

# Neurology in Practice

Fifth Edition

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# **I Approach to Neurological Diagnosis**

Neurology is the branch of medical science which deals with the nervous system in both its normal and diseased states. Clinical neurology is the application of the basic neurosciences, in particular neuroanatomy, neurophysiology, and neurochemistry in patient management.

Most students and practitioners tend to shy away from neurology allegedly because it is perceived to be difficult. In fact, solving a neurological problem can be the most fascinating exercise in detection and logical deduction in clinical medicine. This demands an organized line of thought, a clear plan to be followed, and a specific aim at each stage of the investigation. As long as a proper approach is adopted, neurological diagnosis can be a straightforward and rewarding exercise.

When one approaches a patient with a neurological problem, three vital questions ought to be asked:

1. Where is/are the lesion(s)?
2. What is/are the probable underlying pathological condition(s)?
3. Is the disorder neurological or functional?

## **History**

History taking is not a haphazard activity; it should focus on the three questions. With care, the diagnosis can be made from the history alone in many cases. In others, the history will direct one to focus on certain aspects of neurological examination. This is important, since the patient may not be able to cooperate if one pursues every fine detail of a full neurological examination. In certain diseases, such as epilepsy and headache, the history is crucial for the diagnosis because physical examination and investigation are often negative.

Relatives or eyewitnesses should be interviewed as far as possible since many patients may not be aware of the incident and symptoms, or are unable to give a full history because of impaired cognition and/or dysphasia.

The history can be unnecessarily lengthy if there is no emphasis, but details should be obtained in relevant areas. The following items should be covered:

- Details of the presenting symptom
- Mode of onset: acute, subacute, insidious
- Duration
- Course of illness: static, intermittent, progressive
- Associated symptoms: positive and negative
- Possible causes or risk factors of the disease
- Psychological aspects
- Functional status: how well the patient copes with the disability
- Family history
- Social (including occupational) history

## **Physical examination**

After history taking, one should have a good idea as to which functional aspects of the nervous system are affected, and detailed examination must be directed to the relevant areas. The examination will serve to confirm the diagnosis suggested by the history.

It cannot be over-emphasized that one must be systematic in the neurological examination; otherwise one will get lost or overlook some important tests. A proposed scheme is as follows.

### **General examination**

This includes recognition of abnormal facies and peripheral signs. It may provide clues to the cause, risk factors or associated conditions of the neurological disorder. Examples are:

- Clubbing and lymphadenopathy in cerebral metastasis
- Blood pressure and heart rhythm in syncope
- Goitre and thyroid signs in myopathy and neuropathy
- Skin rash in dermatomyositis
- Nail fold changes in vasculitis

Non-neurological causes of ‘neurological’ symptoms and important co-morbid conditions may also be identified. Examples are:

- Ear and hearing abnormality in patients with dizziness
- Osteoarthritis of the hip in patients with leg weakness

## Neurological examination

Neurological examination begins with observing the patient during history taking. Such observation provides information on higher mental functions. Patient’s own interpretation of symptoms may reveal anxiety, depression, neurosis or delusion.

Components of a practical neurological examination:

- Higher mental functions (relatives’ observations can be very helpful)
  - Assess impaired consciousness using Glasgow Coma Scale (see Table 12.2)
  - Orientation: place, time, person
  - Memory:
    - immediate recall
    - short-term
    - long-term
  - Serial 7: 100-7→93→86→79→72→65
  - Current knowledge
  - Mood
  - Insight
  - Speech
    - Language: ascertain handedness first, then content of speech; dysphasia may be expressive, receptive or global
    - Articulation: dysarthria
  - The Mini-Mental State Examination (MMSE) incorporates many of the above items and is widely used for screening cognitive deficits (see Table 11.2).
- Cranial nerves (see also Chapter 3)
  - I: any change in smell, test each side with aromatic, non-irritant materials
  - II: visual acuity, direct and indirect light reflexes, visual field, fundi
  - III, IV, VI: eye movements in different directions; check for diplopia, nystagmus, and gaze palsy



