Gait Disorders

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Drs Fasano and Bloem discuss the unlabeled use of donepezil and methylphenidate as cognitive enhancers and stimulation of the pedunculopontine nucleus as a surgical indication for the treatment of gait and postural disorders.

ABSTRACT

Purpose of Review: This article provides insight and reviews useful tools for the clinical assessment, understanding, and management of neurologic gait disorders.

Recent Findings: In recent years, our understanding of the physiology of human walking has steadily increased. The recognition of gait as a complex, ''higher-order'' form of motor behavior with prominent influence of mental processes has been an important new insight, and the clinical implications of gait disorders are increasingly being recognized. Better classification schemes, the redefinition of established entities (eg, senile gait), and new insights from research on degenerative disorders primarily affecting gait (eg, primary progressive freezing of gait) have become available.

Summary: Gait disorders are directly correlated with poor quality of life and increased mortality. Because gait is very sensitive to any insult to the nervous system, its assessment should be carried out carefully in routine clinical practice.

Disorders of locomotion are easily discernible to the naked eye. However, when examining gait, clinicians should bear in mind that the clinical phenotype is the net result of changes induced by the disease itself plus any compensations adopted by the patient to improve stability. This review presents a clinically oriented approach to gait disorders based on the dominant phenomenology and underlying pathophysiology, which are tightly connected. The authors conclude by proposing a practical management approach.

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INTRODUCTION

Bipedal locomotion is considered one of the most important human-defining changes in evolution because it allowed for free use of the hands. Many congenital or perinatal psychomotor disturbances first manifest as a delayed initiation of walking. Gait disorders are one of the most common problems encountered in neurologic patients, present in more than half of all non–bed-bound patients admitted to a neurologic service.[1] Gait disorders have devastating consequences, the most notorious being falls and reduced mobility, with subsequent impaired quality of life and a reduced life span (due to a combination of fatal falls, reduced cardiovascular fitness, and death from underlying diseases).

This article briefly describes the physiology of human locomotion and continues in more detail with the methods to clinically evaluate and characterize gait and its disorders based on phenomenology and pathophysiology. The last part of the review will cover the multidimensional strategies to improve gait, aiming to improve mobility and reduce the incidence of falls and fall-related injuries.

THE PHYSIOLOGY OF LOCOMOTION

Walking appears to be a simple, innate ability that we execute seemingly effortlessly every day, but it is an extraordinarily complex and unique motor behavior consisting of three primary components: locomotion, balance, and ability to adapt to the environment (**Figure 8-1**, **Table 8-1**). Normal

gait requires a delicate balance between various interacting systems: three major afferent sensory systems (visual, vestibular, and proprioceptive senses), a locomotor efferent system (including nerves, muscles, bones, joints, and tendons), and the strict surveillance by several structures of the CNS (**Figure 8-2**). The cardiovascular system also participates, providing the ability to stand erect without collapsing because of hemodynamic deprivation.

Suprasegmental Control

Gait control is tightly connected with the attentive resources and other cognitive domains that regulate the strategies employed for goal-directed navigation and management of external challenges, such as balance perturbations, the avoidance of obstacles in the walking trajectory, and other transitions. The main aim of these suprasegmental centers is to preserve stability by integrating the various sources of afferent peripheral information and selecting the strategies that guarantee dynamic stability while minimizing energy expenditure.

Brainstem and Spinal Cord

Cortical locomotor output from the premotor cortex and the supplementary motor area is conveyed to the brainstem locomotor centers via the basal ganglia. The midbrain also receives input from the cerebellar cortex

KEY POINT

- Gait disorders are associated with reduced survival, which can be attributed to a combination of fatal falls, reduced cardiovascular fitness, and death from underlying diseases.

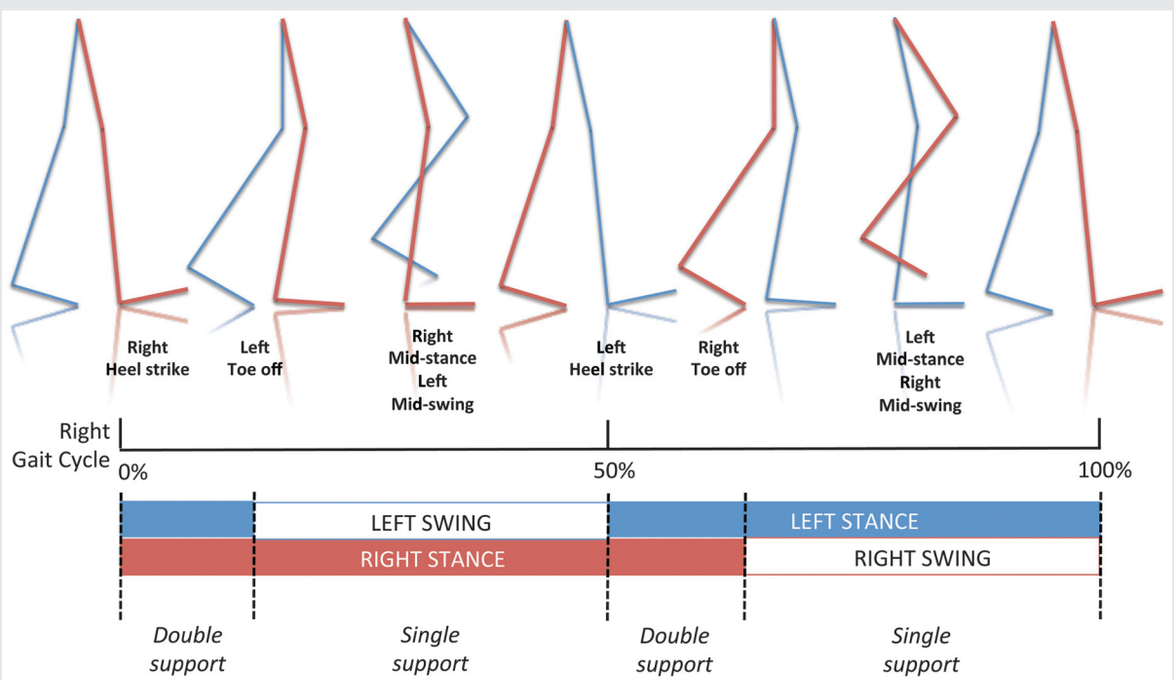

FIGURE 8-1 Locomotion starts with the first shift of the center of body mass over the support foot and tilting of the pelvis in order to lift and swing the first leg. This first step is based on preplanning and execution of a complex motor task and is followed by a more automatic, synchronized, and rhythmic motor planning, which leads to continuous stepping. A *gait cycle* is defined as the period between successive points at which the heel of the same foot strikes the ground (figure illustrates the cycle of the right leg). The stance phase, during which the foot is in contact with the ground, occupies 60% to 70% of the cycle; remaining time is occupied by the swing time, which begins when the toes leave the ground (''toe off'') and, by definition, is equal in time to the single support time of the other leg. During up to 25% of the gait cycle, both feet are in contact with the ground (double-limb support). This cycle period can be virtually absent (as during running), increased (as during cautious/senile gait, weakness, or disequilibrium) or asymmetric (as during limping gait).

TABLE 8-1 Spatiotemporal Indexes of Gait[a]

Index	Definition
Velocity	Walked distance/time (eg, m/s).
Cadence	Number of steps in a given time (eg, steps per minute).
Step length	The distance covered during the swing phase of a given leg (ie, the distance between a toe off and the next heel strike of the same leg).
Stride length	The distance covered during a given gait cycle (ie, the distance between two consecutive heel strikes of the same leg). It is also the summation of the distances of two steps (left and right).
Step width	The distance between the two feet at the perpendicular axis to the walking direction for a given step.
Step height	The maximum distance between the forefoot and ground during the swing time.
Symmetry	The ratio between the step lengths of the two legs.
Spatial/temporal variability	The coefficient of variation of spatial/temporal indices.
Coordination	The timing of leg activation with respect to the other one within a gait cycle.

[a] See Figure 8-1.

to integrate multisensory information (**Figure 8-2**). Interesting new observations (eg, from neuroimaging studies,[2] from postmortem studies,[3] and from recordings of subcuneiform neurons in humans[4]) have extended earlier animal work and established that the mesencephalic locomotor region (MLR) and its descending projections to the pontomedullary reticular formation are involved in gait control in humans as well. The MLR lies just ventral to the inferior colliculus and is composed of the pedunculopontine nucleus (PPN) pars compacta and PPN pars dissipata, the cuneiform nucleus, and the subcuneiform nucleus.[3] MLR is modulated by tonic inhibition coming from the basal ganglia through the globus pallidus internus (GPi) and substantia nigra pars reticulata.

The MLR and other brainstem structures (serotonergic neurons of the median raphe and parapyramidal region, noradrenergic neurons of the locus coeruleus, and dopaminergic neurons of the lateral hypothalamus) send facilitatory signals via the reticulospinal tract to the central pattern generators located in the spinal cord to provide the basic coordinated muscle activation patterns needed to generate locomotion.

Balance

Balance and gait are tightly connected. Control of axial tone is necessary for maintaining upright stance, and lateral weight shifts are needed to free the swing leg and initiate a step.[5] The interdependency between gait and balance is reflected by several clinical observations. Particularly, gait disorders are an established risk factor for falling because (1) impaired stepping

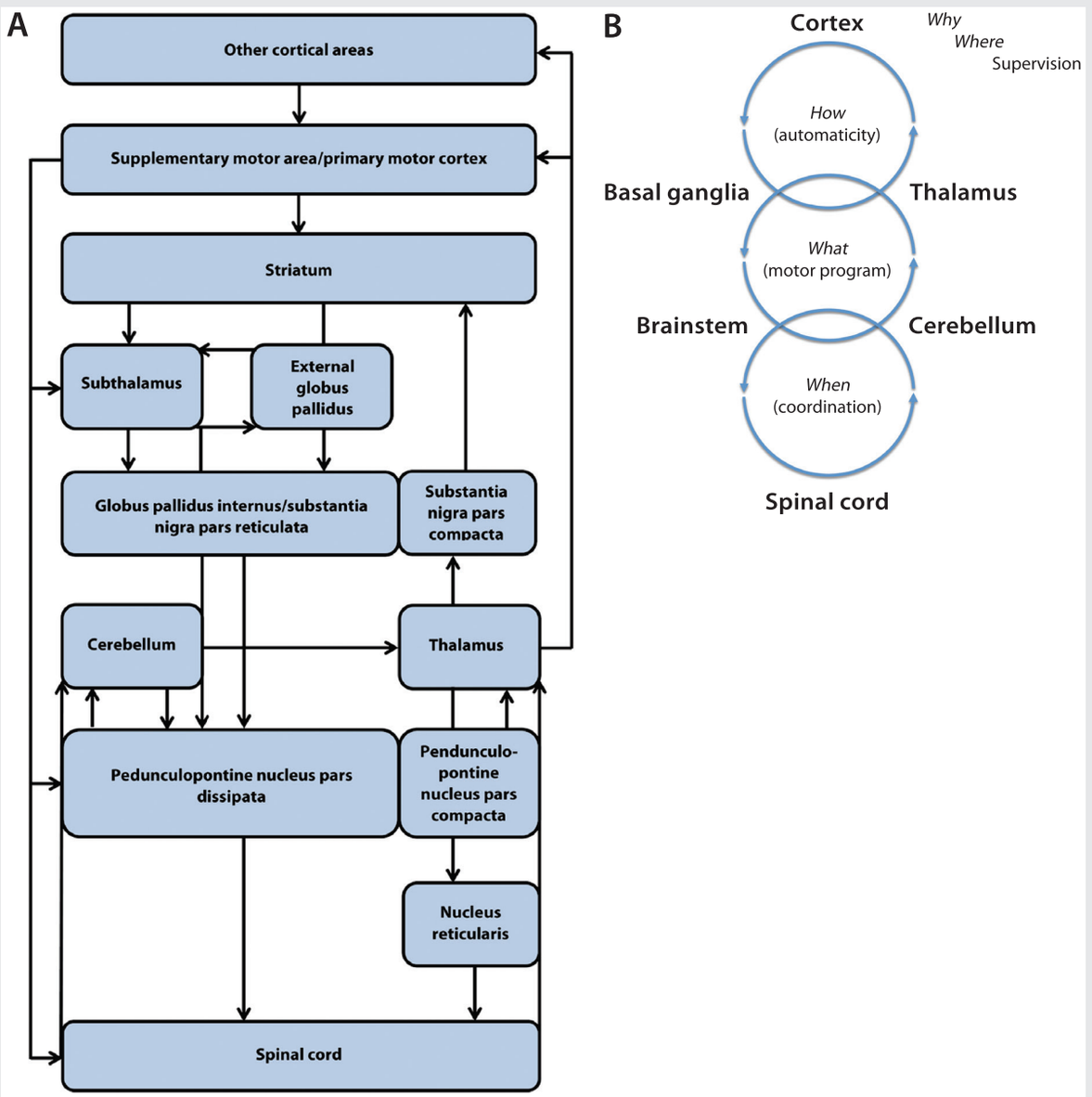

FIGURE 8-2 Anatomic (*A*) and physiologic (*B*) interaction between the CNS centers contributing to locomotion. The most important structures for control of locomotion and balance are the frontal cortex (supplementary motor area especially), basal ganglia, and the mesencephalic locomotor region (and particularly the pedunculopontine nucleus). Following the integration of cognitive, sensory, and limbic cortical inputs, the frontal cortex projects heavily to the brainstem, activating the structures important for both postural control and locomotion. These subcortical structures are modulated by the tonic inhibition exerted by the basal ganglia and cerebellum and exert a supraspinal control of spinal segmental reflexes and alpha motoneurons responsible for movement.

