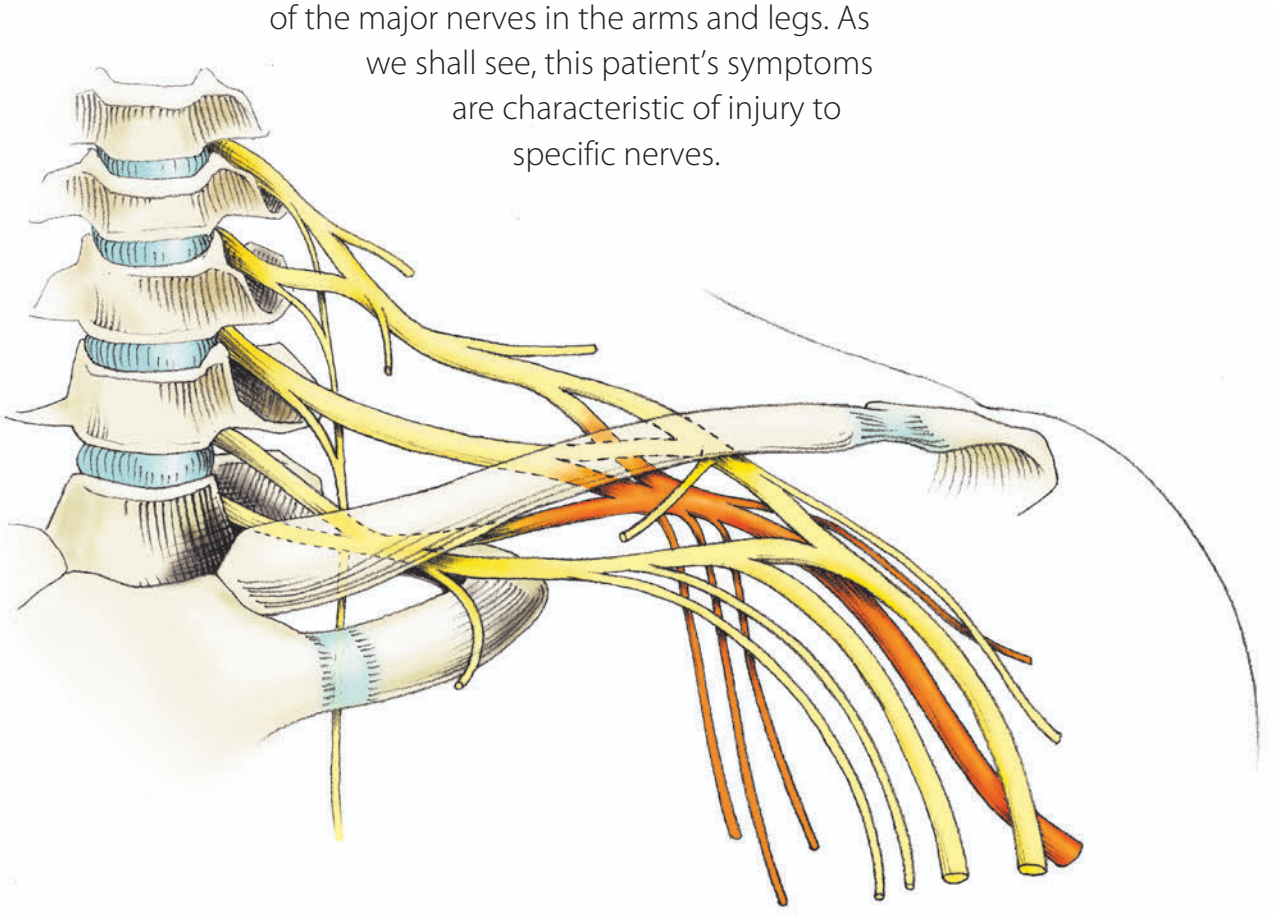
Chapter 9

Major Plexuses and Peripheral Nerves

A 3-week-old infant was not moving her left arm normally. She was a large baby and had endured significant traction on her left shoulder during delivery. Her left arm had decreased tone and appeared internally rotated. She was able to extend her left arm at the elbow and could open and close her hand, but she could not abduct her left arm at the shoulder or flex it at the elbow. The left biceps reflex was absent.

In this chapter, we will learn the sensory and motor functions of the major nerves in the arms and legs. As we shall see, this patient's symptoms are characteristic of injury to specific nerves.



ANATOMICAL AND CLINICAL REVIEW

In this chapter, we will discuss the functions of the brachial plexus, lumbosacral plexus, and nerve branches that arise from them. Knowledge of the motor and sensory territories of the spinal nerve roots (see Chapter 8), major plexuses, and peripheral nerves can be very useful clinically in identifying specific nerve lesions and in distinguishing them from lesions of the central nervous system. Disorders specifically affecting motor neurons were discussed in Chapter 6 (see KCC 6.7). Nerve roots and radiculopathies were discussed in Chapter 8, where we also introduced peripheral nerve and neuromuscular disorders in general (see KCC 8.1). Here we will discuss the most important peripheral nerves in the upper and lower extremities, as well as common localized plexus and nerve syndromes.

Brachial Plexus and Lumbosacral Plexus

The **brachial plexus** is formed by nerve roots arising from the cervical enlargement at **C5**, **C6**, **C7**, **C8**, and **T1** (Figure 9.1). These nerve roots provide the major sensory and motor innervation for the upper extremities. The nerves of the

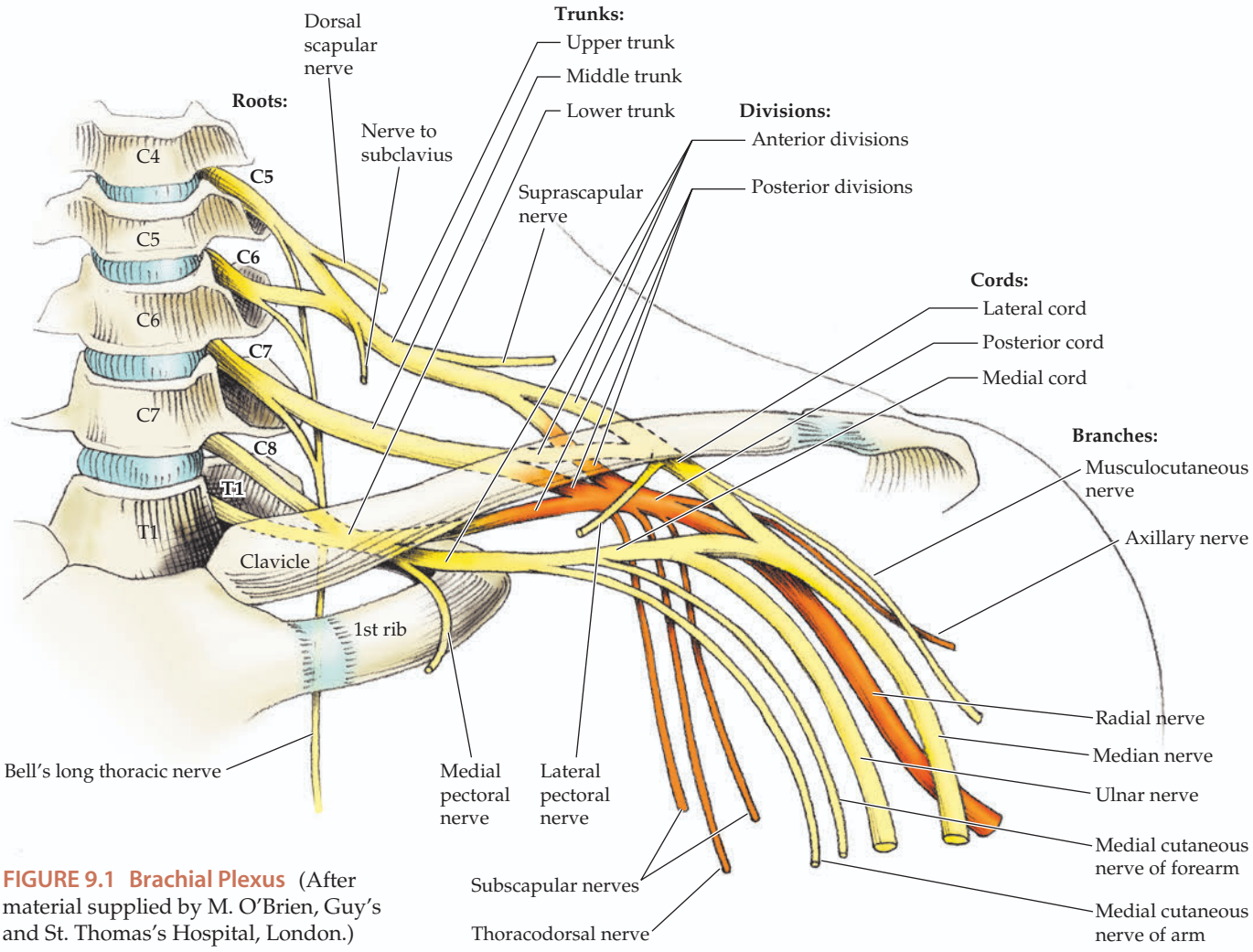


FIGURE 9.1 Brachial Plexus (After material supplied by M. O'Brien, Guy's and St. Thomas's Hospital, London.)

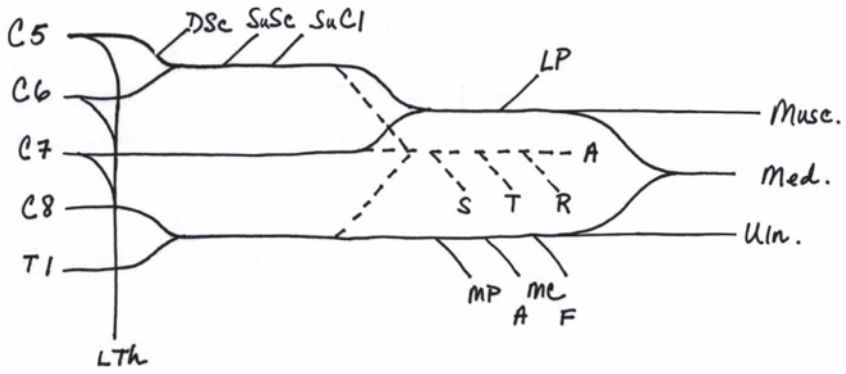


FIGURE 9.2 Brachial Plexus: Simplified Schematic Lth = Bell's long thoracic nerve; DSc = dorsal scapular nerve; SuSc = suprascapular nerve; SuCl = nerve to subclavius; LP = lateral pectoral nerve; A = axillary nerve; R = radial nerve; T = thoracodorsal nerve; S = subscapular nerve; MP = medial pectoral nerve; MC,A = medial cutaneous nerve of arm; MC,F = medial cutaneous nerve of forearm; Musc. = musculocutaneous nerve; Med. = median nerve; Uln. = ulnar nerve.

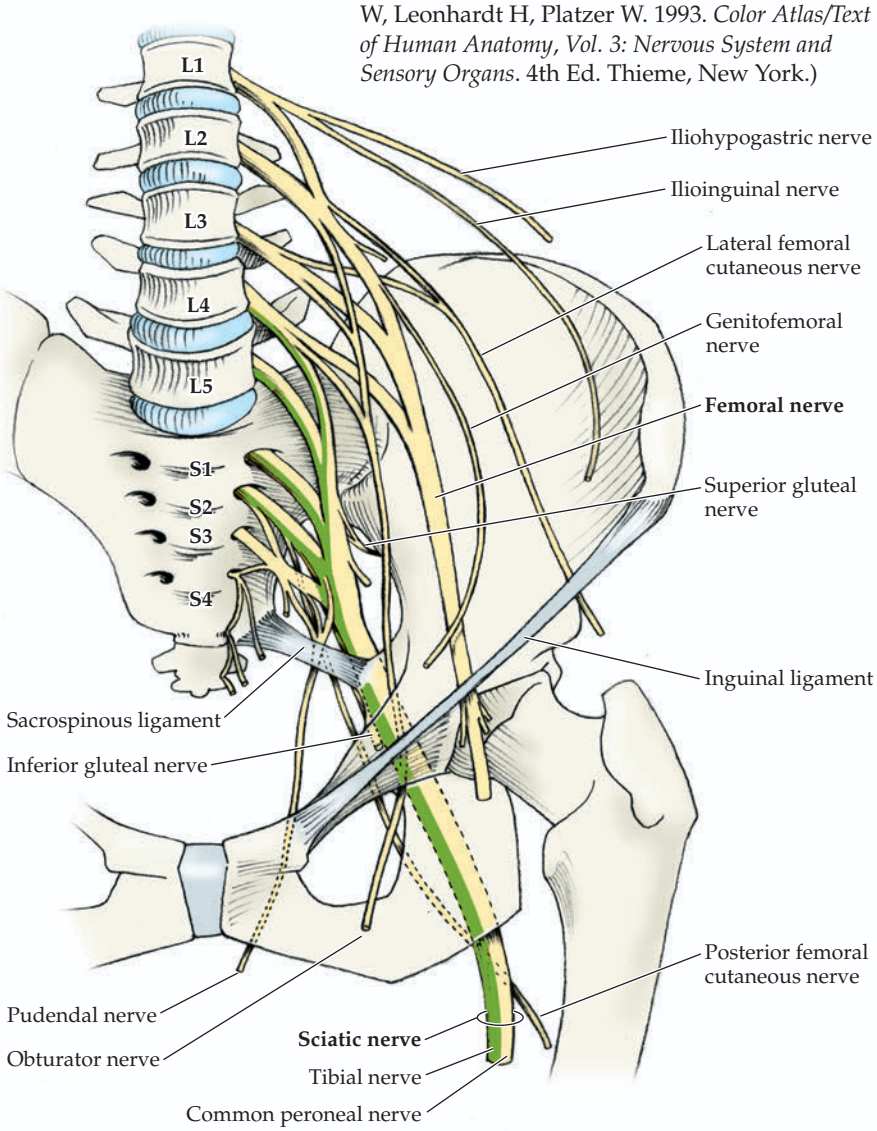
brachial plexus are so clinically important that it is worth committing the structure of the brachial plexus to memory. A simplified schematic can be helpful in this regard (Figure 9.2). The parts of the brachial plexus can be remembered by this mnemonic: Robert (**r**oots) Taylor (**t**runks) Drinks (**d**ivisions) Cold (**c**ords) Beer (**b**ranches). It is also important to know the muscles innervated by each of the nerve branches (see Table 8.1). The five most clinically important nerve branches arising from the brachial plexus are the radial, median, ulnar, musculocutaneous, and axillary nerves (see the next section). A few more mnemonics may be helpful: The nerve branches of the posterior cord can be memorized with the mnemonic **STAR** or **ARTS** (**A**xillary, **R**adial, **T**horacodorsal, **S**ubscapularis). The muscles innervated by the musculocutaneous nerve are represented by the mnemonic **BBC** (**B**iceps, **B**rachialis, **C**oracobrachialis).