- V: facial sensation to pinprick and light touch in all three divisions of the trigeminal nerve, corneal reflex, power of jaw opening and closure, jaw jerk
- VII: facial symmetry, UMN and LMN facial weakness
- VIII: hearing acuity, Weber's and Rinne's test (256 Hz tuning fork)
- IX, X: any hoarseness of voice, symmetry of palatal movements, gag reflex
- XI: power of sternomastoid and trapezius
- XII: any deviation, wasting or fasciculation of tongue
- Motor examination of upper and lower limbs
  - Muscle bulk, tone, power (Tables 1.1 and 1.2), tendon reflexes, plantar response, coordination, gait
  - Differentiate between UMN and LMN signs
  - Segmental levels for reflexes: biceps (C5–6), supinator (C5–6), triceps (C7–8), finger jerks (C8–T1), knee (L3–4), ankle (S1–2)
- Sensations of upper and lower limbs
  - Pain, temperature, vibration (128 Hz tuning fork), joint position
  - Recognize pattern of sensory loss: peripheral nerve vs dermatome (Figure 1.1)
  - C5–T1 dermatomes over the upper limb: shoulder (C5), thumb (C6), middle finger (C7), little finger (C8), inner-upper arm (T1)
  - C4 and T2 are contiguous over sternal angle
  - Over the trunk: nipple (T4), xiphisternum (T7), umbilicus (T10), symphysis pubis (L1)
  - L2–S2 over the lower limb: upper outer thigh (L2), lower inner thigh (L3), inner lower leg (L4), anterior lower leg and foot (L5), lateral lower leg (S1), mid-strip of leg posteriorly (S2)
  - S3 over saddle region
  - S4–5 over perianal region

**Table 1.1 Segmental innervation of upper limb muscles**

Region	Muscle	C4	C5	C6	C7	C8	T1
Shoulder	Supraspinatus	✓	✓				
	Teres minor	✓	✓				
	Deltoid	✓	✓	✓			
	Infraspinatus	✓	✓	✓			
	Subscapularis		✓	✓			
	Teres major		✓	✓	✓		
Arm	Biceps		✓	✓			
	Brachialis		✓	✓			
	Coracobrachialis		✓	✓	✓		
	Triceps			✓	✓	✓	
	Anconeus				✓	✓	
Forearm	Supinator longus		✓	✓			
	Supinator brevis		✓	✓			
	Extensor carpi radialis			✓	✓		
	Pronator teres			✓	✓		
	Flexor carpi radialis			✓	✓		
	Flexor pollicis longus			✓	✓	✓	
	Abductor pollicis longus			✓	✓	✓	
	Extensor pollicis brevis				✓	✓	✓
	Extensor pollicis longus			✓	✓	✓	
	Extensor digitorum communis			✓	✓	✓	
	Extensor indicis			✓	✓	✓	
	Extensor carpi ulnaris			✓	✓	✓	
	Extensor digiti minimi			✓	✓	✓	
	Flexor digitorum superficialis				✓	✓	✓
	Flexor digitorum profundus				✓	✓	✓
	Pronator quadratus				✓	✓	✓
	Flexor carpi ulnaris				✓	✓	✓
	Palmaris longus				✓	✓	✓
Hand	Abductor pollicis brevis				✓	✓	✓
	Flexor pollicis brevis				✓	✓	✓
	Opponens pollicis				✓	✓	
	Flexor digiti minimi				✓	✓	✓
	Opponens digiti minimi				✓	✓	✓
	Adductor pollicis					✓	✓
	Palmaris brevis					✓	✓
	Abductor digiti minimi					✓	✓
	Lumbricals					✓	✓
	Interossei					✓	✓

