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Gait Disorders

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ABSTRACT

Walking is an extraordinarily complex task requiring integration of the entire nervous system, making gait susceptible to a variety of underlying neurologic abnormalities. Gait disorders are particularly prevalent in the elderly and increase fall risk. In this review we discuss an approach to the examination of gait and highlight key features of common gait disorders and their underlying causes. We review gaits due to lesions of motor systems (spasticity and neuromuscular weakness), the cerebellum and sensory systems (ataxia), parkinsonism, and frontal lobes and discuss the remarkably diverse phenomenology of functional (psychogenic) gait disorders. We offer a pragmatic approach to the diagnosis and management of neurologic gait disorders, because prompt recognition and intervention may improve quality of life in affected individuals.
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KEYWORDS: Ataxia; Foot drop; Frontal gait; Functional movement disorder; Gait disorders; Parkinsonism; Spasticity

Gait disorders are common, contribute significantly to morbidity through falls,[1] and may yield clues to diseases occurring at all locations of the nervous system, making the examination of gait one of the most complex and high-yield components of the neurologic examination. In this review we offer a pragmatic approach to examining gait and discuss clinical features of common gait disorders and their underlying etiologies. Abnormal gait is particularly prevalent in the elderly, affecting approximately 1 in 3 community-dwelling individuals older than 60 years. Gait disorders in this population are associated with diminished quality of life[2] and nursing home placement[3] and may be an indicator of progression to dementia in individuals with mild cognitive impairment.[4] A history that includes weakness of the legs, imbalance, unsteadiness on one's feet, or multiple falls may hint at an underlying gait disorder. Prompt recognition, examination, and classification of gait disorders is therefore of paramount importance.

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PHYSIOLOGY AND THE GAIT CYCLE

Normal gait requires precise control of limb movements, posture, and muscle tone, an extraordinarily complex process that involves the entire nervous system. Specialized groups of neurons in the spinal cord and brainstem generate rhythmic activity and provide output to motor neurons, which in turn activate muscles in the limbs. The cerebral cortex integrates input from visual, vestibular, and proprioceptive systems; additional input is received from the brainstem, basal ganglia, cerebellum, and afferent neurons carrying proprioceptive signals from muscle stretch receptors (as may be damaged in peripheral neuropathy). Together, these systems allow individuals to walk not only in a straight, unencumbered line but to adapt their gait to avoid obstacles and adjust posture to maintain balance.[5] Abnormalities of any portion of the nervous system can therefore give rise to a gait disorder.

The gait cycle (**Figure 1**) begins when one heel (illustrated here as right) strikes the ground. Supported by the stance of the right leg, body weight shifts forward as the left leg flexes at the hip and knees and swings forwards, eventually striking the left heel on the ground. Weight then shifts forwards on the left leg, while the right leg swings forward and again strikes the ground. Thus, while one leg is in *stance* phase, the opposite is in *swing* phase. Periods of double support, during which both legs make contact with the ground, normally comprise approximately 10% of the gait cycle[6] but increase as compensation for unsteadiness in many abnormal gaits.

EXAMINATION OF GAIT

The examination of gait begins with observing a patient as he or she walks from the waiting area to an examination room. The ideal setting for a formal gait examination is a long, uncluttered hallway, providing enough distance to reach a comfortable walking speed with good arm swing. Hands should be free except for necessary assistive devices. Observe individuals as they walk in a straight line, but also note any difficulty rising from a chair, initiating gait, or turning. The gait examination provides significant insight into an individual's functional status, and much will be missed if the assessment is limited to the examination room! Make note of *velocity* (distance covered in a given time) and *cadence* (steps per minute). *Stride length* measures distance covered by the gait cycle; *step length* measures the distance covered during the swing phase of a single leg. *Step width* or *base* is the distance between the left and right feet while walking (**Figure 2**). Also make note of posture, arm swing, the height of each step, leg stiffness, or side-to-side lurching. Muscle strength and tone in the legs, sensation, and reflexes may provide further clues as to the etiology of an underlying gait disorder. The Romberg sign is tested by asking patients to stand still with feet together and eyes closed and is considered positive (abnormal) if eye closure provokes a fall. Test tandem gait by asking a patient to take at least 10 steps touching heel-to-toe, as if walking on a tightrope. Heel or toe walking can unmask subtle distal weakness that might be missed by direct confrontational testing.

