CHAPTER 1

1 Neurological assessment and examination

An accurate neurological assessment is fundamental for the correct management of the patient. The basic aim of the neurological examination is to solve the following four questions:

1 Is there a neurological problem?

2 What is the site of the lesion (or lesions) in the nervous system?

3 What are the pathological conditions that can cause the lesions?

4 Having ascertained the neuroanatomical site and the pathological cause from the history, what is the most likely diagnosis?

Answering these four questions in turn will indicate the type of investigation necessary to confirm the diagnosis.

The neurological assessment involves:

- the history of the illness
- clinical examination:
  (a) of the nervous system
  (b) general examination.

The neurological history

As in general medicine and surgery the neurological history is the key to the diagnosis. The history involves not only questioning the patient but also careful observation. Many neurological illnesses can be diagnosed just by observing the patient. The patient's general manner, mood, posture, gait, facial expression and speech are all vital clues to the final diagnosis. In addition, patients who do not have an organic disease may present in a characteristic manner, particularly with an exaggeration of the complaint.

The history and examination commences with observation, and this should begin when first meeting the patient and while taking the history. The way in which the patient walks into the examination room, sits on the chair, answers questions and climbs on to the examination couch will provide vital clues in the search for the diagnosis. Initially it is important to allow the patient adequate opportunity to explain their symptoms in an unstructured and unprompted manner. Direct questioning should then follow.

The questions concerning neurological symptoms are in essence a verbal examination of the neurological system. It is not just the content of the answer that is important but the way in which the patient responds to the questions. The following is a general classification of neurological symptoms.

1 General neurological symptoms:
  (a) headache
  (b) drowsiness (decreased conscious state)
  (c) vertigo
  (d) seizures, blackouts.

2 Symptoms of meningismus:
  (a) headache
  (b) photophobia
  (c) neck stiffness
  (d) vomiting.

3 Symptoms related to the special senses:
  (a) vision
  (b) hearing
  (c) taste
  (d) smell.

4 Symptoms related to speech and comprehension.

5 Motor symptoms:
  (a) power
  (b) coordination.

6 Sensory symptoms.
7 Cognitive symptoms, e.g. memory.
8 Symptoms of other systems which may relate to diseases of the nervous system.

Careful questioning will ascertain the important details concerning each symptom. These include:

- The **time, mode of onset, progression** and **duration** of the symptom. The mode of onset is a valuable clue in discerning the pathological process. Sudden onset of a neurological disturbance is usually due to a vascular or epileptiform cause; a sudden severe headache is characteristic of subarachnoid haemorrhage whereas a slowly progressive headache is more in keeping with a cerebral tumour. Similarly, the abrupt onset of a hemiplegia may result from a vascular catastrophe and slowly progressive weakness may be due to a compressive or infiltrative cause.
- What factors result in **alleviation** or **exacerbation** of the symptom? Headache from raised intracranial pressure is characteristically worse in the morning and on coughing and straining. Patients find the hand pain associated with carpal tunnel syndrome is often worse at night and is alleviated by shaking the hand over the side of the bed.
- Is there a **past history** of any similar event?

It is often helpful to obtain details of the history from the patient's relatives or a witness; it is vital to do this if the patient is a child or if there is impairment of conscious state or memory disturbance. Details of the nature of epileptic seizures should always be obtained from a relative or friend who has witnessed an event.

A thorough understanding of the nature of the illness and symptomatology should have been obtained before the examination is commenced.

Neurological examination

The formal neurological examination should be undertaken in a systemic fashion in the following order.

1 Mental state.
2 Speech.
3 Cranial nerves.
4 Examination of limbs and trunk:
(a) posture
(b) wasting
(c) tone
(d) power
(e) reflexes
(f) sensation
(g) coordination and gait.

Mental state

Examination of the mental state involves an assessment of:

- conscious state
- orientation in time, place and person
- memory
- emotional state
- presence of delusions or hallucinations.

A correct assessment of the mental state is essential prior to the evaluation of the other neurological signs. The remainder of the neurological examination will be undertaken within the context of the patient's mental state. The accurate assessment of conscious state is especially important in neurosurgical disorders and the evaluation of the level of consciousness using the Glasgow coma scale is described in the chapter on head injuries (Chapter 4). Imprecise terms such as 'stuporose' should be avoided and the examiner should objectively assess and describe the patient's response to specific stimuli. **'Drowsiness'**—a depressed conscious state—is the most important neurological sign and indicates major intracranial pathology. As with all neurological symptoms and signs it is essential to obtain an assessment of the progression of the drowsiness by questioning the patient's friends or relatives. **A deteriorating conscious state is a neurosurgical emergency**.

Memory disturbances should be tested formally for both short-term and long-term preservation. Short-term memory should be tested by listing a name, address and type of flower and asking the patient to recall it after 5 minutes. Loss of short-term memory with relative preservation of memory for long-past events is typical of dementia, e.g. Alzheimer's disease. In Korsakoff's psychosis the disturbance of recent memory and disorientation may be so severe that the patient

will make up stories to provide a convincing answer to the questions. This is **confabulation** and is classically associated with alcoholism, although it may rarely be seen as a result of anterior hypothalamic lesions due to trauma or following subarachnoid haemorrhage and vasospasm.

