forehead (Figure 11.23). Look for loss of wrinkling and feel the muscle strength by pushing down against the corrugation on each side. This movement is relatively preserved on the side of an upper motor neurone lesion (a lesion that occurs above the level of the brainstem nucleus) because of bilateral cortical representation of these muscles. The remaining muscles of facial expression are usually affected on the side of an upper motor neurone lesion, although occasionally the orbicularis oculi muscles are preserved. Ask the patient to puff out the cheeks (Figure 11.24). Look for asymmetry.

Figure 11.23 Cranial nerve VII: 'Look up to wrinkle your forehead'

Figure 11.24 Cranial nerve VII: 'Puff out your cheeks'

In a lower motor neurone lesion (at the level of the nucleus or nerve

root), all muscles of facial expression are affected on the side of the lesion.

Next ask the patient to shut the eyes tightly (Figure 11.25). Compare how deeply the eyelashes are buried on the two sides and then try to force open each eye. Check whether Bell's phenomenon[t] is evident. Bell's phenomenon is present in everyone, although not usually visible unless a person has a VII nerve palsy. In this case, when the patient attempts to shut the eye on the side of a lower motor neurone VII nerve palsy, there is upward movement of the eyeball and incomplete closure of the eyelid. Next ask the patient to grin (Figure 11.26) and compare the nasolabial grooves, which are smooth on the weak side.

Figure 11.25 Cranial nerve VII: 'Shut your eyes tight and stop me opening them'

Figure 11.26 Cranial nerve VII: 'Show me your teeth'
(Before asking this question, make sure the patient's teeth are not in a container beside the bed.)

If a lower motor neurone lesion is detected, check quickly for the ear and palatal vesicles of herpes zoster of the geniculate ganglion—the Ramsay Hunt[u] syndrome.

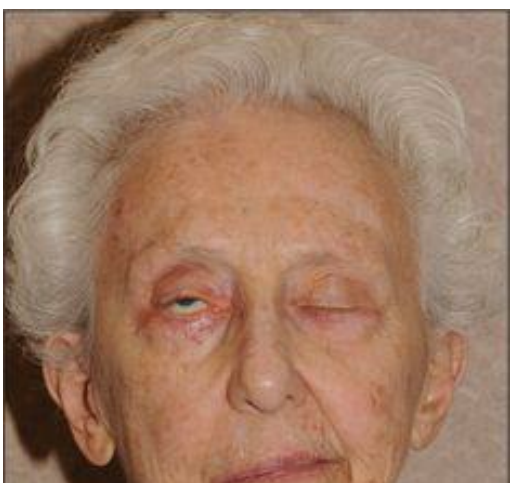
A facial paralysis due to a cortical lesion may spare facial movements due to emotion such as crying or smiling, and indeed these movements may be exaggerated. The opposite abnormality (preservation of voluntary but loss of emotional movements) can also occur as a result of lesions in a number of areas, including the frontal lobes.

Examining for taste on the anterior two-thirds of the tongue is not usually required. If necessary, it can be tested by asking the patient to protrude the tongue to one side: sugar, vinegar, salt and quinine (sweet, sour, saline and bitter) are placed one at a time on each side of the tongue. The patient indicates the taste by pointing to a card with the various tastes listed on it. The mouth is rinsed with water between each sample.

Causes of a seventh (facial) nerve palsy

Vascular lesions or tumours are the common causes of **upper motor neurone lesions (supranuclear)**. Note that lesions of the frontal lobes may cause weakness of the emotional movements of the face alone; voluntary movements are preserved.

In **lower motor neurone lesions**, *pontine* causes (often associated with V and VI lesions) include vascular lesions, tumours, syringobulbia or multiple sclerosis. *Posterior fossa* lesions include an acoustic neuroma, a meningioma or chronic meningitis. At the level of the *petrous temporal bone*, Bell's palsy (an idiopathic acute paralysis of the nerve; Figure 11.27), a fracture, the Ramsay Hunt syndrome or otitis media may occur, while the parotid gland may be affected by a tumour or sarcoidosis. Remember, Bell's palsy is the most common cause (up to 80%) of a facial nerve palsy.[v]



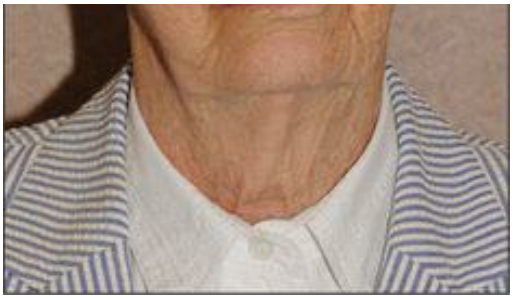


Figure 11.27 Bell's palsy

From Mayo Clinic Images, with permission. ©Mayo Clinic Scientific Press and CRC Press.

Regrowth of the nerve fibres that occurs as the patient recovers from Bell's palsy can lead to aberrant connections. The most striking is the regrowth of fibres meant for the salivary gland to the lacrimal gland in up to 5% of patients. This leads to tear formation when a patient eats—crocodile tears.

Bilateral facial weakness may be due to the Guillain-Barré syndrome, sarcoidosis, bilateral parotid disease, Lyme disease or rarely mononeuritis multiplex. Myopathy and myasthenia gravis can also cause bilateral facial weakness, but in these cases it is not due to facial nerve involvement.

Unilateral **loss of taste**, without other abnormalities, can occur with middle-ear lesions involving the chorda tympani or lingual nerve, but these are very rare.

Irritative changes

Tonic and clonic movements of the facial muscles can occur in seizures. Various abnormal movements of the facial muscles can occur as a result of basal ganglia or extrapyramidal abnormalities (page 396). These include athetoid and dystonic (page 399) movements. Irritative lesions in the brainstem can cause increased secretion of saliva (*sialorrhoea*). This can also occur in Parkinson's disease or accompany attacks of nausea.

The eighth (acoustic) nerve

Examination anatomy

The eighth (acoustic) nerve has two components: the cochlear, with afferent fibres subserving hearing; and the vestibular, containing afferent fibres subserving balance. Fibres for hearing originate in the organ of Corti[W] and run

subserving balance. Fibres for hearing originate in the organ of Corti and run to the cochlear nuclei in the pons. From here there is bilateral transmission to the medial geniculate bodies and thence to the superior gyrus of the temporal lobes. Fibres for balance begin in the utricle and semicircular canals, and join auditory fibres in the facial canal. They then enter the brainstem at the cerebellopontine angle. After entering the pons, vestibular fibres run widely throughout the brainstem and cerebellum.

History

Loss of hearing may have been noticed by the patient or complained of by his or her relatives or friends. Unilateral hearing loss is much more likely to be due to a nerve lesion and must be identified. One should also find out if this has been of gradual or sudden onset, whether there is a family history of deafness, and whether there has been an occupational or recreational exposure to loud noise (e.g. boilermaker, retired rock musician) without hearing protection. There may be a history of trauma or recurrent ear infections.

Examination of the ear and hearing

Look to see if the patient is wearing a hearing aid; remove it. Examine the pinna and look for scars behind the ears. Pull on the pinna gently (it is tender if the patient has external ear disease or temporomandibular joint disease). Feel for nodes (pre- and post-auricular) that may indicate disease of the external auditory meatus.

Inspect the patient's external auditory meatus. The adult canal angulates, so in order to see the eardrum it is necessary to pull up and backwards on the auricle before inserting the otoscope. The normal eardrum (tympanic membrane) is pearly grey and concave. Look for wax or other obstructions, and inspect the eardrum for inflammation or perforation (see Chapter 13).

Next test hearing. A simple test involves covering the opposite auditory meatus with a finger, moving it about as a distraction while whispering a number in the other ear. This should be standardised by the use of set numbers for different tones. For example, the number 68 is used to test high tone and 100 to test low tone. Whispering should be performed towards the end of expiration in an attempt to standardise the volume and at about 60 cm from the ear. The examiner's larynx should not vibrate if the whispering is soft enough. If partial deafness is suspected, perform Rinné's and Weber's tests:

• **Rinné's test**[x]—a 256 Hz vibrating tuning fork is placed on the mastoid process, behind the ear, and when the sound is no longer heard it is placed in line with the external meatus (Figure 11.28). Normally the note is audible at the external meatus. If a patient has nerve deafness the note is audible at the external meatus, as air and bone conduction are reduced equally, so that air conduction is better (as is normal). This is termed Rinné-positive. If there is a conduction (middle ear) deafness, no note is audible at the external meatus. This is termed Rinné-negative.

• **Weber's test**[y]—a vibrating 256 Hz tuning fork is positioned on the centre of the forehead (Figure 11.29). Normally the sound is heard in the centre of the forehead. Nerve deafness causes the sound to be heard better in the normal ear. A patient with a conduction deafness finds the sound louder in the abnormal ear.

Figure 11.28 Cranial nerve VIII, Rinné's test: 'Where does it sound louder?'

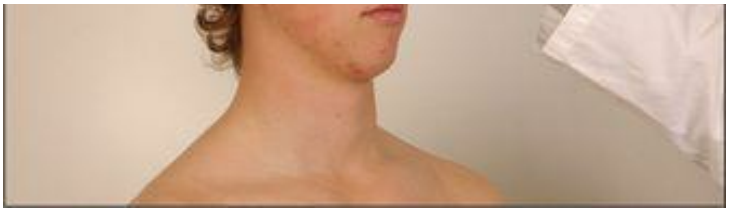


Figure 11.29 Cranial nerve VIII, Weber's test: 'Is the buzzing louder on one side?'

Causes of deafness

Unilateral nerve deafness may be due to (i) tumours, such as an acoustic neuroma; (ii) trauma, such as fracture of the petrous temporal bone; or (iii) vascular disease of the internal auditory artery (rare).

Bilateral nerve deafness may be due to (i) environmental exposure to noise; (ii) degeneration, such as presbyacusis; (iii) toxicity, such as aspirin, streptomycin or alcohol; (iv) infection, such as congenital rubella syndrome, congenital syphilis; or (vi) Ménière's disease.

Brainstem disease is a rare cause of bilateral deafness.

Conduction deafness may be due to (i) wax; (ii) otitis media; (iii) otosclerosis; or (iv) Paget's disease of bone.

Examination of vestibular function

If a patient complains of vertigo, the *Hallpike manoeuvre* should be performed. The patient sits up; having warned him or her what is about to occur, the examiner grasps the patient's head between the hands and gets him or her to lie back quickly so that the head lies 30 degrees below the horizontal. At the same time, the head is rotated 30 degrees towards the examiner. Ask the patient to keep the eyes open. If the test is positive, after a short latent period vertigo and nystagmus (rotatory) towards the affected (lowermost) ear occur for several seconds and then abate and are not reproducible for 10 to 15 minutes. This result is seen in the condition called *benign paroxysmal positioning vertigo* (BPPV). It occurs with repositioning of the head and then abates so that the old name, *benign positional vertigo*, is not really appropriate. It is due to a disorder in the utricle and occurs, for example, following infection, trauma or vascular disease. It is caused by the presence of concretions in the semicircular canals. Inertia of these concretions following movement of the head causes the illusion of movement and nystagmus. If there is no latent period, no fatiguability or the nystagmus persists or is variable, this suggests that there is a lesion of the brainstem (e.g. multiple sclerosis) or cerebellum (e.g. metastatic carcinoma).

multiple sclerosis) or cerebellum (e.g. metastatic carcinoma).

Causes of vestibular abnormalities

Labyrinthine causes include acute labyrinthitis, motion sickness, streptomycin toxicity or, rarely, Ménière's disease.

Vestibular causes include vestibular neuronitis as well as many of the causes of nerve deafness.

In the brainstem, vascular lesions, tumours of the cerebellum or fourth ventricle, demyelination or vasospastic conditions such as migraine may involve the central connections of the vestibular system.

Vertigo may be associated with temporal lobe dysfunction (e.g. ischaemia or complex partial seizures).

The ninth (glossopharyngeal) and tenth (vagus) nerves

Examination anatomy

These nerves have motor, sensory and autonomic functions. Nerve fibres from nuclei in the medulla (Figure 11.30) form multiple nerve rootlets as they exit the medulla. These join to form the ninth and tenth nerves, and also contribute to the eleventh nerve. The nerves emerge from the skull through the jugular foramen (Figure 11.31). The ninth nerve receives sensory fibres from the nasopharynx, pharynx, middle and inner ear and from the posterior third of the tongue (including taste fibres). It also carries secretory fibres to the parotid gland. The tenth nerve receives sensory fibres from the pharynx and larynx, and innervates muscles of the pharynx, larynx and palate.

