

Abnormal gait

Mechanism: Abnormal gait results from failure of any component of the neural or musculoskeletal system (sensory input, central integration, motor pathways, peripheral nerves, muscles, and joints). It is not a single symptom but a final common pathway. The clinical task is to recognize the gait pattern, localize the lesion, and prioritize identifying treatable (normal-pressure hydrocephalus (NPH), myelopathy, vitamin B12 deficiency) or dangerous causes (posterior fossa tumor).

Syndromes:

Spastic gait: limb spasticity leads to knee hyperextension, circumduction, and scissoring.

Cerebellar ataxic gait: wide-based gait ("drunken gait"), dysmetria, and inability to walk in a straight line.

Sensory ataxic gait: loss of proprioception (dorsal columns, neuropathy) worsens gait in the dark and with closed eyes (positive Romberg)

Parkinsonian gait: slow, short, shuffling steps, reduced arm swing, en-bloc turning

Myopathic gait: waddling (Trendelenburg) gait

Steppage gait: leads to high stepping (steppage)

Frontal apraxic gait: dysfunction of frontal-subcortical circuits (NPH, vascular changes) manifests as a "magnetic" shuffling gait

Antalgic gait: pain/mechanical limitation leads to unloading of the limb

Functional gait: inconsistent, incongruent with neurological patterns

Red flags:

- Acute onset of gait disturbance with focal weakness or ataxia (stroke, cerebellar lesion)
- Rapidly progressive or ascending weakness (Guillain-Barré syndrome - GBS)
- Gait change with sensory impairment, back pain, or sphincter dysfunction (spinal cord compression)
- Fever with a newly developed gait disorder (CNS infection, discitis)
- Worsening of gait in a child with headache (posterior fossa tumor)
- Frontal apraxia of gait + cognitive deficit + incontinence - susp. normal-pressure hydrocephalus.

Examination: The main step is determining the type (pathological gait pattern), followed by brain and/or spinal cord imaging immediately upon symptom onset, supplemented by EMG for signs of polyneuropathy, peroneal lesion, and myopathy, and possibly laboratory tests - B12, glycemia, and lumbar puncture including a large-volume tap (tap test).

Treatment: The foundation is therapy for the primary cause (e.g., ventriculoperitoneal shunt in NPH, surgical decompression in myelopathy, vitamin B12 supplementation, dopaminergic therapy in Parkinson disease), supplemented by targeted physiotherapy, rehabilitation aids, and preventive measures against falls.

Onset	Children	Adults
Acute	acute cerebellitis, cerebellar stroke, antalgic gait (trauma, arthritis), intoxication	stroke, vestibular lesion, radiculopathy, trauma
Subacute	ADEM, anti-NMDAR encephalitis, GBS, posterior fossa tumor	GBS, subacute combined degeneration, spinal cord compression
Chronic	cerebral palsy, hereditary ataxia, muscular dystrophy, hereditary spastic paraplegia	Parkinson, cervical myelopathy, neuropathy, NPH, MS

Explanatory notes: ADEM - acute disseminated encephalomyelitis, stroke (CMP), cerebral palsy (DMO), multiple sclerosis (RS).