CNS -1- LOWER LIMB
STEPS OF EXAMINATION

(1) APPROACH THE PATIENT

- Read the instructions carefully for clues
- Approach the right hand side of the patient, shake hands, introduce yourself
- Ask permission to examine him
- Expose the lower limbs completely and keep the patient descent (genital area is covered)

(2) GENERAL INSPECTION

STEPS	POSSIBLE FINDINGS
1. Scan the **bedside**.	➢ Walking stick, shoes-callipers, built-up heels
2. **Scan the patient**.	➢ Nutritional status: under/average built or overweight ➢ Abnormal facies: Sad, immobile, unblinking facies (Parkinson's disease), facial wasting (muscular dystrophy), facial asymmetry (hemiplegia) ➢ Abnormal movement or posture: rest or intention tremors, dystonia, choreoathetosis, hemiballismus, myoclonic jerks, tics, pyramidal posture…*see theoretical notes for description, types, features and causes of abnormal movements* ➢ Abnormal facial movements: hemifacial spasm, facial myokymia, blepharospasm, oro-facial dyskinesia ➢ Scar or deformity underlying an ulnar nerve palsy ➢ Peroneal wasting (Charcot-Marie-Tooth disease) ➢ Pes cavus (Friedreich's ataxia, Charcot-Marie-Tooth disease)
3. Examine the **eyes**	➢ Nystagmus (cerebellar syndrome) ➢ Horner's syndrome (syringomyelia, Pancoast's syndrome)
4. Examine the **hands**: tell the patient "outstretch your hands like this (dorsum facing upwards)"… then "like this (palms facing upwards)"… demonstrate. Feel the radial pulse for AF	➢ wasted hands (MND, Charcot-Marie-Tooth disease, syringomyelia) ➢ AF → consider thromboembolic complications

(3) INSPECTION OF THE LOWER LIMB

Steps	Possible findings
1. Scan the lower limb. 2. Note specifically the quadriceps, the anterior compartment of the shin, the extensor digitorum brevis, and the peroneal muscles. 3. Compare right with left	➢ **Muscle bulk:** Look specifically for: ▪ Generalized disuse atrophy (e.g. severe spastic paraparesis) ▪ Unilateral loss of muscle bulk (old polio) ▪ Distal muscle wasting with preservation of the thigh muscle bulk (inverted champagne bottle) → Charcot-Marie-Tooth disease ▪ Isolated anterior thigh wasting (e.g. diabetic amyotrophy) ▪ Proximal muscle wasting (polyomyositis) ▪ Wasting confined to one peroneal region (lateral popliteal = common peroneal nerve palsy) ➢ **Fasciculations**: irregular twitches under the skin overlying muscle at rest; represent contraction of a motor unit. They occur in LMNL; usually in wasted muscles (nearly always indicate MND). Flicking the skin over wasted muscle may elicit fasciculations. Non-pathological fasciculations occasionally occurs after vigorous exercise in healthy people.

(4) TONE

Steps	Possible findings
1. Tell the patient "Let your legs go loose and let me move them for you" 2. Roll the extended leg on the bed, rotating the **hip** externally and internally (compare right with left) 3. Put your hand behind the **knee** and lift it rapidly (feel for any catch; watch the heel) and let it drops (compare right with left) 4. Hold the knee and ankle. Passively flex and extend the leg at the **knee and hip** joints repeatedly in an irregular and unexpected rhythm (compare right with left)	➢ Normally there is light resistance through whole range of movements. ➢ Normally the heel will lift minimally off the bed when the knee is lifted quickly ➢ *See theoretical notes for abnormalities of the tone*

(5) POWER

Steps	Possible findings
Tell the patient "I am going to test the strength of some of your muscles". Fix the joint proximal to the group of muscles you are testing. Give the patient a space to show his power before resisting him and look at the muscle contracts. Compare right with left	Describe any weakness in terms of the medical research council (MRC) scale from 5 (normal) down to 0 (no visible muscle contraction)…*see theoretical notes for the MRC scale for power* grading
1. **Hip flexion:** tell the patient "keep your leg straight and lift it up into the air. Now keep it up and don't let me push it down"	Action by the iliopsoas (supplied by the femoral nerve & direct branches from the lumbar sacral plexus; **L1,2**)
2. **Hip extension:** tell the patient "now push your leg down into the bed and do not let me stop you"	Action by the glutei (supplied by the inferior gluteal nerve; **L4,5**)
3. **Hip abduction:** tell the patient "push out against my hands"	Action by the glutei (supplied by the superior gluteal nerve; **L4,5**)
4. **Hip adduction:** tell the patient "push in against my hands"	Action by the adductor group (supplied by the obturator nerve; **L2,3,**4)
5. **Knee flexion:** hold the patient's ankle and tell him (her) "bend your knee and pull your heel towards you; don't let me stop you"	Action by the hamstrings (supplied by the sciatic nerve; **L5,S1**,2)
6. **Knee extension**: tell the patient "now straighten your knee out and push my hand"	Action by the quadriceps (supplied by the femoral nerve; **L3,4**)
7. **Ankle dorsiflexion**: tell the patient "pull your foot up to you and push my hand"	Action by the tibialis anterior and long extensors (supplied by the deep peroneal nerve; **L4,5**)
8. **Ankle planter flexion:** tell the patient "push your foot down against my hand"	Action by the gastrocnemius (supplied by the posterior tibial nerve; **S1**)

See theoretical notes for:

- *Features & causes of the different patterns of weakness*
- *Clinical approach to weakness*
- *Motor root values in the lower limb*
- *Examples of mononeuropathies and radiculopathies in the lower limb*

