

INSTRUCTION B

Examine the gait

Instruction B: Examine the gait**What should you do?**

- You will probably be asked to examine the relevant part of the nervous system as you see fit following the gait examination. However, you may be asked to specifically examine part of the nervous system, such as the lower limbs, after the gait examination.
- Have a very quick look all around. Young patients are more likely to have an ataxic or waddling gait, whereas elderly patients probably have Parkinson's disease or stroke.
- Examine the face for any evidence of parkinsonism.
- Look for any evidence of resting or action tremor.
- Look very carefully at the gait base (wide or narrow), the steps, the arms (whether or not they swing), posture (whether or not it is stooped) and the ability to turn.
- Ask the patient to do the tandem walk (heel-to-toe). Demonstrate this to the patient first.
- Stand close to the patient to prevent them from falling during the exam!

Tips

- If you think the patient is ataxic, perform Romberg's test. Ask the patient to stand with their feet together and their arms by their sides. Then ask them to close their eyes.
- Romberg's sign tends to be overrated. All patients with ataxia tend to get worse when they close their eyes. Romberg's sign should only be considered positive if there is a significant degree of worsening of the ataxia after closing the eyes, which indicates sensory ataxia.

FINDINGS

Finding One

The patient's gait is wide-based with difficulty performing the heel-to-toe test ('drunken gait').

Diagnosis

- Ataxic gait due to cerebellar syndrome.

Tips

- Check the cerebellar signs, namely nystagmus, dysarthria, abnormal finger–nose test and abnormal heel–shin test.
- Look for intention tremor (worse on approaching the target).
- Test for dysdiadochokinesia, which is defined as a breakdown of rhythmic, rapid alternating movements (impairment of rapid pronation and supination movements of one hand on the other one).
- Look for any evidence of increased tone, spastic legs or optic atrophy (indicative of multiple sclerosis). Look for any signs of alcoholic liver disease.

Discussion

Q: What are the causes of cerebellar syndrome?

- Multiple sclerosis.
- Alcoholic cerebellar degeneration (usually gait ataxia).
- Drugs such as anticonvulsants (phenytoin and carbamazepine) and lithium.
- Stroke-related causes, such as ischaemia or haemorrhage.
- Paraneoplastic syndrome (usually with lung and breast cancer).
- Spinocerebellar ataxia (genetic ataxia).
- Idiopathic cerebellar ataxia.
- Friedreich's ataxia (pes cavus, absent ankle jerks, upgoing plantars, and scoliosis).
- Posterior fossa tumours.
- Hypothyroidism.

Q: How would you investigate?

- Investigations are based on the likely cause.
- MRI of brain.
- Cerebrospinal fluid analysis, looking in particular for oligoclonal bands.
- Anticonvulsant levels in the blood.
- Paraneoplastic antibodies.
- Genetic testing for spinocerebellar ataxia.
- Chest X-ray or mammogram.
- NCS/EMG and genetic testing for Friedreich's ataxia (DNA testing for GAA repeats of the frataxin gene).

Q: What is the treatment?

- Treat the underlying cause if possible.
- Genetic counselling.

Finding Two

Patient walks with small steps and shuffles. They stoop, with lack of arm swing. The arms are held in flexed positions.

Diagnosis

- Parkinson's disease.

Tips

- Look for pill-rolling tremor.
- Examine the face. It is usually expressionless with lack of blinking.
- Examine for rigidity (cog-wheel if there is tremor), by slow flexion and extension movements at the wrist. If rigidity is mild, ask the patient to flex and extend their fingers in the contralateral hand to reinforce the rigidity.
- Demonstrate bradykinesia by asking the patient to repetitively open and close their thumb and index finger. Observe both the slowness and reduction of the amplitude of the movement.
- Symptoms and signs of Parkinson's disease are more prominent on one side (asymmetry).

Discussion

Q: What is the differential diagnosis of the parkinsonian syndrome?

- Drug-induced parkinsonism, mainly caused by phenothiazines.
- Vascular parkinsonism (multiple cerebral infarct state).
- Progressive supranuclear palsy (check for vertical gaze palsy).
- Multiple system atrophy (check the blood pressure with the patient lying and standing).

Q: How do you make the diagnosis of Parkinson's disease?

- It is a clinical diagnosis.

