7.1 Clinical characteristics of headache syndromes

	Onset	Duration/periodicity	Pain location	Associated features
Primary syndromes				
Migraine	Evolves over 30–120 mins	Usually last <24 h, recurrent with weeks/months symptom-free	Classically unilateral but may be anywhere including face/neck	Aura (usually visual), nausea/vomiting, photophobia and phonophobia
Cluster headache	Rapid onset, often waking patient from sleep	30–120 mins, 1–4 attacks within 24 h, clusters usually last weeks to months, with months to years of remission	Orbital/retro-orbital; always same side during cluster, may switch sides between clusters	Autonomic features, including conjunctival injection, tearing, nasal stuffiness, ptosis, miosis, agitation
Stabbing headache	Abrupt, rarely from sleep	Very brief, seconds or less	Anywhere over head	Common in migraineurs
Secondary syndromes				
Meningitis	Usually evolves over a day or two, can be abrupt	Depends on cause and treatment, usually days to weeks	Global, including neck stiffness	Fever, meningism, rash, false localising signs, signs of raised intracranial pressure
Subarachnoid haemorrhage	Abrupt, immediately maximal, rare from sleep	May be fatal at onset, usually days to weeks	Anywhere, poor localising value	20% isolated headache only; nausea/vomiting, reduced consciousness, false localising signs, III nerve palsies
Temporal arteritis	Gradual onset of temple pain and scalp tenderness	Continuous	Temple and scalp	Usually in those >55 years; unwell, jaw pain on chewing, visual symptoms, tender temporal arteries, elevated erythrocyte sedimentation rate and C-reactive protein

7.2 Features that help discriminate vasovagal syncope from epileptic seizure

Feature	Vasovagal syncope	Seizure
Triggers	Typically pain, illness, emotion	Often none (sleep deprivation, alcohol, drugs)
Prodrome	Feeling faint/ lightheaded, nausea, tinnitus, vision dimming	Focal onset (not always present)
Duration of unconsciousness	<60 s	1–2 mins
Convulsion	May occur but usually brief myoclonic jerks	Usual, tonic–clonic 1–2 mins
Colour	Pale/grey	Flushed/cyanosed, may be pale
Injuries	Uncommon, sometimes biting of tip of tongue	Lateral tongue biting, headache, generalised myalgia, back pain (sometimes vertebral compression fractures), shoulder fracture/ dislocation (rare)
Recovery	Rapid, no confusion	Gradual, over 30 mins; patient is often confused, sometimes agitated/ aggressive, amnesic

7.3 Clinical classification of stroke

Total anterior circulation syndrome (TACS)

- Hemiparesis, hemianopia and higher cortical deficit (e.g. dysphasia or visuospatial loss)

Partial anterior circulation syndrome (PACS)

- Two of the three components of a TACS
- OR isolated higher cortical deficit
- OR motor/sensory deficit more restricted than LACS (see below)

Posterior circulation syndrome (POCS)

- Ipsilateral cranial nerve palsy with contralateral motor and/or sensory deficit
- OR bilateral motor and/or sensory deficit
- OR disorder of conjugate eye movement
- OR cerebellar dysfunction without ipsilateral long-tract deficits
- OR isolated homonymous visual field defect

Lacunar syndrome (LACS)

- Pure motor > 2 out of 3 of face, arm, leg
- OR pure sensory > 2 out of 3 of face, arm, leg
- OR pure sensorimotor > 2 out of 3 of face, arm, leg
- OR ataxic hemiparesis