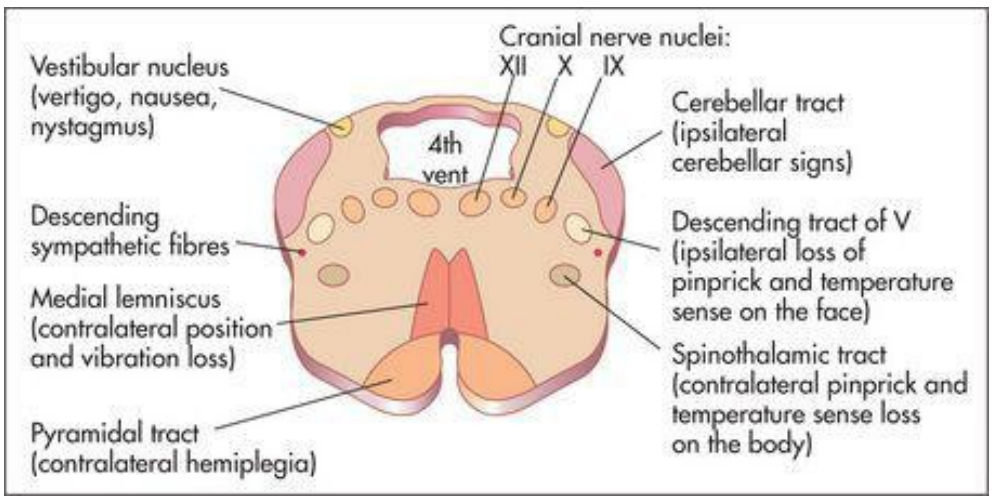


Figure 11.30 Anatomy of the medulla
Shows correlation between lesions and clinical features.

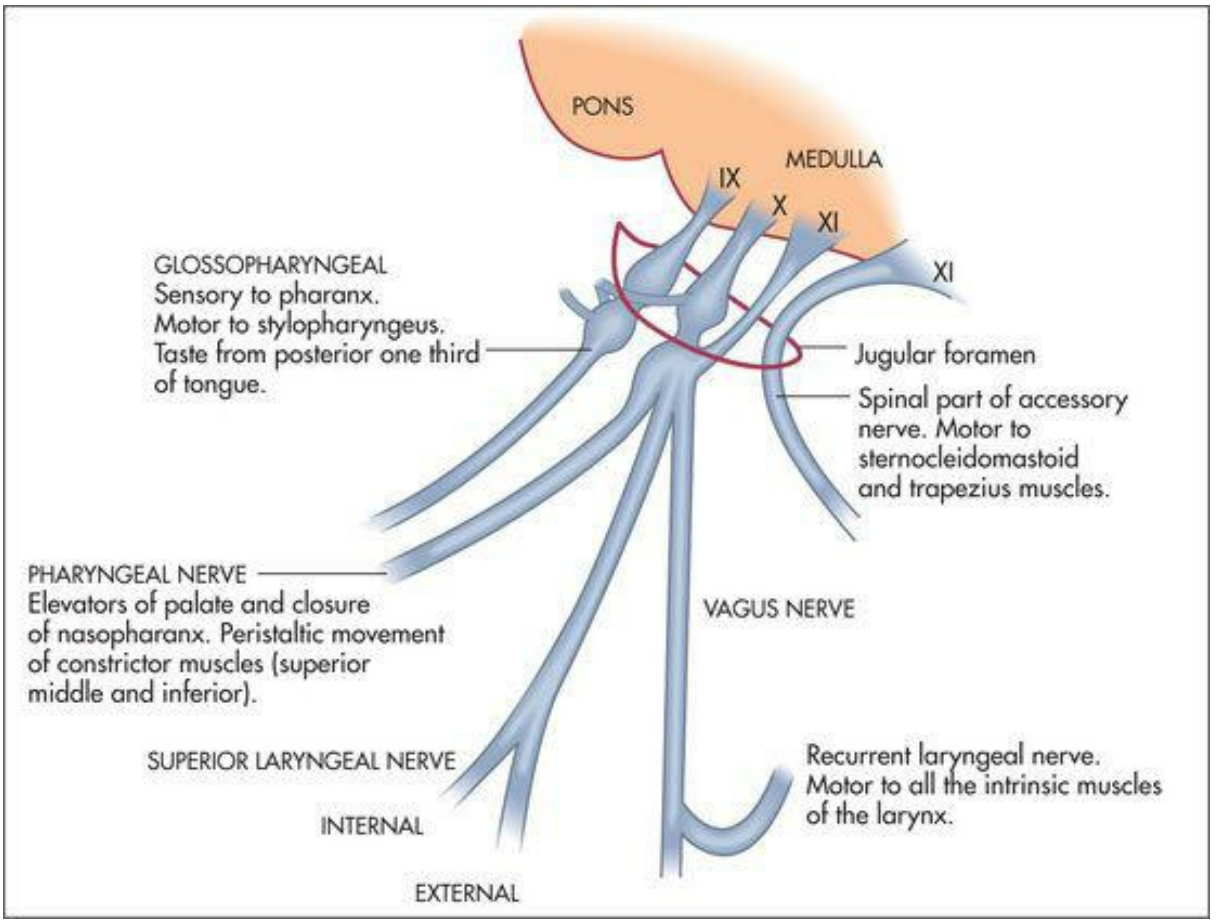


Figure 11.31 The lower cranial nerves—glossopharyngeal (IX), vagus (X) and accessory (XI)

Adapted from Walton JN, Brain's diseases of the nervous system, 10th edn. Oxford: Oxford University Press, 1993.

History

A lesion of the glossopharyngeal nerve may cause the patient no definite symptoms, but difficulty in swallowing dry foods may have been noticed. *Glossopharyngeal neuralgia* is a tic douloureux of the ninth nerve. The patient experiences sudden shooting pains which radiate from one side of the throat to the ear. There may be trigger areas in the throat and attacks can be brought on by chewing or swallowing.

Unilateral vagus nerve paralysis may cause difficulty in initiating the swallowing of solids and liquids and hoarseness.

Examination

Ask the patient to open the mouth and inspect the palate with a torch. Note any displacement of the uvula. Then ask the patient to say 'Ah!' (Figure 11.32). Normally the posterior edge of the soft palate—the *velum*[aa]—rises symmetrically. If the uvula is drawn to one side this indicates a unilateral tenth nerve palsy. Note that the uvula is drawn towards the normal side.

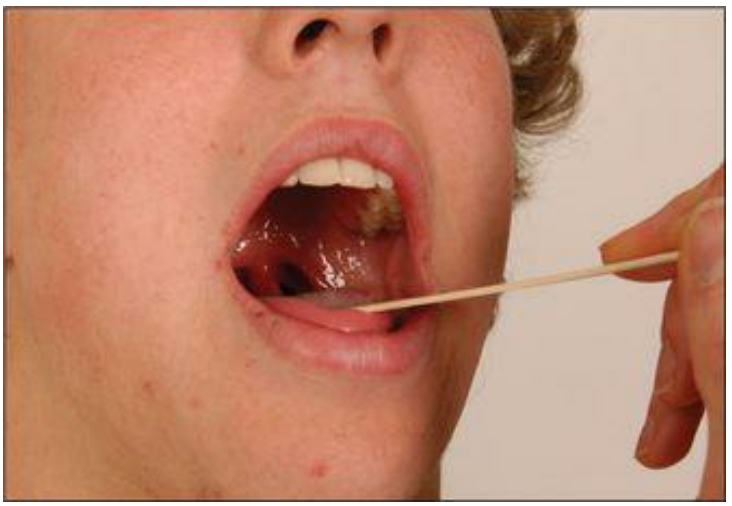


Figure 11.32 Cranial nerve X: 'Say "Ah" '—look for asymmetrical movement of the uvula

Testing for the *gag reflex* (ninth is the sensory component and tenth the motor component) is traditional but not necessary.[11] A better alternative is to touch the back of the pharynx on each side with a spatula (rather than the soft palate). The patient is asked if the touch of the spatula (ninth) is felt each time. Normally, there is reflex contraction of the soft palate. If contraction is absent and sensation is intact this suggests a tenth nerve palsy. The most common cause of a reduced gag reflex is old age. Of more concern to the examiner is the patient with an exaggerated but still normal reflex. This can lead to vomiting onto the examining clinician.

Ask the patient to speak in order to assess hoarseness (which may occur with a unilateral recurrent laryngeal nerve lesion), and then to cough. Listen for the characteristic bovine cough that occurs with recurrent laryngeal nerve lesions. It is not necessary routinely to test taste on the posterior third of the tongue (ninth nerve).

Test the patient's ability to swallow a small amount of water and watch for regurgitation into the nose, or coughing.

Causes of a ninth (glossopharyngeal) and tenth (vagus) nerve palsy

Central causes are vascular lesions (e.g. lateral medullary infarction, due to vertebral or posterior inferior cerebellar artery disease), tumours, syringobulbia and motor neurone disease. **Peripheral (posterior fossa) lesions** comprise aneurysms at the base of the skull, tumours, chronic meningitis or the Guillain-Barré syndrome.

The eleventh (accessory) nerve

Examination anatomy

The central portion of this nerve arises in the medulla close to the nuclei of the ninth, tenth and twelfth nerves and its spinal portion arises from the upper five cervical segments. It leaves the skull with the ninth and tenth nerves through the jugular foramen (Figure 11.31). Its central division provides motor fibres to the vagus and the spinal division innervates the trapezius and sternocleidomastoid muscles. The motor fibres that supply the sternocleidomastoid muscle are thought to cross twice so that cortical control of the muscle is ipsilateral. This makes sense when one remembers that the muscle turns the head to the opposite side. This means that the hemisphere which receives information from and controls one side of the body also turns the head to face that side.

Examination

Ask the patient to shrug the shoulders (Figure 11.33). Feel the bulk of the trapezius muscles and attempt to push the shoulders down. Then instruct the patient to turn the head to the side against resistance (the examiner's hand) (Figure 11.34). Remember that the right sternocleidomastoid turns the head to the left. Feel the muscle bulk of the sternocleidomastoids.

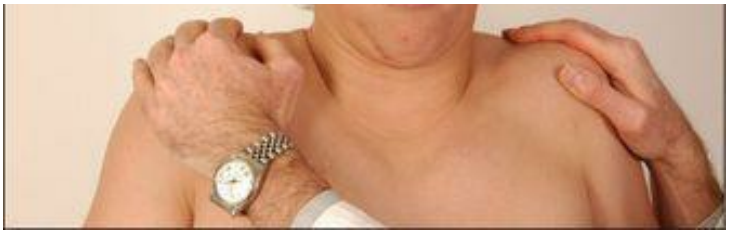


Figure 11.33 Cranial nerve XI: 'Shrug your shoulders—push up hard'

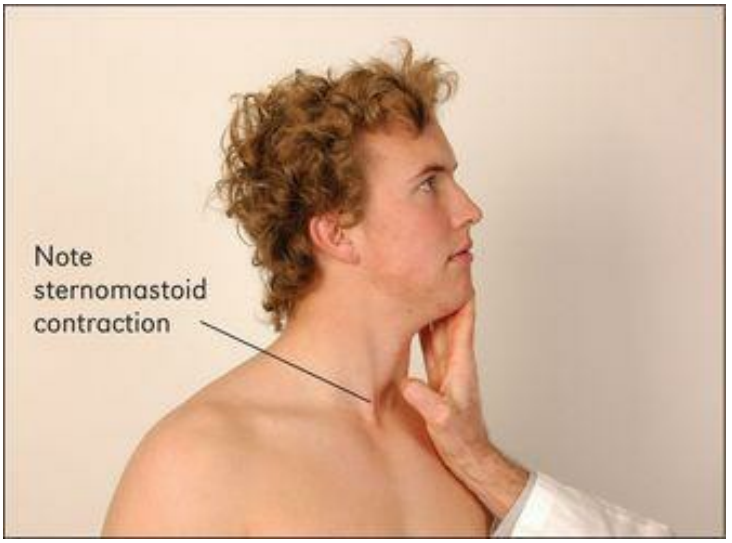


Figure 11.34 Cranial nerve XI: 'Turn your head against my hand'

Weakness of these muscles is less common than *torticollis*, which is due to overactivity of multiple neck muscles. It is a complex movement disorder. The head appears turned to one side either permanently or in spasms. Ask the patient to turn the head to face forwards. This is usually possible at least briefly, but look to see whether he or she needs to use the hands to push the head straight.

Causes of eleventh nerve palsy

Unilateral causes are trauma involving the neck or the base of the skull, poliomyelitis, basilar invagination (platybasia), syringomyelia and tumours near the jugular foramen. **Bilateral** causes comprise motor neurone disease, poliomyelitis and the Guillain-Barré syndrome. *Note:* Bilateral sternocleidomastoid and trapezius weakness also occurs in muscular dystrophy (especially dystrophia myotonica).

The twelfth (hypoglossal) nerve

Examination anatomy

This nerve also arises from the medulla. It leaves the skull via the hypoglossal foramen. It is the motor nerve for the tongue.