(6) REFLEXES

Steps	Possible findings
Explain to the patient. Use a long tendon hammer; flex your wrist and let the hammer fall with its own weight onto the muscle. Compare right with left. If the reflex appears to be absent, ask the patient to clench his teeth as you swing the hammer (reinforcement).	Grade the response from 0 (absent) up to 4+ (clonus)… *see theoretical notes for reflexes grading*
1. **Knee reflex:** place the arm under the knee so that the knee is at 90 degrees. Strike the knee below the patella; watch the quadriceps	Reflex arc through the **femoral** nerve; **L3,4**
2. **Knee (patellar) clonus:** with the knee extended, sharply push the patella downwards with your thumb and forefinger, sustaining the pressure for a few seconds. Do not examine for knee clonus (as it is not expected to be present) if knee reflex is diminished	A rhythmic contraction may be noted. It is always abnormal and indicates 4+ knee reflex grading
3. **Ankle reflex:** hold foot at 90 degrees with a medial malleolus facing the ceiling. The knee should be flexed and lying to the side. Strike the Achilles tendon directly. Watch the muscles of the calf	Reflex arc through the **tibial** nerve; **S1,2**
4. **Ankle clonus**: support the patient's leg with both the knee and ankle resting in 90 degree flexion. Briskly dorsiflex and partially evert the foot and sustain the pressure. Do not examine for ankle clonus (as it is not expected to be present) if ankle reflex is diminished.	A rhythmic contraction may be noted. More than three beats is abnormal and indicates 4+ ankle reflex grading.
5. **Plantar response (Babinski's sign):** Explain to the patient: "I am going to scrape the bottom of your foot". Using a blunt object (orange stick, the end of the reflex hammer or car key), gently scrape the lateral portion of the sole beginning near the heel and moving up towards the little toe then across the foot pad to the base of the big toe. Watch the big toe and the remainder of the foot. N.B. <u>Alternative stimuli to elicit the plantar response</u>: ➢ Chaddock's manoeuver: scrape the lateral portion of the dorsum of the foot beginning near the lateral malleolus and moving up towards the little toe ➢ Oppenheim's sign: with your thumb and index finger, press heavily from above downwards along the medial aspect of the tibia ➢ Gordon's reflex: pinch the Achilles tendon	➢ Reflex arc through **S1,2** ➢ always describe the response as either downgoing, i.e. all the toes flex towards the plantar surface, or upgoing where the big toe extends dorsally (goes up), while the four small toes fan and turn towards the sole ➢ *See theoretical notes for patterns of planter response*
6. **Abdominal reflexes: t**he patient should be supine and relaxed. Using an orange stick lightly scratch the abdominal wall towards the umbilicus in the four quadrants of the stomach.	➢ Normal response is contraction of the recti with the umbilicus moving away from the direction of the scratch. ➢ Afferent: segmental sensory nerves ➢ Efferent: segmental motor nerves ➢ Roots: **T8,9** above the umbilicus, and **T11,12** below the umbilicus ➢ Abdominal reflexes are absent in **UMNL** above their spinal level, and in **LMNL** affecting T8-12. ➢ It is often impossible to elicit abdominal reflexes in anxious patients, elderly, obese, multiparous women, and those who have had abdominal surgery

See theoretical notes for abnormal tendon reflexes, and the root values for reflexes

(7) COORDINATION:

Steps	Possible findings
1. **Heel-shin test**: the patient is lying supine. Tell the patient "Lift your leg and place the point of your heel on your knee and then run it down the sharp part of your shin; now up your shin, now down again…etc." (Demonstrate). Consider repeating the test while the patient's eyes are closed to test for sensory ataxia. Compare right with left. Expect the right leg to be slightly better in right handed persons	➢ In cerebellar **lower limb ataxia**, the patient has difficulty placing or holding the heel on the opposite knee or cannot keep the heel firmly on the tibia as the heel is moved downwards. ➢ Sensory ataxia is similar to cerebellar ataxia but is markedly worse when the eyes are closed
2. **Rapid repeated movements**: Tell the patient "tap your foot quickly on my hand as if listening to fast music" (demonstrate). Compare right with left. Expect the right leg to be slightly better in right handed persons	➢ **Bradykinesia:** slowed movements or break up easily – extrapyramidal sign ➢ **Dysrhythmia**: inability to keep a rhythm - cerebellar sign
3. **Truncal ataxia:** ask the patient to sit up with the arms folded or to rise from a chair with the feet together	➢ **Truncal ataxia** is caused by abnormalities of the midline cerebellar vermis or the flocculonodular lobe. It is usually associated with gait ataxia and symmetric nystagmus in absence of limb incoordination

N.B. do not assess coordination if power < 3 (inability to move against gravity), and tell the examiner that coordination cannot be assessed due to weakness

