the neck, the spinal accessory nerve could be compressed by blunt trauma, traction or surgical procedures such as lymph node biopsy[5,17]or radical neck dissection.[7,11] Trapezius muscle paralysis secondary to war wounds involving the accessory spinal nerve has been widely recognized.[17] Vandeweyer et al.[14] report a case in which a total transsection of the spinal accessory nerve was observed

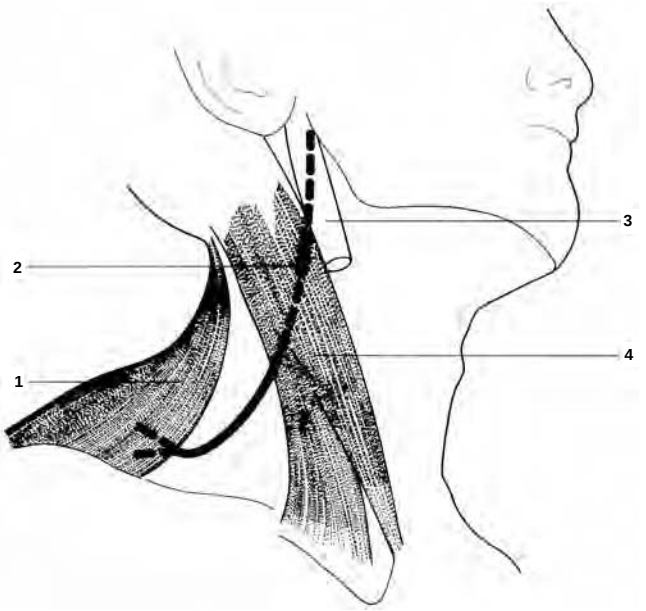

FIGURE 4.1 The spinal accessory nerve may be compressed at multiple sites along its course from the base of the skull along the lateral side of the neck to its endings in the region of trapezius muscle — (1) trapezius muscle; (2) spinal accessory nerve; (3) internal jugular vein; (4) sternocleidomastoideus muscle.

after a glass-penetrating injury. Bodack et al.[2] report spinal accessory nerve palsy as a cause of pain after a whiplash injury. Spontaneous spinal accessory nerve palsy was described by Mariani et al.,[6] who presented a case of isolated unilateral trapezius palsy with acute onset during athletic activity. Spontaneous palsies of unknown cause are very rare, with only seven cases reported.

CLINICAL SYMPTOMS AND SIGNS

Compression of the spinal accessory nerve presents with weakness of the trapezius muscle and the sternocleidomastoideus muscle. Loss of sternocleidomastoid muscle function is not of great practical importance. Tilting and turning of the head can be adequately performed by deep neck muscles. Loss of trapezius muscle function results in a clinically impressive picture. There is pain, deformity and disability.

The trapezius is an important shoulder elevator, but, when paralysed, the scapular alignment is altered. The most impressive clinical sign is a prominent inferior scapular tip. The vertebral margin of the scapula and the inferior scapular tip are no longer parallel to the vertebral column but are obliquely directed toward the mid-axillary line (Figure 4.2). The scapular position should not be mistaken for scapular winging, as seen with the long thoracic nerve palsy with loss of serratus anterior muscle action (Figure 4.3).

With trapezius muscle dysfunction, the patient presents with weakness of shoulder elevation, scapular instability, and weakness of lateral arm elevation (abduction). A positive abduction test results with flaring of the entire vertebral border of the scapula, which resembles a virtual dislocation of the scapula. When performing the test, the examiner holds the patient's wrist firmly at the side. The patient then attempts to abduct against resistance. Clinically, trapezius muscle weakness alters the normally gradual descending nuchal line between the neck and the shoulder. It is replaced with a brisk transition between the lateral neck contour and the horizontal. Patients lose the ability to shrug their shoulder on the involved side. They complain of a dull ache in the shoulder that often extends down the arm. This pain may be due to compensatory overuse of the working shoulder

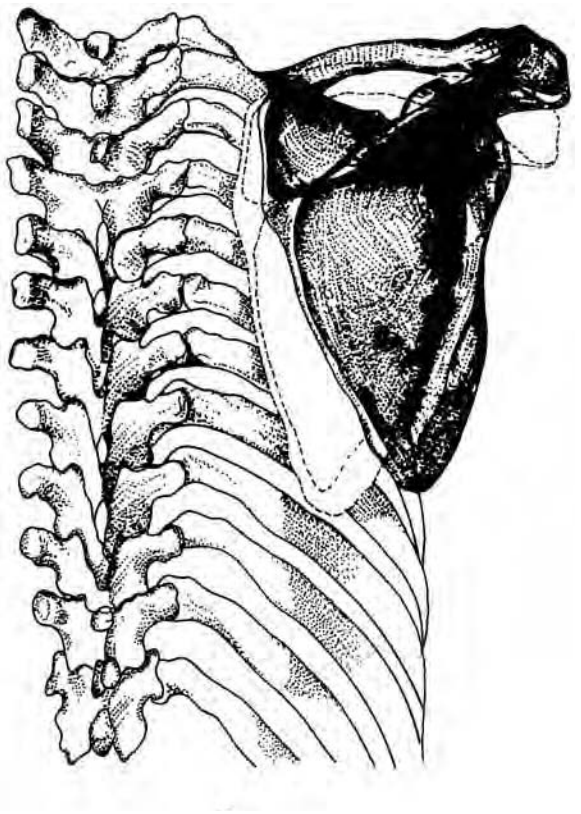

FIGURE 4.2 In spinal accessory paralysis (trapezius muscle) the vertebral margin of the scapula and the inferior scapular tip are obliquely directed toward the mid-axillary line.

girdle muscles. Sunderland[12] believed that traction on the brachial plexus by the drooped shoulder led to the ache. In posttraumatic or post-surgical patients, Bremner-Smith et al.[3] suspected that the pain might originate from the cut nerve itself.

Because of the weakness of the trapezius muscle and the disturbed biomechanics of the shoulder joint, secondary effects appear, including shoulder impingement, rotator cuff tendinitis and adhesive capsulitis. Secondary neuritis of cervical roots or the brachial plexus may also result. Electrodiagnostic investigations are helpful in localizing the site of nerve compression or injury.

TREATMENT

Following blunt trauma, treatment should include physical therapy to maintain shoulder motion. Conservative treatment is aimed at relaxing the paralyzed muscle by elevating the shoulder to prevent excessive stretching of the trapezius. This is most easily achieved by using a sling. The use of reeducating shoulder muscles will only partially compensate for a permanently paralyzed trapezius.[9] In injuries where serial EMG studies show no evidence of regeneration, neurolysis, nerve repair or nerve grafting are appropriate surgical options to consider.[8,15] In late cases, scapula stabilization, either statically or dynamically, can be performed. Static procedures include scapulothoracic fusion and tenodesis of the scapula with fascia to the spine. The results are unpredictable. The repair may stretch out over time. Dynamic transfers, a preferred approach, rearrange the muscle insertions on the scapula or involve tendon transfer. The levator scapulae, rhomboid minor and

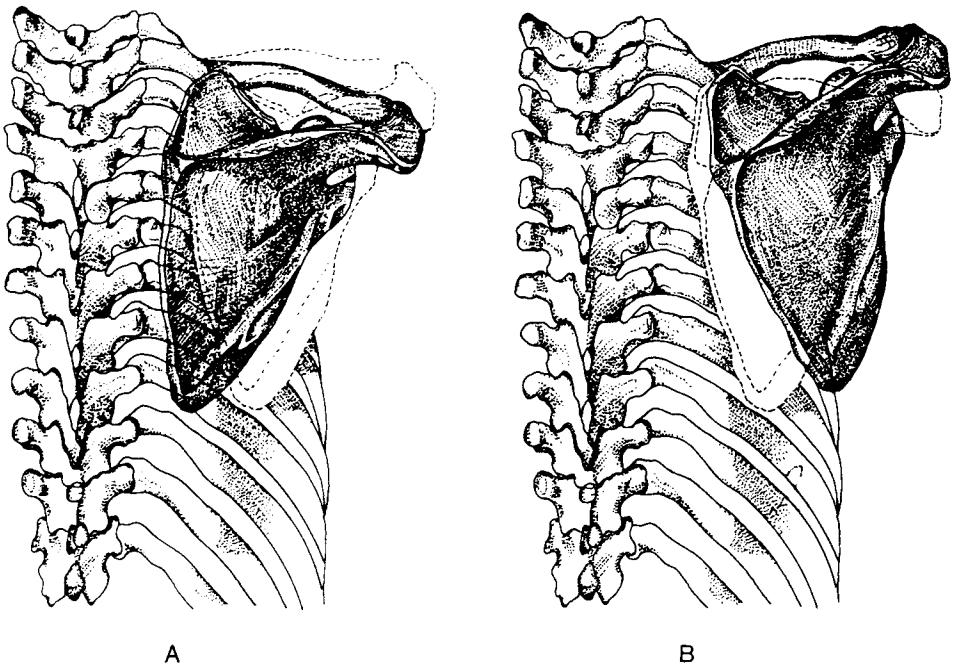

FIGURE 4.3 The type of scapular winging seen in (A) long thoracic nerve paralysis (serratus anterior muscle) and in (B) spinal accessory paralysis (trapezius muscle).

rhomboid major muscles are transferred laterally to substitute for the paralyzed trapezius muscle.[16] According to Bigliani,[1] it is possible to obtain nearly 90% good or excellent results, with improved function and diminished pain.

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5 Thoracic Outlet Syndrome

Thoracic outlet syndrome (TOS) has been investigated extensively to help accurately diagnose, evaluate etiologies, and expediently treat patients presenting with vague symptoms. Compression of the brachial plexus, the subclavian artery, or the subclavian vein before their division and separation, occurs in the area known as the thoracic outlet.[1] Upper-extremity dysfunction may result from upper-limb pain, paresthesias, vascular insufficiency, and motor dysfunction secondary to compression and can be described as TOS. Its clinical presentation varies depending on when and which neurovascular structures are compressed. The term was first used in 1956 by Peet et al.[2] Careful review of the literature reveals descriptions of similar syndromes: the **anterior scalene syndrome** by Adson and Coffey in 1927,[3] the **costoclavicular syndrome** by Falconer and Weddell in 1943,[4] and the **hyperabduction syndrome** by Wright in 1945.[5] The present standard is dependent not only on those authors noted in this chapter, but also on all those whose efforts have yielded this body of knowledge. For didactic reasons, this chapter will present the TOS in separate syndromes, as it has been described chronologically in medical literature.

The clinical diagnosis of TOS remains complex, requiring detailed history, physical examination, and careful selection of appropriate tests. The presence of secondary gain makes patient selection for surgery extremely important. Conservative treatment remains the mainstay of all care. Therefore, close work with a therapist is essential.[68]

The differential diagnosis includes cervical radiculopathy, supraclavicular fossa pathology, trauma, tumors (especially lung), brachial neuritis, distal compressive neuropathies, and complex regional pain syndrome type I (reflex sympathetic dystrophy).[66]

ANTERIOR SCALENE SYNDROME

The brachial plexus and the subclavian artery can be compressed as they pass between the anterior and medial scalene muscles and the first rib. This compression yields a characteristic neurovascular syndrome, the anterior scalene syndrome.

Anatomy

The three scalene muscles originate from the transverse processes of the cervical vertebrae and insert on the first and second ribs. The anterior and medial scalene muscles insert on their respective tubercles on the first rib, sandwiching the subclavian artery (Figure 5.1). The posterior scalenus muscle is fixed to the second rib. A variable scalenus minimus muscle may exist and insert between the anterior and medial scalenus muscles. The scalene muscles elevate the first and second rib during inspiration. Unilateral contraction inclines the head to the side of action and turns the face to the opposite side. Bilateral contraction flexes the cervical spine. The anterior and medial scalene muscles form one side of the posterior scalene foramen, with the sternocleidomastoid muscle and the first rib forming the other sides. Bounded by the anterior scalene muscle, the first rib, and the medial scalene muscle, the posterior scalene foramen admits the brachial plexus and the subclavian artery to the costoclavicular space. The posterior scalene foramen can range from 0.4 to 3.5cm in width.[6]

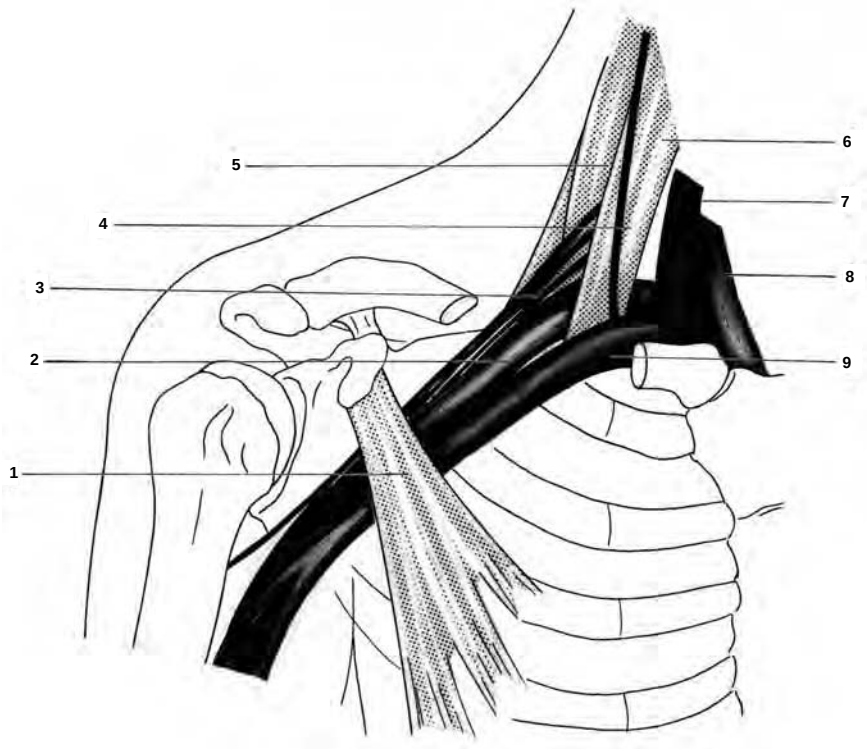

FIGURE 5.1 The region of the thoracic outlet where the brachial plexus lies exposed to bony, muscular, vascular, neoplastic, and traumatic compression — 1: pectoralis minor muscle; 2: subclavian artery; 3: brachial plexus; 4: anterior scalene muscle; 5: phrenic nerve; 6: anterior scalene muscle; 7: internal jugular vein; 8: common carotid artery; 9: subclavian vein.

The subclavian artery arches over the first rib and transverses the sulcus formed by the scalene muscles and first rib. The brachial plexus is composed of nerve roots from C5 to C8 and T1. The plexus may also receive contributions from the C4 (prefixed) or the T2 (postfixed) roots. It innervates the entire upper extremity and lies tautly stretched between the neck and shoulder without bony protection in this region.

Neurovascular compression can occur when disease or anatomical variations narrow this tight foramen. In the development of the anterior scalenus syndrome, anatomical variations are very important.[7] They are described below.

Etiology

Naffziger and Grant[11] and Ochsner, Gage, and Debakey[12,26] have published cases where the anterior scalenus muscle alone, without the existence of the cervical rib, is responsible for the compression of the neurovascular bundle with corresponding clinical symptoms. Komar[13] summarized literature reviews of anterior scalenus syndrome. The anterior scalene syndrome has many similarities to the costoclavicular syndrome, also known as the syndrome of the cervical rib, described by Wilshilre[8] in 1860 and Gruber[9] in 1869.

Normal anatomy provides enough room in the posterior scalene foramen for the brachial plexus and the subclavian artery. However, many anatomical variations and dynamic changes in the anatomy can cause narrowing, lowering the threshold for development of clinical symptoms.[14] Lord and Rosati[9] stress the many embryological, anatomical, and physiological factors that create a disposition for compression.

The insertions of the anterior and medial scalene muscles on the first rib may approach each other, thereby narrowing the sulcus. Fibrous bands may connect the anterior and posterior scalene muscles, producing a sling that elevates the brachial plexus and the subclavian artery over the first rib.[9] Some authors believe that even an unusually strong contraction of the anterior scalene muscle can profoundly elevate the first rib, further narrowing the foramen; however, in a series of hundreds of patients, despite sectioning of the anterior scalene muscle, Telford and Mottershead[27] found the first rib to still be a problem.

The roots of the brachial plexus and the subclavian artery are bent under tension over the first rib, due to the change in posture from that of a quadruped to an erect person.[15] A quadruped's thorax has its largest diameter in the anterior posterior dimension. A person's thorax has its largest diameter in the laterolateral dimension. The asymmetry of the thorax places a human's nerves and arteries in a position of tension.[16] Poor posture, prolonged work above one's head, prolonged wearing of a knapsack, or advanced age can produce a lowered or anteriorly rotated shoulder and further increase the distance the nerves and vessels must travel.[17–21] In adult women, the shoulder has a lower position in relation to the thorax than in men. Carrying heavy burdens on one's arms produces cervicobrachial traction that, when combined with increased respiratory exertion caused by work, results in high degrees of tension through the scalene foramen. Asymmetry of the foramen contributes to the unfavorable situation. The presence of a cervical rib or scalenus minimus muscle plays a role by either raising the floor of the foramen or narrowing the foramen in the anteroposterior dimension. The importance of scalenus muscle hypertrophy in narrowing of the foramen has been noted by Swank and Siomeone[22] and Frankel and Hirata.[23] Chronic vibratory trauma has also been implicated.[24,25]

The vascular symptoms in the anterior scalene syndrome are caused by tension of the artery or vein over the first rib.[28,29] Distal to the area of arterial compression or occlusion, one may find a post-stenotic dilatation. Vegetative nerve fibers are compressed at the same time as the neurovascular bundle.[24,25] These can produce the vague nerve complaints.

Clinical Symptoms and Signs

The neurovascular symptomatology depends on the frequency, duration, and degree of compression of the subclavian artery and the brachial plexus because of their location in the plexus. According to Komar,[13] the symptoms can be arranged by their causes into four groups:

1. Neurological dysfunction
2. Vascular compression
3. Different body postures
4. Functional and anatomical changes of the scalene foramen

The lower roots of the brachial plexus (C8-T1) are at higher risk of compression than the higher roots. The symptoms generally include: pain in the fingers, hand, forearm, arm, and even the shoulder; paresthesias, dysthesias or hyperesthesia (the C8-T1 dermatomes). Numbness appears more often in the fingers, hand, and forearm.

Depending on the degree of arterial compression, ischemic signs of numbness, cold, weakness and skin color changes appear. Gangrene and ulcerations of the fingers may develop in severe cases. Ischemic pain can resemble pain from nerve compression. Weakened grip and impaired finger function could be present.

Neurological symptoms corresponding to the compression of the inferior part of the brachial plexus (C8-T1) result in paresis and atrophy of the hypothenar and interossei muscles. Vascular symptoms are manifested as intermittent ischemic crises similar to Raynaud's phenomenon. Primary Raynaud's phenomenon can coexist with thoracic outlet syndrome.[31] Distal to the site of arterial compression can lie an aneurysm where thrombi may develop. Freed emboli can obliterate one of the terminal finger arteries, which is followed by severe pain.

Adson's sign represents a diagnostic test to elicit symptoms based on body posture.[32,33] The sign utilizes movements that stretch the anterior and medial scalene muscles and potentiate any neurovascular compression in the region of the first rib. The examiner evaluates the strength of the radial pulse in the hanging arm as the patient inspires deeply, extends the neck, and turns the head in both directions. Without prompting, a patient should indicate reproduction of symptoms. Since the pulse may weaken or disappear in normal subjects,[34] one must also examine other signs and perform other tests such as arteriography before proceeding to surgery. Arteriography, ultrasound, or auscultation might allow detection of subclavian artery compression during an Adson's test. With a return to normal posture, the pulse of a normal person with a positive Adson's test will return much quicker than that of a person with anterior scalene syndrome.[13]

French investigators describe an additional test, the signe des plateaux. The arm is abducted and placed parallel to the ground with the palm up. The radial pulse disappears when resistance is applied to the arm. When the patient's arm is supported in this position the pulse remains. Rather than depend on palpation, oscillography can be used. The presence of a cervical rib may be seen on plain radiographs. The relative value of electrophysiological studies in so-called neurogenic TOS has been stressed by many authors,[35–38] but remains uncertain.[65,66] Hypertrophied and taut anterior scalenus muscles and cervical ribs can be palpated in the supraclavicular region. While more than one test is available, none is absolute, thus initial conservative therapy remains mandatory.

Treatment

Treatment depends on the degree of subjective symptoms and objective signs. With mild symptoms or incomplete signs, conservative therapy should be applied even in cases with an identifiable cervical rib. Conservative treatment includes physical therapy, immobilization, ultrasound, and corticosteroid injections. Correct posture is taught. Shoulder loads such as handbags and briefcases should be minimized. Habits and workplace demands should be assessed and corrected if possible. Physical therapy seeks to increase tone in the shoulder muscles to decrease the tone in the cervical musculature. Additionally, conditioning is important. Immobilization is combined with physical therapy to keep the shoulder from continuing to drag between therapy sessions. Women with large breasts may need to use different bras or undergo surgery to reduce the breasts. Cervical pillows and collars may help patients with considerable spasm.[65] If symptoms become severe, surgical decompression consisting of scalenotomy or cervical rib resection[39] has a significant success rate.[40] Some authors advise combining scalenotomy, sympathectomy, and even first-rib resection.[41] To date, neither the best surgical procedure nor the best time for intervention has been found.[42] On the basis of long-term results of primary scalenotomy in 107 patients, Gockel et al.[43] recommended surgical treatment for younger patients with clear evidence of TOS. However, Leffert notes a high rate of recurrence if the anterior scalene muscle was not the sole compressive agent. Therefore, as he believes in a multifactorial compressive picture, Leffert recommends a combined approach of scalenectomy and first-rib resection. In the end, the surgeons' approach depends on their training and desired goals.

COSTOCLAVICULAR SYNDROME

Costoclavicular syndrome occurs with compression of the subclavian artery, subclavian vein, and brachial plexus as they pass between the clavicle and the first rib. Falconer and Weddell[4] describe this syndrome as separate from anterior scalene syndrome because of the vascular involvement.

Anatomy

The costoclavicular space, triangular in shape, connects the cervical spine with the upper extremity; therefore, it bears the name canalis cervicoaxillaris. The boundaries of this space are as follows:

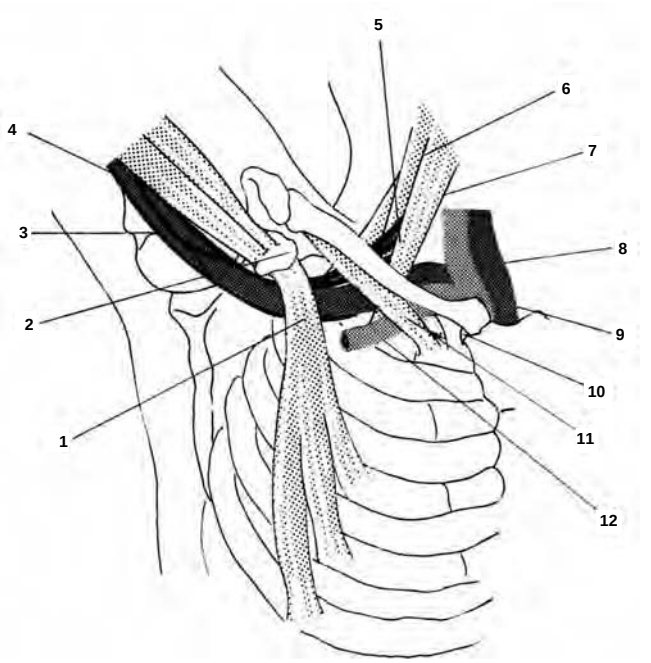

FIGURE 5.2 Dynamic anatomical variations of tunnels have been postulated as possible etiologies for various tunnel syndromes. This figure shows dynamic compression of the brachial plexus in the thoracic outlet — 1: pectoralis minor muscle; 2: coracoid process; 3: median nerve; 4: subclavian artery; 5: brachial plexus; 6: medial scalene muscle; 7: anterior scalene muscle; 8: internal jugular vein; 9: common carotid artery; 10: costoclavicular ligament; 11: subclavius muscle; 12: subclavian vein.

anteriorly, the medial third of the clavicle and the subclavius muscle; posterolaterally, the upper margin of the scapula; and posteromedially, the anterior third of the first rib and the insertions of the anterior and medial scalene muscles (Figure 5.2). The neurovascular bundle runs in the medial angle of this triangle. The subclavian vein lies medially in front of the anterior scalene muscle's insertion on the first rib and deep to the costoclavicular ligament and thickening of the clavipectoral fascia. The fascia extends from the coracoid process to the first rib (costocoracoid ligament). The subclavian artery briefly enters this space via the posterior scalene foramen to lie lateral to the subclavian vein. Passing between the anterior and medial scalenus muscles, the brachial plexus joins the vascular bundle in the costoclavicular space.

Etiology

When the costoclavicular space becomes narrowed by disease or dynamic compression, the neuromuscular structures are compromised.[44-46] Roos and Owens[47] described congenital anomalies associated with TOS. Abnormal anatomy such as congenital fibrous bands in the thoracic outlet predispose an individual for TOS following stress or injury.[48] Functional or dynamic anatomy predominates as an etiology for clinical disease.[49] The space may be narrowed by the following: arm elevation that rotates the clavicle posteriorly; deep inhalation that raises the first rib into the space, because the clavicle does not rise with inspiration; and trauma to the clavicle, first, rib, or retrosternal dislocation of the clavicle.[50,51] Leffert notes that muscle weakness (especially trapezius weakness), depression, obesity, and excessive large breasts can aggravate the symptoms.[66] Although Pollack found cervical ribs in up to 1% of the population (50% bilateral) the presence of a rib does not confirm the diagnosis.

Clinical Symptoms and Signs

Patients with costoclavicular syndrome present with subjective complaints similar to those from patients with anterior scalenus syndrome. While the neurological complaints of pain, paresthesia, and hyperesthesia dominate in the anterior scalenus syndrome, vascular symptoms dominate in the costoclavicular syndrome.[52] Vein compression leads to temporary or permanent edema. The radial artery pulse is evaluated when the patient thrusts his chest forward and pulls the shoulders posteriorly and interiorly. Typically, the pulse weakens or disappears. Measurement using an oscilloscope can verify these changes; however, pulse damping can occur in controls.[53]Komar[13] recommended arteriography to evaluate the changes in flow between positions. Venography and venous pressure measurements may aid in evaluation. Ultrasonography, computed tomography (CT), and magnetic resonance imaging[54] are also useful diagnostic tools.

Treatment

Conservative treatment consisting of physical therapy and temporary immobilization may be tried. Nakatsuchi et al.[55] describe a strapping device for elevation of the shoulder in patients with TOS. If the etiology can be identified, whether it be a pseudoarthrosis of the clavicle, an exuberant callus, or an impinging first rib, surgical decompression and treatment are warranted. Results from surgery vary from 24 to 100% successful outcomes. It is recommended that conservative therapies be tried before surgery is considered.[56] In the operative treatment of TOS, a variety of approaches have been recommended: a supraclavicular approach is used by Cheng and Stoney,[57] a combined scalenectomy and transaxillary first rib resection is used Cina et al.,[58] and Cuypers et al.[59] use only a transaxillary first rib resection. None of these surgeries is risk free. Parziale et al. noted that 21% of malpractice claims against thoracic surgeons involved first-rib resections.[65]

HYPERABDUCTION SYNDROME

Repetitive or prolonged hyperabduction of the arm stretches the neurovascular bundle under the pectoralis minor tendon and the coracoid process. The resultant symptoms of neurovascular compression known as the hyperabduction syndrome was first described by Wright in 1945.[5]

Anatomy

Leaving the costoclavicular space, the three cords of the brachial plexus, the subclavian artery, and the subclavian vein pass under the insertion of the pectoralis minor muscle on the coracoid process. As this neurovascular bundle enters the axillary fossa, the artery and vein become known as the axillary artery and the axillary vein (Figures 5.1 and 5.2). Abduction to 180 degrees stretches the neurovascular bundle around a fulcrum consisting of the pectoralis minor tendon, the coracoid process, and the humeral head. The bundle may reach a 90-degree angle. The neurovascular bundle remains fixed, allowing relatively no motion. The bundle can compensate only by stretching, producing an increased tension along its components. Abduction of the arm narrows by producing 30 degrees of elevation and 35 degrees of posterior displacement of the clavicle, thereby narrowing the costoclavicular tunnel. The tunnel's anterior wall, consisting also of the pectoralis minor muscle, the subclavius muscle, and the costoclavicular ligament (the thickening of the clavipectoral fascia), is stretched and further brought posteriorly, pushing the neurovascular bundle against the fulcrum.

Etiology

Wright[5] describes two critical anatomical points where compression of the neurovascular bundle might occur with the arm in hyperabduction: the first, while passing through the costoclavicular tunnel on slit; and the second, while passing under the pectoralis minor tendon at its insertion on

the coracoid process. During abduction of the arm, the fixed neurovascular bundle can be compressed by the pectoralis minor tendon as well as by the humeral head.[60] The characteristic position for testing is described as 180 degrees of shoulder abduction and elbow flexion. This test reproduces common sleep positions or functional positions of electricians, painters, bricklayers, or masons. Spinner et al.[61] describe a new cause of ulnar nerve compression by the chondroepitrochlearis muscle, arising from the pectoralis major and crossing over the neurovascular bundle in the axilla.

Clinical Symptoms and Signs

Pain, paresthesia, and numbness develop first in the fingers and later in the hand. In some patients, transitory ischemia and edema develop, resembling Raynaud's disease, which has been described by Beyer and Wright[62] in 38% of patients with hyperabduction syndrome. Neurological deficits are usually absent. As paresthesias and pain develop, patients correct their arm position, limiting the duration of nerve compression.

If the arm is abducted to 90 degrees and externally rotated in patients with hyperabduction syndrome, the subjective symptoms can increase, while the radial artery pulse may weaken or disappear. The sensitivity of this test, Wright's maneuver, can be increased by the patient's holding a deep breath.[66] Additionally, further abduction (hyper-) can be added. However, similar to the anterior scalene syndrome or the costoclavicular syndrome, tests can be positive in normal patients. Additionally, while the Adson's test may be positive, Youman and Smiley[63] described the occurrence of TOS with negative Adson's and hyperabduction maneuvers. Strauer and Rastan[64] proposed venography, arteriography, and intra-arterial pressure measurements to accurately assess positional variations in any arm's vascular status. The overhead test is positive when patients hold their arms at full elevation (180 degrees) and open and close their hands repetitively. Symptom reproduction (cramping, fatigue, and numbness) within 30 seconds is significant.[66]

Treatment

Treatment consists of avoiding hyperabduction. This may necessitate changes at work or home. Operative therapy is infrequently indicated; but, if necessary, the pectoralis minor tendon is sectioned at surgery.

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6 Scapulocostal Syndrome

In the shoulder region, especially along the medial border of the scapula, pain may develop and radiate into the neck, the brachium, and eventually the thorax, where it can be mistaken for angina pectoralis. Paresthesia and subjective weakness may complete the symptoms of this syndrome. Michele et al.[1] in 1950 coined the term scapulocostal syndrome; Moseley[2] and Steindler and Marxer[3] in the 1940s described symptoms inherent to it.

ANATOMY

The scapula, the focal point for upper-extremity function, serves as the origin or insertion of no less than 15 muscles and six ligaments that allow man the intricate as well as general functions of the arm. The scapula sits with its superior and inferior angle level with the second and seventh thoracic vertebrae, respectively. Underneath lie the posterior rami of thoracic nerves two through seven, the erector spinae muscles (spinalis, longissimus, iliocostalis), the serratus posterior and superior muscles, and the thoracolumbar fascia. The levator scapulae, major and minor rhomboid, trapezius, serratus anterior, and pectoralis minor muscles all insert on portions of the scapula. These muscles, with the assistance of several ligaments, suspend the scapula and position it in space to allow function. The shoulder gives rise to the deltoid, teres minor and major, biceps, coracobrachialis, supraspinatus, infraspinatus, subscapularis, and triceps muscles. These muscles suspend the upper extremity from the nearly vertical glenohumeral joint with the assistance of the glenohumeral joint. While all these muscles and ligaments must function simultaneously to allow mobility, dysfunction of even one can hamper activity and possibly set up a cycle of pain.

ETIOLOGY

The exact etiology of the scapulocostal syndrome remains unknown, although many hypotheses exist. Russek[4] holds that one major factor is poor posture resulting from one of the following sources: idiopathic poor posture, anatomical or functional deformity secondary to neck or shoulder injury, and static anatomic predilection. Poor posture places the scapula at an unfavorable angle with the chest wall, a situation that could produce dysfunction or pain. Some professions, such as stenography, truck driving, and surgery, place the individual into contorted positions. Michele et al.[1] and McGovney[5] explain the syndrome in light of a segmental reflex mechanism. They propose that nerves from the cervical roots pass through the prevertebral fascia and may be irritated by muscular spasm or fibromyositis. Suspended from and lying in proximity to the cervical spine, the scapula and its associated musculature receive the segmental irritations. Scapulocostal syndrome can develop secondary to trauma or prolonged immobilization, both of which weaken the muscular girdle. The shoulder adjusts with compensatory measures, a process that, over time, leads to dysfunctional motion. Russek[4] also identified shoulder subluxation, humeral fractures, and rotator cuff injuries as predisposing factors to scapulocostal syndrome development. Shull[6] believes the syndrome to be the result of various pathological factors. Localized pain at the posterolateral segments of the ribs as a consequence of the stress fractures of the ribs[7] can be seen as a

scapulocostal syndrome. Ormandy[9] studied 440 patients ranging from 18 to 60 years of age whose altered posture caused deep pain in the shoulder region originating from the medial aspect of the scapular spine. According to Ormandy, the scapulocostal syndrome, myofascitis of the shoulder muscles is caused by altered posture, prolonged immobilization of the shoulder region, or fixed scapular or spinal deformities. Menachem et al.[10] described 22 patients, all young females, with levator scapulae syndrome, presenting with a common clinical picture of pain over the upper medial angle of the scapula.

CLINICAL SYMPTOMS AND SIGNS

In light of the proposed etiologies, which center on the cervical spine and the shoulder girdle, one can understand that patients will present with pain in the neck, upper arm, and chest. The pain and its pattern of radiation can be explained by shoulder-girdle spasm. Pain tends to culminate toward evening. It is not aggravated by arm or shoulder motion. While patients may complain of paresthesia or muscle weakness, examination reveals neither sensory deficit, motor paralysis, nor atrophy. Physical examination may reveal a localized point on the medial scapular border where pressure reproduces the patient's pain.[8] Russek[4] describes another sign: scapular elevation off the chest wall with a forward stretch. In differential diagnosis, the painful scapulothoracic crepitus and scapulothoracic bursitis must be take into consideration.[11]

TREATMENT

Treatment should address the basis of the scapulocostal syndrome; therefore, the circular pattern of spasm and pain can be treated with infiltration of trigger zones with anesthetic followed by physical therapy to decrease spasm. Shull[6] had good results from cooling the trigger zones. Posture can be modified by physical therapy or surgical correction of gross deformity.

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7 Suprascapular Nerve Syndrome

The suprascapular nerve courses through the incisura scapulae (suprascapular notch) bounded by the sharp margins of the scapula and the transverse scapular ligament, and then the nerve courses around the scapular spine, passing through a fibro-osseous tunnel formed by the spinoglenoid ligament and the spine of the scapula. Compression or stretching of the nerve results in the development of suprascapular nerve syndrome, as first described by Andre Thomas in 1936.[1] According to Romeo et al.[57] the suprascapular nerve is vulnerable to compression at the suprascapular notch as well as at the spinoglenoid notch.

ANATOMY

The suprascapular nerve is a mixed motor and sensory peripheral nerve originating from either the C5 or C6 nerve roots of the upper (superior) trunk of the brachial plexus. The nerve passes in a superoposterior fashion through the supraclavicular fossa and then the scapular notch (incisura scapulae) to reach the supraspinatus fossa.The suprascapular vessels cross above the ligament rather than running with the nerve through the notch. The transverse scapular ligament forms a strong, sometimes ossified, bridge over the notch and nerve. Occasionally, veins or even a branch of the suprascapular artery may run through the notch or foramen. At the incisura scapulae, the suprascapular nerve sends branches to the supraspinatus muscle, the acromioclavicular joint and bursa, and the subacromial bursa. The shoulder joint receives branches from the bursa branches and the nerve itself. While most anatomical studies have not identified any cutaneous innervation, Ajmani[60] found a cutaneous branch of the suprascapular nerve supplying the proximal-lateral one third of the arm. The notch can occasionally be replaced by a foramen, the foramen scapulae. This foramen can become stenotic and squeeze the nerve.

Accompanied by vessels in the supraspinatus fossa, the nerve passes through the muscle, sending at least one branch to the muscle. The neurovascular bundle bends around the base of the scapular spine and enters the infraspinatus fossa. In approximately 30% of individuals, the nerve may make a 90-degree turn, run along the scapular spine in the fossa, and give off three branches (Figure 7.1). To complicate matters, a connective tissue band, the ligamentum spinoglenoidale, may exist in up to 50% of people, creating a second fibro-osseous tunnel (spinoglenoid notch) for the nerve to transverse, and terminating in two, three, or four motor branches that supply the infraspinatus muscle.

The suprascapular nerve stretches as the scapula moves. Since the nerve passes around rigid structures, functional anatomy plays a major role in understanding compression. Strong and sudden motions, including protraction and abduction, pull the nerve against the medial wall of the scapular notch or the ligament. External humeral rotation pulls the nerve against the lateral margin of the notch as the infraspinatus muscle interferes with its freedom of motion. Elevation and rotation of the arm also stretch the nerve.

ETIOLOGY

As the scapula moves over the rib cage to optimize its functional position, the suprascapular nerve must adapt or be injured by repetitive stretches. Any disease process that limits its flexibility or

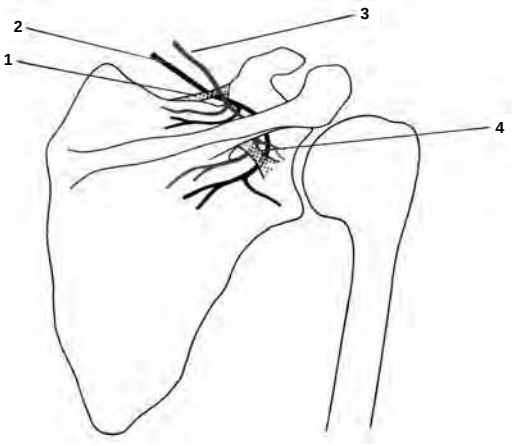


FIGURE 7.1 The suprascapular notch and the spinoglenoid notch with the pertinent anatomical relationships of the scapula and ligament to the neurovascular bundle —1: transverse scapular ligament; 2: suprascapular nerve; 3: suprascapular artery; 4: spinoglenoid ligament.

interferes with its route will place the nerve at risk. Few other nerves are placed in such a position of constant motion that requires an unobstructed course. Fractures of the scapula, including the scapular foramen,[2] may cause direct nerve compression. Bony or soft tissue injuries in the shoulder can alter the muscle tensions and bony relations enough to distort the nerve's course and lead to compression at the foramen. Chronic mechanical irritation due to repetitive nerve compression from stretching can occur, especially in individuals who do overhead work or place their arms into extremes of abduction and external rotation.

Although sleeping with one's arms extended above the head may produce symptoms, the syndrome has been more frequently described in painters, electricians, athletes (especially in volleyball and tennis players, weightlifters, and boxers),[3–8] dancers,[9] and patients in cardiac rehabilitation programs.[10] Ferretti et al.[11] described 12 volleyball players with asymptomatic isolated paralysis of the infraspinatus muscle on their dominant side. These findings were attributed to the repeated stress of the cocking of the arm and follow-through when the athlete serves.

Ganglion cysts (Figure 7.2) causing suprascapular nerve syndrome have been described by many authors.[12–17,52,55,57] Shoulder pain from a variety of etiologies[18] creates a vicious circle of muscle decompensation and altered scapular function that places the suprascapular nerve at risk for compression at the notch. However, Sjostrom and Mjoberg[19] described suprascapular nerve entrapment in an arthrodesed shoulder. Heuss et al.[20] describe an endogenous bilateral compression syndrome of the suprascapular nerve. Asami et al.[58] describe bilateral suprascapular nerve entrapment syndrome associated with rotator cuff tear. Padfua et al.[51] report six cases of a suprascapular nerve lesion: three of them presented a nerve entrapment at the suprascapular notch and three at the spinoglenoid notch.

CLINICAL SYMPTOMS AND SIGNS

Diagnosis of the syndrome of the suprascapular nerve remains difficult and many times unrecognized because the predominant symptom of pain is common to many other disorders. Shoulder pain can come from such varied etiologies as rotator cuff tears, cervical spine disease, scapulocostal syndrome, painful atrophy of shoulder muscles (Personage-Turner's syndrome), and musculoskeletal strain; therefore, the physician must continuously reevaluate the differential diagnosis in light of the patient's history, physical, diagnostic tests, and response to treatment.[21–41,59]

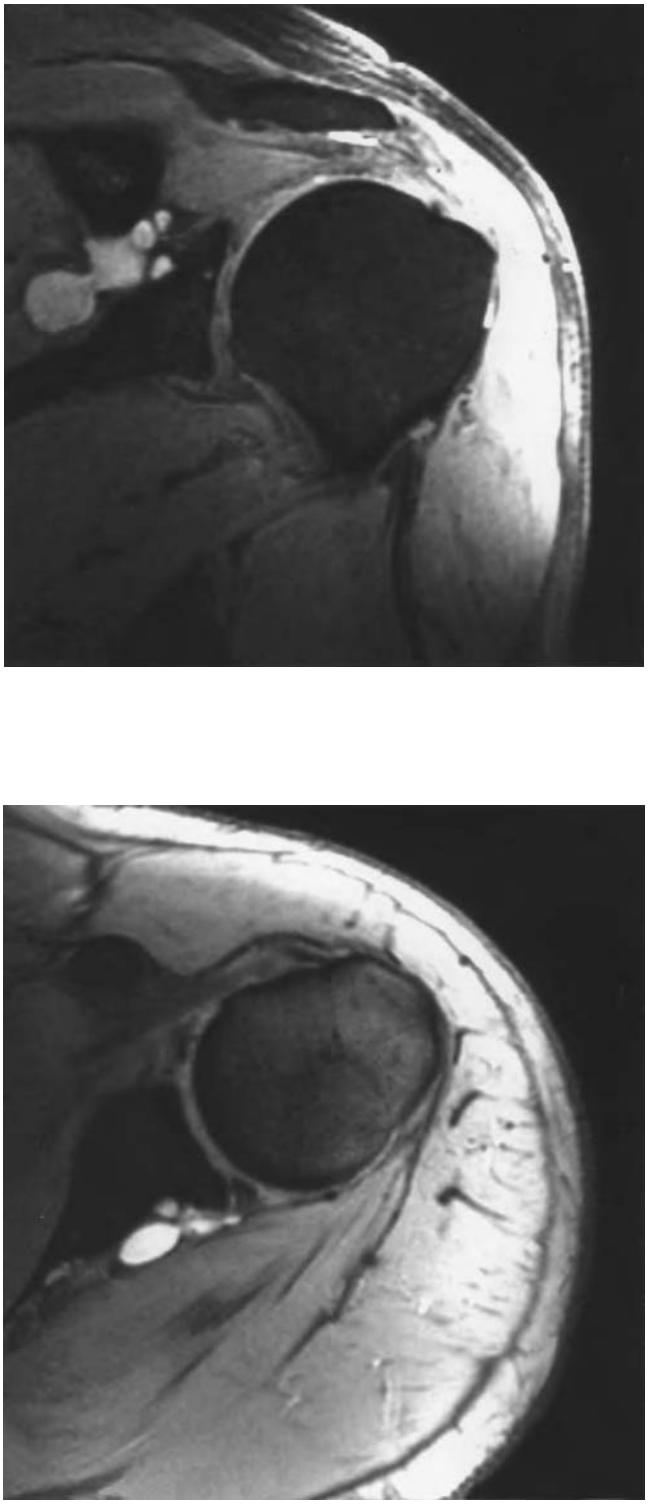


FIGURE 7.2 (top and below) MRI of suprascapular ganglion cyst.

Clinically, the pain does not localize to a specific portion of the shoulder. The pain can range from deep and blunt to sharp with radiation down the distribution of the radial or suprascapular nerves, depending on which shoulder motion elicits pain. The suprascapular and radial nerve have common root origins, so painful stimulation may overlap. Pain is more commonly appreciated over the posterior portion of the shoulder along the border of the trapezius muscle, with pain worsening at night. Once the nerve is damaged, stretching it during daily activities that are as simple as shaving or combing one's hair may aggravate the pain.

Clinical examination reveals a painful spot along the lateral third of the upper border of the trapezius muscle over the scapular notch. The remainder of the shoulder is possibly not painful to pressure. Active and passive range-of-motion exercises will create tension and reproduce the patient's pain. The crossed abduction test seeks to elicit the patient's pain by placing the arm of the painful side on the healthy shoulder, then pulling the elbow along the horizontal toward the healthy shoulder.[30] This will accentuate the nerve's compression in the scapula's notch.

Within 3 to 4 months from the onset of pain, motor symptoms such as supraspinatus and infraspinatus hypotrophy may develop (see Figure 7.3). Henlin et al.[42] report seven clinical and electromyographical cases of pure infraspinatus muscle paralysis. Liveson et al.[43] report three cases of suprascapular nerve lesions at the spinoglenoid notch. Compensation by other muscles in the shoulder girdle covers the loss of strength in abduction and rotation. Esslen et al.[25] found teres minor hypertrophy. Pain in the acromioclavicular joint and reduced sensitivity to vibration in the same region complete the clinical picture of the syndrome. Difficult to access for electrodiagnostic studies, the suprascapular nerve, when compressed, produces electromyographic changes of prolonged latency to the infraspinatus and supraspinatus muscles.[43,54] According to Post,[56] the diagnosis is made by a combined process of exclusion and abnormal electrodiagnostic findings. Today, magnetic resonance imaging[13,45–48,57] and ultrasonography[13,16] are useful diagnostic tools in the diagnosis of suprascapular nerve syndrome.

(A)

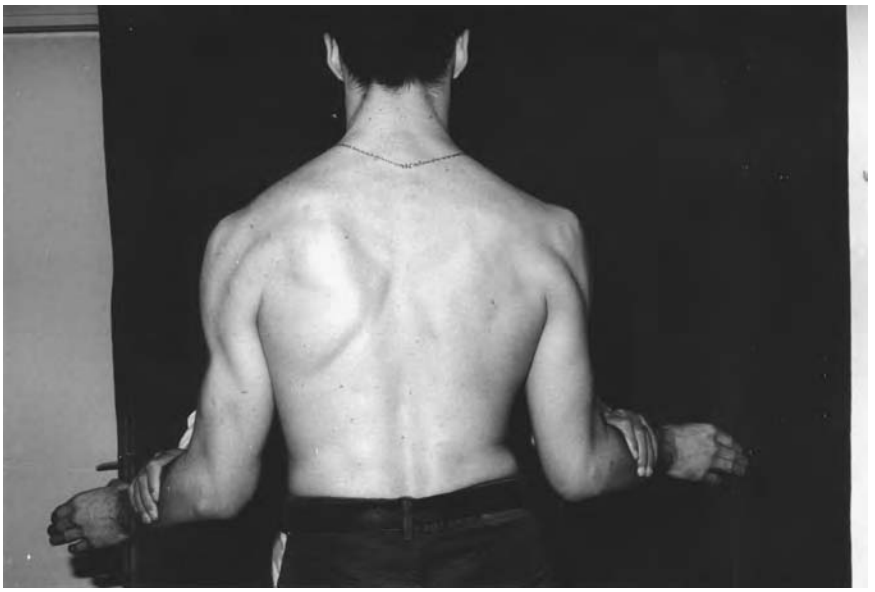
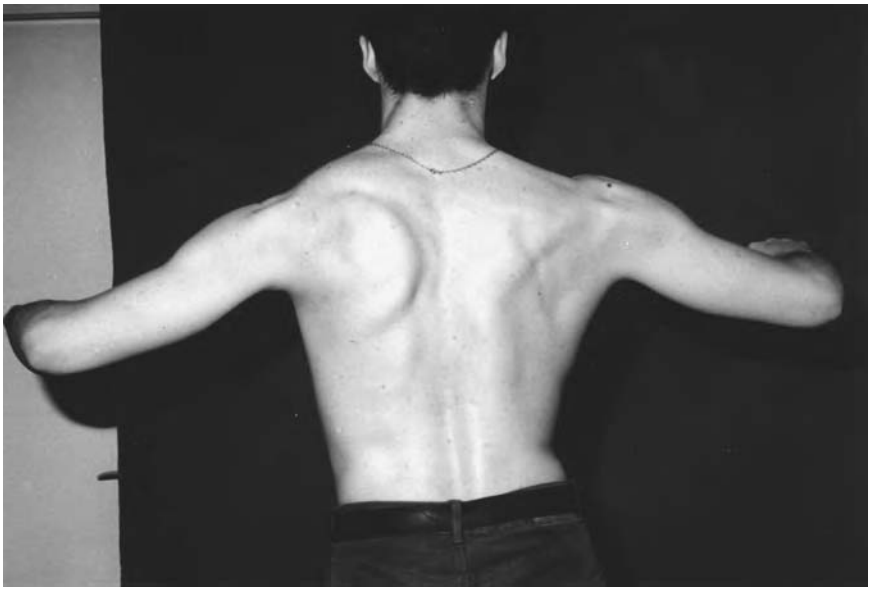


FIGURE 7.3 After intensive weightlifting for a period of several weeks, a 24-year-old athlete noticed a weakness of the right biceps brachii and hypotrophy of the right shoulder muscles. Clinical and electroneurographic tests proved the presence of suprascapular nerve syndrome, lateral axillary hiatus syndrome, and syndrome of the musculocutaneous nerve in the shoulder region. (A) During external rotation in the shoulder against force, hypotrophy of the supraspinatus, infraspinatus, teres minor, and the posterior part of the deltoideus muscles is visible.

(B)



(C)

FIGURE 7.3 (CONTINUED) (B) Hypotrophy of the supraspinatus and deltoid muscles during abduction of the upper arm is visible. (C) The right shoulder is lower than the left, and the right biceps brachii shows marked hypotrophy.

TREATMENT

Initial treatment includes avoidance of all activities that place the nerve under stretch. Infiltration of the scapular notch with anesthetic and corticosteroids using fluoroscopic guidance can be diagnostic as well as therapeutic. Martin et al.[53] reported "excellent" and "good" results with non-operative treatment. The treatment consisted of a program of physical therapy to improve the range of motion of the shoulder and to strengthen the surrounding muscles. Failure to respond to conservative approaches mandates surgical release of the transverse scapular ligament[33,35,40] opening of the scapular foramen if present, and possibly neurolysis.[50] Vastamaki and Goransson[49] evaluated

54 patients, ranging from 2 to 9 years, following surgical release of the suprascapular notch. The most dramatic effect of the operation was prompt disappearance of the pain. However, there were ten poor long-term results. Romeo et al.[57] describe an algorithm for the management of suprascapular neuropathy. According to Cummins et al.,[59] operative decompression of the suprascapular nerve is associated with a high rate of pain relief and functional improvement. However, resolution of muscle atrophy is less predictable.

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8 Lateral Axillary Hiatus Syndrome (Quadrangular or Quadrilateral Space Syndrome)

In this syndrome, first described by Bateman[1] in 1955, the axillary nerve can be compressed while passing through the lateral axillary hiatus (quadrilateral foramen) in the shoulder region.

ANATOMY

The long head of triceps muscle divides the space created by the teres major and minor, the humerus, and the scapula into two spaces: the triangular foramen or medial axillary hiatus and the quadrangular (quadrilateral) foramen or lateral axillary hiatus (Figure 8.1). The medial axillary hiatus lies between the teres minor muscle superiorly, the teres major muscle inferiorly, and the long head of the triceps brachii muscle laterally. The circumflex scapular artery passes through the medial hiatus. The lateral axillary hiatus is limited proximally by the lower margin of the teres minor muscle, distally by the upper margin of the teres major muscle, laterally by the humerus, and medially by the long head of the triceps muscle. The axillary nerve and the posterior circumflex artery pass through the lateral opening. The axillary nerve, a product of the posterior branch of the brachial plexus, enters this space from its position over the subscapular muscle; together with the posterior circumflex artery, the nerve then passes deep to the deltoid muscle. Fractures of the surgical neck of the humerus occur at this point. The axillary nerve supplies the deltoid and teres minor muscles and the skin of the posterolateral region of the shoulder and upper arm via the lateral cutaneous branch of the arm.

ETIOLOGY

Understanding the anatomy of the lateral axillary hiatus, one can foresee the potential risk of axillary nerve damage with upper arm and shoulder trauma. Fractures of the humerus and scapula,[2] as well as shoulder dislocation,[3,4] may traumatize the neurovascular structures along their course. Nerve palsies may remain unnoticed until reduction of shoulder dislocations, because the patients often will not move their shoulders while they are dislocated. Local tumors, organizing hematomas, or simple fracture callus can also narrow the lateral axillary hiatus.

If one abducts an arm while sleeping, the medial and lateral axillary hiatuses decrease in size as the teres major and minor muscles approach each other. Mansat et al.[5] postulated that this functional compression as the arm becomes perpendicular to the body may lead to nerve compression (Figure 8.2). Studying paraplegics with shoulder girdle hypertrophy, Kirby and Kraft[6] described simple teres hypertrophy as an etiology for nerve compression or vascular compromise. Compression of the nerve can occur in the quadrangular foramen from muscle contraction during sporting activities[15] or from abnormal positioning of the arm during anesthesia.[7,8] Baker and Lin[9] even

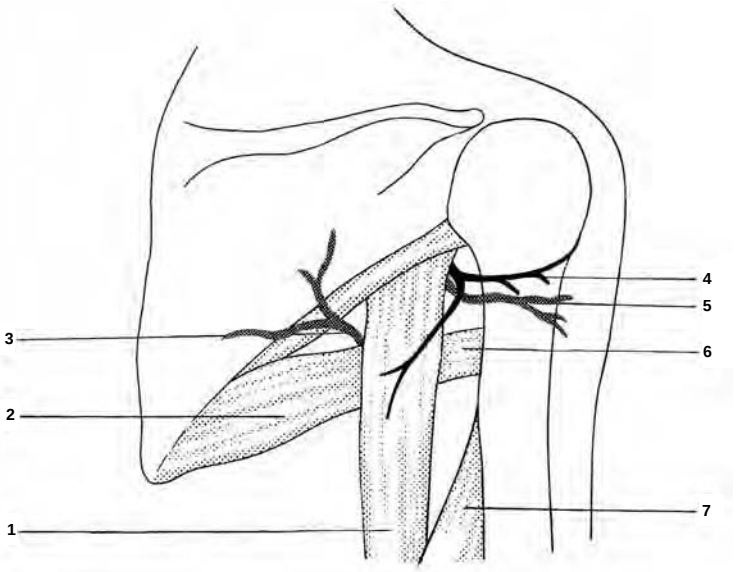

FIGURE 8.1 This quadriangular space is created by the relationships of three muscle bellies and the humerus. The axillary nerve enters this space and can be damaged or compressed —1: long head of the triceps brachii muscle; 2: teres major muscle; 3: circumflex scapular artery; 4: axillary nerve; 5: posterior circumflex humeral artery; 6: teres major muscle; 7: medial head of the triceps brachii muscle.

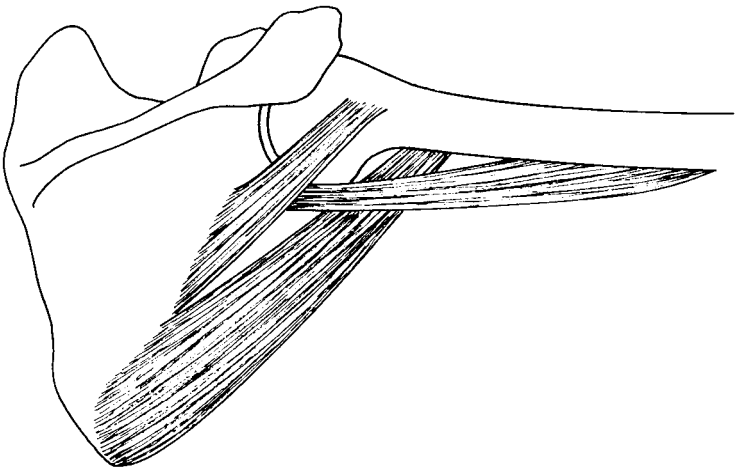

FIGURE 8.2 During abduction of the arm, the medial and lateral axillary hiatuses decrease in size as the teres major and minor muscles approach each other.

consider the quadrangular space syndrome as one of the most commonly recognized neurovascular compression syndromes in the throwing athlete. Spontaneous entrapment of the axillary nerve by fibrous band or muscle in the quadrilateral space may occur.[10]

CLINICAL SYMPTOMS AND SIGNS

Depending on which nerve branches are compromised, the clinical signs may range from paresthesias and hypesthesias around the shoulder and upper arm to deltoid atrophy manifested by contour changes around the shoulder. Compensatory activity of the supraspinatus muscle in con-

junction with the long head of the biceps helps to diminish the functional disability found with deltoid atrophy. Francel et al.[11] present a series of five patients with quadrilateral space syndrome. All patients had the diagnosis made on the basis of tenderness over the quadrilateral space, paresthesia over the lateral shoulder and upper posterior arm, and deltoid weakness associated with decreased shoulder abduction. Additionally, a history of trauma was present in each patient.

Electromyographical studies may indicate not only peripheral nerve lesions proximal to the deltoid muscle, but also the degree of entrapment and reinnervation following removal of the compressive lesion. Angiography might show blockage of the posterior circumflex artery with the arm in 60 degrees of abduction. However, this can possibly be found in individuals without compression if the arm is abducted. Linker et al.[12] present findings of magnetic resonance imaging (MRI) in quadrilateral space syndrome.

TREATMENT

The conservative measures of immobilization (not in abduction) and physical therapy are used prior to local injections of corticosteroids. Mumenthaler and Schiliack[13] emphasize the importance of verifying reduction of the shoulder in preventing the development of this syndrome. Failure to yield improvement within 6 months indicates the need for surgical decompression.[14,18] Mansat et al.[5] suggest that 40% of all patients presenting with axillary nerve compression will need surgical release of the tendinous insertions of the teres major and minor muscles. They recommended a posterior approach. Similarly, Lubahn and Cermak[17] consider the posterior approach a better option. If scarring has left the axillary nerve adherent to the joint, a neurolysis should be done.[19]

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9 Intercostobrachial Nerve Syndrome

The intercostobrachial nerve is the cutaneous branch of T2 (eventually T1-T3). The syndrome of the compression of this nerve presents as pain in the arm predominantly on the postero-medial aspect. While it might localize to the arm, symptoms can be referred to the anterior chest wall.

ANATOMY

The intercostobrachial nerve is the lateral cutaneous branch of T2 (Figure 9.1). It arises from the second intercostal space in the midaxillary line and penetrates the intercostal and serratus anterior muscles where it joins the medial brachial cutaneous nerve. It occasionally joins the lateral cutaneous branches of T3, T1, or T4.[1] The midaxillary exit of intercostobrachial nerve is characteristic. In patients presenting with this compression neuropathy, the nerve tends to exhibit a more anterior exit, thus making the nerve more vulnerable to injury. The medial brachial cutaneous nerve arises from the medial fascicle of C8 and T1 as a separate nerve that may make anastomose with named branches of the intercostobrachial nerve. O'Rourke et al.[4] demonstrated on 28 axillary dissections that the intercostobrachial nerve and its main branch (the posterior axillary nerve) were constant in all dissections, but the nerve's origin, size, and connection to the brachial plexus and medial cutaneous nerve of the arm were variable. In 36% there was a connection to the medial cord of the brachial plexus in the axilla. In the majority, the intercostobrachial nerve supplied at least the proximal half of the arm, and in one third it reached the level of the elbow joint. In 18% there was a connection to the medial cutaneous nerve of the arm.

ETIOLOGY

Anatomical position of the intercostobrachial nerve or nerves places the nerve at risk for compression from different processes in the axillary region or the lateral thoracic wall, as well as from surgical procedures in these regions. Possible causes of intercostobrachial syndrome include primary or metastatic rib tumors, lymphogranulomas, sympathicoblastomas, mediastinal tumors and paravertebral tumors. Iatrogenic lesions could happen from radical mastectomies and axillary dissections, (i.e., first rib resections). Salmon et al.[5] conducted a prospective randomized trial to compare functional results. They assessed the presence of sensory deficits or shoulder pain in those patients whose nerve was preserved versus those patients whose nerve was sacrificed. The conclusion was that preservation of the intercostobrachial nerve, while anatomically preferable, is not functionally necessary during axillary dissection for breast cancer. The compressive lesions following excessive callus formation from humeral and rib fractures might be also causes of intercostobrachial syndrome.

