REVIEW

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Gait disorders: Search for multiple causes

ABSTRACT

Gait disorders predict functional decline in older adults. They are often the result of multiple causes, so a full assessment should consider different sensorimotor levels and should include a focused physical examination and evaluation of functional performance. Exercise and medical and surgical interventions are effective and can reduce the degree of gait disorder, but usually not without some residual impairment. Orthoses and mobility aids are also important interventions to consider.

KEY POINTS

In assessing gait disorders, it is helpful to categorize the problem according to the level of the sensorimotor deficit.

Dementia and depression contribute to gait disorder but may not be the sole causes.

Acute gait disorder may be the presenting feature of acute systemic decompensation in an older adult, and it warrants evaluation for myocardial infarction or sepsis.

A formal neurologic assessment is critical and should include strength and tone, sensation (including proprioception), coordination (including cerebellar function), standing, and gait. Vision screening, at least for acuity, is essential.

The Timed Up and Go (TUG) test is a simple tool for assessing stability. A fall or any difficulty or unsteadiness during the TUG test requires a more extensive evaluation of gait and fall risk factors.

GAIT DISORDERS IN ELDERLY patients often lead to falls and disability. They are a strong predictor of functional decline.

More often than not, a gait disorder represents the combined effects of more than one coexisting condition, so the evaluation should take comorbidities into consideration and should include assessment of different levels of sensorimotor deficits.

In this article, we review the prevalence, impact, and causes of gait disorders in the elderly. We also outline appropriate clinical assessments and interventions known to reduce the severity of gait disorders.

INCIDENCE AND PREVALENCE OF GAIT DISORDERS

At least 20% of noninstitutionalized older adults admit having trouble walking or require the assistance of another person or special equipment to walk.[1] Limitations in walking also increase with age.

In some samples of noninstitutionalized adults age 85 and older, the prevalence of limitation in walking can be over 54%.[1] While age-related changes such as gait speed are most apparent after age 75 or 80, most gait disorders appear in connection with underlying diseases, particularly as disease severity increases. For example, age over 85, three or more chronic conditions, and the occurrence of stroke, hip fracture, or cancer predict "catastrophic" loss of walking ability.[2]

What is normal, and what is not?

Determining that a gait is "disordered" can be difficult, as there are no clearly accepted standards as to what is a "normal" gait in older

adults. Some believe a slowed gait is a disordered gait, and others believe that any aesthetic abnormality—eg, deviation in smoothness, symmetry, and synchrony of movement patterns—constitutes a gait disorder. However, a slowed or aesthetically abnormal gait may in fact provide the older adult with a safe gait pattern that helps maintain independence.

Gait disorders tend to be multifactorial, progressive

In older patients, attributing a gait disorder to a single disease is particularly difficult—and is often not advisable—because many different conditions can result in similar gait abnormalities.[3]

Recent longitudinal studies suggest that certain gait-related mobility disorders progress with age and that this progression is associated with disease and death. When measured by the Unified Parkinson Disease Rating Scale (UPDRS), which includes abnormalities in rising from a chair and turning, gait and postural disorders increased in most (79%) of a nondemented sample of Catholic clergy without clinical Parkinson disease (mean age 75) followed for up to 7 years in a prospective cohort study.[4] This increase was more common in older subjects and was associated with a higher death rate.

These observations raise several questions. The UPDRS may be more appropriate for patients known to have Parkinson disease, but an increased UPDRS score may represent increased parkinsonian signs with age in patients without a diagnosis of parkinsonism, as well as an increase in associated disease and inactivity. It is also unclear whether subjects in this cohort developed other overt neurologic disease, dementia, or both. For example, declining gait speed is one of the factors that can independently predict cognitive decline prospectively in healthy older adults.[5]

Cerebrovascular disease, both subclinical and clinically evident, is increasingly recognized as a major contributor to gait disorders (see discussion of assessment below). Nondemented patients with clinically abnormal gait (particularly unsteady, frontal, or hemiparetic gait) who are followed for approximately 7 years are at higher risk of developing non-Alzheimer dementia, particularly vascular dementia.[6] Of note, at baseline, those with abnormal gait may not have met criteria for dementia but already had abnormalities in neuropsychological function, such as visual-perceptual processing and language skills.

