Chapter 9

PERIPHERAL NERVE INJURIES

MICHAEL D. ROBINSON, M.D.[*]; AND PHILLIP R. BRYANT, D.O.[†]

[*]*Formerly, Major, Medical Corps, U.S. Army, Director of Research, Physical Medicine and Rehabilitation, Walter Reed Army Medical Center, Washington, DC; and Assistant Professor of Neurology, Department of Neurology, Division of Physical Medicine and Rehabilitation, Uniformed Services University of the Health Sciences, Bethesda, Maryland; currently, Assistant Clinical Professor, Department of Rehabilitation Medicine, Mt. Sinai School of Medicine, New York, New York 10029*

[†]*Formerly, Major, Medical Corps, U.S. Army, Physical Medicine and Rehabilitation, Walter Reed Army Medical Center, Washington, DC; currently, Professor and Chairman, Department of Physical Medicine and Rehabilitation, East Carolina University School of Medicine, Greenville, North Carolina 27858-4354*

INTRODUCTION

Nerve injuries are likely to have occurred since the most ancient of recorded battles. Blunt and lacerating trauma to nerves was almost certainly inflicted in alarming numbers in early hand-to-hand combat. It is probable that upper extremity peripheral nerves, including the brachial plexus, were common sites of injury in these ancient conflicts. Mechanized warfare of the current century has also resulted in nerve injuries ranging from mild, isolated nerve trauma to severe injury at multiple sites. Many nerve injuries occur directly from blunt trauma or lacerations, while others are secondary complications due to fractures or compartment syndrome.

Combat-related nerve trauma was well documented in World Wars I and II, as well as in Korea and Vietnam. The experience of neurosurgeons in World War I was recorded by Pollock and Davis.[1] Their text, which contains information on peripheral nerve anatomy, as well as technical methods of nerve exposure, mobilization, transplantation, and suturing, was a popular reference for general surgeons and neurosurgeons involved in World War II.[2]

Woodhall et al[2] note that the most important lesson learned from the World War II experience in managing peripheral nerve injures was application of early nerve repair. Delay was abandoned in favor of prompt exploration within 21 to 90 days after nerve insult in all nerve injuries where any doubt existed regarding spontaneous regeneration. The World War II experience taught neurosurgeons that concomitant soft tissue, bone, and vascular injuries critically affected the timing and techniques of nerve repair.

Electrodiagnosis was in its infancy during World War II. Although it was recognized that it had potential in helping to identify the distribution and severity of nerve lesions and in detecting changes associated with regeneration, electrodiagnosis was only marginally available to physicians during World War II. In addition, at that time, the machines and techniques for assessing nerve integrity were unsophisticated and limited in their applications. A substantial number of soldiers in the Persian Gulf War who were treated by military physical medicine and rehabilitation services were also noted to have sustained peripheral nerve injuries.[3] Many of these soldiers suffered profound and sometimes multiple nerve damage. Although some recorded nerve injuries steadily improved with time, some were so severe that reinnervation activity and functional return were markedly compromised or nonexistent.

Proper management of peripheral nerve injuries requires an understanding of the distribution and function of nerves, the mechanisms of nerve injury, and an appreciation of the regeneration process. It is imperative to astutely examine the patient to obtain the clinical clues necessary for accurate diagnosis. Detection and localization of nerve injury can be complicated in the presence of multiple additional trauma such as fractures or vascular compromise. Carefully planned and performed electrodiagnostic studies based on the history and examination offer additional diagnostic and prognostic information.

Among the most important aspects of care is proper rehabilitation of nerve injuries, regardless of whether surgical intervention is required. Rehabilitation of nerve injuries invariably requires an interdisciplinary approach in order to obtain optimal nerve recovery and function. A physiatrist, neurosurgeon, neurologist, orthopedic surgeon, or plastic surgeon, as well as physical therapist, occupational therapist, and vocational specialist, are among the team members typically involved in the rehabilitative effort.

EPIDEMIOLOGY

Registries of peripheral nerve injuries compiled during the World Wars have shed important light on the severity of injuries, distribution of nerves injured, and associated injuries. Out of nearly 175,000 casualties sustained by American Expeditionary Forces in World War I, 3,129 injuries—nearly 2% of total battle injuries—were documented nerve injuries. Statistical analyses were performed on 2,390 of these, focusing on the nerves most commonly injured. Injuries severe enough to warrant surgical exploration accounted for approximately 45%. A majority, 55%, were felt to be of lesser severity and operative intervention was not undertaken. Of the 1,088 operative cases, 266 were found to be in at least partial continuity. All told, 66% of nerve injuries were partial, that is, they were noted to be anatomically or functionally in continuity. Thirty-four percent were complete injuries (Table 9-1)

requiring surgical coaptation.

Injuries of the sciatic nerve accounted for 23%; radial nerve, 21.5%; peroneal nerve, 17%; median nerve, 11%; tibial nerve, 1.5%; ulnar neuropathies, 21%; and nonspecified, 5% (Table 9-2).[4]

The incidence of peripheral nerve injuries among those injured in combat during World War II has been estimated to be somewhat higher than during the first World War. Spurling and Woodhall[5] suggested 5% to 8% [assuming 500,000 World War II approximately 30,000] to be more likely. Similar to World War I, the percentage of nerve injuries severe enough to warrant surgical exploration was just under 50%, a rate consistent in both the Mediterranean and European theaters. A representative cross section of cases that required surgical exploration when treated in the Mediterranean theater revealed that 40% had complete injuries, 24% had partial injuries, and 36% were in continuity. When combined with those cases not severe enough to require surgical exploration, nearly 80% manifested functional or anatomic continuity. Surgical reconstruction was warranted in only 20%.[5(pp237,238)]

In the European theater, 6,245 peripheral nerve injuries were observed between D-day and V-E day. Again, 2,873 or 46% were severe enough to warrant surgical exploration. Of all nerve injuries, 1,528 or 25% required surgical reconstruction. Nerve injuries in anatomic or functional continuity comprised 75%[5(p248)] (see Table 9-1).

TABLE 9-1

SEVERITY OF NERVE INJURIES

Severity	World War I (%)	World War II* (%)	World War II† (%)
Complete (Explored)	34	20	25
Partial (Not Explored)	55	50	54
Partial (Explored)	11	30	21
Total partial nerve injuries	66	80	75
Percentage of all injuries	2	5-8	NR

NR: not recorded
*Mediterranean Theater, World War II
†European Theater, World War II

TABLE 9-2

PERCENTAGE DISTRIBUTION OF PERIPHERAL NERVE INJURIES

Nerve	World War I	World War II*	Persian Gulf†
Ulnar	21	28	12
Radial	21.5	20	11
Median	11	15	18
Musculocutaneous	NR	0.75	NR
Axillary	NR	0.75	NR
Brachial Plexus	NR	8.5	10
Sciatic	23	8	7
Peroneal	17	11	16
Tibial	1.5	3	8
Femoral	NR	2.5	2
Saphenous	NR	1	NR
L/S Plexus	NR	NR	10
Cranial	NR	NR	3
Not Specified	5	1.8	3

NR: not recorded
*Mediterranean Theater, World War II
†Physical Medicine Service, Persian Gulf War

In the Mediterranean theater, injuries to the ulnar nerve were most common, accounting for 28%; radial nerve, 20%; median nerve, 15%; peroneal nerve, 11%; sciatic nerve, 8%; brachial plexopathies, 8.5%; tibial nerve, 3%; femoral nerve, 2.5%; saphenous nerve, 1%; musculocutaneous nerve and axillary nerves each, 0.75%; and nonspecified, 1.8% (see Table 9-2). Multiple nerve injuries were common, with an incidence ranging from 8% to 20%.[5] The upper extremity manifested multiple nerve injuries more commonly than the lower extremity.

Data from the Mediterranean theater revealed compound fractures to be associated with peripheral nerve injuries in over one third of the cases. Humeral fractures were associated with radial, median, or ulnar nerve injuries 40% of the time. A similar percentage of forearm fractures was related to nerve injuries. Assessment of all nerve injuries requiring surgical correction also showed a 33% rate of association with fractures (Table 9-3).

Vascular injuries not severe enough to cause gangrene were associated with 21% of all nerve inju-

TABLE 9-3

RATES OF CONCOMMITANT INJURIES WITH PERIPHERAL NERVE INJURIES IN WORLD WAR II*

Injury	Percentage
Compound fractures	33
Humeral fractures	40
Radial/Ulnar fractures	39
Vascular injuries	21

*Mediterranean Theater, World War II

ries sustained in the Mediterranean theater (see Table 9-3). A lower rate of 8% was documented when casualties undergoing surgical intervention from all theaters and the zone of the interior were examined.

In the 1990 Persian Gulf War, a substantial number of soldiers, who were treated by Physical Medicine and Rehabilitation services, sustained peripheral nerve injuries. Nerve injuries were associated with 68% of penetrating wounds, 67% of amputations, and 58% of fractures. The high percentages likely reflect the referral pattern of the population seen by the Physical Medicine and Rehabilitation service. Electrodiagnostic evaluation was the primary reason for referral in 40%.

In the Persian Gulf War, median nerve injuries were most common, comprising 18% of injuries. Peroneal nerve injuries followed at 16%; ulnar nerve injuries at 12%; radial nerve injuries at 11%; brachial plexopathies at 10%; lumbar plexopathies at 10%; tibial nerve injuries at 8%; sciatic neuropathies at 7%; femoral nerve injuries at 2%; and cranial neuropathies at 3%[3] (see Table 9-2).

While statistics conveying the magnitude of combat-related nerve injuries are essential, one must also consider that even larger numbers of nerve injuries and neuropathies treated during the world wars were not sustained during battle. When radiculopathies are included with all other peripheral nerve injuries and nerve-associated pain disorders, over 34,000 admissions were logged during World War II that were not battle related.[6] The cumulative loss of manpower and productivity because of such disorders likely impacted on effectiveness and readiness of the fighting force. Thus, a sound understanding of both traumatic and atraumatic nerve injuries is essential for comprehensive management to be undertaken.

PERIPHERAL NERVE ANATOMY AND PHYSIOLOGY

Neuroanatomy

The neuron is the fundamental anatomic unit of the peripheral nervous system (Figure 9-1). Each neuron consists of a cell body (soma), dendrites, and an axon. The cell body contains the nucleus, numerous mitochondria, and essential apparatus for the production of structural proteins. Neurotubules and microfilaments are transported distally and provide the edificial construct that imparts rigidity to the relatively fluid axon. Neurotransmitters are also produced in the cell body and are transported along the axon to synaptic boutons.

Dendrites are branching appendages emanating from the cell body. The multiple phalanges and small spines provide an immense surface area with which to interface with and receive stimuli from other neurons. The chemical signal imparted to the dendrites is transformed into an electrical signal. The membrane potential courses through the cell body, where all the numerous inputs summate, allowing for control of neuronal discharge along the axon.

The axon is an extension of the cell body that arises from the axon hillock. The hillock is devoid of myelin and is likely the summation point for neuronal impulses.[7] Axon length varies from a few millimeters to several feet. Classically, nerve fibers have been divided into unmyelinated and myelinated groups based on the characteristics of axonal ensheathment. The inextricable intercellular relationship between axons and Schwann cells becomes apparent at this level. Developmentally, Schwann cells originate from neural crest cells.[8] The cells develop with the extending neurons. Characteristically, 8 to 15 unmyelinated fibers are circumferentially surrounded by a single Schwann cell, each 200 to 500 μm in length.[7] A column of Schwann cells encases the entire length of the fiber group. The boundaries of individual Schwann cells are difficult to discern due to the myriad interdigitating cytoplasmic phalanges binding the adjacent cells together. Fifty percent to 80% of all nerve fibers are unmyelinated.

Anatomically, myelinated fibers differ from unmyelinated fibers in several ways. One axon is surrounded by one layer of Schwann cells. The cytoplasm of each Schwann cell becomes attenuated and wraps repeatedly around the axonal segment. Ulti-

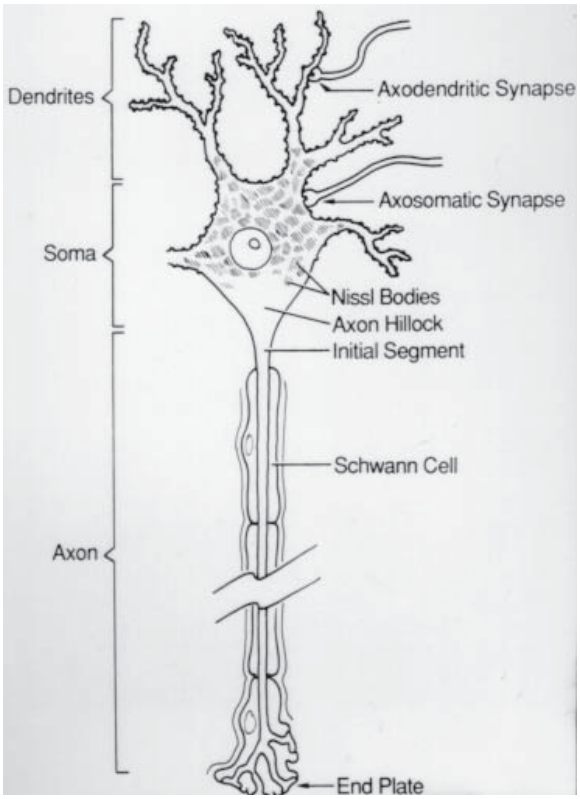

Fig. 9-1. The neuron, including dendrites, soma, and myelinated axon. Adapted with permission from Terzis JK, Smith KL. *The Peripheral Nerve: Structure, Function and Reconstruction.* New York: Raven Press**;** 1990**.**

mately, a multilayered, laminated, lipoprotein sheath is formed. The myelin layer divides the neuron into segments ranging from 0.1 mm to 1.8 mm in length,[9] each forming an internode. These internodes are high resistance, low capacitance insulators that greatly enhance the speed with which an impulse propagates down the length of an axon. The unmyelinated, intersegmental focuses, termed nodes of Ranvier, are highly specialized apparatuses uniquely suited to effect saltatory conduction (Figure 9-2). Sigworth[10] noted that at the nodes, large concentrations of voltage-sensitive sodium ion channels are present. The Schwann cell collar surrounding the edges of the node are rich with mitochondria and may provide the energy mechanism necessary for function of the ionic pumps.[11]

Impulse Propagation

Rapid fluctuations in ionic concentrations traveling along the axolemma predicate neuronal signaling. Embedded in the cell membrane are ion channels, selectively permeable to potassium, sodium, and chloride as well as other ions such as calcium (Ca^{++}) (Figure 9-3). Movement of ions through these channels allows for the establishment of an electrical potential across the cell membrane. A predominance of potassium (K^+) channels allows a relatively large concentration of K^+ to diffuse along its concentration gradient out of the cell. The diffusion of K^+ is held in check by the electrical forces generated by impermeable anions trapped inside the cell, which eventually attract the K^+ back into the cell. An equilibrium between the opposing forces is established. The cell membrane is also permeable to Na^+. The concentration gradient for Na^+ is opposite that noted for K^+. High extracellular concentrations of Na^+ follow the gradient and are attracted into the cell. The anionic forces within the cell also draw Na^+ ions inward. A deluge of Na^+ rushing into the cell is prevented by a relatively lower permeability of the cell to Na^+ ions. How-

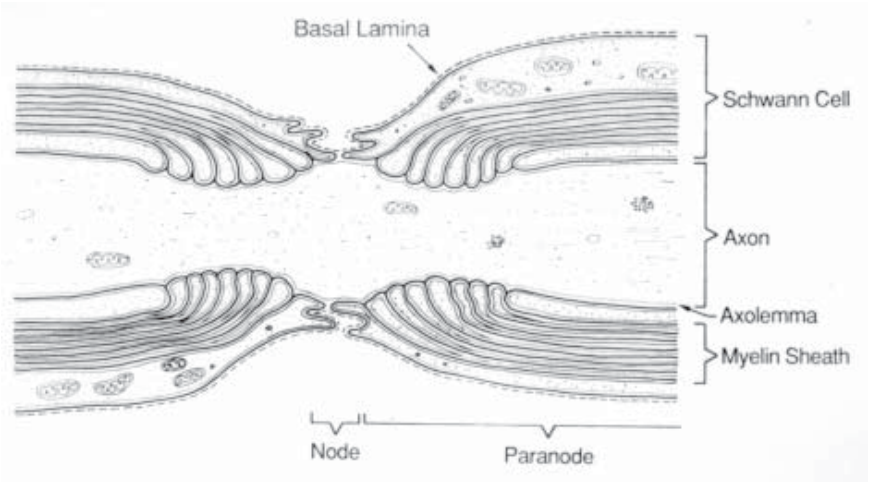

Fig. 9-2. The node of Ranvier. Myelin sheaths form collars at either end of the node, replete with mitochondria, which facilitate impulse transmission. The basement membrane limits ionic diffusion in areas adjacent to the node. Adapted with permission from Terzis JK, Smith KL. *The Peripheral Nerve: Structure. Function and Reconstruction.* New York: Raven Press; 1990.

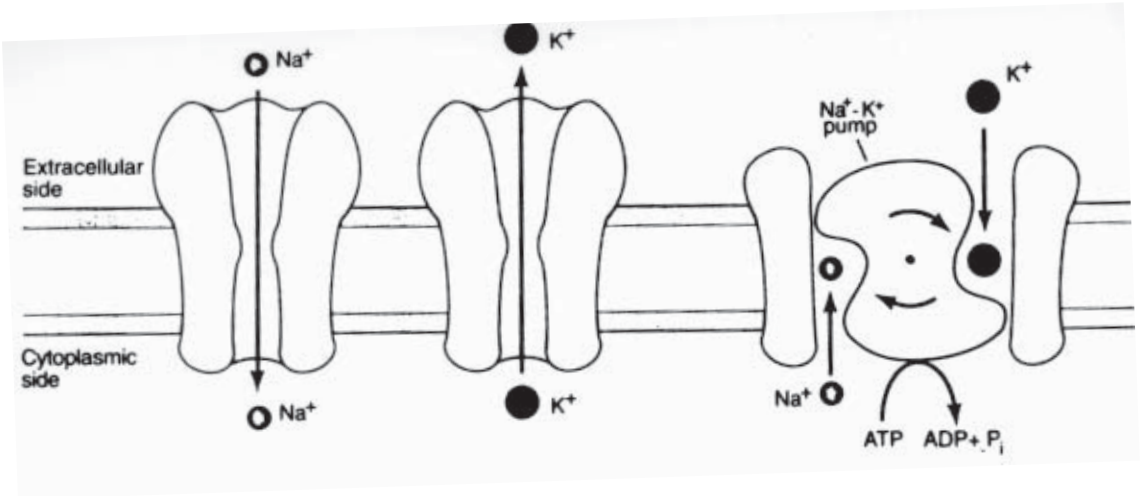

Fig. 9-3. The passive fluxes of Na^+ and K^+ into and out of the cell are balanced by an ATP-dependent Na^+–K^+ pump, which drives Na^+ out of the cell and K^+ into the cell. Adapted with permission from Koester J. Membrane Potential. In: Kandel ER, Schwartz JH, Jessel TM, eds. *Principles of Neural Science.* 3rd ed. Norwalk, Conn: Appleton and Lange; 1991: 87.

ever, as Na^+ flows in, the intracellular fluid becomes less negative and the more freely permeable K^+ is driven out of the cell. A steady state is reached with the electrical gradient across the membrane holding near –60 mV. While the general flux of Na^+ and K^+ are balanced ionically, concentrations must be held in check, otherwise all the intracellular K^+ would be depleted, and the ever-rising intracellular Na^+ would eventually reduce the membrane potential and permanently depolarize the cell. Maintaining the membrane potential requires energy as Na^+ and K^+ are respectively pumped out of and into the cell against their concentration gradients. The Na^+-K^+ ATPase (adenosine triphosphatase) dependent pump extrudes three Na^+ ions from the cell for every two K^+ it brings into the cell.[12] The larger number of ions purged is incompletely offset by the ease with which K^+ diffuses back into the cell. An equilibrium is reached at a resting membrane potential near –70 mV.

Any factor changing the careful balance of ionic flow into or out of the cell will lead to a rapid change in membrane potential. In addition to the nongated ionic channels previously mentioned, the cell membrane also possesses gated ionic channels. While it has not been completely elucidated, that gating mechanism is thought to be positively charged molecules coupled to the lipid matrix that repel cations. Any change in the electrical potential across the membrane will alter the relative strength of the gate (ie, as the inside of the cell becomes less negative, the gate becomes relatively less positive, and its repellent force is diminished).

Tremendous increases in Na^+ permeability occur as the membrane potential becomes less negative (Figure 9-4).The gated ionic Na^+ channels become more permeable when the cell is depolarized by approximately 7mV (ie, to –63mV).[12] Na^+ rushes into the cell down ionic and concentration gradients. As the membrane potential approaches –55mV, the firing level is attained and complete depolarization of the cell occurs. The Na^+ channels quickly close following this rapid depolarization, which is limited to +45mV. Gated K^+ channels then open,

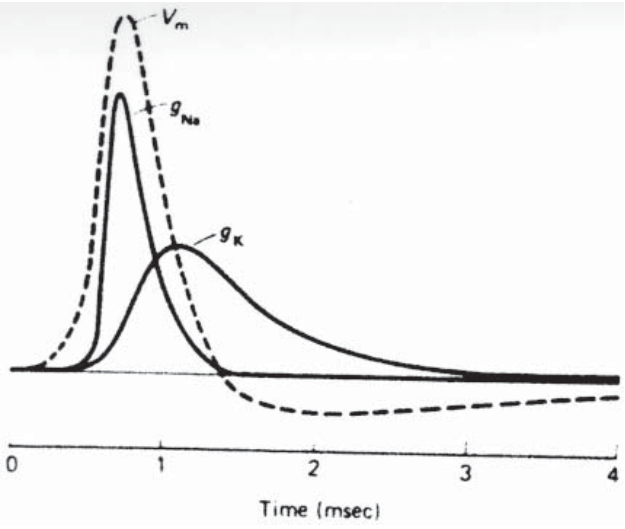

Fig. 9-4. The action potential results from the opening and closing of voltage gaited Na^+ and K^+ channels. Adapted with permission from Koester J. Voltage-Gated ion channels and the generation of the action potential. In: Kandel ER, Schwartz JH, Jessel TM, eds. *Principles of Neural Science.* 3rd ed. Norwalk, Conn: Appleton and Lange; 1991: 110.

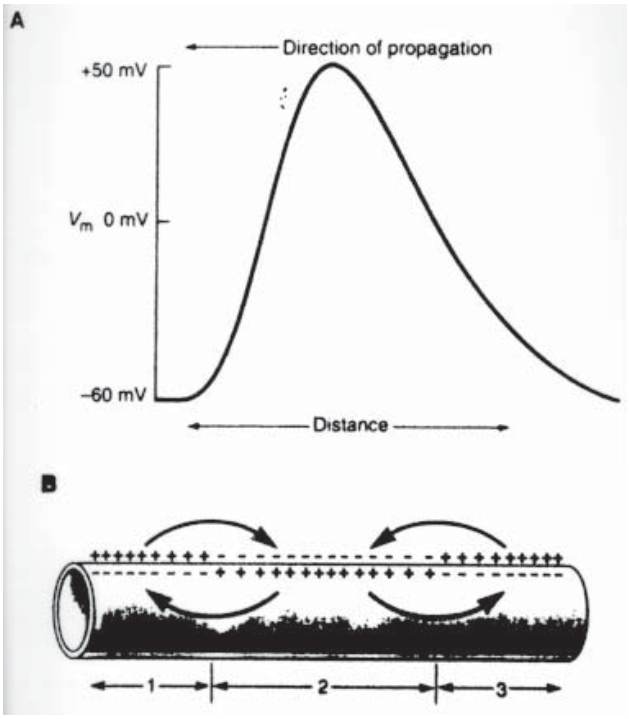

Fig. 9-5. Local changes in polarization (2) change the relative concentrations of ions across adjacent areas of the neuron (1,3). Depolarization will spread in both directions. During normal function however, diffusion of K^+ out of the region, which has already been depolarized (3), balances the local influence and the region does not depolarize or propagate the impulse in a retrograde direction. Adapted with permission from Koester J. Passive Membrane Properties of the Neuron. In: Kandel ER, Schwartz JH, Jessel TM, eds. *Principles of Neural Science.* 3rd ed. Norwalk, Conn: Appleton and Lange; 1991: 100.

allowing K^+ to rush out of the cell, reestablishing the resting polarized membrane potential.

The recorded action potential occurs at one point along the cell membrane (Figure 9-5). To propagate the impulse, depolarization of adjacent portions of the membrane is necessary. As the focus begins to depolarize, it becomes positive in relation to adjacent points along the membrane. The focus acts as an anion sink, "sucking" in the negative charges and causing the adjacent area to become less negative. Eventually, threshold is reached and the adjacent area then becomes the focus as it depolarizes.

The speed and efficiency with which impulses are propagated are radically different between unmyelinated and myelinated fibers. Depolarization proceeds longitudinally along the entire length of the axon in unmyelinated fibers. The segmental myelin sheaths found in myelinated fibers do not allow alteration in the ionic flux to occur. The impulse is propagated by jumping from node of Ranvier to a subsequent node, allowing for very rapid conduction velocities, upward of 70 m/s.

Axonal Transport

The protracted distance between the cell body and the distal reaches of the axon poses unique demands on the neuron. Energy driven systems have developed that maintain constant contact between the soma and the synapse. Three main modes of active transport along the axon have been recognized.

Fast Antegrade Transport

Constant utilization of neurotransmitters at the synaptic cleft and associated loss of cell membrane through pinocytosis requires rapid and constant replenishment. Materials, including neurotransmitter laden vesicles; mitochondria; and membrane components (eg, glycoproteins, glycolipids, lipids), travel along the fast antegrade transport system at a rate of 410 ± 50 mm/day. Ochs and Hollingsworth[13] exhibited the strong dependence of fast transport on oxidative metabolism. Fast transport came to a halt in nerves deprived of oxygen by placing them in a nitrogen environment for no more than 30 minutes.

Slow Antegrade Transport

Slow transport has been delineated into separate constituents, a and b.[14] Slow component a (SCa) is the slower of the two, moving at a rate of 1 to 3 mm/day. Neurofilament triplet proteins and tubulins are the principal materials transported by this system. Slow component b (SCb) travels more rapidly, 3 to 6 mm/day. Proteins such as actin, clathrin, and other microfilaments are carried by SCb. It has been suggested by Wujek and Lasek[15] that SCb is the rate-limiting step in axonal regeneration following nerve injury. The elements provided by SCb form the framework for the growth cone as well as the axon.

Retrograde Transport

Transport of materials from the periphery back to the cell body underlies three main functions: (1) depleted neurotransmitter vesicles are returned to the cell body and restored to full potency; (2) waste materials are cleared from the axonal fringes for degradation; and (3) it has also been postulated recently that trophic substances such as nerve growth factor (NGF) proceed proximally, providing the

soma with information regarding the integrity of the cell. Deprivation of this trophic feedback is felt to be one factor heralding chromatolysis and the subsequent shift to regenerative processes during nerve injury.[16] The rate of retrograde transport varies from 100 to 220 mm/day.[17,18]

Transport Models

While the precise mechanisms of axonal transport have yet to be fully elucidated, several elegant theories have been developed. Taking into account the participation of microtubules, the utilization of ATP, and the need for Ca^{++}, Ochs[19] constructed the transport-filament hypothesis of fast axonal transport. Microtubules form a virtual track system along the length of the axon. A carrier protein binds the material to be transported to the microtubules. In a system reminiscent of the actin-myosin sliding filament complex utilized in muscle contraction, a racheting of the carrier proteins, driven by a Ca^{++}-Mg^{++} ATPase, allows the material to be passed from carrier protein to subsequent carrier protein. Fast antegrade transport employs kinesin as its transport protein, while a form of dynein is used for retrograde transport.[12]

In the past, slow antegrade transport was felt to reflect peristaltic waves inherent to the neuron. Ochs and Brimijan[16] suggest in their unitary hypothesis that the same system envisioned for fast antegrade transport may also be utilized in slow transport. Early detachment of tubulins and other structural proteins from the microfilament track might give the appearance of a slow moving wave.

Architecture of the Nerve

The trinary layers of supporting connective tissues, both individually and collectively, safeguard the neurons from mechanical and biological injury (Figure 9-6).

Fascicular Composition

The structural topography of the nerve trunks imparts a considerable defense against functional loss. Sunderland[20] reflects that at the root level, individual fibers destined to become specific distal branches are inextricably entwined with fibers from other virtual branches (Figure 9-7). As the fascicles extend peripherally, the fibers perpetually interweave, converging and diverging into new and different fascicular assemblies. The rate at which the confluence of fibers changes remains controversial, ranging from 1.5 to 2.5 cm.[7,9] Teleologically, it can be suggested that the plexiform configuration developed to enhance the tensile strength of the nerve trunk and help protect against mechanical deformation. The constantly changing position of individual fibers within the nerve also reduces the likelihood that a focal injury might damage a large

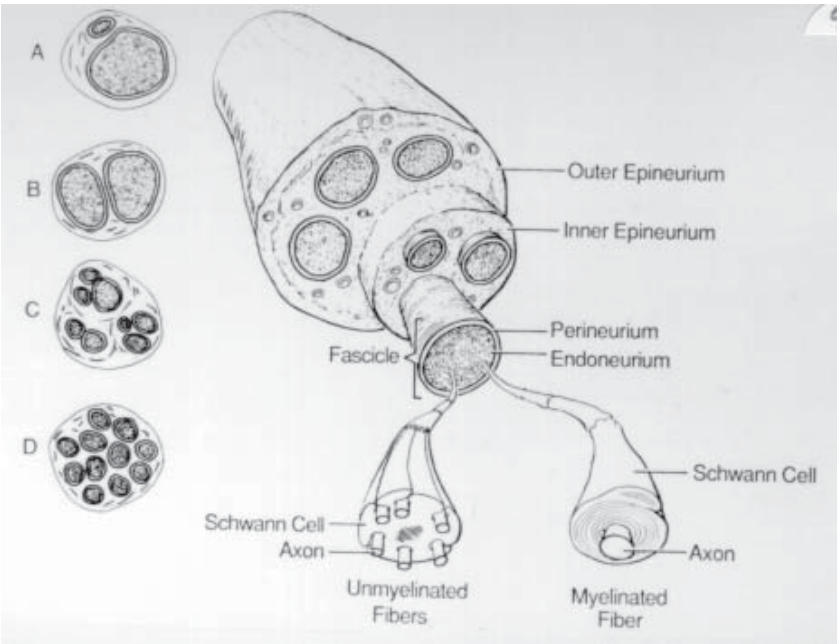

Fig. 9-6. Architecture of the peripheral nerve. Connective tissue elements include the epineurium, perineurium, and endoneurium. A mix of myelinated and unmyelinated fibers are commonly found within the same fascicle. **A**, **B**, **C**, and **D** show cross sections of fascicular composition at different levels. Adapted with permission from Terzis JK, Smith KL. *The Peripheral Nerve: Structure, Function and Reconstruction.* New York: Raven Press; 1990.

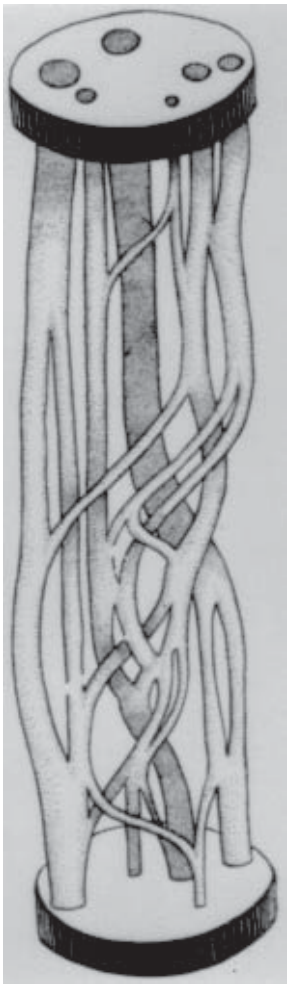

Fig. 9-7. Illustration of the interweaving plexus formations observed in a segment of the musculocutaneous nerve. Adapted with permission from Sunderland S. *Nerve and Nerve Injuries.* Edinburgh, Scotland: Churchill Livingstone; 1978: 32.

number of fibers destined to innervate a specific muscle group or sensory distribution.

Endoneurium. The neuronal complex of axon, Schwann cell, and basement membrane are surrounded by a loose meshwork of collagen fibers, termed the endoneurium. According to Sunderland,[20] the endoneurium imparts some protection to the neuron against stretch injury. Its capacity, however, pales in comparison to that provided by the perineurium.

Perineurium. The perineurium, the investing multiple layers of connective tissue surrounding distinct fascicles, is pivotal in maintaining the integrity and internal milieu of the nerve fibers. Two to five layers of flat, interdigitating cells linked by tight junctions form the blood-nerve barrier.[21] Large proteins, toxins, antigens, and infectious agents are prevented from entering the fascicles. Active transport of essential materials does occur across the blood-nerve barrier. The endoneurial fluid pressure is also maintained by the perineurium. Interdigitating between the lamellae are dense bundles of collagen and elastin fibers.[22] The perineurium is paramount in providing tensile strength and elasticity to the nerve. The response of the nerve to stretch has been examined extensively.[23–26] When a nerve is stretched, the gross undulation of the trunk is initially taken up. Further load leads to straightening of fascicular meandering. At the limits of elastic deformation, the epineurium ruptures. Further tension leads to plastic deformation of the perineurium, all efforts focusing on maintaining the integrity of the fascicles. The events following further loading remain controversial. According to Sunderland and Bradley,[26] the fluid nature of the individual neurons leads to disruption of some fibers prior to disruption of the perineurium. Haftek[24] alternatively suggests that elongation of the perineurium induces a tightening constriction around the fascicles, leading to myelin and eventually axonal injury. Rupture of fibers directly from the increased tension occurs at the perineurial level before the endoneurial level.

Epineurium. The most superficial layer is the epineurium. A loose areolar meshwork of collagen fibers, replete with lipid globules provides fundamental protection against compressive forces. Terzis and Smith divide this stratum into two components. The inner constituent surrounds each fascicle, providing interfasicular support and protection. The outer portion invests the entire nerve trunk.[7] The abundance of epineural tissue varies along the length of the nerve. Areas with larger numbers of fascicles generally possess a greater cross-sectional area of epineurium. For example, 88% of the sciatic nerve at the gluteal level is composed of epineural connective tissue, whereas, the epineurium comprises only 22% of the ulnar nerve at the medial epicondyle.[9] It is also noted that greater amounts of epineurium can be found at levels where nerves cross joints.

Vascular System of the Nerve

The peripheral nervous system is endowed with a multitiered vascular complex that preserves blood flow to the neurons even in the face of mechanical

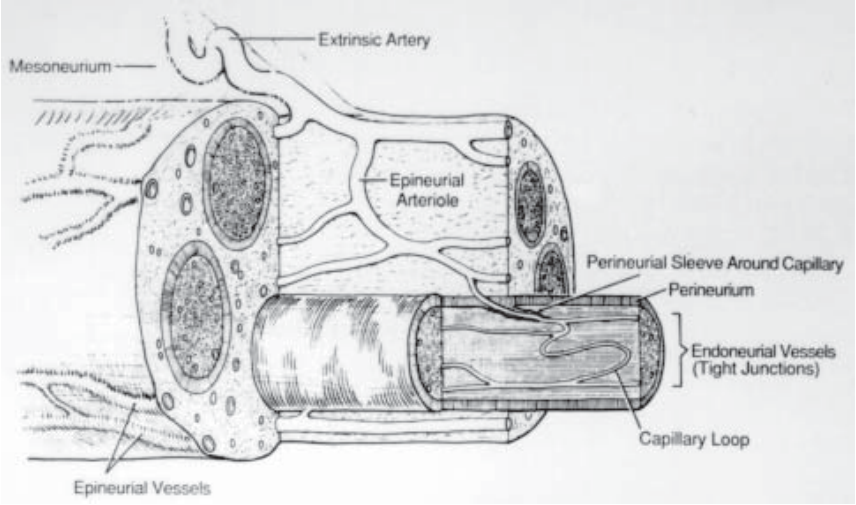

Fig. 9-8. The vascular system of the peripheral nerve. Vessels from periosteum and muscles pierce the epineurium. Branches enter the fascicles, feeding longitudinally aligned vessels beneath the perineurium. Vessels connecting the perineurial and endoneurial plexi follow an oblique path. Endoneurial capillaries form double U–loops, which permit rapid shift in blood flow to accommodate changes in position, pressure, temperature, or injury. Adapted with permission from Terzis JK, Smith KL. *The Peripheral Nerve: Structure, Function and Reconstruction.* New York: Raven Press; 1990.

disturbances. As previously discussed, a constant source of oxygen is necessary to sustain integral energy dependent systems such as axonal transport and impulse propagation. Over the past two decades, the salient works of Lundborg have expanded the current understanding of the neural vascular system.[25,27–29] Vascularization of peripheral nerves can be divided into extrinsic and intrinsic systems (Figure 9-8).

Extrinsic System

The extrinsic system incorporates branches emanating from local arteries in addition to muscular and periosteal vessels.[29] The myriad inputs permit continuous blood flow during movement (eg, translation across moving joints). The tortuosity of the vessels also allows for position changes as slack is taken up when the nerve becomes relatively taut. This rich blood supply becomes increasingly important during mobilization and reparative surgeries. Several studies have shown that significant transposition of several centimeters will ultimately not disrupt blood flow from the extrinsic system.[29,30] Branches from the extrinsic system pierce the epineurium and connect with the intrinsic system.

Intrinsic System

The intrinsic microcirculation incorporates three levels of anastomotic plexuses, each supplied by penetrating branches from more peripheral levels. Longitudinally aligned arterioles and venules make up the epineurial plexus. Vessels course around and between fascicles. Branches penetrate the outer layers of the perineurium, supplying flow to each fascicle. The perineurium provides significant protection to the numerous longitudinally oriented vessels, interdigitating between the lamellae and those at the endoneurial level. The ramifying vessels connecting the perineurial and endoneurial plexuses follow oblique courses through the innermost layer of the perineurium.[27] This configuration might play an important role during nerve injury. Lundborg suggests that in the face of rising endoneurial fluid pressure, focal ischemia may occur as these oblique vessels are compressed.[27] The endoneurial microcirculation consists of abundant capillary beds. Segmented, longitudinally arranged vessels are linked in a double U-loop formation.[29]

Interestingly, blood flow at any level of the intrinsic system responds to the subtle whims of external forces. Rapid shifts in the flow patterns arise following change in position, pressure, temperature, or injury. Thus, efficient utilization of available flow is ensured.[29]

Vascular Innervation

The sympathetic autonomic nervous system innervates the vasa nervosum. Strong stimulation of the sympathetic chain can lead to profound vasoconstriction at the neural level. It has been postulated that extended durations of increased sympathetic tone may lead to abnormal nerve function and may play a role in sympathetically maintained pain syndromes.[27]

PATHOPHYSIOLOGY OF NERVE INJURIES

Classification of Nerve Injuries

Profound insights into the pathophysiology and characterization of nerve injuries were undoubtedly spawned by the innumerable casualties sustained during World War II. Several systems were developed in an effort to clarify the seemingly heterogeneous clinical presentations of nerve injuries in continuity.

Seddon's Classification

Seddon[31] proposed the division of these injuries into three categories. Terms coined by Professor Henry Cohen in 1941 were adopted to describe the levels of injury: (1) neuropraxia, (2) axonotmesis, and (3) neurotmesis.

Neuropraxia. Neuropraxia (literally: nerve nonaction) is characterized by paralysis that occurs in the absence of distal degeneration.[31] Subsequent studies by Denny-Brown and Brenner[32] confirmed focal demyelination as the underlying pathology. Clinical observations at the time suggested that large myelinated fibers were more apt to be involved. Motor loss predominates, with diminution of vibratory and proprioceptive sensation, the most common sensory abnormalities. Less commonly, decreased touch sensation may occur and rarely is deep pain perception completely abolished. Paresthesias are very common and may be the harbinger of returning sensory function. In Seddon's study,[31] the most common etiology was an injury resulting from a compressive force. The time course from onset of injury to complete recovery of motor and sensory function ranged from 1 week to 6 months with 10 weeks as the average. Recovery in Sunderland's population of radial nerve injuries ranged from 17 to 60 days.[33] Recovery is spontaneous. Not uncommonly, several weeks of significant functional loss will be abruptly supplanted by exceedingly rapid recovery.

Axonotmesis. Axonotmesis (literally: axon cutting) suggests an injury to the nerve of such magnitude that distal degeneration occurs. The supporting structures: the epineurium, perineurium, endoneurium, and Schwann cells, however, remain intact. In the population studied by Seddon,[31] 43% of all axonotmetic nerve injuries occurred in the face of closed fractures, dislocations, or fracture/dislocations. Compressive injuries ranked a distant second at 18%. The manifestations of an axonotmetic injury can not clinically be differentiated from a more severe injury in the acute setting. Sensory, motor, and sudomotor functions of the involved axons are abolished. Over time, the excellent levels of recovery help to clarify the modesty of the lesion. Muscle and sensory return is not as rapid as that seen in neuropraxic injuries. The chronicity is directly proportional to the level of injury and can be approximated by dividing the distance to be traversed from the injury focus to the end organ by the rate of regeneration, estimated at 1.5 mm/d.[31]

Neurotmesis. The term neurotmesis (literally: nerve cutting) is something of a misnomer. While it implies total severance of the entire nerve, it was meant to describe an injury in which all functional elements have been severely damaged. The epineurium may in fact be intact at visual inspection. A remarkable 97% of Seddon's group sustaining this level of injury had experienced penetrating wounds, whether from gunshots, stabbings, or lacerations.[31] The prognosis for a neurotmetic lesion is invariably poor. Spontaneous recovery is inimitable. Surgical coaptation may lead to functional return, although resolution of deficits is rarely complete.

Sunderland's Classification

Seddon's classification of nerve injuries was a bold initiative, enhancing the ability to discern clinically salient degrees of injury. Several ambiguities in Seddon's monograph spurred others to develop more elaborate classification systems. While complaints regarding the use of obscure Greek derivatives to describe the different levels seem petty at this time, more significant shortcomings were well founded. Seddon's definition of axonotmesis for example, was specific. It asserted the integrity of the epineurium, perineurium, endoneurium, and Schwann cells. His description of the clinical presentation, however, did not match the presumed anatomical distortion. It was suggested by Sunderland that in cases where more extensive injuries have been sustained (eg, a fusiform neuroma),[31] the strictly defined category of axonotmesis was not truly correct.[34] More precise predictions of functional return, the timing of surgical exploration, and potential microsurgical reconstruction warrant the ability to determine more accurately the severity of an injury. Sunderland's classification system

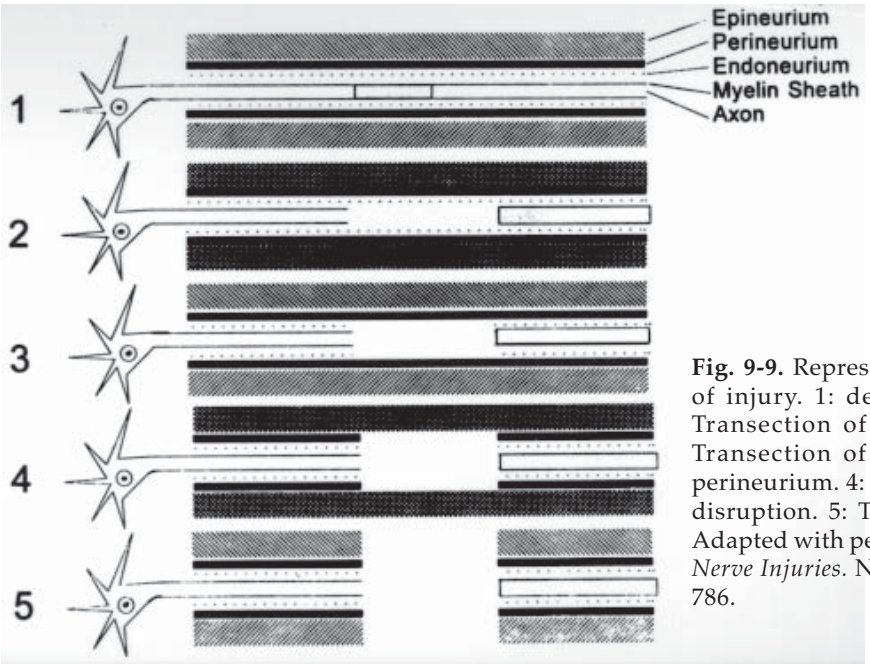

Fig. 9-9. Representation of Sunderland's five degrees of injury. 1: demyelination (conduction block). 2: Transection of axon with intact endoneurium. 3: Transection of the axon and sheath inside intact perineurium. 4: Transection of perineurium, fascicular disruption. 5: Transection of the entire nerve trunk. Adapted with permission from Sunderland S. *Nerve and Nerve Injuries.* New York: Churchill Livingstone; 1978: 786.

was based on detailed anatomical investigations of peripheral nerves and nerve injuries. Five degrees of injury were defined (Figure 9-9).

First-degree injury. A first-degree injury is synonymous with neuropraxia. Focal conduction block is caused by mechanical disruption of the myelin sheath. The axon and all supporting structures remain intact. Wallerian degeneration does not occur. Recovery is rapid and spontaneous.

Second-degree injury. A second degree injury correlates with Seddon's strict definition of axonotmesis. The axon is damaged, but all supporting structures, including the endoneurium and Schwann cell tubes, escape injury. Wallerian degeneration of the distal stump begins expeditiously following the trauma. As the architectural pathways remain unharmed, the regenerating axon has an excellent chance of growing down the proper endoneurial tube, innervating the correct end organ. Recovery is influenced by the level of injury and the rate of advancement of the growth cone. The Hoffman-Tinel sign, the provocation of tingling or lancenating sensations caused by tapping superficially on the sensitive region of a nerve, may be a useful tool providing a gross estimate of the progress and level of axon tip expansion.

Third-degree injury. Intrafascicular derangement typifies a third-degree injury. Within the uninjured perineurium, axonal and endoneurial degeneration transpires. Several potentially confounding obstacles may hamper regeneration. Endoneurial edema might result from alterations in vascular permeability following the insult. The lack of lymphatic channels and the perineurial blood-nerve barrier prevent adequate drainage from the intrafascicular space.[35] Direct injury to the endoneurial capillary plexus or obliquely situated feeder vessels may result in frank hemorrhage. Vascular stasis, ischemia, and, subsequently, fibrosis may block further recovery.

The loss of endoneurial tube continuity leads to a virtual free-for-all, as regenerating axon tips extend randomly toward endoneurial tubes at the distal side of the injury focus. An axon may meet with one of several fates. It may (*a*) pass into the correct distal endoneurial tube and innervate the proper end organ; (*b*) enter the wrong distal tube with ineffectual results; (*c*) axonal sprouting may lead to an axon-endoneurial tube mismatch, with too few tubes available to accommodate all the axons; and, again, the end organ may never be correctly innervated; or (*d*) fibrosis and organizing hemorrhage may entangle the axon, preventing further progression, and local sprouting will result in neuroma formation. The level of injury also has a profound effect on potential functional return. As previously discussed, fibers destined to become specific branches are intermingled at very proximal levels. Therefore, axons injured at high levels are less likely to regain proper end organ contact. As more distinct branches form distally, the likelihood of successful reinnervation improves. In situations where similar muscle groups are erroneously innervated by the wrong axons, neuromuscular reeducation

may be extremely beneficial in improving functional recovery.

As would be expected, the onset of recovery is more delayed than in second degree injuries. Commonly, recovery is incomplete with persistent sensory and motor deficits that are variable in regard to severity.

Fourth-degree injury. Perineurial disruption, in addition to the more internal structures, is the hallmark of a fourth-degree injury. Rarely is any useful function obtained by spontaneous recovery. Neuroma formation is more the rule than the exception. Excision of the affected segment and surgical repair are invariably necessary for any hope of functional return.

Fifth-degree injury. A fifth-degree injury implies complete disruption of the entire nerve trunk, including the epineurium. Ironically, an injury of this magnitude may in fact lead to a better outcome surgically than a fourth-degree lesion, depending on the mechanism of injury. A sharp laceration with very isolated destruction responds well to early surgical intervention. Outcomes following crush or stretch injuries parallel those seen in fourth-degree injuries and are treated similarly.

Utility of Classification Systems

Classifications of peripheral nerve injuries have helped define the severity of injuries, have led to more precise prognostic evaluations, and have guided clinicians toward more rational and appropriate treatment plans. It should be noted, however, that pure lesions, in which all fibers sustain the same degree of injury, are not common. Of 537 nerve lesions in continuity evaluated by Seddon,[31] only 22% were pure. Thus, while gross estimates may be made in regard to recovery, the distribution of injury intensity across the inter- and intrafascicular levels adds another variable that may impact significantly on the return of sensory, motor, or sympathetic function.

END ORGAN CHANGES FOLLOWING DENERVATION

Wallerian Degeneration

Nerve injuries of magnitudes great enough to disrupt the integrity of the axon (ie, second degree or more severe) invariably result in stereotypical degeneration of nerve fibers distal to the focus of injury. This orderly progression was first characterized by Waller in 1852[36] and has been eponymously termed "Wallerian degeneration." The physiologic changes occurring can be divided into those involving the axon, Schwann cells, myelin sheath, and the macrophage response. Wallerian degeneration is erroneously portrayed as merely a destructive process. It is truly an elegant system, initiating the complete destruction of nonviable axonal components, removing all waste products, and establishing a fertile environment that may successfully guide forthcoming regenerating axons (Figure 9-10).

Axon Changes

The earliest axon changes are those seen at the terminal end of the nerve, in motor neurons at the neuromuscular junction. Miledi and Slater[37] demonstrated that an electrical impulse could be propagated along the distal portion of a transected rat phrenic nerve for upward of 8 hours. A rapid decline ensued at this point with complete loss of the ability to evoke an action potential over the next several hours. A strong direct correlation between loss of impulse propagation and cessation of spontaneous end-plate potentials was observed. Histologic changes paralleled those seen physiologically. Within 3 to 5 hours following the loss of spontaneous end-plate potentials, the normal structure of the end plate was severely distorted. Mitochondria became swollen and spherical in shape. Disorganization and eventual fragmentation of the cristae ensued. The axoplasm and its resident organelles disintegrated into amorphous clumps, ultimately engulfed by Schwann cells. Eventually, all that remained was a watery cytoplasm, devoid of functional elements. Schwann cells protruded into the synaptic cleft, completely enveloping the terminal end of the axon, disrupting contact between the nerve and muscle at the neuromuscular junction.[38]

Granular disintegration of the axon rapidly follows failure at the terminal junction. Collapse of neurofilaments and microtubules, which impart structural integrity to the axon, along with diminution of axoplasmic volume, are the cardinal events marking the onset of axonal degeneration.[39] Striking changes within the cell body and proximal stump are also evident within days and peak over the subsequent days to weeks. In motor neurons, the nuclei become eccentric and migrate to the pole opposite the axon hillock. Disappearance of Nissl bodies is noted as well as disintegration of the rough endoplasmic reticulum.[40] There is a virtual halt to

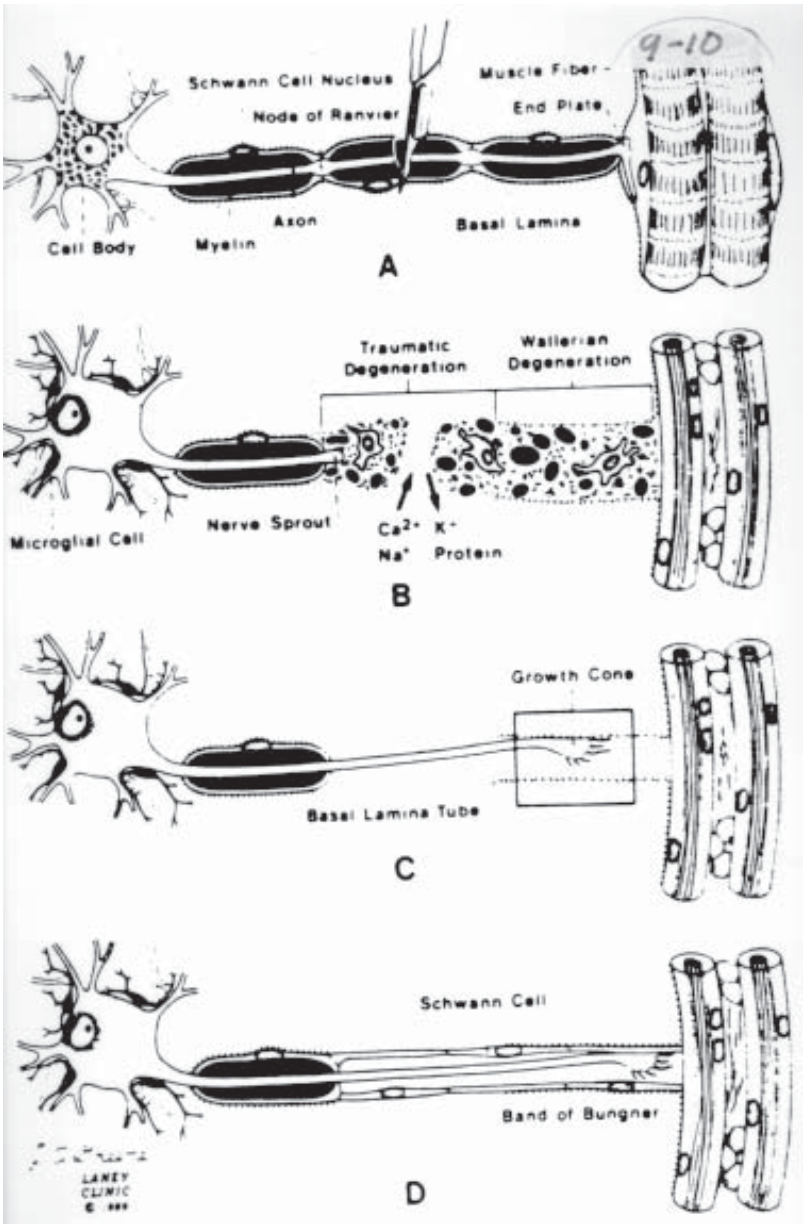

Fig. 9-10. Peripheral nerve degeneration and regeneration. **A**: Injury to normal fiber. **B**: Wallerian degeneration of distal axon. Basal lamina tube remains intact distal to the injury focus. **C**: Growth cone is formed and is attracted by chemotactic agents. **D**: Schwann cell line the basal lamina, attracting axons and eventually laminating the axon in myelin. Adapted with permission from Seckel BR. Enhancement of peripheral nerve regeneration. *Muscle Nerve.* 1990;13:785–800.

the production of neurotransmitter and a down regulation of messenger ribonucleic acid (mRNA).[41] Production of proteins crucial to anticipated regeneration, such as actin and tubulin, which are integral parts of the growth cone, are upregulated[42] while the initial production of structural neurofilament and microtubule proteins is diminished. The cross-sectional area of nerve fibers also decreases and may reflect this loss of neurofilament protein production.[43]

Initiation of Wallerian Degeneration

Debate has raged over the past 150 years as to what triggers the onset of axonal degeneration. Waller summized intuitively that a loss of trophic

support arises following the severance of the axon from its supporting cell body.[44] While the failure of nutritive support may partially explain the onset of chromatolysis and axon disintegration, the rapidity with which degeneration occurs is characteristic of an active, destructive process.[45] Others have suggested that the loss of impulses traveling down the nerve might lead to axonal atrophy, analogous to disuse atrophy seen in immobilized muscle.[46,47] This theory has been recently refuted by several studies in which electrical stimulation mimicking the firing pattern of motoneurons failed to retard axonal degeneration in axotomized nerves in rabbits.[43,48] Gutmann and Holubar[44] suggested an autolytic process as the likely cause of degeneration. Current studies examining ionic fluctuations during axotomy support this hypothesis.

LoPachin et al[49] showed that shifts in the concentration of specific ions precede even the earliest morphologic transformations. The changes in intraaxonal ionic composition follow a sequential pattern. In rat sciatic nerves, loss of K^+ and Cl^- is noted initially at the 8-hour mark. Intracellular levels of Na^+ and Ca^{++} begin to rise rapidly between 16 and 48 hours postinjury. Progressive deactivation of the Na^+-K^+ ATPase pump and potentially an active Ca^{++} pump[39] secondary to the loss of energy input are possible causes. As previously discussed, without the function of the Na^+-K^+ ATPase, K^+ escapes from the cell, along its concentration gradient. The relative impermeability of the neuronal membrane to Na^+ and Ca^{++} accounts for the time delay. As Na^+ rushes into the cell, the gated ion channels for both Na^+ and Ca^{++} become increasingly permeable, allowing for an even greater influx of Na^+ and Ca^{++} ions. The Ca^{++} influx activates endogenous proteases which initiate axonal destruction.

Another school of thought turns the antegrade trophic support theory on its end. Initially, following axotomy, there is down-regulation of neurofilament gene expression. It has been suggested[50,51] that substances akin to NGF may permit specification of the level of neurofilament gene expression at the cell body. The retrograde transport of this factor from the periphery is dependent on intact interactions between the nerve ending and specific target or accessory cells. Thus, the loss of peripheral retrograde feedback might trigger the shift from production of proteins needed to maintain regular neural function and integrity to that necessary for reconstruction of the damaged axon following clearance of axonal debris.

Schwann Cell and Myelin Sheath Changes

Within 2 days following axotomy, profound changes in myelin composition occur. Production of mRNA, sequenced for the production of myelin proteins, is markedly reduced. The internodal segments collapse and become disorganized. Local Schwann cells extend cytoplasmic phalanges through the myelin sheaths, fragmenting them into myelin ovoids. In rats, the ovoids are progressively destroyed over the ensuing several weeks, culminating in disorganized lipid whorls.[45] These Schwann cells, occupying the space previously filled by the internodal myelin are termed bands of Bungner. A proliferation of Schwann cells occurs, providing the groundwork and guidance system for future axonal regeneration. In sensory and sympathetic neurons, the production of NGF as well as NGF receptors has been localized to Schwann cells forming bands of Bungner. Interestingly, it was observed[52] that disruption of axon-Schwann cell contact triggers the expression of NGF receptors of the surfaces of the proliferating Schwann cells.

Role of Macrophages

The mechanism underlying the removal of axonal and myelin debris has been a focus of contention for many decades. In the 1920s, Ramon y Cajal and Swanson[53] described the synergistic roles played by Schwann cells and macrophages. It is only recently, however, that his cooperative paradigm has been confirmed experimentally. Stoll and colleagues[54] observed Schwann cells interrupting, phagocytizing, and degrading myelin sheaths during the earliest hours following axotomy. It was proposed that Schwann cells likely initiate degradation of their own myelin sheaths.[54] They do not, however, participate in the removal of myelin breakdown products. Elimination of myelin is accomplished by macrophages. A vast number of macrophages are chemotactically attracted to the degenerating neuron from the circulation. A paucity of resident macrophages also assist in the clearance. Beuche and Friede's[55] cogent study revealed that when nerve fibers degenerate in an environment devoid of macrophages, collapsed myelin sheaths persist for many weeks. The evacuation of debris is not the only function of macrophages. Synthesis and release of the cytokine interleukin-1 by macrophages is essential for the production of NGF by Schwann cells responding to nerve injury.[56]

Reinnervation

The degenerative phase produces a milieu conducive for regeneration. Cell body protein synthesis has switched to the production of actin and tubulin, essential to the construction of the growth cone. First described by Ramon y Cajal and Swanson,[53] the growth cone is a specialized conical swelling that develops at the distal end of the proximal stump. The growth cone has been described as ameoba-like,[53] extending lamellipodia which facilitate movement toward the endoneurial tube across the focus of injury into the distal stump. Growth cone movement and axonal elongation require several guiding elements. Endoneurial tubes in the distal stump persist following the removal of myelin and axonal debris. As previously mentioned, the disruption of contact between axons and Schwann cells triggers a rapid proliferation of the latter, which line the basal lamina. These Schwann cells produce receptors for growth-associated proteins such as NGF and growth-associated protein-43 (GAP-43), which serve as chemoattractants for the growth cone.[52,57,58] The growth cone follows the weak external chemotactic gradient, which is amplified internally.[59] It has also been shown that there may be some selectivity, improving the likelihood that certain types of neurons will enter matching endoneurial tubes. Contact of sensory growth cones with sympathetic neurons causes a collapse of the cone and redirection of its growth.[60]

The opportunity for axon growth into suitable distal tubes is somewhat self-limited. Schwann cell mitosis will continue only if contact with a growing axon is made.[61] When contact is not established between an extending nerve fiber and Schwann cells within the distal endoneurial tube, the diameter of the tube shrinks to 10% to 20% of its original size, hampering subsequent attempts at reinnervation.[62]

Muscle Changes Following Denervation

Changes in muscle function and morphology following denervation are congruent with those occurring in the nerve fiber. Loss of innervation triggers drastic transfiguration at the nuclear level, which leads to alterations in membrane composition, ionic channels, and neurotransmitter receptors. Macroscopic transformation of muscle tissue constituency and fiber organization ensue. While manifestations of denervation are commonly construed as deleterious, many enhance the tissues' responsiveness to reinnervation. External factors, including the extent and chronicity of nerve injury, the association of concomitant injuries, and age may have more to do with the ultimate functional outcome than the changes caused by denervation within the muscle itself.