Data from Pahapill PA, Lozano MA, Brain.[3] *brain.oxfordjournals.org/content/123/9/1767.long* and Nutt JG, et al, Mov Disord.[5] *onlinelibrary.wiley.com/doi/10.1002/mds.23588/abstract;jsessionid=154012B88A337A599DC35A3C54EB591F.d03t01*.

can destabilize the body's center of mass during walking; (2) disorders can impair CNS regions involved in controlling both gait and postural stability; (3) an impaired ability to step interferes with the ability to rapidly correct for external postural perturbations; and (4) gait impairment can be a secondary

KEY POINTS

- Gait disorders are an established risk factor for falling because (1) impaired stepping can destabilize the body's center of mass during walking; (2) disorders can impair CNS regions involved in controlling both gait and postural stability; (3) an impaired ability to step interferes with the ability to rapidly correct for external postural perturbations; and (4) gait impairment can be a secondary epiphenomenon of disorders primarily affecting postural stability.
- Antalgic gait is a compensatory gait that reduces the stance phase in the affected limb to minimize pain; it is associated with pain and limited range of joint movement.
- Fall phobia is a heterogeneous syndrome clinically characterized by a disproportionate fear of falling after even a single fall or near-fall, which further immobilizes patients and affects their quality of life.
- Patients with reckless gait (''careless gait'') walk too quickly and act impulsively (eg, a very rapid rise from a seated position: the ''rocket sign''). Typically, these patients have frontal-dysexecutive impairment.

epiphenomenon of disorders primarily affecting postural stability.

CLASSIFICATION OF GAIT DISORDERS

Several classifications of gait disorders have been proposed (**Table 8-2**). Nutt and colleagues first classified these hierarchically into lower-level, middle-level, and higher-level disorders.[6] Other classifications have been based on anatomy (eg, frontal gait disorder) or on etiology (eg, vascular), but these are prone to mischaracterization.

A classification based on the dominant clinical phenomenology has been proposed[7]; this review will be guided by both phenomenology and pathophysiology (**Table 8-3**).

Clinical Assessment of Gait Disorders

The bedside examination of gait involves taking a careful history and a detailed physical examination. Falls are a serious complication of gait disturbances, and detecting a possible risk of falls should therefore receive special attention. Indeed, the presence of a prior fall in the preceding 6 to 12 months consistently emerges as an excellent, simple predictor for renewed falls in the future. Note that subjects should be asked about not only falls but also the presence of ''near-falls,'' as these may precede the onset of actual falls and can contribute to a fear of falling and thereby to secondary immobility (**Figure 8-3**, **Table 8-4**, **Table 8-5**).

First rule: consider compensations. The clinical picture of every patient (especially those with gait disorders) is the result of the underlying disease plus any compensatory strategies used by the patient to overcome part of the disability, ranging from subtle (eg, adopting a slower gait speed when balance is subjectively reduced) to very ingenious (eg, a patient grounded by freezing of gait who uses a bicycle to maintain mobility)[8] (**Table 8-6**). Antalgic gait is a typical example of compensatory gait, as it typically ceases once pain is absent.

The perception of postural imbalance can lead to two opposite gait disorders: cautious and reckless gait. Cautious gait is caused by an excessive perception of instability and characterized by slow velocity and short steps with abnormally increased double-support time: patients walk as if on ice, with arms outstretched and a feeling of unsteadiness. Cautious gait is linked to the ''fear-of-falling syndrome'' and ''fall phobia,'' which denotes a heterogeneous syndrome clinically characterized by a disproportionate fear of falling after even a single fall or near-fall.

Reckless gait (also known as careless gait) is the counterpart of cautious gait, caused by the defective perception of instability and typically seen in patients with postural instability who have a poor awareness of their own falling risk.

A common example of compensatory gait is the ataxic gait, an unsteady and uncoordinated walk, with a wide base of support (to compensate for the mediolateral instability) and the feet thrown out with varying step length. Patients with ataxic gait can also show knee locking as another compensatory feature to reduce limb hypermetria and instability,[9] as observed in severe essential tremor.[10] Patients with cerebellar disease display lower limb dysmetria, postural instability, or both (**Supplemental Digital Content 8-1**, *links.lww.com/CONT/A90*). The degree of instability is the major determinant of gait changes,[11] best detectable by asking patients to perform the tandem gait task (**Supplemental Digital Content 8-2**, *links.lww.com/CONT/A91*). These patients' gait is not dramatically worsened by eye closure,

TABLE 8-2 The Proposed Classifications of Gait Disorders and Their Pitfalls

Classification	Examples	Pitfalls
Hierarchical	Lower level Peripheral skeletomuscular problems (arthritic, myopathic, and peripheral neuropathic gait) Peripheral sensory problems (sensory/vestibular/visual ataxic gait) Intermediate level Hemiplegic gait Paraplegic gait Cerebellar ataxic gait Parkinsonian gait Choreic gait Dystonic gait Higher level (higher-level gait disorder)	Overlap in symptoms between different levels (eg, Parkinson disease patients might also display features of higher-level gait disorder) Higher-level gait disorder entity is often abused with confusing nomenclature Higher-level gait disorder subtypes are difficult to recognize in clinical practice
Anatomic	Frontal, cerebellar, etc	Gait patterns are not unequivocally attributed to a specific anatomic lesion (eg, wide-based gait might be caused by either frontal or cerebellar gait)
Etiologic	Vascular, neurodegenerative, etc	Ancillary investigations (eg, MRI) are required
Phenomenologic	Antalgic Paretic/hypotonic Spastic Vestibular Sensory ataxic Cerebellar ataxic Dyskinetic Hypokinetic-rigid Cautious Stiff Higher-level gait disorder	Pathophysiology not taken into account Classification is driven by a mixture of etiologic, anatomic, and clinical features A patient might have one or more types of gait disturbance
Pathophysiologic	See **Table 8-3**	Clinical examination alone cannot always disentangle these conditions An overlap between conditions might be present

[a] Data from Nutt JG, et al, Neurology,[6] *www.neurology.org/content/43/2/268.full.pdf+html?sid=b92aafd5-2187-4f32-8501-6b3ad09a5c54* and Snijders AH, et al, Lancet Neurol.[7] *www.sciencedirect.com/science/article/pii/S1474442206706780*.

in contrast with other conditions such as sensory ataxias or vestibular gait.

Another way to compensate for an underlying condition is to modulate velocity or cadence. Patients with parkinsonian gait typically increase cadence to compensate for the short step length and reduced velocity[12] (**Figure 8-4**).

TABLE 8-3 The Pathophysiologic Classification of Gait Based on the Assessment Approach Described in This Article[a]

Gait	Compensation	Lower Limbs Feature	Velocity	Cadence	Step Length/ Height	Asymmetry
Antalgic	Pain	Limited range of movement	↓	↓	↓	↑↑
Cautious	Instability	None or locking of the knee	↓	↓	↓	↓↑
Higher-level gait disorder	Instability Dysexecutive syndrome Disorders of attention	(Rigidity)	↓/=	↓↑	↓	↓↑
Normal pressure hydrocephalus	Instability	Outward rotated feet	↓	↓↑	↓	=
Vascular parkinsonism	Instability	Rigidity	↓	↑	↓	=
Parkinson disease	(Instability)	Rigidity Tremor (Dyskinesias) (Dystonia)	↓	↑	↓	↑
Multiple system atrophy (MSA)–cerebellar ataxia	Instability	Locking of the knee (Rigidity)	↓	↓/=	↓	=

Step Width	Variability[b]	Arm Swing	Episodic Features	Additional Features	Notes
=	↑[c]	=	Vascular or neurogenic claudication	Orthopedic diseases	Reduced stance phase on affected limb (limping) Improvement after analgesics
=/↑	↑	=	None	Fear of falling	Overlap with higher-level gait disorder and ataxic gait Like ''walking on ice'' Striking improvement by external support Also seen in stiff person syndrome and orthostatic tremor
=/↑	↑↑	↓↑	Freezing of gait	Urinary symptoms, dementia, frontal release signs, depression, no rescue reactions with the pull test (''falling like a log''), motor recklessness	Overlap with cautious gait, vascular parkinsonism, and normal pressure hydrocephalus Poor effect of cueing Inadequate synergies including inappropriate or bizarre foot placement, crossing of the legs, leaning into wrong direction when turning or standing, and difficulty using walking aids Seen in dementia syndromes
↑	↑↑	↑	Freezing of gait (no festination)	Urinary symptoms, dementia	Overlap with higher-level gait disorder and vascular parkinsonism Poor effect of cueing
=/↑	↑↑	=	Freezing of gait	Urinary symptoms, dementia, frontal release signs, depression	Overlap with higher-level gait disorder and normal pressure hydrocephalus Shuffling gait Stepwise progression
↓/=	↑	↓[d]	Freezing of gait Dyskinesias	Rigidity, tremor, bradykinesia also involving upper limbs and face, stooped posture	Overlap with dyskinetic gait, higher-level gait disorder,and other parkinsonian gaits Shuffling gait Response to cueing, dopaminergic drugs, or deep brain stimulation
↑↑	↑	=/↓	(Freezing of gait)	Pyramidal signs, dysautonomia, cerebellar dysarthria, nystagmus, vertical falls (due to syncope)	Overlap with cerebellar ataxic gait

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TABLE 8-3 **The Pathophysiologic Classification of Gait Based on the Assessment Approach Described in This Article**[a] *(continued)*

Gait	Compensation	Lower Limbs Feature	Velocity	Cadence	Step Length/ Height	Asymmetry
MSA-parkinsonism	(Instability)	Rigidity Dystonia	↓	↑	↓	=
Corticobasal syndrome	Instability	Rigidity Dystonia Myoclonus	↓	↑	↓	↑↑
Primary progressive freezing of gait	None	None	↓/=	↑	↓	=
Pure akinesia with freezing of gait	(Instability)	None	↓	↑	↓	=
Progressive supranuclear palsy-parkinsonism	(Instability)	Rigidity	↓	↑	↓	↑
Richardson syndrome	Dysexecutive syndrome Instability	Rigidity	↓	↑	↓	=
Hemiparetic	Leg/trunk stiffness	Weakness Spasticity	↓	↓	↓	↓↓
Paraparetic	Leg/trunk stiffness	Weakness Spasticity	↓	↓	↓	=
Proximal weakness (waddling)	Weakness	Hypotonia Weakness	↓/=	↓/=	=/↑	=