Speech disorders

There are four main speech disorders:
1 Mutism.
2 Aphonia.
3 Dysarthria.
4 Dysphasia.

Mutism

Mutism is characterized by the patient being alert but making no attempt to speak. It may result from lesions affecting the medial aspect of both frontal lobes, classically occurring as a result of vasospasm following subarachnoid haemorrhage from a ruptured anterior communicating artery aneurysm.

Aphonia

Aphonia is said to occur when the patient is able to speak but is unable to produce any volume of sound. It is due to a disturbance of the vocal cords or larynx. If the patient is able to cough normally then it is usually hysterical.

Dysarthria

Dysarthria is due to impaired coordination of the lips, palate, tongue and larynx and may result from extrapyramidal, brainstem or cerebellar lesions. The volume and content of the speech will be normal but the enunciation will be distorted.

Spastic dysarthria. This is due to bilateral upper motor neurone disease due to pseudobulbar palsy, motor neurone disease or brainstem tumours.

Ataxic dysarthria. This is due to incoordination of the muscles of speech; the words are often staccato or scanning and the rhythm is jerky. This type of dysarthria is seen in cerebellopontine angle tumours, cerebellar lesions, multiple sclerosis and phenytoin toxicity.

Dysarthria may result from lesions of the lower motor neurones and the muscles, such as occur in palatal palsies or paralysis of the tongue.

'Rigid dysarthria'. This is characteristic of Parkinson's disease. In severe cases the phenomenon of palilalia is seen, in which there is a constant repetition of a particular syllable.

Dysphasia

Dysphasia may be either expressive or receptive. Patients with expressive dysphasia can understand speech but cannot formulate their own speech. Patients with receptive dysphasia cannot understand spoken or written speech. Although one type of dysphasia may predominate there is frequently a mixture of the two patterns of disability. Dysphasia results from lesions of the dominant hemisphere, which is the left hemisphere in right-handed people as well as in a high proportion of left-handed people.

Expressive dysphasia. This is due to a lesion affecting either Broca's area in the lower part of the precentral gyrus (Fig. 1.1) or the left posterior temporoparietal region. If the latter region is affected the patient may have a nominal dysphasia, in which the ability to name objects is lost but the ability to speak is retained.

Receptive dysphasia. This results from lesions in Wernicke's area, which is the posterior part of the superior temporal gyrus and the adjacent parietal lobe.

Alexia

Alexia is the inability to understand written speech. Alexia with agraphia (inability to write) is due to a lesion in the left angular gyrus. The patient is unable to read or write spontaneously and the condition is often accompanied by nominal dysphasia, acalculia, hemianopia and visual agnosia. Gerstmann's syndrome consists of finger agnosia for both the patient's own finger and the examiner's finger, acalculia, right/left disorientation and agraphia without alexia. It is found in lesions of the dominant hemisphere in the region of the angular gyrus.

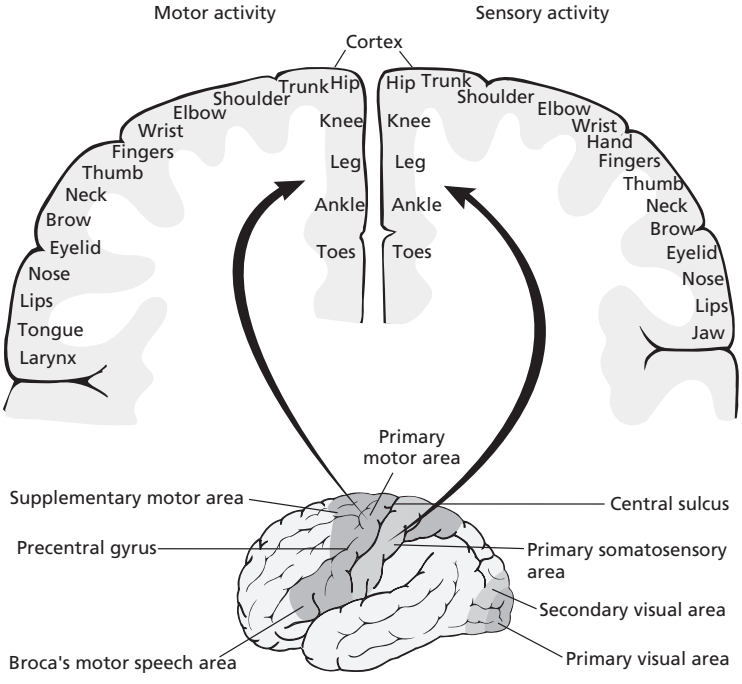

Fig. 1.1 Major areas of somatotopic organization of the cerebrum.