See theoretical notes for types of ataxia and causes of cerebellar ataxia

(8) SENSATION

Steps	Possible findings
1. **Pin prick:** ➢ **Demonstrate** the stimuli to the patient by testing on the sternum (use each end of the hat pin): explain to the patient "this is sharp…and this is blunt…now I'm going to test the sensation in your legs and I want you to close your eyes and say "Sharp" if it feels sharp, and "blunt" if it feels blunt". ➢ **Start distally and move proximally** testing over each dermatome (and each main nerve): lateral foot → medial foot → outer calf → inner calf → inner thigh → outer thigh → inguinal region. Repeat in the other leg then compare right with left ➢ **Map out the boundaries** of any area of reduced, absent or increased sensation; starting from the area of altered sensation and moving towards normal to find the edges, noting any difference between the two sides. ➢ If a stocking sensory loss is present, **demonstrate that the sensory loss is present right round the limb**. ➢ If compression of the cord is suspected, then **demonstrate a sensory level**.	➢ Reduced sensation over the lateral foot → tibial nerve or **S1** lesion ➢ Reduced sensation over the medial foot → common peroneal nerve or L5 lesion ➢ Reduced sensation over the outer calf → **common peroneal nerve** or **L5** lesion ➢ Reduced sensation over the inner calf → **L4** lesion ➢ Reduced sensation over the inner thigh → **L3** lesion ➢ Reduced sensation over the outer thigh → **lateral cutaneous nerve of the thigh** or **L2** lesion ➢ Reduced sensation over the inguinal region → **L1**lesion
2. **Light touch:** ➢ **Demonstrate** "I am going to touch you with this piece of cotton wool and I want you to close your eyes and say (Yes) every time you feel it". Avoid dragging it across the skin or tickling the patient. Time the stimuli irregularly to check the patient reliability. ➢ **Start distally and move proximally** testing the lower limb in the same sequence as for pin prick	
3. **Joint position sense:** ➢ **Demonstrate:** fix the proximal phalanx of the patient's **big toe** with one of your hands. Hold the lateral aspects of the distal phalanx of the patient's big toe between the thumb and index finger of your other hand. Ensure that your thumb and index finger are at 90 degrees to the intended direction of movement. Tell the patient "I'm going to move your finger up and down; this is up (move finger up)…, and this is down (move finger down)…now close your eyes and tell me whether I am moving your finger up or down". ➢ **Start with the IP joint of big toe**. If impaired, move to more proximal joints progressively (IP → MTP → ankle → knee → hip).	
4. **Vibration sense:** ➢ **Demonstrate:** use a 128 Hz tuning fork. Make the fork vibrate and place it on the sternum. Ask the patient "Do you feel it vibrating (buzzing)?" ➢ Tell the patient "Close your eyes". Make the fork vibrate silently and place it on the **terminal phalanx of the big toe** just below the nail bed and ask the patient "Can you feel it now?" If patient cannot feel the vibration, move progressively to more proximal joints (terminal phalanx of the big toe → MTP joint → medial malleolus → tibial tuberosity → ASIS).	**N.B.** vibration sense is commonly reduced or absent in elderly patients

See theoretical notes for:

- *Modalities and tracts of sensation*
- *Patterns, causes and clinical approach to sensory loss*

(9) <u>GAIT AND ROMBERG'S TEST</u>: ask the examiner's permission to examine the patient's gait and perform Romberg's test (*see Ch 9. CNS – Gait*).

(10) <u>ADDITIONAL SIGNS</u>: according to diagnosis, e.g.

- Look for cataract and scar for pacemaker, palpate for local spinal tenderness and examine for sensory level
- Tell the examiner that you would normally extend your examination to examine the upper limb and cranial nerves and woud ask about swallowing (Dystrophia myotonica), bladder symptoms, test the anal tone (spinal cord syndrome), and to dipstick urine for DM.

(11) <u>THANK THE PATIENT AND COVER HIM (HER)</u>

THEORETICAL NOTES

HOW TO DESCRIBE AN ABNORMAL MOVEMENT: abnormal movements are described according to the following fundamental characteristics:

- Is the movement present at rest (rest tremors), with holding of a posture (postural tremors), or during a skilled movement (action or intention tremors)
- Is the movement disorder global (generalised dystonia), focal (focal dystonia), unilateral, or confined to one limb
- Is the movement disorder predominantly distal (choreoathetosis) or proximal (hemiballismus) in the limb
- Is the movement sinuous (choreoathetosis), twitchy (myokymia), or shock like (myoclonus)
- Does the limb hold abnormal postures for several seconds (dystonia)

TYPES OF ABNORMAL MOVEMENTS

1. Tremors **(**rest tremor, postural tremor, action tremor, intention tremor)
2. Asterixis
3. Dystonia
4. Chorea
5. Asthetosis
6. Choreoasthetosis
7. Hemiballismus
8. Myoclonic jerks
9. Tics (habit spasms)
10. Dyskinesia
11. Akathisia

TREMORS

Features	Types	Causes
Tremors are oscillatory distal movements resulting from alternating contraction and relaxation of muscles. Tremors are described according to their speed (**fast or slow**), amplitude (**fine or coarse**) and whether they are maximal at rest, on maintaining a posture or on carrying out an active movement (**rest, postural, action or intention**)	**Rest tremor:** present when limb is at rest and reduced by voluntary movement**.** It is usually rapid, rhythmic, alternating tremor, predominantly in flexion/extension but often with a prominent rotary component between finger and thumb (pill-rolling tremor). It is almost always more severe in the arm than in the leg, and is usually asymmetrical.	1. **Akinetic-rigid syndromes** (parkinsonism)
	Postural tremor: present when limb is maintained in a position against gravity.	1. **Benign essential tremor** (50% familial): usually coarse tremor and often exaggerated in awkward postures, as when the outstretched fingers are held pointing at each other, in full inversion, in front of the patient's nose. 2. **Exaggerated physiological tremor** (fine and rapid): seen with anxiety, hyperthyroidism, excess alcohol or caffeine, or β-agonists.
	Action tremor: present during an action	1. **Lesions of the red nucleus and sub-thalamic nucleus** (most often caused by damage from vascular disease or MS)
	Intention tremor: most prominent during voluntary movement towards a target	1. **Cerebellar disease**

ASTERIXIS

Feature	Causes
Asterixis (negative myoclonus): intermittent inhibition of muscle tone that leads, for example, to a momentary and repetitive partial flexion of the wrists during attempted sustained wrist extension (may superficially resemble a tremor).	1. **Metabolic encephalopathy** (liver failure, uraemia, poisoning with hypnotic drugs) 2. **respiratory failure**