Muscle Fiber Changes

Histological findings. The most characteristic early change in denervated muscle fiber is disruption of sarcolemmal nuclei, which become rounded and hyperchromatic and may become internalized within the myofiber.[63] A gradual reduction in fiber caliber proceeds. While subtle differences exist, the rate of extrafusal muscle fiber atrophy remains uniform across several mammalian species.[64–66] Bowden and Gutmann[65] observed no evidence of degeneration of muscle fibers during the first 3 months following denervation. The internal integrity of the sarcomeres remained intact. The cross-sectional area of muscle fibers decreased precipitously over the initial 60 to 90 days and then stabilized. These findings are consistent with Sunderland's data[9] in opossum and Gutmann and Young's assessment[64] in rabbits, which suggest that atrophy levels off at 70% to 90% by the third month.

Electron microscopy and biochemical studies have revealed that loss of fiber size reflects a loss of myofilaments. Early on, filament disappearance is sporadic and is observed only at the ends of the myofibers. The loss of myosin heavy chains precedes the loss of actin light filaments. By 4 to 6 months, some disorganization of the hexagonal filament arrangement occurs. At this point, changes are readily reversible with reinnervation. Interestingly, a difference in the rate of myosin heavy chain loss has been observed between fast, glycolytic, and slow oxidative muscle fibers. Fast fibers maintain myosin integrity for 4 to 5 times longer than slow fibers.[66] It has been suggested that the firing frequency ratios of the fibers may underlie the difference. Lack of stimulation is unusual in slow fibers, which are recruited early and commonly produce tonic, low-level activity. Thus, they may be more sensitive to the loss of innervation. Fast fibers, alternatively, are recruited late for brief bursts of speed and power and may be more resistant to a paucity of stimulation.[67]

A decrease in mitochondrial size and number, an increase in lysosomes, and a progressive depletion of oxidative and glycolytic enzymes closely follows the loss of myofibrils.[63] Again, these changes are late and not universally discernible for several months. Bowden and Gutmann[65] presumed that deterioration of fibers is incredibly deliberate. It may be up-

ward of 3 years or more before a level of decay is achieved that might confound attempts at reinnervation. Anecdotal reports have shown functional recovery following reinnervation of fibers denervated for 22 years.[68] It should be understood, however, that while the extrafusal fibers maintain their integrity in the face of atrophy for some time, other forces are at work that may compromise the feasibility of reinnervation as time goes on.

Muscle fiber atrophy. Muscle atrophy following denervation is the result of a combination of factors. Muller[69] observed a decrement of strength of 5% per day from initial levels, slowing to a loss of 25% at 7 days of immobilization. Protracted studies assessing strength loss during immobilization over several weeks revealed a leveling off of strength loss. The overall average at the end of 6 weeks was 8% per week with an overall loss of 48% of baseline strength.[70] Loss of contact between nerve and muscle deprives the end organ of trophic substances normally secreted by the motor neuron.[71] Davis and Kiernan[72] formulated two studies to determine relative responsibility for muscle atrophy. The extensor digitorum longus muscles in one group of Wistar rats were immobilized for seven days. Cross-sectional area decreased by 22%, consistent with the effects of immobilization described by other investigators.[69,73,74] In a group in which the muscles were both immobilized and denervated, cross-sectional area decreased by 35%. In a third group in which muscles were immobilized, denervated but injected with extracts of sciatic nerve, cross-sectional area reductions similar to those seen during pure immobilization were achieved. It was determined that approximately 60% of atrophy following denervation can be attributed to disuse, while the loss of neurotrophic influence accounts for the remaining 40%.[75] The importance of this delineation cannot be overstated. While much research is underway to develop techniques to augment reinnervation and the search to isolate neurotrophic substances continues, no clinically significant applications are currently available. Rehabilitative techniques that focus on limiting or overcoming disuse atrophy, however, are well founded and effective methods to enhance functional restoration.

Connective Tissue Changes

While loss of innervation leads to muscle fiber atrophy, collagen biosynthesis is unleashed and flourishes in the absence of neural inhibition. Sherman[76] observed the development of myogenic contractures in immobilized, denervated muscles within 4 to 6 weeks following injury. Myogenic contractures result from fibrosis involving the endomysium and perimysium. Little evidence suggests involvement of the epimysium. Types I and III collagen synthesis markedly increase following denervation. The collagen fibers become coarse, thickened, and enmeshed by the interweaving of cross-linking proteins. The ordinarily loose collagen meshwork surrounding myofibers tightens, mummifying atrophic muscle cells.[77] The effects of disuse, in contrast to muscle fibers, plays no role in the alteration of collagen synthesis in the face of denervation. Savolainen and colleagues[78] showed marked decreases in levels of collagen biosynthetic enzyme activity when muscles were merely immobilized. Denervated muscles, contrarily, expressed biosynthetic enzyme activity two and three times normal following neurectomy. The findings suggest that normal collagen synthesis adapts to the level of muscular activity and is under neural regulation. These findings have recently been confirmed by Virtanen and colleagues,[79] who not only replicated Savolainen's data, but also observed that reinnervation returned collagen biosynthesis to normal levels.

Denervation Supersensitivity

Supersensitivity or membrane instability, as it is termed in electrodiagnostic circles, is a hallmark of denervation. Two processes, the development of ectopic acetylcholine receptors and alterations in the electrical property of the sarcolemma are implicit in its development.

Acetylcholine receptor proliferation. In normal muscle, only the endplate, the region intimately associated with the presynaptic motor neuron bouton, possesses receptors for acetylcholine. A concentration of 3×10^7 receptors is found for every square micrometer. The inherent stability of the acetylcholine receptors is dependent on the influence of the motor neuron. Approximately 20% of receptors turn over rapidly, in less than 72 hours. The majority have half-lives of 6 to 13 days.[80] It is suggested that the rapidly decaying receptors are precursors to the more stable and mature receptors. Following propagation of an action potential, quanta of acetylcholine are released across the synaptic cleft and activate acetylcholine receptor channels at the endplate. The acetylcholine channels are somewhat generic and allow the passage of Na^+, K^+, and Ca^{++} cations. A net influx of Na^+ ions occurs that decreases the polarity of the cell, causing an endplate potential. The acetylcholine channels

work similarly to an automobile starter and are not the direct generators of the muscle action potential. The decreasing negativity caused by the influx at the acetylcholine channel triggers voltage-sensitive Na^+ channels outside the endplate, leading to amplification of depolarization and, ultimately, propagation of the action potential along the length of the muscle fibers.[81]

Denervation leads to profound alterations in muscle cell function. The loss of neurotrophic support induces, at the nuclear level, the production of proteins destined to be extrajunctional acetylcholine receptors. Initially, the extrajunctional acetylcholine receptors form in clusters across the entire membrane. Their density increases, although only to one tenth of that established at the endplate. The entire cell membrane becomes exquisitely sensitive to acetylcholine. During the hiatus between denervation and reinnervation, these extrajunctional acetylcholine receptors remain immature and rapidly turn over every 6 to 35 hours. In contrast to junctional receptors, the extrajunctional receptors are not exposed to acetylcholine esterase.[82]

Alteration of membrane potential. The second major factor promoting the development of denervation supersensitivity is the alteration of the electrical properties of the sarcolemma. The increase in acetylcholine receptors increases the likelihood that even minimal stimulation may lead to depolarization. Changes in the ion channels also play a role. While there is no change in the conductance of Ca^{++} activated K^+ channels, a 60% reduction in the number of these channels has been observed.[83] Alterations in Na^+ conducting channels and a decrease in the activity of the Na^+-K^+ pump also develop.[38] The increased permeability to Na^+ influx and decreased K^+ efflux precipitates a decrease in membrane polarization from –70 to –55 mV. Thus, the muscle fiber teeters perpetually on the brink of depolarization.

Influence on Regenerating Nerve Fibers

During reinnervation, constant feedback between the new nerve fibers and the extrajunctional acetylcholine receptor clusters lead to the development of stable, mature neuromuscular junctions. While the protein has yet to be isolated, several studies suggest that skeletal muscle itself exudes a trophic factor that stimulates and attracts sprouting neurons.[84,85] As many as 4 to 5 neurons may be observed in association with a single muscle fiber during the rudimentary stages of reinnervation. Contact between nerve fibers and extrajunctional acetylcholine receptors leads to the evocation of endplate potentials. Despite their low quantal content, the lack of acetylcholine esterase and the hypopolarization of the cell membrane enable muscle activity.

Repetitive and increasing levels of stimulation to inhibit the synthesis of acetylcholine receptors and eventually only the area of the fiber in contact with the motor neuron retains receptors. The new connections enhance the stability of these receptors and allow them to mature. They also trigger the formation of acetylcholine esterase binding sites within the basal lamina of the synaptic cleft.[86] The trophic influence of the muscle on sprouting nerve fibers eventually wanes. As strong neuromuscular junctions are established and the regions of acetylcholine receptors become constricted and defined, the reinnervated motor fibers will not accept additional innervation from another nerve.[87] Ultimately, a normal relationship between motor neuron and muscle fiber is completed.

MECHANISMS OF NERVE INJURY

Compression Injuries

Compression of peripheral nerves is one of the most common types of injury, exemplified by entrapment neuropathies. Compression may be caused by edematous changes in a nerve as it passes through a narrow space. Alternatively, a normally capacious tunnel may be compromised by soft tissue swelling, fibrosis, fractures, or foreign bodies. Blunt trauma such as that seen in Saturday night palsy is also a common etiology. Despite such familiarity, compression injuries represent complex lesions.

Pathophysiology

The mechanism by which compression disrupts neural function has been intensely studied. Over the past 50 years, somewhat polarized theories have gained popularity ranging from predominantly ischemic injury to pure mechanical deformation. Presently, more integrated explanations have materialized.

Clinical descriptions of motor paralysis with variable sensory deficits, no distal atrophy, and rapid excellent recovery were made by Frazier and Silbert during World War I.[88] Seddon,[31] too, ob-

served a similar clinical pattern, which he termed neuropraxia. It was not until 1944, however, that Denny-Brown and Brenner[89] experimentally confirmed Seddon's clinical hypothesis regarding pathologic changes in nerve fibers. Compression of cat sciatic nerve by either tourniquet or spring clip resulted in similar damage. Initially, edema was observed at the nodes of Ranvier with "powdering" of the myelin sheath. By 7 days after pressure application, the myelin had receded from the nodes closest to the edge of the cuff or clip. Thus, focal demyelination was confirmed.[32,89] Large myelinated fibers were more commonly injured than smaller fibers. Clinically, voluntary motor function had been abolished by pressures of 300 mm Hg applied for two hours. Motor excitability below the level of the lesion was 75% to 100% intact, while that above the level of the lesion had dropped to 0% to 5%. Denny-Brown and Brenner[89] suggested that segmental demyelination was related to ischemia and was not a consequence of direct pressure on the nerve fibers.

Ochoa and colleagues[90] conceded that mild compression, producing a metabolic conduction block that reverses rapidly with release of pressure, is caused by local ischemia. Conduction block lasting weeks to months without significant axonal degeneration was felt to be caused by direct damage to the nerve fibers. The characteristic lesion observed on teased fiber examination was displacement of nodes of Ranvier with invagination of one paranode by an adjacent one. Compression causes movement of the axon and myelin sheath away from the edges of the cuff, along the plane parallel to the nerve. The Schwann cell and basement membrane hold steadfast. A shear force is generated, dissociating the myelin from its parent Schwann cell. The invaginated myelin, torn from its nutrient source, degenerates. A focal barren area, void of myelin, develops.[90]

The findings of Ochoa and colleagues resulted from very high pressures (1,000 mm Hg) applied for several hours across a short segment (5.5 cm). Others employing lower pressures, which more closely resemble clinical situations, have not been able to reproduce these findings.[91] It is likely that a combination of primary mechanical deformation and secondary microvascular damage with edema and impaired local microcirculation produce the common clinical picture of conduction block.

Compression at levels as low as 30 mm Hg have been shown to slow venular flow.[35] Pressures upward of 80 mm Hg can completely arrest endoneurial circulation.[92] Reduction in venous flow further encumbers a normally sluggish endoneurial fluid drainage system. Disruption of oblique penetrating vessels may lead to increased vascular permeability and compromise of the blood-nerve barrier. Endoneurial blood flow, measured by laser doppler, has been observed to decrease by 50% in regions solely displaying focal demyelination.[93]

Axonal transport, which is exquisitely sensitive to ischemia, is disturbed at pressures as low as 20 mm Hg when sustained for upwards of 8 hours. At this level, mechanical distortion of the axon is unlikely. Distal supplies of cytoskeletal proteins, essential for axonal integrity and neurotransmitters utilized in synaptic transmission, may be jeopardized, crippling nerve function. Retrograde transport is also affected, limiting the flow of trophic factors from the periphery back to the cell body and undermining cell homeostasis.[94]

Clinical Considerations

Several studies have addressed the relative significance of ischemia and mechanical deformation in clinical settings. Gelberman et al[95] observed infraretinacular pressures of 32 mm Hg in patients with symptoms suggestive of carpal tunnel syndrome (CTS). Control subjects, without symptoms of CTS, had canal pressures averaging 2.5 mm Hg. This critical pressure was reproduced by Lundborg et al.[96] At 30 mm Hg, abnormal sensations such as numbness and parasthesias were observed 10 to 30 minutes following onset of compression. A decrease in sensory nerve action potential (SNAP) amplitude occurred concurrently. The amplitude decrement was not significant by current electrodiagnostic standards (ie, the amplitude was not decreased by greater than 50% when compared to the baseline amplitude). Gelberman and colleagues[97] noted at 40 mm Hg, decrements in sensory amplitudes of 40% within 10 to 30 minutes. Latencies were minimally delayed. Light pressure and two-point discrimination were not abnormal at this level and only after 4 hours of compression were vibratory sensibility abnormalities, tested at 256 Hz, observed. At 50 mm Hg, a significant drop in SNAP amplitude (ie, greater than 50%) was first observed within 15 minutes. Complete conduction block was achieved within 20 minutes. Motor latency delays were significant within 30 minutes. Abnormal sensibility to vibration and light pressure were documented at 20 minutes. Two-point discrimination measurement expanded beyond four mm at 30 minutes but was not completely lost until all sensation had been com-

pletely ablated. Motor strength faded from normal to fair over several hours. As with lesser levels of pressure, all parameters returned to normal abruptly after pressure release at 240 minutes. Pressures of 60 to 70 mm Hg produced findings similar to those found at 50 mm Hg.[97] Pressures of 90 mm Hg also produced reversible signs and symptoms when maintained for 60 minutes.[96] An animal study[98] employing rabbit nerves compressed at 80 mm Hg produced corresponding amplitude drops; however, maintenance of pressure for greater than 2 hours led to persistent diminution of action potential amplitudes.

A number of important conclusions regarding nerve tissue pressure thresholds can be made from these experiments. Pressure between 30 and 50 mm Hg may produce symptoms including numbness and parasthesias but reveal no objective signs aside from nonsignificant SNAP amplitude drops. This likely reflects mild local ischemic metabolic conduction block and may explain the 20% of patients with symptoms consistent with CTS but normal electrodiagnostic testing results.[99] Pressures greater than 50 mm Hg produce signs reflecting alteration in neurophysiologic function. Symptoms and signs manifest during short durations of pressures less than 80 mm Hg readily reverse with resolution of compression. Higher pressure or longer duration or both may lead to persistent findings, caused not only by ischemia but also by mechanical deformation and focal segmental demyelination. This level of injury is best exemplified by abrupt, focused application of high pressure such as that seen in compression of the radial nerve against the humerus during near-blast injuries, or alternatively, prolonged moderate pressure caused by entrapment of the radial nerve between the humerus and an external force as seen in Saturday night palsy. In these situations, where primarily segmental demyelination has occurred from mechanical deformation, recovery can take an average of 6 to 10 weeks and reflects the period of time required for remyelination.[100] Conduction block lasting greater than 10 weeks without evidence of Wallerian degeneration has been attributed to persistent endoneurial edema and elevated endoneurial fluid pressure. More severe compression may lead to not only demyelination but concomitant axonal injury.

Fascicular anatomy also plays a crucial role in the clinical manifestations of compression injury. Superficial fascicles and fibers are more prone to compression than those found more centrally. While large myelinated fibers are more commonly injured than smaller fibers or unmyelinated fibers, no significant difference in the sensitivity of large motor or large sensory fibers to compression has been consistently observed.[101] The propensity of certain fascicles to be injured based on topography and the variability of fascicular arrangements among individuals may lead to mislocalization of injuries when purely clinical assessments are made (Table 9-4). The utility of electrodiagnostic techniques to determine the focus of injury becomes paramount in these situations.

TABLE 9-4

NERVE FASCICLES ESPECIALLY PRONE TO COMPRESSION

Nerve	Region	Susceptible Fascicles
Sciatic	Proximal thigh Pelvis	Peroneal
Common peroneal	Fibular head	Deep peroneal
Ulnar	Elbow	Sensory to medial hand motor to hand intrinsics
Median	Supracondylar	Anterior interosseus
Median	Carpal tunnel	Sensory
Radial	Radial groove	Variable

While the tibial portion of the sciatic nerve is more susceptible to ischemic injury than its peroneal counterpart, compression injury more commonly affects the peroneal trunk. Sciatic injuries following pelvic or proximal femur fractures or hip dislocations may be mistaken clinically for a more distal injury to the common peroneal nerve. Compression injury of the ulnar nerve at the elbow affects fascicles innervating the hand intrinsics and providing sensation to the ulnar aspect of the hand more than it affects fascicles innervating the flexor carpi ulnaris or providing sensation to the dorsum of the hand. A purely clinical assessment may lead to false localization of injury to the wrist. Injury of the common peroneal nerve at the fibular head is more apt to produce symptoms in a deep peroneal distribution. Trojaborg[102] observed variable involvement of the brachioradialis and specific extensor muscles during compression of the radial nerve during sleep or secondary to blunt trauma. Sensory abnormalities were also variable. Selective injury to specific fascicles at the level of the radial groove

can only explain the heterogenous patterns because individual branch points occur well below the site of injury. While the anterior interosseus branch of the median nerve takes off 5 to 8 cm distal to the lateral humeral epicondyle, supracondylar fractures commonly selectively injure those fascicles. The posterior location of the fibers, close to the bone at the level of the distal humerus, enhances the likelihood of injury. The variability of sensory symptoms in CTS and the usual late finding of motor weakness suggest a more superficial but arbitrary fascicular topography of median nerve fibers beneath the flexor retinaculum.[103]

Ischemic Injuries

Ischemia is unequivocally a major cause of traumatic nerve injury. Yet, the abundant collateral circulation enveloping peripheral nerves has frustrated attempts by researchers to understand the pathomechanics underlying ischemic nerve injury. Results of many studies have been confounded by the mechanism by which cessation of blood flow was instituted. Most studies have employed tourniquet constriction of limbs. Controversy has been raised as to whether pathologic changes observed in these studies reflect ischemic injury, compression injury, or a combination of the two.[32,104–107] More recently, alternative methods of obtaining a relatively pure ischemic insult have been achieved. Korthals and Wisniewski[108] developed a model employing ligation of the abdominal aorta and femoral artery. This technique was modified by Hess and colleagues[109] and Fowler and Gilliatt[110] in several studies. Eloquent studies[111] utilizing infusions of arachidonic acid have also been developed. Clinically, pure ischemic neuropathy has been a consequence of ergot toxicity.[112]

Pathophysiology

The pattern and character of pathologic change from ischemia differs from that inflicted by other causative agents. Ligation of the internal and external iliac arteries of rabbits led to injuries of the sciatic nerves in the study by Hess et al.[109] The tibial portion of the sciatic nerve was predominantly effective relative to the peroneal portion. A junctional zone extending an average of 20 mm was observed early on and was felt to be the site of ischemic axonal injury. The zone revealed histologic changes that distinguished it from more distal Wallerian degeneration. In addition to axonal and myelin destruction, Schwann cells, fibroblasts, and vesicles were also destroyed.[108] Massing of cytoplasmic organelles was also observed in this region.[113] Just proximal to the zone, a region of paranodal demyelination developed. In 92.5% of the fibers, the last node proximal to the junctional zone sustained paranodal demyelination without evidence of axon injury and extended to the penultimate node in 60.5% of fibers and to the third node in only 22% of fibers. Wallerian degeneration was observed distal to the zone of injury within several days after the insult. In a small percentage of fibers, segmental demyelination occurred without evidence of Wallerian degeneration.[109] Segmental demyelination as evidenced by conduction block has also been observed in ischemia caused by ergot toxicity,[112] femoral artery ligation,[114] and arachidonic acid infusion.[115] In contrast to patterns seen in compressive neuropathies, the central core of large fascicles was particularly sensitive to ischemia. These findings suggest that certain regions along the nerve may be more prone to ischemic injury and likely reflect vascular watershed areas.

No consensus has yet developed regarding the tenacity of different types of nerve fibers in the face of ischemia. Lewis et al[116] clinically observed the loss of tactile and position sense early, followed later by the loss of pain and temperature sensation. It was deduced that larger fibers were most susceptible. Yet, it was also observed by several researchers that sensation is always impaired prior to the loss of motor function.[104,106,108,109] Parry and Brown[111] alternatively observed a disproportionate loss of unmyelinated fibers relative to those myelinated. Of interest is that some protection against ischemia has been observed in elderly, diabetics and uremic patients.[107] It is likely that enhanced collateral flow in the face of gradually waning primary vascular channels may help maximize blood flow in these tenuous situations.

Clinical Considerations

While a relatively discrete ischemic neuropathy may be observed in vasculopathies such as polyarteritis nodosum[117] and to some extent diabetes mellitus,[118] it is uncommon to see pure ischemic injury associated with trauma. It has long been suggested, however, that ischemia is the primary cause of peripheral nerve injury associated with compartment syndromes.[105,119,120] A compartment syndrome is defined by Matsen[120] as a condition in which the circulation and function of tissues within a closed space are compromised by increased pressure within that space. Increased compartmental pres-

sure is the fundamental condition underlying many clinical entities, including Volkmann's ischemia, rhabdomyolisis, crush syndromes, and exercise induced ischemia. An initial insult spurs hemorrhage and edema. Compartment pressure rises, collapsing veins and impeding drainage. The arteriovenous gradient is minimized. Decreased blood flow locally leads to ischemia. A vicious cycle of edema, ischemia, and necrosis ensues.

Between 1900 and 1950, several clinicians observed rates of nerve injury associated with compartment syndromes ranging from 50% to as high as 75%.[105] Peripheral nerve tissue is more susceptible to ischemia than other connective tissues.[110] Functional abnormalities commencing in paresthesias and loss of sensation of light touch and pinprick precede motor weakness. Intracompartmental pressures as low as 30 mm Hg have been observed to provoke sensory abnormalities[121] while others have suggested higher pressures in the 40 to 50 mm Hg range.[120] The latitude reflects several variables, including blood pressure, metabolic rate of tissues, and duration of pressure elevation. Sensory abnormalities have been noted within 30 minutes of the onset of ischemia[120] and have been touted as the most sensitive physical findings. They are harbingers of more serious injury if treatment (fasciotomy) is not instituted swiftly.[121] Irreversible changes are observed after 2 to 4 hours of ischemia and have been used as the benchmark for experimental studies.

Electrodiagnosis

Electrodiagnostic findings obtained by Fowler and Gilliatt[110] closely parallel the histologic changes observed by Hess et al.[109] In the majority of cases there was an immediate loss of ability to evoke a motor action potential proximal to the site of injury. Over the course of several days distal stimulation failed to elicit a motor action potential, suggesting Wallerian degeneration. In several animals with less profound functional deficits, distal stimulation was reduced relative to controls, reflecting axon loss; however, no response could be obtained from proximal stimulation. This suggested conduction block in the surviving axons. In general, ischemia produces a mixed injury with axonal loss predominating over demyelination.

While the measurement of intracompartmental pressure is the gold standard, nerve conduction velocity and motor action potentials may provide essential adjunctive information. Matsen and colleagues[119] used themselves as subjects to assess the utility of electrodiagnosis in the monitoring of compartment syndromes. At pressures ranging from 50 to 60 mm Hg, nerve conduction velocity abruptly dropped to 75% of controls at 25 to 30 minutes and paralleled the onset of motor weakness and anesthesia. Amplitudes of motor action potentials also dropped to 75% of distal stimulation in the same time frame. While attempts have been made to establish the minimal pressures at which fasciotomy should be considered, variables including hypotension, impaired vascular flow, concomitant injuries, and increased metabolic demands may place tissues at higher risk of injury at lower than expected compartment pressures. In patients who have sustained multiple trauma and may be hypovolemic or who are obtunded and cannot actively move the limb, electrodiagnostic studies may be helpful. Muscle death occurs earlier than nerve death. However, sensibility and electrodiagnostic correlates of nerve injury in the face of significant compartment pressure occur earlier than muscle injury and are reversible. In other words, nerves are more resilient but their injuries are more readily apparent earlier than muscle injury. Because they reflect the physiologic status of compartmental tissues, they allow for serial, noninvasive monitoring with quantifiable measures and may be the earliest marker of impending tissue injury.

Stretch Injuries

Clinical Considerations

Fractures, dislocations, and fracture-dislocations are commonly complicated by traction injuries to peripheral nerves. While the overall incidence has not been established, the rates of trauma to particular nerves with certain injuries have been assessed. According to Gurdjian and Smathers,[122] upward of 95% of all nerve injuries associated with fractures occur in the upper extremities. Five percent of all shoulder dislocations are complicated by nerve injury (Figure 9-11). In Rowe's series,[123] 30% of injuries were to the ulnar nerve, 18% to the radial nerve, 11% to the axillary nerve, and 4% to the median nerve. Combinations, likely reflecting brachial plexus injury, accounted for 37% of associated nerve injuries and overall are the most common. Mast and colleagues[124] observed an 18% rate of radial nerve palsy associated with humeral shaft fractures (Figure 9-12). Sciatic neuropathy complicates traumatic hip dislocations 11% of the time according to Brav.[125] Peroneal neuropathy occurs in 27% of traumatic knee dislocations (Figure 9-13).[126] The great amount of torque generated by inversion injuries to the

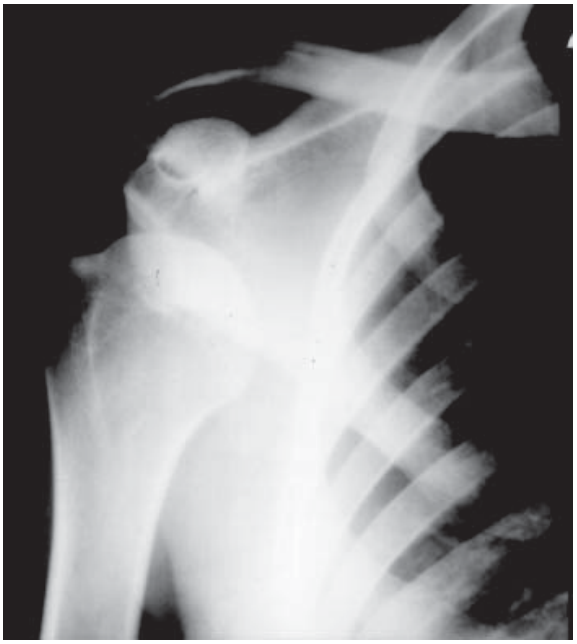

Fig. 9-11. Anterior shoulder dislocation. Stretch injury to major nerve branches or brachial plexus may be associated in 5% of injuries. Photograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

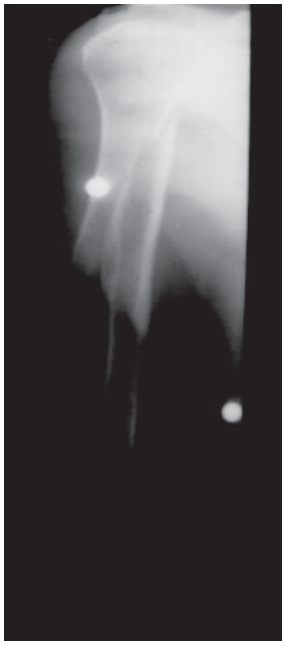

Fig. 9-12. Mid-shaft humeral fracture. Commonly associated with radial nerve injuries. Photograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

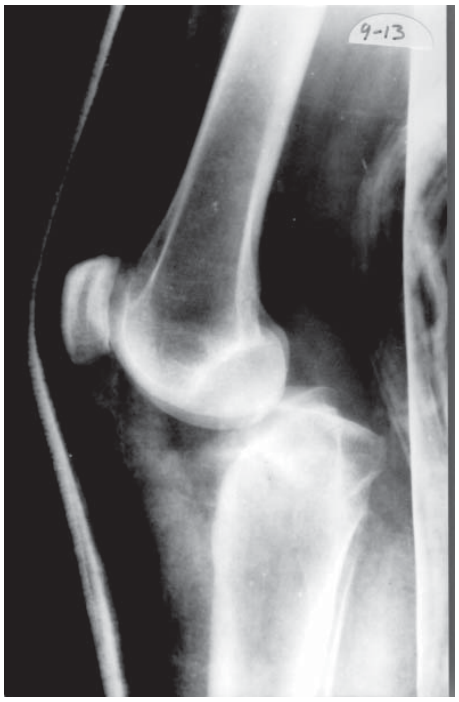

Fig. 9-13. Posterior tibial dislocation following a water skiing accident. Traction injury to the common peroneal nerve or peroneal fascicles of the distal sciatic nerve commonly coexist with this severe injury. Photograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

ankle may lead to proximal fibula fractures and associated peroneal neuropathies (Figure 9-14).[127] Severe varus stress, rupture of the lateral collateral ligament, may also cause a torsion injury of the distal part of the leg resulting in peroneal nerve injury.[128] Stretch is also an element in injuries associated with gunshot wounds[129] and has also been documented[130] as a complication of limb-lengthening procedures utilizing external fixation devices such as those designed by Ilizarov and Wagner.

The distribution of fracture-related nerve injuries has remained remarkably consistent over the years and typifies the intimate relationship of certain nerves and bones. Radial nerve lesions comprise nearly 58% of cases. Injuries to the ulnar nerve constitute 20%, peroneal neuropathies 15%, median neuropathies 5%, and sciatic neuropathies 2%.[122,131,132]

Pathophysiology

The strength and elastic properties of peripheral nerve have been extensively documented.[22,23,25]

Fig. 9-14. Distal tibial fracture with proximal fibular fracture. May be associated with peroneal neuropathy. Photograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

These variables are important not only in the assessment of potential injury associated with trauma, but also in determining when end-to-end anastomoses should be abandoned in favor of grafting in nerve injury repair. As with other tissues, the strength of peripheral nerves is directly proportional to their cross-sectional area. The cable design of fascicular organization imparts even greater strength. Sunderland and Bradley[26] followed the length of several nerves and observed that the maximum load sustained by the nerve was greatest where the fascicular cross-sectional area was greatest. Total cross-sectional area was held constant. Thus, tensile strength varies not only between different nerves but even between different areas of the same nerve.

The severity of nerve injury associated with traction is related to the extent of elongation, the intensity and duration of the deforming stress. Progressive traction forces placed on a nerve lead to a spectrum of functional decline. Lundborg and Rydevik[133] observed the first evidence of injury when nerves were stretched by approximately 8% of their total length. At this stage, microthrombi and emboli appeared in venules with a diminution of venous flow greater than 50%. A complete halt of arteriolar and capillary flow occurred at 15% elongation. Stagnation of endoneurial lymphatic flow followed, leading to increased endoneurial fluid pressure, decreased oxygenation, and cessation of axonal transport. If continued for greater than 30 minutes, axonal degeneration ensued. Lundborg and Rydevik's[133] study revealed no evidence of endoneurial vessel damage or disruption of the blood-nerve barrier imparted by the perineurium. These findings contradict those of Denny-Brown and Doherty[23] who suggested that high levels of endoneurial fluid were transudates emanating from small damaged blood vessels. They also call to question Haftek's[24] findings of perineurial disruption antedating axonal injury, a claim dismissed by Sunderland.[9] The compromise of vascular flow occurs after the slack of the epineurium, perineurium, and endoneurium have been taken up. As the perineurium stretches, the cross-sectional area of the nerve decreases, not unlike Chinese finger traps. The compression may lead to vascular compromise. While it has not been shown experimentally, it is postulated that direct pressure may also lead to deformation of the myelin sheaths, causing first-degree injury in the mildest of cases. At a maximum of 20% elongation, the elastic limit of the nerve is reached. This is commonly paralleled by physiologic failure. Sunderland[9] suggests that, at this juncture, axonal injury transpires, culminating in second-degree injury and Wallerian degeneration. Further tension disrupts endoneurial connective tissue (ie, third-degree injury) with potential for neuroma formation. As a maximum of 30% elongation is approached, the mechanical limit of nerve integrity is reached and the perineurium fails. Further stretch inevitably ends with epineural destruction and loss of continuity of the nerve trunk.[9]

The pivotal role played by the perineurium in the protection of peripheral nerves against traction forces is further validated when comparison to the nerve roots is made. Nerve roots, which are more prone to traction injury than nerve trunks, possess elastic and mechanical limits 5% less than those manifest in nerve trunks. The lack of perineurial sheathing at the root level is felt to underlie this susceptibility to traction injury.[26]

Prognosis

Outcomes following traction injuries, as would be expected, directly relate to the severity of the

injury. Fascicles and individual nerve fibers may sustain widely disparate levels of injury. Integration of fibers of all levels of performance reflects functional outcome. Omer[134] observed nearly 1,000 nerve injuries during his tenure at Brooke Army Hospital, San Antonio, Texas, during the Vietnam conflict from 1966 to 1970. Nerve injury in continuity associated with fractures, fracture-dislocations, or dislocations revealed spontaneous recovery in approximately 85% of cases. Isolated injury above the elbow required on average 2 to 4 months for recovery to ensue. Those below the elbow took 1 to 4 months to recover. Multiple injuries followed similar time frames. While electromyography (EMG) was mentioned in Omer's[134] study, no findings were recorded and thus no objective distinction between neuropraxic and axonotmetic injuries can be made. The fact that 37% of spontaneous recoveries occurred within the first 6 weeks following the injury may be most consistent with a predominant neuropraxia. The rate of recovery is too rapid to be explained by reinnervation. The remaining 63% required 10 to 32 weeks to fully recover. They presumably experienced axonotmetic or second-degree lesions by Sunderland's[34] criteria. These findings are consistent with those of Seddon[31] who also found that nerve injuries associated with fractures were predominantly axonotmetic.

Cold Injuries and Exposure

The ravages of cold-induced injury have been well documented during military campaigns of the last 2 centuries. Barron Larrey, chief surgeon of Napoleon's Grande Armée, documented the effects on French troops during their ill-fated assault on Russia.[135] The 115,000 cases of "trenchfoot" suffered by British soldiers wallowing in the cold, dank mire of the trenches of World War I significantly hampered the army's military effectiveness.[135] Ungley and Blackwood[136] astutely documented the clinical features and pathophysiology of the distal vasoneuropathy termed *immersion foot*, which was sustained by myriad troops exposed to cold sea water for extended periods during World War II. Immersion foot and trenchfoot share the same clinical and pathological presentations and are felt to be the same syndrome. Despite an enhanced insight into cold-induced nerve injuries, they continue to pose a significant threat to both military and civilian populations. Cold injuries accounted for nearly 20% of all British casualties sustained during the Falkland war with Argentina in the early 1980s.[137] More recently, immersion foot has become a problem manifest in the homeless population in the United States.[138] Iatrogenically induced cold injuries have also been documented during cryotherapy for musculoskeletal injuries.[139]

Severe exposure to cold commonly results in freezing of the tissue with subsequent necrosis. Denny-Brown and colleagues[140] observed that direct freezing of rabbit sciatic nerve for 5 minutes led to complete necrosis of all involved fibers. Endoneurial structures were destroyed with the exception of the endothelial lining of penetrating blood vessels. When direct freezing was limited to under 2 minutes, selective involvement of fibers was observed. Large myelinated motor fibers were more susceptible to injury than myelinated sensory fibers and both were more susceptible than unmyelinated fibers.

The absence of neuroma formation or scarring at the site of freezing suggests that relatively good recovery may be anticipated. Denny-Brown et al[140] observed evidence of regeneration within 1 to 2 months and completion by 3 months.

Clinical Considerations

Less intense cooling to temperatures just above freezing leads to a unique syndrome termed *trench foot* in World War I, *immersion foot* in World War II. In the Ungley and Blackwood series,[136] men exposed to immersion of their limbs in sea water at temperatures ranging from 1°C to 9°C for upward of 14 days manifest the now classic syndrome-immersion foot. Three stages have been described (Table 9-5).

Stage I prehyperemic. In the prehyperemic stage, the extremities are cold, swollen, and numb. Anesthesia in a stocking or glove distribution is dense. Distal pulses may or may not be present. Stage I commonly lasts from hours to days.

Stage II hyperemic. Stage I is followed by a hyperemic stage, which may last 6 to 10 weeks. Several findings dominate the clinical picture. Presumed injury to autonomic nerve fibers leads to vasomotor instability. The temperature of the affected limbs remains elevated from 30°C to 34°C, with loss of the distal-proximal temperature gradient. The limb remains erythematous with a violaceous tint. The color change is enhanced by dependency and blanches with elevation. Swelling is observed, especially in limbs that have been inadvertently warmed rapidly.

Sensory abnormalities often reach their nadir at 7 to 10 days after rescue and remain sustained at this intensity for more than 6 weeks. Parasthesias

TABLE 9-5

CLINICAL PRESENTATION OF NONFREEZING COLD INJURY

Stage	Duration	Presentation
Stage I Prehyperemic	Onset to several hours or days	Stocking or glove anesthesia Edema, Hypothermia ± Pulselessness
Stage II Hyperemic	End of stage I to 6–10 weeks	Temperature elevation Erythema, Edema Pain: Dysesthesias, Allodynia, Hyperpathia, Lancinating Pain
Stage III Posthyperemic	End of stage II to years	Normalization of temperature control Persistent hypersensitivity to cold Raynaud's phenomenon Hyperhidrosis Improving sensory and motor function

are universal in a stocking or glove distribution and are intensified by dependent positioning of the limb. Shooting and stabbing pains have also been noted. They are more intense with dependency and may be exacerbated by exposure to cold or warmth, exercise, micturition, defecation, or yawning. Commonly, pain complaints are increased at night. Hyperesthesia and allodynia are also commonly experienced. Sensitivity to pinprick, light touch, and vibration are variably decreased or lost.

Motor strength of muscles within the cooled territory is commonly impaired. Anhydrosis coincides with the extent and distribution of sensory loss. Blistering may be observed in moderate to severe cases as early as 3 days postrescue but are more commonly seen around the 8th to 10th day. Healing may be protracted and take from weeks to several months. Hair, fingernails, or toenails may also be lost.

Stage III posthyperemic. During stage III, which can last for weeks to years, vasomotor control begins to normalize. Skin temperature and color return to normal. There remains, however, a sensitivity to cold that may last for months to years. This abnormal response to cold and emotional stressors is termed Raynaud's phenomenon, and is characterized by vasospasm causing painful blanching, followed by cyanosis. As the vasospasm remits, the digits become erythematous.[141] Hyperhidrosis may develop irrespective of environmental temperature. Sensory and motor functions continue to improve.

Prognosis

Prognosis following immersion foot is generally good. Mild cases resolve within 2 to 5 weeks. In fact, the vast majority of cases in White's[142] series returned to duty within 6 weeks. In those more severely injured, a more protracted convalescence may take place lasting 3 to 12 months. Of the 18 patients followed long-term in the Ungley and Blackwood[136] series with moderately severe courses, 35% returned to full duty. Fifty-four percent returned to light duty, limited by cold sensitivity and standing tolerance. Only 11% were unable to return to some form of active duty. Long-term sequelae included intermittent shooting pains in 64%, paresthesia with prolonged standing in 35%, and intermittent swelling in 43%. Sensitivity to cold remained in the majority up to 2 years postrescue.

Pathophysiology

As with other causative agents, the severity and completeness of cold-induced injuries vary with the duration and intensity of exposure. Susceptibility to injury also differs with nerve morphology. Discrepancies in the literature abound and may reflect the methods by which investigators sought to study these questions. Many investigations based conclusions on clinical findings (eg, loss of movement or response to sensory stimulation) while others have focused on the ability to evoke

SNAPs and compound motor action potentials (CMAPs). Further confusing the picture are conclusions based on findings obtained very soon after the cooling event.

Temperature

The study by Schaumburg and colleagues[143] of direct cryoprobe cooling of cat sciatic nerve evaluated decrementing temperature exposure from 20°C down to 6°C. Motor weakness was observed at 10°C. Little structural damage was noted until 7°C was reached. Response to light touch was lost prior to loss of pinprick, which waned at 7°C. Nerve conduction velocities were measured and noted to be delayed at 7°C. The studies were obtained with needle stimulation and recording electrodes and therefore no assessment of action potential amplitudes can be made. Denny-Brown and colleagues[140] also showed substantial injury to rabbit sciatic nerves when directly exposed to temperatures ranging from 5°C to 8°C. Similar findings were reproduced by other researchers.[144,145] Recently, Kennett and Gilliatt[146] noted a significant difference between rabbit tibial nerves directly cooled at 5°C and 1°C for 2 hours. The amplitude of the compound motor unit action potential (CMUAP) of the former decreased by 50% from baseline by the third postcooling day. The amplitude of the latter dropped by over 70%.

Duration

The duration of nonfreezing cold exposure necessary to effect peripheral nerve injury understandably hinges on whether cold is applied directly or through the insulation of skin, subcutaneous fat, and muscle. Direct cooling of rat sciatic nerve to 3°C for as brief a period as 5 minutes results in an inability to propagate an impulse from a proximal point of stimulation. In the study by Nukada and colleagues,[145] normal latencies returned within 30 to 40 minutes; however, a persistent amplitude drop of greater than 40% remained, suggesting axonal injury. Denny-Brown and colleagues[140] observed a spectrum of injury to fibers exposed directly to temperatures 5°C to 8°C for 30 to 120 minutes. Schaumburg and colleagues[143] observed histologic changes when nerves were directly exposed at 10°C for at least 1 hour but not at 30 minutes. At cooler temperatures, even those exposed for only 30 minutes developed substantial injury. More recent studies[144–147] have used 2 hours as a standard duration to provoke direct nerve injury.

In the more clinically oriented studies during the two World Wars, cold exposure for as short a period as 14 hours was sufficient to cause nerve injury. Most soldiers, however, were exposed for several days.[136,142] This was confirmed experimentally by Kennett and Gilliatt.[146] Rabbit hind limbs immersed in 1°C solution for less than 9 hours developed no appreciable injury. Variable pathology was observed between 9 and 14 hours. Injury similar to that seen with direct cooling at 2 hours was observed when the limb was cooled in excess of 14 hours.[147]

Type of Nerve

As with the other variables discussed, discrepancies in the literature regarding differential sensitivity to cold injuries between different nerve types are widespread and likely reflect different methods used in some of the earlier studies. Denny-Brown and colleagues[140] and Schaumburg and colleagues[143] monitored the loss of motor and sensory function. In both studies, the investigators observed paralysis of muscles innervated by the involved nerves at temperatures below 7°C and 10°C, respectively. Sensory loss occurred later at 0°C and 5°C, respectively. It was inferred that large myelinated fibers were more susceptible to cold than smaller myelinated fibers and both more easily injured than unmyelinated fibers. Recent studies[143,146] employing electrodiagnostic techniques as well as electron microscopy substantiate the clinical findings of Denny-Brown and colleagues.

Morphologic Changes

Direct exposure to nonfreezing cold induces characteristic morphological changes in the peripheral nerve. Axonal swelling with clumping of electron dense material can be seen by electron microscopy as early as 6 hours after exposure. By 24 hours, profound subperineurial and endoneurial perivascular edema is present. Disruption of the myelin sheath into ovoid masses and an invasion by macrophages with associated degeneration of the distal axon commences between the third and seventh day. While patchy demyelination was seen in several axons in a number of studies, the vast consensus is that demyelination is not a primary pathologic process.

Electrodiagnosis

Electrodiagnostic studies performed by Kennett and Gilliatt[146] corroborate the finding histologically

of Wallerian degeneration as the principal pathology. One hour following cooling, motor action potentials could be evoked below the level of the injury. No potential could be obtained during stimulation proximal to the lesion. Prior experiments[146] that did not serially perform nerve stimulations for periods beyond the acute injury may have misconstrued this as conduction block or focal demyelination. Recordings on the second and third postinjury days revealed a progressive loss of motor action potential amplitude. By the third day, distal amplitudes resembled those evoked proximally. Follow-up studies[146] revealed improvement of amplitude both proximally and distally from almost 45% of preinjury at day 3 to nearly 75% of preinjury at 4 weeks. It was felt that the improvement reflected collateral sprouting as not enough time had elapsed for reinnervation from regenerating fibers to occur.

Generalized cooling by immersion revealed intriguing results. The main sites of injury were not at the distal ends of the nerve fibers but at well demarcated areas at the junction of the middle and upper thirds of the tibia, correlating to the area just below surface level. It was postulated that the abrupt temperature gradient may be the affecting variable.[147]

While the type of injury induced by nonfreezing cold has become discernible, the means by which this damage occurs remains obscure. It has been suggested by Bausbaum[144] that several mechanisms may come into play. Low temperatures may increase the viscosity of lipids within the axon membrane and alter its selective permeability properties and ability to propagate impulses. Another mechanism, which has been well described, is the suspension of axonal transport associated with cooling. Nukada and colleagues[145] showed that by 3 to 5 days after cooling, the blood-nerve barrier had been breached. The increased permeability coupled with poor endoneurial lymphatic drainage leads to vasogenic edema, which may also compromise axonal transport and other metabolically driven mechanisms. An inability to clear accumulating waste and toxins may also play a role.

Penetrating Injuries

The passage of high- and low-velocity missiles through soft tissue produces a wide array of devastating injuries. Almost 70% of penetrating wounds evaluated by the Physical Medicine and Rehabilitation Service at Walter Reed Army Medical Center during the Persian Gulf War were associated with peripheral nerve injuries.[3] Injuries caused by projectiles are undoubtedly the most common of all war-related trauma. A complete discussion of this topic is beyond the scope of this chapter but can be found in the Textbook of Military Medicine volume dedicated to injuries associated with conventional warfare, *Conventional Warfare: Ballistic, Blast, and Burn Injuries*.[148]

Pathophysiology

Elegant models developed by Harvey et al[149] at Princeton University during the 1940s simulated the primary and secondary destruction caused by the passage of high-velocity missiles. As the projectile enters the body it pulverizes all soft tissues in its path, transforming them into a bloody pulp. Immediately following the projectile, a large, subatmospheric cavity develops, whose volume is many times that of the primary missile track. The cavity rapidly expands and contracts, producing severe stresses and strains in the adjacent soft tissues. Secondary damage to the soft tissues surrounding the cavity occurs because of the initial rapid stretch and subsequent recoil.[149] Oscillating shock waves, moving at the speed of sound have also been measured in regions of the body distant from the site of impact.[150]

Peripheral nerve injury caused by penetrating wounds can be attributed to three different mechanisms. Large segments of nerves may be destroyed by direct contact with the missile. Commonly, a complete, fifth degree injury occurs in these situations with expansive gaps between nerve endings observed on inspection. Damage can also occur without a direct hit to the nerve. Nerves, like other components of soft tissue, are markedly displaced and deformed by the large cavity following missile passage. A spectrum of stretch and compression injuries ensues. Puckett et al[151] observed effects on the sciatic nerves of cats subjected to the impact of high-velocity spheres whose impact velocity ranged between 3,200 and 4,100 ft/sec. The rate of soft tissue displacement by the temporary cavity was one tenth of the impact velocity (300 to 400 ft/sec). In 4 of 22 cases in which the sciatic nerve was not hit but was within the zone of soft tissue pressure wave expansion, the nerves remained grossly in continuity. The rapid displacement of the nerve led to a wide variety of histological changes. Destruction of axon cylinders with maintenance of surrounding connective tissue (ie, second degree injury) was the mildest form of injury observed. Commonly, re-

gions of intrafascicular and interfascicular fiber destruction was noted (ie, third and fourth degree injuries). Nerve conduction studies (NCSs) performed within several hours of impact revealed an inability to propagate an impulse from the level of the wound or slightly proximal to it. Puckett et al[151] also observed 15 subjects in which the ability to propagate an impulse across the injured region was maintained. It was presumed that no injury occurred in these nerves because they were outside the perimeter of the temporary cavity. The extent to which cavitation causes injury to peripheral nerves remains unclear. Unfortunately, no microscopic evaluations or changes in the morphology of the evoked potentials were mentioned. Thus, it cannot be deduced as to whether milder (eg, first degree) or incomplete injuries to these nerves were truly experienced. An alternate explanation suggests that electromechanical effects caused by cavitation may cause the neurodysfunction.[148]

The prospect of neurological injury in parts of the body well distant from the site of impact remains controversial. Initially raised by Harvey et al[152] and later confirmed by Berlin[153] and Tikka et al,[154] current consensus in the field remains polarized with as many supporting the theory as opposing it. Suneson et al[155] recorded pressure waves of injuriously high energy and amplitude, moving at the speed of sound not only in the abdomen and thorax of pigs wounded in one thigh but also in the brain and contralateral thigh. While macroscopic integrity of the contralateral sciatic nerve was maintained, electron microscopy revealed distortion of myelin sheaths as well as axons. Microtubules were unusually sensitive, experiencing disarray and disintegration.

Clinical Considerations

Prognosis following penetrating injuries depends not only on the integrity of the peripheral nerve but also on the extent of concomitant injury to muscles, bone, and vascular structures. While data obtained in the civilian sector regarding shotgun injuries cannot be directly generalized to reflect the military experience, some important parallels can be made. Fifty-nine percent of shotgun injuries to the upper extremities included nerve damage.[156] Nerve injury occurred at a rate of 40% when all gunshot wounds were included and 50% when all penetrating trauma was assessed.[157] Of the shotgun wound cases, 60% required skin or pedicle grafts to cover soft tissue defects, fractures occurred in 64%, and vascular disruption was observed in 33%.

Other studies[158,159] measuring the association of nerve injury and vascular injury have an observed rate of 37% in close-range shotgun injuries and 51%[158] for all penetrating trauma. According to Visser and colleagues,[157] data from the Vietnam vascular registry documented a 44% incidence of concurrent nerve and vascular injury. When broken down by extremities, both civilian and military sectors convey a preponderance of complex injuries involving the upper extremities. Over 70% of upper extremity vascular injuries from projectiles were complicated by peripheral nerve injuries. Lower extremities sustaining vascular injuries manifest nerve damage in 32% of civilian cases and 25% of military casualties.

Omer[134] assessed outcomes of nerve injuries from high- and low-velocity gunshot wounds sustained during the Vietnam War. Good outcomes were defined as return of sensibility to pressure of less than 3.84 (0.693 F/gm) by Semmes-Weinstein pressure esthesiometry and manual muscle-testing grade of fair or better. Omer's findings were consistent with those obtained by Foerster in World War I and by Sunderland in World War II (as cited in Omer[129]). In all three conflicts, nearly 70% of nerve injuries associated with gunshot wounds recovered spontaneously to a good level.

Isolated low-velocity (1,000–1,500 ft/s at impact) nerve injuries above the elbow required on average 4 to 7 months to recover, while those below the elbow recovered more rapidly, in 3 to 6 months. When more than one nerve was involved above the elbow, recovery averaged 5 to 8 months. Multinerve injuries below the elbow averaged 3 to 7 months to recovery. A bimodal distribution is observed when all low-velocity injuries are included. A majority recover within the first 6 weeks and a second peak is noted at the 5 to 6 month mark. These differences may reflect primarily neuropraxic injuries in the former and axonotmetic lesions in the latter.

High-velocity (2,500–3,000 ft/s at impact) nerve injuries differed little in recovery time in relation to location above or below the elbow. Isolated injuries required 3 to 6 months for recovery and multiple injuries averaged 5 to 9 months.

These findings are more optimistic than those conveyed in the civilian shotgun literature in which transection, nonfunctional, and no recovery groups comprised nearly 80% of outcomes.[156] Eighty-six percent of vascular injuries complicated by peripheral nerve injuries in civilian cases culminated in significant functional impairment.[157]

The differences in outcome may likely be explained by ballistics. Shotgun injuries are of lower velocity, on average 1,200 ft/s. The blast and shock wave effects are less profound than those generated by high-velocity missiles. The local effect is more injurious, however, as upwards of 275 14-mg shots are concentrated into a small area. As the shots encounter different tissue planes and densities, their trajectories may change. A wide area of local trauma occurs. While outcome data are not yet available, injuries caused by modern ordinance, which spray small fragments at low-velocities, may be more similar to those experienced in shotgun injuries than to the high-velocity missile injuries of previous wars.

ELECTRODIAGNOSIS OF PERIPHERAL NERVE INJURIES

General Principles

Electrodiagnosis is a special form of diagnostic test designed to assess the integrity of peripheral nerves, neuromuscular junctions, or muscles. It is an extension of the history and physical examination and is not intended to be performed in isolation. It is, however, intended to augment the information obtained through interview and careful examination of the patient. Electrodiagnostic testing offers qualitative and quantitative information regarding severity and distribution of nerve injury or disease and information that may be helpful in determining prognosis. The quality and accuracy of an electrodiagnostic study is highly dependent on the expertise and experience of the electromyographer.

It is imperative that meticulous attention to proper technique be employed at all times to ensure accuracy and reproducibility. Although guidelines for performing a specific type and number of tests for a suspected clinical problem have been established by the American Association of Electrodiagnostic Medicine (AAEM), each electrodiagnostic evaluation must be tailored to the patient's presentation. It is not uncommon to alter the approach to a clinical problem during the course of the test as new information is gathered and a new differential diagnosis is entertained.

Electrodiagnosis offers a physiologic perspective; imaging studies, such as magnetic resonance imaging or computed tomography scans, offer an anatomic view of suspected pathology. It is important to recognize and document electrical abnormalities in nerves or muscles. It is particularly critical to understand the clinical implications of these electrical abnormalities in order to properly interpret the findings. General categories of pathology can be detected by an appropriately designed electrodiagnostic study; classes of disorders detectable by electrodiagnosis include the following:

- disorders of the motor neuron,
- diseases of the roots and plexus polyneuropathies,
- entrapment and mononeuropathies,
- neuromuscular transmission disorders, and
- myopathies.

Electrodiagnosis is separated into two general types of electrical studies: NCS or EMG. The term EMG is sometimes used to refer to both nerve conduction studies and electromyography. These studies provide the foundation on which more sophisticated studies such as repetitive nerve stimulation, single fiber, macro EMG, and somatosensory studies are performed.

Neurophysiology

Motor Unit

The motor unit is defined as an anterior horn cell, its axon and corresponding terminal branches, the associated neuromuscular junctions, and all muscles fibers innervated by the axon. It is the small functional element of contraction.[159] Discharge of a motor unit results in synchronous contraction of all muscle fibers innervated by the axon. Pathology may occur at any point or segment along this path and is potentially detectable with selective electrodiagnostic studies.

The average size of a motor unit can be expressed as the ratio between the total number of extrafusal fibers and the number of innervating motor axons. This is referred to as the innervation ratio:

$$\text{Innervation ratio} = \frac{\text{Total number of muscle fibers}}{\text{Total number of innervating motor axons}}$$

Motor axons vary in the number of muscle fibers they innervate depending on the type of muscle involved. For example, extraocular muscles have few muscle fibers per axon (ratio of 3:1), while the gastrocnemius muscle has hundreds of muscle fibers

per axon. Those muscles with a small ratio are involved in fine gradations of movement, while those with larger ratios produce relatively coarse movements.[160] Motor units also vary in their size. Some have thick and extensively myelinated axons while some are unmyelinated. The heavily myelinated axons conduct faster than the unmyelinated axons. Large diameter, myelinated axons conduct impulses at about 60 m/s, while small diameter axons with less myelin conduct at about 45 m/s.[161]

The larger motor units have a higher innervation ratio, greater twitch tensions, faster twitch contractions, and a greater tendency to fatigue.[162] The size principle of Henneman[163] proposes that motor units are recruited in an orderly fashion with small motor neurons activated first, followed by progressively larger motor neurons. Thus, mild contraction efforts reflect activation of smaller motor units while progressively increasing contraction force is the result of activation of larger motor units. With few exceptions, the Henneman size principle is applicable to virtually any voluntary activation of motor units.[164,165]

Motor neuron disorders and neuropathy, for example, typically do not alter the size principle. However, an exception to the rule may include the random recruitment pattern generated after reinnervation has occurred in a previously transected peripheral nerve.[166] This loss of the size principle response is attributable to the misdirection of motor axons and the associated impairment or loss of orderly recruitment. It is possible to reestablish the size principle after transection of a nerve if the affected motor axons can reinnervate their respective original muscle fibers.[167] Histologically, all muscle fibers of a given motor unit share the same characteristics. The muscle fiber territories of different motor units overlap so that muscle fibers of a single motor unit are in contact infrequently with fibers innervated by the same motor unit.[160]

Membrane Potential

Muscle membranes polarize at –90 mV while nerve axons polarize at –70 mV.[168] Suprathreshold stimulation results in generation of an action potential due to increased sodium conductance. An all-or-none phenomenon occurs after threshold depolarization, independent of the type of stimulus applied.

An electrical stimulator has a cathode, or negative pole, and an anode, the positive pole. Electrical stimulation generates negative charges under the cathode external to the axon membrane, causing a relative increase in the positive charges internal to the axon membrane at the same point along the axon. This process is referred to as cathodal depolarization. Under the anode, negative charges leave the membrane surface, resulting in greater negativity inside the cell and creating anodal hyperpolarization. With depolarizations of 10-30 mV, threshold of the membrane potential is reached and an all-or-none action potential is generated. The action potential propagates in both directions along the nerve from the point of stimulation.

Strength–Duration Relationship

The shorter the duration of electrical stimulation, the greater the intensity required to produce the same degree of depolarization. The strength-duration curve illustrates this principle graphically. Rheobase is the minimal current strength below which no response occurs even if the current lasts greater than or equal to 300 ms. Chronaxie is the minimum duration of current necessary to excite the cell at twice the rheobase. The use of chronaxie and rheobase to determine nerve excitability is somewhat anachronistic as more precise and useful techniques have been developed over the past several decades.[160]

Motor neurons have an inherent excitability that correlates with the axon diameter, conduction velocity, and motor unit size. An action potential can be propagated at greater speed if the four following conditions are present[160]:

1. faster rate of action potential generation,
2. increased current flow along the axon,
3. lower depolarization threshold, and
4. higher temperature.

Myelinated fibers propagate action potentials via saltatory conduction, which involves current "jump" from one node of Ranvier to another. This is in contrast to the continuous propagation of impulses along unmyelinated fibers. The greater the myelin thickness, the less the internodal capacitance and conductance. As a result, for a given axon diameter, the greater the myelin thickness, the faster the conduction velocity.

In demyelinating conditions, internodal capacitance and conductance increase, inhibiting action potential propagation. If sufficient inhibition occurs to cause failure to activate the next node, conduction block occurs. If inhibition is incomplete, conduction velocity may be slowed with temporal dis-

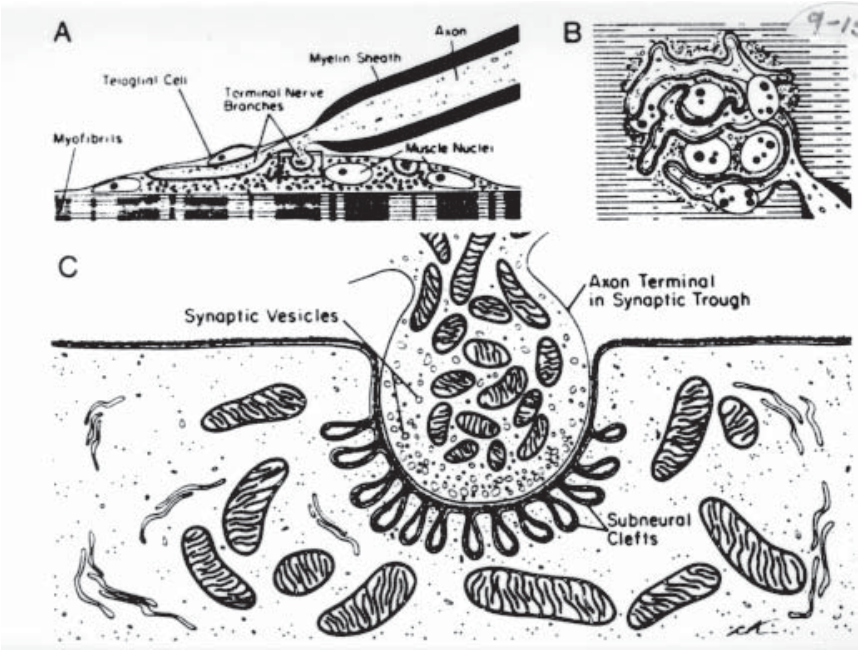

Fig. 9-15. Schematic illustrations of the neuromuscular junction. **A, B**: Light microscopic views of the motor endplate. **C**: Cross-sectional electron microscopic view of the motor endplate. Neurotransmitter is released from the neural synaptic vessels into the cleft. Interactions between neurotransmitter quanta and postsynaptic receptors result in subthreshold depolarization, termed miniature endplate potentials. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 171.

persion of the action potential. In focal demyelinating conditions due to compression, the capacitance of the internodal membrane may actually be decreased due to the pathological narrowing of the fiber diameter. As an isolated change, this would facilitate impulse conduction. However, increased resistance to action potential propagation secondary to the compression greatly outweighs this effect and results in slowing or block of conduction from one node to the next at the site of compression.

