The eighth cranial (auditory) nerve has two components, the cochlear (responsible for hearing) and the vestibular (labyrinthine) responsible for balance and appreciation of movement (Fig. 1).

**COCHLEAR ASPECTS**

Nerve fibres from the Organ of Corti are rapidly joined by the vestibular nerve, travel through the facial canal, pass via the internal auditory meatus into the cerebellopontine angle, and the cochlear fibres then enter the pons. Within the pons, fibres cross the midline so that hearing from both ears can be integrated.

Ward testing of hearing should be carried out in a quiet room and after inspection of the ear drum. Each ear is tested separately whilst the other auditory meatus is occluded by a fingertip. Classically a ticking watch was used for this test despite the fact that the pitch was higher than ideal, but because of the epidemic of digital watches, some other stimulus such as the whispered voice has to be used. If a patient is deaf, and if the tympanic membrane is normal and unobstructed by wax, then tuning fork tests are useful.

**THE RINNE TEST**

If the conduction of sound impulses through the external or middle ear is decreased (by about 30 decibels or more) then the normal advantage of air conduction is lost and bone conduction becomes better than air. The Rinne test is performed to compare air conduction with bone conduction. To perform the test (Fig. 2), the base of a (512 vibrations per second) tuning fork is set vibrating and placed upon the patient’s mastoid process. The patient is then asked to say when the (bone conducted) tuning fork vibrations cease. When this point has been reached, the distal (vibrating end) of the fork is held about 1 cm from the external auditory meatus to test air conduction.

Normally air conduction is better than bone — Rinne positive. If bone conduction is better than air conduction unilaterally, the Rinne test is negative indicating a conductive deafness.

When testing a totally deaf ear it would (spuriously) appear that bone conduction was better than air conduction — a false negative Rinne test. This is because bone conduction to the ear not under test is better than air conduction to the ear under test. This false negative Rinne test should not cause confusion because severe deafness should have already been discovered.

**WEBER’S TEST**

In Weber’s test (Fig. 3) the tuning fork base is placed on the vertex of the skull and the patient asked in which ear the sound is best heard. Normally, the sound is heard in the centre. In conductive deafness the sound is heard in the ear most affected by the deafness (as if the cochlea on that side is perpetually straining to hear and becomes hypersensitive as a result). In sensineural deafness, the tuning fork will be heard best in the unaffected ear.
THE VESTIBULAR APPARATUS

The vestibular apparatus gives the brain information about head position and head movement. After emerging from the internal auditory meatus, the vestibular fibres enter the brainstem and relay with other nervous elements, including structures relevant to eye movements and the vagus nerve.

TINNITUS

Tinnitus is a buzzing, ringing or hissing sound heard by the sufferer. The commonest types of tinnitus are those associated with cochlear dysfunction with sensorineural deafness, especially presbyacusis (poor hearing associated with age).

VERTIGO

Vertigo is a false impression of rotation (not dizziness or swimminess) of either the patient or the surroundings. Vertigo may result from vestibular nerve lesions, lesions of the brainstem connections or lesions of ‘brainstem associated’ tissues — especially the cerebellum. Balance may be impossible and the patient may become ataxic or unable to stand. Vertigo, unless trivial, is almost always associated with nystagmus and because of the numerous connections within the brainstem, nausea, vomiting and sweating may be associated.

Several well recognized vertiginous syndromes exist, with suggestive findings on history or examination.

Benign positional vertigo

Patients with benign positional vertigo are usually middle-aged and have sudden onset of vertigo whilst changing position. To test for this the patient is positioned sitting up so that the head, held by the examiner, can be quickly lowered over the end of the couch below the horizontal, with one ear downwards (Fig. 4). The examiner should look for nystagmus (the patient will surely tell the examiner if vertigo results!). After a resting period this manoeuvre can be repeated with the other ear downwards. Vertigo plus nystagmus tends to occur when the dysfunctioning vestibular apparatus is in the ‘underneath’ position.

In ‘benign’ positional vertigo there is a delay of several seconds before nystagmus, directed towards the lower ear, occurs. On repetition the response is less marked.

In ‘malignant’ positional vertigo there is no delay in onset of nystagmus, and the nystagmus persists as long as precipitating position is maintained. On repetition the response persists unchanged. Such a response may be indicative of a posterior fossa space-occupying lesion.