CLINICAL SIGNIFICANCE

- Gait disorders increase fall risk and often result from an underlying neurologic condition.
- Specific features of abnormal gaits result from a combination of a deficit and attempts at compensation.
- Many gait disorders are readily treatable with specific therapies, such as dopaminergic therapy for Parkinson's disease, or cerebrospinal fluid shunting for normal pressure hydrocephalus.
- Physical therapy and assistive devices may improve mobility and decrease fall risk.

CLINICAL FEATURES AND ETIOLOGY OF GAIT DISORDERS

Gait disorders may be neurologic or nonneurologic in origin. Common nonneurologic causes of abnormal gait include osteoarthritis of the hip and knee, orthopedic deformities, and visual loss[2]; individuals may reduce the stance time of the affected limb to reduce pain, resulting in an asymmetric *antalgic gait*. Common neurologic causes of abnormal gaits are listed in the **Table** and are described here in further detail. Mildly shortened step length, decreased velocity, slightly widened base, and increased double support time are features of normal aging[7] but are also seen as a response to perceived instability, either intrinsic (eg, disequilibrium) or extrinsic (eg, walking on ice). Individuals may walk with hands outstretched in an attempt to steady themselves. This *cautious gait* is nonspecific but may herald an underlying neurologic gait disorder.

Spastic Gait

Spastic gaits are caused by lesions in the corticospinal tract at any level and may be unilateral or bilateral. When unilateral, the affected leg is held in extension and plantar flexion; the ipsilateral arm is often flexed. There is circumduction of the affected leg during the swing phase of each step. Common causes include stroke or other unilateral lesions of the cerebral cortex. If bilateral, the spastic gait may appear stiff-legged or *scissoring* owing to increased tone in the adductor muscles, such that the legs nearly touch with each step

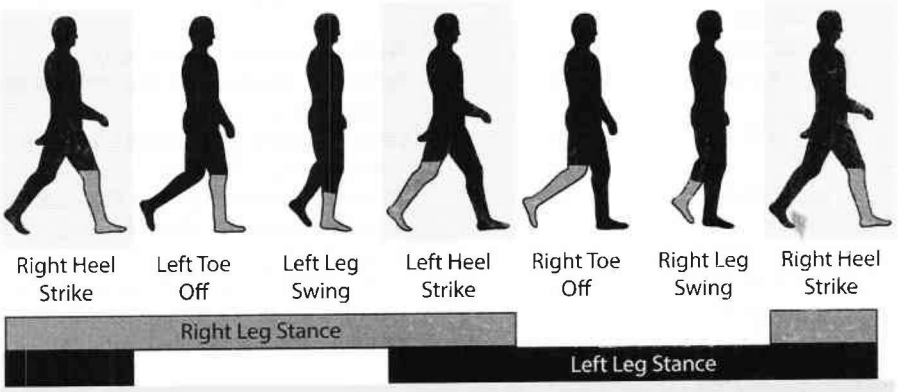

Figure 1 The gait cycle. Right leg is shaded grey. The gait cycle is divided into stance and swing phases. During stance, body weight shifts forward on the supporting leg, while the opposite leg swings forward, eventually making contact with the ground via the heel. Shaded boxes indicate periods of double support, during which both the left and right legs make contact with the ground.

