Evaluation of the Elderly Patient With an Abnormal Gait

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None of the following authors or the departments with which they are affiliated has received anything of value from or owns stock in a commercial company or institution related directly or indirectly to the subject of this article: Dr. Lim, Dr. Huang, Dr. Wu, Dr. Girardi, and Dr. Cammisa.

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J Am Acad Orthop Surg 2007;15:107-117

Abstract

Distinguishing between the normal gait of the elderly and pathologic gaits is often difficult. Pathologic gaits with neurologic causes include frontal gait, spastic hemiparetic gait, parkinsonian gait, cerebellar ataxic gait, and sensory ataxic gait. Pathologic gaits with combined neurologic and musculoskeletal causes include myelopathic gait, stooped gait of lumbar spinal stenosis, and steppage gait. Pathologic gaits with musculoskeletal causes include antalgic gait, coxalgic gait, Trendelenburg gait, knee hyperextension gait, and other gaits caused by inadequate joint mobility. A working knowledge of the characteristics of these gaits and a systematic approach to observational gait examination can help identify the causes of abnormal gait. Patients with abnormal gait can benefit from the treatment of the primary cause of the disorder as well as by general fall-prevention interventions. Treatable causes of gait disturbance are found in a substantial proportion of patients and include normal-pressure hydrocephalus, vitamin B_{12} deficiency, Parkinson's disease, alcoholism, medication toxicity, cervical spondylotic myelopathy, lumbar spinal stenosis, joint contractures, and painful disorders of the lower extremity.

Gait abnormalities exist in approximately 15% of people older than age 64 years. By age 85, the prevalence increases to 40%.[1] In the elderly, gait disorders contribute to the risk of falls and subsequent injury. In a study of 1,103 community-living persons older than age 72 years, nearly 50% of the participants experienced at least one fall during a 2.5-year period.[2] Approximately 25% of the elderly who fall experience a serious injury, and approximately 5% have a fracture. Balance and gait impairment nearly double the risk of falling and the risk of experiencing a subsequent serious injury.[3]

The mere fear of falling contributes to the loss of functional independence in many elderly individuals.[4] In one study, nearly 20% of the elderly acknowledged limiting their activities because of the fear of falling.[5] Falls also contribute to permanent institutionalization of elderly individuals, with concomitant decline in functional status and social/physical activities.[6,7]

Although patients rarely present to the orthopaedic surgeon with gait abnormality as a chief complaint, difficulty walking is a very common secondary symptom. The primary clinical focus of orthopaedic surgeons has been on the injuries conse-

Table 1

Classification of Gait Disorders

Neurologic	Combined Neurologic/Musculoskeletal	Musculoskeletal
Frontal gait	Myelopathic gait	Antalgic gait
Dementias (Alzheimer's disease)	Cervical spondylotic myelopathy	Knee osteoarthritis
Normal-pressure hydrocephalus	Vitamin B_{12} deficiency	Other painful disorders of the lower extremity
Binswanger's disease (subcortical dementia)	Multiple sclerosis	Coxalgic gait
Spastic hemiparetic gait	Thoracic disk herniation	Hip osteoarthritis
Cerebrovascular accident	Stooped gait of lumbar spinal stenosis	Other painful hip disorders
Parkinsonian gait	Steppage gait (foot drop)	Mild hip abductor insufficiency
Parkinson's disease	Acquired or hereditary peripheral neuropathy	Trendelenburg gait
Drug-induced parkinsonism	Sciatic/peroneal neuropathy	Severe hip abductor insufficiency
Progressive supranuclear palsy	Lumbar radiculopathy	Knee hyperextension gait
Cerebellar ataxic gait		Ankle equinus contracture
Alcoholism		Quadriceps deficiency
Phenytoin toxicity		Inadequate knee extension gait
Paraneoplastic syndromes		Knee flexion contracture
Hereditary ataxias		Inadequate knee flexion gait
Sensory ataxic gait		Knee extension contracture or fusion
Tabes dorsalis (syphilis)		Inadequate hip extension gait
Vitamin B_{12} deficiency		Hip flexion contracture
Polyneuropathy (diabetes, HIV, neurotoxic medications)		

HIV = human immunodeficiency virus

quent to falling. However, by identifying causes of gait disorders, orthopaedic surgeons can play an important role in a multidisciplinary effort to prevent falls and improve the quality of life in the elderly.

The identification of the cause of a gait abnormality can be challenging because many abnormal gaits in the elderly look similar. Common compensatory patterns of gait often mask the helpful characteristic features. For example, the cautious gait of the elderly, with short wide steps and increased time in double-limb support, is entirely nonspecific.[8] Following an exhaustive diagnostic workup, many gait disturbances are found to be nonspecific and multifactorial. However, potentially treatable causes of gait impairment can be found in nearly one third of patients. The most common causes of gait disorder in the elderly are cerebral infarcts, painful arthritic disorders of the lower extremities, cervical spondylotic myelopathy, Parkinson's disease, and cerebellar degeneration[9,10] (Table 1).