History

The patient with bilateral hypoglossal nerve paresis may have noticed difficulty in swallowing and a sensation of choking if the tongue slips back into the throat. There are no sensory changes caused by hypoglossal nerve abnormalities, and unilateral disease rarely causes symptoms.

Examination

Inspect the tongue at rest on the floor of the mouth. The normal tongue may move a little, especially when protruded, but is not wasted. Look for wasting and fasciculations (fine, irregular, non-rhythmical muscle fibre contractions). These signs indicate a lower motor neurone lesion. Fasciculations may be unilateral or bilateral (Figure 11.35).

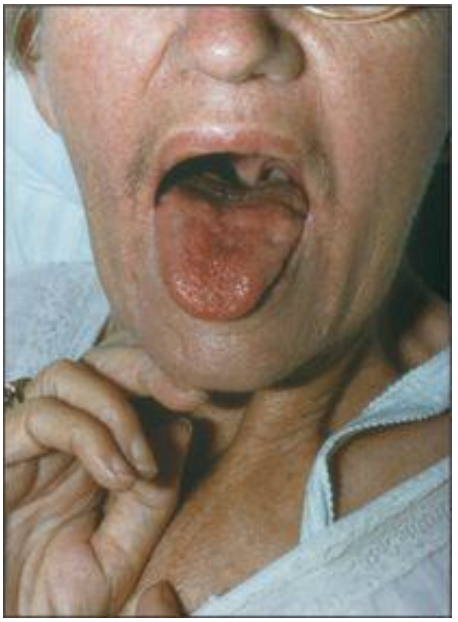


Figure 11.35 Fasciculations of the tongue in motor neurone disease

Ask the patient to poke out the tongue (Figure 11.36), which may

deviate towards the weaker (affected) side if there is a unilateral lower motor neurone lesion (Figure 11.37). The tongue, like the face and palate, has a bilateral upper motor neurone innervation in most people, so a unilateral upper motor neurone lesion often causes no deviation.

Figure 11.36 Cranial nerve XII: 'Stick out your tongue'

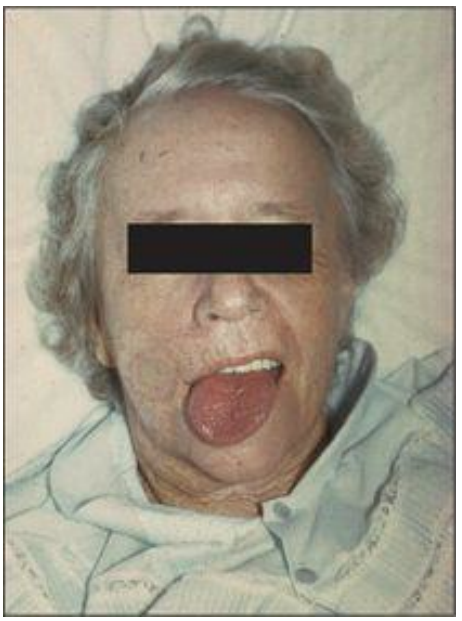


Figure 11.37 Right hypoglossal (XII) nerve palsy—lower motor neurone lesion

A clinically obvious upper motor neurone lesion of the twelfth nerve is usually bilateral and results in a small immobile tongue. The combination of bilateral upper motor neurone lesions of the ninth, tenth and twelfth nerves is called pseudobulbar palsy.

A lower motor neurone lesion of the twelfth nerve causes fasciculation, wasting and weakness. If the lesion is bilateral it causes dysarthria.

Movement disorders may affect the tongue. In Parkinson's disease there may be a coarse tremor of the tongue, made worse by speaking or protruding the tongue. Athetoid, choreiform and tardive dyskinesia can all involve the tongue (page 399).

Causes of twelfth nerve palsy

Bilateral upper motor neurone lesions may be due to vascular lesions, motor neurone disease or tumours, such as metastases to the base of the skull.

Unilateral lower motor neurone lesions with a *central* cause include vascular lesions, such as thrombosis of the vertebral artery; motor neurone disease; and syringobulbia. *Peripheral* causes include: in the posterior fossa, aneurysms or tumours, chronic meningitis and trauma; in the upper neck, tumours or lymphadenopathy; and the Arnold-Chiari malformation.[bb] The Arnold-Chiari malformation is a congenital malformation of the base of the skull with herniation of a tongue of cerebellum and medulla into the spinal canal, causing lower cranial nerve palsies, cerebellar limb signs (due to tonsillar compression) and upper motor neurone signs in the legs.

Causes of **bilateral lower motor neurone lesions** include motor neurone disease, the Guillain-Barré syndrome, poliomyelitis and the Arnold-Chiari malformation.

Multiple cranial nerve lesions

The anatomical courses of the cranial nerves means they can be affected in groups by single lesions that damage them when they run close to each other. Certain disease processes may also interfere with a number of the cranial nerves. There are a number of syndromes that result from abnormalities of groups of cranial nerves:

1. Unilateral III, IV, V and VI involvement suggests a lesion in the cavernous sinus.

2. Unilateral V, VII and VIII involvement suggests a cerebellopontine angle lesion (usually a tumour).

3. Unilateral IX, X and XI involvement suggests a jugular foramen lesion.

4. Combined bilateral X, XI and XII suggests bulbar palsy if lower motor neurone changes are present and pseudobulbar palsy if there are upper motor neurone signs. The clinical features of pseudobulbar and bulbar palsies are

shown in Table 11.7, and the causes of multiple cranial nerve palsies are listed in Table 11.8.

5. Weakness of the eye and facial muscles that worsens with repeated contraction suggests myasthenia.

TABLE 11.7 Clinical features of pseudobulbar and bulbar palsies

Feature	Pseudobulbar (bilateral UMN lesions of IX, X and XII)	Bulbar (bilateral LMN lesions of IX, X and XII)
Gag reflex	Increased or normal	Absent
Tongue	Spastic	Wasted, fasciculations

Tongue	Spastic	Wasted, fasciculations
Jaw jerk	Increased	Absent or normal
Speech	Spastic dysarthria	Nasal
Other	Bilateral limb UMN (long tract) signs	Signs of the underlying cause—e.g. limb fasciculations
	Labile emotions	Normal emotions
Causes	Bilateral cerebrovascular disease (e.g. both internal capsules)	Motor neurone disease
		Guillain-Barré syndrome
	Multiple sclerosis	Poliomyelitis
	Motor neurone disease	Brainstem infarction

UMN = upper motor neurone; LMN = lower motor neurone.

TABLE 11.8 Causes of multiple cranial nerve palsies

Nasopharyngeal carcinoma
Chronic meningitis—e.g. carcinoma, haematological malignancy, tuberculosis, sarcoidosis
Guillain-Barré syndrome (spares sensory nerves)
Brainstem lesions. These are usually due to vascular disease causing crossed sensory or motor paralysis (i.e. cranial nerve signs on one side and contralateral long tract signs). Patients with a brainstem tumour (e.g. in the cerebellopontine angle) may also have similar signs
Arnold-Chiari malformation

Trauma
Paget's disease
Mononeuritis multiplex (rarely), e.g. diabetes mellitus

The head and neck

Inspect and palpate the skull for lumps, such as a meningioma or a sarcoma. Auscultate the skull by placing the diaphragm of the stethoscope on the frontal bone, and then on the lateral occipital bones, and then the bell over each eye (with the opposite eye open). Ask the patient to hold his or her breath each time. Bruits heard over the skull may be due to an arterio-venous malformation, advanced Paget's disease or a vascular meningioma, or they may be conducted from the carotids. Then auscultate over the carotid arteries for a carotid bruit.

The limbs and trunk

History

A variety of symptoms suggest that a patient may have a neurological problem involving the limbs and trunk and that these need to be examined. The exact nature of the symptoms will often suggest the correct diagnosis and where the examination should be directed. A thorough examination is still essential, however, if unexpected findings are not to be missed.

The patient may present with symptoms that are purely or predominately sensory or motor (*Questions box 11.5*), or related to disorders of movement such as tremor. Sensory symptoms include pain, numbness and paraesthesiae (tingling or pins and needles). It is important to find out if there is involvement of more than one modality, something the patient may not have noticed. The distribution, time of onset and duration may give clues to the aetiology of the symptoms or at least as to where the sensory examination should be concentrated.

Questions box 11.5

Questions to ask the patient with muscle weakness

1. Have you felt weakness on both sides of the body?—Suggests spinal cord disease, myopathy or myasthenia gravis (transverse myelitis)

2. Is the weakness just on one side of the body or face?—Transient ischaemic attack or stroke

3. Has the weakness affected just an arm or leg or part of a limb?—Peripheral neuropathy or radiculopathy, stroke or multiple sclerosis

4. Have you had trouble getting up from a chair or brushing your hair or lifting your head?—Proximal muscle weakness (myasthenia gravis, diabetic amyotrophy [involves lower limbs], polymyositis)

5. Have you had trouble swallowing or difficulty speaking?—Myasthenia gravis, polymyositis

6. Have you noticed double vision?—Myasthenia gravis, cranial nerve mononeuritis multiplex

7. Are you taking any medications?—Steroid-induced proximal myopathy

8. Have you had problems with your neck or back or with severe arthritis?—Radiculopathy

9. Have you had a cancer diagnosed at any stage?—Paraneoplastic, Eaton Lambert syndrome

10. Is there any problem like this in the family?—Familial myopathy, Charcot-Marie-Tooth disease

11. Have you had HIV infection?—Various neurological lesions and drug reactions

12. Have you ever had multiple sclerosis diagnosed?

13. Are you a diabetic?—Mononeuritis multiplex, amyotrophy

A family history of a similar problem may help provide the diagnosis in conditions such as muscular dystrophy. A previous injury may be responsible, for example, for a peripheral nerve problem but not remembered until asked about specifically.

Examination anatomy

Muscle weakness has four major causes:

1. Pyramidal or upper motor neurone weakness, which is caused by a lesion in the brain proximal to the 'pyramids' in the brainstem. This is where the nerve fibres decussate or cross to the other side before travelling down the spinal cord (Figure 11.38).

2. Lower motor neurone weakness, which is caused by a nerve lesion within the spinal cord or peripheral nerve.

3. Abnormalities of the neuromuscular junction (myasthenia gravis).

4. Muscle disease.

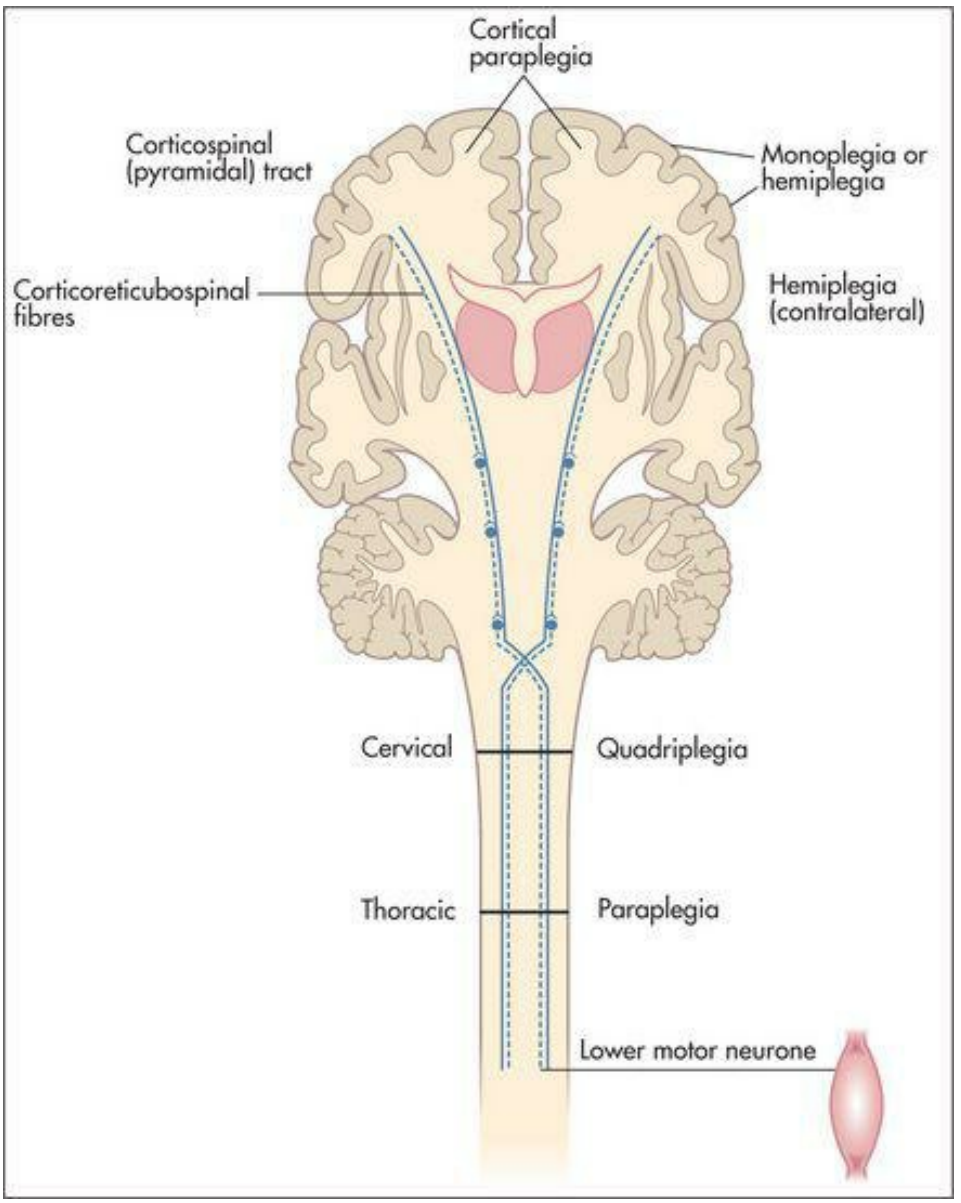


Figure 11.38 Upper and lower motor neurone lesions

Adapted from Lance JG, McLeod JW, A physiological approach to clinical neurology, 3rd edn. London: Butterworths, 1981.

