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Gait Disorders

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ABSTRACT

Walking is an extraordinarily complex task requiring integration of the entire nervous system, making gait susceptible to a variety of underlying neurologic abnormalities. Gait disorders are particularly prevalent in the elderly and increase fall risk. In this review we discuss an approach to the examination of gait and highlight key features of common gait disorders and their underlying causes. We review gaits due to lesions of motor systems (spasticity and neuromuscular weakness), the cerebellum and sensory systems (ataxia), parkinsonism, and frontal lobes and discuss the remarkably diverse phenomenology of functional (psychogenic) gait disorders. We offer a pragmatic approach to the diagnosis and management of neurologic gait disorders, because prompt recognition and intervention may improve quality of life in affected individuals.
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Gait disorders are common, contribute significantly to morbidity through falls,[1] and may yield clues to diseases occurring at all locations of the nervous system, making the examination of gait one of the most complex and high-yield components of the neurologic examination. In this review we offer a pragmatic approach to examining gait and discuss clinical features of common gait disorders and their underlying etiologies. Abnormal gait is particularly prevalent in the elderly, affecting approximately 1 in 3 community-dwelling individuals older than 60 years. Gait disorders in this population are associated with diminished quality of life[2] and nursing home placement[3] and may be an indicator of progression to dementia in individuals with mild cognitive impairment.[4] A history that includes weakness of the legs, imbalance, unsteadiness on one's feet, or multiple falls may hint at an underlying gait disorder. Prompt recognition, examination, and classification of gait disorders is therefore of paramount importance.

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PHYSIOLOGY AND THE GAIT CYCLE

Normal gait requires precise control of limb movements, posture, and muscle tone, an extraordinarily complex process that involves the entire nervous system. Specialized groups of neurons in the spinal cord and brainstem generate rhythmic activity and provide output to motor neurons, which in turn activate muscles in the limbs. The cerebral cortex integrates input from visual, vestibular, and proprioceptive systems; additional input is received from the brainstem, basal ganglia, cerebellum, and afferent neurons carrying proprioceptive signals from muscle stretch receptors (as may be damaged in peripheral neuropathy). Together, these systems allow individuals to walk not only in a straight, unencumbered line but to adapt their gait to avoid obstacles and adjust posture to maintain balance.[5] Abnormalities of any portion of the nervous system can therefore give rise to a gait disorder.

The gait cycle (**Figure 1**) begins when one heel (illustrated here as right) strikes the ground. Supported by the stance of the right leg, body weight shifts forward as the left leg flexes at the hip and knees and swings forwards, eventually striking the left heel on the ground. Weight then shifts forwards on the left leg, while the right leg swings forward and again strikes the ground. Thus, while one leg is in *stance* phase, the opposite is in *swing* phase. Periods of double support, during which both legs make contact with the ground, normally comprise approximately 10% of the gait cycle[6] but increase as compensation for unsteadiness in many abnormal gaits.

EXAMINATION OF GAIT

The examination of gait begins with observing a patient as he or she walks from the waiting area to an examination room. The ideal setting for a formal gait examination is a long, uncluttered hallway, providing enough distance to reach a comfortable walking speed with good arm swing. Hands should be free except for necessary assistive devices. Observe individuals as they walk in a straight line, but also note any difficulty rising from a chair, initiating gait, or turning. The gait examination provides significant insight into an individual's functional status, and much will be missed if the assessment is limited to the examination room! Make note of *velocity* (distance covered in a given time) and *cadence* (steps per minute). *Stride length* measures distance covered by the gait cycle; *step length* measures the distance covered during the swing phase of a single leg. *Step width* or *base* is the distance between the left and right feet while walking (**Figure 2**). Also make note of posture, arm swing, the height of each step, leg stiffness, or side-to-side lurching. Muscle strength and tone in the legs, sensation, and reflexes may provide further clues as to the etiology of an underlying gait disorder. The Romberg sign is tested by asking patients to stand still with feet together and eyes closed and is considered positive (abnormal) if eye closure provokes a fall. Test tandem gait by asking a patient to take at least 10 steps touching heel-to-toe, as if walking on a tightrope. Heel or toe walking can unmask subtle distal weakness that might be missed by direct confrontational testing.

CLINICAL SIGNIFICANCE

- Gait disorders increase fall risk and often result from an underlying neurologic condition.
- Specific features of abnormal gaits result from a combination of a deficit and attempts at compensation.
- Many gait disorders are readily treatable with specific therapies, such as dopaminergic therapy for Parkinson's disease, or cerebrospinal fluid shunting for normal pressure hydrocephalus.
- Physical therapy and assistive devices may improve mobility and decrease fall risk.

CLINICAL FEATURES AND ETIOLOGY OF GAIT DISORDERS

Gait disorders may be neurologic or nonneurologic in origin. Common nonneurologic causes of abnormal gait include osteoarthritis of the hip and knee, orthopedic deformities, and visual loss[2]; individuals may reduce the stance time of the affected limb to reduce pain, resulting in an asymmetric *antalgic gait*. Common neurologic causes of abnormal gaits are listed in the **Table** and are described here in further detail. Mildly shortened step length, decreased velocity, slightly widened base, and increased double support time are features of normal aging[7] but are also seen as a response to perceived instability, either intrinsic (eg, disequilibrium) or extrinsic (eg, walking on ice). Individuals may walk with hands outstretched in an attempt to steady themselves. This *cautious gait* is nonspecific but may herald an underlying neurologic gait disorder.

Spastic Gait

Spastic gaits are caused by lesions in the corticospinal tract at any level and may be unilateral or bilateral. When unilateral, the affected leg is held in extension and plantar flexion; the ipsilateral arm is often flexed. There is circumduction of the affected leg during the swing phase of each step. Common causes include stroke or other unilateral lesions of the cerebral cortex. If bilateral, the spastic gait may appear stiff-legged or *scissoring* owing to increased tone in the adductor muscles, such that the legs nearly touch with each step

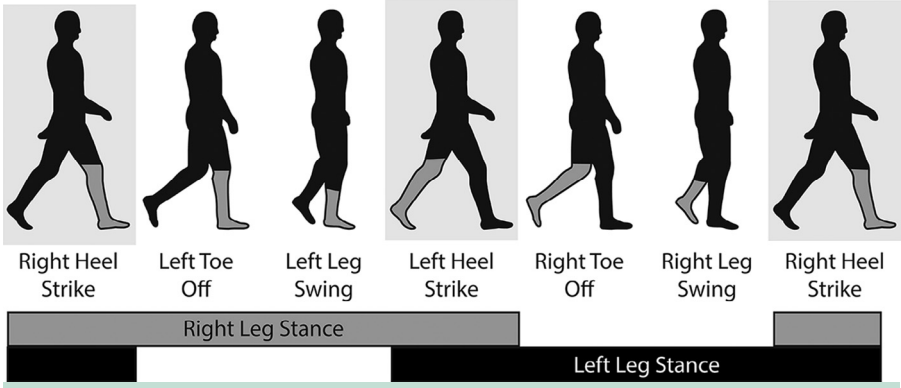

Figure 1 The gait cycle. Right leg is shaded grey. The gait cycle is divided into stance and swing phases. During stance, body weight shifts forward on the supporting leg, while the opposite leg swings forward, eventually making contact with the ground via the heel. Shaded boxes indicate periods of double support, during which both the left and right legs make contact with the ground.