Ask the patient to stand facing away from you with the arms and hands stretched out to touch and push against the wall. Winging of the scapulae is seen typically in fascio-scapular-humeral dystrophy (Figure 11.62).

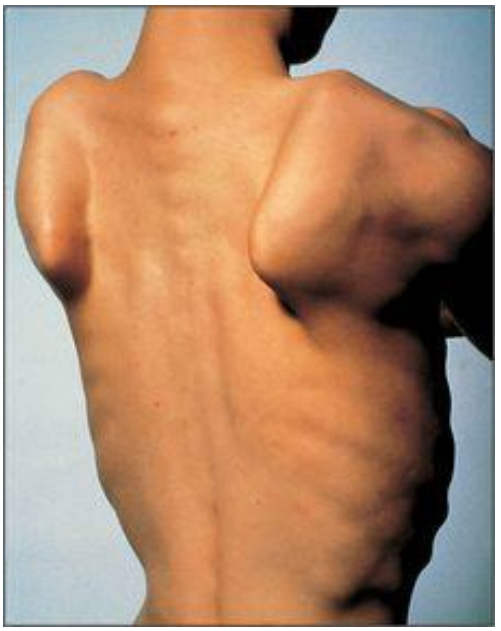

Figure 11.62 Winging of the scapulae, often a result of muscular dystrophy

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Causes of brachial plexus lesions include:

1. Inflammation, autoimmune disorders (more often upper plexus).

2. Radiotherapy (more often upper plexus).

3. Cancer (more often lower plexus). *Cancer* causes a brachial plexus lesion by local invasion. The lower trunk is usually affected first. These plexus lesions are usually painful and progress rapidly. Weakness and sensory loss are both present.

4. Trauma: direct (motor vehicle accident, surgery including sternotomy, lacerations and gunshots), traction (birth injuries, motor vehicle accidents, sporting injuries such as rugby tackles—more often upper plexus), chronic compression (thoracic outlet, 'backpack palsy', fractures with bone displacement).

The lower limbs

Begin by testing gait, if this is possible (see page 276).

Begin by testing *gait*, if this is possible (see page 376).

Inspect the legs with the patient lying in bed with the legs and thighs entirely exposed (place a towel over the groin). Note whether there is a urinary catheter present, which may indicate that there is spinal cord compression or other spinal cord disease, particularly multiple sclerosis.

The motor system

Fasciculations and muscle wasting

Inspect for fasciculations. Look for muscle wasting. Feel the muscle bulk of the quadriceps and calves. Then run a hand along each shin, feeling for wasting of the anterior tibial muscles.

Tone

Test tone at the knees and ankles. Place one hand under a chosen knee and then abruptly pull the knee upwards, causing flexion. When the patient is relaxed this should occur without resistance. Then, supporting the thigh, flex and extend the knee at increasing velocity, feeling for resistance to muscle stretch (tone). Tone in the legs may also be tested by sitting the patient with legs hanging freely over the edge of the bed. A leg (of the patient) is raised by the examiner to the horizontal and suddenly let go. The leg will oscillate up to half a dozen times in a normal person who is completely relaxed. If hypotonia is present, as occurs in cerebellar disease, the oscillations will be wider and more prolonged. If increased tone or spasticity is present, the movements will be irregular and jerky.

Next test for *clonus* of the ankle and knee. This is a sustained rhythmical contraction of the muscles when put under sudden stretch. It is due to hypertonia from an upper motor neurone lesion. It represents an increase in reflex excitability (from increased alpha motor neurone activity).

Sharply dorsiflex the foot with the knee bent and the thigh externally rotated. When ankle clonus is present, recurrent ankle plantar flexion movement occurs. This may persist for as long as the examiner sustains dorsiflexion of the ankle. Test for patellar clonus (which is not so common) by resting the hand on the lower part of the quadriceps with the knee extended and moving the patella down sharply. Sustained rhythmical contraction of the quadriceps occurs as long as the downward stretch is maintained.

Power

Test power next.

Hip

• *Flexion*—psoas and iliacus muscles—(L2, L3): ask the patient to lift up the straight leg and not let you push it down (having placed your hand above the knee) (Figure 11.63).

• *Extension*—gluteus maximus—(L5, S1, S2): ask the patient to keep the leg down and not let you pull it up from underneath the calf or ankle (Figure 11.64).

• *Abduction*—gluteus medius and minimus, sartorius and tensor fasciae latae—(L4, L5, S1): ask the patient to abduct the leg and not let you push it in (Figure 11.65).

• *Adduction*—adductors longus, brevis and magnus—(L2, L3, L4): ask the patient to keep the leg adducted and not let you push it out (Figure 11.66).

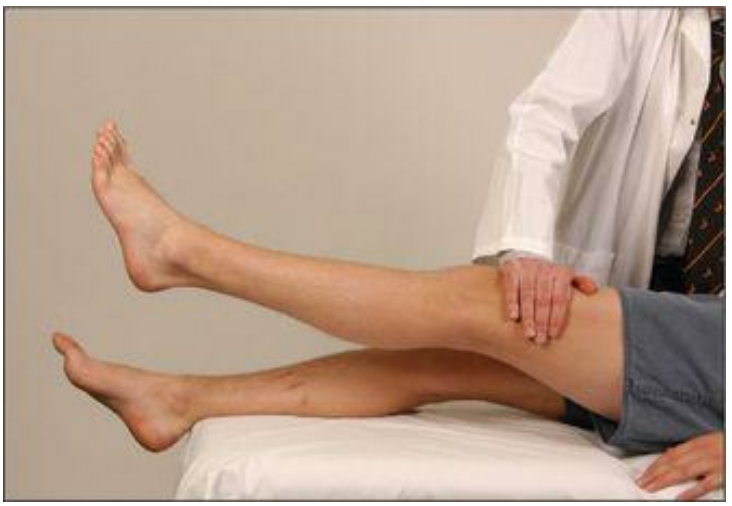


Figure 11.63 Testing power—hip flexion: 'Lift your leg up and don't let me push it down'

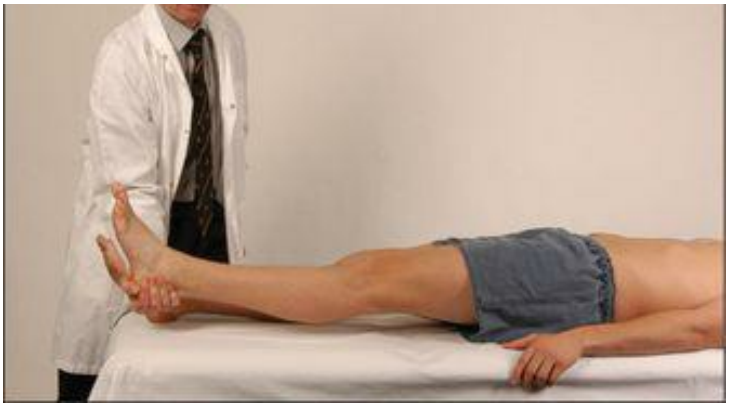


Figure 11.64 Testing power—hip extension: 'Push your heel down and don't let me pull it up'

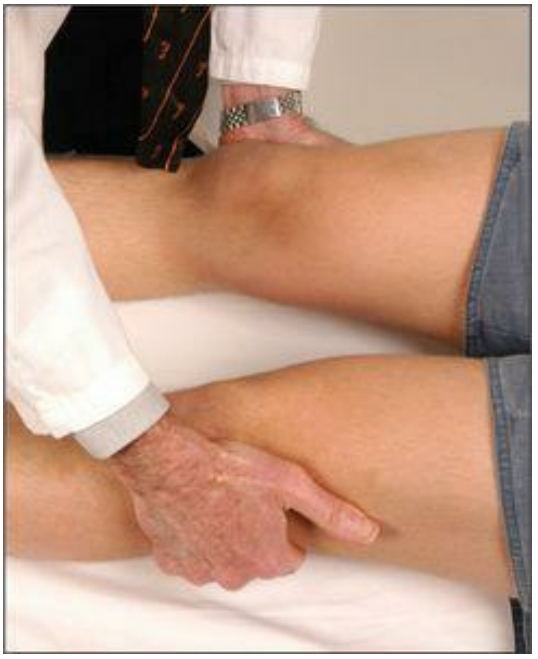


Figure 11.65 Testing power—hip abduction: 'Don't let me push your knees together'

Figure 11.66 Testing power—hip adduction: 'Don't let me push your knees apart'

Knee

• *Flexion*—hamstrings (biceps femoris, semimembranosus, semitendinosus)—(L5, S1): ask the patient to bend the knee and not let you straighten it (Figure 11.67). If there is doubt about the real strength of knee flexion, it should be tested with the patient in the prone position. Here possible help from hip flexion is prevented and the muscles can be palpated during contraction.

• *Extension*—quadriceps femoris (this muscle is three times as strong as its antagonists, the hamstrings)—(L3, L4): with the knee slightly bent, ask the patient to straighten the knee and not let you bend it (Figure 11.68).

Figure 11.67 Testing power—knee flexion: 'Bend your knee and don't let me straighten it'; pull hard

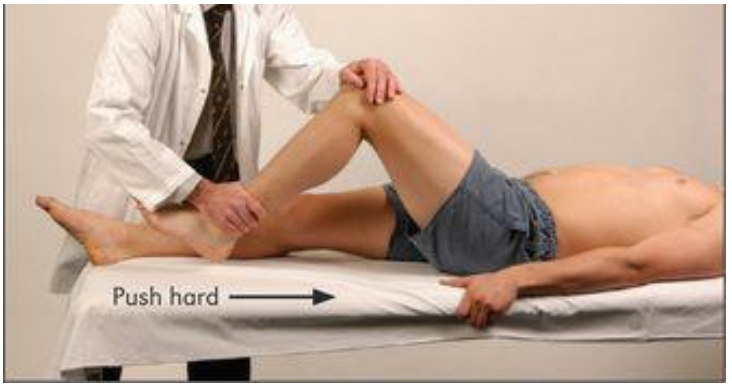


Figure 11.68 Testing power—knee extension: 'Straighten your knee and don't let me bend it'; push hard

Ankle

• *Plantar flexion*—gastrocnemius, plantaris, soleus—(S1, S2): ask the patient to push the foot down and not let you push it up (Figure 11.69).

• *Dorsiflexion*—tibialis anterior, extensor digitorum longus and extensor hallucis longus—(L4, L5): ask the patient to bring the foot up and not let you push it down (Figure 11.70). The power of the ankle joint can also be tested by having the patient stand up on the toes (plantar flexion) or on the heels (dorsiflexion); these movements may also be limited if coordination is impaired.

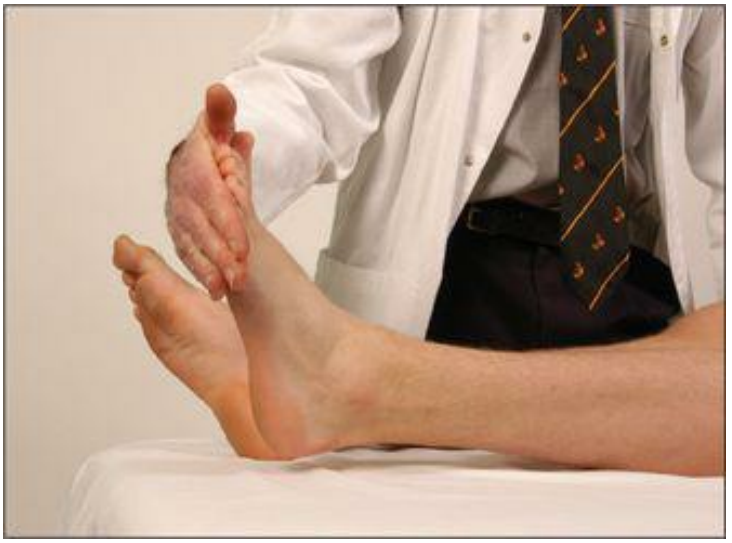


Figure 11.69 Testing power—ankle, plantar flexion: 'Don't let me push your foot up'

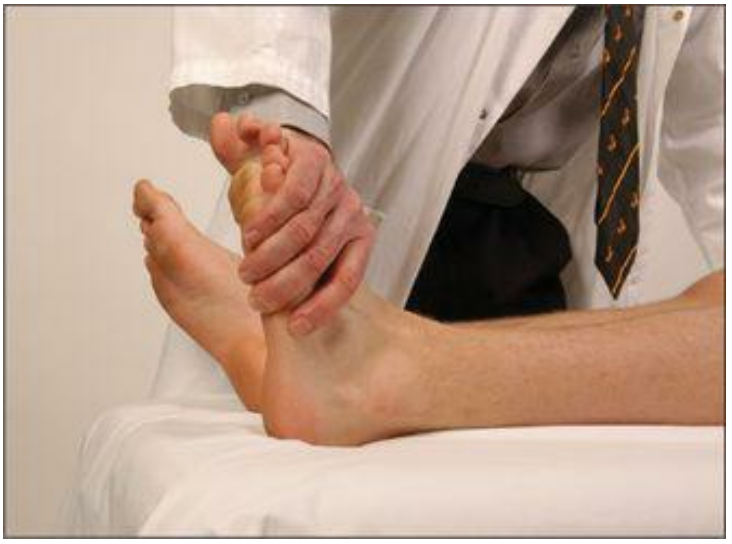


Figure 11.70 Testing power—ankle, dorsiflexion: 'Don't let me push your foot down'

Tarsal joint

• *Eversion*—peroneus longus and brevis, and extensor digitorum longus—(L5, S1): ask the patient to evert the foot against resistance (Figure 11.71).

