Abnormal gait

Balance is the ability to stand, and gait is rhythmic stepping movements for travel (locomotion). Balance and gait problems tend to be found in the same individuals.

Walking messages are initiated by the motor and premotor cortex and modified by the subcortical nuclei, brainstem and cerebellum. These all activate the spine's central pattern generator, which coordinates arm and leg movements into rhythmic gait. Proprioceptive, visual, and vestibular inputs reach the spinal central pattern generator and affect its output. The ability to stand and walk normally is therefore dependent on input from several systems, including visual, vestibular, cerebellar, motor, proprioceptive and sensory. Balance and gait therefore require intact brain, spinal cord, and sensory system.

Some changes in gait, balance, and sensorimotor function occur as a result of disease directly affecting these systems, although many are age-related. Gait and balance abnormalities occur in 8-19% of older people, in 14% of individuals aged over 65 years and in 50% of individuals aged over 85 years [1] [2] . See also the separate Gait Abnormalities in Children article.

The National Institute for Health and Care Excellence (NICE) has issued recommendations on recognition and referral of suspected neurological abnormalities. They recommend that [3] :

- Patients with sudden-onset unsteady gait be assessed for stroke/TIA.
- Patients with rapidly progressive (days-weeks) gait ataxia be referred urgently to a neurologist.
- Adults with gradually progressive gait ataxia should be referred to a neurologist, and the GP should take an alcohol history and check B12/folate/thyroid function, and consider coeliac screen.

- Patients with gait apraxia (difficulty initiating and coordinating walking) should be referred to neurology or elderly care to exclude normal pressure hydrocephalus.
- For patients with gait abnormalities at risk of falls, multifactorial falls assessment should be carried out and referral to falls service should be considered.

Editor's note

Dr Krishna Vakharia, 16th October 2023
Suspected neurological conditions: recognition and referral [3]
NICE has recommended that a person should be referred for facial pain, gait unsteadiness and single limb weakness or hemiparesis in line with NHS England's standard on faster diagnosis of cancer. They should receive a diagnosis or ruling out of cancer within 28 days of being referred urgently by their GP for suspected cancer.

Normal gait

As the body moves forwards, one limb typically provides support while the other limb is advanced in preparation for its role as the support limb. The gait cycle (GC) is composed of stance and swing phases. The stance phase is further subdivided into three segments including:

- Initial double stance.
- Single limb stance.
- Terminal double limb stance.

Duration of each aspect of stance decreases as walking velocity increases. The transition from walking to running is marked by elimination of double support.

A stride is the equivalent of a gait cycle. The duration of a stride is the interval between sequential initial floor contacts by the same limb.

Normal age-related changes in gait

Strength changes with age

- Strength peaks in the mid-20s and declines only a little until the fifth decade, after which it falls off much faster.
- All sensory functions also diminish with age, although again this is slow.
- Gait speed remains stable until the seventh decade and slows modestly after this.
- Age-related changes in the balance of older persons result in compensatory responses that meet routine needs but may be ineffective under demanding circumstances. As a result, the mild age-related decreases in strength and balance may contribute to the increased incidence of falls in older people.
- Loss of function caused by disease is of greater impact than age-related change, but it will be superimposed on that caused by age and thus both may contribute to a failure of mobility [4] .

Walking posture changes with age

- Unless older patients have diseases such as osteoporosis with kyphosis, they walk upright with no forward lean.
- Older people also walk with about a 5° greater 'toe out', possibly due to a loss of hip internal rotation or in a subconscious strategy to increase stability.
- Ground clearance as the foot swings is the same in elderly as in younger people.
- Gait velocity remains stable until about the age of 70, then falls about 15% per decade for normal gait. Velocity is lower because older people take shorter steps:
    - One explanation for this is that calf muscles are weaker and cannot produce sufficient plantar flexion.
    - Another is that the elderly are reluctant to generate plantar flexion power because of poor balance and poor control of the centre of mass whilst standing on one foot momentarily.

