Part 1 – Clinical skills

CHAPTER 2
LOCALIZATION

Dr William P. Howlett
2012

CONTENTS

CHAPTER 2

LOCALIZATION

The site of the lesion

The nervous system can be divided into the central (CNS) and peripheral (PNS) nervous system. In the CNS the main sites of disease are the *cerebral hemisphere, basal ganglia, cerebellum, brain stem and spinal cord* (Fig. 2.1). In the PNS the main sites of disease are the *cranial* and *peripheral nerves*. Diseases of the neuromuscular junction and muscle are included by convention. The main aim of this chapter is to localize abnormal neurological findings to their main site of origin within the nervous system. After reading the chapter the student should aim to distinguish between an upper and lower motor neurone lesion and to be able to localise neurological disorders to their main site of origin. Details concerning localization and the cranial nerves are outlined in Chapter 12.
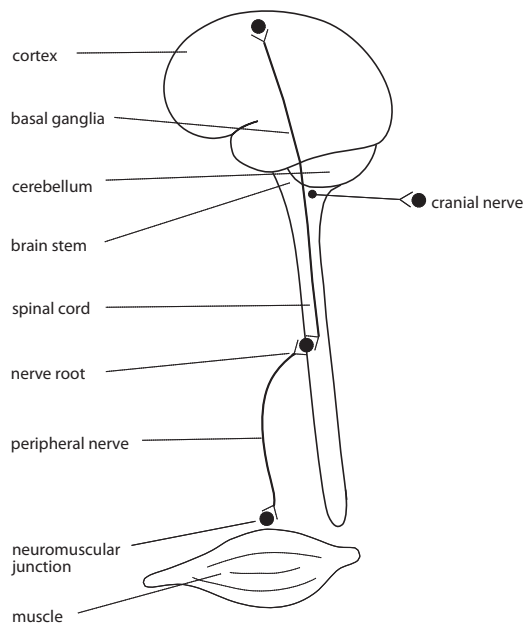
Figure 2.1 Main sites of neurological disorders

MOTOR SYSTEM

CNS

The motor system in the CNS consists of the brain and spinal cord, beginning in the motor cortex and extending down the brain stem and spinal cord to end at the lower border of L1. The motor tract begins in the frontal lobe, descends via the corona radiata on the same side to become the internal capsule. It then descends into the brain stem as the pyramidal tract and mostly (85%) crosses at the lower end of the medulla to the opposite side. From there it descends into the spinal cord as the lateral corticospinal tract and finally synapses with the anterior horn cells at the front (anterior) of the spinal cord on the same side (Fig. 2.2). A lesion anywhere along this pathway in the brain or spinal cord results in an upper motor

neurone lesion (UMNL). The neurological signs of an UMNL are *loss of power, increased tone (hypertonia), clonus, increased reflexes (hyper-reflexia)* and *extensor plantar response (up going toes or Babinski sign)*. The presence of these signs localise the site of the lesion to the CNS. The main UMN disorders presenting with these signs are hemiplegia arising from lesions in the brain and quadriplegia arising from lesions in the brain stem. The other main UMN disorders are quadriplegia and paraplegia arising from the spinal cord depending on the level of the lesion.
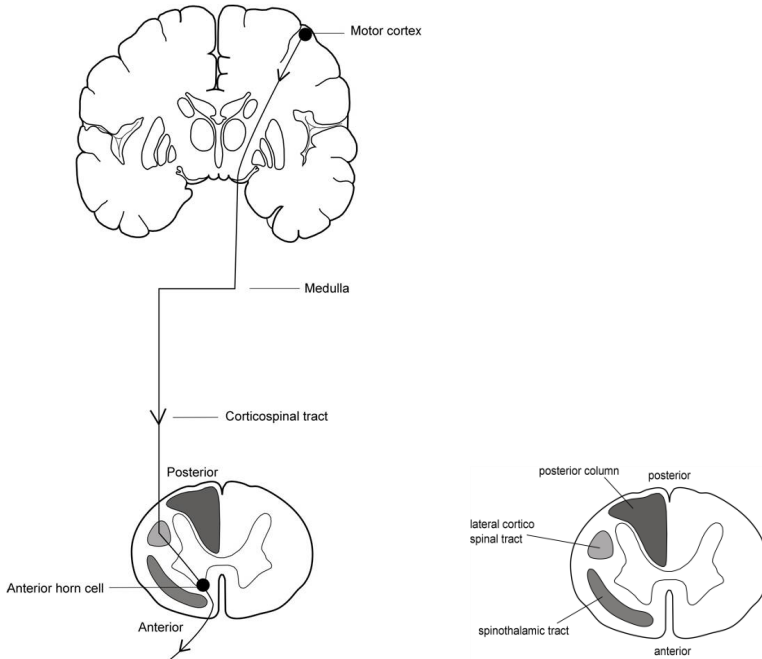
Figure 2.2 The corticospinal tract (left). Spinal cord section. Main motor and sensory tracts (right).