• *Inversion*—tibialis posterior, gastrocnemius and hallucis longus—(L5, S1): with the foot in complete plantar flexion, ask the patient to invert the foot against resistance (Figure 11.72).

Non-organic or hysterical unilateral limb weakness may be detected by Hoover's test. Normally when a patient attempts to resist movement, the contralateral limb works to support the effort. For example when a patient attempts to extend the leg against resistance, the other leg pushes down into the bed. If this movement is absent Hoover's[nn] sign is positive.

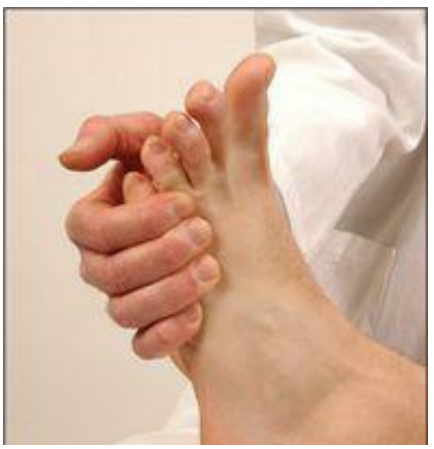


Figure 11.71 Testing power—ankle (tarsal joint) eversion: 'Stop me turning your foot inwards'

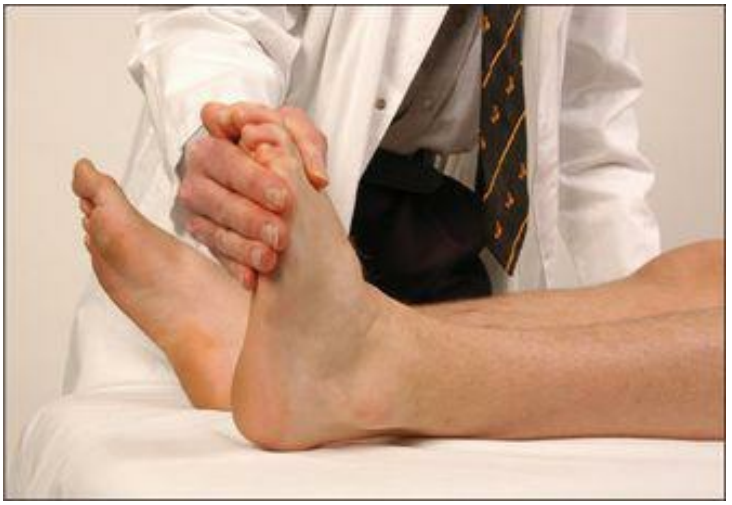


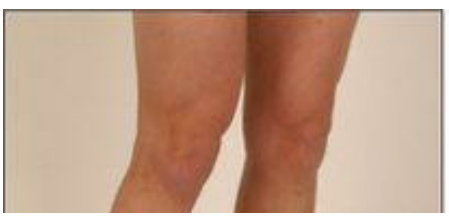
Figure 11.72 Testing power—ankle (tarsal joint) inversion: 'Stop me turning your foot outwards'

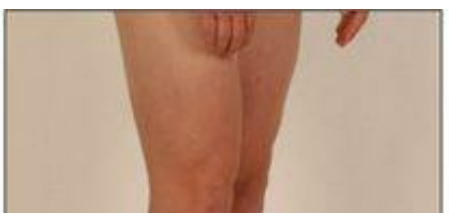
Quick test of lower limb power

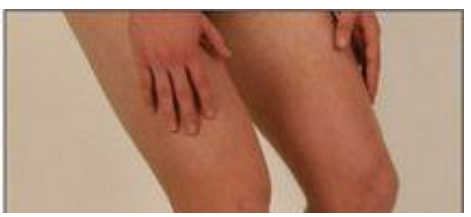
The clinician in a hurry can test lower limb power quickly by asking the patient to:

1. stand up on his or her toes (S1) (Figure 11.73a);
2. stand up on the heels (L4, L5) (Figure 11.73b);
3. squat and stand again (L3, L4) (Figure 11.73c).

This tests ankle, knee and hip power. Inability to perform any of the tests suggests a need to test more formally.







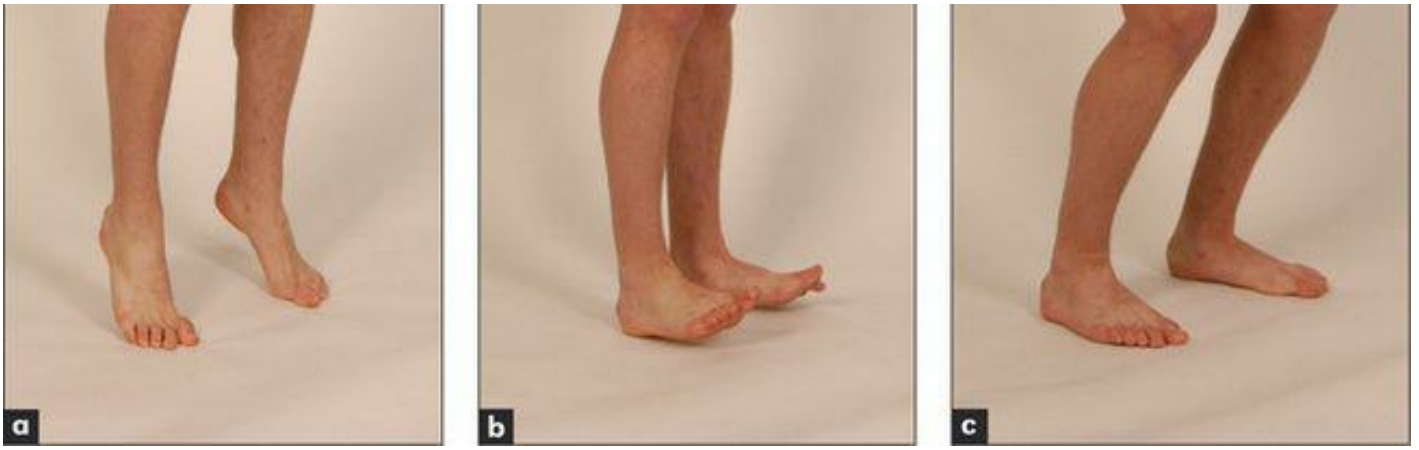


Figure 11.73 Quick test of lower limbs power
(a) 'Stand up on your toes.' (b) 'Now lift up on your heels.' (c) 'Now squat and stand up again.'

Reflexes

Test the following reflexes.

Knee jerk (L3, L4)

Slide one arm under the knees so that they are slightly bent and supported. The tendon hammer is allowed to fall onto the infrapatellar tendon (Figure 11.74). Normally, contraction of the quadriceps causes extension of the knee. Compare the two sides. If the knee jerk appears to be absent on one or both sides, it should be tested again following a reinforcement manoeuvre. Ask the patient to interlock the fingers and then pull apart hard at the moment before the hammer strikes the tendon (Jendrassik's manoeuvre[00]) (Figure 11.75). This manoeuvre has been shown to restore an apparently absent ankle jerk in 70% of elderly people. A reinforcement manoeuvre such as this, or teeth-clenching or grasping an object, should be used if there is difficulty eliciting any of the muscle stretch reflexes.

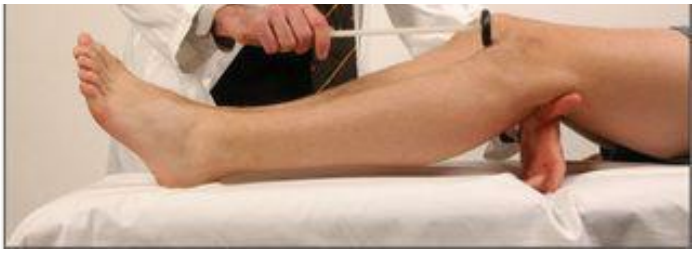


Figure 11.74 The knee jerk examination

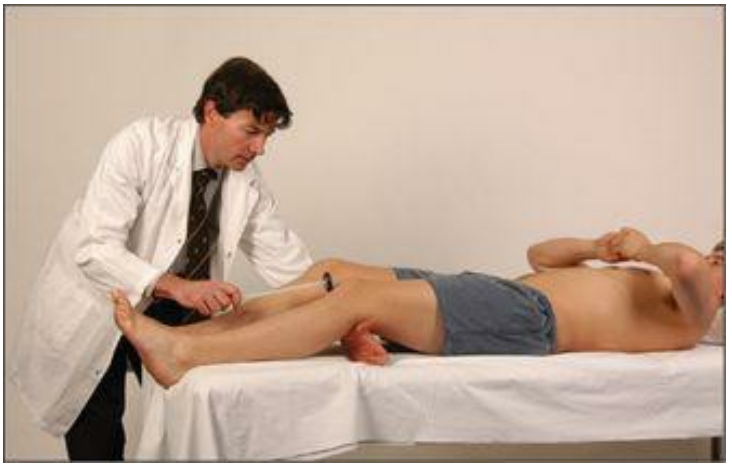


Figure 11.75 The knee jerk with reinforcement: 'Grip your fingers and pull your hands apart'

Ankle jerk (S1, S2)

Have the foot in the mid-position at the ankle with the knee bent, the thigh externally rotated on the bed, and the foot held in dorsiflexion by the examiner. The hammer is allowed to fall on the Achilles tendon (Figure 11.76). The normal response is plantar flexion of the foot with contraction of the gastrocnemius muscle. Again, test with reinforcement if appropriate. This reflex can also be tested with the patient kneeling (page 308) or by tapping the sole of the foot.[16]

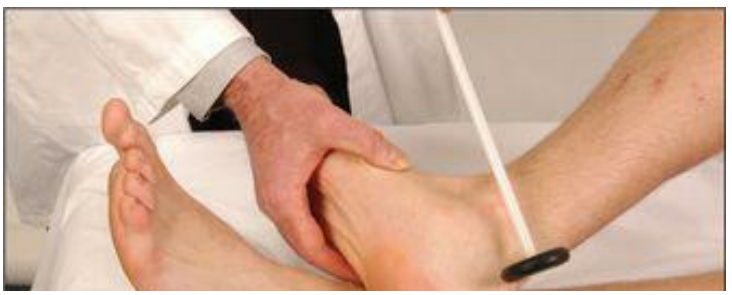


Figure 11.76 The ankle jerk (first method, see also page 308): the examiner dorsiflexes the foot slightly to stretch the tendon

Plantar reflex (L5, S1, S2)

After telling the patient what is going to happen, use a blunt object (such as the key to an expensive car) to stroke up the lateral aspect of the sole, and curve inwards before it reaches the toes, moving towards the middle metatarsophalangeal (MTP) joint (Figure 11.77). The patient's foot should be in the same position as for testing the ankle jerk. The normal response is flexion of the big toe at the MTP joint in patients over one year of age. The extensor (Babinski[pp])[17] response is abnormal and is characterised by extension of the big toe at the MTP joint (the upgoing toe) and fanning of the other toes. This indicates an upper motor neurone (pyramidal) lesion, although the test's reliability can be relatively poor. Bilateral upgoing toes may also be found after a generalised seizure, or in a patient in coma.

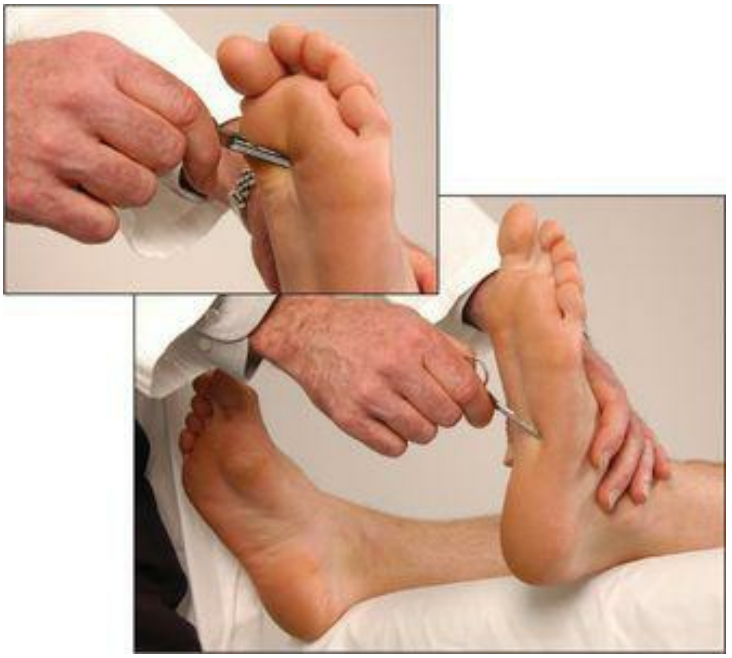


Figure 11.77 The plantar reflex examination

Coordination

Coordination

Test for cerebellar disease with three manoeuvres.

