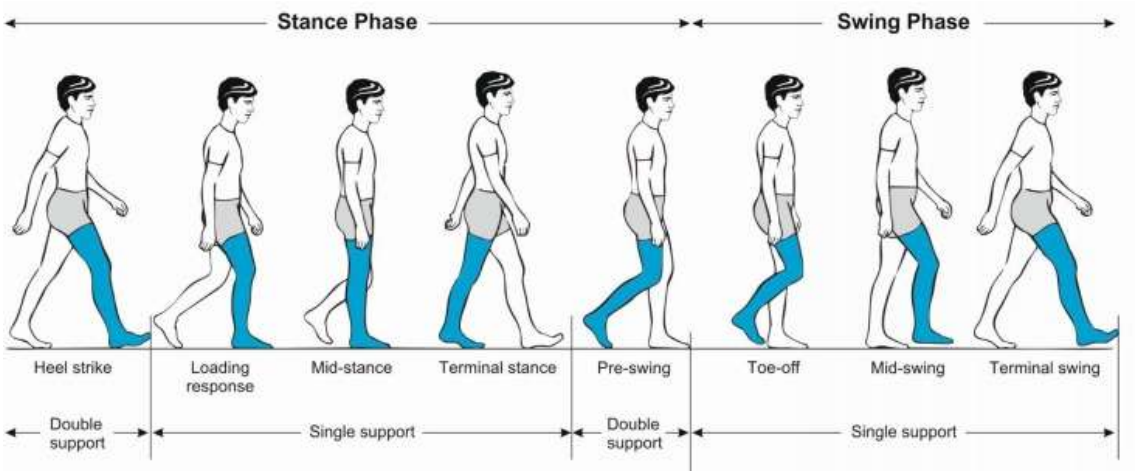
# Gait Variations

Maintaining independent mobility is an important goal of clinical medicine and public health, especially among older persons, who are at the greatest risk for functional decline and disability. People who lose independent mobility are less likely to remain in the community, have higher rates of morbidity, mortality, and self-care disability, and experience a poorer quality of life, with an increased likelihood of depression and social isolation [1].

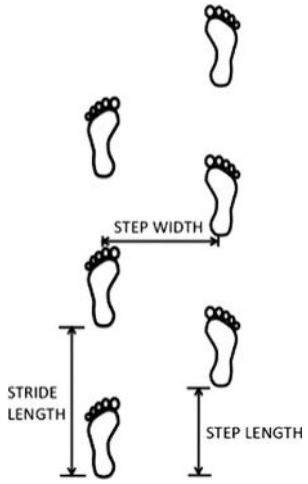
**FEATURES OF SUCCESSFUL GAIT**




[2]

The gait cycle is divided into the stance and swing phase. The stance phase constitutes approximately 60 % of the gait cycle and is subdivided into initial contact (heel strike), loading response, mid-stance, terminal stance and pre-swing. Both feet are on the ground at the beginning and end of the stance phase. Each of these two double support periods lasts for approximately 10–12 % of the gait cycle. The swing phase takes up about 40 % of the gait cycle and is subdivided into initial swing (toe-off), mid-swing (tibia vertical) and terminal swing, terminated by the heel striking the ground [2].

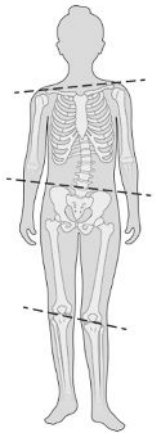
**DEVIATIONS FROM NORMAL GAIT PATTERNS**
(original article: Vazquez-Galliano 2014 Ref 3)




Aging Gait:
Ageing is associated with a decline in gait speed, shorter step length and slower cadence. These changes provide greater stability because both feet are on the ground simultaneously for a greater percentage of the cycle. Elderly subjects prefer a 40 % wider step width than young persons (average step width in elderly women approximately 8 cm and in elderly men 10 cm).

Pathologic Gait:
Pathologic gait results from impaired strength range of motion, proprioception, pain, or balance combined with mechanical compensations, and can have musculoskeletal and neuromuscular aetiologies.