An impulse is conducted electrically from the anterior horn cell to the neuromuscular junction. At the junction, transmission is continued via the presynaptic release of acetylcholine (Figure 9-15). The acetylcholine then binds with acetylcholine receptors on the postsynaptic membrane, generating an electrical impulse. If a sufficient number of acetylcholine molecules are released and successfully bind with the receptors, the impulse then propagates down the muscle fiber at 4 to 7 m/s.[161,169]

Instrumentation

NCSs and EMG are performed on highly sophisticated instruments. Many of these instruments are equipped with computers for rapid acquisition and display of electrodiagnostic data. Some are capable of storing and retrieving both numerical information and graphs. The electrodiagnostic machine is composed of multiple integrated components (Figure 9-16).

At the very minimum, the instrument should be able to display latency and amplitude of a SNAP or CMAP. The duration of the action potential may be

Fig. 9-16. Modern computer based electrodiagnostic machine. Includes preamplifier, differential digital amplifier, stimulating electrode, color monitor, audio speakers, printer, and floppy disk memory storage.

calculated or automatically recorded. The measurement of the area under the curve of the action potential may be desirable and is available on some electrodiagnostic instruments. All such devices have a range of frequencies over which electrical activity is recorded. Some offer only selected ranges, while others permit manual selection of a broad range of low to high frequencies depending upon the desired result.

Electrodiagnostic studies involve the recording, amplification, filtering, processing, display, measurement, and interpretation of biologic electrical data detected by surface or needle electrodes. The data obtained through EMG involve needle electrode pickup of volitional motor unit action potentials and spontaneous potentials arising from muscle membrane instability. Nerve conduction studies involve electrical stimulation of sensory or motor fibers or both, and pickup of time-locked sensory, motor, or mixed action potentials at a separate, accessible point along the nerve or muscle.

Action potentials are generated in the human body, which acts as a volume conductor. Electrical signals detected by the electrodes are first amplified and filtered to eliminate environmental distortion of the desired action potentials. Real time or analog signals are transformed into digital data displayed as waveforms on the monitor of an electrodiagnostic machine. The visually displayed data are accompanied by acoustic data corresponding to the analog signals received.

Waveform display of an action potential, by definition, requires two electrodes because an action potential is defined as the difference between two electrodes locations in a volume conductor. Action potential amplitudes are expressed in millivolts and microvolts; currents are expressed in milliamperes and microamperes; and impedances in kilohms and megohms. Latency and duration of action potentials are measured in milliseconds and microseconds.

Compound motor action potentials are obtained by placing an active electrode on a muscle's motor point. The reference electrode is placed on a relatively electrically inactive site, such as the tendon of the muscle, in order to maximize the waveform amplitude. If the active and reference electrodes are placed in similarly electrically active sites, then little differential amplification occurs, and the resulting waveform amplitude will be significantly attenuated.

Sensory nerve action potential waveforms appear to be optimally displayed when the distance between the active and reference electrodes is greater than the spatial extent of the potential's rise time. Thus, an interelectrode separation of greater than or equal to 4 cm is preferred in order to obtain maximum waveform amplitude. An interelectrode distance less than 4 cm results in reduction of the action potential due to greater common mode rejection.[170,171]

Amplification

Amplification is expressed as a gain or sensitivity factor. The gain is the ratio of the amplifier output to its input signal. An input of 10μV, which results in an output of 1 V, has a gain of 100,000.[171,172] Sensitivity is a ratio of input voltage to the size of deflection of the cathode ray tube (CRT).[171] It is recorded as the number of microvolts or millivolts per division. A division on the CRT grid is typically 1 cm. Electrodes and amplifiers form a circuit dependent on Ohm's Law, which states that voltage (E) is equal to the current (I), multiplied by the resistance (R). Thus:

$$E = I \bullet R$$

Impedance (Z) is substituted for resistance (R) when dealing with alternating currents. Ohm's Law is modified in the following fashion:

$$E = I \bullet Z$$

The electrode and amplifier form a series circuit in which both have an impedance value. Because each part of the circuit has an impedance, the total voltage drop across such a circuit is due to the summation of voltage reduction across each circuit component. The voltage of the observed signal is directly proportional to the impedance of the amplifier and indirectly proportional to the sum of the impedance of the electrode and amplifier.[172] The higher the impedance of the input amplifier, the less the impact of the electrode impedance. Thus, a signal is maximized if the impedance of the amplifier (typically up to hundreds of megohms) is substantially greater than the impedance of the electrode (typically recorded in kilohms).

Differential Amplification

Differential amplifiers are used in electrodiagnostic machines for the purpose of amplifying the difference between input from active and reference electrodes (difference mode signals), while canceling similar signals (common mode signals). The

differential amplifier requires an input terminal for an active and reference recording electrode, as well as an input terminal for a ground electrode. Although no two electrodes have exactly equal impedance, it is important to minimize the electrode impedance difference in order to optimally reduce common interference signals. Also, a high amplifier input impedance serves to minimize the effect of electrode differences in impedance.

The common mode rejection ratio (CMRR) is the ratio of the output of the amplifiers when a signal is amplified differentially vs that present in the common mode.[172] It provides a means of identifying the differential amplification between the signal and the common mode voltage.[160] Most electrodiagnostic equipment has a CMRR greater than or equal to 10,000. According to Kimura,[160] differential amplifiers should have a CMRR exceeding 100,000. However, he points out that even high CMRRs may not completely eliminate extrinsic interference for the following two main reasons:

1. Although electromagnetic interference similarly affects each electrode, there is a variable degree of difference depending on the position of the electrodes.
2. There is also a variable degree of difference between the electrodes depending on their unequal electrical contacts.

Use of short, well-shielded electrode cables and proper grounding of the patient, bed, and electrodiagnostic instrument are important measures to reduce electrogmagnetic interference.

Filters

Action potentials generated in electrodiagnostic studies are the result of a summation of sine waves of variable amplitudes and frequencies.[170,171] High frequencies are manifested by the rapidly changing components of a waveform, while low frequencies are found in the slowly changing portions of a waveform. In waveforms obtained with NCSs, the rapidly changing portions of the waveforms include baseline take-offs, inflection points, rise time, and peaks. Slowly changing components include baseline returns and total potential durations. In EMG, the initial and terminal segments of the CMUAPs and the terminal portions of positive sharp waves constitute low-frequency components.[172]

Filters permit the recording of the desired signal by allowing input of all frequencies of interest contributing to the intended signal, while eliminating as much electrical interference as possible. Thus, this process helps to minimize contamination of the desired signal by extraneous electrical input. A high-frequency (low-pass) filter shunts high frequencies, but passes low frequencies. A low-frequency (high-pass) filter blocks low frequencies, but allows passage of high frequencies.

Different low- and high-frequency filter settings are required for different forms of electrodiagnostic evaluation in order to maximize recording of desired frequencies. The low-frequency value indicates the lowest frequency to which an amplifier will respond, while the high-frequency value indicates the highest frequency detectable by the amplifier.[171] Recommended filter settings include the following: 2 to 10 Hz low frequency to 10,000 Hz high frequency for motor NCSs; 2 to 10 Hz low frequency to 2,000 Hz high frequency for sensory NCSs; 20 to 30 Hz low frequency to 10,000 Hz high frequency for routine EMG; and 500 to 1,000 Hz low frequency to 10,000 to 20,000 Hz high frequency for single fiber EMG. Somatosensory evoked potential filter settings are typically 1–10 Hz low frequency to 500 to 3,000 Hz high frequency (Table 9-6).[172]

Changes in the low- and high-frequency filter settings result in alteration of the sensory and motor action potentials obtained. For instance, progressively increasing the low-frequency filter when evoking a SNAP results in a progressive decrease in its amplitude, peak latency, and negative spike duration, but possible increase in its overall duration, particularly if an extra phase occurs in the terminal portion of the signal. If the low-frequency filter is set too low, it results in an unstable baseline.

TABLE 9-6

COMMON FILTER SETTINGS FOR ELECTRODIAGNOSTIC STUDIES

Test	Low Frequency (Hz)	High Frequency (Hz)
Motor nerve conduction	2 to 10	10,000
Sensory nerve conduction	2 to 10	2,000
Standard EMG	20 to 30	10,000
Single fiber EMG	500 to 1,000	10,000 to 20,000
Somatosensory evoked potentials	1 to 10	500 to 3,000

EMG: electromyography

If the high frequency filter is set too low, the amplitude of the high-frequency or rapidly changing components of the evoked action potential is reduced.[160,171,172]

Frequencies obtained by needle electrode (electromyography [EMG]) may range from 2 Hz to 10,000 Hz, although as noted previously, some prefer 20 to 30 Hz to 10,000 Hz. Increasing the low-frequency cutoff value from 2 Hz to 32 Hz suppresses slowing changing components of a CMUAP. Fibrillations and insertional activity contain primarily high-frequency components and are, thus, not significantly affected by raising the low-frequency cutoff value. However, the tail of the positive sharp wave, a slow-moving component of this potential, will be distorted with progressive increase in the low-frequency component.

Electrodes

Electrodiagnostic evaluations require three electrodes, one active, one reference, and one ground electrode. Surface electrodes placed over muscle motor points in NCSs record summated electrical activity from many motor units. Needle electrodes in EMG pick up individual motor unit action potentials generated within a restricted radius of the recording tip. The ground electrode drains off currents originating from electromagnetic interference. Needle electrodes currently available include monopolar, standard or coaxial concentric, bipolar concentric, single fiber, multielectrode, flexible wire electrodes, and glass microelectrodes.

Monopolar and concentric needle electrodes are most commonly used. The monopolar needle is made of stainless steel and coated with Teflon except at the tip. It is used with a surface reference and ground electrode. It is less painful, less expensive, and records a larger action potential than the concentric needle. The CMUAP amplitude recorded with a faulty monopolar needle is decreased, possibly because the increased surface area of the recording tip has a decreased impedance and expands the region over which potentials are averaged.[172] The duration of the CMUAPs recorded is also decreased when using a monopolar needle with a defective Teflon coating.[171] Monopolar needle impedance is lower than that of concentric needles. Under certain circumstances, it may be advantageous to use a monopolar needle for stimulation of a nerve. Care must be taken to strip back the Teflon coating several millimeters, otherwise the concentrated focus of stimulation produced by a normal needle may cause excessively high local electrical current flow, resulting in focal nerve injury.[171] Alternatively, the use of an unmodified monopolar needle for stimulation with a current duration limited to 0.05 ms reportedly prevents axonal injury.[173,174]

The coaxial concentric needle has a stainless steel cannula with a nichrome, silver, or platinum wire in the center of the shaft. The central wire electrode is separated from the cannula by an insulating material. The needle is designed to register a potential difference between the center wire and the shaft. A separate surface electrode serves as the ground.

The bipolar concentric needle has a cannula which contains two fine stainless steel or platinum wires. This needle is larger in diameter than the coaxial concentric needle. The potential difference between the two inner wires is recorded. The cannula serves as the ground. Thus, electrical activity is recorded from a substantially smaller area than that recorded with the standard concentric needle.

The concentric needle electrodes have increased common mode rejection and record a slightly reduced action potential amplitude relative to the monopolar electrode since the cannula, which serves as a reference, detects similar activity to the active recording surface. The concentric needles also register slightly smaller motor unit amplitudes compared to monopolar needles because there are fewer muscle fibers within the recording range of the active electrode. The concentric needle electrodes generally produce a more stable baseline than that achieved with monopolar electrodes.

The single fiber needle electrode has a significantly smaller leading tip from which to record muscle activity. It specifically records action potentials from individual muscle fibers rather than motor units. It typically has a cannula diameter of 0.5 mm or less and the active recording wire is 25 μm in diameter. The wire is located proximal and opposite to the bevel of the needle. The cannula acts as the reference and a separate surface ground electrode is necessary. Usually only one, but sometimes two muscle fiber action potentials are detectable at any given time with this type of electrode. Monopolar or concentric needles, however, may record 10 or more muscle fibers.[172]

Electromyography

EMG is a means of assessing muscle action potentials using a needle electrode in the extracellular space. The needle examination can be broken down into four separate procedures, each of which conveys significant information regarding the in-

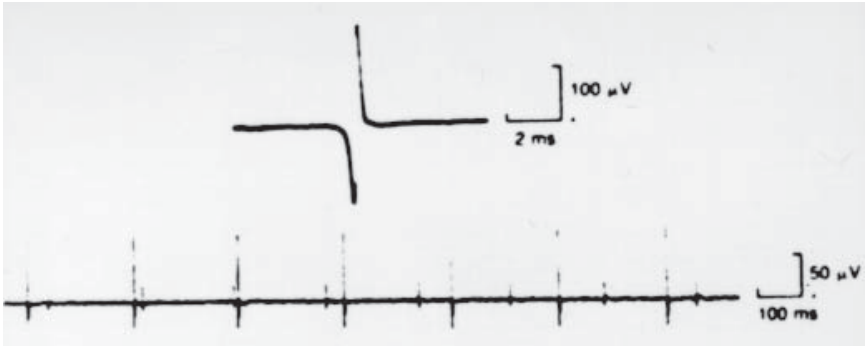

Fig. 9-17. Fibrillation potential. Electrical activity associated with spontaneously contracting muscle fiber. The potentials usually fire at a constant rate. Amplitudes range from 25 μV to 200 μV; durations extend from 0.5 ms to 2.0 ms. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 655.

tegrity of the motor unit: (1) insertional activity, (2) examination at rest, (3) examination at minimal exertion, and (4) examination at maximal exertion.

Insertional Activity

The passage of a needle electrode through a muscle membrane creates a brief electrical discharge that is both visible and audible with appropriate amplification. Insertional activity that is decreased or absent indirectly indicates a decrease in the number of muscle fibers secondary to severe atrophy or fibrosis, or both, of the muscle tissue. If insertional activity is increased, specifically if it lasts longer than the cessation of needle movement in the muscle, this indirectly reflects muscle membrane instability. Increased insertional activity may be found in the presence of a denervation process, myotonic disorder, or myopathic disease.

Examination at Rest

A normal muscle at rest is electrically silent. If electrical activity occurs at rest, it is referred to as spontaneous activity and generally reflects a pathological condition. Fibrillations and positive sharp waves are examples of spontaneous potentials and reflect abnormal discharges originating from single muscle fibers.

Spontaneous activity. Spontaneous activity is the electrical response found after needle electrode insertion activity and volitional muscle contraction has ceased. Spontaneous activity is not typically found in normal muscle at rest; it is also not present in muscles with isolated disuse atrophy; it can occur in paraspinal muscles following myelography, lumbar puncture, surgical intervention, or local trauma. It also may occur in the weak limbs of patients with upper motor neuron lesions. It is present in some myopathic disorders but most often in denervated muscle. Spontaneous activity may present as fibrillations, positive sharp waves, fasciculation potentials, myokymic discharges, or complex repetitive discharges (Figures 9-17 through 9-21).

Fibrillations are the result of spontaneous depolarizing single muscle fibers. They range in amplitude from 25 to 200 μV and in duration from 0.5 to 2.0 ms. They usually have a triphasic morphology and occur at a regular rate.

Positive sharp waves also represent a form of spontaneous activity. They are characterized by a wave with an initial positive deflection followed by more gradual return to baseline. They generally occur at a regular rate and, like fibrillations, are suggestive of

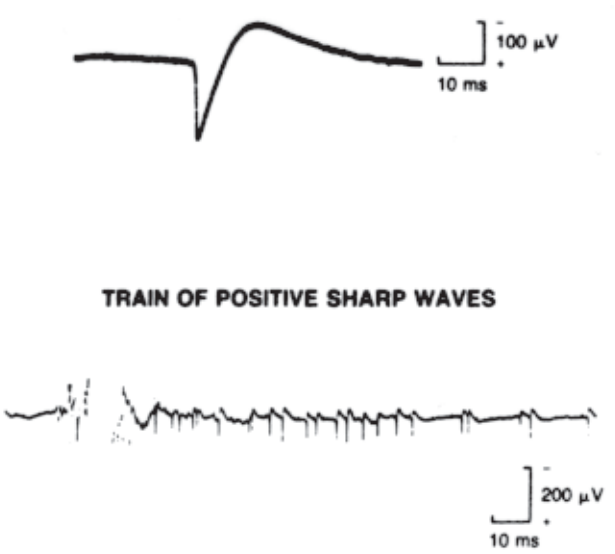

Fig. 9-18. Positive sharp wave. The potential is biphasic with an initial rapid positive deflection. They fire at a constant rate. Amplitudes are up to 1 mv. Duration ranges from 10 to 1,000 ms. Positive sharp waves are not specific for muscle fiber damage. Motor unit action potentials and myotonic discharges may have the same configuration. They do not however, fire at a constant rate. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 656.

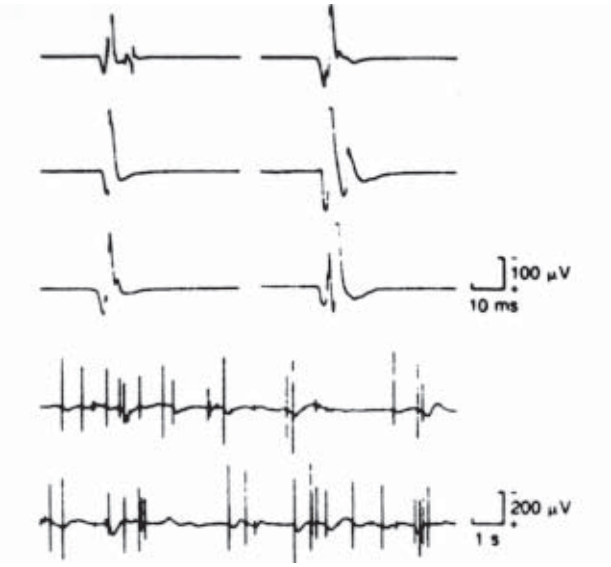

Fig. 9-19. Fasciculation potentials. Often associated with visible fasciculations in the limb, fasciculations have the same configuration as motor unit potentials but fire spontaneously. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 659.

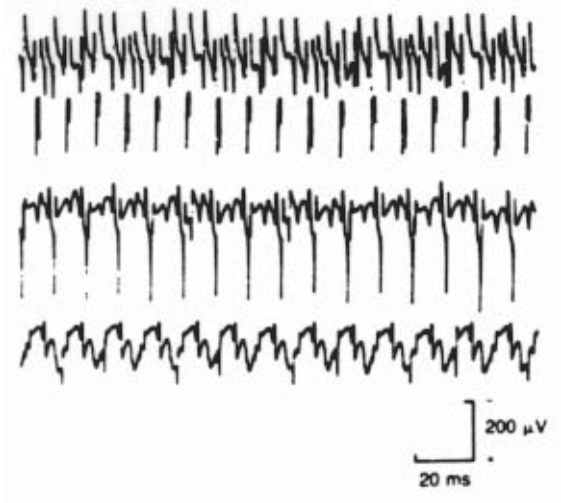

Fig. 9-21. Complex repetitive discharges. A polyphasic action potential that begins spontaneously or after needle movement. They have uniform frequency, shape, and amplitude with abrupt onset and cessation. Amplitudes range from 100 μV to 1 mV. Discharge frequency ranges between 5 Hz and 100 Hz. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 658.

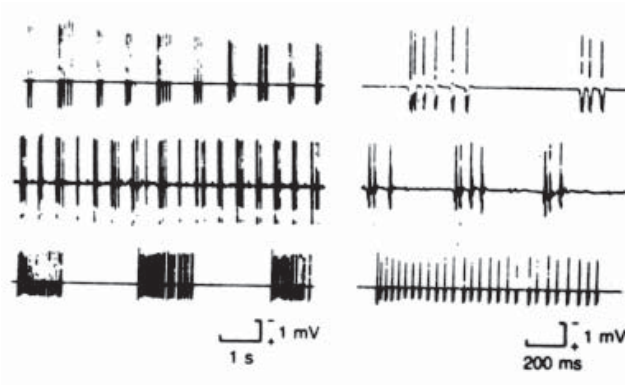

Fig. 9-20. Myokymic discharges. Commonly, discharges are brief, repetitive firings of single motor units for a few seconds at a uniform rate ranging from 2 Hz to 60 Hz. The sequence may be repeated for the same potential. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 660.

TABLE 9-7

QUANTIFICATION OF FIBRILLATION POTENTIALS AND POSITIVE SHARP WAVES

Grade	Definition
1+	Transient but reproducible runs of fibrillation potentials or positive sharp waves in two different sites
2+	Transient but reproducible runs of fibrillation potentials or positive sharp waves in more than two sites
3+	Spontaneous activity at rest in at least two different sites
4+	Profound spontaneous activity, which may fill the screen

Source: Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. 2nd ed. Philadelphia, Pa: FA Davis; 1989: 219.

an unstable muscle membrane. These waves are seen in neuropathic, some myopathic, and even some central nervous system disease (Table 9-7).

Spontaneous activity typically occurs within 2 to 3 weeks after nerve insult. Muscle fiber sensitivity to acetylcholine increases a hundred-fold after denervation and is known as denervation hypersensitivity,[175,176] which possibly contributes to the generation of spontaneous activity, but is not felt to be the sole or even the primary factor.

Examination at Minimal Contraction

Motor units have a distinct appearance, indicative of the extracellular recording of action potentials as they move toward and away from the active and reference electrodes. Specific attributes provide important information regarding particular pathologic processes. Technical factors, including the type of needle electrode employed, the muscle being studied, the temperature, and age of the patient may all influence aspects of motor unit morphology (Figure 9-22).

Motor Unit Electrical Morphology

Duration. The duration of the unit activity is the measurement from the onset of the potential as it leaves the baseline to its final return to baseline.[177] It normally varies from 5 to 15 ms when concentric needle electrodes are used.[160] Different durations will be recorded from the same motor unit depending on the type of electrode used. Duration will be longer with monopolar needles. The use of a surface reference electrode decreases the common mode rejection and more low frequency activity is included. A ratio of monopolar to concentric durations has been documented as 1.86:1.[178] Motor unit duration increases by 10% for each 1°C decrease in temperature between 37°C and 30°C. A 30% increase occurs for each 1°C decrease between 30°C and 20°C.[179]

Phases. The shape of the normal unit recording outside the endplate region has a triphasic appearance. There is an initial positive deflection followed by a negative spike and then another positive deflection before returning to baseline. The section of the motor unit recording between two baseline crossings is termed a phase. Adding one to the number of baseline crossings is one method of calculating the number of phases comprising a motor unit potential.[177]

Polyphasic motor units have more than four phases and reflect a relative asynchrony of muscle fiber firing within the motor unit. According to Kimura,[160] 5% to 15% of motor units may be polyphasic in normal muscles. The acceptable percentage of polyphasic units varies between muscles and with the age of the patient. An extensive compilation has been tabulated and can be found in Chu-Andrews and Johnson's text.[180] A drop in temperature to 25°C will increase the percentage of polyphasic potentials by 10%.[181]

Long duration polyphasic motor unit potentials suggest a significant loss of synchrony and can be

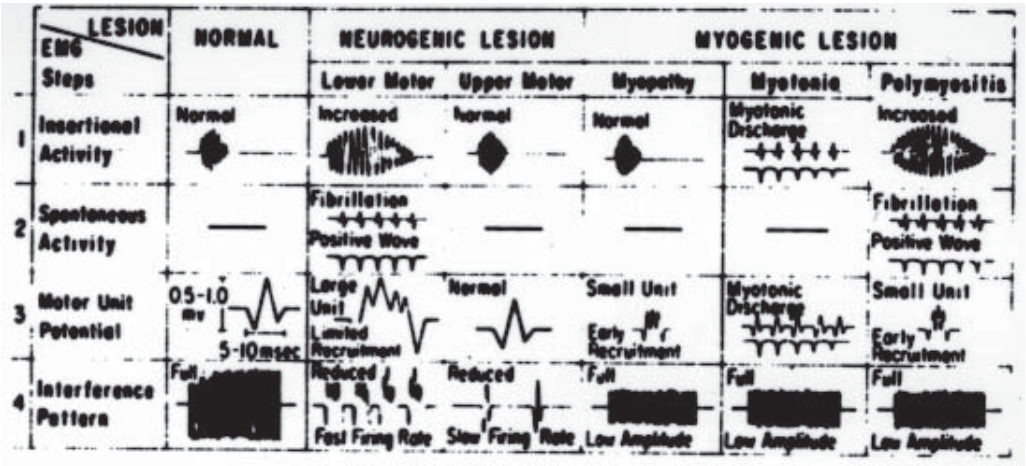

Fig. 9-22. Synopsis of electromyographic findings, including insertional activity, spontaneous activity, motor unit potential morphology, and interference pattern observed normally as well as with neurogenic and myogenic pathology. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 252.

seen during lower motor neuron degeneration and regeneration. Short duration polyphasic potentials are indicative of muscle fiber loss and are classically seen in myopathic processes or during early reinnervation following axonal loss.

Amplitude. The amplitude of the motor unit is the summation of muscle fiber activity within a 1- mm radius from the electrode. Thus, the distance of the tip of the electrode from a group of muscle fibers drastically changes the amplitude of the motor unit potential. To prevent fallacious measurement of potentials too distant to the electrode, motor units potentials suitable for assessment should have a rise time of less than 500 μs and preferably 100 to 200 μs.[160] The rise time is defined as the time elapsed between the initial positive peak to the next negative peak.[177] During minimal contraction, amplitudes range from several hundred microvolts to a few millivolts. During maximal contraction, normal values range from 2 mV to 8 mV. Occasionally, muscles of the hands may generate amplitudes as high as 12 mV and still be considered within the normal range.[180] Amplitudes of motor unit potentials examined with a monopolar needle are greater than those measured by concentric needles. Comparisons documented at minimal contraction measured the mean ratio of amplitudes recorded from monopolar to concentric electrodes at 2.05:1.[180] Temperature differences will also alter motor unit potential amplitudes. Differential slowing of conduction along the fibers summating to generate the potential causes temporal dispersion. Mean amplitudes will decrease by a factor of 2% to 5% for every 1°C decrease in temperature.[181]

Large amplitude motor unit recordings suggest prior lower motor neuron injury. Reinnervation and local sprouting concentrate the muscle fibers comprising the motor unit within a smaller region. Consequently, a larger number of muscle fibers are located near the tip of the electrode and a more significant summation is recorded. Small amplitude motor units are observed when muscle fibers are lost (eg, myopathic processes) or during very early reinnervation after a lower motor neuron injury (Figure 9-23).

Evaluation of motor unit electrical morphology is an indispensable component of the electromyographic evaluation. In situations where quantitative analysis is necessary, strong consideration should be given to use of concentric needle electrodes. Most reference values, including duration, amplitude, shape, and rise time, were established utilizing concentric needles. The use of a surface reference electrode during monopolar needle studies inherently leads to wide variability among the variables examined. Subtle differences in surface reference electrode placement may drastically change the common mode rejection and ultimately the duration and amplitude of the motor unit potentials.

Recruitment

The sole purpose of motor unit recruitment is to generate very specific amounts of force smoothly through muscle contraction. The process follows Henneman's size principle.[163] Small, low-tension units fire initially. As greater amounts of force are generated, larger, faster units also begin to fire. Force generation is the product of an increase in the firing rate of individual motor units and the activation of additional units. Normally, motor unit firing plateaus and the primary mode of increasing force is the recruitment of more motor units.

Assessment of the recruitment rate is an essential technique, allowing for the evaluation of motor unit pathology. First recruited motor units begin firing when contraction levels are barely percep-

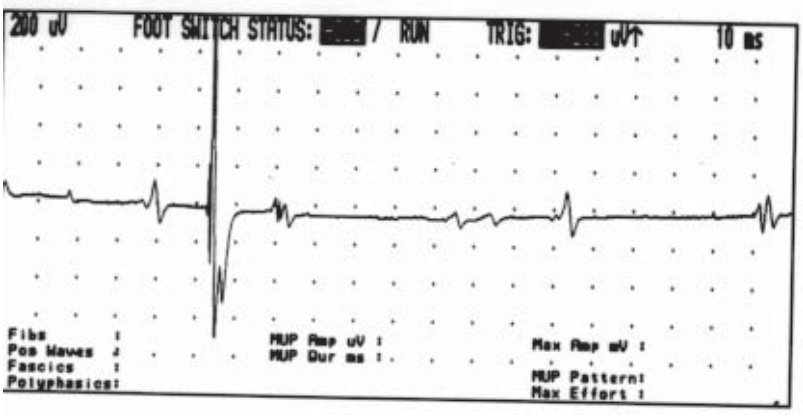

Fig. 9-23. Electromyographic display of a large amplitude polyphasic motor unit potential with associated satellite potential. The entire potential is of long duration. The satellite is not included in the measurement of duration. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

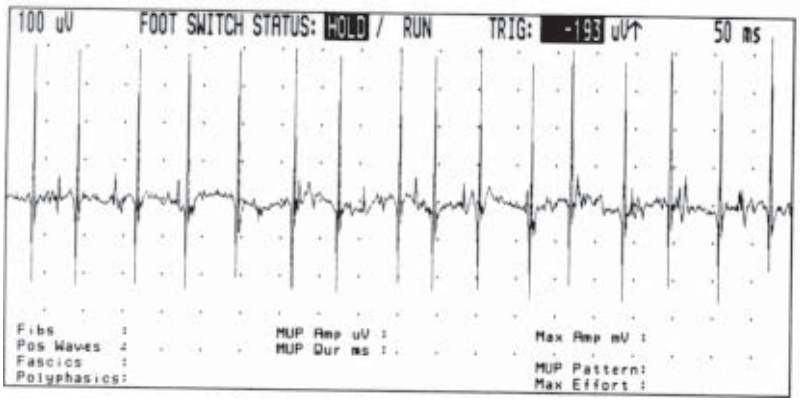

Fig 9-24. Recruitment rate of a first recruited motor unit firing at 16 Hz. This finding may be the earliest suggestion of motor unit pathology. It may occur with axonal loss or conduction block. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

tible. The frequency at which the unit is firing, termed the recruitment rate, is semirhythmic but becomes relatively stable just before the next motor unit begins to fire. Normal firing frequencies range from 5 to 15 Hz (Figure 9-24). On occasion, it is difficult for patients to contract at low enough levels to recruit less than three motor units. An alternative method of describing recruitment is the recruitment ratio. The rate of firing of the fastest unit is divided by the number of units firing. Normal ratios are around 5. A ratio greater than 10 is abnormal.[177]

Recruitment abnormalities may be the earliest evidence of motor unit pathology observed during the electrodiagnostic examination. Motor neuron loss is exhibited as an increase in the firing frequency. As fewer units are available to be recruited, adequate force generation is achieved through higher rates of firing by intact units.[182] Loss of muscle fibers creates a different recruitment pattern. Motor units are intact, yet, because a smaller number of muscle fibers are available, a greater number of normally firing motor units are recruited earlier to attempt adequate force production. The term rapid or early recruitment is used to describe this situation but the term is easily misunderstood. Rapid recruitment conveys only that the timeframe in which many motor units are normally recruited is compressed. The recruitment frequency is normal (ie, recruitment of the second unit does not occur when the first unit is firing at less than 5 to 15 Hz.[177]

Examination at Maximal Contraction

Assessment of motor unit firing during maximum voluntary contraction provides a gross assessment of motor unit integrity.

The interference pattern is a gestalt estimate of the total number of motor units activated per second during maximal contraction.[182] In a healthy individual providing maximal effort, individual motor unit potentials cannot be discerned because the multitude of units firing at upwards of 50 Hz fill the screen and is deemed a full interference pattern. When motor neurons are lost, fewer motor unit potentials are available to summate and the patient will not be able to produce numbers sufficient to fill the screen. The interference pattern is said to be reduced. Individual or discrete motor unit potentials may be observed when axonal injury is severe.[180]

The interference pattern associated with the loss of muscle fibers has been described previously. Low amplitude motor units fill the screen during only a minimal voluntary contraction.

The utility of interference pattern interpretation may be confounded by several variables. A normal patient may not produce a maximal contraction because of pain, poor cooperation, or poor coordination. Patients with severe sensory neuropathies may not be able to gauge the intensity of their muscle contractions due to loss of proprioceptive feedback. Upper motor neuron abnormalities such as spinal cord injury or stroke will also limit the patient's ability to generate a maximal contraction. Incomplete conduction blocks may produce discrete interference patterns and be misinterpreted as severe axon loss if not interpreted in conjuction with the rest of the electrodiagnostic examination.

Nerve Conduction Studies

Conduction velocity may be determined in either motor or sensory nerves. Although some nerves are not readily accessible to study, a large number, including cranial nerves and upper and lower extremity nerves, are available for testing. Electrical stimulation of a sensory or motor nerve results in a re-

cordable action potential. The potential is a summated response of the nerve fibers stimulated. In the case of motor NCSs, motor nerve fibers of a selected nerve are stimulated and the summated electrical response (CMAP) is picked up over a muscle innervated by that nerve. In sensory NCSs, sensory fibers of a selected nerve are stimulated at a point along the length of the nerve. An action potential, which represents the sum of the action potentials generated by these sensory fibers, is obtained with electrode pickup at another site along the course of the nerve.

Latency

Latency is the time in milliseconds between application of the nerve stimulus and the onset of the recorded evoked compound action potential. Thus, latency recordings reflect the speed of the fastest conducting fibers within a nerve. A latency is usually obtained with the most distal stimulation along a nerve. Latencies obtained from more proximal portions of the nerve may be erroneous due to changes in the fascicular composition of the nerve.

Conduction Velocity

Conduction velocity is determined by obtaining latencies from a distal and a proximal stimulation site. It is specifically calculated by dividing the distance between the two sites by the latency difference between the proximal and distal sites:

$$\frac{\text{Distance}}{\text{Time}} = \frac{\text{PD} - \text{DD (mm)}}{\text{PL} - \text{DL (ms)}} = \text{Conduction Velocity(m/s)}$$

where PL is proximal latency, DL is distal latency, PD is distance from site of proximal stimulation to recording electrode, and DD is distance from site of distal stimulation to the recording electrode.

Amplitude

Amplitude of a CMAP is primarily dependent on the number and density of innervated muscle fibers and their synchrony of firing. The integrity of the neuromuscular junction also affects the amplitude of the motor action potential. The SNAP amplitude is primarily dependent on the number of functioning large myelinated axons present.[183]

The amplitude of the CMAP and SNAP are also dependent on the distance between the site of stimulation and the actual position of the nerve, as well as on the intensity of stimulation. The greater the soft tissue thickness, the greater the distance between stimulation site and nerve, which frequently results in attenuation of the action potential amplitude due to incomplete nerve stimulation. If the recording electrode is not close to the nerve or muscle due to fat or other tissue, the signal will also be attenuated.

Duration

Duration of an action potential reflects the degree of variation of latencies between the fastest and slowest conducting fibers. Abnormal temporal dispersion, a reflection of dyssynchrony of conduction, occurs when there is an excessive difference between the fastest and slowest fibers. This finding is typically accompanied by a concomitant reduction in amplitude. If some of the nerve fibers are actually blocked rather than merely slowed by a disease process or by focal injury, the area of the action potential will also be reduced.

Utility of Electrodiagnostic Studies

The potential use of electrodiagnostic techniques to determine the location and severity of nerve injuries has been postulated as far back as World War I.[4] It has only been since the 1970s, however, that the equipment and general expertise in the field have improved to levels at which sensitive information might be provided consistently and reliably. Currently, electrodiagnostic techniques are the most objective and quantitative means of assessing nerve injuries.[184] The complex nature of nerve injuries and the subtleties of interpreting information derived from electrodiagnostic studies require that a physician with special training in the diagnosis and treatment of neuromuscular disorders plan, administer, and interpret such studies. Extensive training in neurophysiology and electrophysiologic techniques is essential to ensure that correct and appropriate diagnoses are obtained by the electrodiagnostic medical consultant.

Determination of Severity

The primary determinant of recovery following nerve injury is the severity of the injury. The predominant electrodiagnostic features observed during Wallerian degeneration are the progressive reduction in the amplitude of evoked potentials along the entire length of the nerve and the appearance of denervation potentials in affected muscles.[45] The electrodiagnostic findings observed following nerve

injury are dependent on three main variables: (1) the time elapsed from the injury, (2) the type of nerve injured, and (3) the length of the injured nerve distal to the focus of injury.

Following transection of a nerve, CMAPs and SNAPs cannot be elicited by stimulation of the nerve proximal to the focus of injury. Stimulation distal to the injury, however, will result in an action potential during a short period until axonal degeneration occurs. In humans, the ability to evoke a distal CMAP begins to wane 3 to 6 days following injury. In a recent study by Chaudhry et al,[45] an ulnar nerve injury at the elbow showed an amplitude drop of 10% at day 3 postinjury, a 90% drop by day 6, and loss of the ability to evoke an action potential at day 9. Diminution of SNAP amplitudes following axonal injury lag behind the loss of CMAP amplitudes by 2 to 3 days. On average, the ability to evoke a SNAP is lost between 9 and 11 days following injury. Following transection of the sural nerve at the calf, a SNAP amplitude drop of 20% was first noted at postinjury day 5, a 60% drop by day 9, and loss of the ability to evoke a SNAP at day 11.[45] The early failure of motor neurons to propagate an evoked potential distal to the site of injury is the electrophysiologic correlate of neuromuscular junction failure. The neuromuscular junction fails prior to axonal degeneration.[37]

The length of the distal stump also has a profound effect on the timing of the loss of ability to evoke an action potential. While the exact mechanism remains unclear, trophic support for the portion of the nerve distal to the focus of injury remains intact for a period of time following injury and is directly proportional to the length of the nerve distal to the focus of injury. Thus, the ability to generate an action potential distal to the site of injury will last for a longer period of time when the distal portion of the nerve is longer. For example, an injury to the ulnar nerve at the elbow with a length distal to the injury of 25 cm revealed a CMAP amplitude drop of 80% by day 6. An injury to the facial nerve, with a length distal to the site of injury of only 12 cm showed an 80% CMAP amplitude drop by day 3.[185] Injuries at more proximal regions along similar nerves will also show the same time differences. An ulnar nerve injury in the arm with a length of 45 cm distal to the focus of the injury may reveal a CMAP amplitude drop of only 30% by day 6 and not be lost until the 11th or 12th day. Electromyographic abnormalities suggestive of axonal injury also lag behind the onset of injury and are related to the length of the nerve distal to the site of injury. The onset of spontaneous activity closely parallels the loss of ability to evoke a SNAP.[184] For example, spontaneous activity may be seen as early as 7 days in facial muscles following axonal injury to the facial nerve. On the other hand, a very proximal injury at the root or plexus level may not manifest evidence of denervation by electromyographic evaluation for upwards of 21 days.

The timing of electrodiagnostic evaluations following nerve injuries may have a profound impact on the interpretation of the study. If NCSs are performed too soon, the ability to elicit a distal action potential may be misinterpreted as a less severe injury such as conduction block (see below) rather than an axonal injury. Inaccurate localization may also occur. If studies are performed during the window when the CMAP is lost but the SNAP is still obtainable, the findings may be interpreted to suggest that a lesion proximal to the dorsal root ganglion is present. A study performed later may reveal that the ability to evoke a SNAP has also been lost, supporting that the location of the injury is distal to the dorsal root ganglion, which is prognostically better than the more proximal lesion and may be amenable to surgical intervention.

One of the major applications of electrodiagnostic medicine is the quantification of each physiologic process that underlies the clinical manifestation of nerve injury. During early evaluations, physical examination tends to overcall the severity of an injury. Late in the course of reinnervation, trick movements may be learned by the patient as compensatory methods of overcoming persistent weakness. In this situation, findings on the physical examination can be misconstrued as greater return of neurologic function than is truly present.

As previously noted by Seddon,[31] it is extremely common to have the coexistence of axonal degeneration, conduction block, and conduction slowing with temporal dispersion following acquired nerve injuries. Analysis of nerve conduction parameters in conjunction with electromyographic findings allows the character of the injury to be determined. Following the initial delay, axonal injury leads to a drop in the amplitude of the evoked action potential along the entire length of the nerve (ie, during stimulation both proximal and distal to the focus of injury). The duration of the potential changes little. The area under the curve is also reduced, reflecting the loss of fibers adding to the summated action potential.

Conduction block is evidenced by a drop in the amplitude of the action potential during stimulation proximal to the site of injury when compared

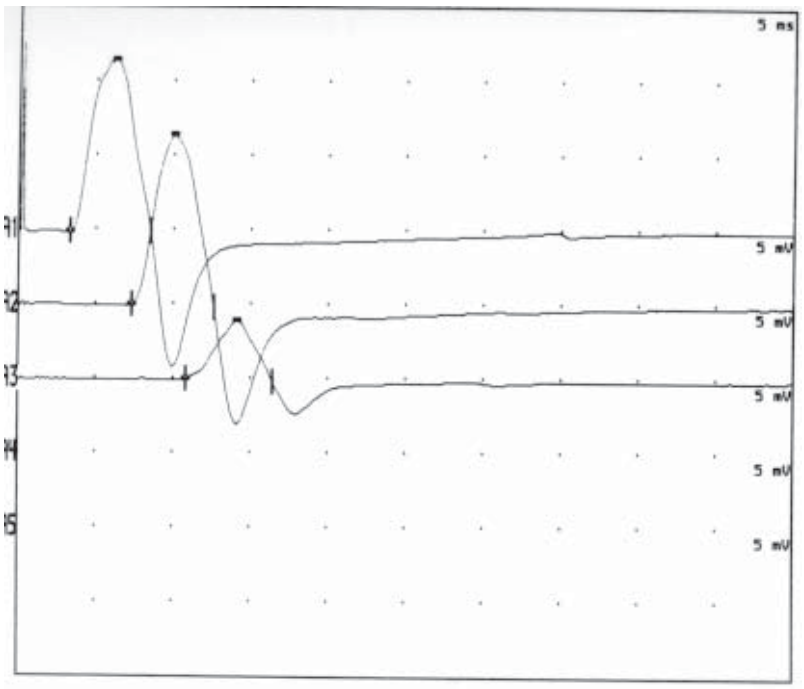

Fig 9-25. Nerve conduction study of the ulnar nerve revealing conduction block at the elbow. Note that both the amplitude and area are markedly decreased with little evidence of temporal dispersion or change in duration, compared to more distally evoked compound motor unit action potentials. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

to stimulation distal to the focus. A comparison of distal CMAP amplitude to proximal CMAP amplitude revealing an amplitude drop of greater than 20% has been suggested to be significant[186] (Figure 9-25). In pure conduction block, the duration of the potential does not change appreciably. Thus, a reduction in amplitude and area are observed.

Conduction slowing with temporal dispersion is the third type of injury that can be discerned. Conduction slowing with temporal dispersion suggests that not only the fastest fibers have been injured but all types of fibers. Differences in the speed of conduction increases the duration of the action potential. The decreased synchrony of firing, manifested by spreading of nerve fiber potentials over a greater timeframe, is reflected in a decrease in the amplitude of the potential as the potentials do not summate. The area under the curve, however, is not decreased because the number of fibers activated is relatively normal (Figure 9-26). Conduction slowing with temporal dispersion may be commonly seen during more insidious, cumulative types of trauma. It may also be seen during reinnervation, whether spontaneous or following surgical nerve repair.

Conduction block and conduction slowing with temporal dispersion are manifestations of demyelinating, neuropraxic injuries. Thus, electromyographic evaluations should not reveal spontaneous activity (Table 9-8). The reader is referred to the discussion on peroneal neuropathies for a clinical example of the electrodiagnostic determination of nerve injury severity.

Localization

The ability to localize nerve injuries requires an in-depth knowledge of neuroanatomy. Comprehensive understanding of dermatomal and myotomal distributions, anomalous innervations, innervations of specific muscles including nerve branch, nerve trunk, course through the plexus of origin, and nerve roots is essential for accurate localization. Electrodiagnostic localization may be especially helpful in combat-related injuries where injuries may occur practically anywhere along the length of the nerve and at multiple sites. Accurate localization also conveys the extent of injury, which may reflect functional recovery and prognosis. Among the factors that affect the results of both natural and surgical recovery is the location of the injury. More distal lesions are more likely to recover in comparison to more proximal injuries. Different nerves also

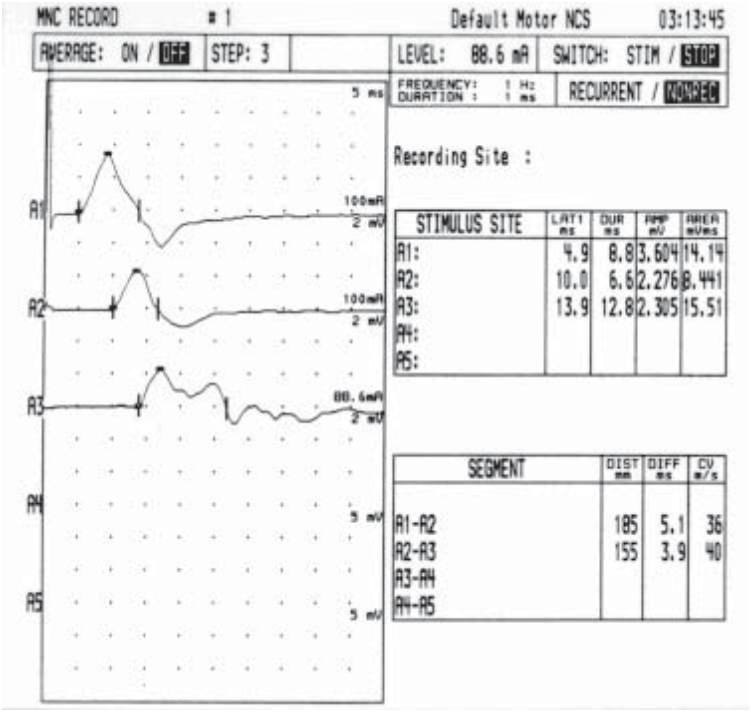

Fig 9-26. Nerve conduction study of the ulnar nerve revealing conduction slowing and temporal dispersion at the elbow. Note that while the amplitude is decreased compared to distally evoked compound motor unit action potentials (CMUAPs), the duration is increased and the area has remained essentially stable. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

TABLE 9-8

ELECTRODIAGNOSTIC PARAMETERS SUGGESTING SEVERITY OF NERVE INJURY

	Axon Loss	Temporal Dispersion	Conduction Block
Nerve Condution Study Parameters*			
Duration	No change	Increased	No change
Amplitude	Decreased	Decreased	Decreased
Area	Decreased	No change	Decreased
Electromyographic Study Parameters			
Spontaneous activity	Present	Absent	Absent
Reduced recruitment	Present	Possible	Present

*Comparison of proximal to distal compound motor action potentials (CMAPs). An increase in duration of greater than 15% and a reduction in amplitude of greater than 20% are considered abnormal when comparing proximal to distal CMAPs.

respond and repair differently when subjected to similar injuries. Radial nerves are reputed[187] to have the best recovery, followed by median and ulnar, and all usually fare better than injuries to nerves in the lower extremities. Lastly, recovery from injury to pure motor or sensory nerves is commonly better than from injuries to mixed nerves. The principles of localization, presented later in this chapter, dealing with more common areas of entrapment injuries can be readily applied to combat-related injuries.

Nerve conduction studies can be employed to differentiate nerve root lesions from more distal injuries. A detailed discussion can be found in the section concerning brachial plexus injuries. Conduction studies are also helpful to localize focal areas of conduction block or conduction delay. The section on ulnar nerve injuries provides a paradigm, including short segment incremental studies and the use of conductions along several different branches, that can be applied in the unique situations encountered with traumatic injuries. Involvement of multiple nerves, even those not clinically suspected by physical examination, can be elucidated by NCSs.

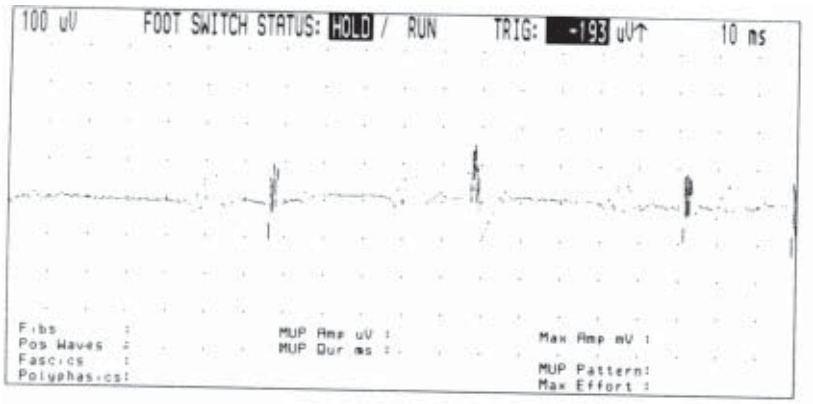

Fig 9-27. Electromyographic display of a low amplitude, long duration polyphasic motor unit potential. The presence of such a motor unit potential may suggest early reinnervation following peripheral nerve injury. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

Electromyographic techniques are especially helpful when the injury primarily involves the loss of axons. Evidence of denervation observed in muscles distal to the lesion with sparing of muscles innervated by the same nerve but proximal to the lesion help to strategically place the lesion. Examples of this can be found in sections discussing the differentiation of radial nerve from posterior interosseus nerve injuries and median nerve from anterior interosseus nerve injuries.

Monitoring Recovery

Electrodiagnostic studies can be used to follow both natural and postsurgical reinnervation. While reinnervation occurs through both collateral sprouting and regeneration, it has been suggested by Buchthal and Kuhl[188] that, in humans, the former is more important. Early evidence of reinnervation can be seen on EMG. Voluntary motor unit activity was observed[189] following end-to-end anastomoses 4 to 7 months after suture. Electromyographic activity was detected after 12 months when grafting was warranted.[189] When a partial injury has been sustained, voluntary motor unit potentials may be evident within several weeks after injury. Low amplitude, polyphasic, long duration motor unit potentials are seen initially and may predate the loss of denervation potentials (Figure 9-27). Single fiber EMG analysis provides a useful technique to explore the earliest signs of reinnervation. Locking one single fiber potential in time (triggering) allows for the assessment of the latency difference between the triggered single fiber action potential and the other single fiber action potential in the motor unit. This latency difference is termed the inter-potential interval (IPI). The tenuous nature of the neuromuscular connection in an immature motor unit is suggested by two findings. *Blocking* occurs when neuromuscular transmission fails intermittently and the action potential of the single motor fiber is not generated. *Jitter* is the variation of the IPIs. A small amount of jitter is seen in normal motor units. Jitter is increased in immature motor units due to fluctuations in the time needed for summation of endplate potential to evoke an action potential at the neuromuscular junction. As the new connections mature, conduction along the sprouted fibers will increase and the potential will become incorporated in the parent motor unit potential (Figure 9-28). Early on, the duration of the motor unit potential will be increased, but as reinnervation proceeds the duration will approach normal and the polyphasic nature of the unit will decrease. Summation will improve as the fibers fire more synchronously. As more fibers are now incorporated in the motor unit, the amplitude may become exceptionally large (Figure 9-29).

Nerve conduction studies may also be used to chart the course of recovery. Very low amplitude, temporally dispersed action potentials may be seen during the initial stages of reinnervation. Conduction velocity across the affected area may be exceedingly slow, in some cases less than 10 m/s. It has been estimated by Buchthal and Kuhl[188] that the action potentials of at least 40 fibers of greater than 7 μm in diameter need to summate before a compound potential of 0.02 μV with a conduction velocity of 10 m/s can be generated. This is felt to be the minimum parameters required to distinguish the potential electrodiagnostically from background noise. While conduction velocity increases and the amplitudes of evoked sensory and motor potentials improve, they commonly do not return to baseline values. Hodes et al[190] observed that even as far out as 12 to 42 months following partial transection, conduction velocities continued to range between 40% to 60% of normal. Similar findings were observed by Cragg and Thomas.[191] Conduction velocities plateaued at 75% of normal at 12 months following injury. Buchthal and Kuhl[188] and Donoso et

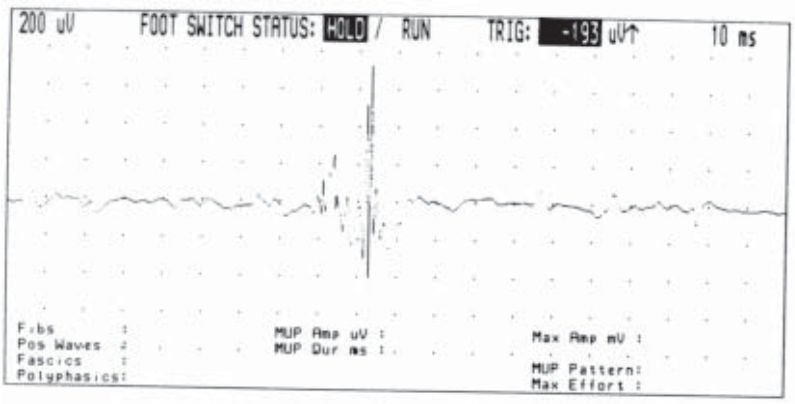

Fig 9-28. Electromyographic display of a normal amplitude, long duration polyphasic motor unit potential commonly observed as reinnervation becomes more stable and synchronization of muscle fiber firing improves. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

al[189] both observed the return of CMAPs 4 to 7 months following nerve suturing. Distal motor latencies improved over time but rarely returned to normal range. SNAPs were first obtainable in Buchthal and Kuhl's[188] study 4 months postsuturing and 7 months following nerve grafting. Only 54% of the Donoso et al[189] groups had detectable SNAPs as far out as 20 months. In both studies, even when SNAPs could be evoked, amplitudes rarely improved to better than 10% of normal.

While these data are intriguing, they become more important when related to the return of sensory and motor function. Return of sensory function does not correlate well with SNAP amplitude changes. According to Buchthal and Kuhl,[188] the ability to perceive tactile stimulation returns when approximately 1% of myelinated sensory fibers reinnervate. This correlated to 5 to 7 months after suturing when it did occur. After 28 months, tactile perception had improved; light touch could be discriminated from pinprick, but the site of stimulation could not be discerned. Three years following suturing, the patient began to localize stimuli. The lack of correlation between SNAP amplitude and return of sensibility can be explained by the temporal dispersion that persists after sensory nerve injury. Also, sensory potentials measure only the fastest and largest myelinated fibers. Thus, sensibility conveyed along small and unmyelinated fibers will not be included in the SNAP. As techniques become more refined in the future, it may be possible to monitor the area of the SNAP, which may better correlate with the return of sensory function.

In contrast to sensory function, a strong correlation between motor function and the amplitude of the CMAP has been determined. Donoso et al[189] observed that a return of CMAP amplitude to over 40% of normal correlated with a return of motor power in the affected muscles to good (4/5) or normal (5/5) strength. The ability of the reinnervated muscle to generate near normal power when fewer than normal CMAPs are obtained and discrete recruitment patterns are observed on EMG, reflects the remarkable resilience of the motor unit. So long as 50% to 75% of motor neurons remain intact, functional recovery may be achievable through collateral sprouting and regeneration.[184]

Limitations of Electrodiagnostic Studies

Nerve conduction studies and EMG convey electrophysiologic abnormalities reflecting periph-

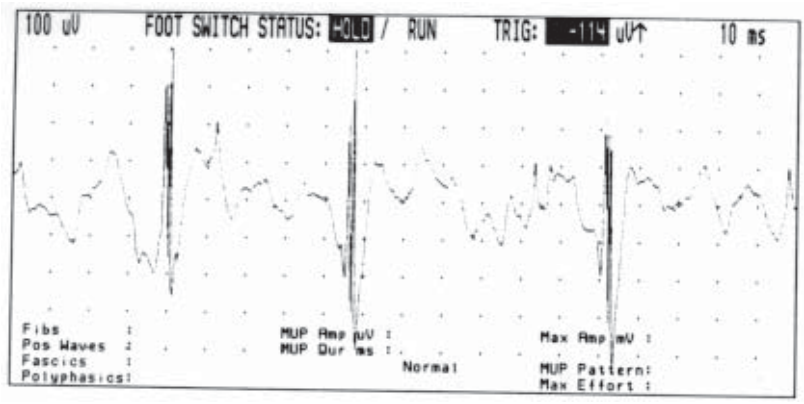

Fig 9-29. Electromyographic display of a normal amplitude, normal duration polyphasic motor unit potential. This type of unit potential may suggest old stable reinnervation. Commonly, as summation improves, these potentials may be of large amplitude. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

eral nerve pathology. It should be understood, however, that these studies cannot be used to infer precise pathological changes (eg, findings may suggest axonal injury but ischemic axonal injury cannot be differentiated from stretch-related axonal injury).

Subtle differences in severity or improvement cannot truly be differentiated by electrodiagnostic studies. Findings suggestive of demyelination can be discerned from those consistent with axonal injury, and to some extent the relative roles of these processes can be inferred. Yet, the hierarchical levels of axonal pathology as outlined by Sunderland (second to fifth degrees) cannot be truly appreciated electrodiagnostically. The ability to evoke an action potential during stimulation proximal to an injury does convey the integrity of the nerve. After an appropriate time delay, action potentials evoked distal to the lesion also reflect continuity. The inability to obtain action potentials does not confer the loss of anatomic continuity. Thus, while electrodiagnostic findings may suggest a very severe axonal injury, they cannot be used to conclude or confirm complete disruption of the nerve.

REHABILITATIVE MANAGEMENT OF PERIPHERAL NERVE INJURIES

General Principles

Rehabilitation of peripheral nerve injuries is absolutely essential to ensure optimal functional recovery. It has been observed that partially transected nerves often recover faster and more completely than those requiring surgical repair.[188] As the vast majority of nerve injuries are in continuity or partial transections, surgical decisions are usually postponed for 8 to 12 weeks.[192,193] Such a hiatus allows for more definitive assessment of severity, resolution of neuropraxia, and potentially, the maturation of the distal nerve stump.[7] Postponement of conservative therapy during this period, however, may lead to secondary injury, which will hamper subsequent functional return. Sharp lacerations are commonly explored within 72 hours as early repair in those situations leads to relatively good outcomes.[192] Yet, even when sharp laceration is the known causative agent, only 15% to 20% are observed to be complete at exploration.[193] Thus, in the vast majority of cases, conservative therapy is the first line of treatment and the cornerstone for any further intervention.

The extent of functional recovery hinges on the extent of injury. Surprisingly, a small number of residual motor units can lead to near normal motor function. Edds' study[47] of reinnervation in partially denervated muscles suggested that reinnervation and axonal sprouting could lead to useful function when 50% to 75% of axons were lost. No functional recovery occurred when greater than 80% of motor units were lost.[9] This finding is consistent with the poor prognosis seen in patients with acute inflammatory demyelinating polyneuropathy, whose electrodiagnostic evaluations reveal CMAP amplitudes less than 20% of the lower limit of normal.[194]

Rehabilitative measures do not improve the rate of reinnervation or reverse the end organ changes occurring specifically from denervation. They are indispensable, however, in the protection of the affected limb from secondary injuries derived from contractures, disuse weakness, edema, pain, and poor positioning. Regardless of whether surgical intervention is warranted early, after a delay, or not at all, these same principles are also employed to facilitate the attainment of maximal functional capacity in the face of residual sensory or motor impairment.

Maintenance of Range-of-Motion

Physiology

The upregulation of collagen synthesis following denervation portends the ominous development of myogenic contractures. Sunderland noted that the development of fibrosis and contractures is so prejudicial to restoration of function if and when reinnervation takes place, that it should be kept to a minimum by appropriate therapy.[9]

Increased flexibility results primarily from stretching the connective tissues within and around the muscle and tendon rather than the contractile elements of muscle. Connective tissues will progressively shorten when not opposed by a stretching force and will elongate when challenged with a constant stress.[195] Stretching, elastic or plastic, occurs when there is linear deformation of the fibers that leads to an increase in length. Elastic stretch occurs when elongation is produced with loading, followed by a recovery to resting length when the load is removed. Plastic stretch occurs when the elongation is maintained after removing the load.[196] Plastic elongation is the type necessary to improve flexibility of connective tissue shortened by edema, injury, or unopposed or imbalanced muscle tension. Four factors influence plastic deformation: (1)

amount of force, (2) type of force, (3) duration of force, and (4) tissue temperature.

Lehmann et al[197] documented that prolonged loading of a tendon leads to significantly greater increases in length than short term loading. This confirmed a more clinically based study by Kottke et al[195] who reported that short duration, high force stretching leads to high tensile resistance and little change in length, while prolonged, low force stretching leads to plastic elongation. Temperature increases to a therapeutic range of 40°C to 43°C decrease the viscosity properties of the connective tissue and maximize the effect of stretching.[197] In the Lehmann et al study,[197] stretching during heating led to significantly greater length increases than stretching after heating was completed.

Clinical Application

The efficacy of passive motion to prevent contractures in the face of peripheral nerve injury is well established (Figure 9-30). Pollock et al[198] brought affected joints through their full range passively for one set of 10 repetitions each day. The end range of each repetition was maintained for several seconds. No contractures developed in either the treated or control groups prior to 30 days postinjury. By 90 days, however, nearly 50% of the control group had developed contractures averaging 35°. Only 15% of the treated group developed contractures. The average severity of contractures in this subgroup was 20°. Institution of passive range-of-motion retarded the development of contractures, diminished their severity, and facilitated their resolution. Passive stretching has also been the principal method of averting contractures in neuromuscular disease. While the experience may not be perfectly applicable to isolated lower motor neuron injuries, the basic premise remains the same. Vigos[199] suggested that passive range-of-motion be initiated immediately and should be undertaken at least once or twice daily. Each session should include 10 to 15 repetitions with the end range held for a 10 to 15 count. Kottke[200] set similar parameters to be done twice a day. As strength improves, the patient should be encouraged to actively assist with range-of-motion. Once strength improves to better than antigravity, active range-of-motion should be continued by the patient even outside of formal therapy sessions.

The intensity of the stretch is crucial to the maintenance or improvement in range-of-motion. In situations where no inflammation is present and sensation is intact, muscles can be stretched vigorously. Stretching can be to just past the point of pain but the pain should abate rapidly with discontinuation of the stretch. Traumatic nerve injuries are commonly complicated lesions, coexisting with bone, vascular, and muscle injuries. Range-of-motion in these situations should be done only by highly trained therapists or physicians. Complications of overstretching, including hemorrhage, myositis ossificans, heterotopic ossification, and disruption of supporting structures, need to be obviated at any cost.[201]

Fig. 9-30. Passive range of motion of the phalangeal joints, administered to a patient sustaining radial nerve injury with weakness of the wrist and finger extensor muscles.