2 Parietal lobe

Dominant side

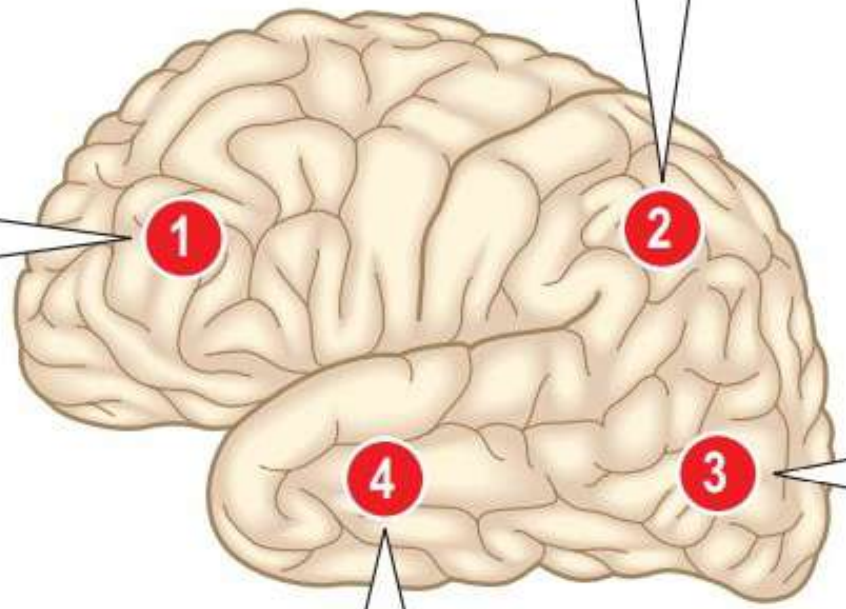
FUNCTION	LESIONS
Calculation	Dyscalculia
Language	Dysphasia
Planned movement	Dyslexia
Appreciation of size, shape, weight and texture	Apraxia
	Agnosia
	Homonymous hemianopia

Non-dominant side

FUNCTION	LESIONS
Spatial orientation	Neglect of non-dominant side
Constructional skills	Spatial disorientation
	Constructional apraxia
	Dressing apraxia
	Homonymous hemianopia

1 Frontal lobe

FUNCTION	LESIONS
Personality	Disinhibition
Emotional response	Lack of initiative
Social behaviour	Antisocial behaviour
	Impaired memory
	Incontinence
	Grasp reflexes
	Anosmia



3 Occipital lobe

FUNCTION	LESIONS
Analysis of vision	Homonymous hemianopia
	Hemianopic scotomas
	Visual agnosia
	Impaired face recognition (prosopagnosia)
	Visual hallucinations (lights, lines and zigzags)

4 Temporal lobe

Dominant side

FUNCTION	LESIONS
Auditory perception	Dysphasia
Speech, language	Dyslexia
Verbal memory	Poor memory
Smell	Complex hallucinations (smell, sound, vision)
	Homonymous hemianopia

Non-dominant side

FUNCTION	LESIONS
Auditory perception	Poor non-verbal memory
Music, tone sequences	Loss of musical skills
Non-verbal memory (faces, shapes, music)	Complex hallucinations
Smell	Homonymous hemianopia

A

7.4 Summary of the 12 cranial nerves

Nerve	Examination	Abnormalities/symptoms
I	Sense of smell, each nostril	Anosmia/parosmia
II	Visual acuity Visual fields Pupil size and shape Pupil light reflex Fundoscopy	Partial sight/blindness Scotoma; hemianopia Anisocoria Impairment or loss Optic disc and retinal changes
III	Light and accommodation reflex	Impairment or loss
III, IV and VI	Eye position and movements	Strabismus, diplopia, nystagmus
V	Facial sensation Corneal reflex Muscles of mastication Jaw jerk	Impairment, distortion or loss Impairment or loss Weakness of chewing movements Increase in upper motor neurone lesions
VII	Muscles of facial expression Taste over anterior two-thirds of tongue	Facial weakness Ageusia (loss of taste)
VIII	Whisper and tuning fork tests Vestibular tests	Impaired hearing/deafness Nystagmus and vertigo
IX	Pharyngeal sensation	Not routinely tested
X	Palate movements	Unilateral or bilateral impairment
XI	Trapezius and sternomastoid	Weakness of scapular and neck movement
XII	Tongue appearance and movement	Dysarthria and chewing/ swallowing difficulties

