# Peripheral nerve lesions

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# Abstract

Compressive peripheral nerve lesions commonly present for surgical consultation and research continually points to an optimal window of opportunity to better functional outcome. Here we provide a review of the anatomy, aetiology, clinical presentation, examination techniques and management of peripheral nerve pathology concentrating in the upper and lower limbs.

We provide table summaries of peripheral nerve lesions and examination of all muscle groups in the upper and lower limbs, for your clinical revision aid.

**Keywords** Carpal tunnel; femoral foot drop; median; neuropathy; radial; sciatic; ulnar

### Introduction

In clinical practice it is the surgeon's prerogative to localize the peripheral nerve lesion though a succinct history and focused clinical examination accompanied by the ability to evaluate and apply the appropriate investigations and management.

### Definitions

Peripheral nerves serve to connect the central nervous system with the motor and sensory, somatic and visceral end organs. They include cranial nerves III-XII, the 31 pairs of spinal nerves, nerves of the extremities and cervical, brachial and lumbosacral plexi.

#### Aetiology

Peripheral neuropathies can be classified into non-compressive and compressive not forgetting it is possible for an element of both. The compressive more commonly present in the surgical setting however, a knowledge of the aetiology of noncompressive peripheral neuropathy is necessary for differential exclusion and for evaluation of timely surgical intervention. Such aetiology is extensive covering systemic disease such as metabolic and endocrine disorders, renal disease, hormonal imbalances, vitamin deficiencies, alcoholism, vascular disease, connective tissue disorders, repetitive stress; toxins, infections and autoimmune disorders such as HIV, Lyme disease,

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**David Choi** is a Consultant Neurosurgeon at The National Hospital for Neurology and Neurosurgery, London, UK. Conflicts of interest: none declared. diphtheria, leprosy, inflammation, and inherited forms of peripheral neuropathy such as Charcot-Marie Tooth deformities.<sup>1</sup>

This article will now focus on compressive peripheral nerve lesions. Typically there will be a history of trauma, operations, previous fractures, abnormal limb posturing, repetitive activities and recent change in body habitus.

# Pathology and pathogenesis

Nerve injuries trigger complex cell–molecular interactions in Schwann cells (SC) which create a growth-permissive environment essential for axonal regeneration. Neurons of the peripheral nervous system are required to regenerate their axons over long distances at a rate of 1 mm/day resulting in re-establishment of the functional motor-unit taking months or years, a condition referred to as chronic axotomy. Therefore optimizing outcome is inversely related to early intervention with neurobiological techniques focusing on accelerating the regenerative phase.<sup>2</sup>

## Diagnosis

The history is accompanied by symptoms of numbness, tingling, pain, general clumsiness and accumulative, inadvertent injuries. Such symptoms can be exacerbated when the limb is in certain positions (often at night) and improved by positional change or 'shaking out the limb'. Few patients will present with florid signs on inspection of a peripheral nerve lesion and therefore signs of a lower motor neuron lesion are elicited via examination manifesting as flaccid tone, absent tendon reflexes, absent pathological reflexes, fibrillation (detected by electromyography) and or fasciculation's (detected by the naked eye).

The differential diagnosis of these symptoms includes upper motor neuron lesions, non-compressive lesions, compartment syndrome and vascular disease.

The investigations to consider for peripheral neuropathy are basic blood tests including thyroid function, glucose,  $B_{12}$  and folate; neurophysiology referral for nerve conduction studies; an MRI scan of the spine (usually cervical or lumbar) to rule out cord or root impingement. In selected cases, where the features of the neuropathy appear atypical, consider an MRI scan of the brain to rule out an intracranial cause.

#### Assessment

Tables 1–3 address how to test the muscles of the upper and lower limb. Box 1 summarizes salient features to elicit in the history and examination.

#### Upper limb peripheral nerve lesions

#### Median nerve

The median never is motor and sensory. It forms from the lateral and medial cords of the brachial plexus. It passes medially in the upper arm, lateral to the brachial artery and enters the forearm via the cubital fossa. In the forearm, the median nerve courses between the two heads of pronator teres, entering the hand via the carpal tunnel. In the upper arm the median nerve does not give off any branches. In the forearm, the anterior interosseous nerve (AIN), a pure motor nerve, arises. The AIN supplies flexor pollicis longus (FPL), flexor digitorum profundus 1 (FDP1), FDP2 and pronator quadratus. Two branches of the median nerve arise

# Summary of peripheral nerve lesions<sup>3-5</sup>

Nerve	Roots	Muscles	Motor deficit	Sensory	Appearance	Sites of damage
Long thoracic Axillary	C5—7 C4—6	Serratus anterior Deltoid	Winged scapula Impaired arm abduction and	Regimental badge area	Wasted shoulder with loss	Chest wall Shoulder dislocation
		Teres minor	external rotation		of normal contour	
Musculocutaneous	C5—6	Biceps Brachialis Coracobrachialis	Elbow flexion	Radial forearm	Wasted anterior arm	
Median	C6-T1	rotator teres, FCR PL FDS FPL, FDP1 and 2, PQ (these are AIN) APB, OP, superficial head FPB, lumbricals 1 and 2	Pronation, wrist flexion, finger flexion, thumb movement, AIN syndrome purely motor		Simian hand, benediction hand, wasted thenar eminence	Axilla Elbow (AIN) Ligament of Struther's Pronator Carpal tunnel
Ulnar	C7-T1	FCU, FDP3 and 4, palmaris brevis, abductor digiti minimi, opponens digiti minimi, flexor difiti minimi, lumbricals 3 and 4, interossei, adductor pollicis, part of flexor pollicis brevis		Ring and little fingers	Clawed hand Ulnar paradox = a higher lesion leads to less clawing than a low lesion	Axilla Elbow Cubital tunnel Forearm Guyon's canal
Radial	C5—8	Triceps, anconeus, brachialis, brachioradialis, ECRL Supinator, ECRB, extensor digitorium, EDM, ECU, APL, EPL, EPB, EI (all PIN)	Elbow extension, wrist and finger extension. Supination, thumb extension and abduction	Dorsal forearm and anatomical snuffbox	Drop wrist	Axilla Spiral groove of humerus Elbow Forearm
Femoral	L1—4	Quadriceps, sartorius, pectineus	Weak hip flexion and leg extension. Weak lateral thigh rotation	Anterolateral thigh and medial leg (saphenous nerve)	Wasted anterior thigh	Pelvis, inguinal area, Hunter's canal
Sciatic	L5-S2	Hamstrings, part of adductor magnus, all muscles of the lower leg and foot (via tibial and peroneal branches)	Weak hip extension. Peroneal = foot drop, weak eversion. Tibial = weak ankle flexion and inversion	Lateral lower leg	Wasted foot muscles, foot drop	Pelvis, popliteal fossa, neck of fibula, leg compartments, tarsal tunnel

AIN, anterior interosseous nerve; PAL, adductor pollicis longus; ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; ECU, extensor carpi ulnaris; EDM, extensor digiti minimi; EI, extensor indicis; EPB, extensor pollicis brevis; EPL, extensor pollicis brevis; EPL, extensor pollicis longus; FCR, flexor carpi radialis; FCU, flexor carpi ulnaris; FDP, flexor digitorium profundus; FDS, flexor digitorum superficialis; FPB, flexor pollicis brevis; FPL, flexor pollicis longus; OP, opponens pollicis; PIN, posterior interosseous nerve; PL, palmaris longus; PQ, pronator quadratus.

Table 1

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