The **lumbosacral plexus** arises from **L1**, **L2**, **L3**, **L4**, **L5**, **S1**, **S2**, **S3**, and **S4** at the lumbosacral enlargement and provides innervation to the lower extremities and pelvis (Figure 9.3). Once again, a simplified schematic may be helpful (Figure 9.4). The muscles innervated by each of the lumbosacral nerve branches should be reviewed (see Table 8.1). The most clinically important nerve branches arising from the lumbosacral plexus are the femoral, obturator, sciatic, tibial, and peroneal nerves, as will be described shortly.

There is also a plexus formed by branches of CN XII and C1 through C5 called the **cervical plexus**, which supplies mainly the neck muscles. We will not discuss this plexus further except to mention that the **phrenic nerve**, which supplies the diaphragm, arises from **C3**, **C4**, and **C5**.

Regions of sensory innervation by cutaneous nerve branches are shown in

FIGURE 9.3 Lumbosacral Plexus (After Kahle W, Leonhardt H, Platzer W. 1993. *Color Atlas/Text of Human Anatomy, Vol. 3: Nervous System and Sensory Organs*. 4th Ed. Thieme, New York.)



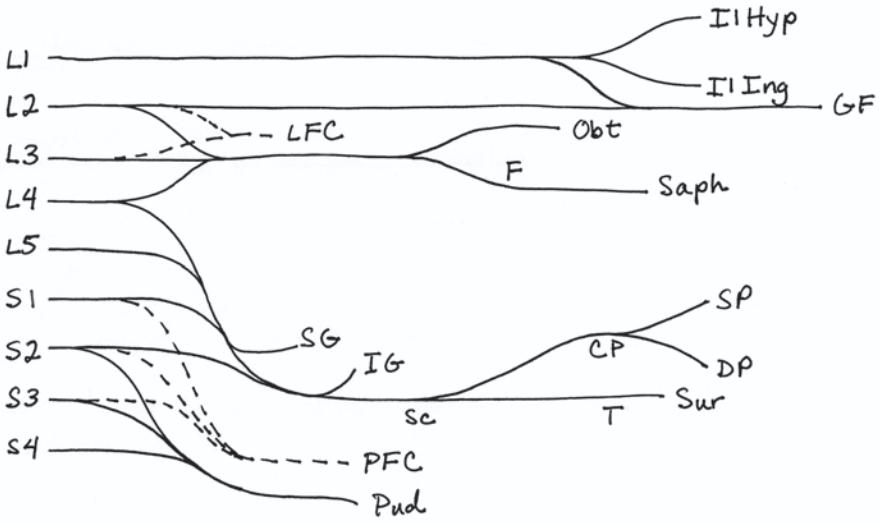
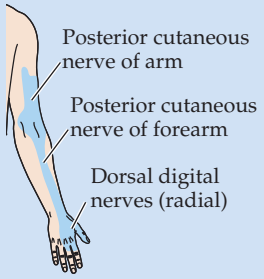
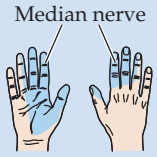
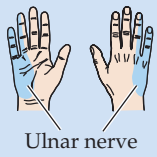
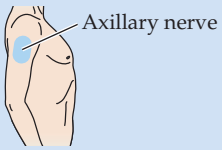
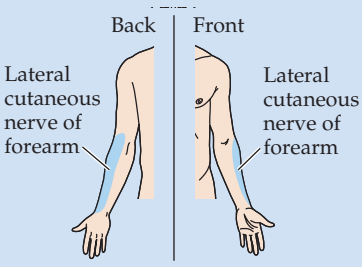


FIGURE 9.4 Lumbosacral Plexus: Simplified Schematic IlHyp = iliohypogastric nerve; IIIng = ilioinguinal nerve; GF = genitofemoral nerve; LFC = lateral femoral cutaneous nerve; F = femoral nerve; Obt = obturator nerve; Saph = saphenous nerve; SG = superior gluteal nerve; IG = inferior gluteal nerve; Sc = sciatic nerve; CP = common peroneal nerve; SP = superficial peroneal nerve; DP = deep peroneal nerve; T = tibial nerve; Sur = sural nerve; PFC = posterior femoral cutaneous nerve; Pud = pudendal nerve.

TABLE 9.1 Five Important Nerves in the Arm

NERVE	MOTOR FUNCTIONS	REGION OF SENSORY LOSS WITH NEUROPATHY
Radial	Extension at all arm, wrist, and proximal finger joints below the shoulder; forearm supination; thumb abduction in plane of palm	
Median	Thumb flexion and opposition, flexion of digits 2 and 3, wrist flexion and abduction, forearm pronation	
Ulnar	Finger adduction and abduction other than thumb; thumb adduction; flexion of digits 4 and 5; wrist flexion and adduction	
Axillary	Abduction of arm at shoulder beyond first 15°	
Musculocutaneous	Flexion of arm at elbow, supination of forearm	

REVIEW EXERCISE

1. Practice drawing the simplified schematic of the brachial plexus shown in Figure 9.2.
2. Practice drawing the simplified schematic of the lumbosacral plexus shown in Figure 9.4.

Figure 9.5. The actual area of sensory loss following a nerve injury is somewhat smaller than the territories shown because of overlap from adjacent nerves. Compare this figure to the dermatomal sensory distribution of nerve roots shown in Figure 8.4.

Simplification: Five Nerves to Remember in the Arm

It is most clinically important to be familiar with the functions of the **radial**, **median**, **ulnar**, **axillary**, and **musculocutaneous nerves** in the arm. Table 9.1 summarizes the motor and sensory functions of these nerves, and they are demonstrated on **neuroexam.com Videos 54** and **55**. Additional details are found in Table 8.1. In general, the radial nerve is important for extension of all joints in the arm and proximal fingers, the median nerve is important for the thumb side of the hand and wrist, and the ulnar nerve is important for the pinky side of the hand and wrist. Note that (1) the sensory terri-

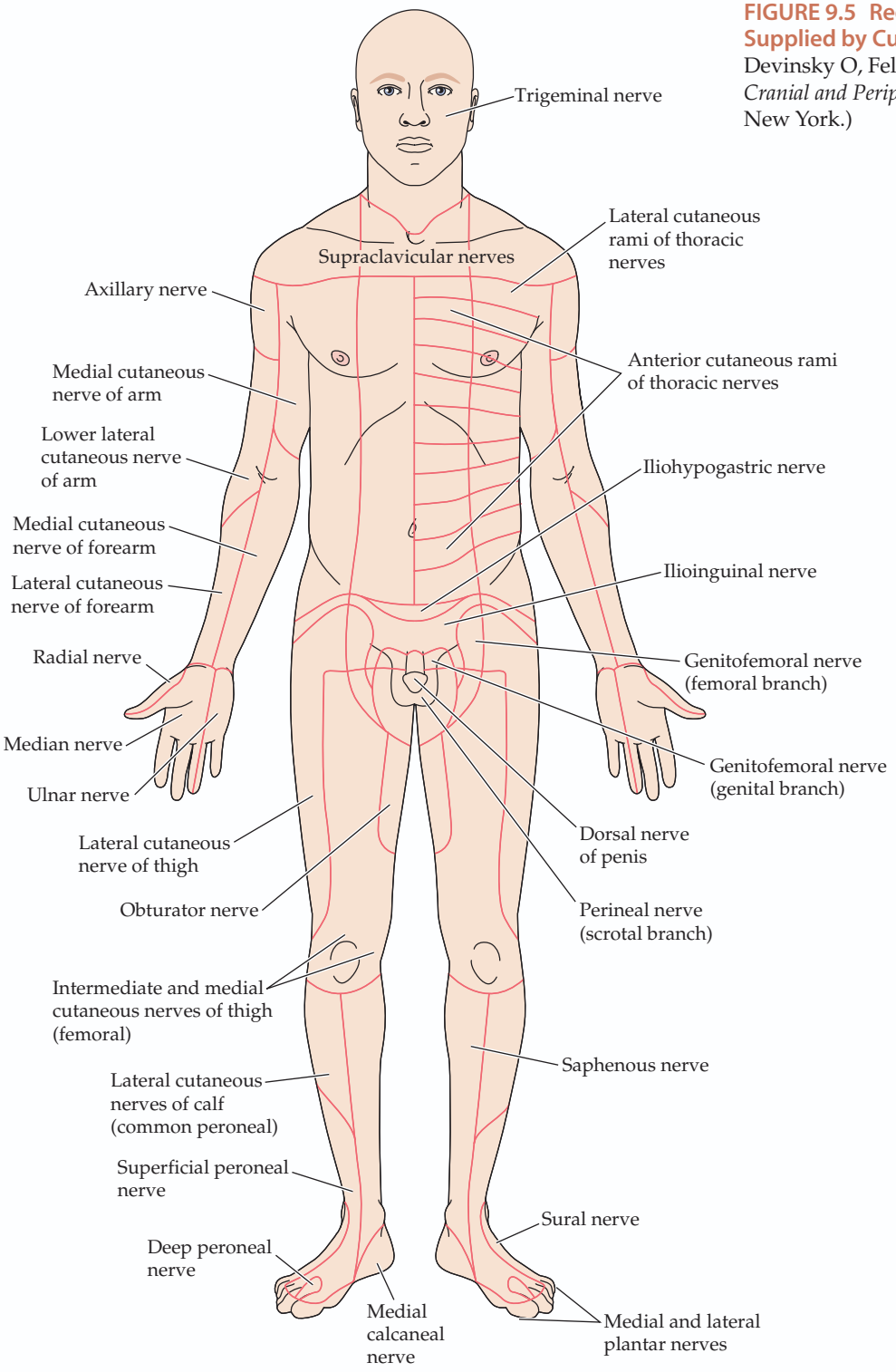


FIGURE 9.5 Regions of Sensory Innervation Supplied by Cutaneous Nerve Branches (After Devinsky O, Feldmann E. 1988. *Examination of the Cranial and Peripheral Nerves*. Churchill Livingstone, New York.)

tories shown in Table 9.1 are smaller than in Figure 9.5 because adjacent nerves overlap somewhat, and (2) Table 9.1 shows regions of *sensory loss* with nerve injury rather than the whole region innervated by the nerve. Finger flexion is best tested at the distal interphalangeal joints (see **neuroexam.com Video 55**), where the flexor digitorum profundus (median nerve for digits 2 and 3; ulnar nerve for digits 4 and 5) acts without significant contributions from other muscles (Table 9.2).

TABLE 9.2 Muscles Contributing to Flexion and Extension at Finger Joints Other than the Thumb

		FLEXION[a]			EXTENSION[a]		
MUSCLE	NERVE	MCP	PIP	DIP	MCP	PIP	DIP
Flexor digitorum profundus	Median (2nd, 3rd digits), ulnar (4th, 5th digits)	X	X	**X**			
Flexor digitorum superficialis	Median	X	**X**				
Flexor digiti minimi (fifth digit)	Ulnar	X					
Lumbricals	Median (2nd, 3rd digits), ulnar (4th, 5th digits)	**X**			**X**	**X**	
Palmar and dorsal interossei	Ulnar	X			X	X	
Extensor digitorum	Radial				**X**	X	X
Extensor indicis (second digit)	Radial				X	X	X
Extensor digiti minimi (fifth digit)	Radial				X	X	X

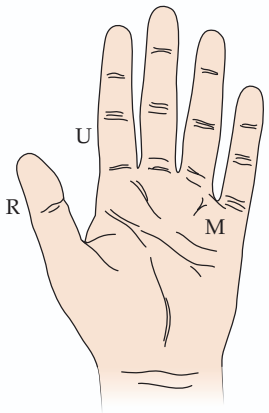
Note: **Bold** text indicates the most important muscles.

[a]MCP, metacarpophalangeal joint; PIP, proximal interphalangeal joint; DIP, distal interphalangeal joint.