Examination of the cranial nerves

Olfactory nerve

The sense of smell should be tested by the patient sniffing through each nostril as the other is compressed. The common causes of anosmia are olfactory nerve lesions resulting from head injury, and tumours involving the floor of the anterior cranial fossa, especially olfactory groove meningiomas. It is important to use non-irritant substances when testing olfaction, as irritating compounds (e.g. ammonia) will cause irritation of the nasal mucosa. The stimulus is then perceived by the general sensory fibres of the trigeminal nerve.

Optic nerve

The optic nerve should be tested by:

- measuring the visual acuity and colour vision
- charting the visual fields
- fundal examination with an ophthalmoscope
- the pupillary light reflex.

Visual acuity

The visual acuity should be tested using the standard Snellen type charts placed at 6 m. The acuity is recorded as a fraction, e.g. 6/6 or 6/12, in which the numerator indicates the distance in metres from the chart and the denominator the line on the chart that can be read. 6/6 is normal vision. Refractive errors should be corrected by testing with the patient's glasses or by asking the patient to view the chart through a pinhole.

Visual fields

The visual fields can be charted by confrontation, with the patient facing the examiner and objects of varying size being moved slowly into the visual field (Fig. 1.2). Formal testing using perimetry should be undertaken in all cases of visual failure, pituitary tumour, parasellar tumour, other tumours possibly involving the visual pathways and demyelinating disease, or if there are any doubts after confrontation that the fields may be restricted.

Perimetry can be performed using either a tangent screen, such as a Bjerrum screen (Fig. 1.3), or

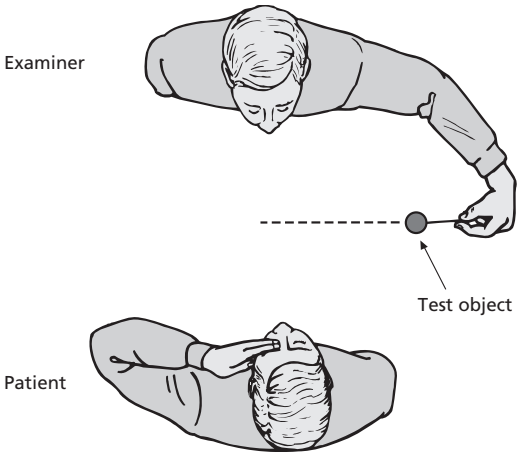

Fig. 1.2 Visual field testing by confrontation.

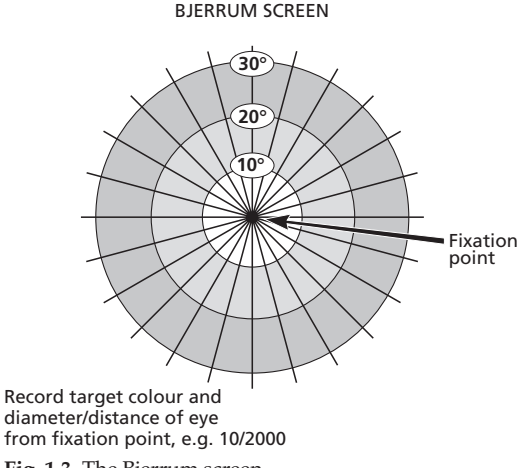

Fig. 1.3 The Bjerrum screen.

a Goldmann perimeter. The Bjerrum screen records the central field of vision. By enlarging the central area out to 30° it is easier to detect scotomas and to measure the blind spot and, provided a small enough target is used, the tangent screen provides an accurate representation of the peripheral fields. An automated perimetry machine will enable an accurate and reproducible field test that is particularly useful in cooperative patients.

The pattern of visual field loss will depend on the anatomical site of the lesion in the visual pathways (Fig. 1.4):

- total visual loss—optic nerve lesion
- altitudinous hemianopia—partial lesion of the optic nerve due to trauma or vascular accident
- homonymous hemianopia—lesions of the optic tract, radiation or calcarine cortex
- bitemporal hemianopia—optic chiasm lesions such as pituitary tumour, craniopharyngioma or suprasellar meningioma.

Fundal examination

The fundus should be examined using the ophthalmoscope with particular attention to the:

- optic disc
- vessels
- retina.

A pale optic disc is due to optic atrophy which may be either primary, as a result of an optic nerve lesion caused by compression or demyelination, or consecutive, which follows severe swelling of the disc. Papilloedema is due to raised intracranial pressure and is evident by:

- blurring of the disc margins
- filling in of the optic cup
- swelling and engorgement of retinal veins, with loss of normal pulsation of the veins
- haemorrhages around the disc margin (if severe).

Third, fourth and sixth cranial nerves

As these cranial nerves are all involved in innervation of the extraocular muscles they are usually examined together. This examination involves assessment of:

- the position of the eyelids
- the pupils
- extraocular movements.

Position of the eyelids

Ptosis is due to paralysis of the levator palpebrae superioris as a result of a 3rd cranial nerve lesion or due to weakness of the tarsal muscle due to a sympathetic lesion (Horner's syndrome).