**Table 1.2 Segmental innervation of lower limb muscles**

Region	Muscle	L1	L2	L3	L4	L5	S1	S2	S3
Hip	Iliopsoas	✓	✓	✓	✓	✓			
	Tensor fascia latae				✓	✓			
	Gluteus medius				✓	✓	✓		
	Gluteus minimus				✓	✓	✓		
	Quadratus femoris				✓	✓	✓		
	Gluteus maximus				✓	✓	✓		
	Obturator internus					✓	✓		
	Piriformis					✓	✓		
Thigh	Sartorius		✓	✓					
	Pectineus		✓	✓					
	Adductor longus		✓	✓					
	Quadriceps		✓	✓	✓				
	Gracilis		✓	✓	✓				
	Adductor brevis		✓	✓	✓				
	Obturator externus			✓	✓				
	Adductor magnus			✓	✓				
	Adductor minimus			✓	✓				
	Articularis genus			✓	✓				
	Semitendinosus				✓	✓	✓		
	Semimembranosus				✓	✓	✓		
	Biceps femoris				✓	✓	✓	✓	
Leg	Tibialis anterior				✓	✓			
	Extensor hallucis longus				✓	✓	✓		
	Popliteus				✓	✓	✓		
	Plantaris				✓	✓	✓		
	Extensor digitorum longus				✓	✓	✓		
	Soleus				✓	✓	✓	✓	
	Gastrocnemius				✓	✓	✓	✓	
	Peroneus longus					✓	✓		
	Peroneus brevis					✓	✓		
	Tibialis posterior				✓	✓	✓	✓	
	Flexor digitorum longus					✓	✓	✓	✓
	Flexor hallucis longus					✓	✓	✓	✓
	Extensor hallucis brevis				✓	✓	✓		
Foot	Extensor digitorum brevis				✓	✓	✓		
	Flexor digitorum brevis					✓	✓		
	Abductor hallucis					✓	✓		
	Flexor hallucis brevis					✓	✓	✓	✓
	Lumbricals					✓	✓	✓	✓
	Abductor digiti minimi						✓	✓	✓
	Flexor digiti minimi brevis						✓	✓	✓
	Opponens digiti minimi						✓	✓	✓
	Quadratus plantae						✓	✓	✓
	Interossei						✓	✓	✓

## Cardiovascular system

- Pulse
- Blood pressure
- Heart sounds and murmurs
- Arterial bruit

## Respiratory system

## Abdomen

## Diagnosis

Upon completion of the examination, it should be possible to arrive at the diagnosis in most cases. There are two stages in the diagnosis: anatomical and pathological.

## Anatomical diagnosis

The lesion(s) may be:

- Single, e.g., tumour in the brainstem
- Two or more but discrete, e.g., optic nerve and spinal cord lesions as in multiple sclerosis
- Diffuse, e.g., neurodegenerative disease or viral encephalomyelitis

Anatomical localization applies to single or multiple discrete lesions. The sites of the central (brain and spinal cord) and peripheral nervous systems are:

- |               |   |                      |                                      |
|---------------|---|----------------------|--------------------------------------|
| • Brain       | { | Cerebral hemispheres | } Anterior and middle cranial fossae |
|               |   | – dominant           |                                      |
|               |   | – non-dominant       |                                      |
| • Brain       | { | Brainstem            | } Posterior cranial fossa            |
|               |   | – midbrain           |                                      |
|               |   | – pons               |                                      |
|               |   | – medulla oblongata  |                                      |
|               |   | Cerebellum           |                                      |
| • Spinal cord |   |                      |                                      |
| • Spinal root |   |                      |                                      |

- Plexus – brachial, lumbosacral
- Peripheral nerve
- Neuromuscular junction
- Muscle

The clinical features and relevant investigations for localization are tabulated (Tables 1.3–1.10). See also Chapter 18 for features of cortical dysfunction.

**Table 1.3 Hemisphere lesion**

---

**Clinical features**

Impaired mentation, dysphasia (dominant), dyspraxia (non-dominant)  
 Homonymous visual field defects  
 Contralateral UMN facial weakness, dysarthria  
 Contralateral UMN limb weakness  
 Contralateral sensory disturbance  
 Conjugate gaze deviation towards lesion  
 Focal seizures

**Investigations**

MRI or CT brain, EEG

---

A UMN lesion refers to a lesion either at the cortex or corticobulbar/corticospinal tract. This may give rise to contralateral facial weakness with relative sparing of the upper facial muscles which are innervated bilaterally. The UMN lesion may also give rise to contralateral limb weakness with spasticity and exaggerated tendon reflexes.

**Table 1.4 Posterior fossa lesion**

---

**Clinical features**

Cranial nerve deficits  
 Bilateral or unilateral UMN limb weakness  
 Bilateral or unilateral sensory disturbance  
 Cerebellar ataxia, nystagmus  
 Conjugate gaze deviation away from lesion

**Investigations**

MRI or CT brain, BAEP

---

NB: Signs of raised intracranial pressure tend to appear early for mass lesions in the posterior fossa.