General examination approach

It is most important to have a set order of examination of the limbs for neurological signs so that nothing important is omitted. The following scheme is a standard approach.

1. **Motor system**
   - General inspection
     - Posture
     - Muscle bulk
     - Abnormal movements
   - Fasciculations
   - Tone
   - Power
   - Reflexes
   - Coordination.

2. **Sensory system**
   - Pain and temperature
   - Vibration and proprioception
   - Light touch.

General inspection

1. Stand back and look at the patient for an *abnormal posture*—for example, one due to hemiplegia caused by a stroke. In this case the upper limb is flexed and there is adduction and pronation of the arm, while the lower limb is extended.

2. Look for *muscle wasting,* which indicates a denervated muscle, a primary muscle disease or disuse atrophy. Compare one side with the other for wasting and try to work out which muscle groups are involved (proximal, distal or generalised, symmetrical or asymmetrical).

3. Inspect for *abnormal movements*, such as tremor of the wrist or arm.

4. Inspect the *skin*—for example, for evidence of neurofibromatosis.

cutaneous angiomata in a segmental distribution (associated with syringomyelia) or herpes zoster. Look for scars from old injuries or surgical treatment. Note the presence of a urinary catheter.

The upper limbs

The motor system[cc]

General

Shake hands with the patient and introduce yourself. A patient who cannot relax his or her hand grip has myotonia (an inability to relax the muscles after voluntary contraction). The commonest cause of this is the muscle disease dystrophia myotonica (page 392). Once your hand has been extracted from the patient's, and after pausing briefly for the vitally important general inspection, ask the patient to undress so that the arms and shoulder girdles are completely exposed.

Sit the patient over the edge of the bed if this is possible. Next ask the patient to hold out both hands, palms upward, with the arms extended and the eyes closed (Figure 11.39). Watch the arms for evidence of drifting (movement of one or both arms from the initial neutral position). There are only three causes for drift of the arms:

1. Upper motor neurone (pyramidal) weakness. The drift of the affected limb(s) here is due to muscle weakness and tends to be in a downward direction. The drifting typically starts distally with the fingers and spreads proximally. There may be slow pronation of the wrist and flexion of the fingers and elbow.

2. Cerebellar disease. The drift here is usually upwards. It also includes slow pronation of the wrist and elbow.

3. Loss of proprioception. The drift here (pseudoathetosis) is really a searching movement and usually affects only the fingers. It is due to loss of joint position sense and can be in any direction.

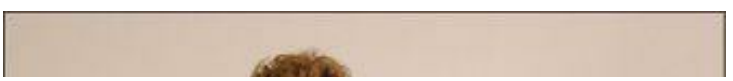


Figure 11.39 Testing for arm drift: 'Shut your eyes and hold your arms out straight. Now turn your palms upwards.'

Ask the patient to relax the arms and rest them on his or her lap. Inspect the large muscle groups for *fasciculations* (Table 11.9). These are irregular contractions of small areas of muscle which have no rhythmical pattern. Fasciculation may be coarse or fine and is present at rest, but not during voluntary movement.[dd] If present with weakness and wasting, fasciculation indicates degeneration of the lower motor neurone. It is usually benign if unassociated with other signs of a motor lesion.

TABLE 11.9 Causes (differential diagnosis) of fasciculations

Motor neurone disease
Motor root compression
Peripheral neuropathy—e.g. diabetic
Primary myopathy
Thyrotoxicosis

Note: Myokymia resembles coarse fasciculation of the same muscle group, and is particularly common in the orbicularis oculi muscles, where it is usually benign. Focal myokymia, however, often represents brainstem disease, e.g. multiple sclerosis or glioma.

Fibrillation is seen only on the electromyogram.

Abnormality is seen only on the electromyogram.

Tone

Tone is tested at both the wrists and elbows. Rotation of the wrists with supination and pronation of the elbow joints (supporting the patient's elbow with one hand and holding the hand with the other) is performed passively, and the patient should be told to relax to allow the examiner to move the joints freely.

If the patient resists these movements the joints should be moved unpredictably and at different rates. When the arm is raised by the examiner and dropped, it will fall suddenly if tone is reduced. With experience it is possible to decide if tone is normal or increased (hypertonic, as in an upper motor neurone or extrapyramidal lesion). Hypotonia is a difficult clinical sign to elicit and probably not helpful in the assessment of a lower motor neurone lesion.

The cogwheel rigidity of Parkinson's disease is an important abnormality of tone in the upper limbs and should be recognised. It is best assessed by having the patient move the other arm up and down as the examiner moves the hand and forearm, testing tone at the wrist and elbow.

Myotonia as described above is also an abnormality of tone that is worse after active movement. In these patients, tone is usually normal at rest but after sudden movements there may be a great increase in tone and the patient is unable to relax the muscle. Tapping over the body of a myotonic muscle causes a dimple of contraction, which only slowly disappears (*percussion myotonia*). This is best tested by tapping the thenar eminence or by asking the patient to make a tight fist and then open the hand quickly. The opening of the fist is very slow when the muscles are myotonic.

Power

Muscle strength is assessed by gauging the examiner's ability to overcome the patient's full voluntary muscle resistance. To decide whether the power is normal, the patient's age, gender and build should be taken into account. Power is graded based on the maximum observed (no matter how briefly), according to the following modified Medical Research Council scheme (although this lacks sensitivity at the higher grades because work against gravity may only make up a small component of a muscle's function, e.g. the finger flexors):

0. Complete paralysis (no movement).

1. Flicker of contraction possible.

2. Movement is possible when gravity is excluded.

3. Movement is possible against gravity but not if any further resistance is added.

4– Slight movement against resistance.

4. Moderate movement against resistance.

4+ Submaximal movement against resistance.

5. Normal power.[ee]

If power is reduced, decide whether this is symmetrical or asymmetrical, whether it involves only particular muscle groups, or whether it is proximal, distal or general. It is also important to consider whether any painful joint or muscle disease is interfering with the assessment (see Chapter 9). Asymmetrical muscle weakness is most often the result of a peripheral nerve, brachial plexus or root lesion, or an upper motor neurone lesion. As each movement is tested the important muscles involved should be observed or palpated.

Shoulder

• *Abduction*—mostly deltoid and supraspinatus—(C5, C6): the patient should abduct the arms with the elbows flexed and resist the examiner's attempt to push them down (Figure 11.40).

• *Adduction*—mostly pectoralis major and latissimus dorsi—(C6, C7, C8): the patient should adduct the arms with the elbows flexed and not allow the examiner to separate them.

Figure 11.40 Testing power—shoulder abduction: 'Stop me pushing your arm down'

Elbow

• *Flexion*—biceps and brachialis—(C5, C6): the patient should bend the elbow and pull so as not to let the examiner straighten it out (Figure 11.41).

• *Extension*—triceps brachii—(C7, C8): the patient should bend the elbow and push so as not to let the examiner bend it (Figure 11.42).

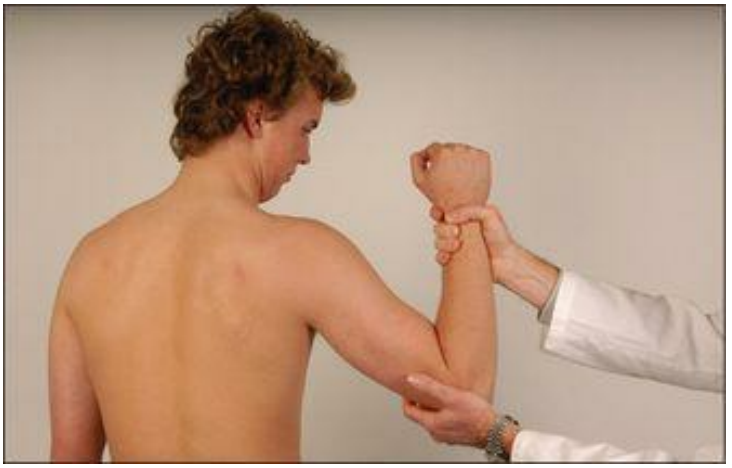


Figure 11.41 Testing power—elbow flexion: 'Stop me straightening your elbow'

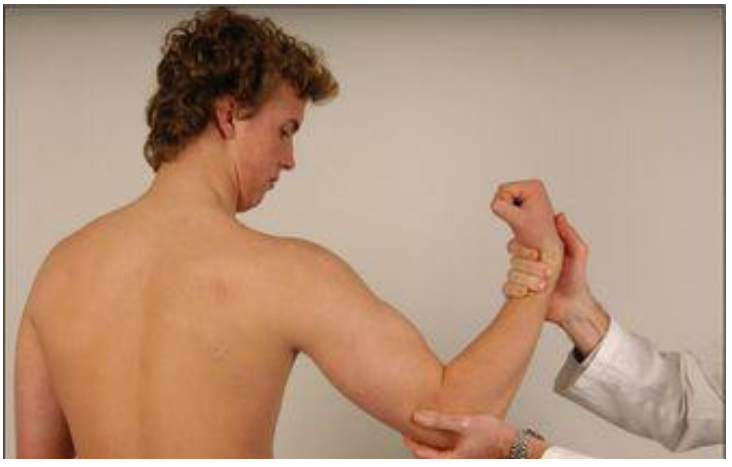


Figure 11.42 Testing power—elbow extension: 'Stop me bending your elbow

Wrist

• *Flexion*—flexor carpi ulnaris and radialis—(C6, C7): the patient should bend the wrist and not allow the examiner to straighten it.

• *Extension*—extensor carpi group—(C7, C8): the patient should extend the wrist and not allow the examiner to bend it (Figure 11.43).

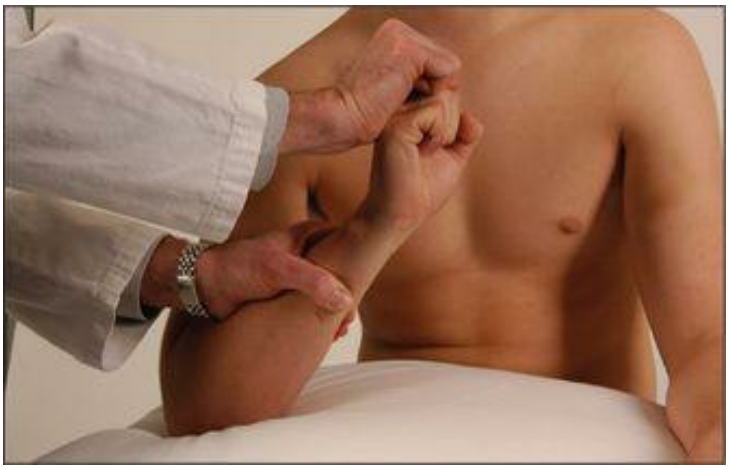


Figure 11.43 Testing power—wrist extension: 'Stop me bending your wrist'

Fingers

• *Extension*—extensor digitorum communis, extensor indicis and extensor digiti minimi—(C7, C8): the patient should straighten the fingers and not allow the examiner to push them down (push with the side of your hand across the patient's metacarpophalangeal joints).

• *Flexion*—flexor digitorum profundus and sublimis—(C7, C8): the patient squeezes two of the examiner's fingers (Figure 11.44).

• *Abduction*—dorsal interossei—(C8, T1): the patient should spread out the fingers and not allow the examiner to push them together (Figure 11.45).

• *Adduction*—volar interossei—(C8, T1): the patient holds the fingers together and tries to prevent the examiner from separating them further.