Heel–shin test

Ask the patient to run the heel of one foot up and down the opposite shin at a moderate pace and as accurately as possible (Figure 11.78). In cerebellar disease the heel wobbles all over the place, with oscillations from side to side and overshooting. Closing the eyes makes little difference to this in cerebellar disease, but if there is posterior column loss the movements are made worse when the eyes are shut—that is, there is ‘sensory ataxia’.

Figure 11.78 The heel–shin test: ‘Run your heel down your shin smoothly and quickly’

Toe–finger test

Unfortunately, a toe–nose test is not a practical way of assessing the lower limbs, so a toe–finger test is used. Ask the patient to lift the foot (with the knee bent) and touch the examiner’s finger with the big toe. Look for intention tremor.

Foot-tapping test

Rapidly alternating movements are tested by getting the patient to tap the sole of the foot quickly on the examiner's hand or tap the heel on the opposite shin. Look for loss of rhythmicity.

The sensory system

As for the upper limb, test for pain sensation first in each dermatome, comparing the right with the left side (Figure 11.79). Map out any abnormality and decide on the pattern of loss.

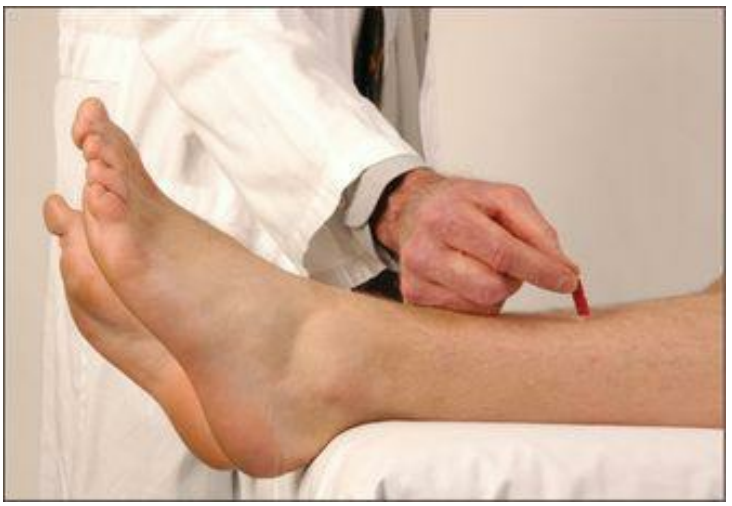
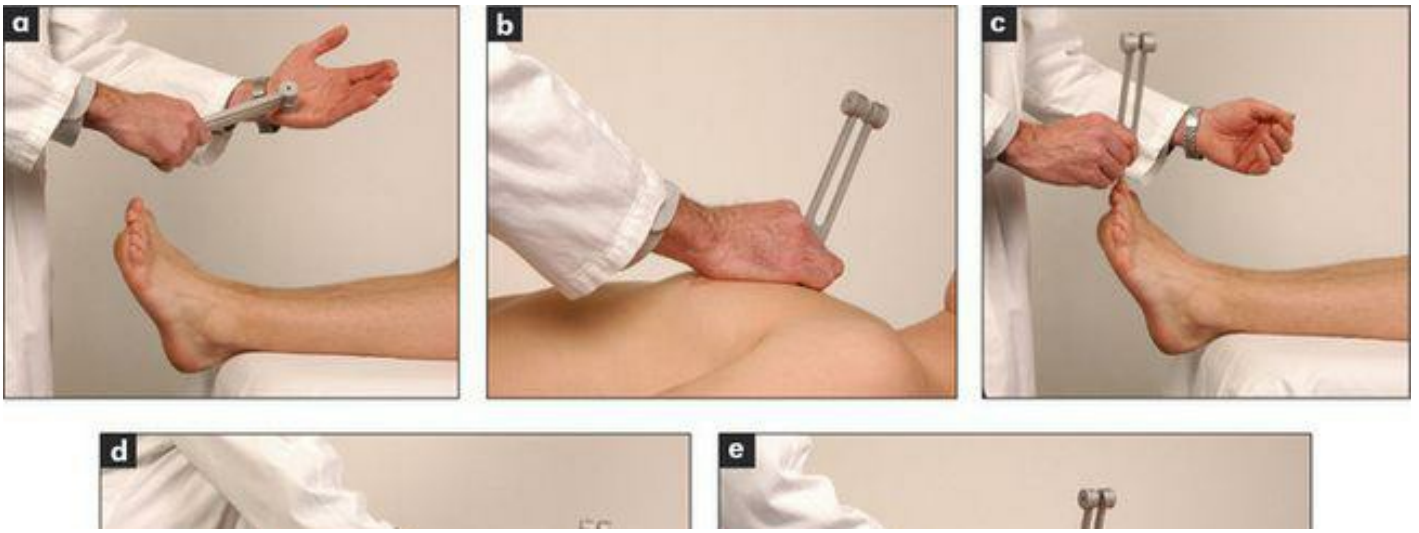


Figure 11.79 Testing pinprick (pain) sensation in the lower limbs (do not draw blood with the pin)

Then test vibration sense over the ankles and, if necessary, on the knees and the anterior superior iliac spines (Figure 11.80). Next test proprioception, using the big toes (Figure 11.81) and, if necessary, the knees and hips.



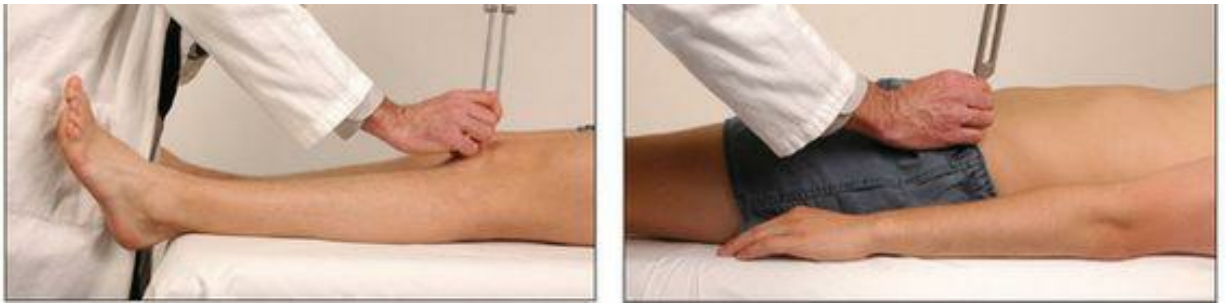


Figure 11.80 Testing vibration sense in the lower limbs
(a) Strike a 128 Hz tuning fork confidently on your thenar eminence. (b) Demonstrate the vibration of the tuning fork on the patient's sternum: 'Can you feel this vibration?' (c) Place the tuning fork on the great toe: 'Can you feel the vibration there? Tell me when it stops.' (d) If vibration sense is absent on the great toe, try testing on the patella. (e) If vibration sense is absent at the knee, try testing on the anterior superior iliac spine.

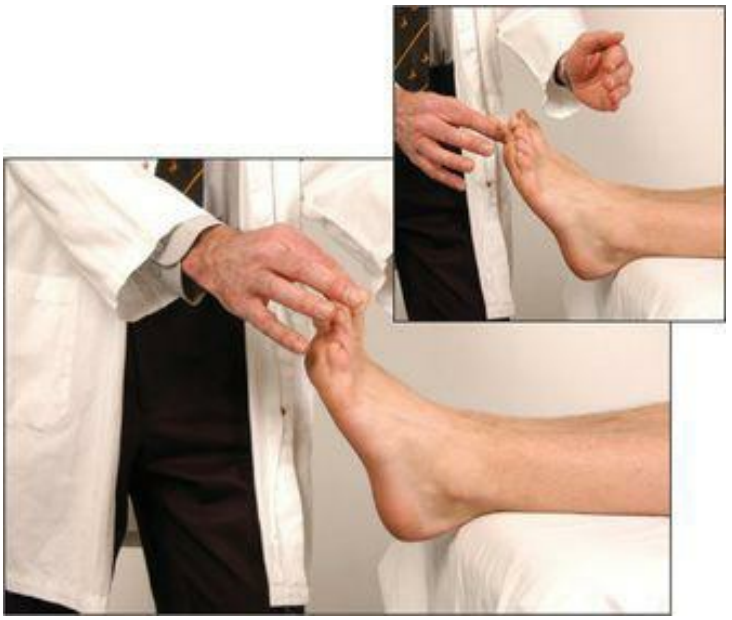


Figure 11.81 Position sense: 'Shut your eyes and tell me whether I have moved your toe up or down'

Finally, test light touch (Figure 11.82).

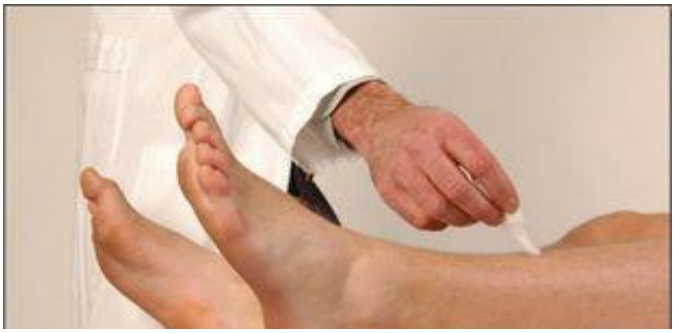


Figure 11.82 Testing light-touch sensation (do not stroke the skin with the cottonwool)

Dermatomes

Memorise the following rough guide (Figure 11.83): L2 supplies the upper anterior thigh; L3 supplies the area around the front of the knee; L4 supplies the medial aspect of the leg; L5 supplies the lateral aspect of the leg and the medial side of the dorsum of the foot; S1 supplies the heel and most of the sole; S2 supplies the posterior aspect of the thigh; S3, S4 and S5 supply concentric rings around the anus.

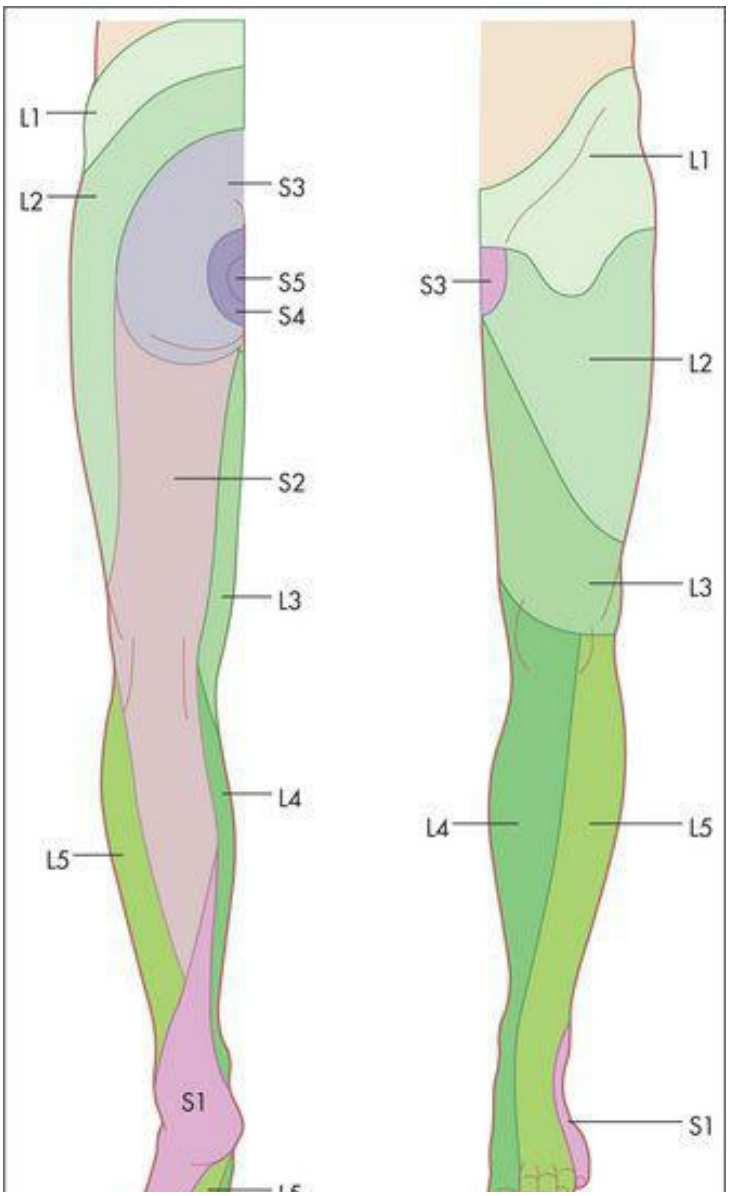


Figure 11.83 Dermatomes of the lower limbs

Sensory levels

If there is peripheral sensory loss in the leg, attempt to map out the upper level with a pin, moving up at 5 cm intervals initially, from the leg to the abdomen, until the patient reports it is sharp. This may involve testing over the abdominal or even the chest dermatomes. Establishing a sensory level on the trunk indicates the spinal cord level that is affected. Remember, a level of hyperaesthesia (increased sensitivity) often occurs above the sensory level and it is the upper level of this that should be determined, as it usually indicates the highest affected spinal segment. Also remember that the level of a vertebral body only corresponds to the spinal cord level in the upper cervical cord because the spinal cord is shorter than the spinal canal. The C8 spinal segment lies opposite the C7 vertebra. In the upper thoracic cord there is a difference of about two segments and in the mid-thoracic cord three segments. All the lumbosacral segments are opposite the T11 to L1 vertebrae.