Vestibular neuronitis

In vestibular neuronitis the patient experiences variable vertigo which is independent of head position. Recovery occurs over several weeks.

Menière’s syndrome

Menière’s syndrome is characterized by episodes of sudden onset severe vertigo with prostration lasting up to 24 hours. There is tinnitus and usually subsequent nerve deafness.

Migraine

Migraine may cause vertigo. The clues are the characteristic features of the associated headache.

Multiple sclerosis (MS)

Vertigo is a possible feature of MS. There may be other brainstem signs including diplopia, numbness of the face, dysarthria, or limb ataxia.

Brainstem ischaemia or temporal lobe epilepsy

Both brainstem ischaemia or temporal lobe epilepsy may cause vertigo.
THE TRIGEMINAL (V) NERVE

The large sensory and a smaller motor root pass forward from the lateral pons to enter a cavity in the dura mater overlying the apex of the petrous temporal bone. There the gasserian ganglion is formed which gives rise to three divisions of the trigeminal nerve — the ophthalmic, the maxillary and the mandibular. The sensory distribution of trigeminal nerve innervation is shown in Figure 1.

Internal sensation is also provided by the trigeminal nerve for the nasal mucosa, the hard and soft palate, teeth, anterior two-thirds of the tongue (via the chorda tympani of the facial nerve) and the buccal mucosa.

After entry to the brainstem trigeminal pain and temperature appreciation fibres descend and may reach the second cervical segment of the spinal cord. There they cross the midline, eventually uniting with the pain and temperature sensory input from the trunk and limbs. Touch sensation crosses at a higher level, and thus dissociated sensory loss may be found if these lower midline-crossing pain and temperature fibres are damaged. This results in trigeminal pain and temperature impairment, whilst touch appreciation is retained.

Examination of trigeminal nerve function

The touch, pain and temperature cutaneous sensation of the three divisions can be tested separately. The corneal blink reflex is in many ways the most crucial sensory function of the trigeminal nerve because an impaired corneal reflex leaves the eye vulnerable to injury by trauma. To test the corneal reflex, a thin wisp of cotton wool should be introduced from the side (so that the patient cannot see it) and be stroked gently across the cornea. A blink (of both eyes) is normally elicited.

To test the motor trigeminal function, the temporalis, masseter and pterygoid muscles are assessed. To test the temporalis and masseter muscles the patient should be asked to clench his teeth so that these two muscles can be seen (or palpated) to be contracting normally (Fig. 2). To test the pterygoids the patient should be asked to protrude the jaw or open the mouth against slight resistance. The jaw will deviate towards the paralysed side (Fig. 3). The jaw jerk reflex should also be elicited. Like all routinely tested reflexes, the jaw jerk is a muscle stretch reflex. The patient lets the jaw flop open and the examiner puts a finger horizontally across the chin and taps the finger with a patellar hammer (it helps if the patient has the eyes shut). If the response is brisker than normal this, like any other abnormally brisk muscle stretch reflex, implies upper motor neurone damage. This reflex has localization value.

If the jaw jerk is pathologically brisk in the presence of bilateral upper motor neurone signs in the limbs, the lesion should be above the pons, whereas if the limb reflexes are brisk but the jaw jerk is normal, the lesion should be below the pons.

THE FACIAL (VII) NERVE

The facial nerve is mostly motor, although for part of its course it carries along with it taste sensation from the anterior two-thirds of the tongue, and some fibres which stimulate salivary secretion. The nucleus of the facial nerve is in the pons. The facial nerve supplies motor impulses to the stapedius muscle, stylohyoid, posterior belly of the digastric, and to the muscles of facial expression.

The forehead muscles are represented bilaterally in the cerebral cortex. Because of this, unilateral cerebral damage does not cause an upper motor neurone type weakness of the forehead muscles of that side. A lower motor neurone lesion of the facial nerve causes weakness of all facial nerve innervated muscles on that side of the face. Thus, there is a paradox in terminology — an upper motor neurone lesion only affects the lower half of the face. Because the orbicularis oculi is weak, overaction of the other eyelid muscles means that the eyelid cannot close. This leads to the possibility of corneal damage.

