Chapter 26: Gait Disorders, Imbalance, and Falls

Jessica M. Baker

PREVALENCE, MORBIDITY, AND MORTALITY

Gait and balance problems are common in the elderly and contribute to the risk of falls and injury. Gait disorders have been described in 15% of individuals aged >65. By age 80, one person in four will use a mechanical aid to assist with ambulation. Among those aged ≥85, the prevalence of gait abnormality approaches 40%. In epidemiologic studies, gait disorders are consistently identified as a major risk factor for falls and injury.

ANATOMY AND PHYSIOLOGY

An upright bipedal gait depends on the successful integration of postural control and locomotion. These functions are widely distributed in the central nervous system. The biomechanics of bipedal walking are complex, and the performance is easily compromised by a neurologic deficit at any level. Command and control centers in the brainstem, cerebellum, and forebrain modify the action of spinal pattern generators to promote stepping. While a form of “fictive locomotion” can be elicited from quadrupedal animals after spinal transection, this capacity is limited in primates. Step generation in primates is dependent on locomotor centers in the pontine tegmentum, midbrain, and subthalamic region. Locomotor synergies are executed through the reticular formation and descending pathways in the ventromedial spinal cord. Cerebral control provides a goal and purpose for walking and is involved in avoidance of obstacles and adaptation of locomotor programs to context and terrain.

Postural control requires the maintenance of the center of mass over the base of support through the gait cycle. Unconscious postural adjustments maintain standing balance: long latency responses are measurable in the leg muscles, beginning 110 milliseconds after a perturbation. Forward motion of the center of mass provides propulsive force for stepping, but failure to maintain the center of mass within stability limits results in falls. The anatomic substrate for dynamic balance has not been well defined, but the vestibular nucleus and midline cerebellum contribute to balance control in animals. Patients with damage to these structures have impaired balance while standing and walking.

Standing balance depends on good-quality sensory information about the position of the body center with respect to the environment, support surface, and gravitational forces. Sensory information for postural control is primarily generated by the visual system, the vestibular system, and proprioceptive receptors in the muscle spindles and joints. A healthy redundancy of sensory afferent information is generally available, but loss of two of the three pathways is sufficient to compromise standing balance. Balance disorders in older individuals sometimes result from multiple insults in the peripheral sensory systems (e.g., visual loss, vestibular deficit, peripheral neuropathy) that critically degrade the quality of afferent information needed for balance stability.

Older patients with cognitive impairment appear to be particularly prone to falls and injury. There is a growing body of literature on the use of attentional resources to manage gait and balance. Walking is generally considered to be unconscious and automatic, but the ability to walk while attending to a cognitive task (*dual-task walking*) may be compromised in the elderly. Older patients with deficits in executive function may have particular difficulty in managing the attentional resources needed for dynamic balance when distracted.

DISORDERS OF GAIT

Disorders of gait may be attributed to neurologic and nonneurologic causes, although significant overlap often exists. The *antalgic gait* results from avoidance of pain associated with weight bearing and is commonly seen in osteoarthritis. Asymmetry is a common feature of gait disorders due to contractures and other orthopedic deformities. Impaired vision rounds out the list of common nonneurologic causes of gait disorders.

Neurologic gait disorders are disabling and equally important to address. The heterogeneity of gait disorders observed in clinical practice reflects the large network of neural systems involved in the task. Walking is vulnerable to neurologic disease at every level. Gait disorders have been classified

descriptively on the basis of abnormal physiology and biomechanics. One problem with this approach is that many failing gaits look fundamentally similar. This overlap reflects common patterns of adaptation to threatened balance stability and declining performance. *The gait disorder observed clinically must be viewed as the product of a neurologic deficit and a functional adaptation.* Unique features of the failing gait are often overwhelmed by the adaptive response. Some common patterns of abnormal gait are summarized next. Gait disorders can also be classified by etiology **(Table 26-1)**.