The superficial or cutaneous reflexes

These reflexes occur in response to light touch or scratching of the skin or mucous membranes. The stimulus is more superficial than the tendon (muscle stretch) reflexes. As a rule these reflexes occur more slowly after the stimulus, are less constantly present and fatigue more easily.

Examples include the palmar or grasp reflex, the cremasteric reflex and the abdominal reflexes and the plantar responses.

The abdominal reflexes (epigastric T6–T9, mid-abdominal T9–T11, lower abdominal T11–L1)

Test these by lightly stroking the abdominal wall diagonally towards the umbilicus in each of the four quadrants of the abdomen (Figure 11.84). Reflex contractions of the abdominal wall are *absent* in upper motor neurone lesions above the segmental level and also in patients who have had surgical operations interrupting the nerves. They disappear in coma and deep sleep,

and during anaesthesia. They are usually difficult to elicit in obese patients and can also be absent in some normal people (20%). Their absence in the presence of increased tendon reflexes is suggestive of corticospinal tract abnormality.

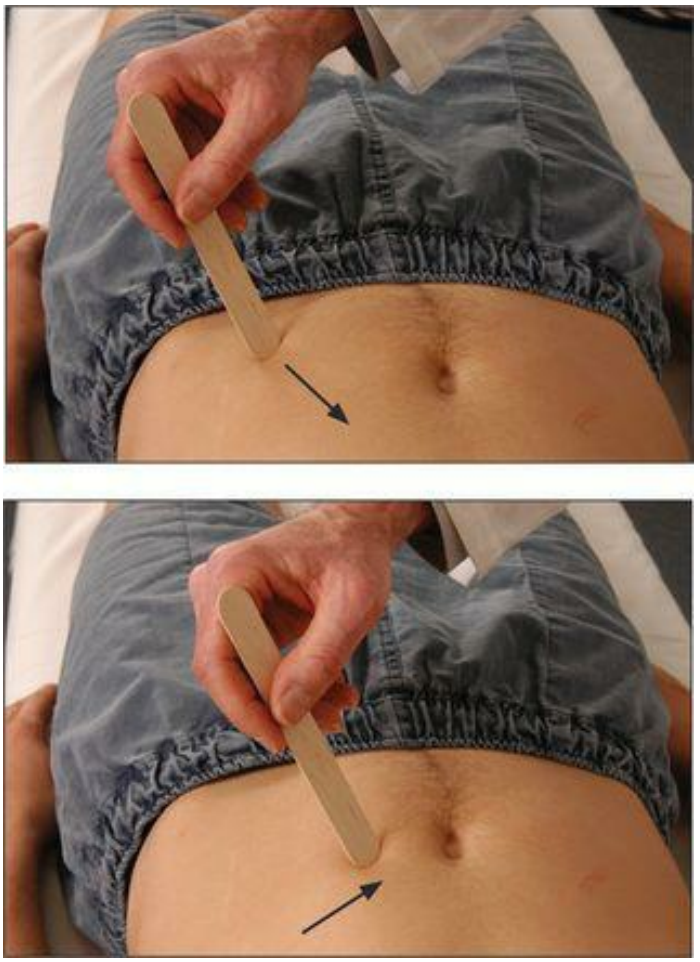


Figure 11.84 Abdominal reflex: stroke towards the umbilicus from each quadrant and watch for abdominal muscle contraction

The cremasteric reflexes (L1–L2)

Stroke the inner part of the thigh in a downward direction; normally contraction of the cremasteric muscle pulls up the scrotum and testis on the side stroked. It may be absent in elderly men and in those with a hydrocele or varicocele, or after an episode of orchitis.

Saddle sensation and anal reflex

Test now for saddle sensation if a cauda equina lesion is suspected (e.g.

Test flow for saddle sensation if a cauda equina lesion is suspected (e.g. because of urinary or faecal incontinence). The only sensory loss may be on the buttocks or around the anus (S3–S5). In this case also test the anal reflex (S2, S3, S4): normal contraction of the external sphincter in response to pinprick of the perianal skin is abolished in patients with a lesion of the sacral segments of the cauda equina. If, however, the lowest sacral segments are spared but the higher ones are involved, this suggests that there is an intrinsic cord lesion.

Spine

Examine the spine and perform the straight-leg raising test.

Examination of the peripheral nerves of the lower limb

Lateral cutaneous nerve of the thigh

Test for sensory loss (Figure 11.85). A lesion of this nerve usually occurs because of entrapment between the inguinal ligament and the anterior superior iliac spine. It causes a sensory loss over the lateral aspect of the thigh with no motor loss detectable. If painful, it is called *meralgia paraesthetica.*

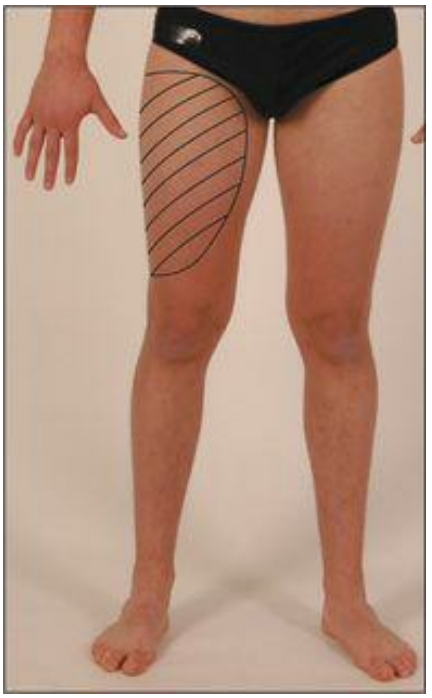


Figure 11.85 Distribution of the lateral cutaneous nerve of the thigh

Femoral nerve (L2, L3, L4)

Test for weakness of knee extension (quadriceps paralysis). Hip flexion weakness is only slight, and adductor strength is preserved. The knee jerk is absent. The sensory loss involves the inner aspect of the thigh and leg (Figure 11.86).

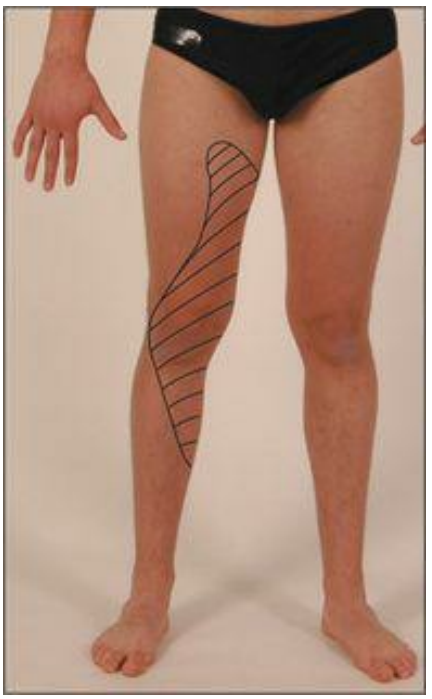


Figure 11.86 Sensory distribution of the femoral nerve

Sciatic nerve (L4, L5, S1, S2)

This nerve supplies all the muscles below the knee and the hamstrings. Test for loss of power below the knee resulting in a footdrop (plantar flexed foot) and for weakness of knee flexion. Test the reflexes: with a sciatic nerve lesion the knee jerk is intact but the ankle jerk and plantar response are absent. Test sensation on the posterior thigh, lateral and posterior calf, and on the foot (lost with a proximal nerve lesion)(Figure 11.87).

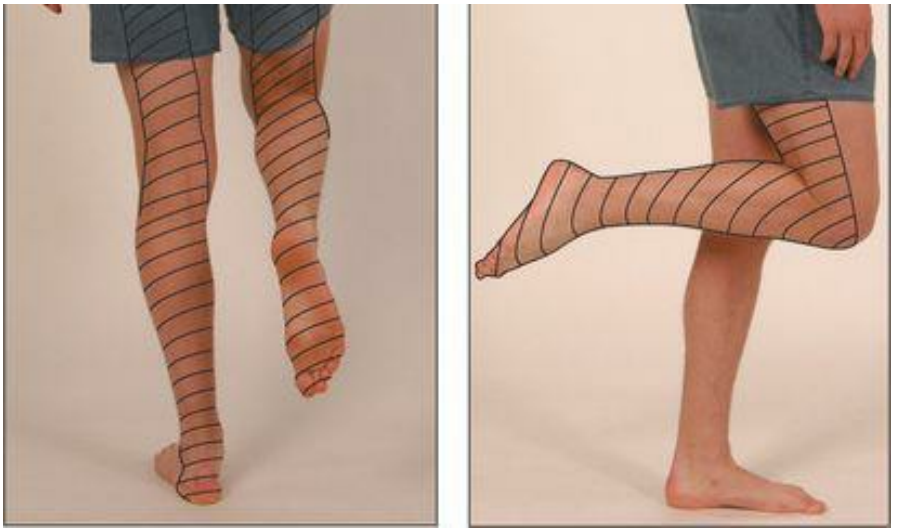


Figure 11.87 Sensory distribution of the sciatic nerve

Common peroneal nerve (L4, L5, S1)

This is a major terminal branch of the sciatic nerve. It supplies the anterior and lateral compartment muscles of the leg (Figure 11.88). On inspection one may notice a footdrop (Table 11.19). Test for weakness of dorsiflexion and eversion. Test the reflexes, which will all be intact. Test for sensory loss. There may be only minimal sensory loss over the lateral aspect of the dorsum of the foot. Note that these findings can be confused with an L5 root lesion, but the latter includes weakness of knee flexion and loss of foot inversion as well as sensory loss in the L5 distribution.

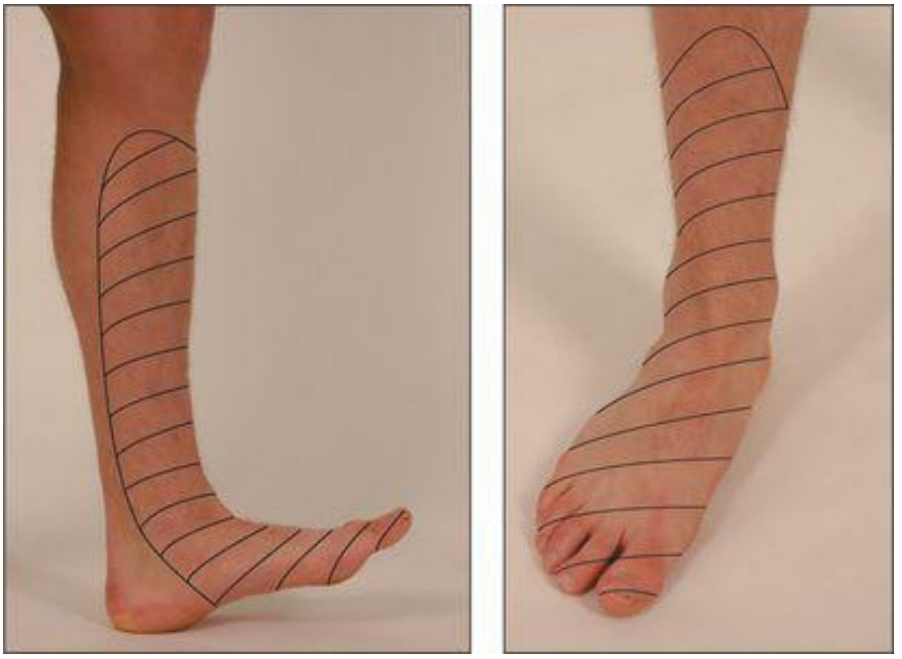


Figure 11.88 Sensory distribution of the common peroneal nerve (compression at the fibular neck)

TABLE 11.19 Causes (differential diagnosis) of footdrop

Common peroneal nerve palsy
Sciatic nerve palsy
Lumbosacral plexus lesion
L4, L5 root lesion
Peripheral motor neuropathy
Distal myopathy
Motor neurone disease
Stroke—anterior cerebral artery or lacunar syndrome (‘ataxic hemiparesis’)

Gait

Method

Make sure the patient’s legs are clearly visible. Now ask the patient to walk normally for a few metres and then turn around quickly and walk back. Then ask the patient to walk heel-to-toe to exclude a midline cerebellar lesion (Figure 11.89). Ask the patient to then walk on the toes (an S1 lesion will make this difficult or impossible) and then on the heels (an L4 or L5 lesion causing footdrop will make this difficult or impossible).