**Table 1.5 Spinal cord lesion**

---

**Clinical features**

UMN limb weakness below lesion – unilateral or bilateral  
LMN limb weakness at level of lesion – unilateral or bilateral  
Pattern of sensory deficits – level, glove and stocking, suspended,  
dissociated  
Sphincter disturbance

**Investigations**

MRI spine\*, XR spine, SEP, CSF analysis

---

\*If MRI not available, myelogram or CT myelogram is an alternative.

A LMN lesion refers to a lesion of the motor neurone or its axons. Depending on the site (brainstem or spinal cord), it may give rise to ipsilateral facial weakness affecting the upper and lower facial muscles or ipsilateral limb weakness with hypotonia and reduced or absent tendon reflexes.

**Table 1.6 Spinal root lesion**

---

**Clinical features**

Segmental LMN weakness and sensory deficits  
Autonomic disturbance

**Investigations**

NCS, EMG, SEP, XR spine, MRI spine\*, CSF analysis

---

\*If MRI not available, myelogram or CT myelogram is an alternative.

**Table 1.7 Plexus lesion**

---

**Clinical features**

Multi-segmental LMN weakness and sensory deficits  
Autonomic disturbance

**Investigations**

NCS, EMG, SEP, MRI, CSF analysis

---

**Table 1.8 Peripheral nerve lesion**

<b>Clinical features</b>	
LMN weakness	Patterns – symmetrical sensorimotor mononeuritis multiplex mononeuropathy
Sensory deficits	
Autonomic disturbance	
<b>Investigations</b>	
NCS, EMG, CSF analysis, nerve biopsy	

**Table 1.9 Neuromuscular junction lesion**

<b>Clinical features</b>
Weakness without wasting – generalized or focal
Fatiguability or post-exertion reinforcement
Autonomic disturbance
<b>Investigations</b>
Tensilon test, Anti-AChR, CK, thyroid function, repetitive stimulation

**Table 1.10 Myopathy**

<b>Clinical features</b>
Weakness, wasting or pseudohypertrophy, generalized or in groups
Muscle pain
Tendon reflexes may be normal or reduced
<b>Investigations</b>
CK, immune markers, muscle biopsy

## **Pathological diagnosis**

Having made the anatomical diagnosis, the pathological diagnosis has to be postulated. The clues are the mode of onset, duration, and progression of symptoms. For example, a vascular lesion is usually of sudden onset and reaches the maximum intensity within a short time. A tumour is usually of insidious onset and progressive deterioration. Other clues are also helpful. For example, a familial history would indicate a hereditary aetiology. A pre-existing collagen disease would suggest an autoimmune basis of the lesion. It is often helpful to go through the list of likely pathology:

- Congenital
- Hereditary

- Inflammatory: infective, granulomatous, autoimmune
- Demyelinating
- Vascular
- Degenerative
- Neoplastic: benign, malignant
- Traumatic
- Idiopathic

## **Investigations**

After a clinical diagnosis has been arrived at, the next steps are to confirm it and to obtain more information in order to plan the management. Investigations (Tables 1.3–1.10) are organized with these aims in mind. To be cost-effective, they should be specific and relevant. Such targeting can only be achieved with a sound clinical diagnosis.

## **Common neurological symptoms and their differential diagnosis**

**Headache** (see Chapter 4)

**Facial pain** (see Chapter 3)

### **Dizziness**

This is a common symptom which may be due to systemic or vestibular disturbance. Distinction has to be made from loss or lapse of consciousness. Associated symptoms of vertigo and disequilibrium, if present, should be elicited.

#### *Systemic disturbance*

presyncope

hyperventilation

anaemia

cardiac arrhythmia

drug-induced, e.g., bromocriptine, levodopa, methyl dopa

electrolyte disturbance, e.g., hyponatraemia



*Vestibular disturbance**Peripheral*

labyrinthitis

Meniere's disease

*Central*

brainstem/cerebellar lesion

vertebrobasilar stroke

cerebello-pontine angle tumours

demyelination

**Altered consciousness**

This state includes complete and partial, prolonged and brief disturbance of consciousness. Details of relevant features during the episode should be obtained from the patient or relatives. Many causes of impaired consciousness apply (see Chapter 12).