PNS

The motor system in the PNS consists of cranial and peripheral nerves, extending from their nerve nuclei in the brain stem and anterior horn cells in the spinal cord to the neuromuscular junction in muscle (Fig. 2.3). A lesion anywhere along this pathway is called a lower motor neurone lesion (LMNL). The neurological signs of a LMNL are *loss of power, muscle wasting, fasciculation, decreased tone (hypotonia)* and *decreased* or *absent reflexes (hyporeflexia or areflexia)*. The presence of LMN signs localise the site of the lesion to the peripheral nervous system. The main clinical disorders causing these signs are peripheral neuropathies, mononeuropathies and cranial nerve palsies.
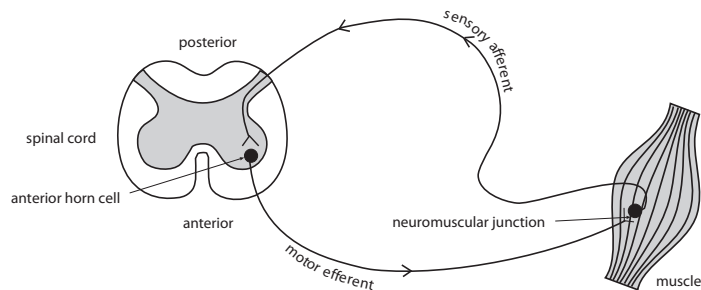
Figure 2.3 The peripheral reflex pathway

The diagram **"the peripheral reflex arc"** outlines the pathway of a peripheral reflex. It is essential to be able to distinguish clinically between an UMNL and a LMNL, in order to be able to correctly localise a neurological disorder to its main site of origin. It is important to note that loss of power is common to both and therefore does not help to distinguish between them, and that these signs may not all be present in any one individual patient. The main differences between them are summarized in the table 2.1 below.

Table 2.1 Key differences between UMNL & LMNL

Neurological findings	UMNL*	LMNL*
wasting		yes
fasciculations		yes
tone		
increased	yes	
decreased		yes
clonus	yes	
reflexes		
increased	yes	
decreased/absent		yes
Babinski sign	yes	

a blank space means no

SENSORY SYSTEM

The sensory system comprises of nerves and tracts which carry stimuli arising from the periphery including skin, joints, muscle and viscera via the peripheral nervous system (PNS) to the brain. In the CNS there are two main sensory pathways, *the dorsal columns* and *the spinothalamic tracts* (Fig. 2.4). The dorsal columns transmit joint position sense, vibration sense

and light touch. The peripheral nerves transmitting these sensations enter via enter the posterior roots of the spinal cord and ascend in the dorsal columns to the lower end of the medulla, where they synapse. They then cross the midline and ascend to reach the thalamus, from where a further relay goes to the sensory cortex in the parietal lobe of the brain on the same side. Sensory symptoms arising from the posterior columns are *numbness, tingling* and *loss of co-ordination*. The spinothalamic tract transmits pain, temperature and crude touch. These enter the posterior spinal cord ascend a few segments, and then cross the midline to ascend in the anterolateral spinothalamic tract via second order neurones to the ipsilateral thalamus, and finally to the parietal lobe on the same side. The main sensory symptoms arising from disorders of the spinothalamic tract are *pain* and *dysaesthesia*. Sensory symptoms arise from disease at different levels in the nervous system. The main sensory sites of clinical interest are at the level of *peripheral nerves, spinal cord* and *brain*.
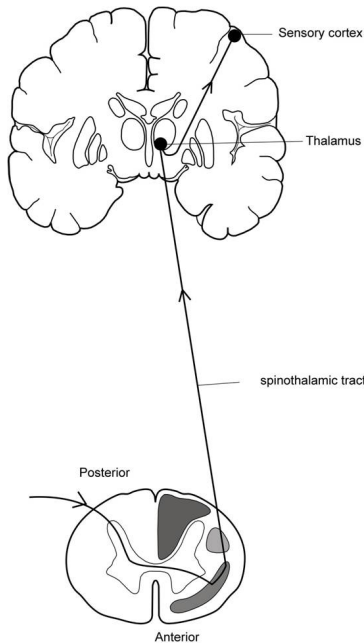
Figure 2.4 The spinothalamic tract (left).
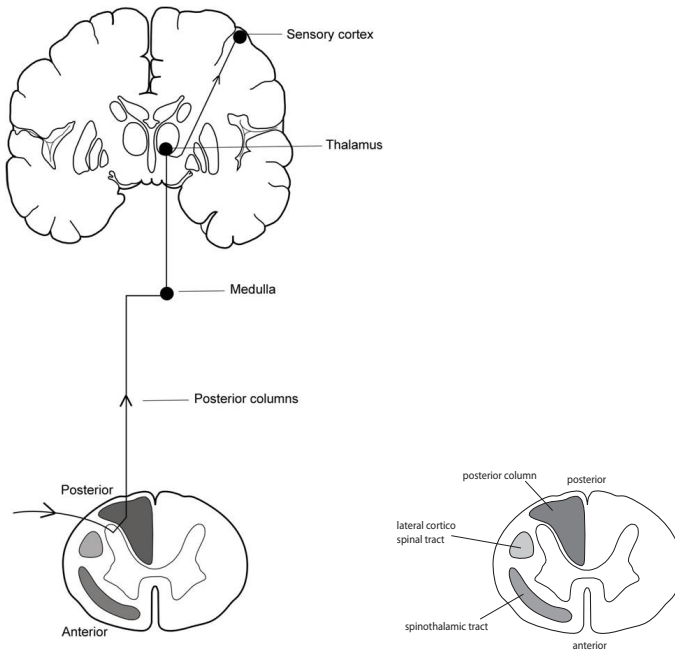
Figure 2.4 continued
Spinal cord section. Main motor and sensory tracts (right). The posterior columns (left).
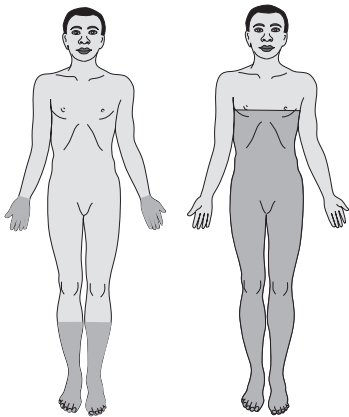
The anatomical pattern of sensory loss helps to localise the site of the underlying disorder. The main patterns of sensory loss are outlined in the following figures 2.5–7.