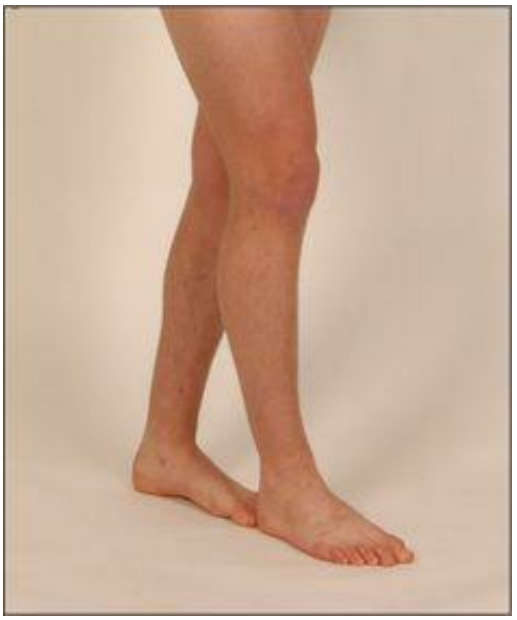
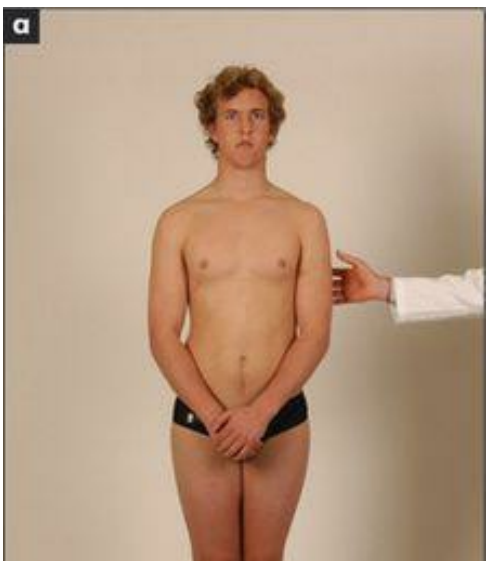


Figure 11.89 Cerebellar testing—heel–toe walking

Test for proximal myopathy by asking the patient to squat and then stand up, or sit in a low chair and then stand.

To test *station* (Romberg[qq] test), ask the patient to stand erect with the feet together and the eyes open (Figure 11.90a). Once the patient is stable ask him or her to close the eyes (Figure 11.90b). Compare the steadiness shown with the eyes open, then closed for up to 1 minute. Even in the absence of neurological disease a person may be slightly unsteady with the eyes closed, but the sign is positive if marked unsteadiness occurs to the point where the patient looks likely to fall. Normal people can maintain this position easily for 60 seconds. The Romberg test is positive when unsteadiness increases with eye closure. This is usually seen with the loss of proprioceptive sensation.



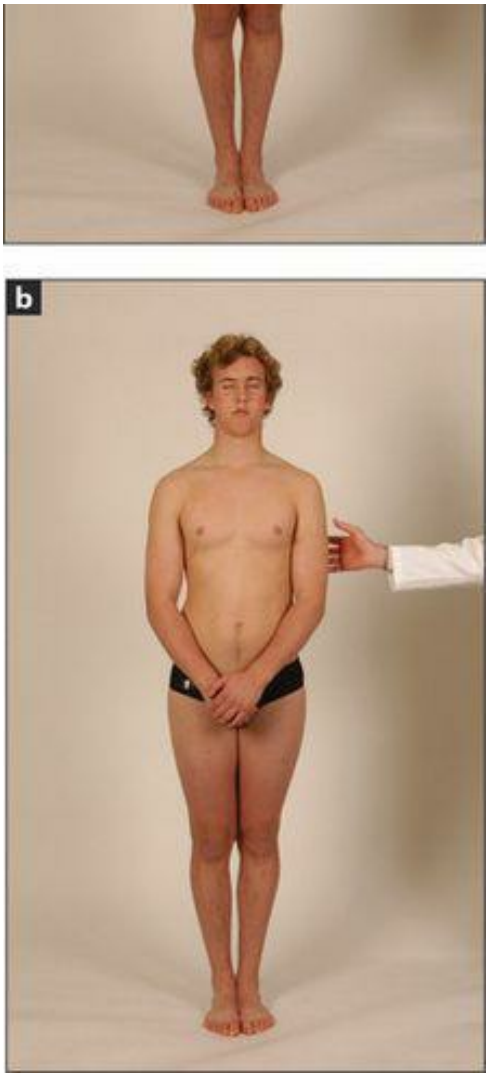


Figure 11.90 Romberg test
(a) 'Stand with your feet together.'
(b) 'Now shut your eyes. I won't let you fall.'

Marked unsteadiness with the eyes open is seen with cerebellar or vestibular dysfunction.

Gait disorders are summarised in Table 11.20.

TABLE 11.20 Gait disorders

Hemiplegia: the foot is plantar flexed and the leg is swung in a lateral arc
Spastic paraparesis: scissors gait
Parkinson's disease: • hesitation in starting • shuffling • freezing • festination • propulsion • retropulsion
Cerebellar: a drunken gait which is wide-based or reeling on a narrow base; the patient staggers towards the affected side if there is a unilateral cerebellar hemisphere lesion
Posterior column lesion: clumsy slapping down of the feet on a broad base
Footdrop: high-stepping gait
Proximal myopathy: waddling gait
Prefrontal lobe (apraxic): feet appear glued to floor when erect, but move more easily when the patient is supine
Hysterical: characterised by a bizarre, inconsistent gait

Speech and higher centres

At this stage of the examination dysarthria (difficulty with articulation), dysphonia (altered quality of the voice with reduction in volume as a result of vocal cord disease) or dysphasia (dominant higher centre disorder in the use of symbols for communication—language) may be obvious. If not, before going on to compartmentalised tests, the clinician should get the patient to talk freely—*propositional* or *free* speech. In a normal clinical encounter this

talk freely—*propositional* or *free* speech. In a normal clinical encounter this comes from history taking. In viva voce or observed standardised clinical examinations (OSCEs), ask the patient to describe the room, his or her clothes, or job or daily activities, in order to promote flowing speech. Then test *comprehension*. This is done first without eliciting language—for example, 'Touch your chin, then your nose and then your ear'; and then with yes and no questions—for example, 'Do you put your shoes on before your socks?' Then test *repetition*—for example, 'Repeat the phrase "no ifs, ands or buts".'

To complete the screening, ask the patient to *name two objects* pointed at, and to *say a phrase* such as 'British Constitution' or 'West Register St' (page 396).

There is no need to examine further if no abnormality of speech is detected in this way.

If there is an abnormality, proceed as outlined in Table 11.21.

TABLE 11.21 Examination of a patient with dysphasia

Fluent speech (receptive, conductive or nominal aphasia, usually)
1. Name objects. Patients with nominal, conductive or receptive aphasia will name objects poorly.
2. Repetition. Conductive and receptive aphasic patients cannot repeat 'no ifs, ands or buts'.
3. Comprehension. Only receptive aphasic patients cannot follow commands (verbal or written): 'Touch your nose, then your chin and then your ear'.
4. Reading. Conductive and receptive aphasic patients may have difficulty (dyslexia).
5. Writing. Conductive aphasic patients have impaired writing (dysgraphia) while receptive aphasic patients have abnormal content of writing. Patients with dominant frontal lobe lesions may also have dysgraphia

may also have dysgraphia.

Non-fluent speech (expressive aphasia, usually)

1. Naming of objects. This is poor but may be better than spontaneous speech.

2. Repetition. May be possible with great effort. Phrase repetition (e.g. 'No ifs, ands or buts') is poor.

3. Comprehension. Often mildly impaired despite popular belief, but written and verbal commands are followed.

4. Reading. Patients may have dyslexia.

5. Writing. Dysgraphia may be present.

6. Look for hemiparesis. The arm is more affected than the leg.

7. As patients are usually aware of their deficit they are often frustrated and depressed.

Dysphasia

There are four main types of dysphasia:[18] receptive, expressive, nominal and conductive. The expressive aphasias are forms of motor *apraxia*. This means the inability to perform deliberate actions in the absence of paralysis.

1. **Receptive (posterior) dysphasia**. This is where the patient cannot understand the spoken (*auditory dysphasia*) or written word (*alexia*). This condition is suggested when the patient is unable to understand any commands or questions or to recognise written words in the absence of deafness or blindness. Speech is fluent but disorganised. It occurs with a lesion (infarction, haemorrhage or space-occupying tumour) in the dominant hemisphere in the posterior part of the first temporal gyrus (Wernicke's area[rr]).

2. **Expressive (anterior) dysphasia**. This is present when the patient understands, but cannot answer appropriately. Speech is non-fluent. This occurs with a lesion in the posterior part of the dominant third frontal gyrus

(Broca's area[ss]). Certain types of speech may be retained by these patients. These include *automatic speech*. The patient may be able to recite word series such as the days of the week or letters of the alphabet. Sometimes *emotional speech* may be preserved so that when frustrated or upset the patient may be able to swear fluently. In the same way the patient may be able to sing familiar songs while unable to speak the words. It is important to remember that unless the lesion responsible for these defects is very large there may be no reduction in the patient's higher intellectual functions, memory or judgment. Some of these patients may incorrectly be considered psychotic, because of their disorganised speech.

3. **Nominal dysphasia**. All types of dysphasia cause difficulty naming objects. There is also a specific type of nominal dysphasia. Here objects cannot be named (e.g. the nib of a pen) but other aspects of speech are normal. The patient may use long sentences to overcome failure to find the correct word (circumlocution). It occurs with a lesion of the dominant posterior temporoparietal area. Other causes include encephalopathy or the intracranial pressure effects of a distinct space-occupying lesion; it may also occur in the recovery phase from any dysphasia. Its localising value is therefore doubtful.

4. **Conductive dysphasia**. Here patients repeat statements and name objects poorly, but can follow commands. This is thought to be caused by a lesion of the arcuate fasciculus and/or other fibres linking Wernicke's and Broca's areas.

To examine for dysphasia in more detail refer to Table 11.21. If the speech is fluent, but conveys information with paraphasic errors, such as 'treen' for 'train' (i.e. a word of similar sound or spelling to the one intended is used),[tt] the main possibilities are nominal, receptive and conductive dysphasia. Test for these by asking the patient to name an object, repeat a statement after you, and then follow commands. Then, if the above are abnormal, ask the patient to read and write, but remember that the occasional patient may be illiterate.

If the speech is slow, hesitant and non-fluent, expressive dysphasia is more likely and exactly the same procedure is followed. It is important to note that many dysphasias will have mixed elements. Large lesions in the dominant hemisphere may cause global dysphasia.

Dysarthria

Here there is no disorder of the content of speech but a difficulty with articulation. It can occur because of abnormalities at a number of levels

articulation. It can occur because of abnormalities at a number of levels. Upper motor neurone lesions of the cranial nerves, extrapyramidal conditions (e.g. Parkinson's disease) and cerebellar lesions cause disturbances to the rhythm of speech.

Ask the patient to say a phrase such as 'British Constitution' or 'Peter Piper picked a peck of pickled peppers'.

Pseudobulbar palsy is an upper motor neurone weakness that causes a spastic dysarthria (it sounds as if the patient is trying to squeeze out words from tight lips), paralysis of the facial muscles and difficulty chewing and swallowing. The cause is infarction in both internal capsules. This causes interruption of the descending pyramidal tracts to the brainstem motor nuclei. The jaw jerk is usually increased. These patients tend to be very emotional and laugh and cry inappropriately. Their facial expressions become very animated at these times in contrast to their inability to control their facial expressions voluntarily.[uu] This phenomenon occurs because the nuclei that control motor responses to emotion do not reside in the motor cortex.

Patients who have bilateral lesions of the ninth and tenth cranial nerves are at risk of aspirating fluids or solids into their lungs if they try to eat or drink. Certain bedside tests can be performed to see if it is safe for them to eat or drink. These traditionally include the level of consciousness, gag reflex, pharyngeal sensation and testing swallowing water. *Good signs guide 11.1* shows the likelihood ratios for these and other tests. The water swallowing test involves asking patient to sip repeatedly 5–10 mL of water. Coughing, choking or a fall in blood arterial oxygen saturation makes the test positive.

GOOD SIGNS GUIDE 11.1 Risk of aspiration after stroke

	Likelihood ratio if:	
	Present	**Absent**
Voice and cough		
Abnormal voluntary cough	1.9	0.6
Dysphonia	1.3	0.4
Examination		
Drowsiness	3.4	0.5
Abnormal sensation—face and tongue	NS	NS
Absent pharyngeal sensation	2.4	0.03
Abnormal gag reflex	1.5	0.6
Tests		
Water swallow test	3.2	0.4
Oxygen desaturation 2 min after swallowing	3.1	0.3

Adapted with permission from McGee S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.

Bulbar palsies cause a nasal speech, while *facial muscle* weakness causes slurred speech. *Extrapyramidal disease* can be responsible for monotonous speech, as it causes bradykinesia and muscular rigidity. Other causes of dysarthria include alcohol intoxication and *cerebellar disease.* These result in loss of coordination and slow, slurred and often explosive speech, or speech broken up into syllables, called scanning speech.

Mouth ulceration or disease may occasionally mimic dysarthria. Each of these causes must be considered and examined for as appropriate.

Dysphonia

This is alteration of the sound of the voice, such as huskiness of the voice with decreased volume. It may be due to laryngeal disease (e.g. following a viral infection or a tumour of the vocal cord), or to recurrent laryngeal nerve palsy.

The cerebral hemispheres

Parietal, temporal and frontal lobe functions are tested if the patient is disoriented or has dysphasia, or if dementia is suspected. If the patient has a receptive aphasia, however, these tests cannot be performed. Their examination is otherwise not routine (Table 11.22). Students should already be familiar with the method of examining the cranial nerves and limbs.