DYSTONIA

Feature	Types	Causes
Dystonia literally means any abnormality of muscle tone, but most neurologists employ it to describe the slow development of an abnormal posture (often of the limb or the neck) which is maintained by co-contraction of both agonists and antagonists. Positions maintained are usually at an extreme of extension or flexion. Common forms are torticollis (the neck twisted to one side), anterocollis (the neck flexed forward), retrocollis (the neck extended backwards), lordosis (the arched back), and scoliosis (twisted back). The arm is usually abducted at the shoulder, extended at the elbow, pronated to an extreme position with the fingers extended. The leg is usually extended at the hip and knee and inverted at the ankle with the toes flexed. The term dystonia frequently is qualified as torsion dystonia, to emphasize the twisted nature of the abnormal postures	**Focal dystonia** (affects only one part of the body): e.g. isolated torticollis or isolated writer's	1. **Idiopathic** 2. **Major tranquilisers**, 3. **Treated Parkinson's** disease on excessive therapy
	Segmental dystonia (affecting two or more adjacent parts of the body): for example torticollis and dystonic posturing in the same arm	1. As for focal dystonia (see above)
	Generalised dystonia (affects parts of the body that are not adjacent): often associated with chorea	1. As for chorea (see below). 2. Rare causes: dystonia musculorum

CHOREA, ASTHETOSIS, CHOREOASTHETOSIS

Feature	Causes
Chorea is a brief, fluid, involuntary movements which frequently appear pseudo-purposeful. In chorea, the ordinary voluntary movements, such as walking or picking up a cup, may be embellished with rapid extra little flourishes of movement. The outstretched upper limbs may assume a hyperpronated posture and little flicks of movement of the digits or wrist, or of the face and tongue may occur.	1. **Drug therapy of Parkinson's disease** (excess treatment) 2. **Wilson's disease** (look for associated liver disease and Keyser-Fleischer rings on cornea) 3. **Huntington's disease** (trace family history) 4. **Post-pill or pregnancy chorea** (chorea gravidarum) 5. **Sydenham's chorea** 6. **Stroke**
Athetosis is a complex irregular slow, writhing movement; occurs nearly continuously in distal muscles. It consists of an interaction between various postures of the hand or foot, especially ***grasping and avoiding***. The fingers are alternately widely extended, the arm following into an extended, abducted and externally rotated posture, and then the fingers clench, often trapping the thumb in the palm, and the limb flexes slightly and rotates internally.	1. The term athetosis was introduced originally to describe the sinuous, writhing digital movements that may follow a **stroke**. Subsequently, athetosis became synonymous with one type of **cerebral palsy**, resulting from brain damage due to anoxia or kernicterus at birth.
Choreoathetosis refers to something between the chorea and athetosis (or uncertainty on the part of the observer!)	

HEMIBALLISMUS, MYOCLONIC JERKS, TICS, DYSKINESIA, AKATHISIA

Feature	Causes
Hemiballismus (Ballism = to throw) is an irregular sudden and often violent flinging movement of a proximal limb, usually an arm. It most often involves only one side of the body (i.e., hemiballism [us]). There is no clear distinction from sever chorea.	1. **Infarction of the contralateral sub-thalamic nucleus** or its connections
Myoclonic jerks are sudden shock-like contractions of one or more muscles which may be focal or diffuse. They may occur singly or repetitively. Myoclonus can occur spontaneously at rest, in response to sensory stimuli, or with voluntary movements.	1. **Healthy individuals** may experience these when falling asleep or surprised by a sudden noise 2. **Metabolic encephalopathies** 3. **Myoclonic epilepsies** 4. **Creutzfeldt-Jakob disease** 5. **Post-anoxic encephalopathy**
Tics (habit spasms) are stereotyped, irresistible movements, which are essentially purposeful movements but recurring involuntarily in repetitive manner (may superficially resemble myoclonic jerks but more stereotyped). Tics may be voluntarily controlled at the expense of mounting inner tension.	1. **Simple tics**: isolated transient or chronic tics in children that may persist to late adolescence (good prognosis) 2. **Gilles de la Tourette syndrome**: tics associated with coprolalia (involuntary and inappropriate obscene speech). The pathophysiology is obscure. A dopaminergic excess has been suggested by the clinical observation that the tics may respond to treatment with dopamine-blocking drugs. 3. **Wilson's disease** (associated with hepatic and renal involvement, Kayser-Fleischer corneal rings, low serum copper and ceruloplasmin levels, and increased 24-h urinary copper excretion) 4. **Post-encephalitic syndromes** 5. **Stimulant or neuroleptic medication**. 6. **In association with obsessive compulsive disorders**.
Dyskinesia literally means any abnormal movement but the term has become synonymous with drug-induced abnormal movements; particularly orofacial dyskinesia. The term tardive (late) dyskinesia is used to distinguish movement disorders that occur as a late complication of treatment with neuroleptic drugs from acute dystonia and extrapyramidal syndrome which develop early.	1. **Neuroleptic drugs** (dopamine blocking agents): phenothiazines (chlorpromazine), butyrophenones (haloperidol), substituted benzamides (metoclopramide), reserpine, tetrabenazine 2. **Antiparkinsonian agents** (dopamine like effects): L-dopa, bromocriptine, lysuride, pergolide
Akathisia: Motor restlessness where the patient constantly shifts crossing and uncrossing his legs and walking on spot.	1. **Late reaction to major tranquilisers** (tardive akathisia)

ABNORMALITIES OF TONE:

Tone	Features	Causes
Normal	▪ Slight resistance through whole range of movement. ▪ Heel will lift minimally off the bed when the knee is lifted quickly	1. Normal 2. Myopathy → normal tone or hypotonia 3. Neuromuscular junction diseases 4. Functional weakness
Hypotonia	▪ Loss of resistance through movement. ▪ Heel does not lift off the bed when the knee is lifted quickly.	1. **LMN Lesion** 2. Cerebellar 3. Myopathy (normal or hypotonia) 4. Spinal shock 5. Chorea
Hypertonia	**Spasticity:** ▪ Resistance is velocity-dependent, detected as a "catch" at the beginning or end of passive movement, has a sudden release after reaching a maximum (the "**clasp-knife**" phenomenon) ▪ Heel easily leaves the bed when the knee is lifted quickly ▪ Predominantly affects antigravity muscles (upper limb flexors and lower limb extensors)	1. **UMN Lesion**
	Rigidity ▪ **Lead pipe or plastic rigidity:** Increased tone through **whole range**, as if bending a lead pipe. ▪ **Cogwheel rigidity**: Increased tone through **whole range, with regular interruption** to the movement giving it a jerky feel (due to associated tremor)	1. **Extrapyramidal syndromes**
	Paratonia or Gegenhalten: ▪ Increased tone through whole range that varies irregularly in response to repetitive passive movements, becoming **worse when the patient tries to relax** (patient apparently opposes your attempts to move his limb)	1. **Bilateral frontal lobe damage** (CVA, dementia)

<u>THE MEDICAL RESEARCH COUNCIL (MRC) SCALE FOR POWER GRADING</u>:

0 = **no muscle contraction** visible
1 = **flicker** of muscle contraction but no movement
2 = **movement** when gravity eliminated (but not against gravity)
3 = movement **against gravity** but not against resistance
4- = **slight** movement **against resistance**
4 = **moderate** movement **against resistance**
4+ = **submaximal** movement **against resistance**
5 = normal power (**full strength**)
N.B. when testing power, allow patient to move the joint through the full range, and look at or feel the muscle contract

FEATURES OF THE DIFFERENT PATTERNS OF WEAKNESS

	UMN Lesion	**LMN Lesion**	**Muscle disease (myopathy)**	**Neuromuscular junction (NMJ) disease**	**Functional weakness**
Muscle bulk	No wasting	Severe wasting	Mild wasting	No wasting	No wasting
Fasciculations	No fasciculations	Fasciculations are common	No fasciculations	No fasciculations	No fasciculations
Tone	Spasticity	Hypotonia	Normal tone / hypotonia	Normal tone / hypotonia	Normal tone
Pattern of weakness	Pyramidal pattern of weakness (weak extensors in the arm, weak flexors in the leg – more distally and more in the abductors than adductors)	Distal/ segmental distribution of weakness	Proximal weakness	Fatiguable weakness	Erratic power
Reflexes	Hyperactive reflexes	Hypoactive/ absent reflexes	hypoactive/ Normal reflexes	Normal reflexes	Normal reflexes
Planter response	Extensor plantar response	Flexor plantar response	Flexor plantar response	Flexor plantar response	Flexor plantar response

CAUSES OF THE DIFFERENT PATTERNS OF WEAKNESS

UMN Lesion	1. **Spinal cord syndromes**; differentiated by sensory signs ➢ Complete transverse lesion: trauma, spinal cord compression by tumour (usually bony secondaries in vertebra), cervical spondylitis, transverse myelitis, MS, intraspinal tumours (e.g. meningiomas), spinal abscess, post infectious (usually viral) ➢ Hemisection of the cord (Brown-Sequard syndrome): Causes as for transection ➢ Central cord: syringomyelia and trauma leading to haematomyelia ➢ Posterior column loss: any cause of complete transection, SCD (vitamin B 12 deficiency), tabes dorsalis ➢ Anterior spinal syndrome: anterior spinal artery emboli or thrombosis. 2. **Brainstem lesions**: brainstem infarction or haemorrhage(elderly), MS(young patients), tumours, trauma 3. **Hemisphere lesions**: infarction, haemorrhage, tumours, trauma, MS
LMN Lesion	1. **Mononeuropathy:** ➢ Compression (Saturday night palsy: compressing radial nerve in spiral groove by leaning arm over chair- also reported to affect sciatic nerve after falling asleep sitting on toilet) ➢ Entrapment, e.g. median nerve in carpal tunnel, common peroneal nerve behind head of the fibula at the knee; more common in DM, RA, hypothyroidism and acromegaly 2. **Radiculopathy:** ➢ Cervical or lumbar disc protrusion, e.g. L5/S1 disc protrusion compresses the S1 root. ➢ At the level of a compressive spinal lesion (secondary tumours, neurofibroma) 3. **Peripheral neuropathies:** ➢ Acute predominantly motor neuropathies: Guillan-Barre syndrome, diphtheria, porphyria ➢ Subacute sensorimotor neuropathy: vitamin B1, B12 deficiencies, heavy metal poisoning (lead, arsenic, thallium), drugs (vincristine, isoniazid), uraemia ➢ Chronic sensorimotor neuropathy: either acquired (DM, hypothyroidism, paraproteinaemia, amyloidosis) or inherited (HSMN, e.g. Charcot-Marie-Tooth disease) 4. **Mononeuritis multiplex:** inflammatory (PAN, RA, SLE, sarcoidosis) 5. **Polyradiculopathy:** indicates lesion of many roots. It is distinct from other peripheral neuropathies because it produces a more proximal weakness. The term is commonly applied to Guillan-Barre syndrome
Muscle disease (myopathy)	1. **Inherited:** muscular dystrophies (Duchene's, Becker's, fascio-scapular-humeral, myotonic dystrophy) 2. **Inflammatory:** polyomyositis, dermatomyositis, PMR 3. **Endocrine:** steroid-induced, hyperthyroid, hypothyroid 4. **Metabolic** (rare): glycogen storage disease (e.g. Pompe's disease), McArdle's disease 5. **Toxic:** alcohol, chloroquine, clofibrate
NMJ	1. **myasthenia gravis**: usually idiopathic; occasionally drug-induced (penicillamine, hydralazine) 2. **Lambert-Eaton syndrome**: paraneoplastic syndrome (usually oat cell carcinoma)
Functional weakness	1. may indicate **hysterical illness**