7.5 Comparison of bulbar and pseudobulbar palsy

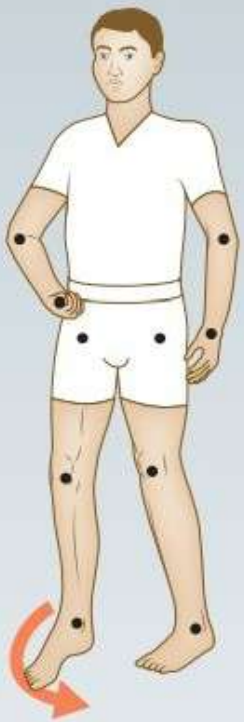
	Bulbar palsy	Pseudobulbar palsy
Level of motor lesion	Lower motor neurone	Upper motor neurone
Speech	Dysarthria	Dysarthria and dysphonia
Swallowing	Dysphagia	Dysphagia
Tongue	Weak, wasted and fasciculating	Spastic, slow-moving
Jaw jerk	Absent	Present/brisk
Emotional lability	Absent	May be present
Causes	Motor neurone disease	Cerebrovascular disease, motor neurone disease, multiple sclerosis

7.6 Features of motor neurone lesions

	Upper motor neurone lesion	Lower motor neurone lesion
Inspection	Usually normal (may be disuse wasting in longstanding lesions)	Muscle wasting, fasciculations
Tone	Increased with clonus	Normal or decreased, no clonus
Weakness	Preferentially affects extensors in arms, flexors in leg	Usually more focal, in distribution of nerve root or peripheral nerve
Deep tendon reflexes	Increased	Decreased/absent
Plantar response	Extensor (Babinski sign)	Flexor

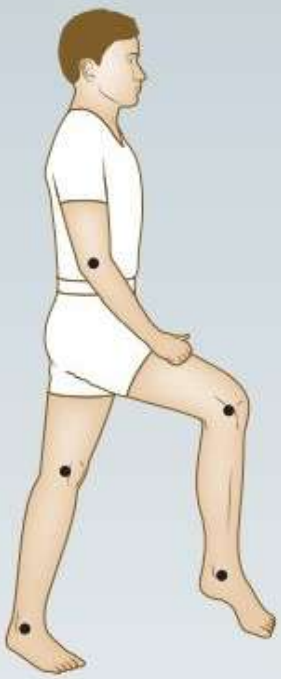
7.7 Common gait abnormalities

Gait disturbance	Description	Causes
Parkinsonian	Stooped posture Shuffling (reduced stride length) Loss of arm swing Postural instability Freezing	Parkinson's disease and other Parkinsonian syndromes
Gait apraxia	Small, shuffling steps (*marche à petits pas*) Difficulty in starting to walk/freezing Better 'cycling' on bed than walking	Cerebrovascular disease Hydrocephalus
Spastic	Stiff 'walking-through-mud' or scissors gait	Spinal cord lesions
Myopathic	Waddling (proximal weakness) Bilateral Trendelenburg signs	Muscular dystrophies and acquired myopathies
Foot drop	Foot slapping	Neuropathies Common peroneal nerve palsy L5 radiculopathy
Central ataxia	Wide-based, 'drunken' Tandem gait poor	Cerebellar disease
Sensory ataxia	Wide-based Positive Romberg sign	Neuropathies Spinal cord disorders
Functional	Variable, often bizarre, inconsistent Knees flexed, buckling Dragging immobile leg behind	Functional neurological disorders



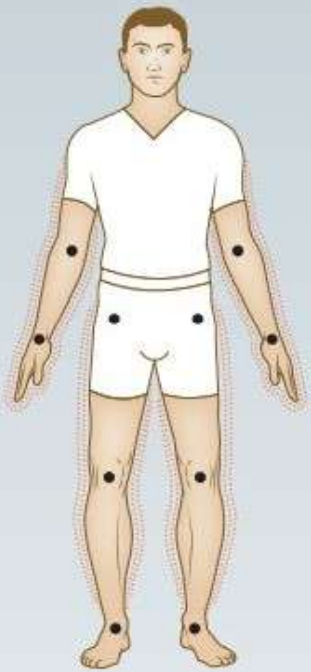
A Spastic hemiparesis

One arm held immobile and close to the side with elbow, wrist and fingers flexed
Leg extended with plantar flexion of the foot
On walking, the foot is dragged, scraping the toe in a circle (circumduction)
Caused by upper motor neurone lesion, e.g. stroke