Normal Gait

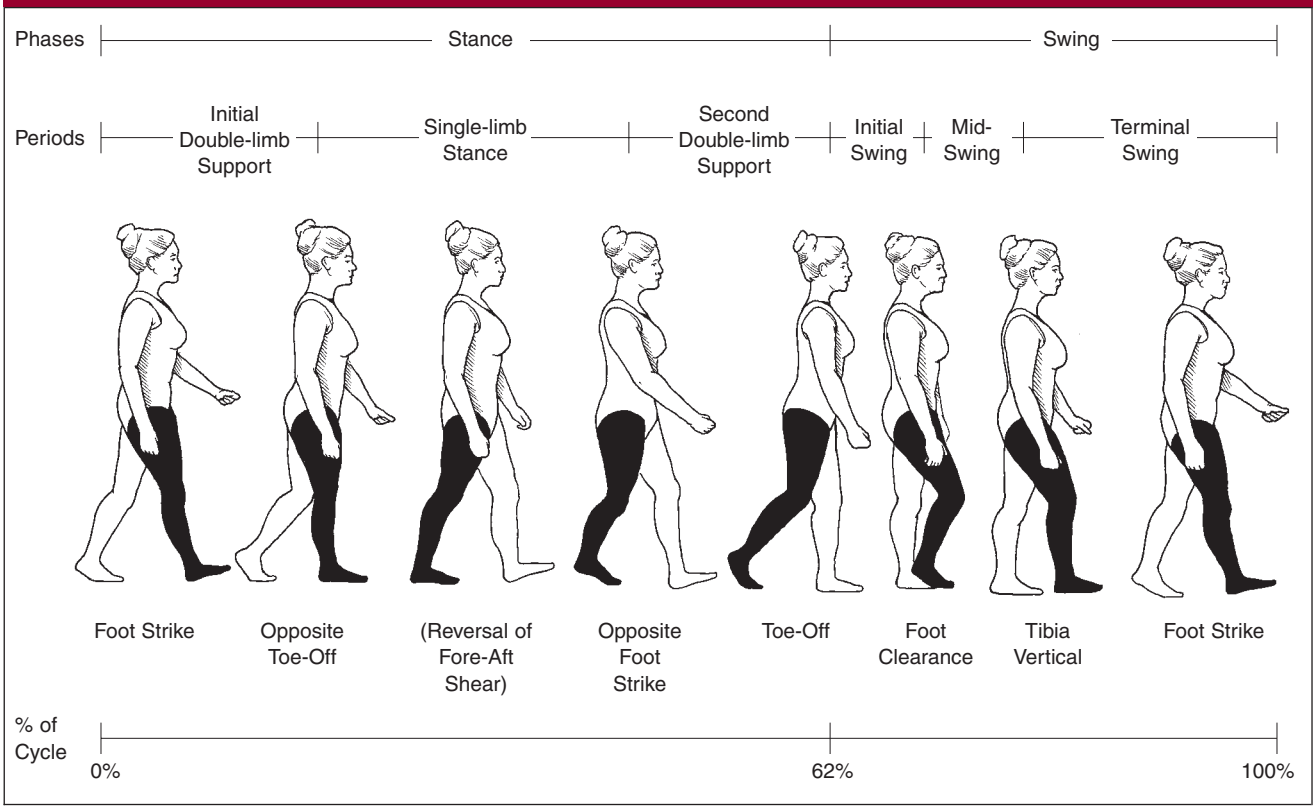
Normal gait is an unconsciously coordinated activity resulting from the interaction between the musculoskeletal and nervous systems. In animals, the center of walking coordination lies in the pattern generator of the spine. Lower order animals have the capacity for stepping when this spinal pattern generator is stimulated. In contrast, walking in higher order primates depends on supraspinal activity. Locomotion centers have been identified in the brainstem. Spontaneous walking requires the basal ganglia and thalamus, as well, but not the cerebral cortex. Clearly, however, the higher centers in the cerebral cortex are important in initiating and directing ambulation.[11,12]

In addition to generating motor activity, the central nervous system also must coordinate the maintenance of balance. Once motor activity is generated, feedback and modulation are relayed through the cerebellum. As the support shifts from one leg to the other in a dynamic equilibrium, reflexive adjustments of the legs and trunk must occur. These reflexive adjustments are based on sensory information from the visual, vestibular, and proprioceptive systems.[11,12]

Gait Cycle

To begin walking, one foot is raised and accelerated forward. The hip and knee flex and the ankle dorsiflexes as the foot clears the ground. Muscle action on the supporting contralateral leg accelerates the body's center of gravity forward. The moving foot is then placed on the ground, and weight is subsequently transferred to that leg. The heel strikes the ground first, followed by gradual transfer of weight to the sole and

Figure 1



The normal gait cycle. (Adapted with permission from Sutherland DH, Kaufman KR, Moitoza JR: Kinematics of normal human walking, in Rose J, Gamble JG [eds]: *Human Walking*, ed 2. Baltimore, MD: Williams and Wilkins, 1994, pp 23-44.)

then to the toes. The other foot is then raised and accelerated forward. The body is held erect, with the head facing forward and the arms swinging equally, smoothly, and loosely. The pelvis and shoulders remain relatively level. As the body passes over the weight-bearing leg, it displaces the center of gravity toward the weight-bearing side, causing a slight side-to-side movement with each step. In addition, the body rises and falls with each step.

Thus, a full gait cycle begins when one foot strikes the ground, and it ends when the same foot strikes the ground again. The cycle is divided into the stance phase and the swing phase. The stance phase begins with the initial contact of the heel on the ground and ends with toe-lift. The swing phase begins when the stance phase ends, and vice versa. Stance takes up about 60% of the time of the gait cycle and the swing phase, about 40%. Both feet are on the ground for two periods during each cycle. Each of these double-support periods lasts for about 10% of the cycle.