Testing facial nerve function

Ask the patient to wrinkle the forehead. This will be unilaterally impaired with a unilateral lower motor neurone lesion.

Ask the patient to close his eyes tightly. With a lower motor neurone lesion this is difficult and in the attempt the eyeball also rolls upwards.

Test taste sensation of the anterior two-thirds of the tongue. Place small quantities of the substance under test (traditionally sugar for sweet, quinine for bitter, vinegar for sour and table salt for salt) onto the patient’s protruded tongue.

The patient should be asked by the examiner to nod his head slightly to indicate a positive reply during the examiner’s serial questions: ‘Is this sweet? Bitter? Sour? Salty?’. The patient obviously cannot speak his reply!

Ask the patient to wrinkle his nose, show his teeth and blow out the cheeks whilst trying to keep the mouth shut.
(‘pretending to play the trumpet’). An upper or a lower motor neurone lesion impairs the performance for all three and, in particular, blowing out the cheeks results in leakage from the corner of the mouth on the affected side.

With an upper motor neurone lesion, emotionally induced movements are usually only slightly impaired.

THE GLOSSOPHARYNGEAL (IX) NERVE

The glossopharyngeal nerve gathers sensation from the back third of the tongue, the fauces, the palate and the upper pharynx, and provides secretory fibres to the parotid gland. It also transmits impulses from the chemoreceptors and baroreceptors of the carotid body and sinus. In addition the glossopharyngeal nerve also supplies the stylopharyngeus muscle which, together with the palatopharyngeus muscle (X nerve), elevates the palate.

To test for pharyngeal sensation, gently touch the soft palate on each side with an orange stick, and then the posterior pharyngeal wall on each side (Fig. 4). After withdrawal of the orange stick the patient should be asked if all four stimuli were similar. If it is deemed necessary to elicit the gag reflex these stimuli should be evoked with a spatula. The gag reflex tests both IX (sensory) and X (motor) functions. It is almost impossible to assess differential taste discrimination on the posterior third of the tongue.

THE VAGUS (X) NERVE

The vagus nerve contributes to various autonomic plexuses. Motor fibres drive the muscles of the palate, larynx, pharynx and also gather a small sensory input from around the external auditory meatus. The recurrent laryngeal nerve (a branch of the vagus) on the right winds posteriorly around the subclavian artery, whereas on the left the recurrent laryngeal nerve winds around the arch of the aorta. Both drive the intrinsic muscles of the larynx: unilateral laryngeal nerve palsies cause dysphonia (impairment of voice production), whilst bilateral palsies cause stridor (p. 145).

Examination of the vagus nerve includes observation of palatal movements, assessment of the patient’s voice and the ability to cough. Weakness of the palate may cause nasal speech and/or regurgitation of food into the nose. If one side of the palate is paralysed, the uvula is pulled over to the intact side when the patient says ‘Aah’.

Weakness of one vocal cord caused by lack of vagal innervation leads to a weak, sometimes hoarse, voice and an inability to cough explosively.

THE ACCESSORY (XI) NERVE

The accessory nerve drives the upper part of the trapezius and the sternomastoid muscle. An accessory nerve lesion causes weakness and wasting of these muscles with impairment of lateral rotation of the head to the opposite side (Fig. 5). Shoulder shrugging is also impaired.

THE HYPOGLOSSAL (XII) NERVE

The hypoglossal nerve is the motor nerve to the tongue. Unilateral lower motor neurone type damage causes weakness, wasting and often unilateral fasciculation of tongue muscle. Weakness of one side enables the muscle component responsible for protrusion of the other side to push over the weak side. Therefore, the protruded tongue deviates towards the weak side (Fig. 6). Upper motor neurone type damage causes a stiff, non-wasted tongue which may be hyperreflexic if percussed.

Other cranial nerves

- An absent corneal reflex predisposes to corneal injury.
- An upper motor neurone facial palsy only affects the lower face.
- With a lower motor neurone facial palsy the upper eyelid cannot be closed because the orbicularis oculi muscle that arches over the eye cannot contract normally.
- A lower motor neurone facial palsy predisposes to corneal injury.
BASIC PRINCIPLES

Neurological examination can be incredibly complex but in an account written for students of medicine more simple screening schemes are more appropriate. This section will therefore concentrate on such techniques before dealing with specific neurological manifestations.