When stretching to correct connective tissue contractures in situations void of inflammation, edema, or hypesthesia, the elevation of tissue temperature should be accomplished through heating modalities. The choice of modality depends on the depth of the tissue to be heated. Deep muscles will require ultrasound diathermy. More superficial tissues such as the finger flexors will be adequately heated by superficial modalities such as moist heat. Concomitant, gentle, prolonged stretching should be applied across the joint once elevated temperatures are attained. The use of heat as an adjunct to stretching is relatively contraindicated in anesthetic regions; regions with impaired vascular supply; and in acutely injured, edematous areas.[201]

Strength Maintenance and Improvement

Physiology

Exercise therapy to preserve or enhance motor strength is integral to the comprehensive rehabilitation of persons sustaining peripheral nerve injuries. As previously discussed, over 60% of weakness experienced during such injuries can be attributed to disuse atrophy.[75] Incomplete reinnervation, whether from intrinsic factors such as muscle fibrosis or extrinsic factors including aberrant innervation by inappropriate axons or lack of reinnervation secondary to neuroma formation, may lead to residual weakness. Much of the understanding of the effects of strengthening partially denervated and reinnervating muscles is derived from experience during the poliomyelitis epidemics of this century. Related studies assessing the utility of strengthening exercise for neuromuscular disease may also be applicable. Herbison and colleagues[202–206] specifically tackled the questions of appropriate intensities, duration, and timing of strengthening exercises following peripheral nerve injury.

Clarifying Overwork Injury

The use of resistance training to improve muscle strength in neurologically intact individuals is unquestioned in the lay and medical literature. Its use in the management of peripheral nerve injury has not been as resolute. Concerns regarding provocation of declines in strength caused by overstressing immature neuromuscular connections or by over taxing a sparsity of competent motor units have been raised. Reports[207,208] of strength loss in individuals with predominantly lower motor neuron injuries undertaking strenuous activity have been largely anecdotal. Lovett[207] surveyed victims of the 1913 polio epidemic, commenting on three persons who presumably lost strength following prolonged overstressing of individual muscles. A highly cited source alleging overwork weakness by Bennett and Knowlton[208] commented on four cases of anterior poliomyelitis and one cervical spinal cord injury.

In contrast, numerous studies have confirmed significant improvements in strength in those who have sustained lower motor neuron injuries. The results of the study by Delorme and colleagues[209] clearly demonstrate that muscles weakened by poliomyelitis and normal muscles respond similarly to progressive resistance exercise. Exercise protocols comprised 2 exercises of 3 sets, 10 repetitions, with a 1-minute rest between sets, done 4 days per week. Similar results were obtained following a regimen of 20 maximum repetitions done at 1-minute intervals, 3 times per week for 5 to 10 weeks.[204] No muscles showed a permanent reduction in power. Of note, it was also observed that slight increases in power frequently accompanied great increases in work capacity.

While the exact pathophysiologic etiologies of postpolio syndrome have yet to be determined, overwork weakness has been touted as a possible cause. Even in this situation, many studies have established that monitored exercise protocols employing submaximal progressive resistance exercises can improve strength and do not precipitate untoward declines in strength.[210–213] A study by Agre and Rodriguez[214] showed that when rest periods were interspersed between isometric contractions of 40% isometric peak torque rather than doing the same amount of work constantly to fatigue, the ability to recover strength after activity was improved.

These polarized conclusions may reflect differences in the classification of weakness. Overwork weakness is defined as a prolonged decrease in both absolute strength and endurance of a muscle subsequent to a period of work. Impairment must be longstanding.[208] Because most of the literature supporting the verity of overwork weakness are case reports and studies of not significant longitude, it is difficult to judge whether they meet the definitional criteria. In several cases in which both upper and lower motor neuron injury was evident, alternative causes for a loss of strength may have also been at play. Lastly, these studies did not show histologic evidence of injury such as central nuclei, fiber splitting, or fiber degeneration.

The type and intensity of activity undertaken more than the activity itself may also explain the disparity. In those cases manifesting weakness af-

ter activity, individuals had engaged in prolonged, strenuous activities lasting hours at a time and were for the most part unsupervised. In the prospective studies, episodic bursts of high resistance maximal or submaximal exercise were performed for short periods each day. Evidence supporting this impression can be found in a series of experiments by Herbison and colleagues.[202–206] Wistar rats were subjected to sciatic nerve crush injuries and either soleus or plantaris tenotomies. Exercise was started at 2 weeks postinjury in one group and 3 weeks postinjury in the other group. The findings revealed increases in muscle weight and protein only in the group postponed 3 weeks. A mild decrement was observed in the group starting only 2 weeks postinjury. Thus, the group participating in the more intense exercise program developed a loss of muscle protein and weight. It should be understood that the exercise undertaken by these animals was prolonged and continuous. Also, all rats were autopsied at 4 weeks postinjury. It is unclear as to whether the changes seen were transient or persistent. It was concluded that a critical relationship existed between the number of contractile units and the intensity of exercise. When the intensity becomes too great for the fibers available, normal physiologic processes may become pathologic.[204]

A follow-up study[205] looked at a less intense form of exercise, swimming. In this situation, denervated rat muscles showed progressive increases in muscle weight and protein concentrations, directly proportional to exercise time and duration. As with most therapeutic applications in medicine, the prescription of strengthening exercise for peripheral nerve injuries must fall within its therapeutic range. The muscle must be overloaded to the extent warranted to overcome disuse atrophy and eventually to improve strength. The stress must also be below levels which may provoke injury.

Clinical Application

The determination of the therapeutic range for strengthening exercise varies directly with the current strength of the individual and the motor units involved. Commonly, electromyographic evidence of reinnervation precludes the onset of clinically observable muscle contraction. In these situations, surface EMG biofeedback might be employed to provide auditory and visual cueing to enable controlled firing of the motor units. Parameters based on the amplitude of the evoked potentials may be used to set goals regarding intensity and duration.[215–217]

Patients with incomplete injuries or those in the process of reinnervation may exhibit strength at all ends of the spectrum. When weakness precludes the voluntary movement of the affected joint through the full range-of-motion against gravity (ie, trace (1/5) or poor (2/5) strength), isometric strengthening exercises are most appropriate. An isometric contraction is a muscle contraction without movement across the joint. Peak isometric strength is the force that can be exerted against an immovable object.[218] In cases of trace and poor strength, the weight of the limb in the face of gravity provides adequate resistance.

While most of the literature regarding strengthening focuses on techniques to attain maximal improvement, Muller[219] assessed the basic requirements to maintain or modestly improve strength in skeletal muscle. All therapeutic prescriptions are founded on four variables that can be manipulated: (1) mode, (2) intensity, (3) frequency, and (4) duration. Mode is the type of exercise to be undertaken. Intensity is the percentage of the individual's maximum capacity. Frequency refers to the number of sessions over a period of time, and duration determines the length of each individual session.

Strength Maintenance

In the early stages following peripheral nerve injury, efforts should be made to maintain strength and overcome disuse atrophy. Commonly, these patients have sustained multiple trauma. Surgical procedures may be impending or the patient may still be in the intensive care unit. Maintenance of strength even at this juncture is vitally important as it will facilitate the rehabilitation process in the future. Surprisingly, very small amounts of exercise are needed to maintain strength. Muller[219] observed that 1 maximal isometric contraction, held for 1 second, performed once a week led to minimal but significant strength improvements. Spacing isometric contractions to 1 every 3 weeks caused moderate decrements from baseline strength. One maximum isometric contraction of 1-second duration done every other week generated mild increases in strength, which slowly returned to baseline by the 14th week.

Strength Improvement

At these modest levels of activity, increases in any variable (ie, intensity, frequency, or duration) will lead to increased strength enhancement. Goals may be attained more rapidly by employing a more rig-

orous program. Modulation of the duration of contraction has led to mixed results. Muller[219] observed no difference in strength outcomes when 1 isometric contraction was held for 1 or 6 seconds. Another study showed comparable strength gains when one maximal isometric contraction was held for 5 or 45 seconds. This paradox was explained by Mundale,[220] who showed that during 1 maximal isometric contraction, maximum tension could be sustained for no longer than 1 second. Yet, when variations in intensity and duration were assessed, differences in each variable affected outcomes. It was observed that one submaximal contraction, 65% of maximum of 1-second duration, performed once a day caused strength gains of 2.5% per week. One daily maximal contraction of 1-second duration improved strength by 3.33% per week. One daily maximal contraction of 6-seconds duration increased strength by 4% per week. Lastly, the greatest improvement of 5% per week was achieved by performing one set of 5 contractions, each 6 seconds in duration, spaced every 2 minutes. A total of 30 seconds of contractions were completed daily.[219] The minimal increase in strength gain observed when one 1-second and one 6-second maximal isometric contractions are compared may reflect additive gains during submaximal contraction. While maximal intensity can only be maintained for 1 second, less powerful contraction during the subsequent 5 seconds may still provide strength improvement.

Once antigravity strength has been achieved, isometric exercise may no longer suffice as the sole method of strengthening. A major drawback of isometric exercise is that strength gains are limited to the joint angle or muscle length at which the muscle is exercised. There is little transference of strength to dynamic activities.[221] It is reasonable to progress to exercises that will enable the production of strength increases throughout the entire range-of-motion. Isotonic exercises involve moving a constant load through a full range-of-motion with or without a changing velocity of movement (Figure 9-31). DeLorme and Watkins[222] are credited with establishing resistive exercises as rehabilitative tools to increase strength.

As previously mentioned, a specific application of DeLorme and Watkins'[222] method had been to improve strength of acute poliomyelitis victims. His program involved the use of progressive resistance exercises with increasing loads. Each individual determined the ten repetition maximum (TRM) for each muscle to be strengthened. The TRM is the greatest weight that can be lifted through the full range of motion 10 times only. Subjects would then perform 3 sets of 10 repetitions daily at 50%, 75%, and 100% of the TRM with 2 minutes of rest between sets. Each week a new TRM would be determined. The drawbacks to this exercise included difficulty in completing the final set of exercises due to fatigue and the fact that full motor unit recruitment was only accomplished during the last set.

The Oxford technique[223] reversed the DeLorme regimen by ordering the exercise sets with 100% TRM first, followed by 75%, and then 50%. With this regimen, fatigue caused by the 100% TRM set is offset by lower loads on the second and third sets.

Both of these techniques were based on the premise that only high weight, low repetition exercises produce strength gains. DeLateur and Lehmann[218] observed that strength gains may be obtained even at levels as low as 30% of maximum voluntary contraction. While there is little evidence to substantiate the provocation of overwork weakness by exercise of intensities described, it should be understood that submaximal exercise levels will improve strength.

Close evaluation of the patient is paramount. Herbison suggests monitoring strength on a daily

Fig. 9-31. Exercising knee extensors. Isotonic strengthening exercises utilize the movement of a constant load through a full range of motion.

basis (Gerald J. Herbison, M.D., Professor and Director of Research, Department of Rehabilitation Medicine, Thomas Jefferson University Hospital, Philadelphia, Pasylvania: Telephone conversation March 1993). Decreases in strength on the day following therapy or a digression in the ability to perform activities of daily living (ADL) should be an admonition that an excessive amount of exercise was attempted. Goals should be lowered and not progressed until they can be achieved without provoking unsatisfactory effects.

These exercise programs can also be used with isokinetic training. Isokinetic strengthening employs equipment that provides a set rate of velocity against which the person can exert maximum torque (Figure 9-32).[218] The velocity chosen becomes very important in isokinetic strengthening.[224] Training at slow velocities generates the greatest torque. Strength gains occur at the training velocity and can be seen when testing is done at even slower velocities. If strength testing is done at faster velocities than the training velocity, no evidence of increased strength is observed.[225]

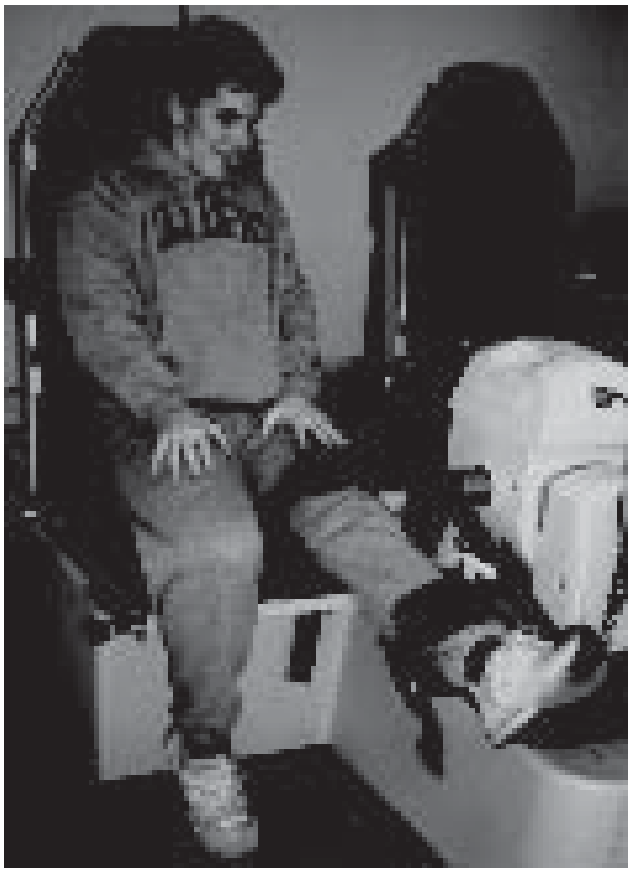

Fig. 9-32. Exercising knee extensors. Isokinetic strengthening requires the use of a machine, which limits the velocity at which a person can generate maximum torque.

A benefit to isokinetic training is that objective measures of torque production can be obtained that may reveal subtle persistent deficits. The biggest drawback is the reliance on special equipment which may not be readily available. There is no evidence that isokinetic strengthening is any more effective than isotonic training. Given their simplicity and universal availability, isotonic exercises are more commonly used.

Edema Control

Physiology

Efforts to ameliorate the development of edema must be expeditious. Traumatized limbs sustaining immobilizing nerve injuries are at great risk for the development of dense, restricting, connective tissue contractures. Edema facilitates the process of fibrosis in the affected region. Histological evidence of fibrosis may be seen as early as 4 days postinjury.[226] Extravascular fluid is also a ripe medium for bacterial growth and infection, which may seriously compromise any likelihood of subsequent functional recovery.

Clinical Application

Several methods to decrease edema, including range-of-motion exercises, elevation, external compression, and massage need to be integrated into an effective treatment plan.

Active range-of-motion. The maintenance of equilibrium between intravascular and interstitial fluid volumes is due in part to the contraction of skeletal muscle.[168] When possible, active motion performed by the patient through the full range-of-motion should be encouraged. Functional electrical stimulation may be a helpful adjunct in situations where strong muscle contractions cannot be performed because of neuropathic weakness (Figure 9-33).

Elevation. Utilization of gravity to decrease edema accumulation is also extremely helpful. Ideally, the affected limb should be kept above the level of the heart to ensure the best passive return of fluid to the central circulatory system. Great lengths should be taken to facilitate the most advantageous positioning throughout the day. When the patient is in bed, the limb should be supported by pillows or foam wedges. Distal portions of the extremities

Fig. 9-33. Electrical muscular stimulation, applied to elicit muscle contraction and decrease the pooling of edematous fluid.

may warrant splinting in a functional position. At the wheelchair level, foam wedge laden arm troughs should be used to support the upper extremities. Affected lower extremities should also be elevated. Elevation above the level of the heart is difficult. At a minimum, the distal aspect of the limb should be kept higher than the proximal end. Dependent positioning should be avoided as much as possible.

External compression. External vascular support from gradient pressure elastic sleeves or stockings should be used continuously when edema is present. Isotonic compression gloves can also be used to combat swelling of the digits and dorsum of the hand.

Intermittent pneumatic compression may also be beneficial. The limb is placed in an inflatable sleeve. Pressure is applied intermittently. Pressures around 30 mm Hg can be applied for 5 to 6 hours in 3-minute cycles (ie, 2.5 min on, 30 s off). Alternatively, 60 mm Hg of pressure can be used for 30 to 60 minutes.[227] It is recommended that the maximum pressure applied should be less than the patient's diastolic blood pressure to prevent vascular occlusion and possible ischemic injury. During each session, the limb should be optimally elevated at a 45° angle. Gradient pressure garments should be applied immediately following treatment to help maintain gains made during mechanical compression.

Centripetal wrapping of the limb is another method commonly used to decrease the accumulation of interstitial fluid. Coban, a semiadhesive, elastic tape is applied distally, and circumferentially wrapped in a proximal direction (Figure 9-34). The limb is elevated for 5 minutes after application. Active range-of-motion is encouraged immediately following its removal. The treatment is repeated 3 times daily.

Massage. According to Knapp, the single best indication for the prescription of massage is reduction of swelling associated with trauma.[228] Effleurage (stroking) and petrissage (compression) methods are applied in a retrograde manner, mobilizing tissue fluids and assisting in proximal return to the intravascular circulation.

Orthotic Management

The rationale for application of orthoses to the nerve-injured limb are numerous. During the early phases of treatment, orthoses are used to limit motion, allowing for healing of the traumatized tissue. Proper positioning helps to prevent the development of contractures in unopposed muscles and overstretching of weakened muscles. Protection against joint and ligamentous injuries to insensate

Fig. 9-34. Centripetal wrapping of the digits with Coban to decrease the accumulation of edema.

and weak limbs can be achieved through orthotic application. In the later phases, static and dynamic orthoses can be fabricated to enhance or substitute for lost function. The principles of orthotic management for musculoskeletal as well as nerve injuries are explored extensively in Chapter 11, Orthotics for the Injured Soldier, which is dedicated solely to the topic.

Hyperesthetic Desensitization

Hyperesthesia is a typical consequence of nerve injury. The instinctive perception of this abnormal sensory experience as injurious compels many patients to protect and not use the affected limb. The ominous consequences of disuse and immobility will further compound the loss of function and mounting anxiety if left untreated.

In 1976, a formal desensitization treatment program was developed at the Downey Hand Center.[229] Integration of psychological and physical principles provided an important foundation. The cognitive-behavioral psychological technique of systemic desensitization has been used in the treatment of emotional and anxiety disorders since the late 1950s.[230] Patients proceed through a hierarchy of anxiety-provoking situations. Each level is experienced repeatedly until the patient is comfortable with the situation. Step-wise progression continues until ultimately patients are able to cope effectively in the face of the circumstances they most fear. Hyperesthetic desensitization marries this concept with the overload principle, which contends that the body must be stressed beyond its current level of activity to adapt and enhance function.[231]

Clinical Application

Patients are exposed to increasingly irritating textures and vibrations through three 10-step modalities. The dowel modality incorporates textures ranging from moleskin to Velcro hooks, wrapped around the ends of ½-in. dowels, which are rubbed, rolled, and tapped on the hypesthetic region (Figure 9-35). Immersion into buckets of particles ranging from cotton to plastic squares is used in the particle modality (Figure 9-36). Exposure to increasing frequencies of vibration ranging from 23 to 100 Hz is used in the vibration modality (Figure 9-37, Table 9-9). Each modality is used three to four times per day. Progression is based on the patient's experience. Each day the patient chooses the level felt to be slightly irritating but tolerable for a 10-minute

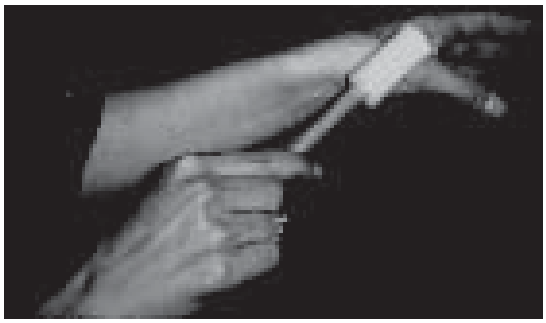

Fig. 9-35. Texture covered dowel, rolled, rubbed, and tapped on hypersensitive region.

Fig. 9-36. Limb immersed in buckets of variably textured particles.

Fig. 9-37. Variable frequency vibrator providing stimulation to hypesthetic area.

TABLE 9-9

HIERARCHICAL STIMULATION FOR DESENSITIZATION THERAPY

Dowel Textures	Immersion Particles	Vibration (Hz)
Moleskin	Cotton	83 near area
Felt	Terrycloth	83 and 23 near area
Quickstick	Dry rice	83 near area, 23 intermittent on area
Velvet	Popcorn	83 and 23 intermittent on area
Semirough	Pinto beans Cloth	83 intermittent, 23 continuous
Velcro loops	Macaroni	83 continuous, 23 intermittent
Hard T-foam	Plastic wire Insulation pieces	100 intermittent, 53 intermittent
Burlap	Small BBs	100 intermittent, 53 continuous
Rug backing	Large BBs	100 continuous, 53 continuous
Velcro hooks	Plastic squares	Unlimited vibration

Source: Barber LM. Desensitization of the traumatized hand. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD, eds. *Rehabilitation of the Hand*. 2nd ed. Princeton, NJ: CV Mosby; 1984: 493-502.

period. In the Downey Hospital study,[229] progression to the next tolerable level usually occurred 2 weeks after initiation of treatment. The length of treatment averaged 7 weeks. Attainment of the highest level of tolerance of the dowel, particle, and vibration modalities was achieved respectively by 62%, 54%, and 22% of patients who had sustained crush injuries to the upper limb. Over 90%, however, noted that their perception of abnormal sensation no longer precluded their return to gainful employment.

The mechanism by which desensitization is achieved remains unclear. It has been suggested that mollifying fears of pain and disability is the most beneficial aspect of desensitization. Down regulation of sensitized nociceptors through stimulation of a different set of sensory fibers analogous to the gait theory, postulated by Melzack and Wall,[232] may provide a physiologic explanation. Neither of these hypotheses has been adequately explored in clinical trials.

Sensory Reeducation

The inherent difference between physical impairment and disability is a basic tenet on which much of rehabilitation medicine is based.[233] Frequently, great functional gains can be achieved despite minimal changes measured grossly on physical examination.

Physiology

The focus of sensory reeducation following nerve injury or repair is to retrain the patient to utilize what residual function remains to its fullest capability. The reserve capacity of sensory perception is not well established. It has been consistently observed, however, that a heightened sensitivity to certain modalities can be achieved as compensation for the loss of another. For example, normal two-point discrimination has been established as 3 to 5 mm.[234] Yet, those with impaired vision, who use tactile sensibility as their main mode of communication and interaction with the environment, may have two-point discrimination as fine as 1.5 mm.[235]

The success of sensory reeducation is founded on a sound understanding of which sensory modalities are essential for function and what level of intensity needs to be perceived. Moberg[236] noted that the presence of most sensory modalities tested on the standard neurologic examination do not correlate with function. Of the tests performed, perception of less than 12 mm on the Weber two-point discrimination test best predicted the ability to perform precision grip. Dellon[237] observed, however, that many persons with static two point discrimination greater than 12 mm functioned extremely well. Further experiments revealed that perception of moving two-point discrimination at the finger tips of less than 6 mm correlated with the ability to identify objects correctly. Sensory fibers which mediate the sensation of touch can be divided into two categories: slow-adapting and fast-adapting. Slow-adapting fibers transmit afferent volleys proportional to the intensity of pressure. They convey information regarding static, constant touch. Fast-adapting fibers respond not to levels of pressure but to abrupt differences and oscillations, experienced as dynamic or moving sensation. Thus, an integration between both static and dynamic pressure sensibility is needed for function.

Clinical Application

Sensory reeducation can be divided into two main categories: protective and discriminative reeducation.

Protective Sensory Reeducation

Patients with profound sensory loss are at great risk for inadvertently injuring the affected limb. Reeducation in this situation is compensatory and education of the patient is paramount. The patient gains an enhanced awareness of situations and stimuli which may place the limb in danger. Problems such as soft tissue injuries and Charcot joints, stemming from lack of proprioception and an inability to self-monitor motor function, and blisters and skin breakdown from overuse and abnormal sudomotor activity are brought to the patient's attention and are monitored scrupulously.[238]

Discriminative Sensory Reeducation

Wynn Parry[239] claims to have established the first formal sensory education program in 1966. Dellon and Jabaley[240] expanded on the concept and have developed a comprehensive approach based on specific levels of sensibility. Pain and temperature sensibilities are the first to return after nerve injury or repair. As discussed, aside from their important protective actions, they do not contribute to the functional capability of the limb. Low frequency (30 Hz) vibratory and moving-light-touch perception are next to return. Over time, constant touch and high frequency (256 Hz) vibratory perceptions return.

The early phase of discriminatory sensory reeducation centers on improving the ability to distinguish moving-light-touch from constant touch. This phase can begin only when low frequency vibration and moving-light-touch can be perceived; when resuturing has been performed, this is usually 4 to 6 months following surgery.[240] The exercises incorporate other sensory inputs to facilitate and reinforce greater awareness of the tactile sensation. Patients first observe visually the intensity, duration, and mode of touch. They then close their eyes and concentrate on the sensation itself. Lastly, they verbalize the sensory experience. Each session is brief, lasting only 10 to 15 minutes because of the great mental concentration needed to perform the exercises correctly. A minimum of one daily session is recommended but two or three are ideal.

Late phase discriminatory sensory reeducation encourages recovery of texture discrimination and object recognition (Figure 9-38). Commencement of the late phase follows return of the ability to differentiate constant and moving-light-touch or moving-light-touch discrimination of less than 6 mm. Fabrics, objects of variable size with rough and smooth edges, and household objects are manipulated in a similar format followed in the early phase. Visual reinforcement is used initially, followed by focused concentration on the tactile perception, and then verbalization.

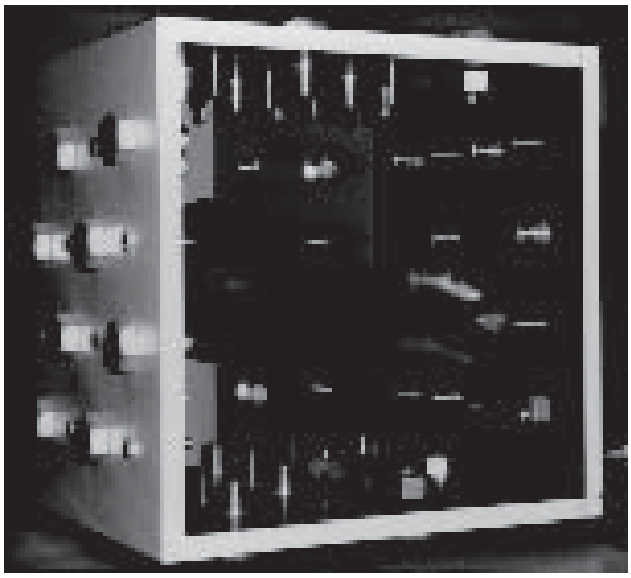

Fig. 9-38. The final phase of sensory reeducation involves sight obstructed manipulation and discrimination of common objects.

As discrimination improves, patients are advanced to activities such as picking objects from a bucket of sand, which requires the ability to discern an object from its background. Ultimately, ADLs with vision occluded are attempted.[238]

The benefits of the addition of sensory reeducation to surgical repair of nerve injuries to the upper extremities are encouraging and have become an integral part of hand therapy following nerve injuries. Several studies using Highet's grading system[241] (Table 9-10) have shown that over 80% of patients undergoing median or ulnar nerve repairs improved function to a grade of S3+ or better.[239,240] These findings strongly support the notion that functional return can be improved by repetitive training of sensory awareness.

Pain Management

Pain is a common perceptual sequela experienced by persons sustaining traumatic nerve injuries. Its

TABLE 9-10

CLASSIFICATION OF SENSORY RECOVERY

Grade	Level of Sensibility Function
S0	Absence of sensibility
S1	Recovery of deep pain sensibility
S1+	Recovery of superficial pain sensibility
S2	Recovery of superficial pain and minimal tactile sensibility
S2+	Same as S2 but with hypersensitivity
S3	Recovery of superficial pain and full tactile sensibility with resolution of hypersensitivity
S3+	Good stimulus localization but imperfect 2-point discrimination
S4	Full recovery of all sensibility

Source: Waylett-Rendall J. Sensibility evaluation and rehabilitation. *Ortho Clin North Am*. 1988;19:43–56.

aversive nature strongly deters interactions with noxious stimulation. The experience of pain during the aftermath of an injury can be extremely distressing to the patient. It is unusual for painful perceptions associated with nerve injuries to evolve into chronic, disabling experiences, per se.[242] They may, however, hamper attempts at rehabilitation and ultimately limit functional restitution. Aggressive early palliative treatment is an integral part of any comprehensive rehabilitation program.

Physiology

Asbury and Fields[243] divided neuropathic pain into two categories: dysesthetic pain and nerve trunk pain. Dysesthetic pain is associated with increased afferent input arising from hypersensitive, damaged, or regenerating nociceptive fibers. The sensation may be constant, intermittent, jabbing, or lancenating and is characterized as burning, tingling, crawling, or electric in nature. The distribution may be difficult for the patient to discern but usually falls within the cutaneous sensory distribution of the injured nerve. In neuromatous formations, the pain may be localized at the focus. Mechanical stimulation provokes discharge of the hypersensitive nerve endings.

Nerve trunk pain occurs in the face of more proximal injuries such as nerve root injuries and plexopathies. It is suggested that increased firing of nociceptive fibers within the sheaths of intact nerve trunks is the underlying cause. The sensation is described as a continuous deep aching and is occasionally noted to be knife-like or "like a toothache." It may follow a dermatomal distribution but is more commonly noted as an ache involving muscles within the myotomal distribution.

A third type of pain may be experienced following complete transection of a nerve. In contrast to the two types of pain previously discussed, deafferentation pain is not dependent on damaged but rather on intact nerve fibers. It has been postulated that the loss of sensory input leads to abnormal somatotopic reorganization. Changes in the receptive fields at the spinal cord, thalamic, and cortical levels may lead to altered, painful perception as normal sensory input is relayed to inappropriate, reorganized regions of the central nervous system. Phantom limb pain is a specific type of deafferentation syndrome that follows amputation. A detailed discussion of the topic, including pathophysiology and potential treatment approaches, which are also applicable to deafferentation pain following complete nerve or nerve lesions, can be found in Chapter 4, Rehabilitation of the Lower Limb Amputee, in *Rehabilitation of the Injured Combatant*. Vol. 1, the first of the two Textbook of Military Medicine rehabilitation books.

Clinical Application

Early attempts at management of neuropathic pain were alarmingly unsuccessful. Maruta and colleagues[244] noted a success rate of only 20% during a 1-year follow-up of patients with chronic neuropathic pain. Current interventions that integrate modulation of both peripheral and central nociceptive transmission hold the promise of greater palliation of this complex and difficult problem.

Pharmacological Intervention

Antidepressants

To the authors' knowledge, no studies have been conducted that confirm the efficacy of antidepressants in the treatment of traumatic, neuropathic pain. Their usage has been intuitive, based on research showing effective relief of pain caused by diabetic neuropathies and postherpetic neuralgia.[245–248]

The mechanism by which antidepressants provide analgesia is poorly understood. It has been hy-

pothesized that chronic pain and depression commonly coexist.[249,250] Blockade of norepinephrine and serotonin re-uptake is postulated to compensate for relative deficiencies of these neurotransmitters. Improvement of the affective disorder is felt to enhance the ability to modulate and tolerate nociceptive inputs. An alternative theory suggests that pain reduction is an epiphenomenon of the sedation that is a common side effect of many antidepressant medications.[251]

A direct analgesic effect is supported by several clinical findings. The onset of pain relief usually occurs within the first 2 to 3 weeks,[245,246] while a large percentage of patients will not manifest improvement of depressive symptoms before four weeks of tricyclic antidepressant use.[252] The dosages needed to modulate pain are also lower than those needed for antidepressant effects. Several studies[245,248,249] have noted analgesic effect at doses ranging from 75 mg to 150 mg per day, while the recommended minimal dose for an adequate trial of a tricyclic antidepressant drug for depression is 150 mg per day and can be increased to as high as 300 mg.[252] Lastly, pain modulation has been confirmed in several placebo-controlled studies that also controlled for depressed patients.[245,248,253] A recent meta-analysis of 39 placebo controlled studies of antidepressant-derived analgesia in chronic nonmalignant pain concluded that the intrinsic analgesic properties and not the sedative or antidepressive effects of the drugs best explained the efficacy of this class of drug in pain modulation.[254] Several researchers have postulated that antidepressants may relieve pain through their ability to weakly bind to central opiate receptors[255]; however, the direct inhibitory action of enhanced levels of norepinephrine and serotonin in descending, pain-modulating pathways is more plausible. The relative importance of the individual neurotransmitters remains controversial. Several earlier studies suggest that those antidepressants that predominantly block serotonin re-uptake produce superior analgesic effects.[255] A more recent placebo-controlled study was undertaken comparing the effects of desipramine and amitriptyline (tricyclic antidepressants possessing both noradrenergic and serotonergic properties) with fluoxetine, a relatively pure serotonin re-uptake inhibitor. Intrinsic analgesic activity in the face of neuropathic pain was observed only in those drugs exhibiting central noradrenergic re-uptake inhibition. Pain relief was no better than placebo in the fluoxetine group. Max et al[245] postulate that norepinephrine is likely the primary metabolite responsible for pain relief, while serotonin is augmentative at best.

The choice of a specific antidepressant medication for the treatment of neuropathic pain is colored more by the side effect profiles of certain drugs than any proven difference in their effectiveness (Table 9-11). Placebo-controlled trials for amitriptyline,[245,247,248] desipramine,[246] nortriptyline,[256] and imipramine[257] have all shown good results in the relief of neuropathic pain. In certain situations, side effects such as sedation may be a corollary to the analgesia provided. Undesirable effects including insomnia, orthostatic hypertension, and anticholinergic effects (eg, dry mouth, constipation, urinary retention) are the most common reasons for choosing or switching to a certain drug. It should be stressed that these drugs are relatively contraindicated for use in persons with cardiac dysrhythmias, closed angle glaucoma, or urinary retention. Con-

TABLE 9-11

HETEROCYCLIC ANTIDEPRESSANT DRUGS USED FOR NEUROPATHIC PAIN

		SIDE EFFECTS			
Drug	**Daily Dosage (mg)**	**Sedation, Hypotension**	**Orthostatic Effects**	**Anticholinergic Dysrythmic Effect**	**Cardiac**
Amitriptyline	50–150	+++	++	+++	Yes
Desipramine	50–150	–	++	+	Yes
Imipramine	50–150	+	+++	++	Yes
Doxepin	50–150	++	++++	++	Yes
Nortriptyline	50–150	+	+	+	Yes
Trazadone	50–150	+++	++	0	Low

Source: Potter WZ, Rudorfer MV, Manji H. The pharmacologic treatment of depression. *N Engl J Med*. 1991;352:622–642.

sultation with an internist should be considered before administration in these instances.

It is recommended that treatment be started at a low dose. Many authors suggest an initial dose of 10 mg to 25 mg for tricyclic antidepressants.[248,253] The dose should be increased every 2 to 3 days, guided by the onset of untoward side effects. Ideally, a dose in the 75 mg to 150 mg range should be achieved. Lower doses have been observed to be beneficial in other pain disorders but, to date, only the higher doses have been used in studies documenting neuropathic pain relief.

Other antidepressant drugs including Doxepin and Trazodone have been shown to be effective in the treatment of chronic pain. No studies have been undertaken as yet to explore their utility in the treatment of peripheral neuropathic pain.

Neural Membrane Stablizers

Anticonvulsant drugs have been observed in noncontrolled trials to be effective in the treatment of shooting, stabbing pain but not background burning, aching pain.[258] They have also served as adjuncts to antidepressants in situations where neuralgic pain is refractory to antidepressant agents alone.[255] Swerdlow and Cundill[258] report rates of pain relief in posttraumatic nerve injury ranging from 64% to 92%. (Five anticonvulsants and one antiarrhythmic drug have been evaluated.)

Carbamazepine. Carbamazepine has been noted to be the most efficacious of the group and is chemically related to amitriptyline. It also causes the most problems in regard to side effects. Dizziness, unsteadiness, drowsiness, and gastrointestinal upset are commonly experienced during initial treatment. Many patients will develop a mild leukopenia, which should be monitored closely but does not necessitate cessation of the drug unless white blood cell levels fall below 3,500 cells/mm^3.[255] Rarely, but ominously, carbamazepine can induce aplastic anemia and agranulocytosis. Dosing should start low, 100 mg per day, and increase by 100 mg every 2 days with a goal of 600 mg per day in divided doses (ie, 200 mg three times daily [tid]). Administration can be increased to as high as 1,800 mg per day, although this is rarely tolerated or warranted. Close monitoring of all hematopoietic cell line levels and liver associated enzymes is essential.

Clonazepam. Clonazepam, a benzodiazepine, revealed similar effectiveness for neuropathic pain relief as carbamazepine but with a much better side effect profile.[258] Its major side effect is sedation, which may be potentiated by concomitant use of tricyclic antidepressants. Clonazepam use is relatively contraindicated in persons with significant renal insufficiency as its active metabolites are excreted by the kidneys. Persons with respiratory compromise may also be placed at risk secondary to enhancement of salivary secretions. The most significant drawback to its use, however, is the habituation and dependency which may develop in prone patients, as with any other benzodiazepine. Dosing should start at 0.5 mg per day and may be increased gradually, 0.5 to 1.0 mg every third day. An average therapeutic dose ranges from 2 to 10 mg per day divided into three doses.

Gabapentin. Recently a new anticonvulsant, gabapentin, has been shown to have potent analgesic properties, particularly in the treatment of neuropathic pain.[259–261] Gabapentin is structurally related to the neurotransmitter gamma-aminobutyric acid (GABA) but does not interact with GABA receptors and does not convert into GABA. In fact, the exact mechanism by which gabapentin produces analgesic effects remains unclear. The greatest advantage of the use of gabapentin for neuropathic pain is its remarkably low level of toxicity and side effects. The most common side effects are somnolence, dizziness, and ataxia. These are not common and were reported primarily when gabapentin was used adjunctively with other antiepileptic drugs. Dosing begins at 300 mg once daily and is increased by 300 mg every 2 to 3 days up to 300 mg tid. Given its relative safety and paucity of side effects, gabapentin has become the first choice among anticonvulsants for the treatment of neuropathic pain.

Phenytoin. Phenytoin is proposed to act at the level of the neurolemma Na^+ channels, enhancing membrane polarization and stabilization. A narrow therapeutic window limits its utility. Side effects commonly encountered include sedation, confusion, and ataxia. Ironically, a sensory polyneuropathy has been observed with chronic usage. Dosing usually begins at 100 mg twice a day and is increased by increments of 100 mg every 2 to 3 days to a daily level of 300 to 500 mg. A common schedule is 200 mg twice a day.[255] If symptoms have not improved by 3 weeks, the dose should be tapered off, as higher doses will only lead to toxicity.

Valproic acid. Valproic acid possesses similar analgesic effects as phenytoin.[258] Given its potential to induce profound, fulminant hepatic failure without warning, it is not commonly used for neuropathic pain relief, especially when more effective agents are available.

Mexiletine. It has been known for some time that intravenous administration of anesthetics such as

lidocaine produce pain relief independent of their ability to block neuronal conduction.[262] Their usefulness in the control of persistent pain has been limited by the logistical difficulties of intravenous infusion of these relatively short-acting agents. The use of mexiletine, an oral lidocaine analogue has recently been reported in the effective management of diabetic neuropathic pain,[263–265] neuromatous pain,[266] and peripheral nerve injury associated pain.[267]

Mexiletine's analgesic properties stem from its ability to block Na^+ channels, stabilizing membrane ionic flow. Experimental studies in rats have revealed two possible sites of action. A peripheral effect was noted by Chabal et al.[266] Sensitivity to mechanical stimulation and spontaneous firing of injured axons was significantly reduced in sciatic neuromas. Woolf and Wiesenfeld-Hallin[268] suggest a central inhibitory effect at the level of the dorsal horn.

A graded response occurs with mexiletine administration, directly related to the dose. Most authors suggest starting at 150 mg per day and increasing by 150 mg every third day until 450 mg per day is achieved (150 mg tid). If satisfactory pain relief is not achieved, the dose can be increased by increments of 150 mg per day at weekly intervals to a maximum level of 750 mg per day (approximately 10 mg/kg/d) in divided doses.[265,267]

The most common side effects experienced by patients are gastrointestinal upset with nausea and occasionally vomiting. Dizziness, tremor, and a general perception of nervousness are also not uncommon. As with other antiarrhythmic agents, the potential for exacerbating underlying cardiac dysrhythmias is very real and potentially dangerous. Thus, all persons should undergo electrocardiographic evaluation prior to the commencement of, and periodically during, treatment with mexiletine. Its use is contraindicated in persons with second- or third-degree A-V block, not using a pacemaker. It is also strongly discouraged for treatment of pain in persons with ventricular dysrhythmias, congestive heart failure, or hypotension.

Substance P Inhibitors

Capsaicin. A naturally occurring alkaloid isolated from capsicum peppers, capsaicin was originally found to relieve the dysesthetic pain associated with herpes zoster,[269,270] and more recently, painful diabetic neuropathy.[271–274] No studies have assessed its potential use in traumatic nerve injuries. Capsaicin's analgesic action is derived from its ability to deplete substance P, a pain-modulating neurotransmitter from the terminals of nociceptive C-fibers. Initial exposure to capsaicin causes excitation of the thermal receptors, manifest as a burning sensation that lasts for 30 to 60 minutes. Repeated exposure, however, leads to the desensitization of these same fibers. Double-blind, vehicle-controlled studies evaluating not only pain relief but also functional capacities of persons with painful diabetic neuropathies revealed significant improvements along all parameters in the treatment groups.[272,274]

Several dosing schedules have been documented in the literature. Anecdotal improvement was seen using topical application of 0.25% capsaicin cream on the affected area tid.[271] Controlled trials have utilized a regimen of 0.75% capsaicin cream applied 4 times per day.[272–274]

As previously noted, the most common adverse effect of capsaicin use is a burning sensation that lasts for 30 to 60 minutes following application. In a study by Simone and Ochoa,[275] all subjects reported a decrease in the intensity and duration of discomfort over several weeks. Patients with severe hypesthesia, unfortunately, may find this early irritative effect intolerable, despite its self-limited course.

Concerns have been raised regarding the potential neurotoxic effects of long-term capsaicin use. High dose systemic administration in neonatal animals has caused irreversible destruction of nociceptive C-fibers.[276] Clinical doses of topically applied capsaicin, however, have not been observed to pose any threat to the integrity of sensory nerve fibers.[277] Apprehension about its use in sensory impaired individuals has also been debated. Capsaicin significantly raises the threshold for heat-pain detection. Thresholds for cold, touch, and mechanical stimulation are not affected.[275] Therefore, despite adequate preservation of protective sensation, patients with impaired baseline sensation should be counseled to take added precautions to prevent thermal exposure and injury. Alteration in heat detection is reversible and returns to baseline with cessation of capsaicin application.

Nonsteroidal Antiinflammatory Drugs

Nonsteroidal antiinflammatory drugs (NSAIDs) are the foundation of many pain management regimens. Their role in the treatment of neurogenic pain has been explored in only a paucity of studies. A single placebo-controlled experiment showed ibuprofen and sulindac to be more effective than placebo in the treatment of painful diabetic neur-

opathy.[278] Several anecdotal reports have also presumed benefit from their use.[279,280]

Following cell injury, mediators of vasodilatation and inflammation are released. Prostaglandins and leukotrienes, metabolites of arachidonic acid, are among the most potent and omnipresent inflammatory instigators. Prostaglandins produce pain by stimulating and sensitizing C-fiber nociceptors. C-fiber activity provokes further vasodilatation and release of inflammatory mediators. Thus, local injury leads to the cascading development of pain and inflammation at sites distant from the focus, termed neurogenic inflammation. Nonsteroidal antiinflammatory drugs' primary mode of action is the inhibition of prostaglandin production from their arachidonic acid antecedents. Nonsteroidal antiinflammatory drugs inhibit the activity of the enzyme cyclooxygenase, which catalyzes the addition of oxygen molecules to arachidonic acids to form prostaglandin precursors.[281]

There are currently over a dozen NSAIDs approved for use in the United States. Interestingly, response to any single agent is unpredictable. The class of agent also has little bearing on the success of pain moderation. The choice of a specific drug is based on side effect profiles, the potential for adverse effects, dosing schedules, and ultimately, cost. Excellent reviews of the pharmacologic properties and physiologic effects can be found in several texts, to which the reader is referred.[281,282]

In addition to the benefits gained from prostaglandin inhibition, many of the adverse effects of NSAIDs can also be ascribed to the impediment of their production. Gastritis and peptic ulceration are associated with the loss of mucosal protection afforded by prostaglandin-E_2. Nephrogenically produced prostaglandins act as endogenous angiotensin II inhibitors, blunting their vasoconstrictor and antidiuretic activity.[63] Patients with severe hypertension or congestive heart failure may be at risk of symptom exacerbation caused by NSAID prostaglandin inhibition. Platelet aggregation inhibition prolongs the bleeding times of patients taking most NSAIDs, with the exception of the nonacetylated salicylates. Nephrotoxicity and rarely hepatotoxicity have occurred with chronic usage. Nonsteroidal antiinflammatory drug use is also relatively contraindicated for treatment of patients with severe allergies or reactive airway disease. Prostaglandin inhibition shunts arachidonic acid precursors toward the 5-lipoxygenase pathway. Leukotrienes, the slow-reacting substances of anaphylaxis, are the ultimate product and may exacerbate symptoms.

Experimental Therapeutics

Experimental treatment with clonidine and calcium channel blockers have proved to be effective pain relievers in some cases, but in others there appears little relief was achieved.

Clonidine. Clonidine, an alpha$_2$-adrenergic agonist, has been reported to decrease pain associated with cancer,[283] arachnoiditis,[284] and herpes zoster.[285] Its precise action is unclear. Possible mechanisms include postsynaptic inhibition at the level of the spinal neurons, presynaptic modulation of nociceptive fiber activity, or inhibition of peripheral or central sympathetic activity.[286,287] A study[286] of the efficacy of transdermal clonidine (0.3 mg/d) for painful diabetic neuropathy failed to show a statistically significant effect. It was suggested by the authors, however, that reproducible pain relief was achieved in a subgroup and that the sample size and study design may have negatively impacted on the study outcomes.

Calcium channel blockers. Studies exploring the effects of calcium channel blockers on nociception are in their infancy. Kavaliers[288] noted that the administration of diltiazem or nifedipine augmented the endogenous production of enkephalins and raised pain thresholds. Calcium channel blockade has also been shown to potentiate the effects of exogenously administered opiates.[289] Gurdal and colleagues[290] showed potential antiinflammatory and nonopioid antinociceptive effects. Nicardipine was found to be highly effective, decreasing pain responses in rats by upward of 90%. Only partial results were obtained with verapamil and diltiazem. The role of cellular calcium ion flux has also been implicated in the actions of tricyclic antidepressants. Administration of nifedipine in conjunction with imipramine has been shown to enhance analgesic effects in Wistar rats.[291] Further controlled trials in human subjects are expected for these drugs and may expand the alternatives in the management of pain.

Electrical Stimulation Intervention

Transcutaneous electrical nerve stimulation (TENS) has been used to modulate pain for more than two decades. Despite controversies regarding mechanisms of action and efficacy, its use in the management of neuropathic pain has become nearly common. Few controlled studies have been performed examining its effectiveness in the face of peripheral nerve injuries. Rates of pain relief ranging from 50% to over 80% have been noted in uncon-

trolled trials.[292,293] Two methods of application are commonly used.

Conventional TENS

Conventional TENS employs high frequency (60 to 200 Hz) impulses of low intensity. Pulse widths range from 2 to 50 μs.[294,295] It is hypothesized that this afferent stimulation inhibits nociceptive inputs at the presynaptic level. Sjolund observed temporary diminution of transmission from nociceptive C-fibers in Wistar rats during peripheral application of TENS. The response was graded. Decreased firing was more pronounced in the 80 to 100 Hz range than at lower frequencies.[296]

The onset of analgesia is virtually immediate, following application of conventional TENS and rapidly abates when stimulation ceases. Of electrotherapeutic modalities available, this is undoubtedly the best tolerated and can be used continuously for hours at a time.

Acupuncture-like TENS

Low-frequency (0.5 to 10 Hz) stimulation at an intensity provoking muscle contraction is utilized in this form of TENS. Pulse widths range from several hundred to 1,000 μs.[294,295] In contrast to conventional TENS, acupuncture-like TENS analgesia has been linked to increased release of endorphins along descending central pain modulating pathways.[296] Acupuncture-like TENS is applied for 20 to 30 minutes. The onset of analgesia is delayed, occurring 30 minutes following completion of the stimulation. Pain relief may last for hours to days. The intensity of stimulation is poorly tolerated by many patients. Its use is frequently reserved for patients not responding to conventional TENS and those with predominantly deep aching pain.

Technique

Electrode placement and stimulation parameters continue to be as much art as science and herein lies the difficulty assessing the efficacy of this modality. Responsiveness to analogous applications may vary widely between individuals with similar pathologies. Electrodes can be placed in any number of configurations. A common method is to apply the electrodes on the periphery of the painful region with stimulation in a parallel or diagonal direction across the painful site. Electrodes can also be placed above or below the painful region. Stimulation along the proximal aspect of the nerve, in a noninjured region or even at the paravertebral levels, has been advocated (Figure 9-39). Pain relief has even been observed with placement of the electrodes on the contralateral limb.[294] Rarely is direct application on the painful region helpful or tolerated by the patient. Once electrodes are secured, the patient using conventional TENS is instructed to increase the intensity until a subtle tingling sensation is perceived. An inability to decrease pain perceptions by 50% or more within 30 to 60 minutes warrants repositioning of the electrodes and a trial at the new position.[295]

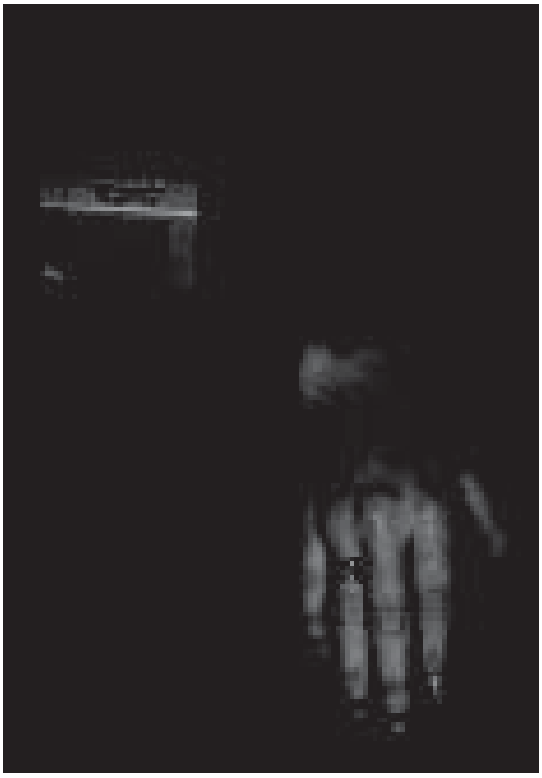

Fig. 9-39. Conventional transcutaneous electrical nerve stimulation (TENS) applied above the level of injury, on the edges of the sensory distribution.

Complications

Medical problems arising from the use of TENS are exceedingly rare. Skin irritation from the electric current, the tape used to secure electrodes, or the electrodes themselves are the most common complaint. These can be easily obviated through the use of self-adherent, disposable electrodes and repositioning the electrodes during subsequent applications. The use of TENS is contraindicated in patients with demand-type pacemakers. The TENS

pulses may be misinterpreted by the pacemaker as heart beats and may inadvertently inhibit or overdrive the pacemaker.[297] It is also suggested that electrodes not be placed on the anterolateral region of the neck to avoid hypotension and bradycardia from stimulation of the carotid body and vagus nerve.

Electrical Muscle Stimulation

Enhancing regeneration of injured nerve fibers and reversing the effects of denervation atrophy have been the rationale for stimulating denervated muscle for nearly a century. There is little consensus in the literature, however, supporting its efficacy,[298] and in fact, several studies[43,299] suggest that it may ultimately impede the regenerative process. Much of the confusion stems from the disparate methods used to study its utility. Variables including the duration, intensity, frequency, and mode of application are far from uniform, differing in practically every study. Subjects, too, range from small amphibians to humans. Unfortunately, size and species do not account for the variability as polarized conclusions have been drawn within species groups.

Aside from the lack of conclusive evidence corroborating its effectiveness, a more basic question needs to be raised. Even if we accept the biased premise that electrical stimulation does retard atrophy and enhances nerve regeneration, are these benefits clinically relevant? If methods are extrapolated from animal studies, a substantial amount of therapy time must be dedicated to the administration of electrical stimulation. Time and labor focused on preventing secondary complications and improving motor and sensory function will be sacrificed. Stimulation levels producing tetanic contractions have been necessary in those studies reporting good outcomes. The loss of innervation requires the application of strong currents to achieve this effect. It is unlikely that many patients will be able to comply with such a regimen for any length of time.

The benefits of electrical stimulation of denervated muscle are equivocal at best. Compelling evidence clearly establishing its usefulness has yet to be presented. Its utility as a component of the rehabilitative treatment armamentarium can not be justified at this time.[9,294,298]

CAUSALGIA (COMPLEX REGIONAL PAIN SYNDROME, TYPE-II)

A unique pain syndrome associated with peripheral nerve injuries was first described in 1864 by Mitchell, Morehouse, and Keen,[300] physicians for the Union Army during the Civil War. In their monograph *Gunshot Wounds and Other Injuries of Nerves*, they established the first description of causalgia:

> It is a form of suffering as yet undescribed and so frequent and terrible as to demand from us the fullest description. In our early experience of nerve wounds, we met a small number of men who were suffering from a pain which they described as "burning" or "mustard hot" or as "a red hot file rasping the skin." In all of these patients and in many later cases, this pain was an associate of the glossy skin.… The part itself is not alone subject to an intense burning sensation, but becomes exquisitely hyperaesthetic, so that a touch or a tap of the finger increases the pain. Exposure to air is avoided by the patient with a care which seems absurd and most of bad cases kept the hand constantly wet, finding relief in the moisture rather than in the coolness of application.[300]

Causalgia (literally: burning pain) specifically applies to a symptom complex of burning pain, hypesthesia, vasomotor instability, and dystrophic changes occurring after injury to a major peripheral nerve or plexus.[301] The terms causalgia and reflex sympathetic dystrophy (RSD) have been used interchangeably in the literature. Recently, the International Association for the Study of Pain (IASP) has developed a revised taxonomy for these disorders. The overall term, Complex Regional Pain Syndrome (CRPS), requires the symptom complex described above for causalgia to be applicable. CRPS-Type I corresponds to RSD and occurs without a definable nerve lesion. CRPS-Type II corresponds to causalgia and requires the presence of a definable nerve injury.[302] The term sympathetically maintained pain (SMP) describes the presentation of continuous burning pain with mechanical allodynia, following a history of physical trauma in the painful area that is relieved by sympathetic blockade.[303] Sympathetically maintained pain can occur in association with either CRPS-Type I or Type II.[302]

The incidence of CRPS-Type II in nerve-related injuries has varied during different conflicts. A rate as high as 32% was originally reported during the Civil War.[300] More recent statistical revisions have lowered that rate to closer to 15%. Mayfield[304] reported that 4% of nerve-injured soldiers developed CRPS-Type II during World War II. Kirklin et al[305] noted an incidence of 2% during the same conflict,

while series collected independently by Nathan[306] and Sunderland and Kelly[307] documented incidences of 14% and 12%, respectively. Rothberg et al[308] observed an incidence of 1.5% during the Vietnam War from 1964 to 1973. In two recent Middle East conflicts, rates closer to those observed during World War II have been documented. The assessment by Jebara and Saade[309] of nerve injuries sustained from 1975 to 1985 during the Lebanese Civil War revealed an incidence of almost 6%. Dillingham et al[3] documented 8% of all nerve injured soldiers evaluated by U.S. Army Physical Medicine and Rehabilitation Services as manifesting symptoms of CRPS-Type II during the Persian Gulf War.

While CRPS-Type II may occur after injury to any nerve, those nerves carrying the lion's share of sympathetic fibers are more commonly associated. In the upper extremity, injuries to the brachial plexus or median nerve are overwhelmingly involved. In the lower extremity, strong associations with sciatic or tibial nerve injuries have been established. Injuries to these nerve trunks accompany 83% of all cases of CRPS-Type II.[310]

Cardinal Characteristics

The vague definition of CRPS has led researchers to further clarify the salient features of this symptom complex. Four cardinal characteristics have been identified: (1) pain, (2) sensory abnormalities, (3) autonomic instability, and (4) trophic changes.

Pain

The pain associated with causalgia is undoubtedly the most disabling component. Classically, it is described as an intense burning sensation. It is nondermatomal in distribution and usually involves the distal aspect of the limb. Cases have been reported in which the pain spreads proximally and even to other limbs.[311] The onset of this type of pain quickly follows the infliction of the inciting wound. A compilation of series from World Wars I and II noted 89% of cases manifest symptoms within the first week following injury.[310] With time, the pain may change to a deep gnawing, aching experience. The pain may be at any intensity but commonly is severe enough to disrupt sleep and encumber the affected limb.

Sensory Abnormalities

Aberrant sensory perceptions are commonly experienced by patients. *Hyperalgesia* describes a lowered threshold to painful stimulation. Noxious stimuli that might normally be perceived as merely uncomfortable are felt to be excruciatingly severe. Another altered sensibility commonly experienced, termed *allodynia,* is the perception of pain from innocuous stimulation such as mechanical or thermal stimuli. Not uncommonly, patients describe unbearable pain from movement of the fine hairs on the limb by the sheets on the bed or a breeze blowing through the room. *Hyperpathia* may also be experienced. In this situation, there is an increasing sensitivity to repetitive stimulation. For example, a finger tap may not be painful initially, but as it is repeated, tolerance decreases and the once tolerable stimulation becomes less bearable. This amplification can be explosive. There is commonly after-sensation and radiation of the discomfort.[312]

Autonomic Instability

Alterations in the function of tissues innervated by the sympathetic nervous system help to differentiate CRPS-Type II from more common neuropathic pain syndromes. Sympathetic cell bodies are located in the intermediolateral gray matter of the spinal cord, extending from the eighth cervical level to the second lumbar level. The myelinated preganglionic axons synapse at the paravertebral ganglia. These white rami may traverse several levels of ganglia before synapsing. Acetylcholine is released at the terminals of all autonomic preganglionic fibers. The postganglionic axons are unmyelinated C-fibers that run with the anterior spinal motor nerves. The majority of postsynaptic fibers release the neurotransmitter norepinephrine. However, sudomotor fibers controlling sweat glands release acetylcholine.[313] Decreased sympathetic activity may be observed very early on, manifest by vasodilatory changes of warmth, erythema, and edema. Hypohidrosis is also observed as vasomotor and sudomotor dysfunction usually coexist. After several weeks to months, sympathetic hyperactivity may also occur. Cool, pale, cyanotic, sweaty limbs, reflecting vasoconstriction and hyperhidrosis, may be discerned.

Trophic Changes

Changes in the integument occur early. Gradual thickening and coarsening of the skin and the development of pitting edema are observed within days to weeks of the initial injury. Hair and nail growth is initially accelerated. In later stages, the skin thins and becomes taut and shiny. Nails be-

come brittle, thickened, and pitted. Ultimately, hair and nails may fall out.

Muscles, ligaments, and tendons shorten and begin to develop contractures. The synergistic effects of immobility due to pain and the presence of edema spur this devastating evolution. As contractures progress and muscles weaken from disuse, joint stiffening and ankylosis may occur.

Bony changes are also seen incipiently and can be seen on bone scan within the first 2 weeks. Patchy, periarticular osteoporosis appears, usually at the metacarpophalangeal (MCP) or metatarsophalangeal (MTP) joints within weeks. Increased vascular flow, possibly related to abnormal sympathetic activity, leads to more generalized involvement. With time, it may become difficult to differentiate diffuse osteoporosis associated with causalgia from that merely secondary to immobility related to pain.

Clinical Staging

DeTakats'[314] descriptive analysis of five cases of RSD (CRPS-TypeI) in 1937 laid the groundwork for further codification of the course of the syndrome. Bonica[315] later grouped the signs and symptoms of untreated cases into three chronological stages. The utility of such staging should be viewed cautiously. Commonly, different signs progress at variable rates. It may become difficult to absolutely classify the severity of the syndrome. The evolution of CRPS-Type II runs along a spectrum, and therefore, the staging system is at best a gestalt view of syndrome severity. Dogmatic application of the staging paradigm may lead to the exclusion of true cases.