Q: What is the treatment?

- Drugs are the main form of treatment.
- Dopamine agonists, especially in the early stages of the disease and in young patients (ropinirole, pramipexole and rotigotine).
- Levodopa is still the main and most effective treatment.
- Monoamine oxidase B inhibitors (rasagiline and selegiline).
- Catechol-O-methyl transferase (COMT) inhibitors (entacapone).
- Apomorphine injection and infusion.
- Surgery, mainly deep brain stimulation. Patient selection is crucial. Patients should have positive responsiveness to dopamine therapy with no cognitive or psychiatric problems.

Q: What are the main problems with levodopa therapy?

- Wearing off: the response period becomes shorter and shorter.
- Dyskinesia: excessive involuntary choreiform movements.
- On/off: rapid and unpredictable shift from 'on' state (good response) to 'off' state (lack of response).

Finding Three

Patients's legs held wide apart. Lumbar lordosis. Trunk moving from side to side with pelvis dropping.

Diagnosis

- Waddling gait due to hereditary muscular dystrophies (Becker muscular dystrophy).

Tips

- A male patient.
- You are unlikely to see Duchenne muscular dystrophy, as patients are usually severely disabled if they reach adulthood.
- Look for pseudohypertrophy of the calves.
- The upper limbs may be involved in the later stages.
- The facial muscles are usually intact.

Discussion

Q: What is the mode of inheritance of Becker muscular dystrophy?

- X-linked (there is not always a family history, as 30% of the cases are new mutations).

Q: What are the other causes of waddling gait?

- Any cause of proximal myopathy (*see* page 101).

Q: How would you investigate?

- Serum creatinine kinase.
- EMG.
- Muscle biopsy.

Q: What is the treatment?

- There is no specific treatment, and therapy is mainly supportive.
- Respiratory and cardiac monitoring.
- Genetic counselling.

PITFALLS

Pitfalls

- Remember that asymmetry of reflexes should be regarded as abnormal.
- When the big toe moves up in the upgoing plantar response (UMN sign), the other four toes fan and turn towards the sole.
- Pyramidal weakness (UMN) predominantly affects hip flexor, knee flexors and ankle dorsiflexion (the flexors).
- Absent ankle reflexes and reduced vibration sense in an elderly patient could be normal.
- Sensory examination should be performed at the end of the examination of the lower limbs. By that stage you should have some idea about the possible diagnosis. The sensory findings should complement the motor ones (e.g. reduced pinprick sensation in stocking distribution in patients with absent ankle jerks).
- Your sensory examination should aim to demonstrate a sensory level (in spastic paraparesis), stocking distribution of sensory impairment (in peripheral neuropathy) and dermatomal abnormalities (L5/S1 in drop foot).
- As you are doing the examination, always think whether the lesion is central (brain and spinal cord) or peripheral (nerves, muscles and neuromuscular junction). UMN signs (increased tone, brisk reflexes and upgoing plantars) are usually an indication of a central lesion. LMN signs (reduced tone, fasciculation, absent reflexes, downgoing or absent plantar response) are usually an indication of a peripheral lesion.
- Patients with sensory ataxia usually have severe impairment of joint position and vibration with positive Romberg's sign. Such patients usually stamp their feet on the floor when walking. Sensory ataxia is usually due to subacute combined degeneration of the cord (vitamin B₁₂ deficiency) and tabes dorsalis (very rare).

- Any cause of severe sensory neuropathy could lead to sensory ataxia, such as paraproteinaemic neuropathy and chronic inflammatory demyelinating polyradiculoneuropathy (CIDP).
- Gait apraxia, marche à petits pas and lower body parkinsonism are different terms used to describe patients who walk with short shuffling steps. This is an indication of diffuse cerebrovascular ischaemic disease (small vessel disease).
- The most common cause of absent ankle jerks and extensor plantars in clinical practice is an elderly patient with diabetes and cervical myelopathy. Other causes include subacute combined degeneration of the cord, taboparesis, Friedreich's ataxia, motor neurone disease and conus medullaris lesion.
- The conus is part of the central nervous system (UMN), whereas the cauda equina is part of the peripheral nervous system (LMN). Lesions involve both parts, producing a mixture of UMN and LMN signs.
- Remember that a short leg with LMN signs and no sensory abnormalities indicates old polio.