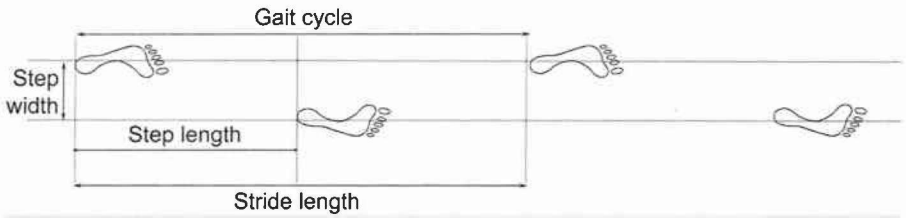

Figure 2 Terminology describing the gait cycle. Reproduced with permission from Pirker and Katzenschlager.[6]

(**Figure 3B**). Common causes of bilateral spastic gait (*spastic paraparesis*) include cerebral palsy, cervical spondylotic myelopathy, and multiple sclerosis, among many others, and are often accompanied by signs of myelopathy, such as bowel and bladder dysfunction, increased reflexes, and Babinski signs. Antispasticity agents such as baclofen or tizanidine are variably effective in improving gait but may reduce painful spasms. Botulinum toxin injections may be useful in cases of focal spasticity.

Neuromuscular Gaits

Weakness of muscles of the lower extremities may manifest as a gait disorder. The *waddling gait* can be seen in cases of proximal muscle weakness, such as myopathy. In normal gait the gluteal muscles serve to stabilize the pelvis, elevating the non–weight-bearing side with each step. With weakness of these muscles, and particularly the gluteus medius, instability of the weight-bearing hip instead causes the non–weight-bearing side to drop (Trendelenburg's sign). This leads to excessive side-to-side trunk motion, giving the gait a waddling appearance.[8] Individuals with proximal muscle weakness often have difficulty rising from a chair without using their arms.

The *steppage gait* is caused by weakness of ankle dorsiflexion, also known as a *foot drop*. Individuals with a steppage gait lift the swinging leg higher to compensate for the toes' inability to clear the ground with each step; the foot landing often has a slapping quality. Weakness of ankle dorsiflexion may be appreciated by direct testing on physical examination, though more subtle weakness may be elicited when an individual is asked to walk on his or her heels. Foot drop may be bilateral, as can be seen in peripheral polyneuropathy, or unilateral. Common causes include an L5 radiculopathy or peroneal neuropathy, which can be differentiated from the former by preservation of ankle inversion on physical examination. Electromyography with nerve conduction studies may aid in diagnosis. Individuals with foot drop may benefit from ankle foot orthoses, which stabilize the ankle in a neutral position.

Parkinsonian Gait

The parkinsonian gait is among the most common gait disorders in the elderly. The classic "shuffling" appearance is

Table Prevalence of Neurologic Gait Disorders in 117 Community-Dwelling Adults

Neurologic Gait Disorder (GD)	Number (Percentage)*	Total Number†	Causes (Number)
Single neurologic GD	81 (69.2)		
Sensory ataxic	22 (18)	46	Peripheral sensory neuropathy (46)
Parkinsonian	19 (16.2)	34	Parkinson's disease (18), drug-induced parkinsonism (8), other (4)
Frontal	9 (7.7)	31	Vascular disease (20), normal pressure hydrocephalus (1), dementia (7), other (3)
Cerebellar ataxic	7 (6.0)	10	Stroke (3), multiple sclerosis (1), essential tremor (3), chronic alcohol abuse (1), other (2)
Cautious	7 (6.0)	7	Idiopathic (7)
Paretic/hypotonic	6 (5.1)	14	Lumbar spinal stenosis (7), peripheral nerve injury (5), other (3)
Spastic	6 (5.1)	7	Ischemic stroke (3), intracerebral hemorrhage (3), congenital (1)
Other	5 (4.3)	10	Vestibular disease (6), dyskinetic (4)
Multiple neurologic GD	36 (30.8)		
Total	117		

Modified and used with permission from Mahlknecht P., Kiechl S., Bloem B.R., Willei J., Scherfler C., Gasperi A., Rungger G., Poewe W., Seppi K. Prevalence and burden of gait disorders in elderly men and women aged 90-97 years: a population-based study. *PLoS ONE*. 2013;8:e69627.

*Percentage represents individuals with a single gait disorder as a proportion of the entire study population.

†Total number of individuals with each gait disorder, includes individuals with multiple causes of gait disorders. For example, 22 of 117 individuals had an *isolated* sensory ataxic gait disorder, 24 individuals had sensory ataxia and an additional neurologic gait disorder.