CLINICAL APPROACH TO WEAKNESS

Features of weakness	Possible diagnosis
Generalized weakness (limbs and cranial nerves)	1. Diffuse disease of nerve: poly-radiculopathy 2. Diffuse disease of muscle: myopathy 3. Diffuse disease of NMJ: myasthenia gravis
Quadriparesis with exaggerated reflexes and extensor plantar response (i.e. **UMN** lesion pattern)	1. Cervical cord lesion 2. Brainstem lesion 3. Bilateral cerebral lesions. Discriminate by sensory testing, cranial nerve signs, and hemisphere signs
Quadriparesis with hypoactive/absent reflexes (i.e. **LMN** lesion pattern)	1. Poly-radiculopathy 2. Peripheral neuropathy 3. Myopathy (normal/hypoactive reflexes): proximal weakness and normal sensations 4. Acute UMNL in the state of spinal shock
Quadriparesis with **mixed** UMNL pattern (in the legs) & LMNL pattern (in the arms)	1. MND (normal sensations) 2. Combined cervical myelopathy and radiculopathy (with sensory loss)
Quadriparesis with **normal reflexes**	1. Myopathy: (normal/hypoactive reflexes): proximal weakness and normal sensations 2. Myasthenia gravis: fatigable weakness with associated cranial nerve abnormalities
Hemiparesis (UMNL pattern with **contralateral sensory** findings	1. Hemisection of cervical cord
Hemiparesis (UMNL pattern with **contralateral cranial nerve** lesion	1. Brainstem lesion ➢ III nerve palsy → midbrain lesion ➢ VI and/or VII → pontine lesion ➢ XII ± IX and XI → medullary lesion
Hemiparesis (UMNL pattern with **ipsilateral UMNL VII** palsy	1. Internal capsule lesion
Hemiparesis (UMNL pattern with **hemisphere signs** (aphasia, higher function deficits, contralateral visual field defects, contralateral inattention or neglect)	2. Cerebral lesion ➢ Parietal lobe lesion → **contralateral lower homonymous quadrantanopia** ➢ Temporal lobe → **contralateral upper homonymous quadrantanopia** ➢ Lesion in the anterior occipital cortex (posterior cerebral artery occlusion) → **congruous homonymous hemianopia** ➢ Bilateral occipital lobe lesions → **cortical blindness**
Paraparesis (**UMNL** pattern)	1. Spinal cord lesion above the root level of the highest motor abnormality. A level may be ascertained with sensory signs (sphincter involvement)
Paraparesis (**LMNL** pattern)	1. Polyradiculopathy 2. Cauda equinal lesion (sphincter involvement) 3. Peripheral neuropathy: ➢ Acute (Guillan-Barre syndrome, porphyria, diphtheria) ➢ Subacute (lead poisoning) ➢ Chronic (HSMN, CIDP)
Monoparesis (**UMNL** pattern)	1. Lesion in spinal cord (above the highest involved level) 2. Brainstem lesion 3. Cerebral lesion. Discriminate by sensory testing, cranial nerve signs, and hemisphere signs.
Monoparesis (**LMNL** pattern)	1. Mononeuropathy 2. Radiculopathy See examples of mononeuropathies and radiculopathies below
Patchy weakness (**UMNL** pattern)	1. Multiple CNS lesions
Patchy weakness (**LMNL** pattern)	1. Polyradiculopathy 2. Multiple single nerves (mononeuritis multiplex)
Variable weakness (non anatomical distribution)	1. If progressively gets worse: consider myasthenia gravis 2. If fluctuates; giving full power at times: consider functional weakness

MOTOR ROOT VALUES IN THE LOWER LIMB

Root value	Muscle action
L**1**,**2**,**3**	Hip flexion
L**2**,**3**	Hip adduction
L**4**,**5**,S**1**	Hip abduction
L**5**,S**1**	Hip extension
L**3**,**4**	Knee extension
L**4**,**5**	Ankle dorsiflexion and inversion
L**5**,S**1**	Ankle eversion
S**1**,**2**	Ankle plantar flexion
L**5**,S**1**,**2**	Knee flexion
L**5**,S**1**	Toes extension
S**2**,**3**	Toes flexion

EXAMPLES OF MONONEUROPATHIES AND RADICULOPATHIES IN THE LOWER LIMB

Common peroneal palsy (compare to L5)	L4 root	L5 root	S1 root
• Foot drop → high stepping gait • Wasting of the muscles on the lateral aspect of the leg (tibialis anterior and peronei) • Weakness of dorsiflexion (tibialis anterior; L5) and eversion (peronei; S1). • Sensory loss over the anterolateral aspect of the lower half of the leg (lateral shin) and dorsum of the foot • Characteristically: ankle reflex is preserved (lost in tibial nerve palsy, sciatic nerve palsy and S1 radiculopathy) • Ankle inversion is intact (lost in tibial nerve palsy, sciatic nerve palsy and L4/5 radiculopathy)	• Weakness of knee extension and foot dorsiflexion • Loss of knee reflex • Sensory loss over the medial shin	• Weakness of foot dorsiflexion, inversion and eversion, extension of the big toe and hip abduction • Sensory loss over the lateral shin and dorsum of foot	• Weakness of plantar flexion and foot eversion • Loss of ankle reflex • Sensory loss over the lateral border of foot and sole of foot