Figure 11.44 Testing power—finger flexion: 'Squeeze my fingers hard' (don't offer more than two fingers)

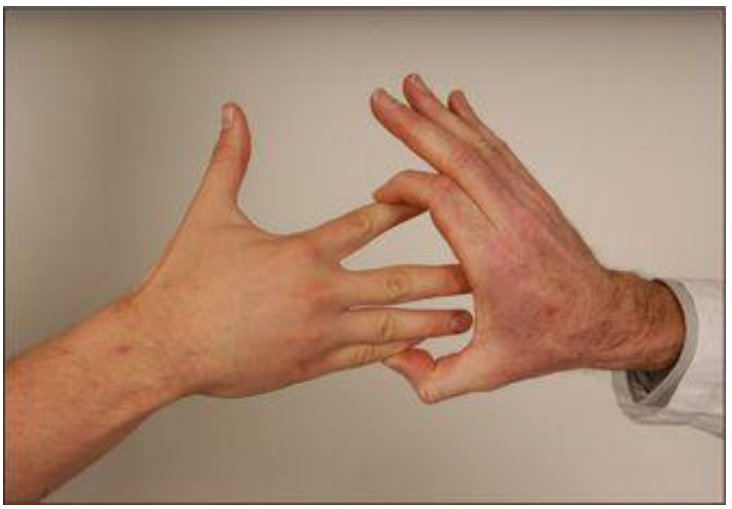


Figure 11.45 Testing power—finger abduction: 'Stop me pushing your fingers together'

Reflexes

The sudden stretching of a muscle usually evokes brisk contraction of that muscle or muscle group. This reflex is usually mediated via a neural pathway synapsing in the spinal cord. It is subject to regulation via pathways from the brain. As the reflex is a response to stretching of a muscle, it is correctly called a muscle stretch reflex rather than a tendon reflex. The tendon merely

called a muscle stretch reflex rather than a tendon reflex. The tendon merely transmits stretch to the muscle.

Tendon hammers are available in a number of designs. Sir William Gowers[ff] used the ulnar side of his hand or part of his stethoscope. In Australia and Britain, the Queen Square hammer[gg] is in common use (Figure 11.46). The Taylor hammer is popular in America; it is shaped like a tomahawk and has a broad rubber edge for most tendons and a more pointed side for the cutaneous reflexes.

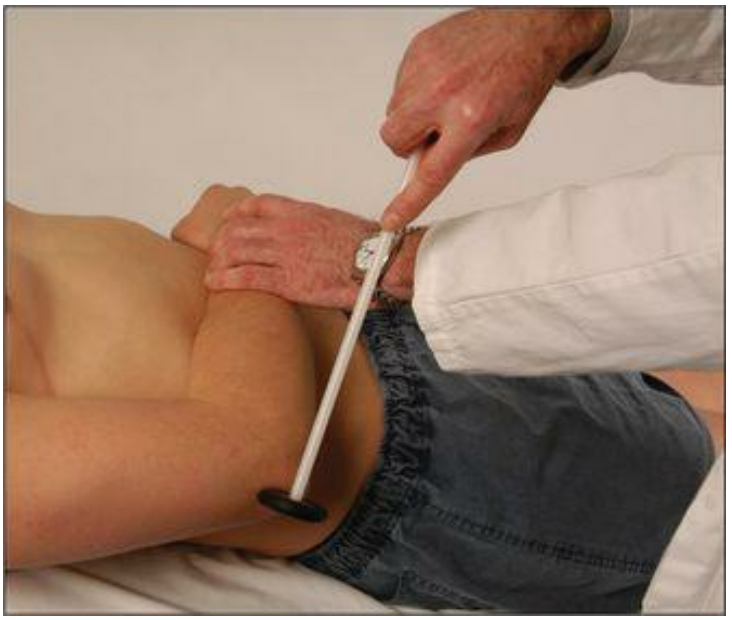


Figure 11.46 A Queen Square patellar hammer

Reflexes are graded from absent to greatly increased (Table 11.10).

TABLE 11.10 Classification of muscle stretch reflexes

0	= absent
+	= present but reduced
	= normal

++	= normal
+++	= increased, possibly normal
++++	= greatly increased, often associated with clonus (page 368)

Make sure the patient is resting comfortably with the elbows flexed and hands lying pronated on the lap and not overlapping one another.

To test the **biceps jerk (C5, C6)**, place one forefinger on the biceps tendon and tap this with the tendon hammer (Figure 11.47). The hammer should be held near its end and the head allowed to fall with gravity onto the positioned forefinger. The examiner soon learns not to hit too hard. Normally, if the reflex arc is intact, there is a brisk contraction of the biceps muscle with flexion of the forearm at the elbow, followed by prompt relaxation. Practice will help the examiner decide whether the response is within the normal range. When a reflex is greatly exaggerated, it can be elicited away from the usual zone.

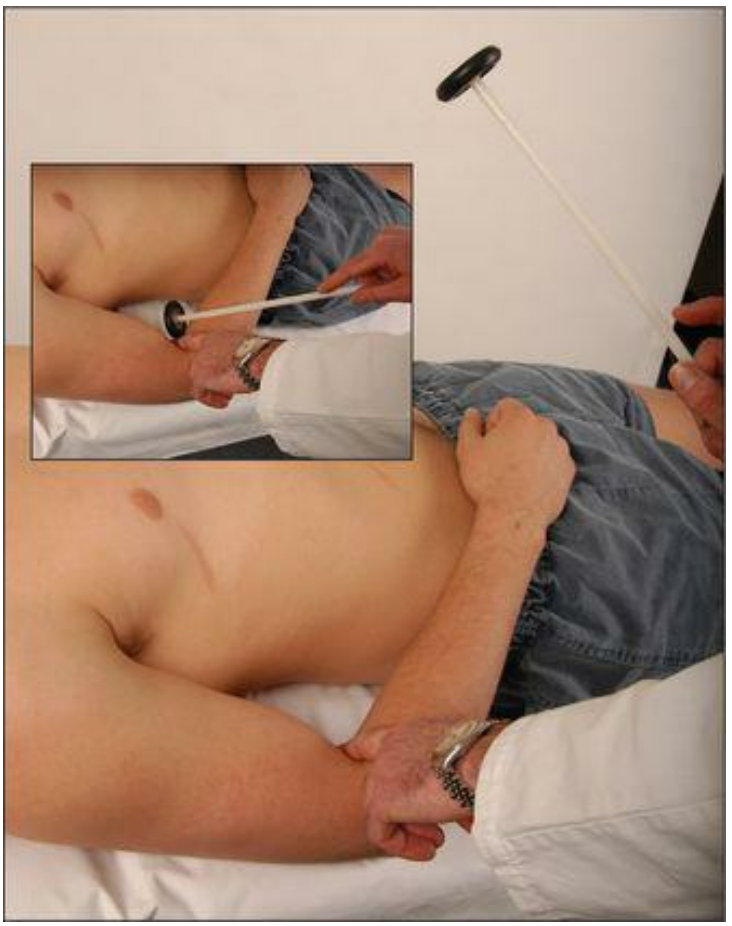


Figure 11.47 The biceps jerk examination

If a reflex appears to be absent, always test following a *reinforcement*

manoeuvre. For example, ask the patient to clench the teeth tightly just before you let the hammer fall. Supraspinal and fusiform mechanisms have been identified to explain reinforcement, but it works partly as a distraction, especially if the reflex is absent because an anxious patient has contracted opposing muscle groups. Merely talking to the patient may provide enough distraction for the reflex to be elicited. Sometimes normal reflexes can be elicited only after reinforcement, but they should still be symmetrical.

An increased jerk occurs with an upper motor neurone lesion (page 354). A decreased or absent reflex occurs with a breach in any part of the reflex motor arc—the muscle itself (e.g. myopathy), the motor nerve (e.g. neuropathy), the anterior spinal cord root (e.g. spondylosis), the anterior horn cell (e.g. poliomyelitis) or the sensory arc (sensory root or sensory nerve).

To test the **triceps jerk (C7, C8)**, support the elbow with one hand and tap over the triceps tendon (Figure 11.48). Normally, triceps contraction results in forearm extension.

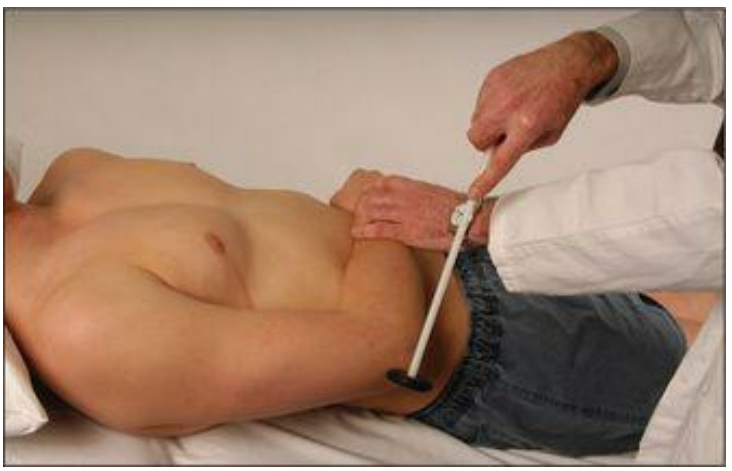


Figure 11.48 The triceps jerk examination

To test the **brachioradialis (supinator) jerk (C5, C6)**, strike the lower end of the radius just above the wrist (Figure 11.49). To avoid hurting the patient by striking the radial nerve directly, place your own first two fingers over this spot and then strike the fingers, as with the biceps jerk. Normally, contraction of the brachioradialis causes flexion of the elbow.

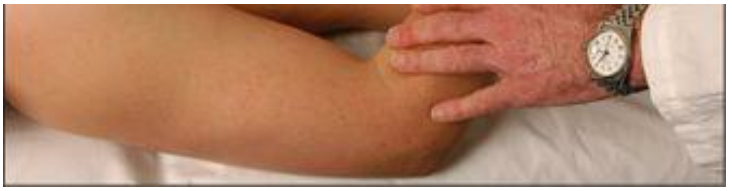


Figure 11.49 The supinator jerk strike zone

If elbow extension and finger flexion is the only response when the patient's wrist is tapped, the response is said to be inverted, known as the *inverted brachioradialis (supinator) jerk*. The triceps contraction causes elbow extension instead of the usual elbow flexion. This is associated with an absent biceps jerk and an exaggerated triceps jerk. It indicates a spinal cord lesion at the C5 or C6 level due, for example, to compression (e.g. disc prolapse), trauma or syringomyelia. It occurs because a lower motor neurone lesion at C5 or C6 is combined with an upper motor neurone lesion affecting the reflexes below this level.

To test **finger jerks (C8)**, the patient rests the hand palm upward, with the fingers slightly flexed. The examiner's hand is placed over the patient's and the hammer struck over the examiner's fingers (Figure 11.50). Normally, slight flexion of all the fingers occurs.

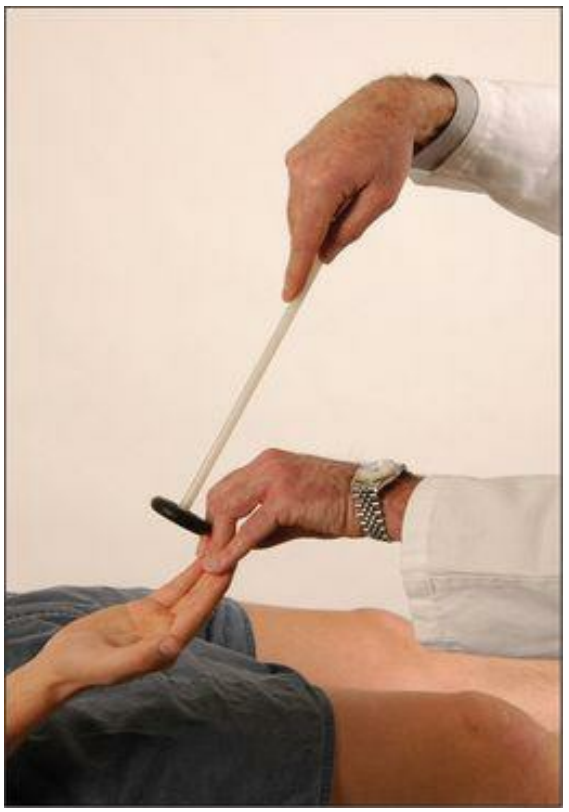


Figure 11.50 The finger jerk examination

Coordination

The cerebellum has multiple connections (afferent and efferent) to sensory pathways, brainstem nuclei, the thalamus and the cerebral cortex. Via these connections the cerebellum plays an integral role in coordinating voluntary movement. A standard series of simple tests is used to test coordination. Always demonstrate these movements for the patient's benefit.