TABLE 11.22 Symptoms and signs in higher centre dysfunction

Parietal lobe
Dysphasia (dominant)
Acalculia,* agraphia,* left–right disorientation,* finger agnosia*
Sensory and visual inattention,‡ construction and dressing apraxia,‡ spatial neglect and inattention,‡ lower quadrantic hemianopia,‡ astereognosis‡
Seizures
Temporal lobe
Memory loss

Upper quadrantic hemianopia Dysphasia (receptive if dominant lobe) Seizures
Frontal lobe Personality change Primitive reflexes, e.g. grasp, pout Anosmia Optic nerve compression (optic atrophy) Gait apraxia Leg weakness (parasagittal) Loss of micturition control Dysphasia (expressive), dysgraphia Seizures
Occipital lobe Homonymous hemianopia Alexia (inability to read; word blindness) Seizures (flashing light aura)

* Gerstmann's syndrome: dominant hemisphere parietal lobe only.
† Non-dominant parietal lobe signs.
‡ Non-localising parietal lobe signs.

Parietal lobe function

The parietal lobe is concerned with the reception and analysis of sensory information.

Dominant lobe signs

A lesion of the dominant parietal lobe in the angular gyrus causes a distinct clinical syndrome called Gerstmann's syndrome. Test for this in the following manner.

1. Ask the patient to perform simple arithmetical calculations, e.g. serial 7s (take 7 from 100, then 7 from the answer, and so on). The inability to do this at least with partial accuracy is called ***A****calculia*.

2. Ask the patient to write—inability is called ***A****graphia*.

3. Test for left–right disorientation by asking the patient to show you his or her right and then left hand. If this is correctly performed, ask the patient to touch his or her left ear with the right hand and vice versa. Inability to do this is called ***L****eft–right disorientation*.

4. Ask the patient to name his or her fingers—inability to do this is called ***F****inger agnosia*. This inability may extend to identification of the examiner's fingers. The *agnosias* are receptive defects involving the inability to understand the meaning of stimulations of different types.

A mnemonic for these four dominant parietal lobe signs is **AALF**. Remember that Gerstmann's syndrome[vv] can be diagnosed only if the higher centres are intact. A demented patient would not be able to perform many of these tests.

Non-dominant and non-localising parietal lobe signs (*cortical sensation*)

Cortical sensations are those that require processing at a higher level than simple sensation. They rely on an intact simple sensation, especially touch and pinprick.

• *Graphesthesia* is the ability to recognise numbers or letters drawn on the skin. Use a pointed object or pencil to draw numbers on the skin (Figure 11.91).

• *Tactile extinction* is the ability to feel a stimulus when it is applied to

each side separately, but not on one side when both sides are stimulated. The patient (with eyes shut) is touched first on one hand and then on the other, and then on both together. Ask on which side the touch is felt. The normal response is 'both' when stimulation is applied to each side. It is important that the hands be touched simultaneously.

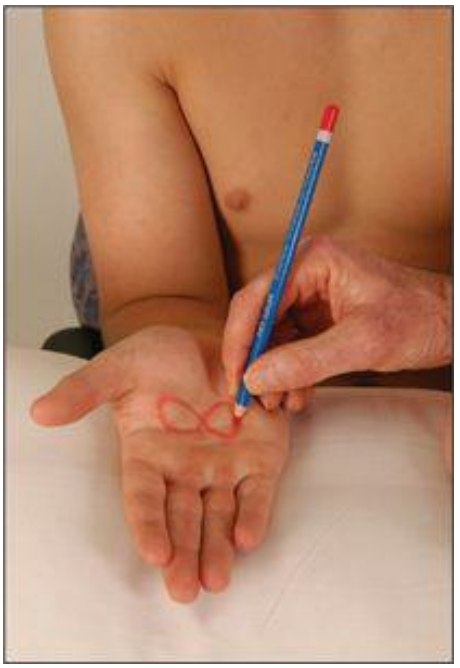


Figure 11.91 Agraphesthesia: 'What number have I drawn?' Patient's reply: 'One' Avoid the use of an indelible pencil.

Now test for general signs of parietal lobe dysfunction.

• Look for *sensory* and *visual inattention*. When one arm or leg is tested at a time, sensation is normal, but when both sides are tested simultaneously the sensation is appreciated only on the normal side. A right-sided parietal lesion will lead to inattention on the left side and vice versa.

• Formal *visual field testing* is also important, as parietal, temporal and occipital lesions can give distinctive defects.

• Examine now for *astereognosis (tactile agnosia)*, which is the inability, with eyes closed, to recognise an object placed in the hand when the ordinary sensory modalities are intact. A parietal lobe lesion results in astereognosis on the opposite side.

• *Agraphesthesia* may also be present; this is the inability to appreciate a number drawn on the hand on the opposite side to a parietal lesion (Figure 11.91).

• *Two-point discrimination* testing involves the ability to distinguish a single point from two points close together (Figure 11.92). The minimal separation that can be distinguished is about 3 cm on the hand or foot and 0.6 cm on the fingertips. A compass can be used for this test. Ask the patient to shut the eyes and then say whether one or two points can be felt. Bring the compass points closer together and test intermittently with just one point.

• Examine for *dressing* and *constructional apraxia*. Dressing apraxia is tested by taking the patient's pyjama top or cardigan, turning it inside out and asking him or her to put it back on. Patients with a non-dominant parietal lobe lesion may find this impossible to do. Constructional apraxia is tested by asking the patient to copy an object that you have drawn (e.g. a tree or a house—Figure 11.93a & b).

• Next test *spatial neglect* by asking the patient to fill in the numbers on an empty clock face (Figure 11.93c). Patients with a right parietal lesion may fill in numbers only on the left side (the other side of the clock face is ignored). Spatial neglect also occurs with dominant parietal lobe lesions but is less common.

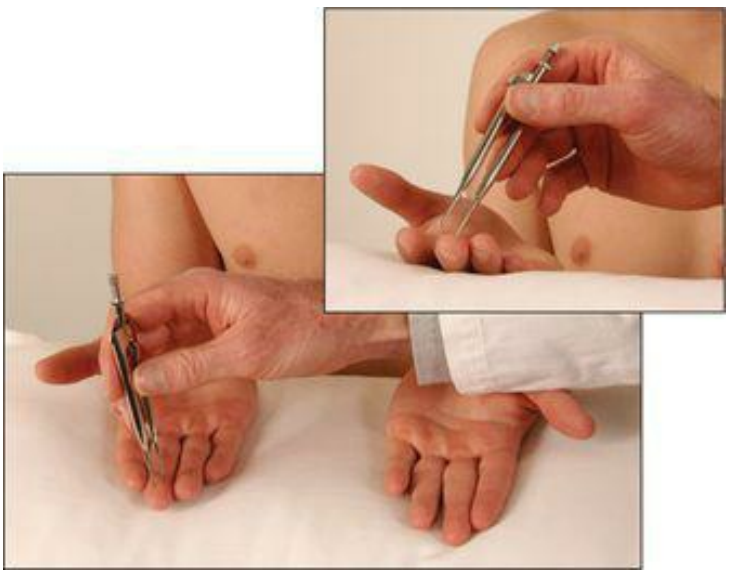


Figure 11.92 Two-point discrimination: 'Can you feel one point or two?'

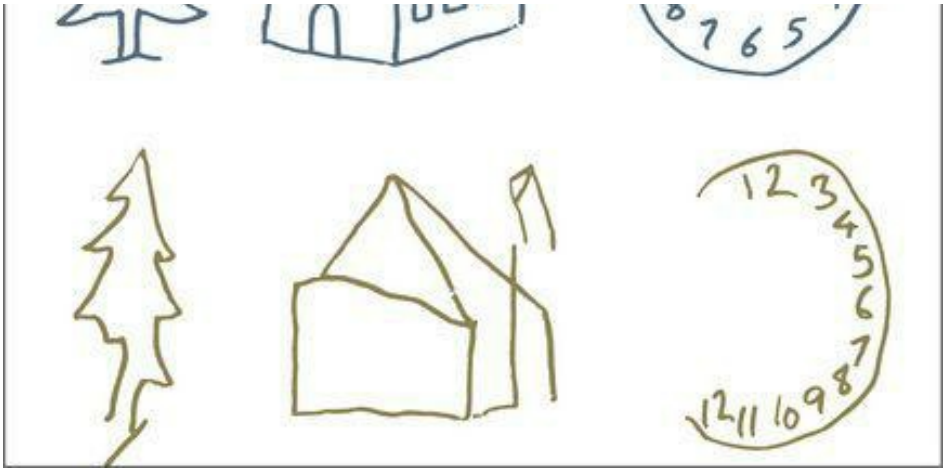


Figure 11.93 Lower figures show constructional apraxia (a & b) and spatial neglect (c)

Temporal lobe function

This lobe is concerned with short-term and long-term memory. Test short-term memory by the name, address, flower test—ask the patient to remember a name, address and the names of three flowers, and repeat them immediately. Then ask the patient five minutes later to repeat the names again. Test long-term memory by asking, for example, what year World War II ended. Memory may be impaired in dementia from any cause.

An alert patient with a severe memory disturbance may make up stories to fill any gaps in his or her memory. This is called *confabulation*, and is typical of the syndrome of Korsakoff's psychosis[ww] (amnesic dementia). Confabulation can be tested by asking the patient whether he or she has met you before. However, be prepared for the very long, detailed and completely false story that may follow.

Korsakoff's psychosis occurs most commonly in alcoholics (where there is loss of nerve cells in the thalamic nuclei and mammillary bodies), and rarely with head injury, tumour, anoxic encephalopathy or encephalitis. It is characterised by retrograde amnesia (memory loss for events before the onset of the illness) and an inability to memorise new information, in a patient who is alert, responsive and capable of problem-solving.

Frontal lobe function

Frontal lobe damage as a result of tumours or surgery (or both), or diffuse disease such as dementia or HIV infection, may cause changes in emotion, memory, judgment, carelessness about personal habits, and disinhibition. There may be persistent or alternating irritability and euphoria.[xx] These

features may be clear when the history is taken but may need to be reinforced by interviewing relatives or friends. Changes of this sort in a previously reserved personality may be obvious and very distressing to relatives.

Assess first the *primitive reflexes.* There is controversy concerning their significance; they are not normally present in adults but may reappear in normal old age.[19] The presence of an isolated primitive reflex may not be abnormal, but multiple primitive reflexes are usually associated with diffuse cerebral disease involving the frontal lobes and frontal association areas more than other parts of the brain. Dementia, encephalopathy and neoplasms are all possible causes.

1. Grasp reflex: the examiner runs his or her fingers across the palm of the patient's hand, which will grasp the examining fingers involuntarily on the side contralateral to the lesion.

2. Palmomental reflex: ipsilateral contraction of the ipsilateral mentalis muscle occurs when the examiner strokes the thenar eminence firmly with a key or thumb nail. Contraction of the mentalis causes protrusion and lifting of the lower lip. This is best considered as the beginning of a wince in response to pain. The response can also be elicited by painful stimulus to other parts of the body. The response is bilateral in about 50% of cases. A unilateral lesion does not necessarily correspond to the side of the lesion in the brain.

3. Pout and snout reflexes: stroking or tapping with the tendon hammer over or above the upper lip induces pouting movements of the lips. This can occur with many intracranial lesions. The sucking reflex is an extension of this. The stimulation may produce sucking, chewing and swallowing movements. It is not a localising sign.

Next ask the patient to *interpret a proverb*, such as 'A rolling stone gathers no moss'. Patients with frontal lobe disease give concrete explanations of proverbs. Test for loss of smell (*anosmia*) and for *gait apraxia*, where there is marked unsteadiness in walking, which can be bizarre—the feet typically behave as if glued to the floor, causing a strange shuffling gait. Look in the *fundi*; you may rarely see optic atrophy on the side of a frontal lobe space-occupying lesion caused by compression of the optic nerve, and papilloedema on the opposite side due to secondarily raised intracranial pressure (Foster Kennedy[vv] syndrome).

Correlation of physical signs and neurological disease

Upper motor neurone lesions

In neurology a clinical diagnosis is made by defining the deficit that is present, deciding on its anatomical level and then considering the likely causes. It is important to be able to distinguish *upper motor neurone* signs (*Good signs guide 11.2*) from *lower motor neurone* signs (Figure 11.38, page 355; see also Table 11.26, page 386). The former occur when a lesion has interrupted a neural pathway at a level above the anterior horn cell: for example, motor pathways in the cerebral cortex, internal capsule, cerebral peduncles, brainstem or spinal cord. When this occurs there is greater weakness of abductors and extensors in the upper limb, and flexors and abductors in the lower limb, as the normal function of this pathway is to mediate voluntary contraction of the antigravity muscles. All muscles, however, are usually weaker than normal. Muscle wasting is slight or absent, probably because there is no loss of trophic factors normally released from the lower motor neurone. The disuse that results from severe weakness may, however, cause some atrophy.

GOOD SIGNS GUIDE 11.2 Unilateral hemisphere lesion

Sign	Positive LR	Negative LR
Upper limb drift	33.0	0.2
Facial weakness	32.3	0.2
Hemiparesis	31.7	0.3
Wrist extensor weakness	29.0	0.3
Babinski response	19.0	0.6
Hemianopia	NS	0.7
Hemisensory disturbance	NS	0.7

NS = not significant.

From McGee S, Evidence-based physical diagnosis. 2nd edn. St Louis: Saunders, 2007.

Upper motor neurone signs occur when the lesion is in the brain or spinal cord above the level of the lower motor neurone (Table 11.23).