First Stage (Acute)

Within days to weeks of the inciting injury, the constellation of pain perceptions and abnormal sensibilities previously described begin. The pain is amplified by emotional stressors, auditory and visual stimulation, and especially by movement of the affected limb. A sodden, pitting edema develops distally. The skin is red, warm, and dry; hair and nail growth is accelerated. During the very early part of the first stage, no abnormalities may be seen on roentgenography. Later, the onset of periarticular osteoporosis is seen. Increased uptake on all three phases of technetium 99m bone scanning is seen in the periarticular regions of the small joints of the affected limb. The acute stage may last for days and resolve spontaneously. Alternatively, it may be recalcitrant, persisting for upward of 6 months.[316]

Second Stage (Dystrophic)

Between 3 and 6 months after onset, the signs and symptoms may evolve into the dystrophic stage. Pain and abnormal sensibility remain similar to that seen during the first stage. The integument becomes cool, pale, and cyanotic. Hair growth wanes and nails become thickened, brittle, and ridged. Edema takes on a firm, nonpitting consistency. Muscle atrophy begins as do contractures of all the soft connective tissues. The spotty osteoporosis seen during the later phase of the first stage becomes more generalized, commonly affecting the epiphyseal regions of the bones. Technetium 99m bone scans reveal normalization of the blood velocity and pool phases with persistently increased uptake during delayed fixation. The second stage can last for many months.[316]

Third Stage (Atrophic)

During the last stage, severe, atrophic changes resistant to treatment develop. Pain and sensory abnormalities may remain steady or rarely decrease in intensity. The skin becomes cool, smooth, glossy, and pale. The principal change however, is atrophy. Marked wasting of muscle and subcutaneous fat occurs, leading to taut stretching of the skin superficially over the osteoporotic bones. Joint contractures progress, culminating in cartilage destruction, joint subluxation, and eventually, ankylosis. Roentgenograms show severe, diffuse osteoporosis, while bone scans reveal decreased uptake during the flow and pool phases with normalization of the later fixation phase.[316]

Pathophysiology

The multitude of theories regarding the etiology of CRPS decries the lack of firm understanding of its pathophysiologic mechanism. The profound diminution of pain following sympathectomy, noted as early as World War I by Leriche (cited in Loh and Nathan),[317] has implicated the sympathetic nervous system as in some way involved in the perpetuation of this pain syndrome.[318] Several heuristic theories have recently been postulated to explain the inciting causes and alterations in the peripheral and central nervous systems that may perpetuate the syndrome even after resolution of the initial injury.

Specific types of sensory inputs are transmitted along particular classes of nerve fibers. Painful, injurious stimulation is conducted afferently along myelinated A-delta fibers and unmyelinated C-fibers. The ability to respond to several types of noxious stimuli or exclusively to a single form of nociceptive input varies between fiber types. Moderately intense mechanical stimulation is transduced through A-delta high threshold mechanoreceptors (HTM). A-beta myelinated mechanothermal nociceptors (MMTN) respond to heat greater than 45°C and to intense mechanical stimulation. Both the A-delta HTM and MMTN fibers terminate along several dorsal horn laminae, predominantly laminae I, IIo, V, and X. C-polymodal nociceptors (C-PMN) are most sensitive to strong prolonged stimulation by temperatures ranging from 45°C to 51°C, intense mechanical stimulation or chemical irritation. C-PMNs terminate at laminae I, IIo, and V. A-beta low threshold mechanoreceptors (LTM) are nonnociceptive, large myelinated sensory afferents. These fibers normally transmit light touch and subtle mechanical stimulation. A-beta fibers terminate at laminae II through V. Some penetrate into the ventral horn, directly synapsing with motor neurons, while others ascend in the dorsal columns.

Of the dorsal laminae mentioned, lamina V is suggested to be integral in the establishment of sympathetically maintained pain syndromes. The most common cells found in the lamina are wide dynamic range (WDR) or multireceptive neurons. These nerve cells will respond to stimulation from not only A-delta and C-PMN but from A-beta LTM fibers. It has been suggested[316] that these WDR neurons may be sensitive to spinal and supraspinal modulation. While much research has focused on the roles played by individual components of the nervous system in the maintenance of sympathetically maintained pain, it is ostensible that peripheral and central alterations are inextricably related and likely confer perpetual changes upon each other.

Central Changes

Roberts and Foglesong[319] hypothesized that sensitization of WDR neurons in the dorsal horn is the pivotal occurrence driving sympathetically maintained pain syndromes. Trauma to a peripheral nerve, in the case of CRPS-Type II, causes nociceptive transmission along unmyelinated C-PMN fibers to the laminae of the dorsal horn, including WDR neurons in lamina V. Kenshalo et al[320] suggested that repeated volleys of C-fiber stimuli increase the sensitivity of WDR neurons to subsequent stimulation. As WDR neurons receive afferent volleys not only from peripheral nociceptors, nonpainful stimulation such as light touch transmitted by A-beta fibers also triggers amplified responses in the primed spinal neurons.

The WDR neuronal response furnishes an important component of ascending nociceptive information. Under normal circumstances, the low frequency and intensity of A-beta stimulation is unlikely to trigger the large bursts of activity in the WDR neurons that are perceived supraspinally as pain. Sensitized WDR cells, however, may produce exaggerated outputs in the face of normal A-beta stimulation. Allodynia and hyperpathia are manifestations of this aberrant, heightened sensitivity to nonnoxious stimulation.

Peripheral Changes

The suggestion that nonnoxious stimulation might be integrally involved in the development of sympathetically maintained pain was put forth initially by Loh and Nathan.[317] Their presumption was further substantiated by Campbell et al.[321] Differential nerve blocks were performed on patients with long-standing nerve-injury–associated hyperalgesia. Selective blockade of large myelinated A-beta fibers led to ablation of the abnormal sensation, corresponding to the loss of light touch sensibility. Sensory input transmitted by C-PMN and A-delta fibers remained intact. Selective blockade of C-PMN and A-delta fibers did not alter the perception of hyperalgesia. The unique susceptibility of A-beta fibers to sympathetic stimulation, as observed in several studies, corroborates their participation in sympathetically maintained pain syndromes.

Sympathetic Nervous System

The precise changes occurring centrally and peripherally, while better understood, continue to be debated. The implication of the sympathetic nervous system, however, has been suspected for close to a century and has rarely been contested. Stimulation of sympathetic ganglia at frequencies as low as 5 Hz have been observed to activate specific subsets of dorsal horn spinal neurons. Sympathetic stimulation failed to trigger neurons receiving input solely from A-delta high threshold neurons. Nearly 50% of WDR spinal neurons were activated.[322] Of the WDR neurons stimulated, studies have observed anywhere from 15% to 56% were composed of those receiving input from A-beta LTM.[320,323] Roberts et al[323] contend that increased

sympathetic output is not necessary to drive the pain cycle. Normal sympathetic tone, as simulated by low-frequency stimulation causes firing of A-beta fibers in the periphery. This is in line with Torebjork's conclusion that there is little evidence from microneurographic recordings to support the contention that sympathetic outflow is increased in sympathetically maintained pain syndromes.[324] In the face of sensitized WDR spinal neurons, normal firing of sympathetically stimulated A fibers would be perceived as painful stimuli.

Roberts[303] has elegantly constructed a paradigm in which central neuronal changes, initiated by a peripheral injury, might be maintained by normal sympathetic activity, even after the original painful stimulation has resolved. It also provokes a number of questions:

1. If anywhere from 54% to 85% of WDR neurons are not triggered by sympathetically stimulated A fibers, are there other peripheral fibers that might be stimulated adrenergically?
2. Are there other peripheral mechanisms that might stimulate sensory fibers, maintaining WDR neuronal sensitization and triggering abnormal pain experiences?

Peripheral C-Polymodal Nociceptors

The strongest evidence for the additional involvement of C-PMN fibers is derived from studies of CRPS-Type II, in which the symptom complex is induced by peripheral nerve injury. The works of Devor and Janig[325] and Wall and Gutnik[326] have shown that afferent nerve fibers entangled in neuromas do respond to sympathetic stimulation as well as to local application of norepinephrine. Sato and Perl[327] observed that normal C-fibers did not respond to adrenergic stimulation. Partial injury of mixed nerves, however, led to situations in which C-PMN fibers developed enhanced sensitivity to both sympathetic stimulation and peripheral exposure of norepinephrine. Sensitization of C-PMN fibers to adrenergic stimulation started as early as 4 days after the inciting injury and peaked during the second week. This time frame mirrors the onset of sensory abnormalities and pain observed in clinical settings of CRPS-Type II.[310] Importantly, sensitization to adrenergic stimulation was not focused at the site of injury. It was noted to be most profound at the nerve endings. While these findings may have important implications in CRPS-Type II, it is unclear whether they can be generalized to nonnerve-injury-associated sympathetically maintained pain syndromes.

The role of peripheral instigators in the development and maintenance of CRPS-Type II cannot be underestimated. Undoubtedly, the overemphasis on ablation of only sympathetic efferents has led to many treatment failures. Following local injury and stimulation of C-PMN fibers, there is commonly the development of edema, caused by a reflex disturbance of vasoconstrictor outflow.[328] As the edema spreads, C-PMN fibers not initially in the region of injury are stimulated, causing a perpetuation of neurogenic inflammation. Less peripheral stimulation is needed to provoke firing of the C nociceptors once they have become sensitized. Thus, an escalation of C-PMN firing ensues, maintaining neurogenic inflammation, causing further sensitization of the C-fibers, and ultimately, sensitizing and perpetuating the firing of WDR spinal neurons. The development of edema, coupled with immobility induced by pain, spawns a new problem: soft tissue contractures. Movement of contracted joints excites muscle and joint nociceptors, adding to the sensitizing volleys converging on the WDR neurons.

Summary

Causalgia is an extremely complicated, sympathetically maintained pain syndrome, which follows a small percentage of peripheral nerve injuries. While its exact pathophysiologic mechanism remains as yet unknown, a collusion of peripheral, central, and sympathetic nervous system activities initiate, maintain, and perpetuate the syndrome. Recent studies support the following hypothesis:

1. Initial peripheral nerve injury stimulates C-PMN fibers, which sensitize WDR spinal neurons.
2. Neurogenic inflammation triggers the firing of additional C-PMNs, which in turn sensitize WDR neurons.
3. Sensitized WDR neurons respond abnormally not only to C-PMN fiber input but also to A-beta fiber input. Thus, both noxious and nonnociceptive stimulation is perceived as hyperintense and painful.
4. Sympathetic adrenergic activity perpetuates WDR sensitization and abnormal firing by triggering the firing of A-beta fibers and also sensitizing and exciting C-PMN fibers.
5. Sympathetic sensitization, peripheral sensitization, and the escalation of nociceptor

involvement as soft tissue pathology develops, perpetuates the pain syndrome even after the initial injury has abated.

A solid understanding of all the potential elements involved may help guide a more rational approach to the management of this syndrome.

Diagnosis

While much of the literature alludes to the notion of CRPS as a unique, well-defined disorder, no studies to date have established firm criteria upon which a diagnosis can be based. Causalgia remains an elusive symptom complex, associated with laboratory findings that may help support but cannot confirm its presence. Most studies examining diagnostic approaches to CRPS do not differentiate Type I from Type II or SMP. Therefore, these terms may be used interchangeably in the next several sections.

Clinical Criteria

Descriptive definitions of CRPS-Type II and CRPS-Type I have been attempted. The IASP classifications for these symptom complexes have already been mentioned.[301] A panel of experts has attempted to develop a more concise definition. The group at Schloss Rettershoff[329] described CRPS-Type I as

> ...a syndrome of continuous diffuse limb pain, often burning in nature and usually consequent to injury or noxious stimulus and disuse, presenting with variable sensory, motor, autonomic, and trophic changes; causalgia represents a specific presentation of CRPS-Type I associated with peripheral nerve injury.[329]

While less vague than the 1986 IASP definition, detractors still lament that this definition of CRPS-Type I can apply to symptoms experienced in any number of posttraumatic situations.

The often-cited criteria established by Kozin et al[330] were developed empirically to assess the efficacy of certain treatment protocols and imaging studies. Kozin's proposed criteria divided patients into four potential groups:

1. Definite reflex sympathetic dystrophy syndrome (RSDS) included patients with pain, tenderness in the distal extremity, signs or symptoms of vasomotor instability, or swelling.
2. Probable RSDS included those with pain and tenderness and either vasomotor instability or swelling.
3. Possible RSDS included patients with vasomotor instability or swelling but without pain.
4. Lastly, the doubtful RSDS category contained those with unexplained pain in an extremity.

Ironically, while many clinicians base diagnoses on the responsiveness to sympathetic blockade, absolutely no patients in Kozin's study, even those deemed as suffering from definite RSDS, achieved a good response to sympathetic blockade. Kozin's scintigraphic results pose another problem when assessed in relationship to the duration of signs and symptoms. The expression of signs and symptoms by patients included in the study ranged from 2 weeks to nearly 6 years. Yet, from 40% to 83% had increased perfusion on the static and flow studies, varying merely by predetermined categorizations. These findings are at odds with the chronological evolution of bone scan findings established by Demangeat et al.[331] It should be understood, therefore, that the efficacy of Kozin's criterion as a diagnostic tool in establishing the diagnosis of CRPS-Type I is dubious at best.

An alternative approach to diagnosis was established by Roberts.[303] Of the signs and symptoms catalogued in other descriptions, only involvement of the sympathetic nervous system distinguishes the syndrome from other posttraumatic pain problems. A diagnosis of sympathetically maintained pain implies that pain relief is obtained during blockade of the sympathetic nervous system. Ganglionic blockade as well as postganglionic regional blockade have been considered. Arner[332] suggested that intravenous infusion of phentolamine, a short-acting alpha-adrenergic blocker (5 mg to 15 mg) over a 5-to-10-minute period could be used diagnostically to determine those patients likely to gain pain relief from more definitive sympathetic blockade. While narrow in focus, the establishment of responsiveness to sympathetic blockade may be exceedingly helpful in the orchestration of a multifaceted, interdisciplinary management approach.

Laboratory Findings

Roentgenography

Kozin et al[330] observed on fine detail roentgenography the nonspecific finding of patchy osteoporosis in 69% of patients meeting his criteria

for definite, possible, and probable RSDS. Bone resorption was observed most commonly in the periarticular regions but also diffusely in a smaller number of cases. As a diagnostic tool, roentgenography has a sensitivity of 69% and a specificity of 79%.[330] Kozin and colleagues[333] cautioned that the development of patchy demineralization reflected the intensity of the resorptive process and not the actual mechanism. The value of roentgenography in their studies are overestimated in that associated problems, such as disuse and immobilization caused by nerve injury or pain, may be independent antecedents of periarticular osteoporosis.[334] The utility of roentgenography may be greatest in the very early stages when the resorption is less likely to be from disuse and more likely caused by accelerated blood flow in the affected region.

Triple-Phase Bone Scan

Several studies have addressed the utility of Technetium 99m (^{99m}Tc) bone scintigraphy as not only a diagnostic but a staging tool. Kozin et al noted the delayed phase (third phase) had a sensitivity of 60% and a specificity of 92%.[330,334] Holder and Mackinnon,[335] observing symptoms only in the hands, documented different levels of sensitivity during each of the three phases. Phase I had a sensitivity of 45%; phase II, 52%; and phase III, 96%. Specificity was virtually the same for all phases at greater than 95%. Davidoff et al[336] explored the predictive value of triple-phase bone scans. Overall sensitivity was rated at 44%, specificity was 92%, positive predictive value was 61%, and negative predictive value, 86%. Differences in outcomes likely reflect the lack of consensus on what constitutes a diagnosis of CRPS-Type I as well as the utilization of subtly different techniques. Other factors that have been noted to decrease the efficacy of triple-phase bone scanning as a diagnostic aid include the manifestation of symptoms for more than 6 months and patient age below 50 years.[337]

Demangeat et al[331] suggested that the variability of sensitivity might be explained if the period of illness during which the bone scan is obtained is considered. Following an initial intravenous bolus of ^{99m}Tc, the time span from the arrival of tracer in the camera field as it passes through the arterial system to the beginning of its disappearance through venous return marks the blood flow phase (phase I). The blood pool phase (phase II) follows quickly, revealing distribution of ^{99m}Tc between the intravascular and interstitial compartments. This phase reflects the vascularization of the soft tissues. By 3 hours after injection, the ^{99m}Tc has left the soft tissue. These delayed, phase III images reflect bone fixation (Figures 9-40 through 9-42).

Distinct patterns of ^{99m}Tc uptake during the three phases paralleled the elapsed time during which symptoms were evident.

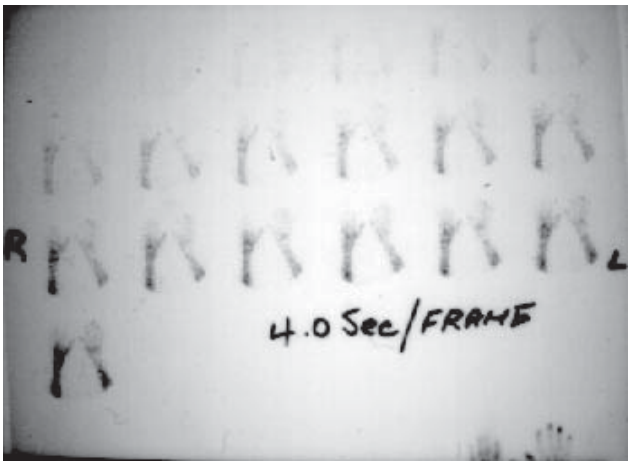

Fig. 9-40. Blood flow phase (phase I) of three phase bone scan. Asymmetrically increased uptake is observed in the left upper extremity of a patient with stage I CRPS-Type I. Scan courtesy of MAJ Antonio Balingit, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

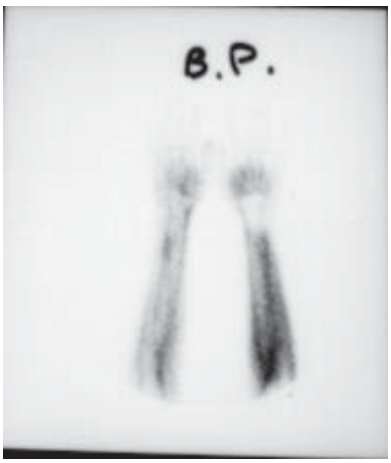

Fig. 9-41. Blood pool phase (phase II) of three phase bone scan. Asymmetrical increased uptake is observed in the right upper extremity of a different patient with stage I CRPS-Type I. Scan courtesy of MAJ Antonio Balingit, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

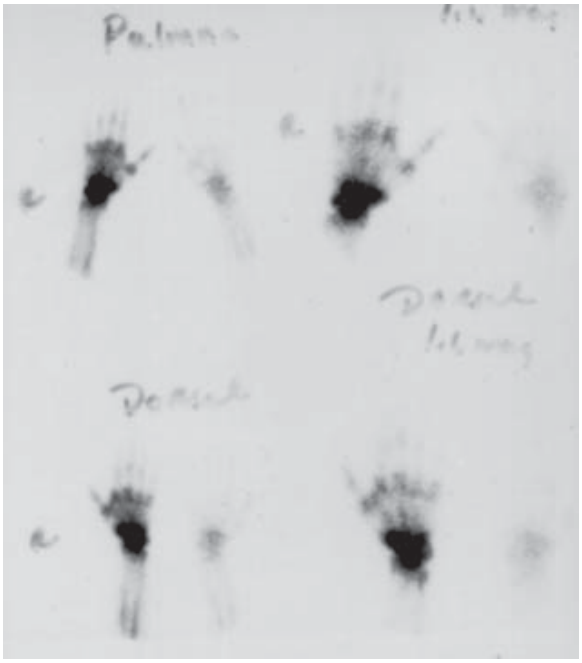

Fig. 9-42. Delayed hyperfixation phase (phase III) of three-phase bone scan. Asymmetrical periarticular increased uptake is observed in the right upper extremity of a patient with stage I CRPS-Type I. Tracing courtesy of MAJ Antonio Balingit, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

During the first 20 weeks from onset of symptoms, increased uptake was observed during all three phases. Normalization of the blood flow and pool phases were seen between 20 and 60 weeks with continued hyperfixation during phase III. After 60 weeks from onset, decreased uptake was observed in the first two phases with normalization in the last phase (Table 9-12).

Triple phase bone scan may be a helpful adjunct in the diagnosis of CRPS-Type I, especially in situations where symptoms, timeframe, and appropriate scintigraphic findings all coincide.

Thermography

Thermography is a noninvasive procedure that measures and displays heat emitted by the superficial tissues of the body. Normally, thermal distributions are symmetrical and follow a generally distinctive pattern. Changes in the digital color representations of temperature distributions or gradients may suggest an underlying pathology. According to Uricchio, thermographic findings in causalgia mirror clinical symptoms. Early in the course, an asymmetrical nonthermotomal temperature increase of several degrees is apparent by thermographic assessment. Later, as the limb becomes cooler, a regional drop in temperature may be seen in the affected limb.[338] While promising, the prohibitively high equipment costs and lack of general availability have kept thermography more in the realm of a research tool. There have been no studies comparing the benefits of thermography to the universally available and less costly scintigraphic studies.

Future Diagnostic Approach

Currently, there is no single test or group of findings that will absolutely confirm the diagnosis of CRPS. Further research is needed to establish the validity (ie, the test accurately measures what it intends to measure) of current diagnostic approaches. A paradigm shift needs to be made away from classification systems that require a person to meet all criteria in order to attain a diagnosis. The overall reliability (ie, capacity to give consistent results when used by different examiners at different times) of each diagnosis is limited by the least reliable criterion. The implementation of a model in which a

TABLE 9-12

TRIPLE PHASE BONE SCAN FINDINGS DURING THREE STAGES OF RSDS

	Bone Scan	
Stage	**Phase**	**Results**
Stage I (0–20 weeks)	Blood flow	Increased
	Blood pool	Increased
	Delayed	Increased
Stage II (20–60 weeks)	Blood flow	Normal
	Blood pool	Normal
	Delayed	Increased
Stage III (60–100 weeks)	Blood flow	Decreased
	Blood pool	Decreased
	Delayed	Normal

RSDS: reflex sympathetic dystrophy syndrome
Source: Davidoff G, Werner R, Cremer S, Jackson MD, Ventocilla C, Wolf L. Predictive value of the three-phase technetium bone scan in diagnosis of reflex sympathetic dystrophy syndrome. *Arch Phys Med Rehabil*. 1989;70:135–137.

person needs to manifest several, but not necessarily all, criteria will improve the overall reliability of each diagnosis and begin the process of honing a generally accepted approach to diagnosis. Such a model has been developed by Wilson[339] and may provide a basis for further diagnostic standardization. At least six criteria would be needed to confirm a diagnosis of CRPS-Type I. Three to five positive criteria may be categorized as possible CRPS-Type I until further evaluation of the criteria can be made. Less than three positive criteria would not constitute CRPS-Type I. Included as criteria would be both clinical and laboratory findings (Table 9-13).

Conclusions regarding the efficacy of specific treatment modalities are contingent on the development of valid diagnostic methods. Until criteria are accepted universally, it will be difficult to accurately assess the effectiveness of therapeutic interventions.

Treatment

No single treatment has been found to be effective in the treatment of CRPS-Type II. A well-coordinated, interdisciplinary approach with a focused goal of functional restoration is the key to successful management. The coupling of peripheral stimulation and central sensitization by sympathetic activity provides three areas that should be treated simultaneously to stymie the self-perpetuating cycle. Initial management should focus on the diagnosis and treatment of the inciting injury. Pain must be brought under control through peripheral, central, and sympathetic treatment approaches and, once decreased to tolerable levels, should be coordinated with aggressive physical therapy.

TABLE 9-13

PROPOSED DIAGNOSTIC CRITERIA FOR COMPLEX REGIONAL PAIN SYNDROME (CRPS)

Clinical Findings	Paraclinical Finding
Burning pain	Roentgenography
Allodynia	Bone scintigraphy
Temperature/color change	Thermography
Edema	Quantitative sweat test
Skin, hair, nail growth change	Quantitative sensory test
	Response to sympathetic blockade

Definite CRPS: More than 5 findings present
Possible CRPS: 3 to 5 findings present
Not CRPS: Less than 3 findings
Source: Wilson PR. Sympathetically maintained pain principles of diagnosis and therapy. In: Stanton-Hicks M, Janig W, Boas RA, eds. *Reflex Sympathetic Dystrophy*. Boston, Mass: Kluwer Academic Publishers; 1988:24–36.

Sympathetic Blockade

Extensive reports collected during World Wars I and II suggest that sympathetic blockade effectively abolishes the pain associated with CRPS-Type II.[316] As an isolated intervention, however, it rarely provides permanent relief.

Ganglionic blockade. Stellate ganglion blocks are used when the upper extremity is symptomatic. The needle is passed in a vertical-dorsal direction along the medial edge of the sternocleidomastoid muscle at the level of the cricoid cartilage. At a depth of 2 cm to 3.5 cm, contact should be made with the transverse process of the C-6 vertebra. The needle is then withdrawn 2 mm. A 5-mL to 10-mL injection of local anesthetic (eg, 1% mepivacaine or 0.5% bupivacaine) is administered following extensive aspiration, ensuring that no vessel has been entered inadvertently. Fluoroscopy may be exceedingly helpful to guide positioning.

Lumbar sympathetic blocks are used for lower extremity symptoms. The needle is passed in the medio-ventral direction, toward the L-2 vertebra. At 6-cm to 8-cm depth, the needle should come in contact with the body of the L-2 vertebra. The needle is withdrawn 1-cm to 2-cm and repositioned cephalo-laterally, placing it 1 cm lateral to the body of the L-1 vertebra. This approach reduces the risk of penetration into the kidney or psoas sheath. A 10-mL to 15-mL local anesthetic solution is injected. Blind technique carries a success rate of 80%. The use of contrast and fluoroscopic guidance improves the success rate to nearly 100%.[340]

An adequate sympathetic blockade should cause a temperature rise in the affected limb of 5°C to 10°C. A Horner syndrome is a common side effect of a successful stellate ganglion block.[341]

Regional blockade. Postganglionic sympathetic blockade can also be achieved through regional intravenous administration of sympatholytic agents.

Guanethidine infusion was introduced in 1974 by Hannington-Kiff[342] and remains the gold standard. Intravenous administration has not been approved in the United States. Alternatives including reserpine and bretylium have also been employed with similar results.[343,344]

Similar results have been achieved with both ganglionic and regional blockade. Bonelli et al[345] observed that symptomatic pain relief and evidence of sympathetic blockade following intravenous regional guanethidine administration every 4 days for a total of 4 blocks was comparable to stellate ganglion blocks performed daily for 8 days. The major advantage of intravenous regional blockade can be seen during treatment of CRPS-Type II in the upper extremity. The ease of intravenous administration easily outweighs the potential hazards of stellate ganglion blockade, which include pneumothorax, intravascular injection, and puncture of the esophagus. The opposite techniques are more easily used in the lower extremity. Lumbar sympathetic blockade is a relatively easy procedure, while tourniquet placement and venous cannulation during regional blockade pose problems in the lower extremity.[346]

Oral Administration

A single uncontrolled study and an anecdotal report[347] discuss the use of oral sympatholytic agents in the treatment of causalgia. Ghostine administered phenoxybenzamine, a long-acting peripheral alpha$_1$-adrenergic blocker, to nerve-injured soldiers with CRPS-Type II during the Lebanese civil war. Initial dosing started at 30 mg per day in three divided doses (ie, 10 mg tid). The dose was increased by 10 mg every 2 days until symptomatic relief was achieved or side effects became unbearable. The average dose was 80 mg daily. The treatment was continued for 6 weeks in the majority of patients with excellent, long-lasting results.[347]

Sympathectomy

In patients who respond to sympathetic blockade, but only transiently, and do not tolerate oral sympatholytic agents, permanent sympathectomy may be considered. Bonica[316] suggests that chemical sympathectomy with 6% aqueous phenol or 50% alcohol can ablate sympathetic activity for several months. It was suggested that this procedure might be better tolerated in older patients than the surgical procedure. Surgical sympathectomy might be tried in younger, healthier patients. Both Bonica[316] and Mayfield[304] warn, however, that sympathectomies rarely provide permanent, long-lasting pain relief. If undertaken, they still must be coordinated with other methods of pain modulation.

Pain relief derived from sympathetic blockade provides three major benefits in the treatment of CRPS-Type II. Aside from direct enhancement of patient comfort, it allows the patient to use the affected limb. It also establishes the diagnosis of sympathetically maintained pain, differentiating the syndrome from other forms of neuropathic pain.

Peripheral Treatment

Injury Management

Injury to peripheral nociceptors and persistent stimulation of nociceptors and mechanoreceptors perpetuate the symptom complex. Diagnosis and rapid treatment of the inciting injury are paramount in the management of CRPS-Type II.

Pain Control

The use of pain-modulating agents that act peripherally has been advocated. While few controlled trials have established their effectiveness, empiric usage stems from results achieved in the treatment of other types of nerve injury.

Corticosteroid administration was found to be highly effective in the Kozin et al study[348] for patients meeting his criteria of definite RSDS. A randomized, placebo-controlled study by Christensen et al[349] also showed improvement beyond that attained with placebo. Thirty milligrams of prednisone was administered daily in divided doses for upwards of 12 weeks. Improvement was noted in all patients.

Nonsteroidal antiinflammatory agents have been included in review articles as potentially effective agents during the acute stage.[350] No controlled studies were noted to have been attempted to establish efficacy.

Case reports attesting to pain relief provided by topical application of clonidine[351] and capsaicin[352] have recently been cited, but lack adequate placebo-controlled trials. Oral administration of membrane stabilizers such as phenytoin,[353] beta-adrenergic blockers including propranolol,[354] gabapentin,[355] and calcium channel blockers have also been reported.[356]

Transcutaneous electrical nerve stimulation and electroacupuncture application have been shown in several small uncontrolled trials to be effective in

the diminution of CRPS-Type II-related pain.[357,358]

Physical Modalities

It has been suggested that, ultimately, the greatest benefit of pain management is that it allows the patient to participate in therapies that will restore normal function. Edema control and the resolution of contractures decrease stimulation of peripheral mechanoreceptors and nociceptors. Repetitive contraction of muscles during progressive resistance training helps to decrease edema accumulation and reverses osteoporotic bony changes. Hypesthesia, a common manifestation of CRPS-Type II, can be effectively dampened through desensitization techniques. While research examining the exact benefits of physical modalities has been severely limited, there is widespread agreement that they are an integral part of the treatment program.[356] All efforts need to be made to encourage use of the affected limb as much as possible.

Central Treatment

Descending central-pain-modulating pathways provide inhibitory impulses at the level of the dorsal horn and may provide an element of pain relief. The use of drugs including tricyclic antidepressants have been recommended despite the lack of clinical trials.[341,356]

As with other chronic pain syndromes, the distress and anxiety caused by the experience of persistent suffering impairs the ability to cope not only with the pain syndrome but also the stressors of day-to-day life. Intense psychologic support is essential and all efforts should be made to allay fears and anxieties about using the affected limb. Affective disorders, which may arise secondarily, should be monitored closely for detection and treated appropriately.[316]

Case Study 1: Penetrating Injury to the Upper Extremity

The patient is a 46-year-old army sergeant. In February 1991, he was struck by an artillery shell fragment which entered his left upper arm, fracturing the humerus and severing the brachial artery. He underwent emergent vascular reconstructive surgery at the evacuation hospital in Saudi Arabia. At the time he was observed to have complete median and radial nerve injuries of the left arm. Surgical clips were attached to the ends of these nerves, marking them for potential nerve grafting in the future. Open reduction and internal fixation was performed to stabilize the humeral fracture.

The Physical Medicine and Rehabilitation Service was consulted on his transfer to Walter Reed Army Medical Center. Initial physical examination of his left upper extremity revealed marked edema, healing surgical wounds, no evidence of infection, and tenderness to palpation. Radial pulse was intact.

His passive shoulder range-of-motion was limited in abduction, internal rotation, and external rotation. Elbow flexion passively was limited to 70° with a firm endpoint. Passive flexion and extension at the wrist, MCP flexion and interphalangeal (IP) joint flexion were limited in all fingers in the left hand.

Strength was good in the proximal shoulder girdle muscles, poor in elbow flexion, and trace in elbow extension. Wrist flexion strength was fair with ulnar deviation observed at the wrist. Wrist extension and MCP joint extension in all fingers was zero as was thumb abduction, extension, opposition, and IP joint flexion. First dorsal interosseus muscle strength was graded as poor. Thumb abduction was good. Distal IP joint flexion was zero in the second digit, trace in the third digit, and fair in the fourth and fifth digits.

Sensation testing to light touch and pinprick revealed absent sensation over the palmar and dorsal aspect of the thumb and second and third digits. Mild sensory loss was also noted in the medial antebrachium. Deep tendon reflexes in the left upper extremity were absent. The patient also complained of severe pain, allodynia, and hypesthesia in the forearm and hand, exacerbated by passive motion and light stroking of the hand and forearm. The distal extremity was cool and cyanotic when compared to the opposite limb.

Plain radiographs of the left elbow and humerus revealed exuberant bony callous formation with heterotopic ossification and a stable, healing fracture. Electrodiagnostic testing 1 month postinjury revealed findings consistent with a severe median nerve injury at the elbow with no evoked potentials obtainable. Electrodiagnostic testing confirmed that the lesion was at a point proximal to the point where the nerve to the flexor carpi radialis arises. Radial motor nerve conductions were not obtainable, suggesting severe axonal injury. Radial SNAPs were obtained, revealing a decreased amplitude but normal latency. Coupled with the clinical finding of absent sensation in the radial distribution, the findings suggested mild to moderate axonal loss with severe conduction block. It was suspected that the focus of injury was likely just distal to the branching point of the superficial radial nerve. A partial, moderately severe left ulnar nerve injury with evidence of axonal loss was also observed. There was also evidence of a mild brachial plexopathy with spontaneous activity in the deltoid and teres minor muscles but with full recruitment. In addition, there was marked soft tissue injury in the brachial region involving the biceps and triceps which complicated electrodiagnostic interpretation.

Triple phase bone scan revealed diffuse increased uptake in the periarticular regions of the left hand and wrist suggestive of CRPS-Type I. In light of his symptoms and associated nerve injuries, a diagnosis of CRPS-Type II was made.

Improving and maintaining adequate functional range-of-motion in his left hand and arm required extensive therapy, including prolonged stretching by occupational therapists and a self-program that the patient followed diligently. The humerus fracture stabilization was strong enough to allow careful, active, assistive range-of-motion at the elbow. Initially, a static dorsal orthosis was fabricated and designed to provide an alternating MCP extension force and finger flexion force. The patient alternated the flexion and extension every few hours to provide prolonged stretch to the MCP and IP finger joints. His range-of-motion in the wrist and hand improved enough to allow fabrication of a functional dynamic orthosis.

Edema of the hand and forearm was treated initially with pneumatic compression followed by the application of a pressure garment sleeve and glove. Centripetal massage and wrapping were also employed. Significant improvement occurred over the course of several weeks.

Muscles not completely denervated—the ulnar innervated muscles, the triceps, the biceps, and the proximal arm and shoulder muscles—were exercised with active assistive, then active resistive exercises. The ulnar innervated muscles (interossei) improved markedly to a level of good (4/5). Good strength in wrist flexion, and good distal IP joint flexion of the fourth and fifth fingers was obtained. Elbow flexion and extension strength had improved to a level of good. Active pronation and supination remained zero. Metacarpophalangeal joint flexion strength in the second through fifth fingers improved to a good level. The proximal shoulder muscle strength improved to normal.

Pain management included administration of the NSAID Naprosyn, as well as the tricyclic antidepressant amitriptyline. The latter was stopped because of anticholinergic side effects. Doxepin was administered as an alternative. Conventional TENS was applied to the proximal aspect of the forearm, providing moderate pain relief. Desensitization, employing progressively harsh textures and vibration, helped to decrease the allodynic and hypesthetic sensations. These treatments in conjunction with reduction in edema and soft tissue contractures decreased the patient's pain to a tolerable level, allowing him to sleep soundly and perform daily tasks.

Sensory reeducation techniques were instituted to enhance the patient's dexterity and sensory discrimination in the ulnar distribution.

A dynamic wrist-hand orthosis was fabricated to provide stabilization and dynamic substitution for movements lost secondary to the multiple nerve injuries. The base orthosis consisted of a dorsal orthoplast plate covering two thirds of the dorsal aspect of the forearm. Wrist extension was achieved through the placement of a hinge at the wrist. Elastic bands provided dynamic extension assistance while allowing for active flexion, driven by the flexor carpi ulnaris. Extension assists were also incorporated, providing dynamic MCP extension for the second through fifth fingers. The extension assists consisted of finger loops attached by nylon cords which ran through eyelets at the distal end of the orthosis. The cords were attached to elastic bands, which were attached to the orthosis with Velcro, allowing for changes in desired tension. A coupling device yoked the second and third fingers, which allowed the third finger to power a three-chuck-jaw grasp. A small hook was attached to the coupler, which was connected to the main dorsal orthosis to provide dynamic extension assistance. A removable sleeve was fabricated to stabilize the thumb. It also was attached to the base orthosis by elastic bands and provided dynamic abduction and opposition. A volar plate provided the base to which the elastic thumb abduction assist was attached (Figures 9-43 and 9-44).

Fig 9-43. Profile view of dynamic wrist-hand orthosis that provides stability and dynamic assistance for the upper extremity of a patient who sustained complete radial and median nerve injuries and a partial ulnar nerve injury.

Extensive patient education and training were required to optimize the patient's hand function with the orthosis.

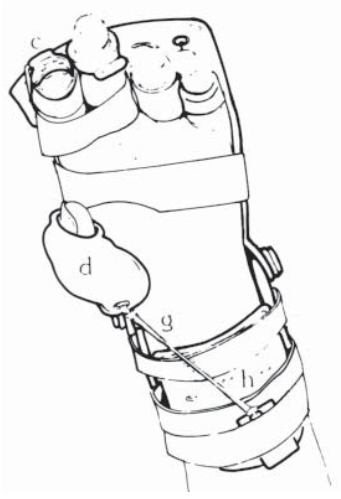

Fig 9-44. Palmar view of the orthosis. **c**: Coupling device yoking the second and third fingers. **d**: Thumb stabilizing sleeve. **g**: Elastic band providing dynamic thumb abduction and opposition. **h**: Volar plate providing base for dynamic thumb assist.

Routine skin inspection was essential to evaluate insensate skin for breakdown. The patient was able to grasp objects and use the orthosis to complete tasks requiring two hands, which he was unable to do without the orthosis.

Six months following his injury, the patient underwent interpositional sural nerve grafting of both the left median and radial nerves. At 2 years following the injury, there was no evidence of return of function or sensation in the distributions of the grafted nerves. During this period, however, the patient continued to use the orthosis while at work and during recreational activities. His pain had subsided and strength in the ulnar distribution had improved to normal levels. With this improvement in strength it was felt that tendon transfers could now be undertaken to improve the stability of the wrist and improve his function.

Transfer of the flexor carpi ulnaris tendon to the extensor digitorum communis and the extensor pollicis longus tendons provided finger and thumb extension. Next, the flexor digitorum profundus III tendon was transferred to the flexor digitorum profundus II tendon to improve index finger flexion. The thumb was stabilized by fusion of the IP joint. Wrist stabilization was achieved by performing an extensor carpi radialis brevis tenodesis.

Further therapy, including maintenance of range-of-motion, strengthening, and muscle reeducation, was necessary following surgery. The patient is currently functional using the left upper extremity.

Case Study 2: Penetrating Injury to the Lower Extremity

The patient is a 20-year-old army specialist. In October 1993, he was struck in the left popliteal region by fragments after his vehicle was hit by a rocket-propelled grenade. The patient was hemodynamically stabilized in Somalia and transferred to Landstuhl Army Medical Center in Germany. Surgical debridement of burned and necrotic tissue was undertaken and a split-thickness skin graft was placed to cover the popliteal region. The patient was then transferred to Walter Reed Army Medical Center for further evaluation and treatment. Upon arrival he was observed to have an ongoing soft-tissue infection and necrosis of the skin graft. The area was sharply debrided. The posterior tibial nerve was burned and contused (Figure 9-45). The common peroneal nerve was avulsed from the sciatic nerve at the bifurcation. The anterior tibial artery was ligated and the popliteal artery was observed to be in continuity but greatly inflamed. Five days later, the patient underwent a left latissimus dorsi free muscle flap to the left popliteal fossa with split-thickness skin grafting.

The Physical Medicine and Rehabilitation Service was consulted upon the patient's transfer to Walter Reed Army Medical Center. Initial physical examination of his left lower extremity, 10 days following surgery, revealed partial thickness burns to the distal medial aspect of the thigh. A 14 x 6 x 16-cm latissimus dorsi musculocutaneous flap covering the popliteal region was intact, edematous but healing well. Capillary refill at the toes was brisk.

Fig 9-45. Surgical debridement of popliteal region following penetrating and burn injury. The tibial nerve is exposed and observed to be badly burned and contused. Photograph courtesy of LTC Gregory A. Antoine, M.D., Plastic and Reconstructive Surgery Service, Walter Reed Army Medical Center, Washington, DC.

Passive range-of-motion at the hip was limited to 90°. Knee flexion was limited to 30°. Knee extension was limited actively by 15° and could not be tested passively because of the recent graft placement. Passive ankle dorsiflexion was limited to neutral.

Strength was good in the proximal hip girdle with the exception of hip extension, which was fair. Knee flexion and extension were fair and significantly limited by pain. No active movement could be observed in ankle dorsiflexion, plantarflexion, inversion, eversion, or toe movement in any direction.

Sensation testing to light touch and pinprick revealed absent sensation along the lateral aspect of the leg, extending from just lateral to the crest of the tibia, posteriorly to the medial third of the posterior aspect of the calf. Sensation was also absent over the entire foot. The findings suggested loss of sensation in the sural, superficial peroneal, deep peroneal, and plantar nerves with sensory sparing in the saphenous nerve distribution (Figure 9-46). Deep tendon reflexes could not be tested at the left patellar tendon and were absent at the left Achilles tendon. The patient also complained of pain not only in the operative site but described a lancinating pain shooting down the back of the thigh and into the toes. He also described a burning, aching sensation in the anesthetic region of the left foot, which felt as though a vise was around his ankle, and his toes were in a contorted posture.

Plane radiographs of the entire left lower extremity revealed no evidence of fractures or joint dislocations. Arteriogram of the left lower extremity revealed no evidence of vascular injury.

Electrodiagnostic testing performed 6 weeks after the injury revealed findings consistent with severe common peroneal and tibial nerve injuries. Evoked potentials could

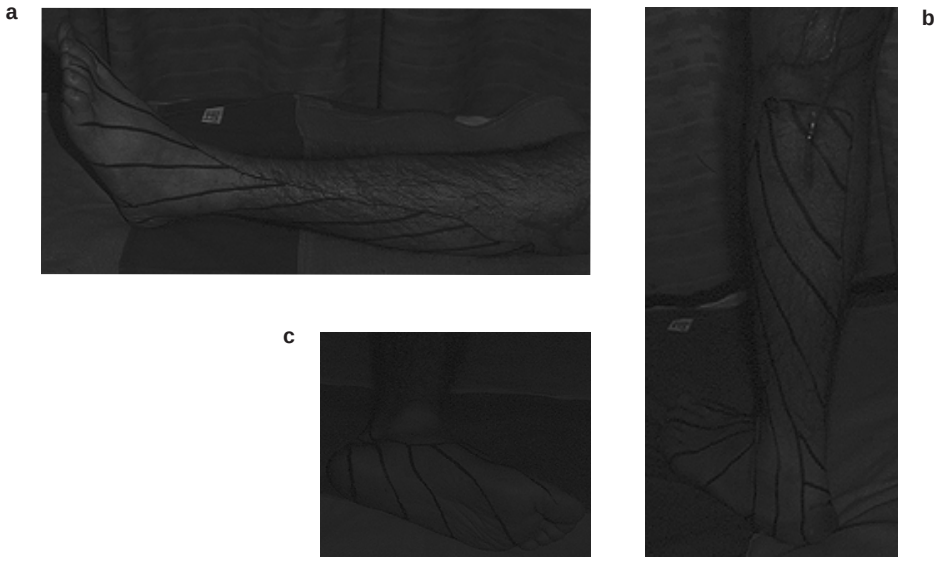

Fig 9-46. a: Anterolateral view**. b:** Sole of the foot**. c:** Posterolateral view**.** Demarcation of sensory loss following injury to the left common peroneal and tibial nerves. Absence of sensation was observed along the sural, superficial peroneal, deep peroneal, and tibial plantar nerves. Sensation in the saphenous nerve distribution was intact.

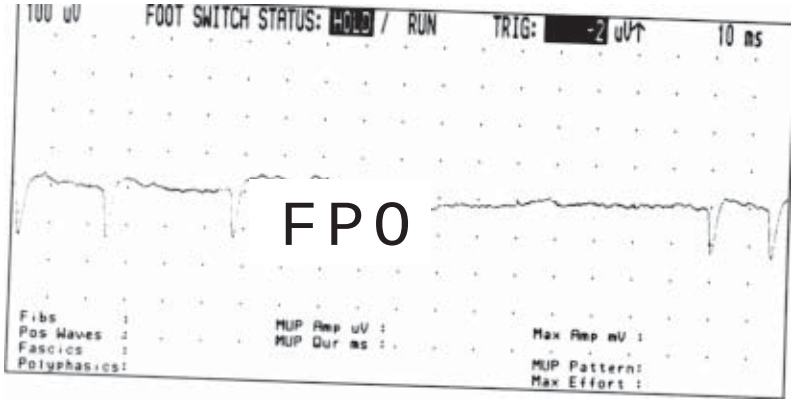

Fig 9-47. Electromyographic display of positive wave potentials observed during assessment for spontaneous activity. Examination was of the left medial gastrocnemius muscle.

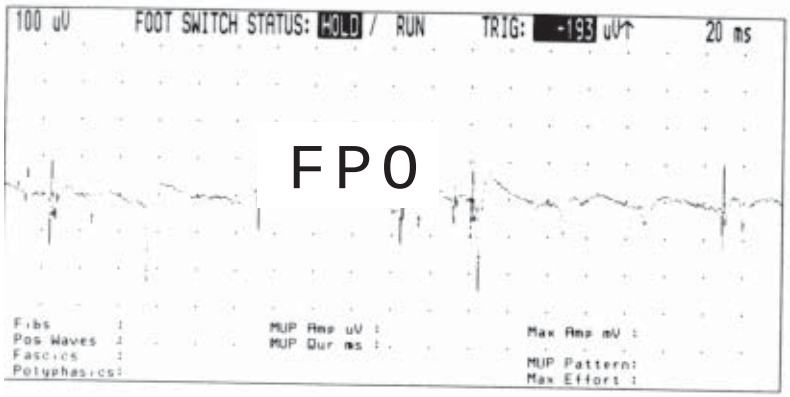

Fig 9-48. Electromyographic display of both positive sharp wave and fibrillation potentials observed during assessment for spontaneous activity. Examination was of the left tibialis anterior muscle.

Fig 9-49. An example of contrast baths that are used to produce hyperemia and to decrease hypersensitivity to hot or cold temperatures. The hot bath is maintained between 40ºC and 43ºC. The cold bath temperature ranges between 15ºC and 20ºC. Immersion protocols include hot bath for 10 minutes, cold bath for 1 minute followed by cycles of 4-minute hot baths alternating with 1-minute cold baths. This is continued for a full 30-minute session. Adapted with permission from Lehmann JF, deLateur BJ. Diathermy and superficial heat, laser and cold therapy. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* 4th ed. Philadelphia, Pa: WB Saunders; 1990: 283–367.

not be obtained during near nerve stimulation proximal and distal to the site of injury. Saphenous sensory conduction was normal. Electromyography revealed evidence of severe, ongoing denervation in muscles of the calf and foot innervated by the superficial peroneal, deep peroneal, and tibial nerves (Figure 9-47 and 9-48). More proximal evaluation suggested the focus of injury to be distal to the branch to the short head of the biceps femoris muscle. There was no evidence to suggest a concomitant plexus or root injury.

Pain management became the major issue following surgery. Trials of ketorolac and meperidine postoperatively did not adequately relieve the patient's pain. The pain precluded his ability to transfer out of the bed, to ambulate or to attempt ADLs. It was recommended that amitriptyline be started, titrating from 25 to 150 mg every night. This was later changed to trazadone 100 mg nightly because of anticholinergic side effects. It was also suggested that Demerol be stopped because of its potential for provoking seizures, and a long-acting morphine sulfate agent to be started at 30 mg twice daily (bid). The patient continued to complain of lancinating pain. The long acting morphine sulfate was increased to 30 mg tid. The NSAID Naprosyn at 500 mg bid was added, as was carbamazepine at a dose titrated up to 200 mg tid, which significantly improved the patient's lancinating pain. The patient was switched to Methadone and gradually weaned off all narcotics over a several-week period without a decrease in pain control. Conventional TENS was applied to the proximal aspect of the thigh, which provided only minimal relief and was subsequently stopped. Desensitization techniques including progressive textures and vibration, and contrast baths helped to decrease the abnormal perception of his toes and ankle (Figure 9-49). Better control of neuropathic and deafferentation pain allowed the patient to participate more aggressively in his therapies.

A plastic molded ankle-foot orthosis was fabricated to provide ankle stabilization and to simulate dorsiflexion and plantar flexion. Trim lines were brought anteriorly to provide mediolateral stability. Medial arch support and lateral flare were built into the foot plate to help stabilize and support the foot. The foot plate was extended to just distal to the metatarsal heads to prevent collapse of the mid-foot and simulate pushoff at the end of the stance phase. The ankle was set in 5° of plantarflexion, which allowed for adequate toe clearance during the swing phase. It also allowed for the most normal forces to be generated at the knee during the stance phase, averting hyperextension or a destabilizing knee flexion moment (Figure 9-50). Extensive gait training was required to optimize the patient's ability to ambulate with the orthosis. Scrupulous skin monitoring was necessary and taught to the patient, as his protective sensation was limited to the saphenous distribution.

After the patient was cleared from the plastic surgery service, active assistive and passive range-of-motion ex-

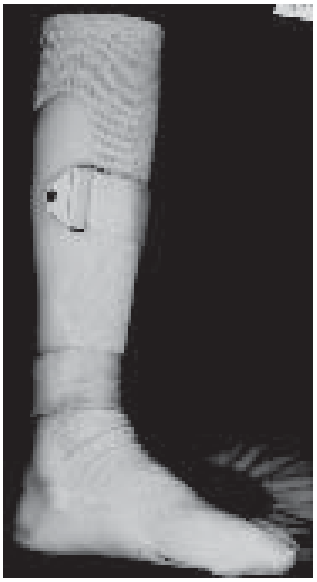

Fig 9-50. Left copolymer, custom molded ankle foot orthosis. The trim lines at the ankle are reinforced, providing mediolateral stability. The ankle is set in 5° platarflexion, allowing toe clearance and knee stability. The foot plate is extended just distal to the metatarsal heads to provide simulated pushoff at the end of the stance phase in the gait cycle.

ercises were instituted. Moist heat application around the left knee was used in conjunction with slow, prolonged stretching. By 10 weeks following his injury, the patient had regained full passive range-of-motion at the knee, hip, and ankle. Active assistive stretching of the shoulder flexors and external rotators was also done to maintain full mobility following harvesting of the latissimus dorsi musculocutaneous flap.

Edema of the lower extremity was treated with centripetal massage and wrapping. Resolution of swelling occurred over the course of several weeks. Compression garments were fabricated not only to maintain edema control but also to decrease hypertrophic scarring which developed in burned regions of the leg.

Progressive resistive exercises of all muscles in the lower extremity with strength of at least fair (3/5) led to marked improvement in strength. By 3 months postinjury the patient had regained normal strength about the hip girdle and knee.

General aerobic training, including cycle ergometry and swimming, improved the patient's endurance to a level at which he could maintain activity at 70% of his calculated maximum heart rate for 45 to 60 minutes.

The patient was reevaluated by the plastic surgery and orthopedic surgery services 4 months following injury. It was suggested that attempts at nerve grafting would not likely be beneficial and were not attempted. The possibility of triple arthrodesis to stabilize the ankle was entertained. The procedure was deferred by the patient who expressed that he was not limited functionally at the time, was experiencing only minimal discomfort in anesthetic regions, and was fully functional using the ankle foot orthosis.

Five months following the injury, the patient was fully independent and had been taken off all medication with the exception of trazadone, which helped relieve his deafferentation pain experienced in the evenings, allowing him to sleep soundly.

UPPER EXTREMITY NERVE INJURIES AND ENTRAPMENT SYNDROMES

Brachial Plexopathies

Anatomic Considerations

Brachial plexopathies are defined as nerve lesions involving any part of the peripheral nervous system distal to the nerve root and proximal to the main nerve branches. Thus, brachial plexopathy refers to postganglionic nerve pathology. The nerve lesion in brachial plexopathies occurs distal to the cell bodies of both the motor and sensory nerve fibers.

The fifth and sixth cervical nerve roots unite between the scalenus medius and scalenus anterior to form the upper trunk of the brachial plexus. The seventh cervical nerve root courses behind the lateral margin of the scalenus anterior to form the middle trunk. The eighth cervical and first thoracic nerve roots unite behind Sibson's fascia with the neck of the first rib between the two spinal nerves. Together they form the lower trunk, which emerges lateral to the fascia (Figure 9-51).

The upper, middle, and lower trunks divide into anterior and posterior divisions lateral to the first rib. The anterior divisions of the upper and middle trunk form the lateral cord and the posterior divisions form the posterior cord. The anterior division of the lower trunk forms the medial cord.

The cords then divide into their respective terminal branches. The lateral cord divides into the musculocutaneous nerve and the lateral head of the median nerve. The medial cord divides into the ulnar nerve and medial head of the median nerve. The posterior cord separates into the radial and axillary nerves. The long thoracic, subclavius, and dorsal scapular nerves branch off directly from the cervical nerve roots.

The long thoracic nerve is derived from nerve fiber contributions from the fifth, sixth, and seventh cervical nerve roots and innervates the serratus anterior muscle. The subclavius nerve arises from the fifth and sixth cervical nerve roots to serve the subclavius muscle. The dorsal scapular nerve is a branch of the fifth cervical nerve root and innervates both the rhomboids and the levator scapulae muscles.

The suprascapular nerve is the sole motor nerve branch emerging from the upper trunk shortly after its formation. It innervates the supraspinatus and infraspinatus muscles.

The subscapular, thoracodorsal, lateral pectoral, medial pectoral, and medial cutaneous nerves of the arm and forearm emerge from the cords of the brachial plexus. The subscapular nerve branches off the posterior cord to innervate the subscapularis and teres major. The thoracodorsal nerve also divides from the posterior cord to serve the latissimus dorsi muscle. The lateral pectoral nerve, a branch of the lateral cord, innervates portions of the pectoralis major and minor muscles. The medial pectoral nerve is a branch off the medial cord. It also contributes nerve fibers to the pectorals major and minor muscles. The medial cutaneous nerves of the arm and forearm are derived from the medial cord and innervate the medial arm and forearm, respectively.

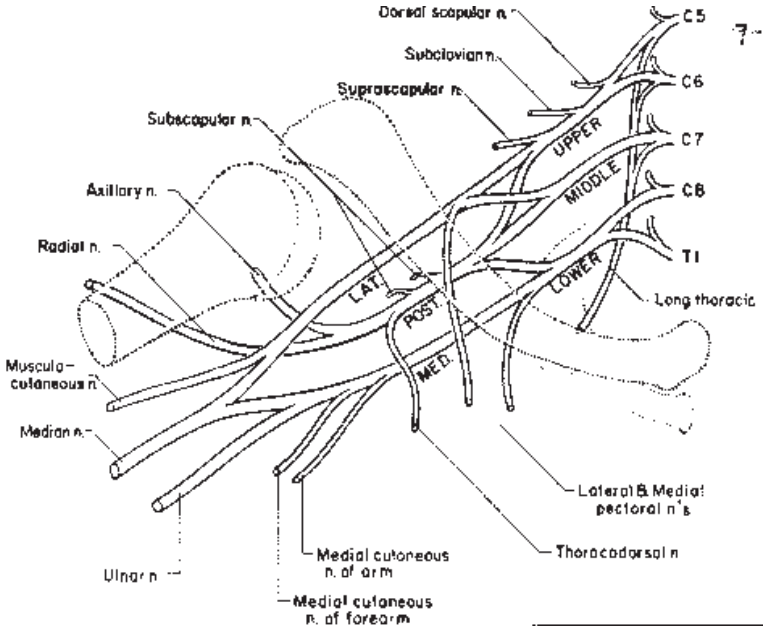

Fig. 9-51. The brachial plexus. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 40.

Etiology

Numerous clinical conditions are associated with plexopathies with an equally wide range of clinical presentations. These clinical conditions include arteriovenous fistula,[359] postradiation therapy,[360,361] postmedian sternotomy,[362–364] Parsonage-Turner syndrome,[365,366] polyarteritis nodosa,[367] viral infections (mononucleosis[368] and parvovirus[369]), herpes zoster,[370] trauma,[371,372] superior sulcus tumors,[373] and coagulopathies.[374]

Brachial plexus injuries may occur at any level and involve a variable degree of the plexus. The majority of lesions, in fact, are not uniform and various degrees of nerve damage typically coexist as a result of an injury.[9]

Brachial plexus injuries may be the result of wounds from knives and other penetrating objects, lacerations, bullet wounds, and shell fragments. The supraclavicular aspect of the brachial plexus is more prone to the latter injuries resulting in primary insult to the upper and middle aspect of the plexus. Lower plexus lesions may concomitantly involve insult to the lungs or great vessels. Thus, individuals with injury to this portion of the plexus are at significantly greater risk of sustaining a fatal wound.

Interestingly, the brachial plexus remains in continuity in a large percentage of open brachial plexus missile and shell fragment wounds.[9] Nelson et al[375] reported on nine cases during the Vietnam War in which patients sustained brachial plexus injury secondary to severe missile wounds of the chest. They found that all of these patients subsequently had rapid, spontaneous recovery of their plexus injury. It should be noted, however, that more severe injuries to the plexus may likely have been associated with significant vascular injuries. Disruption of the subclavian or axillary arteries in a combat setting likely would end in exsanguination. Thus, the authors may have observed only less severe injuries.

Brachial plexus injury can occur as a result of operative intervention due to improper retraction or inadvertent transection, clamping, or ligation of nerves. For example, median sternotomy performed as part of intracardiac surgery may damage the brachial plexus.[376] It may be iatrogenically induced by axillary puncture performed for arteriography as well.[377,378] Closed brachial plexus lesions may result from direct blows or prolonged, excessive pressure applied to the supraclavicular region. It most commonly occurs, however, as a secondary complication. Thus, the plexus can be lacerated by the edge of a fractured bone; by a sharp bone fragment; or from compression by dislocated or fractured bone, aneurysm, or hematoma.[9]

Brachial plexus injury has been documented in a variety of sports activities. Upper trunk damage, for example, has been recorded in hockey and lacrosse due to severe blows directed at the angle between the neck and shoulder.[379] A rifle's recoil has been reported to cause plexus injury by pressing the clavicle against the upper trunk.[380] Rucksack

palsy occurs as a result of upper trunk injury from direct downward pressure on the plexus or by trapping the plexus between the clavicle and the deeper structures, or both.[381] Traction injuries most commonly involve the upper plexus. Wynn Parry[382] reviewed multiple brachial plexopathies attributable to traction injury. He discovered that the upper plexus was 10 times more frequently injured vs the lower plexus. He also found that the great majority of injuries in his series were due to motorcycle accidents, followed by car accidents.

Clinical Presentation

Upper trunk injuries generally result in characteristic deficits. In complete lesions, the arm hangs at the side in an adducted and internally rotated posture. Atrophy is evident in the shoulder abductors, external rotators, and extensors. It is also found in the forearm flexors and supinators. Elbow extension and forearm pronation, as well as wrist and finger flexion strength are essentially preserved. Sensation to light touch and pinprick is generally diminished on the outer aspect of the arm and forearm, extending to the radial side of the hand.

Middle trunk damage occurs only infrequently as an isolated injury. Insult to the middle trunk will result in decreased elbow extension, as well as wrist and long-finger extensor weakness. Sensory deficits occur in the distribution of the C-7 nerve root.

Lower trunk injuries result in atrophy and weakness of the intrinsic muscles of the hand, and less prominent atrophy of the forearm flexor region as well. Sensory impairment is manifested by diminished cutaneous sensation from the medial aspect of the arm at a point just about the elbow extending to the ulnar aspect of the hand and little and ring fingers. In severe injuries, a complete plexus injury or panbrachial plexopathy may occur. In this case the patient presents with complete flaccid paralysis and diffuse loss or impaired sensation of the involved extremity. The patient may also have an ipsilateral Horner's sign consistent with compromise of the sympathetic ganglion at the C-8, T-1 level, as well as vasotrophic changes, particularly involving the distal aspect of the limb.

Injury to Nerve Branches of the Brachial Plexus

Long Thoracic Nerve

Nerve branches of the brachial plexus may be selectively injured. The long thoracic nerve may be damaged by electric shock and trauma, or it may occur as an idiopathic phenomenon (Figure 9-52).[383,384] Long thoracic nerve palsy has been documented to occur in temporal relationship to serum and vaccine injections.[385,386]

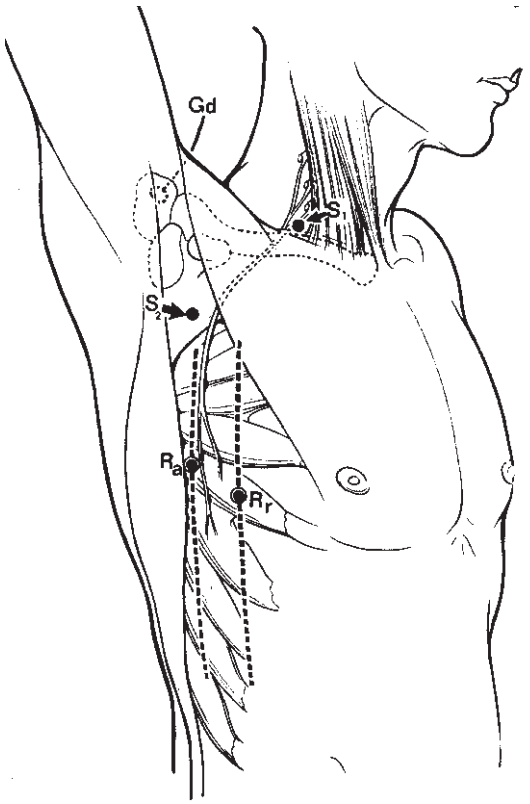

Fig. 9-52. The long thoracic nerve with motor nerve conduction electrode placement. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 48.