CLINICAL SYMPTOMS AND SIGNS

The intercostobrachial syndrome presents with pain because this is solely a cutaneous nerve. Pain localizes in the arm, mainly on the posterior-medial area . This pain may be referred to the anterior

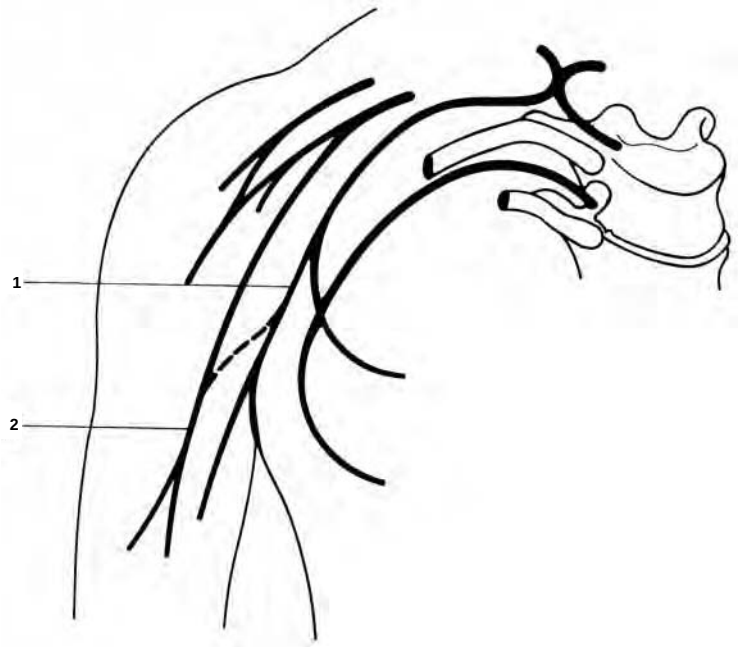

FIGURE 9.1 The intercostobrachial nerve arises from the second intercostal space in the midaxillary line — 1: intercostobrachial nerve; 2: medial brachial cutaneous nerve.

chest wall. The pain might be dull, aching, or burning, with intermittent flashes of sharp, stabbing pain.[6] Abduction of the shoulder and flexion at the elbow can reproduce the symptoms. Placing the hand behind the head and applying pressure just below the second rib, close to the anteroir or mid axillary line can also provoke a response. Additionally, this maneuver can put traction on the nerve. A Tinel sign may be directly elicited by percussion over the nerve as it exits along the mid axillary line. After mastectomy, the activities of daily living that require arm motion can result in the appearence of symptoms or aggravate the symptoms, especially when the nerve is fixed by scarring. The symptoms are characteristically reduced by rest and aggravated by activities, especially toward the end of a day.

TREATMENT

Injection of corticosteroid and local anesthetic at the trigger point may be successful in decreasing symptoms.[3] Surgical treatment consists of exploration of the nerve and neurectomy,[2] which, in refractory cases with intractable pain, may be successful.

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10 Syndrome of the Musculocutaneous Nerve in the Shoulder Region

Wasting and weakness of the biceps and brachialis muscles result when the musculocutaneous nerve is compressed. This usually occurs as the nerve passes through the coracobrachialis muscle. An associated sensory impairment on the lateral aspect of the forearm can occur. While a rare occurrence, this syndrome was described initially by Braddom and Wolfe in 1978, based on their experience with three patients.[4] This chapter will illustrate this syndrome, and a case report will be used to summarize the approach to it.

ANATOMY

Arising from the lateral cord of the brachial plexus, the musculocutaneous nerve contains both motor and sensory fibers from C5, C6, and C7 spinal roots. The motor branches supply the coracobrachialis, biceps brachii, and brachialis muscles. The sensory branch, known as the lateral antebrachial cutaneous nerve, splits to form palmar and dorsal branches that innervate the radial aspect of the forearm distal to the thenar eminence.

The musculocutaneous nerve branches from the lateral cord lie close to the inferior border of the pectoralis minor muscle. After paralleling the axillary artery and the median nerve, the musculocutaneous nerve supplies the coracobrachialis muscle before piercing it to reach the biceps brachii and brachialis muscles (Figure 10.1). Anatomically, another 14% of cases reveal that the musculocutaneous nerve skirts the coracobrachialis muscle instead of piercing it. This usually occurred in conjunction with an anatomical variation, a caput tertium of the brachialis muscle. Almost 50% of the motor supply for the coracobrachialis muscle arises directly from the lateral cord of the brachial plexus. Relatively fixed in its course while supplying motor branches to the biceps brachii and brachialis muscles, the musculocutaneous nerve runs between the muscles, pierces the brachial fascia 2 to 5 cm proximal to the medial cubital crease, and terminates distally as the lateral antebrachial cutaneous nerve. Anatomical variations exist; Kosugi et al.[11] noted a supernumerary head of the biceps and varied branching patterns of the nerve with communication with the median nerve in 57.3% of cases.

ETIOLOGY

Despite the rarity of this syndrome, several factors are consistently found.[1,4,8,10,13–16] The patients are young, active individuals who perform demanding work with flexion of the shoulder and repetitive flexion of the elbow with a pronated forearm. While the occasional patient presents following a single episode of overload,[4] the majority present following repetitive high-demand activities. Some authors[4,13,14] suggest nerve compression occurs with hypertrophy of the coracobrachialis muscle or excessive pressure generated by the muscle. This pressure could cause a neuropraxia or Wallterian degeneration. Isolated musculocutaneous nerve injury complicating closed fracture of the clavicle was described by Bartosh et al.[3]

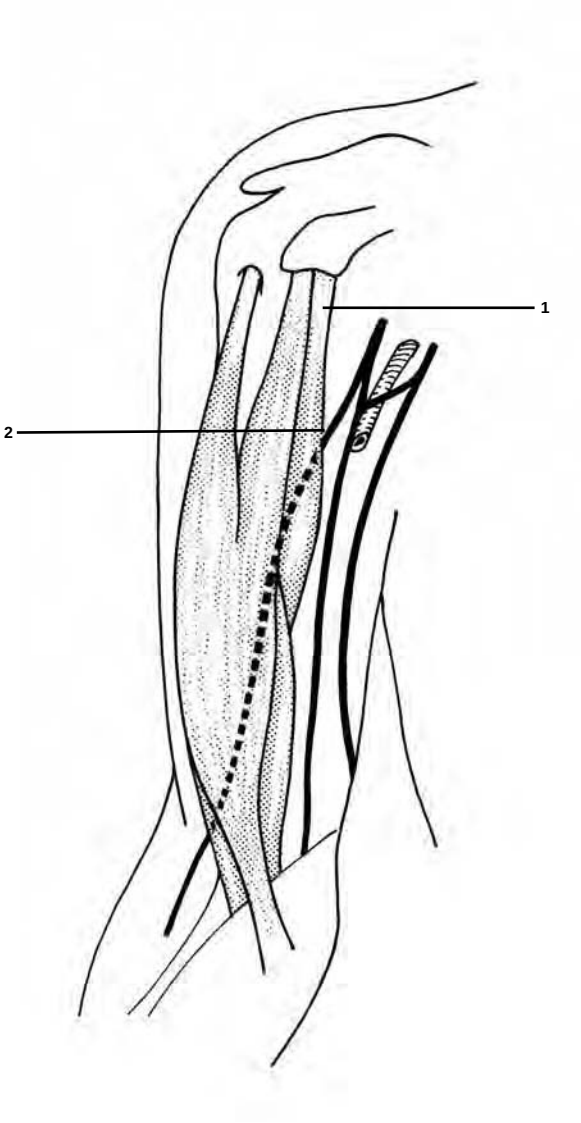

FIGURE 10.1 The musculocutaneous nerve arising from the lateral cord of the brachial plexus and passing through the coracobrachialis muscle — 1: coracobrachial muscle, 2: musculocutaneous nerve.

Some authors[2,6,10,17] have suggested a traction phenomenon. Traction injury may also occur following surgical positioning in abduction and external rotation (Table 10.1). However, except for isolated injuries, compression of the musculocutaneous nerve occurs distal to the motor branch to the coracobrachialis muscle. A functioning coracobrachialis muscle differentiates a musculocutaneous nerve injury from a lateral cord or brachial plexus injury.[19]

CLINICAL SYMPTOMS AND SIGNS

Musculocutaneous nerve compression leads to biceps brachii muscle wasting, which usually leads to the patient's presentation. Sensory complaints are referred to the lateral side of the forearm. Typical symptoms and signs are presented in Table 10.2. Bilateral musculocutaneous nerve palsy was described too.[1,12] Characteristically, biceps brachii and brachialis muscle weakness follow

TABLE 10.1
Proposed Etiologies for Musculocutaneous Nerve Compression or Damage

Authors	Proposed Etiology	Activity
Braddom and Wolfe[4]	Hypertrophy or strong contraction increasing pressure	Weight lifting
Kim and Goodrich[10]	Stretch between origin and fixed area of the nerve	Ball sports (i.e., throwing)
Mastiglia[14]	Entrapment or stretching	Rowing, model airplane flying
Bach et al.[2]	Stretching	Shoulder surgery
Auzou et al.,[1] Felsenthal et al.[8] and Pećina et al.[17]	Entrapment or stretching	Vigorous upper extremity exercise
Caspi et al.[6]	Stretching	Coracoid process transfer

TABLE 10.2
Clinical Symptoms and Signs of Patients with Musculocutaneous Nerve Compression and Possible Differential Diagnoses

General Category	Symptoms and Signs	Diagnoses
Motor	Biceps brachii muscle; weakness, atrophy; absent reflex	Muscle or tendon rupture; C5–C6 radiculopathy; brachial plexus palsy; musculocutaneous; nerve injury
Sensory	Lateral aspect of the forearm; hypesthesia; paresthesias	Brachial plexus; injury
Pain	Present	Acute injuries; not present in musculocutaneous nerve compression
	Absent	Chronic tendon rupture, occasionally with plexus injuries; musculocutaneous nerve injury

intensive athletic activity or strenuous physical work with a flexed arm, elbow flexion-extension, and a pronated forearm.[4,8,11–15] Typically, no pain is associated. Neurologic findings include absent biceps reflex, decreased biceps tone, and hypesthesias and paresthesias on the lateral aspect of the forearm.[1–17]

The clinical syndrome produced by a patient's signs and symptoms should indicate musculocutaneous nerve compression. The differential diagnoses include biceps brachii tendon rupture, a C5 or C6 radiculopathy, or a brachial plexus injury. The value of a thorough history cannot be discounted, because a traumatic event usually precedes these diagnoses.

A detailed physical exam will help define the muscles and the sensory dermatomes involved. A rupture of the long head of the biceps produces muscle weakness and a characteristic deformation of the anterior upper arm. However, no sensory component differentiating musculocutaneous nerve injury from a C5–C6 radiculopathy is associated. An electromyographic (EMG) exam shows that other C5–C6 innervated muscles (the deltoid, supraspinatus, infraspinatus, and teres minor muscles) are uninvolved. EMG studies may show a prolonged latency or decreased amplitude in the involved musculocutaneous nerve.[8,18,20] A brachial plexus injury will involve almost the entire upper extremity rather than isolated motor and sensory findings with equivocal EMG data. Initial denervation indicates a poor prognosis.[15] Thermography can objectively define sensory disturbances (see Color Figure 10.1).*

* Color Figure 10.1 appears after page 174.

TREATMENT

With the limited number of cases available, no randomized study can be used as a guide. However, treatment has been consistently conservative and consistently successful. Felsenthal et al.[8] have used surgical decompression if conservative treatment gave no relief. Cessation of the strenuous inciting action, rest, observation, and a gradual return to activity when symptoms resolve is effective therapy. McIlveen et al.[15] note that iatrogenic surgical injuries recover incompletely, while patients with spontaneous injuries recover with a satisfactory result.

CASE REPORT

A 23-year-old rower without prior history of trauma described a decrease in his left arm's circumference and a painless weakness in elbow flexion (Figure 10.2). His strengthening program required strenuous workouts, including more than 500 pushups daily. Physical examination verified full joint range of motion but reduced biceps muscle strength and size. Neurologically, the biceps tendon reflex was absent, the biceps muscle tone was reduced, and the lateral aspect of the forearm could be hypesthetic. Several diagnostic tests were performed. Ultrasound and radiographic exams of the

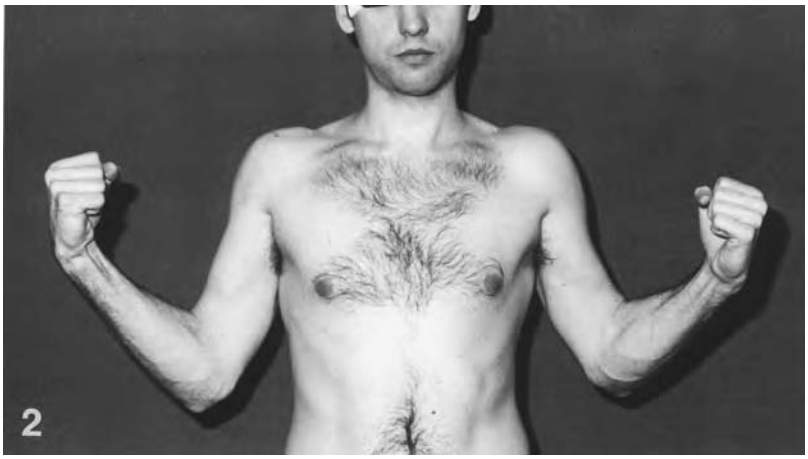

FIGURE 10.2 Patient with athrophy of the left upper arm resulting from musculocutaneous nerve entrapment.

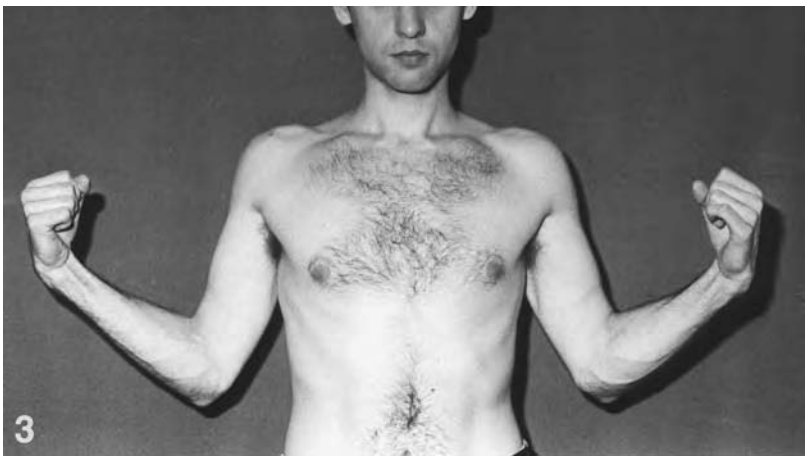

FIGURE 10.3 The appearance of the same patient 3 months later, when the muscle bulk was restored.

cervical spine, shoulder, and elbow were normal. Telethermographic examination demonstrated anisothermy of the forearms, and a computerized multi-isothermal analysis showed hyperthermia of the left forearm in the region innervated by the lateral antebrachial cutaneous nerve. An upper-extremity EMG exam showed an abnormal innervation pattern in his left biceps and brachialis muscles, with prolonged distal latencies and decreased amplitude of the evoked response. Based on a diagnosis of musculocutaneous nerve compression, all upper-extremity exercise was stopped for 6 weeks. Repeat physical and diagnostic exams after 6 weeks showed improvement. Within 3 months, muscular mass and strength had returned, as well as forearm sensitivity (Figure 10.3). Thermography showed no significant temperature difference between forearms. Follow-up EMG examination showed an intermediate innervation pattern producing changes in the action potentials of increased duration, decreased amplitude, and of a polyphasic nature.

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11 Dorsal Scapular Nerve Syndrome

Dorsal scapular nerve syndrome is characterized by weakness of the rhomboid and levator scapulae muscles, which results in scapulae alatae. The mild winging of the scapula is different from the winging seen with serratus anterior muscle injury or weakness.

ANATOMY

The dorsal scapular nerve originates predominantly from C5 but also may receive some contributions from C4 and C6.[2,4] In brachial plexus dissections, Lee et al.[6] found that 21.7% of cases examined had contributions from C4 to T1. Also, 75.8% of the dorsal scapular nerves originated from the C5 ventral ramus. The nerve courses behind the major portion of the brachial plexus to pierce the scalenus medius muscle and pass between the scalenus posterior and levator scapulae muscles before reaching the superior angle of the clavicle. Then, it runs together with the ramus profundus artery transversal colli along the medial margin of the scapula beneath the rhomboid muscles. The nerve supplies the levator scapulae and rhomboid (major and minor) muscles (Figure 11.1). The levator scapulae muscle, inserting on the upper angle of the scapula, elevates and rotates the scapula. The rhomboid muscles pull the scapula medially as well as elevating it through its insertion on the medial border.

ETIOLOGY

While Tackmann et al.[9] describe isolated lesions of the nerve from trauma, the dorsal scapular nerve may be entrapped or compressed by hypertrophy of the scalenus medius muscle, as described by Kopell and Thompson[5] and Nakano.[7]

CLINICAL SYMPTOMS AND SIGNS

Patients may complain of a feeling of abnormal shoulder motion, which might also be decreased. Additionally, Fisher and Gorelick[2] thought that nerve compression might be an unsuspected component of shoulder pain. Clinically, dorsal scapular nerve injury produces a mild form of scapular winging in the resting position. The medial border and inferior scapular spine are lifted off the chest wall. Several tests can further illustrate the weakness of the rhomboid and levator scapulae muscles. Patients will have trouble or find it impossible to try to bring their scapulae together. Additionally, forward elevation of the arm will lift the medial border of the scapula and pull the inferior angle forward off the chest wall; therefore, observation through a range of motion is key to diagnosis. Electromyographic analysis will demonstrate injury to the rhomboid and levator scapulae muscles. An EMG/nerve conduction study is diagnostic.[8]

TREATMENT

Conservative treatment consists of cervical spine stabilization using a collar (may be soft), cervical traction, physical therapy, muscle relaxants, and anti-inflammatories. Neurolysis is rarely considered

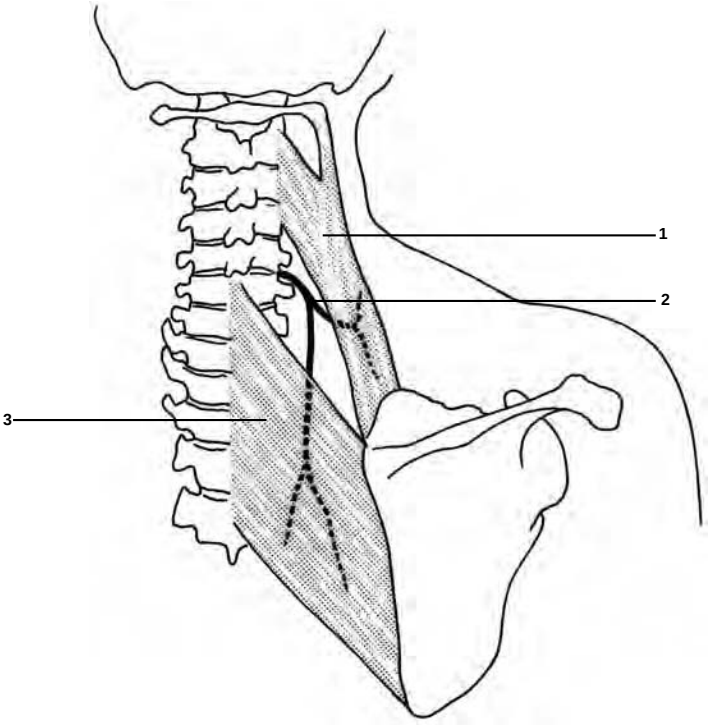

FIGURE 11.1 The course of the dorsal scapular nerve — 1: levator scapulae muscle; 2: dorsal scapular nerve; 3: musculi rhomboidei.

and should be preceded by magnetic resonance imaging (MRI) to further define the compressive anatomy. According Chen et al.,[1] among 24 sides of 22 patients undergoing surgery, the symptoms of 20 sides of 19 patients were completely or partially relieved.

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12 Long Thoracic Nerve Compression

The long thoracic nerve may be compressed at multiple sites along its course, between the clavicle and first two ribs or in the axilla. Nerve compression alters the function of the serratus anterior muscle. Therefore, patients may present with a high-riding scapula and weakness of shoulder abduction and arm elevation.

ANATOMY

The long thoracic nerve receives contributions from C5 to C8. Lee et al.[16] noted that the nerve was formed from C5, C6, and C7 in 76% of cadaver dissections. The roots of C5 and C6 typically pass between the scalenus medius muscle. The contributions from C7 and C8 typically pass between the scalenus medius and minimus muscles. The nerve courses laterally between the clavicle and first rib under the axillary artery before running over the serratus anterior muscle, innervating its individual origins (Figure 12.1). The muscle may be quite large, with nine or ten origins. With an insertion site along the medial border of the scapula, the serratus anterior muscle and the rhomboid muscles act to hold the scapula against the thoracic wall while allowing smooth motion along the wall. Contraction of the serratus anterior muscle pulls the scapula down and out, allowing the elevation of the arm.

ETIOLOGY

While multiple etiologies have been proposed,[5,10,11,21,22,29,33] external nerve compression is the most frequent cause secondary to compression in a patient's axilla (i.e., tight bandage or plaster casts, axillary splints, or poorly adjusted crutches). Axillary masses may compress the nerve against the chest wall. Other causes include falls or blows on the shoulder that shift it suddenly down, or repetitive irritation from carrying heavy loads on one side.[3] These actions lead to repetitive compression of the nerve between the coracoid process and the first or second rib. Tension injuries when the head is tilted suddenly to the contralateral shoulder have been proposed.[4,23,27,34] A variety of athletes from such diverse activities as ballet,[35] tennis, weightlifting,[28] bowling, and golf (to name a few) have presented with symptoms of nerve compression.[1,8,17,23,36] *Borrelia burgdorferi* infections have been reported to produce palsies.[20] Lastly, surgery in the axillary region can damage the long thoracic nerve, either directly or as a result of excessive retraction.[2,24]

CLINICAL SYMPTOMS AND SIGNS

Patients typically present with vague complaints of weakness and pain. Shoulder strength is decreased, especially in abduction and elevation. Range of motion in these planes may not be significantly limited. Patients will have trouble with overhead tasks. The telltale sign of long thoracic nerve compromise is winging of the scapula (Figure 12.2).[17] This can be best demonstrated by having patients do push-ups. If they are unable, they can lean against a wall and push away from the wall (Figure 12.3). Serratus anterior muscle weakness allows the medial margin of the scapula to lift off the chest wall, while the scapula itself shifts up, with the inferior scapular spine swinging medially toward the spine.

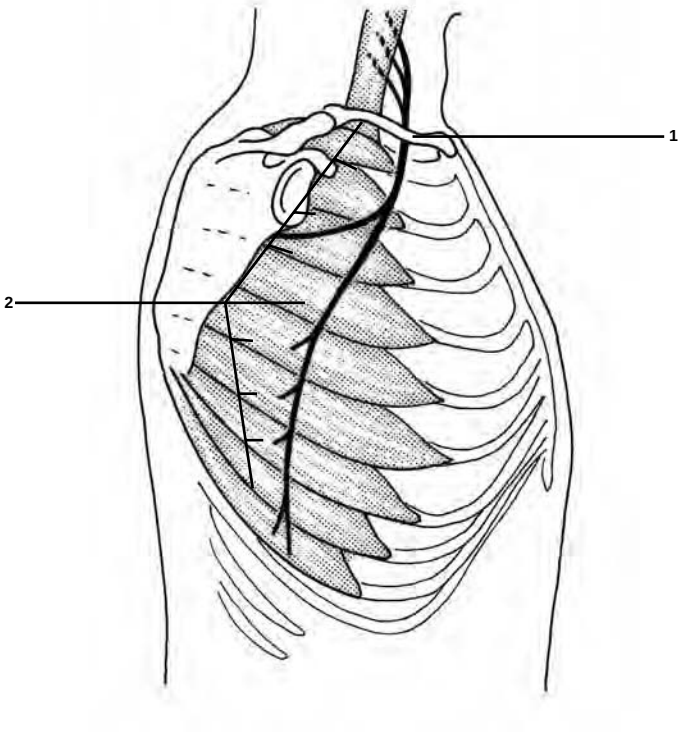

FIGURE 12.1 The long thoracic nerve (1) runs in a lateral and caudal direction along musculus serratus anterior, innervating its indentations (2).

Shoulder pain can appear at the same time as the smooth gliding motion of the scapula over the chest wall is disturbed. Pain is typically diffuse around the shoulder and may spread down the arm as the patient tires during activity. Thus, if a patient complains of pain with activity but does not have it in the office, one should try to reproduce those actions that elicit the pain. These actions may be as simple as having a ballplayer throw for 30 minutes during the visit.

Electrodiagnostic tests are useful in diagnosis of injury and differentiation from other neurologic complaints in the upper extremity, including amyotrophic neuralgia.[7,13–15,24,31] Before significant muscle weakness occurs, amyotrophic neuralgia has a large pain component early in the disease course. Reinnervation and nerve recovery can also be followed with electromyographic studies.

TREATMENT

Conservative treatment consists of activity modification or avoidance of repetitive trauma to the shoulder girdle.[6,17,34,37] Patients should be advised to avoid carrying heavy burdens on their shoulders or pushing heavy objects. Physical therapy seeks to restore range of motion and to stretch the rhomboids and pectoralis minor muscles to avoid contracture. Various braces have been proposed to support the scapula to ease the strain on the serratus anterior muscle.[18,32] Patients should be advised that nerve recovery may take 9 to 24 months. The prognosis for recovery is good in nonpenetrating injuries, as reported by Johnson and Kendall in 111 patients.[12] Schultz

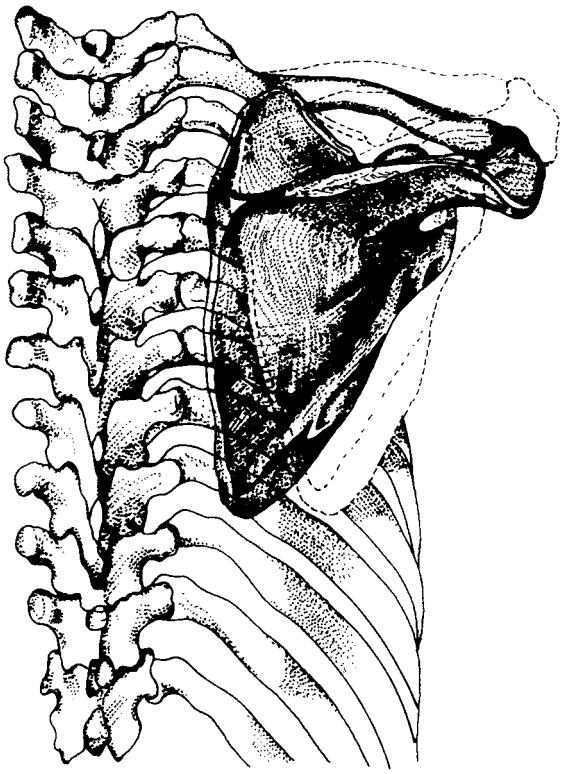

FIGURE 12.2 In long thoracic nerve paralysis (serratus anterior muscle weakness) the characteristic clinical is winging of the scapula.

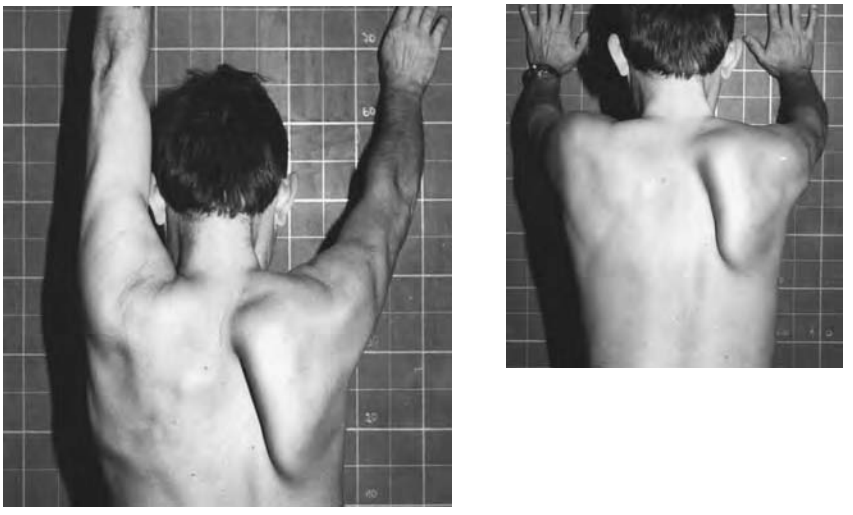

FIGURE 12.3 Scapular winging results from paralysis of the serratus anterior because of long thoracic nerve palsy. Results of the wall test are characteristic clinical signs.

and Leonard[27] report favorable outcomes in four athletes treated conservatively following tension/traction injuries.

Pain relief can be achieved through a combination of physical therapy, anti-inflammatories, and analgesics. Narcotics should be avoided, because of the long time course for recovery. Surgical intervention is warranted only in cases where sharp nerve injury might have occurred. No data are available on the prognosis of nerve repair. Treatment of paralysis consists of either fixation of the scapula to the chest wall or transposition of neighboring musculature to the scapula to replace the action of the serratus anterior muscle.[9,19,26,37]

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13 Radial Nerve Compression in the Upper Arm

The radial nerve in the upper arm can be compressed in three areas along its course: the axilla, the spiral groove, and the area distal to the radial nerve hiatus near the lateral humeral epicondyle. The nerve is most susceptible in the spiral groove as it courses along the posterior surface of the humerus adjacent to the intermuscular septum before exiting through the radial nerve hiatus.

ANATOMY

The radial nerve originates from the posterior branches of the trunks of the brachial plexus. Contributions from C4 to T1 are found in 64.5% of specimens, while 34.5% have contributions from C5 to T1.[13,15] Some authors believe that the contribution may be narrower and limited to C5 to C8[27,33,34] or C6 to T1.[36] After joining together, the posterior branch courses posterior to the axillary artery lying on the subscapular muscle, the tendons of the latissimus dorsi, and teres major muscles before reaching the posterior surface of the humerus. As it crosses the axilla, it divides into the axillary and radial nerves. The radial nerve joins with the profunda brachii artery and vein and enters the spiral groove between the medial and lateral heads of the triceps brachii muscle. The nerve runs along the periosteum in a posteromedial to anterolateral direction before piercing the intermuscular septum at the hiatus, which lies 10 cm proximal to the lateral humeral epicondyle (Figure 13.1).

The radial nerve supplies the triceps brachii and anconeus muscles in the upper arm. In the spiral groove, the posterior antebrachial cutaneous nerve branches off. As the nerve pierces the septum to lie anteriorly, it lies in a sulcus or "canalis" limited by the brachioradialis muscle (laterally) and the brachialis muscle (medially). In this sulcus, the radial nerve branches into its terminal components: the posterior interosseous nerve and the superficial radial nerve. While the superficial radial nerve is predominantly a sensory branch, the posterior interosseus nerve supplies the majority of the extensor muscles in the forearm.

ETIOLOGY

Many etiologies, including anomalous muscles, abnormal positioning (Saturday night palsy), external compression (high-riding crutches), repetitive trauma, and internal compression (tumors/callus), can produce a radial nerve compression picture, as illustrated in Table 13.1. As the radial nerve is exposed in the axilla, continued pressure in this area from the back of a chair, crutches, or tumor can injure the nerve. Most common in drinkers, "Saturday night palsy" occurs when one passes out with an arm stretched across a hard support (typically a chair back) producing long-term radial nerve compression. As normal feedback is diminished because of intoxication, individuals do not alter their position despite painful feedback. This injury can also occur if people sleep with their heads on an outstretched arm for an extended time without position changes. Ill-fitting crutches that ride high up in the axilla can also injure the radial nerve. Because the radial nerve travels a long course, the nerve can be compressed by strictures at its hiatus or in the passageway between muscles or muscle heads. Anomalous muscles or connective tissue can exist in these areas, binding the nerve down and producing tension or

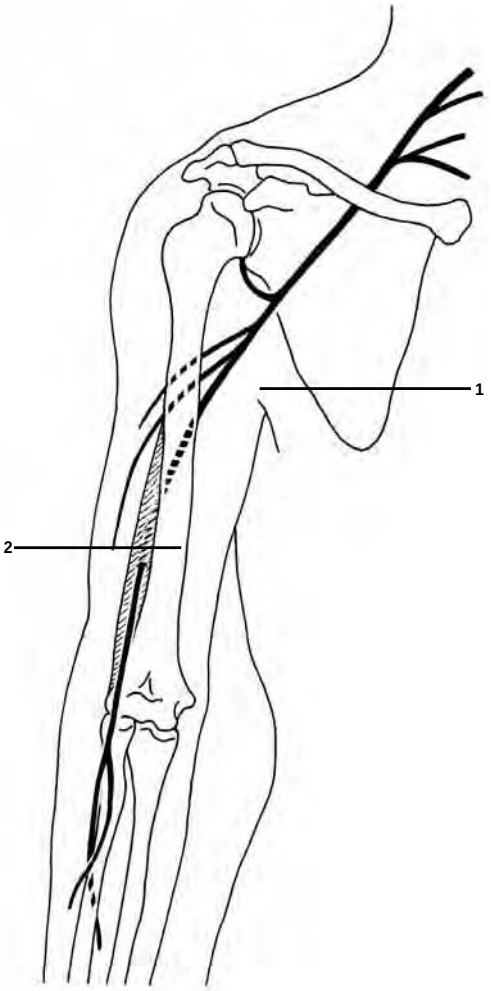

FIGURE 13.1 The radial nerve can be compressed in its course in the upper arm: in the fossa axillaris, in a canalis spiralis up to its issue through hiatus nervi radialis, and in its course in sulcus cubitalis radialis — 1: radial nerve, and 2: hiatus nervi radialis.

compressive forces. Several authors have related nerve injury to triceps brachii muscle contraction or stretching. Wilhelm and Suden[40] described triceps overload in 20 patients, only two of whom had an underlying bony abnormality. Compression in the anterior sulcus distal to the radial nerve hiatus is rare.

CLINICAL SYMPTOMS AND SIGNS

The site of radial nerve compression affects the constellation of signs and symptoms. Knowledge of branch points helps define the area of compression. Proximal compression in the axilla will affect all motor and sensory fields of the radial nerve, producing weakness and numbness. Overlapping sensory fields in the upper arm might mask the sensory impairment. Continued compression in this area can lead to paralysis of elbow, wrist, and finger extension. Finger extension must be tested with the wrist in a neutral position with the patient expected to extend their fingers at the metacarpophalangeal joints to neutral. The combination of intrinsic activity

TABLE 13.1
Etiologies of Radial Nerve Compression

Site	Etiology	Ref.
Axilla	Anatomical anomalies: coracobrachialis minor muscle, long head of the triceps, accessory subscapularis-teres latissimus muscle	12, 18, 22
	Tumors	9
	Trauma	2, 4, 8, 26, 32
	Iatrogenic: crutches, pacemaker	6
	Rifle sling palsy	21
	Tension from a streetcar handle	20
Spiral groove	At the intermuscular septum/hiatus	17, 35
	Saturday night palsy (high backed chair or external compression on the arm)	8, 32
	Prolonged muscle contracture (runners, masons, carpenters, throwing sports, tennis)	5, 25, 31
	Anatomical anomalies: lateral head of the triceps	19, 22, 38–40
	Tumors	23
	Post-traumatic: fracture callus	14

(ulnar nerve) and tenodesis effect (with the wrist flexed) can confuse the observer, who feels that the observed extension effort is evidence of an intact radial nerve, when it is, in effect, only intrinsic action.

Compression at the level of the spiral canal or below will leave the triceps muscle functioning. The brachioradialis muscle and the wrist and finger extensors are affected. Additionally, the superficial radial nerve and the posterior antebrachial cutaneous nerve are affected, leading to numbness. Several authors have noted a variable presentation from compression in this location.[10,33,37] Stewart and Aguayo[29] have proposed that the nerve fasicles might be unequally compressed. Therefore, the brachioradialis muscle may be spared occasionally, with the wrist and fingers demonstrating the weakness or paralysis.

Compression in the spiral canal (Saturday night palsy) presents with paralysis but without pain. Chronic drinkers present frequently with this clinical picture after falling asleep with their arms across a high-backed chair or table. Their deep sleep prevents them from moving to avoid the early irritation of nerve compression. Pain, while not a characteristic symptom, can be found.[1,40] This pain may be described by the patient in the area of the lateral epicondyle, radial styloid, or dorsum of the hand.[1,40]

While posterior interosseous nerve symptoms are described in another chapter, one should realize that nerves are susceptible to injury or compression along their entire course. Inadvertent injury from intravenous access in the antecubital region can produce hematomas locally, which can lead to nerve compression or venous congestion, which may produce, not only weakness in the muscles innervated by the posterior interosseous nerve, but also numbness in the innervation area of the superficial radial nerve.

Diagnosis requires a detailed history and physicial examination, which will indicate a classical picture of muscle weakness and numbness or dysesthesias.[3] Electromyographical (EMG) tests can better define the area of compression. If the cause is not obvious, specialized imaging tests can then be targeted on the area. After plain radiographs to diagnose basic osseous abnormalities or anomalies, one can use MRI or computed tomography (CT). EMG tests also can be used to follow nerve recovery. The differential diagnosis can include the following medical problems: lead poisoning, porphyria, diabetes, and periarteritis nodosa. Therefore, additional lab work may be necessary to define the etiology of radial nerve paralysis.

TREATMENT

Conservative treatment remains the mainstay for radial nerve paralysis unless a penetrating injury is suspected. Initial nerve conduction studies can confirm nerve continuity. Then, a baseline EMG within 3 to 4 weeks can be used to define the level of compression and to compare when assessing recovery. The etiology should be defined and any provocative activities removed. Rest, nonsteroidal anti-inflammatory drugs (NSAIDs), activity modification, and observation usually allow nerve recovery. Saturday night palsies typically recover after a month without sequelae.[10]

If conservative treatment does not yield recovery, the etiology needs to be localized and a surgical decompression done. These procedures can be risky, especially when the radial nerve is compressed in fracture callus. Timing is critical, because denervated muscle will not recover if not re-innervated within 18 months. Thus, the time necessary for nerve regrowth must be considered. Several authors have proposed more-aggressive approaches if no cause is found and the patient has not responded to conservative therapy.[11,28] These proposals have included interfascicular neurolysis, epineurectomy, or even nerve resection and grafting.[11,28] Steudal and Grafin Vitzthum[28] have found that the compressed nerve shows evidence of nearly complete denervation. The recovery following surgical intervention is lengthy, with a variable outcome.

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14 Supracondylar Process Syndrome

On the anteromedial surface of the distal humerus, an atavistic bony formation, the supracondylar process, can exist and be connected to the median epicondyle by a fibrous band (Struthers' ligament) (Figure 14.1). The median nerve can be compressed in this fibro-osseus tunnel, creating the clinical symptoms of the syndrome of the supracondylar tunnel (canalis supracondylaris).

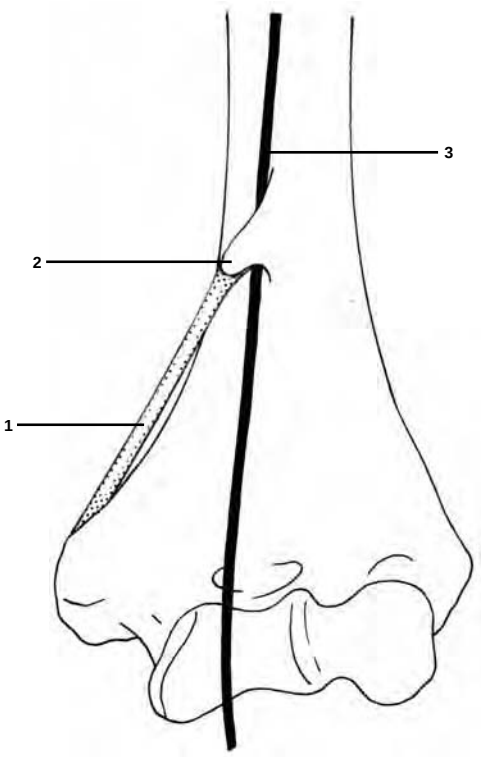

FIGURE 14.1 The location of the supracondylar process and its ligamentous attachments — 1: connective tissue band between the supracondylar process and the medial epicondyle of the humerous (Struther's ligament); 2: supracondylar process; 3: median nerve.

ANATOMY

The supracondylar process represents an anatomical variation that can be regarded as an atavistic formation found in amphibians, reptiles, and mammals.[1] This process is virtually always present in lemurs, but in humans, it is present between only 0.3 and 2.7% of the time.[1–4,27] The bony formation starts from a large base, 7 cm proximal to the median epicondyle on the anteromedial surface of the humerus.[5] Directed distally, its beak-like apex gives rise to a band of connective

tissue that inserts on the medial epicondyle, forming a tunnel. The process can vary from 2 to 30 mm in height.[4-6] Gruber[7] calls it tuberculum musculare. In some lower mammals, the tunnel can be completely ossified, forming a supracondylar foramen. In humans, encrustation with calcium salts creates a radiographically visible tunnel.[8] The median nerve and brachial artery may pass through this tunnel. The ulnar nerve rarely passes under the process (Figure 14.2). Because the compression occurs proximal to all branches of the median nerve in the forearm, all forearm areas (both motor and sensory) innervated by the median nerve are impaired.

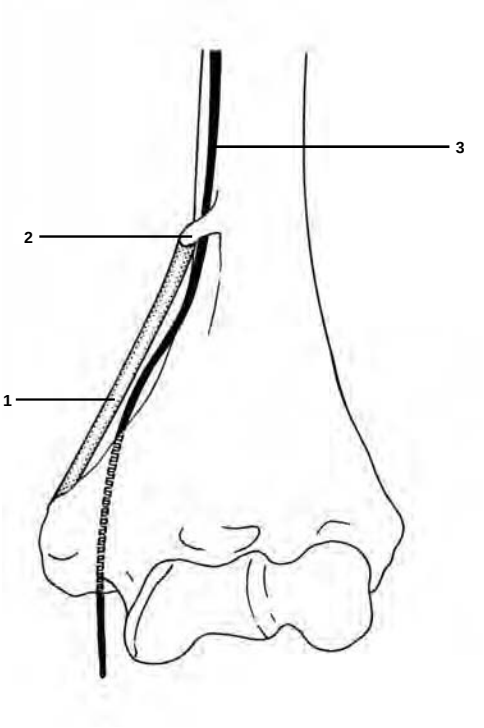

FIGURE 14.3 In a fashion similar to median nerve compression near the supracondylar process, the ulnar nerve may be compressed — 1: connective tissue band (Struther's ligament); 2: supracondylar process; 3: ulnar nerve.

ETIOLOGY

Proposed etiologies for median nerve compression include the following: fractures of the supracondylar process,[1,9–11] brachial artery ischemia,[12] and idiopathic etiologies.[13,14] Domljan[15] found the median nerve to be stretched over the process like a string over the bridge of a violin. Symeonides[14] described a patient who developed the syndrome after prolonged intravenous infusions with the arm fixed in extension and supination. Straub[25] reports the case of an 8-year-old girl with bilateral supracondylar process of the humerus and the compression of the median nerve on the left side. A typical Struthers' ligament was found. After resection of bone and ligament, complete recovery was achieved.

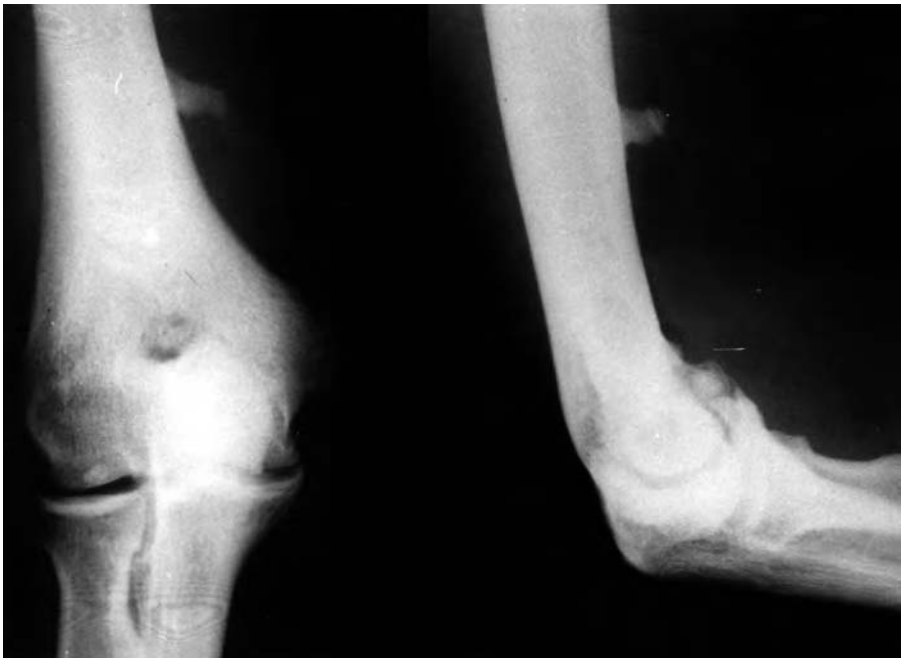

FIGURE 14.3 Radiographic appearance of the supracondylar process.

CLINICAL SYMPTOMS AND SIGNS

Lund[9] first described the clinical findings of this tunnel syndrome. The sensory findings consist of pain and paresthesias in the median nerve dermatomes, with deep blunt pain in the area of compression. The pain increases at night and radiates to the forearm, thumb, and first three fingers. Motor signs include weakness of the involved muscles, decreased thumb opposition, and decreased flexion of the first three fingers. In slender individuals, the supracondylar process can be palpated. Percussion may produce a Tinel's sign, pain, and paresthesias in the median nerve's dermatomes. Thomsen[16] describes signs and symptoms in both the ulnar and median nerve distributions due to compressions in the tunnel of both nerves. Murali et al.[17] describe bilateral compression of the median nerve by supracondylar spurs. Burczak[18] describes a case in which acute median nerve palsy developed after surgical treatment of an intra-articular distal humerus fracture in a patient with an intact supracondylar process. Kessel and Rand[19] feel that elbow extension and forearm supination can eliminate the radial artery pulse caused by compression in the tunnel. Senner et al.[26] and Ivins[24] present cases of patients with the supracondylar process syndrome.

Electromyography can be applied to diagnose impaired conduction velocity across the region.[20] Radiographic studies allow for the possibility of this diagnosis, since plain films would identify the existence of a process (Figure 14.3).

TREATMENT

Conservative therapy starts with immobilization of the forearm in pronation with the elbow in 40 degrees of flexion. Local corticosteroids may be helpful. Surgical decompression remains the definitive treatment, because it removes the supracondylar process (see Color Figure 14.1).[21–23]

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15 Syndrome of the Musculocutaneous Nerve at the Elbow

Compression of the musculocutaneous nerve near the elbow has been described by Basset and Nunley[1] based on 11 patients. Sensory disturbances occur when the nerve is compressed where it pierces the brachial fascia.

ANATOMY

Originating from the lateral cord of the brachial plexus, the musculocutaneous nerve supplies motor branches to the coracobrachialis, biceps brachii, and brachialis muscles and sensory branches to portions of the forearm and wrist. The lateral cord of the brachial plexus crosses into the axilla before dividing into the musculocutaneous nerve and contributing to the median nerve. The musculocutaneous nerve stays lateral to the axillary artery and lies on the coracobrachialis before piercing through the muscle. In about 10% of the cases, the nerve does not pierce the muscle's fascia. The nerve then continues to run between the biceps brachii and brachioradialis muscles before piercing the brachial fascia 2 to 5 cm proximal to the medial cubital crease.

Olson[2] described the nerve as continuing its course under the lateral margin of the biceps tendon until the cubital crease. Shown in Figure 15.1, the musculocutaneous nerve becomes the lateral antebrachial cutaneous nerve, which courses distally under the cephalic vein. While terminology may vary, the lateral antebrachial cutaneous nerve splits to form the palmar and dorsal branches. The palmar branch supplies the anterior skin of the lateral (radial) half of the forearm before terminating at the thenar eminence. The dorsal or posterior branch runs along the radial margin of the forearm to supply the posterior surface of the distal third of the forearm before terminating at the base of the first metacarpal bone.

ETIOLOGY

Compression of the lateral antebrachial cutaneous nerve may occur when the nerve passes below the tendon of the biceps muscle before piercing the brachial fascia. According to Bassett and Nunley,[1] the lateral free margin of the biceps aponeurosis compresses the nerve against the brachial fascia with elbow extension. Pronation further increases nerve compression. Of 11 patients, eight developed symptoms in the dominant hand.[1] All 11 had histories of acute or chronic trauma to the elbow, usually consisting of forced elbow extension coupled with forearm pronation. Other cases have been reported with tennis backhand strokes,[3] repeated pronation and supination of the forearm, and excessive use of a screwdriver during house construction (3000 screws by hand). Hale[4] describes a patient whose symptoms occurred after carrying a heavy bag. Spontaneous compression neuropathy of the musculocutaneous nerve was described by Mathews and Ferlic.[5] Braddom and Wolfe[6] have reported acute entrapment of the musculocutaneous nerve proximally under the edge of a hypertrophied coracobrachialis muscle. However, in addition to sensory losses, the patient had paresis of the biceps, brachialis, and coracobrachialis muscles following very strenuous physical

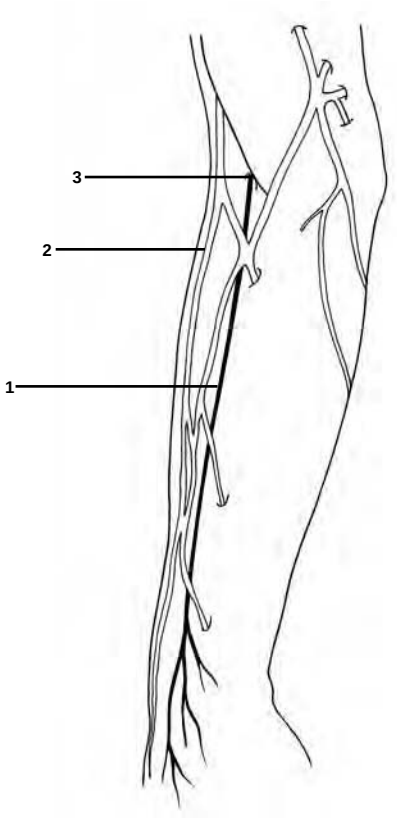

FIGURE 15.1 Because it runs superficially, the terminal portion of the musculocutaneous nerve may be damaged — 1: lateral cutaneous nerve of the forearm; 2: cephalic vein; 3: origin of the musculocutaneous nerve as it exits through a layer of fascia underlying the skin

exercise. Compression of the lateral antebrachial cutaneous nerve by the biceps tendon,[10] by a glomus tumor,[11] or injury of the nerve as a complication of phlebotomy[9] were also described.

CLINICAL SYMPTOMS AND SIGNS

The diagnosis of the syndrome of the musculocutaneous nerve in the region of the elbow can be confused with the symptoms of epicondylitis, especially as both syndromes can be linked to tennis. Pain is always present and usually located in the anterolateral portion of the elbow. In patients who develop acute compression, the pain is usually burning in nature. Patients avoid full elbow extension with forearm pronation, since this motion aggravates their pain. Supination may offer some relief from pain. Patients with chronic irritation present with varying pain and hypesthesia in the wrist and the forearm. Patients do not usually recognize the extent of their hypesthesia; however, they are tender over the musculocutaneous nerve in the region under the biceps muscle where it pierces the brachial fascia. In the subacute and chronic forms, patients have pain with forearm pronation with an extended elbow.

TREATMENT

Treatment options include the following: rest, avoidance of motions that provoke pain, abstinence from sports that use the affected extremity, splinting, corticosteroid injection, and surgical decom-

pression. Splinting and other conservative measures may be used, but surgical decompression should not be deferred if symptoms persist more than 6 months. Decompression releases the musculocutaneous nerve under the biceps tendon and the brachial fascia. Under direct observation, a triangular window should be fashioned in the fascia.[12] There should not be any fascial contact with the nerve regardless of the position of the elbow. The best chance for nerve recovery and relief of symptoms occurs with early decompression. Dailiana et al.[13] describe an operation to relieve compression of the lateral antebrachial cutaneous nerve at the elbow on seven patients, one with bilateral compression; all patients had symptomatic relief. According to Swain, [8] the treatment of the lateral cutaneous nerve syndrome is based on small studies but has included splinting, cortisone injections, and decompression of the nerve.

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16 Medial Antebrachial Cutaneous Nerve Syndrome

Compression or distortion of the medial antebrachial cutaneous nerve most often results from surgical scars or direct injury from surgical procedures in the region of the medial or posterior side of the elbow joint. This can result either in a painful scar or an area of decreased sensitivity inferior to the scar.

ANATOMY

The medial antebrachial cutaneous nerve originates directly from the medial cord (ulnar fascicle, C8, T1 fibers). It runs distally behind the axilary artery, then in front and medial to the neurovascular bundle of the arm. The medial antebrachial cutaneous nerve pierces the fascia together with the basilic vein in the middle of the brachium. It lies subcutaneously on the ulnar side next to the biceps brachii muscle before running distally to branch into an anterior and ulnar ramus in the region of medial cubital crease. This branch point may lie proximal to the crease. The nerve gives one or two branches to the skin of the brachium. The anterior ramus innervates the skin of the antebrachium nearly to the midline. The ulnar ramus innervates the skin on the posterior side of the forearm to the midline (Figure 16.1). The innervation of the medial antebrachial cutaneous nerve also reaches the brachial region and as far distally as the palmar side of the hand.

ETIOLOGY

The medial antebrachial cutaneous nerve innervates the medial and posterior side of the elbow joint. Care should be taken when dissecting around the elbow. Skin incisions can be altered to avoid causing areas of numbness. If the nerve is unavoidable, it should be visualized and protected. The nerve may be injured during ulnar nerve decompression for cubital tunnel syndrome.[1] Because of its anatomical course, the medial antebrachial cutaneous nerve is vulnerable to postoperative scar compression or direct injury during surgical exposure of the medial or posterior part of the elbow.[4]

CLINICAL SYMPTOMS AND SIGNS

Compression or injury of the nerve during surgical interventions results in painful postoperative scars or an area of decreased sensitivity or paresthesias inferior to the scar. Painful neuromas have also been reported.[3] In patients with a complete transection of one or more branches of the nerve, there is an area posterior and distal to the scar of varying size (depending on the cutaneous distribution of the transected nerve), which has less than normal sensitivity. Because of overlap patterns of adjacent cutaneous nerves, there might be no true area of numbness. However, this degree of impaired sensitivity varies from a central zone of greatest impairment, which may approach anesthesia, and gradually merging with surrounding normal skin. The patient with painful scar or “neuromatous” pain has an area of pain in the scar that is usually well localized. This pain may limit the patient’s voluntary elbow extension, as elbow movement aggravates the pain. Palpation of the scar elicits radiating pain in the forearm distribution of the injured nerve. Additionally, the

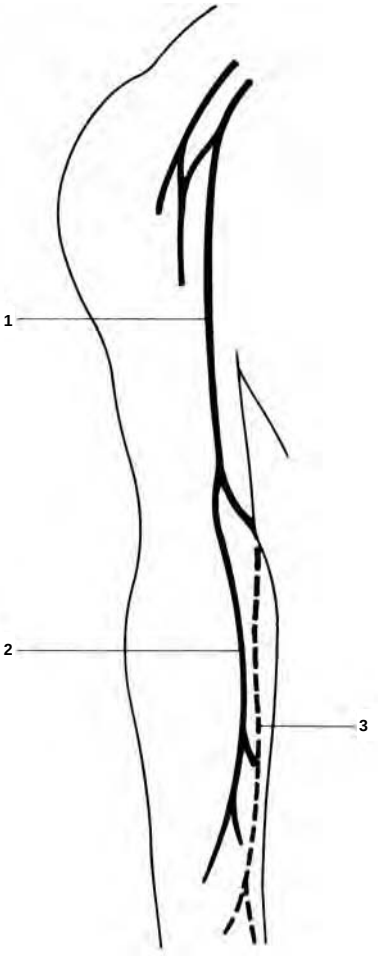

FIGURE 16.1 The medial antebrachial cutaneous nerve originates directly from the ulnar fascicle of the brachial plexus — 1: medial antebrachial cutaneous nerve; 2: ramus anterior; 3: ramus ulnaris.

paresthesias that result might prevent individuals from using the arm in their profession, or might trigger a reflex sympathetic dystrophy syndrome.

Diagnostic nerve block of the medial antebrachial cutaneous nerve is indicated and may relieve the pain and allow increased elbow range of motion if any preblock restricted movement is caused by pain. For a small group of patients, the stimulus of light touch to the area of the medial forearm or elbow results in an exaggerated and painful response. This pain may be so severe that even a shirt sleeve worn over the area causes discomfort. The pain is often described as burning or stinging. Associated with this hyperalgesia in the distribution of the medial antebrachial cutaneous nerve, there is usually considerable pain in the area of the scar. However, the compression of the medial antebrachial cutaneous nerve may occur with compression at the thoracic outlet.[2]

TREATMENT

The treatment always starts with a conservative approach. Occupational therapy can try to decrease the patient's sensitivity in the area of the postoperative scar using contrast baths, silicone gels or sheeting, contrast surfaces and rubs, and in application of local anesthetics or phonophoresis. If conservative therapy does not lead to success, surgical treatment, including exploration and excision

of neuroma, is indicated. The neuroma should be buried in muscle or bone or turned back on itself to avoid regeneration. Several techniques are available for review in current literature.

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17 Sulcus Ulnaris Syndrome

During its course down the arm, the ulnar nerve lies in a sulcus on the posterior surface of the medial humeral epicondyle. Compression of the ulnar nerve in this area presents with symptoms characteristic of the syndrome of the ulnar nerve sulcus, or cubital tunnel syndrome. Ulnar nerve compression at the elbow is commonly accepted as the second most frequently encountered nerve entrapment in the upper extremity, exceeded in prevalence only by carpal tunnel syndrome.[39]

ANATOMY

As shown in Figure 17.1, the ligament connecting the medial epicondyle to the olecranon covers the ulnar nerve, creating a fibro-osseus tunnel. According to Khoo et al.,[29] the space occupied by the ulnar nerve behind the medial epicondyle is the cubital tunnel. The roof of this tunnel was named the cubital tunnel retinaculum (CTR) by O'Driscoll et al.,[11] but is referred to by the eponymous Osborne band or the arcuate ligament of Osborne.[30] The cubital tunnel retinaculum may be absent (type 0); thin and not compress the nerve in full flexion (type Ia); thick and compress the nerve (type Ib); or replaced by the anconeus epitrochlearis muscle (type II).[29] This arcuate ligament of Osborne is about 4mm wide and serves to prevent subluxation of the ulnar nerve with forearm motion. The floor of the cubital tunnel is formed by the joint capsule and the posterior and transverse parts of the medial collateral ligament. The capacity of the cubital tunnel is known to decrease during elbow flexion, possibly leading to a dynamic compression of the ulnar nerve.[29,35] Distal to the cubital tunnel, the ulnar nerve branches to supply the motor innervation to the following muscles:

- Flexor carpi ulnaris
- The ulnar component of the flexor digitorum profundus
- The hypothenar muscles
- The interossei muscles
- The two most ulnar lumbricals
- The adductor pollicis
- The deep head of the flexor pollicis brevis
- Sensory branches to the fingers and hand originate distal to the sulcus.

ETIOLOGY

Trauma, the consequences of trauma, and rheumatic changes in the region of the medial epicondyle can compress the ulnar nerve by narrowing the fibro-osseus tunnel. Vidal et al.[1] describe two peaks: one between the ages of 20 and 30 years, with trauma as the predominating cause, and a second peak between the ages of 50 and 60 years, with rheumatic and degenerative joint disease predominating.

As an etiology, trauma can range from posttraumatic cubitus valgus to chronic nerve compression. Cubitus valgus can cause paresthesias and other sensory disturbances without affecting the cubital joint. Patients who lean on their elbows at work or while confined to bed can develop ulnar nerve compression at the elbow. The basic damage remains nerve damage, whether due to compression or direct contusions.

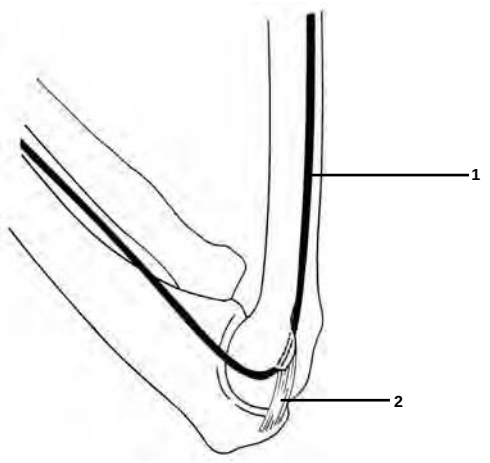

FIGURE 17.1 The ulnar nerve may be compressed proximal to the elbow near the medial epicondyle. A ligament connecting the medial epicondyle with the olecranon can be suspect — 1: ulnar nerve, and 2: epicondylo-olecranon ligament (cubital tunel retinaculum — CTR; arcuate ligament of Osborne).

Microtrauma or repetitive nerve stretches sustained in such varied activities as baseball, construction (jackhammers), boxing, or javelin throwing[2] may traumatize the ulnar nerve. Supracondylar fractures, distal humeral fractures, elbow dislocations, callus formation, and surgical exploration of these areas all may lead to direct and indirect trauma to the nerve in its tunnel. Rheumatic diseases lead to joint degeneration and distort the normal anatomy of the elbow. Rinaldi[3] and Akizuki and Matsui[4] describe the rare incidence of nerve compression caused by tophaceous gout. Ulnar nerve entrapment at the elbow by persistent epitrochleoanconeus muscle is described by Gessini et al.,[5] while Matsura et al.[31] described cubital tunnel syndrome caused by abnormal insertion of triceps brachii muscle. Thus, the ulnar nerve may be stretched or compressed along its course by multiple etiologies.[6,36] According to Artico et al.,[38] the cause of the nerve entrapment is idiopathic in about one quarter to one third of cases.

Compression of the ulnar nerve in the epicondylar or olecranon groove can be caused by a wide variety of lesions and conditions, which can be grouped into three categories: lesions within the groove, conditions outside the groove, and conditions that predispose the nerve to displace from the groove.[39]

While not a true tunnel syndrome, subluxation of the ulnar nerve, since it results in nerve compression, may present with similar symptoms; however, compression occurs secondary to an enlarged tunnel rather than a compressed one.[7] Described by many authors,[8–11] ulnar nerve subluxation occurs as the elbow flexes and the nerve glides medially to be tensioned against the medial epicondyle. Mumenthaler and Schliack[9] described subluxation secondary to traumatic edema of the nerve that forced the nerve to leave the tunnel.

The table below lists the hypotheses of several investigators as to the etiologies of ulnar nerve subluxation.