epileptic seizures, generalized or complex partial  
stroke

cardiogenic syncope

metabolic/endocrine, especially hypoglycaemia

intoxication/drug overdose, e.g., alcohol, substance abuse

neoplastic

functional disorders

**Visual impairment** (see Chapter 3)**Ptosis***Unilateral*

III nerve palsy

Horner's syndrome

MG

*Bilateral*

MG

myopathy

**Diplopia** (see Chapter 3)

**Deafness** (see Chapter 3)

**Dysphagia**

*Structural lesion (symptoms worse with solid food)*  
oesophageal carcinoma or stricture

*Impaired neural control (symptoms worse with fluid)*  
bulbar palsy (see Chapter 3)  
pseudobulbar palsy, e.g., stroke, MND  
neuromuscular junction, e.g., MG, botulism  
neuropathy, e.g., AIDP

**Tremors** (see Chapter 8)

**Gait disturbance in the absence of limb weakness**

Apraxic: diffuse cerebral disease  
subcortical ischaemia or demyelination  
normal pressure hydrocephalus  
Ataxic: cerebellar lesion especially midline  
loss of joint position sense, e.g., neurosyphilis, sub-  
acute combined degeneration of cord, diabetic  
neuropathy  
Shuffling: parkinsonism

**Sensory disturbance in extremities**

peripheral neuropathy  
cervical myelopathy  
functional disorders

**Simulated neurological manifestations**

It is not uncommon for patients with psychogenic disorders to present with various neurological (and/or other somatic) symptoms.

Common simulated neurological features include impaired cognition, amnesia, seizures, blindness, diplopia, aphonia, limb weakness, gait disturbance, tics, tremor, dystonia, pain, and paraesthesia. In these cases, a neurological explanation of the symptoms and signs cannot be found even after thorough examination and investigation. The patient may derive some primary gain from expressing the suppressed unconscious conflict. Avoidance of unpleasant situations, compensation issues, and undue attention from family and carers may constitute secondary gain. A comprehensive psychosocial history from friends and family is essential.

The diagnosis of psychogenic illnesses should only be made after all reasonable steps have been undertaken to exclude organic disorders. Misdiagnosing an organic condition for a psychogenic illness may have serious consequences such as negligence claims (see Chapter 20). Repeated visits may be required before the diagnosis can be made. Do not rush into invasive diagnostic tests or potentially harmful therapeutic interventions as these may bring about more complicated problems. A trial of physiotherapy, psychotherapy, anxiolytic, or antidepressant may be helpful. Consider referral to psychologists or psychiatrists.

The following clues suggest the manifestations are psychogenic in nature:

- The pattern of deficits does not conform to neuroanatomy or neurophysiology.
- Multiple somatic complaints
- Findings on formal examination are inconsistent with functional observation, which is especially useful when the patient is unaware of being observed.
- Variable findings in different examinations

#### Psychogenic limb weakness

- No muscle wasting or atrophy
- Normal reflexes and tone
- Give-way weakness or fluctuating weakness
- Extensor and flexor muscles equally weak
- Simultaneous contraction of the agonist and antagonist muscles when executing a movement

### Psychogenic movement disorders

- Abrupt onset, atypical pattern, paroxysmal symptoms
- Inconsistency over time
- Entrainment of tremor to the examiner's suggested rate
- Spontaneous remissions
- Disappearance when distracted
- Aggravation during formal examination
- Response to placebo, psychotherapy or suggestion

### Psychogenic seizure (see Chapter 7)

Always bear in mind the following:

- Organic and psychogenic disorders may coexist.
- Delirium and dementia are due to organic disorders.
- Cerebrovascular disease, brain tumour, epilepsy, Parkinson's disease, Alzheimer's disease, multiple sclerosis, and Huntington's disease can produce anxiety and depression.
- Substance misuse (e.g., alcohol, recreational drugs) can lead to psychogenic and neurological complications.
- Psychosomatic disorders consist of hysteria, somatization disorders, somatoform pain disorders, and neurasthenia. They commonly present with fatigue, dizziness, headache or pain attributed to physical illness.
- A patient with factitious disorder repeatedly induces the symptoms or signs of disease in the absence of any psychiatric or physical disorder.

# 14 Spinal Cord Disorders

Spinal cord disorders or myelopathies frequently cause severe and permanent disability because the spinal cord contains the entire motor and sensory systems of the trunk and limbs. Therefore, prompt diagnosis and treatment are essential. In particular, acute spinal cord compression is a neurological emergency.