The pupils

An assessment should be made of the **pupil size, shape** and **equality**. The pupils' reaction to light should be tested by shining a beam into the eye and noting the reaction in that eye, as well as the

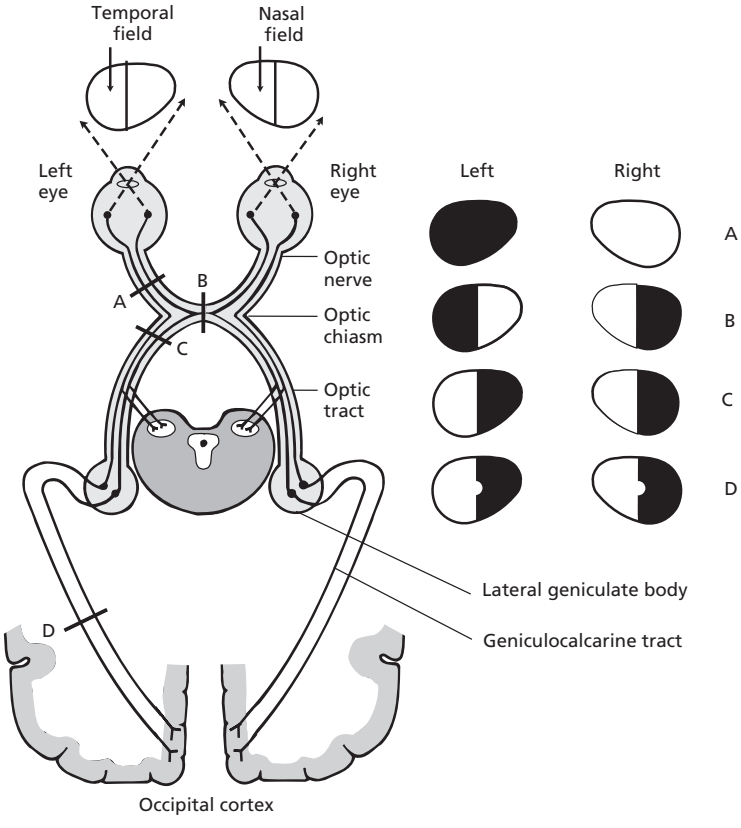

Fig. 1.4 Diagrammatic representation of visual pathways, the common sites of lesions and the resulting field defects.

consensual response in the opposite eye. The reaction to convergence and accommodation for near vision should be tested by asking the patient to fix on a distant object and then placing a pen approximately 12 cm in front of the bridge of the nose.

A unilateral **constricted pupil (miosis**) often indicates a lesion in the sympathetic supply to the pupillary dilator muscle.

Horner's syndrome, in its complete state, consists of miosis, ptosis, enophthalmos and dryness and warmth of half of the face. It is due to a lesion of the sympathetic supply such as results from an intracavernous carotid artery aneurysm, or a Pancoast's tumour of the apex of the lung.

A **dilated pupil (mydriasis)** results from paralysis of the parasympathetic fibres originating from the nucleus of Edinger–Westphal in the midbrain, and is therefore seen in a 3rd nerve palsy. The possible causes are an enlarging posterior communicating artery aneurysm causing pressure on these fibres in the 3rd cranial nerve (Chapter 9) and tentorial herniation resulting from intracranial pressure with the herniated uncus of the temporal lobe compressing the 3rd nerve (Chapter 5).

The Argyll–Robertson pupil is a small, irregular pupil not reacting to light, reacting to accommodation but responding poorly to mydriatics; it is usually caused by syphilis.

The myotonic pupil (Holmes–Adie) usually occurs in young women and presents as a unilateral dilatation of one pupil with failure to react to light. The pupil shows a slow constriction occurring on maintaining convergence for a prolonged period. In the complete syndrome the knee and ankle jerks are absent.

Ocular movement

The following are the general actions of the extraocular muscles.

• Lateral rectus (6th nerve) moves the eye horizontally outwards.
• Medial rectus (3rd nerve) moves the eye horizontally inwards.
• Superior rectus (3rd nerve) elevates the eye when it is turned outwards.
• Inferior oblique (3rd nerve) elevates the eye when it is turned inwards.
• Inferior rectus (3rd nerve) depresses the eye when it is turned outwards.
• Superior oblique (4th nerve) depresses the eye when it is turned inwards.

The patient should be tested for diplopia, which will indicate ocular muscle weakness before it is evident on examination. The following rules help determine which muscle and cranial nerve are involved.

• The displacement of the false image may be horizontal, vertical or both.
• The separation of images is greatest in the direction in which the weak muscle has its purest action.
• The false image is displaced furthest in the direction in which the weak muscle should move the eye.

Disorders of eye movement may be due to **impaired conjugate ocular movement**. The centre for the control of conjugate lateral gaze is situated in the posterior part of the frontal lobe, with input from the occipital region. The final common pathway for controlling conjugate movement is in the brainstem, particularly the median longitudinal bundle. A lesion of the frontal lobe causes contralateral paralysis of conjugate gaze (i.e. eyes deviated towards the side of the lesion) and a lesion of the brainstem causes ipsilateral paralysis of conjugate gaze (i.e. eyes deviated to side opposite to the lesion).

Nystagmus should be tested by asking the patient to watch the tip of a pointer. This should be held first in the midline and then moved slowly to the right, to the left and then vertically upwards and downwards.