Step Width	Variability[b]	Arm Swing	Episodic Features	Additional Features	Notes
=/↑	↑	↓	Freezing of gait	Rigidity and bradykinesia also involving upper limbs and face, pyramidal signs, dysautonomia, Pisa syndrome, antecollis, vertical falls (due to syncope)	Overlap with progressive supranuclear palsy gait, shuffling gait Poor effect of cueing/drugs
=/↑	↑	↓[d]	Freezing of gait	Rigidity, myoclonus, bradykinesia also involving upper limbs and face, alien limb, apraxia, cortical sensory loss	Overlap with MSA and Richardson syndrome gait Shuffling gait Poor effect of cueing/drugs
↓↑	↑	=	Freezing of gait, especially hesitations	None, possible progression to pure akinesia with freezing of gait, progressive supranuclear palsy, Richardson syndrome, corticobasal syndrome, or primary lateral sclerosis	Overlap with pure akinesia with freezing of gait/ progressive supranuclear palsy gait Shuffling gait Poor effect of cueing/drugs
↓/=	↑	↓	Freezing of gait	Akinesia (micrographia, hypophonia, hypomimia), possible progression to progressive supranuclear palsy, or Richardson syndrome	Overlap with primary progressive freezing of gait/progressive supranuclear palsy gait Shuffling gait Poor effect of cueing/drugs
↓/=	↑	↓[d]	Freezing of gait	Rigidity, tremor, bradykinesia also involving upper limbs and face, supranuclear gaze palsy, motor recklessness	Overlap with pure akinesia with freezing of gait and Richardson syndrome gait Shuffling gait Variable effect of cueing/drugs
↓↑	↑	↓	Freezing of gait	Rigidity, bradykinesia also involving upper limbs and face, supranuclear gaze palsy, motor recklessness, pseudobulbar palsy, frontal dementia, early backward falls, retrocollis	Overlap with primary progressive freezing of gait/progressive supranuclear palsy gait Shuffling gait Poor effect of cueing/drugs
=/↑	↑	↓[e]	None	Weakness, spasticity also involving the ipsilateral upper limb, pyramidal signs	Overlap with spastic gait Other features: circumduction
↓↑	↑	=	None	Pyramidal signs, urinary symptoms	Overlap with stiff gait and primary lateral sclerosis Other features: scissoring
=	=/↑[c]	↓/=	Periodic paralysis	Trendelenburg sign, caused by most myopathies, myasthenia gravis or Lambert-Eaton myasthenic syndrome	Usually bilateral

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TABLE 8-3 **The Pathophysiologic Classification of Gait Based on the Assessment Approach Described in This Article**[a] *(continued)*

Gait	Compensation	Lower Limbs Feature	Velocity	Cadence	Step Length/ Height	Asymmetry
Distal weakness (steppage)	Weakness	Hypotonia Weakness	↓/=	↓/=	↓↑	=/↑
Cerebellar ataxic	Instability	Locking of the knee Variable severity of dysmetria	↓	↓	=/↑	=
Sensory ataxic	Instability	Locking of the knee Dysmetria	↓	↓	=/↑	=
Vestibular	Instability	None	↑	↑	=/↑	=
Essential tremor (severe)	Instability	None or locking of the knee	↓/=	↓/=	=	=
Primary lateral sclerosis	Instability Leg/trunk stiffness	Weakness Spasticity	↓/=	↓/=	↓	=
Stiff gait	Leg/trunk stiffness	Spasticity Spasms	↓	↓	↓↑	=/↑

Step Width	Variability[b]	Arm Swing	Episodic Features	Additional Features	Notes
=	=/↑ [c]	=	Periodic paralysis	Caused by neuropathies	Either unilateral or bilateral Other features: drag-to gait, quadriceps gait, foot-drop
↑↑	↑[c]	=	In case of episodic ataxias	Other cerebellar features (including nystagmus, dysarthria)	Other features: staggering
↑↑	↑[c]	=	No	Aggravated by eye closure or darkness (Romberg sign)	Other features: staggering
=/↑	↑[c]	=	In case of Ménière syndrome	Nystagmus, vertigo, nausea, deviation to one side aggravated by eye closure, Unterberger test (in which a patient is asked to walk on the spot with his or her eyes closed; the patient will rotate to one side in the case of a labyrinthine lesion) positive	Other features: staggering
=	=/↑[c]	=	None	Tremor (especially of the head), difficulty performing tandem gait	Improvement with alcohol
=/↑	=/↑	=	(Freezing of gait, especially hesitations)	Weakness, spasticity, and hypokinesia also involving upper limbs, face, and bulbar region; pyramidal signs (possible progression to ALS)	Overlap with primary progressive freezing of gait/paraparetic gait/stiff gait
=	=	=	No	Muscular pain, seen in stiff person syndrome, dystonia, hemiparesis, paraparesis	Overlap with dyskinetic/ hemiparetic/ paraparetic gait Other features: equine gait, circumduction, scissoring

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TABLE 8-3 The Pathophysiologic Classification of Gait Based on the Assessment Approach Described in This Article[a] *(continued)*

Gait	Compensation	Lower Limbs Feature	Velocity	Cadence	Step Length/ Height	Asymmetry
Dyskinetic	Destabilizing movements	Chorea Dystonia	↓	↓/=	=/↑	=/↑

↓ = decreased; = = normal; ↑ = increased; ↓↑ = variable; ↑↑ = markedly increased; () = features in parentheses are not consistently present; ALS = amyotrophic lateral sclerosis.
[a] Psychogenic gait is not included because of its marked variability of presentation; senile gait is not included because it is usually caused by an underlying pathologic condition fulfilling the classification of other gait disorders (eg, vascular parkinsonism or higher-level gait disorder).
[b] Usually worsened by dual tasks.
[c] Usually no worsening under dual tasks.
[d] Asymmetric.
[e] Asymmetric arm swinging with intermittent abduction of ipsilateral arm with each step.

KEY POINTS

- In sensory ataxias, darkness or eye closure markedly worsens gait; in vestibular ataxic gait, eye closure can cause a consistent unilateral deviation of walking.
- Waddling gait is caused by the weakness of proximal muscles of lower limbs; it is characterized by exaggerated alternation of lateral trunk movements with an exaggerated elevation of the hip.
- Steppage gait is seen when the weakness of distal muscles of the lower limbs causes a foot drop (the foot landing loudly on the floor) and the advancing leg is lifted high so that the toes can clear the ground.

Finally, many patients with gait disorders experience loss of gait automaticity (eg, basal ganglia diseases); this can be masked by greater cortical ''voluntary'' gait control, which typically deteriorates under dual tasks (see the section on clinical assessment of gait disorders below).

Second rule: consider limb features and destabilizing factors. Muscular weakness is another condition that frequently impairs gait. Two fundamental types have to be recognized. Proximal weakness is typically seen in myopathies, myasthenia gravis, and Lambert-Eaton myasthenic syndrome and leads to a waddling gait, which is usually bilateral and characterized by exaggerated alternation of lateral trunk movements with an exaggerated elevation of the hip. In contrast, distal weakness is typically seen in neuropathies and leads to a steppage gait. Steppage gait occurs when the weakness of distal muscles of the lower limbs causes a foot drop (the foot landing loudly on the floor) and the advancing leg is lifted high so that the toes can clear the ground (especially when the weakness is severe). This can be unilateral or bilateral. Other examples of gait changes induced by lower limb weakness are drag-to gait (in which the feet are dragged rather than lifted because of a weak push-off, as seen in weakness of the calf muscles) and quadriceps gait (in which at each step on the affected leg the knee hyperextends and the trunk lurches forward) (**Supplemental Digital Content 8-3**, *links.lww.com/CONT/A92*).

Other abnormalities of the legs might also impair gait. Many conditions leading to limb hypertonia cause a stiff gait, characterized by leg extension and plantar flexion of the foot with leg circumduction and hip flexion, accompanied by an intermittent abduction of the ipsilateral arm with each step (when unilateral) or scissoring of the legs with bilateral circumduction (when bilateral). Spasticity is the most common cause of a stiff gait and may be unilateral (as in hemiparesis; **Supplemental Digital Content 8-4**, *links.lww.com/CONT/A93*) or bilateral (as in paraparesis; **Supplemental Digital**

Step Width	Variability[b]	Arm Swing	Episodic Features	Additional Features	Notes
=	=/↑[c]	=	Paroxysmal dyskinesias, myoclonus, task-specific dystonia	Improvement by changing pattern of muscular activation (eg, running or backward walking)	Overlap with stiff gait Scissoring Other features: equine or dromedary gait, cock walk

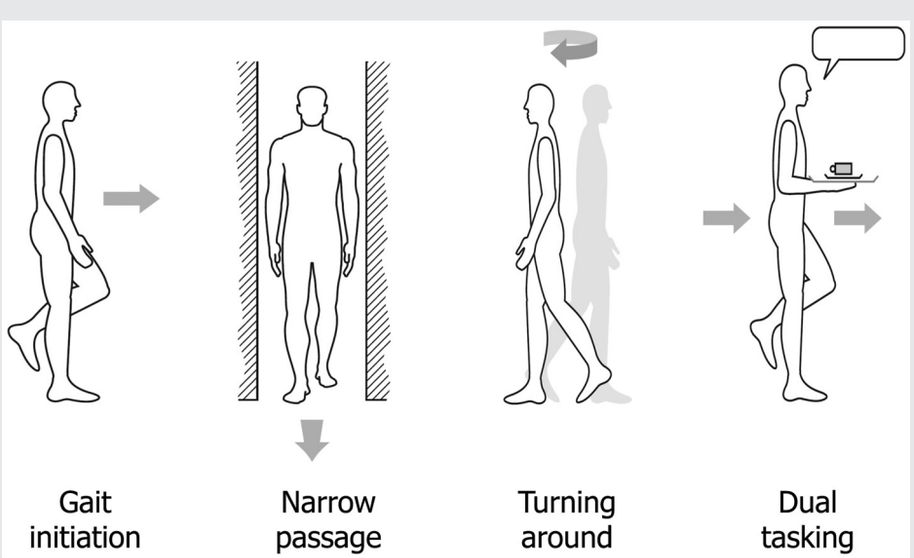

FIGURE 8-3 Several simple tests should complement the clinical assessment of gait. Patients suspected of having freezing of gait often walk normally in the examination room because excitement associated with the doctor's visit can suppress it. However, repeated gait initiations, walking in tight quarters, turning, or performing secondary tasks may provoke freezing of gait. Abnormalities to be noted during turning movements include the ''pivoting'' strategy, where the trunk rotates stiffly (en bloc) with the legs, shuffling of the feet with multiple small steps, or even overt freezing of gait. Furthermore, dual-tasking (the simplest test is to start a routine conversation while walking) has a good predictive value for falls in cognitively impaired older adults because these patients try to perform both tasks equally well, but gait or balance can be compromised (''stop walking when talking''). Other examples of secondary tasks include avoiding obstacles or carrying objects such as a tray while walking.

KEY POINTS

- Because spasticity is a velocity-dependent muscular hypertonia, spastic gait typically aggravates when patients are instructed to hurry up.
- A useful sign that supports the clinical suspicion of dystonic gait is the improvement that occurs in gait when a different motor program is utilized (eg, backward walking or running).

Content 8-5, *links.lww.com/CONT/A94*). Because spasticity is a velocity-dependent muscular hypertonia, spastic gait typically aggravates when patients are instructed to hurry up. Importantly, spasticity is typically accompanied by a variable degree of weakness. Other causes of leg stiffness are stiff person syndrome, neuromyotonia, and myotonia (the latter also characterized by a variable degree of proximal and distal weakness). Equine gait is another type of stiff gait, characterized by lower limb extension and plantar flexion of the foot, so that walking is accomplished mainly by flexing the hip joint.

Involuntary movements (especially chorea and dystonia) might also affect gait, as in dyskinetic gait (**Supplemental Digital Content 8-6**, *links.lww.com/CONT/A95* and **Supplemental Digital Content 8-7**, *links.lww.com/CONT/A96*). These extra involuntary movements might be variable and bizarre, as described in the ''silly walk'' of patients with levodopa-induced dyskinesias.[13] ''Cock-walk'' is another type of dyskinetic gait typical of patients with encephalopathy due to manganese toxicity, mainly as a result of occupational manganese exposure (eg, welders) or methcathinone poisoning, but which can also be seen with other pallidal causes of dystonia, such as pantothenate kinase–associated neurodegeneration. Cock-walk is characterized by a high-stepping gait, strutting on the toes with flexed elbows, and an erect spine. Patients with generalized dystonia (particularly DYT1 dystonia) may display a bizarre gait pattern, such as the dromedary gait, which is characterized by a rolling, high-stepping gait with protrusion of the buttocks due to excessive lordosis. Specific signs, such as the improvement of gait occurring when a different type of locomotor behavior is utilized, might support the diagnosis.

