

CNS - GAIT

STEPS OF EXAMINATION

Step 1: Approach the patient

- Read the instructions carefully for clues
- Shake hands, introduce yourself
- Ask few questions “Could you tell me your name please? Are you right- or left-handed? Are you quite comfortable? Do you feel pain anywhere?”
- Ask permission to examine him

Step 2: General inspection:

- **Bedside:** walking stick, shoes-callipers, built-up heels
- **General appearance:** scan the patient quickly looking for:
 - Nutritional status (under/average built or overweight)
 - Abnormal movement or posture (rest or intention tremors, dystonia, choreoathetosis, hemiballismus, Myoclonic jerks, tics, pyramidal posture)
 - Abnormal facial movements (hemifacial spasm, facial myokymia, blepharospasm, oro-facial dyskinesia)
 - Facial asymmetry (hemiplegia)
 - Nystagmus (cerebellar syndrome)
 - Facial wasting (muscular dystrophy)
 - Sad, immobile, unblinking facies (Parkinson’s disease)
 - Peroneal wasting (Charcot-Marie-Tooth disease)
 - Pes cavus (Friedreich’s ataxia, Charcot-Marie-Tooth disease)
- **Hands:** tell the patient “outstretch your hands like this (palms facing downwards)”... then “like this (palms facing upwards)”
 - Check for wasted hands (MND, Charcot-Marie-Tooth disease, syringomyelia)
 - feel the radial pulse (AF → thromboembolism)

Step 3: Ask the patient “Can you walk without help? I will stay with you in case of any problems”. Notice any cerebellar dysarthria during his reply.

Step 4: Ask him to walk to a defined point, turn and walk back. Look at the patient from behind, in front, and the side. Go through the following questions in sequence to find out the diagnosis:

- **Is the gait obviously asymmetrical:**
 - **Hemiplegic gait:**
 - One leg swing out to the side (abduction and circumduction at the hip)
 - The leg is stiffly extended at the knee and ankle, the foot is inverted and the toes scrape the floor (patient tries to avoid this by contralateral tilt of the trunk)
 - The arm is stiffly flexed at the side (triple flexion posture of the arm) while in the mildest form, the arm is held in a normal position but swings less than the normal arm
 - **Unilateral foot drop:**
 - One knee lifts higher than the other to avoid catching the toe on the floor.
 - The foot hangs down while elevated
 - Patient is unable to walk on the affected heel
 - **Antalgic or painful gait:** The good limb hurries through and the painful limb buckles to cushion the impact on each step
 - **Orthopaedic gait:** bony deformity (shortened limb, previous hip surgery, trauma)
- **Is it short stepping gait:**
 - **Parkinsonian gait:**
 - Delayed initiation of walking. Short shuffling steps with modest flexion at hips and knees. Turns are clumsy and freezing may occur