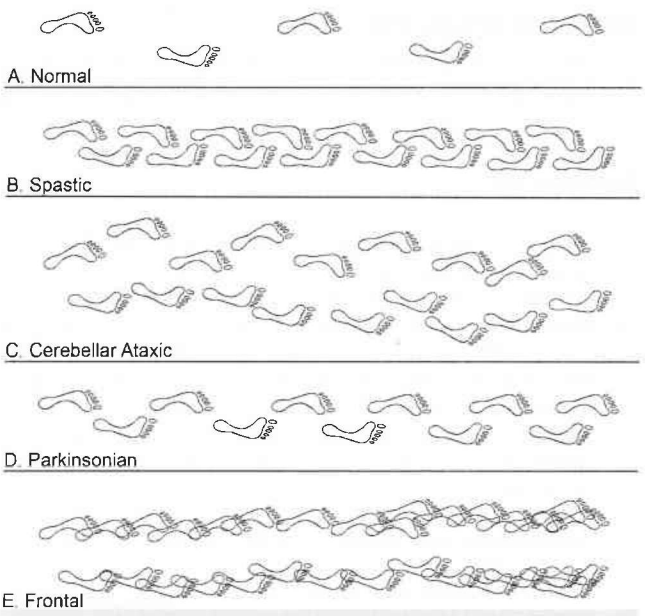

Figure 3 Graphic representation of classical gait disorders. **(A)** Normal step length and width. See text for details. Reproduced with permission from Pirker and Katzenschlager.[6]

caused by a decrease in both step length and height; posture is stooped, arm swing is reduced, and the base is narrow to normal (**Figure 3D**). Parkinsonian turns are characterized by simultaneous rotation of the head, trunk, and pelvis, the so-called *en bloc turn*; in normal individuals, the head rotates first, followed by the trunk then pelvis.[9] Parkinson's disease is typically asymmetric at onset, so arm swing and step length are diminished more on the affected side. Asymmetric shuffling can often be heard as scuffing of one foot more than the other. A parkinsonian (resting) tremor may activate during walking. Step length, velocity, arm swing, and turning speed improve with dopaminergic treatment.[10]

Freezing of gait and *festination* are features of more advanced Parkinson's disease. Freezing is defined as "an episodic inability (lasting seconds) to generate effective stepping" despite the intention to walk.[11] Affected individuals feel as if their feet are stuck to the floor, often associated with alternating trembling of the legs. Freezing is commonly seen while initiating gait, turning, or approaching a destination but can also be provoked by features of one's environment, such as narrow hallways, doorways, or even large crowds.[12] Freezing is a major contributor to fall risk.[13] Freezing may improve with optimization of dopaminergic medications. If freezing persists despite medication adjustment, symptoms may improve with visual or auditory cueing. For example, individuals may avoid or overcome freezing by consciously stepping over a line on the floor, or marching to the beat of a metronome. A laser line produced by an attachment to a cane or walker may be a particularly effective intervention.[14,15] *Festination* describes a phenomenon in which steps become increasingly rapid and short, so that gait takes on the appearance of running. The center of gravity moves forward. Festination may precede freezing but also occurs independently and further contributes to fall risk.[16]

Cerebellar Ataxic Gait

Lesions of the cerebellum cause irregular, uncoordinated movements called *ataxia*. Ataxia of the limbs (appendicular ataxia, as might be assessed with finger-nose-finger testing) is typically caused by lesions of the cerebellar hemispheres, whereas ataxia of gait (truncal ataxia) is caused by midline lesions of the cerebellar vermis.[17] In its mildest form the cerebellar ataxic gait may manifest only as difficulty with tandem gait; individuals may sway or fall when asked to walk heel-to-toe. In more severe form the gait is wide-based (to compensate for instability), step length is variable, turns are unsteady, and there is frequent side-to-side lurching or deviation (**Figure 3C**). Symptoms do not clearly worsen with eye closure. Assistive devices such as a walker may decrease the risk of injury due to falls. The broad differential diagnosis of cerebellar gait ataxia may be narrowed by time course of symptom onset; stroke is a common cause of acute ataxia, autoimmune or other inflammatory disorders are often subacute, and neurodegenerative conditions or alcohol may cause chronic cerebellar ataxia. Imaging of the brain is typically warranted.