CAUSES OF FOOT DROP

1. Common peroneal nerve palsy (ankle eversion is lost while ankle inversion and ankle reflex are preserved)
2. Lumbosacral plexus lesion
3. Sciatic nerve palsy (ankle reflex and ankle inversion are lost)
4. Peripheral neuropathy, especially HSMN type 1
5. L4/5 radiculopathy most commonly due to prolapsed lumbar disc (ankle reflex and ankle inversion are lost)
6. Motor neurone disease (MND)

REFLEXES GRADING

0 = absent
± = present only with reinforcement
1+ = present but depressed (diminished)
2+ = normal
3+ = increased (hyperactive)
4+ = clonus

ABNORMAL TENDON REFLEXES

Reflex response	Causes
Increased reflex or clonus	1. UMNL above the level of the increased reflex
Absent reflexes	1. **Generalized** peripheral neuropathy 2. **Isolated** root lesion or less commonly peripheral nerve lesion 3. **Bilateral absent ankle reflexes** peripheral neuropathy or -less commonly- bilateral S1 root lesions or -very rarely- bilateral sciatic nerve lesions **N.B.** reflexes can be absent in the early stages of severe UMNL (spinal shock)
Reduced reflexes	1. Peripheral neuropathy, muscle disease and cerebellar syndrome.
Inverted reflex: a combination of loss of the reflex tested with spread of the reflex to muscle at a lower level. It is caused by combined spinal cord and root lesion	1. The level of the absent reflex indicates the level of the lesion. For example, in an inverted biceps reflex, when the biceps tendon is tapped there may be no biceps reflex but the triceps contracts; this indicates LMNL at the level of the absent reflex (in this case C5) with UMNL below; indicating spinal cord involvement at the level of the absent reflex. In an inverted supinator reflex, when the supinator reflex is tested there is no response but finger flexion occurs (C5/6 lesion)
Pendular reflex	1. Best seen in the knee jerk where the reflex continues to swing for several beats. This is associated with cerebellar disease
Slow relaxing (myotonic) reflex	1. Especially seen at the ankle reflex and may be difficult to note. It is associated with hypothyroidism.

THE ROOT VALUES FOR REFLEXES

Action	Root values	Reflexes
One two……...buckle my shoe	S **1**,**2**	Ankle reflex
Three four... ….kick the door	L **3**,**4**	Knee reflex
Five six…..……pick up sticks	C **5**	Biceps reflex
	C **6**	Brachioradialis reflex
Seven eight …..shut the gate	C **7**	Triceps reflex
	C **8**	Finger reflex

PATTERNS OF PLANTAR RESPONSE

Response	What it means
Flexor plantar response (negative Babinski's sign): all the toes flex towards the plantar surface	▪ Normal
Extensor plantar response (positive Babinski's sign): The big toe extends dorsally (goes up), while the four small toes fan and turn towards the sole.	▪ UMNL above the S1 level of the spinal cord ▪ Normal in children below the age of 1 year
Withdrawal response: The big toe extends (goes up), the other toes extend and ankle dorsiflexes	▪ Repeat more gently or try alternative stimuli
No response: no movement of the big toe (even if the other toes flex)	▪ profound UMN weakness (toe unable to extend) ▪ Sensory abnormality interfering with the afferent part of the reflex.

TYPES AND CAUSES OF ATAXIA

	Features	Causes	Associated clinical signs
Cerebellar limb ataxia	Abnormalities of the intermediate and lateral portions of the cerebellum typically produce impaired limb movements rather than truncal ataxia. If involvement is asymmetric, lateralized imbalance is common and usually associated with asymmetric nystagmus	**Unilateral** limb ataxia (ipsilateral cerebellar syndrome): ▪ Demyelination, ▪ Vascular disease ▪ Less commonly trauma, tumour or abscess **Bilateral** limb ataxia (bilateral cerebellar syndrome): ▪ Drugs; e.g. anticonvulsants (phenytoin) ▪ Alcohol ▪ Demyelination (MS) ▪ Vascular disease (stroke) ▪ Less commonly hereditary cerebellar degeneration (Friedreich's ataxia), paraneoplastic disorders, or hypothyroidism	▪ Dysmetria and intention tremor (finger-nose and heel-shin tests) ▪ Dysdiadochokinesia (rapid alternating movements) ▪ Muscle tone is often modestly reduced; this contributes to the abnormal rebound due to decreased activation of segmental spinal cord reflexes and also to pendular reflexes, i.e., a tendency for a tendon reflex to produce multiple swings to and fro after a single tap
Cerebellar truncal ataxia	Abnormalities of the midline cerebellar vermis or the flocculonodular lobe produce truncal ataxia which is usually revealed during the process of rising from a chair, assuming the upright stance with the feet together, or performing some other activity while standing.	Lesion of the cerebellar vermis (Midline cerebellar syndrome): cause as for bilateral cerebellar syndrome	▪ Gait ataxia and symmetric nystagmus in absence of limb incoordination
Sensory limb ataxia	Porprioceptive impairment produced sensory limb ataxia, which is similar to cerebellar limb ataxia but is markedly worse when the eyes are closed. Examination also reveals abnormal proprioception and vibratory perception.	Porprioceptive impairment: ▪ Peripheral nerves lesions ▪ Posterior column lesion ▪ Parietal lobe damage	Depends on the site of the proprioceptive impairment: ▪ The peripheral nerves → depressed or absent reflexes ▪ The posterior columns of the spinal cord → spasticity with extensor plantar response ▪ The parietal lobe (rare) → lateralized imbalance

MODALITIES AND TRACTS OF SENSATION

Vibration and joint position	Pin prick and temperature	Light touch
Ascend in the posterior column (remains ipsilateral up to the medulla, where it crosses over)	Ascend in the spinothalamic tract (crosses within one to two segments of entry)	Ascends in both the posterior column and spinothalamic tracts.