Dyskinesias that affect the gait are generally continuously present but can also be episodic, as in paroxysmal dyskinesias[14] or tics involving the lower limbs.[15] Other destabilizing factors are hyperekplexia and other types of myoclonus—for example, as seen in postanoxic encephalopathy (Lance-Adams syndrome), in which the positive and negative action myoclonus produces a bouncing gait and stance.

Other dyskinesias are more static but can still perturb gait from a biomechanical perspective; examples include asymmetrical arm dystonia in patients with corticobasal syndrome (CBS), retrocollis, antecollis, Pisa syndrome, or camptocormia.[16]

Third rule: look at stepping. Even in the absence of a gait analysis, some important spatiotemporal features of gait can be seen in the clinical examination room, and some important features can even be heard (eg, a unilaterally dragging foot, as a subtle sign of spasticity) (**Figure 8-5**).

Step length can be reduced as a sign of akinesia (as occurs in parkinsonism) or pyramidal slowness (motoneuronal disorders). More rarely, step length may be increased by hyperkinesias involving the lower limbs, by hypermetria caused by ataxic dysmetria, or in patients with vestibular instability, who reduce the time spent in stance phases and increase gait velocity to keep balance.[17] The comparison of the step length of both lower limbs allows the detection of asymmetry, a factor for falling and possibly freezing of gait. Changes in cadence may be secondary adaptations to the increased or reduced stride length (**Figure 8-4**).

Variability of step timing is another important gait feature, and two main types can be detected: oscillatory or

TABLE 8-4 Elements of History Taking in Patients With Gait Disorders and/or Falls

- ▶ **Temporal Nature**
  - Continuous
  - Episodic, which can be subdivided into:
    - Random (eg, paroxysmal dyskinesias)
    - Pseudoperiodic (after a given amount of steps, eg, freezing of gait, claudication)
- ▶ **Type of Onset and Progression**
  - Sudden (eg, stroke)
  - Insidious (eg, neurodegenerative disorders)
  - Step-wise (eg, vascular parkinsonism)
- ▶ **Walking Worse in the Dark?**
  - Yes (consider sensory ataxia or vestibulopathy)
  - No
- ▶ **Use of Walking Aids?**
  - Yes (consider latency to using aids: months versus years)
  - No (if not, why not? Embarrassment or inability? Consider higher-level gait disorder)
- ▶ **Medical History**
  - Prior/current diseases
  - Psychoactive medications
  - Intoxication (alcohol)
- ▶ **Protective Factors**
  - Exercise/fitness level
  - Amount of daily walking
  - Adaptation of behavior/activities
- ▶ **Fall History**
  - Frequency of prior falls and near-falls
    - Single (in absence of extrinsic cause, search for risk factors)
    - Recurrent
  - Specific fall pattern?
  - Apparent cause of the fall(s):
    - None (spontaneous, consider intrinsic causes)
    - Extrinsic (environmental, eg, slippery floor)
    - Intrinsic (patient-related)
      - Symptoms preceding the fall:
        - Loss of consciousness (consider syncope, epilepsy, or psychiatric conditions)
        - ''Funny turns'' (vertigo, presyncope)
        - Palpitations, chest pain, breathlessness
        - Sudden weakness of the legs (drop-attack, cataplexy)

Continued on next page

TABLE 8-4 Elements of History Taking in Patients With Gait Disorders and/or Falls *(continued)*

- Behavior:
  - Performing several activities simultaneously
  - Hazardous behavior
  - Inappropriate footwear
- Symptoms after the fall:
  - Confusion (consider epilepsy)
  - Inability to stand up
  - Physical injury
  - Fear of falling

TABLE 8-5 Elements of Physical and Instrumental Examination in Patients With Gait Disorders and/or Falls[a]

Examination	Elements
General	Body mass index (low values indicates frailty)
	Cardiovascular examination
	Measurement of blood pressure in both the recumbent and standing positions (orthostatic hypotension?)
	Joints (ankles, knees, hips)
	Vision (with/without correction)
	General neurologic examination, including:
	Cognition
	Depression and anxiety
	Tendon reflexes and proprioception of the legs
	Upper and lower motor neuron features
	Vestibular tests
Consider compensations	Gait speed
	Fast usually in vestibular imbalance, slow in case of hypokinesia, instability, weakness, stiff gait
	Patients with spasticity or stiff person syndrome worsen when instructed to hurry
	Pain
	Reassess gait after an analgesic
	Imbalance
	Subjective perception
	Too much (fear of falling)
	Too little (reckless gait)
	Striking improvements in gait with external support in patients with cautious gait

Continued on next page

TABLE 8-5 **Elements of Physical and Instrumental Examination in Patients With Gait Disorders and/or Falls**[a] *(continued)*

Physical Examination	Elements
	Transfers
	Righting reactions (rising from chair or bed)
	Sitting or lying down
	Supporting reactions (quiet stance, Romberg test)
	Anticipatory reactions (eg, lifting object from floor)
	Tandem gait
	Climbing stairs
	Instability while turning[b]
	Cadence
	High (compensatory short step length)
	Low (pain, instability)
	Lack of automaticity (cortical/higher-level gait disorder gait)
	Dual tasking[c]:
	Dual tasking (carrying objects, ''stops walking when talking'')
	Avoiding obstacles
	Combinations of the above
Consider limb features	Strength of the legs
	Stand up from a chair (a measure of proximal leg strength)
	Trendelenburg sign[d]
	Leg stiffness
	Spasticity
	Rigidity
	Myotonia
	Spasms
	Dyskinesias
	Tremor (also orthostatic tremor[e])
	Chorea
	Ballism
	Myoclonus (also orthostatic myoclonus[f])
	Tics
	Dystonia
	Can be task-specific, eg, disappears during running or walking backward
Consider perturbations other than dyskinesias	Episodic
	Cataplexy
	Hyperekplexia

Continued on next page

TABLE 8-5 **Elements of Physical and Instrumental Examination in Patients With Gait Disorders and/or Falls**[a] *(continued)*

Physical Examination	Elements
	Static (posture)[g]
	Antecollis
	Retrocollis
	Pisa syndrome
	Camptocormia
	Pusher syndrome[h]
Look at stepping[i] (see also **Figure 8-3** and **Figure 8-5**)	Step height and length
	Short (shuffling)
	Long/high
	Step length symmetry
	Variability
	Oscillatory (sequence effect)
	Variable (also assess dual tasking)
	Stance width
	Narrow (hypokinesia)
	Wide (imbalance)
Recognize specific features	Hesitations
	Gait ignition
	Turnings
	Clockwise
	Counterclockwise
	Narrow passages
	Upon reaching (while walking to a destination)
	Festination
	Freezing of gait
	Effect of cues
	Poor or no improvement generally in higher-level gait disorder and other lower-body parkinsonism
	Arm swing
	Reduction
	Asymmetrically in Parkinson disease, progressive supranuclear palsy–parkinsonism, corticobasal syndrome
	Symmetrically in other degenerative parkinsonism (progressive supranuclear palsy or Richardson syndrome, multiple system atrophy)
	Normal or exaggeration (normal pressure hydrocephalus and other lower-body parkinsonism)

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TABLE 8-5 Elements of Physical and Instrumental Examination in Patients With Gait Disorders and/or Falls[a] *(continued)*

Physical Examination	Elements
Other features	Walking with eyes closed
	Provoke/aggravate ataxia in sensory ataxia
	Consistent deviation to one side in unilateral vestibular loss
	Evaluate footwear
	Appropriateness
	Inspection of the sole
	Look for excessive medial and anterior wear on the same side as a spastic leg
Standardized rating scales	Generic
	Tinetti Mobility Index
	Includes an evaluation of gait features and balance under challenging conditions
	Poor performance is associated with an increased risk of falls
	Gait and Balance Scale
	Berg Balance Scale
	Disease-specific
	Eg, Freezing of Gait Questionnaire
Quantifiable tests	Timed gait and walking distance (eg, distance walked in 6 min)
	These tests have the limitation of not accommodating the quality of gait
	Functional reach test
	With outstretched arms, subjects are instructed to reach forward as far as possible while keeping their feet in place
	Measure of dynamic balance control
	Evaluation of the limits of postural stability in standing
	Timed Up and Go test
	Patients are observed and timed while rising from a high chair with arms, walking 3 m, turning around, walking back, and sitting down again
	Patients requiring more than 13.5 s have an increased risk of falls
	Reactive/protective postural responses
	The retropulsion test (pull test)[j]
	Drawbacks include difficulty in standardizing test execution and the lack of a generally accepted scoring system

Continued on next page

TABLE 8-5 Elements of Physical and Instrumental Examination in Patients With Gait Disorders and/or Falls[a] *(continued)*

Physical Examination Elements	
	The Push and Release Test[k]
	An advantage is that this test allows examiners to apply more consistent perturbation forces
	Time in tandem stance
	Time in single leg standing
Instrumental measurements	Instrumented carpet or walkway sensitive to the pressure changes caused by walking
	A major advantage is that the subject is not required to wear any special shoes, markers, or inserts
	It quantifies stride length, stride width, stride time, and the timing of the gait cycle
	Ambulatory monitoring systems (patient is equipped with accelerometers, footswitches, or other wearable sensors)
	A key advantage is that it allows the measurement of multiple strides, in almost any environment
	Gait analysis in laboratories (quantitative outcome measures, such as kinematics [joint motion], kinetics [reactive forces], and dynamic EMG), with or without treadmill)
	Highly detailed assessment of walking
	Major pitfall is a lack of ecologic validity

[a] Ideally, physicians should also examine patients in the environment where they walk and function in daily life (ie, home and surroundings), but this is not always practical.
[b] Instruct patient to execute slowly and abruptly; note occurrence of freezing of gait.
[c] Multiple task performance also provides insight into actual falling risks because falls in daily life commonly occur while attempting to do two or more things at the same time.
[d] Trendelenburg sign is positive if, when standing on one leg, the pelvis drops on the side opposite to the stance leg due to the weakness of hip abductors (gluteus medius and minimus).
[e] Orthostatic tremor is a fast (14 to 18 Hz) tremor occurring in the legs and trunk immediately after standing; the high frequency and fine amplitude of the tremor makes it nearly invisible and patients usually report unsteadiness.
[f] Orthostatic myoclonus is characterized by leg jerking during upright posture, eventually interfering with gait.
[g] Assess patients while seated and standing, in frontal and lateral view.
[h] *Pusher syndrome* is a disturbance of body orientation perception in the coronal (roll) plane after brain lesions (generally thalamic). Patients experience their body as oriented upright when it is in fact markedly tilted to one side; they use the unaffected arm or leg to actively push away from the unparalyzed side and resist any examiner's attempt to passively correct their tilted body posture.
[i] Use sufficient space during examination. Most examination rooms are too small to assess gait properly. Monitor the patient while walking to and from the examination room, or even follow the patient down the hall.
[j] At our institution, we deliver one shoulder pull without prior warning, as this best mimics daily life where falls are usually unexpected. We then repeat the test several times and regard failure to ''habituate'' to the test as another sign of balance impairment.
[k] The Push and Release Test rates the postural response to a sudden release of a patient who is pressing backward against the examiner's hands, which are placed on the patient's back.

variable. The oscillatory type is typically seen when patients present a progressive reduction of step length, a phenomenon that has also been called the sequence effect. This progressive reduction in amplitude is the hallmark of

TABLE 8-6 **Range of Etiologically Varied Gait Disorders Leading to Similar Gait Phenomenology Due to Compensation**[a]

Compensatory Behavior	Cause of Compensation		Examples
Limping gait	Pain?	Yes	Orthopedic conditions
		No	L5-S1 radiculopathy
	Weakness?	Yes	Hemiparesis
		No	Dystonia
Wide-based gait	Weakness?	Yes	Paraparesis
		No	Ataxia
	Instability?	Yes	Ataxia
		No	Paraparesis, dystonia
Slow velocity	Pain?	Yes	Orthopedic conditions
		No	See below
	Weakness?	Yes	Hemi- and paraparesis
		No	Parkinsonism
	Instability?	Yes	Higher-level gait disorders
		No	Parkinsonism

[a] Velocity is the easiest feature to recognize but it is highly nonspecific.