## **Classification**

Spinal cord disorders may be classified according to their causes. Trauma is the most common cause of myelopathy. For acute non-traumatic myelopathies, transverse myelitis and cord compression are common whereas spinal cord stroke is rare. In chronic non-traumatic myelopathies, spondylosis and benign tumours account for most cases. Syringomyelia, multiple sclerosis, degenerative and paraneoplastic cord lesions should be considered in the differential diagnosis. Subacute combined degeneration of spinal cord and syphilitic myelitis are rare but treatable entities.

## **Congenital and developmental disorders**

Examples are syringomyelia, Arnold-Chiari malformation, and diastematomyelia. These conditions are often associated with spinal bifida.

## **Infection**

Neurotropic viruses, e.g., herpes virus, poliovirus, and rabies may affect the spinal cord and/or the brain. The virus, however, cannot be identified in most cases.

Pyogenic infections of leptomeninges usually affect the sub-arachnoid space around the brain and the spinal cord. Epidural abscess may result from direct or haematogenous spread.

Tuberculous leptomeningitis produces spinal cord ischaemia by inflammatory arteritis. Tuberculous spinal osteitis (Pott's disease), with its associated psoas abscess and bony destruction, may cause cord compression or ischaemia.

Fungal infections (e.g., cryptococcosis) and parasitic infections (e.g., cysticercosis and hydatid disease) are rare in the spinal cord.

### **Neoplastic diseases**

The lesion may be extradural, intradural-extramedullary, or intramedullary in location. The common benign lesions are neurofibroma and meningioma. The malignant lesions are ependymoma, astrocytoma, and carcinomatous meningeal infiltration. Related entities are paraneoplastic and irradiation myelopathy.

### **Diseases of the spine**

- Spondylosis (*vide infra*)
- In longstanding rheumatoid arthritis, atlantoaxial or subaxial dislocation may cause cervical cord compression.

### **Vascular disorders**

Diseases of the aorta, vertebral, intercostal, and radicular arteries may present as cord transection or anterior spinal artery syndrome. Acute spinal venous obstruction is rare and usually secondary to conditions such as tumour, septicaemia, and thrombotic diathesis; the prognosis is poor because of extensive haemorrhagic infarction. Vasculitis, e.g., in SLE, PAN, may give rise to acute spinal cord symptoms. AVM may either present with acute or chronic spinal cord features.

## **Demyelination**

- Multiple sclerosis
- Post-infectious encephalomyelitis

## **Degenerative**

- Spinocerebellar degeneration
- Idiopathic spastic paraparesis

## **Trauma** (see Chapter 20)

## **Clinical picture and diagnosis**

The clinical features depend on the pattern and level of the lesion (Figure 14.1).

### ***Motor deficits***

- UMN weakness below the lesion
- LMN weakness at the level of the lesion
- Mixed LMN and UMN weakness, e.g., upper limbs in cervical cord lesion
- Spinal shock stage: flaccid paralysis

### ***Sensory disturbance***

- Spinothalamic sensations: pain, temperature
- Posterior column sensations: vibration, joint position
- Sensory disturbance may present as a sensory level, glove and stocking distribution, segmental sensory loss, or dissociated sensory loss.
- Pain may arise from vertebral collapse or malignant infiltration of spinal root (radicular pain) or spinal cord.

**Autonomic dysfunction**

- Bladder: spastic or paralytic
- Bowel: incontinence  $\pm$  constipation
- Sexual dysfunction

**Differential diagnosis**

- Guillain-Barré syndrome may present as a rapidly evolving tetraparesis. Distinguishing features include the presence of bulbar palsy, paresis of extraocular muscles and areflexia.
- Acute neuropathies: usually due to vasculitis or toxin.
- Myasthenia gravis: no sensory disturbance; ocular involvement is common.
- Acute occlusion of the terminal aorta may cause ischaemia of the cauda equina or proximal sciatic and femoral nerves. Rarely the spinal cord may be involved. Pale cold skin and loss of pulses in the lower limbs are clues to the diagnosis.
- Occlusion of anterior cerebral artery may cause bilateral infarction of the paracentral lobule, resulting in acute paraplegia, sphincter dysfunction, and loss of position sense with intact pain perception. The last feature distinguishes this entity from acute spinal cord syndrome.
- Falx meningioma with pressure effect on the leg areas of the cerebral cortex

**Specific investigations**

Investigations are guided by the clinical diagnosis, especially for the spinal level where the lesion is likely to be present.