PATTERNS AND CAUSES OF SENSORY LOSS

Sensory lesion	Pattern	Causes
Mononeuropathy	Single nerve lesion most commonly median, ulnar, peroneal, lateral cutaneous nerve to the thigh	➢ Entrapment neuropathy (e.g. median nerve in carpal tunnel, common peroneal nerve behind head of the fibula at the knee, more common in DM, rheumatoid arthritis, hypothyroidism) ➢ May be presentation of more diffuse neuropathy (see cause of peripheral neuropathy below)
Mononeuritis multiplex	Multiple single nerve lesions	DM, connective tissue disease (SLE, RA), vasculitis (PAN, Churg-Strauss), infection (HIV), malignancy, or may be presentation of more diffuse neuropathy (see above)
Radiculopathy	Root or roots lesions most commonly C5, C6 and C7, L4, L5 and S1	Compression by prolapsed intervertebral discs, tumours (e.g. neurofibromatosis)
Peripheral neuropathy	Peripheral nerves lesions: sensory loss either in both legs or in both arms and legs - without clear upper level → distal glove and stocking deficit	DM, Vitamin B1, B12 deficiencies, Uraemic neuropathy, Alcohol, Drugs (vincristine, isoniazid), Carcinomatous neuropathy (especially CA bronchus), Amyloidosis, and Leprosy
Spinal cord lesion → sensory loss in both legs (or both arms and both legs) with clear upper level.	Complete transverse lesion → hyperaesthesia at the upper level with loss of all modalities a few segments below the lesion.	Trauma, spinal cord compression by tumour (usually bony secondaries in vertebra), cervical spondylitis, transverse myelitis, MS, intraspinal tumours (e.g. meningiomas), spinal abscess, post infectious (usually viral)
	Hemisection of the cord (Brown-Sequard syndrome) → loss of joint position sense and vibration sense on the same side as the lesion and pain and temperature on the opposite side a few levels below the lesion.	As for transection
	Central cord lesion → loss of pain and temperature sensation at the level of the lesion, where the spinothalamic fibres cross in the cord, with other modalities preserved (dissociated sensory loss).	Syringomyelia and trauma leading to haematomyelia
	Posterior column lesion → loss of joint position sense and vibration sense with intact pain and temperature.	Any cause of complete transection, SCD (vitamin B 12 deficiency), tabes dorsalis
	Anterior spinal syndrome → loss of pain and temperature below the level with preserved joint position sense and vibration sense.	Anterior spinal artery emboli or thrombosis
Brainstem lesion	Hemisensory loss including face on the contralateral side): loss of pain and temperature on the face and on the opposite side of the body	Demyelination (young patients), brain stem stroke (lateral medullary syndrome), brain stem tumours
Thalamic sensory loss	Hemisensory loss of all modalities including face on the same side)	Stroke, cerebral tumour, MS, trauma
Cortical loss	Hemisensory loss including face on the same side): parietal lobe- the patient is able to recognize all sensations but localizes them poorly- loss of two point discrimination, astereognosis, sensory inattention	As for thalamic sensory loss
Functional loss	This is suggested by a non-anatomical distribution of sensory deficit frequently with inconstant findings	May indicate hysterical illness; however, this is a difficult diagnosis to make in the absence of appropriated psychopathology

CLINICAL APPROACH TO SENSORY LOSS

Features of sensory loss	Possible diagnosis
Sensory loss in the distribution of a **single nerve**	Mononeuropathy; most commonly median, ulnar, peroneal, or lateral cutaneous nerve to the thigh
Sensory loss in the distribution of a **single root**	Radiculopathy; most commonly (C5, C6, C7) or (L4, L5 and S1)
Sensory loss affecting **both legs** with clear upper level	Spinal cord lesion
Sensory loss affecting **both legs** (or **both arms and both legs)** without clear upper level (glove and stocking)	Peripheral neuropathy
Sensory loss affecting **both arms and both legs** with clear upper level	Cervical spinal cord lesion
Hemisensory loss including the face on the same side (all modalities)	Thalamic lesion
Hemisensory loss including the face on the same side (not all modalities)	Internal capsule lesion
Hemisensory loss including the face on the contralateral side	Brain stem lesion

SCIATIC NERVE (L4,5,S1,2,3): most important branch of lumbosacral plexus and largest nerve in the body. It terminates by dividing into **medial** popliteal **(tibial)** and **lateral** popliteal (common **peroneal**) nerves

LATERAL POPLITEAL (COMMON PERONEAL) NERVE (L4,5,S1,2)**:**

- Passes through the popliteal fossa, winding around the head of the fibula (more susceptible to injury than the tibial nerve)
- Divides into terminal pressures: deep peroneal (anterior tibial) and superficial nerves
- Common peroneal nerve palsy (CPNP):
  - Foot drop → high stepping gait
  - Wasting of the muscles on the lateral aspect of the leg (**tibialis anterior** and **peronei)**
  - Weakness of dorsiflexion (**tibialis anterior**; L5) and eversion (**peronei**; S1).
  - Sensory loss over the anterolateral aspect of the lower half of the leg and dorsum of the foot
  - Characteristically: ankle reflex is preserved (lost in tibial nerve palsy, sciatic nerve palsy and S1 radiculopathy)
  - Ankle inversion is intact (lost in tibial nerve palsy, sciatic nerve palsy and L4/5 radiculopathy)