B Steppage gait

Foot is dragged or lifted high and slapped on to the floor
Unable to walk on the heels
Caused by foot drop owing to lower motor neurone lesion



C Sensory or cerebellar ataxia

Gait is unsteady and wide-based. Feet are thrown forward and outward and brought down on the heels
In sensory ataxia, patients watch the ground. With their eyes closed, they cannot stand steadily (positive Romberg sign)
In cerebellar ataxia, turns are difficult and patients cannot stand steadily with feet together whether eyes are open or closed
Caused by polyneuropathy or posterior column damage, e.g. syphilis

D Parkinsonian gait

Posture is stooped with head and neck forwards
Arms are flexed at elbows and wrists. Little arm swing
Steps are short and shuffling and patient is slow in getting started (festinant gait)
Caused by lesions in the basal ganglia

7.8 Medical Research Council grading of muscle power

Grade	Description
0	No muscle contraction visible
1	Flicker of contraction but no movement
2	Joint movement when effect of gravity eliminated
3	Movement against gravity but not against resistance
4[a]	Movement against resistance but weaker than normal
5	Normal power

[a]*May be further classified as 4+ or 4–.*

7.9 Nerve and muscle supplies of commonly tested movements

Movement	Muscle	Nerve and root
Shoulder abduction	Deltoid	Axillary C5
Elbow flexion	Biceps[a] Brachioradialis (supinator reflex)[a]	Musculocutaneous C5[a]/6 Radial C6[a]
Elbow extension	Triceps[a]	Radial C7
Wrist extension	Extensor carpi radialis longus	Posterior interosseous C6
Finger extension	Extensor digitorum communis	Posterior interosseous C7
Finger flexion	Flexor pollicis longus (thumb) Flexor digitorum profundus (index and middle fingers) Flexor digitorum profundus (ring and little fingers)	Anterior interosseous C8 Ulnar C8
Finger abduction	First dorsal interosseous	Ulnar T1
Thumb abduction	Abductor pollicis brevis	Median T1
Hip flexion	Iliopsoas	Iliofemoral nerve L1/2
Hip extension	Gluteus maximus	Sciatic L5/S1
Knee flexion	Hamstrings	Sciatic S1
Knee extension	Quadriceps[a]	Femoral L3[a]/4
Ankle dorsiflexion	Tibialis anterior	Deep peroneal L4/5
Ankle plantar flexion	Gastrocnemius and soleus[a]	Tibial S1[a]/2
Great toe extension (dorsiflexion)	Extensor hallucis longus	Deep peroneal L5
Ankle eversion	Peronei	Superficial peroneal L5/S1
Ankle inversion	Tibialis posterior	Tibial nerve L4/5

[a]*Indicates nerve root innervation of commonly elicited deep tendon reflexes.*

7.10 Primitive reflexes

Snout reflex

- Lightly tap the lips. Lip pouting is an abnormal response

Grasp reflex

- Firmly stroke the palm from the radial side. In an abnormal response, your finger is gripped by the patient's hand

Palmomental reflex

- Apply firm pressure to the palm next to the thenar eminence with a tongue depressor. An abnormal response is ipsilateral puckering of the chin

Glabellar tap

- Stand behind the patient and tap repeatedly between their eyebrows with the tip of your index finger. Normally, the blink response stops after three or four taps