Limb motion changes with age

- Cadence (rhythm) does not change with age:
    - Everyone has a preferred cadence, which relates to leg length and usually represents the most energy-efficient rhythm for individual body structure.
    - Tall people take longer steps at a slower cadence.
    - Short people take shorter steps at a faster cadence.
- Double stance (the time when both feet are on the ground) increases with age - from 18% of a total gait cycle in young adults to approximately 26% in healthy elderly people:
    - During double stance, the centre of mass is between the feet, which is a stable position.
    - Increased time in the double stance position reduces momentum and therefore reduces time for the swing leg to advance, contributing to short step length.
    - Increased double stance may be needed on uneven ground or when balance is impaired, so that step length is compromised for stability. Elderly people with a fear of falling increase their double stance time.

Joint motion changes with age

- Ankle plantar flexion is reduced during the late stage of stance, just before the back foot lifts off.
- Maximal ankle dorsiflexion is not reduced.

Studies have found a strong association between the severity of age-related white matter changes and the severity of gait and motor compromise. Increasing physical activity may reduce the associated risks [5] .

The presence of neurological gait abnormalities in the elderly (without dementia) is a significant predictor of the risk of development of dementia - especially non-Alzheimer's dementia [6] .

Gait and stability examination

- First, observe the patient entering the room - speed, stride, balance.
- Ask them to walk across the room, turn, and come back.
- Ask them to walk heel-to-toe in a straight line. This may be difficult for older patients even in the absence of disease.
- Ask them to walk on their toes in a straight line, and then to walk on their heels in a straight line.
- Ask them to hop in place on each foot.
- Ask them to do a shallow knee bend.
- Ask them to rise from a chair and walk forwards across the room, turn and come back to you.
- Assess cerebellar function - Romberg's test, finger-nose pointing, dysdiadochokinesia, heel-to-shin testing

Significant observations

- Difficulty rising from a chair (could suggest proximal muscle weakness, balance problems or difficulty initiating movements).
- Shuffling gait which may suggest Parkinsonism. This is also associated with a flat foot strike in heel-to-toe testing with a reduced loading at the heel.
- Balance - do they veer off course? This suggests cerebellar dysfunction.
- Postural sway: this is a feature of late-stage Parkinson's disease and other conditions of poor balance, when balance cannot be maintained through standing, turning or walking. In Parkinson's disease it relates to lack of flexibility in shifting postural responses.
- Rate of walking - note whether they start off slowly and then speed up (a Parkinsonian characteristic) or whether there is general slowness (eg, joint degenerative disease, weakness).

- Nature of steps - look for a steppage gait due to foot drop (loss of dorsiflexion) leading to needing to lift the leg higher than normal when walking. This is associated with conditions such as peroneal nerve injury, fibular injury, multiple sclerosis, Guillain-Barré syndrome, and prolapsed intervertebral disc.
- Difficulty turning: turning problems are common with any gait disorder; turning is generally more difficult than walking. People without balance or gait problems usually can do an 'about-face' in one or two steps. Those with nonspecific problems may need three or four steps. People needing five or more steps are likely to have cerebral or basal ganglia dysfunction. If a patient has less trouble turning than walking forwards, a psychogenic disturbance is likely.
- Widened base. With frank ataxia, base width is about 12 inches. If base width approaches two feet, the likelihood of psychogenic gait disorder rises, unless the patient has morbid obesity or an obvious structural explanation.

The assessment and examination of gait and balance need to be supplemented by appropriate history and examination of all systems. In particular, attention must be paid to speed of onset and rate of any deterioration.

Acute deterioration in gait is a feature of many serious conditions requiring urgent assessment and intervention, amongst them vascular, infective, neoplastic, neurological, metabolic and toxicological conditions and acute confusional states.

Patterns of abnormal gait

Antalgic gait

An antalgic gait is one in which the patient avoids certain movements to avoid acute pain. Typically there is:

- Limited joint range of motion with an inability to bear full weight on an affected extremity.
- Stance phase duration shortened to compensate pain in the affected leg.
- Resultant limp with slow and short steps.

It can be seen as a feature of:

- Trauma
- Osteoarthritis
- Pelvic girdle pain

Gait in cerebellar disease

Ataxia is the most prominent manifestation of cerebellar disease. Ataxic gait consists of arrhythmic (irregular) steps, unsteadiness, wide base, and highly impaired tandem gait. There may be associated cerebellar signs (eg, dysarthria, intention tremor, nystagmus).

Aetiology may include trauma, toxic and metabolic causes, neoplasms, immune mechanisms and genetic diseases. It may also arise from multiple sclerosis. It may also arise developmentally as congenital ataxia, most commonly a non-progressive disorder of children in which co-ordination will usually improve with age. Children aged between 2 and 10 years can present with subacute reversible ataxia stemming from a viral infection.