The gait cycle can be further characterized by subdividing each of the phases. The stance phase is subdivided into initial double-limb support, single-limb stance, and second double-limb support, or, more commonly, initial contact, loading response, midstance, terminal stance, and preswing. The swing phase is subdivided into initial swing, midswing, and terminal swing (Figure 1). Gait is also described in terms of walking velocity (distance per time), cadence (steps per time), step length (longitudinal distance between the two feet), step width (side-to-side distance between the two feet), and stride length (longitudinal distance covered during a complete gait cycle, representing the sum of the left and right step lengths).[12]

Normal Gait of Elderly

The globally degenerative changes of aging have deleterious effects on gait. Therefore, it may be challenging to distinguish between "normal" and pathologic gaits in the elderly. In general, the elderly use a shorter, broader-based stride. There is an approximately 10% to 20% decrease in walking velocity. This is caused by a decrease in step length rather than by a change in cadence.[13] Pelvic rotation and lower extremity joint excursions are also reduced.[8] Elderly men tend to walk with a slightly flexed posture, with slightly flexed

elbows and knees and decreased arm swing. Elderly women tend to walk with a slight waddling quality.[11]

Neurologic Gait Disorders

Frontal Gait

Frontal gait associated with dementia is common in the elderly and has a variety of causes.[14] Patients with this gait stand with their feet widely apart. The feet appear to be stuck to the floor (the so-called magnetic foot response). There is difficulty in initiating ambulation and picking the feet off the floor. Several small shuffling steps are taken for a short distance, followed by a few moderate steps. The patient then suddenly stops, and the cycle is repeated. The neurologic examination is often normal because patients usually lack muscle weakness, reflex abnormalities, or sensory changes. Because the ability to perform coordinated movements is lost in the absence of motor or sensory impairment, this pattern is considered a form of gait apraxia.

Communicating normal-pressure hydrocephalus is a treatable cause of frontal gait and dementia. The diagnostic triad is slowness of thought and action, incontinence, and unsteady gait. The gait disturbance may be the only symptom in the early stages. Brain magnetic resonance imaging (MRI) shows ventricular enlargement, an enlarged flow void about the cerebral aqueduct, and a variable degree of periventricular white matter changes. However, many elderly with or without gait impairment have enlarged ventricles. To confirm the diagnosis of normal-pressure hydrocephalus, a neurologic consultation should be obtained for a dynamic lumbar puncture. The cerebrospinal fluid pressure should be <180 mm Hg, and the patient's symptoms should improve after the removal of 40 to 50 mL of cerebrospinal fluid. The treatment is ventriculoperitoneal shunt implantation.

Binswanger's disease, also known as subcortical dementia, is another cause of frontal gait. It is characterized by multiple cerebrovascular lesions in the deep hemispheric white matter. The clinical syndrome consists of hypertension, loss of memory and cognition, dysarthria, lack of facial expression, increased tone in the lower extremities, and frontal gait. There is no specific treatment.

Spastic Hemiparetic Gait

A hemorrhagic or ischemic lesion of the corticospinal tract in the contralateral cerebrum or brainstem causes a spastic hemiparetic gait. Characteristically, the affected arm is adducted at the shoulder, flexed at the elbow, and flexed at the wrist and fingers. The upper extremity does not swing and is held up against the chest or abdomen. Because of spasticity, it is difficult to flex the hip and knee and to dorsiflex the ankle. The affected stiff spastic leg is then swept outward to avoid the foot dragging on the floor (circumduction). The upper body rocks slightly to the contralateral side during circumduction.

Hypertension and hypercholesterolemia are the most common treatable risk factors for strokes. The rate of stroke recurrence is high but can be lowered by aggressive treatment with antihypertensives, statins, and/or antiplatelet drugs.[15]

Parkinsonian Gait

A flexed posture and shuffling gait are distinctive and readily identifiable features of parkinsonian gait. Patients stand immobile with a lack of spontaneous movements. They assume a flexed posture with the spine flexed; head bent down; and elbows, hips, and knees flexed. Initiating ambulation from a standing or sitting position is difficult. When the patient begins ambulation, the trunk bends further forward, and the lower extremities remain flexed. The upper extremities do not swing. Step length is reduced to the point that the feet barely clear the floor and a distinctive shuffle results. Forward movement may lead to successively more rapid steps (ie, festination). Walking may then suddenly stop involuntarily. These patients also may freeze in an attempt to pass through a doorway or over an obstacle. The body tends to turn en bloc, as a single unit.

Parkinson's disease results from the idiopathic degeneration of dopamine-producing neurons in the substantia nigra of the midbrain. It affects approximately 1.5% of the population older than age 65 years. In addition to the poor balance and difficulty walking, tremors are characteristic. However, some elderly present only with axial rigidity and gait disorder. Drug-induced parkinsonism can be caused by use of neuroleptic drugs (eg, droperidol), particularly in nursing home patients.

Progressive supranuclear palsy, a rare, rapidly progressive degenerative disorder, also can present with a parkinsonian gait. In contrast with patients with Parkinson's disease, those with progressive supranuclear palsy have eye-movement paralysis and an extended neck posture, and they display gait disturbance early in the course of disease.

Cerebellar Ataxic Gait

Patients with lesions of the cerebellum present with an unsteady gait and tendency to fall. In cerebellar ataxic gait, the feet are placed farther apart than normal (ie, wide base) and tend to be externally rotated. This foot positioning is thought to be a compensatory strategy to improve balance. The patients stagger from side to side and often search for a form of mechanical support. There is an accompanying fore-and-aft tremor of the head and/or trunk (ie, titubation). The steps are of varying length, and the feet are placed erratically. Tandem gait exhibits the most striking abnormalities in even mild cases of cerebellar ataxia. Patients quickly lose balance and must place the foot to the side to prevent falling. Whether the eyes are open or

closed, patients with cerebellar ataxia cannot stand with their feet together. Compared with normal control subjects, there is no difference in spontaneous gait velocity and stride length. The mean range of motion (ROM) of the hip, knee, and ankle are also the same. However, stance and double-limb support phases are prolonged.[16]

The most common cause of cerebellar ataxia is alcoholism. In alcoholic cerebellar degeneration, the gait disorder may be present without other signs of cerebellar dysfunction (eg, nystagmus, dysarthria). Other causes of cerebellar dysfunction include toxins (eg, phenytoin), paraneoplastic syndromes, vitamin E deficiency, and hereditary ataxias.