Cause	Author(s)
Rupture of the epicondylo-olecranon ligament	Platt, 1926;[12] Arkin, 1940;[13] Godshall and Hansen, 1971;[14]
Cubitus valgus	Marinescu and Danalia, 1968[15]
Shallow sulcus	Rolfsen, 1970[7]
Congenital anomalies of the medial epicondyle	Wachsmuth and Wilhelm, 1968[16]; Murakami and Komiyama, 1978[17]

CLINICAL SYMPTOMS AND SIGNS

In the first stage of the syndrome, paresthesias, hyperesthesia, hypesthesia, and pain develop in the sensory dermatome of the ulnar nerve. Paresthesias and pain also may be found proximally near the shoulder as the compression progresses. Tinel's sign, produced by tapping over the ulnar sulcus, may not be positive. Hypotrophy and atrophy might be found if the nerve compression remains unrelieved. Muscle wasting may be first seen in the web space between the first and second metacarpals, followed by interossei and hypothenar wasting. Loss of ulnar innervation will lead to a claw-hand appearance.

Radiographic studies allow visualization of rheumatic, arthritic, or posttraumatic changes around the elbow. Serology and laboratory testing will distinguish between several rheumatologic etiologies, if present. Electromyography and nerve conduction velocity studies determine the location of the compression. High-resolution ultrasonography can detect morphologic changes in the ulnar nerve accurately, and it could therefore be useful as a screening and even follow-up modality in patients with cubital tunnel syndrome.[32]

TREATMENT

Conservative therapy remains similar to that of many other tunnel syndromes: rest, splinting,[18] removal of the causative agent, and local corticosteroid injection. Splints include nighttime extension splints, elbow pads, and reversed elbow pads that limit flexion. These splints try to prevent repetitive nerve irritation. The sulcus may be injected into the center of an imaginary line connecting the medial epicondyle to the median edge of the olecranon. Surgical treatment consists of releasing the ligament connecting the epicondyle and the olecranon, ligament release and medial epicondylectomy,[19–23] or anterior transposition of the ulnar nerve. First performed successfully in 1898 by Curtis,[24] transposition has been successfully adopted by many surgeons,[1,25–27,34] including the authors, but some authors consider that cubital tunnel syndrome does not require transposition of the ulnar nerve.[33] Greenwald et al.[37] demonstrated the effectiveness of surgical therapy in patients with cubital tunnel syndrome identified by clinical examination without electrodiagnostic testing. Studying clinical and electromyographical results following various surgical decompressions of the ulnar nerve, Deutinger et al.[28] found that anterior transposition of the ulnar nerve yielded the best results. One should note that surgical decompression must occur before permanent nerve damage results; otherwise, the type of surgical release performed will not change the outcome. Marin Braun and Foucher[23] reviewed 51 patients operated on for ulnar nerve entrapment at the elbow. With an average follow-up of 4.6 years, 39% of the patients were cured, 27% improved, 31% were unchanged, and none worsened. According to Posner,[40] a variety of operative procedures have been described in the literature. Deciding on the most effective procedure can be difficult, given the excellent results claimed by proponents of each. Unfortunately, there is a paucity of information based on prospective randomized clinical studies comparing the different surgical methods. Black et al.[41] described the operative technique of stabilized subcutaneous anterior transposition of the ulnar nerve with immediate range of motion. Patients were able to return to their occupations sooner when their affected elbows had been mobilized immediately.

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18 Pronator Teres Muscle Syndrome

The median nerve leaves the cubital fossa (fossa cubiti) by passing between the two heads of the pronator teres muscle and under the tendinous arch of the flexor digitorum superficialis (FDS) muscle (Figure 18.1). Compression in this region produces sensory-motor symptoms of tunnel syndrome, the syndrome of the pronator teres muscle.

ANATOMY

The median nerve passes 2.5 to 4 cm below the level of the medial epicondyle between the humeral and ulnar head of the pronator teres muscle. As demonstrated by Ilic, Lolic, and Dimcevic[1] and Beaton and Anson,[2] the median nerve varies in its course through the pronator teres (Table 18.1). Upon leaving the pronator teres, the median nerve gives rise to the anterior interosseous nerve.

Having transversed the pronator teres muscle, the median nerve divides under the tendinous arch of the FDS into the layer between the FDS and the flexor digitorum profundus (FDP). It later runs between the FDS and the FDP and then between the flexor carpi radialis (FCR) and the palmaris longus (PL) to reach the carpal tunnel. In the forearm, the median nerve branches multiple times to supply all wrist and hand flexors, except the flexor carpi ulnaris and the ulnar portion of the FDP. Prior to entering the carpal tunnel (5 to 8 cm), the median nerve gives rise to the palmaris

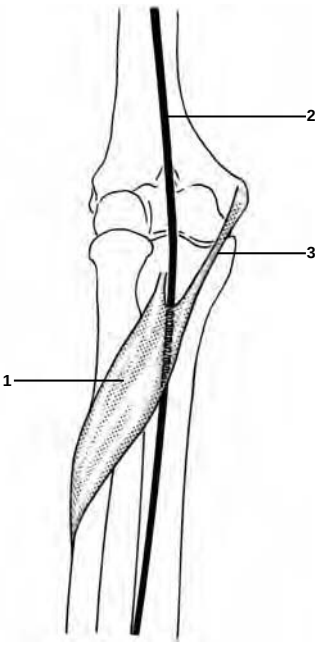

FIGURE 18.1 The median nerve can run through the pronator teres muscle or between its heads during its course into the hand — 1: pronator teres muscle; 2: median nerve; 3: humeral head of the pronator teres muscle.

TABLE 18.1
Variations of the Median Nerve in its Course Through the Pronator Teres Muscle

Humeral and Ulnar Heads Both Present		Ulnar Head Missing	
Location	**Percent**	**Location**	**Percent**
Between the heads	56	Behind humeral head	25
Behind the heads	11	Pierces muscle	3
Through humeral head	3		
(per Beaton and Anson, 1939[2])	2		
Through ulnar head	2		

branch, which pierces the forearm fascia to innervate the thenar eminence and the radial aspect of the wrist.

ETIOLOGY

Multiple etiologies have been proposed to account for median nerve compression in the region of the pronator teres muscle (Table 18.2). These etiologies have a common denominator: mechanical compression secondary to static or dynamic stenosis. The presence of a fibrous band or a scarred lacertus fibrosus can create a static compression.[3] Nebot-Cegarra et al.[4] proposed the following four anatomical variations responsible for development of pronator teres syndrome:

- Short and tendinous ulnar head
- Ulnar head joined to the arch of the FDS
- Ulnar head with three origins
- Humeral head perforated by the median nerve

Lacey and Soldatis[5] describe bilateral pronator syndrome associated with anomalous heads of the pronator teres muscle.

Trauma leading to Volkmann's contracture or prolonged external compression as in "honeymoon paralysis" have been noted to produce median nerve symptoms. Stal et al.[31] describe pronator syndrome in 23 female users of milking machines, mostly in the hands, which usually were statically loaded with heavy equipment. Many investigators propose a dynamic compression of the median nerve during supination or elbow extension (Figure 18.2). In this arm position, the tendinous portions of the muscle heads approach each other and compress the nerve.[6] In a study of 16 patients explored surgically, Hartz et al.[7] found 15 to have a compressive aponeurotic prolongation of the biceps brachii and 13 to have a compressive tendinous arch of the FDS. According to Hill and Hall,[33] the median nerve at pronator arch was constricted at the site of potential entrapment; appeared swollen proximal to this site, or exhibited neither swelling nor constriction.

CLINICAL SYMPTOMS AND SIGNS

Subjective symptoms and objective signs are present in the whole area of median nerve innervation distal to the site of compression. In contrast to carpal tunnel syndrome, which characteristically involves the muscles of the thenar eminence, the pronator teres syndrome involves not only the thenar muscles, but also the wrist and finger flexors. Patients will complain of impaired thumb, index finger, and middle finger flexion. Sensory disturbances occur along the volar and dorsal surfaces of the hand, the palm, and several fingers.[8]

TABLE 18.2
Proposed Causes for the Syndrome of the Pronator Teres Muscle

Etiology	Author(s)
Myositis (irritating nerve)	Seyffarth, 1951[15]
Fibrous band	Fearn and Goodfellow, 1965[16]
	Johnson et al., 1970[3]
	Thompson and Kopell, 1959[21]
	Kopell and Thompson, 1963[17]
	Pesserini and Valli, 1968[19]
	Seyffarth, 1951[15]
	Sharrard, 1968[20]
	Kopell and Thompson, 1963[17]
	Ollivierre et al., 1995[18]
Forearm trauma	Komar, 1977[22]
	Thompson and Kopell, 1963[17]
	Anto and Aradhya, 1996[30]
Dynamic relationship of the nerve and muscles in the forearm; Strenuous activities; occupations (repetitive pronation of the forearm with the elbow extended)	Bora and Osterman, 1982[23]
	Domljan, 1969[10]
	Pećina, 1979[6]
	Thompson and Kopell, 1959[21]
	Lubahn and Cermak, 1998[32]
Anatomical (at the aponeurosis of the FDS and biceps brachii; humeral head perforated by the median nerve; hypertrophy of the muscle)	Hartz et al., 1981[7]
	Martinelli et al., 1982[24]
	Flory and Berger, 1985[25]
	Olehnik et al., 1994[26]
	Spinner et al., 1991[27]
	Tulwa et al., 1994[28]
	Anto and Aradhya, 1996[30]

Since the median nerve gives off the palmaris branch prior to entering the carpal tunnel, sensory disturbances in the palm indicate compression proximal to the carpal tunnel. This is an important diagnostic sign for differentiating between carpal tunnel syndrome and pronator teres syndrome.

Spinner[9] described muscle stressing tests to localize the area of compression and entrapment. Pain and paresthesias evoked by resisted pronation of the forearm with the elbow extended implicates the heads of the pronator teres; simultaneous resisted flexion of the elbow and supination of the forearm implicate the lacertus fibrosus; and resisted flexion of the proximal interphalangeal joint of the middle finger implicates the arch of the FDS muscle belly. Pain and a Tinel's sign can also be elicited by direct pressure to the area between the two heads of the pronator teres; therefore, patients can present after pushing cars with extended arms,[6] sudden lifting of heavy burdens,[10] or doing jobs that require repetitive pronation and supination.

Beyond basic clinical tests, several technical tools are available. Radiographic studies may be able to identify posttraumatic lesions in the cubital region. Electromyographic examination will reveal motor unit damage and decreased conduction velocity of the FCR, FDS, and FPL. Mysiew and Colachis[11] evaluated the dynamic maneuvers improving electrodiagnostic sensitivity.

TREATMENT

Since the majority of pronator syndromes are intermittent and mild, conservative treatment should be tried initially.[9] Treatments consist of reduction in physical activity, forearm immobilization for a limited time in neutral between supination and pronation, and local corticosteroid injection.[12] If

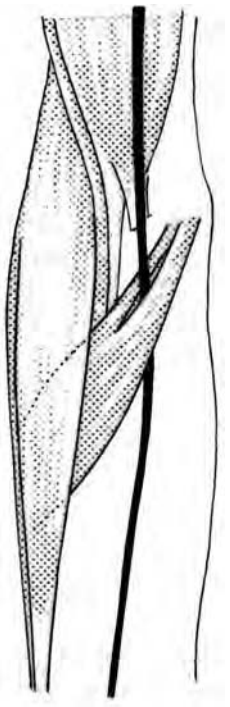

FIGURE 18.2 This figure demonstrates the anatomical changes during pronator teres muscle activity that brings its two heads together; therefore, the median nerve loses its space to run without compression.

symptoms persist, surgical decompression is required, preferably within 6 months of presentation. Wide exposure of the proximal forearm allows not only identification and release of the compressive structure, but also neurolysis of the median nerve, if necessary. Haussmann and Patel[29] recommend decompression of the nerve fascicles by epineurotomy, microsurgical interfascicular dissection, and removal of the constricting outer layer of the perineurium above and below the elbow. The tendinous arch between the origins of the FDS should be sectioned with release of the humeral head of the pronator teres from its radial insertion. Reviewing the results of surgical decompression in[39] patients, Hartz et al.[7] found 87% to have satisfactory results. Because the skin incision can leave an unsatisfactory scar, Tsai and Syed[13] use a transverse skin incision, and Gainar[14] uses two off-set linear incisions to modify the scar.

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19 Supinator Syndrome

The terminal motor branch of the radial nerve, the posterior interosseous nerve, passes under the tendinous arch of the supinator muscle. The nerve compression results in the clinical picture of the supinator syndrome described by Kopell and Thompson[1] and Mumenthaler and Schliack.[2] This syndrome has also been known as **posterior interosseous nerve paralysis** or **traumatic progressive paralysis of the deep branch of the radial nerve**. Roles and Maudsley[3] describe this syndrome as **radial tunnel syndrome**, since they define the radial tunnel as the course of the radial nerve, from its piercing of the lateral intermuscular septum through the radial cubital sulcus to its entrance into the supinator canal.

ANATOMY

Having perforated the lateral intermuscular septum, the radial nerve passes from posterior to anterior in the brachial sulcus and enters the sulcus cubitalis radialis, which lies between the brachialis and brachioradialis muscles. At the level of the capitellum, the radial nerve gives branches to the brachialis, brachioradialis, and extensor carpi radialis longus muscles, the periosteum of the lateral epicondyle, the humeroradial joint, and the annular ligament.[4] Within the radial cubital sulcus, the radial nerve divides into two terminal branches, the deep branch (ramus profundus) and the superficial branch (ramus superficialis). The superficial branch of the radial nerve supplies sensory innervation to the dorsum of the hand, the first two fingers, and the radial half of the third finger.

The deep branch enters distal to the origin of the extensor carpi radialis brevis (ECRB) in the supinator canal (Figure 19.1). Spinner[5] noted that the margin of the ECRB during pronation may dynamically compress the deep branch before its entrance into the supinator canal. According to investigations of Laulan et al.,[6] the medial edge of the ECRB is a real fibrous arch in 95% of cases and crosses over the posterior interosseous nerve 9mm more proximally than the arcade of Frohse. Before passing between two layers of the supinator muscle, the deep branch passes under the tendinous arch of the superficial supinator muscle layer, Frohse's arcade.[7] Frohse's arcade may exist in only 30% of adults and not at all in the fetus.[5] Entering the canal, the deep branch supplies the ECRB and the supinator muscles. The deep branch continues its lateral course around the radius to reach the dorsum of the forearm. At the distal edge of the supinator muscles, the deep branch typically divides into two divisions:

1. The muscular branch to the extensor carpi ulnaris, the extensor digitorum communis, and the extensor digiti minimi muscles.
2. The posterior interosseous nerve to the abductor pollicis longus, the extensor pollicis longus, the extensor pollicis brevis, and the extensor indicis muscles. The terminal branches also supply the ligaments and the capsule of the wrist.

ETIOLOGY

As listed in Table 19.1, multiple agents can compress the radial nerve in the supinator tunnel; however, dynamic compression due to muscular activity and anatomical relations has become one

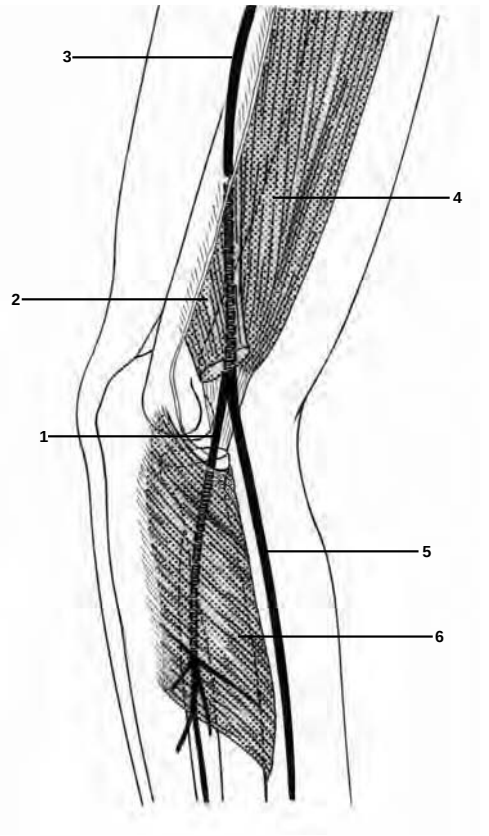

FIGURE 19.1 The radial nerve runs in proximity to multiple muscle bellies in the arm and forearm. Along its course, the nerve or its branches risk compression — 1: deep branch (profundus) of the radial nerve; 2: brachioradialis muscle; 3: radial nerve; 4: brachialis muscle; 5: superficial branch of the radial nerve; 6: supinator muscle.

of the currently favored etiologies.[8–11] Compression and stretching of the nerve over Frohse's arcade occurs with repeated pronation, forearm extension, and simultaneous wrist flexion. Multiple activities have been implicated. Kopell and Thompson[1] propose that ECRB compression could lead to the supinator syndrome. Describing 10 years of experience with radial tunnel syndrome, Ritts et al.[12] found the compressive agent to be the arcade of Frohse in 34 cases (57%), the ECRB in 12 cases (20%), the leash of recurrent radial vessels in eight cases (13%), and the fibrous bands anterior to the radial head in six cases (10%). Sponseller and Engber[13] describe compression of the posterior interosseous nerve at both the entrance to the supinator canal and the exit from the supinator muscle. Rahimizadeh[14] describes two patients with delayed radial nerve palsy caused by a traumatic aneurysm of a collateral radial artery.

Thus, in summary, anatomical compressive etiologies include the following: thickened fascia superior to the elbow capsule, the leash of Henry (vessels), the fibrous edge of the ECRB, and the supinator muscle (proximal arcade of Henry, distal fibrous edge).[51,57]

CLINICAL SYMPTOMS AND SIGNS

Deep pain in the posterior part or dorsum of the forearm, followed by gradual fist weakness and local pain on compression distal to the lateral humeral epicondyle, compose the clinical picture of

TABLE 19.1
Proposed Agents that Could Compress the Radial Nerve in the Supinator Tunnel

Cause	Author(s)
	General Trauma
Radial Subluxation	Grigoresco and Jordanesco, 1931[34]
Monteggia Fracture	Spinner et al., 1968[27]
Distal Humeral Fracture	Esposito, 1972[35]
Violent Motion	Sharrard, 1966[26]
	Tumors
Fibromas, Lipomas, Ganglions, Multiple Myeloma, Myxoma	Capellini, 1958;[36] Mulholland, 1966;[37] Richmond, 1953;[38] Bowen and Stone, 1966;[39] Goldman et al., 1969;[16] Catanzariti and Cesari, 1968;[40] Blakemore, 1979;[41] Rayan and Comer, 1982;[42] Ogino et al., 1991;[18] Steiger and Vogelin, 1998;[53] Valer et al., 1993[43]
	Inflammation
Neuroma	Whiteley and Alpers, 1959[44]
Bursitis	Weinberger, 1939[45]
Rheumatoid Arthritis	Marmor et al., 1967;[46] Chang et al., 1972[47]
	Anatomical Position
Dynamic Compression	Koppel and Thompson, 1963;[1] Spinner, 1968;[5] Espositio, 1972;[35] Younge and Moise, 1994;[10] Portilla et al., 1996[11]
Conductor	Ritts et al., 1987[12]
Balloonists	Guillain and Courtellemont, 1905[48]
Violinists	Silverstein, 1937[49]
Swimmers	Kruse, 1958[50]

the supinator syndrome. Unilateral and gradual in appearance, the syndrome has been described by Komar[15] as first giving rise to finger weakness, with the thumb being the last digit affected. There are no sensory deficits, since the posterior interosseous nerve has no cutaneous sensory component. The superficial branch of the radial nerve contains the sensory fibers and branches before the radial nerve enters the supinator tunnel. Since branches to the brachioradialis and the extensor carpi radialis longus muscle also leave before the tunnel's entrance, elbow and wrist motion remain unaffected. If the compression is stronger or longer in duration, the hand adopts the position of a hanging hand, characteristic of radial nerve paralysis.[16] Symptoms are aggravated by the simple motion of wringing a wet cloth.

In spite of a characteristic picture, the supinator tunnel syndrome often is not recognized or is mistaken for lateral epicondylitis (tennis elbow).[19–22] These two syndromes may appear together; Moss and Switzer[23] have described a variety of symptoms associates with resistant tennis elbow and resistant radial tunnel pain. The same motions of pronation, forearm extension, and wrist flexion common to the supinator syndrome also occur at the end of the serve in tennis. Pain over the lateral epicondyle with resisted middle finger extension with an extended arm can be confused with the local tenderness distal to the lateral epicondyle found in a patient with supinator syndrome. Direct recording of local pressure in the radial tunnel during passive stretch and active contraction of the supinator muscle was described by Werner et al.[24] without evidence of the dynamic compression of the nerve. The same was concluded by Verhaar and Spaans[25] on the basis of electrophysiological investigations.

While electromyographic studies can show neurological compromise, Zeuke et al.[17] believe that surgical treatment can be based on the characteristic clinical picture alone. Barnum et al.[55] did not

find electrodiagnostic studies to be conclusive. In some patients, ultrasonography, computed tomography, and magnetic resonance imaging reveal the cause of the nerve compression in the supinator tunnel.[18] Suematsu and Hirayama[54] characterized posterior interosseous nerve compression based on the effect on the hand. Type I, affecting the fingers and thumb, had compression along the supinator. Type II, affecting the fingers (recurrent branch), and Type III, affecting the thumb (descending branch) were compressed at the distal edge of the supinator.

TREATMENT

Treatment seeks to avoid repetitive trauma to the nerve in the tunnel. Physical therapy and local corticosteroid injections give good results if dynamic compression is not a factor. In our opinion, the nerve should not be injected. An anesthetic block may help with diagnosis. Surgical treatment should not be postponed too long, since irreversible nerve damage could occur.[26–28] Palazzi et al.[29] recommend surgical intervention within the first 4 months of symptoms. Roles and Maudsley[3] recommended decompression of the deep branch of the radial nerve in the supinator tunnel when treating resistant epicondylitis. Barnum et al.[55] recommended release of all constricting areas through the brachioradialis-ECRL interval. Believing that 30% of patients with radial epicondylitis have posterior interosseous compression, Jalovaara and Lindholm's primary surgical approach is decompression of the posterior interosseus nerve when presented with resistant lateral epicondylitis.[30] Raimbeau et al.[31] describe excellent results after surgical treatment, while Atroshi et al.,[32] on the basis of 37 patients with radial tunnel release followed for 1 to 5 years, concluded that the symptoms and signs used as diagnostic criteria for radial tunnel syndrome may be unreliable; therefore, the results of posterior interosseous nerve decompression may be unpredictable. Surgical results vary and expectation must be guarded, especially in work-related cases and chronic pain.[52,57]

A case of bilateral thoracic outlet syndrome combined with bilateral radial tunnel syndrome was reported.[33] Persisting complaints in the upper extremities after bilateral first-rib resection and scalenotomy were caused by radial nerve entrapment in the radial tunnel. Although this bilateral double-crush phenomenon is extremely rare —in fact, has not been reported previously — persistence of symptoms after initial treatment of nerve entrapment is an indication to search for another site of compression.

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20 Anterior Interosseous Syndrome

A motor branch of the median nerve in the cubital region, the anterior interosseous nerve (AIN), risks compression throughout its course in the forearm (Figure 20.1). Kiloh and Nevin[1] first described two patients with classical prevention of the decreased function of the distal phalanx of both the thumb and the index finger.

ANATOMY

From the posterior surface of the median nerve 2 to 8 cm distal to the medial humeral epicondyle originates an exclusively motor nerve branch, the anterior interosseous (palmaris) nerve. While initially paralleling the median nerve, the AIN soon dives under the deep fascial layer of the flexor digitorum superficialis (FDS) to run along the interosseous membrane between the flexor pollicis longus (FPL) laterally and flexor digitorum profundus (FDP) medially. It is accompanied by interosseal vessels. Distally, the nerve ends under the pronator quadratus (PQ), branching 2 to 5 cm distal to its origin. The anterior interosseous nerve supplies the FPL, the FDP to the second finger, and the PQ.[2] Some authors mention the possibility of direct innervation by the median or ulnar nerve. As a separate entity or a component, the AIN may accompany the median nerve and course between the heads of the pronator teres or between the tendinous arch of the FDS. Because of these anatomical relationships, some authors[3,4] do not differentiate the pronator teres syndrome from the anterior interosseous syndrome; however, the Kiloh-Nevin syndrome defines only anterior interosseous nerve involvement, and thus is only a motor deficiency.

ETIOLOGY

The description of the anterior interosseous syndrome as a tunnel syndrome remains a question of definition. In which tunnel does the nerve run? Several authors, as shown in Table 20.1, have reported varied etiologies for nerve compression.

These etiologies can range from median nerve tumors that cause isolated paralysis to fibrous anomalies of the FPL and FDS. Englert[5] and Haussmann[6] have described this syndrome as arising without any external compression. Operatively, the fibers of the AIN have been found to be compressed within the main trunk of the median nerve proximal to the elbow. These two cases of intratruncal fascicular compression were treated in one case by neurolysis and in the other by translocation.

Suso et al.[7] describe Kiloh-Nevin syndrome resulting from an extrinsic compression of the AIN from a Robert-Jones type bandage in a patient with a distal-end clavicle fracture. Compression of the AIN after use of a sling for dislocation of the acromioclavicular joint (two cases) was reported by Vailas.[8] Enzenauer and Nordstrom[9] describe the AIN syndrome associated with forearm band treatment of lateral epicondylitis.

Penkert[10] mentions the possibility that trauma along the course of the nerve may lead to segmental demyelination and paresis. Paresis disappears gradually with remyelinization. While the anterior interosseous nerve may be compressed as it pierces the fascia of the FDS, most authors reject this hypothesis, because this fascia is a loose structure. Because the nerve passes under the same radial portion of the tendinous arch as the FDS, Luppino et al.[2] and Penkert[10] describe the

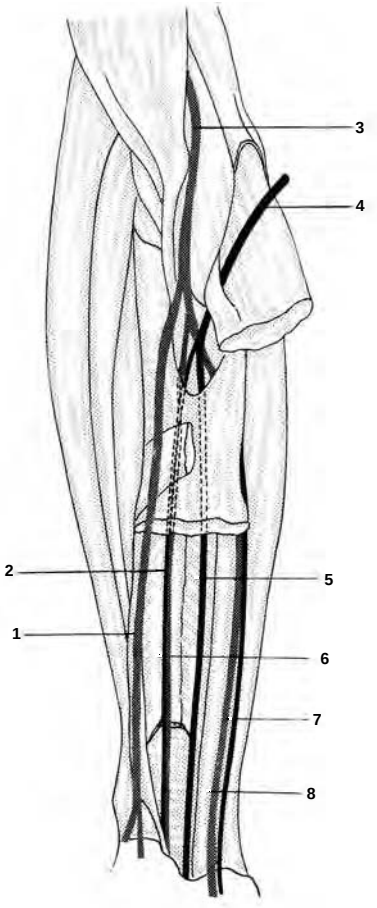

FIGURE 20.1 The forearm contains multiple muscle groups that are intertwined with the entire neurovascular supply to the hand. The proximity of all of these structures dictates that variations in the normal structure of one will affect the other associated structures — 1: radial artery; 2: anterior interosseous nerve; 3: brachial artery; 4: median nerve; 5: median nerve; 6: anterior interosseous nerve; 7: ulnar nerve; 8: ulnar artery.

upper margin of this arch as the compressive agent of the tunnel. Nerve recovery after arch sectioning confirms this hypothesis. However, as Eren et al.[11] described, only half of the 40 surgical patients reported in the literature had identifiable compressive causes.[10–19]

Spinner et al.[20] present an entrapment of the median nerve caused by compression in the distal arm because of an accessory bicipital aponeurosis. It is important to differentiate this syndrome from the classic anterior interosseous syndrome, and electrodiagnostic studies are also important in establishing the site of the entrapment. Carmant and Veilleux[21] describe AIN syndrome that occurred in two young women within the month following parturition. A patient with AIN syndrome was found at operation to have the median artery passing through the AIN just below the elbow.[22] Wong and Dellon[44] describe a patient with brachial neuritis presenting as anterior interosseous nerve compression syndrome.

CLINICAL SYMPTOMS AND SIGNS

While developing acutely or gradually, the clinical picture is manifested by an inability of the patient to pinch between his thumb and index finger. This characteristic pinch sign (Figure 20.2)

TABLE 20.1
Various Etiologies for Nerve Compression

Etiology	Author(s)
Trauma/forearm fractures	Warren, 1963;[34] Luppino et al., 1972;[2] Penkert, 1983;[10] Seror, 1996;[43] Joist et al., 1997[45]
Post-traumatic thrombosis of the antibrachial vessels	Haussmann, 1982[6]
Vascular anomalies	Eren et al., 1983;[11] Proudman and Menz, 1992;[22] Franzini et al., 1995[35]
Neuroma of the median nerve	Spinner, 1970[30]
Anatomical anomalies	Spinner, 1970;[30] Benini and Tedeschi, 1974;[36] Gardner-Thorpe, 1974;[37] Leven and Hauffman, 1976;[38] Chan and Lamb, 1984;[39] Spinner et al., 1991[20]
Intratruncal fascicular compression	Englert, 1976;[5] Haussmann, 1982;[6] Haussmann and Patel, 1996[42]
Segmental demyelination	Penkert, 1983[10]
Metastatic tumor	Peters and Todd, 1983[40]

develops from impaired flexion of the terminal phalanges of the thumb and of the index finger. Thus, the patient pinches with extended distal interphalangeal joints. Opposition of the thumb and finger flexion of the 3rd, 4th, and 5th digits remains intact; however, the ability to write is usually lost.[23] Additionally, the patient cannot clench his fist (Figure 20.3). The syndrome may be complete with both the thumb and finger affected, or incomplete with either the thumb or finger affected. Hill et al.[24] have reported the largest series of incomplete syndromes with a total of 26 patients from both the U.S. and Canada. Occasionally, the patient will have weakness of the pronator quadratus muscle (PQ). The PQ must be examined with the elbow flexed to eliminate the effect of the pronator teres.[25] Patients feel dull pain in the proximal third of the forearm that is aggravated by radial pressure at the level of the tendinous arch of the FDS. Characteristically, there is no sensory loss, nor are there symptoms. The presence of sensory symptoms should indicate median nerve compromise.

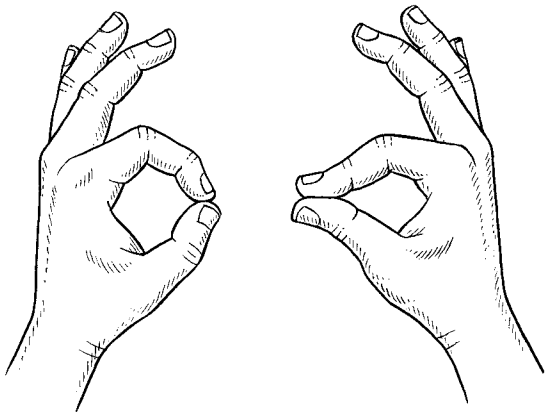

FIGURE 20.2 Comparison of the pinch of an unaffected hand with the pinch of a hand where the anterior interosseous nerve is compromised (pinch sign).

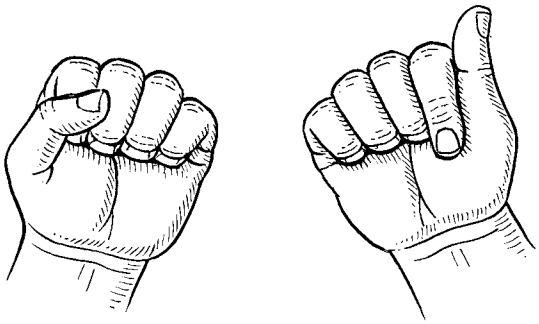

FIGURE 20.3 Inability to form a fist because of problems incorporating the thumb and index finger may give the investigator a solid clinical sign for nerve compression.

Electromyography may show not only denervation of the affected muscles, but also reinnervation if it occurs. Eren et al.[11] have found a tendency for spontaneous recovery within 6 months; however, Farber and Bryan[26] did not find denervation in clinically manifest AIN syndrome.

TREATMENT

As in most tunnel syndromes, conservative therapy may be applied early in the course of the disease, with a time limit for achieving relief. This limit is typically 6 months after the onset of symptoms if there is no evidence of recovery, either clinically or electrodiagnostically. Eren et al.[11] found that five of seven patients in their series recovered spontaneously. Therefore, in the absence of a definitive cause of compression, conservative therapy may allow time for re-innervation.[27,41]

Assmus et al.[28] recommend surgical decompression if there is no evidence of recovery within 2 months. Other surgeons extend their follow-up to 3 to 6 months, with electromyographic evaluation.[29,30,46] Surgical treatment might include exploration with fibrous band or tendon release, removal of anomalous muscles, or neurolysis.[30-32] If no improvement occurs after decompression, tendon transfer should be performed.[46] Schantz and Riegels-Nielsen[33] conclude that exploration of the AIN is the treatment of choice. Of 15 patients, the AIN was explored with objective signs of compression in 9; 11 of these patients showed satisfactory function, and three had a tendon transfer.

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21 Flexor Carpi Ulnaris Muscle Syndrome

Commonly known by some as the cubital tunnel syndrome, the syndrome of the flexor carpi ulnaris muscle includes ulnar nerve compression, since it courses not only in its sulcus behind the medial humeral epicondyle, but also between the two heads of the flexor carpi ulnaris muscle. However, Fiendel and Stratford[1] described ulnar nerve compression in the latter situation as the cubital tunnel syndrome. To avoid confusion, this book will use the more inclusive definition to pinpoint accurately the region in question.

ANATOMY

The ulnar nerve leaves the ulnar sulcus behind the medial humeral epicondyle and passes between the humeral and ulnar heads of the flexor carpi ulnaris (Figure 21.1) as the nerve enters the forearm. The arcuate ligament, a tendinous arch that is a continuation of the fibroaponeurotic covering of the epicondylar groove,[45] connects these heads and defines the tunnel's entrance and roof. Extending from the medial epicondyle to the medial border of the olecranon, the triangular arcuate ligament additionally serves as a common origin for both the humeral and ulnar heads of the flexor carpi ulnaris muscle. The ulnar nerve passes distally within the flexor carpi ulnaris and, approximately 5 cm beyond the medial epicondyle, it penetrates the flexor-pronator aponeurosis, the fibrous common origin of the flexor and pronator muscles.[44]

Once in the forearm, the ulnar nerve lies between the flexor digitorum superficialis, the flexor digitorum profundus, and the flexor carpi ulnaris muscles. The ulnar vessels join the ulnar nerve in this region.

Sensory and motor branches leaving the canal at the level of the origin of the flexor carpi ulnaris muscle are identical to those branches originating in the ulnar sulcus behind the medial epicondyle; therefore, the differential diagnosis between compression in the ulnar sulcus and the flexor carpi ulnaris becomes difficult.

ETIOLOGY

The anatomical relationships around the elbow affecting the ulnar nerve, both in the ulnar sulcus and between the two heads of the flexor carpi ulnaris muscle, are similar. Therefore, several of the etiologies are similar. However, an idiopathic form of the syndrome of the flexor carpi ulnaris relies on the anatomical relationship of its heads and its tendinous arch. First described by Feindel and Stratford,[1] the fibrous arch compresses the nerve as elbow flexion tenses the arch. Several other investigators have described the importance of the arch.[2–4] Consistent with Esposito's[5] finding of a 10% tunnel volume reduction in flexion, cadaver studies have demonstrated an increase in compartment pressures from 7 to 24 mmHg when the elbow changes from extension to flexion.[6] Using this finding, Buehler and Thayer[7] have endorsed the "elbow flexion test" as a clinical test for nerve compression. Vanderpool et al.[8] described a change in the anatomical head relationship when the elbow flexes. Extrapolating from this finding, it is understandable how this syndrome might appear more frequently in those who work for many hours with their elbows flexed, as described by Kenneth.[9] Another cadaver study by Amadio and Beckenbaugh[10] found that the deep

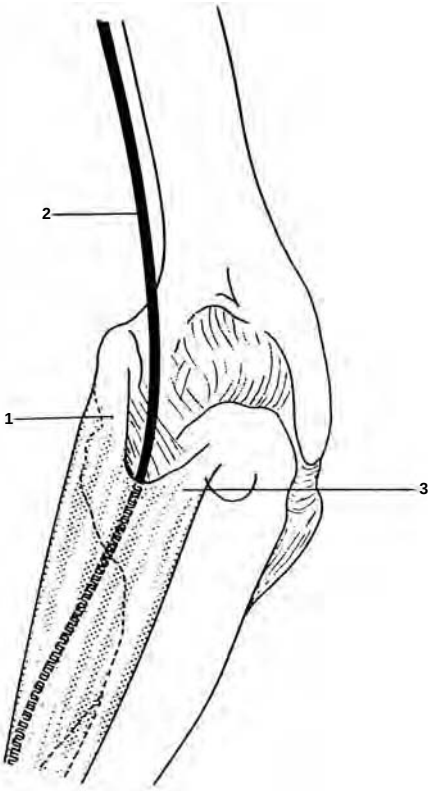

FIGURE 21.1 The ulnar nerve also may been compressed just distal to the elbow by the heads of the flexor carpi ulnaris muscle — 1: humeral head of the flexor carpi ulnaris muscle; 2: ulnar nerve; 3: ulnar head of the flexor carpi ulnaris muscle.

aponeurosis of the flexor carpi ulnaris muscle was a potential site for nerve compression. Green and Rayan[47] found that elbow flexion increases the pressure in the "distal tunnel" and that releasing the arcuate ligament alone does not decompress the ulnar nerve in the distal tunnel.

While functional anatomical changes may account for many of the patients with this syndrome, other etiologies have been postulated. Thompson and Kopell[11] emphasize the importance of external trauma, since the nerve lies quite superficially. Arthritis of the elbow may produce a compressive proliferative synovitis and bony osteophytes. Konishüke et al.[12] describe cubital tunnel syndrome in a patient in long-term hemodialysis. Loose bodies and other synovial changes have also been submitted as causes. Tumors, ganglions, perineural cysts, and lipomas,[13] in addition to anomalous muscles[14] and variations in the anconeus muscle, could be rare causes of nerve compression.[15] Vidal et al.[16] described three cases that did not fit any aforementioned etiology.

CLINICAL SYMPTOMS AND SIGNS

Gradual development of paresthesias, pain, and muscle weakness have led to the description of this syndrome as tardy ulnar nerve palsy. In the end stage, paresis of ulnar innervated musculature leads to the development of the **Froment's sign** (Figure 21.2). This sign requires dysfunction of the adductor pollicis muscle. While having the patient hold a piece of paper between the thumb and index finger, the physician will observe compensation by the flexor pollicis longus muscle. Thus, the interphalangeal joint of the thumb will be held in flexion. While this finding is diagnostic of ulnar nerve damage, atrophy and this sign occur more frequently in the syndrome of the flexor

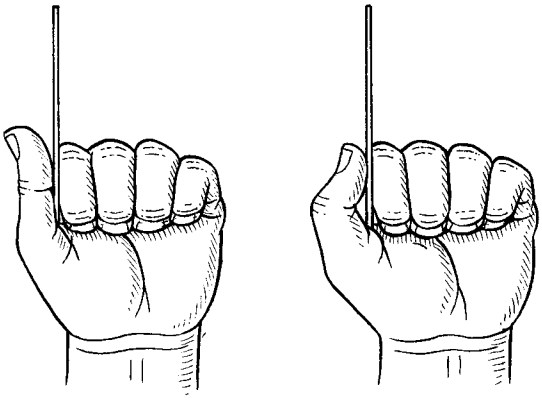

FIGURE 21.2 Froment's sign.

carpi ulnaris muscle than in any other ulnar nerve compressive syndrome.[17] According to Novak et al.,[18] the most sensitive provocative test in the diagnosis of cubital tunnel syndrome is elbow flexion when combined with pressure on the ulnar nerve. A Tinel's sign will be appreciated more distally in this syndrome than in the syndrome of the ulnar sulcus. Radiographic studies and a thorough history may indicate when prior fractures in development have led to an elbow deformity that alters the course of the nerve.[19] Magnetic resonance imaging is not essential for either diagnosing a neuropathy or determining appropriate treatment. Perhaps in the future, with continuing technical advancements, it will become more useful for detecting early nerve damage.[45] Electromyographic studies will usually indicate a pathology near the elbow. Ushida and Sugioka[20] describe the value of electrophysiological examination of the flexor carpi ulnaris muscle in the diagnosis of cubital tunnel syndrome. Szendroi et al.[21] postulate that Dupuytren's contracture is a late consequence of this syndrome. According to Folberg et al.,[22] cubital tunnel syndrome can arise without an obvious compression injury or can be secondary to nerve enlargement or narrowing of the cubital tunnel.

Neurologic symptoms and signs similar to ulnar nerve entrapment can be caused by brachial plexus compression from thoracic outlet syndrome or an occult apical lung tumor. However, neither a Tinel's sign nor a localized electromyographical change will be appreciated at the elbow.[23] This absence will help with the differential diagnosis. Epicondylitis may be mistaken for this syndrome;[24] however, epicondylitis (or epitrochleitis, since the medial epicondyle lies above the humeral trochlea) presents as a localized pain on palpation without paresis or sensory changes in the ulnar nerve distribution.

TREATMENT

Advised for patients with intermittent mild symptoms without neurological deficits,[25] conservative therapies include short-term immobilization, physical therapy, and local corticosteroid infiltration. Splinting can include night extension splints and daytime flexion blocking pads. Where conservative therapy fails or more definite neurological signs of compromise are present, one must proceed immediately with surgical release.[26] Except with respect to sectioning of the tendinous arch of the flexor carpi ulnaris muscle, no absolute consensus exists with respect to what release to perform; therefore, the releases performed are as follows: anterior subcutaneous or submuscular transposition of the ulnar nerve,[27–30] medial epicondylectomy,[31] and simple tunnel decompression if this is the only site of entrapment.[32,33] Release of the flexor's tendinous arch usually disturbs the ligament connecting the olecranon and epicondyle. Because compression in the ulnar sulcus can masquerade as flexor carpi ulnaris muscle compression, decompression in both locations and anterior translo-

cation of the ulnar nerve are recommended. If ulnar nerve decompression is not verified, many patients will continue to have symptoms and ulnar nerve impairment. Reoperation is more difficult, with a greater risk of complications. Higher success rates have been found with translocation and decompression than with simple decompression.[29,31,34] Surgical intervention must be planned according to the specific indications of each patient,[35–38] because no single surgical intervention can be recommended as ideal. According to Posner,[46] operative procedures include decompression without transposition of the nerve (*in situ* or by means of medial epicondylectomy) and decompression with transposition of the nerve carried out in a subcutaneous, intramuscular, or submuscular fashion. Complications of surgical intervention include the following: neuroma of the medial antebrachial cutaneous nerve, subluxation or dislocation of the nerve after release or transposition, inadequate protective covering for the nerve after transposition, unrecognized residual tethers that may lead to new sites for compression, iatrogenic nerve injury, incomplete decompression, and compression secondary to postoperative formation.[8,39] Dellon et al.[40] describe the musculofascial lengthening technique for submuscular transposition as the only surgical strategy that reduced intraneural ulnar pressure. Gabel and Amado[41] explore the nerve from the supracondylar process to the tendinous arch of the flexor carpi ulnaris muscle. Tsai et al.[42] use a new endoscopic technique that allows the release of all involved structures up to 10 cm both proximally and distally through a 3-cm skin incision. In conclusion, we agree with Assmus' opinion about surgery:[43] "Most important for the outcome of surgery is an early operation."

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22 Carpal Tunnel Syndrome

Compression of the median nerve occurs most commonly in a fibro-osseus canal on the palmar surface of the wrist, the carpal tunnel. The syndrome has been described frequently in the literature and excessively diagnosed.[119] In 1854, James Paget[1] first described chronic compression of the median nerve secondary to an old radius fracture at the level of the carpal tunnel. Other authors described disturbances ranging from vasomotor neurosis to brachialgia paresthetica nocturia and from neurobrachialgia mechanica to acroparesthesia.[2] These disturbances can be explained by Marie and Foix'[3] discussion in 1913, which implicated the transverse carpal ligament (flexor retinaculum) as the compressive agent. They described thenar atrophy due to median nerve compression, which could be relieved by sectioning the ligament. While Moersch[4] and others used this approach, it was not until 1946 that Cannon and Love[5] published their results on nine patients. Due to the work of Brain, Wright, and Wilkinson[6] and Phalen,[7] carpal tunnel surgery has become a refined and successful treatment of carpal tunnel syndrome. Carpal tunnel syndrome (CTS) is common.[91] A survey of physicians in California estimated that 515 of 100,000 patients sought medical attention for carpal tunnel syndrome in 1988. In the Netherlands, a prevalence of 220 per 100,000 has been reported.

ANATOMY

The median nerve courses through a fibro-osseus tunnel surrounded by the carpal bones, the transverse carpal ligament, and the flexor tendons. The scaphoid's and trapezium's tubercles bound the tunnel radially. The ulnar border consists of the pisiform and the hamate (Figure 22.1). The inelastic transverse carpal ligament connects the radial and ulnar eminence of the wrist (Figure 22.2) and lies just below the skin. The ligament can course 2 to 5 cm longitudinally, 2 to 3 cm in width, and 0.5 cm in thickness. The tunnel itself narrows distally. The median nerve and tendons are relatively close to the ligament's posterior surface. The tendons of the flexor digitorum superficially (FDS) course above the tendons of the flexor digitorum profundus (FDP) and the flexor pollicis longus (FPL) (Figure 22.3). The FPL runs within its own synovial sheath, the radial digitocarpal sheath. The finger flexors run together in the ulnar digitocarpal sheath.

The median nerve lies superficial to the flexor tendons and remains the most sensitive to pressure of all structures within the carpal tunnel. The anatomy of the median nerve and its branches varies so significantly that a cautious surgical approach is warranted to avoid a disabling sensory or motor deficit.[8] Before entering the tunnel, the median nerve gives off a palmar branch to supply the skin of the palm and thenar eminence. The palmar cutaneous branch originates 5 to 8 cm proximal to the wrist and typically runs on the radial side of the median nerve. Within the tunnel, the median nerve may branch into a radial and ulnar component. The radial component of the median nerve supplies sensory branches to the palmar surfaces of the first and second fingers and motor branches to the abductor pollicis brevis, the opponens pollicis, and the superficial head of the flexor pollicis brevis. In 33% of individuals, the entire flexor pollicis brevis receives median nerve innervation. In 2% of the population, the adductor pollicis also receives median nerve innervation. Damage, whether surgical or compressive, leads to thenar atrophy and loss of abduction and opposition, as in an ape's hand. The ulnar component of the median nerve provides sensory branches to the palm surface of the second, third, and radial side of the fourth finger. Additionally, the median nerve can supply the dorsal surfaces

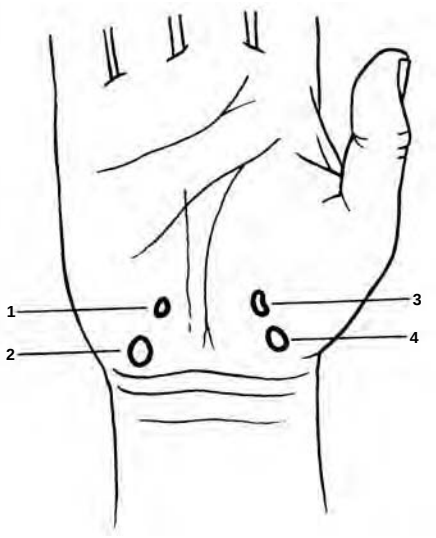


FIGURE 22.1 Palpation can help identify the bony boundaries of the carpal tunnel — 1: hook of the hamate; 2: pisiform bone; 3: tubercle of the trapezium; 4: tubercle of the scaphoid.

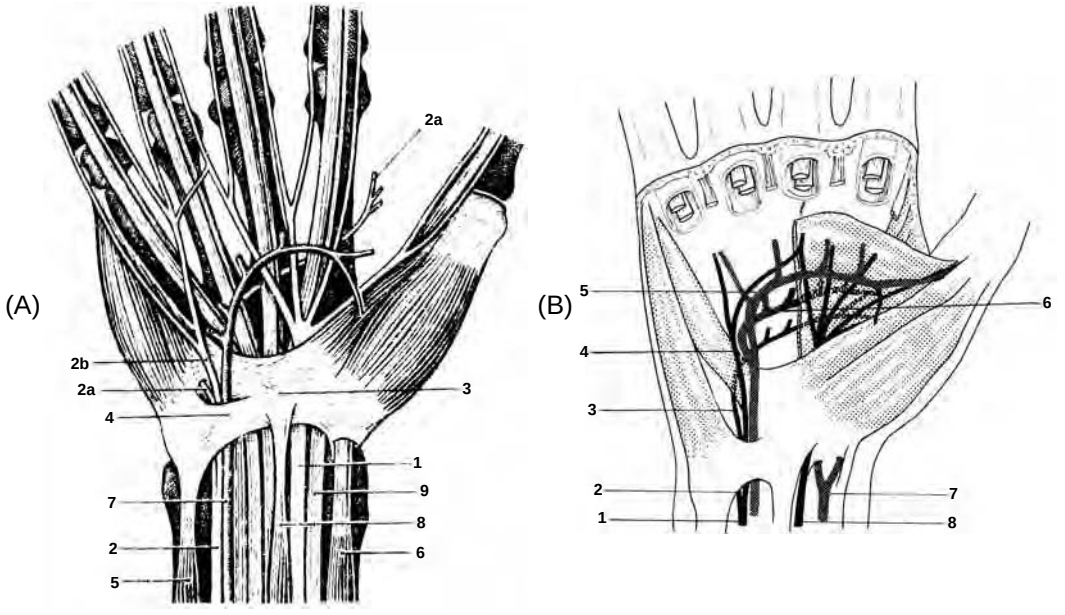


FIGURE 22.2 (A) Detailed anatomy of the palm — 1: median nerve; 2: ulnar nerve; 2a: deep branch (profundus) of the ulnar nerve; 2b: superficial branch of the ulnar nerve; 3: flexor retinaculum; 4: aponeurosis of the flexor carpi ulnaris muscle (tendinous end plate); 5: flexor carpi ulnaris muscle; 6: flexor carpi radialis muscle; 7: ulnar artery; 8: palmaris longus muscle; 9: flexor pollicis longus muscle. (B) Further delineation of the vascular arcades: 1: ulnar nerve; 2: ulnar artery; 3: deep branch of the ulnar nerve; 4: superficial branch of the ulnar nerve; 5: superficial palmar arterial arcade; 6: deep palmar arterial arcade; 7: radial artery; 8: median nerve.

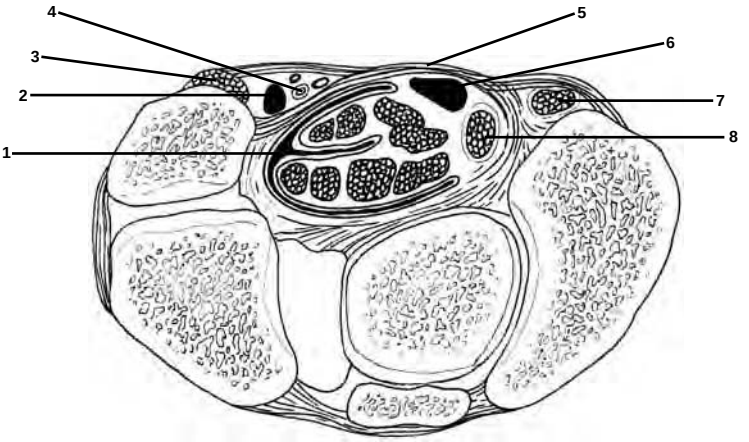


FIGURE 22.3 Content of the carpal tunnel, which includes nine tendons and the median nerve. Changes in the bony floor or ligamentous roof decrease the space available for the median nerve in the carpal tunnel. The ulnar nerve runs outside of the carpal tunnel — 1: digitocarpal synovial invagination, ulnar side; 2: ulnar nerve; 3: flexor carpi ulnaris muscle; 4: ulnar artery; 5: flexor retinaculum or transverse carpal ligament; 6: median nerve; 7: flexor carpi radialis muscle; 8: flexor pollicis longus muscle.

of the second, third, and fourth fingers distal to proximal interphalangeal joint. Patients commonly complain of sensory disturbances, pain, and cramps in these fingers. Spontaneous and provoked sensory disturbances, paresthesias, pain, and cramps commonly occur in these areas following a Tinel test of the wrist or hyperextension or hyperflexion of the wrist (Phalen's test).

Surgeons have performed extensive anatomical studies of the median nerve to improve surgical approaches and to decrease the risk of nerve injury (Table 22.1).[9,10]No simple approach exists. One must divide each layer and carefully examine for nerve branches. Unusic[11] recommends exposure of the whole transverse ligament before release from the ulnar side. Pappathanassiou[12] noted that motor branches might originate from the ulnar side of the ligament. Stancic et al.[92] explored the median nerve in 100 hands to find that only 47.7% of them showed the standard textbook anatomy. Additionally, the smaller hand muscles may be fully innervated by either the median or ulnar nerve, as in the "all-median hand" or "all-ulnar hand" of Jušić and Šoštarko.[13]

TABLE 22.1
Variation of the Median Nerve

Location	Innervation	Author(s)
Ulnar origin of motor branch	Thenar motor	Pappathanassiou, 1968[12]
High division into ulnar and radialcomponents	Hand	Kessler, 1969[62]
Ligament	Two motor branches, one Pierces ligament	Linburg and Albright,1970[10]
Forearm	Motor branches exist high, then merge with additional median nerve branch	Ogden, 1972[63]
Entire area of ligament	(1) Variation of thenar nerve; (2) Accessory branch at distal portion; (3) High division; (4) Accessory branch proximal to tunnel	Lanz, 1977[8]

ETIOLOGY

More than 80 years ago, Marie and Foix[3] suggested sectioning of the transverse carpal ligament to relieve several causes of carpal tunnel syndrome. The success of surgical intervention underscores the mechanical nature of the syndrome.[5,14] The carpal tunnel represents a limited space containing bone, tendon, connective tissue, synovium, and nervous tissue. Therefore, disease of, or trauma to, any of these components decreases the potential space and increases the pressure in the tunnel.[15–17] The median nerve, the most pressure-sensitive component, manifests the disease's involvement of the wrist (Figure 22.4). Table 22.2 lists some of the literature describing pathological causes of carpal tunnel syndrome, which is three times more frequent in women than men and peaks between 30 and 50 years of age. Rheumatoid arthritis produces synovitis and intrinsic muscle atrophy in the hand. Synovitis can be seen to cause asymmetric joint swelling.

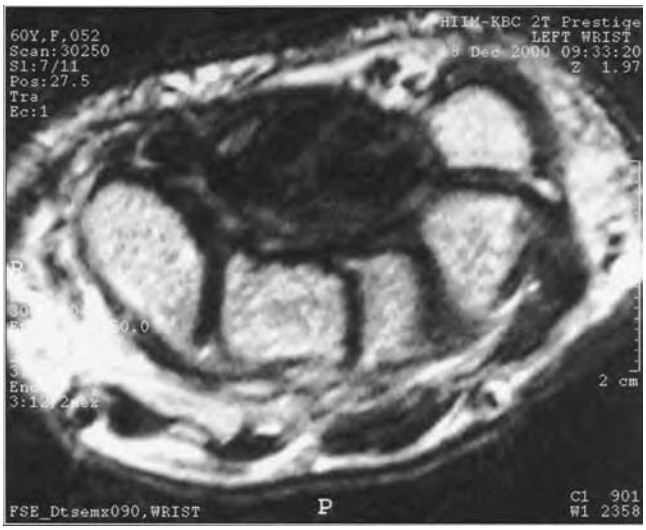


FIGURE 22.4 MRI demonstrates high signal intensity as a sign of swelling of the median nerve on FSE T2-weighted image with flattening of the nerve.

To use this information to guide surgical intervention, one would like to correlate disease states with their impact on connective tissue hypertrophy, synovial swelling, vascular supply (venous or arterial),[18–20] tissue edema, anatomic relationships, or general carpal tunnel pressure. Barnes and Currey[21] were unable to correlate synovial swelling on the palmar wrist surface with the appearance of carpal tunnel syndrome. Brain et al.[6] have investigated pressure variations in the carpal tunnel that range from 0 to 300 mmHg, depending on position. Chaise and Witovet[22] found that patients had a pressure of 25 mmHg, when a control group had a pressure of 4 to 6mmHg. With maximal extension and flexion, the control group had pressures of 30mmHg, while the patients developed pressures up to 100 mmHg. Brain et al.[6] suggested that extension develops the biggest pressure. Other authors have duplicated these findings.[23] Leven and Huffman[24] found that conduction velocity was impaired most in positions of extreme flexion and extension. Thus, they recommended immobilization in a neutral position. Repetitive occupational tasks have currently come to the forefront in discussions of carpal tunnel syndrome.[6,25] Workers spend a large portion of their time in flexion or extension, which may place their median nerve into prolonged periods of high pressure. Phalen[7] proposed that ischemia combined with compression led to the development of carpal tunnel syndrome.

TABLE 22.2
The Multitude of Etiologies for Carpal Tunnel Syndrome

Category	Specific Etiology	Author(s)
Autoimmune/hematologic	Amyloidosis	Goldman, 1970[28]
	Paraproteinemia	Huth et al., 1972[28]
	Psoriasis	Schambaugh et al., 1974[28]
	Sarcoidosis	Rosenbaum and Ochoa[109]
	Multiple Myeloma	Swinton et al., 1970[28]
	Lupus Erythematosus	Sidiq et al., 1972[64]
	Dermatomyositis	Quinones et al., 1966[65]
	Polyneuritis	Isaacs, 1972[28]
	Blood Dyscrasias	Bloget et al., 1962[66]
	Rheumatoid Disease	Michaelis, 1950;[67] Smukler et al., 1963;[68] Phalen, 1966;[7] Barnes and Currey, 1967;[21] Phillips, 1967;[69] Androic, 1979;[70] Domljan, 1969;[50] Bilić and Pećina, 1981;[17] 1986;[37] Todorovic and Smiljanic, 1982;[71] Moneim and Gribble, 1984[72]
	Hemophilia	Jabaley, 1978[77]
Congenital	Anatomical anomalies	Dekel and Coates, 1979;[73] Barfred and Ipsen, 1985;[74] Leslie and Ruby, 1985;[75] Asai et al., 1986;[76] Zeiss and Jakab, 1995;[110] Nogueira et al., 1999[111]
Idiopathic	Primary Tunnel Stenosis (essential)	Dekel and Coates, 1979;[73] Brain et al., 1947;[6] Leven and Huffman, 1972;[24] Chaise and Witovet, 1984[22]
Infectious/Inflammatory	Tenosynovitis	Phalen, 1966[7]
	Tuberculosis	
Metabolic/Hormonal	Pregnancy, Menopause	Mletzko, 1962;[78] Tobin, 1967[79]
	Oral Contraceptives	Rosenbaum and Ochoa[109]
	Diabetes Mellitus	Rosenbaum and Ochoa[109]
	Renal Failure/Dialysis	Allieu et al., 1983;[80] Benoitet al., 1988[81]
	Anticoagulant Therapy	Hartwell and Kurtay, 1966[28]
	Acromegaly/Myxedema	Johnson and Shrewsbury, 1970;[82] Skanse, 1961[83]
Neoplasms	Nerve Sheath Tumors	
	Bone Tumors/Cysts	
	Ganglions	Seddon, 1952[84]
	Palmar Lipomas	Robbins and Lubahn, 1994[93]
	Neurofibromatosis	Rosenbaum and Ochoa[109]
	Metastatic Disease	Tachmann et al., 1989[42]
Trauma	Fractures	Waters et al., 1994[94]
	Repetitive Action, Occupation Induced	Hunt, 1910;[108] Rossignol et al., 1998;[112] Marti, 1960;[85] Masear et al., 1986[25]
	Degenerative Joint Disease	Nigst, 1981;[86] Leviet et al., 1984[87]
	Pseudoarthrosis of the Scaphoid	
Vascular	Circulatory Disturbances	Baasch, 1951;[88] Pećina, 1974;[16] Vaine, 1957[89]
	Raynaud's Phenomenon	Ivkovic et al., 1976[18]

CLINICAL SYMPTOMS AND SIGNS

As listed in Table 22.3, patients present with a constellation of symptoms and signs that depend on the duration of nerve compression.[26] Phalen[7] noted that 80% of patients present with sensory complaints. Spinner et al.[27] found that 100% of their patients had median paresthesias. These sensory

TABLE 22.3
Signs and Symptoms of Carpal Tunnel Syndrome

Sensory	Motor	Signs
Hypoesthesia	Hypotrophy	Trophic Ulcers
Hyperesthesia	Weakness	Edema (21%)
Numbness	Atrophy	
Burning		
Pain		
Night		
Stiffness		

complaints range from night pain that awakens the patient to numbness and tingling in any of the sensory dermatomes of the median nerve. Since sensory fibers are more pressure sensitive than motor fibers, only 40% of patients will present initially with thenar hypertrophy or atrophy. Patients will complain of problems grasping or pinching.