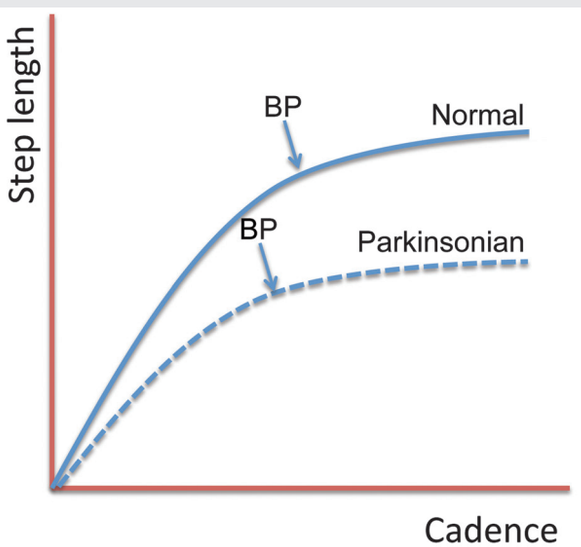

FIGURE 8-4 Humans can increase the velocity of locomotion by increasing both step length and cadence. Accelerated walk is associated with shortening of all gait phases but proportionally more double-limb support time. Depending on leg length, at a certain point, referred to as break point (BP), step length can no longer be increased and a higher cadence becomes the only way to increase velocity. Patients with parkinsonism can modulate both variables but, similarly to people with short legs, they more rapidly reach the break point, resorting to an increase in cadence.

Modified from Morris ME, et al, J Neurol Neurosurg Psychiatry.[12] © 1994 with permission from BMJ Publishing Group Ltd. *jnnp.bmj.com/content/57/12/1532.long.*

KEY POINT

- Dual tasking is a useful examination technique for the assessment of patients at risk for falls because it worsens impairment of gait automaticity and may cause freezing of gait, especially when subtle or subclinical.

akinesia and can involve any repetitive movement (eg, finger or foot tapping). This sequence effect can result in a new cycle of progressive step length reduction (hence the oscillatory nature of the phenomenon [**Figure 8-6A**]). When the step amplitude reduction during walking is pronounced, freezing of gait can emerge[18] (**Figure 8-6B**, **Supplemental Digital Content 8-8**, *links.lww.com/CONT/A97*). This knowledge can be used in clinical practice by asking patients to purposefully walk with rapid, small steps to elicit freezing of gait.

The variable variability of gait is seen in cerebellar or sensory ataxia as well as in patients with impairment of gait automaticity (eg, patients with basal ganglia disorders). In this case, patients try to maintain the regularity of their steps by focusing their attention on the motor task of walking (cortical gait). However, such patients are particularly susceptible to the "dual-task cost": when the patient is asked to perform another cognitive task (eg, backward counting) or motor task (eg, carrying a tray), gait becomes visibly irregular and slow or culminates into a full stop (eg, as seen in the "stops walking while talking" phenomenon).[19] Dual tasking is a useful examination technique for the assessment of patients at risk for falls because it worsens impairment of gait automaticity and may cause freezing of gait, especially when subtle or subclinical.

Another form of dual-task impairment occurs when subjects fail to get their priorities right.[20] Under complex circumstances, young, healthy people purposefully neglect the secondary task and lend relatively more priority to walking safely. This prudent posture-first strategy is diminished in elderly people and diminished further in patients with parkinsonism

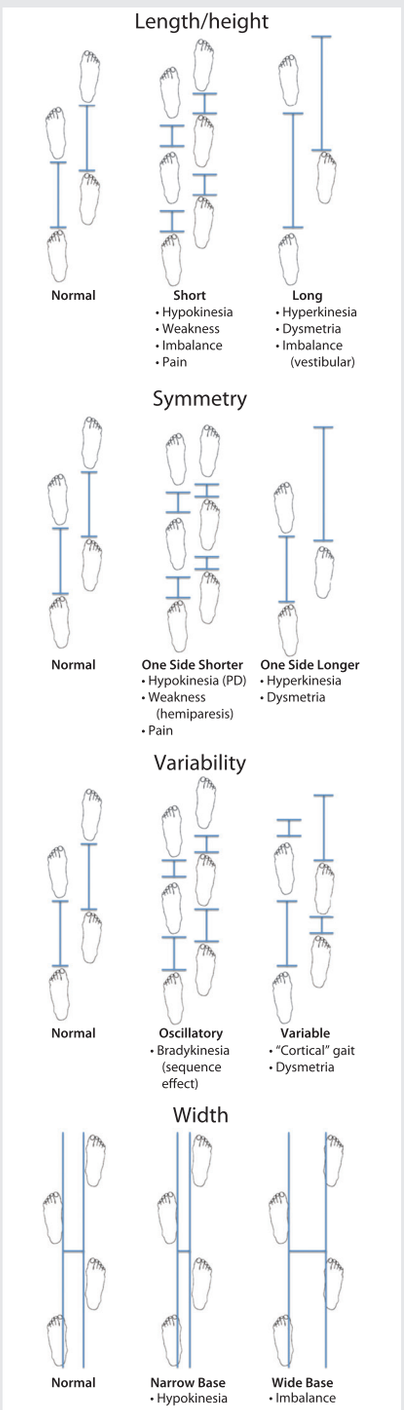

FIGURE 8-5 Some important spatiotemporal features of gait can be appreciated by the naked eye. The figure shows the major determinants of changes in step length/height, symmetry, variability, and width.

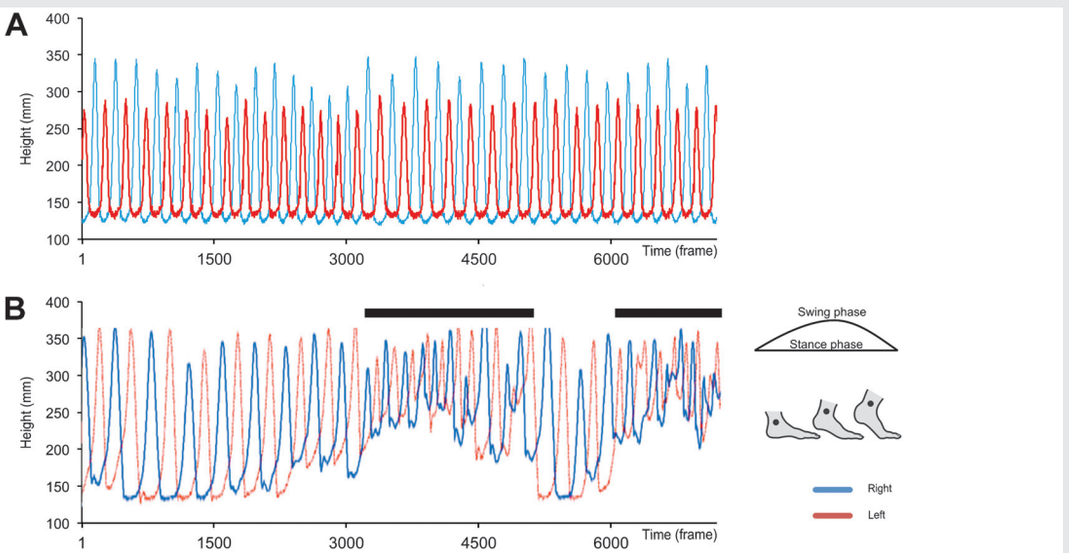

FIGURE 8-6 Gait analysis recordings of the trajectories along the sagittal plane of the right (*blue line*) and left (*red line*) ankle in two different patients with Parkinson disease. *A*, The typical sequence effect without motor block (''oscillatory'' variability); *B*, Two sequences with progressive reduction of step length followed by freezing-of-gate episodes (*dark lines*).

(posture-second strategy), in severe cases resulting in recklessness.[20] A marked dual-task decrement while walking has been observed in many conditions, including idiopathic falls in elderly patients and in those with parkinsonism, higher-level gait disorder, or dementia.

In most patients with postural instability, the step width is wide and, as such, is a nonspecific sign, although it can be seen particularly in patients with instability along the mediolateral axis (patients with ataxia, Huntington disease, severe essential tremor, or one of the forms of atypical parkinsonism). A normal to even narrow base of support is typically seen in patients with Parkinson disease (PD), who indeed do not display instability along the mediolateral axis.[21]

Fourth rule: recognize specific gait abnormalities. Freezing of gait is a common and disabling feature of many forms of parkinsonism, most commonly experienced during gait initiation, turning, and when negotiating obstacles or other tasks (dual tasking). Patients with freezing of gait experience sudden and often unexpected episodes during which their feet subjectively become ''glued to the floor'' while their trunk continues to move forward. Focused attention and external stimuli (cues) can help to overcome the episode.[22] Because of its sudden and unpredictable nature (**Figure 8-6B**), freezing of gait often leads to falls and injuries (**Supplemental Digital Content 8-9**, *links.lww.com/CONT/A98*). Freezing of gait is accompanied by different phenomena.[23] Hesitations are characterized by the inability to generate effective stepping at the beginning of walking (start hesitation) or in tight quarters such as passing through a narrow doorway (tight quarters hesitation) or turnings (turning hesitation). Trembling in place is characterized by an alternating shaking of the knees at a frequency of about 3 Hz to 8 Hz,

KEY POINT

- Freezing of gait is most commonly experienced during gait initiation, turning, and when negotiating obstacles or other tasks (dual tasking).

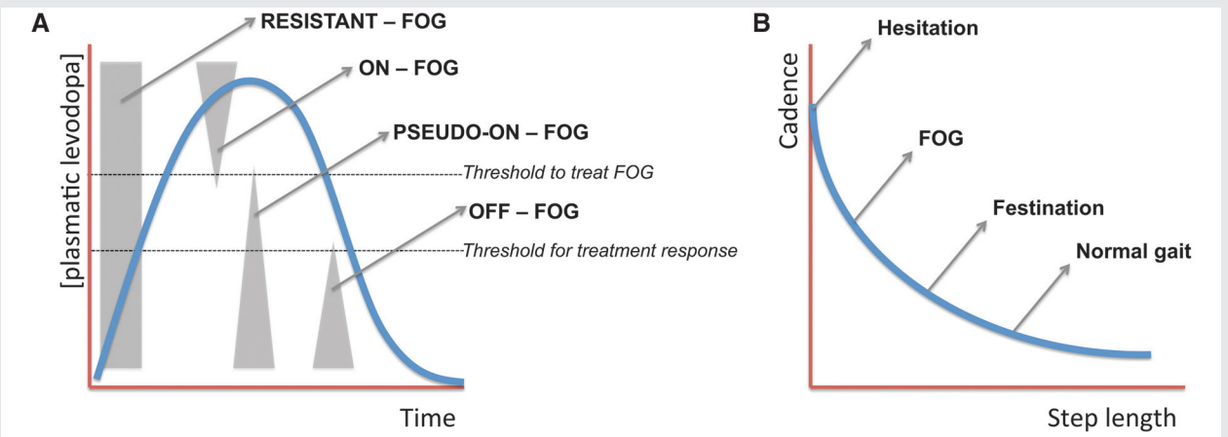

FIGURE 8-7 Pharmacologic and pathophysiologic classification of freezing of gait. *A*, Freezing of gait (FOG) can be classified according to the effect of levodopa treatment: patients with Parkinson disease (PD) usually have a levodopa-responsive FOG, which only appears during the levodopa off states (OFF–FOG). If patients have FOG while being under levodopa effect, three options have to be considered: PSEUDO-ON–FOG is caused by an insufficient amount of levodopa (sometimes it is a transition state characterized by festination); ON–FOG is directly caused by levodopa itself, ie, patients do not have (or have less) FOG during off states; finally, RESISTANT–FOG is not improved by levodopa and invariably affects off and on states. *B*, Other pathophysiologic features point to a common underlying mechanism for FOG: the different clinical expression can be interpreted along the relationship between cadence and step length. These phenomena are often coexisting in the same patient and follow a gradient of severity: turning hesitation (especially toward the least affected side) is the earliest sign (and one of the most common), followed by narrow hesitation, festination, and FOG while walking.[31] The most severe subtype is a complete akinesia, without any observable motion of the legs.