Sensory Ataxic Gait

Balance during ambulation depends on afferent input from the visual, vestibular, and proprioceptive systems. Sensory ataxia results from a disruption anywhere along the afferent pathways. In patients with sensory ataxia, proprioception is lost, and patients become unaware of limb position. Strength is preserved. Patients stand and walk with their feet set widely apart, keeping their eyes on the ground. They are able to stand with eyes open but become unstable when their eyes are closed (Romberg's test). In ambulation, the legs are lifted high in the air and allowed to slap on the ground to increase sensory feedback. The gait disturbance becomes worse when other sensory modalities become compromised, such as in the dark when vision is impaired.

Sensory ataxia of tabes dorsalis is a classic example. Today, the most common cause of sensory ataxia likely is diabetes, with polyneuropathy and combined deficits in vision, proprioception, and vestibular function. Human immunodeficiency virus and neurotoxic medications (eg, cisplatin, isoniazid) are other causes of polyneuropathy. Ototoxic medications (eg, gentamicin) also can cause imbalance from bilateral vestibular loss.

Combined Neurologic/ Musculoskeletal Gait Disorders

Myelopathic Gait

Cervical spondylotic myelopathy is one of the most common identifiable causes of gait disturbance in the elderly. Degenerative osteophytes and ligamentous hypertrophy narrow the cervical spinal canal, causing mechanical compression of the spinal cord. The primary clinical manifestations are gait/balance disturbance with spasticity and hyperreflexia. Most patients have neck pain, but 15% do not.[17] Compression of the cervical spinal cord causes a lower motor neuron lesion at the level of the compression and upper motor neuron lesions below that level. Patients often have numbness and paresthesias in the upper extremities and report loss of fine motor skills, such as a change in their handwriting or the inability to handle coins. Lower extremity weakness affects the proximal musculature. There is loss of position and vibration sense because of dorsal column dysfunction. In severe cases, changes in bowel and bladder function (primarily, urinary urgency) occur.

The spastic paraparetic gait in cervical spondylotic myelopathy is described as stiff-legged, wide-based, and jerky. Movements at the hip and knee are slow and stiff, requiring considerable effort. Toe clearance in the swing phase is compromised by the reduced knee flexion and spastic plantar flexion of the ankle. Consequently, there is a tendency to circumduct or scuff the feet. Patients also may move from side to side to compensate for the slow and stiff movements of the leg. Walking speed is reduced because of a decrease in step length. Normal cadence is preserved. The time of double-limb support and the step width are increased.[18,19]

In addition to cervical spondylotic myelopathy, vitamin B_{12} deficiency is another cause of myelopathy in the elderly. Vitamin B_{12} deficiency is easy to identify and is readily treatable. Thoracic disk herniations or demyelinating disease (eg, multiple sclerosis) are other causes of myelopathy.

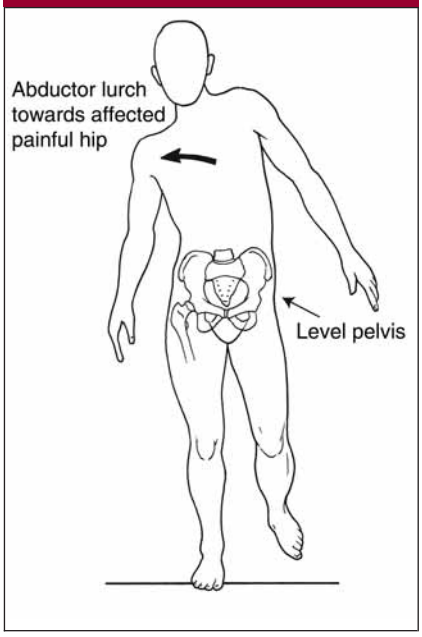
Stooped Gait of Lumbar Spinal Stenosis

Lumbar spinal stenosis is a common cause of difficulty in ambulation. Degenerative osteophytes and ligamentous hypertrophy narrow the lumbar spinal canal, causing mechanical compression of the cauda equina. Patients primarily present with a long history of back pain with progression to leg pain. A feeling of tightness, heaviness, pain, and subjective weakness in the legs (ie, neurogenic claudication) is exacerbated by standing, walking, and exercising in an erect posture. This symptom complex is rapidly relieved by sitting down or leaning forward (such as on a shopping cart).

On examination, patients tend to assume a "simian" posture, with shoulders translated anterior to the pelvis.[20] Thigh pain can be elicited following 30 seconds of lumbar extension.[21] Patients walk slowly in a stooped gait with decreased velocity, shortened stride, and the lumbar spine bent forward. The stooped gait is apparent as soon as gait is initiated, before the onset of pain. Factors related to the style of walking, such as symmetry, smoothness, and rhythm, also are abnormal.[22]

In lumbar spinal stenosis, epidural pressure rises intermittently during ambulation. The severity of this rise in pressure has been shown to depend on the posture, speed, and stride of ambulation. The stooped gait observed in spinal stenosis is thought to be an adaptive mechanism to minimize lumbar epidural pressure.[22] Proprioceptive loss and bowel/bladder dysfunction are uncommon.

Figure 2



Coxalgic gait. The gait caused by a painful hip is characterized by shifting of the upper torso toward the painful side during the single-limb stance phase on the affected hip. This coxalgic gait pattern results from an unconscious adaptive response to decrease the joint reaction force on the painful hip. (Adapted with permission from Hoppenfeld S: *Physical Examination of the Spine and Extremities*. Upper Saddle, NJ: Pearson Education, 1976, p 139.)