Komar[28] and Ford and Ali[29] discuss the two forms of carpal tunnel syndrome: acute and chronic. The acute form presents with severe pain, wrist or hand swelling, a cold hand, or decreased finger motion. Finger-motion loss is caused by a combination of pain and paresis. The chronic form presents with either a predominating sensory dysfunction or motor loss with trophic changes. Proximal pain may be present in carpal tunnel syndrome.[30]

To avoid permanent nerve damage, diagnosis and treatment of carpal tunnel syndrome must be prompt. Several basic clinic tests are available in the office. These tests seek to reproduce pain or paresthesias in the median nerve's distribution within 30 to 60 seconds of testing (Table 22.4). Tinel's sign[31] uses percussion over the transverse carpal ligament, Phalen's test uses maximal flexion of the wrist, and Wormser's test[32] (or reverse Phalen's) uses hyperextension of the wrist. Using the theory that compressed nerves are more sensitive to ischemia, Gilliatt and Wilson[33] and Wilson[34] introduced the use of a tourniquet to temporarily cut off arterial supply to the hand. Phalen[35] has found that 83% of his patients developed symptoms within 1 minute. De la Caffinière and Theis[36] found significant nerve damage when symptoms appeared in the first 15 seconds of tourniquet time. Bilić and Pećina[37] describe a sensitive pressure test that places digital pressure on the median nerve in the carpal tunnel when the patients place their entire extremity weight on the carpal tunnel. The tourniquet can be used in conjunction but is not necessary. If symptoms appear after 10 seconds, the damage to the nerve appears reversible. If paresthesias, hyperesthesia, or hypoesthesias appear

TABLE 22.4
Various Tests Used to Diagnose Carpal Tunnel Syndrome

Test	Findings	Sensitivity	Time	Prognosis
Tinel's	Paresthesias or pain	80%		—
Phalen's	Paresthesias or pain	80%	Less than 1 min	—
Wormser (reverse Phalen)	Paresthesias or pain		Less than 1 min	—
Tourniquet	Paresthesias or pain	83%	15s	—
Thenar Atrophy	Decreased muscle bulk	36%		—
Pressure Test	Paresthesias		60 s (if > 10 s < 5 s)	—
	Hyperesthesia			Not Severe
	Hypoesthesia			Severe
Durkan's	Paresthesias or pain		Less than 30 s	—

in less than 5 seconds, the damage appears to be severe, with low operative success. Bilic's pressure test can be modified and performed to assess other tunnel syndromes. Other tests include Green's[38] description of the diagnostic and therapeutic value of carpal tunnel injection and Braun's[39] description of proactive testing in the diagnosis of dynamic carpal tunnel syndrome. Durkan[95] describes a new diagnostic test, called the carpal compression test, in which a pressure of 20kPa (150mmHg) is applied to the area of the carpal tunnel for as long as 30 seconds. A recent review of diagnostic tests in the literature was undertaken by Massy-Westrapp et al.[113] Definitions were standardized. They exposed the wide variations in specificity and sensitivity. The predictive values were not calculated. No one test was consistently superior. Our preference is to combine the findings of a Phalen's test, a Durkan compression test, and a simplified Semmes-Weinstein monofilament with a detailed history.

While previously of limited value, radiographic studies of the carpal tunnel using magnetic resonance imaging (MRI) may yield an accurate anatomical assessment of the tunnel.[40] Hart and Gaynov,[41] Hartwell and Kurtay,[28] and Barthold[43] describe axial X-rays of the hand in maximal dorsal flexion with the beam parallel to the fourth metacarpal bone and at 25 to 30° off a perpendicular line of the film (Figure 22.5). Figure 22.6 demonstrates the carpal tunnel with the hand in ulnar abduction. Mergarzadeh et al.[44,45] described the anatomy of the tunnel as seen by MRI (Figure 22.4). Four general findings in carpal tunnel syndrome are described as follows:

1. Swelling of the median nerve
2. Flattening of the median nerve
3. Palmar bowing of the flexor retinaculum
4. Increased signal intensity of the median nerve on T2-weighted images

Liang[46] found that computed tomography (CT) images revealed sectional tunnel areas that were smaller than controls in idiopathic carpal tunnel syndrome but were larger than controls in secondary

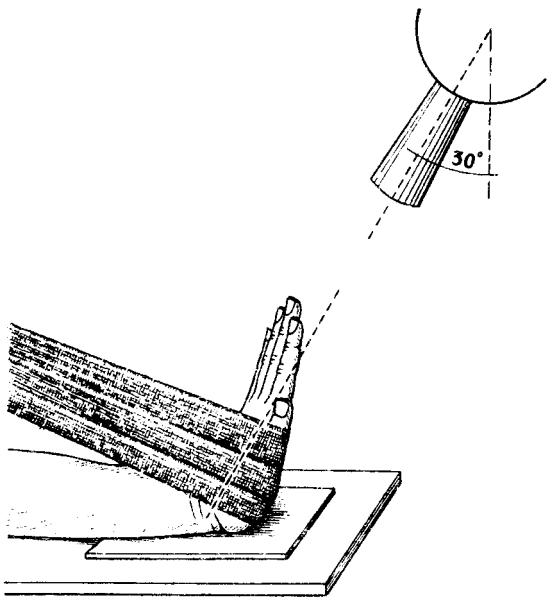
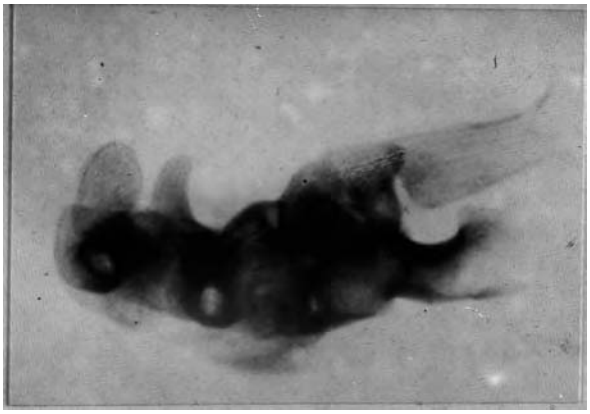
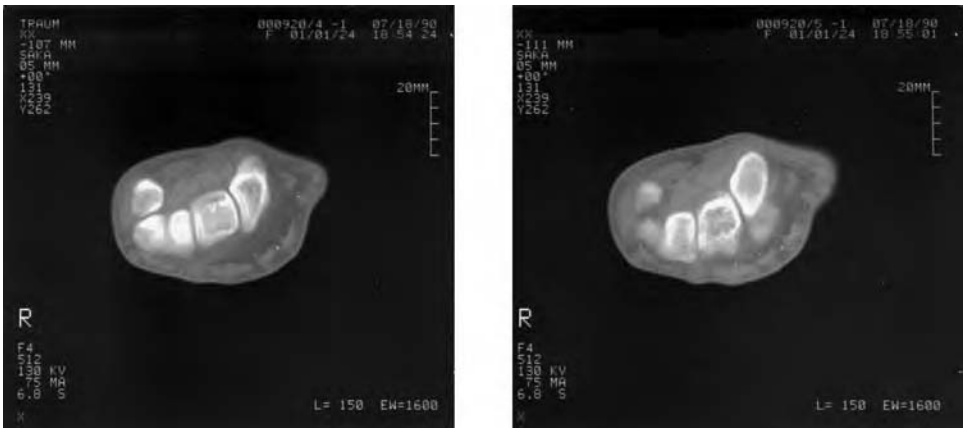


FIGURE 22.5 Because the bony anatomy of the carpal tunnel may influence the development of the carpal tunnel syndrome, radiographic analysis of the carpal bones and arches complements the exam. Hart and Gaynov[90] and Barthold[43] proposed that a radiographic shot at 30° to an extended wrist would best visualize the carpal tunnel.



(A)



(B)

FIGURE 22.6 (A) The film shot as suggested in Figure 22.5. (B) A computerized tomogram of the carpal tunnel.

carpal tunnel syndrome. In our practice, imaging studies are performed only when history indicates trauma, tumor, or rheumatological diseases.

Before proceeding with surgery, any other causes of median nerve impairment prior to the carpal tunnel must be identified. Electromyographic studies bear an important role in differentiation among the possible affected areas.[47] The median nerve may be damaged from its roots in the cervical spine, through the division in the brachial plexus, or along its course in the arm. For example, involvement above the pronator teres (PT) damages the palmaris branch, whereas involvement below, at the CTS, spares this branch. Vascular disease, toxic neuritis, and polyneuropathies may confuse the clinical picture. Radicular damage will present segmentally, whereas polyneuropathies will appear symmetrically or in areas of circulation. Characteristic electromyographic (EMG) findings in carpal tunnel syndrome are prolonged latency of motor impulses (more than 4 msec) and sensory impulses across the canal.[13,48] Even in the face of negative EMG findings, Phalen[35] believed that the diagnosis was warranted if the patient has:

- Sensory changes in the median nerve's distribution
- Positive Tinel's and Phalen's tests
- Tenderness to pressure over the wrist
- Hypotrophy of the thenar muscles

Thenar atrophy actually indicates severe nerve injury that may not recover despite release. Electrodiagnostic tests should be done from the neck down to eliminate double crush diagnoses. The surgeon must be comfortable with the neurologist's skills, consistent test environment, and objectivity. Massey-Westrapp et al.[113] found variability in EMG criteria. However, the use of EMG/NCV values in the Worker's Compensation patient allow objective criteria against which to base future claims.

TREATMENT

Treatment of carpal tunnel syndrome depends on the etiology, duration of symptoms, and intensity of nerve compression. If the syndrome is secondary to endocrine, hematologic, or other systemic disease, the primary disease should be treated. Conservative measures can be initiated. The surgeon should follow and confirm reversal of median nerve compression. If this reversal does not occur or if the symptoms and signs progress, a timely treatment course should be monitored.

Conservative therapy includes avoidance of trauma or repetitive actions, immobilization, anti-inflammatory medication, improved ergonomics and corticosteroid injections (Figure 22.7). Duncan et al.[49] reported that, in the U.S., 50 to 75% of all patients diagnosed with carpal tunnel syndrome have relief of symptoms without surgical treatment. The wrist is immobilized in the physiological position of slight extension with a dorsal splint for 3 to 6 weeks. Some patients respond better to a neutral position.

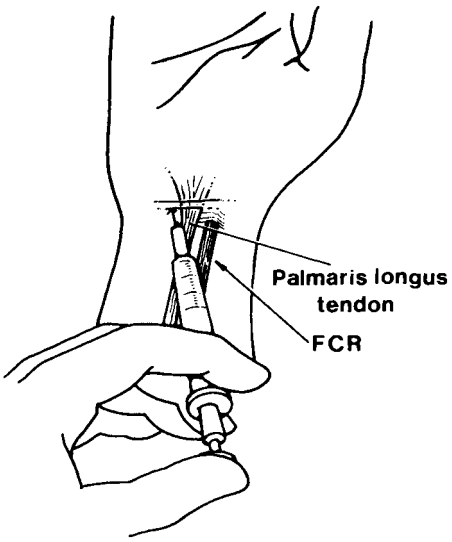


FIGURE 22.7 Conservative therapy, which includes corticosteroid injection into the carpal tunnel, may be tried within the framework of a planned course of intervention. The placement of the injection to allow entry into the carpal tunnel is illustrated.

While many patients have relief, the symptoms often recur. Corticosteroid injection into the canal may be offered to these patients.[50,51] Thin-needle injections are placed into the carpal tunnel from the ulnar side of the palmaris longus tendon or the flexor carpi radialis tendon at an angle of 30 degrees at the level of the proximal skin crease. The anesthetic and steroid mixture should not be injected into the nerve or tendons. Neither resistance nor electric shocks would be appreciated

upon injection. Patients frequently suffer uneasiness, pain, and stiffness for 48 hours, because the additional fluid increases nerve compression in the tunnel.

While injections can be repeated in 7 to 10 days for a total of three or four injections, failure to bring relief should be seen as an indication for surgical decompression. In our practice, one injection will determine the diagnosis and potential surgical prognosis. Patients should be numb and warned to protect their fingers for a day after injection. Failure to sense any relief following injection produces a guarded prognosis for complete recovery following release. Prolonged conservative therapy trials of greater than 6 to 8 months can lead to less optimal results from surgery.[52]

Mild cases of carpal tunnel syndrome (EMG and clinical) can exceed 6 months. Moderate or severe degrees of compression should not simply be observed. While results vary, Semple and Cargill[53] show a 97% success rate for symptoms persisting less than 6 months, and Hybbinette and Mannerfelt[54] show a 98 and 90% success rate in relieving pain and sensory disturbances, respectively. Their studies described an objective improvement in motor symptoms. Despite the interest in occupationally induced carpal tunnel syndrome, studies on ergonomic designs have failed to provide conclusive results on effect.[14] Cultural variations dramatically affect the claim and diagnosis rate.[119] This variation contradicts occupation-based theories.

Failure to respond within 6 months of conservative therapy or worsening of symptoms make surgical decompression necessary in spite of normal electromyography.[55] The carpal tunnel is approached paralleling the thenar crease to cross the palmar crease obliquely (Figure 22.8).[17] Care

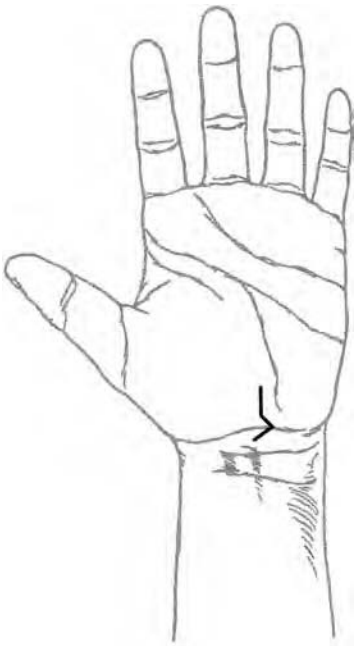


FIGURE 22.8 There are many approaches to the carpal tunnel, including a one-incision (classic), two-incision, and endoscopic approach to relieving compression of the median nerve. This is the classic one-incision approach, which stays ulnar to the thenar crease and crosses the wrist crease obliquely.

must be taken to preserve the palmaris branch of the median nerve.[56] While not uniformly present, the tendon of the palmaris longus muscle is identified and usually split. It should be preserved in younger patients to maintain a tendon source for reconstructive procedures. It can also be repaired as a protective layer over the median nerve.

The transverse carpal ligament or the flexor retinaculum can be approached from either the distal or proximal section as long as constant watch is continued for the variable branches of the median nerve. The variations of the thenar branches alone range from numbers of branches to their points of departure from the carpal tunnel. The transverse carpal ligament must be fully released or partially removed to prevent recurrence of a tight fibrous band and thus carpal tunnel syndrome.[57] Unusic[11] recommends exposure of the whole transverse ligament before release from the ulnar side. Exploration of the median nerve usually reveals an hourglass deformity in the region of stenosis. A characteristic vascular blush is typically present. If these lesions cannot be found, a longitudinal incision can be made in the epineurium.

Neurolysis and tenosynovectomies remain controversial. Unusic suggested microscopic interfascicular neurolysis. Curtis and Eversmann[58] recommend intrafascicular neurolysis when thenar atrophy or long-standing symptoms are present. We do not recommend routine neurolysis. In the presence of thenar atrophy, the motor branch should be traced and decompressed. Rheumatoid patients typically require synovectomies to decompress the area. This may require enlarging the incision. Additionally, tendon function should be verified, since tendon damage or rupture is common in the rheumatoid population. This can be done preoperatively. Take-down of the individual flexors may lead to tendon rupture and function loss.

Bilić and Pećina's review[37] found that surgery brought pain relief, improved function and esthetics features, and promoted deceleration of the disease process (see Figure 22.9 and Color Figure 22.1). Following decompression, the wrist should be dorsally splinted in extension and elevated for 24 to 48 hours to reduce swelling. The time of immobilization varies from 1 to 3 weeks, depending on the degree of exploration. Immediate finger motion is encouraged to avoid spot welding of the tendons to the median nerve by scar. Postoperative pillar pain (base of the thumb) remains for up to 3 months regardless of treatment.[115] Also, carpal tunnel syndrome may recur, with a less favorable prognosis.[116,118]

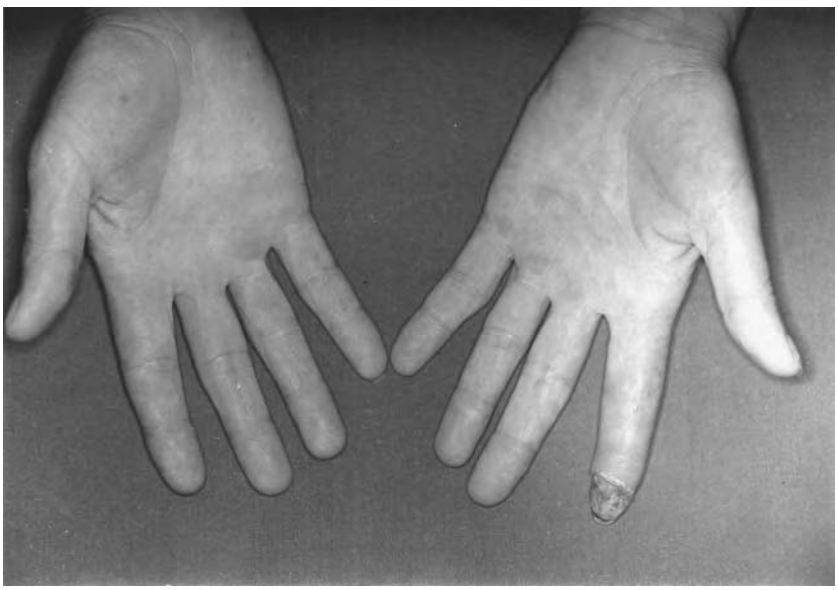


FIGURE 22.9 The hands of a female patient with a defect on the index finger resulting from median nerve compression in the carpal tunnel — the preoperative finding (see Color Figure 22.1 for the postoperative result).

McDonald et al.[59] describe the complications of surgical decompression as follows:

- Recurrence of compression due to incomplete sectioning of the transverse carpal ligament
- Sympathetic dystrophy
- Trauma to the sensory or motor branches of the median nerve[60]
- Hypertrophic scar
- Palmar hematoma
- Flexor tendon prolapse
- Adhesions limiting tendon function

These complications can be reduced by precise technique, postoperative care, and rehabilitation.[30,61]

Endoscopic carpal tunnel release remains a popular method. Multiple studies have looked at the advantages and disadvantages of this method.[96–107,121,122] Several different endoscopic procedures are currently used, but should only be employed by surgeons familiar with them. The risk profile is suggested to be decreased by this approach. Care must be used, as the ulnar nerve and artery lie on the ulnar border of the transverse carpal ligament. Endoscopic release allows earlier motion and decreases the size of the incisions. Limited open approaches that avoid the palm produce smaller incisions than a classic open procedure and may also allow earlier motion. If anatomical anomalies or crossing nerves are seen, a full open approach is employed. Despite all current therapies, patients with prolonged compression or work-related claims have a slower recovery and are less satisfied by surgery.[117,120]

While conservative measures may benefit some patients, the risk of prolonged compression combined with the high success rate of surgical decompression make a definite treatment schedule necessary. Such a treatment schedule will allow trials of conservative therapies prior to surgical decompression without exposing the patients to the risk of prolonged median nerve compression.

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23 Ulnar Tunnel Syndrome (Guyon's Canal Syndrome)

Analogous to median nerve compression at the wrist, ulnar nerve compression at the wrist occurs in Guyon's canal, producing ulnar tunnel syndrome. Guyon,[1] a French surgeon, described the fibro-osseus tunnel shown in Figures 22.2 and 22.3 of the previous chapter. While not as common as carpal tunnel syndrome, ulnar tunnel syndrome should be suspected in patients who complain of hand numbness.

ANATOMY

The ulnar tunnel, or Guyon's canal, lies at the level of the proximal carpal bones along the ulnar border. The transverse carpal ligament forms the floor of the tunnel, with the tendinous insertion of the flexor carpi ulnaris muscle forming the roof. The ulnar and radial borders consist of the pisiform and hamate bones, respectively. Triangular in shape, as seen in Figure 23.1, the tunnel narrows from a large volar base to a dorsal point. Anatomical studies have found the tunnel's height to range from 8 to 15mm.

Blood vessels and the palmar branch of the ulnar nerve pass through the tunnel. Proximal to the canal, the ulnar nerve divides into a dorsal branch and a palmar branch that further subdivides into a superficial and a deep branch prior to entering to the tunnel (Figure 22.2). Only the superficial and deep palmar branches of the ulnar nerve run in the ulnar tunnel. Therefore, it is clinically significant that the dorsal branch is spared in ulnar tunnel syndrome. However, it is affected by ulnar nerve compression in the ulnar sulcus behind the medial epicondyle (cubital tunnel syndrome).

The superficial branch of the palmar branch innervates the palmaris brevis muscle, the palmar skin of the fifth finger, and the skin on the ulnar side of the fourth finger. The deep branch innervates the hypothenar muscles, the two ulnar-sided (lateral) lumbricals, all the interossei muscles, the adductor pollicis muscle, and the deep head of the flexor pollicis brevis muscle. Therefore, the clinical picture of compression of the superficial branch yields mixed motor and sensory symptoms, while compression of the deep branch yields only motor symptoms.

ETIOLOGY

Shea and McClain[2] describe three different types of ulnar nerve compression in the ulnar tunnel or Guyon's canal based on motor and sensory disturbances. As shown in Figure 23.2, the compression occurs in the tunnel, and symptoms distal depend on which branches are compromised. The Type I compression, also known as the "upper form," occurs quite proximally and prevents any ulnar nerve innervation, motor or sensory. Resisted fifth-finger abduction will not create palmar creases because the palmaris brevis does not function; therefore it produces a positive Mumenthaler's sign. Type II compression produces only motor dysfunction, so only the deep branch is compromised. The "middle form" implies compression prior to the hypothenar muscle branches. The "lower form" of the second type implies deep-branch compression distal to the hypothenar muscle branches, thereby sparing them. The third form of compression has predominantly sensory symptoms, since the superficial branch is compromised, affecting the dermatome as shown in Figure 23.2. We disagree with the sign typically described with the upper form, as a lesion in this area

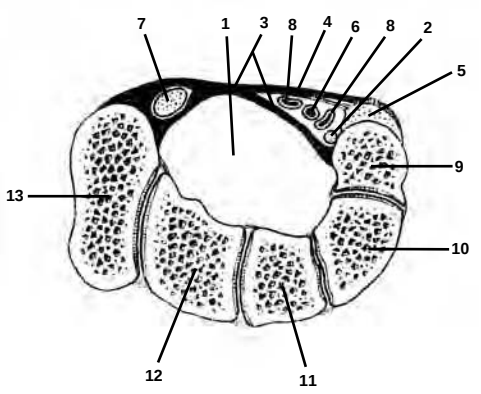

FIGURE 23.1 Not at risk from compression in carpal tunnel syndrome, the ulnar nerve does not run in its own tunnel with the inherent risks of compression when pathology affects the components of the tunnel — 1: carpal tunnel; 2: ulnar nerve; 3: flexor retinaculum; 4: aponeurosis of the flexor carpi ulnaris muscle (tendinous insertion); 5: flexor carpi ulnaris muscle; 6: ulnar artery; 7: flexor carpi radialis muscle; 8: ulnar veins; 9: pisiform; 10: triquetrum; 11: hamate; 12: capitate; 13: scaphoid (navicular).

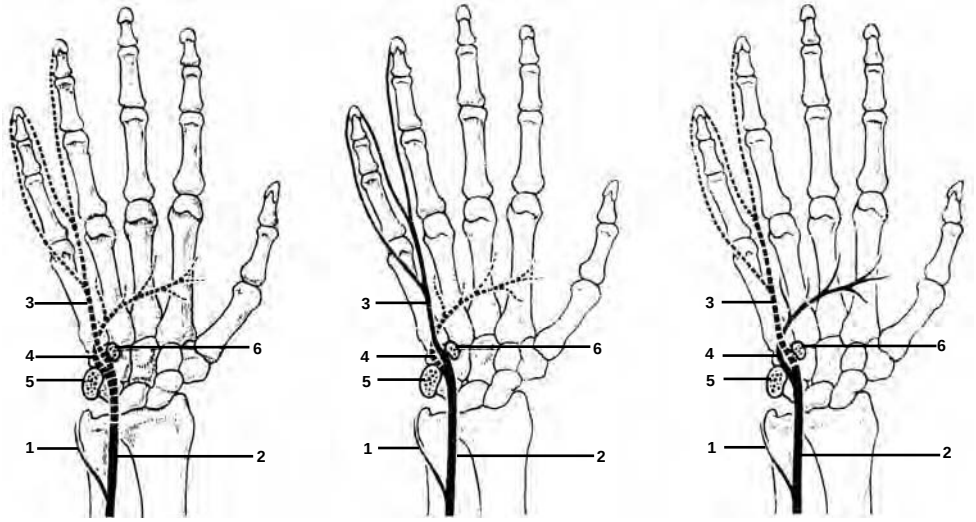

FIGURE 23.2 Various forms of ulnar nerve compression and the branches affected — 1: dorsal branch of the ulnar nerve; 2: ulnar nerve; 3: superficial branch of the ulnar nerve; 4: deep (profundus) branch of the ulnar nerve; 5: pisiform; 6: hook of the hamate.

should knock out all intrinsic motor function in the hand that is due to the ulnar nerve. Wu et al.,[60] break ulnar neuropathy into five categories based on a literature review.

There are multiple etiologies of mechanical irritation and damage to the nerve in the ulnar tunnel.[3] Posttraumatic compression and anatomical anomalies represent some of the more common etiologies. Nerve irritation may come from events as different as hook of the hamate fractures,[4] Galeazzi fracture-dislocations,[48] a bipartite hamulus, [5] or riding bicycles.[6–8,54] In war time, German soldiers rode bicycles over long distances, which led to nerve compression from the handlebars that was described in the literature as *Radfahrerlähung.* Anomalies may include the passage of the fourth-finger flexor tendon through the tunnel. Ganglions,[9,55] typically arising from degenerative arthritis, are present in 28.7% of patients with compressive symptoms. Ulnar artery aneurysms,[23,24] ulnar artery false aneurysm[25,26] or thromboangitis,[27] or lipoma in Guyon's tunnel[28] may produce

the syndrome. Included in the category of muscular disturbances leading to compression is hypertrophy of the flexor carpi ulnaris muscle.[29] The proximity to the carpal tunnel is a concern, as two patients were found to have a partial release of Guyon's canal after attempted endoscopic carpal tunnel release.[30] Thus, ulnar nerve injury may be iatrogenic. Occupationally related external compression,[10,11] vascular disturbances, Dupuytren's contracture,[58] pisotriquetral joint arthritis,[12–14] fractures,[52] bursitis near the pisiform bone,[15] giant cell tumor of tendon sheath,[16,17,57] giant cell tumors,[53] inflammatory diseases (including rheumatic diseases), edema,[18] aberrant or anomalous muscles,[19–22,49,50,59] and idiopathic cases complete the list of proposed etiologies.

CLINICAL SIGNS AND SYMPTOMS

While hypothenar atrophy may not be present,[56] patients describe uncertainty in their hand movements, including grasping. Pain may arise early and be aggravated by wrist extension. Numbness, tingling, and paresthesias may be found in the appropriate dermatomal distribution. Worse at night, this involvement of the third, fourth, and fifth fingers might force patients to stop using the hand. Forced palmar (Phalen's sign) and dorsal flexion (Wormser's or reverse Phalen's sign) cause paresthesias in the fourth and fifth fingers, with occasional involvement of the second and third fingers.[31,32] Adduction of the fifth finger and abduction and adduction of the thumb and other fingers are not impaired (a fact important for differential diagnosis). Tests used to elicit symptoms in carpal tunnel syndrome are also used to evaluate patients with ulnar tunnel syndrome. Pressure and tapping on the carpal tunnel or the ulnar tunnel may produce paresthesias in the palmar branch of the dermatome of the ulnar nerve (Tinel's sign). Use of a blood pressure cuff to induce ischemia might reproduce pain and paresthesias within minutes that may spread proximally and persist after cuff release. Late signs of compression are hypotrophy and atrophy leading to cramping and weakness of grip.

Other studies are available to identify compression in the ulnar tunnel. Hart and Gaynov[33] and Barthold[34] recommended special radiographic axial views of the carpal tunnel to evaluate abnormal configurations. Electromyographic and conduction velocity studies can help identify the level of nerve dysfunction.[35,47] While a positive test supports a clinical diagnosis, negative or borderline-negative results must be kept in perspective. Anomalous innervation must be kept in mind.[42] Hogue suggests that EMG data also help show recovery after decompressive surgery.[61] Thermography[36] and Doppler sonography[37]can also be used in diagnosis of ulnar tunnel syndrome (see Color Figure 23.1). Additionally, the presence of a coexisting carpal tunnel syndrome may be appreciated, allowing both neuropathies to be treated simultaneously.[38,39] Laboratory examinations will identify rheumatologic diseases that will need to be addressed to optimize treatment.[40,41]

TREATMENT

Conservative therapy remains the first line in treating ulnar nerve compression in the ulnar tunnel or Guyon's canal. Trials of conservative therapies should not be continued indefinitely. Prolonged compression may lead to permanent nerve damage. While conservative therapy can take up to 6 months to work, the absence of relief within 6 months should be addressed surgically without delay. Avoidance of repetitive trauma, rest immobilization (splinting), local corticosteroid injection, and anti-inflammatory medication may be tried individually and in combination to achieve relief. Any underlying diseases leading to nerve damage must be treated.

Muscle atrophy or weakness, persistent symptoms (past 6 months), or failure of conservative therapy to bring permanent relief define patients with severe neurological deficits. These patients require surgical decompression with complete release of both the motor and sensory branches.[43,44] The approach to the tunnel removes the tendinous floor, including the transverse carpal ligament (flexor retinaculum). The tunnel is cleared and adhesions removed. The nerve below the tendinous

arch of the hypothenar muscles must also be released. Following surgery, patients are immobilized in wrist splints for 10 days to 2 weeks before therapy begins. Finger motion is allowed. Surgical results will be poorer when definitive therapy is postponed or when the patient is older than 65. Release of the transverse carpal ligament with carpal tunnel syndrome will relax the pressure in the ulnar tunnel.

While less common, ulnar tunnel syndrome may occur in conjunction with carpal tunnel syndrome; therefore, one should assess the status of the median nerve before proceeding to ulnar tunnel release. Wissinger[45] described a limited approach to both tunnels. If involved, the median nerve should be released.

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24 Syndrome of the Deep Branch of the Ulnar Nerve

The deep branch of the ulnar nerve risks compression as it passes through a fibro-osseus tunnel formed by the tendinous origin of the hypothenar muscles (Figure 24.1). While similar to ulnar nerve compression in Guyon's tunnel (if only the motor branch is compressed), the abductor digiti minimi muscle is unaffected, as its innervation originates before the tendinous arch. Therefore, at times, this syndrome can be known as the syndrome of the tendinous arch of the hypothenar muscle or the syndrome of the deep branch of the ulnar nerve; it is also known to Uriburu et al.[1] as the *piso-hamate hiatus syndrome*.

ANATOMY

The fibro-osseus tunnel in the region of the hypothenar muscles has two bony walls and two fibrous walls. The ulnar and radial walls consist of the pisiform bone and the hamate bones, respectively. Both the piso-hamate ligament that spans the floor and the tendinous arch that forms the roof have been implicated in nerve compression. Using cadaver studies as a basis, Hayes et al.[2] emphasized the importance of the piso-hamate ligament in ulnar nerve compression. Connecting the pisiform and the hook of the hamate bone, the tendinous arch serves as the origin for a portion of the hypothenar muscles: the adductor digiti minimi, flexor digiti minimi brevis, and the opponens digiti minimi muscles.

The interossei muscles, the two ulnar lumbrical muscles, the adductor pollicis muscle, and the deep head of the flexor pollicis brevis muscle receive innervation from the deep branch of the ulnar nerve. This deep branch passes through the tunnel and gives motor fibers to the hypothenar muscles — except the palmaris brevis muscle, which receives its innervation from the superficial branch. Sparing of the abductor digiti mini muscle, whose motor branch originates proximal to the tunnel, allows differentiation of this syndrome from ulnar nerve compression proximally (i.e., Guyon's tunnel in ulnar tunnel syndrome).[3]

ETIOLOGY

While some etiologies are similar to those leading to ulnar tunnel compression,[4] compression of the deep branch of the ulnar nerve occurs most frequently with ganglion cysts.[2,5] It also occurs with anomalous muscles,[6,7] intraneural cysts,[8] giant cell tumor,[9] carpal bone fractures including hamate fractures,[10] doubled hamulus of hamate bone, [11] ulnar artery anomalies and diseases,[12] and chronic wrist trauma[13,14] (including occupational neuritis).[15] Compression of the deep branch of the ulnar nerve due to hand edema was described by Leslie.[16] Loten et al.,[17] Pastacaldi et al.,[18] and Stern and Vice[19] describe compression of the deep branch of the ulnar nerve by the fibrotic arch or a fibrotic band around the nerve. Antuna et al.,[20] note nerve compression caused by hypertrophy of the pisiform, which was termed pseudotumor. Netscher and Cohen[21] describe compression of the deep branch of the ulnar nerve by a tight fibrous arch at the origin of the flexor digiti minimi.

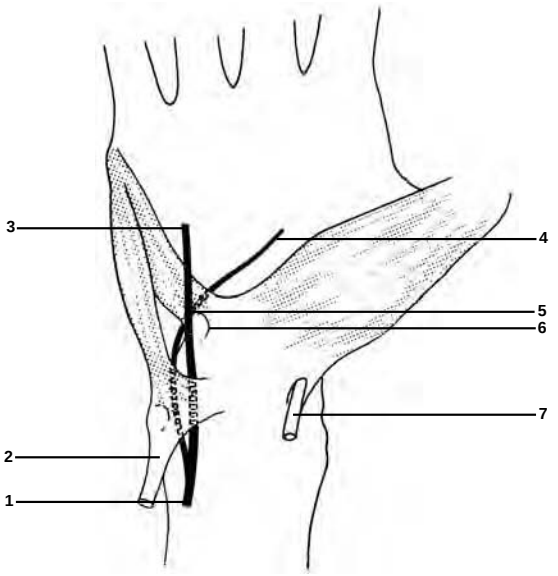

FIGURE 24.1 Anatomy along the hypothenar area of the palm — 1: ulnar nerve; 2: flexor carpi ulnaris muscle; 3: superficial branch of the ulnar nerve; 4: deep branch of the ulnar nerve; 5: tendinous origin of the hypothenar muscles; 6: tendinous arch of the hypothenar muscles; 7: flexor carpi radialis muscle.

CLINICAL SYMPTOMS AND SIGNS

Patients present with poorly localized pain in the area of innervation of the deep branch of the ulnar nerve. Since the deep branch itself has no sensory component, the patients do not have paresthesias or hypesthesias. The palmaris brevis and abductor digiti minimi muscles typically continue to function. In contrast to ulnar tunnel syndrome, this difference is secondary to their innervation, which is proximal to the tendinous arch but distal to the ulnar tunnel. Since deep-branch compression presents with motor signs, the differential diagnosis must include ulnar tunnel syndrome (type II), progressive spinal muscle atrophy, or lateral amyotrophic sclerosis. Electromyographic studies play an important role in localizing the lesion.[22]

TREATMENT

Conservative therapy consists of avoidance of repetitive wrist trauma, short-term immobilization, and anti-inflammatory medication. Local corticosteroid injections should be tried before surgical decompression. Surgical release of the tendinous arch, from which the hypothenar muscles take their origin, is usually sufficient for decompression.

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25 Syndrome of the Tendinous Arch of the Adductor Pollicis Muscle

The syndrome of the tendinous arch of the adductor pollicis muscle occurs when the terminal portion of the deep branch of the ulnar nerve gets compressed between the tendinous arch connecting the transverse and the oblique heads of the adductor pollicis muscle (Figure 25.1). Midpalm trauma is a common etiology producing palmar pain with intrinsic muscle atrophy.

ANATOMY

The transverse head of the adductor pollicis muscle originates from the anterior margin of the third metacarpal bone. The oblique head and the deep head of the flexor pollicis brevis muscle share a common origin — the volar ligaments covering the trapezoid and the capitate bones. Connecting the heads of the two adductor pollicis muscles, the tendinous arch forms the roof of a tunnel, the floor of which is the third metacarpal bone. The terminal branch of the deep branch of the ulnar nerve dives through this tunnel to innervate the adductor pollicis.

ETIOLOGY

Repetitive trauma to the mid palm may lead to the development of nerve compression. This type of trauma is common in professions and sports that require prolonged gripping, such as cycling. In cycling, flat rectangular grips may be worse offenders than round handles.[1] Fractures, tumors, or any disease that affects the third metacarpal bone may elevate the floor, narrow the tunnel, and lead to nerve compression.[2–4] Comtet et al.[5] describe compression of the deep palmar branch of the ulnar nerve by the arch of the adductor pollicis. According to Ruder and Wood,[6] this type of ulnar nerve compression neuropathy is present in fewer than 1% of those ulnar neuropathies that occur at wrist and hand.

CLINICAL SYMPTOMS AND SIGNS

Patients may present with blunt midpalmar pain without paresthesias and isolated intrinsic weakness or atrophy of the first dorsal interosseous muscle and adductor pollicis muscle. Pressure at the base of the third metacarpal bone may reproduce the pain. Electromyographic analysis shows that conduction time is prolonged for the adductor pollicis muscle but not for other hypothenar muscles.

TREATMENT

Treatment consists of removal of the cause by conservative methods. Local injection of corticosteroids usually relieves the compression;[7] however, abnormalities of the tunnel's bony structure may necessitate surgery.

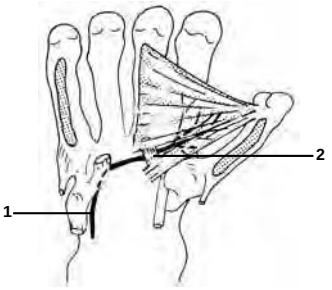

FIGURE 25.1 As the deep branch of the ulnar nerve (1) transverses the palm; it may be compressed under the tendinous arch (2), connecting the oblique and transverse heads of the adductor pollicis muscle.

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26 Syndrome of the Superficial Branch of the Radial Nerve

Making an analogy to paresthetic meralgia, Wartenberg in 1932[1] suggested the name *cheiralgia paresthetica* to define isolated neuropathy of the superficial branch of the radial nerve. Also known as Wartenberg's disease, radial nerve compression can occur throughout its course in the forearm, especially in the tunnel region beneath the tendon of the brachioradialis muscle. The work of Schlesinger,[2] Stopford,[3] and Matzdorff[4] helped Wartenberg[1] clarify the syndrome. Sprofkin,[5] Wartenberg,[6] and Bora and Osterman[7] suggested that cheiralgia paresthetica was more common than originally thought.

ANATOMY

The superficial ramus, or branch, divides from the radial nerve as it lies in the radial cubital sulcus. As shown in Figure 26.1, this branch travels distally over the supinator muscle and the pronator teres. Passing over the pronator teres muscle, the nerve dips under the ulnar border of the brachioradialis muscle and bends dorsally to run along the radius. The superficial branch, at a point between the middle and distal thirds of the radius, arches dorsally through a tunnel in the antebrachial fascia to terminate in the skin beyond the radial styloid. The dorsal digital nerves, the terminal branches of the superficial branch, supply sensation to the dorsal skin of the first, second, and radial side of the third digits to the base of the second phalanx. As skin dermatomes overlap, the only autonomous region of the superficial radial nerve is the dorsal web space closest to the thumb.

ETIOLOGY

While predominantly injured by trauma to the radius, the superficial branch of the radial nerve can also be injured by multiple reported causes including:

- Surgical trauma (especially during operations for De Quervin's disease) or other trauma to the wrist[8,9,25]
- Compressive plaster casts
- Intravenous infusion in the forearm and wrist
- Osteoarthritic changes of the wrist or a scaphoid exostosis[24]
- Chronic irritation from tight cuffs or watch straps[3,10,15]

In addition, Fossati et al.[16] describe lymphatic compression of the superficial branch of the radial nerve, and Margles[17] presents a case of palmar ganglion compressing the radial digital nerve of the thumb. Superficial radial nerve compression at the elbow due to an accessory brachioradialis muscle was described by Spiner and Spiner.[26] This list leaves many cases unexplained except by the dynamic relationships of the nerve and brachioradialis tendon near the radius. Wartenberg's syndrome caused by a split tendon of the brachioradialis muscle is described by Turkof et al.[18,27] and Zoch and Rothmund.[19] Brown and Chung[28] describe quadrangular space syndrome associated with superficial radial sensory neuropathy in a patient who improved after surgical decompression of the quadrangular space.

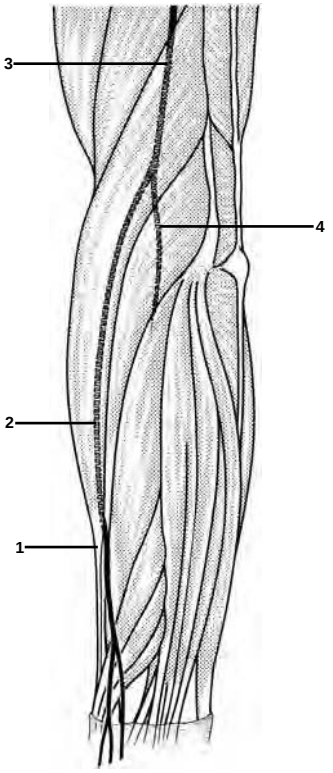

FIGURE 26.1 The course of the radial nerve in the forearm — 1: brachioradialis muscle; 2: superficial branch of the radial nerve; 3: radial nerve; 4: deep branch of the radial nerve.

CLINICAL SYMPTOMS AND SIGNS

Characteristically, patients present with paresthesias without weakness or atrophy. Burning pain, sensory changes, and night pain are felt along the dorsal wrist, thumb, and web space (Figure 26.2). The simple irritation of clothing in this region may be enough to produce paresthesias. The examiner generally will find a positive Tinel's test. Chronic cases will present with skin changes: a thin shiny skin surface and a lack of hair. Lanzetta and Foucher[20] found that De Quervain's disease was associated in 50% of cases. To avoid unexpected postoperative complications and medicolegal problems, it is important to diagnose Wartenberg's syndrome before operating on the tenosynovitis.

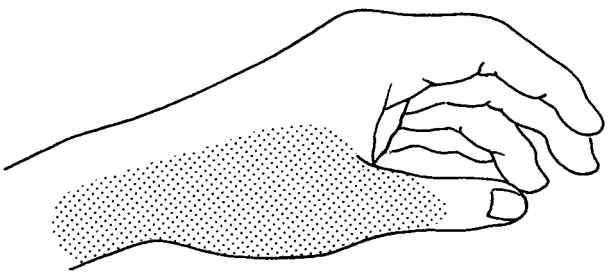

FIGURE 26.2 The region of the forearm supplied by the superficial branch of the radial nerve. Nerve compression in this area leads to paresthesias consisting of burning pain, commonly known as *cheiralgia paresthetica*.

To differentiate cheiralgia paresthetica from a cervical radiculopathy, one needs only to note that there are neither motor signs nor any effect on other areas of radicular involvement.

TREATMENT

Damage to the radial nerve can lead to disability, especially if the patient's dominant hand is involved; therefore, extreme caution must be exercised when dissecting proximal to the base of the thumb and near the brachioradialis tendon. If the syndrome has been caused by external compression, conservative therapy can be successful. Local application of corticosteroids, anesthetic creams, histamine, and ionophoresis might yield some success. Surgical exploration may be necessary if conservative therapy fails.[12,21] If the nerve is trapped by scar tissue, then release, neurolysis, and coverage with healthy tissue should be performed. The presence of a neuroma produces a guarded prognosis for recovery and relief.[22]

Hypesthesia following nerve resection may be acceptable when faced with continued burning pain and paresthesias. However, the loss of hand sensation could disable a patient and increase the risk of injury even in daily activities. Lanzetta and Foucher[20] treated 52 cases (50 patients) of entrapment of the sensory branch of the radial nerve in the forearm (Wartenberg syndrome) between 1988 and 1992. Conservative treatment achieved 71% excellent and good results. Operative treatment achieved 74% excellent and good results. A modified technique involving longitudinal plication of the brachioradialis tendon was used in seven cases operated on by Zoch and Aigner.[23]

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27 Distal Posterior Interosseous Nerve Syndrome

The posterior interosseous nerve can be compressed or irritated as it travels along the fourth extensor compartment. The nerve courses along the (radial side of the compartment) floor adjacent to Lister's tubercle. The syndrome presents with a dull ache that can be reproduced or aggravated by wrist dorsiflexion or by pressure over the compartment.

ANATOMY

The trunk of the radial nerve divides within the sulcus cubitalis radialis into two terminal branches: the superficial branch (ramus superficialis) and the deep branch (ramus profundus). A mixed sensory and motor branch, the deep branch enters the supinator canal and divides at the distal end of the supinator muscle into two parts: *the muscular branch*, which supplies the extensor carpi ulnaris, extensor digitorum communis, extensor digiti minimi, abductor pollicis longus, extensor pollicis brevis muscles and *the posterior interosseous nerve*, which innervates the extensor pollicis longus and extensor indicis proprius muscles. The posterior interosseous nerve is actually the terminal branch of the deep branch of the radial nerve. The posterior interosseous nerve runs along the interosseous membrane distally as a sensory branch. Distally, the posterior interosseous nerve continues its course along the radial side of the floor of the fourth extensor compartment adjacent to the Lister's tubercle (Figure 27.1). At this level, it is accompanied by the posterior branch of the anterior interosseous artery. The tendons of extensor digitorum communis and extensor indicis proprius muscles run through the fourth compartment. The nerve is situated deep to the extensor digitorum communis tendon and on top of the dorsal joint capsule. It ends in a bulbous expansion over the dorsal wrist joints before multiple sensory twigs are sent to the dorsal wrist capsule and dorsal intercarpal ligaments.

ETIOLOGY

Repetitive dorsiflexion activities in the wrist can result in irritation or in compression of the posterior interosseous nerve. Trauma to the wrist capsule may also produce nerve irritation. Carr et al.[1] found ten patients with wrist pain who were engaged in vigorous athletic activities (gymnastics) or occupations (secretarial) that required repetitive dorsiflexion. Two patients with distal posterior interosseous nerve syndrome described trauma to the wrist. One developed dorsal wrist pain 18 months after successful closed treatment of a scaphoid fracture. The second patient fell during a gymnastic routine onto a dorsiflexed hand 3 months before the onset of his dorsal wrist pain. According to Dellon,[2] postoperative neuromas may be a source of the pain in the dorsal wrist. Capsule manipulation during operative procedures as common as ganglion excision or as complex as wrist stabilization procedures may produce nerve injury. Compression of the nerve into the fourth dorsal compartment might also result from anatomical anomalies such as an accessory muscle (i.e., extensor brevis manus muscle). Some patients following repetitive dorsiflexion maneuvers suffer compression of the posterior interosseous nerve as it enters the dorsal wrist capsule, inciting symptomatic inflammation and eventually undergoing a reactive fibrosis of the nerve. Dorsiflexion of the wrist can cause nerve irritation at the distal edge along

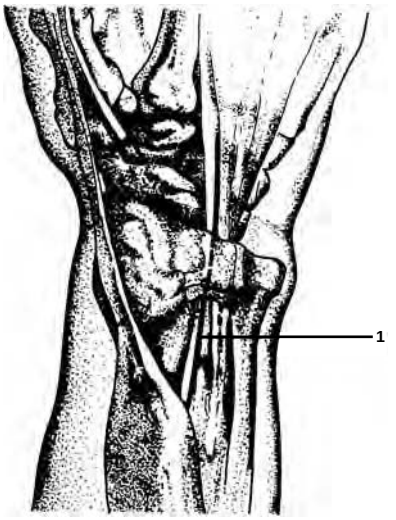

FIGURE 27.1 The distal posterior interosseous nerve courses along the radial side of the fourth dorsal extensor compartment —1: distal part of the posterior interosseous nerve.

the dorsal carpal ligaments or by compression between the floor and the roof of the fourth dorsal extensor compartment. Surgical excision of the nerve has shown reactive fibrosis several centimeters proximal to the site of compression.

CLINICAL SYMPTOMS AND SIGNS

In *distal posterior interosseous nerve syndrome*, patients present with dull ache, which could be reproduced by dorsiflexion of the wrist and by pressure over the fourth extensor compartment.[3] Physical examination reveals no palpable mass or ganglion. Motor and sensory examination is entirely normal, because the nerve is innervating the wrist capsule only deep to the skin. Results of PA, lateral and oblique radiographs, stress views, and tomograms of the wrist are negative. A diagnostic test that can be therapeutic in this syndrome is to inject local anesthetic with corticosteroids in the radial side of the fourth extensor compartment 3 cm proximal and 1 cm ulnar to Lister's tubercle. Immediate elimination of discomfort after the injection suggests that the posterior interosseous nerve is the origin of the patient's pain. Care must be taken to avoid an intra-articular wrist injection. Wrist joint pathology might exist to complicate the interpretation of the test. If present, pain relief after excision may be incomplete. If no pain relief occurs, the diagnosis of distal posterior interosseous nerve syndrome is less likely, and an alternative explanation must be found. These unresponsive symptoms are often attributed to dorsal wrist synovitis, occult dorsal ganglions, or "dorsal wrist syndrome" as described by Watson et al.

TREATMENT

Treatment can be conservative or operative. The conservative treatment can be divided into two stages. Stage one consists of a palmar wrist splint in the neutral position, nonsteroidal anti-inflammatory medications, and avoidance of inciting activities for 3 to 6 weeks. Patients who fail to respond are injected with local anesthetic and corticosteroids in the same manner as described above.[1,2] Patients who experience temporary relief of pain after injection, but whose symptoms return to the point of significantly compromising employment are candidates for surgical treatment. Surgical treatment[2,4] means excision of the posterior interosseous nerve in its entirety from 3 cm

proximal to the wrist joint to its termination. Some authors actually recommend excision of the terminal portions of both the posterior and anterior interosseous nerves through a dorsal incision.

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28 Digital Collateral Nerve Syndrome

The terminal branches of the median and the ulnar nerves can be compressed in the region of the metacarpophalangeal joints. Pain and paresthesias may result in the corresponding digits. The terminal nerve branches supply sensation to the skin and joints of the fingers; therefore, signs of sensory dysfunction indicate nerve compression. This syndrome is also known as *metacarpal tunnel syndrome*.

ANATOMY

The heads of the second to the fifth metacarpal bones are connected by superficial and deep transverse metacarpal ligaments near the metacarpophalangeal joints. These ligaments form tunnels through which the digital nerves, the common palmar digital arteries and veins, and the tendon of the lumbrical muscles pass (Figure 28.1). The digitorum palmaris communis nerves (the branches of the ulnar and the median nerves) branch before or within the tunnels to provide terminal branches (the digitorum palmaris proprius nerves) to neighboring surfaces of corresponding fingers. The branches provide innervation to the lumbricals proximal to the tunnels. The third digital branch, a branch of the median nerve, anastomoses with the ulnar nerve via a ramus communicans. Variations in and overlapping areas of innervation exist; therefore, few areas of the hand can be assessed as being innervated solely by the median, ulnar, or even radial nerve.

ETIOLOGY

Compression, swelling, and trauma to the metacarpal space compromise the digital nerve. Inflammation of the flexor tendon sheaths, especially in rheumatoid arthritis, represents one of the most frequent causes of nerve compression. Trauma to the hyperextended finger forces the digital nerves or their branches against the transverse metacarpal ligament.[14] This damage may be compounded in occupations that continuously place the fingers into hyperextension. Kopell and Thompson[1] postulate that occupations using vibrating devices (saws and jackhammers) favor the development of the digital nerve compression. Cases involving neoplasms and digital nerve aneurysms[2] have been reported. Poulenas and Verdan[3] encouraged the use of the term “defile syndrome” rather than “tunnel syndrome” to define the compression of the digital nerves, because their anatomical investigations revealed very narrow defiles rather than tunnels. Goldwirth and Goodwin discuss compression from carrying heavy grocery bags (the “plastic bag syndrome”).[12]

Compression of the digital nerves to the thumb is considered a separate entity, secondary to anatomical differences. Digital nerve compression of the thumb occurs most frequently in bowlers, due to direct nerve compression from handling and throwing the bowling ball. This damage requires repetitive injuries. Termed “bowler’s thumb,” the anatomical finding of perineural fibrosis secondary to repeated microtrauma has been reported by several investigators.[4–7] Calder et al.[8] describe a 68-year-old woman who developed digital pain. This was secondary to a mass of hyperplastic Pacinian corpuscles compressing the digital nerve. DeSmet[13] documents digital nerve compression from a fascial band on the radial side of a ring finger. Dobyns[9] noted that the principal lesions involved

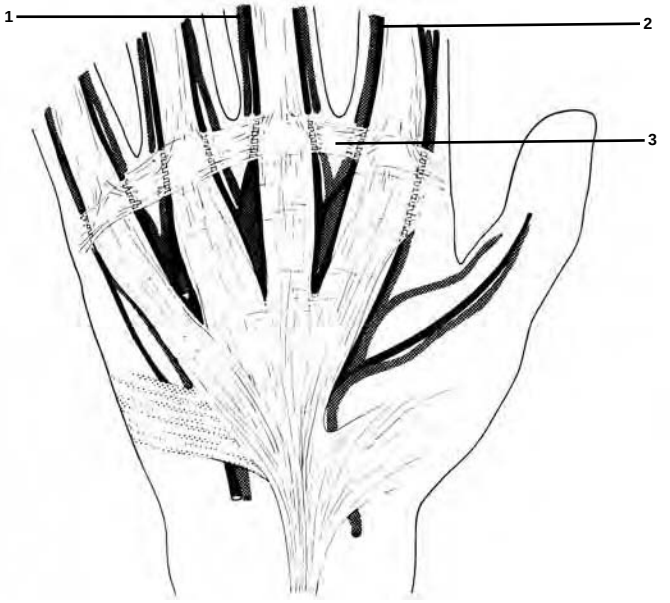

FIGURE 28.1 The arrangement of the neurovascular bundles as they leave the palm and enter the digits — 1: proper palmar digital artery; 2: proper palmar digital nerve; 3: superficial transverse metacarpal ligament.

in digital nerve compression are of two types: (1) fibrosis about, around, and in the nerve; and (2) reactive hyperplasia of the nerve support elements, special end organs, Pacinian corpuscles, or even the nerve fibers themselves. Many personal and professional devices and techniques can place the digital nerves at risk. These elements are present in all parts of our society, including the home and in occupational, avocational, and medical situations. Recognition of these factors requires detailed patient histories.

CLINICAL SYMPTOMS AND SIGNS

Depending on whether the digitalis palmaris communis or the digitalis palmaris proprius is compressed, the sensory findings will vary from sharp burning pain, hypesthesia, or paresthesias in two neighboring fingers or a solitary finger respectively. Several fingers may be involved if more than one metacarpal tunnel is involved. Physical signs include reproduction of pain or paresthesias by finger hyperextension or adduction, or by applying pressure between the metacarpal heads. These maneuvers aggravate nerve compression by narrowing the tunnel. Nerve signs and symptoms, often accompanied by a mass in chronic cases, are diagnostic.

TREATMENT

Since the predominant etiology is inflammatory in nature, treatment should be directed at reducing perineural inflammation. Immobilization, anti-inflammatory medications, and perineural corticosteroid injections are usually successful.[10,11] Dobyns[9] prefers conservative options: education, risk awareness, and avoidance of risk activities. Surgery, when indicated, is effective. If compression persists, some authors propose neurolysis of the affected nerves. It consists principally of lysis of the damaged nerve and positioning it in a protected position or, on occasion, excision of terminal or end-organ lesions.

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Section III

Tunnel Syndromes of the Trunk

29 Notalgia Paresthetica

Notalgia paresthetica presents as a rare compression syndrome of the dorsal nerve branches of thoracic roots at their passage through the multifidus spine muscle. Analogous to meralgia paresthetica or cheiralgia paresthetica, notalgia paresthetica is derived from the Greek *noton* meaning back and *algia* meaning pain. The notalgia paresthetica, pain in the back or dorsalgia, was described by Astwazaturow in 1934.[8] Notalgia paresthetica is characterized by interscapular burning pain, itching and demonstrable paravertebral paresthesias in the T2 through T6 dermatomes.

ANATOMY

Spinal nerves arise from the spinal cord by a ventral root and a dorsal root, including the spinal ganglion. Both roots are joined to a mixed spinal nerve. There are 31 pairs of spinal nerves: cervical, dorsal, lumbar, sacral and coccygeal. These mixed nerves divide into a ventral and a dorsal division. The ventral divisions are large and form general plexuses: cervical, brachial, lumbar, sacral and pudendal. The branches of the dorsal division are smaller. The dorsal branches of the thoracic roots pass over the transverse processes medial to the costotransversal ligament and intertransversal muscle, which is often missing. The dorsal branches of the thoracic roots divide into medial and lateral branches. Above the level of the T6, the cutaneous branches originate from the medial branches. They emerge in the groove between spinalis and longissimus muscle, pursue a right-angled course through the multifidus spinae muscle and supply the skin of the back (Figure 29.1). The lateral branches supply the longissimus and iliocostalis muscles. Below the T6, the medial branches supply the rotatores, multifidus, spinalis and semispinalis muscles, and lateral branches supply the skin of the rest part of the back. The lateral branches emerge in the groove between the longissimus and iliocostalis muscles.

ETIOLOGY

The posterior rami of spinal nerves arising from T2 through T6 are unique in that they pursue a right-angled course through the multifidus spinal muscle. This particular circumstance may predispose them to harm from otherwise innocuous insults of a varied nature. Massey and Pleet[6] described electromyographic findings consistent with paraspinal denervation in patients with notalgia paresthetica. They suggested nerve entrapment as the underlying cause of notalgia paresthetica. In six patients with notalgia paresthetica described by Pleet and Massey the cause remained unknown.[8] In one of their patients, the sensory loss appeared in conjunction with a postvaccinal brachial neuritis. In one female patient, there was a temporal relationship to a surgical procedure, perhaps to being positioned on her back on the operating table. While some hereditary cases have been noted, predominantly in young patients associated with multiple endocrine neoplasia type 2A,[1] notalgia paresthetica mainly occurs in older patients. Most are sporadic pathologies linked with musculoskeletal compression of spinal nerves.[2,9] Notalgia paresthetica has also been reported in patients with a hystory of neuritides, thereby suggesting an underlying predisposition to peripheral neuropathy.[8]

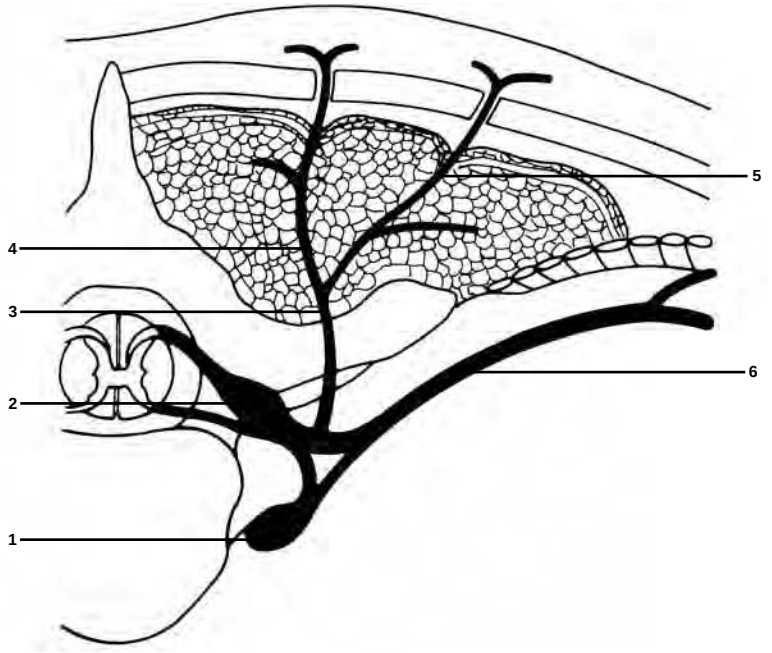

FIGURE 29.1 Notalgia paresthetica is a rare compression syndrome of the dorsal nerve branches of thoracic roots at their passage through the multifidus spine muscle — 1: ganglion trunci sympathici; 2: spinal ganglion; 3: dorsal branch of the thoracic root; 4: medial branch; 5: lateral branch; 6: ventral branch of the thoracic root.

CLINICAL SYMPTOMS AND SIGNS

The symptoms are characterized by interscapular burning pain and demonstrable paravertebral paresthesias. These paresthesias are similar in nature to the pain experienced in meralgia paresthetica or cheiralgia paresthetica — hence, the name.[5,8] The patient does not have a functional disability; however, the pain may restrict shoulder and trunk motion. There may be a zone of hypesthesia medial to the scapular margin in the distribution of the second through sixth thoracic dermatomes. Astwazaturow's patients may have both hypesthesia for touch and hyperalgesia in the affected zone. Tenderness can be found over the spinous processes of T2 to T6. In some other patients, intermittent "tingling," and persistent burning or itching over the scapular region were described. Patients can describe a gradual onset of a "crawling" sensation, itching or burning in the upper back just medial to the scapula. Depending on their jobs, the patients' perception of their sensory abnormality can affect their ability to be productive. Pigmented patches on the skin and friction amyloidosis can arise with irritation.[9] A skin biopsy shows intraepithelial necrotic keratinocytes with melanin and melanophages in the papillary and mid dermis. This is consistent with a clinical diagnosis of notalgia paresthetica.[12] Sometimes, sensory neuropathies and or electrical disorders can be present.[9]

TREATMENT

Treatment with paravertebral anesthetic and steroid injections at the trigger points are usually successful.[2,3,4,7] Topical capsaicin (Zostrix) has shown some (but only transient) efficacy in relieving notalgia paresthetica symptoms.[2,9,11] Antidepressants have also been used in the treatment of pruritus. According to some authors, patients with notalgia paresthetica could benefit from spinal or paraspinal ultrasound or radiation physiotherapy.[9]

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30 Medial Superior Cluneal Nerve Syndrome

The medial branch of the superior cluneal nerve is confined within a tunnel consisting of the thoracolumbar fascia and the superior rim of the iliac crest as it passes over the iliac crest. The nerve can be entrapped between the rigid fibers of the thoracolumbar fascia and the iliac crest. The syndrome is manifested by the pain localized to the iliac crest projecting a pattern of pain into the buttock.

ANATOMY

Three groups of cluneal nerves exist:

1. The superior cluneal nerves from dorsal branches of L1–L3 innervate the skin in the gluteal region, the skin over the gluteus maximus and medius muscles.
2. The middle cluneal nerves originate from the dorsal branches of S1–S3 and perforate the gluteus maximus muscle to innervate the gluteal region (skin over the sacrum and buttock).
3. The inferior cluneal nerves originate from the posterior femoral cutaneous nerve originate from the sacral plexus and emerge under the inferior margin of gluteus maximus muscle and innervate the lower buttock skin.

The superior cluneal nerve has three branches: medial, intermediate, and lateral superior cluneal nerves. However, the contributing roots vary as high as T11 or T12.[6] According to Maigne and Doursounian,[6] innervation of the gluteal region can come from T11 and T12. In general, the medial branch of the superior cluneal nerve originates from L1, the intermediate branch from T12 and the lateral branch from T11. The medial branch rarely originates from L2, the intermediate from L1, and the lateral branch from T12.