Simplification: Three Nerves Acting on the Thumb


MNEMONIC

Different thumb muscles are innervated by the radial, ulnar, and median nerves. It is easiest to remember these by the mnemonic RUM (**R**adial, **U**lnar, **M**edian), as shown in Figure 9.6. Thumb abduction in the plane of the palm (abductor pollicis longus) is mediated by the **R**adial nerve, adduction (adductor pollicis) by the **U**lnar nerve, and opposition (opponens pollicis) and flexion (flexor pollicis longus and superficial head of the flexor pollicis brevis) by the **M**edian nerve. It should also be recalled that thumb abduction perpendicular to the palm (see Table 3.4; **neuroexam.com Video 55**) is mediated by the abductor pollicis brevis, which is innervated by the median nerve after it passes through the carpal tunnel.




MNEMONIC

FIGURE 9.6 Three Nerves Acting on the Thumb The radial nerve abducts the thumb in the plane of the palm. The ulnar nerve adducts the thumb in the plane of the palm. The median nerve opposes the thumb. Note also that the abductor pollicis brevis (median nerve) abducts the thumb perpendicular to the plane of the palm (not shown).

Intrinsic and Extrinsic Hand Muscles

The **intrinsic hand muscles** include the muscles of the **thenar eminence** at the base of the thumb (opponens pollicis, abductor pollicis brevis, flexor pollicis brevis, adductor pollicis), the muscles of the **hypothenar eminence** at the base of the pinky finger (opponens digiti minimi, abductor digiti minimi, flexor digiti minimi), the **lumbricals**, and the **interossei**. Intrinsic hand muscles are innervated by the ulnar nerve, except for the LOAF (**L**umbricals I and II, **O**pponens pollicis, **A**bductor pollicis brevis, **F**lexor pollicis brevis—superficial head) muscles, which are innervated by the median nerve after it passes through the carpal tunnel. All intrinsic hand muscles are supplied by **C8** and **T1** (see Table 8.1).

In addition to the intrinsic hand muscles, **extrinsic muscles** in the forearm are important for finger movements (see Table 8.1). Intrinsic and extrinsic muscles contributing to flexion and extension at finger joints other than the thumb are summarized in Table 9.2. As we have already mentioned, it should be clear from this table that the flexor digitorum profundus (median nerve for digits 2 and 3; ulnar nerve for digits 4 and 5) is best tested at the distal interphalangeal joints, since other muscles participate in flexion at the other joints. Similarly, the extensor digitorum (radial nerve and C7) is best tested at the metacarpophalangeal joints (see Table 9.2). This is because other muscles, most notably the lumbricals, are predominantly responsible for finger extension at the proximal and distal interphalangeal joints (median nerve for 2nd and 3rd digits; ulnar nerve for 4th

and 5th digits). See Tables 8.1 and 9.1 for muscles contributing to finger adduction, abduction, and opposition. Note, for example, that the **palmar interossei** adduct the fingers, while the **dorsal interossei** abduct them.

Simplification: Five Nerves to Remember in the Leg

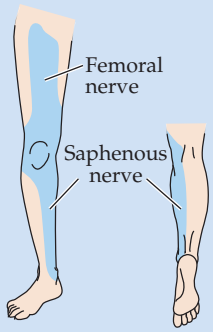
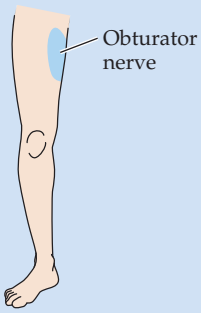
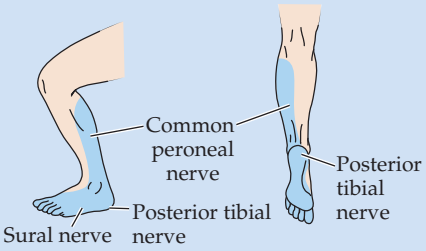
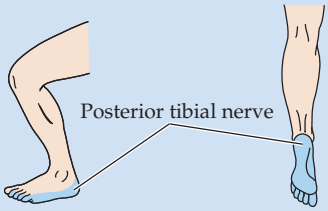
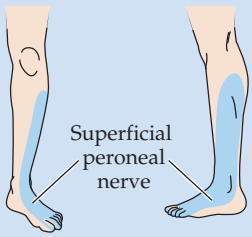
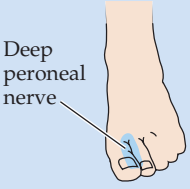
It is most clinically important to be familiar with the functions of the **femoral**, **obturator**, **sciatic**, **tibial**, and **peroneal nerves** in the leg. Table 9.3 summarizes the motor and sensory functions of these nerves, and they are demonstrated on **neuroexam.com Videos 56** and **57**. Table 8.1 provides additional details. Note again that the sensory territories shown in Table 9.3 are smaller than in Figure 9.5, since here we are interested in regions of sensory loss.

The tibial and common peroneal nerves are the two most important branches of the sciatic nerve. The **hamstring muscles** (semitendinosus, semimembranosus, and biceps femoris) are innervated by the sciatic nerve itself before it divides into the tibial and common peroneal nerves. The common peroneal nerve divides further to give rise to the **superficial** and **deep peroneal nerves** (see Figures 9.3 and 9.4; Table 9.3).

REVIEW EXERCISE

1. Turn back to Tables 3.4–3.6 in Chapter 3, where we discussed strength and reflex testing (see also **neuroexam.com Videos 54–58**). In these tables, cover all columns except for the left-most column. For each action or reflex, list the appropriate muscle, nerves, and nerve roots being tested (refer to Table 8.1).
2. In Tables 9.1 and 9.3, cover the columns showing the regions of sensory loss and sketch the region of sensory loss for each of the five nerves in the arm and the leg.

TABLE 9.3 Important Nerves in the Leg

NERVE	MOTOR FUNCTIONS	REGION OF SENSORY NERVE LOSS WITH NEUROPATHY
Femoral	Leg flexion at the hip, leg extension at the knee	
Obturator	Adduction of the thigh	
Sciatic	Leg flexion at the knee (see also tibial and peroneal nerves, in column at left)	
Tibial	Foot plantar flexion and inversion, toe flexion	
Superficial peroneal	Foot eversion	
Deep peroneal	Foot dorsiflexion, toe extension	

KEY CLINICAL CONCEPT

9.1 COMMON PLEXUS AND NERVE SYNDROMES

In Chapter 8, we introduced the general causes of neuropathy (see KCC 8.1). Here we focus on clinical localization of common mononeuropathies and plexus syndromes caused mainly by mechanical factors or diabetes. Note that in some cases trauma to a limb produces nerve injury, with the exact mechanism of such injury remaining unclear.

Upper-Extremity Nerve Injuries

BRACHIAL PLEXUS, UPPER TRUNK INJURY (ERB–DUCHENNE PALSY) Common causes include traction on an infant's shoulder during a difficult delivery as well as motorcycle accidents. Damage to the upper trunk of the brachial plexus (see Figures 9.1 and 9.2) causes loss of function in C5- and C6-innervated muscles, resulting in prominent weakness of the deltoid, biceps, infraspinatus, and wrist extensors (see Table 8.1). The arm assumes a characteristic "bellman's tip" or "waiter's tip" pose, held at the side, internally rotated, and with the wrist flexed (Figure 9.7). Finger and hand movements are relatively spared. Most infants recover fully, but prognosis depends on the severity of the injury. Surgical repair of the plexus is occasionally pursued. Differential diagnosis includes traumatic avulsion of the C5 and C6 nerve roots, or other causes of a C5 and C6 radiculopathy (see KCC 8.3).

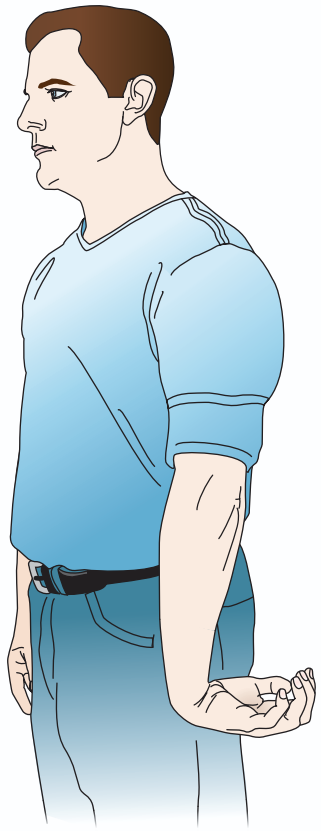


FIGURE 9.7 "Bellman's," or "Waiter's Tip," Pose Assumed in Upper-Plexus Lesions

BRACHIAL PLEXUS, LOWER TRUNK INJURY (KLUMPKE'S PALSY) Common causes include upward traction produced by grabbing a branch during a fall from a tree, thoracic outlet syndrome, and Pancoast's syndrome. Damage to the lower trunk of the brachial plexus (see Figures 9.1 and 9.2) causes weakness of C8- and T1-innervated muscles, resulting in hand and finger weakness, and atrophy of the hypothenar muscles, together with sensory loss on the ulnar aspect of the hand and forearm. If the T1 nerve root is damaged proximal to the sympathetic trunk (see Figure 6.13), there may also be an associated Horner's syndrome (see KCC 13.5; see also Figure 13.10). Differential diagnosis includes ulnar neuropathy or C8–T1 radiculopathies.

In **thoracic outlet syndrome**, the lower brachial plexus is compressed as it passes between the clavicle and the first rib (see Figure 9.1). Symptoms may be increased by raising and external rotation of the arm, which may also decrease brachial arterial pulses. EMG and X-rays (looking for a cervical rib or other bony abnormalities) are important diagnostically. Treatments include exercises to strengthen shoulder muscles and surgical decompression for well-documented refractory cases. Treatment for equivocal cases has been a source of controversy.

In **Pancoast's syndrome**, an apical lung tumor (usually non-small cell carcinoma) extends into the lower brachial plexus. In addition to lower-plexus signs (sometimes including Horner's syndrome), the recurrent laryngeal nerve is occasionally involved as it loops downward into the thorax, producing hoarseness (see KCC 12.8). Ultimately, the entire brachial plexus may be invaded, producing a flail, insensate upper extremity.

AXILLARY NEUROPATHY Dislocation or fracture of the proximal humerus can compress the axillary nerve, causing deltoid weakness and numbness in the shoulder (see Table 9.1). Differential diagnosis includes C5 radiculopathy (although axillary neuropathy does not involve the biceps, while C5 radiculopathy does).

BRACHIAL PLEXITIS (BRACHIAL NEURITIS, PARSONAGE-TURNER SYNDROME) This is a disorder of unknown, possibly inflammatory cause with onset in adult-

hood and slightly more commonly in males, of burning shoulder or lateral neck pain followed by weakness of muscles innervated by the brachial plexus. Most patients recover fully within 6 to 12 weeks of onset.

RADIAL NEUROPATHY Common causes include sleeping with the arm slung over a park bench ("Saturday night palsy"), compression in the axilla by improper crutch use ("crutch palsy"), or fracture of the humerus damaging the nerve as it travels in the spiral groove. There is weakness of all extensors of the arm, hand, and fingers below the shoulder, weakness of forearm supination, loss of the triceps reflex, and sensory loss in a radial nerve distribution (see Table 9.1). A **wrist drop** is often present (Figure 9.8A). The triceps may be spared, depending on how distal the lesion is in the arm. The posterior interosseous nerve is a purely motor branch of the radial nerve. Trauma or entrapment of the posterior interosseous nerve results in weakness of radial nerve-innervated muscles, sparing the triceps (see Table 8.1) and with no sensory loss. Tight wrist bands or handcuffs can sometimes compress the superficial branch of the radial nerve, causing isolated sensory loss in the dorsal lateral hand called **cheiralgia paresthetica**, or handcuff neuropathy.

MEDIAN NEUROPATHY Causes include sleeping with a lover's head resting on the upper arm ("honeymooner's palsy"). Fractures of the humerus or distal radius can occasionally injure the median nerve. In addition, entrapment (see KCC 8.1) can occur as the nerve passes through the pronator teres muscle in the forearm. There is weakness of wrist flexion and abduction, opposition of the thumb, and flexion of the second and third digits, together with sensory loss in a median nerve distribution (see Table 9.1). In an attempt to make a fist, the hand may assume a "preacher's hand" or "orator's hand" pose (Figure 9.8B).

CARPAL TUNNEL SYNDROME This common entrapment syndrome is caused by compression of the median nerve as it passes together with the tendons of the hand under the flexor retinaculum on the flexor surface of the wrist. It is seen most often in women over age 30 and can be associated with activities such as typing or housepainting that can cause repetitive stress injury, edema, and inflammation in the wrist. Other causes include pregnancy, oral contraceptives, hypothyroidism, arthritis, wrist fracture, acromegaly, uremia, diabetes, and amyloidosis. Recall that after the median nerve passes through the carpal tunnel, it innervates the LOAF (**L**umbricals I and II, **O**pponens pollicis, **A**bductor pollicis brevis, **F**lexor pollicis brevis—superficial head) muscles. The best muscle to test in suspected carpal tunnel syndrome is the abductor pollicis brevis, which abducts the thumb perpendicular to the plane of the palm (see **neuroexam.com Video 55**), although thumb flexion and opposition (see Figure 9.6) may also be weak. Symptoms often include sensory loss in the first, second, and third digits, as well as prominent paresthesias (see KCC 7.1) that are most bothersome at night and can sometimes radiate into the upper arm. Patients often report shaking the hand to try and relieve symptoms (flick sign). In advanced cases, thenar atrophy may be present. Wrist flexion, flexion of the second and third digits, and sensation over the thenar eminence are typically spared, since nerves providing these functions branch off proximal to the carpal tunnel.

