TABLE 32-2 FEATURES OF CEREBELLAR ATAXIA, SENSORY ATAXIA, AND FRONTAL GAIT DISORDERS

Feature	Cerebellar Ataxia	Sensory Ataxia	Frontal Gait
Base of support	Wide-based	Narrow base, 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

typically exhibit proximal weakness. Weakness of the hip girdle may result in some degree of excess pelvic sway during locomotion.

TOXIC AND METABOLIC DISORDERS

Alcohol intoxication is the most common cause of acute walking difficulty. Chronic toxicity from medications and metabolic disturbances can impair motor function and gait. Mental status changes may be found, and examination may reveal 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.

PSYCHOGENIC GAIT DISORDER

Psychogenic disorders are common in neurologic practice, and the presentation often involves gait. Some patients with extreme anxiety or phobia walk with exaggerated caution with abduction of the arms, as if walking on ice. This inappropriately overcautious gait differs in degree from the gait of the patient who is insecure and making adjustments for imbalance. Depressed patients exhibit primarily slowness, a manifestation of psychomotor retardation, and lack of purpose in their stride. Hysterical gait disorders are among the most spectacular encountered. Odd gyrations of posture with wastage of muscular energy (astasia–abasia), extreme slow motion, and dramatic fluctuations over time may be observed in patients with somatoform disorders and conversion reactions.

APPROACH TO THE PATIENT: 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, though neurologic and orthopedic problems may coexist. Characteristic patterns of abnormality are sometimes seen, though, 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 state in which opposing physical forces cancel one another out. In physiology, this term is taken to mean the ability to control the center of mass with respect to gravity and the support surface. In reality, people are not consciously aware of their center of mass, but everyone (particularly gymnasts, figure skaters, and platform divers, for example) move so as to manage it. Disorders of balance present as difficulty maintaining posture while standing and walking and as a subjective sense of disequilibrium, which is a form of dizziness.

The cerebellum and vestibular system organize antigravity responses needed to maintain an upright posture. These responses are physiologically complex, and the anatomic representation they entail is not well understood. Failure, resulting in disequilibrium, can occur at several levels: cerebellar, vestibular, somatosensory, and higher-level disequilibrium.

Patients with *cerebellar* ataxia do not generally complain of dizziness, though 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. 28) 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. 122). 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. In prospective studies, dementia and sedating medications substantially increase the risk for falls.

FALLS

Falls are common in the elderly; 30% of people older than 65 who are living in the community fall each year. Modest changes in balance function have been described in fit older individuals as a result of normal aging. Subtle deficits in sensory systems, attention, and motor reaction time contribute to the risk, and environmental hazards abound. Many falls by older adults are episodes of tripping or slipping, often designated *mechanical falls*. A fall is not a neurologic problem per se, but there are events for which neurologic evaluation is appropriate.