Pain in the middle of the gluteal region is transmitted through the medial superior cluneal nerve (Figure 30.1). This nerve is situated 64.7 ± 5.3mm from the posterior superior iliac spine and 81.0 ± 9.2mm from the midline.[5] The perforating point of the nerve on the thoracolumbar fascia is 5.8 ±1.8mm inferiorly for the medial branch, 2.2 ± 1.8mm superiorly for the intermediate branch and 12.0 ± 4.4mm superiorly for the lateral branch. The medial branch is confined within a tunnel formed by the thoracolumbar fascia and the superior rim of the iliac crest. The other two branches perforate the thoracolumbar fascia or pass through a fissure in the fascia without bony contact. The medial branch can be entrapped in the tunnel between the iliac crest and the thoracolumbar fascia.

ETIOLOGY

Showing its distribution and iliac crest crossing patterns in 37 dissected cadavers Maigne et al.[4,5] presented a comprehensive study of the anatomy of the superior cluneal nerve. They were the first to describe and present photographic evidence of a superior cluneal nerve entrapment syndrome. Maigne and Doursounian[6] published the results of surgical neurolysis in 19 patients with unilateral

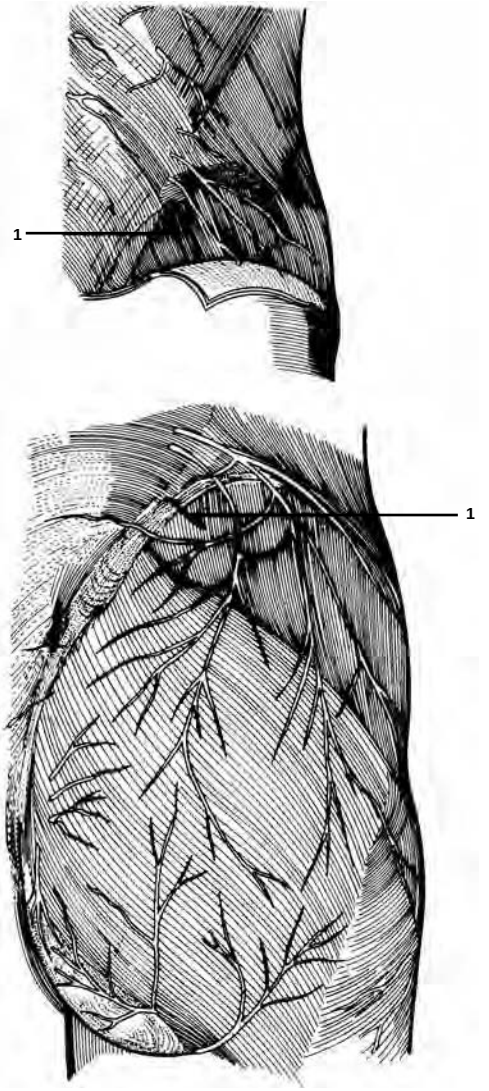

FIGURE 30.1 The cutaneous innervation of the gluteal region by the cluneal nerves. The medial superior cluneal nerve can be entrapped between the rigid fibers of the thoracolumbar fascia and the iliac crest — 1: medial superior cluneal nerve.

low back pain related to medial superior cluneal nerve entrapment. In 15 cases, a constricting fascial ring was found around the medial superior cluneal nerve. Compression was severe in seven cases, with poststenotic swelling of the nerve. It was moderate in eight cases. The nerve appeared normal, without compression, in four cases. Lu et al.[3] used 15 cadavers for the anatomic relationship of the superior cluneal nerve in the posterior iliac crest region. In two specimens they found the medial branches of the superior cluneal nerve constricted within the osteofibrous tunnel. The nerve was entrapped between the rigid fibers of the thoracolumbar fascia and the iliac crest. According to Lu et al., the superior cluneal nerves are at risk when a surgeon does a subperiosteal dissection in the region of the posterior iliac crest. This relationship is important for the surgeon harvesting autogenous bone from the posterior crest.

CLINICAL SYMPTOMS AND SIGNS

Maigne and Doursounian[6] postulated an entrapment neuropathy of the medial superior cluneal nerve, and proposed the following diagnostic criteria: first, pain in the distribution of the nerve; second, a trigger point over the posterior iliac crest, located 7 cm from the midline (corresponding to the nerve compression zone); and third, relief of symptoms by nerve block.

Patients considered to have medial superior cluneal syndrome should have unilateral pain originating from a clearly identifiable point on the posterior iliac crest, approximately 7cm from the midline. Pressure applied to this point should trigger the pain. The remainder of the iliac crest should be nontender. To confirm the diagnosis, the medial superior cluneal nerve block test must be positive. Lidocaine is injected along and above the iliac crest. This injection should result in temporary complete or almost complete relief of both pain and tenderness. Berthelot et al.[1] describe patients with episodes of flashing pain induced by lateral bending toward the opposite side, i.e., by rotation of the trunk. Hence, the possibility of entrapment neuropathy should be considered only when other causes of pain have been reasonably ruled out and when pain relief by local iliac crest blocks is constant and dramatic.

TREATMENT

Surgery should be proposed only when patients fail to improve after local blocks with anesthetic and steroids. According to Maigne and Doursounian,[6] surgery under general anesthesia was performed with the patient in the prone position. Through a 6-cm incision, the medial superior cluneal nerve was exposed as it exited through the osseofibrous tunnel prior to crossing the posterior iliac crest. This point was located at the lateral border of the erector spinae muscles. The nerve was surgically released by generous enlargement of the fascial part of the orifice. At follow-up, clinical results were rated excellent in 13 cases (19 total cases including the seven cases of severe nerve compression). The positive anesthetic test in the cases not improved by surgery may be related to a placebo effect or to anesthetic spreading to other pain sites.[6] It is well known that the number of pain syndromes may localize to the posterior iliac crest.[2] The performance of two consecutive blocks, separated by a week, should reduce the number of false positive results.

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31 Syndrome of Musculus Rectus Abdominis

Compression of the anterior cutaneous terminal branches of the last six intercostal nerves (T6 to T12) within the sheath of rectus abdominis muscle has been described by Applegate as the "abdominal cutaneous nerve entrapment syndrome."[2] Komar and Varga described it as the "syndrome of the rectus abdominis muscle."[8]

ANATOMY

Ventral branches of thoracic nerves are called intercostal nerves, and they live within the intercostal spaces. Running anteriorly along the ribs, the neurovascular bundle consists of a nerve, vein, and artery. The nerve lies inferior to the artery and vein. The first six intercostal nerves reach the sternum, while the six lower nerves run obliquely to the abdomen. They pierce the diaphragm, run between the oblique and transverse abdominal muscles, and reach the linea alba after passing through the rectal sheath. These nerves consist of motor and sensory fibers that innervate the surrounding abdominal musculature, as well as the skin (Figure 31.1).

The ramus cutaneous lateralis leaves the nerve root, pierces the external intercostal muscles between the axillar and medioclavicular line, and supplies the skin of the lateral trunk wall. Terminal branches of the six lower intercostal nerves, rami cutanei anteriores abdominales, run between the obliquus abdominis internus and transversus abdominis muscles, reaching the internal side of the sheath surrounding the rectus abdominis muscle. They change direction and bend at 90 degrees to pierce the sheath, entering the muscle itself and finally transversing the external layer of the sheath. Beneath the skin, the nerve divides into medial and lateral branches. Rami cutanei anteriores abdominales supply the skin of the anterior abdominal wall from the level of the xiphosternal line to the symphysis pubis and laterally to the prolongation of the mammillary line (Figure 31.2). Hammond et al.[5] discuss the implications of the rectus abdominis muscle innervation and thoracoabdominal flap elevation for breast reconstruction.

ETIOLOGY

From its origin at the spinal cord to its termination at the skin, the intercostal nerve transverses half of the body's circumference. The nerve is fixed at its origin and at the point where it passes through the rectus sheath, especially at its abrupt entry into the sheath. During the investigation of microanatomy of the structures contributing to abdominal cutaneous nerve entrapment syndrome Applegate and Buckwalter[3] paid special attention to the fibrous ring in the rectus muscle, through which the neurovascular bundle travels. If this bundle is pushed or pulled too far from behind or in front, however, compression of the bundle against the ring causes nerve ischemia and symptoms of abdominal cutaneous nerve entrapment. The nerve's abrupt entry into the muscle produces a fulcrum on which external forces can act. According to Komar and Varga,[8] an increase of the volume of the abdominal cavity produces a large increase in the distance between the two fixed points of the nerve. This increases the tension on the nerve. This phenomenon may occur during pregnancy.[8,12] Peleg[11] describe the case of 15-year-old girl who came to the hospital with abdominal pain of 3 months' duration. It was proposed that oral contraceptive therapy might have caused

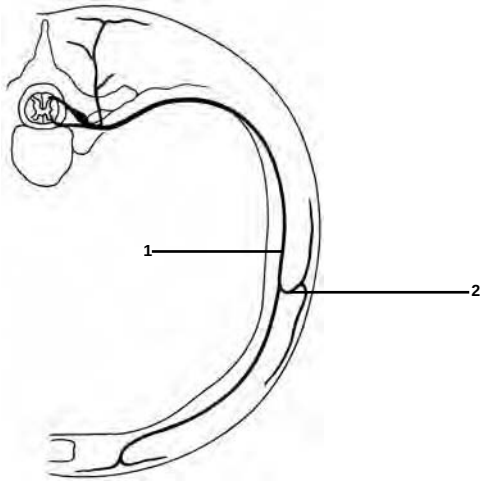

FIGURE 31.1 The intercostal nerve running along the rib — 1: intercostal nerve, and 2: ramus cutaneous lateralis.

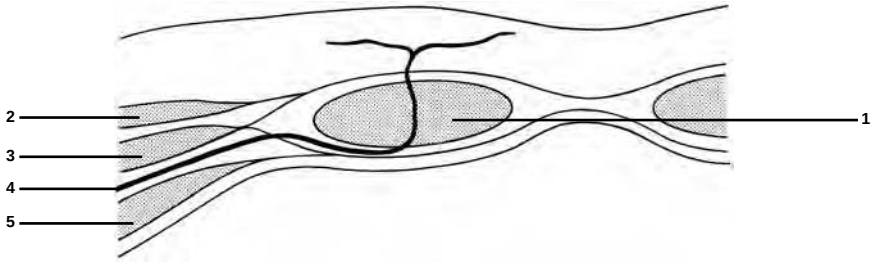

FIGURE 31.2 Compression of the anterior cutaneous terminal branch of the intercostal nerve occurs within the sheath of musculus rectus abdominis — 1: rectus abdominis muscle; 2: obliquus abdominis externus muscle; 3: obliquus abdominis internus muscle; 4: ramus cutaneous anterior abdominalis of the intercostal nerve; 5: transverse abdominal muscle.

changes in the abdominal wall that led to nerve entrapment. Heavy physical activity that overloads the abdominal muscles can result in this syndrome, because the contraction of the muscle compresses the nerve directly. Tung et al.[15] emphasized the fact that abdominal or spinal surgery might play an increasing role in the baseline activity of the rectus abdominis muscle. Muscle hyperactivity may cause tension or compression of the nerve at its entry into the muscular sheath.

CLINICAL SYMPTOMS AND SIGNS

Patients present with acute burning pain in the area of the rectus abdominis muscle. This can be unilateral or bilateral. Movement that tenses this muscle increases the pain, such as changing positions (lying to sitting), coughing, and the Valsalva maneuver. Neurological symptoms might occur as well: hypesthesia, loss of vibration sense, and lack of two-point discrimination. Intra-abdominal pressure may cause protrusion of the abdominal wall in the painful region because of decreased muscle tone. The symptoms may appear segmentally along the rectus muscle depending on the injured nerve.[14] The most painful pressure point is in the region where the nervous branch lies beneath the skin (3 cm lateral to the umbilicus). According to Applegate,[2] who reported on 62 patients with entrapment syndrome of the anterior intercostal nerves, 75% of the cases are women. Infiltration of local anesthetic into the painful region reduces or stops

the pain, serving as a diagnostic test. Electromyography may show changes in the involved muscle segment.

Due to involvement of the sensory branches of the intercostal nerves, the differential diagnosis must include problems with abdominal viscera, diabetes,[9,13] and rib abnormalities ("slipping rib syndrome," "rib-tip syndrome," "slipping-rib cartilage syndrome," or "clicking-rib syndrome").[1,6,7,9] A detailed physical examination and history can help exclude these disease entities. Rib abnormalities noted above occur at the rib–cartilage interface (T8 to T10) when the junction becomes loose. Pain initiated by manipulation of the inferior part of the costal arch helps confirm this diagnosis.

TREATMENT

Treatment of the syndrome of the musculus rectus abdominis should address the cause of an increase in nerve tension. Decreasing intra-abdominal pressure by removing the cause may relieve the pain. If the cause cannot be treated, reduced activity should be recommended. Corticosteroids and anaesthetic injections can be used to further decrease nerve irritation. Surgical intervention is rarely indicated.

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32 Iliohypogastricus Syndrome

The iliohypogastric nerve may be compressed along its course through the posterior abdominal wall, where it pierces the aponeurosis of the transversus and internal oblique muscles and then runs between the internal and external oblique muscles. Compression can result in motor and sensory dysfunction (see Figure 32.1.).

ANATOMY

The iliohypogastric nerve originates from the L1 nerve root with contributions from the T12 nerve root. The nerve passes between the bundles of the psoas muscle to arch over the quadratus lumborum muscle to run superiorly and laterally behind the kidney. It pierces the aponeurosis at the origin of the transversus abdominis muscle and internal oblique muscle, innervates these muscles, and turns inferiorly to run between the internal and external oblique muscles above the inguinal ligament. In this area, the lateral cutaneous branch separates from the main trunk to pierce the internal and external oblique muscles above the middle of the iliac crest before innervating the area of skin overlying the gluteus medius and tensor fasciae latae muscles. It terminates near the rectus abdominus muscle with one final branch, the anterior cutaneous nerve. This sensory branch tracts medially above the annulus inguinalis subcutaneous, pierces the anterior sheath of the rectus abdominus muscle, and innervates the skin above the inguinal ligament and symphysis.

ETIOLOGY

Tumors, bleeds, surgical injury (direct, indirect, or from scars), trauma, pregnancy, and traction injuries have been proposed as potential causes of nerve injury or compression.[1–3,6,8–10,12] Tumors in and around the kidneys can compress the nerve. Surgery to treat these pathological processes can either directly or indirectly injure the iliohypogastric nerve. Scar formation can fix the nerve or compress it.[6,8] As the nerve courses along the iliac crest, it is vulnerable to trauma, tumors, or iatrogenic injury from surgery (biopsies or bone graft harvest). Farther along in its course, the nerve is at risk for surgical injury from incisions in the inguinal region. The ilioinginual and genitofemoral nerves are also at risk from surgical approaches in this area. Blunt abdominal trauma can lead to bleeds that could compress the nerve. If the nerve is relatively fixed along its course, surgical procedures at the hip or pelvic region could produce traction injuries. According to Ziprin et al.,[13] athletes' groin pain may be due to nerve entrapment in the external oblique aponeurosis. An awareness of this nerve entrapment may reduce delays in operating, which would lead to an earlier return to sport.

CLINICAL SYMPTOMS AND SIGNS

Compression of the nerve proximally produces ipsilateral abdominal muscle weakness that worsens when the patient stands or strains. Sensory dysfunction depends on the site of compression. If proximal to the lateral cutaneous nerve branch point, the patient will have paresthesias, both near the hip and above the inguinal ligament. If after the branch point, the patient will note sensory changes near the inguinal ligament. If the nerve is injured near the iliac crest, only the dermatome

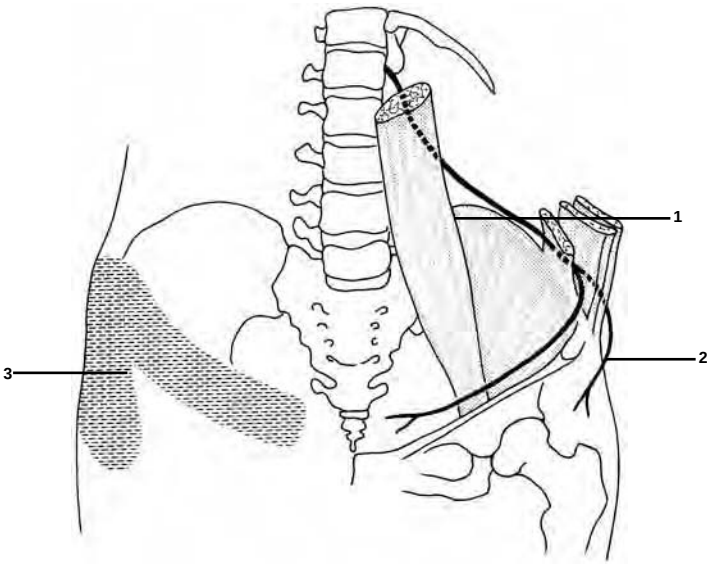

FIGURE 32.1 The iliohypogastricus nerve can be compressed when it pierces, as well as passing between, the abdominal muscles — 1: iliohypogastricus nerve; 2: lateral cutaneous nerve; 3: the skin area innervated by the iliohypogastricus nerve.

of the lateral cutaneous nerve will be affected. These sensory changes are often described as burning, pain, or pins and needles, which can be aggravated by hip extension.

Patients might actually walk with a bent posture to minimize the traction irritation of the nerve. This posture and symptom development are usually gradual in development; however, iatrogenic injuries will present acutely. Testing of the lateral cutaneous nerve distribution will help allow differentiation of iliohypogastric nerve injury from ilioinguinal and genitofemoral nerve injury.

Touzard et al.[11] and Maigne et al.[7] describe the pain in the trochanteric region caused by tunnel compression of the lateral cutaneous perforating branch of the ilio-hypogastric nerve.

Knockaert et al.[4] assessed the value of abdominal muscle electromyography in 41 patients with a clinical syndrome suggestive of ilioinguinal-iliohypogastric nerve entrapment. Electromyographic abnormalities were detected in 60% of cases with definite diagnosis and in 37% of those with probable diagnosis of ilioinguinal-iliohypogastric nerve entrapment syndrome.

TREATMENT

Treatment consists of removing offending agents, if possible. Initial observation following surgical insult is prudent unless the problem is recognized and correctable at the time of surgery. Anesthetic injection, transcutaneous stimulation, abdominal muscle strengthening, gait correction, and posture modification may alter the patient's symptoms as well as decrease nerve irritation. Success is variable. Surgical options include neurolysis, rhizotomy, and nerve resection.[3,5,12] Rhizotomy, in a recent study, appeared to produce more satisfactory results than neurectomy.[12]

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33 Ilioinguinal Syndrome

The ilioinguinal nerve can be compressed as it passes through the abdominal wall between the transversus abdominis muscle and the internal and external oblique abdominis muscles. This produces both muscular and sensory dysfunction.

ANATOMY

The ilioinguinal nerve originates from the L1 nerve root (rarely L2) and descends along the quadratus lumborum and the iliacus muscles. Piercing the transversus abdominis muscle and the internal oblique muscle, the nerve comes to run under the external oblique muscle. As it runs between the internal and external oblique muscles distal and medial to the anterior superior iliac spine, the nerve changes direction and either accompanies the spermatic cord in men or the round ligament in women, the ligamentum teres of the uterus (Figure 33.1). Sensory fibers branch off to innervate the skin of the scrotum in men or the skin of the labia major in women. An additional sensory branch supplies the skin over the inguinal ligament.

ETIOLOGY

Described as neuralgia by Cramer in 1933,[1] nerve injury during surgery has been described by many authors.[2–5] Stark et al.[20] describe the nerve irritation after laparascopic hernia repair. Since the ilioinguinal nerve courses through the retroperitoneal space to come anteriorly over the abdominal wall, the nerve lies at risk for compression from muscular, retroperitoneal, renal, and urogenital pathology.[6–8] Retroperitoneal endoscopic neurectomy for nerve entrapment after hernia repair was described by Krahenbuhl et al.[18] Additionally, pathology involving the spermatic cord or the round ligament may damage the nerve. Inguinal hernias can also distort the muscular tunnel. Ilioinguinal nerve entrapment in a patient with systemic sclerosis was described by Langevitz et al.[9] Kopell et al.[10] describe the ilioinguinal syndrome as a tunnel syndrome based on the functional anatomy.

The ilioinguinal nerve has been described as being susceptible to stretching, as its length is fixed between its origin and where it pierces through the abdominal musculature at the level of the anterior superior iliac spine.[10] This muscular tunnel fixes the nerve; therefore, dynamic or permanent changes in the position of the hip or the pelvis will create tension on the nerve. Prolonged or recurrent stretches will damage the nerve. Additionally, if hip motion and gait are affected, compensatory movement can further alter abdominal muscle motion during gait. Altered abdominal movements can also compress a stretched nerve. Other investigators have noted the interdependence of osteoarthritis of the hip joint and development of the ilioinguinal syndrome.[11,12] Compression caused by prolonged coughing in individuals with bronchial asthma has been postulated, as the function of the abdominal musculature endures repeated stresses.

Chronic groin pain on the ventral surface of the scrotum and the proximal ventro-medial surface of the thigh, especially in athletes, has been diagnosed in various ways — recently, the concept of "sports hernia" has been advocated in Europe.[19] Entrapment of the ilioinguinal and genitofemoral nerves may be a reasonable candidate for the cause of chronic groin pain.

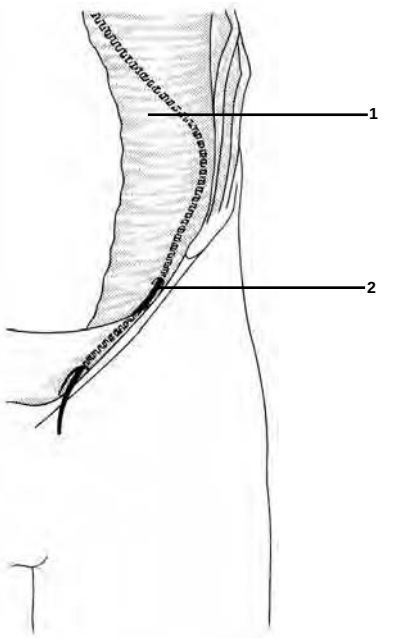

FIGURE 33.1 The ilioinguinal nerve (2) lies in a susceptible position for nerve compression and damage as it pierces the transversus abdominis muscle (1) and its fascia.

CLINICAL SYMPTOMS AND SIGNS

Patients present with pain in the inguinal region that may irradiate to the hip. Abdominal wall tension or erect posturing may increase their symptoms. Occasionally, their pain may have begun after lifting a heavy weight.

Pressure medially and distal to the anterior superior iliac spine will reproduce their pain as radiation along the inguinal ligament. Sensory disturbances (hypesthesis, hypalgesia, and dysthesia) will be felt along the ilioinguinal dermatomes. Abdominal muscular weakness and atrophy can be identified by having the patient try to rise from a supine position. Contraction of the abdominal muscles will produce a protrusion of abdominal contents above the inguinal ligament. To help themselves rise, patients will pull their legs toward their bodies as they bend forward. A sudden crooked gait secondary to the syndrome has also been described.[10,11]

For technical reasons, the sensory-nerve action potential from the ilioinguinal nerve cannot be recorded; however, needle EMG examination of the transversalis muscle or the internal oblique muscle may reveal denervation if the motor branch is involved.[17,21]

The differential diagnosis must contain retroperitoneal and renal pathologies, genitofemoral nerve damage or entrapment, and meralgia paresthetica. Occurring after inguinal hernia repair, appendectomy, or cesarean sections, entrapment or damage to the genitofemoral nerve creates chronic pain and paresthesias over the upper thigh distal to the femoral triangle and in the lateral scrotum or labia,[13] as well as the loss of the cremaster muscle reflex. One should be able to distinguish meralgia paresthetica by affected dermatome examination.

TREATMENT

Removal of compressive causes as well as those causes that place the nerve under tension are necessary to relieve the syndrome. Static changes in the hip and pelvis are more difficult to treat; however, a trial of physical therapy, local corticosteroid injection, and gait modification may be

effective. If conservative therapy does not succeed, surgical therapy,[12–16] which might include sectioning of the nerve, is indicated with 78% excellent results, according to Lee and Dellon.[22] According to Benini,[16] in the frequent cases attributable to previous surgery the retroperitoneal resection of the nerve is advocated instead of a local revision, which is usually unsuccessful because of the impossibility of finding and restoring the fine nerve in a dense scar.

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34 Genitofemoral Nerve Syndrome

The genitofemoral nerve may be compressed where it passes through the psoas muscle or where its femoral and genital rami pass through the abdominal wall. Compression or traction in these areas will produce sensory changes along the skin of the medial thigh, scrotum/labia majora, and the abdominal wall below the inguinal ligament (see Figure 34.1).

ANATOMY

The genitofemoral nerve receives contributions from L1 and predominantly L2 nerve roots. The nerve passes through the psoas muscles before branching into a femoral and genital ramus. This division point is variable.

The lateral branch of the genitofemoral nerve is the femoral ramus which runs on the lateral surface of the psoas muscle behind the spermatic artery and vein and lateral to the external iliac artery covered by the iliac fascia. It crosses the deep circumflex iliac artery, passes through the lacuna vasorum and saphenous hiatus, and reaches the subinguinal region before branching to anastomose with the genital ramus and the lateral femoral cutaneous nerve.

The medial branch of the genitofemoral nerve is the genital ramus, which runs along the psoas muscle in front of the common iliac artery and external iliac artery and behind the ureter, spermatic artery, and spermatic vein. It enters the deep inguinal ring, passes through the inguinal canal, and emerges from the superficial inguinal ring, innervating the cremaster muscle, the tunica dartos, scrotal/labia majora, and the skin in the subinguinal region. In 13% of cases, the genital branch and the ilioinguinal nerve are united in the inguinal canal.[1]

ETIOLOGY

Nerve compression can occur from postsurgical scars and adhesions from herniorrhaphy (conventional or laparascopic approach), appendectomy or gynecological surgery,[2–8,10,16] psoas abscesses,[4] blunt abdominal trauma[14,15] or external compression from tight clothes or bicycling.[8] The femoral branch is usually affected by appendectomy scars.[3] Recent use of laproscopic hernia repairs have led to reports of injury to the genitofemoral and lateral femoral cutaneous nerves.[7,8]

CLINICAL SYMPTOMS AND SIGNS

Patients may complain of intermittent pains in the region below the inguinal ligament, into the scrotum or labia majora, and into the medial thigh. The affected areas depend on which nerves are affected.[5]

Signs of nerve injury include: hypesthesia in the dermatome, absent cremaster reflex, increased tenderness or symptomatology with hip extension or rotation, and radiation of pain with pressure over the deep inguinal annulus.

One must consider a list of differential diagnoses when assessing sensory disturbances in the lower abdomen (Table 34.1). Key to diagnosis is a careful delineation of what skin area is affected.[17] Ilioinguinal nerve symptoms are appreciated above the inguinal ligament, symphysis, and root of

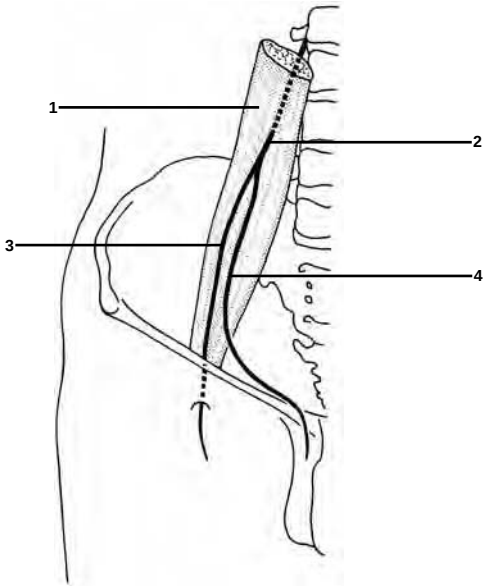

FIGURE 34.1 The genitofemoral nerve (2) can be compressed either when it passes through the psoas muscle (1) or when its ramus femoralis (3) and ramus genitalis (4) pass through the abdominal wall.

TABLE 34.1
Genitofemoral and Ilioinguinal Neuralgia

	Entrapment	
	Genitofemoral	**Ilioinguinal**
Site	Posterior abdominal wall, inguinal or femoral region	Medial to anteroposterior iliac spine
Pain	Groin, scrotum, upper thigh	Groin, scrotum, back
Sensory change	Hyperalgesia in distribution of nerve	Hypoesthesia or hypalgesia in inguinal region
Point of tenderness	± Internal inguinal ring	Medial to anteroposterior iliac spine
Hip joint movement	Hyperextension or rotation of hip increases pain	Limitation of internal rotation, extension of hip
Treatment	Excision of portion of main trunk of genitofemoral nerve	Nerve block, neurolysis, neurectomy

the penis but below the anterior superior iliac spine. Harms et al.[3] and Perry[13] recommend selective nerve block of the ilioinguinal nerve for diagnosis. If no relief is obtained, a paravertebral block of L1 and L2 is done. If eradication of pain is successful, Harms et al.[3] recommend genitofemoral nerve resection. Electrodiagnostic tests and radiological procedures may help in several of the differential diagnoses.

TREATMENT

Conservative treatment options are limited. It is not always possible to remove the causes of the compression, irritation, or tension. Local infiltration of a combination of anesthetic and corticosteroids may result in disappearance of the sensory disturbances. Local infiltration of the femoral branch is performed in the region of the lacuna vasorum, lateral to the wall of the femoral artery. Epidural infiltration of the L1 root may also be effective.[8]

When symptoms are caused by prior surgical interventions, Harms et al.[3] recommend resection of the genitofemoral nerve if conservative treatment has brought no relief. While earlier literature[10,11] recommended a transabdominal approach for resection, Laha et al.[9] have more recently described an extraperitoneal approach as used in lumbar sympathectomy. The genitofemoral nerve, as well as its branches, are identified on the psoas muscle. Two to three centimeters of nerve proximal to the bifurcation should be removed. Krahenbuhl et al.[6] recommend the retroperitoneal endoscopic technique as a new surgical approach for treating entrapment neuralgia of genitofemoral and ilioinguinal nerves. According Starling et al.[14,15] neurectomy of the genitofemoral nerve proximal to the entrapment controlled the persistent pain in 77% of the patients.

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35 Pudendal Nerve Syndrome (Syndrome of Alcock's tunnel)

The pudendal nerve is a mixed nerve innervating the perineal and genital region including the pelvic diaphragm. The nerve can be compressed when passing through the foramen infrapiriforme, the foramen ischiadicum minus, or the region of Alcock's canal. Alcock's canal is situated in the lateral wall of the ischiorectal fossa in the doubling of the fascia obturatoria. The compression of the nerve results in sensory disturbances, often of a neuralgic type.

ANATOMY

The pudendal nerve is the one of the longest nerves of the pudendal plexus. This plexus is also known as the sacral plexus. It originates predominantly from the ventral branches of the second, third, and fourth sacral nerve; but also receives tributaries of other ventral branches of the sacral nerves as well as sympathetic and parasympathetic fibers. The plexus is situated in front of the coccygeus muscle and the inferior margin of the piriformis muscle. The sacral artery and sacral veins run in front of the nerve. The plexus gives off small branches, nervi pelvici. These branches contain sympathetic and parasympathetic fibers that innervate the rectum, the inferior bladder, the urethra and the genital organs. The pudendal nerve leaves the pelvis through the foramen infrapiriforme, the distal portion of the greater sciatic foramen. It makes a loop around the spine of the ischium, behind the internal pudendal artery and reenters the pelvis together with the artery through the foramen ischiadicum minus (the lesser sciatic foramen) to reach the lateral wall of the ischiorectal fossa. In the frontal section, the ischiorectal fossa has the shape of a triangle, with the base caudal and the apex pointing cranially (Figure 35.1). The lateral limit of the fossa is the internal surface of the ischium covered by the obturator internus muscle, its fascia, the medial aspect of the levator ani muscle and its fascia. The base of the fossa is formed by subcutaneous fat covered by fascia lunata and skin. The apex corresponds to the line joining the obturator internus muscle and the levator ani muscle.

In its course through ischiorectal fossa, the nerve is situated in the doubling of the obturator fascia, also known as Alcock's canal, where the nerve is accompanied by the pudendal artery and vein. At the level of the tuberosity, the nerve gives off its branches. The inferior rectal nerve branches at the entrance of the fossa to innervate the rectum and perineal skin. The perineal nerve continues along the course of the nerve. Its superficial branch gives off not only lateral branches that innervate the anterior part of the perineal region, but also medial branches that end as scrotal branches or labial majus branches. Muscular branches from the deep branch of the perineal nerve innervate the anterior part of the sphincter ani externus muscle, the bulbocavernosus muscle, the ischiocavernosus muscle, the transversus perinei profundus muscle, and the membrane of the sphincter urethral (Figure 35.2). The terminal branch of the pudendal nerve is the dorsal nerve of the penis or clitoris. The dorsal nerve of the penis (or clitoris) passes through the urogenital diaphragm and runs together with the dorsal artery of the penis (or clitoris) to the dorsum of the penis (or clitoris) covered by corresponding fascia.

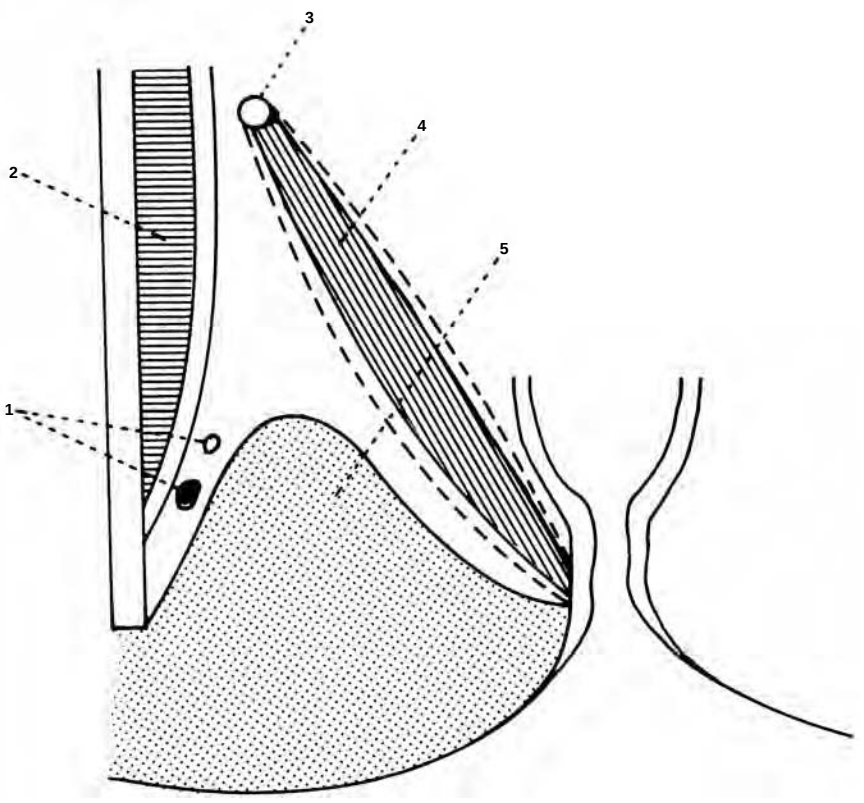

FIGURE 35.1 In the frontal section, the ischiorectal fossa has the shape of a triangle with the base caudal and the apex pointing cranially — 1: pudendal nerve and vessels in Alcock's canal; 2: obturator internus muscle; 3: tendinous arch for origin of levator ani muscle; 4: levator ani muscle; 5: fat tissue covered by fascia lunata.

ETIOLOGY

Different expansive processes in the pelvic region, especially tumors, may cause compression of the pudendal nerve. These tumors include primary or metastatic tumors of the urogenital system, tumors of the cauda equina, neurofibromas of Recklinghausen's disease or solitary neurofibromas as described by Tognetti and col.[4] Trauma to the pelvic region with or without hematomas or surgery[2] can result in compression of the pudendal nerve, especially when the nerve runs through Alcock's canal. Analogous to other tunnel syndromes, thickening of the obturator fascia, which surrounds the nerve, may cause compression of the nerve in neuralgias assumed to be idiopathic. Factors causing peripheral neuropathies including diabetes, alcocholism, rheumatoid arthritis and hypothyroidism should be assessed while taking a patient's history. Some sport activities (bicycling, motorcycling and horseback riding) increase pressure in, produce blows to, or create repetitive vibrations in the pelvic region.[1]

CLINICAL SYMPTOMS AND SIGNS

Pudendal nerve syndrome is manifested clinically with sensory disturbances, pudendal neuralgias, or chronic pain. Characteristically, pudendal neuralgia has paroxysmal attacks that may be spontaneous or caused by touching the innervated region. These aggravating factors can be as simple as sitting, coitus, or the increase of intrapelvic pressure with urination or defecation. The syndrome

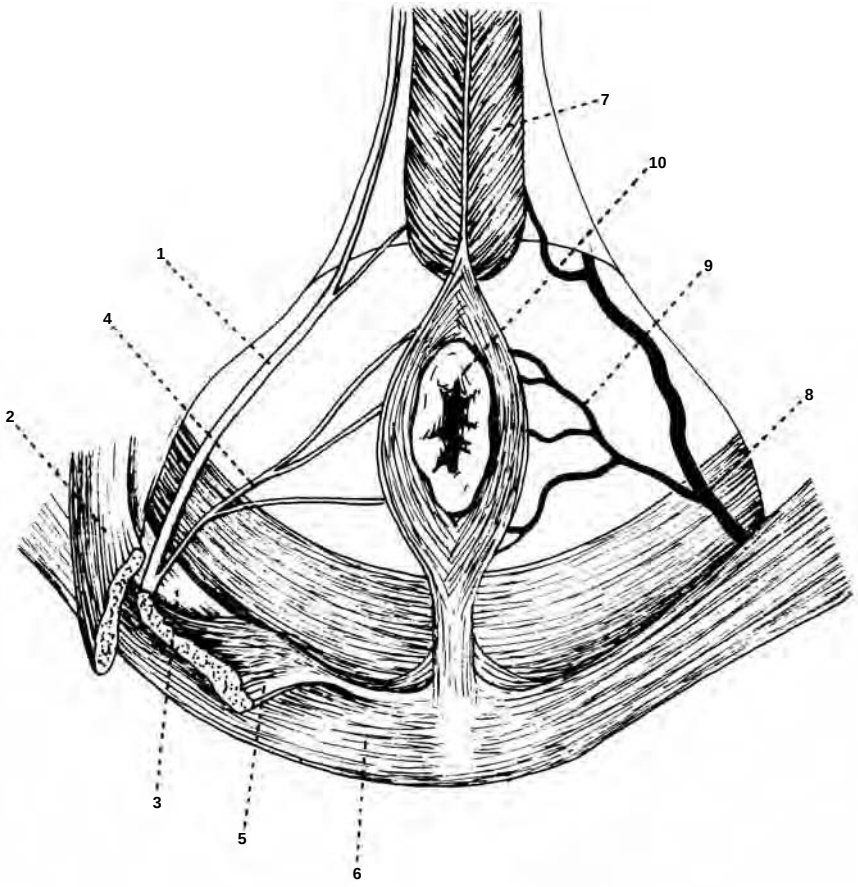

FIGURE 35.2 At the level of the tuber ossis ischii, the pudendal nerve gives off its branches — 1: pudendal nerve; 2: gluteus maximus muscle; 3: sacrospinal ligament; 4: perineal nerves; 5: gluteus maximus muscle; 6: gluteus maximus muscle; 7: bulbospongiosus muscle; 8: arteria pudenda interna; 9: arteria rectalis inferior; 10: anus.

may result in permanent burning or paresthesias radiating to the pelvic floor. Disturbances are intensified in the sitting position and diminish with an upright posture and walking. This situation is because sitting places the nerve in tension during its course through Alcock's canal. Motor disturbances have not been mentioned in the literature, although such disturbances could be expected as result of longer compression. A multitude of diagnostic tools (i.e., plain radiographs, CT, MRI, teletermogaphy, EMG/NCV, somatosensory evoked potentials) may be necessary to exclude other causes of this regional pain. The best objective diagnostic method is electrodiagnostic testing, which can exclude psychogenic neuralgias.

Electrodiagnostic testing is a very delicate method used to evaluate the muscles of the pelvic diaphragm, the latency of the bulbo-cavernous and bulbo-anal reflexes (by stimulating afferent sensory fibers). Registration of these evoked somatosensory potentials, cortical as well as medullary, make it possible to differentiate the disturbances in the medullary region among the roots of the pudendal plexus. It is also possible to measure the distal motor latency on pudendal muscle by endorectal stimulation of the nerve. This occurs at the level of the ischial spine and registers in the bulbocavernous muscle, the sphincter urethrae, or sphincter ani muscles. However, the diagnosis is often primary or pure (idiopathic) pudendal neuralgia.

TREATMENT

Conservative measures need to address the initiating factors. This intervention might be as simple as adjusting one's bike seat position, its padding, or one's training regime. With the failure of simple adjustments, initial treatment is anesthetic infiltration of the ischiorectal fossa by anesthetics under radiographic control (X-rays or ultrasound). The infiltration can be done through sacral openings or through the sacral hiatus. Electrostimulation has also been tried. In the case of unsuccessful conservative treatment, or in the case of evident compression of the nerve, operative treatment is indicated by transgluteal or transperineal approach.

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Section IV

Tunnel Syndromes in the Lower Extremities

36 Introduction

Nerve compression in the lower extremities represents a large proportion of a physician's practice. The list of differential diagnoses can be quite long, and without appropriate testing, many true diseases may be missed and many backs operated upon unnecessarily. Mignoucci and Bell[1] discuss the differential diagnosis and approach to the lower back in great detail. The aproach should still follow the steps outlined in the introduction. Yielding a treasure of detail, the history will indicate the avenues to search.

Lower back pain can occur simultaneously with tunnel syndromes in the lower extremity, further confusing the situation. Patients will complain of sciatica, pain radiating down from the back into the legs. The overlapping presentations of many diseases further cloud the issue. Degenerative or traumatic disc disease, spinal stenosis, referred pain from bony disease in this extended region, peripheral vascular disease, malingering, psychiatric disturbances, trauma, infection, inflammation, tumors (intrinsic or extrinsic to the nerve), neuropathies, or hormonal and metabolic disturbances can masquerade as peripheral nerve entrapment. Saal et al.[2] suggested that nerve irritation leads to substance release into the spinal cord, increasing the sensitivity of nearby nerves to pain.

Initial treatment of lower back pain involves bed rest, anti-inflammatories, analgesics, physical therapy, modalities, education, and job modification. Typically, 50% of patients with lower back pain recover within a month.[3] In fact, fewer than 3 to 10% of all patients with unrelenting sciatica ever require surgical intervention.[4] Evaluation of patients with chronic back pain typically does not yield a surgically correctable lesion;[4] therefore, cautious intervention is required. However, these same patients become quite familiar with physicians' expectations and can present quite intriguing symptoms. From among all these patients comes the select group with tunnel syndromes of the lower extremity.[5]

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37 Lumbosacral Tunnel Syndrome

Compression of the fifth lumbar nerve root represents one of the most common complaints of patients with lower back pain and sciatica. While this nerve root is commonly involved by a herniated disc or spinal stenosis, the fifth nerve root can be compressed after it leaves the intervertebral foramen and crosses the alla of the sacrum under the lumbosacral ligament (Figure 37.1). This region is commonly known as the lumbosacral tunnel.

ANATOMY

With its large fibrous band forming the ventral side of the fibro-osseus tunnel, the lumbosacral ligament originates from the fifth lumbar vertebra and inserts on the upper border and anterior surface of the alla of the sacrum. Present in everyone, the ligament varies in width, thickness, and shape.[1] Its origin may be the body of L5 and its transverse process (73%), the body solely (20%), or the transverse process solely (7%). The alla or the wing of the sacrum forms the posterior wall of the fibro-osseus tunnel. Briggs and Chandraraj,[8] and Hardy[9] reported about variations in the lumbosacral ligament and associated changes in the lumbosacral region resulting in compression of the fifth dorsal root ganglion and spinal nerve. Sixty-five lumbosacral regions from adult cadavers were disected by Briggs and Chandraraj[8] and the position and relations of the lumbosacral ligament noted. The lumbosacral ligament was present in all specimens; in 22 (34%) it extended medially across the ventral ramus of the fifth lumbar nerve, and in six (9%) of these the underlying nerve was compressed and visibly flattened.

The fifth lumbar nerve leaves the intervertebral foramen and runs inferolaterally, crossing the upper border of the sacrum before reaching the anterior surface of the sacrum. Over this path, the nerve courses under the lumbosacral ligament. Sympathetic nerve branches pierce the superior border of the ligament and join with the nerve. The branch of the fourth lumbar nerve root runs over the anterior surface of the lumbosacral ligament to join the fifth nerve root as it exits from the inferior border of the ligament forming the lumbosacral trunk. The lumbosacral tunnel also contains branches of the iliolumbar arteries and veins and may even contain a venous plexus. Lumbosacral nerve-root anomalies are not rare in this region.[2] The lumbosacral tunnel may be considered as an extension of the L5–S1 intervertebral foramen, through which pass the L5, the sympathetic ramus communicans to this nerve, and branches of the iliolumbar arteries and veins.

ETIOLOGY

As in all tunnel syndromes, nerve compression occurs when disease processes alter the volume of the lumbosacral tunnel. Marginal osteophytes on L5 and S1 can be so exuberant that they can form a compressive anteromedial wall.[3,4] Thickening of the ligament can flatten the nerve against the posterior sacrum wall.[1] Intrinsic and extrinsic neural tumors will decrease the space in the tunnel. Additionally, disorders of the tunnel's vascular components may compress the nerve. These include aneurysms, tortuosities, and venous dilatations.[1] Regional inflammation can lead to local tissue edema and compression. Bony changes, including primary and secondary tumors of the sacrum, ilium, and spine; fractures of the pelvic ring and sacrum; and motion of the sacroiliac and lumbosacral spine may produce symptomatic nerve compression.[5]

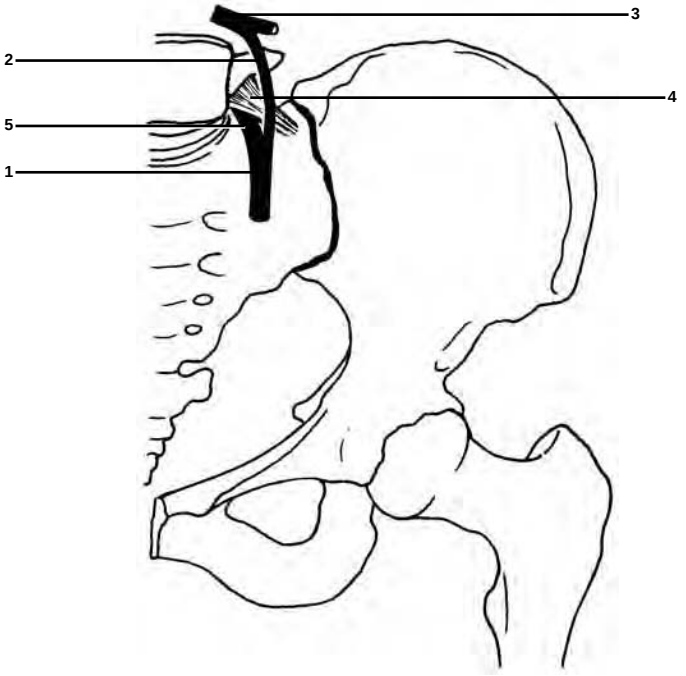

FIGURE 37.1 The anatomical relationships of the lumbosacral tunnel — 1: lumbosacral trunk; 2: branch of L4; 3: L4 root; 4: lumbosacral ligament; 5: L5 root.

CLINICAL SYMPTOMS AND SIGNS

The symptoms and signs of lumbosacral tunnel syndrome are found in the L5 distribution. One will find decreased sensation and pain in the L5 dermatome. Weakness and atrophy usually are not observed; therefore, the diagnosis of the lumbosacral tunnel syndrome requires exclusion of the other causes of L5 radiculopathy, as mentioned earlier.

TREATMENT

When the syndrome is not caused by tumors or aneurysms, conservative therapy should be applied along the accepted courses for relief of radicular pain.[6] Chayen et al.[7] describe the use of anesthethic blocks. Surgical release remains the last resort, with care being taken not to include the branch of the fifth lumbar root while sectioning the lumbosacral ligament.

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38 Gluteal Nerve Syndromes

The superior and inferior gluteal nerves originate from the sacral plexus and course through the infrapiriform and suprapiriform foramen (Figure 38.1). Compression of these nerves results in muscular dysfunction, whether loss of function or decreased strength of the gluteal muscles, and occasionally the tensor fascia lata muscle.

ANATOMY

The superior gluteal nerve originates from the lumbosacral trunk formed by the roots of L4 and L5, which combine with the sacral plexus before separating again. Akita et al.[1] found this origin to be proximal and dorsal to the origin of the inferior gluteal nerve. The nerve leaves the pelvic region with the superior gluteal artery through the suprapiriform foramen. The boundaries of this foramen are the superior edge of the piriformis muscle, the lower edge of the gluteus medius muscle, and the ischium in the area of the greater sciatic notch. The piriformis muscle splits the greater sciatic notch in two, producing a superior and an inferior foramen. As the nerve passes through the superior foramen, it branches to supply the gluteus medius and minimus muscles, with an inferior ramus supplying the tensor fasciae latae muscle. Chiba[4] notes the number of variations in the nerve course. In the most common case, the muscle was pierced only by the superior gluteal nerve (60%).

The inferior gluteal nerve originates in a similar fashion from the lumbosacral trunk but has its innervation from the S1 and S2 roots. The nerve enters the infrapiriform foramen with the sciatic nerve, which lies laterally and courses inferiorly. The inferior gluteal nerve exits the foramen and ramifies to innervate the gluteus maximus muscle and the hip capsule.

ETIOLOGY

Isolated compression injury of these nerves is rare. Their course and position render them susceptible to injury from pelvic fractures, hip surgery, and intramuscular injections.[3–5,7,9,10] Traction during hip replacement surgery can stretch and injure these nerves. Stohr[17] reviewed 53 patients with lumbosacral plexus injuries and found that 31 were caused by pelvic trauma and 22 by hip surgery. This review emphasizes the need for careful evaluation in the trauma situation and avoidance of prolonged or excessive traction during total hip replacement procedures. Dhillon and Nagi note that limb lengthening more than 4 cm may have an effect.[7]

Additionally, lumbosacral injuries may involve a variety of nerves. Twenty-seven cases of incomplete paresis of the sacral or lumbosacral plexus were found to affect the common peroneal nerve (78%), the gluteal nerves (11%), the tibial nerve (7.8%), and the obturator nerve (3.5%).[17]

Isolated compression of the inferior or the superior gluteal nerves is rare and has been reported by several authors, as shown in Table 38.1.[6,8,13,15–17] Nerve compression from piriformis muscle hypertrophy has been reported,[12] although it remains controversial. The piriformis muscle syndrome is detailed in another chapter. Patients with coagulopathy can have nerve compression from hematomas either spontaneously or following intermuscular injections.[15,16] Blunt trauma to the gluteal region can injure these nerves but typically produces a mixed picture. Stohr noted sciatic nerve

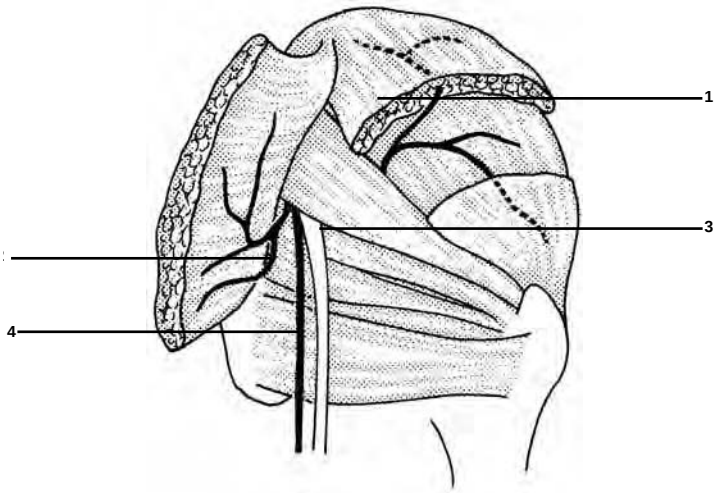

FIGURE 38.1 The gluteal nerves can be compressed in their course through the foramen suprapiriforme and/or foramen infrapiriforme — 1: superior gluteal nerve; 2: inferior gluteal nerve; 3: sciatic nerve; 4: cutaneous femoris posterior nerve.

TABLE 38.1
Several Etiologies of Gluteal Nerve Compression

Nerve Affected	Site or Cause	Presumed Initial Etiology	Author(s)
Superior gluteal	Foramen/muscle borders	Spondylolisthesis	Rask[13]
Superior/inferior	Piriformis muscle bellies	Spondylolisthesis	de Jong and van Weerden[6]
Superior/inferior and sciatic	Direct pressure	Coma/anesthesia	Stohr[16,17]
Superior gluteal		tumor	Paik et al.,[11]
		scar tissue	Gulec and Buyukbebeci, [8]
Superior/inferior and sciatic	Hematoma	Injection	Stohr[16,17]

injury in five patients, with two having gluteal nerve injury following comas or general anesthesia.[15,16] Nerve injury was presumed to have come from direct pressure. Four of these patients had an incomplete and delayed recovery. Pelvic fractures can distort the anatomy, leading to stretch injuries to the lumbosacral plexus and gluteal nerves.[8,9]

CLINICAL SYMPTOMS AND SIGNS

Gluteal nerve compression leads to a weakness or loss of function in the muscles of innervation. The gluteus medius and minimus muscles and the tensor fasciae latae muscle (superior gluteal nerve) abduct the leg and stabilize the pelvis during a one-legged stance. Therefore, injury to the inferior gluteal nerve would produce a pelvic tilt to the opposite side during gait or while one stands on the affected leg, producing the Trendelenburg sign. During gait, affected patients will lean toward the affected side to balance their center of gravity, which is also known in the French literature as the *boiterie de l'épaule*.[14] Clinical examination both in gait and in stance will confirm the diagnosis. Tackmann et al.[18] recommend also observing patients as they try to hop on the affected leg. One should make sure that patients do not try to stabilize themselves by propping

their good legs against the affected legs during testing. All of these signs are best seen from the rear of the patient.

The gluteus maximum functions to extend the hip and to maintain an upright posture. Inferior gluteal nerve injury affects this action. Patients typically complain of problems with climbing stairs, standing from a sitting position, or rising from a couch. One can isolate the gluteus maximum by placing patients prone on a table and having them try to extend their hips. Limited extension can be initiated by the gluteus medius muscle. However, one can apply resistance to the thigh and determine the patients' strength. As this muscle is key in maintaining one's posture and climbing stairs, one can assume that its strength should be proportionate.

TREATMENT

As isolated gluteal nerve compression is rare, its treatment should be based on a conservative approach, including observation and physical therapy, which may include electrotherapy of the muscles. Physical therapy is key to preserving one's range of motion should the nerves recover. Reports of reinnervation over 4 to 8 months have been reported.[9,15–17] Physical therapy might need to continue for up to 3 years. The best treatment is to protect the patient who is comatose or under anesthesia from having direct pressure in this region. Direct penetrating trauma, iatrogenic injury, or severe pelvic misalignment may require early surgical intervention to correct. Dhillon's series of sciatic nerve injuries following total hip arthroplasty did not fare as well as peroneal nerve palsies.[7] Gulec and Buyukbebeci[8] describe late superior gluteal nerve palsy following posterior fracture-dislocation of the hip. The palsy resulted from traction by scar tissue formation, and excision of the scar tissue and decompression of the superior gluteal nerve led to complete recovery.

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39 Iliacus Muscle Syndrome

The femoral neurovascular bundle and the iliopsoas muscle pass under the inguinal ligament to supply the leg. The iliac fascia forms an iliopectineal arch that connects the inguinal ligament to the iliopubic or iliopectineal eminence and divides the space beneath the inguinal ligament (Figure 39.1). The lacuna vasorum or vascular tunnel lies medial to the arch. Through the lateral space of the lacuna muscularis runs the femoral nerve and the iliopsoas muscles. Relatively rigid, the lacuna muscularis represents a tunnel, the walls of which are the iliac bone, iliopsoas muscle, iliopectineal arch, and inguinal ligament. Described by Aichroth and Rowe-Jones in 1971,[1] the syndrome of the iliacus muscle, or the iliacus tunnel syndrome, occurs with femoral nerve compression.

ANATOMY

The femoral nerve originates from roots L2, L3, and L4 below the psoas muscle. Passing between the iliacus and psoas muscles, the nerve supplies both muscles and enters the leg with the combined iliopsoas tendon under the inguinal ligament. One of two terminal divisions of the femoral nerve, the superficial nerve branch supplies motor rami to the pectineus and sartorius muscles, as well as sensation to the anterior thigh via the anterior cutaneous ramus. The deep branch supplies the quadriceps femoris musculature and produces the saphenous nerve, which supplies sensation to the medial thigh, leg, and foot. All anterior thigh muscles except the tensor fascia lata are innervated by the femoral nerve. Sensory disturbances due to femoral nerve compromise would be seen over the anterior thigh, the anterior medial area of the knee, the medial leg, and the medial portion of the foot.

ETIOLOGY

Since the femoral nerve lies in the pelvic basin, surgical procedures within the pelvis are the most common cause of femoral nerve compression. Hematomas and dynamic relationships can also compress the nerve. Bleeding within the nerve sheath produces intrinsic compression, whereas bleeding external to the sheath can still narrow the tunnel significantly (Table 39.1).

One might postulate that arteriovenous malformations, vascular aneurysms, femoral vessel catheterization,[36] muscle tumors, retroperitoneal sarcoma,[37] bony disruptions due to traumas, subperiostal hematoma,[38] or hernias also can lead to femoral nerve compromise. A case report of post-irradiation femoral neuropathy was also described.[39]

Femoral compressive neuropathy can complicate anterior lumbar interbody fusion. This is a result of tight constriction of the nerve by a muscular portion of the psoas muscle, when both the lumbar spine and the hip are immobilized in the position of the maximum stretch of the muscle.[40]

CLINICAL SYMPTOMS AND SIGNS

Symptoms can be predicted using one's knowledge of the femoral nerve's anatomy and the level of compressive lesion. Patients with high lesions (often termed paresis of the superior type) have difficulty standing from a seated position due to iliopsoas muscle weakness. High lesions will

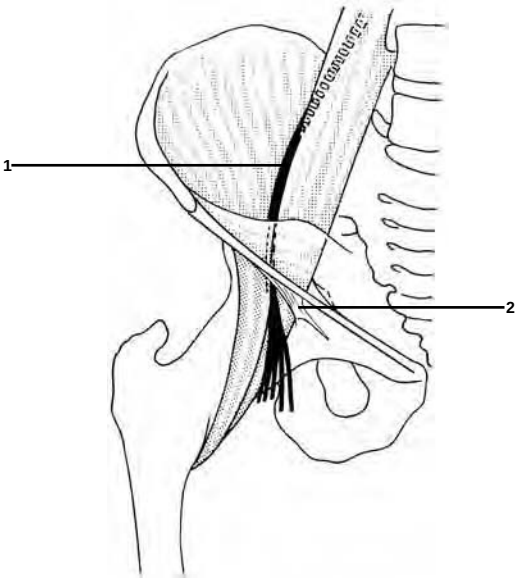

FIGURE 39.1 The course of the femoral nerve (1) as it crosses the iliacus muscle and pelvis to pass near the iliopectineal arch (2) to supply the leg.

TABLE 39.1
Proposed Etiologies for Femoral Nerve Compression

Etiology	Author(s)
Extrinsic	Gumpertz, 1896[3]
Hysterectomy and gynecological operation	Adler et al., 1956;[4] Krone, 1972;[5] Komar, 1977;[2] Kvist-Poulsen and Borel, 1982;[6] Raber and Schneider, 1993;[7] Helbling et al., 1994[8]
Renal transplantation	Vaziri et al., 1976;[9] Sisto et al., 1980;[10] Probst et al., 1982[11]
Retractor placement	Buchbender and Weiss, 1961[12]
Spontaneous retroperitoneal bleed	Seddon, 1930;[13] Tellroth, 1939;[14] Hall, 1961;[15] Lang, 1966;[16] Fearn, 1968;[17] Cianci and Piscatelli, 1969;[18] Mastroianni and Roberts, 1983;[19] Merrick et al., 1991[20]
Iliacus or psoas muscles bleed	Goodfellow et al., 1967;[21] Kubacz, 1971;[22] Stören, 1978;[23] Uncini et al., 1981;[24] Nobel et al., 1980;[25] Berlusconi and Capitani, 1991;[26] Niakan et al., 1991;[27] Rosset et al., 1991;[28] Kumar et al., 1992;[29] Jarnjoom et al., 1993;[30] Guivarc'h, 1997;[41] Guivarc'h, 1997[41]
Intrinsic (hematoma within the nerve)	Bigelow and Graves, 1952[31]
Functional (in sport or action)	Aichroth and Rowe-Jones, 1971[1]
Hip hyperextension	Koll, 1957;[32] Luft, 1963;[33] Brozin et al., 1982;[34] Milgram et al., 1984[35]

also compromise lower femoral nerve function. Patients with iliacus tunnel syndrome (paresis of the inferior type) have difficulty extending their knees. They also have hypotrophy of the anterior thigh compartment. Hip extension aggravates the pain, while other movements of the hip are painless. The patellar reflex typically disappears. Sensory disturbances will be appreciated throughout the femoral nerve's dermatome. Radiculopathy would present segmental sensory disturbances. Quadriceps hypotrophy has neither the sensory disturbances nor the pain associated with iliacus tunnel syndrome.

TREATMENT

Komar[2] recommends conservative therapy, as the risks of surgical exploration outweigh its benefits. Physical therapy may stabilize the quadriceps hypotrophy. The best treatment lies in avoidance of surgical damage during pelvic procedures and reversal of coagulopathies and other causes. Guivarc'h[41] recommend surgical treatment in case of hematoma of the iliopsoas muscle.