Panel A modified with permission from Espay AJ, et al, Neurology.[30] © 2012 American Academy of Neurology. *www.neurology.org/content/78/7/454.abstract?sid=fdbb9e4b-62f1-43af-9677-5364846c725a*.

typically with the forefoot stuck to the floor and the heel up in the air. One hypothesis is that this shaking represents multiple anticipatory postural adjustments in the absence of effective stepping (cadence without step).[24] Festination is characterized by taking increasingly rapid and small sequential steps during walking; it is usually seen upon reaching a destination and is accompanied by a progressively forward trunk.

The precise pathophysiology underlying freezing of gait remains unknown, but the following hypotheses are currently taken into account[23]:

1. A dopaminergic deficiency plays a major role in levodopa-responsive cases, possibly in regions outside the putamen (as dopaminergic cell implants into the putamen of PD patients improve bradykinesia and rigidity but do not improve freezing of gait).
2. Norepinephrine deficiency might contribute to freezing-of-gait pathogenesis.
3. Freezing of gait reflects dysfunction in an organized network involving the frontal lobes (supplementary motor area) and its connections with the GPi and brainstem (PPN).
4. Basal ganglia dysfunction leads to failure in generating adequate movement amplitude, a phenomenon known as the "sequence effect."[18]
5. A disturbance in the fine regulation of interlimb coordination has been also found for the upper limbs of patients with freezing of gait.[25]
6. A disorder of temporal and spatial gait symmetry has also been found in patients with freezing of gait,[26,27] which explains why highly asymmetric tasks (turning or initiating walking) are provoking factors.

7. The failure to initiate compensatory stepping could be due to impairment of anticipatory postural adjustments,[24] which explains the finding that patients with freezing of gait can walk better when assisted.
8. Freezing of gait is associated with cognitive (especially executive) deficits,[28] as also underlined by neuroimaging studies finding different degrees of cortical involvement in patients with freezing of gait.
9. Misperception of optic flow could play a role, especially in patients with left-side impairment,[29] which explains why vision can have a profound impact on gait (eg, doorways or obstacles).

Freezing of gait might be further classified according to the effect of levodopa treatment as ''off–freezing of gait'' (only present during the off periods), ''pseudo-on–freezing of gait'' (present during off and on episodes but responding to higher levodopa doses), ''on–freezing of gait'' (absent or mild during off episodes and present or worsened during on periods), and ''levodopa-resistant freezing of gait'' (persisting after a supratherapeutic dose of levodopa).[30] (**Figure 8-7**).

Levodopa-resistant freezing of gait may complicate late stages of PD and is far more frequent in other parkinsonisms or in conditions without overt parkinsonism, such as primary progressive freezing of gait, normal-pressure hydrocephalus (NPH), or—more rarely—higher-level gait disorder (see the section on higher-level gait disorder below).

Other episodic changes of gait exist. In the elderly, walking difficulties after exercise are often due to fatigue (cardiopulmonary or neuromuscular disease) but may also reflect vascular claudication, which is typically accompanied by muscular pain relieved by any type of rest (including standing still). Another important cause is neurogenic claudication, which usually accompanies lumbar spinal stenosis and is characterized by back and leg pain that is exacerbated after prolonged standing and relieved when seated or when bending forward.

SELECTED DISORDERS AFFECTING GAIT

Parkinson Disease and Other Degenerative Parkinsonisms

Many features of parkinsonian gait are related to the impairment of scaling function and defective internal cueing that are characteristic for these conditions: amplitude reduction (smaller step length, causing slowness of gait), reduced step height (causing the typical shuffling gait), and reduction or abolishment of the automatic synkinetic arm swing during walking (asymmetrical in PD, but more symmetrical in atypical parkinsonism) (**Table 8-3**). Gait is also more variable due to lack of automaticity. Other typical features are step length asymmetry and an increased cadence to compensate for the reduced step length.[12] All degenerative parkinsonisms typically present with this hypokinetic/rigid gait, with some specific differences—ie, a marked and early involvement of gait impairment, poor responsiveness to levodopa or cueing, early appearance of freezing of gait, and postural imbalance (especially along the mediolateral axis) (**Table 8-3**). These features are very rarely present in drug-induced parkinsonism.

Retrospective cross-sectional studies have suggested that freezing of gait is present in around 7% of patients with PD in the first 2 years of their disease, 28% in 5 years, 39% in 10

KEY POINTS

- An accurate distinction of the different freezing-of-gait subtypes in Parkinson disease requires a comprehensive motor assessment in three medication states: before levodopa (''off–freezing of gait''), after a normal levodopa dose (''pseudo-on–freezing of gait'' or ''on–freezing of gait''), and after a supratherapeutic dose (resolution of pseudo-on–freezing of gait, persistence of levodopa-resistant freezing of gait, worsening of freezing in patients with on–freezing of gait).
- Parkinsonian syndromes other than Parkinson disease and drug-induced parkinsonism tend to show marked and early involvement of gait impairment, poor responsiveness to levodopa or cueing, early appearance of freezing of gait, and postural imbalance (especially along the mediolateral axis).

KEY POINT

■ *Higher-level gait disorder* refers to a specific gait phenotype characterized by backward-directed postural instability, absence of response to sensory cues, troubles adapting with walking aids, and a variable combination of cognitive decline and parkinsonian, frontal release, or pyramidal signs.

years, and 58% after 10 years.[32] Disease duration increases the risk of developing freezing of gait, but the presence of tremor is associated with a lower risk. PD patients may restore fairly normal stepping amplitude by using other internal resources (attention) or external triggers (visual, auditory, or tactile cueing).

Lower Body Parkinsonism

Specific conditions are characterized by a parkinsonian gait with freezing of gait and no (or relatively few) signs in the upper body. These conditions are characteristically also accompanied by (1) normal or exaggerated swinging of the upper limbs, (2) high step variability (especially under dual tasks), (3) early postural instability, and (4) poor response to sensory cues.

Higher-Level Gait Disorder

Higher-level gait disorder is the nomenclature introduced by Nutt and colleagues[6] to solve the confusion generated by the introduction of the terms ''frontal gait disorder'' or ''gait apraxia.''[33] The latter was originally used to describe patients who were better able to perform cycling leg movements while lying recumbent, thus suggesting that their loss of control over leg movements was task-specific. Several lines of evidence[34] indicate that gait apraxia is a misnomer and should not be used: (1) leg apraxia has not been routinely studied in literature, and its relationship with gait apraxia is misleading, as they do not necessarily coexist in a single patient; (2) the term ''gait apraxia'' has been abused in literature using different definitions to indicate various gait disorders with overlapping features; (3) gait apraxia typically exists without any evidence of other forms of apraxia, and patients with bilateral limb apraxia can have a normal gait; (4) in the strictest of definitions, apraxia is the inability to perform ''skilled or learned motor acts,''[34] whereas locomotion is not a consciously learned motor act but rather a repetitive motor pattern generated by spinal mechanisms and modified by suprasegmental structures.

Historically, many other terms have been confusingly applied to the higher-level gait disorder, such as ''isolated gait ignition failure,'' ''cautious gait,'' ''subcortical disequilibrium,'' ''frontal disequilibrium,'' and ''frontal gait disorder.''[6]

The core features of higher-level gait disorder are reduced cadence, short steps with marked variability, preservation of arm swing, freezing of gait in absence of response to sensory cues, en bloc or wide-based turns, backward-directed postural instability, and trouble adapting with walking aids. A variable combination of other features might be present, such as cognitive decline and parkinsonian, frontal release, or pyramidal signs.

Normal walking critically depends on the interaction between the executive control dimension (integration and decision of action) with the cognitive dimension (eg, navigation, visuospatial perception or attention) and the affective dimension (mood, cautiousness, and risk-taking).[7] Higher-level gait disorder encompasses various disorders that share dysfunction of the highest integrative sensorimotor systems, but with intact basic motor and sensory functions, signaling a disconnect between frontal and subcortical structures involved in gait control and postural stability.

Vascular Parkinsonism

Vascular parkinsonism was initially described as arteriosclerotic parkinsonism and is caused by three main mechanisms (**Figure 8-8** A–C). Although an acute onset is possible in

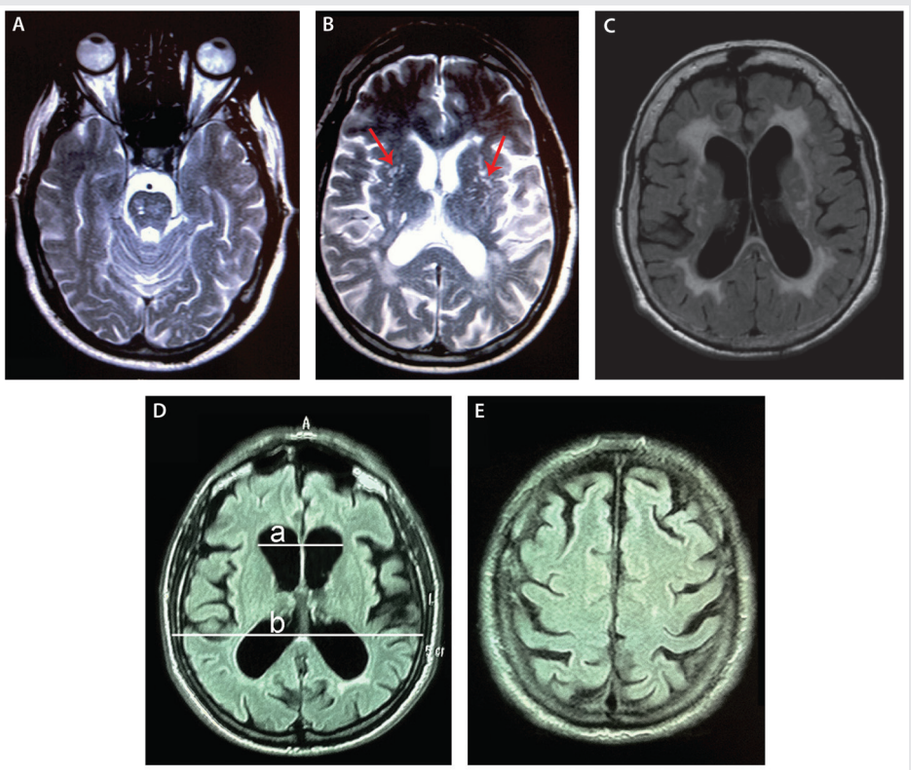

FIGURE 8-8 Brain MRI may be particularly useful for the diagnosis of gait disorders when vascular parkinsonism or normal pressure hydrocephalus are suspected. Vascular parkinsonism with gait disorder might be caused by a strategic lesion in the brainstem (*A*), in basal ganglia (*B*), and/or due to diffuse chronic vascular damage of the subcortical white matter, as in Binswanger disease (*C*). Normal pressure hydrocephalus is suspected in the setting of ventriculomegaly disproportionate to any degree of parenchymal atrophy, radiologically defined by an Evans index of greater than 0.3 (*D*). The Evans index is calculated as the ratio of the maximum width of frontal horns of the lateral ventricles (*line ''a''*) to the transverse inner diameter of the skull (*line ''b''*). Normal pressure hydrocephalus is also supported by narrowing of the CSF spaces at the high convexity (*E*), dilatation of the Sylvian fissure (''mismatch'' sign),[35] upward bowing of the corpus callosum, and empty sella. Furthermore, white matter hyperintensities surrounding dilated ventricles can be seen and are thought to represent transependymal fluid due to elevated CSF pressure (and have been documented to be reversible in some cases).

case of strategic lesions, disease onset is usually insidious. A stepwise progression is suggestive but not universal or specific. These patients might have a positive history of other vascular events and additional findings such as pyramidal signs, pseudobulbar palsy, or urinary incontinence; dementia can also be present, but visual hallucinations must be absent.[36]