TABLE 26-1

Prevalence of Neurologic Gait Disorders

NEUROLOGIC GAIT DISORDER	NO. (%)[a]	TOTAL NUMBER[b]	CAUSES (NO.)
Single neurologic gait disorder	81 (69%)		
Sensory ataxic	22 (18%)	46	Peripheral sensory neuropathy (46)
Parkinsonian	19 (16%)	34	Parkinson's disease (18), drug-induced parkinsonism (8), dementia with parkinsonism (4), parkinsonism (4)
Higher level	9 (8%)	31	Vascular encephalopathy (20), normal pressure hydrocephalus (1), severe dementia (7), hypoxic ischemic encephalopathy (1), unknown (1)
Cerebellar ataxic	7 (6%)	10	Cerebellar stroke (3), cerebellar lesion due to multiple sclerosis (1), severe essential tremor (3), postvaccinal cerebellitis (1), chronic alcohol abuse (1), multiple system atrophy (1)
Cautious	7 (6%)	7	Idiopathic, associated fear of falling (7)
Paretic/hypotonic	6 (5%)	14	Neurogenic claudication (7), diabetic neuropathy (1), nerve lesion due to trauma or surgery (4), distal paraparesis after Guillain-Barré syndrome (1), unknown (2)
Spastic	6 (5%)	7	Ischemic stroke (3), intracerebral hemorrhage (3), congenital (1)
Vestibular ataxic	4 (3%)	6	Bilateral vestibulopathy (3), recent vestribular neuronitis (1), recent Ménière's attack (1), acoustic neuroma with surgery (1)
Dyskinetic	1 (1%)	4	Levodopa-induced dyskinesia (3), chorea (1)
Multiple neurologic gait disorders	36 (30%)		
Total	117		

[a]Percentage of individuals with a single gait disorder. [b]Includes individuals with multiple gait disorders.

Note: Of 117 patients with a neurologic gait disorder, 81 had a single neurologic gait disorder; the remainder (36) had multiple neurologic gait disorders.

Source: Reproduced with modifications from P Mahlknecht et al: PLoS One 8:e69627, 2013.

CAUTIOUS GAIT

The term *cautious gait* is used to describe the patient who walks with an abbreviated stride, widened base, and lowered center of mass, as if walking on a slippery surface. Arms are often held abducted. This disorder is both common and nonspecific. It is, in essence, an adaptation to a perceived postural threat. There may be an associated fear of falling. This disorder can be observed in more than one-third of older patients with gait impairment. Physical therapy often improves walking to the degree that follow-up observation may reveal a more specific underlying disorder.

STIFF-LEGGED GAIT

Spastic gait is characterized by stiffness in the legs, an imbalance of muscle tone, and a tendency to circumduct and scuff the feet. The disorder reflects compromise of corticospinal command and overactivity of spinal reflexes. The patient may walk on the toes. In extreme instances, the legs cross due to increased tone in the adductors ("scissoring" gait). Upper motor neuron signs are present on physical examination. The disorder may be cerebral or spinal in origin.

Myelopathy from cervical spondylosis is a common cause of spastic or spastic-ataxic gait in the elderly. Demyelinating disease and trauma are the leading causes of myelopathy in younger patients. In chronic progressive myelopathy of unknown cause, a workup with laboratory and imaging tests may establish a diagnosis. A structural lesion, such as a tumor or a spinal vascular malformation, should be excluded with appropriate testing. **Spinal cord disorders are discussed in detail in Chap. 442.**

With cerebral spasticity, asymmetry is common, the upper extremities are usually involved, and dysarthria is often an associated feature. Common causes include vascular disease (stroke), multiple sclerosis, motor neuron disease, and perinatal nervous system injury (cerebral palsy).

Other stiff-legged gaits include dystonia **(Chap. 436)** and stiff-person syndrome **(Chap. 94)**. Dystonia is a disorder characterized by sustained muscle contractions resulting in repetitive twisting movements and abnormal posture. It often has a genetic basis. Dystonic spasms can produce plantar flexion and inversion of the feet, sometimes with torsion of the trunk. In autoimmune stiff-person syndrome, exaggerated lordosis of the lumbar spine and overactivation of antagonist muscles restrict trunk and lower-limb movement and result in a wooden or fixed posture.