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40 Obturator Tunnel Syndrome

While leaving the pelvis and entering the thigh, the obturator nerve passes through a fibro-osseus tunnel, the obturator tunnel. Compression of the obturator nerve can mimic many different disorders, ranging from herniated discs to nerve compression at other levels in the pelvis.

ANATOMY

Originating from the second, third, and fourth lumbar nerve roots of the lumbar plexus, the obturator nerve passes below the psoas muscle, crosses over the sacroiliac joint, and runs along the pelvic wall to reach the obturator tunnel (Figure 40.1).

The obturator sulcus of the pubic bone forms the roof of the tunnel. The floor consists of the internal and external obturator muscles. The obturator membrane separates the muscles and joints with their fascias to create an inelastic floor of the oblique tunnel. The obturator artery, two veins, and multiple lymph nodes can be found alongside the obturator nerve in the tunnel. Within the tunnel, the nerve divides into an anterior or superficial branch, a posterior or deep branch, and a branch to the external obturator muscle.

As the nerve exits the tunnel, the anterior and the posterior branches are separated by the adductor brevis muscle. The anterior branch innervates the pectineus, adductor longus, adductor brevis, and gracilis muscles. Its terminal branch supplies sensory fibers to the skin of the medial thigh. The posterior branch supplies the adductor brevis and the adductor magnus muscles. The obturator nerve also supplies sensation to the knee joint and the medial side of the knee. Lolić-Draganić and Illić[1] estimate that 9% of individuals have an accessory obturator nerve that leaves the pubis through either muscular or vascular passages (lacunae musculorum or vasorum) (Figure 40.2).

ETIOLOGY

The course of the obturator nerve places it at risk from disorders of all organ systems in the pelvis as well as from compression within the tunnel. The nerve can be compressed within the true pelvis by pelvic fractures, pelvic hematomas secondary to anticoagulation or trauma,[2] retroperitoneal masses,[3] and intrapelvic tumors. Normal life events such as pregnancy can create complications that lead to obturator nerve or obstetrical palsy.[4–6,17]

While the obturator tunnel is well protected from direct trauma by neighboring muscles, neuropathy due to direct damage may occur. Complications of genito-urological surgery or total hip arthroplasty[7–9] may traumatize the nerve in its tunnel. Anticoagulation leading to minor bleeds within the tunnel, [2] aneurysms of the obturator artery,[10] bone osteophytes, [11] obturator hernias,[12] bone cement after total hip arthroplasty,[21] or tissue edema due to inflammation decrease the amount of space available for the nerve. Of all the tunnel components, the obturator nerve is the least tolerant of compression and can also lead to nerve compression. Kopell and Thompson[13] describe obturator tunnel syndrome related to inflammatory changes of the pubic bone in osteitis pubis. Bradshaw et al.[18,19] described sport-related entrapment of the obturator nerve.

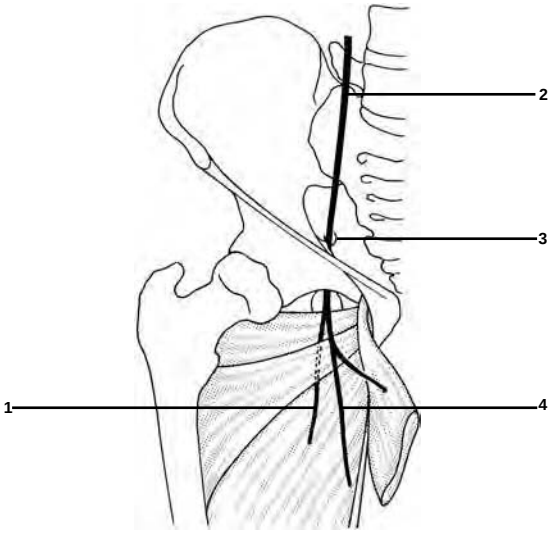

FIGURE 40.1 The relationship of the obturator nerve and its branches to the internal rotators of the hip and the adductor muscles — 1: posterior (profundus) branch of the obturator nerve; 2: obturator nerve; 3: obturator canal; 4: anterior (superficial) branch of the obturator nerve.

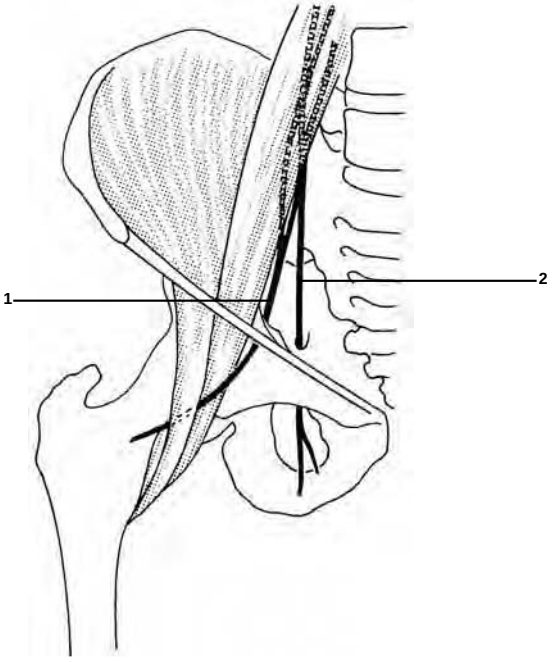

FIGURE 40.2 Variations in the obturator nerve can occur with an accessory obturator nerve (1) crossing the pelvis in a separate location from the obturator nerve (2).

CLINICAL SYMPTOMS AND SIGNS

While osteitis pubis causes mild local pain, obturator tunnel syndrome causes strong nonlocalized pain and resting pain with radiation from the symphysis pubis to the knee. Medial knee pain, known as Howship Romberg's symptom,[14,15] develops with nerve compression. Occasionally, sharp pain

may be felt in the posteromedial knee. This area receives innervation from the posterior obturator nerve branch. With prolonged compression, adductor paresis may develop; however, the adductor longus muscle and the adductor magnus muscle receive some innervation from the femoral and sciatic nerves, respectively. Adductor paresis produces a characteristic gait with circumduction of the affected leg. Deep non-localized pain and spasm may develop among the adductor muscles. Some investigators question whether adductor spasm can lead to obturator tunnel syndrome. In review, medial knee pain[3] or adductor spasm combined with medial thigh and knee pain[16] can be considered characteristic of obturator tunnel syndrome.

TREATMENT

Primary obturator tunnel syndrome can be treated conservatively with physical therapy and rest; however, obturator tunnel syndrome secondary to pelvic pathology requires treatment of the cause. With the exception of coagulopathies, these causes must be treated surgically via an intra or extraperitoneal approach.[20,21]

CASE REPORT

In a female patient with osteoarthritis a total hip replacement with cemented prosthesis was performed. Immediately after the surgical intervention, the patient complained of debilitating pain spreading from the pubic region along the medial side of the thigh to the knee. The weakness of the adductor muscles and hypoesthesy of the distal medial part of the left thigh were found. The symptoms indicated a possible lesion of the obturator nerve. Standard postoperative X-ray pictures displayed extrusion of the bone cement in a medial direction, while axial pictures of the hip joint showed the exact localization of the extrusion of cement, medially in the anterior part of incisura acetabuli at the exit of the obturator tunnel (Figure 40.3). Electromyoneurography (EMNG)

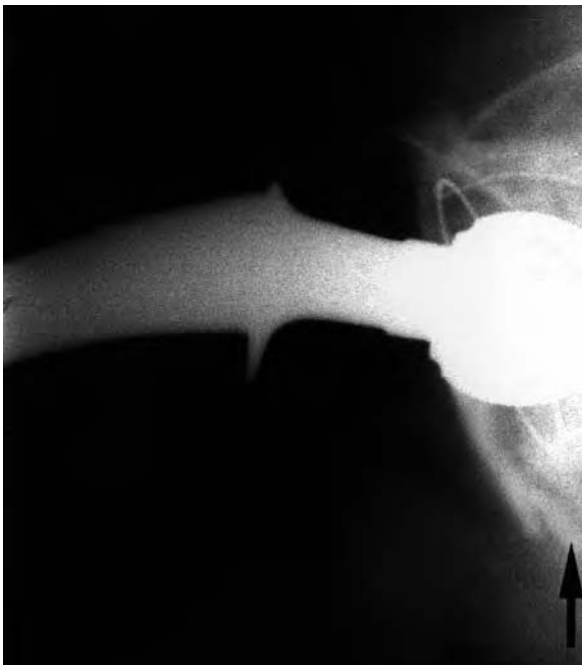

FIGURE 40.3 The axial X-ray picture of the hip joint shows the extrusion of cement medially in the anterior part of incisura acetabuli at the extra-pelvic exit of the obturator tunnel (black arrow).

demonstrated the lesion of the obturator nerve. As physical treatment gave no results, and electromyographic examination similarly brought no improvement, surgical intervention, i.e., inspection of the obturator nerve, was indicated. Surgical intervention was performed by an extra-abdominal approach to the extra-pelvic exit of the obturator tunnel. There, a sliver of bone cement was found to have compressed the nerve at the exit of the obturator tunnel. The integrity of the nerve and its conduction were still intact, based on intraoperative stimulation above the level of the cement. A 2.5-cm-long piece of cement was chiseled off from its base in the region of the incisura acetabuli and removed. Immediately following the surgical procedure, the patient ceased to complain of pains spreading from the inguinal region to the knee. A weakness of the muscles remained as well as limping. An aggressive continuous rehabilitation program was applied. Six months after decompression of the obturator nerve, both clinical and electromyographic results were above expectation, allowing the patient to return to her job. Clinical and EMNG findings taken 1 year after decompression of the nerve showed signs of further recovery. Clinical findings were normal when compared with the other extremity.

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41 Cutaneous Femoris Posterior Nerve Syndrome

Nervus cutaneous femoris posterior is a cutaneous nerve innervating the distal part of the gluteal region, perineum, and posterior part of the thigh. It can be compressed when passing through foramen infrapiriforme (see Figure 38.1) and in its course under musculus gluteus maximus, resulting in sensitive disturbances in the innervation area of the nerve.

ANATOMY

Nervus cutaneus femoris posterior originates from the posterior surface of the sacral plexus by two or three roots,[6] alone or together with the nervus gluteus inferior, in which case it is called (according to Bichat) nervus gluteus minor. Running between the piriformis muscle and plexus sacralis, it leaves the basin together with the nervus gluteus inferior, posterior to the nervus ischiadicus, and through the foramen infrapiriforme. In the gluteal region, the nerve is covered by the gluteus maximus muscle, posterior to the ischiadicus nerve. In the thigh, it runs between the biceps femoris and semitendinosus muscles below the fascia lata. The nerve gives off terminal branches that pierce the fascia lata and ramify in the skin of the posterior surface of the thigh. Besides its terminal branches, the nerve gives off collateral branches: clunium inferiores nervi for the skin and distal gluteal area and rami perineales for the perineum and posterior part of the labia majora.

ETIOLOGY

Etiological factors producing spasm or prolonged contraction of the piriformis muscle, hypertrophy of the pirioformis muscle, and narrowing of the foramen infrapiriforme[7] (described in detail in Chapter 42) may lead to compression of the cutaneus femoris posterior nerve. Arnoldussen and Korten[1] describe three patients with compression of the cutaneus femoris posterior nerve due, in their opinion, to compression of the nerve below the gluteus maximus muscle as a result of a long-held sitting position.

CLINICAL SYMPTOMS AND SIGNS

Clinical symptoms of nervus cutaneus femoris posterior syndrome are sensitive disturbances characteristic of the cutaneous nerve in its innervation area, i.e., in the posterior surface of the thigh to the knee, in the distal and internal part of the gluteal region, and in the posterior part of the perineum, labia majora, or scrotum (Figure 41.1). These disturbances can vary from hypo- to hyperesthesia and a sensation of burning, similar to meralgia paresthetica. These symptoms were reported in a 56-year-old man with discomfort after falling on his right buttock 10 years earlier.[2] The electrophysiological technique to assess the posterior femoral cutaneous nerve was described by Dimitru and Nelson.[3] Somatosensory-evoked potentials have also been used to assess this nerve.[4] In localization of sensitive disturbances, telethermography is of great help. In a differential diagnosis of lumboischialgia, the same signs can be applied as described for piriformis muscle syndrome (Chapter 45). According to Reid and Cros[8] the differential diagnosis includes a sacral radiculopathy

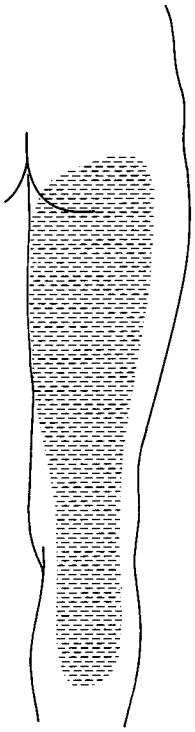

FIGURE 41.1 Localization of sensitive disturbances when the cutaneous femoris posterior nerve is compressed.

and this can be distinguished from posterior femoral cutaneous neuropathy by a hypoactive ankle jerk and the presence of myotomal muscle weakness.

TREATMENT

Treatment is, in general, conservative and consists of avoiding the possible causes of the syndrome such as sitting for a long time on a hard base, physiotherapy, and application of anesthetics and corticosteroids.[5]

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42 Piriformis Muscle Syndrome

The sciatic nerve passes through the greater sciatic foramen in proximity to the piriformis muscle (Figure 42.1). Compression in this fibro-osseus tunnel may result in the clinical picture of the piriformis syndrome. In 1928, Yeoman[1] described the importance of the neuromuscular relationship in the development of lumbosacral neuralgias. Since the L4–L5 and S1–S2 distributions are affected, the list of differential diagnoses can be quite long.

ANATOMY

The sacrospinal and the sacrotuberous ligaments connect the ischial spine and the ischial tuberosity, respectively, to the sacrum and delineate two foramens: the greater (foramen ischiadiacum majus) and the lesser (foramen ischiadiacum minus) sciatic foramens. The greater (incisura ischiadica major) and the lesser (incisura ischiadica minor) sciatic notches lie at the apex of each foramen along the posterior ischial border. The majority of important structures connecting the gluteal region with the pelvis run through the greater sciatic foramen; therefore, this area has been known as the hilus of the gluteus or the Gibraltar of the gluteus. The piriformis muscle splits the foramen into the suprapiriformis region and the infrapiriformis region. The superior gluteal nerve and vessels leave through the suprapiriformis region.

The infrapiriformis region or foramen is triangular in shape, delineated by the inferior margin of the piriformis muscle superiorly, the sacrospinous ligament inferiorly, and the bony margin of the greater sciatic notch laterally. Two groups of neurovascular structures leave the pelvis through this region. The medial group includes the pudendal neurovascular bundle. The lateral group consists of the sciatic nerve, the inferior gluteal nerve, the posterior cutaneous nerve of the thigh, and the inferior gluteal vessels. The sciatic nerve represents two terminal nerves of the sacral plexus: the tibial nerve, which is the ventral portion of the plexus (L4–L5; S1–S2), and the common peroneal nerve, which is the dorsal portion of the sacral plexus (L4–L5; S1–S2). While considered an extremely important nerve, the sciatic nerve enters the thigh as one or two nerves. Studies by Pećina[2] revealed intrapelvic division in 26.5% of the cases, postforamen division in 4.6% of the cases, division at the inferior border of the gluteus maximus muscle in 11.5% of the cases, and division in the thigh in the rest of the cases (Figure 42.2). Occasionally, the common peroneal nerve can pass through the piriformis muscle (long, flat, or pear-like), separating the muscle into two bellies.[3–9] The piriformis muscle has been shown to exist in two bellies in 18% of the population.[2] The sacral plexus and branches of the internal iliac artery lie between the rectum and the piriformis muscle, which covers the anterior surface of the sacrum.

Surgical approaches in the gluteal region, especially with hip surgery, use the piriformis muscle for orientation, since it connects the sacrum and the greater trochanter of the femur. The position and direction of the upper muscular margin can be found by following the superior iliotrochanteric line, which connects the posterior superior iliac spine with the apex of the greater trochanter (Figure 42.3). The inferior iliotrochanteric line runs parallel and 3 cm distal to the superior iliotrochanteric line. The inferior line will indicate the upper margin of the infrapiriformis foramen or region.

The contents of the foramen and their relative volumes play major roles in the development of nerve compression. The size of the piriformis muscle belly may vary greatly, thereby narrowing

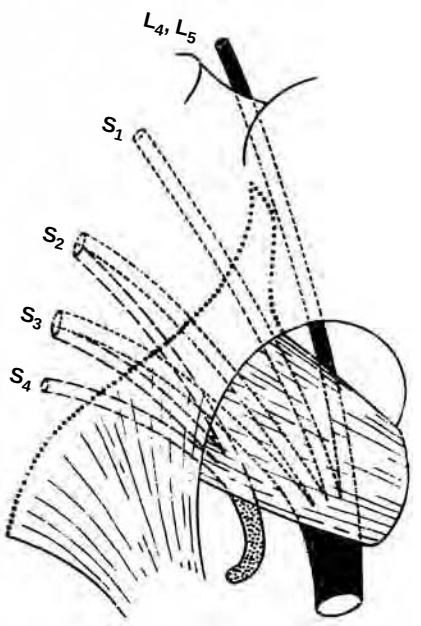

FIGURE 42.1 The sciatic nerve lies near the piriformis muscle and can even run through it in several variations.

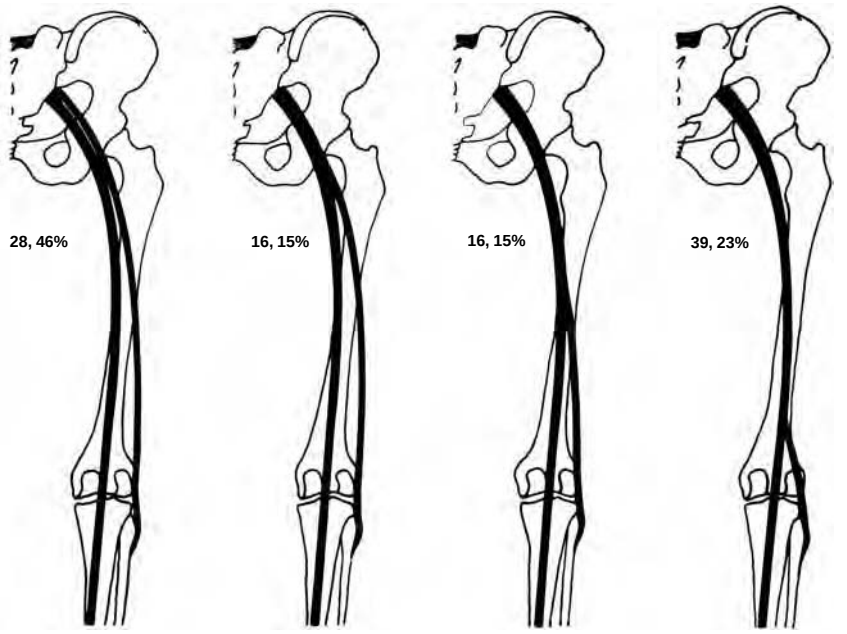

FIGURE 42.2 The most common variations in the form taken by the sciatic nerve as it courses down the lower extremity. The most common forms occur with the sciatic nerve dividing before leaving the pelvis or just proximal to the knee.

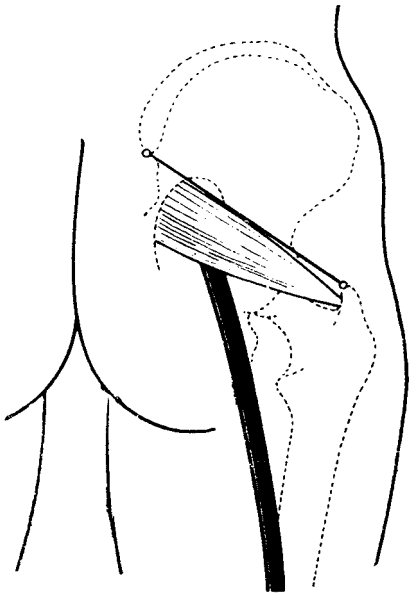

FIGURE 42.3 Projection of the piriformis muscle on the surface anatomy of the gluteal region.

the foramen. In up to 50% of the population, a synovial bursa may exist between the tendon of the piriformis muscle and the bone. The piriformis muscle functions to externally rotate the hip as well as to help extend the hip. With the hip already flexed, the piriformis muscle acts as a hip abductor. If the thigh is fixed, the muscle acts to rotate the pelvis to the activated side and backward. The common peroneal nerve can pass through the piriformis muscle in 21% of the population (Figure 42.4); however, in only 5% of these people does the muscle actually divide to allow the nerve to pass (Figures 42.5 and 42.6). The divided muscle actually plays an important role in the development of the piriformis syndrome.

ETIOLOGY

Multiple etiologies have been proposed to explain the compression or irritation of the sciatic nerve as it courses distally. While all have been proposed as independent etiologies, basic anatomical relationships, direct and indirect trauma, inflammation, and local ischemia probably combine to induce the piriformis muscle syndrome.

Yeoman[1] emphasized the anatomical relationship of the sciatic nerve and the piriformis muscle. The development of the syndrome secondary to muscle irritation can be divided into the four groups shown in Table 42.1. Many authors have discussed the relationship of sacroiliac joint disease and the lumbosacral plexus leading to ischialgia;[10,11] however, Yeoman was the first to link sacroiliac disease with piriformis muscle spasm. Freiberg and Vinkle[12] and others have since seconded this opinion.[6,13,14] Levin[15] described the anatomical position of the piriformis muscle during the Lasegue test. At approximately 20 degrees, the muscle is stretched and compression can occur. In some patients with a clinical picture of lumboischialgia of unknown etiology, the test becomes positive at small angles, where only the muscle, not the nerve, is stretched. Static disorders of the lumbosacral spine and hip can produce symptoms.[16] Flexion contracture at the hip may produce lumbar lordosis. Increased tension of the pelvifemoral muscles develops as the muscles try to stabilize the pelvis and spine in the new position. The involved muscles hypertrophy to handle the tension; however, the bony foramens do not enlarge. This leads to a decrease in available space for the neurovascular structures. With neural tissue being the least tolerant to compression of the neurovascular bundle,

FIGURE 42.4 One of the variations in the form of the sciatic nerve. This specimen shows the common peroneal nerve passing through the piriformis muscle.

neurological signs develop earlier than vascular signs. The infrapiriformis foramen narrows with piriformis hypertrophy and may lead to sciatic nerve compression.[2,17,18] If the sciatic nerve passes between the two tendinous heads of the piriformis, compression occurs with muscle stretch during internal hip rotation rather than muscle contraction (Figure 42.6).

Trauma, either direct or indirect, can lead to compression of the piriformis muscle by scarring, spasm, or simply the muscle mass of the gluteus maximus muscle. Robinson[19] and Pace and Nagle[20] postulated that trauma in the sacroiliac or gluteal region can lead to piriformis syndrome. According to Benson and Schutzer,[52] patients who have blunt trauma to the buttock and then have signs and symptoms that are suggestive of lumbar nerve-root compression may have posttraumatic piriformis syndrome. Steiner et al.[14] believed that adhesions between the neural and the muscular fibrous sheaths would lead to compression when the now-combined unit distorts with muscular contraction.

Inflammation can lead to changes in the relationships between all the structures in this area. As shown in Table 42.2, many authors have discussed the possibility of muscular spasm with changes in the synovial bursa. Inflammation of any structure in a narrow area can lead to compression or scarring, which will lead to distortion with motion.

Besides the direct compression of the nerve, compression of the vessels supplying the nerve can lead to the piriformis syndrome.[2] A branch of the inferior gluteal artery (arteria commitans nervi ischiadici) and its corresponding veins join the nerve as it crosses the inferior margin of the piriformis. Constant or repeated muscle spasm can compress the vessels and lead to vascular congestion or ischemia in the sheath. These changes can produce pain along the nerve. Unilateral piriformis syndrome in a patient with previous melanoma was described by Lam et al.,[22] with

FIGURE 42.5 A second variation in the form taken by the common peroneal nerve as it courses between the tendinous portions of the piriformis muscle.

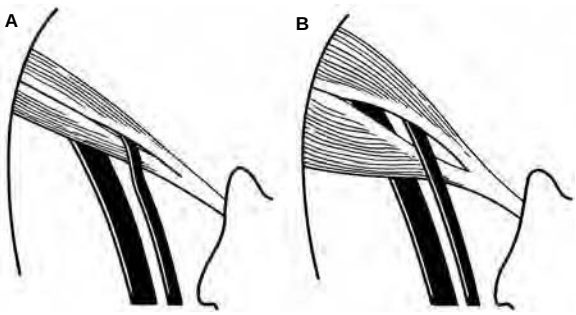

FIGURE 42.6 Dynamic or functional anatomy has been postulated as a cause of the piriformis syndrome. This figure illustrates the change in the piriformis muscle when stretched passively or contracted actively. (A) In passive stretching of the divided piriformis muscle by internal thigh rotation, the tendinous portions compress the nerve; (B) in active contraction of the muscle in external rotation, the space between the tendons enlarges and the nerve is free to pass. These changes must be noted, as these relationships depend on the actual course taken by the nerve.

haematoma of the piriformis muscle by Katati et al.,[49] and with myositis ossificans of the piriformis muscle by Beauchesne and Schutzer.[46] Uchio et al.[47] describe bilateral piriformis syndrome after total hip arthroplasty, while Nagi et al.[55] think that constriction by a tightened piriformis tendon may be a cause of sciatic nerve injury during reconstruction procedures at the hip joint.

TABLE 42.1
Postulated Etiologies for the Development of the Piriformis Muscle Syndrome Due to Irritation of the Nerve

Muscle spasm secondary to irritation of the piriformis at either its origin by sacroiliac disease or at its insertion by bursitis or trochanteric disease
Inflammatory or degenerative changes of the muscle, tendon, or fascia
Degeneration or deformities affecting the bony origin or insertion of the muscle
Anomalies in the course of the nerve through the muscle or between the tendon of the muscle and the bone (the vascular structures may also be affected)

TABLE 42.2
Several Etiologies of the Piriformis Syndrome

Etiology	Author(s)
Piriformis muscle spasm secondary to sacroiliac disease	Yeoman, 1928[1]
Trauma in the sacroiliac or gluteal region	Robinson, 1947;[19] Benson and Schutzer, 1999[52]
Trauma, fibrous adhesions	Solheim et al., 1981[38]
Inflammation of the synovial bursa	Hoff, 1949[43]
Inflammation of the synovial bursa	Toplič anec and Dürrigl, 1966[44]
Passage of the common peroneal nerve through the muscle belly or anomalous relationship	Kopell and Thompson, 1960;[6] Chen, 1994;[21] Ozaki et al., 1999[54]
Vascular compromise due to direct compression	Pécina, 1969[2]
Anatomical relationship of the muscle and sciatic nerve: compression due to discordance between the size of the sciatic foramen and the muscle, position of the nerve under the tendon and above the bone, or between the two heads of the divided piriformis muscle	Pécina, 1975, 1979[17,18]
Hip surgery	Uchio et al., 1998;[47] Nagi et al., 1999[55]

CLINICAL SYMPTOMS AND SIGNS

The piriformis syndrome has many symptoms similar to and overlapping the symptoms of lower back pain, ischialgias, vascular disease, and lower extremity pathologies; therefore, piriformis muscle syndrome remains an under-diagnosed cause of sciatica.[23,45,50,51,53] Pain and paresthesias can present along the entire sciatic nerve. In time, burning sensations, hypesthesia, or anesthesia may develop. Motor weakness or hypotrophy may be included in the presentation. If the sympathetic branches are compressed, trophic changes in the skin will be appreciated. Sciatic nerve dysfunction and reflex sympathetic dystrophy as a consequence of the piriformis syndrome can occur.[24] Frequently, diagnosis requires eliminating other causes of sciatic nerve impingement or irritation based on the history and physical, as described in Table 42.3. A literature review reveals no consensus on the diagnosis or treatment of piriformis syndrome.[50]

Pain in the sacral or gluteal region remains the most constant symptom. The pain increases with sitting or walking and decreases with lying supine. However, not all authors concur. Lam[25] stressed that lumbosacral pressure is not present but that nerve irritability in the gluteal region exists. Robinson[19] described the pain and irritability as originating in the sacroiliac joint and the region of the greater sciatic foramen and the piriformis. Infiltration of the muscle with anesthetic may alleviate the pain and eliminate the symptoms by relieving muscle spasm. Rectal examination

TABLE 42.3
Differentiating Symptoms and Signs of the Piriformis Syndrome

No pain in the lumbosacral region
Peripheral nerve distribution irritation, not radicular in origin
Irritability on palpation of the greater sciatic foramen (region between the greater trochanter and the posterior superior iliac spine)
Palpable mass (or swelling) over the region of the piriformis muscle with exacerbation of pain
Piriformis muscle spasm appreciated by rectal exam
Positive Lasegue sign at 25 degrees
Increased pain with internal rotation of the hip or hip flexion with an extended knee
Decreased pain with external rotation of the hip

with palpation of the piriformis provides an objective evaluation of the muscle's spasm.[26,27] Additionally, a rectal examination allows a quick screening for rectal and lower pelvic pathology. Pelvic and lower back disease may require further studies, including laboratory and radiographic evaluations. Karl et al.[28] describe the scintigraphic appearance of the syndrome in one patient. Jankiewicz et al.[29] describe the appearance of the piriformis muscle syndrome in computed tomography and magnetic resonance imaging, while Fishman and Zybert[30] describe electrophysiologic evidence of piriformis syndrome.

Several physical examination maneuvers elicit symptoms.[17,31,32] The Gowers-Bonnet test seeks to elicit pain with hip flexion, knee flexion, and internal rotation. While not pathognomonic, this test seeks to stretch the piriformis. A modification of this test, as described by Pećina[17] and Komar,[32] has the patient bend forward while standing with knees extended and lower extremity in internal rotation to passively stretch the piriformis and the sciatic nerve.

TREATMENT

Conservative therapy consisting of physiotherapy (including a home program of prolonged piriformis muscle stretching[33]) including rectal massage and rectal diathermy, corticosteroid and anesthetic injections, and anti-inflammatory medication can be tried to alleviate muscle spasm.[19,20,34–37,56] Corticosteroid injections into the synovial bursa under the piriformis as its inserts into the greater trochanter could be repeated to bring relief. Therefore, direct injection of the muscle with local anesthetic and steroid can be considered both diagnostic and therapeutic.[20] Fishman et al.[58] describe injection of the piriformis muscle by fluoroscopic and electromyographic guidance. Caudal epidural steroid injection for treatment of piriformis syndrome was also performed.[56] Hanania and Kitain[48] describe a perisciatic injection of steroid using simple lendmarks and utilizing a nerve stimulator to locate and inject near the sciatic nerve and into the piriformis muscle. Treatment of sacroileitis, if present, will bring relief. Surgical therapy must be initiated within 6 to 8 months of presentation. If the compression is due to muscle position, stretch, or size, sectioning of the piriformis muscle tendon near its insertion will release the nerve.[38–42] Kouvalchouk et al.[57] describe four athletes treated surgically by section of the piriformis muscle and neurolysis of the sciatic nerve. Loss of piriformis action does not cause any noticeable functional disturbance and, in most patients, will lead to disappearance of symptoms. Benson and Schutzer[52] treated 15 cases of piriformis syndrome (in 14 patients) with an operative release of the piriformis tendon and sciatic neurolysis. They obtained 11 excellent and four good results. All of the patients returned to their usual daily activities at an average of 2.3 months postoperatively.

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43 Meralgia Paresthetica

To reach the anterolateral thigh, the lateral femoral cutaneous nerve passes through two fibrous tunnels consisting of the inguinal ligament and the fascia lata. Described initially as Rooth's meralgy[1] and Bernhardt's syndrome,[2,3] meralgia paresthetica occurs with nerve compression in the fibrous tunnels or nerve stretching. Mumenthaler and Schliack[4] used its anatomical location to categorize the syndrome of the inguinal ligament.

ANATOMY

Solely a sensory nerve, the lateral femoral cutaneous nerve originates from the L2 and L3 nerve roots, passes under the psoas muscle, and emerges laterally to cross the ilium buried in a fibrous tunnel formed by a doubling in the fascia of the iliacus muscle. As shown in Figure 43.1, the nerve courses toward the medial aspect of the anterior superior iliac spine and bends through the inguinal ligament at an angle of 70 to 90 degrees. At this point, the nerve divides into a thick anterior and thin posterior branch. Leaving the pelvis in this fashion, the anterior branch pierces the fascia lata to innervate the skin of the lateral thigh (Figure 43.2). The posterior branch runs deep under the tensor fascia lata muscle to innervate the skin of the gluteal region.

Stevens[5] and Ghent[6,7] describe the following four variations of the lateral femoral cutaneous nerve:

1. The nerve passes under the inguinal ligament.
2. The nerve makes a sharp turn when crossing the iliac fascia.
3. The nerve passes through the sartorius muscle.
4. The nerve courses laterally and behind the anterior superior iliac spine.

ETIOLOGY

Vulnerable to compression and stretching from its origin under the psoas muscle until it exits through the inguinal ligament and the fascia lata, the lateral femoral cutaneous nerve can be damaged by multiple etiologies, including tumor, trauma, and surgical complications, as shown in Table 43.1. Compression above the fascial tunnels and in the tunnels can be considered upper and lower forms of the syndrome.[8]

To explain meralgia paresthetica that has no identifiable cause, Kopell and Thompson[9] postulate that the nerve can be damaged by acute or chronic stretching. Since the lateral femoral cutaneous nerve is fixed at both its origin and at the fascial tunnels, leg length changes, scoliosis, increased tension of the abdominal musculature and fascia lata (due to long periods of standing), trunk or leg hyperextension,[35] and surgery may damage the nerve. In cadaver studies, Nathan[10] found that approximately 50% of all lateral femoral cutaneous nerves were thickened where they had pierced the inguinal ligament.

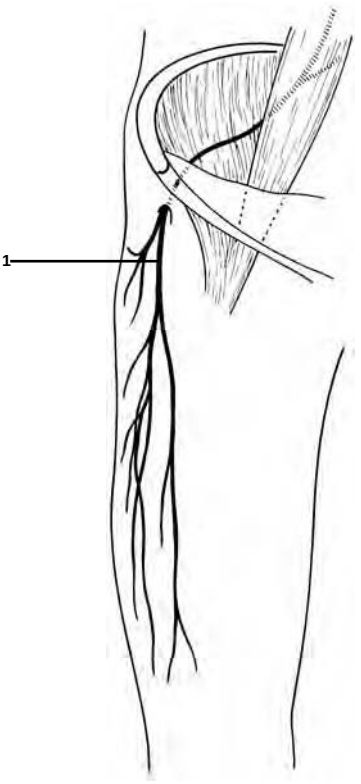

FIGURE 43.1 Easily damaged in surgical procedures around the hip, the lateral femoral cutaneous nerve (1) passes anteriorly to the anterior superior iliac spine before coursing laterally over the thigh.

CLINICAL SYMPTOMS AND SIGNS

Characteristic symptoms consist of paresthesia, burning pain, and dysthesia aggravated by even the touch of clothing, or leg extension while sleeping. Patients even avoid placing keys or objects over the affected thigh.[11] Some investigators encourage the limitation of meralgia paresthetica's definition to the sensory complaints, not the signs.[8] Clinical examination will reveal hypesthesia, trophic skin changes with long-standing compression, hair loss, and a positive inversed Lasegue's sign. Seeking to elicit pain over the thigh, the inversed Lasegue's sign is performed by flexing the knee and extending the hip with the patient in a lateral position (similar to Menel's procedure). Local pressure over the inguinal ligament, especially close to the anterior superior iliac spine in the lower form of this syndrome, may produce pain or local irritation in the nerve's distribution, similar to a Tinel's sign. Using patients' unaffected leg as a control, electromyographic studies effectively show nerve compression on the affected side.[12] The experience of 12 cases highlights the usefulness of antidromic sensory nerve conduction in the diagnostic and prognostic aspects of meralgia paresthetica.[32] Seror[34] compared sensory nerve conduction with somatosensory evoked potentials, and demonstrated that sensory nerve conduction is the more reliable method for electrodiagnosis of meralgia paresthetica. The differential diagnosis should include intra- or extra-spinal radiculopathies of the L2 and L3 nerve roots and lumbar plexus pathology.[13–15,30] Etiologies causing the upper form of the syndrome may be difficult to identify without a computed tomography (CT) or magnetic resonance imaging (MRI) of the abdomen and pelvis. Thermography is also useful in diagnosing meralgia paresthetica.[16]

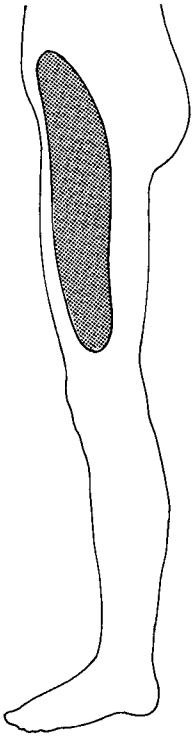

FIGURE 43.2 When the lateral femoral cutaneous nerve gets compressed, the lateral skin of the thigh typically develops paresthesia, burning pain, and disturbances in sensation. Some patients find the area so sensitive they will not keep anything in their pockets on the affected side.

TABLE 43.1
Proposed Etiologies of Meralgia Paresthetica

Etiology	Author(s)
Retroperitoneal hematoma or tumor or metastasis	Flowers, 1968[20] Tharion and Bhattacharji, 1997[31]
Iliopsoas muscle abscess	Komar, 1977[8]
Abdominal aortic aneurysm	Carayon and Gruet, 1969[21]
Abdominal/inguinal surgery	Moscona and Hirshowitz, 1980;[22] Yamont et al., 1994[23]
Direct injury, stretching, or scar formation	Rhodes, 1957;[17] Peterson, 1952[25]
Iliac graft harvest	Mandic, 1982;[17] Massey, 1980[26] Nahabedian and Dellon, 1995[29]
Seat belt trauma	Beresford, 1971;[27] Mandic, 1982[17]
Anomalies of passage, i.e., through the sartorius muscle	Ghent, 1961[7]
Tight girdle/clothing	Bora and Ostermann, 1982[11]
Obesity	Mandic, 1982[17]
Weight loss	Baldini et al., 1982[28] Limb length discrepancy Goel, 1999[33]

TREATMENT

Treatment must address the underlying compression. Compression in the upper form requires decompression. The lower form, affecting the fascial tunnels, requires relief of tension and compression in the fascia lata and inguinal ligament. Local application of anesthetics and corticosteroids may bring immediate relief in the lower form of the syndrome. At the same time, this injection serves in the differential diagnosis. Conservative therapies for the lower form of compression include histamine therapy, ionophoresis, and use of anesthetic ointment on the affected skin. Mandic[17] recommended local anesthetics, weight loss in obese patients, optimal diabetic control, removal of compressive garments, and physical or electric therapies (i.e., transcutaneous electrical nerve stimulation, or TENS). A multimodality regimen is recommended by Dureja et al.[18] If conservative therapy fails, neurolysis or nerve resection is indicated,[29] as patients prefer hypesthesia to burning pain. Edelson and Stevens[19] described 21 cases of meralgia paresthetica in children treated surgically.

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44 Saphenous Nerve Syndrome

The saphenous nerve passes through the adductor canal and penetrates the vasto-adductor membrane to run superficially (Figure 44.1). Compression or stretching of the nerve in this area produces pain along its dermatomes.

ANATOMY

The anatomical relations of this canal have been studied by Jo and Solter.[1] The longest sensory branch of the femoral nerve, the saphenous nerve, leaves the femoral triangle to enter the adductor canal together with the femoral artery and vein. The vastus medialis muscle and adductor longus muscle, the walls of the canal, are connected by a vasto-adductor membrane, the roof of the canal. The sartorius muscle also covers the proximal portion of the canal and later covers the two terminal branches of the saphenous nerve: the infrapatellar and the descending branches.

The infrapatellar branch bends at 90 degrees to the longitudinal axis of the femur at the level of the knee's joint line. Sending a branch between the superficial and deep layers of the medial collateral ligament, the infrapatellar branch supplies the medial portion of the joint and the overlying skin. This small branch can be injured in surgery or meniscal dislocation. The descending branch accompanies the saphenous vein to supply the skin of the medial leg and foot.

ETIOLOGY

Extensively studied by many investigators[2–5] trauma along the adductor canal or vasto-adductor membrane underlies most of the described etiologies. Mumenthaler and Schliack[6] and Jones[7] described saphenous nerve damage in association with femoral arteriography and vascular surgery, respectively. Inflammatory disorders such as saphenous vein thrombophlebitis may reduce the space within the canal and lead to compression. Entrapment of the saphenous nerve by branches of the femoral vessels and pes anserine bursitis have been described;[8,9] however, trauma predominates. Surgery around the knee — meniscectomy, arthrotomy,[10] ligament reconstruction, arthroscopy, knee arthroplasty and saphenous vein surgery (for varicosities) — has increased the incidence of nerve compressions or injury in recent times.[11,12] Direct trauma to the canal in sports (i.e., soccer or rugby) can damage the nerve.

Functional anatomy has been used to explain nerve compression. Kopell and Thompson[13] described changes in knee position (especially genu varum), deformation with torsion, or direct trauma following trauma that led to stretching and mechanical irritation of the nerve in the region of the vasto-adductor membrane. Dumitru and Windsor[14] have described subsartorial entrapment in a bodybuilder. Widmer and Gerster[26] described the medial meniscal cyst imitating a tumor, with compression of the saphenous nerve.

CLINICAL SYMPTOMS AND SIGNS

Patients present with constant medial leg pain when walking and climbing, leading to possible confusion of this syndrome with vascular disorders.[15] Pressure over the adductor canal or where

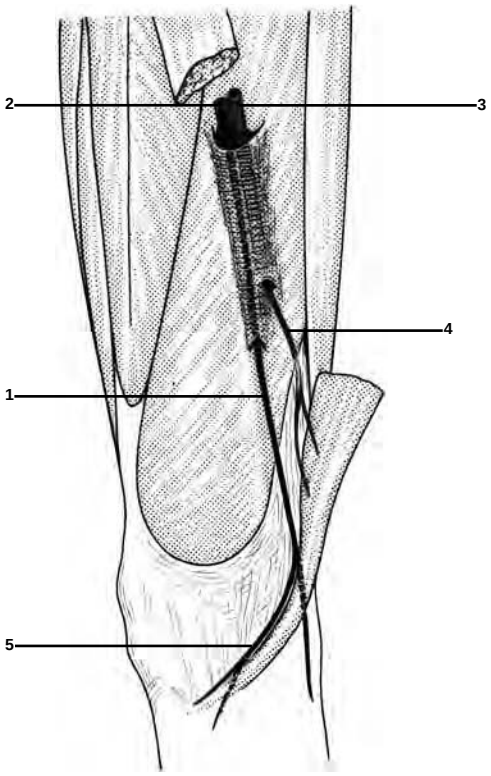

FIGURE 44.1 Compression of the saphenous nerve typically occurs in the region depicted here — 1: saphenous nerve; 2: femoral vein; 3: femoral artery; 4: descending genicular artery; 5: infrapatellar branch of the saphenous nerve.

the nerve crosses the medial femoral condyle produces strong pain radiating down to the medial malleolus. Compression of the ramus infrapatellaris is the cause of medial knee pain;[16,17,18] Wartenberg[19] named this syndrome "gonyalgia paraesthetica." Physical examination typically reveals hyperalgesia and hyperesthesia in the infrapatellar region and hypalgesia and hypesthesia along the medial leg and foot. Resisted adduction produces pain in the canal.[13] Pain with knee hyperextension leads patients to walk without fully extending their knees. This altered gait loads the foot abnormally and may lead to the development of metatarsalgia.[13] The value of somatosensory-evoked potentials in saphenous entrapment neuropathy is discussed by Trainier et al.[20]

Pain relief following a local novocaine injection into the adductor canal, despite provocation, may isolate this syndrome.[21] Occasionally, injury to the infrapatellar branch occurs simultaneously with medial meniscal damage, producing Turner's symptom of hypesthesia. As Mandi and Kalmar[22] found, 4.2% of all arthrotomies for suspected meniscal tears were found to have saphenous nerve compression instead of a meniscal tear.[5] Therefore, in cases where a torn meniscus is questionable, one can consider testing with local anesthetic injections into the canal for diagnostic confirmation.

TREATMENT

Conservative therapy is often sufficient.[27] These measures include the following: rest, physiotherapy, anti-inflammatory medication, and local corticosteroid injection. Surgical treatment consists of sectioning of the vasto-adductor membrane from the site of compression proximally. If the tendon of the sartorius muscle compresses the infrapatellar branch, the tendon may require partial release.

While surgical exploration and neurolysis may be necessary initially, recurrent symptoms might require neuroma or saphenous nerve resection.[23–25]

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45 Popliteal Entrapment Syndrome

The tendinous arch of the soleus muscle and the popliteus muscle delineate the popliteal fossa, through which pass the popliteal artery, vein, and tibial nerve (Figure 45.1). In contrast to the rest of the syndromes in this book, the tibial nerve does not get compressed in the popliteal fossa. Limited data exist in the medical literature concerning neurovascular compression of the popliteal artery in this region, which has been described as either popliteal artery entrapment syndrome[1] or popliteal entrapment syndrome. In 1959, Hamming[2] focused attention on it by describing an intraoperative finding in a patient with intermittent claudication. The most frequent causes of arterial compression are anatomical variations of the popliteal artery or the muscles crossing the fossa. Described originally in 1879 by Stuart,[3] these variations convert the popliteal fossa into a tunnel.

ANATOMY

Continuing the femoral artery's course after leaving the opening in the adductor magnus muscle tendon, the popliteal artery angles toward the lateral side of the fossa, reaches the middle of the fossa midway through the fossa, and then courses distally between the medial and lateral heads of the gastrocnemius muscle to dive under the tendinous arch of the soleus muscle. The popliteal artery divides into the anterior and posterior tibial arteries after crossing the tendinous arch. The popliteal artery lies deep to the popliteal vein and deep and medial to the tibial nerve in the fossa. The artery lies in near the popliteal surface of the fossa, the knee joint capsule, and the posterior surface of the popliteal muscle. The popliteal fossa is bounded as follows: laterally by the biceps femoris; proximally by the plantaris; distally by the lateral head of the gastrocnemius; and medially by the semitendinosus, semimembranosus, and medial head of the gastrocnemius; "the roof is covered by the fascia lata."[4] While these are the classic relationships, variations do arise.

Stuart[3] and Hamming[2] described arteries that, instead of passing between the heads of the gastrocnemius, passed between the medial head of the gastrocnemius and the medial femoral condyle. The proximal origin of the muscle head is due to embryonal development. Chambardel-Dubreuil[5] described an artery coursing through the medial head (Figure 45.2). These variations place the artery at risk for intermittent compression with muscle activity. The popliteal vein may follow the artery's anomalous course.[6]

ETIOLOGY

Many investigators describe intermittent claudication associated with a popliteal artery that is compressed between the gastrocnemius and medial femoral condyle or by the gastrocnemius tendinous origin itself.[7,8] Based on these varied relationships, Insua et al.[9] classified the anomalies into four categories that have since been modified:

1. In Type I, the artery passes along the internal margin of the normal medial head of the gastrocnemius before turning over the margin to reach the fossa from under the head of the muscle.

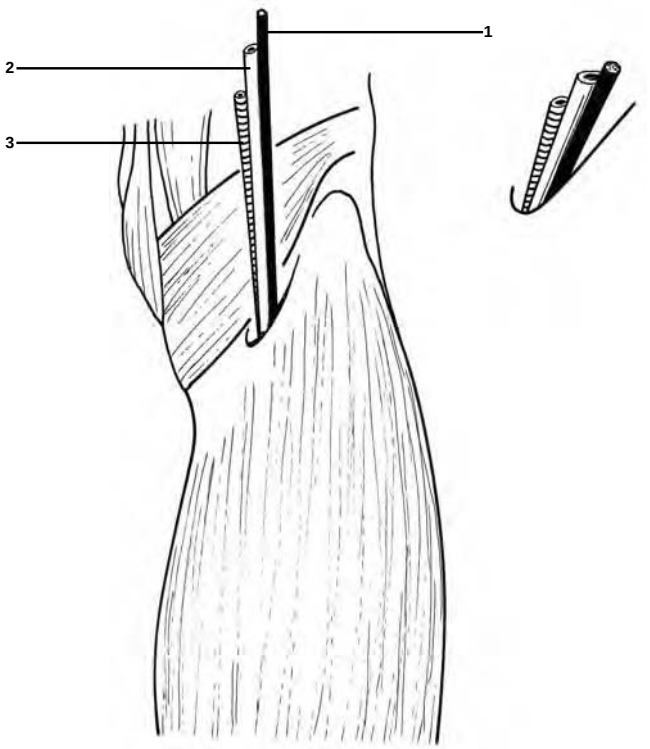

FIGURE 45.1 Typical relationship of the popliteal artery (3), the popliteal vein (2), and the tibial nerve (1) as they course over the popliteus muscle and under the medial head of the gastrocnemius muscle.

2. In Type Ia, the medial head of the gastrocnemius originates more proximally on the femur, thus covering the minimally displaced popliteal artery.
3. In Type II, the gastrocnemius has an accessory head or aberrant tendinous bundle, lateral to the medial head, which covers a nondisplaced popliteal artery.
4. In Type IIa, a medially placed plantaris unites with the medial head of the gastrocnemius over normal popliteal artery.

Modifications have been made by multiple investigators describing more than 160 cases on entrapment (Table 45.1).[10–14] Becquemin et al.[14] emphasize the possibility of compression due to anomalies in the semimembranosus, semitendinosus, adductor magnus, or tendinous arch of the soleus. However, the tendinous arch is mentioned only as a possibility. Compression by the arch would compress the popliteal artery and vein and the tibial nerve, providing a neurologic presentation of the popliteal entrapment syndrome. Turnipseed and Pozniak[15] describe popliteal entrapment as a result of neurovascular compression by the soleus and plantaris muscle. Popliteal artery entrapment syndrome is very often described in young athletic patients.[16–19,32]

A new syndrome: the pseudo-entrapment of the popliteal artery and tibial nerve caused by kinking of the neurovascular bundle is described by Psathakis.[20] The kinking in pseudo-entrapment is caused by one or more transverse fibrous bands, resulting in calf pain and distress. Chernoff et al.[21] demonstrated by magnetic resonance imaging that functional impairment of popliteal arterial flow during plantar flexion occurred in subjects who had no symptoms of popliteal entrapment syndrome. Popliteal vascular compression in a normal population can be induced in 53% of subjects with simple leg positioning caused by myofascial compression.[22] This must be considered when evaluating patients for interventions on the basis of physiologic testing of the popliteal vessels.

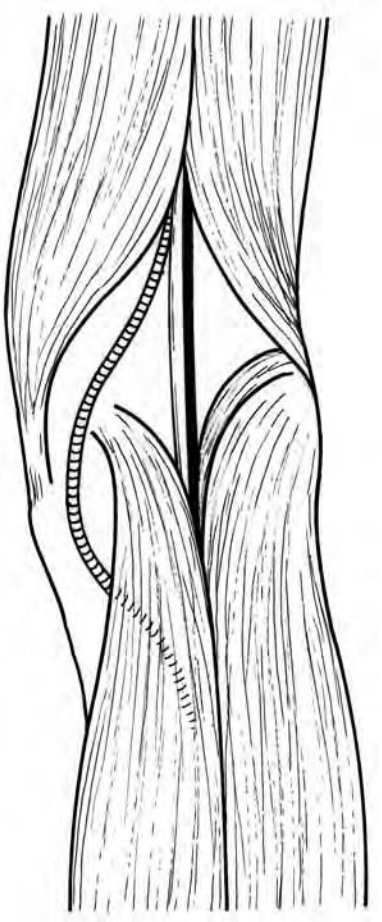

FIGURE 45.2 Anomaly in the popliteal artery as it courses around the medial head of the gastrocnemius muscle.

TABLE 45.1
Modified Classification of Popliteal Artery Entrapment

Type	Muscle	Artery	Associated Vessels
I	Normal	Anomalous course	None
Ia	Proximal origin	Minimally displaced	None
II	Aberrant origin or accessory head	Normal	None
IIa	Medially placed plantaris joins the medial head	Normal	None

CLINICAL SYMPTOMS AND SIGNS

Popliteal entrapment syndrome should be suspected in young, active patients who present with intermittent claudication. Their clinical picture depends on the presence of thromboses, aneurysms, or only dynamic compression with activities.

Ischemic symptoms may involve only the foot or the entire leg. Since nerves are the most sensitive tissue, ischemia leads to numbness, tingling, and feelings of cold, pain, and paresthesia. The leg muscles can cramp intermittently. Claudication remains the most constant symptom. The

other symptoms might develop only after intensive exercises or just after walking but not running. Weakening or disappearance of the dorsalis pedis pulse with maximal dorsiflexion or plantar flexion with an extended knee implicates popliteal artery compression. Relief of symptoms and reappearance of the arterial pulse with rest strengthen the argument for compression. Thromboses or aneurysms of the popliteal artery may lead to acute ischemic attacks or emboli, which are more severe than the temporary ischemic changes of cramps and sensory dysfunction.[6,13,14,23–26]

Invasive and noninvasive vascular exams aid in diagnosis. Doppler ultrasound allows dynamic arterial testing during active muscular contraction in plantar flexion and dorsiflexion. Color Doppler ultrasonographic imaging in the diagnosis of popliteal artery entrapment syndrome is also a useful exam[27] (see Color Figure 45.1*). Set et al.[28] describe a new technique of leg muscle scintigraphy using 99mTc methaxy isobutyl isonitrile with single photon emission tomography in detection of popliteal artery entrapment. Impedance plethysmography evaluates ankle, calf, and thigh pressures and pulses and can be used in conjunction with treadmill tests. Appearance of symptoms during testing confirms the suspicion. The combination of all data from these tests differentiates popliteal entrapment syndrome from peripheral vascular disease, neurogenic claudication, psychoneurosis, and chronic compartment syndrome. Allen et al.[17] describe a female long-distance runner with popliteal entrapment syndrome misdiagnosed and surgically treated as compartment syndrome. Bell[26] advocated the use of compartment pressure measurements to clearly define patients with presumed compartment syndrome, McDonald et al.[24] recommended angiography to detail medial dislocation (i.e., behind the medial femoral condyle) and selective biplane angiography (anteroposterior, lateral) with changes in foot position from dorsiflexion to neutral and then to plantar flexion. Maximal dorsiflexion may require assistance. Arterial narrowing or obliteration can occur with popliteal entrapment syndrome. Stress magnetic resonance imaging,[29] magnetic resonance tomography, and magnetic resonance angiography[30] are useful techniques for the diagnosis of popliteal entrapment syndrome, and conventional angiography will be unnecessary in the future.

TREATMENT

Surgical decompression might require maneuvers that do not affect muscle function, e.g., decompression of the artery by dividing the compressive structures and resection of the damaged arterial segment. The segment can be replaced by a saphenous vein graft or repaired by performing an endarterectomy. If the artery is normal, removal of the compressive agent is sufficient. Steurer et al.[31] described an alternative therapeutic approach to popliteal artery entrapment syndrome with vascular complications. Combined catheter treatment, consisting of percutaneous transluminal thrombembolectomy, local thrombolysis, and percutaneous transluminal dilatation, was performed for thrombotic and embolic obstruction of popliteal or crural vessels. The aberrant tendomuscular structures were surgically corrected at a later date.

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* Color Figure 45.1 follows page 174.

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46 Peroneal Tunnel Syndrome

The common peroneal nerve runs in an exposed fibro-osseus tunnel at the level of the fibular neck. Compression produes a characteristic clinical picture that, while it bears many names in the medical literature (restless legs, crossed-legs palsy, isolated peroneal nerve palsy at the head of the fibula), will be discussed here as the peroneal tunnel syndrome.

ANATOMY

The common peroneal nerve usually branches from the sciatic nerve in the proximal portion of the popliteal fossa. Variations occur where the nerve leaves proximally or distally.[1] The nerve runs along the biceps femoris muscle and tendon to the popliteal fossa (Figure 46.1). It then encircles the head of the fibula, passes below the tendinous origin of the peroneus longus, and enters the peroneal tunnel near the fibular neck (Figure 46.2). Proximal to the fibular head, the lateral sural cutaneous nerve branches from the common peroneal nerve.

As the nerve enters the tunnel, the common peroneal nerve divides into the deep, superficial, and recurrent peroneal nerves. Additional branches to the peroneus longus and anterior tibialis muscles can be found at the tunnel's entrance. Bogdanović et al.[2] described branch points at the level of the fibular neck in 67% of the cases, at the level of the fibular head in 22% of the cases, and more proximal in the popliteal fossa in 11% of the cases.

In the peroneal tunnel, the peroneal nerve and its branches stretch over the periosteum of the fibular neck and are covered by the tendinous origin of the peroneus longus muscle. This tendinous origin can be found in the shape of a hook or J on the left leg or reversed J on the right leg.[3] Plantar flexion or foot inversion (adduction, supination, and plantar flexion) tenses the peroneus longus muscle and brings it closer to the fibula. This motion decreases the tunnel's space and compresses the nerve against the fibular neck. The deep and superficial branches are stretched and bend over the lower hook of the tendinous arch.

The superficial peroneal nerve (superficial peroneus) runs between the fibula and the peroneus longus muscle. As it courses distally, it lies on the anterior intermuscular septum between either the peroneus longus muscle proximally and the extensor digitorum longus muscle, or the peroneus longus muscle and the peroneus brevis muscle. The superficial peroneal nerve supplies both the peroneus brevis and longus muscles. At the junction of the middle and distal third of the tibia, the superficial peroneal nerve pierces the crural fascia and splits into two cutaneous branches. Compression by the crural fascia produces the syndrome of the superficial peroneal nerve. The terminal branches — the medial dorsal cutaneous pedal and the intermediate dorsal cutaneous pedal branches — supply the anterolateral skin of the leg and the dorsal skin of the great, second, third, and medial fourth toes. The dorsum of the lateral fourth and fifth toes are innervated by the sural nerve via the lateral dorsal cutaneous pedal nerve.

Dividing from the common peroneal nerve, the deep peroneal nerve (peroneus profundus) pierces the anterior intermuscular septum and travels with the anterior tibial vessel between the tibialis anterior muscle and either the extensor digitorum longus muscle proximally or the extensor hallucis longus muscle distally. In this fashion, these muscles receive their innervation from the deep peroneal nerve. The nerve enters the foot under the cruciform ligament (anterior tarsal tunnel

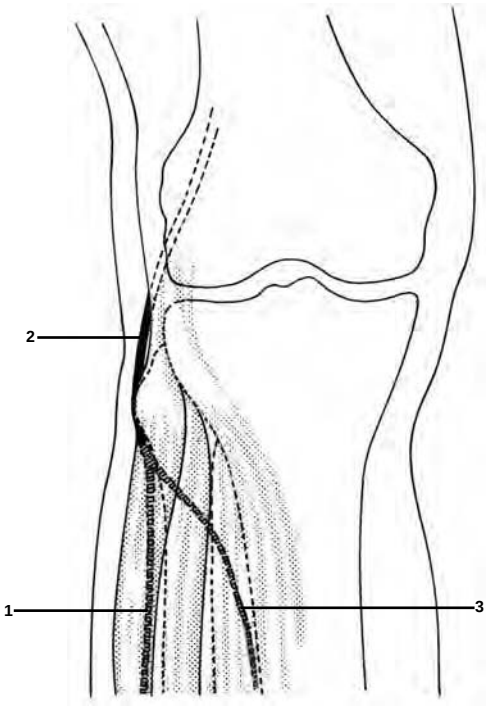

FIGURE 46.1 Coursing around the fibular head, the common peroneal nerve (2) divides into a superficial (1) and a deep (profundus) (3) branch. The deep branch dives deep to run along the intercompartmental fascial plane.

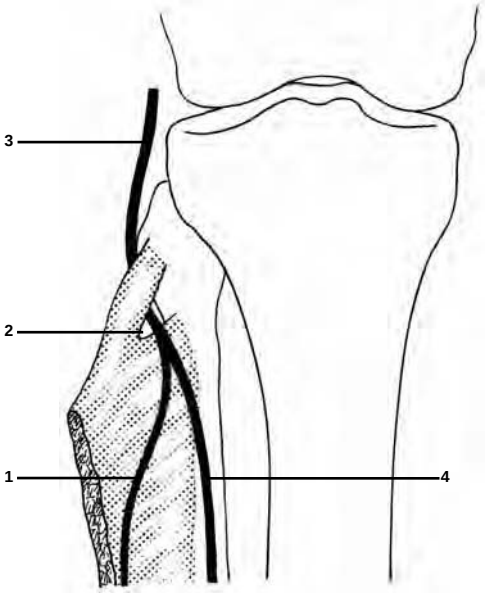

FIGURE 46.2 Occasionally, the common peroneal nerve (3) or one of its branches — the superficial (1) or the deep branch (4) — can be compressed by the tendinous arch of the peroneus longus muscle (2).

syndrome), provides dorsum of the articular branches, and ends in the first metatarsal space with sensory branches to the skin between the great and the second toes.