Normal Pressure Hydrocephalus

NPH is a slowly evolving neurologic disease generally seen in the elderly, which is associated with an excessive volume of intraventricular CSF that is not explained by cerebral atrophy (**Figure 8-8D**). The full clinical picture includes the sequence of gait impairment followed by urinary incontinence (frequency and urgency followed by frank incontinence) and eventually by dysexecutive-predominant dementia.[37] Gait is always affected first in true NPH, when the disorder is defined by the sustained response to CSF shunting. By contrast, the presence of dementia antedating or

TABLE 8-7 Clinical and Instrumental Features for the Diagnosis of Normal Pressure Hydrocephalus[a]

▶ **Principal Criteria**
Gait dysfunction *plus* urinary[b] or cognitive dysfunction[c]
▶ **Supportive Criteria**
Early cognitive decline[d]
Age >40 y old
Insidious progression of symptoms over a period of at least 3 mo
CSF opening pressures between 70 and 245 mm H_2O
Ventriculomegaly: MRI/CT with an Evan index of ≥0.3 (Figure 8-8D)[e]
Increased stroke volume of CSF[f] at the cine phase flow MRI
▶ **Exclusion Criteria**
Structural lesion or congenital aqueductal stenosis
CSF opening pressures above 245 mm H_2O[g]
Papilledema[g]
Resting tremor
Early postural impairment
Early cognitive decline[h]
Late urinary symptoms[h]
Excellent response to levodopa[i]
Age at onset <40 y old
Acute onset
Onset of a cognitive decline before gait disorder[d]
Visual hallucinations[d]
Early presence of cortical deficits such as aphasia, apraxia, or agnosia
Back pain (suggesting lumbar canal stenosis)
▶ **Suggestive of an Alternative Diagnosis**
Parkinsonism (found in up to 71% of patients with normal pressure hydrocephalus)[39]
Bradykinesia (in 62% of patients with normal pressure hydrocephalus)[39]
Postural tremor
Cerebrovascular changes of white matter (comorbid in over 60% of patients with normal pressure hydrocephalus)

[a] Modified from the evidence-based guidelines established through a systematic review of 653 references dating from 1966 to 2003.[40]
[b] Abnormal urinary urgency or frequency is sufficient to document urinary dysfunction.
[c] Impairments of two or more domains, such as psychomotor speed, fine motor speed or accuracy, attention, short-term recall, executive function, or behavioral/personality change constitute cognitive dysfunction.
[d] Also consider Lewy body dementia.
[e] Other neuroimaging features include temporal horn enlargement, periventricular signal changes, periventricular edema, and aqueductal/fourth ventricular flow void.
[f] The volume of CSF passing through the cerebral aqueduct in systole and diastole.
[g] This should prompt investigation for a secondary cause of normal pressure hydrocephalus.
[h] Consider other neurodegenerative disorders.
[i] Patient experiences a >30% reduction of Unified Parkinson Disease Rating Scale-III after an acute challenge with levodopa. If a second, 25% to 50% higher dose of levodopa fails to substantially attenuate the motor symptoms, a slow dose titration up to approximately 1000 mg/d over 4 to 6 weeks should be administered.[39,41]

Case 8-1

A 63-year-old man presented with a 20-month history of freezing of gait consisting mainly of hesitations with starting, turning, and crossing narrow passages. On neurologic examination, he had mild lower limb spasticity and bilateral extensor plantar responses. No signs of parkinsonism were found, and a diagnosis of primary progressive freezing of gait was formulated. 123I-FP-CIT single-photon emission computed tomography (SPECT), brain MRI, nerve conduction studies, and EMG were normal. High doses of levodopa did not improve gait. One year later, he presented with a spastic paraparesis, dysarthria, and pseudobulbar affect with emotional lability consistent with a diagnosis of primary lateral sclerosis (**Supplemental Digital Content 8-10,** *links.lww.com/CONT/A99*).

Comment. A phenotype of primary progressive freezing of gait but with normal nigrostriatal dopaminergic projections suggests primary lateral sclerosis.

KEY POINT

■ Gait is always affected first in true normal pressure hydrocephalus, if the disorder is defined by sustained response to CSF shunting. Presence of dementia antedating or concurrently developing with gait impairment is a red flag against the diagnosis of normal pressure hydrocephalus.

concurrently developing with gait impairment is a red flag against the diagnosis of NPH. In patients with NPH, gait is characterized by slow, small, shuffling steps with freezing of gait and deterioration under dual-task conditions and may resemble that of patients with PD, although with preserved arm movements (lower-body parkinsonism), broad-based gait with outwardly rotated feet, and a lack of response to sensory cueing.[38]

The diagnosis is based mainly on the sequential development of NPH symptoms (ideally before frank dementia ensues) combined with a fitting MRI picture (**Table 8-7**), and can be best supported by the response to external lumbar drainage via a spinal catheter at a rate of approximately 10 mL/h for 36 to 72 hours. This procedure has a sensitivity of 50% to 100% and a specificity of 60% to 100%.[37] Other, less accurate tests are the CSF infusion test in order to measure the increased resistance to CSF absorption, and high-volume (30 mL to 50 mL) CSF removal via lumbar puncture, which may improve gait for a few hours.[37]

In recent years, the dignity of NPH as a separate entity has been challenged by a number of studies. A recent retrospective study[42] in 13 patients with suspected NPH who underwent surgical shunt placement found a definite gait improvement in 75% of them at 3 to 6 months, although this rate dropped to 50% at 1 year and to 33% at 3 years. Additional or alternative neurologic diagnoses, particularly progressive supranuclear palsy (PSP), later surfaced in five patients. Since patients with moderate to severe postural instability are known not to experience sustained improvement after surgery, postural instability might be the hallmark of another underlying neurodegenerative disease. This view is consistent with recent pathologic studies, which found that patients receiving a diagnosis of NPH during life had pathology consistent with neurodegenerative disorders (usually PSP or Alzheimer disease).[43,44]

Primary Progressive Freezing of Gait

Primary progressive freezing of gait refers to a distinct clinical syndrome seen in patients with progressive freezing of gait evolving in isolation of any other neurologic abnormalities for the first 3 years.[45] This clinical syndrome is considered one of the clinical variants of PSP but in rare cases may also be due to pallidonigroluysian degeneration (''primary'' primary progressive freezing of gait[45]), CBS, or primary

TABLE 8-8 Suggestive Features of Psychogenic Gait Disturbance

Suggestive Features	Caveats and Pitfalls
Variable, inconsistent pattern which is usually worsened when bystanders are present	Episodic weakness in myasthenia gravis, cataplexy, and paroxysmal dyskinesias
Incongruous with known gait disorders (bizarre)	Especially if consistent over time, might be caused by less-known conditions such as cataplexy, task-specific dystonic gait, or drug-induced dyskinesias[13]
Rarely falls/injuries	Exceptions with sometimes severe injuries have been described[51]
Momentary fluctuations of stance and gait, often in response to suggestion[a]	
Excessive slowness (without sequence effect) or hesitation[a]	Pyramidal slowness
''Psychogenic'' Romberg test with a build-up of sway amplitudes after a silent latency or with improvement by distraction[a]	
Nonphysiologic pattern with uneconomic postures and wastage of muscular energy[a]	Dystonia
''Walking on ice'' gait pattern (small, cautious steps with fixed ankle joints)[a]	Cautious gait is often the presenting feature of organic diseases (eg, stiff person syndrome or orthostatic tremor)
Sudden buckling of the knees, usually without falling (the most common type of pure psychogenic gait disorder)[a]	
Abrupt onset	Stroke, etc
Incongruous affect (la belle indifference)	Not easily assessable
Secondary gain	Not easily assessable, especially with long disease duration
Prior or actual history of psychiatric disease	Common in many organic conditions (eg, dementia), ''fear of falling'' in case of balance disorders

[a] These features proved most valuable for diagnosis of psychogenesis, as they occurred alone or in combination in 97% of subjects enrolled in one study.[50]

lateral sclerosis.[46] A recent study with ^{123}I-FP-CIT single-photon emission computed tomography (SPECT) in patients with clinically defined primary progressive freezing of gait found that nigrostriatal denervation predicted possible disease progression toward pure akinesia or PSP.[47] By contrast, normal SPECT findings were associated with possible development of CBS or primary lateral sclerosis[47] (**Case 8-1**).

Senile Gait

The term ''senile gait'' originally referred to ambulatory problems that would develop with advanced aging in very old people, without identifiable neurologic explanation.[33] This view has been challenged. Vascular disease appears to be the most common neurologic cause of senile gait.[48] Other studies have shown that the most common causes were sensory ataxia, myelopathy, multiple strokes, and parkinsonism.[33] Because most patients with senile gait in fact had an underlying degenerative or cerebrovascular disorder, the term ''senile gait'' is discouraged for use as a specific gait category.

From a clinical perspective, senile gait is characterized by two basic patterns with variable combinations: a phenotype resembling the cautious gait pattern (wide-based and slow,

Case 8-2

A 55-year-old man presented with a rapidly evolving gait disorder. Five years before this evaluation, he presented with right-hand resting tremor and was diagnosed with Parkinson disease, achieving a satisfactory improvement with low doses of a dopamine agonist. Unfortunately, this drug induced compulsive eating, and he gained 10 kg in a few years. Because of the onset of gait impairment, the treating physician increased the doses of the dopamine agonist, which demonstrated no clinical benefit and caused a further gain of weight. At the current evaluation, his gait was characterized by small steps, with flexed knees, and was accompanied by dyspnea and knee joint pain. The final diagnosis was a comorbid antalgic gait due to knee arthrosis in a patient with concurrent Parkinson disease and obesity. He reduced the dopamine agonist dose and started dietary restrictions, rehabilitation, and a nonsteroidal anti-inflammatory drug to reduce pain. A few months later, he had lost 5 kg and gained a significant improvement of mobility.

Comment. A comprehensive approach to the patient's conditions and a careful examination of gait allows for recognition of conditions that are potentially manageable.

with an increased double-support time), and a higher-level gait disorder phenotype in more advanced cases. In recent years, the idea of senile gait as a specific condition of the elderly related to the inevitable consequences of aging has been challenged.

Functional (or Psychogenic) Gait Disorders

Functional (or psychogenic) disorders of posture and gait are common and are the major manifestation in 8% to 10% of patients with medically unexplained neurologic symptoms.[49] Like other functional movement disorders, functional gait disorders feature incongruous neurologic signs (a clinical picture not compatible with known organically determined gait patterns) as well as an inconsistent presentation (with variations over time and susceptibility to distraction), and several features are fairly typical (**Table 8-8**) (**Supplemental Digital Content 8-11**, *links.lww.com/CONT/A100*). Anxiety and depression are common but are not required for a diagnosis.

THERAPEUTIC ISSUES

The management of gait disorders should mirror the same stepwise approach used for the diagnosis. The first step is to address the disorders that lead to compensatory changes of gait. As shown by **Case 8-2**, a critical review of medication is also important, because disorders of gait and balance can be a side effect of medications.

The second step is the management of limb features or other destabilization factors—for example, to reduce stiffness (caused by spasticity, spasms, or dystonia) or alleviate hyperkinesias (eg, treatment of myoclonus). The third step is represented by the introduction of interventions focused on the primary etiology of the gait disorder (eg, levodopa for freezing of gait in parkinsonism, or surgical shunting for NPH). Once pharmaceutical or surgical strategies are optimized and tailored for the specific condition and patient, rehabilitation should be started (fourth step). Multidisciplinary rehabilitation is particularly useful for those conditions that are otherwise not treatable (eg, ataxia

KEY POINTS

- Up to 20% of very old individuals walk normally; therefore, even when isolated, gait disorders in the elderly are not inevitable consequences of aging and may reflect underlying cerebrovascular or neurodegenerative disease.
- Functional gait disorders have the same core features as other functional movement disorders: inconsistency (changing patterns over time, with susceptibility to distraction) and incongruence (clinical picture not compatible with known organically determined gait patterns).

KEY POINTS

- High doses of antispasticity drugs might hinder the supportive abilities of the legs, especially when the patient relies on spasticity for standing.
- For focal spasticity, chemodenervation with botulinum neurotoxin is recommended as a first-line agent.

or myopathies). Treatment is focused on the underlying primary deficit (eg, strength training for weakness), support of the compensatory mechanisms (eg, cueing strategies for freezing of gait), the prevention of secondary complications (eg, fall prevention strategies, promotion of mobility to reduce dependence), and increasing the volume of physical activities.