Finger–nose test

Ask the patient to touch his or her nose with the index finger and then turn the finger around and touch the examiner's outstretched forefinger at nearly full extension of the shoulder and elbow (Figure 11.51). The test should be done both briskly and slowly, and repeated a number of times with the patient's eyes open and later closed. Slight resistance to the patient's movements by the examiner pushing on his or her forearm during the test may unmask less-severe abnormalities.

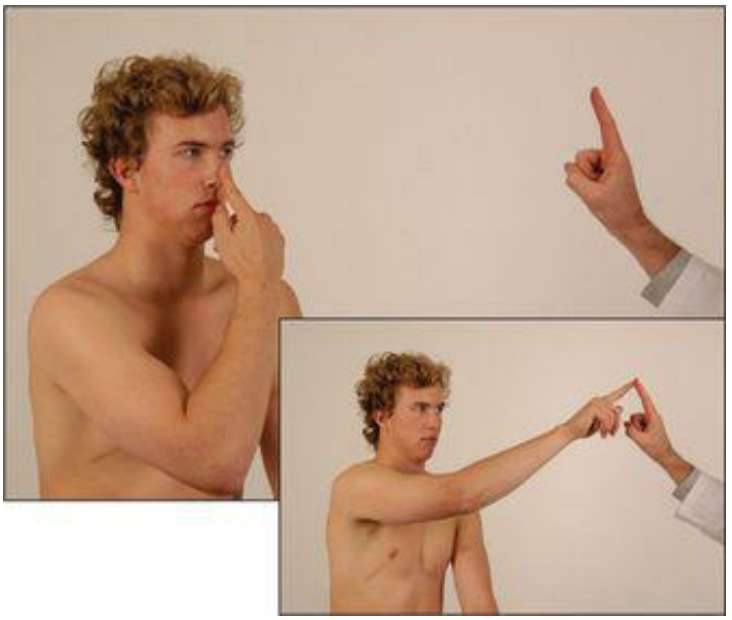


Figure 11.51 Finger–nose test: 'Touch your nose with your forefinger and then reach out and touch my finger'

Look for the following abnormalities: (i) intention tremor, which is tremor increasing as the target is approached (there is no tremor at rest); and (ii) past-pointing, where the patient's finger overshoots the target towards the side of cerebellar abnormality. These abnormalities occur with cerebellar disease.

disease.

Rapidly alternating movements

Ask the patient to pronate and supinate his or her hand on the dorsum of the other hand as rapidly as possible (Figure 11.52). This movement is slow and clumsy in cerebellar disease and is called dysdiadochokinesis.[hh]

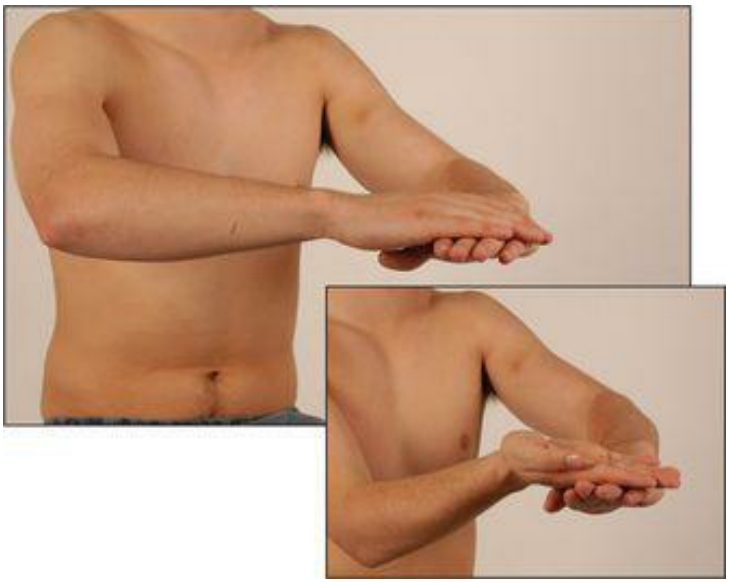


Figure 11.52 Testing for dysdiadochokinesis in the upper limbs: 'Turn your hand over, backwards and forwards on the other one, as quickly and smoothly as you can'

Rapidly alternating movements may also be affected in extrapyramidal disorders (e.g. Parkinson's disease) and in pyramidal disorders (e.g. internal capsule infarction).

Rebound

Ask the patient to lift the arms rapidly from the sides and then stop. Hypotonia due to cerebellar disease causes delay in stopping the arms. This method of demonstrating rebound is preferable to the more often used one where the patient flexes the arm at the elbow against the examiner's resistance. When the examiner suddenly lets go, violent flexion of the arm may occur and, unless prevented, the patient can strike himself or herself in the face. Therefore, only medical students trained in self-defence should use this method.[ii]

Muscle weakness may also cause clumsiness, but motor testing should have revealed any impairment of this sort.

The sensory system

To test sensation, which can be a difficult assessment and frustratingly time-consuming, use the following routine.[12]

Spinothalamic pathway (pain and temperature)

Pain and temperature fibres enter the spinal cord and cross, a few segments higher, to the opposite spinothalamic tract (Figure 11.53). This tract ascends to the brainstem.

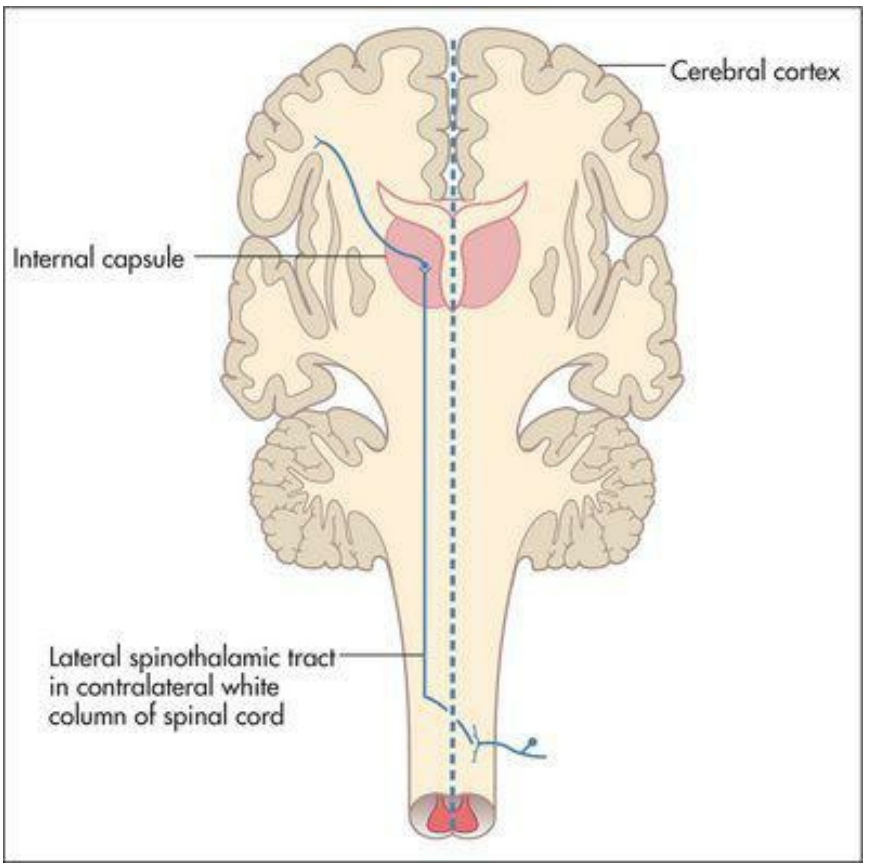


Figure 11.53 Pain and temperature pathways

Adapted from Snell RS, Westmoreland BF, Clinical neuroanatomy for medical students, 4th edn. Boston: Little Brown, 1997.

Pain (pinprick) testing

Using a new pin,[10] demonstrate to the patient that this induces a relatively

sharp sensation by touching lightly a normal area, such as the anterior chest wall. Then ask the patient to say whether the pinprick feels sharp or dull. Begin proximally on the upper arm and test in each dermatome—the area of skin supplied by a vertebral spinal segment (Figure 11.54). Also compare right with left in the same dermatome. Map out the extent of any area of dullness. Always do this by going from the area of dullness to the area of normal sensation.

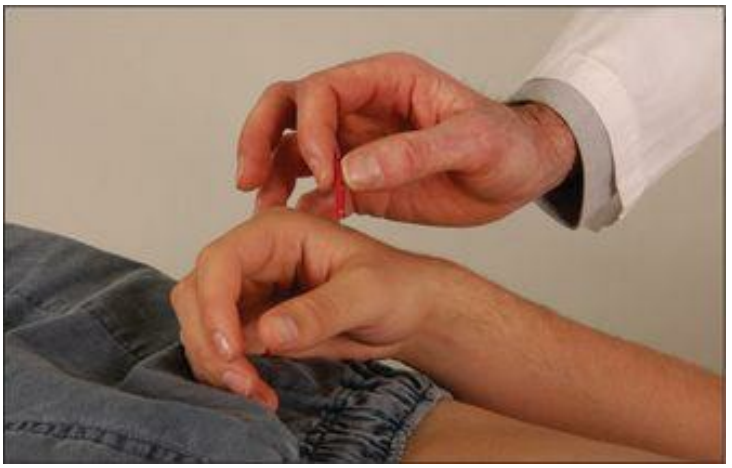


Figure 11.54 Testing for pinprick (pain) sensation with a disposable neurology pin: 'Does this feel sharp or blunt?'

Temperature testing

This can be done in a similar fashion, using test tubes filled with hot (40–45 degrees) and cold (5–10 degrees) water. Cold sensation can also be tested with a metal object, such as a tuning fork. Absence of ability to feel heat is almost always associated with inability to feel cold. Temperature differences of 2–5 degrees can usually be distinguished. These tests are performed only in special circumstances—for example, for suspected syringomyelia.

Posterior columns (vibration and proprioception[ii])

These fibres enter and ascend ipsilaterally in the posterior columns of the spinal cord to the nucleus gracilis and nucleus cuneatus in the medulla, where they decussate (Figure 11.55).

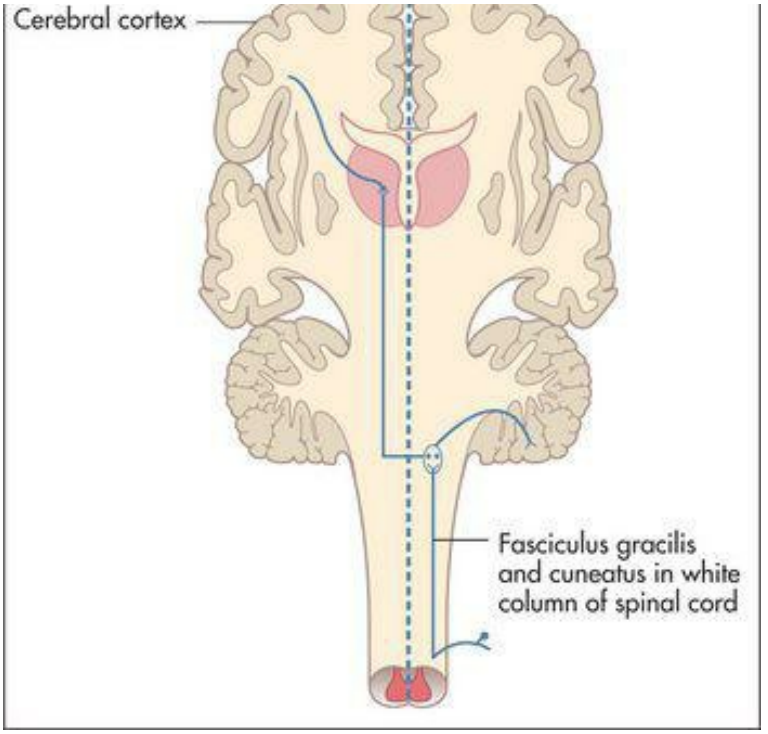


Figure 11.55 Vibration and joint position sense pathways

Adapted from Snell RS, Westmoreland BF, Clinical neuroanatomy for medical students, 4th edn. Boston: Little Brown, 1997.

Vibration testing

Use a 128 Hz tuning fork (not a 256 Hz fork). Ask the patient to close the eyes, and then place the vibrating tuning fork on one of the distal interphalangeal joints. The patient should be able to describe a feeling of vibration. The examiner then deadens the tuning fork with the hand, and the patient should be able to say exactly when this occurs. Compare one side with the other. If vibration sense is reduced or absent, test over the ulnar head at the wrist, then the elbows (over the olecranon) and then the shoulders to determine the level of abnormality. Although the tuning fork is traditionally placed only over bony prominences, vibration sense is just as good over soft tissues.