Jerk nystagmus is the common type, consisting of slow drift in one direction and fast correcting movement in the other.

Horizontal jerk nystagmus is produced by lesions in the vestibular system which may occur peripherally in the labyrinth, centrally at the nuclei, in the brainstem or in the cerebellum. In peripheral lesions the quick phase is away from the lesion and the amplitude is greater in the direction of the quick phase. In cerebellar lesions the quick phase is in the direction of gaze at that moment but the amplitude is greater to the side of the lesion. By convention the quick phase is taken to indicate the direction of the nystagmus, so that if the slow phase is to the right and the quick phase to the left the patient is described as having nystagmus to the left.

Vertical nystagmus is due to intrinsic brainstem lesions such as multiple sclerosis, brainstem tumours or phenytoin toxicity. The so-called 'downbeat' nystagmus, which is characterized by a vertical nystagmus exaggerated by downgaze, is particularly evident in low brainstem lesions as caused by Chiari syndrome, where the lower brainstem has been compressed by the descending cerebellar tonsils (Chapter 11).

Trigeminal nerve

The 5th cranial nerve (trigeminal nerve) is tested by assessing facial sensation over the three divisions of the cranial nerve; corneal sensation should be tested using a fine piece of cotton wool. The motor function of the 5th nerve can be tested by palpating the muscles while the patient clenches their jaw, testing the power of jaw opening and lateral deviation of the jaw (Fig. 1.5).

Facial nerve

The facial nerve is tested by assessing facial movement. In an **upper motor neurone** facial weakness the weakness of the lower part of the

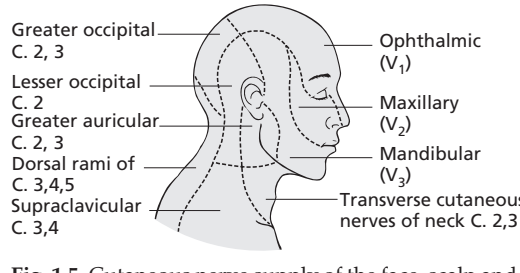

Fig. 1.5 Cutaneous nerve supply of the face, scalp and neck.

face is very much greater than the upper, with the strength of the orbicularis oculis being relatively preserved. This is due to a lesion between the cortex and the facial nucleus in the pons. **Lower motor neurone** weakness is evident by equal involvement of the upper and lower parts of the face and is due to a lesion in, or distal to, the facial nerve nucleus in the pons.

The chorda tympani carries taste sensation from the anterior two-thirds of the tongue and this should be examined using test flavours placed carefully on the anterior tongue.

Vestibulocochlear nerve

The 8th cranial nerve consists of:

- the cochlear nerve—hearing
- the vestibular nerve.

The cochlear nerve

Hearing can be examined at the bedside by moving a finger in the meatus on one side, to produce a masking noise, and repeating words at a standard volume and from a set distance in the other ear. Differentiation between conduction and sensorineural deafness can be aided using tests with a tuning fork.

The **Rinne's test** involves holding a vibrating tuning fork in front of the external meatus and then on the mastoid process. In nerve deafness both air and bone conduction are reduced, but air conduction remains the better. In conductive deafness bone conduction will be better than air conduction.

In **Weber's test** the vibrating tuning fork is placed on the centre of the forehead. In nerve deafness the sound appears to be heard better in the normal ear, but in conductive deafness the sound is conducted to the abnormal ear.

Formal audiometry should be performed if there are symptoms of impaired hearing.

The vestibular nerve

The simplest test of vestibular function is the caloric test, which is usually performed in patients suspected of having a cerebellopontine angle tumour or as a test of brainstem function in patients with severe brain injury. The test is described in Chapter 4, p. 44.

Glossopharyngeal and vagus nerves

The glossopharyngeal and vagus nerves can be most easily assessed by testing palatal movement and sensation from the pharynx and soft palate. If necessary the vocal cords (vagus nerve) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be tested.

Accessory nerve

The accessory nerve supplies the motor power to the upper part of the trapezius and sternocleidomastoid. The latter muscle can be tested by turning the patient's head against resistance and watching and palpating the opposite sternomastoid muscle. The trapezius muscle is best tested by asking the patient to shrug the shoulders and attempting to depress the shoulders forcibly.

Hypoglossal nerve

The hypoglossal nerve is responsible for movements of the tongue. The tongue should be inspected to detect wasting and movements from side to side should be observed to detect weakness. The tip of the protruded tongue will deviate toward the side of weakness.

Examination of the periphery

Posture and general inspection

The patient's posture may indicate an underlying neurological disability, or an abnormal posture may result from pain. A patient with sciatica will often lie on the opposite side with the affected leg flexed at the hip and knee. The decerebrate posture is discussed in Chapter 4.

The limbs should be inspected to compare size and shape and to detect deformity; long-standing neurological lesions may result in impaired growth or wasting. Lesions of lower motor neurone in infancy, such as a brachial plexus palsy or poliomyelitis, will cause marked retardation in limb growth. Upper motor neurone lesions of long standing, such as acute infantile hemiplegia and cerebral birth trauma, will also cause retardation in growth, but of a lesser degree, with a hemiplegic posture and exaggerated reflexes.

