CAUTIOUS GAIT

The term *cautious gait* is used to describe the patient who walks with an abbreviated stride and lowered center of mass, as if walking on a slippery surface. 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. Upper motor neuron signs are present on physical examination. Shoes often reflect an uneven pattern of wear across the outside. 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 family history should suggest hereditary spastic paraplegia (Chap. 452); genetic testing is now available for some of the common mutations responsible for this disorder. Tropical spastic paraparesis related to the retrovirus human T-cell lymphotropic virus 1 (HTLV-1) is endemic in parts of the Caribbean and South America. 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. 456.**

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, and perinatal injury to the nervous system (cerebral palsy).

Other stiff-legged gaits include dystonia (Chap. 449) and stiff-person syndrome (Chap. 122). 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 AND FREEZING GAIT

Parkinson's disease (Chap. 449) is common, affecting 1% of the population >55 years of age. The stooped posture and shuffling gait are characteristic and distinctive features. Patients sometimes accelerate (festinate) with walking, display retropulsion, or exhibit a tendency to turn en bloc. A National Institutes of Health workshop defined freezing of gait as "brief, episodic absence of forward progression of the feet, despite the intention to walk." Gait freezing occurs in 26% of Parkinson's patients by the end of 5 years and develops in most such patients eventually. Postural instability and falling occur as the disease progresses; some falls are precipitated by freezing of gait.

Freezing of gait is even more common in some Parkinson's-related neurodegenerative disorders, such as progressive supranuclear palsy, 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. Falls within the first year suggest the possibility of progressive supranuclear palsy.

Hyperkinetic movement disorders also produce characteristic and recognizable disturbances in gait. In Huntington's disease (Chap. 449), 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.

FRONTAL GAIT DISORDER

Frontal gait disorder, sometimes known as *gait apraxia*, 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. 36).

The most common cause of frontal gait disorder is vascular disease, particularly subcortical small-vessel disease. Lesions are frequently found in the deep frontal white matter and centrum ovale. Gait disorder may be the salient feature in hypertensive patients with ischemic lesions of the deep-hemisphere white matter (*Binswanger's disease*). The clinical syndrome includes mental changes (variable in degree), dysarthria, pseudobulbar affect (emotional disinhibition), increased tone, and hyperreflexia in the lower limbs.

Communicating hydrocephalus in adults also presents with a gait disorder of this type. Other features of the diagnostic triad (mental changes, incontinence) may be absent in the initial stages. MRI demonstrates ventricular enlargement, an enlarged flow void about the aqueduct, and a variable degree of periventricular white-matter change. A lumbar puncture or dynamic test is necessary to confirm hydrocephalus.

CEREBELLAR GAIT ATAXIA

Disorders of the cerebellum 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 (Chaps. 449 and 454) and various forms of hereditary cerebellar degeneration (Chap. 450). A short expansion at the site of the fragile X mutation (*fragile X pre-mutation*) has been associated with gait ataxia in older men. Alcoholic cerebellar degeneration can be screened by history and often confirmed by MRI. In patients with ataxia, 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 32-2 compares sensory ataxia with cerebellar ataxia and frontal gait disorder. Some frail older patients exhibit a syndrome of imbalance from the combined effect of multiple sensory deficits. Such patients have disturbances in proprioception, vision, and vestibular sense that impair postural support.

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 footdrop, and the sole of the foot may slap on the floor during weight acceptance. Neuropathy may be associated with a degree of sensory imbalance, as described earlier. Patients with myopathy or muscular dystrophy more