Steppage Gait

Steppage gait results from weakness in ankle dorsiflexion (ie, foot drop). Patients drag their feet and/or compensate by lifting the foot as high as possible, with increased flexion of the hips and knees. The gait may be unilateral or bilateral. The toe of the affected leg hits the ground before the heel or sole of the foot, and the heel tends to strike the ground with a characteristic slapping sound.

A foot drop can be caused by acquired and hereditary peripheral neuropathies, sciatic or peroneal neuropathy, or lumbar radiculopathy. Initially, the slapping-foot pattern of steppage gait may appear to be similar to that of sensory ataxia. However, the two problems are distinguishable on further physical examination. In sensory ataxia, patients may report gait worsening in the dark, and they will have preservation of ankle dorsiflexion strength and an abnormal Romberg test. In contrast, patients with steppage gait or foot drop demonstrate weakness of ankle dorsiflexion.

Musculoskeletal Gait Disorders

Antalgic Gait

Antalgic gait is characterized by a decreased time in the stance phase on the affected limb. The affected limb is placed on the ground just long enough to quickly swing the unaffected limb to stance. This gait allows the patient to diminish pain by minimizing the weight-bearing time on that limb. Antalgic gait can be caused by any painful condition of the lower extremity, such as knee osteoarthritis, ankle sprains, and stress fractures of the foot.

In knee osteoarthritis, the antalgic gait is accompanied by a decreased walking velocity and a shortened step length. Normal cadence is preserved. In addition to the decreased dynamic knee flexion ROM, patients with knee osteoarthritis also have less ROM at the hip and ankle joints.[23] Avoidance of forceful quadriceps contraction also is observed and is thought to help reduce articular loads. These subtle abnormal knee kinematics are exaggerated during paced walking.[24,25]

Coxalgic Gait

Patients with hip pain present with a characteristic coxalgic gait. This gait is characterized by shifting of the upper torso toward the painful hip during the single-limb stance phase on the affected hip (Figure 2). This shifting of the torso in ambulation is often referred to as an abductor lurch, gluteus medius lurch, or the Duchenne sign. Coxalgic gait results from an unconscious adaptive response to decrease the joint reaction force on the painful hip.

A simplified free body diagram illustrates this adaptive response (Figure 3). During single-limb stance phase on the affected hip, the hip joint is a fulcrum, with the pull of the abductors on one side and the weight of the body on the other. The joint reaction force is the sum of these two forces. To maintain a static equilibrium during single-limb stance, the sum of the moments about the hip fulcrum must equal zero. Therefore, the distance from the body's center of gravity to the hip joint multiplied by body weight equals the distance from abductors to the hip joint multiplied by the force of the abductors. When the torso is shifted toward the affected hip, the moment arm of the body of gravity is decreased. Less abductor force is then required to maintain a static equilibrium. The decrease in abductor force leads to a decrease in the joint reaction force.

A common cause of coxalgic gait in the elderly is hip osteoarthritis. Aside from the characteristic coxalgic gait, most of the other gait changes associated with hip osteoarthritis are subtle and not easily detectable by visual inspection alone. There may be an antalgic component to the coxalgic gait (ie, a minimization of weight bearing on the affected limb). There is also a decrease in dynamic ROM of the hip during ambulation and an increase in anterior pelvic tilt and lumbar lordosis. This increased extension of the spine and pelvis is believed to compensate for the loss of hip extension.[26]

The appearance of a coxalgic gait also can be caused by painless, mild hip abductor insufficiency. In mild abductor insufficiency, the compensatory trunk lurch decreases the force required by the abductors to maintain a level pelvis during single-limb stance. Abductor weakness that can follow an anterolateral approach to total hip replacement is

associated with a postoperative painless "coxalgic" gait.[27] L5 radiculopathy also can cause abductor weakness with a subsequent coxalgic gait pattern.

Trendelenburg Gait

A Trendelenburg gait is very similar in appearance to a coxalgic gait, with one main distinguishing feature—the tilt of the pelvis. With Trendelenburg gait, the contralateral hemipelvis drops during the single-limb stance phase on the affected side (Figure 4). As the contralateral hemipelvis drops, greater knee flexion is required to clear the swing-phase foot off the ground. Similar to the coxalgic gait pattern, the torso also lurches toward the affected side. However, in the coxalgic gait pattern, the pelvis remains level.

A Trendelenburg gait is caused by severe abductor insufficiency and is most commonly seen in patients with polio. In the painless "coxalgic" gait pattern caused by mild abductor insufficiency, the torso lurches toward the affected hip during single-limb stance. This compensation brings the body's center of gravity closer to the hip joint, decreasing the required abductor force necessary to maintain a level pelvis in static equilibrium. Although the abductors are weak, the compensatory lurch permits the weak abductors to maintain a level pelvis. In severe abductor insufficiency, however, even with the compensatory lurch, the abductors are unable to generate sufficient force to keep the pelvis level. Therefore, during single-limb stance, the hemipelvis contralateral to the severely weak abductor drops, resulting in the characteristic Trendelenburg gait.[28,29]