Typical characteristics of cerebellar gait include:

- Broad-based gait.
- Lurching quality.
- Difficulty with turning.
- Difficulty walking in a straight line.
- Broad-based posture.

Assessment should include asking the patient to walk heel-to-toe. The duration of the problem should be established and knowledge of previous and early motor abilities may be helpful. Diurnal variation with morning unsteadiness which decreases later in the day may suggest raised intracranial pressure.

Patients can present with cerebellar ataxia of extremely acute onset and this may be accompanied by headache, vertigo, vomiting, altered consciousness and neck stiffness. In this case, urgent referral for diagnosis is needed to exclude stroke, cerebral haemorrhage and acute infection. Cerebellar ataxia is one of the rare neurological complications of varicella.

Gait in Parkinsonism

The bradykinesia and slowness of postural adjustments, together with a forward-flexed posture, produce the 'festinant gait' typical of Parkinson's disease. Parkinsonian gait has several features, including:

- Short steps ('petit pas', in which the heel lands less than one foot-length ahead of the toes of the other foot).
- Reduced arm swing.
- Stooped posture.
- Anteropulsion/retropulsion (centre of gravity is ahead of or behind the feet, causing forward or backward acceleration).
- Festination (hasty but short steps attempting to compensate for displaced centre of gravity).
- Postural instability, evidenced when the patient attempts to stand up without the use of his or her arms (he or she tends to fall back into the seat) or when the physician pushes on the chest or back of the standing patient (the patient will have more difficulty than most in maintaining position).

Frontal gait disorder

This describes a combination of findings seen in patients with cerebral tumours, subdural haematomas, dementing illness, normal pressure hydrocephalus, and multiple lacunar infarcts. Characteristic findings are:

- Slow, shuffling, wide-based gait ('marche a petit pas').
- Hesitation in starting to walk.
- Difficulty picking feet off the floor.
- Poor postural control.

Motor function of the legs is sometimes much better when seated or lying, suggesting an element of gait apraxia.

Some of these findings resemble Parkinsonism, but the distinguishing features of frontal gait disorder are its wide base, normal arm swing, absence of other Parkinsonian features, more upright posture, and higher incidence of dementia and urinary incontinence. Causes include frontotemporal dementia, frontal lobe degeneration and normal pressure hydrocephalus.

Cautious gait

- This is a careful gait, slow and wide-based with abducted arms, similar to that of walking on ice.
- It is often age-related.
- Causes include prior falls, deconditioning, and sensory deficit (eg, low sight).

Hemiparetic gait

After a cerebrovascular event:

- The affected limb is generally extended and circumducted.
- The strong gluteal and quadriceps muscle groups are generally spastic.
- The hip flexors, hamstrings and dorsiflexors of the foot are generally weak.
- The hip and knee will thus be stiff and slightly flexed.
- The foot will be plantar-flexed and tending to drag along the floor.

This indicates pyramidal pathway damage - and the residual power left is dependent on non-pyramidal pathways and there being enough residual cortical function.

Paraparetic gait

Damage to the descending corticospinal tract (eg, by a tumour) may present initially with a generalised stiffening of the legs. The patient may find it impossible to walk quickly or run. Features are:

- A stiff, scissor-like walk with leg adduction and extension.
- Ankle clonus may be present, and it develops into a spastic, foot dragging 'shoe-scuffing' gait.

The condition is associated with bilateral leg weakness and hyperreflexia.

Causes include spinal cord lesions and bilateral cerebral hemisphere abnormalities.

Scissor gait

Scissor gait is usually seen in spastic cerebral palsy, usually diplegic and paraplegic varieties. Characteristic features include:

- Legs flexed slightly at the hips and knees, giving the appearance of crouching, with the knees and thighs hitting or crossing in a scissor-like movement.
- Often mixed with, or accompanied by, spastic gait - a stiff, foot-dragging walk caused by one-sided, long-term muscle contraction.
- The individual being forced to walk on tiptoe unless the dorsiflexor muscles are released by an orthopaedic surgical procedure.
- Muscle contractures of the adductors, resulting in thighs and knees rubbing together and crossing in a manner analogous to scissors.
- Complicated assisting movements of the upper limbs when walking, which may also be evident.