PARKINSONISM, FREEZING GAIT, AND OTHER MOVEMENT DISORDERS

Parkinson's disease **(Chap. 435)** is common, affecting 1% of the population >65 years of age. The stooped posture, shuffling gait, and decreased arm swing are characteristic and distinctive features. Patients sometimes accelerate (festinate) with walking, display retropulsion, or exhibit a tendency to turn en bloc. The step-to-step variability of the parkinsonian gait also contributes to falls, which are a major source of morbidity, particularly later in the disease course. Dopamine replacement improves step length, arm swing, turning speed, and gait initiation. There is increasing evidence that deficits in cholinergic circuits in the pedunculopontine nucleus and cortex contribute to the gait disorder of Parkinson's disease. Cholinesterase inhibitors such as donepezil and rivastigmine have been shown in early studies to significantly decrease gait variability, instability, and fall frequency, even in the absence of cognitive impairment, perhaps through improvement in attention.

Freezing is defined as a brief, episodic absence of forward progression of the feet, despite the intention to walk. Freezing may be triggered by approaching a narrow doorway or crowd, may be overcome by visual cueing, and contributes to fall risk. Gait freezing is present in approximately one-quarter of Parkinson's patients within 5 years of onset, and its frequency increases further over time. In treated patients, end-of-dose gait freezing is a common problem that may improve with more frequent administration of dopaminergic drugs or with use of monoamine oxidase type B inhibitors such as rasagiline or selegiline **(Chap. 435)**.

Freezing of gait is also common in other neurodegenerative disorders associated with parkinsonism, including progressive supranuclear palsy (PSP), multiple-system atrophy, and corticobasal degeneration. Patients with these disorders frequently present with axial stiffness, postural instability, and a shuffling, freezing gait while lacking the characteristic pill-rolling tremor of Parkinson's disease. The gait of PSP is typically more erect compared with the stooped posture of typical Parkinson's disease, and falls within the first year also suggest the possibility of PSP. The gait of vascular parkinsonism tends to be broad-based and shuffling with reduced arm swing bilaterally; disproportionate involvement of gait early in the disease course differentiates this entity from Parkinson's disease.

Hyperkinetic movement disorders also produce characteristic and recognizable disturbances in gait. In Huntington's disease **(Chap. 436)**, the unpredictable occurrence of choreic movements gives the gait a dancing quality. Tardive dyskinesia is the cause of many odd, stereotypic gait

disorders seen in patients chronically exposed to antipsychotics and other drugs that block the D_2 dopamine receptor. *Orthostatic tremor* is a high-frequency, low-amplitude tremor predominantly involving the lower extremities. Patients often report shakiness or unsteadiness on standing and improvement with sitting or walking. Falls are common. The tremor is often only appreciable by palpating the legs while standing.

FRONTAL GAIT DISORDER

Frontal gait disorder, also known as higher-level gait disorder, is common in the elderly and has a variety of causes. The term is used to describe a shuffling, freezing gait with imbalance, and other signs of higher cerebral dysfunction. Typical features include a wide base of support, a short stride, shuffling along the floor, and difficulty with starts and turns. Many patients exhibit a difficulty with gait initiation that is descriptively characterized as the "slipping clutch" syndrome or gait ignition failure. The term *lower-body parkinsonism* is also used to describe such patients. Strength is generally preserved, and patients are able to make stepping movements when not standing and maintaining their balance at the same time. This disorder is best considered a higher-level motor control disorder, as opposed to an apraxia **(Chap. 30)**, though the term *gait apraxia* persists in the literature.

The most common cause of frontal gait disorder is vascular disease, particularly subcortical small-vessel disease in the deep frontal white matter and centrum ovale. Over three-quarters of patients with subcortical vascular dementia demonstrate gait abnormalities; decreased arm swing and a stooped posture are particularly prevalent features. The clinical syndrome also includes dysarthria, pseudobulbar affect (emotional disinhibition), increased tone, and hyperreflexia in the lower limbs.

Normal pressure (communicating) hydrocephalus (NPH) in adults also presents with a similar gait disorder **(Chap. 431)**. Other features of the diagnostic triad (mental changes, incontinence) may be absent in a substantial number of patients. MRI demonstrates ventricular enlargement, an enlarged flow void about the aqueduct, periventricular white matter change, and high-convexity tightness (disproportionate widening of the sylvian fissures versus the cortical sulci). A lumbar puncture or dynamic test is necessary to confirm a diagnosis of NPH. Neurodegenerative dementias and mass lesions of the frontal lobes cause a similar clinical picture and can be differentiated from vascular disease and hydrocephalus by neuroimaging.