Musculoskeletal aetiologies:



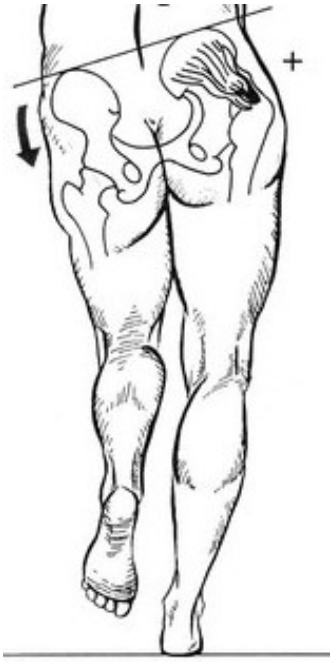
1) Antalgic gait

Pain with weight bearing. Characterised by a shorter step length and stance time on the side of the painful lower extremity. It may be accompanied by ipsilateral trunk lean with hip pain or a contralateral trunk lean with knee and foot pain.

2) Leg length discrepancy

True or apparent leg length discrepancy. Shortened side: pelvic drop, decreased hip and knee flexion, ankle plantar flexion (vaulting, toe walking). Lengthened side: hip hiking, circumduction, excessive hip and knee flexion (steppage gait), foot hyperpronation.

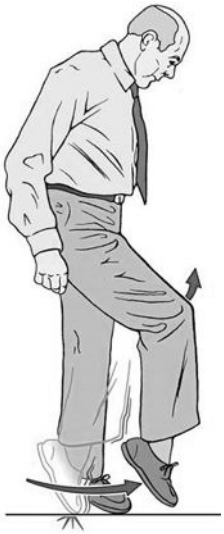
Neuromuscular Aetiologies: Lower Motor Neuron Lesion or other causes of muscular weakness:



1) Hip abductor weakness (Trendelenburg gait):
Excessive downward drop of the contralateral pelvis during stance. Referred to as positive Trendelenburg sign if present during single-limb standing. Compensated gait causes the trunk to lean laterally toward the stance lower extremity of the weak side. If bilateral, it is referred to as a waddling gait.

2) Hip extensor weakness (Posterior lurch gait):
Backward trunk lean with hyperextended hip during stance phase of affected limb. This action moves the line of gravity of the trunk behind the hip and reduces the need for hip extension torque. Use of iliofemoral ligament to lock hip in extension to prevent trunk falling forward.

3) Knee extensor weakness:
Knee buckling (uncompensated), genu recurvatum (compensated). Knee remains fully extended throughout the stance. An associated anterior trunk lean in the early part of stance moves the line of gravity of the trunk slightly anterior to the axis of rotation of the knee. This keeps the knee extended without action of the knee extensors. This gait deviation may lead to an excessive stretching of the posterior capsule of the knee.

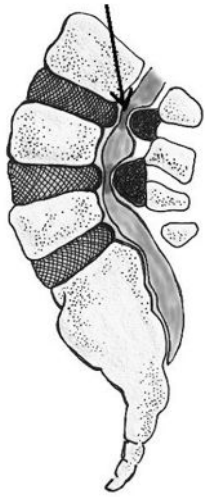


4) Ankle dorsiflexion weakness (Steppage gait and foot slap):

When there is severe weakness, the initial contact with the ground is made by the forefoot followed by the heel region. Mild weakness, the initial contact is made with the heel followed by a rapid ankle plantar flexion, causing a “foot slap” During the swing phase, toes may drag/catch. Compensatory is “steppage gait” with excessive hip and knee flexion or body shift to clear foot.

5) Ankle Plantar Flexor Weakness (Calcaneal gait):

Heel remains in contact with the ground late in terminal stance. Lack of eccentric contraction of ankle plantar flexor causes GRF to pass behind knee, creating knee flexion moment with excess tibial motion over ankle during mid to late stance. Increased quadriceps contraction needed to prevent knee buckling. Contralateral leg step length (swing duration) reduced by unstable stance of affected limb. Heel off delayed and push off phase decreased.



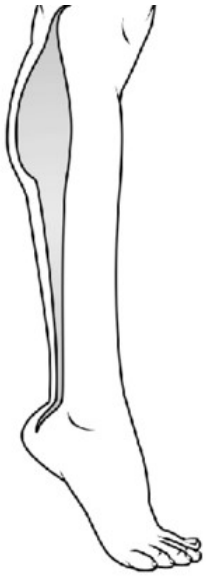
6) Myopathic gait (Waddling gait):

Proximal muscle weakness (hip girdle muscles) resulting in combination of posterior lurch and bilateral Trendelenburg, “waddling gait”. Toe-walking with disease progression. Compensatory mechanism moves GRF anterior to knee joints to prevent knee buckling from extensor weakness.