Figure 2. 5 Sensory loss in peripheral neuropathy glove and stocking distribution (left)

In **peripheral nerve disorders** the most common pattern of loss is that seen in peripheral neuropathies where the *loss* is mainly *distal* and affects *the feet* and *hands* in a *glove* and *stocking distribution.* The main causes of this are HIV and diabetes mellitus.

Figure 2.6 Sensory loss in paraplegia (right)
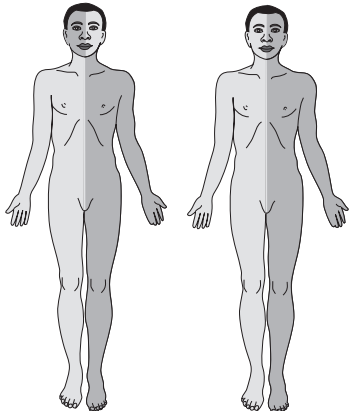
In **spinal cord disorders** the *loss involves* the *limbs* (usually the legs) and the *trunk below* the *level* of the *lesion.* The extent and pattern of loss depends on the underlying lesion, e.g. complete or partial cord involvement resulting in paraplegia. The main causes are trauma and infection.

Figure 2.7 Sensory loss in brain stem lesion (left). Sensory loss in hemiplegia (right).

In **brain disorders** sensation is *lost* or more commonly *altered* on the *side of the body opposite* the site of *lesion*. The main causes are vascular and space occupying lesions.

CEREBRAL HEMISPHERES

The brain has two hemispheres, each containing a frontal, parietal, temporal and occipital lobe, each lobe with their own distinctive functions. The main neurological signs indicating a lesion on one side of the brain are a *loss of power* or less frequently *sensation* on one side of the body, *a loss of speech* if the dominant hemisphere is involved and *a loss of vision* to one side if the optic pathway is involved. The presence of these focal neurological signs help to localize the site of the lesion to a cerebral hemisphere on one side and also to an individual lobe within that hemisphere (Fig. 2.8).

Frontal lobe
hemiparesis
expressive dysphasia (dominant)
social disinhibition
urinary incontinence

Parietal lobe
sensory impairment
receptive dysphasia (dominant)
apraxia sensory inattention
contralateral lower homonymous quadrantanopia

Temporal lobe
receptive dysphasia (dominant)
memory loss
contralateral upper homonymous quadrantanopia

Occipital lobe
contralateral homonymous hemianopia

Figure 2.8 Cerebral hemispheres

Frontal lobe

The frontal lobe contains the motor cortex, which is responsible for motor function and movements of the opposite half of the body. Disorders affecting either frontal lobe result in *weakness* or a *loss of power* involving the opposite side of the body and also a *loss* or *impairment (dysphasia) of speech (aphasia, expressive),* if the speech area (Broca's area) in the dominant hemisphere is affected. *Personality changes* with features of *social disinhibition* and *urinary incontinence* may also occur. *Frontal lobe release signs,* including the grasp reflex may also be present.