CEREBELLAR GAIT ATAXIA

Disorders of the cerebellum **(Chap. 439)** have a dramatic impact on gait and balance. Cerebellar gait ataxia is characterized by a wide base of support, lateral instability of the trunk, erratic foot placement, and decompensation of balance when attempting to walk on a narrow base. Difficulty maintaining balance when turning is often an early feature. Patients are unable to walk tandem heel to toe and display truncal sway in narrow-based or tandem stance. They show considerable variation in their tendency to fall in daily life.

Causes of cerebellar ataxia in older patients include stroke, trauma, tumor, and neurodegenerative disease such as multiple-system atrophy **(Chap. 440)** and various forms of hereditary cerebellar degeneration **(Chap. 439)**. A short expansion at the site of the fragile X mutation (*fragile X premutation*) has been associated with gait ataxia in older men. Alcohol causes an acute and chronic cerebellar ataxia. In patients with ataxia due to cerebellar degeneration, MRI demonstrates the extent and topography of cerebellar atrophy.

SENSORY ATAXIA

As reviewed earlier in this chapter, balance depends on high-quality afferent information from the visual and the vestibular systems and proprioception. When this information is lost or degraded, balance during locomotion is impaired and instability results. The sensory ataxia of tabetic neurosyphilis is a classic example. The contemporary equivalent is the patient with neuropathy affecting large fibers. Vitamin B_{12} deficiency is a treatable cause of large-fiber sensory loss in the spinal cord and peripheral nervous system. Joint position and vibration sense are diminished in the lower limbs. The stance in such patients is destabilized by eye closure; they often look down at their feet when walking and do poorly in the dark. **Table 26-2** compares sensory ataxia with cerebellar ataxia and frontal gait disorder.

TABLE 26-2

Features of Cerebellar Ataxia, Sensory Ataxia, and Frontal Gait Disorders

FEATURE	CEREBELLAR ATAXIA	SENSORY ATAXIA	FRONTAL GAIT
Base of support	Wide-based	Wide-based, looks down	Wide-based
Velocity	Variable	Slow	Very slow
Stride	Irregular, lurching	Regular with path deviation	Short, shuffling
Romberg test	+/–	Unsteady, falls	+/–
Heel → shin	Abnormal	+/–	Normal
Initiation	Normal	Normal	Hesitant
Turns	Unsteady	+/–	Hesitant, multistep
Postural instability	+	+++	++++ Poor postural synergies rising from a chair
Falls	Late event	Frequent	Frequent

NEUROMUSCULAR DISEASE

Patients with neuromuscular disease often have an abnormal gait, occasionally as a presenting feature. With distal weakness (peripheral neuropathy), the step height is increased to compensate for foot drop, and the sole of the foot may slap on the floor during weight acceptance, termed the *steppage gait*. Patients with myopathy or muscular dystrophy more typically exhibit proximal weakness. Weakness of the hip girdle may result in some degree of excess pelvic sway during locomotion. The stooped posture of lumbar spinal stenosis ameliorates pain from the compression of the cauda equina occurring with a more upright posture while walking and may mimic early parkinsonism.

TOXIC AND METABOLIC DISORDERS

Chronic toxicity from medications and metabolic disturbances can impair motor function and gait. Examination may reveal mental status changes, asterixis, or myoclonus. Static equilibrium is disturbed, and such patients are easily thrown off balance. Disequilibrium is particularly evident in patients with chronic renal disease and those with hepatic failure, in whom asterixis may impair postural support. Sedative drugs, especially neuroleptics and long-acting benzodiazepines, affect postural control and increase the risk for falls. These disorders are especially important to recognize because they are often treatable.

FUNCTIONAL GAIT DISORDER

Functional neurologic disorders (formerly “psychogenic”) are common in practice, and the presentation often involves gait. Sudden onset, inconsistent deficits, waxing and waning course, incongruence of symptoms with an organic lesion, and improvement with distraction are key features. Phenomenology is variable; extreme slow motion, an inappropriately overcautious gait, odd gyrations of posture with wastage of muscular energy, astasia–abasia (inability to stand and walk), bouncing, and foot stiffness (dystonia) have been described. Falls are rare, and there are often discrepancies between examination findings and the patient’s functional status. Preceding stress or trauma is variably present, and its absence does not preclude the diagnosis of a functional gait disorder. Functional gait disorders may be challenging to diagnose and should be differentiated from the slowness and psychomotor retardation seen in certain patients with major depression.