TABLE 46.1
Etiologies and Investigators for Peroneal Tunnel Syndrome

Etiology	Author(s)
	External
Plaster casts (short leg, PTBs), traction splint	Mumenthaler, 1973[6] Mihalko et al., 1999[42]
Crossed-legs or squatting (prolonged)	Woltmann, 1929;[7] Nagler and Rangel, 1947;[8] Marwah, 1967[9]; Babayev et al., 1998[37]
Sleeping positions	Bora and Osterman, 1982[10]
Unknown	
Idiopathic	Osborne, 1957;[11] Fettweis, 1968[12] LeGeyt and Ambrose, 199;[39]
Repetitive trauma (functional anatomy)	Kopell and Thompson, 1960;[3] Moller and Kladin, 1987[13] (runners); Leach et al., 1989[14] (runners); Mitra et al., 1995;[34] (athletes) Loredo et al., 1998;[40] Ihunwo and Dimitrov, 199;[45]
	Internal
Bony changes	
Exostosis	Theodorou et al., 1978;[15] Watson and Torch, 1993;[19] Gallagher-Oxner et al., 1994[20]
Osteophyte	Sheman et al., 1983[16]
Fibular fractures	Casselt and Dürrschmidt, 1969;[17] Komar, 1977[18]
Knee arthroplasty	Rose et al., 1982[4]
Ganglions	Brooks, 1952;[21] Parkes, 1961;[22] Clark, 1961;[23] Muckart, 1976;[24] Bora and Osterman, 1982;[10] Firooznia et al., 1983;[25] Evans et al., 1994[26] Kabukcuoglu et al., 1997;[35] Ramelli et al., 1999[41]
Synovial cysts	Kopell and Thompson, 1963;[3] Edwards et al., 1995[27] Gayet et al., 1998[38]
Other	
Hemophilia	Large et al., 1983[28]
Enlarged fabella	Mangieri, 1973;[29] Takebe and Hirohata, 1981[30]
Aneurysm	Kars et al., 1992[31]
Muscle herniation	Alhadeff and Lee, 1995[32]
Fibrous histiocytoma	Linssen et al., 2000[46]

ETIOLOGY

Compression in the area of the peroneal tunnel frequently comes from an external source, as shown in Table 46.1. These external causes can be as simple as a tightly placed short leg cast with the knee in hyperextension or prolonged knee bracing.[43] The close relationship of the common peroneal nerve to the fibula places it at risk with fibular fractures or correction of valgus knees during knee arthroplasty.[4] Synovial cysts or ganglions can displace the nerve from its course and place it into a prolonged stretch.[5,27,38] The cysts can actually enter into the tunnel and directly compress the nerve (Figure 46.3) While idiopathic forms still exist, many previously idiopathic cases can be explained by functional anatomical changes. Repetitive actions requiring inversion or pronation (e.g., runners and machine operators using pedals) stretch the common

peroneal nerve against the fibula and the lower margin of the tendinous arch. The concurrent weakness in dorsiflexion and eversion as well as sensory complaints may be confused with chronic subtalar synovitis or chronic talar subluxations.

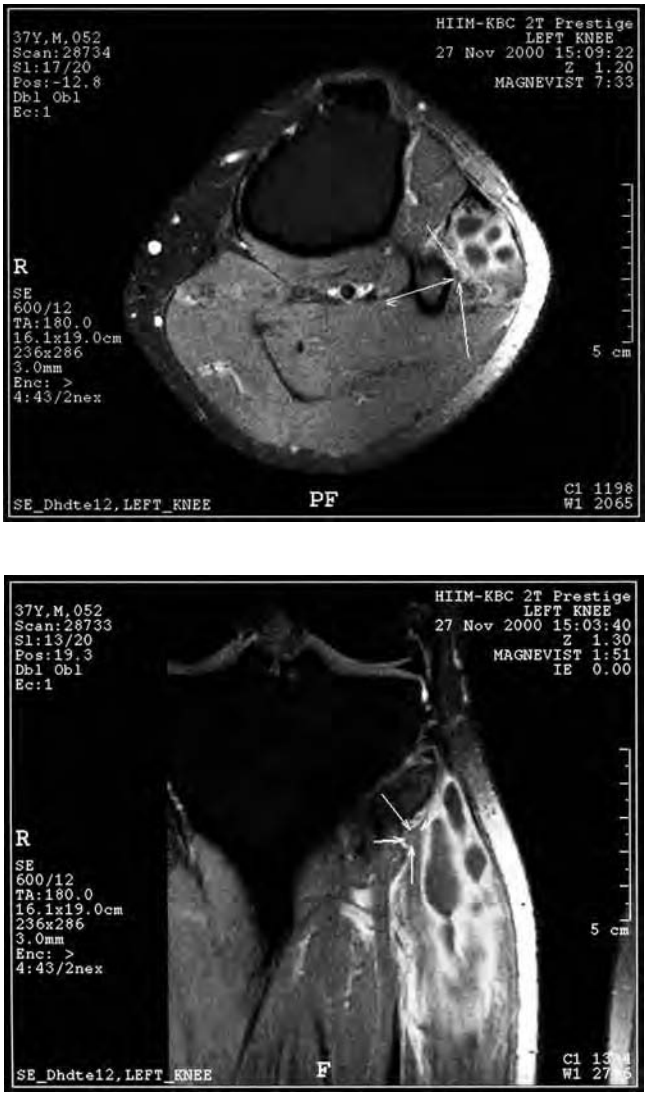

FIGURE 46.3 "Coronal and axial MRI show the compression of the peronal nerve (arrows) by the ganglion.

CLINICAL SYMPTOMS AND SIGNS

Pain appears initially in the compressed region before spreading distally into the common peroneal nerve's dermatome, which includes the dermatomes of the deep and the superficial peroneal nerves. Radiation of pain into the thigh may occur. Additional palpation or pressure over the tunnel will increase the patient's pain. This pain will not be felt in lumbar stenosis, root entrapment, or more proximal compression. Presenting gradually after the appearance of lateral leg and dorsal foot pain, motor weakness and atrophy can lead to a full-blown, dropped-foot presentation. Patients will have weakness on dorsiflexion and inversion. Forced inversion will actually increase their pain. Trophic

changes in the bones of the foot may occur with neural dysfunction. Electromyographic and nerve conduction velocity studies will aid in differentiating peroneal tunnel syndrome from sciatica, lumbar stenosis, syndrome of the piriformis muscle, polyneuropathies, and anterior tibial syndrome.[33,44] Close examination of the involved dermatomes helps greatly. If compression occurs proximal to the thigh, other nerve areas including the tibial nerve may be involved. In the piriformis muscle syndrome, pain is localized to the gluteal region without reproduction of pain with peroneal tunnel palpation. Conduction velocity studies will verify normal conduction across the peroneal tunnel if nerve compression is occurring proximally. Patients with anterior tibial syndrome typically will present with a swollen red leg, strong pain, and a missing dorsalis pedis arterial pulse. The pain in polyneuropathies is sharp, burning, and independent of motion, with electromyographic studies implicating the spinal cord as the source. The sensory changes are circular and the leg is hypotonic without reflexes but with trophic skin changes.

TREATMENT

Identification of a specific peroneal tunnel syndrome etiology allows appropriate treatment (see Color Figure 46.1). External compressive causes must be relieved. Repetitive motions that irritate the nerve must be avoided. Local corticosteroid injections may bring relief. Physical therapy allows strengthening of atrophied muscles following either conservative or surgical relief of nerve compression. Failure of conservative therapy to relieve the symptoms necessitates surgical decompression of the tunnel. Fabre et al.[36] recommend operative decompression when symptoms persist or recovery remains incomplete for 3 to 4 months, provided that the diagnosis has been confirmed with electrophysiological studies.

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47 Superficial Peroneal Nerve Syndrome

Compression or distortion of the superficial peroneal nerve can occur where the nerve abandons the muscular layer of the leg and pierces the crural fascia at the level between the middle and distal third of the leg. The syndrome of the superficial peroneal nerve was first described in 1945 by Henry[1] under the term "mononeuralgia in the superficial peroneal nerve" and is also known as "superficial peroneal nerve entrapment."

ANATOMY

At the entrance to the peroneal tunnel near the head of the fibula, the common peroneal nerve divides into two terminal branches, the deep and superficial peroneal nerves. This terminal branch point can vary, as can the course of the superficial peroneal nerve. The superficial branch continues distally between the fibula and the peroneus longus muscle lying on the intermuscular septum of the anterior compartment. The nerve also lies between the peroneus longus and the extensor digitorum longus muscles proximally and the peroneus longus and brevis muscles distally. At the level between the middle and distal thirds of the leg, the nerve pierces the crural fascia and continues subcutaneously as the cutaneous dorsalis medialis and the cutaneous dorsalis intermedius nerves (Figure 47.1). Prior to piercing the fascia, the superficial peroneal nerve supplies the peroneus longus and brevis muscles. The cutaneous branches of the superficial nerves supply the skin of the anterolateral side of the leg, the dorsum of the foot, the dorsum of the first, second, and third toes, and the medial side of the fourth toe. The sural nerve via the cutaneous dorsalis lateralis nerve supplies the lateral sides of the fourth and fifth toes.

ETIOLOGY

Trauma represents the most frequently proposed etiology of the rarely diagnosed syndrome of the superficial peroneal nerve.[2–4] Surgical trauma, lipomas, [5] muscular hernias,[6,7] tight boots,[8] repetitive compression at the foot in sports,[9] everyday life,[10] and dynamic compression in the narrow fascial tunne[11,2,5] have all been postulated as etiologies. Styf and Korner[11] and Styf[12] have described the development of the syndrome after fasciotomy for chronic anterior compartment syndrome. Trauma in this area could lead to local inflammation, reactive swelling, and, eventually, compression in the fascial tunnel. Rubin et al.[13] described entrapment of an accessory superficial peroneal sensory nerve.

Dynamic compression based on the functional anatomy of the leg places the nerve at risk.[14,15] The superficial peroneal nerve is fixed; therefore, forced inversion and extension of the foot further stretches the nerve over the fascial border.[1,2,5] Surgical data revealed marked narrowing in 12 of 19 subjects, poststenotic edema in five of 19 subjects, and muscular hernia in the tunnel in six of 19 subjects.[12] While typically noted as 1 cm in length, surgical evidence has shown that the tunnel might actually run 3 to 11 cm in length (10 of 19 subjects).[12] Repetitive activities may cause scarring of the nerve or fascial borders, further narrowing the tunnel.

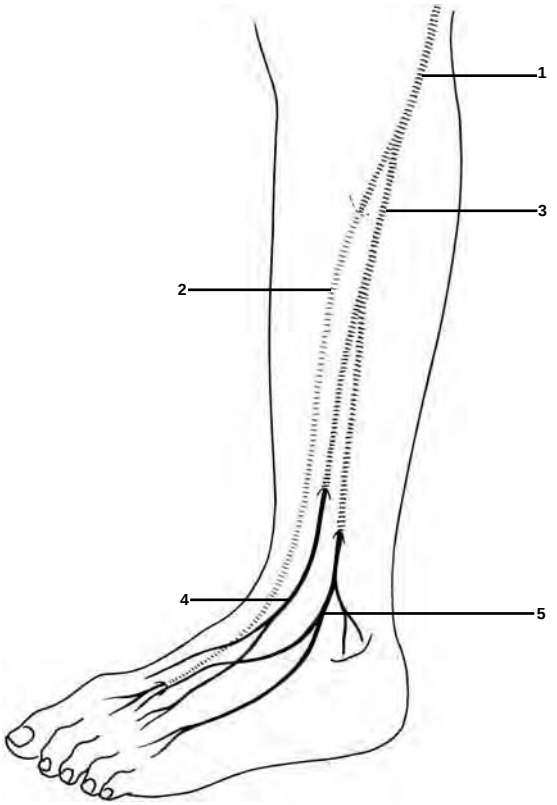

FIGURE 47.1 The relationship of the peroneal nerves as they reach the foot — 1: common peroneal nerve; 2: deep peroneal nerve; 3: superficial peroneal nerve; 4: medial dorsal cutaneous nerve; 5: intermediate dorsal cutaneous nerve.

CLINICAL SYMPTOMS AND SIGNS

Described as mononeuralgia in 1945 by Henry,[1] pain caused by compression or damage to the superficial peroneal nerve appears on the dorsum of the foot accompanied sometimes by dysesthesias or complete anesthesia in the nerve's dermatome. Styf[12] suggested three tests for evaluating patients for the syndrome:

1. Resisted dorsiflexion and eversion with pressure applied over the tunnel
2. Passive plantar flexion and inversion
3. Stretching of the nerve as in the second test with percussion over the tunnel

A positive result for these provocative tests is the production of pain or parasthesias over the dermatome of the nerve. Electromyographic studies of the peroneal and anterior tibial muscles and conduction velocity examination help identify the syndrome.[16] Radiographic examination will help to eliminate other pathological states in the area such as stress fractures or tumors. Magnetic resonance imaging confirmed the diagnosis of the fascial entrapment of the superficial peroneal nerve in one 16-year-old female athlete.[18] Since lumbosacral spine pathology can easily involve various nerve roots, one should not be misled and jump to conclusions when presented with lower-extremity neurological symptoms. Bannerjee and Koons[5] described a case of superficial peroneal compression treated only after L4/L5 discectomy failed to resolve an individual's pain. An invasive

but diagnostic test using local injection of anesthetic over the tunnel will temporarily relieve pain due to compression in the tunnel.

TREATMENT

Since trauma ranks as a common etiology, removal of trauma should be included in the treatment protocol. Many of the movements that stretch the nerve, however, are among normal daily activities. Therefore, conservative therapy might not succeed. Passive changes in the foot's position can be beneficial. Physical therapy and local corticosteroid injections at the fascial tunnel might also be effective. If these fail to relieve the symptoms, fasciotomy of the nerve's tunnel should free the nerve and give lasting results.[17–20]

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48 Sural Nerve Syndrome

The sural nerve is vulnerable to compression or tension injuries as it courses posteriorly along the lower leg and behind the lateral malleolus, resulting in pain and burning in its dermatome. Compression or distortion occurs especially where the nerve pierces the crural fascia at the level between the middle and distal third of the calf (Figure 48.1).

ANATOMY

Many variations in the composition of the sural nerve exist. According to Gremigni,[6] the standard description of the sural nerve occurs in 79% of cases. Based on cadaver evidence, Williams[17] concludes that, "no true sural nerve occurred in 16.34% cases." Classically, the nerve originates from two branches: the medial cutaneous sural nerve from the tibial nerve and the ramus communicans of the lateral sural cutaneous nerve from the common peroneal nerve. Recent descriptions by Lawrence and Botte[9] found that the nerve lay close to the Achilles tendon 7 cm proximal to the malleolus and within 1.4 cm of the malleolus as it coursed posteriorly and inferiorly.

The sural nerve begins with its main component, the medial sural cutaneous nerve. This nerve branches from the tibial nerve in the popliteal fossa and runs distally between the two heads of the gastrocnemius muscles beneath the crural fascia. Coert and Dellon[4] noted that the lateral sural nerve and lateral cutaneous nerve of the calf pierce the deep fascia near the fibular head. Between the middle and distal thirds of the calf, the sural nerve pierces the fascia and runs distally near the small saphenous vein close to the Achilles tendon. It anastomoses at different levels with the communicating branch of the common peroneal nerve. The sural nerve arches behind the lateral malleolus to supply the ankle joint, the posterior calf, and the lateral side of the heel and foot out to the fifth toe, as the lateral dorsi pedis cutaneous nerve. As a cutaneous nerve, it lies superficial to the peroneus longus and brevis tendons.[9] It rarely supplies the fourth toe and the lateral border of the third toe. Additionally, it may anastomose with the intermedius dorsi pedis cutaneous nerve, a branch of the superficial peroneal nerve. Lawrence and Botte[9] found an anastomotic branch entering the sinus tarsi in 24% of specimens.

ETIOLOGY

While this is a rare syndrome,[16] one should be aware of the problem and its multiple etiologies, both intrinsic and extrinsic (Table 48.1). One of the first cases described was reported by Pringle et al. in 1974.[12] The crural fascia may act as either a compression point or a fixation point for the sural nerve. Coert and Dellon concur.[4] Heuser[8] noted nerve compression at the communicating branch from the common peroneal nerve. If the nerve is fixed by the fascia, extreme inversion of the foot may lead to a traction injury. Occasionally, the cause of symptoms cannot be identified.

CLINICAL SYMPTOMS AND SIGNS

Patients may present with pain and burning in the sural nerve's dermatome. Use of the Hoffman's or Tinel's signs can help localize the site of compression, though these signs are less specific.

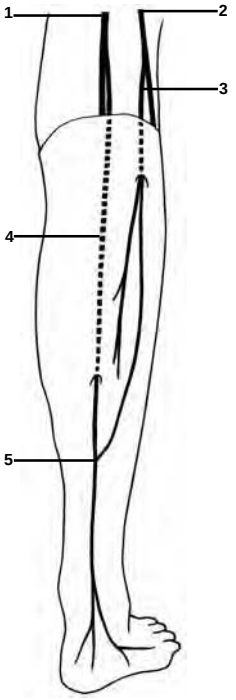

FIGURE 48.1 The compression or distortion of the sural nerve may occur especially where the nerve pierces the crural fascia — 1: tibial nerve; 2: peroneal nerve; 3: nervus cutaneous surae lateralis; 4: nervus cutaneous surae medialis; 5: sural nerve.

TABLE 48.1
Etiologies of Sural Nerve Compression

Intrinsic	Extrinsic
Baker's cyst[11]	Boots/tight lacing[8]
Lipoma	Prolonged compression (table edge,[3] casts)
Scar[2]	Occupational neuropathy[3]
Crural fascia[4]	External pressure[13]
Ganglion (joint, tendon sheath)[12]	Direct blow[14]
Thrombophlebitis	
Trauma (recurrent ankle sprains)[1]	

Unfortunately, neurological signs cannot always be found. Electromyography is not always helpful, as only a few patients will show a decrease in nerve conduction velocity.[5,7,15] In differential diagnosis, the other entrapment syndromes of the sciatic nerve must be taken in consideration.[10]

TREATMENT

Identification of the cause of nerve compression is essential in treatment. After removal of the offending agent, spontaneous regeneration or nerve recovery with the disappearance of symptoms usually occurs. If a protracted course of conservative treatment fails to relieve symptoms, surgical treatment may be considered. Removal of ganglions, Baker's cysts, or scar tissue can help nerve recovery if identified as the intrinsic cause. If the nerve is bound in scar, it may need to be transposed

onto a healthier bed. One might also consider a fasciotomy, when manipulation of the ankle and foot seem to tension the nerve where it pierces the fascia.

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49 Anterior Tarsal Tunnel Syndrome

When passing under the inferior extensor retinaculum (ligamentum cruciforme) on the dorsum of the foot, the deep peroneal nerve (peroneus profundus) can be compressed, resulting in pain through the dermatome, which receives branches originating distal to the retinaculum. Marinacci described the clinical symptoms of the anterior tarsal tunnel syndrome in detail.[1]

ANATOMY

The anterior tarsal tunnel lies under a thickening of the dorsal pedis fascia and above the talus bone. These fascial thickenings form a retinaculum that fixes and redirects the extensor tendons. While at times consisting of four branches in a cruciform shape, the inferior extensor retinaculum typically has three branches that form a Y transversely across the foot's dorsum. The base of the Y, the lateral portion, originates in the sinus tarsi on the lateral side of the calcaneus. As it passes over the tendons for the extensor digitorum longus, the retinaculum divides into two rami: the superior ramus, which inserts on the medial malleolus, and the inferior ramus, which inserts on the dorsal surface of the navicular and first cuneiform bones. Occasionally, a superfluous second lateral branch may exist, which inserts on the lateral malleolus, producing a retinaculum with a cruciform shape, the ligamentum cruciforme. Medially and below the superior and inferior rami, the tendons and the tendon sheaths of the anterior tibialis and extensor hallucis longus muscles run accompanied by the dorsal pedis artery and vein. The deep peroneal nerve joins these structures in the anterior tarsal tunnel after innervating all the foot extensors, except for the extensor digitorum brevis muscle, which is the only muscle affected by anterior tarsal tunnel compression. Within the tunnel, the nerve divides into a lateral and a medial branch (Figure 49.1).

The lateral branch passes under the tendon of the extensor digitorum brevis muscle to innervate the tarsal and metatarsal joints via articular branches. Passing under the tendon of the extensor hallucis brevis muscle, the medial branch continues distally to terminate in the space between the great toe and the second toe, supplying sensory innervation via the dorsalis hallucis lateralis nerve and the second medial digital nerve.

ETIOLOGY

Anatomical relationships play a major role in the development of anterior tarsal tunnel syndrome. The tunnel consists of a tight retinaculum overlying mobile soft-tissue structures that lie near the bony floor. This tight band fixes the structures; therefore, bony, joint, vascular, neural, or muscular disorders will alter the volume of the tunnel. The soft-tissue structures in the tunnel act distal to the tunnel; sudden movement or repetitive actions far from the tunnel can stretch and damage the nerve.[2] Distortion of the talonavicular joint or underlying bony surface by osteophytes, synovial pseudocysts, ganglions, anomalous muscle or anomalous communication and fractures compress the tunnel.[1–5,14–16] Repetitive compressive trauma caused by shoe straps, and extensive stretching from the prolonged plantar flexion of high-heeled shoes have been postulated as etiologies.[1,6,7] Inflammation of the tendons or development of tendon sheath or retinaculum ganglions can also

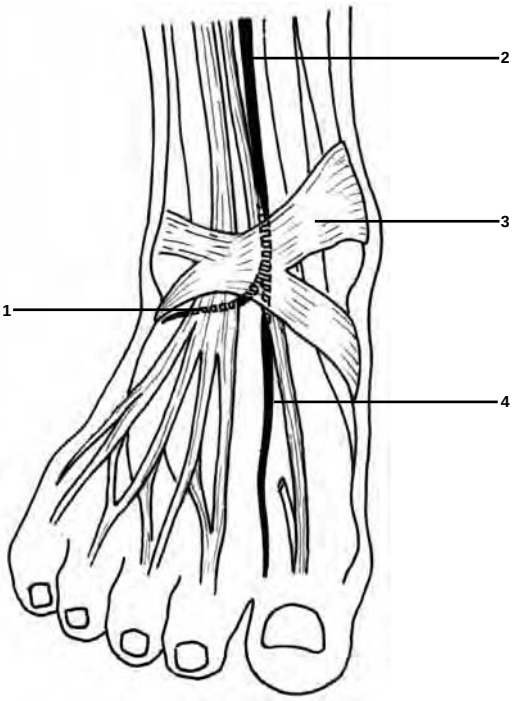

FIGURE 49.1 The deep peroneal nerve may be compressed as it enters the dorsum of the foot under the extensor retinaculum — 1: lateral branch of the deep peroneal nerve; 2: deep peroneal nerve; 3: inferior extensor retinaculum (cruciform ligament); 4: medial branch of the deep peroneal nerve.

narrow the tunnel. Finally, primary neurovascular diseases such as neuromas or aneurysms could decrease the tunnel's volume. Lin et al.[8] reviewed 10 patients treated for anterior tarsal tunnel syndrome. The causes of onset of the syndrome included contusion of the dorsum of the foot, tight shoelaces, talonavicular osteophytosis, ganglion, and pes cavus.

CLINICAL SYMPTOMS AND SIGNS

The clinical picture varies, depending on whether the sensory or motor fibers of the deep peroneal nerve are affected. Characteristic of sensory compromise, burning pain will be localized to the dorsal space between the great and second toes. Nocturnal exacerbations have been reported and may awaken patients from sleep.[9] A positive Tinel's sign may be elicited over the deep peroneal nerve under the inferior extensor retinaculum. In addition, ankle plantar flexion, toe dorsiflexion, or direct pressure over the nerve could precipitate the symptoms.[10] Blunt, undefined pain felt deep in the foot coexists with disturbed extensor digitorum brevis muscle function when the motor fibers are compressed. Trophic changes of the metatarsal bones (i.e., osteoporosis) may occur with motor fiber compromise. To assess extensor digitorum brevis muscle function, the foot is maximally dorsiflexed, eliminating the action of the long extensor. Then the patient is asked to further extend the great toe. Maximal plantar flexion may reproduce foot pain but fails to be pathognomonic for anterior tarsal tunnel syndrome. Electromyographical (EMG) studies might help differentiate this syndrome from proximal peroneal nerve compression or L5 nerve root lesions.[9,17] If present, an accessory peroneal nerve, which does not pass through the tunnel, can mask EMG findings in the extensor digitorum brevis.[12,13]

TREATMENT

Initially, conservative therapy should be used to treat anterior tarsal tunnel syndrome. Rest, immobilization of the foot at 90 degrees to avoid compression over the tunnel, physical therapy, anti-inflammatory medication, or local corticosteroid injections may relieve the symptoms. In extreme cases, surgical decompression or even sectioning of the nerve at the entrance of the tunnel has been suggested to avoid neuroma formation and tendon fixation. Operative decompression in nine feet of eight patients gave successful results at follow-ups of 1.5 to 4 years.[8]

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50 Tarsal Tunnel Syndrome

Accompanied by their corresponding arteries and veins, the tibial nerve's two terminal branches, the medial and the lateral plantar nerves, pass around the medial malleolus through a fibro-osseus tunnel, the tarsal tunnel (Figure 50.1). Compression of the nerve produces a clinical picture that was simultaneously described by Keck[1] and Lam.[2,3] Multiple names exist for this tunnel: canal calcaneén de Richet, canal tibio-astragalo-calcaneén, canalis malleolis, and canalis plantaris. Anatomical studies suggest that most of the nerve compression occurs in the medial tarsal tunnel.[4,5] To clarify its analogy to the carpal tunnel syndrome, this chapter will discuss compression of the tibial nerve in the tarsal tunnel as tarsal tunnel syndrome.[5,6]

ANATOMY

The fibro-osseus tarsal tunnel has bony walls consisting of a bony sulcus on the medial side of the calcaneus, the posterior talar process, and the medial malleolus (Figure 50.2). Pećina et al.[5] found on 103 specimens that, at the lower margin of the calcaneal sulcus for the flexor hallucis longus tendon, is a long ridge (variable in development) to which are attached the deep layer of fibers for the abductor hallucis arch. The sulcus of the nerve, in the calcaneus, can vary from shallow (8 to 10 mm, 23%) or medium (11 to 13 mm, 64%) to deep (14 to 16 mm, 13%).[5] Nerves running in a shallow sulcus are possibly more susceptible to compression. The medial wall of the tunnel is formed by the ligamentum lacinatum and tendinous arch of the abductor hallucis muscle. Tendons overlie this fibro-osseus vault. The ligamentum lacinatum has two layers, a deep and a superficial layer. The superficial layer is a thickening of the crural fascia between the medial malleolus and passes over the sustentaculum tali and posterior talar process to insert in the crural fascia. The deep layer divides the tunnel into two lacunae: a tendinous lacuna for the tendons of the posterior tibial, the flexor digitorum longus and the flexor hallucis longus muscles, and a neurovascular lacuna.[7]

Lying below the ligamentum lacinatum, the tendinous arch of the abductor hallucis muscle also consists of a superficial and a deep fibrous layer. The superficial layer originates from the calcaneal tuberosity, crosses over the calcaneal sulcus, and reaches the medial malleolus. The deep layer follows the same course to the middle of the tunnel before branching to reach the bony ridge of the calcaneus.[5] When an individual is standing, the deep layer divides the neurovascular structures into an upper and lower section (Figure 50.3), which, respectively, supply the medial and the lateral plantar areas via their plantar sulcus. Just as the posterior tibial artery in the neurovascular lacuna divides into a lateral and a medial plantar artery, both of which are accompanied by their corresponding veins, the tibial nerve divides into a medial and a lateral plantar nerve, which accompany their vascular supply separated only by a thin membrane. The tarsal tunnel can be considered the hilum of the plantar surface, since virtually all of the neurovascular supply enters through it. These divisions within the tarsal tunnel allowed Heimkes et al.[8] to describe a proximal and a distal syndrome.

Before entering the tarsal tunnel, the tibial nerve sends a calcaneus branch to supply the heel's skin. This nerve may be compressed at the edge of the lacinate ligament and lead to the heel pain of calcaneus nerve entrapment.[9] However, not all heel pain can be considered calcaneal nerve

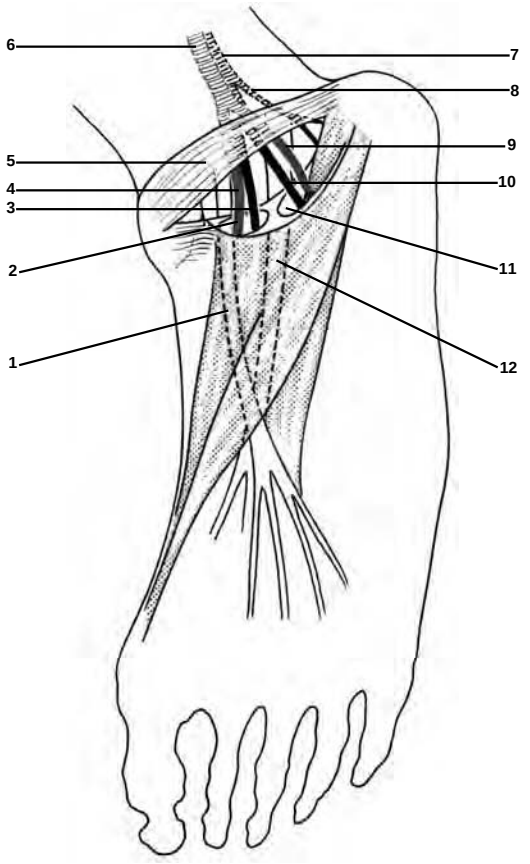

FIGURE 50.1 The complex anatomy of the tarsal tunnel — 1: flexor digitorum muscle; 2: upper (medial) tarsal tunnel; 3: medial plantar nerve; 4: medial plantar artery; 5: ligamentum lacinatum; 6: posterior tibial artery; 7: tibial nerve; 8: calcaneal branches of the tibial nerve; 9: lateral plantar artery; 10: lateral plantar nerve; 11: lower (lateral) tarsal tunnel; 12: flexor hallucis longus muscle.

entrapment. Compression of the lateral plantar nerve as it passes below the heel to supply the abductor digiti quinti muscle also produces heel pain.

Investigations by Pećina et al.[5] reveal that the upper section of the tarsal tunnel is narrower than the lower section. Therefore, the medial plantar neurovascular structures are not only closer to the tendinous lacuna but also are in a narrower tunnel than the lateral plantar vessels. These anatomical facts place the medial plantar vessels in the medial tarsal tunnel at higher risk for compression. The plantar neurovascular distribution can be seen in Figure 50.4.

ETIOLOGY

While having nerve compression or irritation in common, the etiologies for tarsal tunnel syndrome are quite diverse, as shown in Table 50.1. Mechanical pressure from changes in the tissue relationships within the tunnel remains the common denominator of the proposed etiologies. Therefore, trauma and congenital or acquired anomalies predispose affected individuals to a higher risk of nerve compression, since their tarsal tunnels are abnormal in configuration. Rather than changing the bony components, autoimmune and inflammatory diseases affect the tunnel's soft tissues and decrease the volume of the tunnel.[10–13] Since the neural components of the tunnel remain most sensitive to increased pressure, changes in sensory and motor function are among the first symptoms

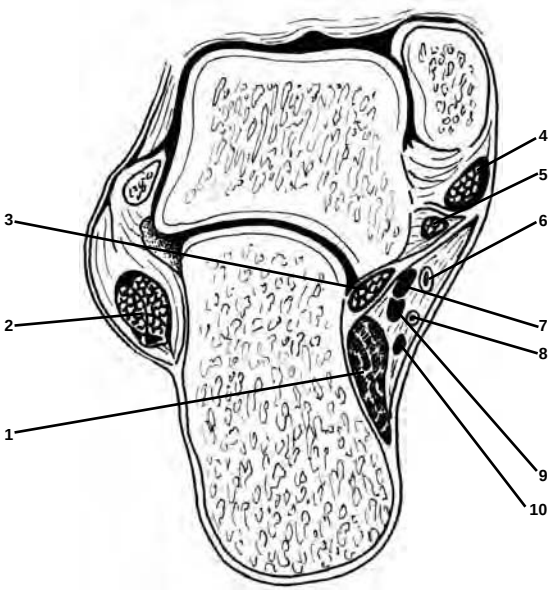

FIGURE 50.2 Cross-section of the tarsal tunnel — 1: abductor hallucis muscle; 2: peroneus longus muscle; 3: flexor hallucis longus muscle; 4: posterior tibial muscle; 5: flexor digitorum longus muscle; 6: medial plantar artery; 7: medial plantar nerve; 8: lateral plantar artery; 9: lateral plantar nerve; 10: calcaneal branches of the tibial nerve.

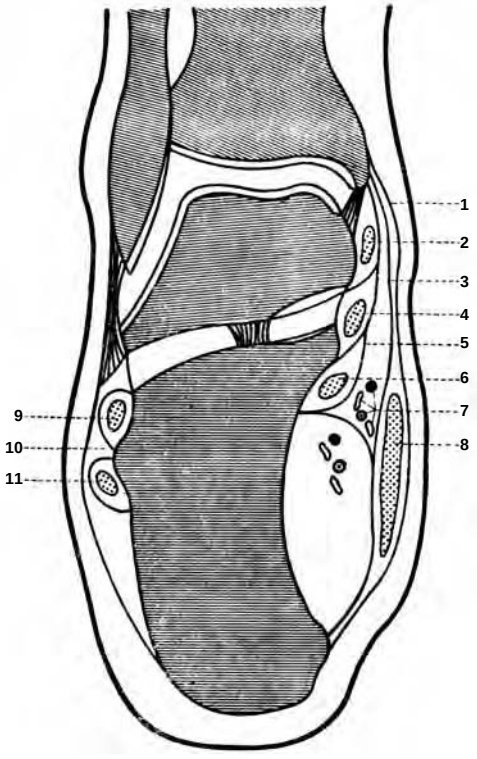

FIGURE 50.3 Cross-section of the tunnel with attention to the relationships of the upper tarsal tunnel — 1: ligamentum lacinatum; 2: posterior tibial muscle; 3: tendinous arch of the abductor hallucis muscle; 4: flexor digitorum longus muscle; 5: deep layer of the ligamentum lacinatum; 6: flexor hallucis longus muscle; 7: the neurovascular bundle in the upper tarsal tunnel; 8: abductor hallucis muscle; 9: peroneus brevis muscle; 10: canal of the peroneal muscles; 11: peroneus longus muscle.

FIGURE 50.4 This schematic delineates the dermatomes of the nerves passing through the tarsal tunnel: the calcaneal branches (black), the medial plantar nerve (hatched), and the lateral plantar nerve (dotted).

of tunnel damage.[14,15] The upper section of the tunnel containing the medial plantar neurovascular structures remains more sensitive to volume changes than the lower section containing the lateral plantar neurovascular structures. The tarsal tunnel syndrome in athletes[16–19] and occupational tarsal tunnel syndrome[20] are being described more frequently. Tarsal tunnel syndrome secondary to cosmetic silicone injection is described by Frey et al.[21] and recurrent tarsal tunnel syndrome by Zahari and Ly.[22]

The tibial nerve, like the median nerve, has a rich vascularity but is sensitive to ischemia. Compression of the vasa vasorum surrounding the nerve will lead to ischemia and neurological symptoms.[23–25] Increased vascular compromise during standing and walking account for the crises experienced by patients with tarsal tunnel syndrome. In several idiopathic cases that have been relieved after surgery, the nerves were found to be normal in appearance. These cases have been proposed to be vascular in nature; however, idiopathic cases remain as such until their causes become clarified.

CLINICAL SYMPTOMS AND SIGNS

The clinical picture of tarsal tunnel syndrome is characterized by pain and paresthesia, especially in the dermatome of the medial plantar nerve, the medial plantar surface, and the great, second, and third toes (Figure 50.4). Predominantly affecting the toes, the pain is accompanied by burning, numbness, pressure, or feelings of pins and needles. These symptoms become crises when they worsen at night or after long periods of standing or walking. Proximal irradiation of pain can lead to an erroneous diagnosis of sciatica, lumbosacral spine disease, or proximal neurological compromise. Patients may even present with bilateral tarsal tunnel syndrome.[26,27]

Objective signs can be difficult to detect. While anesthesia or hyperesthesia is rare, hypesthesia and loss of two-point discrimination are early signs of nerve compression. Tinel's sign, one of the more constant objective findings, usually can be provoked in tarsal tunnel syndrome. Sometimes, sweating might be reduced. Inspection of the ankle could reveal a retromalleolar or submalleolar swelling. Forced eversion (pronation) and dorsi-flexion of the foot can reproduce pain and paresthesia in the distribution of the nerve.[28] Abduction of the toes may also cause pain. While occasionally felt throughout the plantar surface, the symptoms are usually confined to the dermatome of the medial plantar nerve. This indicates compression of the upper section of the tunnel. However,

TABLE 50.1
Proposed Etiologies of Tarsal Tunnel Syndrome

Category	Action	Specific Etiology
Trauma	Change in anatomical structures	Exostoses (Carayon and Courbil, 1965[52]); medial malleolar fracture; talus fracture — posterior process; dislocation of the ankle; joint changes (Kenzora et al., 1982[53]); posttraumatic arthritis (Komar and Banky, 1966;[58] Mosimann and Mumenthaler, 1969[59]; I.V. infusions and scarring in the saphenous vein (Serre et al., 1965[12]); shallow sulcus (Pećina et al., 1968[5]); anomalies of the ankle (vertical talus) valgus deformity/varus heels/pronated splayed forefeet (Radin, 1983;[60] O'Sullivan et al., 1992;[54] Bruns and Hermann, 1992;[55] Wallenbock and Plecko, 1993;[56] Stefko et al., 1994[57])
Congenital/acquired	Initial tunnel configuration	Malformation of posterior process talus (Mosimann and Mumenthaler, 1969[59]); hypertrophy of abductor hallucis muscle (Edwards et al., 1969;[50] Kim et al., 1997[93]); anomalous muscle (Ho et al., 1993;[61] Sammarco and Conti, 1994[62]); accessory soleus muscle (Pla et al., 1996[89]); flexor digitorum accessorius longus muscle (Canter and Siesel, 1997[94])
Autoimmune	Connective tissue disease, spatial relationship	Rheumatoid arthritis (McGuigan et al., 1983[63]); amyloid; sarcoidosis; dermatomyositis; gout; scleroderma; systemic disease (Oloff et al., 1983[13])
Inflammatory tenosynovitis	Space in the tunnel	Ankylosing spondylitis (Enright et al., 1979;[64] Kucukdeveci et al., 1995[65]); tendonitis (Kenzora et al., 1982;[53] Wilman and Patel, 1995[66])
Metabolic/hormonal	Tissue effects	Diabetes; pregnancy; myxoedema; acromegaly osteoporosis (Byrd, 1981[67]); hyperlipidemia (Ruderman et al., 1983[68])
Tumors	Space-occupying lesions	Ganglion (Matricali, 1980;[69] Brown, 1982;[70]Takakura et al., 1998;[97] Nagaoka and Satou, 1999[104]); lipoma; neurilemoma (Dowling and Skaggs, 1982;[71]Cancilleri et al., 1999[106]); cysts; neurofibroma (Marinacci, 1957, 1968;[79,80] Myerson and Soffer, 1989;[72] Aydin et al., 1991;[73] Tedder et al., 1992;[74] Chen, 1992;[75] Belding, 1993;[76] Cancilleri et al.,1999[106])
Vascular	Space in tunnel	Varicose veins (Gould and Alvarez, 1983;[81] Pećina, 1987[82]); venous plexus (Keck, 1962[1]); peripheral occlusive disease (Greiter and Wilde, 1970[83]) stasis secondary to occupations requiring long periods of standing; ischemic changes in nerve (Moazami-Gondarzi and Khodadadyan, 1992;[77] Boyer et al., 1995[78])
Idiopathic		Lam, 1962;[2] Keck, 1962;[1] McGill, 1964;[84] Kopell and Thompson, 1960;[85] Marinacci, 1957;[79] Bora and Osterman, 1982;[86] Takakura et al., 1991;[87]Kohno et al., 2000[107]

to avoid confusion between complete and partial tarsal tunnel syndrome, both presentations will be considered tarsal tunnel syndrome.

Muscular dysfunction can be difficult to detect by a superficial examination. Since the long flexors of the foot and toes are preserved, patients have no difficulty standing or walking. While the effects of carpal tunnel syndrome on one's hands are well noticed, the effects of tibial nerve compression may go unremarked, as the foot typically is not used for precise movements.

The clinical picture does not include primary vascular signs, since the vascular structures are more resistant to compression; however, several vascular disorders have been postulated to lead to neural compromise. Diagnosis of tarsal tunnel syndrome requires a full evaluation, including radiographic, sonographic,[105] computerized tomography, magnetic resonance imaging (MRI), [105] telethermographic, and electromyographic studies. Axial radiographs of the calcaneus reveal the

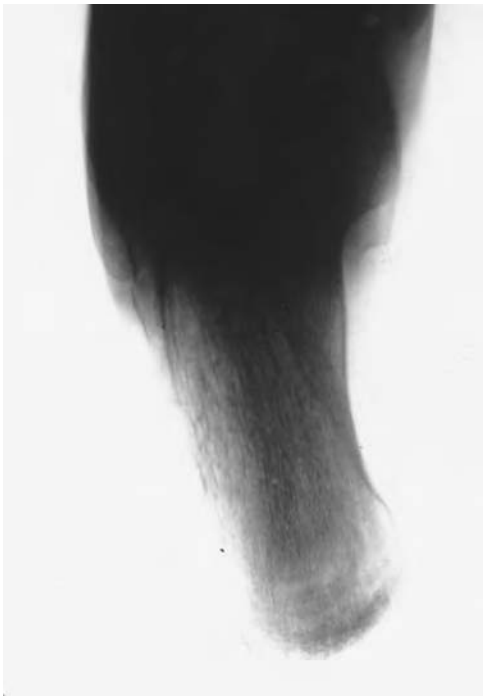

FIGURE 50.5 The components of the tarsal tunnel run along the calcaneus and are bound to the bone by ligaments and tendons accompanying the nerves into the foot. Bony abnormalities can deform the tunnel and lead to nerve compression.

bony structure of the tunnel (Figure 50.5), but today MRI is the inevitable method for tarsal tunnel diagnosis.[29–33] Early in confusing cases, electrodiagnostic studies may be of considerable value in evaluation, progression of compression, and nerve recovery.[34–37,91,99,102] Kaplan and Kernahan[38] emphasize the importance of decreased amplitude and increased duration of evoked potentials rather than distal motor latency. Oh et al.[39] use these studies to rule out S1 root compression from the differential diagnosis. Negative electromyographic and normal conduction velocity studies do not exclude the possibility of tarsal tunnel syndrome.[28,40,42] A vascular evaluation in nondiabetic patients will clarify the vascular supply to the lower extremity. Screening for tarsal tunnel syndrome can be done by utilizing the measurement of the two-point static-touch thresholds for pressure and distance.[88] The differential diagnosis includes arch problems, Morton's metatarsalgia, lumbosacral spine disorders (i.e., bony disorder, spurs), calcaneodynia, and plantar fasciitis.

TREATMENT

Conservative therapy removes the causes of compression and treats the primary disease (Figure 50.6). Rest, avoidance of repetitive trauma, immobilization (plaster casts), use of orthotics, physical therapy, anti-inflammatory medication, and local corticosteroid injection can be tried in several combinations[43] to yield success rates up to 79% (19/24) as shown by Androić.[44]

Surgical therapy should be initiated if the symptoms have persisted for 6 months, conservative therapy has failed to bring relief, or muscle atrophy exists. While not without risk, corticosteroid injections can be repeated up to three times within 2 months before considering surgery.

Operative treatment can relieve the compression by releasing the ligamentous bands, removing the offending agents (i.e., inflamed synovium, ganglions, exostoses, osteophytes), and occasionally performing a neurolysis.[45–49,100,103] Sectioning of the superficial layer of the lacinate ligament is not

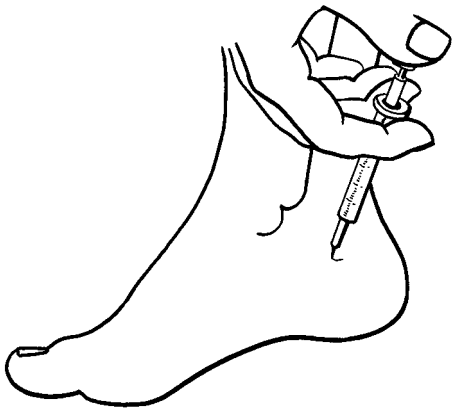

FIGURE 50.6 Conservative therapy for tarsal tunnel may include injection of corticosteroid into the tunnel.

sufficient.[98] The deep layer must be sectioned but preserved, since it is important not only as an origin for the abductor hallucis muscle, but also as a foot stabilizer. Following surgical release, patients should be immobilized up to 2 weeks before starting motion. Operative results can be as excellent as 88% (14/16), as described by Edwards et al.[50] According to Turan et al.,[96] surgical decompression was beneficial in most patients with longstanding (median, 60 months) tarsal tunnel syndrome. Evaluation of surgical results using multivariate analysis[95] showed that the outcome is influenced, in order of importance, by fibrosis around the nerve, the preoperative severity of the condition, a history of sprained ankle, Worker's Compensation, a long history and heavy work. The results were favorable when there was a short history, the presence of a ganglion, no sprains, and light work. According to Mahan et al.,[90] surgical success rates vary in the literature from 44% to 100%.In the study of Herbsthofer et al.,[100] 21 of 32 patients who had a decompression operation were reexamined at an average follow-up time of 12 years. Using the criteria described by Kaplan, 10 postoperative results were rated as very good, six as good and five as poor.

Novotny et al.[92] described two patients with recurrent tarsal tunnel syndrome who were treated with re-release of the retinaculum, followed by nerve coverage with a radial forearm free flap. Autogenous saphenous vein graft wrapping of the tibial nerve has been described as an effective treatment option for failed tarsal tunnel decompression.[101]

An endoscopic approach to the tarsal tunnel syndrome might be an option.[51]

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51 Medial Plantar Nerve Syndrome (Jogger's Foot)

Medial plantar nerve compression may occur in the region of the navicular tuberosity when the nerve passes through an osseo-fibro-muscular tunnel. This tunnel lies between the abductor hallucis muscle and the navicular tarsal bone. The patient, usually a middle-aged jogger, complains of aching or shooting pain in the medial aspect of the arch during running.

ANATOMY

The tibial nerve enters the tarsal tunnel and divides into a medial and a plantar nerve. They accompany their vascular supply separated only by a thin membrane (see Figures 50.1 to 50.3). The innervation of the plantar medial nerve on the planta pedis can be compared with the innervation of the median nerve in the hand (see Figure 50.4). Having passed the medial (upper) canal of lacuna vasonervorum of the tarsal tunnel, the nerve lies behind the vessels situated above the abductor hallucis muscle. The nerve crosses below the talus and the navicular bone. Then, the nerve crosses the lateral margin of the abductor hallucis muscle lying between the fibro-muscular part of the muscle and the navicular tuberosity (i.e., the nerve is lying within the osteo-fibro-muscular tunnel, where it can often be compressed). The medial plantar nerve follows the medial margin of the flexor digitorum brevis muscle under the flexor hallucis longus tendon and the flexor digitorum longus muscle. At the level of the metatarsal bones, the nerve branches into the digital plantar communis nerves (Figure 51.1). Before its ramification, the nerve provides cutaneous branches to the medial part of the planta pedis and muscular branches to the abductor hallucis muscle, the flexor digitorum brevis muscle, the medial head of the flexor pollicis brevis muscle, and to the two medial lumbrical muscles. The digital plantar communis nerves branch at the base of the toes into the digital plantar proprii nerves, which supply the plantar side of the first, second, third and medial side of the fourth toe.

ETIOLOGY

Medial plantar nerve entrapment has been described.[2] Its connection to long-distance running was not established until 1978, when Rask[5] described jogger's foot. Running with the ankle in valgus for long distances may damage the medial plantar nerve. The valgus position increases the tension on the nerve as it is fixed behind the navicular tuberosity on the medial aspect of the jogger's foot.[5] Eversion of the foot stretches the medial plantar nerve between the fibromuscular tunnel formed by the abductor hallucis muscle and its border with the navicular tuberosity. Repeated trauma to the nerve causes inflammation of the nerve at the entrapment point. Arch supports must be avoided in the jogger's shoe to prevent further trauma to the medial plantar nerve along the longitudinal arch. Because the medial plantar nerve runs in proximity to the flexor digitorum longus and flexor hallucis longus muscles, it is sometimes difficult to distinguish tendon problems from neuritis.[6]

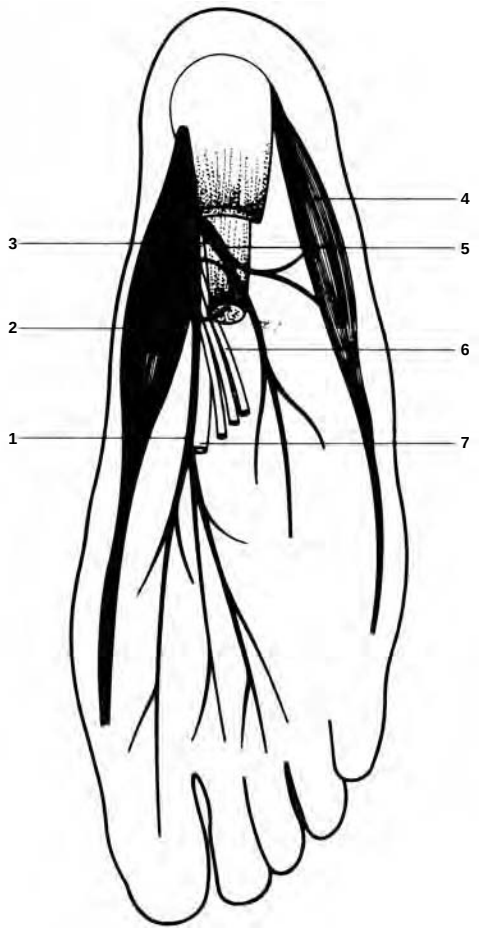

FIGURE 51.1 Medial plantar nerve compression may occur when the nerve passes through an osseo-fibro-muscular tunnel between the abductor hallucis muscle and the navicular tarsal bone — 1: medial plantar nerve; 2: abductor hallucis muscle; 3: lateral plantar nerve; 4: abductor digiti quinti muscle; 5: the first branch of the lateral plantar nerve; 6: flexor digitorum longus muscle; 7: flexor halucis longus muscle.

CLINICAL SYMPTOMS AND SIGNS

Typically, the patient will complain of aching or shooting pain along the medial aspect of the arch during running. Jogger's foot is characterized by exercise-induced neuritic pain at the medial arch radiating into the toes along the distribution of the medial plantar nerve.[1] Most characteristically, the onset of pain is associated with the use of a new arch support[4] or new shoes, without a change being made in the training regime. The pain, numbness, or tingling will worsen with high arch supports, especially rigid orthoses. The characteristic neuritic symptoms consist of burning, shooting, and sharp pain radiating from the arch toward the plantar aspect of the second and great toe.

Physical examination demonstrates tenderness along the plantar aspect of the medial arch in the region of the navicular tuberosity. A positive Tinel's sign may be found just behind the navicular tuberosity with reproduction of the paresthesias. Pain can be reproduced by everting the heel or having the patient stand on the ball of the foot. Flexion of the toes against resistance should not induce pain, nor should passive toe hyperextension. These two findings help distinguish between nerve irritation and flexor tenosynovitis. Evaluation of a runner's shoes and orthoses helps to assess

heel, arch, and foot position as well as shoe wear. A runner can also be evaluated after a training run. An injection combined with a dynamic test can help confirm the diagnosis.

TREATMENT

Nonoperative treatment usually is successful.[3–6] Treatment consists of eliminating any high-arched orthoses, especially if they are rigid. Matching shoe design to the foot or the gait pattern or adjusting break-in patterns can limit irritation. Training regimes might also be scrutinized and modified. Uneven surfaces can aggravate a runner's symptoms. Injecting the entrapment point (usually Lidocaine and cortisone) can be done for diagnostic purposes or to relieve the localized inflammatory reaction. Rest or limiting the distance run assists in healing. Anti-inflammatory medications have a definite place in the overall therapy.[5] If conservative treatment is unsuccessful, electromyography or nerve conduction velocity testing can be done to evaluate for neuropathy or more proximal compression.[6] Surgical neurolysis should be reserved for those patients who do not respond to conservative measures.[5] At surgery, the area of maximum tenderness should be addressed by releasing the fascia over the nerve in the affected zone.[6]

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52 Syndrome of the First Branch of the Lateral Plantar Nerve

Chronic plantar heel pain may be caused by compression or entrapment of the first branch of the lateral plantar nerve as it passes around the inferior medial border of the heel. This nerve is also known as the nerve to the abductor digiti quinti muscle. In 1940, Roegholt[9] first proposed that entrapment of this nerve branch was the cause for heel pain. Tanz[13] in 1963 presented the first compelling evidence that linked nerve entrapment to heel pain. Przyluki and Jones[8] reported on three patients with chronic heel pain whose symptoms were relieved by excision of the lateral plantar nerve branch described previously by Tanz. They demonstrated that the nerve innervated the abductor digiti quinti muscle, traveling across the heel anterior to the medial tuberosity of the calcaneus.[8]

ANATOMY

The tibial nerve, known at this level as the posterior tibial nerve, divides into its calcaneal sensory branches and the medial and lateral plantar nerve branches. The plantar nerve branches provide intrinsic motor function and sensibility to the plantar aspect of the foot. The lateral plantar nerve runs between the flexor digitorum brevis muscle and the quadratus plantae muscle forming an arch directed laterally and distally. It gives off muscular branches (rami), ramus profundus (deep branches) and ramus superficialis (superficial branches).[14] The muscular branches supply the abductor digiti quinti, flexor digiti quinti, and quadratus plantae muscles. The first branch of the lateral plantar nerve innervates the abductor digiti quinti muscle and is also known as the nerve of the abductor digiti quinti (Figure 52.1). Entrapment of this nerve has been postulated to cause heel pain. This has led to special investigations dedicated to its anatomic variations. Hamm and Sanders[5] found that the nerve to the abductor quinti muscle was present in 100% of specimens. It originates as the posterior branch of a trifurcation of the posterior tibial nerve in 46% of specimens, a branch of the lateral plantar nerve in 49% of specimens, and a direct branch of the posterior tibial nerve in 5% of specimens. In most specimens, the nerve's origin was either at the level of the medial malleolus, the sustentaculum tali or between the two sites (79%). Rhondhuis and Husson[10] demonstrated that, as the first branch passes across the heel, it inervated not only the abductor digiti quinti muscle, but also the quadratus plante muscle, the flexor brevis muscle, and the periosteum overlying the medial calcaneal tuberosity. The first branch thus represents a mixed nerve containing both motor and sensory fibers.[10]

ETIOLOGY

Entrapment of the first branch can occur as the nerve changes from a vertical to a horizontal direction around the medial plantar aspect of the heel.[1] Compression takes place between the thick deep fascia of the abductor hallucis muscle and the medial caudal margin of the medial head of the quadratus plantae muscle. Another potential site of entrapment of the first branch of the lateral plantar nerve is where the nerve passes just distal to the medial calcaneal tuberosity. A spur or inflammation at the origin of the flexor digitorum brevis muscle or plantar fascia might provide sufficient compression of the nerve against the long plantar ligament and the calcaneus. The chronic

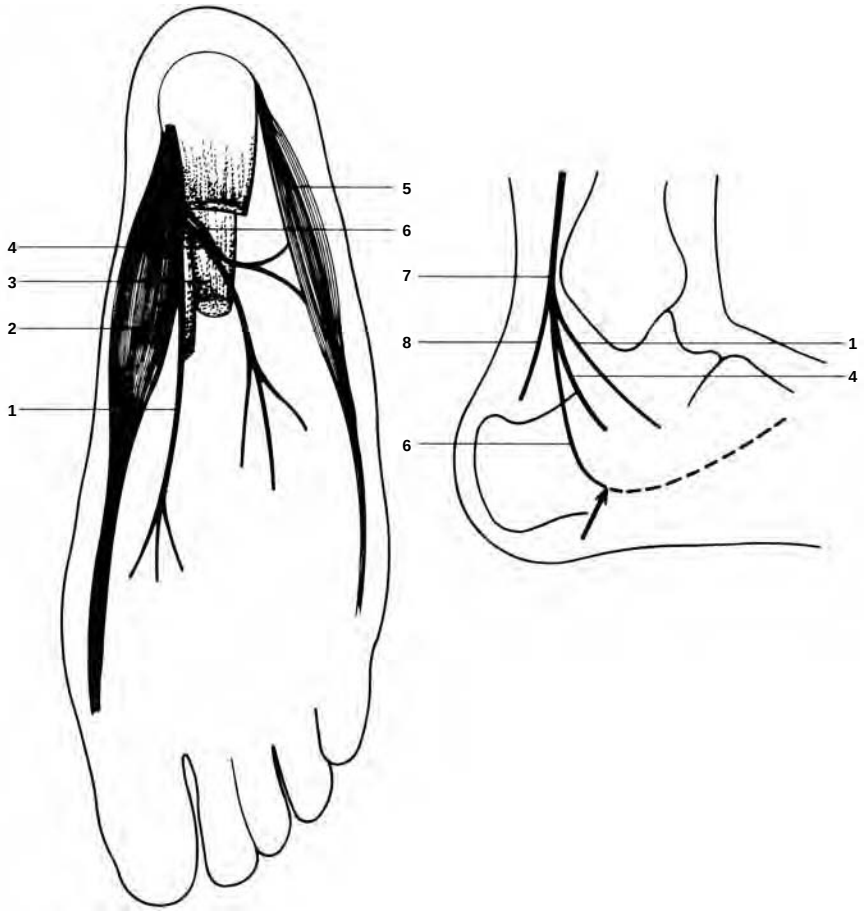

FIGURE 52.1 The first branch of the lateral plantar nerve innervates musculus abductor digiti quinti and is also known as the nerve of the abductor digiti quinti muscle — 1: medial plantar nerve; 2: abductor hallucis muscle; 3: quadratus plantae muscle; 4: lateral plantar nerve; 5: abductor digiti quinti muscle; 6: the first branch of the lateral plantar nerve (the nerve of the abductor digiti quinti); 7: tibial nerve (posterior tibial nerve); 8: medial calcaneal nerve.

inflammatory changes might predispose to chronic entrapment. Nerve entrapment may be a contributing factor in as many as 20% of patients with chronic heel pain.[1] This occurrence is more likely in patients who have edema of the flexor digitorum brevis muscle in conjunction with microtears and edema of the plantar fascia. Fifty percent of the patients operated on by Baxter and Pfeffer[2] had developed pain in direct relation to sports. The most common sport reported was long-distance running.[2]

CLINICAL SYMPTOMS AND SIGNS

Accurate diagnosis is dependent on close attention to the anatomy and to the known problem areas. Although a Tinel's sign is not always reliable, consistent pain at the plantar medial aspect of the heel over the nerve is diagnostic.[3] On physical examination, reproduction of neuritic pain with palpation of the plantar medial aspect of the heel along the proximal abductor hallucis or plantar fascia is characteristic.[12] The pathognomonic sign of the entrapment of the first branch of the lateral plantar nerve is maximal tenderness in the area where the nerve is compressed between the taut deep fascia of the abductor hallucis muscle and the medial caudal margin of the quadratus plantae

muscle.[2] Some patients may have paresthesias elicited with pressure over the nerve at the entrapment site. In advanced cases, diminished ability to actively abduct the small toe may be evident.[3] The utility of electromyography (EMG) or nerve conduction velocity (NCV) studies in diagnosis has been discussed.[4,7,11] While the sensitivity of electrodiagnostic evaluation of individual patients with suspected plantar nerve entrapment remains uncertain, complete electrodiagnostic evaluation may improve the clinical assessment and provide objective evidence of neurologic abnormalities.[11] Mabin[7] believes that EMG or NCV studies are essential to evaluate peripheral nerve injuries to differentiate focal lower-extremity nerve entrapment from ischemic mononeuropathies, lumbar radiculopathies or plexopathies, and generalized peripheral neuropathies.

In the absence of consistent objective findings for nerve compression, it is, therefore, incumbent on the examiner to differentiate first branch entrapment from the other more common causes of heel pain: plantar fasciitis, heel pain syndrome, and fat pad disorders. The use of diagnostic injections (nerve blocks) should be combined with the observed clinical signs and described symptoms to improve diagnostic accuracy.

TREATMENT

Most patients respond to conservative treatment. This includes the use of heel cups or pads with or without a heel lift in the shoe. A stretching program focused on the Achilles tendon and the plantar fascia should be done concurrently.[12] Additionally, non-steriodal anti-inflammatory medications may be useful. Local corticosteroid injection should be considered before surgery. The injection can be not only diagnostic but also therapeutic. If limited pain relief is obtained, surgical decompression should be approached with limited expectations. Surgery to decompress the nerve[1–3,6] should include release of the superficial and deep fascia of the abductor hallucis and a partial proximal medial plantar fasciectomy. Baxter and Pfeffer's overall success rate after surgery is approximately 89% excellent or good results.[2]

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53 Morton's Metatarsalgia

Metatarsalgia has become a common name for multiple disorders arising from pain in the forefoot. Debating the true meaning of metatarsalgia goes beyond the scope of this chapter; however, compression of nerves in the metatarsal tunnels produces forefoot pain commonly known as Morton's neuroma, Morton's metatarsalgia, neuroma plantaris, or Morton's disease.

ANATOMY

The metatarsal tunnels lie between the superficial and deep transverse metatarsal ligaments (ligamentum metatarseum transversum profundum and superficiale), which connect the metatarsal heads. The medial and lateral plantar nerves and the medial and lateral plantar arteries and veins produce the common digital neurovascular bundle that transverses the metatarsal tunnels. The medial and the lateral plantar nerves supply branches to the first, second, and third metatarsal spaces and the third and fourth metatarsal spaces, respectively. These fields on the plantar surface have minimal overlap. The medial plantar nerve also supplies the great toe, while the lateral plantar nerve supplies the fifth toe (Figure 53.1). The branch to the great toe originates as the medial plantar nerve leaves the tarsal tunnel. The common digital branches from the medial plantar nerves originate at a branch point over the first and second metatarsal bases. Anesthetic injection into this area distal to the medial cuneiform bone will produce complete anesthesia of the first and second metatarsal spaces, since the third space receives some innervation from the lateral plantar nerve. Within the tunnels, the common digital nerves branch into the digitales proprii nerves for the medial and lateral plantar skin of the corresponding toes (i.e., the first metatarsal space lies between the great and second toe; thus, there is a lateral branch to the great toe and a medial branch to the second toe). The tendons of the flexor muscles and the interossei muscles contribute to the tunnel formed by the transverse ligaments and the metatarsal heads.