Differential diagnosis of carpal tunnel syndrome includes radiculopathy of C6 and C7 or compression of the median nerve proximal to the carpal tunnel, although these conditions usually include more proximal symptoms. Tests to provoke paresthesias in a median nerve distribution may be helpful, but they are not very sensitive or specific. These include Tinel's sign, in which the median nerve is percussed in the carpal tunnel, and Phalen's sign, in which the dorsal surfaces of the hands are pressed together, flexing the wrists for about

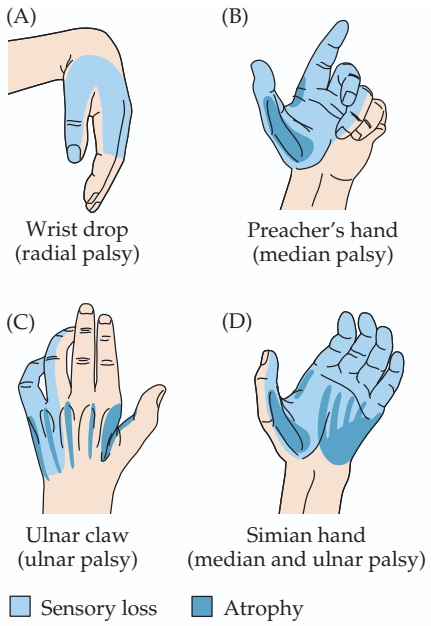


FIGURE 9.8 Classic Hand Poses in Lesions of the Radial, Median, and Ulnar Nerves (A) "Wrist drop" in radial nerve lesions, resulting from weakness of wrist extensors. (B) "Preacher's hand," or "orator's hand," in proximal median nerve lesions, resulting from weakness of the hypothenar and first two flexor digitorum profundus muscles when the patient is asked to make a fist (flex fingers). (C) "Ulnar claw," or "benediction posture," in chronic ulnar nerve lesions, resulting from weakness of the lumbricals (see Table 9.2) for digits 4 and 5 when the patient is asked to extend the fingers. (D) "Simian hand" in chronic median plus ulnar nerve lesions, resulting from thenar and hypothenar atrophy and loss of thumb opposition.


MNEMONIC

1 minute. Treatments include immobilization by a removable wrist splint, steroid injections, and surgical decompression of the carpal tunnel.

ULNAR NEUROPATHY The medial epicondyle of the elbow carries the name "funny bone" because mild trauma to the ulnar nerve as it passes over the elbow in the **ulnar groove** (between the olecranon and medial epicondyle) produces transient paresthesias (see KCC 7.1) in an ulnar distribution. A common cause of ulnar neuropathy is entrapment (see KCC 8.1) at the elbow in the cubital canal, which lies in the region of the ulnar groove. This condition is sometimes called tardy ulnar palsy, a delayed result of a posttraumatic, degenerative, or congenital increased carrying angle at the elbow. The ulnar nerve can also be damaged acutely by fractures of the medial epicondyle, or it may be compressed by a habit of resting the elbows on a hard table.

Findings include weakness of wrist flexion and adduction, finger adduction and abduction, and flexion of the fourth and fifth digits, together with sensory loss and paresthesias in an ulnar distribution (see Table 9.1). As with most neuropathies, motor findings may be absent in mild cases. Severe cases may include atrophy and fasciculations in the hypothenar eminence. Because of weak lumbricals at the fourth and fifth digits, these fingers may assume a characteristic "ulnar claw" or "benediction posture" (Figure 9.8C). Differential diagnosis includes C8 and T1 radiculopathy, Pancoast's syndrome, thoracic outlet syndrome, or other lesions of the brachial plexus inferior trunk or medial cord (see Figures 9.1 and 9.2). Unlike ulnar neuropathy, these conditions sometimes produce a Horner's syndrome, sensory changes in the T1 dermatome of the upper medial arm (see Figure 8.4) or involvement of hand muscles innervated by the median nerve. Entrapment in the cubital canal at the elbow can be treated surgically by translocation of the ulnar nerve to the flexor side of the elbow.

Compression of the ulnar nerve in the hand as it passes over the hamate bone in Guyon's canal can occur from prolonged leaning forward while cycling. The result is weakness of finger adduction and abduction without sensory loss because the cutaneous branches of the ulnar nerve are given off more proximally.

Combination of chronic median and ulnar nerve lesions leads to thenar and hypothenar atrophy with lack of thumb opposition, resulting in a "simian hand," or "monkey's paw" (Figure 9.8D).

Lower-Extremity Nerve Injuries

FEMORAL NEUROPATHY The femoral nerve can occasionally be injured during pelvic surgery or compressed by a retroperitoneal hematoma or pelvic mass. Abnormalities include weakness of thigh flexion and knee extension, loss of the patellar reflex, and sensory loss in the anterior thigh (see Table 9.3). Differential diagnosis includes L3 or L4 radiculopathy. L3 or L4 radiculopathy, however, may include weakness of thigh adduction (obturator nerve), a feature not associated with femoral neuropathy (see Table 8.1).

SCIATIC NEUROPATHY Causes of sciatic neuropathy include posterior hip dislocation, acetabular fracture, and intramuscular injection placed too medially and inferiorly in the buttocks. There is weakness of all foot and ankle muscles and of knee flexion, loss of the Achilles tendon reflex, and sensory loss in the foot and lateral leg below the knee (see Table 9.3). Differential diagnosis includes lesions in the foot area of the motor cortex (see KCC 6.3; Figure 6.14F).

The term "sciatica" is vague and refers to all disorders causing painful paresthesias in a sciatic distribution. The most common cause is compression of lumbosacral roots by disc material and osteophytes (see KCC 8.3). Rarely, the sciatic nerve may be compressed more distally by muscular or skeletal elements.

PERONEAL NERVE PALSY As the common peroneal nerve passes around the fibular head near the skin surface, it is vulnerable to laceration, stretch injury by forcible foot inversion, or compression by tight stockings, a cast, crossed legs, or trauma. In peroneal nerve palsy, there is **foot drop**, with weakness of foot dorsiflexion and eversion, and sensory loss over the dorsolateral foot and shin. Most patients recover spontaneously when the mechanical cause is removed. A foot brace may improve function if foot drop is significant. Differential diagnosis includes L5 radiculopathy. However, L5 radiculopathy includes weakness of foot dorsiflexion, eversion, and inversion, while in peroneal palsy, foot inversion is normally spared because this function can be carried out by the tibialis posterior (innervated by the tibial nerve) (see Table 9.3; see also Table 8.1).

OBTURATOR NERVE PALSY The obturator nerve (originating from L2–L4; see Figure 9.3 and Table 9.3) can be compressed in women during complicated delivery or occasionally during pelvic trauma or surgery. Deficits include gait instability due to weakness of the leg adductor muscles and pain and numbness in the medial thigh.

MERALGIA PARESTHETICA The lateral femoral cutaneous nerve (which originates in L2 and L3; see Figures 9.3 and 9.4) can be entrapped as it passes under the inguinal ligament or fascia lata, producing paresthesias and loss of sensation in the lateral thigh (see Figure 9.5). This entrapment syndrome includes no motor involvement or reflex changes. Common causes include obesity, pregnancy, weight loss, or heavy equipment belts, and symptoms may be worse after prolonged walking, standing, or sitting. Differential diagnosis includes L2 or L3 radiculopathy, although, unlike meralgia paresthetica, these conditions usually include motor changes or decreased patellar reflex. Symptoms most often resolve spontaneously or by avoidance of mechanical precipitants; however, surgical decompression is occasionally attempted in refractory cases.

MORTON'S METATARSALGIA Tight-fitting shoes can compress the digital nerves, especially of the third and fourth toes, producing patches of numbness and paresthesias.

In conclusion, familiarity with the patterns of sensory and motor loss in the common plexus and nerve syndromes discussed in this section, as well as those seen in radiculopathies (see KCC 8.3) and other disorders of nerves, muscles, and the neuromuscular junction (see KCC 8.1), can greatly aid in localizing neurological deficits and in distinguishing disorders of the peripheral and central divisions of the nervous system. When the diagnosis remains uncertain, electrodiagnostic tests can often be helpful, as discussed in the next section. ■

KEY CLINICAL CONCEPT

9.2 ELECTROMYOGRAPHY (EMG) AND NERVE CONDUCTION STUDIES

Electromyography (EMG) and nerve conduction studies are valuable diagnostic tools that can help localize and determine the causes of nerve and muscle disorders. In **nerve conduction studies**, stimulating electrodes are placed on the skin overlying a nerve, and recording electrodes are placed either at a different point along the same nerve or overlying a muscle innervated by the nerve (Figure 9.9A–C). When a stimulus is given to the nerve, a **compound motor action potential** (**CMAP**) can be recorded over the belly of a muscle innervated by that nerve, resulting from the summated electrical activity of muscle cells (Figure 9.9D). If a distal nerve branch with purely sensory function is used for

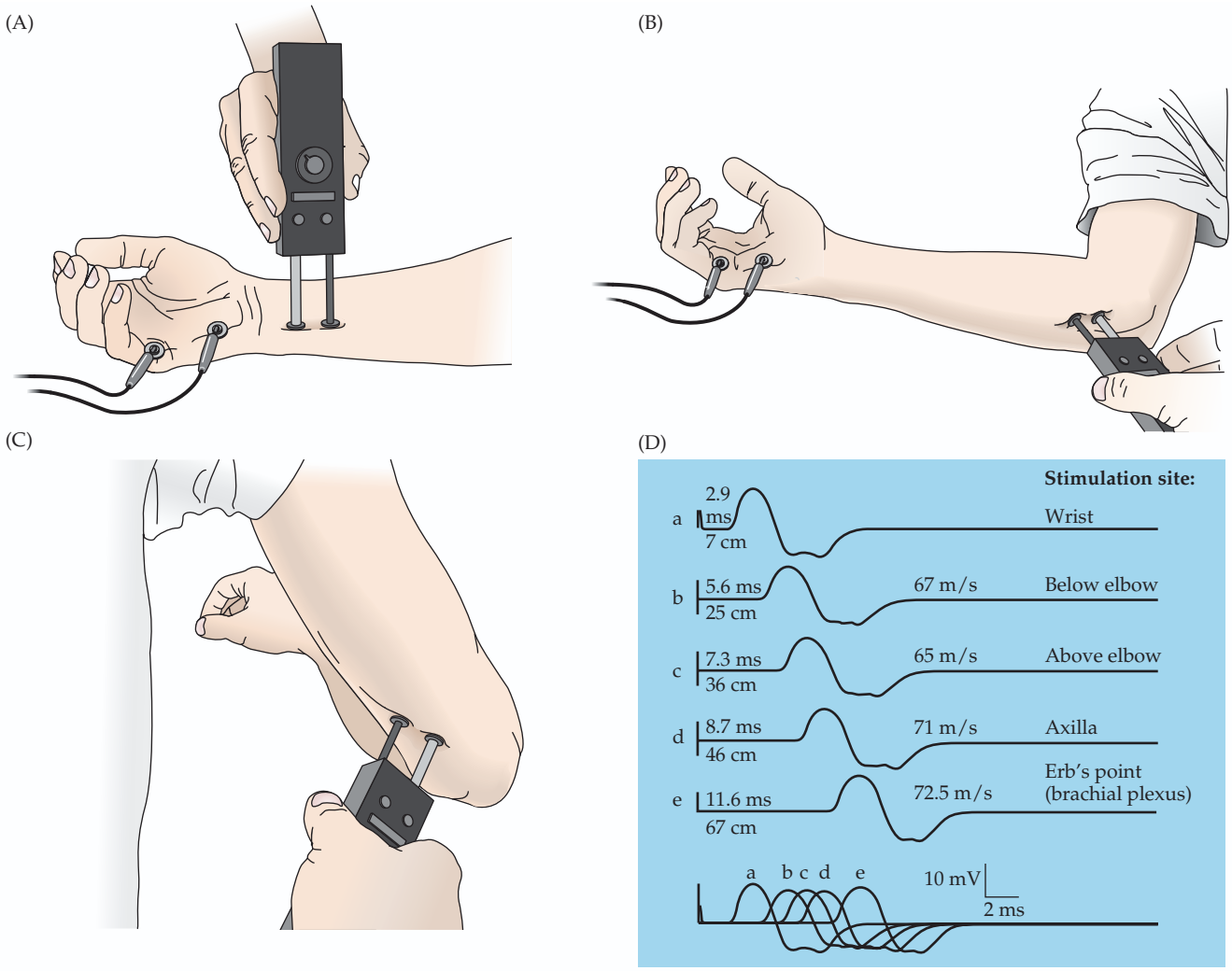


FIGURE 9.9 Nerve Conduction Study
The compound motor action potential (CMAP) of the ulnar nerve was recorded in this example from a normal subject at several stimulus locations. (A) Stimulation of the ulnar nerve at the wrist and recording of CMAP from the abductor digiti quinti in the hypothenar eminence. (B) Ulnar nerve stimulation just distal to the ulnar groove. (C) Ulnar nerve stimulation just proximal to the ulnar groove. (D) CMAP recordings for different stimulation sites. For each trace, the distance from the recording electrode (in centimeters) and the latency to CMAP onset (in milliseconds) are indicated to the left. Conduction velocities between adjacent stimulation sites (in meters per second) were calculated by dividing the distance between the two stimulation sites by the difference in motor latencies for the two sites. (After Rajesh KS, Thompson LL. 1989. *The Electromyographer's Handbook*. 2nd Ed. Little, Brown, Boston.)

recording or stimulation and a second set of stimulating or recording electrodes is placed somewhere along the nerve, a **compound sensory nerve action potential** (**SNAP**) can be recorded over the nerve, resulting from summated electrical activity in the sensory neuron axons of the nerve.