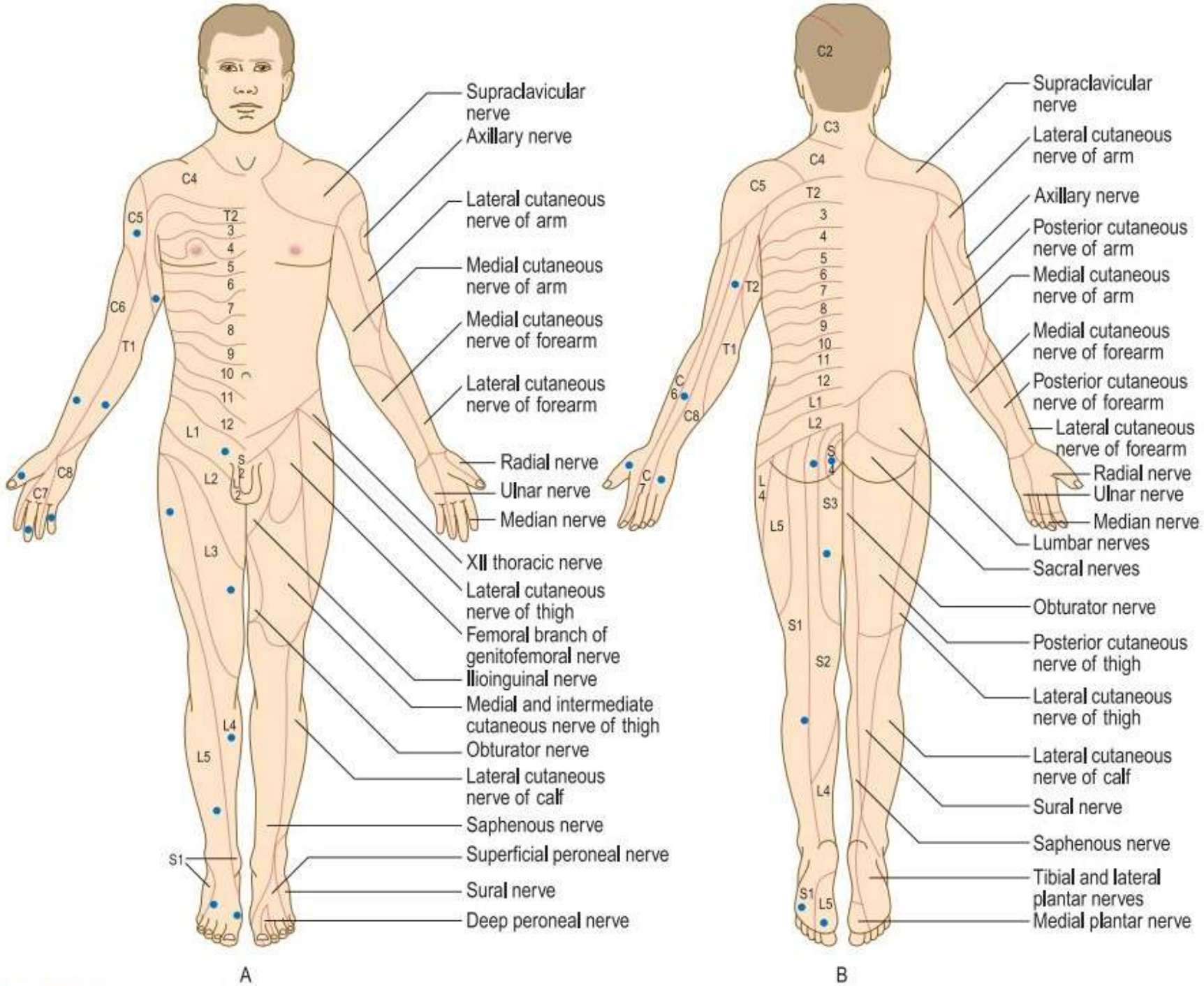


Fig. 7.26 Dermatomal and sensory peripheral map innervation. Points (shown in blue) for testing cutaneous sensation of the limbs. By applying stimuli at the points marked, both the dermatomal and main peripheral nerve distributions are tested simultaneously. **A** Anterior view. **B** Posterior view.

7.11 Common features of carpal tunnel syndrome

- It is more common in women
- There is unpleasant tingling in the hand
- It may not observe anatomical boundaries, radiating up the arm to the shoulder
- Weakness is uncommon; if it does occur, it affects thumb abduction
- Symptoms are frequently present at night, waking the patient from sleep
- The patient may hang the hand and arm out of the bed for relief
- There is thenar muscle wasting (in longstanding cases)
- It is commonly associated with pregnancy, diabetes and hypothyroidism