Suprascapular Nerve

Rarely, the suprascapular nerve is entrapped in the suprascapular notch(Figure 9-53).[387–390] The suprascapular nerve may be entrapped at the level of the spinoglenoid notch, resulting in isolated weakness of the infraspinatus muscle.[391,392] It may be specifically injured secondary to compression by ganglia at the spinoglenoid notch.[393]

Medial Antebrachial Cutaneous Nerve

The medial antebrachial nerve injury has occurred as a complication of surgical repair of a cubital tunnel syndrome.[394] It also may be damaged as a consequence of a stretch injury (Figure 9-54).[395]

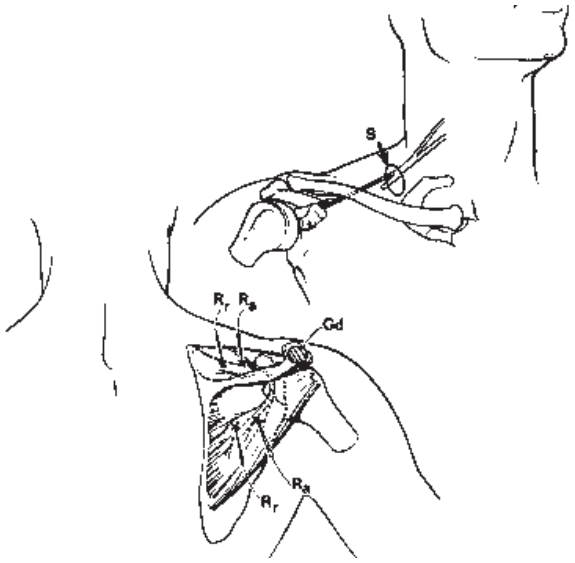

Fig. 9-53. The suprascapular nerve with electrode placement for motor nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 55.

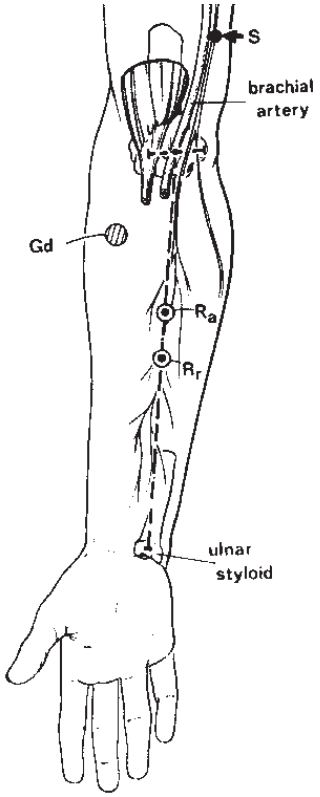

Fig. 9-54. The medial antebrachial cutaneous nerve with electrode placement for sensory nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985:147.

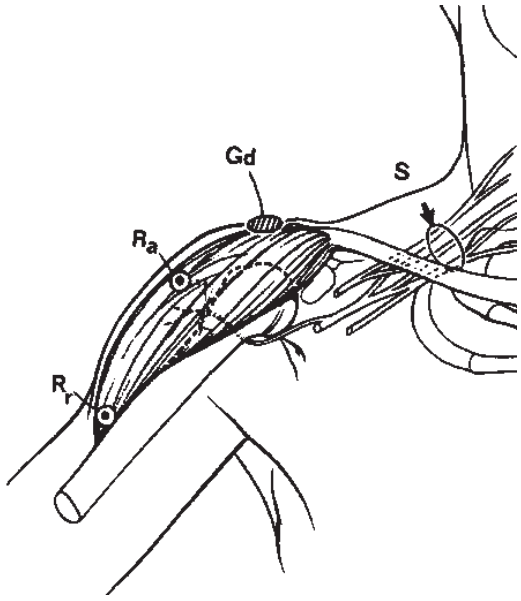

Fig. 9-55. The axillary nerve with electrode placement for motor nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 68.

Axillary Nerve

Isolated axillary nerve injury may occur following humeral head fractures, posterior dislocations or manipulations (Figure 9-55).[396,397] It may also occur after intramuscular injection into the posterior shoulder area.[398] Shoulder dislocation may also cause injury to the posterior cord, resulting in weakness in muscles innervated by the axillary and radial nerves (Figure 9-56).[399]

The axillary,[400] long thoracic,[401] and musculocutaneous nerves[402] may be injured secondary to surgical positioning or as a complication of obstetrical deliveries.

Musculocutaneous Nerve

The musculocutaneous nerve may be lacerated as a complication of a midshaft humeral fracture[403] or entrapped by the biceps aponeurosis and tendon at the level of the elbow during exercise activities.[404]

Electrodiagnosis of Brachial Plexopathy

One of the most important challenges an electromyographer encounters in evaluation of a brachial plexopathy is to distinguish between pre- and postganglionic lesions. In isolated preganglionic lesions,

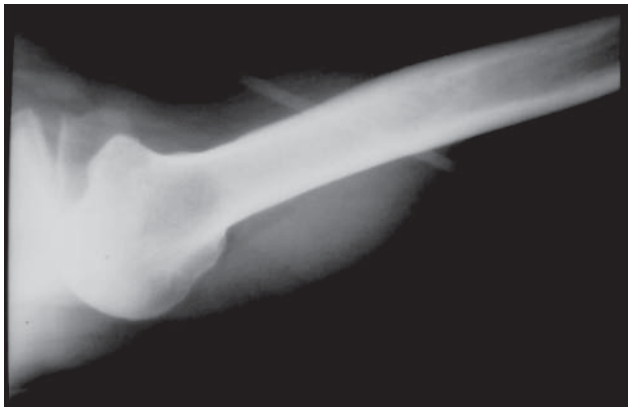

Fig. 9-56. Axillary view of posterior dislocation of the humeral head, which may be associated with injuries of the axillary nerve and posterior cord of the brachial plexus. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center. Washington, DC.

corresponding sensory NCSs are normal. In postganglionic nerve insult, however, pertinent SNAPs are either significantly reduced in amplitude or absent. This phenomenon occurs as a result of proximal axonal injury with resulting axonal degeneration distal to the site of insult.

It is important to recognize that SNAP amplitudes will not be abnormal for at least the first 3 to 5 days following proximal postganglionic nerve insult and may continue to conduct impulses for up to 2 weeks. It is also significant that temporal dispersion of the SNAP is not characteristic of most plexopathies, assuming there is not a concomitant systemic disorder or underlying polyneuropathy or both, or an isolated peripheral entrapment neuropathy. Sensory NCSs may provide valuable clues regarding the presence of a postganglionic brachial plexopathy, but are limited by the fact that sensory nerve studies may not be available for the suspected level of brachial plexus pathology. In addition, it is recognized that needle electrode evaluation is more sensitive in detecting and localizing nerve pathology than sensory NCSs.

Axonotmesis appears to be the most frequent pathological process associated with plexopathies.[405] Conduction abnormalities occurring in plexopathies include proximal focal slowing or conduction block, or both. Distal motor and sensory conduction velocities are not usually significantly affected in the presence of incomplete proximal injury, although slightly decreased conduction velocities may be recorded.

Thoracic Outlet Syndrome

Anatomic Considerations

The thoracic outlet syndrome (TOS) typically involves the lower trunk of the brachial plexus and may be secondary to neurologic or vascular disturbance, or both. It may, on occasion, affect the middle trunk as well. It has been described in various terms based on the proposed site and mechanism of presumed compression injury. These include compression secondary to cervical rib anomaly[406] and scalene anticus,[407] costoclavicular,[408] and hyperabduction syndromes.[409]

Three classifications of TOS are currently recognized[410]:

1. True, classic, or neurogenic TOS with objective neurologic impairment.
2. Disputed or symptomatic TOS with evanescent neurogenic symptoms and normal physical exam.
3. TOS secondary to vascular compression.

True neurogenic TOS is rare.[411] Neurologic impairment as a result of lower brachial plexus injury is felt to occur potentially at several sites[9]:

- posterior border of Sibson's fascia;
- crescentic tendinous fibers of the scalenus medius and scalenus anterior muscle;
- between the narrow tendinous angle between the scalenus medius and scalenus minimis, or between the scalenus medius and scalenus anterior muscles;
- sharp tendinous posterior border of the scalenus anterior muscle;
- abnormal rib or ligamentous extensions associated with the rib;
- bony prominence on the first rib;
- between the clavicle and a normal or abnormal rib; and
- between the aneurysm of the subclavian artery and underlying structures.

Etiology

The fundamental cause of TOS appears to be due to intermittent or chronic compression injury. This may occur from malalignment of normal structures, or may be secondary to an anomalous fibrous band. This band may attach to a cervical rib, but has also been noted to attach to the first thoracic rib or other

osseous structures. The anterior scalene syndrome is felt to result from lower plexus compression injury between the anterior and medial scalene muscles. This appears to be a particular problem for competitive swimmers in whom both scalene muscle hypertrophy and repetitive arm action predispose the athlete to plexus injury.[412] The costoclavicular syndrome implies lower plexus injury secondary to compression injury by the ribs or clavicle or both. An additional "subsyndrome" is the pectoralis minor syndrome, which is defined as plexus injury as it enters the axilla between the upper ribs and the pectoralis minor muscle. This subset of TOS is also felt to be precipitated or exacerbated by repetitive arm activity in the presence of pectoralis minor hypertrophy.[412]

Clinical Presentation

The actual frequency of TOS is unknown. Early reports of TOS may not have been lower trunk compression injury but CTS or cervical radiculopathy instead.[9,413]

The signs and symptoms of TOS are often vague. Physical examination findings are typically poorly defined, but it should be in the differential diagnosis in patients presenting with obscure upper extremity pain and numbness, along with weakness and atrophy of hand intrinsic muscles (Table 9-14).

Musicians, particularly flutists and violinists, are felt to be at particular risk for developing TOS.[414,415] There is a significantly greater incidence of TOS in females. The ratio is 9:1 for neurogenic TOS. Posture may play a role in this female predilection since drooping shoulders are more common in women.[410,416] True neurogenic TOS often initially presents with paresthesias, which may be followed by persistent pain along with development of weakness and muscle atrophy. The distribution of signs and symptoms generally follow the distribution of the medial cutaneous nerve of the forearm and the ulnar nerve. Pain is described as a poorly localized aching discomfort but may at times affect the whole arm. The pain is usually exacerbated with repetitive or heavy lifting or prolonged playing of selected musical instruments. There is usually an accompanying sensory deficit in the areas innervated by the medial cutaneous nerve of the forearm and the ulnar nerve.

The physical exam in TOS may offer few objective clues. The Adson maneuver, consisting of ipsilateral shoulder abduction and external rotation along with scapular retraction and neck rotation while monitoring for a decrease in the radial pulse, is felt to be an unreliable test, especially for neurogenic TOS.[412]

TABLE 9-14

MASQUERADERS OF THORACIC OUTLET SYNDROME

Pathology	Conditions
Neurologic	Multiple Sclerosis
	Parsonage Turner syndrome
	Traumatic brachial plexopathy
	Brachial plexopathy associated with Pancoast tumor
	Carpal tunnel syndrome
	Ulnar neuropathy
	Complex region pain syndrome
Intramedullary	Syringomyelia
	Spinal cord infarction
	Gliomas
Extramedullary	Neurofibromas
	Meningiomas
	Cervical spondylosis
	Herniated cervical intervertebral disk
Vascular	Atherosclerosis
	Systemic lupus erythematosus
	Aortic dissection

A variety of conditions and pressure from various soft tissue masses potentially mimic TOS and may need to be excluded. These include intramedullary and extramedullary disease processes as well as pathologies which may cause vascular insufficiency (see Table 9-14).

Electrodiagnosis

Nerve conduction studies have limited usefulness in identifying TOS. They are most helpful in excluding other conditions, particularly CTS and ulnar neuropathy. Sensitive nerve conduction findings supportive of a diagnosis of TOS include decreased ulnar SNAP amplitude at the wrist and decreased mixed nerve potential amplitude at the elbow. This drop in amplitude is attributable to axonal injury specifically affecting nerves at the level of the brachial plexus, which are destined to emerge as ulnar sensory fibers.[417] Other studies[417,418] have indicated that patients with TOS may show de-

creased median CMAP along with a decreased ulnar SNAP amplitude. The latter nerve conduction findings are currently felt to be the most sensitive criteria for neurologic TOS.

Proximal NCSs from Erb's point to an ulnar innervated muscle may have some merit if delayed conduction can be documented.[413,419] Some authors,[420,421] however, have not documented any abnormality with NCSs. It is not always clear whether the actual lesion is below, at, or above this site of stimulation. Furthermore, supramaximal stimulation is required that may result in stimulation of the nerve distal to the intended site, and there is significant potential for error when measuring from site of stimulation to the active electrode.

C-8 nerve root simulation, which has the advantage of definite stimulation proximal to Erb's point, has been advocated.[412] However, this technique is primarily useful in identifying focal demyelination injury, which is not characteristic of TOS. Nerve root stimulation is not currently an established technique for confirming TOS.[421] Weber and Kahn suggest that ulnar F-waves may be an effective way of studying conduction across the thoracic outlet.[412] Dawson and colleagues[422] acknowledge that there may be prolonged F-wave latencies in unambiguous TOS, but feel this slowing is not a sensitive measurement for this syndrome. Electromyographic studies may be helpful in confirming the presence of chronic denervation activity in the thenar and hypothenar muscles.

Treatment

A number of nonsurgical measures have been advocated to treat TOS. Arm restraints to prevent excessive shoulder abduction during sleep[422] and selective upper body exercises[423] have been suggested. A number of authors[424–426] have documented significant success with a formal exercise program.

Sunderland[9] has identified specific criteria for determining failure of conservative management. These include signs of muscle wasting, intermittent paresthesia followed by sensory loss, and progressive pain to the point of incapacitation.

Surgical management of TOS most commonly involves first cervical rib resection via the transaxillary approach, but this procedure remains controversial and indications are quite variable and not well-established. When constricting bands are identified during the procedure, they are released. Supraclavicular exploration is the procedure of choice for some surgeons.[413,427] It has the advantage of visualizing a cervical rib directly within the field and if constricting bands are present, they may be dissected without the need to remove the first rib. However, its disadvantages are less acceptable cosmesis, potential phrenic or long thoracic nerve injury or both, and more extensive dissection.[422] Unfortunately, severe, persistent CRPS-Type II and functional deficit can result from surgical intervention. Thus, it is prudent to limit surgical management to those patients with unequivocal clinical evidence of true neurogenic or true vascular compromise.[422]

Radial Nerve Injuries and Compression Syndromes

The radial nerve is especially vulnerable to injury at several sites. These include injury to the main trunk of the radial nerve at the level of the spiral groove, posterior interosseous injury at the elbow, and entrapment or injury of the superficial radial nerve in the distal third portion of the forearm or at the wrist.

Injuries and Compression in the Spiral Groove Area

Anatomical considerations. The radial nerve is a branch of the posterior cord of the brachial plexus with neural fiber contributions from the fifth to eighth cervical and first thoracic nerve roots (Figure 9-57). It begins at the lower border of the pectoralis minor. In the arm, it winds around the posterior aspect of the humerus and passes along the musculospiral groove. It emerges anterior to the distal arm about proximal to the lateral epicondyle. In the upper arm, the radial nerve branches innervate the three heads of the triceps, the anconeus, and the upper portion of the forearm extensors and supinator muscles. Sensory branches of the radial nerve include the posterior brachial cutaneous, posterior antebrachial cutaneous, and superficial radial nerves. The posterior aspect of the upper arm is supplied by the posterior brachial cutaneous nerve. The dorsal surface of the forearm is supplied by the posterior antebrachial cutaneous nerve. The superficial radial nerve provides sensory innervation to the dorsal aspect of the radial half of the hand.

Radial nerve branches to the long and medial heads of the triceps arise in the axilla. The radial nerve may be accompanied in the spiral groove by the branches to the medial and lateral heads of the triceps. The main radial nerve trunk typically lies directly against the humerus in the groove area, but its branches lie in a plane between the heads of the triceps. Thus, the main radial nerve bundle is more

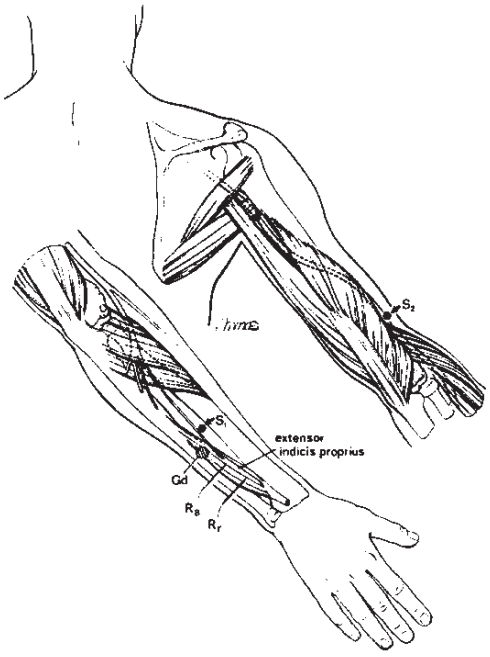

Fig. 9-57. The radial nerve with electrode placement for motor nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 73.

vulnerable to injury secondary to mid-third fractures of the humeral shaft. This is in contrast to crutch palsy in which both the radial nerve branches and the main radial nerve suffer compression injury between a crutch and the underlying muscle tissue.[9]

The posterior cutaneous nerve of the arm and forearm accompanies the radial nerve to the spiral groove but lies more posterior relative to the humeral shaft. Thus, it too is less vulnerable to injury than the main radial nerve in the event of mid-third humeral fractures.[9]

As the radial nerve emerges from the spiral groove, it sends a branch to the brachioradialis, followed slightly more distally by a branch to the extensor carpi radialis longus. It then passes between the brachialis and brachioradialis before entering the forearm.

Etiology. The radial nerve may incur injury as a result of direct, prolonged pressure, such as in Saturday night palsy.[428] This form of nerve lesion characteristically occurs in cachectic and fatigued persons who are intoxicated or suffering from drug narcosis. Thus, they are in a state in which they fail to perceive and react to the stages of progressive nerve injury.

Humeral fractures, as previously noted, may be associated with radial nerve injuries in the upper arm.[429,430] Injury may occur as a result of direct injury associated with the trauma causing the fracture or by the fracture edges. Also, the radial nerve is relatively fixed where it leaves the spiral groove. This predisposes it to traction injury when the fractured segments of the humerus are traumatically separated.[9] Patients sustaining fractures of the distal third of the humerus are also at risk for radial nerve injury.

Radial nerve entrapment has been documented to occur in an area of drug-induced (pentazocine) fibrotic changes within the lateral head of the triceps.[431] Radial nerve injury has also been attributed to compression by this muscle during severe muscular exertion.[432] Lotem et al[433] reported three cases of radial nerve injury manifested by transient paresis following intense muscular effort, specifically elbow extension. Sunderland cites Gowers in 1892 who wrote,

> I have three times seen paralysis from a violent contraction of the triceps, once during the act of pulling on a tight pair of boots, once from throwing a stone with energy, and once from grasping a lamp-post to avoid a fall during a severe attack of giddiness. In each the nerve was at once completely paralyzed; and in the second, in which the palsy was severe, a bruised appearance was observed over the lower part of the triceps.[9]

Clinical presentation. Radial nerve compression at the spiral groove may cause wrist drop due to wrist and finger extension weakness, along with weakness of the brachioradialis. The triceps muscles are typically spared. Sensory impairment may involve the dorsal interspace between the thumb and index finger and/or the proximal halves of the thumb, index finger, and middle finger.[389]

Treatment. Treatment is directed at surgically removing the offending compressive mass or eliminating the precipitating posture. Wrist-hand splints with the hand and wrist placed in a position of function may be prescribed (Figure 9-58). Any splinting should be coupled with a daily range-of-motion exercise program, both passive and active assistive depending on the degree of weakness present.

Posterior Interosseous Nerve Entrapment

Anatomic considerations. The posterior interosseous nerve is a motor branch of the radial nerve (Figure 9-59). The radial nerve bifurcates into

Fig. 9-58. Dynamic dorsal wrist hand orthosis with low profile outriggers assisting metacarpophalangeal extension and thumb abduction. The wrist is set in a functional position.

a sensory branch and the posterior interosseous nerve, usually at the level of the radiocapitellar joint. In some cases, it may separate 2-5 cm proximal or distal to this joint.[434] When the nerve is directly anterior to the radiohumeral joint capsule and the radial head, it lies lateral to the biceps tendon and bicipital bursa and medial to the supinator muscle.[9] It then passes between the two heads of the supinator muscle and innervates this muscle as it courses through it. It proceeds dorsolaterally around the neck of the radius within the substance of the supinator muscle. The extensor carpi radialis brevis is innervated by the radial nerve at or distal to the radial head.

The posterior interosseous nerve subsequently separates into two groups comprised of multiple branches. One group innervates the superficially lying forearm extensor muscles, including the extensor digitorum communis, extensor digiti quinti, and extensor carpi ulnaris. The other innervates the deeper lying forearm extensors, including the abductor pollicis longus, extensor pollicis longus and brevis, and the extensor indicis proprius.

The posterior interosseous nerve enters the supinator through an inverted, fibrous arch known as the arcade of Frohse. It is formed by the thickened tendinous edge of the proximal border of the superficial head of the supinator.

Etiology. The posterior interosseous nerve is susceptible to both intrinsic and extrinsic compression injury. It can be entrapped by the aforementioned arcade of Frohse,[435–437] the supinator,[438] tumors (most commonly lipomas),[439,440] ganglia, and rarely, elbow synovitis.[422,441] It has also been documented to occur after fracture or dislocation of the radial head and specifically with Monteggia fractures (radial head dislocation along with fracture of the ulna)[442–444] and as a complication of Canadian forearm crutch use.[445] In some of patients, the posterior interosseous nerve is said to lie directly on the periosteum of the proximal radius, making it particularly susceptible to damage by a fracture of the proximal radius or by a proximally placed metal plate used to stabilize the fracture.[434] Vascular leashes enveloping the nerve have been found to be potential causes of posterior interosseous nerve compression.[422]

Clinical presentation. Patients present with compromised wrist extension strength. They also have finger extension weakness at the MCP joints and thumb extension and abduction paresis. They retain radially deviated wrist extension because the extensor carpi radialis longus and brevis muscles are spared, each receiving a branch from the radial nerve proximal to the supinator. The brachialis and triceps are also spared. Prior to developing the combination of wrist, thumb, and finger extension weakness, these patients may present with isolated inability to fully extend their little and ring fingers.

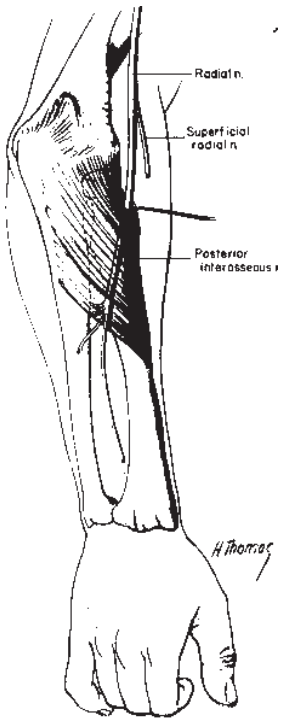

Fig. 9-59. The posterior interosseous branch of the radial nerve. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 35.

Muscles that are spared following a posterior interosseous compression neuropathy include extensor carpi radialis longus, the extensor carpi radialis brevis, and the supinator. The muscles that are affected are the extensor carpi ulnaris, including the extensor digitorum communis, extensor indicis proprius, abductor pollicis longus, extensor pollicis brevis, and the extensor pollicis longus.

Although the posterior interosseous nerve is a motor nerve, compression injury has caused paresthesias in some cases.[446] Pain begins at the elbow or proximal forearm area in about half of the patients.[447] Pain is usually acute, although it may also be gradual in onset. There may be reproducible tenderness with palpation over the forearm extensor muscle mass just distal to the radial head.

Electrodiagnosis. Nerve conduction studies of the radial nerve in the forearm have shown a prolonged motor latency consistent with focal slowing involving the posterior interosseous nerve. The study is performed by stimulating the radial nerve at the level of the elbow with active electrode pickup of the action potential on the extensor digitorum communis muscle.[448] The superficial radial NCS is normal. Electromyography may reveal spontaneous activity consistent with muscle membrane instability and suggestive of denervation activity involving all radially innervated forearm muscles except for the extensor carpi radialis longus and brevis and supinator muscles.

Treatment. In most cases, clinically or electrodiagnostically or both, documented posterior interosseous nerve entrapment is managed surgically. This includes exploration and resection of the offending mass, such as a tumor or ganglia, or the release of compressive fibrous bands.

A wrist-hand splint fabricated in a position of function may be used pre- or postoperatively to address the incomplete wrist weakness and finger and thumb weakness. A static splint may be used at rest and a low-profile dynamic extension splint used during the day. Daily passive and active wrist flexion and extension to the degree capable should also be included in the therapeutic program.

Superficial Radial Nerve Compression

The superficial radial nerve courses subcutaneously along the lateral aspect of the radius. It is particularly vulnerable to extrinsic pressure because of its superficial position. Excessively tight wristbands or handcuffs may cause focal nerve compression injury resulting in sensory impairment involving the radial aspect of the hand. This nerve may take an aberrant course or there may be variations in the distribution of cutaneous innervation, thus confusing the clinical picture.

There are multiple documented causes of superficial radial nerve compression injury[412,434,449]: constricting wrist bands, handcuffs, forearm hemorrhage, excessively tight surgical gloves, vein cutdown procedure, operative repair of deQuervain's tenosynovitis, casting for Colle's fracture, schwannoma of the superficial radial nerve, and fibrous tendon joining brachioradialis and extensor carpi radialis.

Injury to the superficial radial nerve results in isolated numbness, paresthesias, or painful dysesthesia without motor deficits. There may be a positive Tinel's sign in the lateral forearm area. Symptoms may be enhanced with placing the forearm in end-range pronation coupled with ulnar wrist flexion.[450]

Nerve conduction studies of the superficial radial nerve may result in an absent response, or prolonged latency with attenuation of the amplitude and temporal dispersion of the sensory action potential. Electromyography is of no value in this syndrome except to exclude other nerve pathology.

Treatment is directed at removing the offending constriction or focal pressure. Causalgias related to superficial nerve compression have been documented as a complication of some superficial radial nerve lesions.[451] Nerve repair, excision of an associated neuroma, and cervical sympathectomies have been performed to address this problem with some success.[434]

Median Nerve Injuries and Compression Syndromes

The median nerve is vulnerable to compression injury at three major sites. At the elbow; at the ligament of Struthers, where the main trunk of the median nerve may be entrapped; or between the two heads of the pronator teres. In the forearm, the anterior interosseous nerve, a motor branch of the median nerve, is subject to compression. The most common entrapment neuropathy, CTS, occurs secondary to compression at the distal edge of the transverse carpal ligament or, less commonly at the intermetacarpal tunnel.[160] A number of less common sites of median nerve compression have been observed[411]:

- post stupor with proximal median nerve compression at entrance to canalis brachialis at inferior border of pectoralis major;

- injury secondary to fracture of distal third of humerus;
- entrapment by lacertus fibrosis (fibrous band connecting biceps tendon to flexor carpi radialis);
- injury secondary to elbow dislocation;
- injury secondary to fracture—dislocation of distal radioulnar joint;
- entrapment by hemodialysis loop grafts at both the elbow and in the forearm;
- entrapment by lateral border of flexor digitorum superficialis;
- distal forearm fibrovascular band;
- radial artery puncture at the wrist with direct trauma or injury secondary to bleeding; and
- compression injury of palmar cutaneous branch secondary to ganglia.

Supracondylar Process Syndrome

Anatomic considerations. The ligament of Struthers is an anomalous fibrous band (Figure 9-60). It has been documented to have an incidence of 0.7% to 2.7%.[452] It extends from its attachment on the supracondylar process of the distal anteromedial humerus (usually via a bony spur) to the medial epicondyle.[160] The bony spur may be palpable and is evident on a radiograph.[453] The ligament may be bilateral[411] and is not always associated with a bony spur.[454]

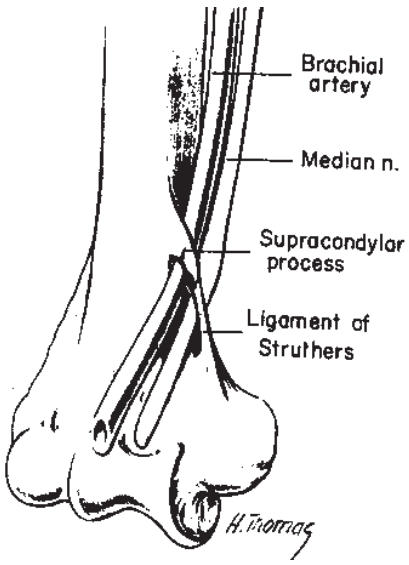

Fig. 9-60. The median nerve and brachial artery passing between the distal humerus and the ligament of Struthers. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 28.

Etiology. The median nerve may be entrapped under the ligament of Struthers just proximal to the antecubital space. Some feel the compression injury is due to a combination of static impingement and dynamic injury associated with repetitive motion at the elbow.[412]

Clinical presentation. This syndrome may result in weakness of all median nerve innervated forearm and hand muscles, including the pronator teres. It may also result in sensory deficits in the hand in the distribution of the median nerve. The brachial artery also passes under the ligament of Struthers when it is present. If it too is compressed, a patient may present with distal vascular and neuropathic changes.[411]

Electrodiagnosis. Median nerve conduction velocity across the site of presumed entrapment at the elbow may be slowed.[455,456] An EMG of selected forearm and hand muscles innervated by the median nerve may show evidence of denervation activity.

Pronator (Teres) Syndrome

Anatomic considerations. The median nerve crosses the elbow in close approximation to the brachial artery and biceps tendon. The biceps tendon is lateral, the median nerve is medial, and the brachial artery courses between these two structures. The median nerve enters the forearm and passes under the lacertus fibrosus, a thick fascial band extending from the biceps tendon to the forearm fascia. It then courses between the superficial and deep head of the pronator teres and passes beneath the fibrous arch of the flexor digitorum superficialis.

Etiology. The median nerve may be compressed by a thickened lacertus fibrosus, by a hypertrophied pronator teres or fibrous band within this muscle, between the superficial and deep heads of the pronator teres, or by the edge of the fibrous arch of the flexor digitorum superficialis (Figure 9-61).[422] Fractures of the forearm as well as elbow dislocation may secondarily injure the median nerve (Figure 9-62). Median nerve injury in this forearm area has also been documented in association with compartment syndrome.[457] A persistent median artery as a cause of pronator teres syndrome, secondary to compression of the nerve by the artery, was reported by Jones and Ming.[458] Luce et al[459] noted that in patients on anticoagulants, repeated attempts at brachial artery puncture may also cause median nerve injury. This compressive injury secondary to bleeding may develop insidiously.

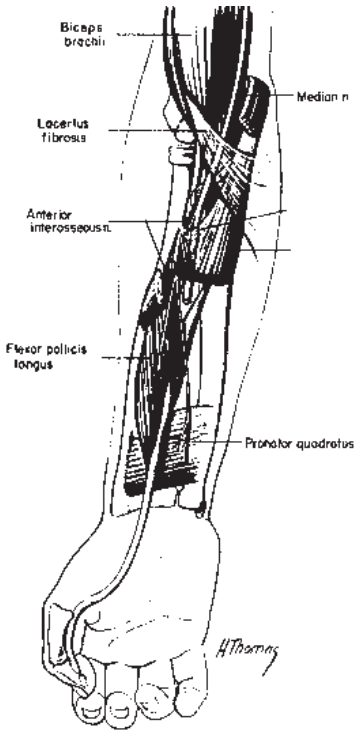

Fig. 9-61. The median nerve may be entrapped by the lacertus fibrosis or the fibrous arch of the flexor digitorum sublimis (superficialis) as well as between the deep and superficial heads of the pronator teres muscle. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 25.

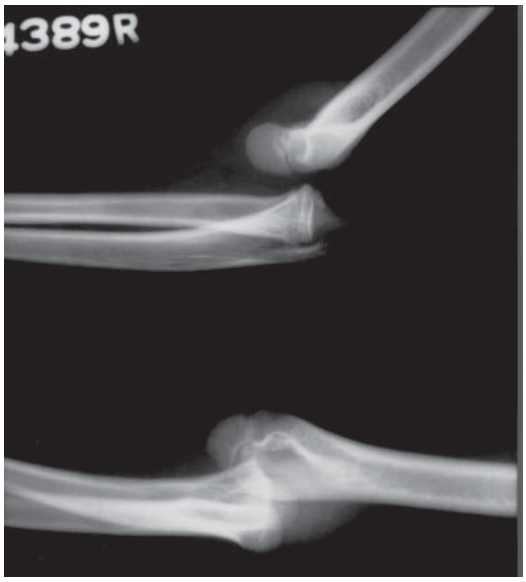

Fig. 9-62. The median nerve may be injured following dislocation of the elbow. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

Clinical presentation. Patients usually complain of an aching discomfort in the proximal forearm, exacerbated by activities requiring repetitive or strenuous forearm pronation. There may be proximal radiation to the elbow or even the shoulder, along with a sense of heaviness and easy fatigability with use of the arm. Poorly localized paresthesias may be present, usually intermittently if at all. Nocturnal exacerbation of symptoms is not characteristic.

Physical examination usually reveals dull tenderness with palpation over the pronator teres muscle mass. The pronator teres may appear enlarged upon palpation. Occasionally, a sharp pain will be elicited instead, and a tap over the pronator teres may produce a Tinel's sign.

Muscle strength testing reveals variable weakness of the median nerve innervated forearm flexors, including the palmaris longus, flexor carpi radialis, flexor digitorum profundus (radial portion), flexor digitorum superficialis, flexor pollicis longus, and pronator quadratus. The pronator teres, on the other hand, is typically spared because branches to this muscle emerge from the median nerve prior to the aforementioned entrapment sites. There is also variable weakness of the median innervated hand intrinsics.

Spinner describes three tests that may be helpful in clinically supporting the presence of pronator teres syndrome.[434]

1. One test is performed by placing the forearm in full pronation and the wrist in flexion. An examiner then passively supinates the forearm and extends the wrist while the patient actively resists this maneuver. A positive test is manifested by precipitation or exacerbation of pain in the proximal forearm and suggests median nerve compression in this area by the pronator teres.
2. A second test involves full supination of the forearm concomitantly with elbow flexion. The examiner passively pronates the forearm while the patient resists the action. The patient's active biceps contraction causes tightening of the lacertus fibrosus. A positive test is manifested by precipitation or exaggeration of symptoms and suggests that the lacertus fibrosus is the compressive structure.
3. A third test directs the patient to flex the proximal IP joint of the middle finger against resistance, thus activating the flexor digitorum sublimis muscle. If this

test precipitates or increases the patient's discomfort, it is suspected that the arch of the flexor digitorum sublimis may be causing median compression injury.

Electrodiagnosis. The pronator teres syndrome is primarily a clinical diagnosis. Electrodiagnostic testing detects abnormalities in only a minority of patients studied.[422] Median motor and sensory conduction velocities may be slowed in the forearm segment. The sensory action potential amplitude distally may be reduced with compression injury of the median nerve in the forearm. Nerve conduction studies are infrequently abnormal, but can be helpful in excluding other neuropathic conditions in the differential, including CTS. Electromyography provides the most useful information when abnormal findings, which are suggestive of denervation activity, are found in both median-innervated forearm and hand intrinsic muscles, excluding the pronator teres.[454]

Treatment. Conservative management includes avoidance of precipitating or exacerbating activity and use of NSAIDs. Temporary use of static elbow and wrist splints in a position of rest of the biceps, supinator, pronator teres, and forearm wrist flexors, followed by gently progressive range-of-motion activities to restore pain-free mobility, may be helpful in some patients.

In patients who sustain acute median nerve compression in the proximal forearm secondary to crush injury, bleeding, or other causes of increased intracompartmental pressure, fasciotomy with median nerve decompression is advocated. If this surgical intervention is performed in a timely fashion, there is excellent prognosis for median nerve recovery. This may not be the case for individuals suffering from chronic median nerve compression. Their prognosis for recovery is guarded.[422]

Anterior Interosseous Syndrome

Anatomic considerations. The anterior interosseous branches off the main median nerve approximately 5 to 8 cm distal to the lateral epicondyle. It is predominantly a motor nerve. Although it has no superficial sensory fibers, it does carry pain and proprioception fibers from deep forearm soft tissue structures and from the wrist joints, including the radiocarpal, intercarpal and carpometacarpal, and distal radioulnar joints.[422] It innervates three forearm muscles, the flexor pollicis longus, the flexor digitorum profundus (radial portion), and the pronator quadratus.

Etiology. The anterior interosseous syndrome is also known as the syndrome of Kiloh and Nevin.[460] The site of injury is just distal to the pronator teres muscle. The anterior interosseous nerve may be selectively compressed by any number of structures or directly injured secondary to trauma.

Causes of anterior interosseous syndrome[411,422] include

- tendinous origin of deep head of pronator teres;
- tendinous origin of flexor digitorum sublimis to the middle finger;
- accessory muscles and tendons from the flexor digitorum, flexor pronator quadratus, and other forearm muscles;
- fibrous bands of the flexor digitorum sublimis or flexor digitorum profundus;
- aberrant vessels and thrombosed collateral vessels;
- excessive forearm exertion;
- direct trauma;
- forearm fractures, lacerations, gunshot wounds;
- drug injections;
- postoperative complication of open reduction internal fixation of forearm fractures;
- venus cutdowns; and
- extrinsic pressure (cast, heavy handbag, prolonged leaning on forearm).

Collins and Weber[461] describe actual avulsion of the anterior interosseous nerve secondary to trauma. Anterior interosseous syndrome has also been documented to occur in association with metastatic bronchiogenic carcinoma involvement of the forearm.[462]

The anterior interosseous syndrome may be clinically mimicked by more proximal inflammatory nerve lesions or more proximal compression injury. For example, the anterior interosseous neural fibers can be selectively involved in brachial neuritis (neuralgic amyotrophy or Parsonage-Turner syndrome).[463,464] A pseudo-anterior interosseous nerve syndrome involves partial median nerve injury secondary to catheterization at the antecubital fossa level, presumably selectively involving nerve fibers specifically destined to form the anterior interosseous nerve. Elbow dislocations have been documented to produce a similar presentation (see Figure 9-62).[465,466]

Rupture or entrapment of the flexor pollicis longus tendon will cause isolated inability to flex the IP joint of the thumb. This tendon, along with the

flexor digitorum profundus to the index finger, is especially vulnerable to rupture in rheumatoid arthritis on the sharp edge of an eroded scaphoid tubercle. This phenomenon can mimic a partial anterior interosseous syndrome. A history of locking or snapping of the thumb at this joint and the absence of any weakness of the pronator quadratus or flexor digitorum profundus muscle to the middle finger will help to differentiate this problem from the anterior interosseous syndrome. If due to rheumatoid arthritis, one may detect clinical evidence of prominent flexor tenosynovitis, and wrist radiographs may reveal scaphoid bone erosion.[422]

Clinical presentation. Patients classically present with acute pain in the proximal forearm region and sometimes in the elbow area as well. They do not typically have any sensory symptoms, although poorly localized and described sensory complaints may be noted on occasion. Weakness is variable and limited to the flexor pollicis longus, flexor digitorum profundus to the index and middle fingers, and the pronator quadratus. These patients experience difficulty making the circular "OK" sign with their thumb and index finger. They form a triangle instead because of the weakness at the IP joint of the thumb and the distal IP joint of the index finger. For the same reason, they are unable to pinch with the tips of their thumb and index finger effectively. They typically have variable loss of dexterity in fine motor tasks as well.

Electrodiagnosis. Conventional median motor NCSs, which involve stimulation at the wrist and from above the elbow with pickup at the abductor pollicis brevis muscle, are normal. However, median motor conduction from the elbow to pickup over the pronator quadratus may reveal a prolonged latency and temporal dispersion of the CMAP.[467] Median sensory NCS is normal and its value is primarily the exclusion of other median nerve pathology. Elecromyography may reveal evidence of variable degrees of denervation activity involving the flexor digitorum profundus I and II, flexor pollicis longus, and pronator quadratus.

Treatment. Management of anterior interosseous nerve injury is highly dependent on the cause. If a penetrating wound is the cause, immediate surgical exploration and repair is warranted. If a crush injury with impending Volksmann ischemic contracture is the cause, immediate decompression is clearly indicated. If it is spontaneous or clearly related to a given activity, relative rest with avoidance of the precipitating or exacerbating activity may be sufficient to provide relief. Temporary splinting of the forearm may offer greater assurance of adequate immobilization and rest. Nonsteroidal antiinflammatory medications and local steroid injections in the region of the pronator teres may be helpful in alleviating persistent discomfort.

There is little consensus regarding timing of surgical intervention in patients who suffer persistent discomfort despite conservative management. However, the surgical literature tends to favor surgical exploration in those patients with spontaneous onset of a complete anterior interosseous nerve deficit if they show no significant improvement after 12 weeks.[468,469] Most favor waiting an even longer period of time in patients who present with only incomplete anterior interosseous nerve palsy.

Dawson and colleagues[422] suggest a period of at least 6 months of conservative treatment in patients with spontaneous anterior interosseous syndrome. This rationale is based on their experience that most cases of spontaneous anterior interosseous nerve palsy are due to a neuritis rather than compression injury and that these patients characteristically recover completely within about ten months after onset.[470]

Carpal Tunnel Syndrome

Anatomic considerations. Prior to entering the carpal tunnel, the palmar cutaneous nerve is the last major branch of the median nerve in the forearm, usually arising about 3 to 4 cm proximal to the proximal edge of the transverse carpal ligament. Thus, it is spared in CTS. It serves the thenar region of the hand and a variable portion of the palm.

The median nerve becomes more superficial at the distal forearm between the tendons of the palmaris longus and the flexor carpi radialis. It then passes through the fibroosseous carpal tunnel (Figure 9-63). The radial wall of this tunnel is formed by the scaphoid and trapezium and ulnar wall by the pisiform and hamate. The lunate, capitate, trapezoid, and associated ligamentous structures form its floor or dorsal surface. The transverse carpal forms the roof or volar surface. The transverse carpal ligament attaches to the tubercle of the scaphoid and trapezium laterally and the hamate medially. Nine extrinsic wrist flexor tendons accompany the median nerve through this tunnel. They include the flexor pollicis longus (1), flexor digitorum superficialis (4), and flexor digitorum profundus (4) tendons. The distal volar skin crease of the wrist corresponds to the proximal border of the carpal tunnel, which extends 3 cm distal to this crease.[422,434] The carpal tunnel narrows in cross-section at 2 to 2.5 cm distal to the entrance

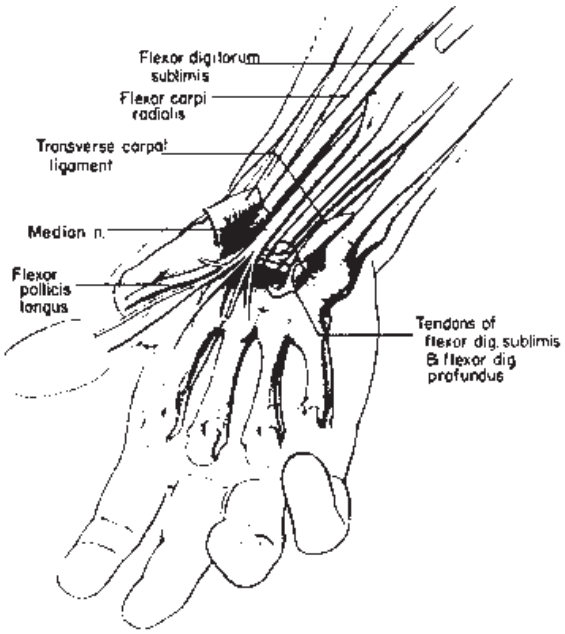

Fig. 9-63. The carpal tunnel. The median nerve, tendons of both the flexor digitorum profundus and superficialis, and flexor pollicis longus travel beneath the transverse carpal ligament which has been reflected. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 20.

of the tunnel.[471] In CTS, the size of the myelinated nerve fibers are significantly reduced at this point.[472]

As the median nerve emerges from the carpal tunnel, it splays outward into both motor and sensory fibers. The motor nerve, in close proximity to the common palmar digital nerve to the thumb, curves over or through the flexor pollicis brevis. It supplies the superficial head of the flexor pollicis brevis as it courses over or through it and subsequently divides to serve the other thenar muscles, the abductor pollicis brevis and the opponens pollicis.

The sensory fibers classically serve the skin of the radial aspect of the palm and volar surface of the thumb, index, and middle fingers, and the radial half of the ring finger. Dorsally, sensory fibers from the median nerve supply the skin distal to the proximal IP joints of the index and middle fingers and the radial aspect of the little finger. The dorsal surface of the distal thumb may be innervated by either the median or radial nerve.[422,473] It is clinically important to be aware that a multitude of anatomic variations exist regarding median sensory supply to the hand and digits.

Etiology. Carpal tunnel syndrome is the most common entrapment neuropathy. Space-occupying lesions within the carpal tunnel are the most common cause of this syndrome. A wide variety of conditions are associated with CTS. All of them, to some degree, compromise the limited space within this relatively unyielding fibro-osseous structure.

Acute CTS occurs as a complication of multiple traumatic injuries involving the wrist or hand. It has been associated with Colle's fracture (Figure 9-64).[474] It has also been documented to occur after epiphyseal fracture of the distal radius, after fracture of both the ulna and radial bones, and after dislocation or fracture-dislocation of the carpus.[475,476] Suppurative flexor tenosynovitis has been documented to cause abrupt onset of CTS as well. It may be complicated by acute median nerve paralysis requiring immediate decompression to prevent irreparable damage.[422]

Crush injury of the hand or wrist results in marked swelling and may be complicated by Volkmann's ischemic contracture. Such patients are at high risk for developing acute CTS. Individuals taking anticoagulants are at increased risk for bleeding into the carpal tunnel[477] or into the median nerve itself,[478] with consequent development of acute or chronic CTS.

Chronic CTS may occur as result of any of the aforementioned traumatic injuries. It may also develop secondary to prolonged or repetitive extrinsic trauma, such as occasionally occurs after walking with axillary crutches. Crutch walking in a susceptible patient has been documented to cause

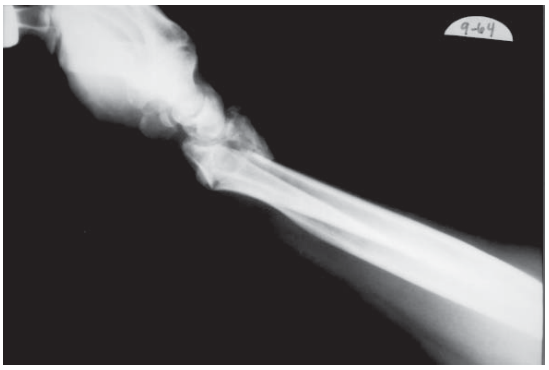

Fig. 9-64. Distal radial fracture with dorsally deviated distal segment (Colle's fracture), which may be associated with median nerve entrapment at the wrist. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

chronic compression injury of the motor branch of the median nerve.[422]

Clinical presentation. Carpal tunnel syndrome is a consideration in any patient who presents with numbness, painful dysesthesias, weakness, and impaired dexterity of the hand. In most patients, there is a characteristic presentation, but a significant number present in an atypical fashion and require electrophysiologic testing for definitive diagnosis. Carpal tunnel syndrome affects females more often than males. The most common age of onset is in the fifth or sixth decades, although many present earlier, particularly if their daily activities or occupation involves extensive manual labor. Symptoms most often affect the dominant hand,[479] but bilateral complaints are not uncommon.

Numbness classically involves the thumb, index finger, and radial half of the ring finger. Some have found that numbness is most commonly present in middle finger or both the middle and index fingers.[422] Pain often occurs at the wrist with proximal radiation but may be present in the hand, forearm, elbow, and shoulder as well. The pain is described as an aching, cramping, or burning discomfort. Shaking or rubbing the hand may offer momentary relief. Weakness is not a typical complaint, although patients may complain of tiredness and easy fatigability when performing manual activities. They may also complain of dropping items with increasing frequency. However, this is most likely related to their sensory deficit rather than weakness. There may be an increased sensitivity to cold, and in some individuals with advanced disease, vasotrophic changes, including excessive sweating, edema, and skin color changes, may occur.

Early in the course of CTS, there are generally no objective physical findings. Later, slight hypesthesia develops in the median nerve distribution. Several investigators have noted that a significant percentage of patients initially present with decreased sensation involving the middle finger.[422,480] As the compression neuropathy advances, progressive loss of sensation, including impaired two-point discrimination, occurs.

A Tinel's sign may be elicited. It is produced by tapping gently over the volar wrist skin crease. A positive sign is manifested by a tingling or electrical shock sensation radiating into one or more of the median innervated digits. It most often is elicited in patients with severe CTS. However, it can produced in a number of patients without CTS. For this reason, it is not considered a sensitive or specific test for CTS. Nevertheless, when Tinel's sign is positive in the context of other characteristic signs and symptoms, it is supportive of the diagnosis. The Phalen's test is produced by placing the wrist in full flexion for 30 to 60 seconds. Symptoms will be exacerbated by this maneuver in a positive test. Phalen[480] found the test positive in 80% of 484 hands tested. Although a number of patients with definite CTS may not have a positive Phalen's test, when it is positive, it is a relatively reliable sign.

Dawson and colleagues[422] have grouped patients with CTS into three categories based on the severity of their symptoms. This grouping permits clearer identification of the patterns of presentation and thus aids in diagnosis. It also offers guidelines for treatment and prognostication.

Group I. Patients in Group I present with mild symptoms of intermittent numbness, tingling, and pain the median nerve distribution. They have nocturnal exacerbation of their symptoms and may be awakened from their sleep by pain. They may also relate that their symptoms are increased with driving, holding up a newspaper to read, and with manual labor, particularly sewing or other tedious hand activities. They have a positive Flick sign, which is manifest by vigorous shaking of their affected hand to "get the circulation going" with momentary, partial relief of their symptoms. Symptoms are initially sporadic, followed by increasing frequency. The physical examination is essentially normal, except perhaps for a positive Tinel's sign at the wrist or a positive Phalen's sign.

Group II. Group II CTS patients complain of persistent symptoms. Pain is their primary complaint. They also have hypesthesia, a sense of clumsiness along with actual decrease in dexterity with fine motor tasks. They have prominent nocturnal exacerbations and characteristically experience a notable increase in symptoms with manual activities. They complain of numbness, burning pain, and a sense of swelling in their hands. They usually have positive Flick, Tinel's, and Phalen's signs. They may rub their hands together or place the affected hand under water in an effort to obtain relief. On examination, these patients have objective weakness in thumb abduction and opposition, and may show thenar atrophy. They typically have sensory impairment in the median nerve distribution and possible vasotrophic changes.

Group III. Group III CTS patients have the most severe symptoms. They usually have longstanding complaints of painful dysesthesias and impaired dexterity with associated functional disability. Examination reveals severe sensory loss, including loss of two-point discrimination, and skin atrophy. They have marked thenar muscle wasting with

significant weakness of thumb abduction and opposition. Prognosis is considered very poor in patients who present with these symptoms, regardless of therapeutic intervention.

Electrodiagnosis. Simpson[481] is credited as being the first investigator to demonstrate focal slowing of median nerve conduction across the wrist. In doing so, he provided the first neurophysiologic evidence of entrapment neuropathies. Nerve conduction across the wrist is a highly sensitive means of confirming the clinical diagnosis in most patients. In some cases, conduction studies across the wrist detect abnormalities in asymptomatic patients or in patients who have CTS but present with confusing symptoms, such as isolated shoulder or neck pain.

Median sensory nerve conduction across the wrist is a sensitive electrophysiologic means of diagnosing CTS.[422,482] The orthodromic stimulation technique involves use of ring electrodes, typically on the index or middle fingers, for stimulation. The active and reference surface electrodes are placed just proximal to the wrist crease with 14 cm conventionally used as the distance between stimulating and pickup electrodes in an adult. The antidromic technique involves stimulation proximal to the wrist crease with pickup of the sensory potential with ring electrodes on the index or middle fingers. The distance between the site of stimulation and the active electrode is again, conventionally, 14 cm. Although the latter technique characteristically results in larger sensory amplitudes, it also has the potential disadvantage of concomitant stimulation of a muscle action potential at the wrist, which may distort the sensory response.

More sensitive nerve conduction tests to detect CTS do exist. One particularly sensitive method is to compare the median vs ulnar sensory latencies to the ring finger in the same hand.[483]

The technique touted as being the most sensitive is comparison of the median and ulnar latencies with palmar stimulation. Palmar stimulation of the median and ulnar nerves generates a mixed nerve action potential for each nerve. These potentials are recorded at the wrist for comparison.[484] One other sensitive test is comparison of the median sensory latency across the wrist to the thumb with the radial sensory latency to the same digit.[485,486] Kimura[487] has performed serial stimulation from midpalm to distal forearm at 1-cm increments, known as the "inching" technique. Normally, median sensory axons show a predictable latency change of 0.16 to 0.20 ms/cm from one stimulation point to another. A localized latency increase across a 1-cm segment significantly greater than the other segments is suggestive of a focal median neuropathy.[160] Kimura[487] noted that the increase in latency in CTS occurred most often between 2 to 4 cm distal to the proximal border of the transverse carpal ligament. Identification of focal pathology rather than more diffuse involvement of the median nerve helps distinguish CTS from a distal neuropathy involving digital nerves, such as sometimes found in diabetics.

A reduction in amplitude of the SNAP may provide evidence to support the presence of CTS. Although not as sensitive as delayed conduction and of no localized value, reduced amplitude or absence of the SNAP does frequently occur in CTS. The range of normal amplitudes for the median SNAP is large, however, so it is advocated that the ratio of the median SNAP amplitude to that of the ulnar SNAP amplitude be obtained instead.[422]

The incidence of prolonged median motor distal latency is high in CTS, but it is not as sensitive as median sensory conduction across the wrist.[482,488] Segmental stimulation of median motor axons across the wrist can be done; however, this technique is technically more challenging than segmental stimulation of median sensory axons described above because the recurrent course of the thenar nerve anatomically varies among subjects.[489] A decrease in the amplitude of the median motor action potential does occur in CTS, but is not a sensitive indicator. Temporal dispersion of the action potential is also seen in some cases. Slowing of median motor nerve conduction in the forearm segment does not necessarily exclude CTS. It can be observed in more severe cases of CTS.[490]

The median NCS results on the clinically affected side may be compared with corresponding studies on the opposite side; however, because of the high incidence of bilateral involvement, this technique may have limited usefulness in improving the sensitivity of conduction studies in CTS.

Residual latency refers to the calculated time difference between the measured distal latency of a motor nerve and the expected distal latency. It is calculated by dividing the distance between the stimulus cathode and the active recording electrode by the maximum conduction velocity measured in a more proximal segment of the nerve. The residual latency is derived from the combination of the neuromuscular transmission time and the slowing of conduction in the terminal axons due to decreasing diameter and unmyelinated segments. The residual latency is increased in patients with CTS.[491]

Electromyography may be useful in identifying electrical evidence of motor axonal degeneration, but it is less helpful than the NCSs for diagnosing

CTS. More advanced cases of CTS may show increased insertional and spontaneous activity (fibrillations and positive sharp waves) in median innervated hand intrinsic muscles consistent with muscle membrane instability, along with a decreased recruitment pattern of motor unit action potentials, suggestive of denervation activity.

Current practice recommendations by the AAEM for electrodiagnosis of CTS suggest that sensory conductions of the median nerve across the wrist reflect a high degree of clinical certainty and should be standard practice. If the median sensory conduction is abnormal, one other sensory nerve in the symptomatic limb should be tested for comparison. If the median sensory conduction distance is greater than 8 cm and the latency results fall in the normal range, additional studies are necessary. A median conduction across the wrist of a distance less than 8 cm or comparison of the median conduction with radial or ulnar conduction in the same limb should be standard practice. Motor conduction studies of the median nerve, recording over the thenar eminence with comparison to one other motor nerve in the symptomatic limb is also recommended. It is felt however, that the motor conduction study reflects only moderate clinical certainty. An EMG of median innervated muscles of the thenar eminence is felt to be of unclear clinical utility and is left as an option at the electromyographer's discretion.[492]

Treatment. Conservative management of CTS is advocated for patients with mild or intermittent symptoms. Relative rest from the potentially precipitating or exacerbating activity, volar splinting of the wrist in neutral, and NSAIDs are often the initial measures used and are frequently helpful (Figure 9-65). Additional nonsurgical forms of treatment include local steroid injection of the wrist and a short course of oral steroids or a trial of diuretics, or both. Although pyridoxine at 100 to 200 mg per day has been prescribed, its actual benefits are inconclusive.[493,494] Doses as low as 300 mg/d have been shown to cause a progressive generalized sensory neuropathy.[495]

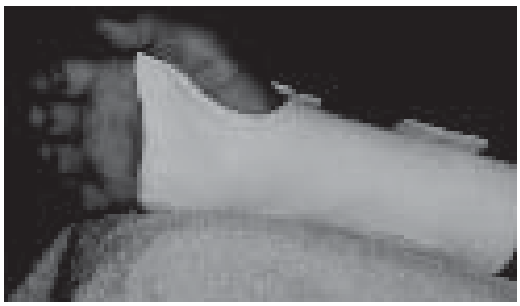

Fig. 9-65. Volar wrist hand orthosis (resting hand splint) may be very effective in the treatment of mild to moderate carpal tunnel syndrome.

Local steroid injections are used for patients with persistent but mild or intermittent symptoms, as well as for patients with painful symptoms who are considered poor surgical risks. Steroid injections are most successful in patients with minimal or intermittent symptoms and least successful in those with persistent numbness and thenar atrophy. Thus, injections are generally contraindicated in patients with significant or persistent sensory loss and thenar atrophy and weakness.[422] Green[496] found that a positive response to steroid injection is an excellent predictor of a successful surgical response. An acceptable method is to mix 1 to 2 mL of 1% lidocaine with 20 mg of methylprednisolone and inject this preparation proximal to the wrist crease and ulnar to the palmaris longus tendon. Care is taken to avoid direct injection into the median nerve or the transverse carpal ligament. Partial or complete relief usually occurs within 3 days. If no relief or exacerbation of symptoms occur, do not reinject. If partial relief occurs, consider a repeat injection. Do not inject more than four times due to the increased potential for tendon rupture and direct median nerve injury.[422]

When performed correctly, surgical management of CTS is generally highly successful. Absolute indications include failure of conservative management in a patient who is not a poor surgical risk or who manifests clinical evidence of thenar atrophy and weakness, or both. Persistent, longstanding sensory loss is considered a relative indication for surgical intervention.[422]

Postoperatively, a bulky hand dressing and wrist splint are applied. Most patients are permitted to begin to incompletely flex their digits within 48 hours. Finger flexion is gently progressed over the next several days. At 4 to 5 days, many patients are able to do light functional activities, but the wrist splint is maintained for up to 2 weeks, at which time the sutures may be removed. Strenuous activity is deferred for 2 to 3 months, but light clerical work can be progressively instituted after 2 weeks.

Acute CTS occurring in association with a fracture or fracture-dislocation, such as Colles' fracture, requires immediate closed reduction after documenting the median nerve deficit. If the symptoms of CTS persist or worsen after the reduction, immediate carpal tunnel release (CTR) is indicated. If symptoms of CTS occur after comminuted or un-

stable forearm or wrist fractures and persist greater than 24 to 48 hours despite operative management of the fractures, CTR is indicated. In the case of Volkmann's ischemic contracture secondary to a crush injury, CTR is done in combination with a more proximal fasciotomy.[422]

Ulnar Nerve Injuries

The ulnar nerve is derived from the medial cord of the brachial plexus, which formed from contributions from the C-8 and T-1 nerve roots. It carries both motor and sensory fibers. It supplies the flexor carpi ulnaris, the medial portion of the flexor digitorum profundus in the forearm, and the majority of the intrinsic muscles of the hand. The hand intrinsic muscles innervated include the following: first dorsal interosseous, adductor pollicis, deep head of the flexor pollicis brevis, flexor digiti minimi, abductor digiti minimi, opponens digiti minimi, volar interossei, dorsal interossei, and ulnar lumbricals (Figure 9-66). Sensory innervation includes the following: an articular branch to the elbow; the palmar cutaneous branch, which serves the palmar aspect of the hypothenar region; the dorsal cutaneous branch, which innervates the medial dorsum of the hand and the medial 1½ or 2½ digits; and the superficial branch, which serves the volar aspect of the medial 1½ digits.

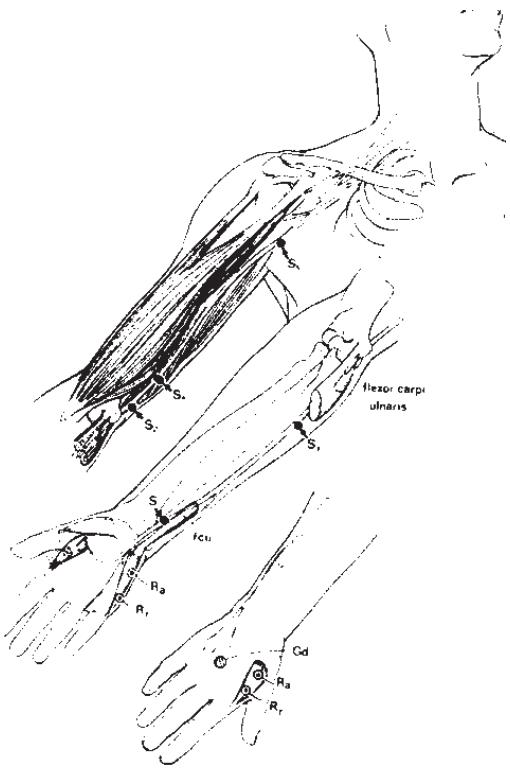

Fig. 9-66. The ulnar nerve with electrode placement for motor nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 152.