7) Neurogenic Claudication:

Pain and neurological deficits with ambulation. Usually secondary to lumbar spinal stenosis. Will have hyperlordosis with stenosis and vertebral shift if walking/standing. Pain and neurologic deficits better with spinal flexion – better walking uphill or cycling. Symptoms worse on hyperextension.

Neuromuscular Aetiologies: Upper Motor Neuron Lesion or other causes involving the central nervous system:



1) True Equinus Gait:

Ankle in plantar flexion throughout stance and hips/knees extended related to spasticity/contracture of ankle plantar flexors or weakness of ankle dorsiflexors. May present with compensatory genu recurvatum.

2) Jump Gait:

The ankle is in equinus, the knee and hip are in flexion, there is an anterior pelvic tilt and an increased lumbar lordosis. May be related to hamstring/hip flexor spasticity/contractures. May present with “stiff knee gait” from hamstring quadriceps co-contraction.

3) Apparent Equinus:

Not true equinus but walk in equinus position to compensate for severe hip and knee flexion throughout the stance phase; the ankle has a normal range of dorsiflexion. May be related to spasticity/contractures of hip/knee flexors.

4) Crouch Gait:

Excessive dorsiflexion or calcaneus at the ankle in combination with excessive flexion at the knee/hip. Common with severe diplegia and spastic quadriplegia, especially in older or overweight children. Most common cause in spastic diplegia: isolated lengthening of heel cord in younger children without control of spasticity/contracture of hamstrings/iliopsoas, leading to rapid increase in hip/knee flexion. Result is energy-inefficient gait, with anterior knee pain, patellar pathology in adolescence. Also caused by botulinum injections to gastroc-soleus without addressing hamstrings/iliopsoas or providing adequate orthotic support.

5) Parkinsonian Gait:
Rigidity (pelvis/thorax rotating en bloc, reduced arm swing), postural instability (trunk flexion, protracted shoulders, flexed hips/knees), bradykinesia (slow motion, inability to change direction) with possible festination.

6) Cautious Gait:
Associated with older age-related brain disease or dysfunction and fear of falling. Slow, wide based gait, with stooped posture and reduced arm swing.

7) Spastic Gait: Usually secondary to brain injury

Hemiparetic: Paretic side with flexor muscle synergy noted. Typical features are as follows for the affected side: Upper arm is adducted and internally rotated with flexion at the shoulder, elbow, and wrist. Hip and knee flexed. Foot inverted and plantarflexed. Lower leg circumducts during swing phase, also known as "Wernicke-Mann gait."

Paraparetic: similar to hemiparetic gait but involves bilateral lower legs. Gait is spastic, spastic and ataxic and/or scissoring.

8) Cerebellar Ataxic Gait:
Usually secondary to cerebellar injury. Balance worse than leg placement. Differentiate from sensory ataxia which is proprioceptive deficits from dorsal column injury and worse with Romberg testing. Cerebellar gait initiation is normal. However, leg coordination is inconsistent and "broad-based, insecure, and wobbly." Unilateral cerebellar lesions have ipsilateral ataxia, whereas vermis causes truncal ataxic gait.

9) Frontal Gait Ataxia
Usually secondary to injury to frontal lobe and connecting networks. May be secondary to vascular disease or hydrocephalus. May involve other lobes of brain and associated with Alzheimer's disease. Difficult to classify condition. Not Parkinsons disease or any neurologic tremor. Patients have lost the ability to walk, change positions from sit to stand, and have a broad base gait with short stride length and are unable to initiate gait on own. Will also have en bloc gait, freezing episodes, and retropulsion.

1. Gill TM, Gahbauer EA, Murphy TE, Han L, Allore HG. Risk factors and precipitants of long-term disability in community mobility: a cohort study of older persons. Ann Intern Med. 2012 Jan 17;156(2):131-40.
2. Pirker W, Katzenschlager R. Gait disorders in adults and the elderly : A clinical guide. Wien Klin Wochenschr. 2017 Feb;129(3-4):81-95.
3. Vazquez-Galliano J, Kimawi I, Chang L. Biomechanics of gait and treatment of abnormal gait patterns. Physical Medicine and Rehabilitation. 2014.