APPROACH TO THE PATIENT WITH SLOWLY PROGRESSIVE DISORDER OF GAIT

When reviewing the history, it is helpful to inquire about the onset and progression of disability. Initial awareness of an unsteady gait often follows a fall. Stepwise evolution or sudden progression suggests vascular disease. Gait disorder may be associated with urinary urgency and incontinence, particularly in patients with cervical spine disease or hydrocephalus. It is always important to review the use of alcohol and medications that affect gait and balance. Information on localization derived from the neurologic examination can be helpful in narrowing the list of possible diagnoses.

Gait observation provides an immediate sense of the patient's degree of disability. Arthritic and antalgic gaits are recognized by observation, although neurologic and orthopedic problems may coexist. Characteristic patterns of abnormality are sometimes seen, although, as stated previously, failing gaits often look fundamentally similar. Cadence (steps per minute), velocity, and stride length can be recorded by timing a patient over a fixed distance. Watching the patient rise from a chair provides a good functional assessment of balance.

Brain imaging studies may be informative in patients with an undiagnosed disorder of gait. MRI is sensitive for cerebral lesions of vascular or demyelinating disease and is a good screening test for occult hydrocephalus. Patients with recurrent falls are at risk for subdural hematoma. As mentioned earlier, many elderly patients with gait and balance difficulty have white matter abnormalities in the periventricular region and centrum semiovale. While these lesions may be an incidental finding, a substantial burden of white matter disease will ultimately impact cerebral control of locomotion.

DISORDERS OF BALANCE

DEFINITION, ETIOLOGY, AND MANIFESTATIONS

Balance is the ability to maintain equilibrium—a dynamic state in which one's center of mass is controlled with respect to the lower extremities, gravity, and the support surface despite external perturbations. The reflexes required to maintain upright posture require input from cerebellar, vestibular, and somatosensory systems; the premotor cortex and corticospinal and reticulospinal tracts mediate output to axial and proximal limb muscles. These responses are physiologically complex, and the anatomic representation they entail is not well understood. Failure can occur at any level and presents as difficulty maintaining posture while standing and walking.

The history and physical examination may differentiate underlying causes of imbalance. Patients with *cerebellar* ataxia do not generally complain of dizziness, although balance is visibly impaired. Neurologic examination reveals a variety of cerebellar signs. Postural compensation may prevent falls early on, but falls are inevitable with disease progression. The progression of neurodegenerative ataxia is often measured by the number of years to loss of stable ambulation.

Vestibular disorders **(Chap. 22)** have symptoms and signs that fall into three categories: (1) vertigo (the subjective inappropriate perception or illusion of movement); (2) nystagmus (involuntary eye movements); and (3) impaired standing balance. Not every patient has all manifestations. Patients with vestibular deficits related to ototoxic drugs may lack vertigo or obvious nystagmus, but their balance is impaired on standing and walking, and they cannot navigate in the dark. Laboratory testing is available to investigate vestibular deficits.

Somatosensory deficits also produce imbalance and falls. There is often a subjective sense of insecure balance and fear of falling. Postural control is compromised by eye closure (*Romberg's sign*); these patients also have difficulty navigating in the dark. A dramatic example is provided by the patient with autoimmune subacute sensory neuropathy, which is sometimes a paraneoplastic disorder **(Chap. 94)**. Compensatory strategies enable such patients to walk in the virtual absence of proprioception, but the task requires active visual monitoring.

Patients with *higher-level disorders of equilibrium* have difficulty maintaining balance in daily life and may present with falls. Their awareness of balance impairment may be reduced. Patients taking sedating medications are in this category.

FALLS

Falls are common in the elderly; over one-third of people aged >65 who are living in the community fall each year. This number is even higher in nursing homes and hospitals. Elderly people are not only at higher risk for falls but are also more likely to suffer serious complications due to medical comorbidities such as osteoporosis. Hip fractures result in hospitalization, can lead to nursing home admission, and are associated with an increased mortality risk in the subsequent year. Falls may result in brain or spinal injury, the history of which may be difficult for the patient to provide. The

proportion of spinal cord injuries due to falls in individuals aged >65 years has doubled in the past decade, perhaps due to increasing activity in this age group. Some falls result in a prolonged time lying on the ground; fractures and CNS injury are a particular concern in this context.