Lesions proximal to the dorsal root ganglia will leave the cell bodies and distal axons of sensory neurons intact (see Figure 8.1B). Therefore, SNAPs will be preserved. In contrast, proximal lesions of motor nerve roots will cause degeneration of the distal motor neuron axons, reducing or abolishing CMAPs. There are standard values for SNAP and CMAP latencies or conduction velocities for each major nerve when stimulated at various points along its course. These values allow nerve conduction studies to be used to determine if there is evidence for slowed nerve conduction—for example, in the case of **demyelination** (see KCC 8.1). In addition, there are standard values for SNAP amplitudes. Decreased SNAP amplitudes suggest that conduction in some axons of the nerve has been interrupted, as is the case in **axonal damage**.

CMAP studies can be used to evaluate the function of the neuromuscular junction by the use of **repetitive stimulation**. Slow, repetitive stimulation (2–3 Hz) depletes presynaptic stores of acetylcholine; faster repetitive stimulation (>5 Hz) increases presynaptic calcium, facilitating neurotransmitter release. Under normal conditions, repetitive stimulation does not significantly affect

CMAP amplitude because there is a "safety factor," meaning that every presynaptic action potential results in a postsynaptic potential well above the threshold needed to produce a muscle cell action potential. Under pathologic conditions, however, failures in neuromuscular transmission occur. Therefore, for example in **myasthenia gravis** (see KCC 8.1), in which there is a decrease in postsynaptic acetylcholine receptors on muscle cells, slow repetitive stimulation results in a gradual **decrement** in CMAP amplitude. Decrement of >10% is considered abnormal. In **Lambert–Eaton myasthenic syndrome** and in **botulism**, in which there is decreased presynaptic neurotransmitter release, fast repetitive stimulation (or active volitional muscle contraction) causes CMAPs to **increment** in amplitude from an abnormally low starting point.

In **electromyography** (**EMG**), an electrode is inserted directly into a muscle, and motor unit action potentials (MUPs) are recorded from muscle cells. The EMG pattern provides information useful in distinguishing weakness caused by **neuropathic disorders** (nerve or motor disease) from that caused by **myopathic disorders** (muscle disease). In neuropathic disorders, **increased spontaneous activity** (fibrillation potentials and positive sharp waves) often is recorded on EMG and is sometimes also visible on physical examination as **fasciculations** (see KCC 6.1). Fasciculations and other forms of spontaneous activity can occur due to chronic deinnervation of muscle cells. Deinnervation also causes adjacent motor axons to sprout and reinnervate a larger region, resulting in abnormally large motor units (a motor unit consists of all the muscle cells innervated by a single motor neuron axon). Therefore, with neuropathic disorders, MUPs are of abnormally large amplitude and duration. Reduced MUP amplitude and duration suggests a **myopathic** disorder is present.

When a muscle is voluntarily contracted, the EMG normally shows a pattern of continuous firing of motor units referred to as a normal **recruitment pattern**. In neuropathic disorders, the recruitment pattern has normal amplitude but shows interrupted firing, since some motor units are not successfully activated. This phenomenon is referred to as decreased, reduced, or incomplete recruitment. In myopathic disorders, the recruitment pattern is continuous or even increased (since more motor units need to be activated for a given force), but the amplitude is often decreased. ■

CLINICAL CASES

CASE 9.1 COMPLETE PARALYSIS AND LOSS OF SENSATION IN ONE ARM

CHIEF COMPLAINT

A 60-year-old man with a history of lung cancer gradually developed severe pain, weakness, and numbness in his right arm.

HISTORY

The patient smoked for 34 years. Two years ago he was diagnosed with lung cancer and underwent a right upper-lobe lung resection followed by radiation and chemotherapy. Six months ago he developed shooting **pain and swelling of the right arm**. He gradually **lost all strength and sensation in the entire right arm up to the shoulder** but continued to have severe burning pain. Past medical history was notable for right eye surgery following an assault with a baseball bat 20 years ago.

PHYSICAL EXAMINATION

Vital signs: T = 99.4°F, P = 110, BP = 130/80.

Neck: Supple; no tenderness.

Lungs: Clear.

Heart: Regular rate with no murmurs, gallops, or rubs.

Abdomen: Normal bowel sounds; soft, no masses.

Extremities: Right arm swollen, with two firm, 5 cm discolored masses—one in the right axilla and one in the upper right chest wall. Also marked clubbing of the fingers bilaterally.

(*continued on p. 370*)

CASE 9.1 (*continued*)

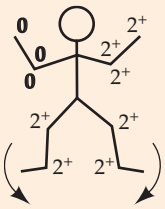
Neurologic exam:

MENTAL STATUS: Alert and oriented × 3.

CRANIAL NERVES: Intact, except for the right eye, which had an irregular pupil and diminished vision in both the lateral and medial fields.

MOTOR: Normal tone except for the **right arm**, which was **flaccid**. 5/5 power throughout, except for **0/5 power in the right shoulder**, **arm**, **and hand**.

REFLEXES:



COORDINATION: Normal on finger-to-nose (except unable to test right arm) and heel-to-shin testing.

GAIT: Normal.

SENSORY: **Absent light touch, pinprick, and vibration sense in the entire right arm up to the deltoid** (Figure 9.10)**.** Sensation otherwise normal.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

1. On the basis of the symptoms and signs shown in **bold** above, where is the lesion?
2. What is the most likely diagnosis?
3. This patient had an abnormal right eye due to prior trauma. If his right eye had been normal previously, what additional finding might be present on exam that would help with the localization?

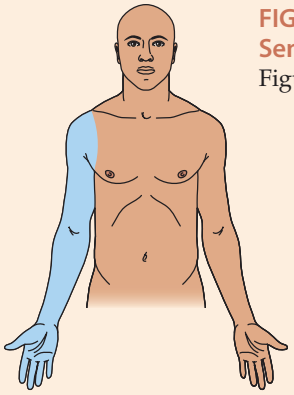


FIGURE 9.10 Region of Sensory Loss Compare to Figures 8.4 and 9.5.

Discussion

1. The key symptoms and signs in this case are:
   - **Paralysis, with decreased tone and absent reflexes in the entire right arm and hand**
   - **Absent light touch, pinprick, and vibration sense in the entire right arm up to the deltoid**
   - **Pain and swelling in the right arm**

   Weakness in one arm can be caused by peripheral nerve lesions or by lesions in the arm region of the motor cortex (see KCC 6.3; Figure 6.14E). However, it is unlikely that a cortical lesion would produce complete paralysis and sensory loss in the entire arm ending sharply at the shoulder, with no face or leg weakness at all. In addition, no single peripheral nerve lesion could produce this pattern. Therefore, the lesion must involve the entire right brachial plexus, or all right-sided nerve roots from C5 through T1.
2. The history of right apical lung tumor supports the possibility of a lesion invading the right brachial plexus from below (see the description of Pancoast's syndrome in KCC 9.1), as does the presence of swelling in the arm, suggesting obstruction of venous return. Because of the past history of radiation therapy, **radiation plexitis** is another possibility, in which numbness and sometimes weakness can develop in a limb months to years after treatment due to radiation-induced nerve injury.

3. A lesion of the proximal portion of the lower brachial plexus involving the T1 nerve root can cause a Horner's syndrome (see Figures 6.13, 13.10; KCC 13.5). This condition could be difficult to appreciate in this patient because of his prior history of right eye trauma.

Clinical Course and Neuroimaging

A **brachial plexus MRI** (Image 9.1, page 372) showed extensive invasion of the apical lung mass into the region of the right brachial plexus. The cancer in this patient, unfortunately, was no longer amenable to treatment. However, his pain was managed by a multidisciplinary team using oral, intravenous, and epidural medications to provide adequate pain relief.

CASE 9.2 A NEWBORN WITH WEAKNESS IN ONE ARM

MINICASE

A 3-week-old infant girl was brought to the pediatrician because of left arm weakness. She was born at 42 weeks (2 weeks past the due date) weighing 10 pounds 11 ounces, and the delivery was complicated by shoulder dystocia (difficulty delivering the shoulder) resulting in significant traction on the left neck and shoulder during delivery. Left arm weakness was noted at birth that improved slightly but was still present at the appointment. Exam was normal except for the **left upper extremity**, which had **decreased tone and lay internally rotated at the infant's side with decreased spontaneous movements**. **She did not abduct the left arm or flex it at the elbow** but did have spontaneous opening and closing of the hand with normal grip strength, normal elbow extension, and some wrist flexion. **The left biceps reflex was absent**, and other reflexes were 2^+ throughout.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

Discussion

The key symptoms and signs in this case are:

- **Weakness of left arm external rotation, abduction, and elbow flexion, with decreased tone and absent biceps reflex**

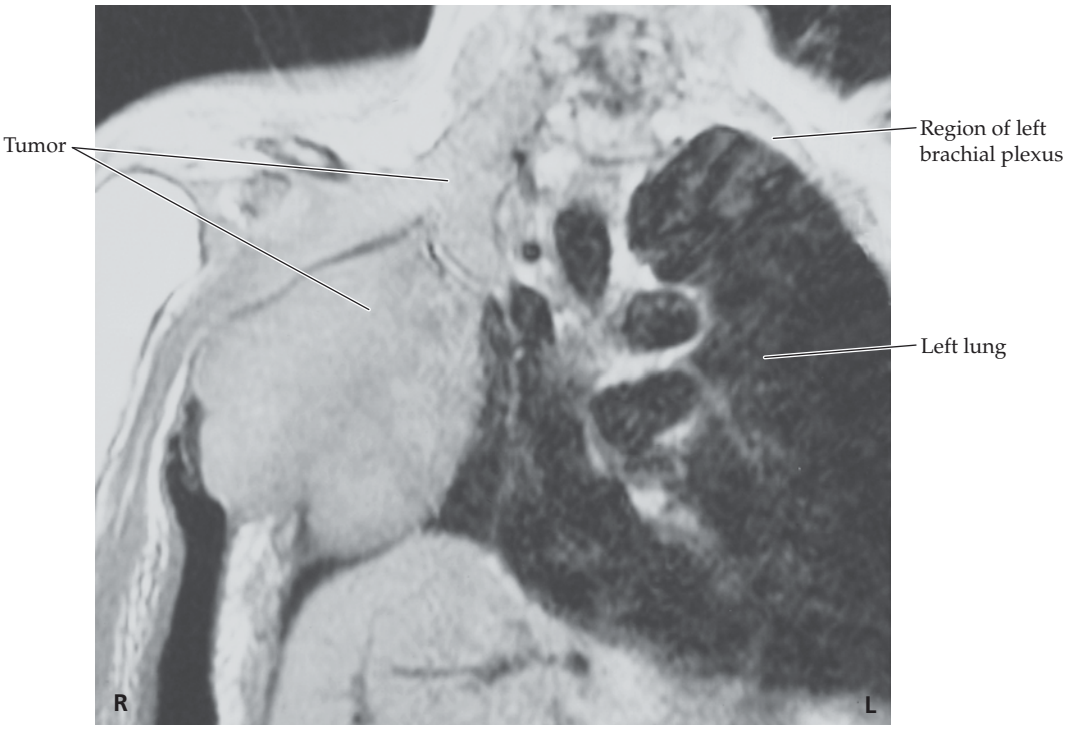
The patient has typical findings consistent with a left brachial plexus upper trunk injury (Erb–Duchenne palsy), affecting C5 and C6 innervated muscles, caused by left shoulder traction at birth (KCC 9.1).

Clinical Course

A physical therapy program was initiated to preserve range of motion during recovery. At age 7 weeks the patient was able to lift her arm off the table and had some external rotation of the arm, as well as slight spontaneous elbow flexion. The left biceps reflex was still absent. By age 4 months she was able to reach for objects well with either hand, although she preferred to use her right hand, and she had $4^+/5$ left biceps strength when pulled to a seated position. Continued improvement was anticipated.

CASE 9.1 COMPLETE PARALYSIS AND LOSS OF SENSATION IN ONE ARM

IMAGE 9.1 Right Apical Lung Cancer Extending into the Region of the Brachial Plexus T1-weighted coronal MRI scan of the chest.



CASE 9.3 A BLOW TO THE MEDIAL ARM CAUSING HAND WEAKNESS AND NUMBNESS

MINICASE

A 38-year-old alcoholic man was seen to fall, catching his right arm on a garbage can. He was brought to the emergency room. He had an **abrasion and tenderness of the right upper medial arm**. Neurologic exam was normal except for **4/5 strength of right thumb opposition, second and third finger flexors, and wrist flexion and abduction. There was also decreased pinprick and light touch sense along the lateral surface of the right hand and first, second, and third fingers** (Figure 9.11)**.**

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

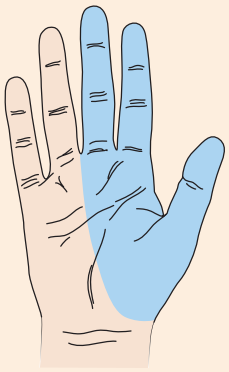


FIGURE 9.11 Region of Sensory Loss

Discussion

The key symptoms and signs in this case are:

- **Weakness of right thumb opposition, second and third finger flexors, and wrist flexion and abduction**
- **Decreased pinprick and light touch sense along the lateral surface of the right distal forearm, hand, and first, second, and third fingers**
- **Abrasion and tenderness of the right upper medial arm**

The pattern of weakness and sensory loss in this patient is consistent with a median nerve injury (see Table 9.1; Figure 9.5; see also Table 8.1). The most likely cause is compression of the nerve in the upper medial arm, as evidenced by the tenderness in this area and the mechanism of injury.