ETIOLOGY

While the pathology was first described by Durlacher in 1894,[1] in 1876, Morton[2] first postulated compressive neuropathy as the etiology. Tubby[3] provided operative data detailing a neuroma-like swelling between the metatarsal heads. Prolonged compression and irritation of the neurovascular bundle leads to endoneural edema, nerve finger degeneration with reparative changes and thickening of the nerve, and hyaline changes and thickening of the walls of the vessel.[4] Multiple authors have described the etiology as that of an entrapment neuropathy.[5–9]

While many etiologies have been proposed, most require a change in the metatarsal tunnel size. Trauma, chronic overuse, inflammatory diseases, functional anatomy, and degenerative changes of the tunnel's tissues probably provide the changes necessary to compress or to irritate the nerve.[10–13,47] Hormonal factors that produce edema, which can be found in pregnant or premenstrual woman,[14,15] and nerve ischemia[16] have also been suggested as etiologies. However, the interdependence of all of these factors makes it statistically difficult to isolate the basic causes. Since the neurovascular structures are nearly at their terminal point, local nerve ischemia can easily result from compression.

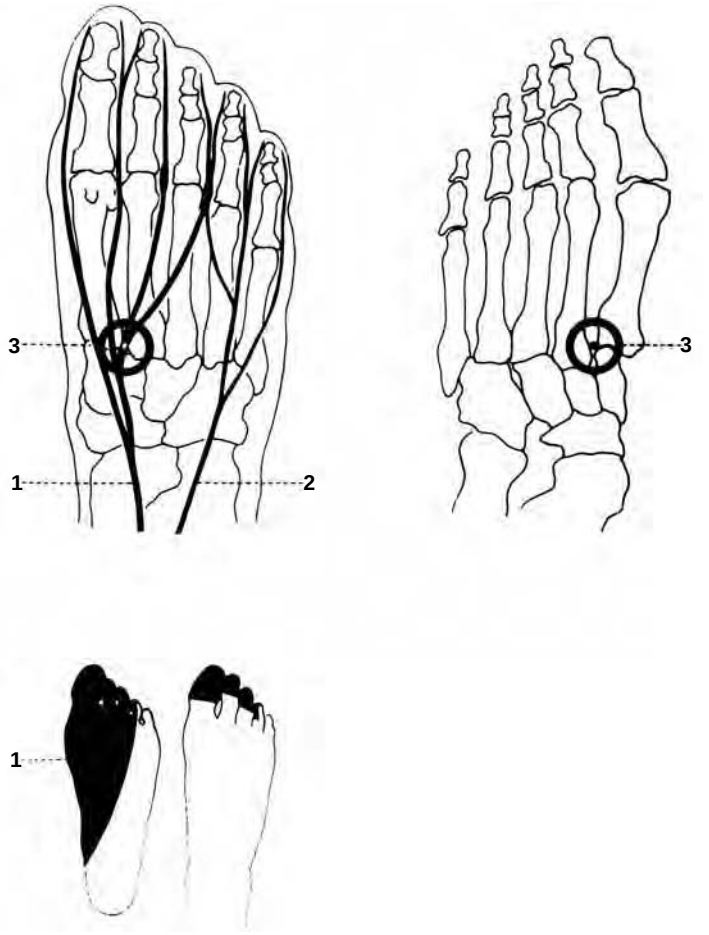

FIGURE 53.1 Course of the medial (1) and lateral (2) plantar nerves. A diagnostic intervention may include the injection of anesthetic into the space indicated (3). The dermatome of the medial plantar nerve typically is involved and is highlighted on the plantar and dorsal views of the foot.

In the development of the metatarsalgia, distortion of the metatarsophalangeal joints caused by trauma plays a major role. Fractures or subluxation of the joint changes the anatomy of the tunnel. Osteochondritis of the second metatarsal head (Morbus Kohler-Frieberg) can lead to tunnel narrowing.[17] Minimal variations in the tunnel anatomy could lead to metatarsalgia if only repetitive nerve irritation from the anterior margin of the transverse metatarsal ligament is needed.[18] Other causes of compression include rheumatic inflammatory diseases, ganglions, and synovial cysts.[19]

The anatomical relationships of the metatarsal canal have been proposed as the basic uniting theory.[5,20] Because all of the above etiologies affect the static and, therefore, the dynamic anatomy of the metatarsal tunnel, simple anatomical relationships cannot be the sole cause of the metatarsalgia. However, foot motion and position alter the anatomy of the tunnel. Distortion and variation in relationships of the metatarsal heads will affect the size of the tunnel. Congenital and acquired abnormalities of the entire leg may alter the weight-bearing status of the foot, predisposing the nerve to compression in the region of the metatarsal heads. Due most frequently to shoes with high heels or narrow toe boxes,[21] hyperextension of the toes narrows the metatarsal tunnels. Crouching duplicates the position created by high-heeled shoes; therefore, individuals whose professions require long periods of crouching may present with metatarsalgia. Flexion contractures of the hip and knee, pes equinus, digiti mallei, and other foot deformations also lead to toe hyperextension to compensate for the more proximal abnormalities.

CLINICAL SYMPTOMS AND SIGNS

Morton[2] described metatarsalgia as follows: "In walking, paroximal pain appears, pain that goes to the heart and provokes unbearable sensations with cold sweat and finally prevents the individual to direct his spirit and will to any other subject but to this unbearable pain." This picture remains accurate today. Pain appears cutting or electrical in sensation but never diffuse and burning, like plantar fasciitis. Toe hyperextension in high-heeled shoes or while crouching aggravates the pain. Patients will even describe having to stop work to remove their shoes. Avoiding shoes with high heels or narrow toe boxes may eliminate the pain; however, trouble may persist, regardless of an individual's footwear.

Clinical examination reveals a trigger point at the metatarsal heads, especially between the third and fourth heads (the third metatarsal space). The second and fourth metatarsal spaces are less frequently involved, and the first metatarsal space is rarely involved. Compression of the metatarsal heads against each other provokes and increases the pain. While typically masked by the pain, hypoanalgesia or analgesia on the skin of the involved toes may be felt. Morton's metatarsalgia should not be confused by name or clinical findings with Morton's foot (Neanderthal foot, short first metatarsal bone), when the worst pain localizes to the basis of the first and second metatarsal bones.[22] In an attempt to improve the accuracy of diagnosis, Guiloff et al.[9] conducted clinical and electrophysiological examinations of 16 patients with atypical foot pain. Many authors[9,23,24] concluded that nerve conduction velocity studies were helpful in clarifying the diagnosis. The diagnosis of Morton's neuroma has been improved by using computerized tomography,[25] ultrasonography,[26,27] and magnetic resonance imaging.[27–31]

TREATMENT

Removal of the provocative causes can be accomplished by several approaches.[45,46] Conservative methods include avoidance of high-heeled shoes, shoes with narrow toe boxes, and prolonged plantar flexion or toe extension. Additionally, support of not only the metatarsal heads with a pad proximal to the heads but also support of the arch with a longitudinal foot arch should be provided. Physical therapies, anti-inflammatory medication, and local corticosteroid injections have been found to be effective (Figures 53.1 and 53.2).[32,33] Rather than inject the tunnel with the additional

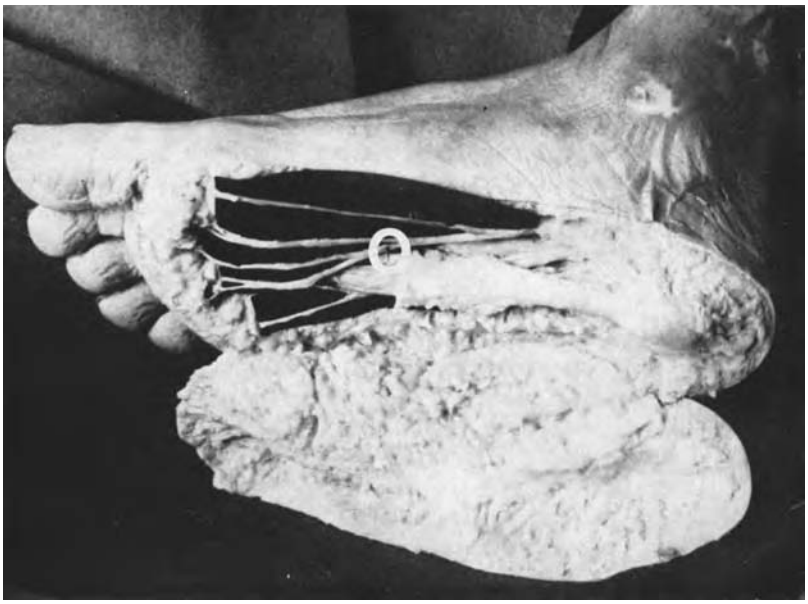

FIGURE 53.2 This pathologic specimen has been dissected to show the nerve branch point that is injected when a needle is placed in the interspace between the first and second metatarsal bones.

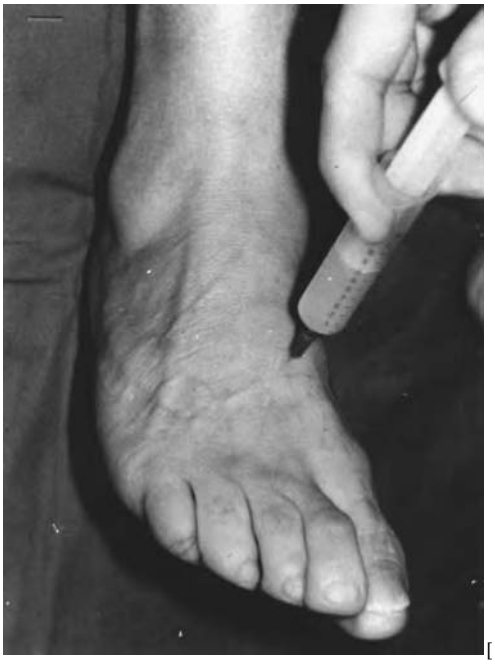

FIGURE 53.3 Actual location of the injection on the dorsum of the foot.

fluid, Krmpotić-Nemanić et al.[32,33] recommended injections distal to the leading edge of the medial cuneiform bone, as shown in Figure 53.3. While the needle may come close to the medial plantar nerve and produce pain through its distribution, the corticosteroid injection should be placed around the nerve. The local anesthetic included will provide sudden but temporary disappearance of the symptoms and temporary anesthesia in the dermatome of the nerve. If removal of aggravating factors is continued, the use of local corticosteroid injection may be repeated, achieving a success rate of 70%. Krmpotić-Nemanić et al.[32,33] found a 70% success rate in women treated with 3 to 10 local injections. Surgical removal of the offending neuroma,[34–38] treatment with carbon dioxide laser surgery,[39] endoscopic decompression,[44] or neurolysis[40] can be used (see Figure 53.1). If no neuroma is experienced, the transverse metacarpal ligament should still be sectioned. Unfortunately, surgery may not yield complete satisfaction. Johnson et al.,[41] Young and Lindsey,[42] and Amis et al.[43] describe reoperation because of persistent pain after excision of an interdigital neuroma.

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Section V

Tunnel Syndromes in Athletes

54 Tunnel Syndromes in Athletes*

In describing individual tunnel syndromes in the upper and lower extremities, acute trauma and especially long-term repetitive microtrauma in areas of specific tunnels have been indicated as instigating agents.[1–10] Certain sports or physical activities have been mentioned that lead to specific tunnel syndromes — for example, cyclist's palsy and bowler's thumb.

Unlike tunnel syndromes (nerve entrapment syndromes), vascular and neurovascular syndromes in athletes seem to be more common and have been described in greater detail, while tunnel syndromes in athletes have been reported only recently. This chapter provides some information about tunnel syndromes in particular athletic activities, with specific reference to each individual tunnel syndrome presented in this book.

THE UPPER EXTREMITIES

The spinal accessory nerve is the cranial nerve most susceptible to injury. The clinical picture is primarily characterized by loss of power of trapezius muscle and difficulty in lifting the arm. During sports, the spinal accessory nerve may be injured by blunt stretching trauma.[121] Stretch injuries can also be caused by lifting heavy objects, or by vigorous training programs that include floor pushups.[122] The nerve may be damaged by a direct blow, for example, with a hockey stick or as a result of direct contact between athletes. Another mechanism that has been described is depression of the shoulder with the head forced in the opposite direction.[121] Such an injury occurs most commonly in wrestling, the result of a cross-face maneuver.

The syndrome of upper-limb pain, paresthesias, vascular insufficiency, and motor dysfunction stemming from compression of the brachial plexus, the subclavian artery, or the subclavian vein before their division and separation, bears the name *thoracic outlet syndrome*. This syndrome has been found among athletes, especially those who engage in certain sports such as swimming and other activities that require a swinging motion of the arm (throwing).[11–14] Compression in the outlet can occur at any one of three levels: interscalene triangle, costoclavicular space, and pectoralis minor muscle insertion on the coracoid process. Abnormal structural variations (cervical rib, fibrous band, "abnormal" scalene muscle development) may compress or cause friction of the plexus or vessels at the level of the interscalene triangle.

Compression of neurovascular structures through the costoclavicular space is usually caused by dynamic changes, especially in shoulder girdle mechanics, i.e., in the functional anatomy of the shoulder girdle.[15] The coracoid process and the pectoralis minor muscle insertion act as a fulcrum over which the neurovascular structures change direction when the arm is elevated. This site has been implicated as a source of neurovascular compression among athletes who repetitively hyperabduct the arm — swimmers, tennis players, pitchers. In swimmers, neurovascular compression in this area could develop as a result of pectoralis minor muscle hypertrophy.[16] Different patterns of clinical presentation are seen, depending on where and which neurovascular structures are compressed.

The patients' symptoms are created by neurological compression.[13] Typical symptoms are pain, numbness, or paresthesias. Sensory loss and muscle atrophy are rare, but can occur. Vascular

* Additional contributions by Ivan Bojanić.

symptoms of thoracic outlet syndrome are quite rare. Venous obstruction may cause arm edema, cyanotic discoloration, and venous collaterization across the shoulder and chest wall, whereas arterial obstruction produces symptoms of coolness, numbness, ischemic pain, and exertional fatigue. A very common symptom pattern in thoracic outlet syndrome is that of "mixed involvement." In the mixed symptom pattern, patients have symptoms of both upper- and lower-trunk compression, along with variable degrees of vascular insufficiency.

Thoracic outlet syndrome remains a clinical diagnosis, based almost entirely on the history and physical examination.[13] Therefore, thoracic outlet syndrome should be a diagnosis of exclusion. Initial treatment of patients with thoracic outlet syndrome should be nonoperative and should include rest from athletic activities, as well as shoulder musculature rehabilitation to diminish the soft tissue mass and improve posture.[7,12,13] Surgical treatment is indicated in patients with significant neurological or vascular involvement that does not respond to nonoperative treatment.[7,12,13]

Compression of the brachial plexus may arise from intrinsic or extrinsic factors, both of which can occur in association with athletic activity. The most common external agent creating compression of the brachial plexus is a knapsack.[17–19] In Hirasawa and Sakakida's series,[19] most of the observed brachial plexus lesions were described as *backpack paralysis*. Brachial plexus compression results when large, heavy backpacks are carried for long periods. The axillary straps create a compression force around the plexus, with the clavicle as a firm strut against which compression can occur. The shoulder girdle is pulled posteriorly by the heavy pack, adding a component of traction. Treatment of backpack paralysis combines avoiding the mechanisms thought to have caused it with physical therapy. Physical therapy restores the patient's strength, allowing complete recovery.

Long thoracic nerve entrapment has been described in a wide variety of sports, including tennis, golf, cycling, gymnastics, soccer, bowling, weightlifting, basketball, and football.[20–24] Clinical features may include pain around the shoulder girdle, decreased active shoulder motion, and a winged scapula. Electromyography and nerve conduction studies can be used to exclude other causes of scapular winging and confirm the diagnosis. Treatment involves physical therapy and cessation of the instigating activity. Prognosis is quite favorable, but recovery could take up to 2 years.[20–23]

Suprascapular nerve entrapment is an infrequently observed disorder that is often misdiagnosed. The manner of presentation of this syndrome depends on the anatomical site of compression. Entrapment usually happens at the suprascapular notch. Patients, mostly throwing athletes, suffer poorly localized pain, intact sensation, weakness of external rotation and abduction, and atrophy of the supraspinatus and the infraspinatus muscle.[25–29] Occasionally, entrapment is seen distally at the spinoglenoid notch. Patients may be asymptomatic or might describe mild pain and weakness of the shoulder because of denervation of the infraspinatus.[33–32,129,131,133] Ferretti et al.[30] found 12 top-level volleyball players with isolated asymptomatic paralysis of the infraspinatus muscle. Sandow and Ilić[130] describe the suprascapular nerve rotator cuff compression syndrome in volleyball players. Ganzhorn et al.[33] described the case of a weight lifter who, while posing in front of a mirror, came for treatment after noticing wasting in the region of the dorsal scapula. Zeiss et al.[128] and Zuckerman et al.[127] also described suprascapular entrapment neuropathy in weight lifters. When the entrapment is localized to the suprascapular or spinoglenoid notch, restraint from athletic activities, nonsteroidal anti-inflammatory medication, and local steroid injections may be useful.[25,26,34] If such nonoperative measures are unsuccessful, surgical exploration is indicated.

Entrapment of the axillary nerve in the quadrilateral space is rare in athletes.[35] It has been noted in baseball pitchers.[36] Paladini et al.[134] described two case of isolated neuropathy, not consequent to acute trauma, of the axillary nerve of young volleyball players. The lesion site is thought to be in the quadrilateral space. The quadrilateral space syndrome also has been seen in tennis players.[135]

Compression of the musculocutaneous nerve as it passes through the coracobrachialis muscle has been seen in weightlifters secondary to muscle hypertrophy.[22,26,37–40] This lesion results in biceps

brachii and brachialis muscle wasting and weakness. Sensory complaints are referred to the lateral side of the forearm. Typically, no pain is associated. Cessation of the strenuous inciting action, rest, and a gradual return to activity when symptoms resolve are components of an effective therapy.[37–40,136]

The *lateral (antebrachial) cutaneous nerve* is compressed between the distal biceps tendon and the brachialis muscle. This entity has been described in racquetball and tennis players, and in a windsurfer.[22,41,155] Symptoms include pain, paresthesias, and numbness over the radial aspect of the forearm. Treatment consists of rest, nonsteroidal anti-inflammatory medication, and a posterior elbow splint that prevents full extension. After 12 weeks of unsuccessful nonoperative treatment, surgical decompression is indicated.[41,136]

Entrapment of the radial nerve above the elbow is rare in athletes.[42] Cases have been described after throwing a discus and serving in tennis.[137] The site of compression is in the region of the lateral intermuscular septum, which the radial nerve pierces as it enters the anterior aspect of the arm. It has been reported after strenuous activities.[138,139] In most cases, the symptoms will disappear within several weeks. Persistent or progressive symptoms suggest the need for surgical decompression.[42,43]

In their study of a group of patients with chronic tennis elbow, Roles and Maudsley[44] recognized the compression of the radial nerve within its tunnel and called it *radial tunnel syndrome*. The syndrome differs from a posterior interosseous nerve syndrome, in which the problem is localized to compression of the nerve at one particular site, the arcade of Frohse, and results in only a motor deficit.[45–50] In radial tunnel syndrome, there may be a spectrum of complaints, including pain, paresthesias, and weakness.[51] A motor deficit is not nearly as common as in a posterior interosseous nerve syndrome. There are five potential sites of compression within a radial tunnel: fibrous bands at its proximal portion, a fibrous medial edge along the extensor carpi radialis brevis, a "fan" of radial recurrent vessels, the arcade of Frohse, and, last, a fibrous band at the distal edge of the supinator muscle. The radial tunnel syndrome most commonly affects tennis players but may also be seen in rowers and weightlifters.[42] Nonoperative measures should be the first form of treatment. Such measures include rest of the elbow and wrist from repetitive stressful activity and a course of anti-inflammatory medication. Surgical exploration with neurolysis is indicated if nonoperative treatment fails.[42,52,140]

Athletes participating in sports requiring repetitive pronation-supination ulnar flexion may acquire *Wartenberg's disease*, entrapment of the superficial sensory branch of the radial nerve in the forearm.[53,54] The wearing of wristbands, as in racquet sports, has been implicated as a cause of this syndrome, also known as handcuff neuropathy.[46,55,56] The patients' main complaints are burning pain or numbness and tingling over the dorsoradial aspects of the wrist, thumb, and web space, usually associated with or aggravated by wrist movement. If the syndrome stems from external compression, nonoperative therapy can be successful. It may take several months for the symptoms to resolve. Surgical exploration is necessary if nonoperative therapy fails.[53,54]

The *distal posterior interosseous nerve syndrome* occurs when the nerve gets compressed or irritated traveling in the fourth extensor compartment. The repetitive dorsiflexion activities in the wrist can result in irritation or in compression of the nerve. In the statistics of Carr et al.,[123] ten patients were employed in occupations or engaged in vigorous athletic activities, such as gymnastics, that required repetitive dorsiflexion of the wrist. The patients present with dull ache, which can be reproduced by dorsiflexion of the wrist and by pressure over the fourth extensor compartment.

Ulnar nerve entrapment at the elbow is most frequently encountered among throwing athletes, such as baseball pitchers, tennis players, and javelin throwers, but it is also observed in those engaged in skiing, weightlifting, and stick-handling sports.[57–61] Because of its position in the cubital tunnel, the ulnar nerve is vulnerable to repetitive tension or traction stresses in athletes. This stress can be compounded by subluxation or instability of the nerve. Childress[62] reported that 16.2% of the general population suffered recurrent dislocation of the ulnar nerve when the elbow was flexed

and extended. Repeated stress and injury may lead to inflammation, adhesions, and progressive compressive neuropathy.

The intermittent nature of athletic endeavors might confound the presentation of the athlete with entrapment of the ulnar nerve at the elbow. Sometimes, the first symptom will consist of pain along the medial joint line that is either associated with or exacerbated by overhead activities. As the inflammation of the nerve progresses, pain and paresthesias will be noted down the ulnar aspect of the forearm to the hand. Sensory changes definitely precede motor changes; however, a careful evaluation of the intrinsic musculature of the hand is essential to detect any weakness. Initial treatment consists of avoidance of underlying cause (e.g., throwing), nonsteroidal anti-inflammatory medication, and occasionally immobilization. Quite often, recalcitrant ulnar nerve entrapment at the elbow requires surgery.[141] However, many transient episodes can be treated nonoperatively.[61,62] Del Pizzo et al.[57] reported on 19 baseball players with ulnar nerve entrapment at the elbow who underwent surgery. The surgery consisted of anterior transfer of the nerve deep to the origin of the flexor muscles.

Entrapment of the ulnar nerve in Guyon's canal (ulnar tunnel syndrome) is seen in cyclists and racquetball players as a result of chronic external compression. The first report of ulnar neuropathy as a complication of long-distance cycling was published in 1896.[63] Several reports since have described this complication, called cyclist's or handlebar palsy.[64–71] Factors reported in the literature as contributing to the development of neuropathies in cyclists include the use of worn-out gloves, unpadded handlebars, an improperly adjusted bicycle seat, and vibratory trauma from rough roads. Symptoms include weakness of grip and occasionally numbness of the fourth and fifth digits. Jackson[69] recently studied 20 cyclists who rode more than 100 miles per week and found that nine complained of either hand or finger numbness during cycling that resolved after completion of the ride. They reported that their hand numbness or pain was alleviated by adjusting their hand position. Conventional treatment for nerve compression syndrome at the wrist consists of changes in cycling techniques, including frequently varying hand position, the use of properly padded gloves and handlebars, and adjustments to the bicycle that ensure a proper fit.[69,70] These changes frequently will relieve symptoms in most cases without the need for surgical decompression of the Guyon's canal.

Friedland and St. John[143] described entrapment of the ulnar nerve at the wrist in a video-game enthusiast who put excessive pressure on his palm.

Repetitive trauma to the heel of the palm can cause ulnar artery spasm, thromboses, or aneurysms, leading to compromised ulnar nerve function with a more vascular type of presentation. This condition, known as *hypothenar hammer syndrome*, has been described in athletes engaged in several sports, including karate, judo, tennis, and lacrosse.[54,72–74] Nonunion of the hook of the hamate or of the pisiform, which may be fractured during a tennis, baseball, or golf swing, can also cause entrapment within the ulnar tunnel (Guyon's canal).[46] It is important to keep in mind the possibility of a double-crush injury of the ulnar nerve in conjunction with the syndrome of the flexor carpi ulnaris muscle (cubital tunnel syndrome) and ulnar tunnel syndrome.[69,75]

Entrapment of the median nerve at the elbow is termed the *pronator teres syndrome* and may stem from repetitive exercise, with resultant hypertrophy of the flexor-pronator muscle group.[22,75–77] This entity has been seen in baseball players and weightlifters.[142] Patients complain of pain and tenderness in the volar aspect of their forearms over the area of compression, which worsens with exertion. Sensory complaints are common, consisting of numbness and paresthesias in part or all of the median nerve distribution of the hand. The pronator teres syndrome is often a difficult diagnosis and must be distinguished from carpal tunnel syndrome. Because most cases are intermittent and mild, nonoperative treatment should be tried first.[22,69,75] Persistent or progressive symptoms suggest the need for surgical intervention.[22,75]

Anterior interosseous syndrome (Kiloh-Nevin syndrome) has been described in association with repetitive activities, such as throwing, racquet sports, or weight lifting.[8,22,42,78] It is characterized by a vague feeling of discomfort in the proximal forearm that might mimic a pronator teres syndrome.

However, because the anterior interosseous nerve is a pure motor division of the median nerve, there are no sensory complaints or deficits as in pronator teres syndrome. The classic finding is that the patient loses the ability to pinch his thumb and index finger. However, this symptom is not always present. Initial treatment should be nonoperative, because in many cases there will be spontaneous improvement; however, if there is no improvement after 8 to 12 weeks, surgical decompression and neurolysis should be performed.[7,22,75]

The incidence of *carpal tunnel syndrome* as a sports-related problem is surprisingly low when compared with the incidence in the general population. Carpal tunnel can be seen in athletes who play sports that require gripping, throwing, cycling, or repetitive wrist flexion-extension. It can also stem from direct trauma.[19,22,28,46,47,54,75,77,79–82]

Digital nerve entrapment syndromes in athletes are less common than those entrapment syndromes at the wrist level. Digital nerves may be compressed in the distal palm or at the proximal digit. Bowler's thumb is the most common syndrome of the digital nerve in the hand in athletes.[46,47,54,77,80,83–87] Repetitive compression of the ulnar side digital nerve to the thumb from direct pressure on the nerve at the thumb hole of a bowling ball has been implicated as a cause of bowler's thumb, which has also been reported in a baseball player.[46] On physical examination, patients have tenderness over the ulnar volar aspect of the metacarpophalangeal joint of the thumb and a positive Tinel's sign in this area, with paresthesias radiating to the ulnar aspect of the tip of the thumb. There is no motor involvement; however, grip strength may be somewhat diminished because of pain.

Bowler's thumb should be treated nonoperatively with rest, cessation of activity, nonsteroidal anti-inflammatory medication, and modification of equipment and technique.[54,83] In advanced cases, a molded plastic thumb guard is recommended to prevent trauma. Surgical treatment is indicated for those with persistent significant symptoms.[54,83,86] Surgical options include resection of the neuroma, primary repair of the nerve, neurolysis, and transfer to a new location. Compression of digital nerves in tennis players has recently been reported.[78,88] Symptoms include numbness along the volar surface of the index finger of the racquet hand and an abnormal sweat pattern, especially in players who have recently started playing or increased their amount of playing. Physical findings usually include calluses over the second metacarpal head, which implies rubbing of the digital nerve between the fixed bone and the racquet handle. Early recognition, improved technique, better equipment, and protective measures are helpful in treating this problem.[78,88] Surgery is very rarely indicated.

THE LOWER EXTREMITIES

Groin pain in athletes can be a consequence of nerve entrapment syndrome.[144–147]According to Akita et al.,[144] chronic groin pain, especially in athletes, has been diagnosed in various ways. In Europe, recently, the concept of "sports hernia" has been advocated. Entrapment of the ilioinguinal nerve and the genitofemoral nerve may have a very important role in chronic groin pain produced by groin hernia. Bradshaw et al.[145,146] describe a case of obturator nerve entrapment, a previously unreported cause of chronic groin pain in athletes. They report 32 cases of "obturator neuropathy," a fascial entrapment of the obturator nerve where it enters the thigh. There is a characteristic clinical pattern of exercise-induced medial thigh pain commencing in the region of the adductor muscle origin and radiating distally along the medial thigh. Needle electromyography demonstrates denervation of the adductor muscles. Surgical neurolysis treatment provides the definitive cure of this problem, with athletes returning to competition within several weeks of treatment. The surgical findings are entrapment of the obturator nerve by a thick fascia overlying the short adductor muscle. The role of conservative treatment in the management of this condition is unknown at present.[146]

Piriformis muscle syndrome is not discussed with specific reference to athletes, although athletic activities may cause changes that significantly contribute to the development of the syndrome. Basic anatomical relationships of the muscle and sciatic nerve, direct and indirect trauma, muscular

hypertrophy or anatomical nerve variations,[147] inflammation, and local ischemia probably combine to induce the piriformis muscle syndrome.[5–7,89,90] The piriformis syndrome has many similarities to, and overlaps with symptoms of, low back pain, ischialgias, vascular disease, and lower-extremity pathologies.[7,91] Pain in the sacral or gluteal region remains the most constant symptom. The pain increases with sitting, walking, or running and decreases with lying supine. Frequently, diagnosis requires eliminating other causes of sciatic pain. Nonoperative treatment includes physiotherapy, nonsteroidal anti-inflammatory medication, and local steroid injection.[7,89] Surgical treatment consists of sectioning of the piriformis muscle at its tendinous origin and external neurolysis of the sciatic nerve.[7] Kouvalchouk et al.[150] report four athletic patients (two cyclists, two long-distance runners) who have been treated surgically by section of the piriformis muscle and neurolysis of the sciatic nerve. Pećina treated several athletes (soccer players, track and field athletes, fencer) with piriformis muscle syndrome conservatively and surgically.

Pudendus nerve syndrome is caused by compression of the nerve when it passes through the foramen infrapiriforme, foramen ischiadicum minus and especially in the region of Alcock's canal situated in the lateral wall of the ischiorectal fossa. The compression of the nerve results in sensitive disturbances, often of a neuralgic type, in the innervation field of the nerve. The etiology of pudendus nerve syndrome includes some sport activities, such as bicycling, motorcycling and horseback riding or all three, causing pressure, blows or vibration in the pelvic region.[124] Perineal numbness is well known among cyclists and often is attributable to fixing the saddle in a "nose-up" position. This can be alleviated by using a softer saddle and placing it either horizontal or "nose-down."[148] Impotence and nerve entrapment in long-distance amateur cyclists was reported by Andersen and Bovim.[149]

Meralgia paresthetica is an entrapment syndrome of the lateral femoral cutaneous nerve as it enters the thigh through or under the superolateral end of the inguinal ligament, causing burning sensations, paresthesias, and dysesthesias of the anterior and lateral thigh. This entrapment could be due to direct or repetitive trauma. Among athletes, it has been found primarily in gymnasts.[92] Meralgia paresthetica usually responds to nonoperative treatment, including avoidance of repetitive trauma or pressure sources, nonsteroidal anti-inflammatory medications, and steroid injections.[6,89,92] Resistant cases require surgical intervention, neurolysis, or nerve resection.

In athletes, the *saphenous nerve* can be compressed within the adductor canal (Hunter's or subsartorial canal) or where it exits the fascia during strong contraction of the surrounding musculature, such as may occur with knee extensions or squats.[89,93,94] Hemler et al.[95] reported saphenous nerve entrapment caused by pes anserine bursitis. Entrapment of the saphenous nerve causes medial knee pain, dysesthesia, and hypesthesia in the distal distribution of the nerve. Relief is usually obtained with nonoperative measures, but surgical exploration and neurolysis may be necessary.[89,93]

Sural nerve entrapment can occur anywhere along the course of the nerve. It is most often seen in runners.[89,96,97] Recurrent ankle sprains could lead to fibrosis and subsequent nerve entrapment.[98] Patients complain of shooting pain and paresthesias along the lateral border of the foot, sometimes extending proximally to just behind the lateral malleolus and up the posterior lateral aspect of the lower leg. Nonoperative treatment usually is successful.[89,96,97] Several cases of sural nerve entrapment have been described in athletes who sustained avulsion fractures of the base of the fifth metatarsal bone.[99] In these cases, persisting symptoms and nonunion of the fracture made surgical excision of the nonunited fragment and a neurolysis of the sural nerve necessary.

Only a few cases of *common peroneal nerve entrapment* in runners have been reported.[100,101] Leach et al.[102] have reported the cases of eight athletes (seven runners and one soccer player) with common peroneal nerve entrapment. In all reported patients, running induced pain and numbness. Examination after running confirmed muscle weakness and a positive Tinel's test where the nerve winds around the fibular neck. Because of the failure of varying nonoperative treatments, all of the patients were treated surgically by neurolysis of the peroneal nerve as it travels under the sharp fibrous edge of the peroneus longus muscle origin. Leach et al.[102] reported that seven of eight operated athletes returned to their previous level of activity without any further symptoms.

Superficial peroneal nerve entrapment occurs most commonly in runners but also can be seen in soccer, hockey, and tennis players, bodybuilders, and dancers.[103–107] According to clinical and anatomical studies, the point of entrapment of the nerve is at its exit point from the deep fascia, ~12 cm above the tip of the lateral malleolus. Loss of or disturbances in sensation during exercise over the outer border or distal calf and over the dorsum of the foot, including the second to fourth toes, is a common sign of the entrapment. Occasionally, patients complain only of pain at the junction of the middle and distal third of the leg, with or without local swelling. The symptoms typically worsen with any physical activity, including walking, jogging, running, or squatting. Relief by conservative measures is uncommon. Decompression by local fasciectomy and fasciotomy of the lateral compartment has been reported to give good results.[106,107] One wrestler was successfully operated on by the first author of this book. Daghino et al.[151] describe the fascial entrapment of the superficial peroneal nerve in a 16-year-old female athlete. Magnetic resonance imaging confirmed the diagnosis of this neuropathy. Limited fasciotomy, at the point where the nerve becomes subcutaneous, relieved all symptoms.

Entrapment of the deep peroneal nerve (syndrome of the anterior tarsal tunnel) has been described in runners, soccer players, skiers, and dancers.[89,96,97] Patients frequently have a history of recurrent ankle sprains or previous trauma. Tight, high-heeled shoes or ski boots have also been implicated as inciting factors.[108,109] An osteophyte on the dorsum of the talus or of the intermetatarseum at the tarsometatarsal joint can also press on the nerve.[110] Baxter et al.[89,96,97,110] described this entrapment in joggers who tie keys under the tongues of their running shoes and in athletes who do sit-ups with their feet hooked under a metal bar. The patients complained of dorsal foot pain, numbness, and paresthesias over the first web space. The pain usually occurs during athletic activities. Most patients will respond well to nonoperative therapy with local steroid injections, alteration of footwear, and orthotic devices.[96,97,111] Occasionally, when these measures fail, a patient may require surgical decompression.

Tarsal tunnel syndrome is an uncommon condition in athletes. It has been described in runners, ballet dancers, and basketball players.[112–116] The most common etiology is alteration of the normal spatial relationships stemming from space-occupying lesions such as lipomas, ganglion cysts, neurilemomas, nerofibromas, varicose veins, and enlarged venous plexus. Other causes have included severe pronation of the hindfoot, chronic flexor tenosynovitis, posttraumatic scarring, and inflammatory collagen vascular disease.

According to the literature, many cases are idiopathic. Athletes usually suffer burning, sharp pain at the medial malleolus radiating into the sole of the foot, the heel, and sometimes the calf. They may notice numbness and burning paresthesias on the plantar aspect of the foot and in the toes that radiate up the calf. Initially, symptoms may be intermittent but become constant over time. The symptoms are accentuated by prolonged standing, walking, and especially running.

Treatment should be directed toward identifying and correcting the etiology of the syndrome. Nonoperative treatment of the athlete with tarsal tunnel syndrome includes rest, nonsteroidal anti-inflammatory medication, local steroid injection, flexibility exercises, well-fitting shoes, and custom-made foot orthotics to help control abnormal mechanics.[112–116] Failure of nonoperative treatment necessitates surgical exploration and decompression of the nerve.

One of the most commonly overlooked causes of chronic heel pain in athletes is entrapment of the first branch of the lateral plantar nerve (nerve to the abductor digiti quinti muscle). Although runners and joggers account for the overwhelming majority of cases, this entrapment has been reported in athletes who participate in soccer, dance, tennis, and track and field events.[97,117–119] Entrapment occurs between the heavy, deep fascia of the abductor hallucis muscle and the medial caudal margin of the medial head of the quadratus plantae muscle.

Athletes complain of chronic heel pain that intensifies with walking and especially running. Tenderness over the course of the nerve, maximal in the area of entrapment, is a characteristic and pathognomonic finding. In plantar fasciitis, tenderness is localized at the calcaneal origin of the plantar fascia. Infrequently, the patient may have paresthesias along the course of the nerve.

Park and DelToro[152] described a case of an isolated neuropathy of the first branch of the lateral plantar nerve proven with electrodiagnosis. Treatment is similar to that given for other forms of heel pain — rest, nonsteroidal anti-inflammatory medication, heel cups, stretching programs, and, occasionally, local steroid injections.[97,117–119] If 6 to 12 months of nonoperative therapy fail to relieve the symptoms, and other possible causes of heel pain have been ruled out, surgical intervention is indicated.[126]

Medial plantar nerve entrapment, or medial plantar nerve compression syndrome ("jogger's foot"), occurs in the region of the Master Knot of Henry.[97,110,120] The patient, usually a middle-aged jogger, complains of aching or shooting pain in the medial aspect of the arch during running.[125] Most characteristically, the onset of pain is associated with the use of a new arch support. Physical examination will find point tenderness of the plantar aspect of the medial arch in the region of the navicular tuberosity. The pain can be reproduced by everting the heel or having the patient stand on the ball of the foot. Park and Del Toro[153] described a method of nerve conduction technique for study of the medial plantar nerve. Differentiation from posterior tibial tendinitis should be considered. Nonoperative treatment usually is successful.[97,110,120] At surgery, the area of maximum tenderness should be addressed by releasing the fascia over the nerve in the affected zone.[97]

Interdigital neuromas (metatarsalgia) are not uncommon in athletes, especially runners and dancers.[8,22,89,96,97,110,154] Patients usually complain of plantar or forefoot pain associated with sprints or long-distance running. The pain is described as burning or sharp, frequently radiating to the toes. Patients might also notice numbness or tingling in the affected toes. They will often tell of many shoe changes in an attempt to seek relief. Typically, the pain is relieved by rest, removal of the shoes, and massage of the forefoot. A variety of metatarsal pads and orthotic devices have been suggested, but they are usually uncomfortable and rejected by athletes. A small percentage of interdigital neuromas respond to local steroid injections. Most require surgical excision of the neuroma.[8,22,89,96,97,110]

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Glossary

Abduction Displacing from the body; shifting away
Aberrant Having an unusual course
Accessory Additional formation
Accuracy Conformity to truth or to a standard or model; exactness
Afferent nerve Nerve that transmits impulses from the periphery to the central nervous system
Amplify To increase
Amyotrophy Pertains to muscular atrophy (lack of muscular tone)
Antidromic Propagation of an impulse along an axon in the opposite direction from normal
Aponeurosis A flat fibrous sheet of connective tissue that serves to attach muscle to bone or other tissues
Axon Process of a neuron that conducts impulses away from the cell body
Axonal Pertaining to axon
Axonotmesis Nerve injury damaging nerve tissue without severing nerve sheath
Caudal Toward the tail, inferior, or posterior
Cheiralgia From Greek, meaning pain in the arm or hand
Compound muscle action potential (CMAP) Potential resulting from stimulation of a nerve; recorded from the muscle that is innervated by the stimulated nerve
Conduction time Elapsed time for a nerve impulse to travel between two points
Cranial Toward the head or superior
Crura A pair of elongated or diverging masses
Crural Pertaining to the leg or thigh; femoral
Demyelination Loss of myelin sheath
Denervation Removal of a nerve from its supplied muscle
Dysfunction Abnormal, inadequate, or impaired action of an organ or part
Efferent nerve Nerve that transmits impulses from a nerve center to the periphery
Electrode Medium used between an electric conductor and the object to which the electric energy is applied
Electromyography (EMG) Method of recording electrical activities generated in muscle
EMNG Electromyoneurography; electromyography and electroneurography
End Plate Noise Normal spontaneous electrical activity associated with end plate spikes
Evoked potential Muscle or nerve potential evoked by stimulation
Fascia Fibrous membrane
Fasciculation Spontaneous muscle activity, contraction of muscle fasciculi
Fasciculus (fasciculi (pl.)**)** Muscle fiber group
Fibrillation Spontaneous muscle activity, contraction of muscle fibers
Flaccid Relaxed, flabby, having defective or absent muscular tone, reflexes; a reaction to nerve damage, usually lower motor neurons of the spinal cord
Fossa A furrow or shallow depression
F-wave Response of anterior horn cells stimulated by antidromic conducting impulses derived from peripheral nerve stimulation
Galvanometer An instrument that measures current by electromagnetic action
Ganglion Cystic tumors developing on a tendon, capsule, ligament, or aponeurosis

Giant motor unit Two to four times greater muscle action potential than normal; higher frequency of driving; sign of spacious and temporal compensation; sign of remaining nerve fibers sprouting after injury to innervate a larger group of muscle fibers

Graphanesthesia Inability to recognize figures traced on the skin

Heteresthesia Variation in degree of sensory response to cutaneous stimuli

Hoffman's sign Presence of this sign is shown by flicking the nail of the second, third, or fourth fingers and observing flexion, which indicates hyperactive tendon reflexes

H-reflex Monosynaptic reflex usually obtained from human soleus muscle or forearm flexor muscle

Hyperabduction Extreme displacement from the body; extreme shifting away

Hyperesthesia Increased sensitivity to sensory stimuli such as pain or touch

Hypesthesia Lessened sensibility to touch

Hypodermal needle electrode Electrode in the form of a needle that is placed into the muscle through the skin

Hypoesthesia Dulled sensitivity to touch

Idiopathic Unknown cause; of a disease, for example

Innervative To stimulate a part, as the nerve supply of an organ

Insertional muscle activity Electromyographic registration of muscle activity during insertion of the hypodermic electrode into the muscle

Lower motor neuron Motor nerve cell that innervates the skeletal muscles

Meralgia From Greek, meaning pain in the region of the thigh

Mixed nerve potential Nerve potential obtained by stimulation of sensory and motor fibers; sensory nerve fibers are usually stimulated orthodromically and motor nerve fibers antidromically

Motoneuron Motor nerve cell

Motor nerve conduction velocity (MNCV) Conduction velocity of motor nerve fibers

Motor nerve fiber Axon of the motor nerve cell

Motor unit Motor neuron and muscle fibers that its branches innervate

Motor unit action potential Electromyographic registration of single motor unit activity; each action potential has parameters such as amplitude, duration, frequency of driving, and shape

Muscle action potentials Electromyographic registration of voluntary muscle activity as a result of synchronous activation of single motor units, biphasic, triphasic, or polyphasic if there are more than three phases; polyphasic muscle action potentials are signs of asynchronous activation of muscle fibers.

Myelin sheath Lipoproteinaceous envelop around nerve fibers

Myocenic Relating to the origin of muscle cells

Myocenic potentials Related to muscle activity

Myokimia Hyperkinesis that has very small amplitude; usually benign

Myositis, polymiositis Myopathy with inflammatory and immunological features

Myotonia Delayed relaxation of a muscle after initial contraction

Neoplasm A new and abnormal formation of tissue, as a tumor or growth serving no useful function

Nerve conduction velocity Neurophysiological analysis of the peripheral motor or sensory nerve fibers

Nerve fiber An axon with its sheath(s)

Neuralgia Severe sharp pain along the course of a nerve; neurodynia

Neuraxis The cerebrospinal axis

Neurectasia Surgical nerve stretching

Neurexeresis Ripping or tearing out of a nerve to relieve neuralgia

Neuritis Inflammation of a nerve or nerves, usually associated with a degenerative process

Neuroanastomosis Surgical attachment of one end of a severed nerve to another end
Neurocenic Relating to the origin of nerves
Neurolysis Loosening of adhesions surrounding a nerve, disintegration of nerve tissue
Neuroma General term for a tumor of nerve origin
Neuromuscular junction Connection between terminal part of the motoneuron axon and muscle fiber
Neuromyotonia Continuous muscle fiber activity
Neuron Nerve cell consisting of the cell body and its processes, dendrites, and one long axon
Neuropraxia The condition where trauma has led to the loss of nerve conduction despite maintaining anatomical continuity
Neurotmesis Nerve injury resulting in complete loss of function, despite little apparent damage anatomically
Nociceptive Pertaining to painful stimuli
Objective Perceptible to other individuals; can be measured, seen, heard, or felt
Obturator Structure that closes an opening
Orthodromic Propagation of an impulse along an axon in the normal direction
Paralysis Temporary suspension or permanent loss of sensation or voluntary motion
Paresthesia Sensation of numbness, prickling, or tingling; heightened sensitivity
Piriform Having the shape of a pear
Polyneuritis A kind of peripheral neuropathy
Positive Sharp Waves Electrical activity that indicates denervated muscle cells; monophasic
Predictive value The ability of a statistical test to accurately identify individuals with and without a certain trait (i.e., true positives and true negatives)
Probability The ratio that expresses the likelihood of the occurrence of a specific event
Receptor Sensory nerve endings in the skin, deep tissues, viscera, and special sense organs
Recording electrode Surface or needle electrode that picks up muscle or nerve signals
Reflex An involuntary response to a stimulus
Reinnervation Regrowth of the nerve fibers spontaneously or after anastamosis
Reproducible An event or result that can be produced repetitively
Retinaculum Fibrous strap, layer of connective tissue typically holding tendons down to bone
Retroperitoneal Under or behind the peritoneal membrane
Saltatory conduction Leaping conduction of nerve impulse along myelinated nerve fibers
Saphenous From Arabic, meaning hidden
Sensitivity The statistical ability of a test to identify individuals with a disorder (positives) from a population
Sensory nerve conduction velocity (SNCV) Conduction velocity of sensory nerve fibers
Sensory nerve evoked potential (SNEP) Potential stimulated by a nerve impulse; normal is a triphasic pattern that changes to polyphasic when demyelination occurs
Sensory nerve fiber Axon of the sensory nerve cell
Sign Any objective evidence or manifestation of an illness or a disordered function of a body indicating pathology or disease
Somatosensory potentials Obtained by stimulation of superficial or deep receptors and then surface recording the evoked potentials over the cerebral cortex region
Specificity The statistical ability of a test to identify those without the disorder (negatives) from a population
Spasticity Increased tone or contractions of muscles causing stiff or awkward movements; related to upper neuron lesions
Spontaneous muscle activity Sign of the pathological condition, registered electromyographically when muscle is relaxed
Stimulation electrode Surface or needle electrode used for stimulation of nerve fibers or skin receptors

Surface electrode Electrode placed on the surface of an object, usually shaped as a plate

Symptom Any perceptible change in the body or its functions that indicates disease; may be cardinal, subjective, or constitutional

Syndrome A group of signs and symptoms that collectively characterize or indicate a particular disease or abnormal condition

Terminal latency (TL) Time, in milliseconds, for a nerve impulse to travel from distal nerve stimulation to the muscle; also measured in centimeters as the distance from the distal nerve stimulation electrode to the recording electrode that is over or in the muscle

Tone Normal tension or responsiveness of tissues to passive stretch or elongation (tonicity: hypo- or hyper-)

Transection A cutting made across a long axis; a cross-section

Transposition A transfer of a structure from one position to another

Upper motor neuron Motor nerve cell that is located in the cerebral cortex and sends impulses to lower motor neurons

Voluntary muscle activity Electromyographic registration of muscle activity during its voluntary contraction

Wallerian degeneration Nerve fiber degeneration proximal to the zone of injury (i.e., when severed from its cell body and innervated muscle)

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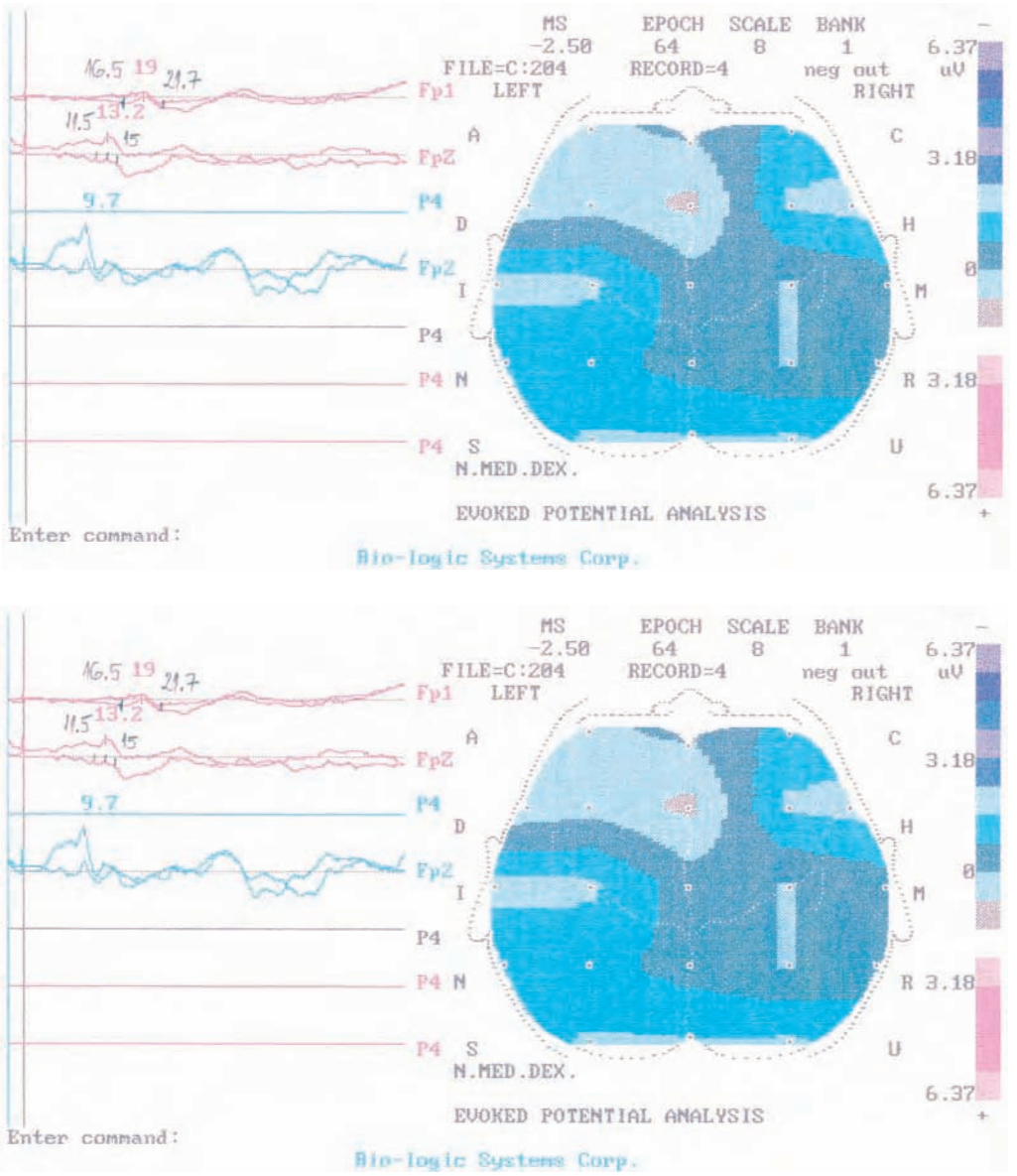
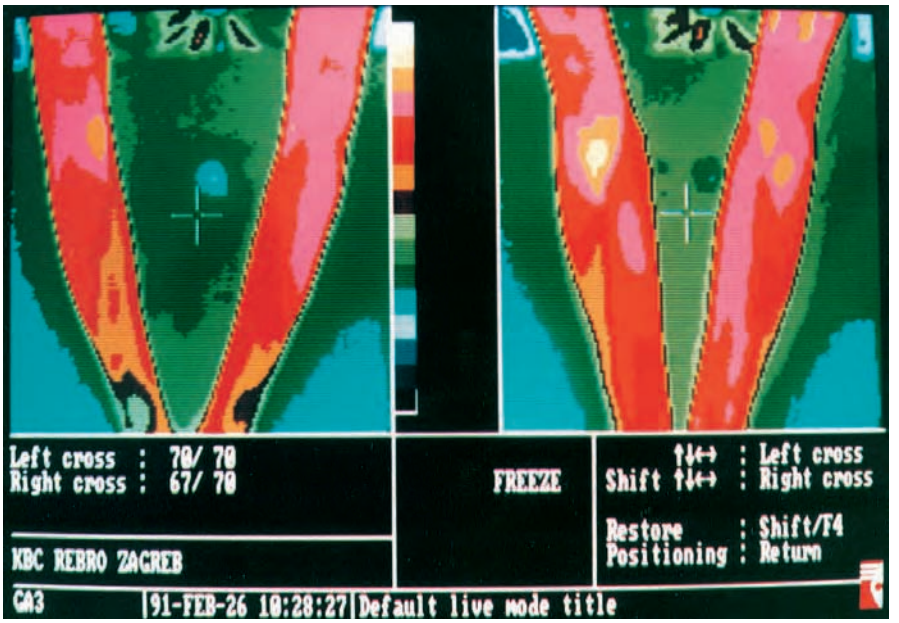
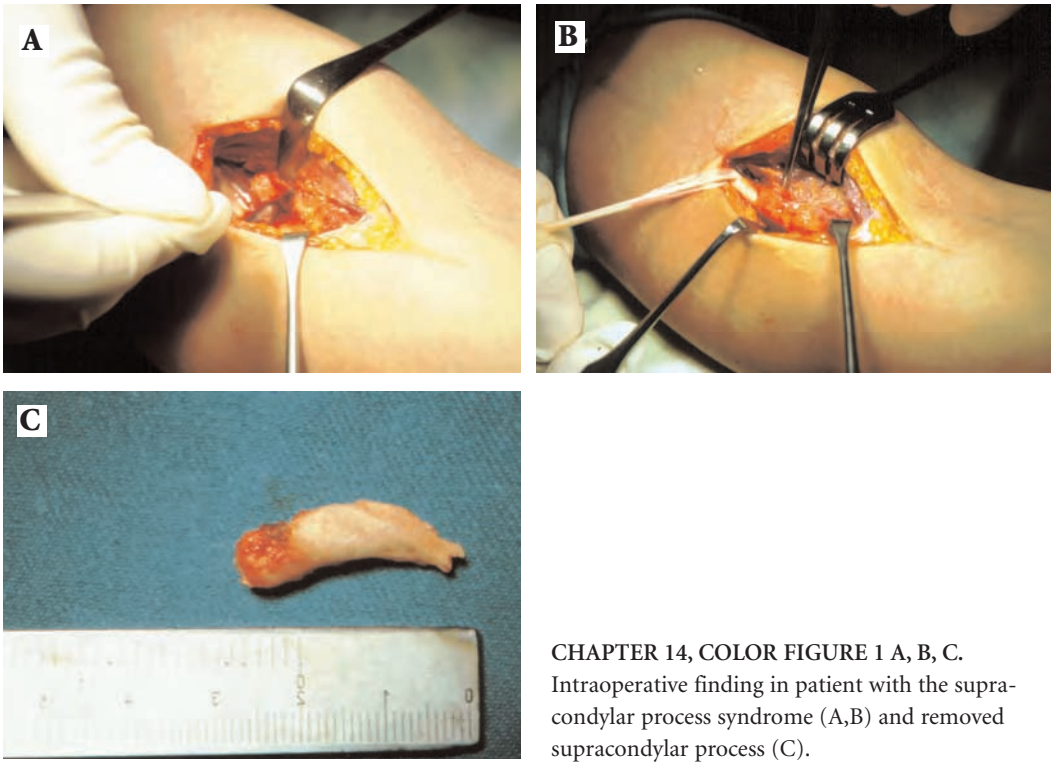


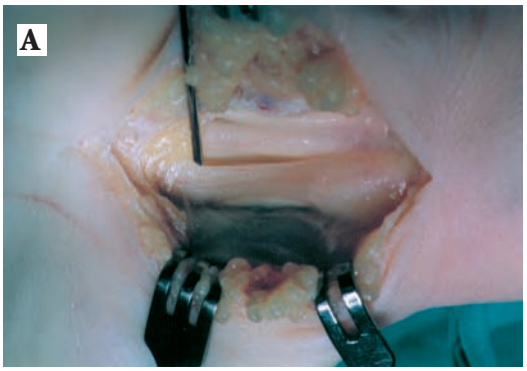
CHAPTER 2, COLOR FIGURE 1. Two examples of median nerve somatosensory-evoked potential.

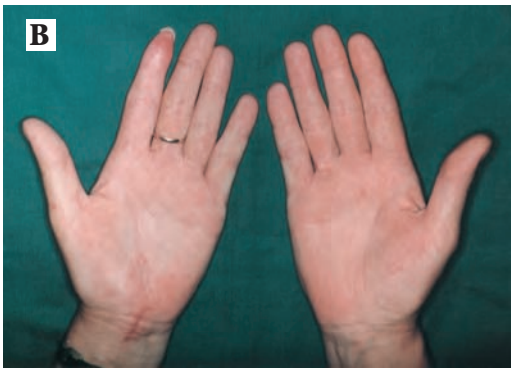


CHAPTER 10, COLOR FIGURE 1. Telethermography shows an increase in temperature in the region innervated by the lateral cutaneous nerve of the affected forearm.



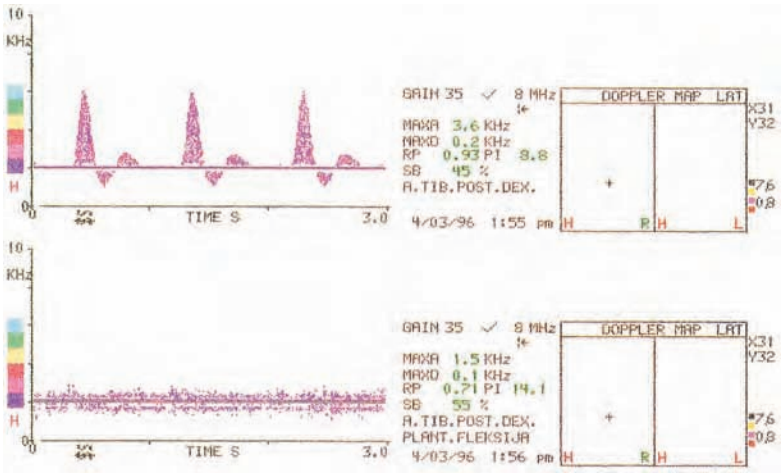
CHAPTER 14, COLOR FIGURE 1 A, B, C. Intraoperative finding in patient with the supracondylar process syndrome (A,B) and removed supracondylar process (C).



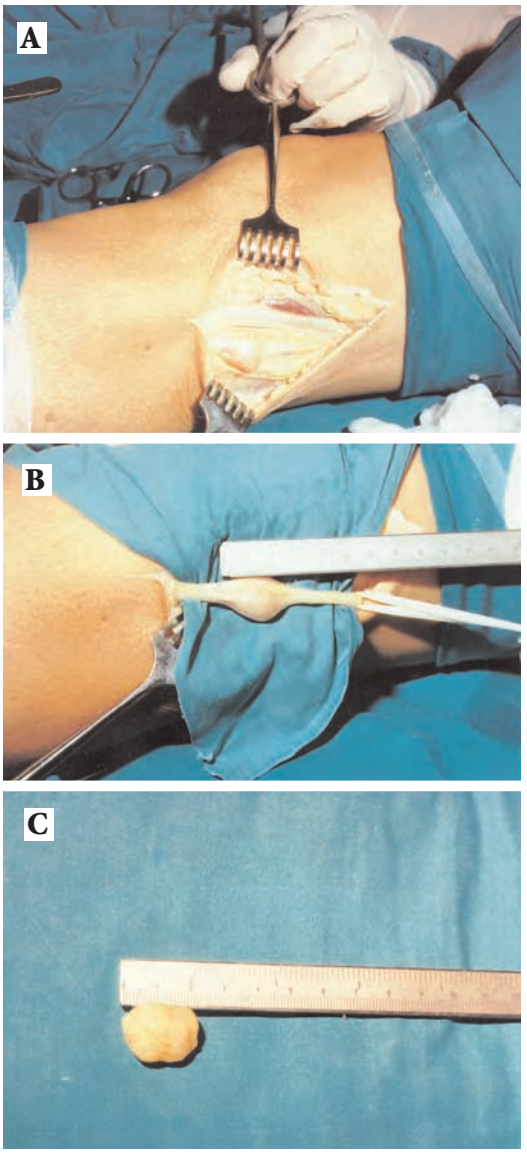


CHAPTER 22, COLOR FIGURE 1 A, B.
Hands of a female patient with defect on the index finger resulting from median nerve compression in the carpal tunnel. Intraoperative finding of significant median nerve compression (A). Postoperative finding and visible healing of index finger defect (B).

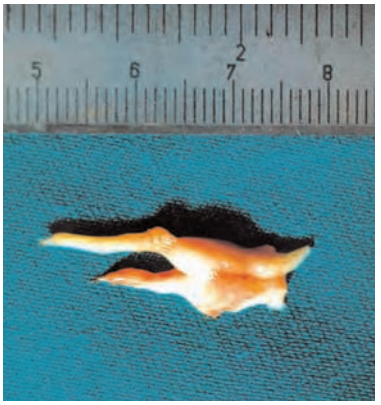
CHAPTER 23, COLOR FIGURE 1. Thermographic "amputation" of the ulnar sides of the fourth and fifth fingers in a patient with ulnar tunnel syndrome (syndrome of Guyon's canal).



CHAPTER 45, COLOR FIGURE 1. Doppler waveforms of the posterior tibial artery with the leg in the neutral position and then with knee extension and active plantar flexion of the foot.



CHAPTER 46, COLOR FIGURE 1 A,B,C. Intraoperative finding in patient with the peroneal tunnel syndrome (A,B), and extirpated ganglion (C).



CHAPTER 53, COLOR FIGURE 1. Morton's neuroma or neuroma plantaris

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