CLASS 8: Syndromes of PNS lesions: lesions of radial, femoral, sciatic and other peripheral nerves.

Radial N. (C5−C8)

- **Anatomy.** Radial nerve provides motor innervation to the *triceps brachii*, *brachioradialis*, and *supinator mm.*, as well as all of the *extensors of the wrist, thumb, and finger joints.* Its sensory innervation is to the dorsal skin of the upper arm and forearm aswell as the dorsum of the hand, with an autonomic zone located between the first and second metacarpal bones.
- **Typical deficits.** The clinical manifestations of radial nerve palsy depend on the level of the lesion:
- **Lesion in the upper arm:** the radial n. is particularly vulnerable to injury in the radial nerve canal of the humerus, because it lies directly on the bone at this location. The corresponding, readily apparent neuro-logical deficit is a **wrist drop** (Fig. 12.**31**), attributable to loss of action of the wrist and finger extensors. In addition, sensation is diminished on the radial portion of the dorsum of the hand.

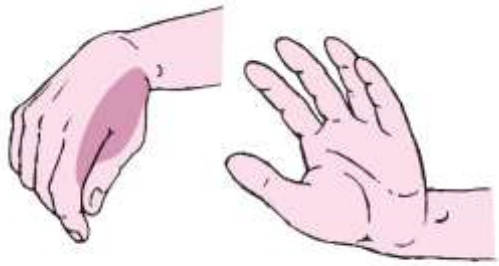

Fig. 12.31 **Right wrist drop** due to a radial nerve lesion. The shading indicates the sensory autonomous area in the distribution of the superficial branch of the radial n.

- **"High radial nerve lesion":** if the nerve is injured more proximally in the upper arm or in the axilla, the triceps brachii m. is also weak and the elbow can no longer be actively extended against resistance.
- **Supinator canal syndrome:** if the radial n. is compromised at the site of its passage through the supinator m., only its deeply penetrating *motor terminal branch* is affected. The resulting deficit is purely motor. The branch to the extensor carpi radialis m. and the brachioradialis m., which leaves the nerve proximal to its passage through the supinator m., is unaffected, but all of the other forearm muscles innervated by the radial n. are paretic. Finger extension is impaired, but wrist extension is preserved, particularly on the radial side (Fig. 12.**32**).

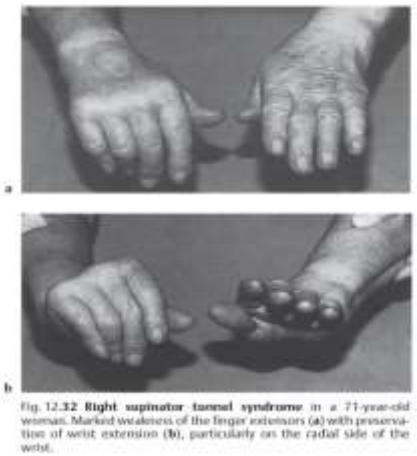

Fig. 12.32 **Right supinator tunnel syndrome** in a 71-year-old woman. Marked weakness of the finger extensors (**a**) with preservation of wrist extension (**b**), particularly on the radial side of the wrist.

- **Causes.** Radial nerve lesions can be produced by *trauma* and by *pressure*, e. g., by the use of crutches that press in the axilla, or by external pressure on the upper arm (humerus). The supinator canal syndrome is an anatomical bottleneck (*entrapment*) syndrome.

Median N. (C5−T1)

- **Anatomy.** All of the muscles innervated by this nerve are distal to the elbow. In the forearm, these include most of the *long flexors of the fingers* (with the exception of the deep flexors of the fourth and fifth fingers, which are innervated by the ulnar n.), as well as the *flexor carpi radialis*, *pronator teres*, and *pronator quadratus mm.* After the nerve passes through the carpal tunnel together with the long flexor tendons, it innervates most of the *thenar muscles* (abductor pollicis brevis and opponens pollicis m. and the superficial head of the flexor pollicis brevis m.), as well as the *first and second lumbrical mm.* Its sensory innervation is to the radial side of the palm, the volar surface of the fingers from the thumb to the radial half of the fourth finger, and the dorsal surface of the terminal phalanges of these fingers.
- **Typical deficits.** In median nerve lesions, too, the clinical manifestations depend on the level of the lesion:
- **Median nerve lesion in the upper arm** (i. e., proximal to the origin of its motor branches to the forearm flexors): the typical clinical appearance is that of the "pope's blessing hand," as depicted in Fig. 12.**34**, caused by weakness of the radial finger flexors.

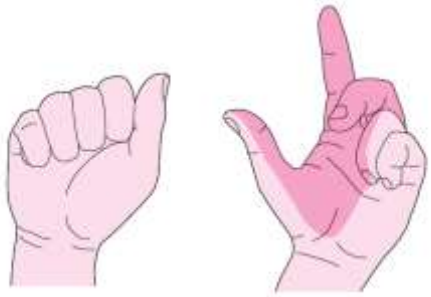

Fig. 12.**34** **"Preacher's hand" due to a proximal left median nerve lesion.** The hypesthetic area is shaded dark red.