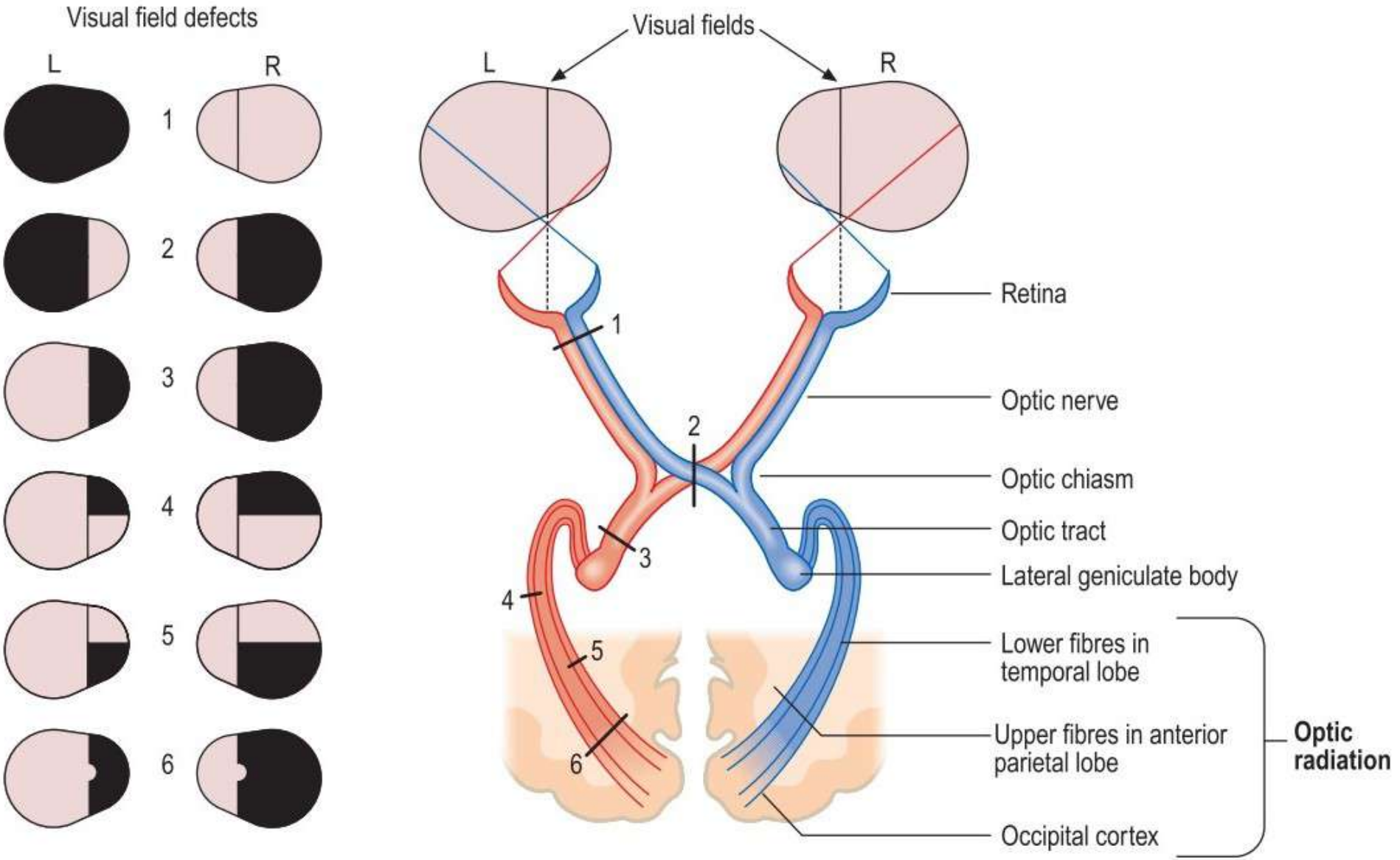


Fig. 8.5 Visual field defects. 1, Total loss of vision in one eye because of a lesion of the optic nerve. **2,** Bitemporal hemianopia due to compression of the optic chiasm. **3,** Right homonymous hemianopia from a lesion of the optic tract. **4,** Upper right quadrantanopia from a lesion of the lower fibres of the optic radiation in the temporal lobe. **5,** Lower quadrantanopia from a lesion of the upper fibres of the optic radiation in the anterior part of the parietal lobe. **6,** Right homonymous hemianopia with sparing of the macula due to a lesion of the optic radiation in the occipital lobe.