TABLE 11.23 Level of upper motor neurone lesion

1 Leg affected: L1 or above
2 Arm affected: C3 or above
3 Face affected: pons or above
4 Diplopia: midbrain or above

Spasticity occurs because of destruction of the corticoreticulospinal tract, resulting in stretch reflex hyperactivity. *Monoplegia* is paralysis affecting only one limb, when there is a motor cortex or partial internal capsule lesion. *Hemiplegia* affects one side of the body due to a lesion affecting projection of pathways from the contralateral motor cortex.

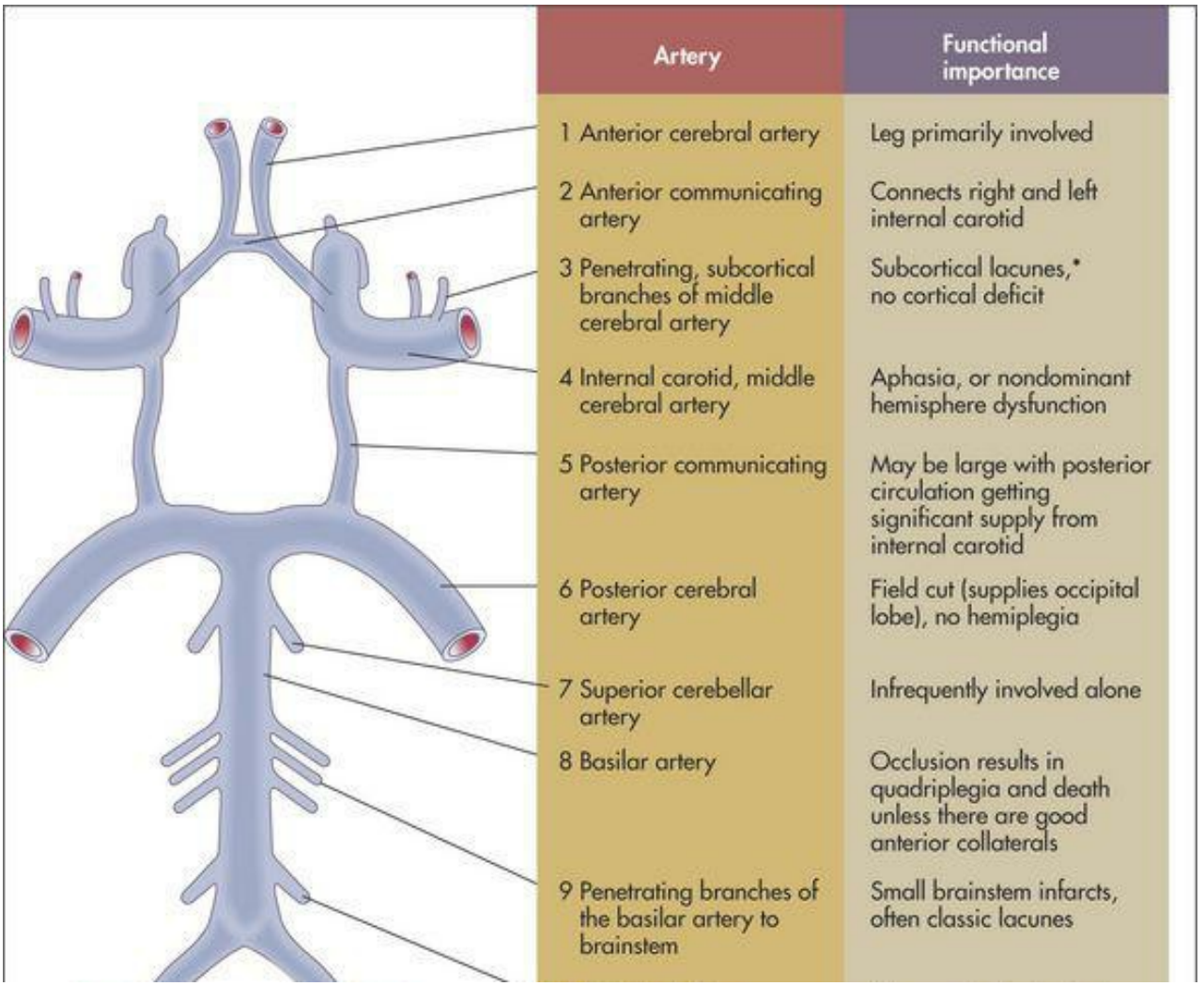
Paraplegia affects both legs, while *quadriplegia* affects all four limbs, and is the result of spinal cord trauma or, less often, a brainstem lesion (e.g.

basilar artery thrombosis).

Causes of hemiplegia (upper motor neurone lesion)

Vascular disease (stroke or TIA)

Thrombosis, embolism or haemorrhage occur in specific vascular territories (Figure 11.94).[20] Thrombus induces symptoms and signs in a slow stepwise progression. Embolic strokes are worse at the onset. Symptoms lasting less than 24 hours are called transient ischaemic attacks (TIAs). Lesions in the territory of the *internal carotid artery* result in hemiplegia on the opposite side of the body if a large area of the internal capsule or hemisphere is involved. Homonymous hemianopia, hemianaesthesia and dysphasia may occur (Table 11.24). Stenosis of the internal carotid artery in the neck may be associated with a bruit.[21]



Artery	Functional importance
1 Anterior cerebral artery	Leg primarily involved
2 Anterior communicating artery	Connects right and left internal carotid
3 Penetrating, subcortical branches of middle cerebral artery	Subcortical lacunes,* no cortical deficit
4 Internal carotid, middle cerebral artery	Aphasia, or nondominant hemisphere dysfunction
5 Posterior communicating artery	May be large with posterior circulation getting significant supply from internal carotid
6 Posterior cerebral artery	Field cut (supplies occipital lobe), no hemiplegia
7 Superior cerebellar artery	Infrequently involved alone
8 Basilar artery	Occlusion results in quadriplegia and death unless there are good anterior collaterals
9 Penetrating branches of the basilar artery to brainstem	Small brainstem infarcts, often classic lacunes

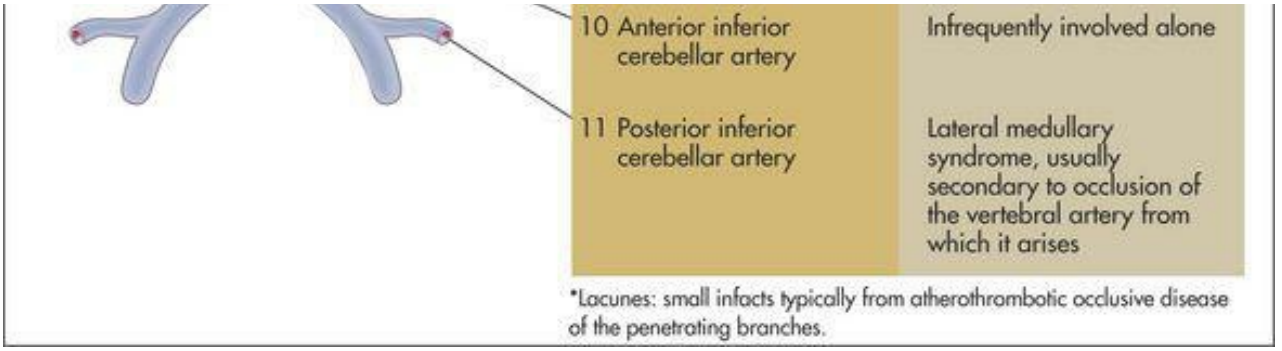


Figure 11.94 Anatomy of the circle of Willis, showing the functional importance of the arterial blood supply

Adapted from Weiner HL, Levitt LP, Neurology for the house officer, 6th edn. Baltimore: Williams & Wilkins, 2000.

TABLE 11.24 Intracerebral thrombosis or embolism: clinical features

Middle cerebral artery	Posterior cerebral artery	Anterior cerebral artery	Vertebral/basilar (brainstem)
Main branch Infarction middle third of hemisphere: UMN face, arm>leg; homonymous hemianopia; aphasia or non-dominant hemisphere signs (depends on side); cortical sensory loss	Main branch* Infarction of thalamus and occipital cortex: hemianaesthesia (loss of all modalities); homonymous hemianopia (complete); colour blindness	UMN leg>arm; cortical sensory loss leg only; (if corpus callosum affected) urinary incontinence	'Crossed' motor/sensory (e.g. left face, right arm); bilateral extremity motor/sensory; Horner's syndrome (Chapter 13); cerebellar signs; lower cranial nerve signs
Perforating artery Internal capsule infarction: UMN face, UMN arm>leg			

UMN = upper motor neurone lesion.
* Effects variable because of anastomoses with distal middle cerebral artery branches and supply from posterior communicating artery, but one would examine particularly for occipital and temporal lobe dysfunction.

Haemorrhagic strokes often involve the internal capsule and putamen (causing a contralateral hemiparesis and often sensory loss), or the thalamus (causing a contralateral hemianaesthesia).

Lesions in the territory of the *vertebrobasilar artery* may produce cranial nerve palsies, cerebellar signs, Horner's syndrome (Chapter 13) and sensory loss, as well as upper motor neurone signs (often bilateral because of the close proximity of structures in the brainstem). For example, a lesion in the midbrain may be associated with a third nerve paralysis and upper motor neurone signs on the opposite side. Hemianaesthesia and homonymous

hemianopia may occur if the posterior cerebral arteries are affected. An important syndrome to recognise is the *lateral medullary syndrome* (Table 11.25). Atheroma in the ascending aorta is increasingly recognised as a source of cerebral emboli.

TABLE 11.25 Lateral medullary syndrome ('Wallenberg's* syndrome')

Occlusion of the vertebral, or posterior inferior cerebellar or lateral medullary arteries causes ipsilateral and 'crossed' neurological signs
• Cerebellar signs (ipsilateral)
• Homer's syndrome (ipsilateral)
• Lower cranial nerves (IX, X)—palate and vocal cord weakness (ipsilateral)
• Facial sensory loss of pain (ipsilateral)
• Arm and leg sensory loss of pain (contralateral)
• No upper motor neuron weakness

* Adolf Wallenberg (1862–1942), professor of medicine, Danzig.

Compressive and infiltrative lesions

Tumours tend to occur in the lobes of the brain, and focal signs will depend on the tumour site. Signs localised to the parietal, temporal, occipital or frontal lobe suggest this disease process (see Table 11.22).

There may, however, be false localising signs in the presence of raised intracranial pressure: for example, a unilateral or bilateral sixth nerve palsy (because of the nerve's long intracranial path). Papilloedema is usually associated if there is raised intracranial pressure.

Demyelinating disease

Multiple sclerosis results in lesions in different areas usually with a relapsing and remitting course.

Infection

Human immunodeficiency virus (HIV) infection.

Lower motor neurone lesions

Lower motor neurone lesions interrupt the spinal reflex arc and therefore cause muscle wasting, reduced or absent reflexes and sometimes fasciculations. This results from a lesion of the spinal motor neurones, motor root or peripheral nerve (Table 11.26).

TABLE 11.26 Upper and lower motor neurone lesions (see also 8, page 355)

Signs of upper motor neurone (pyramidal) lesions
1. Weakness is present in all muscle groups, but in the lower limb may be more marked in the flexor and abductor muscles. In the upper limb, weakness may be most marked in the abductors and extensors. There is very little muscle wasting. 2. Spasticity: increased tone is present (may be clasp-knife) and often associated with clonus. 3. The reflexes are increased except for the superficial reflexes (e.g. abdominal), which are absent. 4. There is an extensor (Babinski) plantar response (upgoing toe).
Signs of lower motor neurone lesions
1. Weakness may be more obvious distally than proximally, and the flexor and extensor muscles are equally involved. Wasting is a

prominent feature.

2. Tone is reduced.

3. The reflexes are reduced and the plantar response is normal or absent.

4. Fasciculation may be present.

Motor neurone disease

This disease of unknown aetiology results in pathological changes in the anterior horn cells, the motor nuclei of the medulla and the descending tracts. It therefore causes a combination of upper motor neurone and lower motor neurone signs, although one type may predominate.

Importantly, fasciculations are almost always present. The muscle stretch reflexes are usually present (often increased) until late in the course of the disease, and there are rarely any objective sensory changes (15%–20% of patients report sensory symptoms).

Peripheral neuropathy

Distal parts of the nerves are usually involved first because of their distance from the cell bodies, causing a distal loss of sensation or motor function, or both, in the limbs. A typical sensory change is a symmetrical glove and stocking loss to all modalities (see Figure 11.95). This is unlike the pattern found with individual nerve or nerve root disease, which should be suspected if sensory loss is asymmetrical or confined to one limb. Peripheral muscle weakness may be present due to motor nerve involvement. Occasionally motor neuropathy may occur without sensory change. In the latter case, reflexes are reduced but may not be absent in the distal parts of the limbs (Table 11.27).