Wasting

The limbs and shoulder girdles should be inspected to detect wasting and fasciculation. As well as palpating for specific muscle wasting in each limb the circumference of the limbs should be measured at clearly identifiable positions, such as 8 cm above or below the olecranon, 10 cm above the patella and 8 cm below the tibial tuberosity.

The pattern of wasting will be an important clue as to the underlying neurological disease.

Wasting of the forearm and small muscles of the hand. This results from lower motor neurone lesions affecting particularly the C7, C8 and T1 levels and may be due to lesions of the:

- spinal cord—motor neurone disease, syringomyelia, cervical cord tumours
- cervical nerve root—cervical disc prolapse
- brachial plexus—trauma, cervical rib, axillary tumour
- peripheral nerve—ulnar nerve compression at the elbow, carpal tunnel syndrome (median nerve).

Wasting of the muscles of the lower leg. This will result from compression of the cauda equina or lumbosacral nerve roots caused by a lumbar disc prolapse or tumour.

Muscular dystrophies. These are genetically determined inherited degenerative myopathies and cause particular patterns of muscle wasting.

- Facioscapulohumeral dystrophy involves the face and shoulder girdle.
- Proximal limb girdle dystrophy involves both shoulder and hip girdles.
- Dystrophia myotonica involves the face, sternomastoids and quadriceps femoris. Myotonia (the failure of muscle to relax after contraction) is present, particularly in the peripheral muscles and tongue.
- Peroneal muscular atrophy, with predominant involvement of the lower limbs, causes the 'inverted bottle appearance' with similar but less striking changes in the upper limbs.
- Duchenne's muscular dystrophy occurs mainly in young boys and affects the arms and legs; the muscles have a pseudohypertrophic appearance.

Tone

The tone in the upper limbs should be tested using a flexion–extension movement of the wrist, by holding the patient's terminal phalanges and by pronation–supination of the forearm. The tone in the lower limbs should be tested by flexion of the hip, knee and ankle.

Decreased tone

This is due to:

- a lower motor neurone lesion involving the spinal roots or anterior horn cell of the spinal cord
- lesions of the sensory roots of the reflex arc, e.g. tabes dorsalis
- cerebellar lesions, which cause ipsilateral hypotonia
- myopathies
- spinal shock (the acute phase of a severe spinal lesion usually due to trauma).

Increased tone

This will be produced by any upper motor neurone lesion involving the corticospinal tracts above the level of the anterior horn cell in the spinal cord.

There are three major types of hypertonicity.

1 'Clasp knife' spasticity, in which the resistance is most pronounced when the movement is first made. It is usually more marked in the flexor muscles of the upper limbs and extensor muscles of the lower limbs and is a sign of an upper motor neurone lesion.

2 'Lead pipe' rigidity, in which there is equal resistance to all movements. This is a characteristic feature of a lesion of the extrapyramidal system but is also seen in severe spasticity from an upper motor neurone lesion.

3 'Cog wheel' rigidity, in which there is an alternating jerky resistance to movement and which occurs in degenerative lesions of the extrapyramidal system, particularly Parkinson's disease.

'Clonus' is best demonstrated by firm rapid dorsiflexion of the foot and is indicative of marked increased tone.

Power

The power should be tested in all limbs, comparing each side. A systematic evaluation will enable the recognition of a particular pattern of weakness that will be in keeping with either a cerebral, spinal cord, plexus or peripheral nerve weakness. The major nerve and main root supply of the muscles are shown in Table 1.1.

The Medical Research Council classifies the degree of weakness by recording power, ranging from 0 to 5 (Table 1.2). It is apparent that there is a considerable range of power between grades 4 and 5 and some clinicians make their own further subclassification in this region.

Weakness due to a **corticospinal tract lesion** is most marked in the abductors and extensors of the upper limbs and the flexors of the lower limbs. It is normally associated with increased tone and exaggerated reflexes.

Weakness due to **lower motor neurone lesions** is usually more severe than when the upper motor neurone is involved and is seen in the distribution of the nerve affected. It is associated with wasting, hypotonia and diminished reflexes.

Fasciculation is an irregular, non-rhythmical contraction of muscle fascicles which is most easily seen in the deltoid or calf muscles. It occurs classically in motor neurone disease but may also occur in lower motor neurone lesions, e.g. in the lower limbs following long-standing lumbar root compression.

Reflexes

The deep tendon reflex requires the stimulus, sensory pathway, motor neurone, contracting muscle and the synapses between the neurones in order to elicit a response.

Reduced or absent tendon reflex

This may occur due to any breach in the reflex arc:

- sensory nerve—polyneuritis
- sensory root—tabes dorsalis
- anterior horn cell—poliomyelitis
- anterior root—compression
- peripheral motor nerve—trauma
- muscle—myopathy.