- X-ray spine: vertebral lesions, e.g., collapse, spondylosis
- MRI spine: investigation of choice since the cord, subarachnoid space, and adjacent tissues can be clearly visualized. CT spine or myelogram may be indicated if MRI is not available.
- CSF analysis: useful in infection, demyelination, and neoplastic meningitis

NB: LP may cause further deterioration if there is cord compression.



## **SELECTED ENTITIES**

### **Acute spinal cord compression**

It is a neurological emergency caused by:

- Malignancy: carcinomatous metastasis, lymphoma, myeloma
- Infection: tuberculous or pyogenic abscess, vertebral collapse
- Epidural haematoma: spontaneous, traumatic
- Acute disc protrusion

Urgent MRI is indicated to confirm or exclude spinal cord compression.

High-dose IV steroids can be given prior to confirmation with neuroimaging. Upon confirmation, urgent neurosurgical decompression or radiotherapy should be arranged. Delay of treatment is associated with poor recovery of function.

### **Transverse myelitis**

The causes include viral infection, SLE, post-infectious demyelination, NMOSD, and MS. Note any history of viral infection or vaccination and previous episodes of neurological deficits. Acute spinal cord compression must be excluded.

MRI is indicated to visualize the lesion and exclude cord compression.

CSF analysis commonly shows lymphocytosis, normal glucose, normal or slightly raised protein. Oligoclonal IgG indicates intrathecal synthesis of IgG; it is a non-specific finding although commonly present in demyelination.

Treatment depends on the underlying cause.

## **Degenerative disease of the spine**

If the spinal canal is constitutionally narrow, degenerative tissues of the spine (e.g., osteophytes, discs, ossified posterior longitudinal ligament, hypertrophied ligamentum flava) are more likely to cause compression of the spinal cord or roots.

### **Cervical spondylotic myelopathy (CSM)**

Cervical spondylosis as a radiological feature is very common, and CSM is the most common cause of cervical cord lesion in subjects over age 50.

Males are more affected than females. A history of neck injury, neck pain, and stiffness can be elicited in some patients. The symptomatology is that of a cervical cord lesion, with or without spinal root lesion.

The mechanisms by which cervical spondylosis brings about cord damage are complex. Compression due to acquired spondylotic changes on top of a constitutionally narrow canal (sagittal diameter < 10 mm) is the most important factor. In addition, dynamic factors, viz. friction with osteophytes, pressure from the ligamentum flava, vertebral subluxation, and hyperextension injury also play a part. These mechanisms lead to impairment of microcirculation at the arteriolar level resulting in ischaemic cord damage. The differential diagnosis is that of other cervical cord lesions, e.g., neurofibroma, syringomyelia. Motor neurone disease, at its early stage, may mimic CSM.

Cervical spine radiography provides information on the sagittal diameter of the spinal canal, the presence and degree of spondylotic tissues, and the presence of vertebral subluxation. MRI (Figure 14.2) shows the degree and site of spinal cord and root compression and is the investigation of choice. SEP study may document posterior column deficits and is helpful in monitoring progress.

Treatment is conservative for patients with mild symptoms and/or a long history with little or no progression. However, surgery is indicated for patients with significant symptomatology or progression, and for those in whom conservative treatment has failed.

### **Cauda equina compression by lumbar prolapsed intervertebral disc (LPID)**

LPID is common but in most cases, it either causes compression of a single spinal root (e.g., sciatica) or no neurological damage. Central protrusion of the disc into the spinal canal occurs uncommonly but may lead to acute or subacute cauda equina compression. It occurs more often at L4/5 and L5/S1 levels, hence the lowermost lumbar roots and sacral roots are affected.

The clinical picture is typical, and features include low back pain with or without radiation to both lower limbs; reduced sensation in the buttocks, perineum, and posterior thighs; absent ankle jerks; and sphincter disturbances with urinary retention, constipation and erectile dysfunction. Weakness in the lower limbs, especially of foot movements, may be present.

Diagnosis is made on clinical grounds and confirmed by MRI. Early diagnosis and prompt surgical decompression may partially or completely reverse the deficits.