For each person who is physically disabled, there are others whose functional independence is limited by anxiety and fear of falling. Nearly one in five elderly individuals voluntarily restricts his or her activity because of fear of falling. With loss of ambulation, the quality of life diminishes, and rates of morbidity and mortality increase.

RISK FACTORS FOR FALLS

Risk factors for falls may be *intrinsic* (e.g., gait and balance disorders) or *extrinsic* (e.g., polypharmacy, environmental factors); some risk factors are modifiable. The presence of multiple risk factors is associated with a substantially increased risk of falls. **Table 26-3** summarizes a meta-analysis of studies establishing the principal risk factors for falls. Polypharmacy (use of four or more prescription medications) has also been identified as an important risk factor.

TABLE 26-3

Meta-Analysis of Risk Factors for Falls in Older Persons

RISK FACTOR	MEAN RR (OR)	RANGE
Muscle weakness	4.4	1.5–10.3
History of falls	3.0	1.7–7.0
Gait deficit	2.9	1.3–5.6
Balance deficit	2.9	1.6–5.4
Use assistive device	2.6	1.2–4.6
Visual deficit	2.5	1.6–3.5
Arthritis	2.4	1.9–2.9
Impaired ADL	2.3	1.5–3.1
Depression	2.2	1.7–2.5
Cognitive impairment	1.8	1.0–2.3
Age >80 years	1.7	1.1–2.5

Abbreviations: ADL, activity of daily living; OR, odds ratio from retrospective studies; RR, relative risk from prospective studies.

Source: Reproduced with permission from Guideline for the Prevention of Falls in Older Persons. J Am Geriatr Soc 49:664, 2001.

ASSESSMENT OF THE PATIENT WITH FALLS

The most productive approach is to identify the high-risk patient prospectively, before there is a serious injury. All community-dwelling adults should be asked annually about falls and whether or not fear of falling limits daily activities. The Timed Up and Go ("TUG") test involves timing a patient as they stand up from a chair, walk 10 feet, turn, and then sit down. Patients with a history of falls or those requiring >12 s to complete the TUG test are at high

risk for falls and should undergo further assessment.

History

The history surrounding a fall is often problematic or incomplete, and the underlying mechanism or cause may be difficult to establish in retrospect. Patients should be queried about any provoking factors (including head turn, standing) or prodromal symptoms, such as dizziness, vertigo, presyncopal symptoms, or focal weakness. A history of baseline mobility and medical comorbidities should be elicited. Patients at particular risk include those with mental status changes or dementia. Medications should be reviewed, with particular attention to benzodiazepines, opioids, antipsychotics, antiepileptics, antidepressants, antiarrhythmics, and diuretics, all of which are associated with an increased risk of falls. It is equally important to distinguish *mechanical falls* (those caused by tripping or slipping) due to purely extrinsic or environmental factors from those in which a modifiable intrinsic factor contributes. *Recurrent falls* may indicate an underlying gait or balance disorder. Falls associated with loss of consciousness (syncope, seizure) may require appropriate cardiac or neurologic evaluation and intervention **(Chaps. 21 and 425)**, although a patient's report of change in consciousness may be unreliable.

Physical Examination

Examination of the patient with falls should include a basic cardiac examination, including orthostatic blood pressure if indicated by history, and observation of any orthopedic abnormalities. Mental status is easily assessed while obtaining a history from the patient; the remainder of the neurologic examination should include visual acuity, strength and sensation in the lower extremities, muscle tone, and cerebellar function, with particular attention to gait and balance as described earlier in this chapter.

Fall Patterns

The description of a fall event may provide further clues to the underlying etiology. While there is no standard nosology of falls, some common clinical patterns may emerge and provide a clue.

DROP ATTACKS AND COLLAPSING FALLS

Drop attacks and collapsing falls are associated with a sudden loss of postural tone. Patients may report that their legs just "gave out" underneath them or that they "collapsed in a heap." Syncope or orthostatic hypotension may be a factor in some such falls. Neurologic causes are relatively rare but include atonic seizures, myoclonus, and intermittent obstruction of the foramen of Monro by a colloid cyst of the third ventricle causing acute obstructive hydrocephalus. An emotional trigger suggests cataplexy. While collapsing falls are more common among older patients with vascular risk factors, drop attacks should not be confused with vertebrobasilar ischemic attacks.