The last step is educating patients and caregivers about individual exercise routines beyond the physical therapy program. The introduction of walking aids should be also considered and trained with the patient if needed. Indeed, the ultimate goal is always to give patients the tools to mobilize themselves independently.

An exception to the aforementioned staged approach is the treatment of psychogenic gait disorder, for which optimal management is still a challenge. Some patients respond to psychological management and rehabilitation therapies, but persistence for more than 6 to 12 months is frequently associated with an unfavorable prognosis and long-term disability.

MEDICAL TREATMENT

In general, drugs that can improve gait can be subdivided into seven categories.

1. Antispasticity drugs are used to reduce muscular hypertonia. First-line drugs are baclofen, dantrolene, and tizanidine, although the off-label prescription of benzodiazepines can be also considered. At low doses, antispasticity drugs have a modest clinical impact on gait abilities, as they mostly improve painful spasms rather than spasticity. Patients not responding to one drug may respond to another, but a combination of drugs should also be tried. Because of their wide distribution, orally administered medications have the advantage of reducing spasticity in muscles throughout the body. However, their wide distribution also has the disadvantage of producing systemic side effects. Moreover, high doses of antispasticity drugs might hinder the supportive abilities of the legs, especially when the patient relies on spasticity for standing.

   For focal spasticity, focal chemodenervation with botulinum neurotoxin (BoNT) or phenol injections may be considered, with BoNT as a first-line agent. The most commonly targeted muscles are the medial popliteal for spastic ankle flexion, the tibialis posterior for foot inversion, and obturator muscles for scissoring gait. Both treatments might be implemented by the guidance of an ultrasound scan, nerve stimulator, or EMG to locate the appropriate neural target for denervation.
2. Antidyskinetic drugs can be used to treat dystonic or choreatic gait. Anticholinergics, baclofen, and BoNT can be used to treat dystonia. Choreatic movements and tics might be managed by neuroleptics, but these agents may cause parkinsonism. The vesicular monoamine transporter-2 inhibitor tetrabenazine works as a dopamine depleter with minimal antagonism of dopaminergic receptors. Antiepileptics and clonazepam can be used for the treatment of paroxysmal dyskinesias and myoclonus.
3. Dopaminergic drugs are used to treat the hypokinetic elements of gait in patients with parkinsonism. Treatment for freezing of gait deserves a special consideration because levodopa generally alleviates freezing of gait but might also have a negative impact

(**Figure 8-7A**). Dopamine agonists have also been associated with the appearance of freezing of gait,[52] suggesting a pathogenic role of unbalanced dopaminergic stimulation on D2-like receptors without corresponding D1 stimulation or, alternatively, excessive stimulation in extranigrostriatal circuitries.

4. Other drugs can be considered for the treatment of levodopa-resistant freezing of gait. Methylphenidate—a psychostimulant originally used for the treatment of attention deficit disorder—has received growing interest in recent years. Although a randomized pilot trial reported no beneficial effect, another recent multicenter, double-blind, placebo-controlled, randomized trial found that methylphenidate (1 mg/kg/d) improved gait hypokinesia and freezing of gait in patients with PD receiving deep brain stimulation (DBS) of the subthalamic nucleus.[53] The main side effects are increased heart rate, weight loss, anxiety, and insomnia. Methylphenidate is mainly seen as a dopaminergic agent, but may also improve freezing of gait by influencing noradrenergic systems or improving attention. L-Threo-3, 4-dihydroxyphenylserine, a synthetic norepinephrine precursor, has produced slight to marked improvement of freezing of gait in 64% of patients with pure akinesia with freezing of gait.[54] Similarly, a marked improvement with duloxetine, a reuptake inhibitor of serotonin and norepinephrine, has been reported in a case of primary progressive freezing of gait.[55] High-dose selegiline, possibly working as an amphetamine, has also been successfully used in a similar patient.[56] BoNT injection in the calf muscles was initially proposed on the basis of their early contraction observed before a freezing-of-gait episode; however, a double-blind, placebo-controlled pilot study found that it did not improve freezing of gait and increased fall risk.[57]
5. Psychostimulants can be also used with several purposes other than treating freezing of gait: they improve fatigue, somnolence, and weakness but also promote attention and cognitive resources. A single dose of 20 mg of methylphenidate was found to improve gait function as well as measures of executive functions in 26 older adults without freezing of gait.[58] Donepezil (an acetylcholinesterase inhibitor specifically used to enhance cognition) has been shown to be beneficial in some patients with levodopa-resistant freezing of gait.
6. Drugs can be used to improve postural stability; however, imbalance is rather difficult to treat, and with the exception of dopaminergic drugs in PD, antivertiginous drugs for vestibular diseases, and acetazolamide for episodic ataxias, the majority of drugs have disappointing results. Nonetheless, the observations that falls are common in patients with dementia and that a dysexecutive syndrome might be the primary cause of idiopathic falls in elderly patients have supported the use of cognitive enhancers to improve stability. This has been proposed for methylphenidate[58] and donepezil.[59]
7. Drugs to improve muscular strength can be used for gait disorders caused by weakness, particularly for those conditions associated with the proximal type. With the exception of peripheral cholinesterase inhibitors and 3,4-diaminopyridine or

Case 8-3

A 79-year-old woman came to the clinic with an abnormal gait, which had slowly developed over the past year, accompanied by several falls; she also reported urinary incontinence and had developed cognitive impairment. Gait was characterized by shuffling steps, but without evidence of parkinsonism. A head CT scan revealed ventriculomegaly that seemed out of proportion to the amount of concurrent atrophy. On lumbar puncture, the opening pressure was normal, and high-volume CSF removal did not improve the clinical picture. As a result, the patient did not undergo ventriculoperitoneal shunting. A few months later, her cognition rapidly worsened, and a clinically based diagnosis of Alzheimer disease was made.

Comment. Clinicians commonly face patients with the full (nonspecific) triad of normal pressure hydrocephalus, with ventriculomegaly on brain imaging but who fail to respond to CSF drainage. True normal pressure hydrocephalus is a rare condition mimicked by a number of neurodegenerative disorders.

4-aminopyridine (4-AP) for the treatment of weakness caused by myasthenia gravis and Lambert-Eaton syndrome, respectively, few other drug treatments can be similarly effective in treating weakness (eg, immunomodulation treatments for autoimmune causes of weakness). 4-AP blocks the voltage-gated potassium channels, thus enhancing neural transmission by improving axonal conduction and synaptic neurotransmitter release. It was initially proposed for gait impairment due to spinal cord injuries, but a randomized clinical trial was negative. It has been found effective in patients with multiple sclerosis, but its use is limited by seizures associated with fluctuating serum drug levels. More recently, the extended-release formulation of 4-AP (dalfampridine) dosed at 10 mg twice daily was approved by the US Food and Drug Administration to improve walking in people with multiple sclerosis. The most common adverse events include increased falls, urinary tract infections, dizziness, insomnia, and headaches.[60]

SURGICAL INTERVENTIONS

Selected surgical interventions can be of benefit, including orthopedic interventions to improve range of motion at affected joints, dorsal rhizotomy as antispasticity intervention, and intrathecal baclofen for lower limb spasticity refractory to oral treatments. Ventriculoperitoneal shunt placement improves patients with NPH whose external lumbar drainage procedure suggests an improvement in gait with fluid diversion (**Case 8-3**).

For PD, evidence suggests that subthalamic nucleus stimulation improves freezing of gait, but only when it occurs in the off state. Moreover, concerns exist about the development of secondary gait worsening or postural deficits postoperatively—not immediately after surgery but only after several years, even in the face of a persistent beneficial effect on appendicular motor control.[61] Reducing the stimulation frequency (60 Hz to 80 Hz instead of 130 Hz or above) has been suggested to optimize DBS treatment.[62] DBS of GPi remains a good alternative, especially because it seems associated with fewer motor side effects over time.[63]

However, the initial improvement induced by GPi DBS may not be retained in the long term.[64]

Other targets for either superficial (eg, spinal cord or motor cortex stimulation) or deep (eg, PPN) stimulation are currently being explored, but it is unclear whether they will emerge as a therapy for gait impairment, as the bulk of evidence is only weakly favorable, at best.[65]

CONCLUSION

Since gait disorders are a major determinant of quality of life and mortality, they should receive great attention in the care of neurologic patients. This review has presented a clinically oriented approach to gait disorders based on the dominant phenomenology and underlying pathophysiology. Gait is very sensitive to any insult to the nervous system; therefore, a careful diagnostic approach is needed to disentangle the underlying causes, the necessary step to guide the therapeutic management of any gait disorder.

USEFUL WEBSITES

American Academy of Neurology
www.aan.com

European Parkinson's Disease Foundation
www.epda.eu.com

Functional and Dissociative Neurological Symptoms: A Patient's Guide
www.neurosymptoms.org

International Society of Posture & Gait Research
www.ispgr.org

Life NPH: Hope for People With Normal Pressure Hydrocephalus
www.lifenph.com

The Movement Disorders Society
www.movementdisorders.org

National Ataxia Foundation
www.ataxia.org

Parkinson Research Foundation
www.parkinsonresearchfoundation.org

Parkinson's Disease Foundation
www.pdf.org

Rescue Consortium
www.rescueproject.org

WE MOVE: Worldwide Education and Awareness for Movement Disorders
www.wemove.org

VIDEO LEGENDS

Supplemental Digital Content 8-1

Ataxic gait. Video shows ataxic gait in a patient with alcoholic ataxia. Note the wide base of support and feet dysmetria during stepping.

links.lww.com/CONT/A90

Supplemental Digital Content 8-2

Difficulty performing tandem gait in a patient with severe essential tremor. Video shows a patient with severe essential tremor having difficulty performing tandem gait. Despite the lack of any obvious ataxic feature of gait, the patient displays a series of missteps.

links.lww.com/CONT/A91

Supplemental Digital Content 8-3

Quadriceps gait in a patient with cervical myelopathy. Video shows the gait in a patient with cervical myelopathy causing spastic paraparesis. In detail, the weakness of quadriceps causes the knee hyperextension at each step on both legs.

links.lww.com/CONT/A92

Supplemental Digital Content 8-4

Hemiparetic gait. Video shows hemiparetic gait in a patient with left-hemisphere stroke. Note lower limb circumduction.

links.lww.com/CONT/A93

Supplemental Digital Content 8-5

Scissoring gait. Video shows scissoring gait in a patient with secondary dystonia due to juvenile cerebral palsy.

links.lww.com/CONT/A94

Supplemental Digital Content 8-6

Dyskinetic gait. Video shows dyskinetic gait in a patient with lower limb dystonia due to a mitochondrial disorder. Note the left foot inversion.

links.lww.com/CONT/A95

Supplemental Digital Content 8-7

Dyskinetic and stiff gait. Video shows dyskinetic and stiff gait in a patient with Parkinson disease experiencing disabling levodopa-induced dyskinesia. Note the dystonic features of the left lower limb.

links.lww.com/CONT/A96

Supplemental Digital Content 8-8

Slow-motion kinematic of sequence effect. Video shows the slow-motion kinematic of feet markers in a patient with Parkinson disease experiencing freezing of gait allows detection of the progressive reduction of step length typical of the sequence effect.

links.lww.com/CONT/A97

Supplemental Digital Content 8-9

Small-stepped gait, tight-quarters hesitation, and freezing of gait. Video shows small-stepped gait, tight-quarters hesitation, and freezing of gait in a patient with long-standing Parkinson disease.

links.lww.com/CONT/A98

Supplemental Digital Content 8-10

Stiff gait and freezing of gait. Video shows the patient with primary lateral sclerosis described in **Case 8-1** exhibiting stiff gait due to spastic paraparesis and freezing of gait during turning.

links.lww.com/CONT/A99

Supplemental Digital Content 8-11

Sudden buckling of the knees in functional/psychogenic gait. Video shows the sudden buckling of the knees in a girl with functional/psychogenic gait. Note the inconsistency and variability of the disorder over time.

links.lww.com/CONT/A100

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