### **Intermittent claudication of cauda equina**

The underlying pathology is stenosis of the lumbar spinal canal, often due to spondylotic tissues superimposed on a constitutionally narrow canal.

The clinical features consist of weakness, reduced tendon reflexes, and sensory disturbance in the legs after walking a certain distance. At rest, these features may be absent. The differential diagnosis is peripheral vascular disease in which case the pulses in the legs are weak or absent.

X-ray of lumbar spine shows the degenerative bony changes and alignment. MRI confirms the diagnosis and provides information for surgical management.

Surgical decompression is indicated for patients with severe or progressive symptomatology.

## **Multiple sclerosis** (see Chapter 9)

### **Syringomyelia**

This is a classic example of an intramedullary lesion, with cavitation of the central part of the spinal cord, often extending vertically in the central grey matter over many segments. It presents with segmental sensory impairment of spinothalamic sensations due to disruption of decussating fibres at the anterior commissure. Extension of the syrinx to the anterior horns causes segmental amyotrophy, whereas extension to the posterior horns causes segmental loss of posterior column sensations. Functional disturbances of the legs and sphincters occur at a late stage.

Syringobulbia is due to extension of syrinx to the brainstem, resulting in lower cranial nerve palsy and disturbance of facial sensations.

About half of all idiopathic cervical syringomyelia cases are associated with type I Arnold-Chiari malformation in which the neck may be short and congenital abnormalities of the cervical spine and base of skull may be present. Secondary syringomyelia may complicate obstruction of the foramen magnum by localized arachnoiditis, cyst or tumour.

X-ray cervical spine and base of skull may show skeletal abnormalities. In MRI cervical spine (Figure 14.3), the location and extent of the syrinx, as well as cerebellar tonsillar herniation, can be visualized. MRI brain shows syringobulbia and other associated abnormalities.

Decompression of the syrinx is the treatment for symptomatic patients, particularly for those with Arnold-Chiari malformation.

## **Spinocerebellar ataxia (SCA)**

In most cases, including the SCA type 3, the inheritance is autosomal dominant. Sporadic cases are far less common. The symptoms start in the 30s or earlier. The progression is usually slow, and disability is moderate to severe. Degeneration of corticospinal and spinocerebellar tracts results in cerebellar ataxia and pyramidal signs. Associated features are uncommon and include peripheral neuropathy, optic neuropathy, cardiomyopathy, and cardiac arrhythmia. Cognition is intact. There is no curative treatment. Genetic diagnosis (including preimplantation) for the affected family is available.

## **Subacute combined degeneration of spinal cord**

This is a classical and treatable condition due to vitamin B<sub>12</sub> deficiency which may occur in pernicious anaemia, dietary insufficiency, and gastric or ileal resection. The pathological changes are degeneration of the lateral and posterior columns of the spinal cord, and to a lesser extent the brain and peripheral nerves. The patient presents with spastic tetraparesis, impaired posterior column sensations, peripheral neuropathy, and occasionally encephalopathy.

The diagnosis is made upon confirmation of vitamin B<sub>12</sub> deficiency (e.g., megaloblastic anaemia, low serum B<sub>12</sub>) and exclusion of other spinal cord lesions. The cause of vitamin B<sub>12</sub> deficiency should also be elucidated.

Treatment is by parenteral vitamin B<sub>12</sub> replacement. Hypokalaemia may develop during treatment. Early treatment confers a better chance of complete recovery.

## **Paraneoplastic myelopathy**

This is not caused by spinal cord compression or invasion by carcinoma. The clinical syndrome is a rapidly developing non-inflammatory myelopathy with motor and sensory dysfunction.

Pathologically, there is necrosis of the tracts of the spinal cord without evidence of neoplasm. Anti-tumour antibody cross-reactive to the spinal cord has been proposed as the mechanism.

## **Irradiation myelopathy**

The lesion evolves over a period of weeks and then becomes permanent. The total dose, fractionation, and size of radiated field are important factors. In general, fractionated irradiation up to a total dose of 3,500 rad is relatively safe. Irradiation induces obliterative endarteritis and thus ischaemic necrosis of the spinal cord tissues. The time interval between irradiation and the first spinal cord symptom varies, but usually ranges from 6–48 months. CSF is either normal or shows a slightly elevated protein level.

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