8.8 Causes of eyelid ptosis

Cause	Diagnosis	Associated distinguishing features
Neurogenic	Horner's syndrome	Ptosis, miosis, eye movement spared
	Cranial nerve III palsy	Dilated pupil, eye movements affected (see Fig. 8.10)
Myogenic	Myotonic dystrophy	Frontal balding, sustained handgrip
	Chronic progressive external ophthalmoplegia	Bilateral ptosis and impairment of eye movements, often without diplopia, sparing of pupil reflexes
	Oculopharyngeal dystrophy	History of swallowing abnormalities
Neuromuscular junction	Myasthenia gravis	History of variable muscular fatigue
Mechanical	Eyelid tumour	Evident on inspection
	Eyelid inflammation/ infection	Evident on inspection
	Trauma	Scarring/history of trauma
Degenerative	Levator aponeurosis degeneration	Often unilateral, eye movement normal
	Long-term contact lens wear	History of contact lens use

8.9 Causes of anisocoria

Dilated pupil

- Cranial nerve III palsy
- Pharmacological treatment with a dilating agent (e.g. tropicamide or atropine)
- Physiological
- Post-surgical
- Adie's tonic pupil

Constricted pupil

- Horner's syndrome
- Mechanical, e.g. secondary to posterior synechiae in iritis or trauma
- Physiological
- Late-stage Adie's tonic pupil
- Pharmacological treatment with a constricting agent (e.g. pilocarpine)

9.5 Tuning fork tests

	Weber test	Rinne test
Bilateral normal hearing	Central	AC > BC, bilateral
Bilateral symmetrical sensorineural loss	Central	AC > BC, bilateral
Unilateral or asymmetrical sensorineural loss LEFT	Louder right	AC > BC, bilateral[a]
Unilateral conductive loss LEFT	Louder left	BC > AC, left AC > BC, right
Bilateral conductive loss (worse on LEFT)	Louder left	BC > AC, bilateral

[a] *Patients with a severe sensorineural loss may have BC > AC due to BC crossing to the other better-hearing cochlea that is not being tested (false-negative Rinne test).*

AC, *air conduction;* BC, *bone conduction.*

19.14 Glasgow Coma Scale

Eye opening	
Spontaneous	4
To speech	3
To pain	2
No response	1
Verbal response	
Orientated	5
Confused: talks in sentences but disorientated	4
Verbalises: words, not sentences	3
Vocalises: sounds (groans or grunts), not words	2
No vocalisation	1
Motor response	
Obeys commands	6
Localises to pain, e.g. brings hand up beyond chin to supraorbital pain	5
Flexion withdrawal to pain: no localisation to supraorbital pain but flexes elbow to nail bed pressure	4
Abnormal flexion to pain	3
Extension to pain: extends elbow to nail bed pressure	2
No response	1

Record the GCS as a total and its three separate components: e.g. GCS 9/15: E3, V2, M4

11.20 Definitions of paralysis

Term	Definition
Paresis	Partial paralysis
Plegia	Complete paralysis
Monoplegia	Involvement of a single limb
Hemiplegia	Involvement of one-half of the body
Paraplegia/diplegia	Paralysis of the legs
Tetraplegia	Paralysis of all four limbs

11.24 Monosynaptic (deep tendon) reflexes and root innervation

Reflex (muscle)	Nerve root
Biceps	C5
Supinator (brachioradialis)	C6
Triceps	C7
Knee (quadriceps)	L3, 4
Ankle (gastrocnemius, soleus)	S1

2. Symptoms and definitions

Paraesthesia	Tingling, or pins and needles Spontaneous or provoked Not unduly unpleasant or painful
Dysaesthesia	Unpleasant paraesthesia
Hypoaesthesia	Reduced sensation to a normal stimulus
Analgesia	Numbness or loss of sensation
Hyperaesthesia	Increased sensitivity to a stimulus
Allodynia	Painful sensation resulting from a non-painful stimulus
Hyperalgesia	Increased sensitivity to a painful stimulus