Clinical Course

X-rays of the right arm revealed no fractures. The patient was discharged from the emergency room and did not return for follow-up.

CASE 9.4 NOCTURNAL PAIN AND TINGLING IN THE THUMB, POINTER, AND MIDDLE FINGER

MINICASE

A 38-year-old man who works in a cola factory developed **pain and tingling in his right thumb**, **index**, **and middle fingers** over the past 2 months that occasionally radiates into the right arm and forearm. His symptoms are worse at night or when the arm is relaxed. He has also noticed some **decreased sensation of the fingertips of the same fingers** while buttoning his shirt. Exam was notable only for obesity and **4^{+}/5 weakness of the right opponens pollicis and decreased pinprick sensation in the palmar aspect of the right first**, **second**, **and third fingers**, **sparing the thenar area** (Figure 9.12). Tinel's and Phalen's signs were not present.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

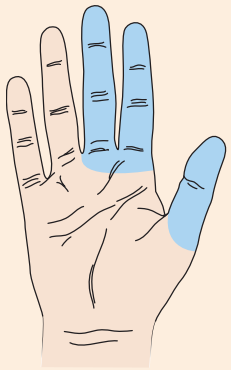


FIGURE 9.12 Region of Sensory Loss

Discussion

The key symptoms and signs in this case are:

- **Mild weakness of the right opponens pollicis**
- **Pain, tingling, and decreased pinprick sensation in the palmar aspect of the right first, second, and third fingers, sparing the thenar area**

The opponens pollicis is supplied by the median nerve (Table 9.1; see also Table 8.1). The fact that wrist flexion and abduction are spared, as is sensation over the thenar area, suggests a lesion of the median nerve after the branch to the flexor carpi radialis and the palmar cutaneous branch are given off, such as in the carpal tunnel (see KCC 9.1). Carpal tunnel syndrome of the right wrist is thus the most likely diagnosis. Note that the abductor pollicus brevis is usually affected in carpal tunnel syndrome (see KCC 9.1) but was not specifically tested in this patient. Other, less likely pos-

sibilities include a mild right C6 and C7 radiculopathy or a more proximal right median nerve lesion.

Clinical Course

Thyroid function tests and routine blood chemistries were normal. The patient was given a removable splint to hold his wrist in slight extension at night, and his symptoms gradually improved.

CASE 9.5 HAND AND WRIST WEAKNESS AFTER A FALL

MINICASE

A 20-year-old male waiter tripped while working at a restaurant and broke his fall by extending his left hand onto a table. That night he had pain in his left arm that resolved by the next day, but he then noticed **weakness of the left hand and wrist** and came to the emergency room. Exam was normal except for **3/5 strength in the left wrist extensors**, **finger extensors**, **and thumb abduction in the plane of the palm**, **and 4/5 strength in forearm supination**. Strength in all other muscles was intact, as was sensation.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

Discussion

The key symptoms and signs in this case are:

- **Weakness of left forearm supination, wrist extensors, finger extensors, and abduction of the thumb in the plane of the palm**

These muscles are all supplied by the radial nerve (Table 9.1; see also Table 8.1). In particular, the facts that the triceps was spared and that there was no sensory loss suggest a lesion of the posterior interosseous nerve, a purely motor branch of the radial nerve. The posterior interosseous branch of the radial nerve was apparently injured during the fall, with the exact mechanism being unclear (see KCC 9.1).

Clinical Course

X-rays of the left arm did not reveal a fracture. The patient was given a splint to avoid developing contracture deformities and was followed by an occupational therapist as his strength gradually recovered. An EMG done 2 months after the injury was consistent with a lesion of the left radial nerve distal to the fibers innervating the triceps. By 4 months after the injury, strength had returned to $4^{+}/5$ in the affected muscles and was continuing to improve gradually. (Note: In posterior interosseous nerve injuries, the extensor carpi radialis is usually spared, so extension of the wrist in the radial direction is preserved. This was not tested for in this case.)

CASE 9.6 NUMBNESS AND TINGLING IN THE PINKY AND RING FINGER

MINICASE

A 32-year-old computer programmer developed 2 months of worsening **tingling and numbness in his left fifth digit, in the medial aspect of his left fourth digit, and along the medial surface of his left hand and forearm**. The symptoms were worse upon awakening in the morning and were exacerbated after resting his elbows on a hard surface. Exam was normal except for **4/5 strength in left fifth finger abduction, and decreased pinprick sensation in the left fifth digit and the medial half of the left fourth digit** (Figure 9.13). Symptoms were not worsened by arm abduction plus elevation.

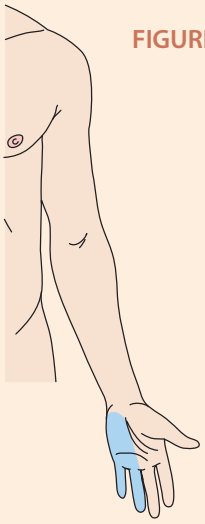


FIGURE 9.13 Region of Sensory Loss

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

Discussion

The key symptoms and signs in this case are:

- **Weakness of left fifth finger abduction**
- **Paresthesias and decreased pinprick sensation in the left fifth digit and the medial half of the left fourth digit**

The sensory and motor deficits in this patient could be caused by mild dysfunction in the ulnar nerve, the brachial plexus lower trunk (e.g., thoracic outlet syndrome), the brachial plexus medial cord, or the C8 or T1 nerve roots (see Table 9.1; KCC 9.1; see also Table 8.1). The fact that the symptoms are worse after pressure on the elbow, are not accompanied by neck pain (common in cervical radiculopathy; see KCC 8.3), and are not exacerbated by arm abduction plus elevation (characteristic of thoracic outlet syndrome) suggests, but does not prove, that the ulnar nerve is the culprit.

Clinical Course

Nerve conduction studies (see KCC 9.2; Figure 9.9) were normal except in the ulnar nerves. To measure conduction velocity in the ulnar nerve, a recording electrode was placed on the skin over the belly of the abductor digiti minimi muscle and a stimulating electrode was placed on the skin at various points on the arm over the course of the ulnar nerve. When a stimulus was given to the nerve, a compound motor action potential (CMAP) could be recorded over the muscle. Distal conduction velocity in this patient was normal when the ulnar nerve was stimulated below the medial epicondyle of the elbow (see Figure 9.9A,B), but was decreased when the ulnar nerve was stimulated just above the elbow (see Figure 9.9C), suggesting a conduction problem at the elbow.

The nerve conduction studies showed that both ulnar nerves were affected, although only the left one had produced symptoms. Nerve conduction studies of the median nerves were normal bilaterally. The patient was given elbow pads to wear while sleeping or while working at the computer, and he was instructed to avoid resting on his elbows. Two months later his paresthesias had improved markedly, strength in his finger abductors was normal, and he had only mildly decreased sensation in a left ulnar distribution.

CASE 9.7 SHOULDER WEAKNESS AND NUMBNESS AFTER STRANGULATION

MINICASE

A 39-year-old woman was assaulted by strangulation. She escaped serious asphyxia but over the subsequent days developed progressive swelling in the neck area and swallowing difficulties. She went to the emergency room ten days later and a CT scan revealed a hematoma and abscess in the retropharyngeal space extending into her left sternocleidomastoid muscle. She was taken to the operating room for drainage of the abscess and was treated with antibiotics. Following surgery her swallowing was improved, but she reported difficulty raising her left arm to put on a shirt or to apply deodorant. She also noticed some numbness over her left shoulder area. Neurological examination was normal except for **3/5 strength on abduction of the left arm at the shoulder**, and **diminished light touch and pinprick sensation in a patch over her left lateral shoulder** (Figure 9.14). Of note, left biceps strength and reflexes were normal.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

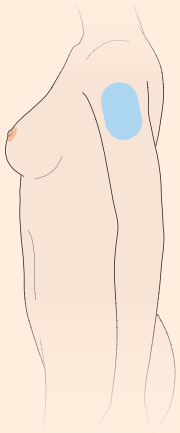


FIGURE 9.14 Region of Sensory Loss

Discussion

The key symptoms and signs in this case are:

- **Weakness of left arm abduction**
- **Decreased sensation to light touch and pinprick on left lateral shoulder**

Weakness of arm abduction and shoulder numbness could be caused by a left C5 radiculopathy (see Figure 8.4; Table 8.5) or by a left axillary nerve injury (see Table 9.1; Figure 9.5). Sparing of the biceps, which receives important innervations from the C5 nerve root, makes a C5 radiculopathy less likely. Therefore, the most likely diagnosis is a left axillary nerve injury (see KCC 9.1). Axillary nerve injury is usually caused by dislocation or traction of the shoulder, and can also been seen as a complication of surgery in the neck and shoulder region.

Clinical Course

The nerve injury was discussed with the patient, and follow-up was advised since in some cases when prompt recovery does not occur, axillary nerve injury can benefit from treatment by nerve grafting or decompressive surgery. However, after a course of intravenous antibiotics in the hospital, the patient was discharged home and did not return for follow-up appointments.

CASE 9.8 UNILATERAL THIGH PAIN, WEAKNESS, AND NUMBNESS IN A DIABETIC

MINICASE

A 45-year-old man spent several weeks in the intensive care unit for diabetic ketoacidosis and severe bilateral pneumonia. When he finally stabilized and was transferred to a regular hospital floor, he noticed **weakness and numbness in the left leg**, **with numbness and tingling over the anterior thigh down to the medial calf above the foot**. A neurology consult was called, and on exam he had **4/5 strength in the left iliopsoas and quadriceps**, with preserved strength in all other muscle groups, including the thigh adductors. There was **decreased pinprick sensation in the left anterior thigh and medial calf** (Figure 9.15). Reflexes were normal and symmetrical except for an **absent left patellar reflex**.

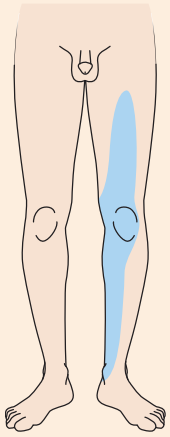


FIGURE 9.15 Region of Sensory Loss

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

Discussion

The key symptoms and signs in this case are:

- **Weakness of the left iliopsoas and quadriceps with absent patellar reflex**
- **Paresthesias and decreased pinprick sensation in the left anterior thigh and medial calf**

The pattern of weakness, reflex loss, and sensory changes in this patient could be caused by an L4 radiculopathy or a femoral neuropathy (see Table 9.3; see also Tables 8.1, 8.6; Figure 8.4). The fact that thigh adduction is spared suggests a femoral neuropathy, since L4 contributes significantly to both the obturator and femoral nerves. The most likely diagnosis is left femoral neuropathy caused by diabetes (see KCC 8.1, 9.1). Less likely, insertion of a femoral vein catheter during the patient's stay in the intensive care unit may have caused an undetected hematoma compressing the femoral nerve. Compare this case to Case 8.9.

Clinical Course

The patient gradually recovered strength in the left leg. When seen 1 year later, he had 5/5 power in all muscle groups, but he had persistent sensory loss in a left femoral nerve distribution and an absent left patellar reflex.

CASE 9.9 TINGLING AND PARALYSIS OF THE FOOT AFTER A FALL

CHIEF COMPLAINT

A 30-year-old woman presented to the emergency room after a fall with **tingling and paralysis of the right foot**.

HISTORY

Two days ago the patient slipped on a wet floor in the supermarket and fell backward, landing on her back. She initially noticed no symptoms, but she awoke at 3:00 A.M. to feed her 2-month-old baby and was **unable to move her right foot**. She also had a **tingling sensation in her right lateral lower leg and foot**. These symptoms did not resolve over the next 2 days, so she came to the emergency room. There was no back pain, and there were no bowel or bladder symptoms.

(*continued on p. 378*)

CASE 9.9 (*continued*)

PHYSICAL EXAMINATION

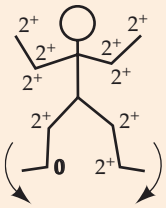
Vital signs: T = 98°F, P = 84, BP = 136/68.
Neck: Supple with no bruits.
Lungs: Clear.
Heart: Regular rate.
Abdomen: Soft.
Extremities: Normal.
Back and spine: No tenderness.
Rectal: Normal tone with no masses.
Neurologic exam:

MENTAL STATUS: Alert and oriented × 3. Fluent speech.

CRANIAL NERVES: Intact.

MOTOR: No drift. Normal tone, except for **diminished tone in the right foot**. 5/5 power throughout, except for **0/5 power in the right tibialis anterior**, **extensor hallucis longus**, **foot invertors**, **foot evertors**, **and gastrocnemius**, **and 3/5 power in the right hamstrings**.