The more complex a screening neurological test requested of a patient, the more likely it is to reveal an abnormality. However, the precise identification of the underlying abnormality almost invariably requires more specific testing.

Testing at the periphery of limbs rather than centrally is more likely to reveal a neurological defect. For example, if the patient can maintain the position of an outstretched upper limb whilst the examiner presses down upon the out-stretched fingers, muscle weakness in shoulder, elbow and wrist muscles is unlikely.

Screening for sensation defects is best performed at the peripheries, because the nerves supplying the peripheries are longer and thus more vulnerable to diffuse pathologies such as polynuertitis or neuropathies (which often first manifest by changes in the feet).

Diffuse metabolic or degenerative nervous system pathologies usually produce symmetrical signs rather than isolated or unilateral signs.

When testing sensation move from areas of normal sensation to areas of reduced sensation as patients find it easier to be precise about boundary definition.

The five neurological elements of limb and trunk examination are:
- tone
- power
- sensation
- coordination
- reflexes.

Abnormalities of two or more of these elements may be integrated to produce clinical signs. For example, normal coordination may require integration of proprioception, muscle power, intact higher mental function and vision.

The five neurological elements of limb and trunk examination are:
- tone
- power
- sensation
- coordination
- reflexes.

Normally if the knee and hip are passively flexed by the examiner the lower limb folds so that the heel touches the patient’s buttock.

Abnormal

Normal

Normally if the knee and hip are passively flexed by the examiner the lower limb folds so that the heel touches the patient’s buttock.

If there is an upper motor neurone lesion the lower limb remains stiff, does not fold and, because of the sudden muscle stretching, the knee jerk reflex may be evoked with a kicking action of the hypertonic limb.

EXAMINATION OF LIMBS AND TRUNK — TONE AND POWER

TONE

Clinical assessment of tone

Clinical examination of tone should be conducted by means of passive limb movements. It is important to ensure that the patient does not have joint immobility or arthritis. Over vigorous passive movements may cause pain or fractures!

In the upper limb the most useful screening test involves holding hands with the patient, flexing and extending the wrist joints, and then flexing and extending the elbow joint whilst simultaneously rotating the forearm gently.

In the lower limb, the hip joint, the knee joint and the ankle joint can be passively flexed and extended. The lower limb can be ‘rolled’ on the bed and, in the absence of arthritis of the hip, stiffness will be caused by increased tone. A useful technique is to place your hand beneath the patient’s relaxed and extended knee and then lift up the knee. Normally the patient’s heel will almost come to touch the buttocks as the legs ‘fold up’. However, if there is hypertonia this ‘folding-up’ will not occur and the whole lower limb will remain more or less straight. If the knee lifting is performed rapidly, the abrupt stretching of the quadriceps may elicit a pathological knee jerk reflex which results in a kicking-like action (Fig. 1).

Normal muscle tone is maintained by negative and positive feedback mechanisms.

Hypotonia

Hypotonia is produced by:
- lower motor neurone lesions leading to lack of motor input to the muscles
- acute neurological lesions affecting the spine — ‘spinal shock’
- lesions of the cerebellum
- chorea which may be caused by degenerative lesions or by neurological toxins
- intrinsic muscle disease
- some conditions such as tabes dorsalis in which sensory impulses derived from the muscles are lacking.

Hypertonia

Hypertonia is produced in muscles from which the higher neurological ‘relaxatory’ influences have been removed.

With upper motor neurone type rigidity there is ‘lead-pipe’ type spastic rigidity, often with rigidity predominantly manifest in either the agonist or antagonist muscles. There may also be sudden
reduction of hypertonia on passive movements — the ‘clasp-knife phenomenon’.

Damage to extrapyramidal pathways causes a persisting hypertonia which affects agonist and antagonist muscles equally; this often causes difficulties in initiating bodily movement. Additionally, there may be a typical coarse compound tremor superimposed to give a ‘cog-wheel’ type rigidity.

RARELY, hypertonia may be caused by certain intrinsic muscle disease (e.g. dystrophia myotonica), anxiety, lack of patient cooperation or hysteria.