In the proximal upper arm, the ulnar nerve lies medial to the brachial artery. It remains in this relationship until the mid portion of the upper arm. At that point, it pierces the medial fascial septum and courses posterior to this septum in the posterior compartment. At the elbow, the ulnar nerve passes posterior to the medial epicondyle. It subsequently courses between the two heads of the flexor carpi ulnar, which constitutes the "cubital tunnel." It passes to the medial volar forearm area where it lies between the flexor carpi ulnaris and the flexor digitorum profundus. It lies medial to the ulnar artery in the distal forearm. In the distal forearm, the ulnar nerve is relatively superficial and courses between the flexor carpi ulnaris and flexor digitorum profundus tendons.

At the level of the wrist, the ulnar nerve passes through Guyon's canal, which is formed by connective tissue bounding the pisiform and the hook of the hamate. After passing through the canal, the ulnar divides into a superficial and a deep branch. The superficial branch supplies the palmaris brevis muscle and the skin of the hypothenar eminence and medial 1½ digits. The deep branch supplies the aforementioned intrinsic muscles of the hand.

Ulnar Nerve Injury at the Elbow

Ulnar nerve injuries and entrapment at the elbow are relatively common. Cubital tunnel syndrome and tardy ulnar nerve palsy are two terms used to describe the clinical presentation associated with ulnar nerve injury at the elbow. In order to clarify etiology, the term *cubital tunnel syndrome* is best restricted to those cases where the ulnar nerve is compressed by the aponeurosis of the flexor carpi ulnaris. *Tardy ulnar palsy*, on the other hand, is associated with antecedent trauma, precipitating joint deformity or recurrent nerve subluxation. Many clinicians, however, continue to use the term tardy ulnar palsy to describe any ulnar entrapment neuropathy at the elbow.

Anatomic considerations. The cubital tunnel at the elbow is formed by the following structures: its sides are formed by the two heads of the flexor carpi ulnaris, its floor by the medial ligaments of the elbow joint; the aponeurotic arch, which bridges the two heads of the flexor carpi ulnaris, forms the roof

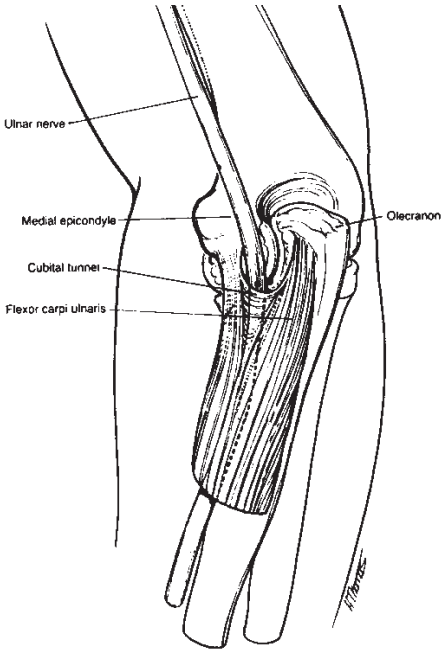

Fig. 9-67. The ulnar nerve passing through the cubital tunnel. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 30.

(Figure 9-67).[497] The cubital tunnel is a frequent site of entrapment.[498,499]

Clinical presentation. Patients with an ulnar nerve lesion at the elbow primarily present with complaints of intermittent hypesthesia in the ulnar distribution. Their symptoms are often exacerbated by sustained elbow flexion, particularly if also resting on the elbow, or by activities requiring repetitive flexion and extension of the elbow. Symptoms are generally intermittent at first. As symptoms progress, patients may experience nocturnal exacerbations with disturbance of sleep. They may awaken with elbow pain along with radiation of paresthesias from the elbow down the medial aspect of the hand to the little finger.

As noted above, sensory complaints are the initial symptoms. Impaired light touch and two-point discrimination, as opposed to pinprick and thermal sensations, are most often affected. The sensory deficit may involve the ulnar aspect of the palm, volar surface of the little finger and ulnar half of the ring finger, the medial dorsum of the hand, and the dorsum of the little and ring fingers. Note, however, that the ulnar nerve may supply the entire ring and ulnar half of the middle finger in about 20% of cases.[422] Most often, the sensory deficits and symptoms involve the superficial terminal branches to the palmar aspect of the little finger and medial half of the ring finger, along with the dorsum of these fingertips. In longstanding ulnar nerve entrapments, trophic changes may occur at the fingertips of the little or ring fingers. The medial cutaneous nerve branch of the brachial plexus serves the medial forearm up to a point proximal to the skin crease at the wrist. It is not involved in ulnar nerve lesions at the elbow.

Although most patients start with sensory complaints, an occasional patient will develop symptoms of weakness and impaired dexterity as their initial problem. In the majority of cases, progressive weakness of hand grip and pinch strength, as well as decreasing dexterity evolve slowly over time. With chronic compression, symptoms may be slowly progressive over months prior to developing objective motor deficits. With acute, traumatic compression injuries, sensory deficits and motor weakness may be immediate or variably delayed.

A positive Tinel's sign may be elicited at the elbow, typically with radiation down the medial aspect of the forearm. This, however, is a nonspecific sign that can be elicited in a number of patients without documented ulnar neuropathies. A more specific test for ulnar neuropathy is the elbow flexion test. In this test, elbow flexion reproducibly precipitates or exacerbates elbow pain or painful dysesthesias or both in the distribution of the ulnar nerve. The Tinel's sign and elbow flexion test are performed bilaterally for comparison.

Point tenderness, nerve subluxation, or nerve thickening may be palpable at the elbow. If the tenderness is detected several centimeters proximal to the medial epicondyle, it may be due to compression of the ulnar nerve by the arcade of Struthers or within the medial intermuscular septum. Tenderness elicited immediately posterior to the medial epicondyle is typically secondary to trauma. Tenderness reproduced 2 to 3 cm distal to the medial epicondyle in the region of the flexor carpi ulnaris aponeurosis is suggestive of cubital tunnel syndrome. If the tenderness is obtained even more distal, that is, greater than 3 cm distal to the medial epicondyle, suspect ulnar compression by the deep aponeurosis of the flexor carpi ulnaris.[422]

Subtle weakness may manifest as lateral instability of the index finger and increasing abduction and progressive weakness of the little finger. Weakness of ulnar innervated hand intrinsic muscles is also reflected by flexion contracture of the proximal IP joint of the little finger or ring finger, or both, along with hyperextension of the MCP joints of these digits.

Ulnar nerve lesions at the elbow may significantly affect a patient's fine motor function. The pinch strength between the thumb and fingers may be reduced due to weakness of the adductor pollicis, ulnar innervated portion of the flexor pollicis brevis, and first dorsal interosseous. The adductor pollicis and flexor pollicis brevis muscles normally stabilize the thumb MCP joint. A positive Froment's sign occurs when a patient uses the action of the flexor pollicis longus muscle (active flexion at the thumb IP joint), to compensate for weakness of the first dorsal interosseous or adductor pollicis muscle or both when attempting to perform a lateral pinch maneuver.

In the presence of ulnar neuropathy at the elbow, there is weakness of the flexor digitorum profundus muscles of the little finger and ring finger, as well as weakness of the ulnar innervated hand intrinsics. This results in both decreased hand grip power and impaired coordination in fine motor tasks, especially those requiring precision. Power grasp is approximately 100 to 140 lbs of pressure in an adult male and 50 to 60 lbs in an adult female. The dominant hand normally generates 15% to 20% more pressure than the nondominant limb.[422] A patient's power grasp strength may drop to as low as 20% to 25% of normal in the presence of a severe ulnar nerve lesion.[422]

One reason why some patients lack coordination when performing fine motor tasks is that weakness of the interosseous muscles compels them to initiate digit flexion at the MCP joints with the long finger flexors rather than the interossei. As the interosseous weakness progresses, a patient may develop variable degrees of a claw hand deformity involving the ring and little fingers.

Etiology. Ulnar nerve lesions at the elbow may occur as the result of sustained external pressure, direct trauma, chronic subluxation, or bony or scar impingement.

As previously mentioned, cubital tunnel syndrome refers to entrapment of the ulnar nerve by the aponeurosis of the flexor carpi ulnaris. This syndrome should be entertained when ulnar sensory or motor deficits or both develop without a readily identifiable cause. The cubital tunnel narrows and the medial collateral ligament bulges medially with elbow flexion. This accounts for why repetitive or sustained elbow flexion is implicated as a precipitating or exacerbating factor in some patients with symptoms of ulnar nerve compression.

Classic tardy ulnar nerve may occur when there is chronic stretch of the nerve secondary to a cubitus valgus deformity. This elbow malalignment often occurs after a capitular fracture with arrest of lateral humeral epiphyseal growth. A cubitus valgus deformity and tardy ulnar nerve palsy may also occur following a supracondylar fracture.[422] A compressive lesion at the elbow may affect different fascicles of the ulnar nerve. The most commonly affected are the fascicles carrying the fibers to the terminal digital nerves and intrinsic hand muscles rather than to the forearm muscles.[500] Fibers destined for the flexor carpi ulnaris are more lateral and posterior than the fibers to more distal muscles and are, therefore, more protected against trauma or compression.[422]

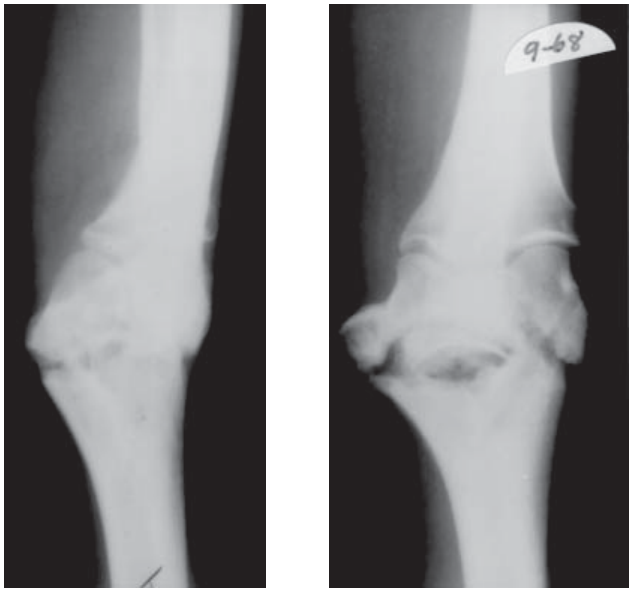

Fig. 9-68. Comminuted intracondylar distal humeral fracture, which may be associated with ulnar nerve injury. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

External pressure from trauma (Figure 9-68) or bony or soft tissue compression at the elbow, can be either acute or chronic and may be recurrent or sustained. A host of etiologies have been identified as a cause of ulnar neuropathy at the elbow[411,422,501–511]:

- Recurrent trauma
- Direct, blunt trauma, including elbow fracture and/or dislocation
- Recurrent subluxations
- Cubitus valgus deformity
- Ganglia
- Gouty tophus
- Accessory or aberrant anconeus muscle

- Synovial cyst
- Callus formation secondary to elbow fracture
- Posttraumatic calcification
- Scar formation post dislocation or other trauma
- Anomalous vessels, fibrous bands, or tumors at the elbow

Ulnar neuropathy at the elbow has been associated with rheumatoid arthritis.[512] It has also been documented in bedridden patients.[422] Chan et al[513] discovered that ulnar neuropathy tended to occur most often on the nondominant side in laborers, presumably because of their tendency to rest on their nondominant elbows for support while performing with the dominant arm.

Differential diagnosis of ulnar neuropathy at the elbow includes exclusion of an ulnar nerve lesion at the wrist. In the latter case, normal or only mildly slowed nerve conduction velocity would be present across the elbow or in the forearm segment, while slowing across the wrist would be present. Also, the dorsal cutaneous NCS would be normal with wrist lesions in contrast to the abnormal ulnar sensory nerve conduction across the wrist. In more severe ulnar injury at the wrist, EMG abnormalities would be present in ulnar innervated hand intrinsics while sparing the ulnar innervated forearm muscles. TOS, C-8/T-1 radiculopathy, high (more proximal) ulnar nerve lesion, medial cord lesion (eg, secondary to a superior sulcus tumor), syringomyelia, and early amyotrophic lateral sclerosis may also require exclusion.

Electrodiagnosis. Simpson[481] has been credited with being the first to demonstrate that slowed conduction across the elbow segment is helpful in localizing an ulnar nerve lesion at the elbow. Slowing of the ulnar motor nerve conduction velocity across the elbow segment is the most specific diagnostic criterion for identifying an ulnar compression neuropathy at the elbow.[498,503,514,515] Although it may be helpful to compare the conduction velocity across the elbow segment with that obtained in the above- and below-elbow segments, the absolute conduction velocity is felt to be a more sensitive indicator of ulnar conduction abnormality than the relative velocity.[503,515] The degree of slowing, however, does not necessarily correlate with severity.[422]

Focal demyelination injury of the ulnar nerve at the elbow will cause temporal dispersion of the CMAP when stimulation occurs proximal to the lesion. A decrease in amplitude may also be seen. A decrease in amplitude of greater than 10% is felt to be abnormal.[515] In a study by Pickett and Coleman,[516] a decrease in amplitude of greater than 25%, with stimulation proximal to the elbow compared to distal to the elbow, was the best criterion for confirming an ulnar nerve conduction deficit across the elbow.

Whenever assessing amplitude changes with median and ulnar NCSs, it is important not to overlook the possible presence of a Martin-Gruber anastomosis, seen in up to 20% of the population (Figure 9-69). In this anomaly, some nerve fibers from the median nerve cross over to the ulnar nerve in the forearm and course with the ulnar nerve fibers through Guyon's canal. This process will result in a larger ulnar CMAP amplitude with stimulation at the wrist rather than at the elbow, which, in turn, may cause an erroneous assumption that there has been an amplitude drop due to a partial conduction block at the elbow. Assuming that there is not

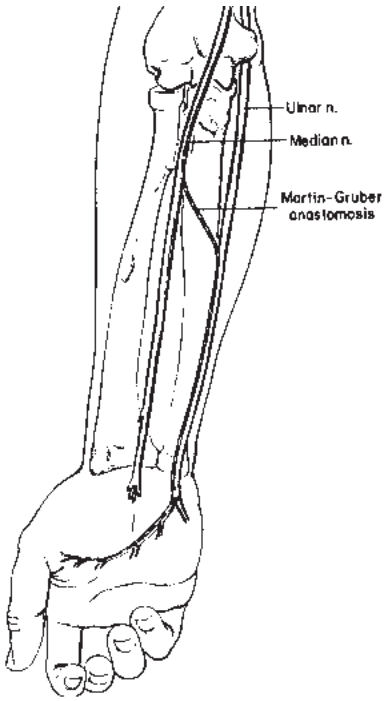

Fig. 9-69. Martin-Gruber anastomosis. Fascicles from the median nerve cross over in the forearm and run with the ulnar nerve. Stimulation of the median nerve at the wrist will generate a compound motor unit action potential (CMUAP) of lower amplitude than stimulation at the elbow. Stimulation of the ulnar nerve at the wrist will evoke a larger amplitude than stimulation around the elbow. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 27.

a concomitant ulnar neuropathy actually present at the elbow, a similar small CMAP amplitude would be obtained with stimulation below and above the elbow in the presence of a Martin-Gruber anastomosis.

When performing ulnar NCSs across the elbow, it is important to maintain a consistent elbow position as the length of the nerve changes with movement of the arm into different positions. While different angular elbow positions have been advocated, Bielawski and Hallet[517] observed no major difference in the ability to diagnose a lesion at the elbow between flexed or extended positions. They did, however, stress that the same technique should be used consistently by physicians in the same electrodiagnostic laboratory. It is also crucial to avoid too short an interstimulus distance in order to prevent inaccurate conduction velocity calculations. Some argue that the cathode for below-elbow stimulation should be at least 4 cm distal to the medial epicondyle and the above-elbow stimulus site at least 10 cm proximal to the below-elbow site.[422] Adherence to this technique helps to ensure that the focal site of compression is included in the across-the-elbow segment and that a sufficiently long segment is assessed. It is particularly important if cubital tunnel syndrome is suspected, as the site of nerve constriction is felt to be 1.5 to 3.5 cm distal to the medial epicondyle.[499] Surface pickup over the abductor digiti minimi muscle is the most common technique for ulnar motor studies. However, it may be preferable to place the surface pickup electrode over the first dorsal interosseous muscle instead, because motor fibers to this muscle are more likely to be involved in an ulnar nerve lesion at the elbow, and thus more likely to demonstrate conduction slowing or block.[500]

A decrease in the ulnar SNAP amplitude obtained distally across the wrist confirms the presence of an ulnar nerve lesion and correlates with severity, but, as an isolated finding, is of no localizing value.[498,503] If the ulnar dorsal cutaneous SNAP is unobtainable or its amplitude reduced, particularly if its latency is within normal limits, it is supportive of a more proximal ulnar nerve lesion. However, it cannot be used to definitely localize the site to the elbow area. Because the amplitude of a SNAP reflects the number of functioning nerve fibers, a decreased SNAP amplitude indicates more severe injury and possibly a poorer prognosis.[422,500]

Recently, a short segment incremental study of the ulnar nerve across the elbow has been shown to be a sensitive tool in further localizing areas of entrapment[518] (Figure 9-70). Stimulation at 1-cm in-

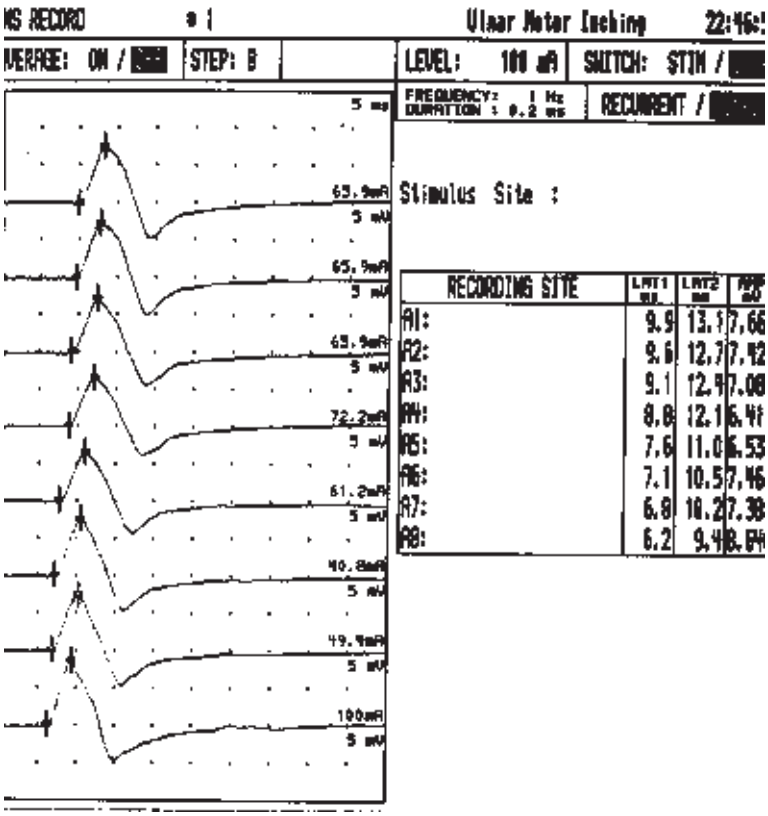

Fig 9-70. Short segment incremental study of the ulnar nerve across the elbow. A 1.2-ms latency delay was observed 2 cm distal to the medial epicondyle. The finding suggested compression at the cubital tunnel. Tracing courtesy of CPT Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

crements along the path of the ulnar nerve, extending above and below the elbow, enabled the differentiation of entrapment at the retroepicondylar groove from entrapment at the humeroulnar aponeurotic arcade (cubital tunnel). A latency change over a 1-cm segment of greater than 0.4 ms was found to be significant. Latency delays at the medial epicondyle reflected entrapment at the retroepicondylar groove. Slowing 1 to 3 cm distal to the medial epicondyle was consistent with compression in the cubital tunnel. Latency prolongation at a focus more distal than 3 cm was felt to be secondary to entrapment at the exit from beneath the flexor carpi ulnaris.[518]

Electromyography is not as sensitive as NCSs in identifying and localizing an ulnar nerve lesion at the elbow. However, it can be a useful adjunct to these studies, particularly if EMG abnormalities suggestive of denervation activity are found in both the ulnar innervated forearm and hand muscles. Electromyography may also be helpful in excluding a C-8/T-1 radiculopathy.

Treatment. Conservative management is appropriate for patients with mild symptoms. This includes not resting on the elbow (especially when flexed), as well as avoidance of sustained or extreme elbow flexion and activities requiring repetitive flexion and extension of the elbow. Some advocate use of an elbow splint, such as orthoplast splints or long-arm, bivalved casts to maintain the elbow in extension or partial flexion, but tolerance and compliance may be a problem. A well-cushioned elbow pad is well tolerated and minimizes the opportunity for inadvertent trauma to the elbow. A lumbrical bar orthotic is prescribed for selected patients showing signs of developing a claw-hand deformity or who already demonstrate this deformity but have not yet developed fixed contractures. This particular orthotic is helpful in preventing progression of the deformity, particularly if used in combination with daily range-of-motion exercises of the involved digits.

Various surgical procedures are available to treat ulnar neuropathy at the elbow. These include release of the flexor carpi ulnaris aponeurosis, medial epicondylectomy, and anterior transplantation of the nerve to a subcutaneous position or under the flexor and pronator muscles.[422] Surgical management is indicated for patients with persistent or progressive signs and symptoms of ulnar neuropathy, particularly progressive weakness, despite a trial of conservative management.

In the event cubital tunnel syndrome is identified, a cubital tunnel release may be sufficient to relieve symptoms. Cubital tunnel release involves the release of the flexor carpi ulnaris aponeurosis. When indicated, it has the advantage of being a relatively simple procedure with low morbidity. It can be done under local anesthesia and does not require postoperative splinting. It is not, however, recommended for patients with osseous or joint pathology at the elbow or for those with congenital subluxation of the nerve.[422]

For patients who fail to receive satisfactory results from a simple release, a medial epicondylectomy or anterior transplantation may be warranted.[519] Excision of the medial epicondyle removes a potential or actual cause of nerve compression, requiring less dissection with less chance of injuring nerve branches than the anterior transplantation procedure. The disadvantage is that the ulnar nerve is still in a subcutaneous position and, therefore, remains vulnerable to trauma.

Postoperatively, the elbow is protected with a posterior splint for two weeks, at which time gently progressive range-of-motion exercises are instituted. Results have generally been good with this procedure.[422,520–522]

Anterior transplantation is designed to remove the ulnar nerve from compressive forces in the cubital tunnel. The nerve is placed in a less vulnerable, anterior position. This position also decreases the tension on the nerve during elbow flexion and places the nerve in a well-vascularized intermuscular bed. Anterior transplantation is, however, a more challenging procedure than either the simple release or medial epicondylectomy. Since it involves use of a large skin flap, there is a risk of injury to the posterior branch of the medial antebrachial cutaneous nerve.

Except in those rare cases where isolated cubital tunnel syndrome is identified and simple cubital tunnel release is sufficient, Dawson and colleagues[422] prefer medial epicondylectomy to either the subcutaneous or submuscular anterior transposition procedure. It is specifically preferred because of its relatively good success rate and because it requires less dissection and devascularization of the nerve.

Ulnar Neuropathy at the Wrist

Anatomic considerations. At the level of the wrist, the ulnar nerve passes through a closed space referred to as Guyon's canal (Figure 9-71). Within the canal, the nerve passes over the transverse carpal ligament. The volar carpal ligament roofs the nerve at this point. The pisiform and the hook of

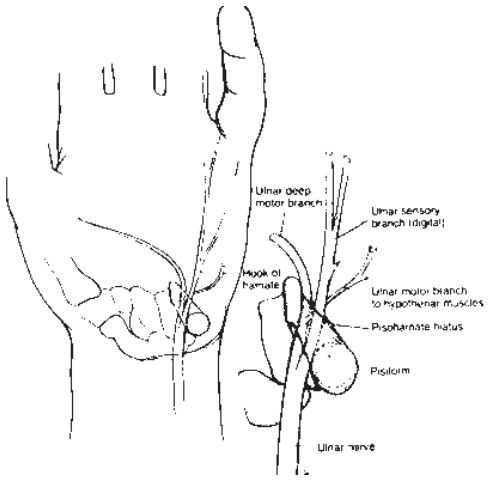

Fig. 9-71. Guyon's canal and its relationship to the distal branches of the ulnar nerve. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 31.

the hamate form its bony margins. The ulnar artery and vein also pass through Guyon's canal. The ulnar nerve branches into its terminal superficial and deep branches within the canal.

The superficial terminal branch of the ulnar nerve supplies the palmaris brevis, innervates the skin on the distal medial aspect of the palm, and gives off two palmar digital nerves. In the majority of cases, the ulnar nerve provides sensory innervation to the little finger and the medial half of the ring finger. In a minority, sensation is provided to the little and ring fingers and the medial half of the middle finger. The deep terminal branch supplies the hypothenar muscles (abductor digiti minimi, flexor digiti minimi brevis, and opponens digiti minimi) and then dives into the palm under the flexor tendons to supply the palmar and dorsal interossei, the third and fourth lumbricals, a portion of the flexor pollicis brevis, and finally the adductor pollicis.

The palmar cutaneous branch of the ulnar nerve arises at 7 cm proximal to the wrist, descends near the ulnar artery, pierces the deep fascia, and supplies the skin over the hypothenar eminence. The dorsal cutaneous ulnar nerve branch arises 5 to 10 cm above the wrist, passes posteriorly deep to the tendon of the flexor carpi ulnaris, pierces the deep fascia, and supplies the skin on the medial dorsum of the hand, as well as the dorsum of the little and ring fingers and the medial dorsum of the middle finger (Figure 9-72).

The most common site of ulnar nerve compression at the wrist is within Guyon's canal.[422,523] The nerve is also vulnerable to compression just proximal or distal to the canal. Rarely, an isolated compression injury of the dorsal ulnar cutaneous nerve may occur proximal to the wrist.

Clinical presentation. The distribution of sensory impairment and weakness is dependent on the level of ulnar nerve compression injury. If there is total ulnar sensory loss involving both the dorsal and palmar surfaces of the medial aspect of the hand, as well as the fourth and fifth digits, along with weakness of all ulnar innervated hand intrinsics, then a lesion proximal to the wrist is suspected.

A lesion within Guyon's canal will spare the sensory distribution of the palmar cutaneous branch and the dorsal ulnar cutaneous branch. If a lesion occurs proximal in the canal, however, it may cause sensory impairment in the distal medial aspect of the palm and the volar surfaces and tips of the fourth and fifth digits. It may also cause weakness of the hypothenar muscles, as well as the interossei, third and fourth lumbricals, flexor pollicis brevis (deep head), and adductor pollicis.

Compression injury isolated to sensory fibers within Guyon's canal can occur. In this case, sensation is preserved in the distribution of the dorsal ulnar cutaneous and palmar cutaneous branches, but sensory loss typically occurs on the volar aspect of the little finger and medial half of the ring finger. With a lesion more distal in the canal, at or distal to the hook of the hamate, innervation to

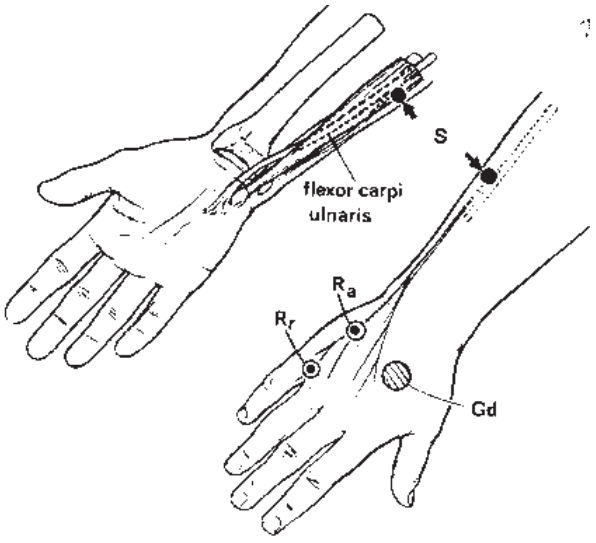

Fig. 9-72. The dorsal cutaneous branch of the ulnar nerve with electrode placement for sensory nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 178.

hypothenar muscles is preserved, but other ulnar-innervated hand intrinsic muscles will be weak.

A lesion of the deep palmar branch of the ulnar nerve, distal to Guyon's canal, will spare the hypothenar muscles and all ulnar sensory fibers. However, it will cause weakness of the remaining ulnar-innervated hand intrinsics, including the fourth and fifth lumbricals, interossei, flexor pollicis brevis (deep head), and adductor pollicis muscles.

Based on the aforementioned potential sites of ulnar nerve compression at or near the wrist, patients may have mixed motor and sensory deficits, isolated sensory loss, or pure motor deficits. Wrist pain with radiation proximal or distal may occur. There may be nocturnal exacerbations and cold intolerance. Clinical examination may reveal local swelling, induration, and a palpable mass, or a positive Tinel's sign or both.

Etiology. A multitude of causes of ulnar nerve compression at or distal to the wrist have been reported. Extrinsic pressure in the form of repetitive trauma, such as experienced in some occupations or sports activities, or chronic compression secondary to masses, have been documented to cause ulnar neuropathies of the wrist and hand.

Hunt,[524] in 1908, was the first to identify occupational trauma as the cause of an ulnar compression neuropathy, specifically a neuritis of the deep palmar branch of the ulnar nerve. Chronic compression neuropathy of the ulnar nerve may occur in pipe cutters, metal polishers, mechanics, and professional cyclists.[422] In the cyclists, ulnar neuropathy has been attributable to chronic pressure from leaning on the handlebars.[525,526] A variety of causes of ulnar nerve compression at or distal to the wrist have been reported[527–542]:

- Occupational trauma
- Bicycling (handlebar palsy)
- Edema
- Wrist fracture
- Ganglion
- Calcium deposits
- Tumor
- Fibrous band
- True aneurysms
- False aneurysms
- Rheumatoid synovial cyst

An alternate list of causes has been documented by Shea and McClain[543] in order of highest to lowest occurrence among 137 patients:

- Ganglia
- Occupational Neuritis
- Laceration
- Ulnar artery disease
- Arteritis
- Thromboangiitis
- Fracture of carpal bones
- Soft tissue contractures
- Fracture of metacarpal bones
- Fracture of radius
- Aberrant muscles
- Neurolemoma
- Anomalous relationship of nerve to Guyon's canal
- Pisiform bursitis
- Carpal osteoarthritis
- Accessory ossicle
- Dislocation of distal ulna
- Lipoma
- Hemophiliac cyst
- Dislocation of pisiform

Differential diagnosis of ulnar neuropathy at the wrist includes C-8, T-1 radiculopathy, TOS, amyotrophic lateral sclerosis, ulnar neuropathy at the elbow or in the forearm, CTS, and isolated fractures of the carpal bones. Ulnar neuropathy in the wrist area may be due to fractures of the metacarpals, pisiform, or body or hook of the hamate. Thus, these fractures should be excluded when a patient has a history of trauma to the wrist, point tenderness over the affected carpal or metacarpal bones, and ulnar nerve sensory and especially motor deficits. In the event of a fracture of the pisiform or hook of the hamate, special radiologic views, including a carpal tunnel view, lateral tomograms, or oblique views of the hand, will be necessary to identify these fractures as, conventional radiograph views are typically negative.

Electrodiagnosis. The most common ulnar motor NCS performed across the wrist is stimulation at the wrist with surface pickup at the abductor digiti minimi. However, the most common site of ulnar nerve compression in the wrist area involves the deep palmar branch distal to the branch to the abductor digiti minimi. Thus, nerve conduction to the abductor digiti minimi alone is not sufficient to exclude an ulnar motor neuropathy in the wrist region. Motor nerve conduction across the wrist to the first dorsal interosseous, however, would demonstrate a prolonged latency or unobtainable evoked response in the presence of compression of the deep palmar branch. Electromyography, in this case, would be normal in the hypothenar muscles

but show evidence of denervation activity in other ulnar-innervated hand intrinsics.

In more proximal lesions within Guyon's canal, ulnar motor nerve conduction to both the abductor digiti minimi and the first dorsal interosseous would be absent or prolonged and EMG evidence of denervation activity could be detected in all ulnar-innervated hand intrinsics.

If an ulnar nerve lesion is sufficiently proximal within Guyon's canal or proximal to Guyon's canal, then both the deep and superficial terminal branches of the ulnar nerve may be involved. In this case, both ulnar motor and sensory NCSs would be abnormal. Ulnar sensory studies, either orthodromic or antidromic, would reveal a prolonged latency or absent SNAP. If obtainable, the SNAP may also be reduced in amplitude. Ulnar nerve lesions at or distal to the wrist spare the dorsal ulnar cutaneous nerve. Thus, sensory nerve conduction of this nerve may have localizing value.

Isolated compression neuropathy of the ulnar sensory fibers is the least common type of nerve lesion at the wrist.[543] If present, only the ulnar sensory nerve conduction across the wrist is abnormal, manifest by a prolonged latency, reduced SNAP amplitude, or unobtainable response.

The dorsal ulnar cutaneous branch neuropathy is uncommon. When it occurs, it is usually secondary to blunt trauma or lacerations. It is less likely to be injured than the superficial radial nerve since it lies in a more protected position and because it is subject to less iatrogenically induced surgical trauma. In the event this nerve is injured, isolated abnormality of the dorsal ulnar cutaneous NCS will be present, manifest by a prolonged latency, reduced SNAP amplitude, or unobtainable response.

Treatment. Treatment of ulnar compression injuries in the region of the wrist depend on the site, etiology, and duration of the lesion. Conservative management is reserved for mildly symptomatic patients. This includes relative rest in the event of isolated trauma or avoidance of repetitive or chronic compressive trauma. In certain predisposing occupations, relative rest or modifications, such as padded gloves when performing work, may be sufficient to relieve symptoms.

Fig. 9-73. Lumbrical bar hand orthosis compensates for weakness of the ulnar innervated lumbrical muscles and limits metacarpophalangeal hyperextension, placing the fingers in a functional position.

In the event a patient experiences extrinsic trauma that does not respond to conservative management, surgical intervention may be indicated. This may include exploration and decompression or neurolysis, or both. Tumors and ganglia are removed. A fracture of the hook of the hamate is excised in combination with decompression and neurolysis of the nerve. A fracture of the pisiform may also require excision.

If clawing of the lateral two digits occurs secondary to weakness of ulnar-innervated intrinsic muscles, a lumbrical bar orthotic may be helpful in preventing fixed contractures (Figure 9-73). This is most successfully employed when used in conjunction with daily range-of-motion exercises of the affected digits.

NERVE INJURIES AND ENTRAPMENT NEUROPATHIES IN THE LOWER EXTREMITY

Lumbosacral Plexopathy

Anatomic Considerations

The lumbar plexus is produced by the union of the ventral rami of the first three lumbar nerves and the greater part of the fourth. There is a contribution from the subcostal nerve as well. The lumbar plexus lies anterior to the lumbar vertebral transverse processes. It is embedded in the posterior aspect of the psoas major muscle. The lower part of the ventral ramus of the fourth lumbar nerve joins the ventral ramus of the fifth to form the lumbosacral trunk.[473]

The sacral plexus is formed by the lumbosacral trunk and the ventral rami of the first three sacral nerves and the upper part of the fourth sacral ramus. The sacral plexus gives rise to multiple branches prior to the greater sciatic foramen. Its remaining fibers emerge from the greater sciatic foramen as the sciatic nerve (Figure 9-74).[473]

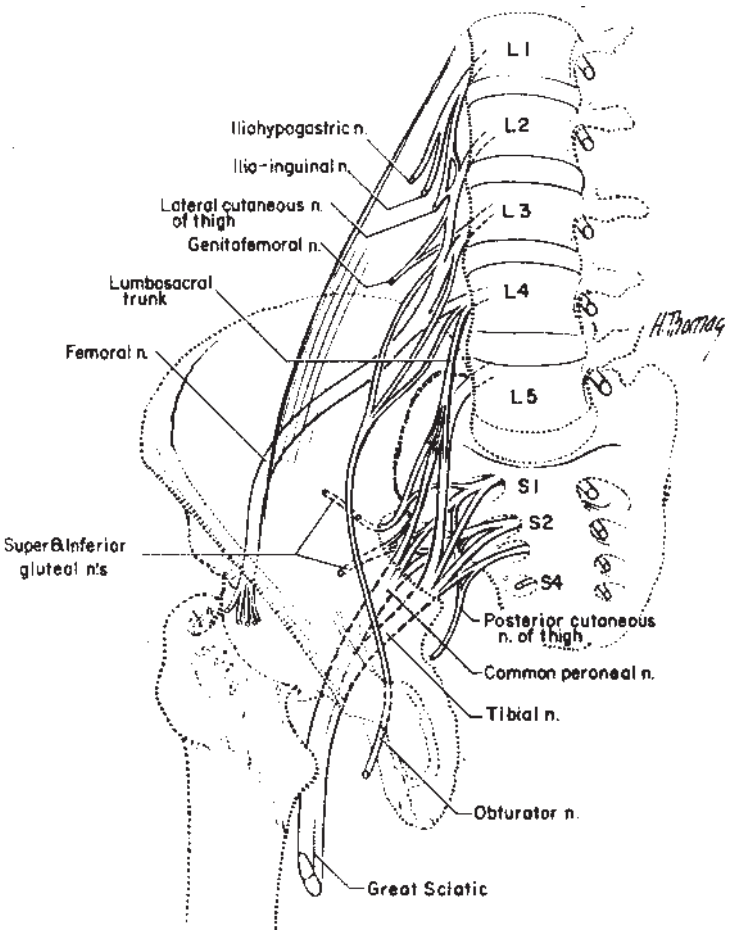

Fig. 9-74. The lumbar and sacral plexuses. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis*. 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 47.

Clinical Presentation

Lumbosacral plexopathies usually occur secondary to trauma. Thus, onset of weakness is typically acute with variable distribution and severity of weakness and sensory deficits depending on the specific portion of the plexus injured. Hyporeflexia or areflexia may also be present in the affected limb at the patella or ankle or both, depending upon site and extent of nerve pathology. Bowel or bladder dysfunction may occur if fibers from the lower sacral nerve roots are injured.

Etiology

Lumbosacral plexopathies are uncommon. Two of the most common causes include pelvic fractures and retroperitoneal hemorrhage. Pelvic fractures usually injure the sciatic nerve with the peroneal component most significantly affected. However, fracture of the pelvic ring or a fracture near the sacroiliac joint may directly damage the plexus or one or more of its branches (Figure 9-75).[411,544,545] Such fractures have been recorded to occur as a complication of cancer or postradiation therapy.[546]

Electrodiagnosis

Electromyography is the most helpful electrodiagnostic test. Findings consistent with denervation activity can be demonstrated by sampling clinically weak muscles of the affected extremity. An EMG of the lumbosacral paraspinals, on the other hand, will be normal unless both nerve roots and plexus are concomitantly involved. If sensory deficits are present in the distribution of the lateral femoral

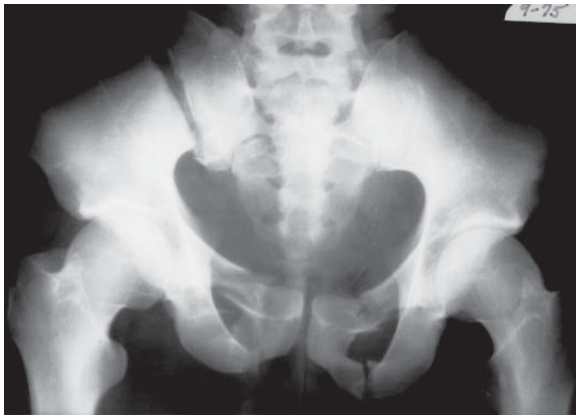

Fig. 9-75. Fractures of the left ileum and right and left ischial arches, associated with traumatic lumbosacral plexopathy. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

cutaneous, saphenous, or sural nerves, sensory NCS of these nerves may be supportive of a plexopathy if they are abnormal, assuming the clinical examination and EMG also suggest a plexopathy. The sensory action potentials of these nerves may be attenuated in amplitude or unobtainable in the presence of a plexopathy affecting their nerve fibers. The soleus H-reflex may also be supportive if prolonged or unobtainable, assuming an S-1 radiculopathy has been excluded clinically or electrodiagnostically.

Sciatic Neuropathy

Anatomic Considerations

The fourth lumbar to the third sacral roots contribute to the formation of the sciatic nerve (Figure 9-76). The nerve lies anterior to the piriformis in the lesser pelvis. In the majority of cases, the nerve passes below the piriformis muscle. In a small percentage, it passes through the piriformis. The nerve then courses laterally beneath the gluteus maximus and passes along the posterior surface of the ischium. It then runs midway between the ischial tuberosity and the greater trochanter and downward over the gemelli, the obturator internus tendon, and the quadratus femoris. The latter muscle separates the sciatic nerve from the hip joint. The nerve then enters the posterior thigh beneath the lower border of the gluteus maximus.

The sciatic nerve courses down the middle of the posterior thigh. At a point just above the apex of the popliteal fossa, it is overlapped by the margins of the biceps femoris and semimembranosus muscles. In the great majority of cases, the sciatic nerve divides into its tibial and peroneal branches near the apex of the popliteal fossa. In a smaller percentage, the nerve divides into these two branches more proximally. In rare cases, the tibial and peroneal branches arise independently from the sacral plexus and course in parallel until they reach the apex of the popliteal fossa where they divide.

The sciatic nerve supplies an articular branch to the hip. It also supplies branches to the semimembranosus, semitendinosus, ischial head of the adductor magnus, and to both heads of the biceps femoris. The branch to the short head of the biceps femoris actually arises from the peroneal portion of the sciatic nerve. All other posterior thigh muscles innervated by the sciatic nerve arise from the tibial division.

In injuries involving the sciatic nerve, the peroneal nerve fibers are more often and more severely

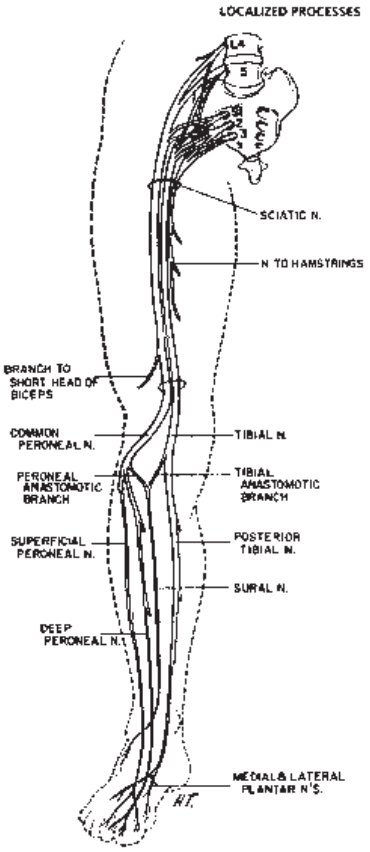

Fig. 9-76. The sciatic nerve with its distal branches. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 49.

affected than the tibial fibers. This is explained by the fact that the peroneal division contains fewer and larger funiculi and has less adipose tissue available to protect the nerve from trauma.[547]

Etiology

Entrapment of the sciatic nerve as it passes through the piriformis or between the piriformis and underlying gemelli, is a well known but clinically rare entity.[548] The piriformis syndrome is presumed to result from compression of the sciatic nerve between the two tendinous heads of the pirformis muscle. Irritation of the nerve may also occur with provocative positioning, especially flexion-adduction-internal rotation.

Other potential causes of sciatic neuropathies include retroperitoneal hemorrhage,[549] fibrous bands spanning the biceps femoris and adductor magnus,[550] and gluteal compartment syndrome.[551] Association with proximal femoral fracture and dislocation has been documented[552] and was observed during the Persian Gulf War (Figure 9-77). Association of sciatic neuropathies with acetabular fractures has been noted to be as high as 13%.[553] Recently, Kaplan and Challenor[554] observed the development of a sciatic neuropathy caused by the development of a posttraumatic osseous tunnel surrounding the nerve. Sciatic nerve palsies occur in association with total hip replacement in less than 2% of patients.[555] Its presence, however, significantly impacts on the rehabilitation and ultimate functional outcome of these patients. In Schmalzried's study, of 36 patients who sustained sciatic nerve injuries (followed for 24 months postsurgery), 64% had persistent mild neurologic deficits and 16% suffered from severe nerve injury.[556]

Fig. 9-77. Comminuted proximal femur fracture following open reduction internal fixation. The patient sustained a concomitant complete sciatic nerve injury.

Clinical Presentation

Proximal sciatic neuropathies may manifest in a number of ways. Pain in the gluteal region, radiating down the posterior aspect of the leg and calf are characteristic. In the piriformis syndrome, symptoms are aggravated by prolonged sitting and with resisted external rotation of the hip. Severe injury to the sciatic nerve will compromise control of the foot and ankle, severely affect knee flexion and hip extension, and variably ablate sensation below the knee, except in the medial malleolar region, which is supplied by the saphenous nerve.

Electrodiagnosis

Stewart's[557] criteria for piriformis syndrome can be generalized to other proximal sciatic neuropathies. Evidence of denervation in muscle supplied by the sciatic nerve including the hamstrings, tibial, and peroneal-innervated muscles may be observed. Muscles in the same myotomal distribution but innervated by the superior gluteal or inferior gluteal nerves are normal. Lumbar paraspinal muscles also show no evidence of denervation. The electromyographic studies are most helpful distinguishing the neuropathy from the much more common L-5 and S-1 radiculopathies. Nerve conduction studies are of lesser utility. Two techniques deserve mention. MacLean[558] developed a method for measuring latencies across the sacral plexus as well as the proximal sciatic nerve. The L-5 and S-1 roots are stimulated just medial and slightly caudal to the posterior superior iliac spine. Recording electrodes are placed on the abductor hallicus. Stimulation at the root level is followed by near nerve stimulation at the sciatic notch. Subtraction of the distal latency

from the proximal latency establishes the latency across the proximal nerve segment. Normal latency obtained by MacLean[558] for the sciatic plexus/sciatic nerve was less than 5.3 ms (mean plus two standard deviations). Side-to-side latency differences should not be greater than 0.9 ms. Fishman and Zybert[559] have developed a provocative test that measures latency shifts in H-reflexes before and during flexion-adduction-internal rotation positioning of the hip.

Femoral Nerve Injury

Anatomic Consideration

The posterior divisions of the second, third, and fourth lumbar roots entwine at the level of the psoas muscle. Piercing the muscle, the femoral nerve courses between the iliacus muscle and the psoas tendon. The nerve then crosses the pelvic brim, beneath the inguinal ligament (Figure 9-78). Before entering the femoral triangle, motor branches to the quadriceps, sartorius, and pectineus separate off. Cutaneous sensory branches arborize across the anterior thigh. The saphenous nerve, the terminal branch of the femoral nerve, dives deep with the femoral vessels and penetrates the adductor canal. The nerve becomes superficial in the popliteal region and extends along the medial aspect of the leg, providing sensation down to the medial malleolus and occasionally to as distal as the great toe (Figure 9-79).

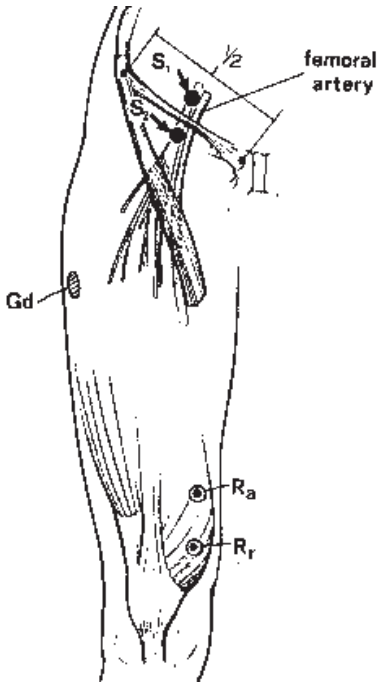

Fig. 9-78. The femoral nerve, passing beneath the inguinal ligament. Electrode placement for motor nerve conduction studies are noted. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 190.

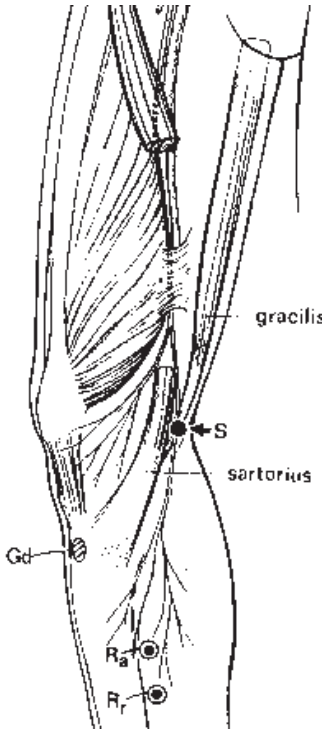

Fig. 9-79. The saphenous nerve, the terminal sensory branch of the femoral nerve, passing through the adductor canal, transmitting sensation from the medial aspect of the leg. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 193.

Etiology

Retroperitoneal hemorrhage, involving the iliopsoas muscle is a common cause of this uncommon injury (Figure 9-80). Associations with hemophilia and anticoagulation are obvious.[560,561] Traumatic injury to the iliopsoas with subsequent femoral nerve injury has been observed following efforts to regain balance during severe hyperextension at the hip.[562,563] Other related traumas have included falls, gunshot wounds, and pelvic fractures.[564] Iatrogenic femoral neuropathies have occurred following pelvic surgery, attributed to the surgery itself as well as to prolonged lithotomy positioning of the pa-

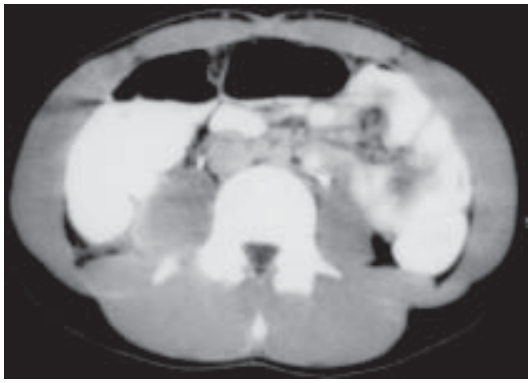

Fig. 9-80. Computed tomography of the pelvis revealing prominent asymmetry of the right psoas vs the left, consistent with psoas hemorrhage. Soft tissue density extending along the right ileum is the residua of retroperitoneal hemorrhage. Femoral nerve entrapment was associated with this traumatic injury.

tient.[564,565] Cases have also been reported of injuries occurring after total hip arthroplasty and femoral vessel catheterization.[566–568]

Clinical Presentation

Weakness of the knee extensors is repeatedly noted as the dominant clinical finding. Weakness of the hip flexors and sensory abnormalities along the anterior thigh and medial aspect of the lower leg are also commonly seen. Severe injuries may reveal a loss of the patellar reflex on the affected side.

Electrodiagnosis

Femoral nerve motor conduction studies have been described by Johnson et al.[569] In the electrodiagnostic laboratory Walter Reed Army Medical Center, recording electrodes are placed over the vastus medialis 32 cm distal to the inguinal ligament. The reference electrode is placed 4 cm distal to the active electrode. Stimulation below the inguinal ligament is 30 cm from the active electrode. Stimulation above the inguinal ligament is 35 cm from the active electrode. The ground electrode is placed between the stimulating and active electrodes. Abnormal latencies are greater than 7.5 ms and 8.4 ms below and above the inguinal ligament, respectively. Proximal, neuropraxic injuries following iliopsoas hematomas may not reveal significant findings on the distal motor-evoked potentials, although compression by the inguinal ligament may show a conduction delay across the segment. Sensory conductions can be obtained along the saphenous nerve. Stimulation occurs at the intersection of the medial gastrocnemius and the tibia. The active electrode is placed 14 cm from the stimulation point, between the medial malleolus and the tibialis anterior tendon. The reference electrode is placed 3 cm distal to the active electrode. Amplitudes less than 0.5 mV and latencies greater than 4.4 ms are greater than two standard deviations from the mean and are considered abnormal.[570] Saphenous nerve somatosensory-evoked potentials are likely the only helpful conduction study when a very proximal conduction block is suspected.[571] An EMG provides a more reliable assessment of femoral nerve pathology. Injuries proximal to the inguinal ligament will commonly involve the iliopsoas muscle. Variable abnormalities may be seen when the focus of injury lies from just proximal to the inguinal ligament to just above the femoral triangle, as many variations of motor branching to the quadriceps, pectineus, and sartorius muscles may occur in this region. However, at this relatively distal level, the iliopsoas muscle should be spared.

Treatment

Most reports suggest that conservative treatment is satisfactory in the majority of patients. Return to normal levels of function ranged from several months to a year. In the rare situation where retroperitoneal hemorrhage leads to progressive weakness, surgical exploration and decompression may be warranted.

Peroneal Nerve Injury

Anatomic Consideration

The common peroneal nerve fascicles incorporate the lateral portion of the sciatic nerve. At a variable level, just above the popliteal region, the sciatic nerve splits into its distinct branches, the common peroneal and tibial nerves (Figure 9-81). The common peroneal nerve courses laterally along the medial border of the biceps femoris muscle, crossing the proximal end of the lateral gastrocnemius muscle. The nerve becomes superficial as it passes between the fibular head and the peroneus longus muscle. Two branches diverge as the trunk passes anteriorly (Figure 9-82). The superficial peroneal nerve dives between the peroneus longus and the extensor digitorum longus and provides innerva-

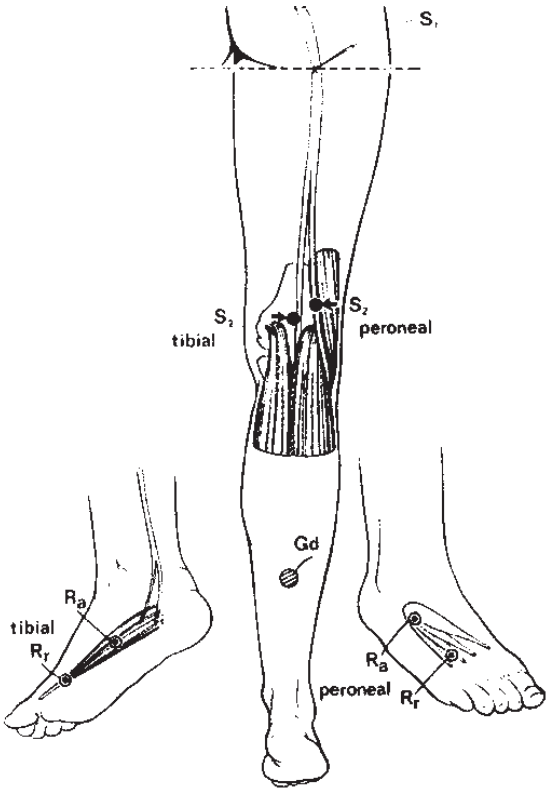

Fig. 9-81. The sciatic nerve dividing in to common peroneal and tibial branches above the popliteal fossa. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 201.

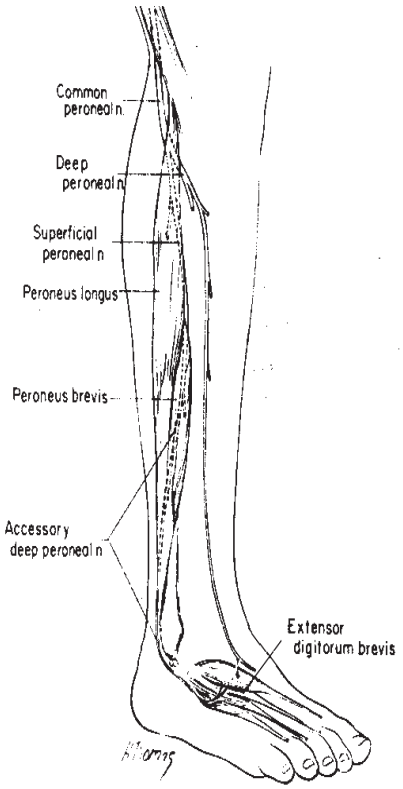

Fig. 9-82. Branches of the common peroneal nerve including the deep peroneal, superficial peroneal, and accessory deep peroneal nerves. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 208.

tion to the peroneus longus and brevis. The sensory portion continues distally, becoming superficial at the distal third of the leg. The superficial peroneal nerve transmits sensation from the anterolateral aspect of the distal half of the leg. Distal branches, including the intermediate and medial dorsal cutaneous nerves, supply sensation to the dorsum of the foot. Sensation along the first web space and the lateral border of the foot is provided by the deep peroneal and sural nerves, respectively, and not by the superficial peroneal nerve.

The deep peroneal nerve continues to run inferomedially along the medial aspect of the fibula, deep to the extensor digitorum longus muscle, anterior to the interosseus membrane. It provides articular branches to the knee and motor innervation to the tibialis anterior, extensor digitorum longus, and extensor hallicus longus muscles. The nerve dives beneath the extensor retinaculum between the extensor hallicus longus and tibialis anterior tendons. The lateral branch of the deep peroneal nerve innervates the extensor digitorum brevis and peroneus tertius muscles. The medial branch provides sensory innervation to the first web space.

Etiology

The course of the peroneal nerve makes it particularly susceptible to both compression and stretch injuries. As it passes beneath the head of the fibula, it lies intimately with the fibular periosteum for 10 cm. The initial 4 cm are covered only by skin and superficial fascia. Passage through a tunnel formed by the peroneus longus muscle, the fibula, and intermuscular septum limits the nerve's longitudinal excursion to merely 0.5 cm.[572] While entrapment within this tunnel is exceedingly rare, repetitive injuries may lead to adhesions, further limiting the mobility of the nerve (see Figure 9-82).

The vast majority of common peroneal nerve injuries occur at the nerve's most vulnerable point, at the fibular head. Mild injuries have been noted to occur spontaneously.[563,573] It is likely, however, that they developed secondary to inadvertent pro-

longed compression, possibly from limited movement during sleep, extended or repetitive leg crossing, or kneeling. Persons who have had an abrupt loss of weight are felt to be particularly susceptible, in light of the loss of protective superficial and epineural fat.[421] Ten percent of all sports-associated nerve injuries involve the peroneal nerve.[574] Minor trauma resulting in peroneal nerve injury during participation in sports including soccer, running, rugby, racquetball, mountain climbing, and bungee cord jumping have been anecdotally reported.[575–578] More severe injuries are associated with more profound trauma. Inversion injury at the ankle generates traction forces on the peroneal nerve at the fibular head. The Nitz et al[579] series revealed a 17% incidence of peroneal nerve injury with grade II lateral ankle sprains and an astonishing 86% incidence with grade III injuries. Trauma such as that sustained during motor vehicle accidents, which generate varus forces at the knee powerful enough to disrupt the lateral collateral ligaments, fracture the distal femur or proximal tibia, or dislocate the knee, commonly injure the peroneal nerve.[575,580]

Iatrogenic injuries to the peroneal nerve have also been documented. Application of casts and orthoses may compress the nerve against the fibular head. Prolonged compression due to poor positioning can occur during operative procedures. Injury following application of sequential pneumatic compression devices for deep vein thrombosis prophylaxis has been reported.[581]

Palsy of the deep peroneal branch is a frequent complication of external fixation during proximal tibial osteotomies, performed as treatment for severe varus deformities.[582] Limb lengthening through application of Ilizarov external fixation requires repeated elongation of 0.25 mm every 6 hours.[583] The tension-stress developed by distension of the fixator stimulates bone formation, a process described by Ilizarov as distraction osteogenesis.[584] Stretching of the soft tissues spanning the focus of lengthening is a potentially injurious corollary. A small series involving patients undergoing tibial lengthening revealed mild axonal sensorimotor peroneal neuropathies in all subjects.[585]

The ubiquitous use of arthroscopy in the diagnosis and treatment of knee injuries has led to a small but significant number of peroneal nerve injuries. The rate of serious neurological complications is low, less than 1% in several retrospective studies.[586,587] Rodeo et al[588] suggested that the position of the common peroneal nerve at the level of the joint line places it at risk during passage of large bore needles and especially by suture placement during lateral meniscal repair. Esselman and colleagues[589] caution that the topography of the common peroneal nerve at the level of the knee places the deep peroneal fascicles at greater risk than the superficial peroneal fibers. Great care needs to be taken during electrodiagnostic evaluations to prevent the erroneous diagnosis of a more distal lesion.[589]

Clinical Presentation

Weakness of the ankle dorsiflexors, especially the tibialis anterior muscle, is the most profound consequence of injury to the peroneal nerve. Incomplete paralysis impairs the ability of the dorsiflexors to contract eccentrically during heelstrike. The uncontrolled plantar flexion segue to footflat produces the foot-slap gait abnormality. Severe or complete paralysis forces the patient to adopt a steppage gait. Exaggerated hip and knee flexion is needed during the swing phase to clear the plantar flexed foot.[590] Weakness of the foot evertors and toe extensors are also commonly found on examination and may cause mediolateral instability during the stance phase. Sensory deficits were perceived by 79% of subjects in Katirji and Wilbourn's[591] study and variably involved the lateral aspect of the leg and dorsum of the foot.