Proprioception testing

Use the distal interphalangeal joint of the patient's little finger. When the patient has his or her eyes open, grasp the distal phalanx from the sides and move it up and down to demonstrate these positions. Then ask the patient to close the eyes while these manoeuvres are repeated randomly. Normally,

movement through even a few degrees is detectable, and should be reported correctly. If there is an abnormality, proceed to test the wrists and elbows similarly. As a rule, sense of position is lost before sense of movement, and the little finger is affected before the thumb.

Light-touch testing

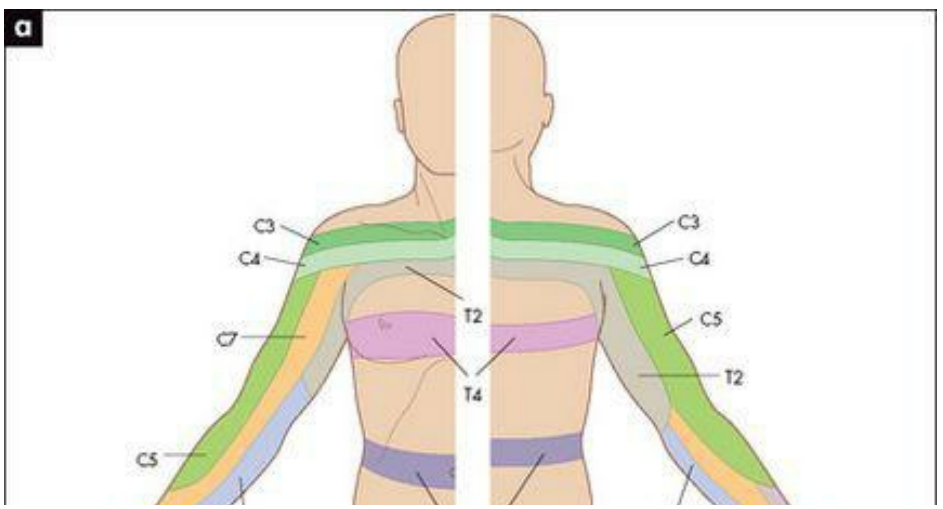
Some fibres travel in the posterior columns (i.e. ipsilaterally) and the rest cross the middle line to travel in the anterior spinothalamic tract (i.e. contralaterally). For this reason light touch is of the least discriminating value. Irritation of light-touch receptors is probably responsible for paraesthesiae—for example following ischaemia of a limb.

Test light touch by touching the skin with a wisp of cottonwool. Ask the patient to shut the eyes and say 'yes' when the touch is felt. Do not stroke the skin because this moves hair fibres. Test each dermatome,[kk] comparing left and right sides.

Interpretation of sensory abnormalities

Try to fit the distribution of any sensory loss into a dermatome (due to a spinal cord or nerve root lesion), a single peripheral nerve territory, a peripheral neuropathy pattern (glove distribution, page 386), or a hemisensory loss (due to spinal cord or upper brainstem or thalamic lesion).

Sensory dermatomes of the upper limb (Figure 11.56) can be recognised by memorising the following rough guides: C5 supplies the shoulder tip and outer part of the upper arm; C6 supplies the lateral aspect of the forearm and thumb; C7 supplies the middle finger; C8 supplies the little finger; T1 supplies the medial aspect of the upper arm and elbow.



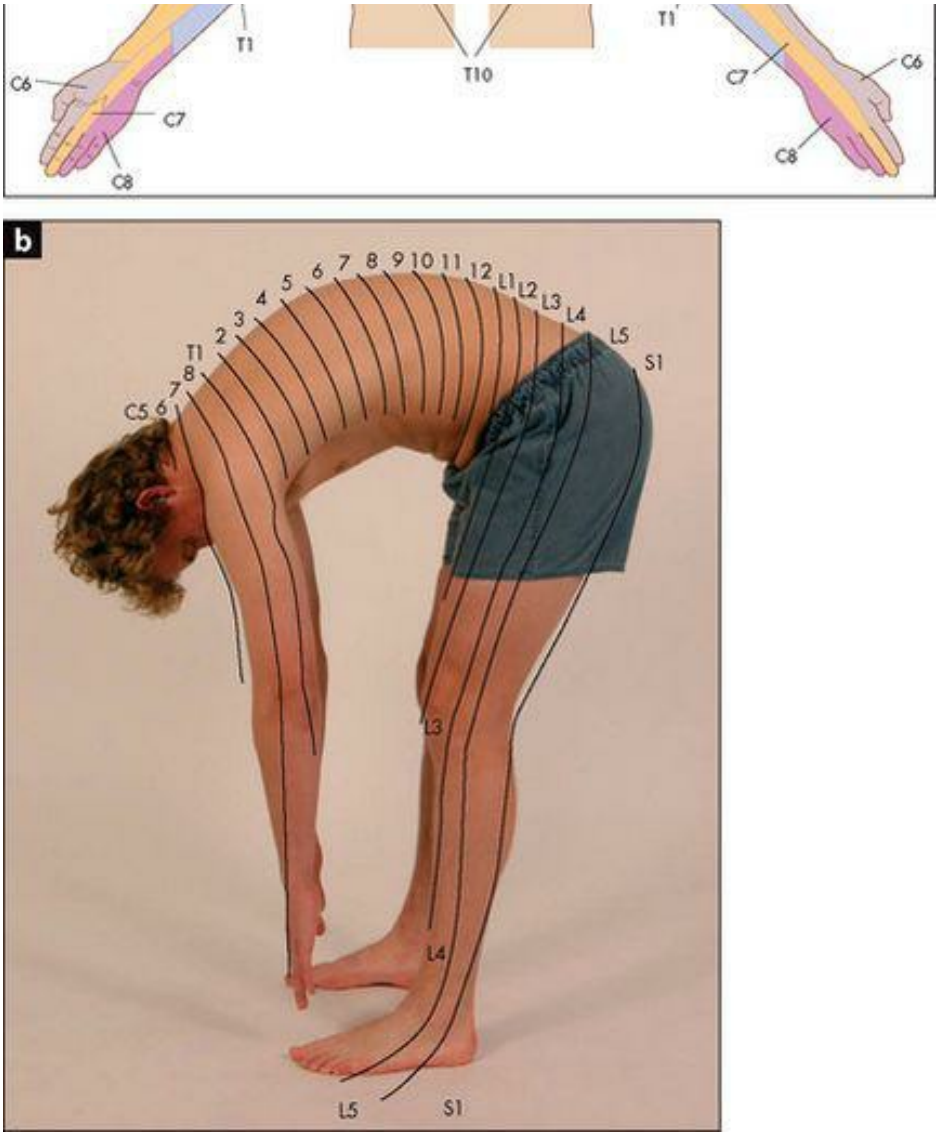


Figure 11.56 Dermatomes of the upper limb and trunk
(a) The dermatomes explained. (b) The distribution of the dermatomes makes more sense if we are thought of as quadrupedal

Examination of the peripheral nerves of the upper limb

A lesion of a peripheral nerve causes a characteristic motor and sensory loss.[13] Peripheral nerve lesions may have local causes, such as trauma or compression, or may be part of a mononeuritis multiplex, where more than one nerve is affected by systemic disease.

The radial nerve (C5–C8)

This is the *motor nerve* supplying the triceps and brachioradialis and the

extensor muscles of the hand. The characteristic deformity that results from radial nerve injury is *wrist drop*. To demonstrate this, if it is not already obvious, get the patient to flex the elbow, pronate the forearm and extend the wrist and fingers. If a lesion occurs above the upper third of the upper arm, the triceps muscle is also affected. Therefore test elbow extension, which will be absent if the lesion is high.

Test *sensation* using a pin over the area of the anatomical snuff box. Sensation here is lost with a radial nerve lesion before the bifurcation into posterior interosseous and superficial radial nerves at the elbow (Figure 11.57).

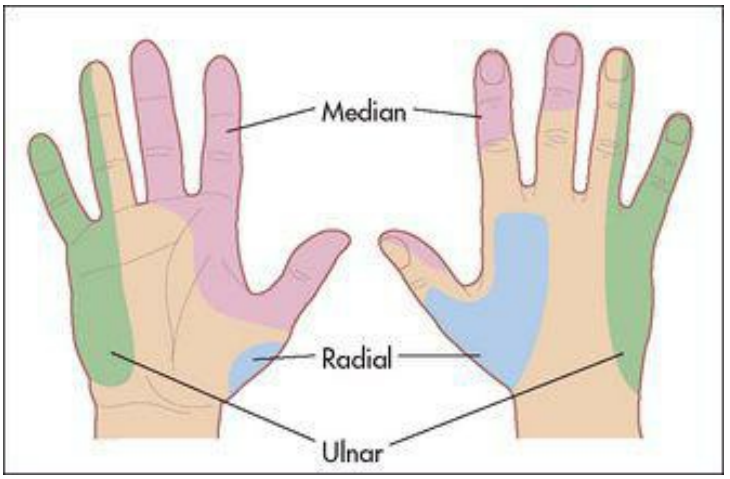


Figure 11.57 Average loss of pain sensation (pinprick) with lesions of the major nerves of the upper limbs

The median nerve (C6–T1)

This nerve contains the *motor* supply to all the muscles on the front of the forearm except the flexor carpi ulnaris and the ulnar half of the flexor digitorum profundus. It also supplies the following short muscles of the hand (LOAF)—the ***L****ateral* two lumbricals, ***O****pponens* pollicis, ***A****bductor* pollicis brevis, and in many people the ***F****lexor* pollicis brevis.

Lesion at the wrist (carpal tunnel).[14,15]

Use the pen-touching test to assess for weakness of the abductor pollicis brevis. Ask the patient to lay the hand flat, palm upwards on the table, and attempt to abduct the thumb vertically to touch the examiner's pen held above it (Figure 11.58). This may be impossible if there is a median nerve palsy at the wrist or above. Remember, however, that most patients with the

palsy at the wrist or above. Remember, however, that most patients with the carpal tunnel syndrome have normal power and may indeed have symptoms but no signs at all.

Figure 11.58 Pen-touching test for loss of abductor pollicis brevis: 'Lift your thumb straight up to touch my pen'

Lesion in the cubital fossa

Ochsner's clasping test[ll] (for loss of flexor digitorum sublimis). Ask the patient to clasp the hands firmly together (Figure 11.59a)—the index finger on the affected side fails to flex with a lesion in the cubital fossa or higher (Figure 11.59b).

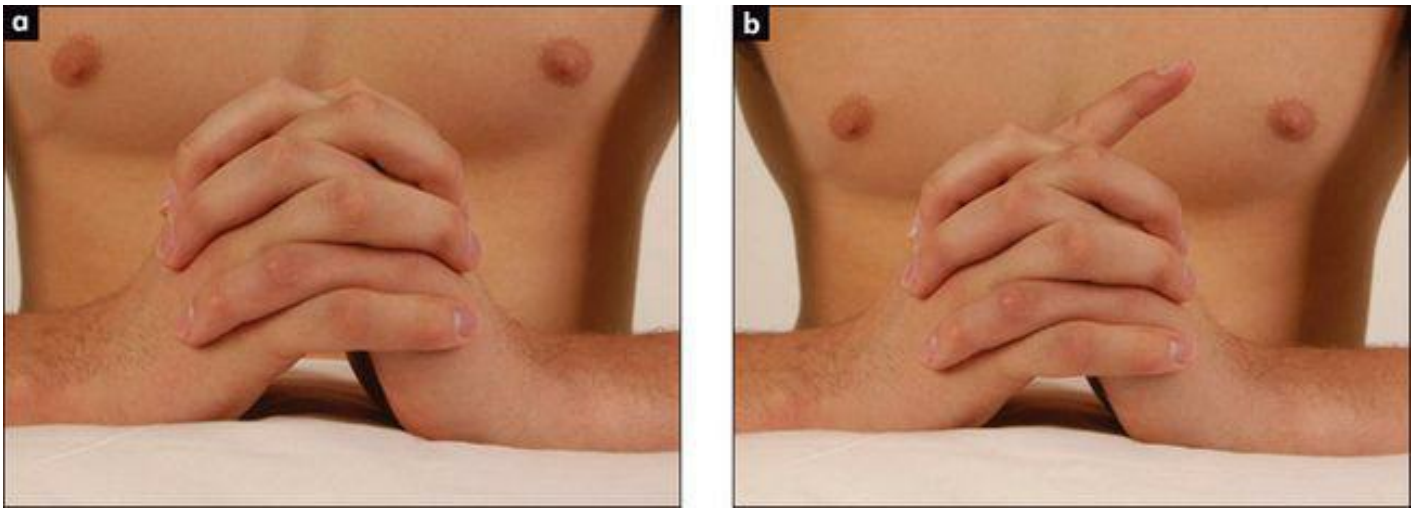


Figure 11.59 Ochsner's clasping test
(a) Normal. (b) Abnormal due to loss of function of the flexor digitorum (simulated demonstration).