Increased deep tendon reflexes

Due to lesions of the pyramidal system, increased deep tendon reflexes may be excessively prolonged, with a larger amplitude in a cerebellar lesion. In myxoedema the relaxation phase of the reflex is retarded.

Each deep tendon reflex is associated with a particular segmental innervation and peripheral nerve as listed in Table 1.3.

The superficial abdominal reflex has a segmental innervation extending from T9 in the upper abdominal region to T12 in the lower area. The reflex may be absent in pyramidal lesions above the level of segmental innervation, particularly in spinal lesions. However, the reflex may also be difficult to elicit when the abdominal muscles have been stretched or damaged by surgical operations, or in a large, pendulous, obese abdomen.

Plantar reflex

This should result in the great toe flexing the metatarsophalangeal joint. The Babinski response consists of extension of the great toe at the metatarsophalangeal joint, and usually at the interphalangeal joint, and indicates disturbance of the pyramidal tract.

Sensation

The modalities of sensation which should be tested are:

- light touch
- pinprick (pain)
- temperature
- position (proprioception)
- vibration.

Sensory testing involves an accurate understanding of the anatomical pathways of sensation. All modalities of sensation travel by the peripheral nerve and sensory root to the spinal cord, or via the cranial nerves to the brainstem. The fibres for pain and temperature sensation enter the posterolateral aspect of the spinal cord, travel cranially for a few segments and then cross to the opposite anterolateral spinothalamic tract. This tract ascends to the brainstem and is joined by the quintothalamic (trigeminothalamic) tract in the pons. The fibres end mostly in the ventrolateral nucleus of the thalamus and from here the

Table 1.1 Nerve and major root supply of muscles.

	Spinal roots
Upper limb	
Spinal accessory nerve	
Trapezius	C3, C4
Brachial plexus	
Rhomboids	C4, C5
Serratus anterior	C5, C6, C7
Pectoralis major	
Clavicular	**C5**, C6
Sternal	C6, **C7**, C8
Supraspinatus	**C5**, C6
Infraspinatus	**C5**, C6
Latissimus dorsi	C6, **C7**, C8
Teres major	C5, C6, C7
Axillary nerve	
Deltoid	**C5**, C6
Musculocutaneous nerve	
Biceps	C5, C6
Brachialis	C5, C6
Radial nerve	
Triceps	
Long head	
Lateral head	C6, **C7**, C8
Medial head	
Brachioradialis	C5, **C6**
Extensor carpi radialis longus	C5, **C**6
Posterior interosseous nerve	
Supinator	C6, C7
Extensor carpi ulnaris	**C7**, C8
Extensor digitorum	**C7**, C8
Abductor pollicis longus	**C7**, C8
Extensor pollicis longus	**C7**, C8
Extensor pollicis brevis	**C7**, C8
Extensor indicis	**C7**, C8
Median nerve	
Pronator teres	C6, C7
Flexor carpi radialis	C6, C7
Flexor digitorum superficialis	C7, **C8**, T1
Abductor pollicis brevis	C8, **T1**
Flexor pollicis brevis*	C8, **T1**
Opponens pollicis	C8, **T1**
Lumbricals I and II	C8, **T1**
Anterior interosseous nerve	
Flexor digitorum profundus I and II	C7, **C8**
Flexor pollicis longus	C7, **C8**
Ulnar nerve	
Flexor carpi ulnaris	C7, **C8**, T1
Flexor digitorum profundus III and IV	C7, **C8**
Hypothenar muscles	C8, **T1**
Adductor pollicis	C8, **T1**
Flexis pollicis brevis	C8, **T1**
Palmar interossei	C8, **T1**
Dorsal interossei	C8, **T1**
Lumbricals III and IV	C8, **T1**
Lower limb	
Femoral nerve	
Iliopsoas	**L1, L2**, L3
Rectus femoris	
Vastus lateralis (Quadriceps femoris)	L2, **L3, L4**
Vastus intermedius (Quadriceps femoris)	
Vastus medialis (Quadriceps femoris)	
Obturator nerve	
Adductor longus	**L2, L3**, L4
Adductor magnus	
Superior gluteal nerve	
Gluteus medius and minimus	**L4, L5**, S1
Tensor fasciae latae	
Inferior gluteal nerve	
Gluteus maximus	**L5, S1**, S2
Sciatic and tibial nerves	
Semitendinosus	L5, **S1**, S2
Biceps	L5, **S1**, S2
Semimembranosus	L5, **S1**, S2
Gastrocnemius and soleus	S1, S2
Tibialis posterior	L4, L5
Flexor digitorum longus	L5, **S1, S2**
Flexor hallucis longus	L5, **S1, S2**
Small muscles of foot	S1, S2
Sciatic and common peroneal nerves	
Tibialis anterior	**L4**, L5
Extensor digitorum longus	**L5**, S1
Extensor hallucis longus	**L5**, S1
Extensor digitorum brevis	**L5**, S1
Peroneus longus	L5, S1
Peroneus brevis	L5, S1

* Flexor pollicis brevis is often supplied wholly or partially by the ulnar nerve.

sensory impulses pass through the posterior limb of the internal capsule to the postcentral sensory cortex (see Chapter 19, Fig. 19.1). Fibres carrying light touch, proprioception and vibration sensation ascend mainly in the ipsilateral posterior columns of the spinal cord on the same side to the nuclei gracilis and cuneatus. The fibres cross the midline to ascend through the brainstem in the medial lemniscus, to synapse in the thalamus and then on to the sensory cortex.