These features are typical and are usually present to some degree regardless of the mildness or severity of the cerebral palsy.

Vestibular gait

Patients with unilateral vestibular disorders:

- Veer to the affected side (as opposed to a generalised instability with bilateral vestibular or cerebellar disorders).
- There is a wide-based gait and their difficulties are exaggerated by asking them to walk heel-to-toe.

Many patients respond well to a simple home programme of vestibular rehabilitation head movement exercises. This results in reduced symptoms of imbalance during stance and gait.

Trendelenburg gait

The gluteus medius is very important during the stance phase of the gait cycle to maintain both hips at the same level. One leg stance accounts for about 60% of the gait cycle. During the stance phase of the gait cycle, there is approximately three times the body weight transmitted to the hip joint. The hip abductors' action accounts for two thirds of that body weight. A Trendelenberg gait is the result:

- During the standing phase, the weakened abductor muscles allow the pelvis to tilt down on the opposite side to decrease the workload on the hip abductors. To compensate, the trunk moves to the weakened side to attempt to maintain a level pelvis through the gait cycle.
- It can occur when there is muscular dysfunction (weakness of the gluteus medius or minimus) or pain.
- When standing on the right leg, if the left hip drops, this is a positive right Trendelenburg sign (the contralateral side drops because the ipsilateral hip abductors do not stabilise the pelvis to prevent the droop).
- When the patient walks, the body swings to the other side to compensate for hip drop, leading to a compensated Trendelenburg gait.

Trendelenburg gait is also seen in L5 radiculopathy and after poliomyelitis, in which case there is usually also a foot drop.

Strain to the gluteus maximus and gluteus minimus can be caused by overuse of the gluteus medius by sportsmen using glute-isolating equipment. Tendonitis or tears of the gluteus medius can occur after sports injury or with long-term wear and tear. These tears generally cause pain and weakness on the side of the hip (not the groin). The role of the gluteus medius during activities such as walking and running is to dynamically stabilise the pelvis in a neutral position during single leg stance. The muscle tear itself may be relatively painless, and athletic patients are often masters of compensation and able to keep the pelvis in neutral while the lower leg adducts and internally rotates, making diagnosis tricky. Ultrasound may be helpful for diagnosis.

Waddling gait

- This is a swaying, symmetrical, wide-based gait with toe walking.
- This is associated with proximal muscle weakness in the legs and is seen in pregnancy, particularly in the presence of pubic symphysis diathesis, and in multiple sclerosis.

Gait in neuropathic disorders

Typically unsteady, gait in neuropathic disorders is often high-stepping, this being an almost diagnostic feature. Patients may fall over if asked to close their eyes.

Neuropathic disorders can arise from:

- Diabetes.
- Alcohol dependency (also see under 'Gait in psychiatric disorders', below).
- HIV.
- Toxin exposure.
- Metabolic abnormalities.
- Vitamin deficiency.
- Adverse effects of certain drugs (also see under 'Medication-related gait disturbance', below).
- 32-70% of all peripheral neuropathies are idiopathic.

Dementia-related gait

In vascular dementia there is early disturbance in gait, with unsteadiness and frequent, unprovoked falls. Early in the condition this is typically more marked than in Azheimer's disease. There may be focal neurological abnormalities such as visual disturbances (eg, field defects), sensory or motor symptoms (eg, dysphasia, hemiparesis, visual field defects) or extrapyramidal signs (eg, dystonias and Parkinsonian features).

Cautious gait is seen in early Alzheimer's disease. Changes to gait may be subtle at first, presenting initially with a reduction in the speed and stride of walking. Balance disturbance, short-stepping gait and apraxia increase with the severity of disease. Frontal gait disorder is also more common in Alzheimer's disease patients. The degree of impairment is associated with factors related to the severity of the disease (low Mini Mental State Examination (MMSE) and low Activities of Daily Living (ADL) scores), but also to factors such as age, sex, depression, obesity, and the presence of comorbidities[7] .