Clonus

Clonus is usually tested at the same time as tone. Clonus is a rhythmical involuntary contraction of a muscle placed under tension. Pathological clonus (which implies damage to the corticospinal tract relevant to the affected side) increases with increased tension of the muscle concerned, whereas normal ‘physiological’ clonus is abolished with persistent or increased muscle tension. Pathological clonus is often associated with upper motor neurone type hypertonia and hyperreflexia. Quadriceps clonus is best elicited by a firm and rapid distal displacement of the patella, whilst ankle clonus is best elicited by a firm dorsiflexion of the ankle (Fig. 2).

Fig. 2 Testing for clonus.

MUSCLE POWER

Muscle power is graded on an MRC (Medical Research Council) scale of 0–5.

0 = no visible contraction
1 = visible contraction without active movement
2 = movement possible, but not against gravity
3 = movement possible against gravity
4 = movement possible against gravity and resistance, but weaker than normal
5 = normal power.

Marked muscle weakness should be initially apparent from the patient’s gait, posture or limb movements.

Formal testing of muscle power

Ask the patient to extend forwards the whole of both upper limbs symmetrically with the palms upwards. Ask the patient to close both eyes (to abolish visually initiated compensation for muscle weakness) and then ask the patient to maintain the position of the limbs. A weak arm will drift downwards under its own weight. If the drift downwards is corrected when the patient’s eyes are open then this implies that there was also some impairment of joint position sense. There is often a slow pronation of the hand if the weakness is due to an upper motor neurone lesion (Fig. 3). Ask the patient to keep centrally with their arms outstretched arms whilst you push both hands upwards, downwards, medially and laterally to assess and compare the power which the patient can utilize in each upper limb to maintain the original position. Hypotonia can also be assessed at the same time by tapping the hand upwards, downwards or sideways (in the presence of hypotonia there tends to be excessive excursion of the pushed limb with ‘overshoot’ when the patient attempts to return the limb to the original position).

In the lower limbs, similar principles apply. Ask the patient to keep his legs straight and lift each ankle in turn off the bed and assess the strength of each leg by pressing downwards at the base of the toes.

If a weakness is demonstrated, determine whether it is a global or localized weakness. A global weakness affects all muscles to a greater or lesser extent and usually indicates an upper motor neurone type weakness. A localized weakness confined to an individual muscle or groups of muscles sharing common innervations usually indicates a lower motor neurone lesion. If a lower motor neurone is partially damaged there may be fasciculation, a visible ‘wriggling’ of muscles, either spontaneously or induced by gentle tapping of the muscle.
SENSATION
There are five cutaneous sensory modalities which can usefully be tested (Fig. 1): pain, temperature (both pain and temperature cross the midline soon after entry into the spinal cord), touch (most often crosses the midline at higher levels than pain or temperature), joint position sense and vibration sense.

Patients use many colloquial descriptions of their sensory experiences. Always be sure you derive correctly the medical definition of the patient’s symptoms. Pain due to neuritis or nerve irritation is often described as ‘burning’ or ‘stabbing’. Some patients often describe total loss of sensation as ‘numbness’ but for others numbness is reduced but abnormal sensation.

The purpose of testing cutaneous sensation is to determine whether dysfunction is caused by peripheral nerve dysfunction (either focal or generalized), or by upper motor neurone damage (caused by spinal or more highly situated neurological damage). A dermatome pattern of sensory loss usually occurs in spinal cord lesions, or in nerve root lesions just after the nerves have left the spinal cord, but injury distal to this causes sensory impairment of peripheral nerve distribution (Fig. 2). With a sensory peripheral neuropathy there is ‘glove and stocking’ sensory loss, usually with approximately symmetrical cutaneous sensory loss, with a horizontal upper border.

Pain
Superficial pain is tested by pin-prick and deep pain by deep muscle or tendon pressure. When testing by pin-prick never use the same pin on different patients as this constitutes a definite infection risk. Always ask if the pin-prick feels like a pin-prick should (to avoid testing for touch — a different sensation). An opened out paper clip for pin-prick sensation is recommended. The point is not too sharp, the convex ends can be used for bluntness appreciation and the two ends can be used for two point discrimination. They are also cheap, safe and disposable. Deep pain is tested (only if indicated) by firm squeezing of muscles or tendons. Assessment of response is rather subjective, but asymmetry of response is likely to be significant.