REFLEXES:



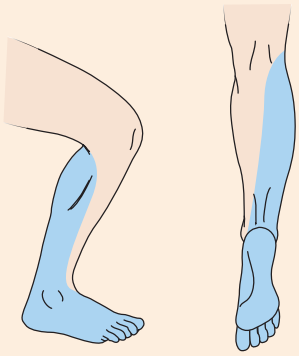


FIGURE 9.16 Region of Sensory Loss

COORDINATION: Normal on finger-to-nose testing.

GAIT: **Flailing movements of the right foot while raising it off the floor with each step.**

SENSORY: **Decreased light touch, pinprick, vibration, and joint position sense in the right lateral calf and in the entire right foot** (Figure 9.16)**.** Sensation otherwise normal.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

Discussion

The key symptoms and signs in this case are:

- **Weakness of the right tibialis anterior, extensor hallucis longus, foot invertors, foot evertors, gastrocnemius, and hamstrings, with decreased right foot tone and absent right Achilles tendon reflex**
- **Paresthesias and decreased light touch, pinprick, vibration, and joint position sense in the right foot and lateral calf**

Right leg weakness, decreased tone, and hyporeflexia could be caused by a peripheral nerve lesion in the right leg or by an acute upper motor neuron lesion in the motor cortex or spinal cord (see KCC 6.1, 6.3; Figure 6.14F). The pattern of sensory loss is not consistent with a spinal cord lesion (see KCC 7.4) or cortical lesion (see KCC 7.3), but it does match that of a sciatic nerve lesion (see KCC 9.1; Table 9.3), as do the details of the motor exam. The most likely diagnosis is right sciatic neuropathy, probably caused by the fall, although the exact mechanism of injury is unclear.

Clinical Course and Neuroimaging

X-rays of the lumbosacral spine and pelvis revealed no fractures. A **lumbar plexus MRI** (Image 9.9A,B, page 380) revealed T2 bright signal in the right sciatic nerve, consistent with edema. The patient was treated with physical therapy and a right foot brace to aid ambulation. An EMG done 1 week after the onset of symptoms revealed inability to activate the muscles of the right

sciatic nerve, including the gastrocnemius, tibialis anterior, flexor hallucis brevis, and medial hamstrings. A follow-up study was not done. The patient's strength gradually improved, and by 5 months after onset she felt almost back to normal. On exam 1 year after onset, she had 4/5 to $4^+/5$ strength in the right tibialis anterior, foot evertors, and invertors. Gastrocnemius strength was 5/5 bilaterally, and she had recovered sensation and the Achilles tendon reflex in the right foot.

CASE 9.10 A LEG INJURY RESULTING IN FOOT DROP

MINICASE

A 27-year-old man slipped on a wet tile floor and twisted his right foot toward the left, resulting in acute foot pain, followed by weakness. He was seen in the emergency room and had **0/5 power in his right tibialis anterior and extensor hallucis longus and 3/5 power in his right foot evertors**. Power was otherwise 5/5, including the right foot invertors and gastrocnemius. He had **decreased sensation to pinprick on the dorsum of the right foot, which was especially pronounced in the web space between the first and second toes** (Figure 9.17).

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

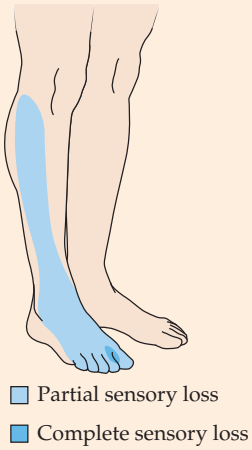


FIGURE 9.17 Region of Sensory Loss

Discussion

The key symptoms and signs in this case are:

- **Weakness in the right tibialis anterior and extensor hallucis longus; moderate weakness of the right foot evertors**
- **Decreased sensation to pinprick on the dorsum of the right foot, especially between the first and second toes**

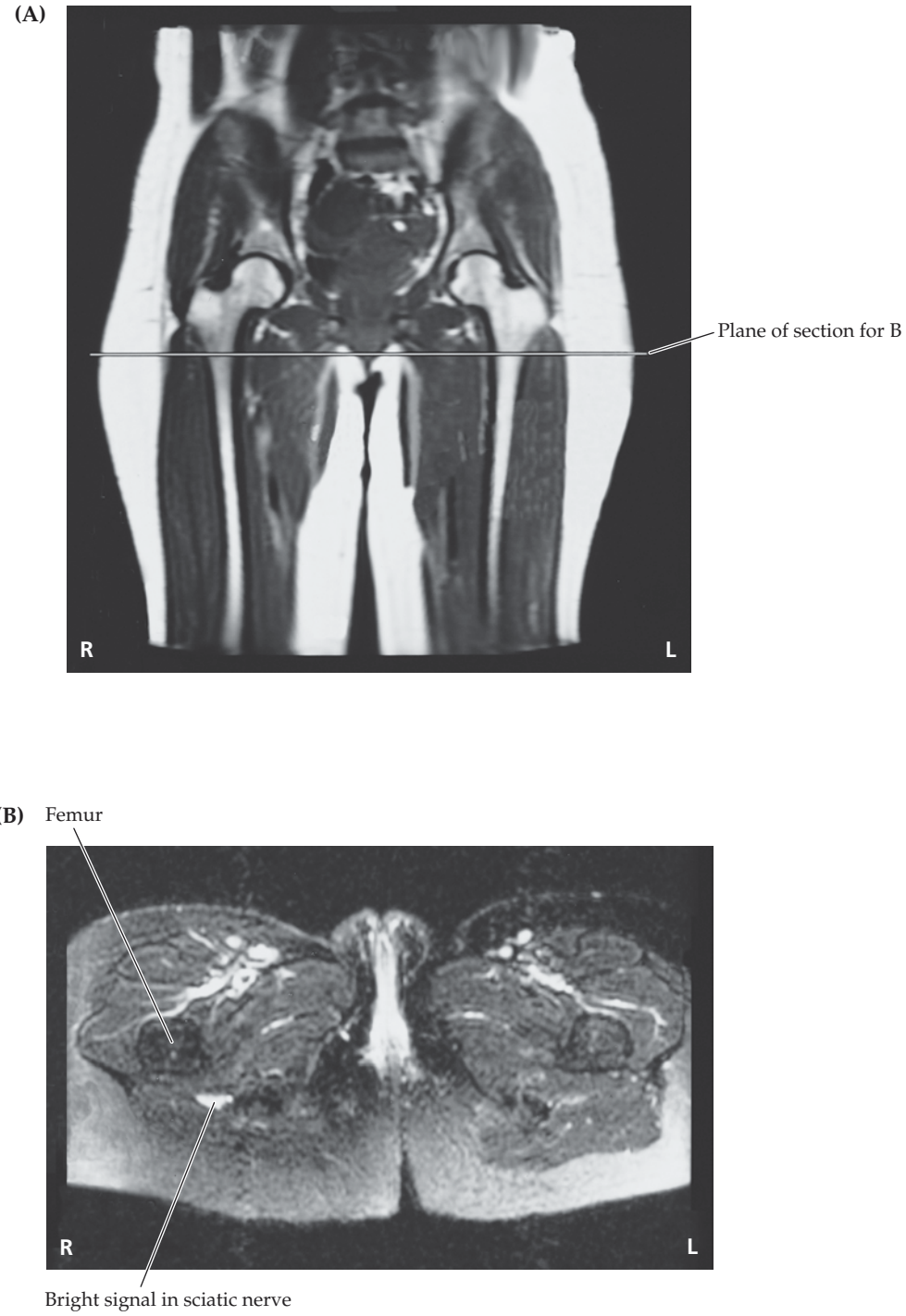
The pattern of weakness and sensory loss is consistent with injury to the common peroneal nerve (see Table 9.3; KCC 9.1; see also Table 8.1), which most likely occurred during the fall. Note that the deep peroneal nerve (tibialis anterior, extensor hallucis longus, sensation between first and second toes) appears to be more severely involved in this patient than the superficial peroneal nerve (foot eversion, sensation on dorsal foot and lateral shin). In addition, this should be distinguished from an L5 radiculopathy, in which there may also be weakness of foot inversion, not seen in the present case (compare to Case 8.10).

Clinical Course

An EMG (see KCC 9.2) 2 days after the injury showed abnormally low recruitment of motor unit action potentials in the right tibialis anterior, extensor hallucis longus, extensor digitorum brevis, and peroneus muscles, suggesting a neuropathic process. Motor nerve conduction studies showed decreased amplitudes when the right peroneal nerve was stimulated just above the fibular neck and normal amplitudes when stimulated just below the fibular neck, suggesting nerve injury at the fibular neck. The patient gradually improved over the following months.

CASE 9.9 TINGLING AND PARALYSIS OF THE FOOT AFTER A FALL

IMAGE 9.9A,B Abnormal Bright Signal in the Right Sciatic Nerve, Compatible with Sciatic Neuropathy
MRI of the lumbar plexus. (A) Coronal T2-weighted MRI showing the plane of section for B. (B) Axial T2-weighted section showing abnormal bright signal in the right sciatic nerve as it passes dorsal to the femur.



CASE 9.11 LATERAL THIGH PAIN AND NUMBNESS AFTER PREGNANCY

MINICASE

Two days after giving birth, a 24-year-old woman developed burning **pain and numbness in the right lateral thigh**, which was worsened by walking. Exam was normal except for a patch of **decreased sensation to light touch**, **pinprick**, **and cold on the right lateral thigh** (Figure 9.18). Importantly, her reflexes and motor strength were normal.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

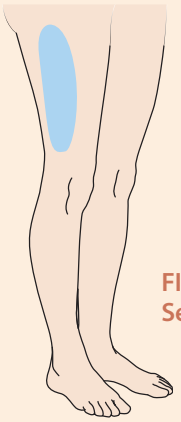


FIGURE 9.18 Region of Sensory Loss

Discussion

The key symptoms and signs in this case are:

- **Pain, paresthesias, and decreased sensation to light touch, pinprick, and cold on the right lateral thigh**

A purely sensory disorder affecting the lateral thigh is consistent with dysfunction of the lateral femoral cutaneous nerve, or meralgia paresthetica (see Figure 9.5; KCC 9.1). An L2 or L3 radiculopathy could be considered; however, there were no motor deficits, reflex abnormalities, or back pain to support this possibility.

Clinical Course

The patient was reassured that her symptoms were caused by injury to a sensory nerve that would likely improve with time. Her symptoms gradually resolved over the following 5 months and required no specific treatment.

CASE 9.12 DYSARTHRIA, PTOSIS, AND DECREASED EXERCISE TOLERANCE

CHIEF COMPLAINT

A 35-year-old woman saw a neurologist because of worsening dysarthria and muscle fatigue.

HISTORY

The patient worked as a nurse, and over the course of four months she noticed that at the end of her dictations, she had profound **difficulty enunciating her words**. This was **most apparent at the end of her work day**. Also, toward the end of her work day she had **difficulty producing a full smile**. Her symptoms disappeared with rest. She also noticed some mild neck discomfort and felt that it was **difficult at times to hold her head up**. In addition, she had **reduced exercise tolerance**, becoming short of breath sooner than previously when using the treadmill at her gym.

PHYSICAL EXAMINATION

Vital signs: T = 98°F, P = 80, BP = 90/70.

Neck: Supple, no bruits.

Lungs: Clear.

Heart: Regular rate.

Abdomen: Soft.

Extremities: Normal.

Neurologic exam:

MENTAL STATUS: Alert and oriented × 3. Fluent speech. Recalled 3/3 words after 5 minutes. Normal calculations.

CRANIAL NERVES: Intact visual fields and acuity. Pupils equal and reactive to light and accommodation. Extraocular movements intact with no nystagmus. **On prolonged**

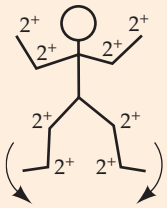
(*continued on p. 382*)

CASE 9.12 (*continued*)

upgaze she developed ptosis of the left eyelid. Facial sensation was intact. Face movements were symmetrical. Hearing was normal. Palate elevation was normal and tongue was midline. **While reading a long passage aloud, her speech gradually became dysarthric.**

MOTOR: Normal tone. No fasciculations or tremor. 5/5 strength throughout.

REFLEXES:



COORDINATION: Normal finger-to-nose and heel-to-shin testing.

SENSORY: Normal pinprick, temperature, vibration, and joint position sense. No extinction.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

1. Based on the symptoms and signs shown in **bold** above, where is the lesion?
2. What is the most likely diagnosis?

Discussion

The key symptoms and signs in this case are:

- **Dysarthria, worse at the end of prolonged speech**
- **Difficulty producing a full smile at the end of the day**
- **Ptosis of the left eye on prolonged upgaze**
- **Difficulty holding head up at the end of the day**
- **Reduced strength and shortness of breath on treadmill**

1. Dysarthria can be caused by cranial nerve or upper motor neuron disorders (see KCC 12.8), as can facial weakness during smiling (see Figure 12.13). Ptosis can result from disorders of CN III supplying the levator palpebrae superior or from Horner's syndrome (see KCC 13.6). Weakness of the neck muscles, legs, and respiratory muscles, likewise, can have central or peripheral causes. However, the weakness in these multiple locations, in the absence of any sensory findings, would be unusual for a neuropathy. In addition, there are no upper motor neuron signs to suggest a multifocal disorder affecting the central nervous system. Therefore, a peripheral disorder affecting the neuromuscular junction or muscles involved in speech, eyelid elevation, respiratory muscles, and proximal respiratory, neck, and leg muscles is most likely.
2. This pattern of weakness with no sensory loss and intact reflexes, together with the fact that the weakness was worse toward the end of the day or with repeated use of the muscles, is most suggestive of myasthenia gravis (see KCC 8.1). Other possible causes of diffuse, slowly progressive weakness without sensory loss, reflex loss, or upper motor neuron signs include Lambert–Eaton syndrome and myopathic disorders (see KCC 8.1).