The sensory loss involving nocioceptive stimuli (pain and temperature) should conform to a particular pattern:

- peripheral nerve
- dermatome (nerve root)
- spinal cord—resulting in a sensory level
- 'glove and stocking' due to peripheral neuropathy
- hemianalgesia—thalamic or upper brainstem
- loss of pain and temperature on one side of the face and the opposite side of the body—lesion of the medulla affecting the descending root of the 5th nerve and the ascending spinothalamic tract from the remainder of the body.

Table 1.2 Medical Research Council classification of power.

0	Total paralysis
1	Flicker of contraction but no movement of limb
2	Muscle only able to make normal movement when limb is positioned so that gravity is eliminated
3	Normal movement against gravity but not against additional resistance
4	Full movement but overcome by resistance
5	Normal power

Coordination

Coordination should be tested in the upper and lower limbs. In the upper limb it is best assessed using the 'finger–nose' test and in the lower limb using the 'heel–knee' test. It is important to determine whether abnormalities of coordination are due to defects in:

- cerebellar function
- proprioception
- muscular weakness.

Gait

An essential part of the examination is to observe the patient's gait. This is best done not only as a formal part of the examination but also when the patient is not aware of observation. The type of gait is characteristic of the underlying neurological disturbance.

A hemiparesis will cause the patient to drag the leg and, if severe, the leg will be thrown out from the hip, producing the movement called **circumduction**.

A **high stepping gait** occurs with a foot drop (e.g. L5 root lesion due to disc prolapse, lateral popliteal nerve palsy, peroneal muscular atrophy). The patient raises the foot too high to overcome the foot drop and the toe hits the ground first. In tabes dorsalis the high stepping gait is due to a profound loss of position sense but

Table 1.3 Deep tendon reflexes, peripheral nerve and segmental innervation.

Tendon reflex	Major segmental innervation	Peripheral nerve
Biceps jerk	C5(6)	Musculocutaneous
Supinator jerk	C5/C6	Radial
Triceps jerk	C7(8)	Radial
Flexor finger jerk	C6–T1	Median and ulnar
Knee jerk	L3/L4	Femoral
Ankle jerk	S1(2)	Medial popliteal and sciatic

a similar gait, of lesser severity, will result from involvement of the posterior column of the spinal cord or severe sensory neuropathy which interferes with position sense. The gait is worse in the dark and the heel usually strikes the ground first.

In **Parkinson's disease** or other extrapyramidal diseases the patient walks with a stooped, shuffling gait. The patient may have difficulty in starting walking and stopping. A slight push forward will cause rapid forward movement (protopulsion).

In the **ataxic gait**, the patient is unstable due to cerebellar disturbance. A midline vermis tumour will result in the patient reeling in any direction. If the cerebellar hemisphere is involved then the patient will tend to fall to the ipsilateral side.

A **waddling gait** is associated with congenital dislocation of the hips and muscular dystrophy.

The hysterical gait is often bizarre and is diminished when the patient is unaware of any observation.

Following the clinical assessment, a presumptive diagnosis is made and further investigations can be performed to confirm the diagnosis. These laboratory investigations and radiological procedures are described in the following chapter.

Brain death

The use of donor organs for transplantation and the advent of improved intensive care facilities have resulted in the necessity of medically and legally accepted criteria of brain death.

If there is irrecoverable brainstem damage and the tests described below show no evidence of brainstem function, then the patient is medically and legally dead. If artificial ventilation is continued the other organs may continue to function for some time. However, continued prolonged ventilation of the patient after the diagnosis of brain death is not only undignified for the dead patient and distressing to the relatives, but is also wasteful of expensive medical resources that are often in short supply.

The diagnosis of brain death relies on:

- preconditions before testing can be performed
- brain death tests.

The preconditions are that all reversible causes of brainstem depression have been excluded. These include:

- depressant drugs
- hypothermia (temperature must be greater than 35°C)
- neuromuscular blocking drugs
- metabolic or endocrine disturbance as a cause of the patient's condition.

Brain death testing must be delayed until these preconditions are absolutely satisfied.

The tests for brainstem function are:

- lack of pupil response to light
- lack of corneal reflex to stimulation
- lack of oculocephalic reflex
- failure of vestibulo-ocular reflex (caloric testing)
- failure of a gag or cough reflex on bronchial stimulation
- no motor response in the face or muscles supplied by the cranial nerves in response to painful stimulus
- failure of respiratory movements when the patient is disconnected from a ventilator and the P_aCO_2 is allowed to rise to 50 mmHg.

The tests should be repeated after an interval of 30 minutes and it is essential that they should be carried out by two doctors of adequate seniority and with expertise in the field.

Further reading

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