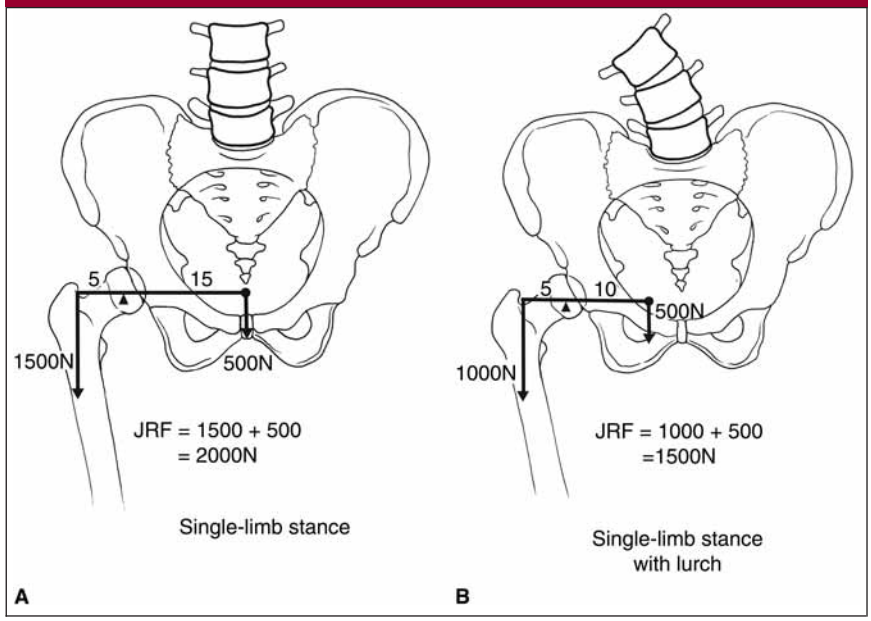
For example, in Figure 3, B, with the torso lurched toward the affected hip, 1,000 N of abductor force is necessary to maintain a level pelvis in single-limb stance. If the abductors are only mildly weak and still able to generate 1,000 N of force, the pelvis will remain level during the single-limb stance of ambulation, and a coxalgic gait will result. However, if the abductors are severely weak and unable to generate 1,000 N of force, the contralateral hemipelvis will drop during the single-limb stance of ambulation, and a Trendelenburg gait will result.

The Trendelenburg gait also should be distinguished from the Trendelenburg test. The presence of a Trendelenburg gait indicates dynamic abductor insufficiency. In contrast, the Trendelenburg test is a static test, also used to demonstrate abductor insufficiency. In the Trendelenburg test, the patient is first asked to stand in double-limb stance. The dimples of the posterior superior iliac spine are observed as a landmark for the pelvis and should be level. The patient is then asked to stand on one leg. As one limb leaves the ground, the abductors on the contralateral stance phase limb should contract and the pelvis should remain level (a negative Trendelenburg test or sign). If, however, the hemipelvis contralateral to the stance limb drops, the abductors on the stance limb are severely insufficient (a positive Trendelenburg test).[30] A Trendelenburg gait pattern and a positive Trendelenburg test usually coexist because they indicate the same pathology.

Knee Hyperextension Gait

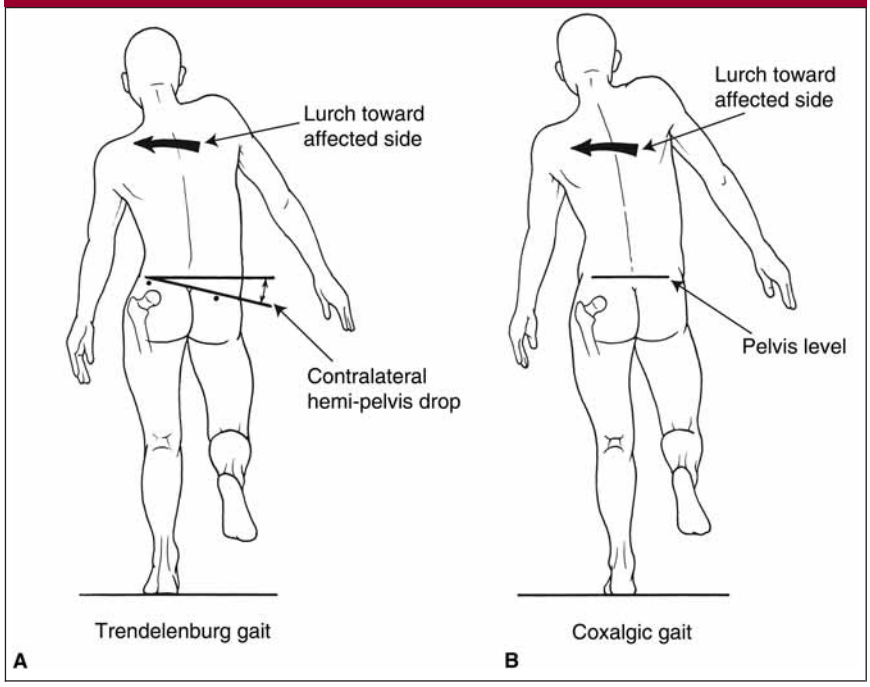
Knee hyperextension gait can result from an ankle equinus contracture or from quadriceps deficiency. With an ankle equinus contracture,

Figure 3



Free body diagram of the hip in single-limb stance. **A,** Assume that the distance from the hip to the abductors is 5 cm and that the distance from the hip to the body center of gravity is 15 cm. If the weight of the body is 500 N, the abductors must exert 1,500 N of force to maintain the static equilibrium. (The sum of the moments about the hip fulcrum must equal zero, ie, 1,500 N × 5 cm = 500 N × 15 cm.) In this case, the joint reaction force (JRF) is the sum of the abductor force and the body weight, totaling 2,000 N. **B,** When the upper body is lurched toward the affected hip, the body center of gravity is effectively shifted in the same direction. Thus, if the distance from the hip to the body center of gravity is decreased to 10 cm, the abductors need exert only 1,000 N to maintain static equilibrium: 1,000 N × 5 cm = 500 N × 10 cm. Therefore, the lurch reduces the JRF to 1,500 N.