TOPPLING FALLS

Some patients maintain tone in antigravity muscles but fall over like a tree trunk, as if postural defenses had disengaged. Causes include cerebellar pathology and lesions of the vestibular system. There may be a consistent direction to such falls. Toppling falls are an early feature of progressive supranuclear palsy, and a late feature of Parkinson's disease, once postural instability has developed. Thalamic lesions causing truncal instability (*thalamic astasia*) may also contribute to this type of fall.

FALLS DUE TO GAIT FREEZING

Freezing of gait is seen in Parkinson's disease and related disorders. The feet stick to the floor and the center of mass keeps moving, resulting in a disequilibrium from which the patient has difficulty recovering, resulting in a forward fall. Similarly, patients with Parkinson's disease and festinating gait may find their feet unable to keep up and may thus fall forward.

FALLS RELATED TO SENSORY LOSS

Patients with somatosensory, visual, or vestibular deficits are prone to falls. These patients have particular difficulty dealing with poor illumination or walking on uneven ground. They often report subjective imbalance, apprehension, and fear of falling. These patients may be especially responsive to a rehabilitation-based intervention.

Patients who lack strength in antigravity muscles have difficulty rising from a chair or maintaining their balance after a perturbation. These patients are often unable to get up after a fall and may have to remain on the floor for a prolonged period until help arrives. If due to deconditioning, this is often treatable. Resistance strength training can increase muscle mass and leg strength, even for people in their eighties and nineties.

TREATMENT OF INTERVENTIONS TO REDUCE THE RISK OF FALLS AND INJURY

Efforts should be made to define the mechanism underlying falls in a given patient, as specific treatment may be possible once a diagnosis is established. Orthostatic changes in blood pressure and pulse should be recorded. Medications (including over-the-counter) should be reviewed, reevaluating benefits and burdens of medications that might increase fall risk. Treatment of cataracts and avoidance of multifocal lenses could be considered for patients whose falls result from vision impairment. A home visit to look for environmental hazards can be helpful. A variety of modifications may be recommended to improve safety, including improved lighting, installation of grab bars and nonslip surfaces, and use of adaptive equipment.

Home- and group-based exercise programs focusing on leg strength and balance, physical therapy, and use of assistive devices reduce fall risk in individuals with a history of falls or disorders of gait and balance. Rehabilitative interventions aim to improve muscle strength and balance stability and to make the patient more resistant to injury. High-intensity resistance strength training with weights and machines is useful to improve muscle mass, even in frail older patients. Improvements realized in posture and gait should translate to reduced risk of falls and injury. Sensory balance training is another approach to improving balance stability. Measurable gains can be made in a few weeks of training, and benefits can be maintained over 6 months by a 10- to 20-min home exercise program. This strategy is particularly successful in patients with vestibular and somatosensory balance disorders. The National Institute on Aging provides online examples of balance exercises for older adults. A Tai Chi exercise program has been demonstrated to reduce the risk of falls and injury in patients with Parkinson's disease. Cognitive training, including dual-task training, may improve mobility in older adults with cognitive impairment.

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FURTHER READING

American Geriatrics Society, British Geriatrics Society, American Academy of Orthopedic Surgeons Panel on Falls Prevention: Guideline for the prevention of falls in older persons. J Am Geriatr Soc 49:664, 2001. [PubMed: 11380764]

Ganz D, Latham N: Prevention of falls in community-dwelling older adults. N Engl J Med 382:734, 2020. [PubMed: 32074420]

National Institute on Aging: Exercise and Physical Activity. Available from *https://www-nia-nih-gov.kaplanmc.idm.oclc.org/health/exercise-physical-activity*. Accessed April 25, 2021.

Nutt JG: Classification of gait and balance disorders. Adv Neurol 87:135, 2001. [PubMed: 11347216]

Pirker W, Katzenschlager R: Gait disorders in adults and the elderly. Wien Klin Wochenschr 129:81, 2017. [PubMed: 27770207]