Gait in psychiatric disorders[2]

In psychiatry, gait disturbances reflecting cortical and subcortical dysfunction are often seen. Specific examples include:

Alcohol dependency

- Alcoholism affects gait at every level of the nervous system.
- Major alcohol-related deficits include cognitive deficits, weakness due to myopathy, asterixis (sudden loss of muscle tone), cerebellar ataxia, chorea, and loss of position sense (sensory ataxia).
- The Wernicke-Korsakoff syndrome of thiamine deficiency includes confusion and ataxia, both of which impact gait (the third is extraocular movement problems).
- Alcoholic neuropathy is a distal, predominantly sensory or sensorimotor polyneuropathy. The dysaesthesia of alcoholic neuropathy sometimes discourages walking.
- Alcoholic cerebellar degeneration affects mostly the vermis, leading to a wide-based gait, poor tandem gait, and perhaps leg ataxia, but usually no arm ataxia.

Schizophrenia

- Schizophrenia is consistently associated with mild Parkinsonism and ataxia, regardless of medications.
- Often the gait is slower, stride length shorter, and tandem gait mildly impaired.

Depression

Depressed patients occasionally have noticeable Parkinsonism that resolves with recovery from the depression. Gait normalises as the mood disorder improves.

Psychogenic or 'hysterical' gait disorders

- These have been described in the literature over a period of 150 years, when astasia (inability to stand) and abasia (inability to walk) were noted in patients with intact leg function, sometimes characterised by acrobatic near-falls that appear to require more strength and balance than normal standing and walking.
- Gait may be very slow, and buckling of the knees is common, although turns are often normal.
- Useful clues suggesting psychogenic balance and gait disorders are abrupt onset, selective disability, relation to minor trauma, and improbable longitudinal courses.
- Usually there are anatomical inconsistencies in the features. Presentation may be bizarre or dramatic, often with severe weakness or sudden collapses in the presence of normal physical findings. The related affect may seem inappropriately unperturbed, or unusually hostile to investigatory questions.

Choreic gait

- Wide-based gait, with slow leg raising and simultaneous knee flexion.
- Associated with choreoathetoid movements of the upper limbs.
- Causes include Huntington's chorea and dopaminergic medication.

Medication-related gait disturbance [2]

- Medications are a factor in at least 30% of the elderly with balance or gait problems.
- Polypharmacy (more than four medications) is a risk factor for falls, and psychiatric medications are major offenders
- Selective serotonin reuptake inhibitors (SSRIs) are associated with falls and can cause more postural instability than tricyclics.
- Most medication-related falling can be attributed to cognitive impairment, ataxia, Parkinsonism, and hypotension.
- Ataxia is typically caused by sedative-tranquilisers and anticonvulsants.
- Parkinsonism is usually caused by antipsychotics, less often by other dopamine-blocking agents such as metoclopramide and prochlorperazine, still less often by SSRIs, valproate, and calcium-channel blockers.
- Hypotension can be caused by any number of agents, including several antipsychotics, antidepressants and, of course, antihypertensive drugs.
- Clozapine and valproate, by causing asterixis (negative myoclonus), can cause falling when the legs lose tone. Gabapentin may also cause asterixis-related falls.
- Vestibular functioning can be suppressed to bad effect by meclizine and benzodiazepines and can be ablated by aminoglycosides.

Gait Abnormality Rating Scale[8]

The Gait Abnormality Rating Scale (GARS) is a videotape-based analysis of 16 facets of human gait: the total score represents a rank ordering of risk for falling, based on the number of gait abnormalities recognised and the severity of the gait abnormality[9] . GARS comprises three categories:

- Five general categories.
- Four lower limb categories.
- Seven trunk, head and upper limb categories.

Each item has a score range from 0 (good function) to 3 (poor function). The total GARS score is the sum of the 16 individual items. The total score represents a rank ordering of risk for falling, based on the number of gait abnormalities recognised and the severity of any abnormality identified.

Investigation

Investigation of gait abnormality will depend on the history and examination findings, and therefore the differential diagnosis. Options will of course include MRI and CT, X-rays, ultrasound, blood tests and dynamic electromyography (EMG).

Specialist investigations include three-dimensional kinematic and kinetic studies which can define and describe abnormal muscular activity during the gait cycle, allowing effective treatment. This may be helpful in rehabilitation - for example, after spinal injury or stroke [10] .

Dr Mary Lowth is an author or the original author of this leaflet.

Authored by:	Peer Reviewed by: Dr Laurence Knott	
Originally Published: 20/11/2023	Next review date: 10/03/2020	Document ID: doc_1740

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