Sensory Ataxic Gait

Individuals with deficits of proprioception (limb and joint position sense) are unable to sense the position of their feet relative to the ground, resulting in an unsteady gait. The stance is wide-based, with a shortened step length and stomping quality as the foot hits the ground. Visual cues may partially compensate for proprioceptive deficits, so affected individuals often look down at their feet while walking. Gait therefore worsens dramatically in the dark or with eyes closed, a feature useful for differentiating sensory from cerebellar ataxia. Romberg's sign is present. Sensory ataxic gaits are commonly caused by lesions of peripheral nerves or the dorsal columns of the spinal cord, such as with syphilis or vitamin B12 deficiency. Joint position and vibratory sense are diminished in the lower extremities; loss of reflexes suggests the presence of a peripheral neuropathy. Many patients improve with physical therapy.[18]

Frontal Gait

Higher-level gait disorders encompass a class of gaits not caused by lesions of the corticospinal tract, basal ganglia, cerebellum, or neuromuscular systems.[19] Among the most common of higher-level gait disorders is the *frontal gait*, typically caused by lesions of the frontal lobes (**Figure 3E**). Impaired balance is a core feature, and like cerebellar and sensory ataxia, step width is also widened. Step length is decreased and variable, and step height is diminished. Failure to initiate gait is a prominent feature; feet may appear glued to the floor when an individual attempts to begin walking, a feature that may also occur in isolation.[19] Freezing with turns is common. These features lead to the classic description of this gait as "magnetic." Common causes include microvas-

cular white matter disease, vascular or neurodegenerative dementias, and normal pressure hydrocephalus (NPH), the latter of which may be accompanied by cognitive decline and urinary incontinence and should be excluded by imaging. Only a small proportion of individuals with NPH present with the complete triad, so suspicion should be high in the setting of enlarged ventricles and a compatible gait disorder. Gait abnormalities are an early feature of NPH and are also the most likely to respond to cerebrospinal fluid shunting.[20] For individuals with frontal gaits from disorders other than NPH, physical therapy and assistive devices may improve ambulation and decrease fall risk.

Functional (Psychogenic) Gait Disorder

Functional gait disorders, formerly referred to as "psychogenic," frequently co-occur with other functional neurologic disorders and are common in clinical practice. Though their presentation is heterogeneous, functional gait disorders are typically abrupt in onset, fluctuate over time, and are both suggestible and easily distractible. Common patterns include excessive slowing of gait or buckling of the knees, usually without falls.[21] Abnormal twisting or muscle contractions may superficially resemble dystonia. *Astasia-abasia* describes an inability to stand or walk without support, despite ability to otherwise use the legs normally. Bizarre, inefficient postures that appear unsteady yet do not result in falls are another feature of functional gait disorders.[22] Mood disorders are present in a substantial number of patients but are not required for diagnosis.[21]

The diagnosis of a functional gait disorder should be made not purely by exclusion of organic disease but by positive identification of internal inconsistencies or distractibility. For example, functional gait disorders or postural instability may normalize when an individual is asked to walk while talking on the phone. Sharing these inconsistent features with the patient highlights their potential reversibility and may be therapeutic. Communicating the diagnosis should focus more on positive features than diseases that have been excluded and emphasize mechanism over etiology. The metaphor of "a problem with the software, not the hardware" may be particularly effective.[23] Treatment often involves a multidisciplinary team of neurologists and psychiatrists; consensus-based guidelines for physical therapy have been published.[24]

CONCLUSIONS

Gait disorders are a major source of disability, morbidity, and mortality in the elderly and may be neurologic or nonneurologic in origin. When neurologic in origin, gait disorders may arise from lesions in any part of the nervous system. This review has provided an overview of the clinical features of various gait disorders, emphasizing clinical features allowing for prompt recognition, offering an opportunity for effective intervention and improvement in quality of life.

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