- **Median nerve lesion at the wrist.** A lesion of the median n. in the carpal tunnel causes weakness of the thenar muscles. Clinically, pain and paresthesia are the most prominent symptoms. Carpal tunnel syndrome is discussed separately, in detail, because of its special clinical importance (see below).
- **Kiloh−Nevin syndrome.** An *isolated lesion of the anterior interosseous n.* is a rare event. This nerve is the motor terminal branch of the median n., which innervates the flexor pollicis longus m., the radial portion of the flexor digitorum profundus m. (flexion of the terminal phalanges of the 2nd and 3rd fingers), and the pronator quadratus m. A lesion of this terminal branch—due either to trauma or, occasionally, to entrapment—mainly impairs flexion of the terminal phalanges of the thumb and index finger. The patient can no longer form an "O" with these two fingers.
- **Causes.** The median n. is the nerve most frequently injured by *direct trauma*, often by a cut in the wrist. *Pressure palsies* of the median n. also occur, both in the upper arm (due to prolonged maintenance of an awkward position, or to an Esmarch tourniquet) or in the palm of the hand (e. g., in occupational injuries). *Compression at anatomical bottlenecks* (entrapment) is a further cause of median nerve lesions. In many individuals, a bony spur is present just above the medial epicondyle of the humerus (the *supracondylar process*). A fibrous band (of Struther) may run from this spur to the medial epicondyle, forming a tunnel through which the median n. passes. The nerve can be compressed either by the supracondylar process or by the fibrous band. Further compression syndromes affecting the median n. are the *Kiloh−Nevin syndrome* and the *carpal tunnel syndrome*.

Ulnar N. (C8–T1)

- **Anatomy.** Among the muscles innervated by this nerve, the ulnar flexors of the wrist and fingers (the flexor carpi ulnaris m. and the ulnar portion of the flexor digitorum profundus m.) are functionally much less important than the ulnar-innervated intrinsic *muscles of the hand.* The ulnar n. is, indeed, the most important nerve for finger function: it innervates not only the *hypothenar mm.*, but also all of the *interossei*, the *3rd and 4th lumbrical mm.*, and, in the thenar region, the *adductor pollicis m.* and the *deep head of the flexor pollicis brevis m.* It provides sensory innervation to the ulnar edge of the hand, the volar surface of the fifth finger, and ulnar half of the fourth finger. A sensory branch arising from the ulnar n. in the distal third of the forearm innervates the skin on the ulnar side of the dorsum of the hand, as well as on the dorsal surface of the fifth finger and the ulnar half of the fourth finger.
- **Typical deficits.** The typical clinical picture of ulnar nerve palsy is a **claw hand** (Fig. 12.**39**): because the interossei and lumbrical muscles cannot contract, the ulnar digits are hyperextended at the metacarpophalangeal joints, while the remaining digits are flexed at these joints. The long fingers can no longer be fully adducted against one another, the fingers cannot be strongly spread apart, and the patient cannot flick the middle finger against the examiner's palm with full, normal strength. A key finding is that, when the patient grasps a flat object (such as a piece of paper) between the thumb and the index finger, weakness of the adductor pollicis m. (ulnar n.) leads to functional substitution by the flexor pollicis longus m. (median n.), and therefore to flexion of the thumb on the affected side at the interphalangeal joint. This finding, called *Froment sign*, is highly characteristic of ulnar nerve palsy (Fig. 12.**40**).

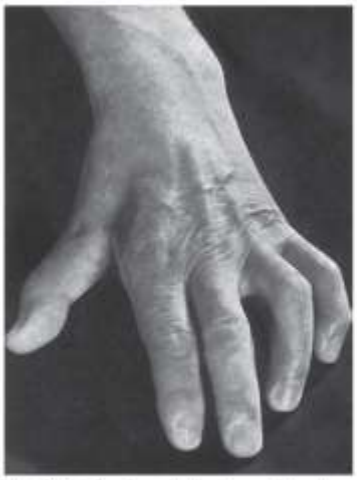

Fig. 12.**39** **Claw hand due to a right ulnar nerve lesion at the elbow.** Typical features include hyperextension at the metacarpophalangeal joints and hyperflexion at the interphalangeal joints, particularly on the ulnar side of the hand. There is marked atrophy of the interossei and of the hypothenar muscles.

Fig. 12.**40** **Froment sign in right ulnar nerve palsy.** Flexion of the interphalangeal joint of the thumb when the patient pulls on a flat object (piece of paper).

Femoral nerve (L1–L4)

Anatomy. This nerve provides:

- motor innervation to the hip flexors (iliacus and psoas major muscles) and the knee extensors (quadriceps femoris muscle)

- sensory innervation by the anterior cutaneous branches to the anterior surface of the thigh and by the saphenous nerve to the medial quadrant of the anterior surface of the lower leg.