Temperature
Temperature appreciation is assessed by using warm and cool tubes of water applied to the skin in random sequence. This test is usually only employed if dissociated sensory loss is suspected (touch intact but with loss of pain and temperature appreciation). This situation occurs if the pain and temperature fibres, which cross soon after entry into the spinal cord, are damaged by lesions in the centre of the spinal cord, with the touch fibres (which do not cross until they have ascended) intact. Affected patients typically burn themselves on hot objects. Syringomyelia, in which expanding cavities destroy the centre of the spinal cord, is a common cause.

Touch
Touch is usually tested by using a small point of cotton wool which is gently dabbed onto the skin. Stroking the skin, the neurological appreciation of which utilizes different spinal pathways, may provide misleading information. Always ask the patient if the cotton wool feels like cotton wool should feel and not just ‘can you feel this?’. It is also useful to request the patient to close his eyes to avoid vision-related positive responses.

Joint position sense
Joint position sense, if intact at the peripheries, is almost always intact proximally. Grip the innermost and outermost surfaces of the end of the big toe between your thumb and first finger. Show the patient, by moving the toe, which is ‘up’ and which is ‘down’.

Joint position sense
Also do Romberg’s test which measures integrity of proprioception.

Vibration sense
Fig. 1 Testing for sensation.

Fig. 2 Dermatome and peripheral nerve innervation.
Then ask the patient, with eyes closed, to report the direction of your random movements of the toe (remember that a guessing patient will achieve a 50% success rate). A similar technique can be used on the thumb and other peripheral joints. If there is peripheral impairment proceed to test more proximal joints.

‘Internal’ position sense is tested by Romberg’s test in which a patient (who can stand securely with feet together when the eyes are open) sways or falls when the eyes are shut — thus demonstrating that equilibrium is dependent on vision and there is inadequate input from the internal position sensors. Always stand by the patient and terminate the test if the patient sways such that he/she might fall. Patients will fail Romberg’s test if there is cerebellar incoordination but in this case visual input will not steady the patient. If the patient has a proprioceptive deficit (sensory ataxia) the swaying only occurs when the eyes are shut.

**Vibration sense**

Vibration sense, if intact at the peripheries, is almost always intact proximally. To test vibration sense, place the base of a large 128 cycle per second tuning fork on a bony prominence (the medial malleolus is the traditional site although the base of the big toe is more peripheral). The patient should be told that it is not the sensation of touch that is being tested but rather the ‘buzzing’ feeling of vibration. The patient is then asked to close the eyes and the tuning fork is removed, tapped briskly (and preferably silently). The base is replaced on the bony prominence and the patient is asked if the buzzing can be appreciated. In theory when this occurs, place the base of the (still vibrating) tuning fork on the tip of the big toe. This test can be used on the thumb and other peripheral joints. If there is vibration sense impairment at the peripheries the patient probably has neuropathy. Other ways of assessing coordination include the successive rapid movements including walking, eating and writing. When examining coordination be aware that any previously detected deficit in tone, power or certain types of sensation may affect your assessment of coordination. This is the reason why coordination should only be assessed after evaluating tone, power and sensation. However if complex coordination is normal the case of the neuropathy is likely to be normal.

To test coordination ask the patient to tap the back of one hand rapidly with the fingers of the other hand (dysdiadochokinesis). Other ways of assessing coordination include the successive touching of the thumb tip with each of the fingers of the same hand. Another easily understood request is to ask the patient to ‘play the piano’ with outstretched fingers of both hands.

Then there is the famous finger nose test which is one of the more idiotic requests that doctors make of their patients! If the patient is incoordinated or has a tremor he may well miss the nose and poke himself in the eye! It is more sensible to ask the patient to touch the point of the jaw. With the patient’s eyes closed, move one of his hands to various positions and ask the patient to touch the point of the jaw. Other ways of assessing coordination include the successive touching of the thumb tip with each of the fingers of the same hand. Another easily understood request is to ask the patient to ‘play the piano’ with outstretched fingers of both hands.