Parietal lobe

The parietal lobe contains the sensory cortex whose main function is discriminatory sensation involving the opposite half of the body. Patients with lesions in the parietal lobe have subtle sensory impairments, which require higher sensory testing to demonstrate. They have an *inability* to *recognise familiar shapes, textures* and *numbers* and an *impairment of fine touch* when tested on the opposite hand on either side. Lesions involving the dominant hemisphere result in *difficulty* with *calculation, writing* and *apraxia (difficulty performing task related movements)* and a *receptive dysphasia* if the dominant hemisphere is affected (Wernicke's area). Lesions involving the non dominant hemisphere result in a lack of *visuo-spatial awareness* with *hemi-neglect* of the opposite side of the body. This can result in an inability to dress or wash on the

affected side. A lesion in either parietal lobe may result in an *inferior quadrantic visual field defect* or a loss of the lower half of the visual field coming from the opposite side.

Temporal lobe

The temporal lobe contains Wernicke's receptive speech area in the dominant hemisphere. Damage to it results in loss of understanding of speech and writing *(aphasia, receptive)* and *loss of memory. Seizures* originating in the temporal lobe may begin with a characteristic *hallucinatory prodrome* of *smell, taste, vision, hearing* or *emotion.* A lesion in either lobe may result in a *superior quadrantic visual field defect* or loss of the upper half of the visual field coming from the opposite side.

Occipital lobe

The occipital lobe is responsible for vision. Lesions of the occipital lobe may result in a *contralateral homonymous hemianopia* or loss of the visual field coming from the opposite side.

SPEECH DISORDERS

There are three main types of speech disorder: *dysphonia, dysarthria* and *dysphasia.*

Dysphonia

This is a *disorder* of *voice production* of *sound* as air goes through the vocal cords. It results in inability to produce a normal volume of speech or sound. It is usually recognized during the history taking, because the sound the voice generates is low, hollow or hoarse. It arises from failure of adduction of the vocal cords due either to paralysis or to local disease in the larynx. It can be suspected by asking the patient to cough, when instead of the normal sharp explosive cough there is a characteristic husky or bovine like cough. The diagnosis is confirmed by inspection of the larynx and vocal cords. The main causes are a local lesion e.g. a tumour, recurrent laryngeal nerve paralysis, and myasthenia gravis.

Dysarthria

This is an *inability* to *coordinate* the *movements* of *tongue, lips* and *pharynx* to *articulate* or produce understandable sounds. This makes words sound slow and slurred and leads to difficulty in their understanding. Any neurological disorder which affects the muscles or movements involved in speech production can produce a dysarthria. The main causes are stroke, cerebellar disease, and cerebral palsy.

Dysphasia

This is a *disorder of language production* resulting in either a *loss* of *understanding* or *expression* of words or *both.* It arises because of damage to the speech areas in the brain in the dominant hemisphere. The main speech centres are situated on the left side of the brain in >90% of right handed people and also in about two thirds of left handed people. Dysphasia and aphasia are clinically classified as either receptive, expressive or global.

Key points

- listening determines the type of speech disorder
- it can be dysphonia, dysarthria or dysphasia
- pts with dysphonia can't make the sounds
- pts with dysarthria can make the sounds but the words don't sound normal
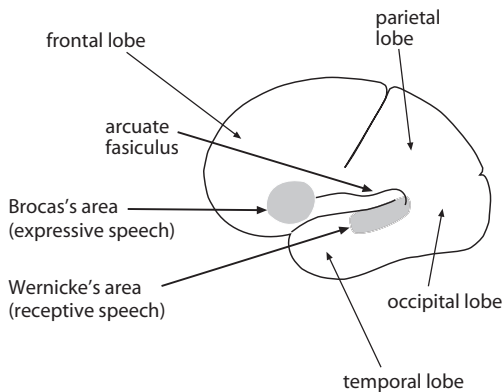- pts with dysphasia cannot either understand or say words normally or both

Figure 2.9 Main speech areas of the brain

Key points

- establish whether the patient is right or left handed and his/her language
- listen to speech for fluency, content & meaning
- assess reception by simple commands/questions e.g. "close your eyes"
- assess expression by asking the patient to name 3 familiar objects; e.g. pen, watch & glasses

Receptive

A patient with receptive aphasia *loses understanding* for both the *spoken* and *written word,* and is unable to follow even simple bedside questions and commands. The speech content is fluent but meaningless and many words are either incorrect or newly created. It is caused by a lesion in Wernicke's area (Fig. 2.9).

Expressive

A patient with expressive aphasia understands normally but *has difficulty* in *finding* the *words*. In this type of aphasia there are often great gaps between the words, with non fluent telegraphic or monosyllabic speech and lack of rhythm. Writing may be poor. The patient is usually aware, though frustrated and may use gestures to try to help to express. It is caused by a lesion in Broca's area in the dominant frontal lobe (Fig. 2.9).