Figure 4



Trendelenburg gait. The Trendelenburg gait is very similar in appearance to a coxalgic gait, with one main distinguishing feature–the tilt of the pelvis. In the Trendelenburg gait **(A)**, the contralateral hemipelvis drops during the single-limb stance phase on the affected side because of severe abductor insufficiency. In the coxalgic gait **(B)**, the pelvis remains level. (Adapted with permission from Hoppenfeld S: *Physical Examination of the Spine and Extremities*. Upper Saddle, NJ: Pearson Education, 1976, p 164.)

increased ipsilateral knee and hip flexion are required to help the foot clear the floor. Therefore, during the initial contact portion of the stance phase, the combination of the plantarflexed ankle and the flexed knee directs the forefoot to the floor instead of the heel. Therefore, the heel rocker action of the stance phase is lost. In addition, during midstance and terminal stance, the contact of the plantarflexed foot on the ground leads to an extension moment on the knee, resulting in knee hyperextension.

The quadriceps-deficient knee hyperextension gait results when the quadriceps fails to perform its usual stabilizing function during the loading response of gait. The loading response occurs immediately after initial contact in the stance phase. During the normal loading response, the ground reaction force (GRF) is posterior to the ankle, anterior to the hip, and posterior to the knee. The posterior force gives the knee joint a flexion moment. In normal gait, the quadriceps counteracts this flexion moment to preserve joint stability. However, when the quadriceps is deficient, other compensatory mechanisms must be used to achieve joint stability. These compensatory mechanisms include increasing hip extension and ankle plantar-flexion forces to provide an extension moment to the knee. The trunk may lean forward to move the GRF anteriorly, closer to the center of the knee, and thus reduce the flexion moment. Also, initial contact may occur with a flatfoot instead of with the heel. Using the entire foot for initial contact moves the point of application of the GRF anteriorly. This places the GRF closer to the knee joint, and the knee flexion moment is additionally reduced. These compensatory mechanisms all contribute to hyperextension of the knee observed during stance in a patient with a deficient quadriceps mechanism.[31]

Inadequate Knee Extension Gait (Flexion Contracture)

Knee flexion contractures lead to inadequate knee extension during gait. Inadequate extension primarily affects the stance phase of the gait cycle. The flexed knee during midstance interferes with the normal mechanisms used to realign the body vector over the supporting foot. Without compensation, the flexed knee would place the body behind the foot. Compensatory increased ankle dorsiflexion and/or premature heel rise allows the body to be aligned directly over the foot during midstance. Also, stance-phase weight bearing on an excessively flexed knee requires greater extension forces from the quadriceps to preserve joint stability. The increased functional demands placed on the quadriceps explain early ambulation fatigue in patients with knee flexion contractures.[31]

Inadequate Knee Flexion Gait (Extension Contracture)

Inadequate knee flexion primarily affects the swing phase of the gait cycle. In patients with a severe extension contracture or a knee fused in excessive extension, the swing-phase limb cannot be shortened sufficiently to clear the foot. In compensation, the ipsilateral hip may be abducted to circumduct the limb. Circumduction may be accompanied by elevation of the ipsilateral hemipelvis (ie, hip hiking) and leaning of the trunk to the contralateral side. In addition, the contralateral stance phase ankle may be plantarflexed to elevate the entire body and clear the swing-phase foot (ie, vaulting).[31]

Inadequate Hip Extension Gait

Inadequate hip extension caused by a flexion contracture reduces stride length and the duration of single-limb support during gait. Inadequate hip extension keeps the thigh forward and prevents the body from advancing over the supporting foot. Exaggerated lumbar lordosis is used to compensate for the lack of hip extension. The pelvis is tilted anteriorly to accommodate the flexed hips to extend the femur while the trunk is tilted posteriorly to balance the body directly over the feet. In patients with limited lumbar mobility, a forward-leaning trunk posture may be seen. This posture requires a form of external support such as a cane or a walker to support ambulation. Alternatively, hip flexion contractures can be compensated for during gait by knee flexion using a crouched gait. A crouched gait allows the femur to roll forward, but this adaptive mechanism can result in concomitant knee flexion contractures and early ambulation fatigue resulting from the increased functional demands on the quadriceps.[31]

Approach to the Elderly Patient With a Gait Abnormality

History

The presenting history can provide many diagnostic clues. An insidious nature of onset suggests a degenerative disorder, whereas sudden stepwise progressions suggest cerebrovascular disease. Accompanying reports of pain in the neck, low back, or lower extremities suggest degenerative conditions in, respectively, the cervical spine, lumbar spine, and lower extremities. Gait abnormality with urinary urgency and incontinence suggests multiple cerebral infarcts, Binswanger's disease, normal-pressure hydrocephalus, or severe cervical spondylotic myelopathy.[1] A history of alcohol and medication intake also may be helpful in establishing a diagnosis. Benzodiazepines, phenytoin, and neuroleptic drugs are known to affect, respectively, the vestibular system, cerebellum, and basal ganglia. Toxic/metabolic causes of gait abnormality are important to identify because they are generally reversible. Symptoms of depression also should be elicited because depressed patients may exhibit slowness from psychomotor retardation, manifesting as a lack of purpose in stride and a lifting action of the legs during gait.[32]

In the setting of a gait disorder and recent fall, the nature of the fall should be carefully detailed. Crumpling at the knees suggests syncope or loss of strength from a transient ischemic attack. Falling stiffly backward is common in Parkinson's disease. The location and activity of the patient during the fall may point to environmental or patient-related causes. If the fall occurred shortly after the patient had just arisen from sitting or lying down, orthostatic hypotension may have resulted. Recently adjusted cardiac medications, especially antihypertensive diuretics or vasodilators, should be reviewed. Falls that occur shortly after meals may reveal postprandial hypotension as a cause. Recurrent tripping may be caused by a foot drop or increased tone from spasticity.[33]

Physical Examination

Although the modern gait analysis laboratory can provide a multitude of objective measurements, the most revealing features of a patient's gait often can be garnered by careful systematic observation alone. Some gaits readily suggest a specific disorder, as in Parkinson's disease, cerebellar ataxia, or hip osteoarthritis.