- Occasional festinant gait (indicates impairment of postural reflexes): the pace tends to accelerate as the upper body gradually leans further ahead of the feet (propulsion). Similarly, the patient takes several steps backward (retropulsion) when given a gentle pull from behind.
- Stooped posture (flexion dystonia), arms adducted at shoulders and flexed at the elbow with reduced arm swing,
- Additional signs: sad, expressionless, unblinking facies. The hands may show coarse (4- to 6-Hz) pronation-supination rest tremor (pill-rolling)
- **Marche a petit pas:**
 - Upright posture, marked arm swing, short, quick tapping steps
 - Additional signs: dementia, pseudobulbar palsy, emotional lability
- **Apraxic gait:**
 - When asked to walk while standing, a long pause often occurs before any attempt to walk, as if the patient is glued to the ground (sticky feet/ magnetic gait). After few steps, walking is stopped again for several seconds. The process is then repeated
 - Disjointed movement (slow, shuffling, unsteady, short steps) as if forgotten how to walk, neither turning or straight walking are fluent, and there is tendency to retropulsion
 - Stooped posture
- **Is the gait Broad based or scissoring:**
 - **Cerebellar ataxia:**
 - Broad-based, unsteady (ataxic), high-stepping gait that veers towards the side of lesion. Turns are clumsy.
 - Difficulty walking heel-to-toe
 - Additional signs: scanning/staccato dysarthria and nystagmus
 - **Sensory ataxia:**
 - Broad-based, unsteady (ataxic), high-stepping, slapping (stamping) gait, with clumsiness on the turns (patient watch the floor intently and is more ataxic when eyes closed)
 - Difficulty walking heel-to-toe
 - Positive Romberg's test
 - **Scissoring (paraparetic) gait:**
 - Stiff legged gait: The patient has difficulty in bending the knees, so the foot is raised from the ground by tilting the pelvis (abduction and circumduction at the hip). The stiff leg is then swung forward, dragging the inverted foot along the floor, so that the foot tends to cross (scissor)
 - The arms are held in flexed and pronated position
 - Additional signs: bedside wheelchair and/or walking sticks, diffuse atrophy and contractures (if chronic), scars in the back or spinal deformity
- **Is the gait high-stepping (but not broad based):**
 - **Bilateral foot drop:**
 - High stepping with the feet slapping the ground (knee lifted high to avoid catching the toe on the floor)
 - Unable to walk on heels
- **Is the gait waddling:**
 - **Waddling (myopathic) gait:**
 - The body sways from side to side with each step, due to marked rotation of the pelvis and the shoulder
 - Additional signs: The body is often tilted backwards, with an increased lumbar lordosis
- **Is the gait bizarre or chaotic:**
 - **Functional gait (Astasia-abasia):** Bizarre, elaborated movement, worse when watched, however they do not fall and hurt themselves (veer toward the examiner's arms or a nearby bed. Inconsistent with rest of examination)
 - **Choreoathetotic gait:**
 - Chaotic walking (shuffling, twitching and spasmodic), due to Intermittent, irregular movements that disrupts the flow of the gait and unpredictable flexion or extension movements at the hip (pelvic lurch)
 - Unusual foot placement responses may occur so that the toes may extend away from the floor (avoiding response) or the feet may appear glued to the floor (grasping response)
 - Involuntary movements are usually exaggerated during walking

Step 5: If you suspect **sensory ataxia**, tell the patient “Close your eyes while walking”. In case of sensory ataxia, he will become more ataxic with eyes closed

Step 6: Heel-to-toe (tandem) gait: tell the patient “can you walk as if on a tight rope like this”. This will exacerbate ataxia (note the side to which the patient tends to fall).

Step 7: Walking on toes: tell the patient “Can you walk on your toes like this” (if unable → weakness of gastrocnemius - S1 lesion).

Step 8: Walking on heels: tell the patient “Can you walk on your heels like this” (if unable → foot drop - L5 lesion). **N.B.** if the patient has a spastic gait or a hemiparesis he may find both walking on toes and walking on heels difficult to perform

Step 9: Romberg’s test: have the patient between you and a wall and tell him “stand with your feet together like this; I am ready to catch you if you fall” allow him to stand like this for a few seconds. If he does not fall with his eyes open tell him “close your eyes”. Watch for unsteadiness with eyes opened and with eyes closed:

- Severe unsteadiness with eyes open → cerebellar disorders, particularly those involving the vermis.
- Patient is able to stand with eyes open and tends to fall with eyes closed or patient is more unsteady (tends to fall) with the eyes closed more than with them open → positive Romberg’s test (**sensory ataxia** – posterior column lesion or peripheral neuropathy)
- False-positive Romberg’s test
 - In vestibular (labyrinthine) disorders, the patient has consistent unsteadiness which is worse with eyes closed. It is different from sensory ataxia in that the imbalance appears after an interval and consists of a slow swaying to one side (side of the lesion), while in sensory ataxia, the swaying begins as soon as the eyes are closed, rapid, and occurs in all directions
 - Patients with hysteria tend to sway from the hips rather than the ankles. However they do not fall and hurt themselves

Step 10: Additional signs according to suspected diagnosis:

- Parkinson’s disease: check for extrapyramidal signs (expressionless unblinking face, rest tremors, cogwheel rigidity, glabellar tap sign)
- Cerebellar ataxia: check for cerebellar signs (nystagmus, staccato dysarthria, finger-nose incoordination, dysdiadochokinesia)
- Sensory ataxia: check joint position sense and vibration sense, look for Argyll Robertson pupils and clinical anaemia
- Paraparetic (scissoring) gait: look for scars in the back or spinal deformity, examine tone, reflexes, plantar reflex and sensation
- Apraxic gait: frontal lobe signs (dementia, grasp and suck reflexes)

Step 11: Thank the patient and cover him (her)

THEORETICAL NOTES

Phases of gait: for each leg, gait has two phases:

- **Swing phase:** from toe-off to heel-strike (foot clears the ground)
- **Stance phase:** from heel-strike to toe-off (foot on the ground and load bearing). When both feet are on the ground this is double stance