Global

This occurs when the patient is neither able to *understand* the *spoken* or *written word* or able to *express* himself. It is caused by an extensive lesion of the dominant hemisphere, affecting both temporal and frontal speech areas. The main cause of aphasia is stroke.

> Key points
>
> · pts with receptive aphasia do not understand
> · pts with expressive aphasia cannot express the right words
> · pts with global aphasia can neither understand nor express the right words

BASAL GANGLIA

The basal ganglia and their connections control movement. Diseases affecting them cause movement disorders, which are characterised by either too *little* or too *much movement*. Parkinson's disease (PD) causes too little movement. The main clinical features of which are *bradykinesia, rest tremor, rigidity* and *gait disorder*. Disorders that result in too much movement include *dystonia* and *chorea*. The main causes are medications and stroke.

CEREBELLUM

The cerebellum and its connections coordinate voluntary movement. Disorders affecting the cerebellum cause incoordination. The main symptoms and signs of cerebellar disease are *dysarthria, nystagmus* and *incoordination* of the *limbs* and *gait*. Hypotonia and pendular reflexes are additional signs but may not always be present. Cerebellar signs are localizing and the presence of unilateral cerebellar signs help to localise a lesion to the cerebellum to the same side. The main causes are stroke, drugs including alcohol, hereditary disorders, tumours and forms of neurodegenerative disease.

BRAIN STEM

The brain stem (Fig. 2.10) comprises the midbrain, pons and medulla. It is responsible for the lower ten cranial nerves, the ascending motor and descending sensory tracts, integrating co-ordination and balance and the central regulation of heat, respiration, circulation and consciousness. The clinical features of a brain stem disorder will depend on the site and the extent of the lesion. In general a brain stem lesion is suggested when *cranial nerve palsies* and *ataxia* occur on *one side* of the *head* and *a loss of power* and *sensation* occurs on the *opposite half* of the *body*. An alteration or loss of consciousness and quadriplegia occurs with extensive brain stem lesions. The main causes are stroke, trauma and mass lesions.
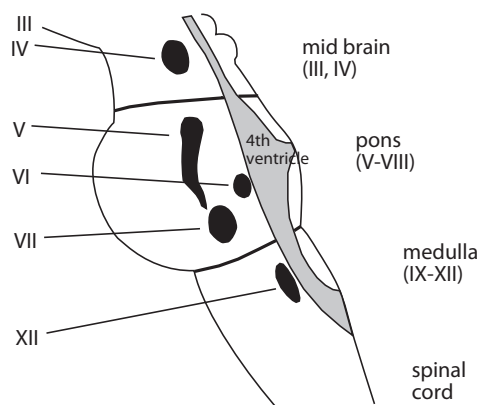

Figure 2.10 Brain stem. Main cranial nerve nuclei in the brain stem

SPINAL CORD

The spinal cord (CNS) extends from the top of C1 vertebra down to the end of L1. It is then continuous with the cauda equina (PNS) which extends from L1 down to S5 (Fig. 2.11).

The spinal cord is surrounded by three layers, a thick dura, an arachnoid and a pia which is adherent to the cord. The subarachnoid space contains cerebrospinal fluid and extends down to S5. The cord is made up of a large H shaped grey area in the centre containing many nerve cells and a peripheral white area which contains the ascending and descending axons or tracts (Fig. 2.12).
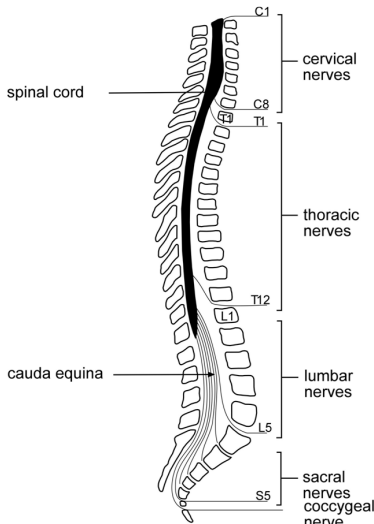

Figure 2.11 Spinal cord.
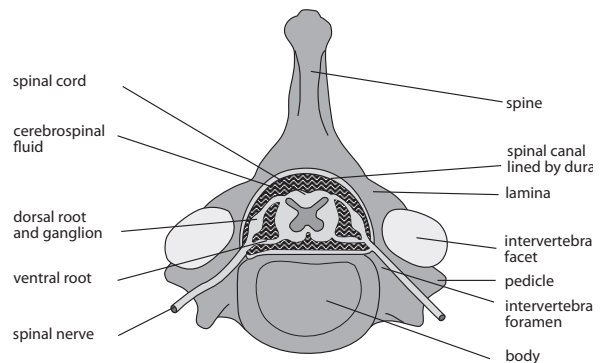Spinal nerve and vertebra coloumn