Diagnostic Studies and Clinical Course

The neurologist performed a "Tensilon" (edrophonium) test (see KCC 8.1) by evaluating the patient's ability to read a long passage aloud. Her dysarthria was markedly reduced after administration of edrophonium, so the test was considered positive. Acetylcholine receptor antibodies were also positive at 1.73 nmol/L (normal is less than 0.3 nmol/L). **Repetitive stimulation** (see KCC 9.2) of the ulnar nerve at 3 stimuli per second produced a 23%

decrement of the CMAP amplitude recorded over the abductor digiti minimi muscle (Image 9.12, page 385). Decrement of greater than 10% is considered abnormal and supports the diagnosis of myasthenia gravis (see KCC 9.2). Pulmonary function tests were normal. The patient underwent a chest CT, which revealed a 7 × 5 cm lobulated mass in the right anterior mediastinum extending over to the right side of the pericardium, consistent with a thymoma (see KCC 8.1). She was treated with the anticholinesterase medication pyridostigmine (Mestinon) and underwent surgical resection of the thymic mass, which was confirmed histologically to be a thymoma. Following surgery, her dysarthria and fatigue resolved completely, and she had a normal neurologic exam, including no dysarthria and no ptosis, even after prolonged upgaze.

CASE 9.13 GENERALIZED WEAKNESS AND AREFLEXIA

CHIEF COMPLAINT

A 70-year-old woman came to the emergency room because of progressive weakness, gait difficulty and shortness of breath.

HISTORY

The patient was well until about 2 weeks previously when she developed intermittent diarrhea. About 8 or 9 days prior to admission she noticed **weakness in her arms and legs**, and had a few falls. She was sent for physical therapy, but her weakness progressed so that for the past 4 days she was **unable to walk**. She also noticed **tingling in her feet and finger tips**. Finally, she developed **breathing problems** and came to the hospital.

PHYSICAL EXAMINATION

Vital signs: T = 98.2°F, P = 74, BP = 132/74, RR = 20.

Bedside pulmonary function tests: **Vital capacity 1.6 L** (normal is greater than ~3.5 L for adult females, ~4.5 L for males); **Negative inspiratory force –35 cm H_2O** (normal is larger than –80 cm H_2O).

Neck: Supple, no bruits.

Lungs: Clear.

Heart: Regular rate, no murmurs.

Abdomen: Normal bowel sounds, soft, nontender.

Extremities: Normal, no edema

Neurologic exam:

MENTAL STATUS: Alert and oriented × 3. Normal language, attention and memory.

CRANIAL NERVES: Visual fields intact. Pupils equal and reactive to light. Extraocular movements intact. Facial sensation intact V_1 –V_3. **Facial movements appeared weak bilaterally. Palate and pharyngeal movements appeared weak. Weak shoulder shrug.** Tongue normal.

MOTOR: Normal bulk and tone. **Power in bilateral deltoids was 4/5, biceps 3/5, triceps 4^-/5, wrist extensors 3/5, finger abductors 4^-/5, hip flexors 3/5, knee flexors 4^+/5, foot dorsiflexors 4^-/5.**

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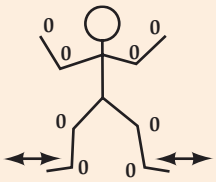


GAIT: **Unable to stand unsupported.**

SENSORY: **Decreased vibration sense in both feet up to the ankles.** Pinprick intact.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

1. Based on the symptoms and signs shown in **bold** above, where is the lesion?
2. What is the most likely diagnosis?

Discussion

The key symptoms and signs in this case are:

- **Progressive weakness of the face, palate, arms and legs leading to inability to walk**
- **Breathing difficulty, with reduced vital capacity and inspiratory force**
- **Absent reflexes**
- **Tingling in her fingertips and feet, and decreased vibration sense in both feet**

1. Weakness affecting bilateral cranial nerves, arms, legs and breathing muscles could be seen in a central disorder such as a brainstem lesion involving corticobulbar and corticospinal tracts, in widespread lower motor neuron disease, or in a diffuse disorder of the peripheral nerves (polyneuropathy), neuromuscular junctions, or muscles themselves (see KCC 6.3, Generalized Weakness or Paralysis). Cranial nerve involvement suggests the lesion is not in the spinal cord. An upper motor neuron lesion above the spinal cord is also unlikely since there is no hyperrflexia despite presence of deficits for over a week. In addition, the absent reflexes along with bilateral distal sensory involvement is not compatible with lower motor neuron disease, neuromuscular or muscle disorders since these conditions are not associated with sensory deficits, and do not usually cause reflex loss unless weakness is profound (dropped reflexes are typically due to sensory nerve involvement). Therefore, the most likely localization is a diffuse symmetric polyneuropathy.
2. Neuropathy can have many causes, but when it occurs days to weeks following an acute illness, with progressive motor greater than sensory involvement and absent reflexes, the most likely diagnosis is Guillain–Barré syndrome (see KCC 8.1), also known as acute inflammatory demyelinating polyneuropathy (AIDP). There are a few other causes of generalized rapidly progressive predominantly motor weakness including myasthenia gravis, heavy metal or organophosphate toxicity, diphtheria, botulism, Lyme polyradiculitis, porphyria, poliomyelitis, and tick paralysis. However, these other disorders can usually be distinguished from Guillain–Barré syndrome based on clinical features (see KCC 8.1) and diagnostic tests including nerve conduction studies and lumbar puncture (see KCC 5.10, 9.2).

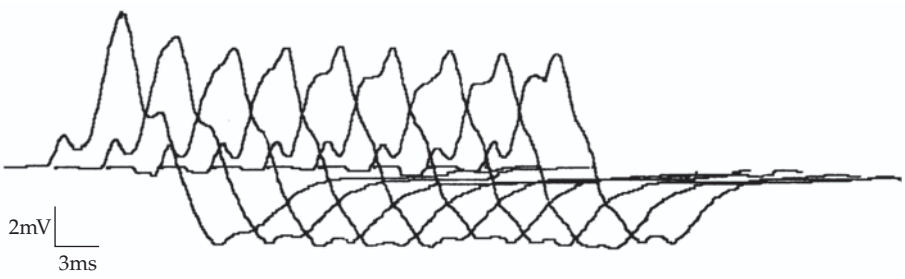
Diagnostic Studies and Clinical Course

Although there appeared to be cranial nerve involvement, since the patient was elderly and had some degenerative disease of the spine, an urgent MRI of the cervical spine was performed to rule out spinal cord compression as the cause of her progressive weakness and sensory loss. The MRI did not reveal cord compression, enabling a **lumbar puncture** (see KCC 5.10) to be performed safely. Cerebral spinal fluid showed 2 white blood cells/mm^3 (67% lymphocytes and 31% monocytes), 2 red blood cells/mm^3, normal glucose of 69 mg/dl, and elevated protein of 115 mg/dl (see Table 5.7). This elevated CSF protein with normal cell count is referred to as **albuminocytologic dissociation** and is evidence of the autoimmune response against nerves and nerve roots characteristically seen in Guillain–Barré syndrome. **Nerve conductions studies** (see KCC 9.2) revealed markedly reduced conduction velocities, and somewhat reduced compound motor action potential (CMAP) amplitudes, compatible with greater demyelination than axonal loss, and confirmed the diagnosis of Guillain–Barré syndrome.

The patient was treated with a course of intravenous immunoglobulin daily for five days, but by the second hospital day, her vital capacity had dropped to 800cc and negative inspiratory force to –20 mm H_2O, so she was electively intubated. Her strength gradually improved, and by hospital day 5 she was successfully extubated and was able to breathe on her own. By the time of discharge from the hospital, 9 days after admission, her strength was 4/5 to $4^+/5$ in the upper and lower extremities, and she no longer had diminished vibration sense in the feet. She was last seen as an outpatient seven months later and had normal nerve conduction velocities, and a normal neurological exam including 5/5 strength throughout, 2^+ reflexes and a normal gait.

CASE 9.12 DYSARTHRIA, PTOSIS, AND DECREASED EXERCISE TOLERANCE

IMAGE 9.12 Decrement on Repetitive Stimulation
Repetitive stimulation testing was performed by stimulating the ulnar nerve of the right arm and recording the CMAP over the right abductor digiti minimi muscle (see Figure 9.9; KCC 9.2). Stimulation was repeated at 3 per second for a total of 9 stimuli. Successive stimuli are shown displaced sequentially to the right (3 ms displacement per stimulus) to allow comparison of amplitudes of successive CMAPs: The first CMAP in the series is displayed farthest to the left, and the ninth CMAP is displayed farthest to the right. There was a decrement in CMAP amplitude of 23%.



CASE 9.14 MYSTERIOUS WEAKNESS AFTER DINNER*

MINICASE

One evening after dinner, a 58 year old woman began to have **swallowing difficulties** and **double vision**. Over the next few hours she developed **ptosis**, **facial weakness**, and **difficulty breathing** so her family brought her to the emergency room. Examination was notable for normal mental status, **severely limited horizontal and vertical eye movements with normally reactive pupils**, **facial diplegia**, **dysarthria**, **weakness of the bilateral arms and legs worse proximally than distally, 1+ patellar reflexes but otherwise undetectable deep tendon reflexes**, and a normal sensory exam. **Vital capacity was 600 cc** (normal is greater than ~3.5 L for an adult woman) so she was intubated.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What are some possibilities for the diagnosis?

*A description of this patient was published previously in Shapiro BE, Soto O, Shafqat S and Blumenfeld H. 1997. *Muscle and Nerve*, 20: 100–102.

Discussion

The key symptoms and signs in this case are:

- **Diplopia, limited horizontal and vertical eye movements, ptosis, facial diplegia, dysarthria and dysphagia**
- **Proximal arm and leg weakness**
- **Breathing difficulty, with reduced vital capacity**
- **Diminished reflexes**

Generalized weakness without sensory loss can be caused by a brainstem lesion (see KCC 14.1) or by a widespread disorder of the lower motor neurons, peripheral nerves, neuromuscular junctions, or muscles (see KCC 6.3, Generalized Weakness or Paralysis). Proximal greater than distal weakness suggests a muscle or neuromuscular junction disorder, but the diminished reflexes suggest a possible acute polyneuropathy such as Guillain–Barré syndrome (see KCC 8.1). Another possibility is an acute upper motor neuron lesion, which can sometimes be associated with decrease rather than in-

creased reflexes (see Table 6.4). In summary, the differential diagnosis is large and includes an acute brainstem lesion such as infarct or hemorrhage, and rapidly progressive peripheral disorders such as Guillain–Barré syndrome, myasthenia gravis, heavy metal or organophosphate toxicity, botulism, porphyria, poliomyelitis, and tick paralysis (see KCC 8.1).

Clinical Course

The patient was intubated and admitted to the intensive care unit. MRI with MRA did not reveal any brainstem abnormalities, routine blood tests and cerebrospinal fluid were normal. The family and patient repeatedly denied any possible toxin exposure. **Nerve conduction studies** demonstrated normal conduction velocities but markedly diminished compound motor action potential (CMAP) amplitudes (see KCC 9.2). In addition, fast repetitive stimulation or strong voluntary muscle contraction caused CMAP amplitudes to increase 2- to 3-fold. Increment of this kind is usually seen in presynaptic disorders of the neuromuscular junction, such as Lambert-Eaton myasthenic syndrome or botulism (see KCC 8.1; 9.2).

On further questioning, the family admitted to operating a home canning operation. They revealed that the patient had prepared a spaghetti dinner using their canned tomato sauce on the day of admission. Stool specimens and residual sauce brought in by the family were positive for botulinum toxin type B. She was treated with botulinum antitoxin, and had a prolonged course in the intensive care unit, but eventually recovered fully. When she was extubated and able to talk again, she explained that she had opened a dented can, and the sauce did not smell right, so she tried to feed it to the dog. The dog refused to eat it (wise choice), but the patient tasted some of the raw sauce which seemed alright, so she then cooked it and fed the whole family a spaghetti dinner. Fortunately, she cooked the sauce long enough to inactivate the toxin.

Additional Cases

Other chapters describe related cases for the following topics: **radiculopathy** (Cases 8.1–8.11); **distal symmetric polyneuropathy** (Cases 6.5 and 10.3); and **cranial neuropathy** (Cases 12.2–12.7, 13.1–13.3, and 13.5). Other relevant cases can also be found using the **Case Index** located at the end of this book, and new cases are also available through the **Online Review and Study Guide**.

Brief Anatomical Study Guide

1. The **brachial plexus** arises from **C5 through T1** (see Figure 9.2), while the **lumbosacral plexus** arises from **L1 through S4** (see Figure 9.4).
2. The most clinically important nerves in the upper extremity are the **radial, median**, **ulnar**, **axillary**, **and musculocutaneous nerves**; Table 9.1 summarizes the sensory and motor functions of these nerves.
3. The most important nerves in the lower extremity are the **femoral**, **obturator**, **sciatic**, **tibial**, **and peroneal nerves**; Table 9.3 summarizes the sensory and motor functions of these nerves.