Features and causes of abnormal gaits:

Abnormal gaits	Causes
Hemiplegic gait	Unilateral UMNL (stroke , MS)
Unilateral foot drop	<ul style="list-style-type: none"> ▪ Common peroneal nerve palsy (ankle eversion is lost while ankle inversion and ankle reflex are preserved) ▪ Lumbosacral plexus lesion ▪ Sciatic nerve palsy (ankle reflex and ankle inversion are lost) ▪ Peripheral neuropathy, especially HSMN type 1 ▪ L4/5 radiculopathy most commonly due to prolapsed lumbar disc (ankle reflex and ankle inversion are lost) ▪ MND
Antalgic or painful gait	Arthritis, trauma
Orthopaedic gait	Shortened limb, previous hip surgery, trauma
Parkinsonian gait	Extrapyramidal syndromes (basal ganglion dysfunction), e.g. Parkinson's disease or drug induced (major tranquilizers: phenothiazines)
Marche a petit pas (senile gait)	Bilateral diffuse cortical dysfunction (multilacunar infarct)
Apraxic gait	Abnormal cortical integration of the movement, usually with frontal lobe pathology, e.g. NPH or CVA
Cerebellar ataxia	<ul style="list-style-type: none"> ▪ Ipsilateral cerebellar syndrome: demyelination, vascular disease, trauma, tumour or abscess ▪ Bilateral cerebellar syndrome: drugs (phenytoin), alcohol, demyelination (MS), vascular disease (stroke), hereditary cerebellar degeneration (Friedreich's ataxia), paraneoplastic disorders, hypothyroidism ▪ Midline cerebellar syndrome (truncal ataxia and gait ataxia, in absence of limb incoordination): lesion of the cerebellar vermis (causes as for bilateral cerebellar syndrome)
Sensory ataxia	<ul style="list-style-type: none"> ▪ Posterior column lesion: <ul style="list-style-type: none"> ○ Cord compression (e.g. cervical spondylosis, tumour) ○ Tabes dorsalis ○ Vitamin B12 deficiency (subacute combined degeneration of the cord) ○ Degenerative spinal cord disease ▪ Peripheral neuropathy (predominantly sensory): DM, Vitamin B1, B12 deficiencies, Uraemic neuropathy, Alcohol, Drugs (vincristine, isoniazid), Carcinomatous neuropathy (especially CA bronchus), Amyloidosis, and Leprosy.
Scissoring (paraparetic) gait	Spastic paraparesis (corticospinal lesion): <ul style="list-style-type: none"> ▪ Cerebral palsy ▪ MS ▪ Cord compression ▪ Hereditary spastic paraplegia
Bilateral foot drop	Peripheral neuropathy (predominantly motor): acute (Guillan-Barre syndrome, porphyria, diphtheria), subacute (lead poisoning), chronic (HSMN, CIDP)
Waddling (Myopathic) gait	Weak proximal muscles (proximal myopathies, bilateral congenital dislocation of the hip)
Functional gait (Astasia-abasia)	Hysteria
Choreoathetotic gait	Huntington's disease

• **Pathophysiologic basis of Romberg's test:**

- Central postural control (equilibrium) is dependent on input from three peripheral modalities:
 1. Vision
 2. Vestibular apparatus
 3. Proprioception (joint sense and sense of position)
- Disturbance in any one of these modalities can be compensated for (completely or incompletely) by input from the other two systems.
- Impaired proprioception can be overcome by visual and vestibular feedback. Asking the patient to close his eyes during the Romberg's test helps uncover any disordered proprioception that may have been masked by vision.

• **Causes of positive Romberg's test (loss of joint position sense):**

- **Posterior column lesion in the spinal cord**
 - Cord compression (e.g. cervical spondylosis, tumour)
 - Tabes dorsalis
 - Vitamin B12 deficiency (subacute combined degeneration of the cord)
 - Degenerative spinal cord disease
- **Peripheral neuropathy (predominantly sensory):** DM, Vitamin B1, B12 deficiencies, Uraemic neuropathy, Alcohol, Drugs (vincristine, isoniazid), Carcinomatous neuropathy (especially CA bronchus), Amyloidosis, and Leprosy.
- **N.B.** causes of predominantly motor neuropathies are: acute (Guillan-Barre syndrome, porphyria, diphtheria), subacute (lead poisoning), chronic (HSMN, CIDP)