Figure 2.12 Vertebra showing spinal cord, nerve roots and spinal nerves

The main ascending tracts are the *spinothalamic* and *dorsal columns* (Fig 2.4) and the main descending tracts are the *corticospinal tracts* of which the *lateral* (LCST) is the main one (Fig 2.2). These main tracts all cross the spinal cord to supply the opposite side. The spinothalamic tract crosses shortly after entry and the posterior columns and the corticospinal tract cross at the lower end of the medulla. Any lesion affecting the spinal cord may result in *loss of motor, sensory* and *autonomic function below* the *level* of the *lesion.* Disorders affecting the spinal cord result in either a *quadriplegia* or *paraplegia,* depending on the site and level of injury or disease. The main causes are trauma, TB, infections, myeloneuropathies and peripheral neuropathies.

Paraplegia

Paraplegia means *paralysis of the legs*. This results from a lesion affecting either the spinal cord or the cauda equina. It can very rarely arise from a lesion within the brain. When the lesion affects the spinal cord above the level T12, L1 the clinical findings are those of *spastic paraplegia*. These **UMNL** signs are almost always combined with loss of sensation at and below the level of the lesion. The *sensory level* below which sensation is lost or reduced is very important as its upper limit usually indicates the site of the lesion. When the lesion affects the spinal cord below the level L1 it involves the cauda equina and the neurological findings are those of a *flaccid*

paraplegia. In this case the findings are all **LMNL** signs which when combined with upper limit of sensory loss in the legs and perineum will help to determine the site of the lesion.

Quadriplegia

Quadriplegia indicates *paralysis* of all *four limbs*. The clinical findings are the same as for paraplegia except in this case all four limbs are involved. When the site of the lesion is in the lower half of cervical cord there may be radiculopathy or lower motor neurone signs involving the arms (C5-T1) at the level of the lesion and upper motor neurone signs and sensory loss below the level of the lesion. Both spastic and flaccid forms of quadriplegia and paraplegia are associated with loss of bladder and bowel control.

Bladder

The bladder is innervated by the autonomic and somatic or voluntary nervous system. The autonomic supply comprises of the parasympathetic fibres from S2-4 which are involved in emptying the bladder and the sympathetic fibres from T11-L2 which are involved with urine retention. The voluntary nerve fibres (S2-4, pudendal nerve) supply the external bladder neck sphincter. Loss of control of bladder function or neurogenic bladder arises primarily because of lesions in the spinal cord or cauda equina. Patients with spinal cord lesions present with a spastic paraparesis and spastic bladder with frequency, urgency and incontinence. They may develop satisfactory reflex bladder emptying or require intermittent self-catheterization and anticholinergic drugs. Patients with cauda equina lesions present with flaccid paraparesis and flaccid bladder with urine retention and usually require a permanent urinary catheter. Constipation is a feature of both types of paraplegia.
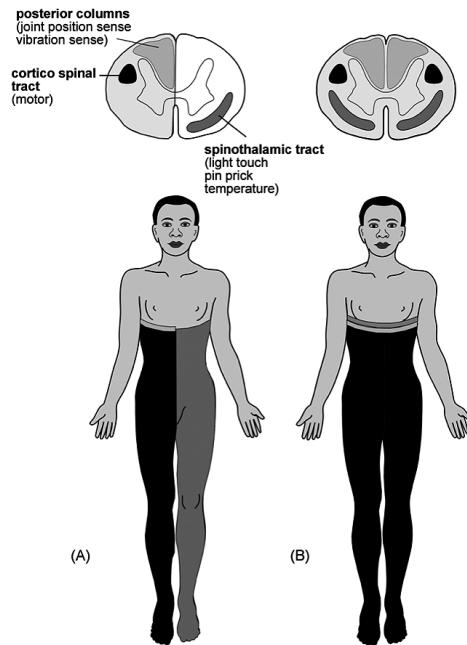

Figure 2.13 Sensory loss associated with spinal cord lesions. A: Hemisection of cord. B: Complete transverse section of cord

Localization

A lesion affecting one half of the spinal cord results in a hemi section or a *Brown-Sequard syndrome* (Fig. 2.13). This has three main and diagnostic neurological features all occurring below the level of the lesion. These are a *loss of power (UMNL)* and a *loss of joint position sense* and *vibration* occurring on the **same side** as the **lesion** and a *loss of pain* and *temperature* on the **opposite side** the **lesion**. A lesion causing a complete transverse section of the cord results in a *total loss of power* and *feeling* **below** the **level** of the **lesion** and *loss of bowel* and *bladder control* (Fig. 2.13).

PERIPHERAL NERVOUS SYSTEM

Peripheral nerves

The peripheral nervous system is made up of mixed motor and sensory fibres comprising the cranial and peripheral nerves. Details concerning cranial nerve disorders and localization are presented later in chapter 12. In general disorders affecting the peripheral nerves are divided into two main groups called neuropathies: *mononeuropathies* which involve single nerves and *polyneuropathies* which involve all nerves. The main causes of these are HIV, diabetes and leprosy.