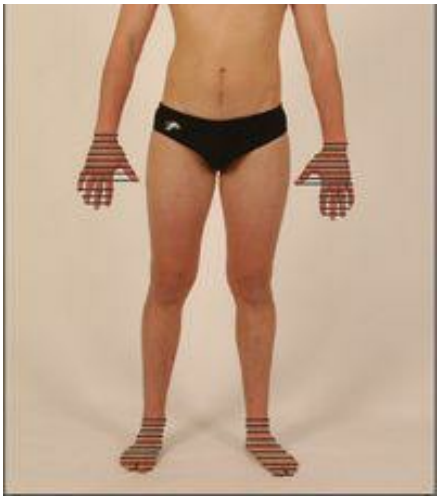


Figure 11.95 Peripheral neuropathy: glove and stocking sensory loss

TABLE 11.27 Peripheral neuropathy (Figure 11.95)

Causes (differential diagnosis) of peripheral neuropathy
1. Drugs—e.g. isoniazid, vincristine, phenytoin, nitrofurantoin, cisplatinum, heavy metals, amiodarone
2. Alcohol abuse (with or without vitamin B_1 deficiency)
3. Metabolic—e.g. diabetes mellitus, chronic kidney disease (renal failure)
4. Guillain-Barré syndrome
5. Malignancy—e.g. carcinoma of the lung (paraneoplastic neuropathy), leukaemia, lymphoma
6. Vitamin deficiency (e.g. B_{12}) or excess (e.g. B_6)
7. Connective tissue disease or vasculitis—e.g. PAN, SLE
8. Hereditary—e.g. hereditary motor and sensory neuropathy

9. Others, e.g. amyloidosis, HIV infection 10. Idiopathic
Causes of a predominant motor neuropathy 1. Guillain-Barré syndrome, chronic inflammatory polyradiculoneuropathy 2. Hereditary motor and sensory neuropathy 3. Diabetes mellitus 4. Others—e.g. acute intermittent porphyria, lead poisoning, diphtheria, multifocal conduction block neuropathy
Causes of a painful peripheral neuropathy 1. Diabetes mellitus 2. Alcohol 3. Vitamin B_1 or B_{12} deficiency 4. Carcinoma 5. Porphyria 6. Arsenic or thallium poisoning

PAN = polyarteritis nodosa; SLE = systemic lupus erythematosus; HIV = human immunodeficiency virus.

Guillain-Barré syndrome (acute inflammatory polyradiculoneuropathy)

This disease, thought to have an immune basis, may begin 7 to 10 days after an infective illness. It results in flaccid proximal and distal muscle paralysis, which typically ascends from the lower to the upper limbs. Wasting is rare

which typically ascends from the lower to the upper limbs. Wasting is rare. The reflexes are reduced or absent. The cranial nerves can be affected; occasionally disease is confined to these. Sensory loss is minimal or absent. Unlike transverse myelitis, the sphincters are little affected. Weakness of the respiratory muscles can be fatal but the disease is usually self-limiting. HIV infection can cause a similar syndrome.

Multiple sclerosis

This disease with unknown cause is characterised by scattered areas of inflammation in the central nervous system (CNS). A careful history is necessary as the diagnosis depends on the occurrence of at least two neurological episodes separated in time and place within the CNS; see Table 11.28.

TABLE 11.28 Clinical manifestations suggestive of multiple sclerosis

• Internuclear ophthalmoplegia (affected eye—weak adduction; other eye on abduction horizontal nystagmus)
• Optic neuritis (central visual loss, eye pain, pale optic disc)
• Marcus Gunn pupil
• Upper motor neurone weakness
• Cerebellar signs
• Posterior columns sensory loss
• Faecal/urinary incontinence

The examination

The signs can be very variable. Look particularly for signs of spastic paraparesis and posterior column sensory loss as well as cerebellar signs.

Examine the cranial nerves. Look carefully for loss of visual acuity

Examine the cranial nerves. Look carefully for loss of visual acuity, optic atrophy, papillitis and scotomata (usually central). *Internuclear ophthalmoplegia* is an important sign and is almost diagnostic in a young adult. Internuclear ophthalmoplegia is weakness of adduction in one eye as a result of damage to the ipsilateral medial longitudinal fasciculus; there may be nystagmus in the abducting eye. Bilateral internuclear ophthalmoplegia is almost always caused by MS.

Other cranial nerves may rarely be affected (III, IV, V, VI, VII, pseudobulbar palsy) by lesions within the brainstem. Charcot's triad for multiple sclerosis consists of nystagmus, intention tremor and scanning speech, but occurs in only 10% of patients.

Look for Lhermitte's[zz] sign (an electric-shock-like sensation in the limbs or trunk following neck flexion). This can also be caused by other disorders of the cervical spine, such as subacute combined degeneration of the cord, cervical spondylosis, cervical cord tumour, foramen magnum tumours, nitrous oxide abuse, and from mantle irradiation.

Thickened peripheral nerves

If there is evidence of a peripheral nerve lesion, peripheral neuropathy or a mononeuritis multiplex (Table 11.29), palpate for thickened nerves. The median nerve at the wrist, the ulnar nerve at the elbow, the greater auricular nerve in the neck and the common peroneal nerve at the head of the fibula are the most easily accessible. If nerves are thickened consider the following diagnoses:

- Acromegaly
- Amyloidosis
- Chronic inflammatory demyelinating polyradiculoneuropathy
- Leprosy
- Hereditary motor and sensory neuropathy (autosomal dominant) (Table 11.35, page 394)
- Other—e.g. sarcoidosis, diabetes mellitus, neurofibromatosis.

TABLE 11.29 Mononeuritis multiplex

Definition: mononeuritis multiplex refers to the separate involvement of more than one peripheral (or less often cranial) nerve by a single disease
Acute causes (usually vascular)
Polyarteritis nodosa
Diabetes mellitus
Connective tissue disease—e.g. rheumatoid arthritis, systemic lupus erythematosus
Chronic causes
Multiple compressive neuropathies
Sarcoidosis
Acromegaly
HIV infection
Leprosy
Lyme disease
Others—e.g. carcinoma (rare)

Spinal cord compression (Figures 11.96 to 11.100)

It is important to remember that a spinal cord lesion causes lower motor neurone signs at the level of the lesion and upper motor neurone signs below that level (Table 11.30). Don't forget the spinal cord's anatomy and vascular supply (Figure 11.96). Examine any suspected case as follows.

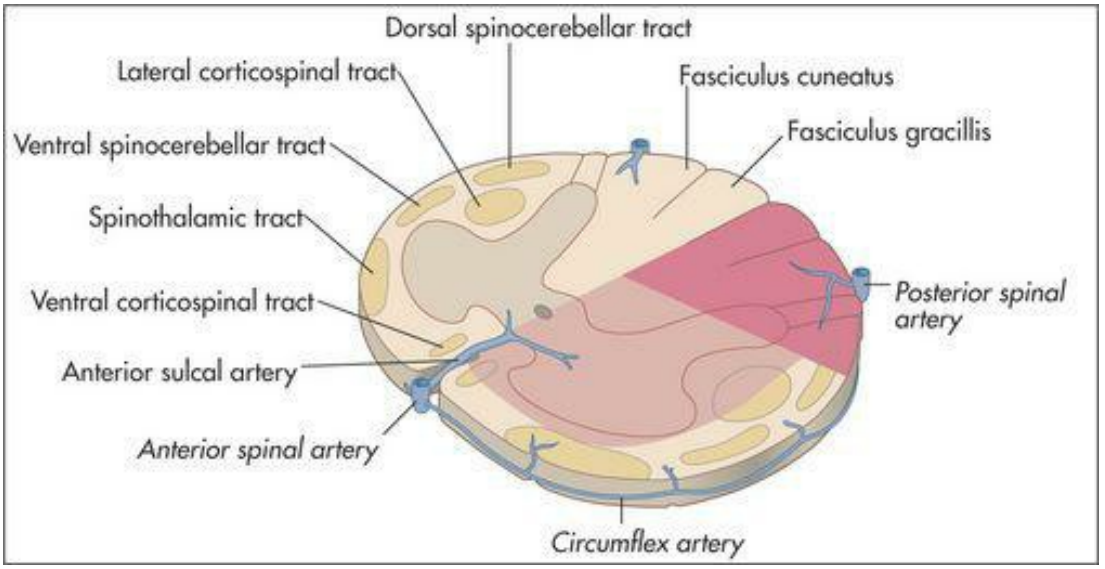


Figure 11.96 Anatomy and vascular supply of the spinal cord
Note: Anterior spinal artery occlusion spares posterior column function.

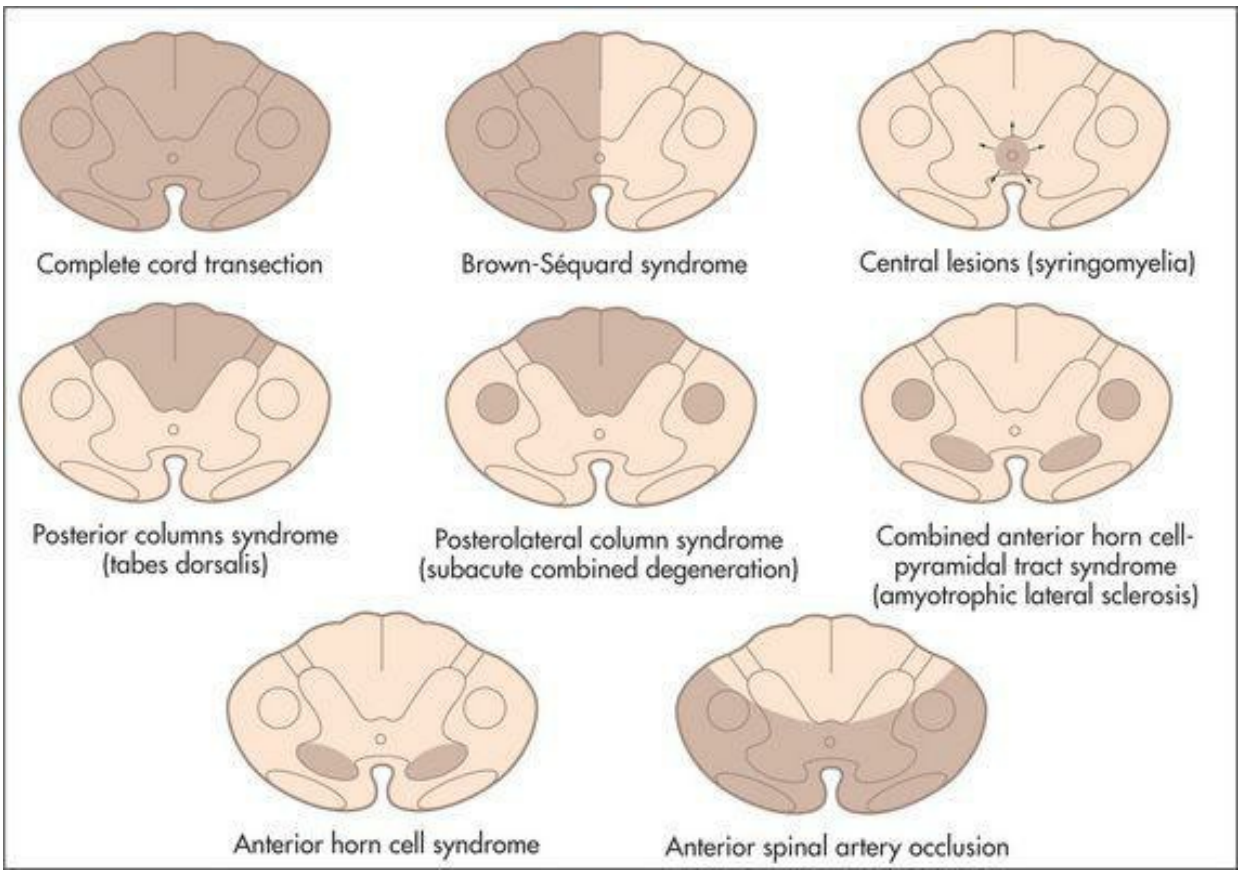


Figure 11.97 Spinal cord syndromes

Adapted from Brazis PW, Localisation in clinical neurology, Philadelphia: Lippincott, Williams & Wilkins, 2001.

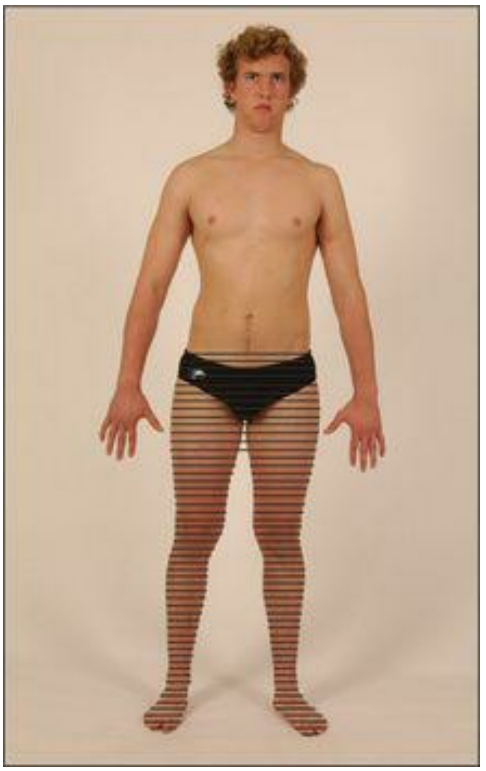


Figure 11.98 Sensory loss with transverse section of the spinal cord

Figure 11.99 Pattern of sensory loss with intrinsic spinal cord disease
For example, central tumour or less commonly with extrinsic compression of the spinal cord —sacrum is spared.