

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