The diagrams below are to help to remind the student of the following, a typical peripheral *reflex arc* using the knee jerk as an example (Fig. 2.14), the main *segmental motor movements* and their nerve roots (Fig. 2.15), the main *peripheral reflexes* and their *nerve root* of origin (Fig 2.16), and the main *sensory dermatomes* (Fig. 2.17).

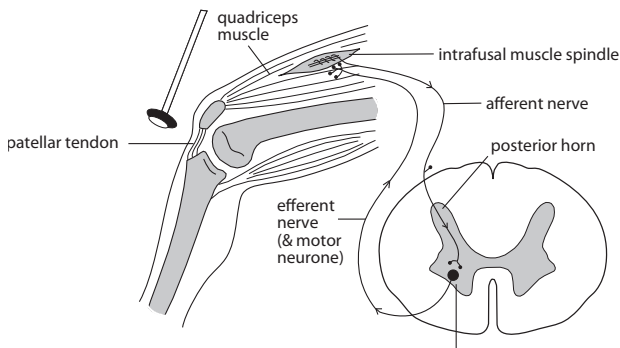**Figure 2.14** The knee reflex

The diagram entitled "the segmental limb movements" shows the main movements tested during motor neurological examination and their *nerve roots innervations*.
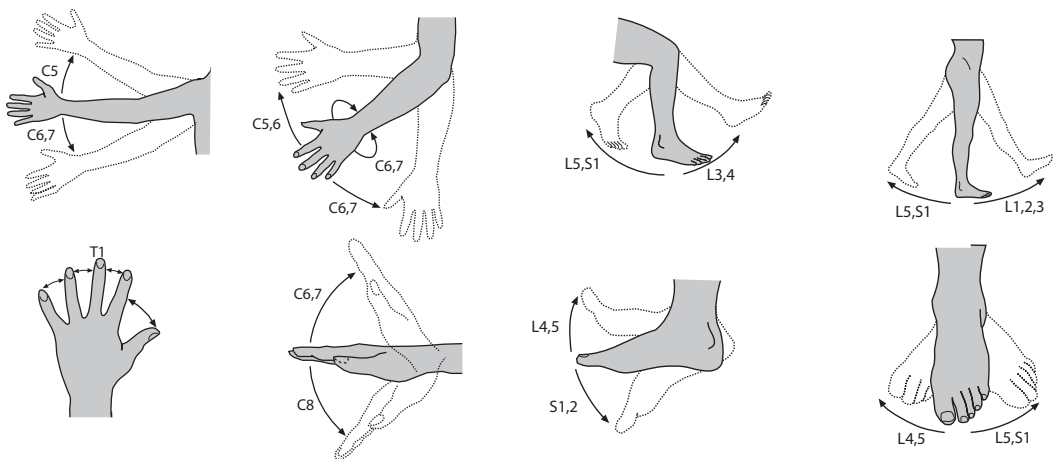
Figure 2.15 The segmental limb movements

The diagram marked *"reflexes"* shows a simplified method of remembering the nerve root origins of the main peripheral reflexes. This is done by "counting from 1 to 8 from below up".
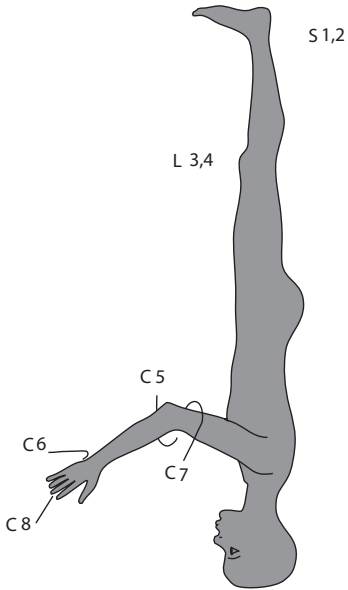

Figure 2.16 Reflexes. Count from the ankle

The diagram marked skin territories shows the *main sensory dermatomes* and their nerve root origin.
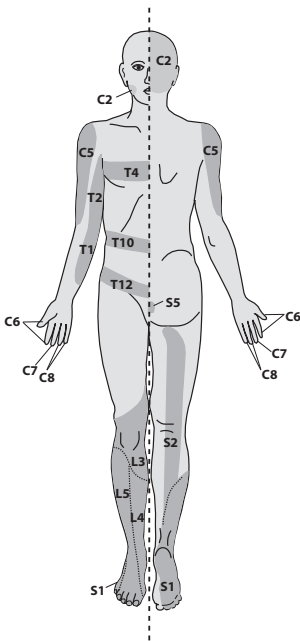

Figure 2.17 Skin territories of landmark nerve roots

The main sites of neurological disorders and the resulting patterns of motor and sensory loss are summarized in Tables 2.2 & 3.