Deficits. Motor dysfunction – the lesion of the nerve impairs hip flexion and knee extension. The hip flexors are examined with the patient sitting up and the knee extensors are examined with the patient supine. In the standing patient, a low-lying patella is seen on the side of the lesion.

The patellar tendon reflex is absent. The patient cannot climb stairs with the affected leg and the leg is in a hyperextended position whilewalking.

Sensation is diminished on the anteromedial surface of the thigh and medial surface of the lower leg.

Lesions of the femoral nerve can be traumatic or iatrogenic (surgery). The nerve can also be damaged by a pelvic tumor or by a hematoma in the psoas sheath, e.g., in an anticoagulated patient.

Sciatic nerve (L4–S3)

Anatomy. The sciatic nerve is the common trunk of the fibular (=peroneal or common peroneal) and tibial nerves. It is the longest and thickest nerve in the human body. The proximal portion of sciatic nerve gives off cutaneous branches to the buttock and the posterior surface of the thigh (the inferior cluneal nn. and the posterior femoral cutaneous n.). It gives off motor branches to the knee flexors (the semimembranosus, semitendinosus and biceps femoris muscles).

Deficits. Proximal lesions produce hypesthesia on the buttock and the posterior surface of the thigh and impair knee flexion

The sciatic nerve trunk can be injured by fractures of the pelvic ring or proximal portion of the femur, by surgical procedures in the region of the hip, or by faultily delivered injections. Tumors are a less common cause of sciatic nerve palsy.

Fibular nerve (L4–S2)

Anatomy. The fibular (peroneal or common peroneal) nerve travels to the lateral margin of the popliteal fossa, winds around the fibular neck, and then enters into the body of the fibularis longus (peroneus longus) muscle, where it divides into the superficial and deep fibular (peroneal) nerves. The superficial fibular (peroneal) nerve provides motor innervation to the fibular (peroneal) muscles and sensory innervation to the lateral surface of the lower leg and the dorsum of the foot, with the exception of the space between the first and second toes (the first interosseous space). The latter is supplied by the deep fibular (peroneal) nerve, which also innervates the dorsiflexors of the foot and toes and the intrinsic muscles of the dorsum of the foot.

Deficits. The clinical manifestations of a lesion of the deep fibular nerve include foot drop and steppage gait. Sensation is impaired on the dorsum of the foot and completely abolished in the first interosseous space. A lesion of the superficial fibular n. causes weakness of pronation of the foot (i.e.,inability to elevate the lateral edge of the foot);when the patientwalks, the lateral edge of the foot hangs downward. Sensation is impaired in the lower legand on the dorsum of the foot.

The fibular nerve can be injured by penetrating or blunt trauma, e.g., by knee fractures. Injection palsies of the sciatic nerve usually affect its fibular portion. The most common cause of fibular nerve palsy is compression of the nerve at the fibular neck by local, external pressure (faulty surgical positioning) that is spontaneously reversible.

Tibialis anterior syndrome. It is caused by infarction (due to compression) of the muscles in the anterior compartment of the lower leg, because of overuse, trauma, or a hematoma. Clinical manifestations are an intense local pain and then a muscle swelling. The pain increases on passive extension of the muscles by plantar flexion of the foot. The muscles become necrotic in 12 to 24 hours and are later replaced by connective tissue. The resulting contracture prevents the appearance of the flaccid foot drop that is otherwise characteristic of fibular nerve palsy. In the acute phase the deep fibular nerve can be damaged, because its course passes through the anterior compartment of the lower leg.

Tibial N. (L4–S3)

Anatomy. This nerve innervates the plantar flexors of the foot and toes in the lower leg, as well as all of the intrinsic muscles of the foot, except those on the dorsum. It provides sensory innervation to the heel and sole.

Deficits. Weakness of plantar flexion makes tiptoe walking impossible, while weakness of the intrinsic muscles of the foot makes the patient unable to fan the toes. The sensory deficit on the sole of the foot is particularly troublesome.

Tarsal tunnel syndrome is an entrapment neuropathy affecting the terminal branch of the tibial nerve as it passes under the medial malleolus. It is seen almost exclusively after fractures or sprains of the upper ankle joint. Its typical feature is local pain behind the medial malleolus or on the sole of the foot, which increases when the patient walks. The nerve trunk is tender to palpation behind the medial malleolus. Sensation is diminished on the sole of the foot and the plantar skin is abnormally smooth and dry. The patient can no longer fan the toes.

Morton metatarsalgia. A painful neuroma can develop on a digital nerve (a sensory terminal branch of the tibial nerve) if the nerve is chronically injured by being compressed between two adjacent metatarsal heads. It is called Morton metatarsalgia, causes pain in the forefoot, which is initially felt only on walking, but later also at rest.