The gait examination begins by observing the undressed patient as he or she rises from the chair to stand. The ease of the transition and the standing posture should be noted. A flexed posture can be a sign of Parkinson's disease or lumbar spinal stenosis. A rigid spinal posture may indicate a bony spinal deformity. The width of the feet during stance can be a sign of imbalance, seen in cerebellar ataxia, sensory ataxia, frontal lobe disease, and cervical spondylotic myelopathy. Also, standing balance in single-limb stance and in tandem stance should be observed. Next, with the patient comfortably standing, ask the patient to close his or her eyes (Romberg's test). The clinician should remain close to the patient in case he or she begins to fall. Patients with sensory ataxia caused by proprioceptive loss will have dramatic stance instability with eye closure. In contrast, standing stability is unaffected by eye closure in cerebellar ataxia.

The patient is then asked to walk at a normal pace. Difficulty in initiating the first step can be indicative of Parkinson's or frontal lobe disease. Once the walking is under way, the patient is observed from the side, front, and back. The overall pattern of body movement should first be noted. Next, the patient should be observed from the feet upward, with the observer relating the action of the two limbs, the torso, and then the upper extremities. The observer should now be able to differentiate a focal problem in an extremity to a generalized movement disorder. The velocity, step length, step width, rhythm, and cadence all should be noted. The fluidity and symmetry of arm swing should be noted, as should the use of any mechanical assistive devices. Asking the patient to walk at a faster pace may elicit more subtle characteristics of abnormal gait. The patient then is asked to walk in a straight line heel to toe (tandem gait) and to perform the heel-to-shin test. In the heel-to-shin test, the supine patient is asked to touch the heel of one foot to the contralateral knee and drag it in a straight line down the shin and up again. These two tests often bring out subtle cerebellar and sensory ataxias.

The remaining routine neurologic and musculoskeletal examina-

tions should focus on evaluating muscle bulk and strength, sensation, proprioception, deep tendon reflexes, spinal deformity, limb-length inequalities, and joint motion. Increased muscle tone and hyperreflexia indicate an upper motor neuron lesion. Muscle weakness and areflexia indicate a lower motor neuron lesion. Subtle ankle dorsiflexion or plantar-flexion weakness can be elicited by observing the patient walking on the heels and toes, respectively. Finally, brief screening examinations of vision and hearing also should be performed.[34]

Imaging

Clinical suspicion of a neurologic cause of gait disturbance warrants a referral to a neurologist for evaluation and possible brain imaging. MRI and/or computed tomography of the brain are sensitive for infarcts, demyelinating disease, mass lesions, hydrocephalus, and cerebellar atrophy. A patient with a recent fall and disturbance in alertness should be imaged emergently to rule out a subdural hematoma.

Patients suspected to have cervical spondylotic myelopathy or lumbar spinal stenosis should be imaged with plain radiography and MRI. In patients with coxalgic or Trendelenburg gait, antalgic gait, knee hyperextension gait, or other gaits related to limited joint mobility, plain radiographs of the involved regions should be obtained.

Treatment

Even when no identifiable cause of the disorder is found, many patients with a gait disorder and a history of falls can benefit from evaluation for possible interventions to prevent falls. A home visit can be arranged to eliminate environmental hazards, such as inadequate lighting, tripping obstacles, and slippery surfaces. Consideration may be given to the installation of grab bars and stairwell modifications. A referral to a physical therapist may be made for gait training, use of assistive devices, high-intensity resistance strength training, and sensory balance training. The practice of tai chi also has been shown to be beneficial in fall prevention.[35,36]

Medications that adversely affect gait may be eliminated or the dosage changed in consultation with the primary care physician. Benzodiazepines affect postural control and increase the risk of falls and hip fractures. Serotonin-reuptake inhibitors, tricyclic antidepressants, neuroleptic agents, anticonvulsants, and class IA antiarrhythmics (eg, procainamide) are other medications known to increase the risk of falling.[35]

Neurologic causes of gait disturbances are treated under the care of the neurologist. Levodopa is the mainstay of therapy for Parkinson's disease, but gait and balance are not always improved with the medication.[37] Because vitamin B_{12} supplementation is inexpensive and has very limited potential for harm, it may be initiated in patients suspected to have B_{12}-deficient myelopathic or ataxic gait.[38] Ventriculoperitoneal shunting is effective in improving gait in 85% of patients with normal-pressure hydrocephalus.[39]

In cervical spondylotic myelopathy, multiple studies using objective gait analysis have demonstrated improved walking velocity, step length, balance, and an overall normalization of ambulation after surgical decompression.[19,40-42] Surgical decompression for lumbar spinal stenosis has been demonstrated to improve speed, stride, cadence, and other factors related to the style of walking.[22] In the setting of radiculopathy from lumbar spinal stenosis or herniated nucleus pulposus, surgical decompression improves or eliminates foot drop in most patients.[43] For osteoarthritis of the hip and knee, total joint arthroplasty can improve gait to nearly normal.[44,45]

Summary

Evaluation of an elderly patient with a gait disturbance is challenging. Failing gaits resulting from most causes often appear to be similar because adaptive changes mask the underlying unique characteristics. Also, it is often difficult to distinguish between the normal, slow, wide-based gait of the elderly and many pathologic gaits. Despite this, treatable causes of gait disturbance can be found in a substantial proportion of patients. Treatable neurologic causes include normal-pressure hydrocephalus, vitamin B_{12} deficiency, Parkinson's disease, alcoholism, and medication toxicity. Treatable musculoskeletal causes include cervical spondylotic myelopathy, lumbar spinal stenosis, joint contractures, and painful disorders of the lower extremity. Orthopaedic surgeons should be a key component of a multidisciplinary effort to identify and treat these disorders and thereby contribute to improving the quality of life of elderly patients.

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