Table 2.2 Main sites and patterns of motor loss

Site	Pattern of motor loss (weakness)
CNS (UMNL)	
hemisphere	hemiparesis
brain stem	cranial nerve palsies one side & hemiparesis on other side. quadriparesis & LOC*
spinal cord (C1-T12)	quadriparesis paraparesis
PNS (LMNL)	
spinal cord (L1-S5)	paraparesis
anterior horn cell	localised/generalized
nerve root	root distribution
nerve plexus	>one nerve root
mononeuropathy (cranial or peripheral)	single peripheral nerve
polyneuropathy	feet and legs > hands and arms

* these may occur in an extensive brain stem lesion

Table 2.3 Main sites and patterns of sensory loss

Site	Sensory loss
PNS	
single nerves	nerve distribution
all peripheral nerves	glove and stocking
spinal nerve roots	dermatomal
cauda equina	both legs & perineum
CNS	
Spinal cord	
complete transection	all below level of the lesion
hemisection (Brown Sequard Syndrome)	joint position/vibration/light touch on same side & pain/temperature on opposite side
Brain	
brain stem	face on same side as lesion & limbs on side opposite lesion
parietal lobes*	on side opposite the lesion a. numbness b. agnosia c. loss of two point discrimination d. astereognosis e. graphanaesthesia f. sensory inattention

* cortical sensory testing

Cortical sensory testing: Definitions

agnosia: abnormality of perception despite normal sensory pathways

two point discrimination: ability to determine whether one or two points are being applied at the same time (normal discrimination on finger tips: 2-3 mm)

astereognosis: inability to recognise familiar shapes and textures when felt in either hand with both eyes shut

graphanaesthesia: inability to identify numbers drawn on the palms with both eyes shut

sensory inattention: inability to correctly recognise and report a stimulus (visual or tactile) coming from the side opposite the lesion when the two stimuli are presented together to both sides at the same time

NEUROMUSCULAR JUNCTION

Neuromuscular disease results in a pattern of weakness which is characteristically *fatigable* on repeated testing. The neurology examination is usually normal apart from the muscle weakness. The degree of weakness ranges from *drooping* of the *eye lids* and *diplopia* in mild cases to *difficulty* to *talk, swallow, breathe, move eyes* and *limbs* in severe cases. The main cause of neuromuscular junction weakness is myasthenia gravis.

MUSCLE

Muscle disease results in weakness and sometimes muscle wasting. Neurology examination is normal apart from the muscle weakness and reflexes are usually preserved until late in the disease when they may be lost. The clinical characteristic of myopathic weakness is that the *proximal muscles* are more involved than the *distal ones*. This is most obvious whilst asking the patient to repeatedly elevate the arms over the head or whilst attempting to stand up from a seated position on the ground or in a chair. The main causes of muscle weakness are myopathy, polymyositis and muscular dystrophy.

GAIT DISORDERS

A normal functioning gait requires the coordinated action of an intact sensory and motor system, which in turn relies on normal balance and muscles. Neurological causes of gait disorder may arise from any one or all of these. Gait disorders are classified on the basis of their *main clinical presentation*. The main types of abnormal gait are outlined below in Table 2.4. The site of abnormality is suggested by the characteristic gait and confirmatory neurological signs.

Table 2.4 Common gait disorders seen in clinical practice

Gait disorder	Clinical features
Hemiparetic gait	arm adducted, leg extended, stiff & circumducting
Spastic paraparesis	slow, stiff, jerky with scissoring/adduction at knees & dragging of toes
Cerebellar	wide based & unsteady, can't walk a straight line
Parkinson's	slow & stiff with small & shuffling steps, neck & trunk & limbs semi flexed & decreased or no arm swing
Sensory ataxic	high stepping gait with stomping feet
Neuropathic	high stepping with foot drop
Myopathic	waddling or rolling

Key points

- neurological history & examination are essential
- most important question is whether the lesion is in the CNS or PNS
- neurological findings help to correctly localize the site of the lesion
- localization determines the likely cause, investigations & management

Selected references

Fitzgerald M.J.T. & Folan-Curran Jean, *Clinical Neuroanatomy and related neuroscience*. 4th edition. Elsevier Harcourt Publishers Ltd, 2002.

Wilkinson Iain & Lennox Graham, *Essential Neurology*. Blackwell Publishing 4th edition 2005.

Harrison Michael, *Neurological Skills, A guide to examination and management in Neurology*. Butterworth's 1st edition 1987.

Fuller Geraint, *Neurological examination made easy*. Churchill Livingstone, 3rd edition 2004.

O'Brien MD, *Aids to the examination of the peripheral nervous system*. Saunders 1st edition 2000.