Electrodiagnosis

Katirji and Wilbourn's series[591] of over 100 patients revealed four distinct patterns of peroneal nerve injury. Electrodiagnostic studies are essential in delineating these injuries, which prognostically are very different. They are also key tools in the differentiation of peroneal nerve injuries at the fibular head from more proximal problems including sciatic neuropathies, lumbar plexopathies, and L-5 radiculopathies.

The most important information can be gleaned from the peroneal motor NCS. Active electrodes are commonly placed on the extensor digitorum brevis muscle. Stimulation is applied just below the fibular head and at the popliteal fossa. In situations where a CMAP cannot be elicited from the extensor digitorum brevis muscle because of atrophy or local trauma, electrodes should be placed over the tibialis anterior muscle. Of the parameters assessed, the amplitude of the evoked potential is of the greatest utility. Decreased amplitudes above and below the fibular head suggest axonal loss, while normal amplitudes below the fibular head, but with decreased or absent amplitudes at the popliteal fossa,

are more indicative of conduction block. Prolongation of conduction velocities across the fibular head of greater than 10 ms relative to more distal conduction velocities have been touted as significant. They have not, however, been shown to be clinically relevant in terms of dorsiflexor weakness or ultimate prognosis.[573,591,592]

Sensory nerve action potentials can be obtained by stimulation of the superficial peroneal nerve. The active electrode is positioned one finger breadth medial to the lateral malleolus. Stimulation is applied 12 cm proximal to the active electrode, along the medial border of the peroneus longus muscle and the edge of the fibula.[593] Again, the amplitude of the action potential provides the most significant information. Normal amplitudes in the face of foot drop suggest either a lesion proximal to the dorsal root, conduction block at the fibular head or, less commonly, isolated injury to the deep peroneal fascicles with sparing of the superficial fibers. Decreased amplitudes or inability to obtain a potential suggests axonal injury. Posterior tibial motor and sural sensory NCSs as well as H-reflexes should be performed to help differentiate more diffuse and proximal etiologies.

Electromyography is essential in the evaluation of peroneal neuropathies. Two muscles innervated by the deep branch should be tested, including the tibialis anterior and either the extensor hallucis or the extensor digitorum brevis. One superficial peroneal innervated muscle, either the peroneus longus or peroneus brevis should also be examined. Testing of muscles within the L-5 myotome but not in the peroneal distribution such as the tibialis posterior or flexor digitorum longus is helpful in determining the likelihood of more proximal injuries such as proximal sciatic neuropathies, lumbar plexopathies, and most importantly, L-5 radiculopathies. Electromyographic evaluation of the short head of the biceps femoris muscle should always be performed. Innervated by the peroneal portion of the sciatic nerve, it is the only peroneal-innervated muscle above the knee. Its involvement is exceedingly helpful in injury localization.

According to Wilbourn,[592] the most common presentation is axonal injury at the fibular head. Nerve conduction studies reveal low amplitude CMAP during stimulation at both the popliteal fossa and distal to the fibular head. Sensory nerve action potentials are either not obtainable or of very low amplitude. Electromyography helps to localize the lesion. Evidence of denervation observed in muscles innervated by the tibial nerve, containing fascicles arising from the L-5 nerve root is normal. Needle examination of the short head of the biceps muscle helps to determine whether the injury is distal to its branch site in the mid thigh. Denervation of the short head of the biceps suggests a more proximal lesion involving the peroneal portion of the sciatic nerve.

The second most common type of injury is focal conduction block at the fibular head. CMAPs distal to the fibular head are normal, while those attained by popliteal stimulation are either not obtained or are of decreased amplitude. Interestingly, incomplete conduction block in this situation rarely causes temporal dispersion. Sensory action potentials are normal. Electromyographic findings are of little help and may in fact be confusing if not considered in relation to the NCSs. An injury severe enough to cause complete conduction block frequently injures at least a small number of axons. Sparse evidence of denervation may be seen in several muscles. It is important to realize that the discrete interference pattern and minimally increased recruitment frequency are products of focal conduction block and not severe axon loss.

A relatively rare presentation is a mixed axon loss-conduction block injury. Motor conduction studies reveal decreased action potential amplitudes below the fibular head and either absent or more severe attenuation of the amplitude during stimulation at the popliteal fossa. SNAPs are absent. Electromyography suggests significant denervation of muscles innervated by the deep and superficial peroneal branches.

Isolated axonal injury of the deep peroneal fascicles exclusively is extremely unusual. Proximal and distal CMUAP are of low amplitude or not obtainable. The superficial peroneal sensory potential is normal. Sensory NCSs of the deep peroneal nerve may be helpful.[594] While technically difficult, assymetry between affected and nonaffected limbs may suggest a focus distal to the dorsal root ganglion. Electromyography is important in this situation. Denervation will be observed just within muscles innervated by the deep peroneal nerve.

Treatment

Initial electrodiagnostic studies are of value in predicting the outcome of peroneal palsies and may help guide treatment plans. Berry and Richardson[572] observed a correlation between persistent, moderate sensorimotor deficits and initial distal motor conduction velocities of less than 30 meters per second. Smith and Trojaborg[573] also observed that nor-

mal sensory and motor conduction parameters below the fibular head invariably lead to a good prognosis. Abnormal distal latencies or significantly decreased amplitudes were associated with incomplete recovery. Wilbourn's[592] findings parallel those of the previous researchers. Focal conduction block at the fibular head typically resolved completely, within 8 to 12 weeks. Axon injury at the fibular head resulted in satisfactory but incomplete recovery and usually took upward of 6 months. More proximal axon loss such as those exhibited in sciatic neuropathies and L-5 radiculopathies portended poor outcomes. Mixed axon loss-conduction block injuries recovered in a bimodal fashion, with the conduction block resolving in weeks and reinnervation occurring several months later. Incomplete functional return was common.

Treatment of peroneal neuropathies varies with the severity of the injury. Maintenance of full range-of-motion at the ankle is of paramount importance. Prolonged stretching of the gastrocnemius-soleus complex should be performed several times a day. Progressive resistance strengthening should also be started early and include not only the dorsiflexors but the foot everters as well. Proprioceptive retraining at the ankle should be instituted prior to return to normal activities. Ankle foot orthoses are not necessary in situations where adequate toe clearance can be achieved. In cases of severe weakness of the foot dorsiflexors, moderate dorsiflexor weakness but with rapid fatigue or mediolateral instability, an ankle foot orthosis is indicated. An exhaustive discussion of this subject can be found in chapter 11, *Orthotics for the Injured Soldier.*

NERVE INJURIES OF THE FOOT AND ANKLE

Entrapment neuropathies of the foot and ankle are uncommon. The actual incidence is unknown.[595] They include entrapment of the superficial peroneal, sural, deep peroneal, and posterior tibial nerve or its branches, the medial and lateral plantar nerves, the calcaneal nerve, and the interdigital nerves. Entrapment of the deep peroneal nerve is also known as the anterior tarsal tunnel syndrome. Entrapment of the posterior tibial nerve or its branches is typically referred to as the tarsal tunnel syndrome. It is also referred to by some as the posterior tarsal tunnel syndrome. The term *entrapment* in this discussion will include those nerve injuries due to direct trauma as well as chronic compression.

These nerve injuries are likely underdiagnosed.[595,596] There may be a number of reasons for this situation. First, many patients present with a history of vague or fleeting symptoms, initially accompanied by only minimal or no definite neurologic deficits. If the patient presents to coaches or trainers, he may find that they are typically much more aware of soft-tissue injuries of the foot and ankle than they are of nerve injuries; thus, the index of suspicion may be relatively low, particularly on initial evaluation. This lack of awareness regarding the types and nature of entrapment neuropathies of the foot and ankle may also hold true for many physicians and podiatrists not experienced in this type of injury. The neural problems often coexist with soft-tissue injuries. The latter conditions typically receive initial treatment while the nerve injury either receives delayed attention or goes unnoticed altogether. In addition, the signs and symptoms of nerve injury may be temporally related to a given activity for only a brief period of time immediately following the activity. Thus, the nerve insult may not be appreciated when the individual is assessed well after the precipitating activity. Finally, the diagnosis may be difficult to detect by objective findings. Electrodiagnostic studies, including both NCSs and needle EMG, can be particularly helpful in confirming and localizing the site of nerve entrapment and may also be helpful in assessing the severity of the injury while offering clues regarding the prognosis of the nerve lesion. However, electrodiagnostic studies are primarily an extension of the physical examination and may not be sensitive enough to pick up subtle or transient nerve pathology. In addition, they require appropriate timing to obtain meaningful information for accurate diagnosis and prognostication.

Although the number of neurologic injuries at the foot and ankle are undoubtedly a small percentage of the total, it is worthwhile examining the type and percentage of acute and overuse injuries of the foot and ankle, since they appear to play a role in causing nerve damage. Garrick and Requa[597] assessed 16,754 injuries occurring in a variety of sports and found that one quarter of the injuries were to the foot and ankle; 9.7% involved the ankle, 15.5% the foot.

They noted that over half (50.4%) of the ankle injuries were acute sprains. Volleyball and basketball had the highest proportion of acute ankle sprains at 82% and 79% of participants, respectively. Football and racquetball each had more than 70%.[597]

The highest proportion of overuse injuries of the ankle occurred in cycling (70%); ice skating (40.5%); and ballet and running, each with more than 35%.

Volleyball had the highest percentage of overuse injury of the foot at 70%; followed by running at 59%; and then by tennis, gymnastics, racquetball, and skiing, each at 50% or above.[597]

According to Schon and Baxter,[596] neurologic injuries of the foot and ankle occur most commonly in runners or joggers (60%) and dancers (20%). They add that the most common neurologic problems in athletes are interdigital neuromas, followed in descending order by entrapment of the first branch of the lateral plantar nerve, medial plantar nerve, tibial nerve, lateral plantar nerve, deep peroneal nerve, superficial peroneal nerve, and sural nerve.

Tarsal Tunnel Syndrome

Posterior tarsal tunnel syndrome is perhaps the best known of the entrapment neuropathies of the foot and ankle. Tarsal tunnel syndrome is defined as the entrapment of the tibial nerve or one of its branches by the flexor retinaculum. Kopell and Thompson[598] were credited with the first description of this nerve injury in 1960. Keck and Lam, however, were independently the first to coin the term tarsal tunnel syndrome (as cited by Radin[599]).

Anatomic Considerations

The tarsal tunnel is formed by fibrous and osseous structures, including the flexor retinaculum or laciniate ligament, which forms the roof. The medial wall of the calcaneus, the posterior aspect of the talus, the distal tibia, and the medial malleolus complete the tunnel. The tendinous arch of the abductor hallucis muscle contributes to the medial wall of the tunnel along with the laciniate ligament.[600] The latter ligament has both a superficial and a deep layer. The superficial layer is a thickening of the crural fascia between the medial malleolus and the calcaneal tuberosity and the deep layer extends from the medial malleolus to insert on the crural fascia. The deep layer lies over the sustentaculum tali and posterior talar process (Figure 9-83).[600]

The tibial nerve, the posterior tibial artery and vein, and tendons of the posterior tibialis, flexor digitorum longus, and flexor hallucis longus all pass through the tunnel. The posterior tibial nerve typically divides 1 to 2 cm proximal to an imaginary line drawn from the tip of the medial malleolus to the calcaneal tuberosity. The medial and lateral plantar nerves, branches of the tibial nerve, each then enter one of the two tunnels located within the abductor hallucis muscle.

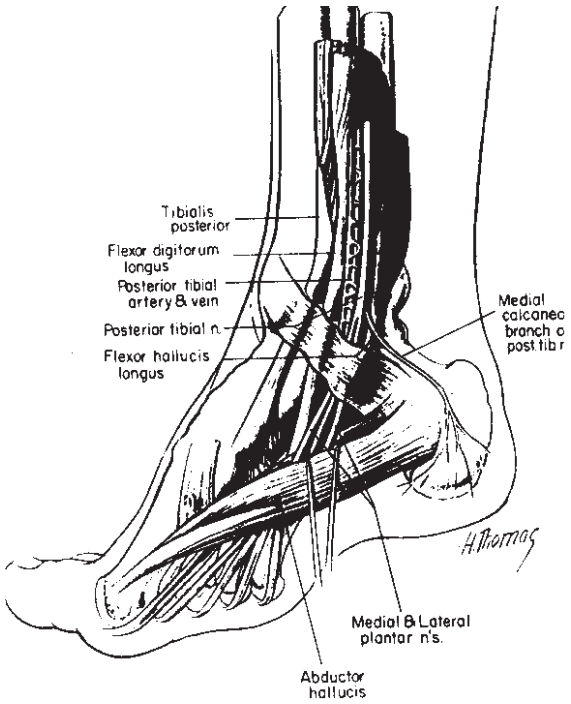

Fig. 9-83. The tarsal tunnel. The tibialis posterior, flexor digitorum longus, flexor hallucis longus, posterior tibial nerve, artery, and vein pass beneath the flexor retinaculum. Reprinted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 54.

The medial plantar nerve supplies sensation to the medial aspect of the plantar surface of the foot and the medial half of the fourth toe. It also provides a motor branch to the abductor hallucis, the short toe flexors, and the first lumbrical muscle.

The lateral plantar nerve provides sensation to the fifth toe and the lateral half of the fourth toe. It also supplies sensory innervation to the lateral sole and motor fibers to the remaining intrinsic muscles of the foot, except for the short toe extensor. As previously noted, the medial and lateral plantar branches pass via separate openings in the medial superior origin of the abductor hallucis muscle. This anatomic structure predisposes these nerves to selective entrapment.[601] The first branch of the lateral plantar nerve passes obliquely between the abductor hallucis muscle and the quadratus plantae. It subsequently divides into three branches. One innervates the periosteum of the medial process of the calcaneal tuberosity; it also frequently innervates the long plantar ligament and occasionally the quadratus plantae muscle. Another supplies the

flexor digitorum brevis muscle. The terminal branch innervates the abductor digiti minimi muscle.[596]

The calcaneal nerve branch lies posterior to the aforementioned branches and may pierce the laciniate ligament or exit between it and the fascia of the abductor hallucis muscle. Branches of the calcaneal nerve piercing the laciniate ligament innervate the medial aspect of the calcaneus, while other branches travel the length of the tarsal tunnel to innervate the plantar aspect of the heel.[601] Just proximal to the tarsal tunnel, a calcaneus branch splits from the tibial nerve to innervate the skin of the heel. This branch is susceptible to nerve compression at the edge of the laciniate ligament. If calcaneal nerve entrapment does occur, a patient typically complains of heel pain.[602]

Etiology

Tarsal tunnel syndrome has been attributed to a multitude of etiologies. Increased pressure within the tarsal tunnel is felt to be the common denominator in causing nerve injury. This may occur as the result of direct compression injury. It may also result from decreased volume due to space-occupying changes within this nonyielding, fibroosseous tunnel. The synovial proliferation associated with tenosynovitis; soft-tissue masses such as ganglion, lipomas, neurilemomas, and neurofibromas, as well as local bleeding; and venous engorgement, edema, or both, are space-occupying conditions that may cause focal nerve injury within the tunnel.[600]

Bony anomalies may cause compression neuropathy. Schon and Baxter[596] report two cases in which bony abnormalities of the posterior talus caused such nerve compression. Metabolic disorders or hormonal changes, such as those that occur with diabetes, pregnancy, myxedema, acromegaly, and hyperlipidemia, predispose an individual to compression neuropathy.[600]

The medial plantar neurovascular structures pass through the upper section of the tarsal tunnel. This upper section is reportedly narrower than the lower and accounts for the suggestion that the medial plantar vessels and nerves are more sensitive to volume changes than the lateral plantar neurovascular structures which pass through the lower section.[600] Jackson and Haglund[595] state, however, that entrapment of the tibial nerve most commonly occurs at the anterior, inferior aspect of the tarsal tunnel at the point the nerves wind around the medial malleolus. They agree with Kaplan and Kernahan[603] that the lateral plantar nerve branch is more frequently subject to compression neuropathy than the medial branch.[595]

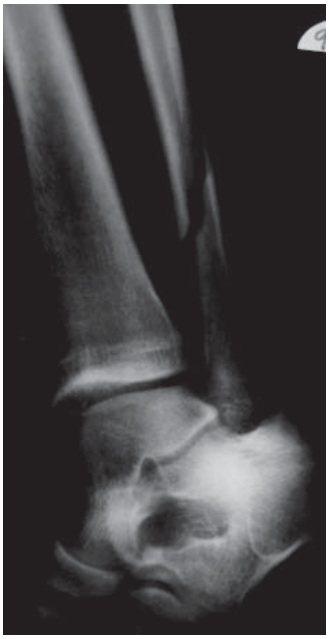

Fig. 9-84. Pronation–external rotation injury at the ankle, resulting in tibial nerve injury, rupture of the anterior talofibular ligament, incomplete tear of the deltoid ligament, transverse fracture of the medial maleolus, spiral fracture of the distal fibula, and fracture of the posterior lip of the distal tibia. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

The majority of nerve injuries involving the tarsal tunnel have been recorded as posttraumatic complications.[422] Many of these have been considered to be the result of scar formation after an ankle sprain and others have been thought to be due to secondary osseous changes. Ankle sprains involving the deltoid ligament and calcaneal or medial malleolus fractures or both have been cited as possible causes (Figure 9-84).[604]

Kraft[605] notes that the tibial nerve is especially vulnerable during ambulation because of its location and the structure of the tarsal tunnel. He feels that the distal branches of the tibial nerve are sometimes injured as a result of repetitive trauma rather than chronic, steady compression. The repetitive action at the ankle experienced with running, especially when associated with excessive pronation, is thought to place the tibial nerve "on stretch" on a repetitive basis.[595] Rask[606] reports that excessive valgus or external rotation of the foot, particularly that experienced with running, potentially causes

excessive stretch of the medial plantar nerve and referred to this problem as "jogger's foot." The fact that prolonged standing or walking typically precipitates or exacerbates symptoms supports the contention that fluid stasis or engorgement within the tarsal tunnel is a factor in causing compression.[422]

Other suspected causes or predisposing conditions include inflammatory soft tissue conditions associated with rheumatoid arthritis, ankylosing spondylitis, Reiter's disease, collagen vascular diseases, and isolated flexor tenosynovitis secondary to acute or chronic injury.[596,604] Chronic thrombophlebitis has also been included in the list of potential causes.[422,604] Ricciardi-Pollini et al[607] reported eight cases of tarsal tunnel syndrome and found a thickened laciniate ligament, varices, exostoses, and adhesions to be causative factors. Jackson and Haglund[595] add that gout with urate deposits, connective tissue changes associated with aging, as well as fluid retention and bone spurs may also have a role in exacerbating or precipitating nerve injury.

Some have implicated severe pronation of the hindfoot as a potential cause.[596] DeLisa and Saeed[604] summarized multiple reports in the literature that indicate that abnormal calcaneal eversion relative to an adducted talus and an abducted forefoot may place increased tension on the neurovascular structures within the tunnel and cause this syndrome. Hypertrophy of the abductor hallucis muscle or an accessory muscle belly of the short digital flexors, rapid weight gain, and constant squatting are additional causes reported in the literature.[595,604]

Fewer cases have been noted as the consequence of benign tumors, such as neurilemomas, ganglion, and lipomas. Others have been documented as the result of varicosities beneath the flexor retinaculum.[602]

Jackson and Haglund[595] reported two selected cases of tarsal tunnel syndrome. One occurred as the result of direct, extrinsic compression attributable to a tight alpine ski boot. The other occurred in a female runner with bilateral plano valgus deformities and increased Q-angles.

Schon and Baxter[596] purport two mechanisms for tarsal tunnel syndrome. They include vascular compromise of the nerve by pressure on the vasa nervorum and direct compression neuropathy. These authors feel that the former mechanism results in primary sensory deficits without motor symptoms or signs while the latter results in both sensory and motor signs and symptoms. Pecina and colleagues[600] note that the tibial nerve, though rich in vascularity, is sensitive to ischemic insult and agree that compression of the vasa nervorum and the resulting ischemia may manifest as neurologic symptoms consistent with tarsal tunnel syndrome.

Clinical Presentation

Patients typically present with complaints of numbness and tingling involving the toes and plantar aspect of the foot. Onset is usually insidious.[599] The distribution of sensory involvement is quite variable, reflecting the variable involvement of the tibial nerve or its branches. Sensory distribution of the medial or lateral plantar nerves or both are characteristically involved.[598] Individuals may also complain of burning pain and dysesthesias in a similar distribution. At times, particularly with prolonged activity, symptoms may be relatively constant with activity-induced exacerbations. In general, however, symptoms are intermittent and precipitated or exacerbated by prolonged standing, walking, running, or similar activities involving dynamic action at the foot and ankle. Rest usually diminishes such symptoms. Nocturnal paresthesias and pain are common and may be severe enough to prohibit sleep or, more typically, awaken the patient from sleep.[604] Not infrequently, patients complain of pain radiating up the medial aspect of the calf and this presentation may lead one to erroneously presume the presence of a radiculopathy.[600]

Sensory impairment classically involves the sole of the foot. Anesthesia and hyperesthesia are reportedly rare, while hypesthesia and loss of two-point discrimination are early signs of nerve compression.[600] A positive Tinel's sign may be elicited over the tarsal tunnel site and passive placement of the heel into an end-range valgus and everted posture[604] or eversion with dorsiflexion at the ankle[600] may exacerbate symptoms. The Valleix phenomenon, the situation where a Tinel's sign is obtainable in the tarsal tunnel region and tenderness is elicited both proximal and distal to the compression site, may be demonstrated on occasion.[608] Passive varus alignment of the heel, on the other hand, typically reduces the symptoms presumably due to creation of "slack" in the laciniate ligament.[604] There is often tenderness with palpation at a point posterior and inferior to the medial malleolus. There is, however, a lack of tenderness with palpation in the area of the sole of the foot where the patient is typically most symptomatic.[599] A variable degree of retro- or submalleolar swelling may be noted upon inspection.[600] If motor impairment is present, it usually manifests as weakness of the toe flexion at the metatarsophalangeal joints and toe extension at the IP joints of all or some of the toes.[604] In contrast,

strength of the long flexors of the foot and toes is preserved.

Differential Diagnosis

The differential diagnosis should include Achilles tendinitis, plantar fasciitis, prolapsed metatarsal heads, plantar callosities, longitudinal arch sprain, localized rheumatoid disease, other arthritic conditions, sciatica, peripheral neuropathy, peripheral vascular disease, old fractures, bone spicules, accessory ossicles, and an S-1 radiculopathy.[595] Diabetic neuropathy with burning paresthesias of both feet may be particularly difficult to distinguish from bilateral tarsal tunnel syndrome by clinical exam alone.[422]

Radiographic and electrodiagnostic studies are of potential value in the evaluation for tarsal tunnel syndrome. Radiograph examination of the ankle may detect the presence of bony anomalies, ankle fractures, accessory ossicles, and ankle malalignment. These abnormalities may be factors in precipitating the syndrome or any one may be the primary source of the symptoms. Careful clinical evaluation will assist in making this distinction. Ricciardi-Pollini et al[607] documented a case involving a young woman who was noted by radiograph to have an exostosis located at the medial site of the talus. At surgery to remove this osseous lesion, it was noted to be compressing the posterior tibial nerve. Its removal, coupled with neurolysis, resulted in complete relief of her symptoms.

Electrodiagnosis

Electrodiagnostic studies may be of particular importance not only in reliably diagnosing a peripheral neurologic deficit, but in providing information regarding the severity and specific distribution of nerve involvement. Such studies may be particularly helpful in distinguishing a peripheral neuropathy from a bilateral tarsal tunnel syndrome.

Multiple studies are cited by DeLisa and Saeed[604] which demonstrate prolonged sensory or motor distal latencies, or both, across the tarsal tunnel region. These authors found the mixed nerve conduction technique as described by Saeed and Gatens[609] substantially superior to the medial and lateral plantar orthodromic sensory nerve conduction technique employing a ring electrode stimulator at the great toe and fifth toe, respectively. The recording electrode in the latter case is located just proximal to the flexor retinaculum. This method can be difficult to perform technically, particularly because one typically requires an averager to obtain an evoked potential of acceptable amplitude. On the other hand, the mixed nerve conduction technique involves orthodromic stimulation of the medial or lateral plantar nerves in the midsole of the foot. This stimulation is done at a predetermined distance with the recording electrode located proximal to the flexor retinaculum. This technique is typically reliable and reproducible.

Motor NCSs through the tarsal tunnel are an additional means of assessing the integrity of the medial or lateral plantar nerves. In the case of the medial plantar nerve study, the active pickup electrode is located over the motor point of the abductor hallucis muscle. The lateral plantar nerve motor NCS is performed with the active pickup electrode placed over the abductor digiti minimi. In both cases, the site of stimulation is 10 cm proximal to recording electrodes at a point just proximal to the flexor retinaculum.[604]

Needle electrode study of foot intrinsic muscles served by either the medial or lateral plantar nerves may detect electrodiagnostic abnormalities consistent with muscle membrane instability and suggestive of a denervation process. DeLisa and Saeed[604] considers the abductor hallucis and the first dorsal interosseus muscles especially useful in the initial screening process.

In the presence of tarsal tunnel syndrome, one will typically obtain either prolonged or absent sensory or CMAP. In addition, there may be evidence of denervation activity, along with reduced recruitment and interference patterns involving the foot intrinsics.

Treatment

Conservative management is the initial treatment of choice. A trial of an NSAID and custom-molded orthotics are typically offered. Injection of the tarsal tunnel site with a mixture of a local anesthetic and corticosteroid compound may be performed for both diagnostic and therapeutic purposes. Pecina and colleagues[600] feel repeat corticosteroid injections may be performed at the same site up to three times over a two-month period. If unsuccessful and no other conservative measure offers relief, they then feel a surgical option should be entertained. Ice and ultrasound have also been employed with variable success.

Appropriate alteration in footwear may be warranted for improved ankle and foot stability. A custom-molded foot orthotic may be added to provide optimal heel stability and prevention of excessive forefoot pronation. Use of a medial arch sup-

port as well as an external heel counter on the shoe are two additional means of achieving appropriate foot alignment. Rask,[606] however, notes that arch supports should be avoided because they may cause compression injury to the medial plantar nerve in the region of the longitudinal arch. The latter issue is controversial and will receive further elaboration later in the discussion of medial plantar entrapment neuropathy in the region of the longitudinal arch.

Relative rest with elimination of the suspected precipitating or exacerbating activities is a particularly important measure one should consider. This especially includes elimination of repetitive trauma in the region of the tarsal tunnel, as well as repetitive or marked stretch of the tarsal tunnel elements. Complete immobilization has not been required in this author's experience, although some advocate short-term use of plaster casts.[600] Pecina and colleagues[600] reported a study by Androic that purportedly documented success as high as 79% with conservative measures.

For refractory cases, to include those patients suffering from tarsal tunnel syndrome as the result of varices, exostoses, and adhesions, surgical decompression along with neurolysis may be warranted. Ricciardi-Pollini and colleagues[607] reported a small series of eight patients undergoing surgical decompression due to the latter problems as well as to a thickened laciniate ligament. All but one received complete relief of their symptoms within 24 hours after surgery. Radin[599] studied a series of 14 patients with documented tarsal tunnel syndrome with associated varus heels and pronated, splayed feet. He argued that surgical release of the flexor retinaculum posterior to the medial malleolus is the treatment of choice in individuals with tarsal tunnel syndrome and a planovarus deformity. He reported successful outcomes with surgical intervention in more than 90% of the cases. Kaplan and Kernahan[603] surgically managed 18 patients with tarsal tunnel syndrome and reported complete or partial improvement in all.

Lateral Plantar Nerve Branch-I Entrapment

Entrapment of the first branch of the lateral plantar nerve should be considered in individuals suffering from chronic heel pain. This appears especially applicable for the athletic population. Schon and Baxter[596] report that about 10% to 15% of athletes with chronic, persistent heel pain have entrapment neuropathy of this particular nerve branch. Pecina and colleagues[600] note that runners and joggers make up the great majority of reported cases, but acknowledge that athletes in soccer, dance, and tennis also experience this problem.

Etiology

The site of entrapment is purported to occur between the deep fascia of the abductor hallucis muscle and the medial caudal margin of the head of the quadratus plantae muscle.[596,600] Chronic repetitive trauma at the latter site is the presumed cause. Local inflammation secondary to chronic pressure reportedly may also occur where the nerve passes over the plantar side of the long plantar ligament or in the osteomuscular canal between the calcaneus and the flexor digitorum brevis.[596] A review of the literature by Schon and Baxter[596] indicates that a hyperpronated foot, hypertrophied abductor hallucis or quadratus plantae muscle, accessory muscles, abnormal bursae, and phlebitis in the calcaneal venous plexus may all play a role in precipitating this nerve entrapment.

Clinical Presentation

Chronic heel pain is the typical presentation. Characteristically, the patient is an athlete involved in a sport requiring a running activity. Pain is typically exacerbated by the running activity or even by walking and is variably relieved with rest. Although radiation of pain to the ankle is not uncommon, numbness in the heel or foot is atypical.[596,600]

Reproducible point tenderness along the course of the nerve, particularly at the site of suspected entrapment, is present. This site is deep to the abductor hallucis muscle. A Tinel's sign may be elicited, but is reportedly an atypical finding.

Differential Diagnosis

Achilles tendinitis, plantar fasciitis, and bursitis should be included in the differential diagnosis. A roentgenogram should be obtained to exclude the possibility of heel spurs, stress fractures, and bone tumors. Occasionally, a bone scan may be warranted to clarify the diagnosis. Less common causes of heel pain that merit consideration include heterotopic calcification, Paget's disease, Strumpell-Marie disease, venereal disease, Sever's apophysitis, rheumatoid arthritis, and gout.[602] More proximal involvement of the tibial nerve, sciatic nerve, and an S-1 radiculopathy should be excluded. Electrodiagnostic testing, coupled with the history and physi-

cal examination, may prove helpful in ruling out the latter possibilities.

Treatment

Patients typically receive a trial of conservative therapy similar to that provided for heel pain due to other causes. This usually includes a trial of an NSAID, relative rest, ultrasound, stretching, heel cups, heel doughnut pads, and cortisone injections with local anesthetic. The latter treatment regime is sometimes successful. However, in refractory cases, typically those that persist after 6 to 12 months of conservative therapy, surgical release of the first branch of the lateral plantar nerve is indicated.[600]

Medial Plantar Nerve Entrapment at the Longitudinal Arch

Isolated entrapment of the medial plantar nerve in the longitudinal arch is a rarely diagnosed condition. Rask described such an entrapment neuropathy in joggers, and as previously mentioned, coined this clinical syndrome the "jogger's foot."[606]

Anatomy

The course of the medial plantar nerve has been previously discussed.

Etiology

The entrapment of the medial plantar nerve in this particular syndrome is felt to occur in the region of the longitudinal arch, also known as the master knot of Henry.[600,606] It has been attributed to chronic, focal inflammation due to repetitive trauma in the latter area. Rask[606] contends that long-distance running is the typical precipitating activity, particularly if excessive valgus displacement and external rotation occurs with this activity. He feels the latter presentation causes excessive stretch of the medial plantar nerve against the fibromuscular tunnel through which it passes.

Clinical Presentation

The typical patient is a middle-aged individual who jogs on a regular basis.[600] Rask[606] notes that chronic burning pain in the heel region is the typical presentation. Pecina and colleagues[600] note that onset of discomfort is characteristically temporally related to using a new arch support.

Impaired sensation involving the medial aspect of the sole of the forefoot and point tenderness at the site of entrapment in the arch of the foot, posterior to the navicular tuberosity, is characteristic.[600,606] A Tinel's sign may also be elicited.

Differential Diagnosis

More proximal entrapment of the medial plantar nerve in the tarsal tunnel, as well as tibial or sciatic nerve pathology, S-1 radiculopathy, and peripheral neuropathy should be excluded. Electrodiagnostic assessment, performed as an extension of the history and examination, usually permits one to confidently exclude these possibilities. Local anesthetic nerve block at the site of presumed entrapment typically results in temporary relief of symptoms and arguably excludes the possibility of calcaneal bursitis and plantar fasciitis.[606]

Treatment

Pecina et al[600]and Rask[606] note that this form of nerve entrapment typically responds to conservative management alone. A trial of relative rest, NSAID, and local anesthetic and cortisone injections have been implemented with some success. The injections are performed at the site of presumed entrapment just posterior to the navicular tuberosity and, as previously alluded, may have both diagnostic and therapeutic benefits.[606]

Rask[606] also advocates having the jogger modify his running style, specifically avoiding excessive valgus and external rotation when running. The jogger is encouraged to run on the lateral aspect of his foot with a very slight toe-in sprint in order to relieve pressure from the medial plantar nerve. As previously noted, Rask feels that arch supports should be avoided in joggers who develop medial plantar nerve entrapment neuropathy in the longitudinal arch area. He believes that such supports can cause further trauma to the medial plantar nerve in the latter region.[606] If the arch support is fabricated so that it is too high or causes focal pressure in the longitudinal arch, then his recommendation appears reasonable. However, if the arch support is custom-designed to accommodate the individual's arch and to evenly distribute pressure over a broad rather than focal area, then direct pressure trauma is likely to be minimal. In addition, the benefit derived in preventing hyperpronation of the forefoot and in stabilizing the foot and ankle arguably outweigh the likelihood of direct pressure trauma to the nerve.

In refractory cases, surgical neurolysis is advocated.[606] However, literature regarding the efficacy of surgical intervention for this particular form of entrapment neuropathy is lacking.

Anterior Tarsal Tunnel Syndrome

This syndrome is described as an entrapment of the deep peroneal nerve beneath the inferior extensor retinaculum. There has been a paucity of reports regarding this form of entrapment in the literature to date.[610–612] Kopell and Thompson[598] have been credited with identifying the syndrome in 1963.

This syndrome has been documented most commonly in runners. Other sports in which this problem has been identified include soccer, skiing, and dancing.[596]

Anatomic Considerations

In the proximal third of the leg, the deep peroneal nerve passes between the extensor digitorum longus and tibialis anterior muscles. In the middle third, it passes deep to the extensor hallucis longus muscle and tendon. At a point 3 to 5 cm above the ankle joint, the nerve courses between the extensor digitorum longus and extensor hallucis longus muscles. At 1 cm above the ankle joint, underneath the oblique superior medial band of the inferior extensor retinaculum, a branch to the extensor digitorum brevis emerges laterally. The medial branch of the deep peroneal nerve passes under the oblique inferior medial band of the inferior extensor retinaculum along with the dorsalis pedis artery. At this point the deep peroneal is susceptible to compression injury between the talonavicular joint ridges and the retinaculum (Figure 9-85).[596]

The inferior extensor retinaculum typically has three branches and forms a Y-shaped pattern transversely across the dorsum of the foot. The deep peroneal nerve has branches innervating all foot extensors except for the extensor digitorum brevis at the level of the anterior tarsal tunnel. This accounts for the fact that the only muscle typically observed to be involved in anterior tarsal tunnel syndrome is the extensor digitorum brevis.[600] The deep peroneal nerve also innervates the first dorsal interosseous muscle, but objective determination of atrophy and weakness involving this muscle is obviously difficult to assess accurately.[422]

In its distal course, the deep peroneal nerve pierces the dorsal aponeurosis of the foot where it becomes superficial. It then provides cutaneous innervation to the first web space, along with sensory supply to the lateral aspect of the great toe and medial aspect of the second toe.[596] A number of variations in the pattern of nerve branches off the deep peroneal nerve have been documented and are reportedly not uncommon.[610]

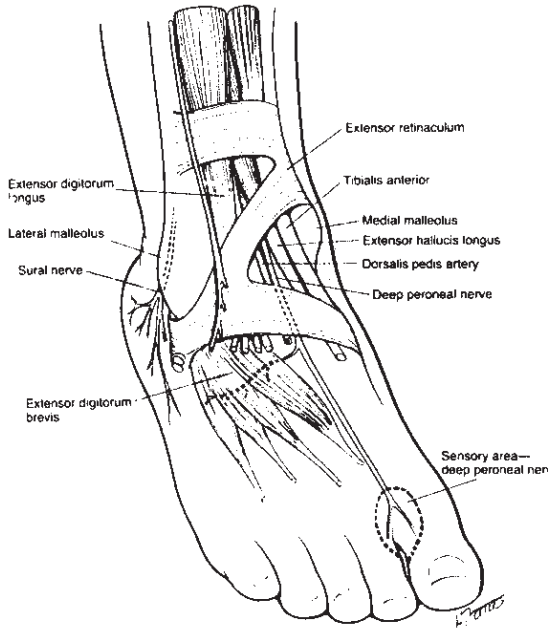

Fig. 9-85. The anterior tarsal tunnel. Branches of the deep and superficial peroneal nerve; tendons of the tibialis anterior and extensors digitorum longus and hallucis longus pass beneath the superior, oblique, and inferior bands of the extensor retinaculum. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 53.

Etiology

Anterior tarsal tunnel syndrome is usually attributed to chronic or repetitive compression injury at the ankle. Pecina and colleagues[600] contend that anatomical factors, specifically a tight, unyielding retinaculum overlying a bony structure, predisposes the deep peroneal nerve to such injury. Ankle edema; fractures; subluxations and sprains; as well as osteophytes, synovial pseudocysts, neuromas, ganglia, aneurysms, and tenosynovitis have been implicated as possible causes.[600] Bony anomalies of the talonavicular joint; repetitive compressive trauma from tight shoes, high boots, or shoe straps; chronic stretch injury due to prolonged ankle plantar flexion while wearing high heels; and direct

ankle trauma have also been described as potential causes.[422,610] Maximal stretch of the deep peroneal nerve at the ankle has been documented to occur when end-range plantar flexion is coupled with dorsiflexion of the toes.[422] Schon and Baxter[596] cite from their experience that most patients have a history of recurrent ankle sprains, specifically those sprains in which the ankle is forced into plantar flexion and supination. They also note that they have observed this syndrome to occur in joggers who wear keys under the tongue of their shoes when running and soccer players who receive direct, often repetitive trauma to the dorsum of the foot. They additionally suggest that the deep peroneal nerve may sustain repetitive trauma in those individuals who perform sit-ups with their feet hooked under a hard surface.

Clinical Presentation

The typical patient complains of burning pain in the region of the web space between the great toe and second toe. There may also be subjective complaints of numbness in the latter area. The patient does not typically complain of any focal weakness. Nocturnal pain and paresthesias of the foot are not uncommon. Patients may point out that prolonged standing or walking, tight-fitting shoes, or walking in high heels precipitates or exacerbates their symptoms, while extension or eversion of ankle relieves their symptoms to some degree.[422,600]

Decreased sensation to light touch and pinprick is typically noted in the web space between the great toe and second toe. A Tinel's sign may be elicited with a tap over the anterior tarsal tunnel site. There may be also be variable weakness and atrophy of the extensor digitorum brevis muscle. The latter muscle is best tested with the ankle maximally dorsiflexed to eliminate the actions of the extensor hallucis longus and extensor digitorum longus muscles.[600]

Differential Diagnosis

The primary conditions which must be differentiated from anterior tarsal tunnel syndrome include more proximal peroneal nerve compression injury, especially at the fibular head, and an L-5 radiculopathy. If the lateral branch of the distal deep peroneal nerve is selectively injured, then sensory impairment will not be present. Instead, a patient may simply complain of foot pain. Under such circumstances, it is difficult to definitely exclude the possibility of arthritic pathology or ligamentous injuries in the ankle region. Focal atrophy involving the extensor digitorum muscle is one clue that helps to make this distinction. If more proximal peroneal nerve injury is present, then delineation of muscle weakness and atrophy above the level of the ankle may be present.[422]

Electrodiagnosis

Electrodiagnostic studies remain particularly helpful in excluding the possibility of more proximal peroneal nerve injury and an L-5 radiculopathy. A prolonged distal latency when stimulating at the ankle and recording at the extensor digitorum brevis, along with normal proximal conduction velocity, supports the presence of the anterior tarsal tunnel syndrome. In addition, needle electrode examination of the extensor digitorum muscle may yield evidence of muscle membrane instability suggestive of denervation activity, while sparing other lower extremities typically served by the deep peroneal nerve. It should be noted, however, that needle electrode abnormalities suggestive of denervation activity may be found in a small percentage of patients who are asymptomatic or without nerve conduction deficits or both, presumably due to the local muscle trauma that occurs with regular shoewear. Gessini and colleagues[610] reported four cases of anterior tarsal tunnel syndrome. Three of the four demonstrated significantly prolonged motor distal latency and the fourth was at the upper limits of normal. In addition, all four patients had needle electrode findings of denervation activity isolated to the extensor digitorum brevis muscle.

Roentgenograms of the ankle and foot may be helpful in excluding osseous lesions that may potentially compress the deep peroneal nerve, such as an osteophyte on the dorsum of the talus where it articulates with the navicular bone.[596]

Treatment

Conservative management is typically advocated and includes avoidance of constricting compression in the anterior tarsal tunnel region by tight shoes, boots, or shoelaces. Also, avoidance of high heels may eliminate the posture of ankle plantar flexion and toe dorsiflexion, a position felt to precipitate or exacerbate the symptoms of anterior tarsal tunnel syndrome. Use of shin guards with a lip over the dorsum of the foot when playing soccer may assist in preventing direct trauma to the anterior

ankle. Relative rest and use of NSAIDs, local anesthetic, and corticosteroid injections may provide a variable degree of relief. Use of orthotics to maintain the ankle at 90° has been suggested, although there is no convincing evidence that this truly alters the patient's status. Surgical decompression is generally reserved for refractory cases only. Schon and Baxter[596] reported a case involving a 19-year-old college sprinter with anterior tarsal syndrome attributed to an osteophyte on the dorsum of the talus, which was surgically excised. The patient's symptoms resolved, and he has reportedly returned to training. Literature documenting the efficacy of surgical intervention otherwise appears to be lacking at this time.

Superficial Peroneal Neuropathy

Superficial peroneal entrapment neuropathy was reportedly first described by Henry in 1945 and termed "mononeuralgia in the superficial peroneal."[600,613,614] It is an infrequently diagnosed and documented condition. The average age is reported to be 36 years, but ranges from 15 to 79 years. In a subpopulation, specifically in athletes, superficial peroneal neuropathy has been diagnosed at an average age of 28 years. Men and women have been documented to experience this problem with equal incidence. Of the athletes, runners have been most frequently diagnosed with this neuropathy. It has also been documented in individuals engaging in soccer, tennis, racquetball, hockey, and dancing.[596,613–616] The most common site of compression injury is typically described as occurring at the junction of the middle and distal thirds of the leg where the superficial peroneal nerve surfaces through the crural fascia.[596,600,615]

Anatomic Considerations

The superficial peroneal nerve, a branch of the common peroneal nerve, passes through the anterolateral compartment of the leg and innervates the peroneus longus and brevis muscles. The nerve lies between the peroneus longus and the extensor digitorum muscles proximally. Its subsequent course takes it between the anterior intermuscular septum and the fascia of the lateral compartment. It then pierces the deep fascia and emerges to a subcutaneous route in the leg about 10.5 to 12.5 cm above the lateral malleolus.[596] This point is typically at the level between the middle and distal thirds of the leg. Thereafter, it divides into lateral and medial terminal cutaneous branches, also referred to as the intermediate dorsal cutaneous and the medial dorsal cutaneous nerves.[596,617] The medial terminal branch supplies the medial side of the dorsum of the foot, as well as the dorsal aspect of the medial side of the great toe and of the adjacent sides of the second and third toes. The lateral terminal division supplies sensory fibers to the lateral aspect of the dorsum of the foot and the adjacent sides of the third, fourth, and fifth toes.[617] The innervation of the toes typically extends to the level of the IP joints.[422] The cutaneous innervation of the superficial peroneal nerve also includes innervation to the skin of the anterolateral aspect of the leg.[600]

Etiology

Pecina and colleagues[600] point to trauma as the most common proposed etiology of superficial peroneal neuropathy. Styf cites literature indicating that local trauma has been reported in about 25% of patients with compression of this particular nerve.[613]

Schon and Baxter[596] note that most cases have reportedly shown that the edge of the deep fascia compresses against the superficial peroneal nerve as it pierces this latter structure. In the presence of muscle herniation through the fascia due to fascial defects, they feel the nerve is likely to experience focal compression injury.[596,611] Styf[613] points out that exercise creates sufficient pressure to herniate muscle tissue through fascial defects with resulting compression of the nerve. McAuliffe and colleagues[618] reported a case of a young woman who experienced bilateral superficial peroneal nerve entrapments as the result of fat herniation through fascial defects in the lateral compartments of both legs. In each leg, the nerve was entrapped against the fascia by a nodule of fat and was noted to have focal, fusiform swelling. McAuliffe and colleagues[618] also cite work by Banerjee and Koons[619] who documented unilateral superficial peroneal nerve entrapment exacerbated by exercise, reportedly attributable to ankle inversion injury or to the wearing of high boots.

Styf and Korner[620] note that there is a fibrotic, relatively unyielding tunnel through which the superficial peroneal nerve passes. It is located in the corner between the anterior intermuscular septum and the fascia over the lateral compartment. In the presence of edema following trauma, it is their contention that this anatomic feature predisposes an individual to a local "mini compartment syndrome." This tunnel is typically described as being of low compliance and about 1 cm in length; how-

ever, Styf[613] and Pecina et al[600] cite surgical evidence indicating it may extend from 3 to 11 cm in length.

Schon and Baxter[596] feel that the recurrent stretching of the superficial peroneal nerve that ostensibly occurs with chronic ankle sprains is a major factor in causing injury to this nerve. Pecina and colleagues[600] and Lowdon[614] also support the role of stretch injury in causing superficial peroneal nerve injury. They note that the fact that the nerve is fixed makes it vulnerable to the stretch trauma that purportedly occurs with forced, abrupt inversion and plantar flexion at the ankle. Styf and Korner[620] have documented superficial peroneal nerve entrapment as a complication of fasciotomy performed to address chronic anterior compartment syndrome. Schon and Baxter[596] presented a case of a female tennis player who had a superficial peroneal nerve entrapment most likely attributable to compression by a ganglion cyst. Styf reported one case of surgically documented superficial peroneal nerve entrapment by scar tissue following an earlier fasciotomy to decompress the latter nerve.[613]

Clinical Presentation

The typical patient presents with longstanding complaints of pain involving the lateral leg and dorsum of the foot. About one third reportedly have additional complaints of numbness and paresthesias in the latter distribution.[596] As noted previously, approximately 25% have a history of preceding trauma.[613] Ankle sprains are cited as the most common form of possible precipitating injury.[596] Although ankle eversion weakness may be associated with entrapment of this nerve, a review of the literature fails to document weakness as a presenting complaint. It is possible that the presence of chronic compression injury of the superficial peroneal nerve actually predisposes an individual to frequent ankle sprains. The sprains would then be an indirect manifestation of ankle weakness. Exercise usually exacerbates the symptoms. Nocturnal exacerbations are reportedly uncharacteristic.[596]

Patients may present with hypesthesia in the sensory distribution of the superficial peroneal nerve, but Schon and Baxter[596] report that diminished sensation to light touch and pinprick is actually atypical. Patients may also have ankle eversion weakness. A Tinel's sign may be elicited with a light tap over the site where the nerve emerges from the deep fascia, with radiation to the dorsum of the foot and toes. Point tenderness and a palpable bulge or fascial defect may also be detected at the point the nerve pierces the fascia.[596]

Styf[613] advocates the use of three provocative tests to determine if a superficial peroneal entrapment neuropathy is present. One involves applying pressure at the point the nerve emerges from the fascia (at the junction of the middle and distal thirds of the lateral leg) while the patient simultaneously actively dorsiflexes and everts the foot against resistance. The second test involves passive plantar flexion and inversion of the ankle without applying pressure at any point along the nerve course. The final and third test is performed by maintaining passive stretch in plantar flexion and inversion while the nerve is percussed along its subcutaneous route. A positive test with any one of these maneuvers is manifested by the precipitation of pain or paresthesias in the lateral leg or dorsum of the foot and ankle. Styf contends that a positive test suggests superficial peroneal entrapment neuropathy and should prompt further neurophysiologic investigation for confirmation.[613] His diagnostic criteria for diagnosis of the latter nerve entrapment include decreased sensibility and pain over the dorsal aspect of the foot at rest or during exercise, along with at least one positive test among the three aforementioned tests.[613]

Differential Diagnosis

Anteroposterior, lateral, and oblique radiographs of the leg and ankle should be performed to rule out the possibility of a stress fracture or bone tumor. An electrodiagnostic study should be performed to confirm the presence of a superficial peroneal nerve entrapment neuropathy, as well as to exclude the possibility of a more proximal peroneal nerve injury or an L-5 radiculopathy. A measure of the sensory action potentials of the cutaneous branches serving the dorsum of the foot can be performed. This technique is potentially beneficial in identifying entrapment of the superficial peroneal nerve at the point it emerges from the fascia or in isolating more distal involvement of the cutaneous branches.[422] Injection of a local anesthestic, such as 1% xylocaine, along with cortisone in the region of suspected entrapment may be both diagnostic and therapeutic.

Treatment

Avoidance of potential sources of repetitive trauma or stretch injury should be the initial goal. Running activities, in particular, may need to be curtailed or reduced. If signs and symptoms are mild and there is a perceived need to continue such activity by an athlete, then use of supportive foot-

wear or stabilizing foot orthotics may be sufficient to address the problem. Specifically, shoes or foot orthotics providing appropriate arch supports and external heel counters may add sufficient ankle stability to minimize the potential for recurrent ankle sprains. Occasionally, an aircast brace may be warranted if only mild additional ankle mediolateral stability is necessary. If signs and symptoms persist, or if more significant impairment is noted, complete avoidance of exacerbating exercises or sport participation may be necessary. Injection of a local anesthetic with cortisone at the site of suspected entrapment may provide prompt and lasting relief. A trial of NSAIDs may offer some relief.

Extrinsic compression of the cutaneous branches of the superficial peroneal nerve is rarely documented. However, if such is suspected by history and examination, avoidance of overly tight lacing of shoes, excessively tight boots, or other poorly fitting footwear may prove beneficial.

If conservative measures fail, fasciotomy of the fascial tunnel through which the superficial peroneal nerve passes is advocated as a potentially effective means of treating this problem.[600] The aforementioned young woman presenting with bilateral superficial peroneal nerve entrapment neuropathies was successively managed with decompression of the nerve by bilateral, local fasciotomies.[618] Styf[613] studied 21 patients with documented superficial peroneal nerve entrapment. Each had been managed with fasciotomy and neurolysis. Of the 19 reviewed at a later date, 9 reported complete satisfaction, while another 6 had improvement but were unsatisfied since improvement was insufficient to allow performance of their athletic activity. Three of the 19 reported no change and one was actually worse.

Sural Entrapment Neuropathy

Sural entrapment neuropathy is infrequently diagnosed. It reportedly has no particular age or gender distribution.[596] Although entrapment may occur at any point along the course of the nerve, it most commonly occurs at the point the nerve exits the fascia in the leg 20 to 25 cm above the base of the heel.[422,600]

Anatomic Considerations

The sural nerve is a branch of the distal sciatic nerve. It courses through the posterior compartment of the leg and surfaces through the fascia. As previously noted, this occurs at 20 to 25 cm above the base of the heel.[422] It then passes down the lateral aspect of the calf and posterior to the lateral malleolus. It innervates the lateral part of the heel and sole of the foot up to the base of the fifth toe.

Etiology

Sural nerve injury may occur as the result of laceration trauma or compression injury. It reportedly occurs most frequently in runners.[596] The nerve is particularly vulnerable to compression at the point it pierces the fascia. This is a common site for partial or complete excision biopsy of the nerve.[422] Sural nerve compression has also been reported in athletes with fractures of the base of the fifth metatarsal incurred with severe ankle plantar flexion and inversion injuries.[600,621]

Recurrent ankle sprains with accompanying scar formation have been postulated to cause sural entrapment neuropathy.[600] Schon and Baxter[596] report a case of a male middle-distance runner with a two-year history of pain along the Achilles tendon. In the latter case, the sural nerve was found to be tightly adherent to an aponeurotic band behind the Achilles tendon. The patient responded successfully to surgical release of the aponeurosis and lateral displacement of the sural nerve.

Another potential cause of sural nerve entrapment includes ganglion of the peroneal sheath or calcaneal cuboid joint.[596,622] Schon and Baxter[596] also cite a case report by Husson and colleagues in which an individual was found to have entrapment of the sural nerve by myositis ossificans circumscripta at the musculotendinous junction of the Achilles.

Clinical Presentation

Patients typically complain of progressive increase in symptoms of paresthesias and shooting pain down the lateral aspect of the leg with radiation to the lateral aspect of the ankle and foot. They may have a history of recurrent ankle sprains. As previously noted, they may present with a history of running for exercise or sport. An increase in mileage or intensity of running may be temporally related to onset of their symptoms.

There may be decreased sensation to light touch or pinprick in the distribution of the sural nerve, but it is typically limited to an area a few centimeters below the lateral malleolus.[422] A Tinel's sign may be elicited with percussion at the site of entrapment. This sign most commonly occurs at the nerve's exit from its fascia. It is characteristically manifested by radiating paresthesias beyond the area of sensory loss to the lateral aspect of the foot.[422] After nerve

biopsy, permanent sensory loss or persistent painful paresthesias are potential complications.

Differential Diagnosis

Electrodiagnostic studies, specifically a sural sensory NCS, may be helpful in confirming the presence of the entrapment neuropathy and in excluding the possibility of a sciatic nerve lesion, an S-1 radiculopathy, or a peripheral neuropathy. Radiographic evaluation of the leg, ankle, or foot may be warranted if pain is the sole or primary complaint. The latter studies would serve to exclude the possibility of a stress fracture or bone tumor.

Treatment

Cessation or modification of a suspected precipitating activity, such as running, should be recommended. The use of orthotics or bracing has not been advocated in the literature, presumably because it has limited application in this form of entrapment neuropathy. A trial of NSAIDs is warranted. If unresponsive to conservative management, surgical exploration and decompression at the site of entrapment merits consideration.

Digital Neuropathies

Digital neuropathies secondary to interdigital neuromas are relatively common, particularly in the athletic population. Runners and dancers appear to be the most susceptible to this problem.[596,600] The elderly population and women appear to manifest this form of neuropathy with greater frequency than others. In the former, it may simply reflect the effects of chronic repetitive nerve irritation that may come with increasing age, and in women presumably due to wearing of high heels and tight, pointed shoes.[596]

Morton's toe neuroma is a specifically described entrapment of an interdigital nerve. This interdigital neuroma is typically described as occurring between the third and fourth digits of the foot, although it may also occur in the second web space.[422,596]

Anatomic Considerations

The medial plantar nerve is the larger of the two terminal branches of the tibial nerve. In its distal segment, it divides into four plantar digital nerves. The plantar digital nerves are distributed to the plantar aspect of the medial three and one-half toes. In addition, they also innervate the distal aspect of the extensor surface of these toes. The first plantar digital nerve innervates the medial surface of the plantar aspect of the great toe, while the other three provide nerve supply to adjacent sides of the great, second, third, and fourth toes, respectively. The second plantar nerve also supplies the first lumbrical muscle.[617]

The lateral plantar nerve divides into a superficial and a deep branch in its distal segment. The superficial branch then divides into two plantar digital nerves. One of the latter nerves supplies the lateral side of the little toe, the flexor digiti minimi muscle, and the two interosseus muscles of the fourth space. The other digital nerve innervates the adjacent sides of the fourth and fifth toes, as well as the distal part of the extensor surface of the corresponding distal phalanges. The deep branch of the lateral plantar supplies the interossei of the first three spaces, the two or three lateral lumbricals, and the adductor hallucis muscle.[617]

Etiology

Morton is noted to have described the condition of Morton's toe neuroma in 1876 and felt that it was caused by pinching of the lateral plantar nerve. However, it took until the 1940s before it was recognized that the signs and symptoms were actually due to a neuroma of the interdigital nerve secondary to entrapment.[422] Review of the literature by Guiloff and colleagues[623] indicate that an early investigator felt that this nerve lesion was due to ischemic insult, noting that a number of investigators since that earlier time support the contention that it is, in fact, an entrapment neuropathy.

The sharp anterior edge of the deep plantar fascia forms the intermetatarsal ligament and it is this structure that is felt to cause focal injury to a digital nerve.[422] As previously noted, the toes are forced into dorsiflexion at the metatarsophalangeal joint when high heels and tight, pointed shoes are worn. This posture is felt to force the nerve against the intermetatarsal ligament with resulting compression neuropathy. The repetitive, extreme dorsiflexion that occurs at the metatarsophalangeal joints of dancers and runners presumably causes repetitive microtrauma to the plantar nerve when it is repeatedly forced up against the distal edge of the intermetatarsal ligament.[596] Repetitive or sustained squatting is also associated with excessive dorsiflexion at the metatarsophalangeal joints and thus, is considered a possible cause of plantar nerve entrapment neuropathy.[422]

It has also been suggested that runners with a hypermobile first metatarsal are more susceptible to developing calluses under the second and third metatarsal heads. A neuroma may develop as a consequence of the pressure created by the calluses.[596] Schon and Baxter[596] feel excessive pronation may result in increased dorsiflexion of the third metatarsal relative to the fourth. As a consequence, the nerve is felt to be more vulnerable to impingement in the presence of hyperpronation.

Clinical Presentation

Burning pain is characteristic of digital neuropathies due to interdigital neuromas. The pain is typically in the plantar region and exacerbated with prolonged standing or running, particularly sprinting or long-distance running.[422,596] Some relief is typically obtained with rest, gentle foot massage, and elevation of the affected lower extremity. Aching, cramping, or sharp pain with radiation to the toes has also been recorded.[596] Others have reported intermittent pain described as a stabbing, shooting, or pricking discomfort "like needles." Still others complain of a piercing, hot pain.[623]

Point tenderness with palpation over the metatarsal head or more typically with pressure in the space between the metatarsal heads, usually between the third and fourth toes, is a reproducible finding. Less frequently, the tenderness may be elicited with pressure between the second and third toes.[422,596] Focal swelling may be detected at the site of tenderness. Exquisite pain may be elicited by squeezing the metatarsal heads together.[596] Other provocative tests to reproduce symptoms also include passive dorsiflexion of the toes along with firm palpation of the intermetatarsal space and squatting by the patient. There may be a hypermobile first metatarsal and calluses under the second and third metatarsal head, conditions that have been suggested to predispose individuals to developing interdigital neuromas. Although not consistently present, impaired sensation in the medial half of the fourth toe may be present; however, other objective neurologic deficits are reportedly not typically found.[422]

Differential Diagnosis

Stress fractures, arthritic disease, and bone tumors should be excluded with appropriate radiographs. Metatarsal capsulitis due to strain, contusion, or a subluxed metatarsophalangeal joint, as well as adventitious bursas, sesamoiditis, plantar fasciitis, and peripheral neuropathy may mimic some of the signs and symptoms found with interdigital neuromas; however, an appropriate history and careful physical examination will generally exclude these conditions.

Electrodiagnosis

An electrophysiologic technique for assessing the integrity of the interdigital nerves has been developed by Oh and colleagues.[624] It includes electrical stimulation of the great toe and fifth toe with ring electrodes. The recording electrode is provided by placement of a near-nerve needle at the ankle. This technique permits simultaneous stimulation of two branches of the interdigital nerves.[422,624] Dawson and colleagues[422] point out that decrease in the amplitude of the sensory action potential is characteristic of an affected interdigital nerve. Guiloff applied a similar electro-physiologic technique to assess patients presenting with Morton's metatarsalgia. They concluded that this form of assessment with the technique they employed was not advisable for routine testing of such patients nor a replacement for a sound clinical examinaton.[623] Katz[625] acknowledges that such electrophysiologic studies are available, but notes that success in obtaining consistent results is variable.

Treatment

Conservative treatment for interdigital neuromas initially includes avoidance of the physical activity presumed to precipitate or exacerbate the symptoms. It also includes prescription of an NSAID, local massage, as well as shoe modifications and the use of foot orthotics. Typically, metatarsal pads and shoes with an ample toe-box are prescribed. At times, extra-depth shoes are employed. Use of high-heels and pointed shoes are discouraged. A trial injection of cortisone with local anesthestic occasionally offers lasting relief. However, Lillich and Baxter[626] point out that use of metatarsal pads and local anesthetic and cortisone injections are successful in relieving symptoms less than 50% of the time.

If a patient's symptoms are refractory to the aforementioned conservative measures, then excision of the neuroma in toto is advocated. Dawson and colleagues[422] report that this particular surgical intervention has a high success rate with the primary residual deficit limited to the sensory distribution of the excised nerve. As previously noted however, the sharp anterior edge of the deep plantar fascia forms the intermetatarsal ligament and it

is this structure that is felt to cause focal injury to a digital nerve.[422] This constitutes the rationale for releasing the latter ligament without excision of the neuroma as advocated and practiced by Gauthier. He reportedly obtained successful results in over 200 patients managed in this fashion.[422,627]

CONCLUSION

Traumatic injuries to the peripheral nerves pose complex challenges to the military physician. While the etiology and specific pathology may be wide ranging, it is not uncommon for even mild injuries to be severely disabling, if not properly diagnosed and treated. Comprehensive management requires an extremely detailed assessment, and may require both electrodiagnostic testing and surgical exploration.

Treatment of nerve injuries must be multidisciplinary and must consider all aspects of the inherent disability. Pain control is of primary importance. Little else in terms of functional restoration will be accomplished until pain is brought down to tolerable levels. As neural regeneration may take months to become clinically evident, even after surgical intervention, protection of the affected area from complications of disuse and immobility are essential. Rehabilitation efforts, including the enhancement of strength, flexibility, sensory discrimination, and dexterity, must commence early in the treatment course to harness and improve residual function and to set the stage for more complete and successful recovery as reinnervation occurs.

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