For the *sensory* component of the median nerve, test pinprick sensation over the hand. The constant area of loss includes the palmar aspect of the thumb, index, middle and lateral half of the ring fingers (Figure 11.57). The palm is spared in median nerve lesions in the carpal tunnel.

The ulnar nerve (C8–T1)

This nerve contains the *motor* supply to all the small muscles of the hand (except the LOAF muscles), flexor carpi ulnaris and the ulnar half of flexor digitorum profundus. Look for wasting of the small muscles of the hand and for partial clawing of the little and ring fingers (a claw-like hand). Clawing is hyperextension at the metacarpophalangeal joints and flexion of the interphalangeal joints. Note that clawing is more pronounced with an ulnar nerve lesion at the wrist, as a lesion at or above the elbow also causes loss of the flexor digitorum profundus, and therefore less flexion of the interphalangeal joints. This is the 'ulnar nerve paradox', in that a more distal lesion causes greater deformity.

Froment's sign[mm]

Ask the patient to grasp a piece of paper between the thumb and lateral aspect of the forefinger with each hand. The affected thumb will flex because of loss of the adductor of the thumb.

Causes of a true claw hand are shown in Table 11.11, while causes of wasting of the small muscles of the hand are shown in Table 11.12; see also Figure 11.60.

TABLE 11.11 Causes (differential diagnosis) of a true claw hand (all fingers clawed)

Ulnar and median nerve lesion (ulnar nerve palsy alone causes a claw-like hand)
Brachial plexus lesion (C8–T1)
Other neurological disease—e.g. syringomyelia, polio

Other neurological disease – e.g. syringomyelia, polio
Ischaemic contracture (late and severe)
Rheumatoid arthritis (advanced, untreated disease)

TABLE 11.12 Causes (differential diagnosis) of wasting of the small muscles of the hand

Spinal cord lesions Syringomyelia Cervical spondylosis with compression of the C8 segment Tumour Trauma
Anterior horn cell disease Motor neurone disease, poliomyelitis Spinal muscular atrophies, e.g. Kugelberg-Welander* disease
Root lesion C8 compression
Lower trunk brachial plexus lesion Thoracic outlet syndromes Trauma, radiation, infiltration, inflammation
Peripheral nerve lesions Median nerve lesion

Median and ulnar nerve lesions Peripheral motor neuropathy
Myopathy Dystrophia myotonica—forearms are more affected than the hands Distal myopathy
Trophic disorders Arthropathies (disuse) Ischaemia, including vasculitis Shoulder hand syndrome

Note: Distinguishing an ulnar nerve lesion from a C8 root/lower trunk brachial plexus lesion depends on remembering that sensory loss with a C8 lesion extends proximal to the wrist, and the thenar muscles are involved with a C8 root or lower trunk brachial plexus lesion. Distinguishing a C8 root from a lower trunk brachial plexus lesion is difficult clinically, but the presence of a Horner's syndrome or an axillary mass suggests the brachial plexus is affected.

* Eric Klas Kugelberg (1913–83), professor of clinical neurophysiology at the Karolinska Institute in Stockholm, and Lisa Welander (1909–2001) described this in 1956. Lisa Welander was Sweden's first woman professor of neurology.

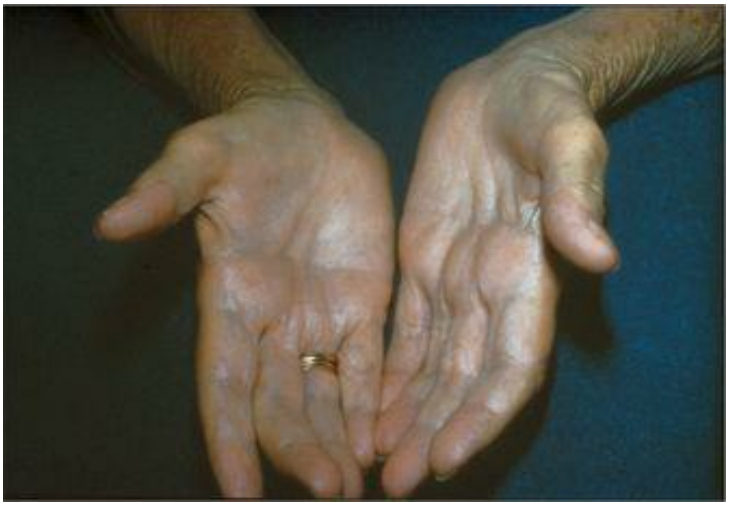


Figure 11.60 Motor neurone disease
Shows wasting of the small muscles of the hand.

For the *sensory* component of the ulnar nerve, test for pinprick loss over the palmar and dorsal aspects of the little finger and the medial half of the ring finger (Figure 11.57).

The brachial plexus

Brachial plexus lesions vary from mild to complete; motor and/or sensory fibres may be involved. Nerve roots form trunks, which divide into cords and then form peripheral nerves (see Tables 11.13 and 11.14). The anatomy is shown in Figure 11.61.

TABLE 11.13 Nerve roots and brachial plexus trunks

Nerve roots	Trunks	Muscles supplied
C5 and 6	Upper	Shoulder (especially biceps and deltoid)
C7	Middle	Triceps and some forearm muscles
C8 and T1	Lower	Hand and some forearm muscles

TABLE 11.14 Brachial plexus cords, nerves and their supplied muscles

Cords	Nerves formed	Muscles supplied
Lateral	Musculocutaneous, median	Biceps, pronator teres, flexor carpi radialis
Medial	Median and ulnar	Hand muscles
Posterior	Axillary and radial	Deltoid, triceps and forearm extensors

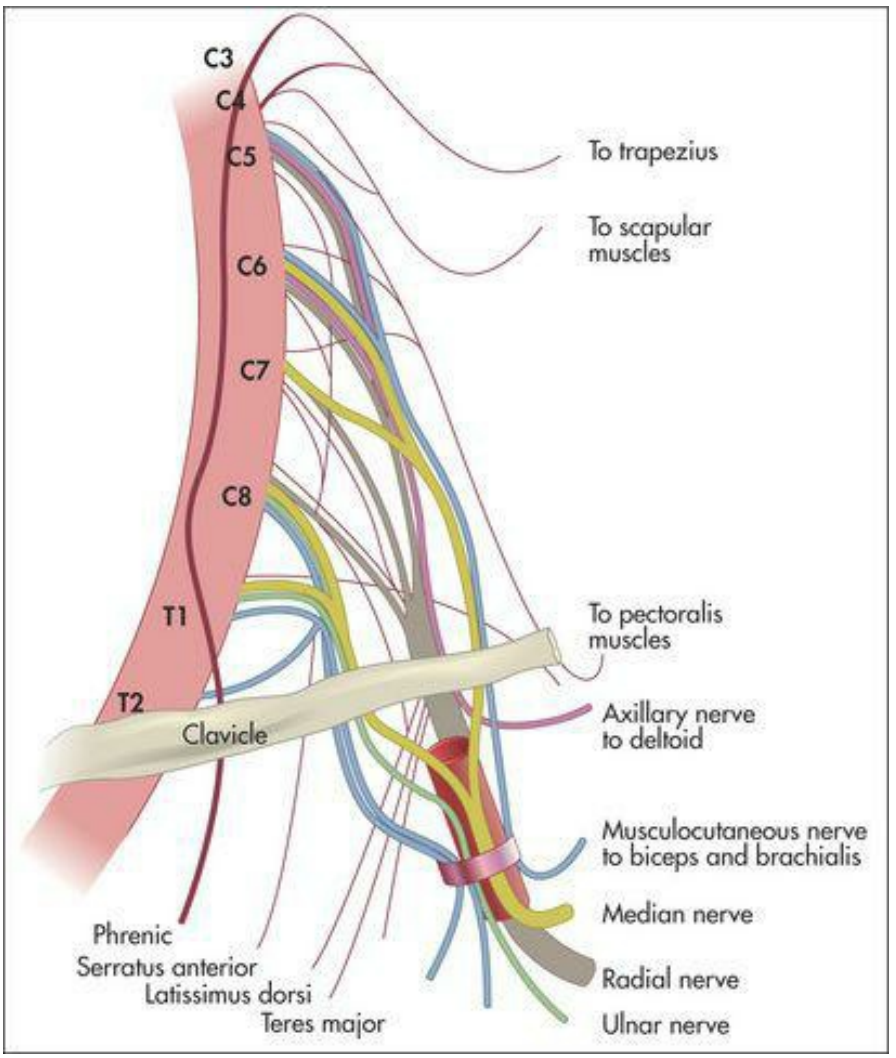


Figure 11.61 The brachial plexus.

Figure 11.01 The brachial plexus

Adapted from Chusid JG, Correlative neuroanatomy and functional neurology, 19th edn. Los Altos: Lange Medical, 1985.

Patients with brachial plexus lesions may complain of pain or weakness in the shoulders or arms. Pain is often prominent, especially when there has been nerve root avulsion. A neurological cause is more likely if there is dull pain that is difficult to localise, if the pain is not related to limb movement and is worse at night, and if there is no associated tenderness. The patient may be unable to get comfortable. An orthopaedic or traumatic cause is more likely if the pain is much worse with movement, or there are signs of inflammation, joint deformity or local tenderness. Most plexus lesions are supraclavicular (i.e. proximal), especially when they occur after trauma. When infraclavicular (i.e. distal) lesions occur they are usually less severe.

Examine the arms and shoulder girdle (Table 11.15). Remember that the dorsal scapular nerve (which supplies the rhomboid muscles) comes from the C5 nerve root proximal to the upper trunk, and so rhomboid function is usually spared in upper trunk lesions. Typical lesions of the brachial plexus are described in Table 11.16. The cervical rib syndrome may cause a lower brachial plexus lesion (Table 11.17) . Table 11.18 suggests a scheme for distinguishing plexus and nerve root lesions.

TABLE 11.15 Shoulder girdle examination

Method
Abnormalities are likely to be due to a muscular dystrophy, single nerve or a root lesion. Inspect each muscle, palpate its bulk and test function as follows:
1 *Trapezius (XI, C3, C4):* ask the patient to elevate the shoulders against resistance and look for winging of the upper scapula

against resistance and look for winging of the upper scapula.
2 *Serratus anterior (C5–C7):* ask the patient to push the hands against the wall and look for winging of the lower scapula.
3 *Rhomboids (C4, C5):* ask the patient to pull both shoulder blades together with the hands on the hips.
4 *Supraspinatus (C5, C6):* ask the patient to abduct the arms from the sides against resistance.
5 *Infraspinatus (C5, C6):* ask the patient to rotate the upper arms externally against resistance with the elbows flexed at the sides.
6 *Teres major (C5–C7):* ask the patient to rotate the upper arms internally against resistance.
7 *Latissimus dorsi (C7, C8):* ask the patient to pull the elbows into the sides against resistance.
8 *Pectoralis major, clavicular head (C5–C8):* ask the patient to lift the upper arms above the horizontal and push them forward against resistance.
9 *Pectoralis major, sternocostal part (C6–T1) and pectoralis minor (C7):* ask the patient to adduct the upper arms against resistance.
10 *Deltoid (C5, C6) (and axillary nerve):* ask the patient to abduct the arms against resistance.

TABLE 11.16 Brachial plexus lesions

Complete lesion (rare)
1. Lower motor neurone signs affect the whole arm
2. Sensory loss (whole limb)
3. Horner's syndrome (an important clue)

Note: This is often painful.
Upper lesion (Erb Duchenne*) (C5, C6) 1. Loss of shoulder movement and elbow flexion—the hand is held in the waiter's tip position 2. Sensory loss over the lateral aspect of the arm and forearm
Lower lesion (Klumpke†) (C8, T1) 1. True claw hand with paralysis of all the intrinsic muscles 2. Sensory loss along the ulnar side of the hand and forearm 3. Horner's syndrome

* Wilhelm Heinrich Erb (1840–1921), Germany's greatest neurologist.

† Auguste Déjérine-Klumpke (1859–1927), French neurologist, described this lesion as a student. She was an American, but was educated in Switzerland. As a final-year student she married the great French neurologist Jules Déjérine.

TABLE 11.17 Cervical rib syndrome

Clinical features
1 Weakness and wasting of the small muscles of the hand (claw hand)
2 C8 and T1 sensory loss
3 Unequal radial pulses and blood pressure
4 Subclavian bruits on arm manoeuvring (may be present in normal people)
5 Palpable cervical rib in the neck (uncommon)

TABLE 11.18 Distinguishing brachial plexus lesions and nerve root compression

	Root	**Plexus**
Previous trauma	Occasionally	Some types
Insidious onset	Usually	Some types
Neck pain	Yes	No
Unilateral interscapular pain	Yes	No
Weakness	Mild–moderate	Often severe
Pattern of weakness	Most commonly triceps (C7 lesions, the most commonly affected root)	Usually shoulder and biceps or hand