Cerebellar incoordination cannot be usefully compensated for by vision, although incoordination caused by pure muscle hypotonia or weakness may be compensated for to some extent. Incoordination must be distinguished from visiospatial impairment. In the latter, coordination may be normal but the patient cannot draw a clock, or lay a table or draw a star.

**Sensation and coordination**

- Always ensure that you interpret the patient’s description of sensory symptoms correctly.
- Pain and temperature fibres cross soon after entry into the spinal cord.
- Touch fibres ascend the spinal cord and cross later.
- Incoordination caused by impaired joint position sense will be minimized if the patient has his eyes open.
- Cerebellar incoordination is not helped by vision.
TENDON (MUSCLE STRETCH) REFLEXES

Despite the fact that any striated muscle rapidly stretched will give a reflex, only five muscle stretch reflexes are usually tested routinely (Fig. 1).

To elicit muscle stretch reflexes the patient must be relaxed, with attention distracted from the fact that you are about to strike, albeit gently, with a hammer! If necessary, the patient should close the eyes to abolish visually initiated involuntary muscle tensing.

The tendon should be struck firmly with the head of a 12-inch length patellar hammer (the miniaturized versions are not adequate) so that maximum stretching of the muscle is elicited (Fig. 2). It is important to provide identical stimuli to each side when comparing the reflexes. A useful technique to confirm asymmetry of reflexes is to diminish progressively a stimulus on one side until there is no response. If an identical stimulus provokes a response on the other side then there is asymmetry (either hyporeflexia on one side or hyperreflexia on the other). When percussing a tendon it is helpful to have the hammer head at right angles to the tendon. Figure 3 details the practical order of percussion of the upper limb reflexes.

Hyperreflexia
Pathologically brisk reflexes occur with upper motor neurone lesions or in patients with painful limbs. Anxiety may cause brisk reflexes. There is a well-recognized syndrome (to the author at least) of symmetrical markedly brisk reflexes, normal plantar responses and bitten finger nails which signifies an anxiety state.

Hyporeflexia
Hyporeflexia is found if either sensation or motor innervation is defective, or if there is spinal cord dysfunction at the level of the reflex concerned. If the defect is motor, there will usually be muscle wasting, but if the hyporeflexia is caused by sensory impairment, muscle wasting is unusual.
**Points to note**

The gracilis reflex (L2, 3) is useful in collapsed patients, or in patients who cannot flex their knees. A finger or thumb is placed transversely across the gracilis muscle just above the knee and percussed with a patellar hammer. The gracilis contraction even if not visible, can be felt by the finger or thumb (Fig. 4).

Lesions at C7, 8 sometimes cause inversion of the triceps reflex — which is a flexion rather than extension of the elbow joint — almost as if the absent triceps response allows the transmitted stretching stimulus to be transmitted to the biceps which then contracts.

In hypothyroidism, reflex contractions may be sustained with a slow relaxation phase (Fig. 5).

**THE ABDOMINAL REFLEXES**

The normal abdominal reflexes (Fig. 6) consist of a contraction of the relevant quadrant muscles when the skin of that quadrant is gently scratched with a pointed object. A normal response requires an intact upper motor neurone relevant to the affected side, intact cutaneous sensation and lower motor neurone supply to the contracting muscles. This reflex may be absent in old people and in some normal individuals, but any asymmetry of response would be significant.

**THE PLANTAR RESPONSES**

The most useful way of eliciting the plantar response is to use the prongs of a tuning fork which provide two stimuli for the price of one. The prongs should be gently drawn along the lateral border of the foot from the heel towards the little toe (S1 cutaneous innervation). It is important that the toes are in the resting position at the start of the test. A normal response is flexor with plantar flexion of the big toe. A pathological response is extensor with extension of the big toe (often with a fanning of the other four toes). The crucial part of the extensor plantar response is an initial contraction affecting the extensor hallucis longus tendon.

An extensor response is found in lesions of the upper motor neurone relevant to that foot. In comatose patients both plantar responses may be extensor and of no diagnostic significance, but any asymmetry of response would imply an asymmetrical causative lesion. Absent plantar response may be also found in peripheral neuropathy. Equivocal responses are found in infants under the age of 12 months.

A convenient method of recording the findings on examination of the reflexes and plantar responses is shown in Figure 7.