Gait disorders with no apparent cause ("idiopathic" or "senile" gait disorder) are associated with a higher death rate, primarily from cardiovascular causes; these cardiovascular causes are likely linked to concomitant and possibly undetected cerebrovascular disease.[7]

CONDITIONS THAT CONTRIBUTE TO GAIT DISORDERS

Disordered gait, defined as a gait that is slowed, aesthetically abnormal, or both, is not necessarily an inevitable consequence of aging but rather a reflection of the increased prevalence and severity of age-associated diseases.[8] These underlying diseases, neurologic and nonneurologic, contribute to disordered gait. Elderly patients usually have more than one condition contributing to their gait disorder.

When asked what makes walking difficult, patients most often cite pain, stiffness, dizziness, numbness, weakness, and sensations of abnormal movement.[9] Conditions seen in the primary care setting that can contribute to gait disorders include degenerative joint disease, acquired musculoskeletal deformities, intermittent claudication, impairments following orthopedic surgery and stroke, and postural hypotension.[9]

Declining gait speed predicts cognitive decline in healthy older adults

In a group of community-dwelling adults over age 88, joint pain was by far the most common contributor, followed by stroke and visual loss.[8]

The diagnoses found in a neurological referral population were primarily neurologically oriented[10,11] and included frontal gait disorders (usually related to normal-pressure hydrocephalus and cerebrovascular processes), sensory disorders (also involving vestibular and visual function), myelopathy, previously undiagnosed Parkinson disease or parkinsonian syndromes, and cerebellar disease.

Conditions that cause severe gait impairment, such as hemiplegia and severe hip or knee disease, are often not mentioned in these neurologic referral populations. Thus, many gait disorders, particularly those that are clas-

sic and discrete (eg, related to stroke and osteoarthritis) and those that are mild or related to irreversible disease (such as vascular dementia), are presumably diagnosed in a primary care setting and treated without referral to a neurologist. Dementia and fear of falling also contribute to gait disorders.

Less common contributors to gait disorders include metabolic disorders related to renal or hepatic disease, tumors of the central nervous system, subdural hematoma, depression, and psychotropic medications. Case reports also document reversible gait disorders due to clinically overt hypothyroidism or hyperthyroidism and deficiency of vitamin B_{12} and folate.[3]

DISEASE-RELATED FACTORS THAT AFFECT GAIT

Factors that slow gait speed are also considered contributors to gait disorders. These factors are often disease-associated—eg, related to cardiopulmonary or musculoskeletal disease—and include reductions in leg strength, vision, aerobic function, standing balance, and physical activity, as well as joint impairment, previous falls, and fear of falling.[12–18] In combination, these factors may have an effect greater than the sum of the single impairments, such as when leg weakness is superimposed on impaired balance.[17] Furthermore, the effect of reduced strength and aerobic capacity on gait speed may be nonlinear: ie, for very impaired patients, small improvements in strength or aerobic capacity yield relatively larger gains in gait speed, whereas small improvements may yield little gait speed change in a healthy older patient.[15,19]

Factors that slow gait speed may also contribute to gait disorders

While older adults may maintain a relatively normal gait pattern well into their 80s, some slowing occurs, and decreased stride length becomes a common feature described in gait disorders in the elderly.[3]

Some authors have proposed the emergence of an age-related gait disorder without accompanying clinical abnormalities, ie, "essential senile gait disorder."[20] This gait pattern is described as broad-based, with small steps, diminished arm swing, stooped posture, flexion of the hips and knees, uncertainty and stiffness in turning, occasional difficulty initiating steps, and a tendency toward falling. These and other nonspecific findings, such as the inability to perform tandem gait, are similar to gait patterns found in a number of other diseases, and yet the clinical abnormalities are insufficient to make a specific diagnosis. This "disorder" may be a precursor to a still asymptomatic disease (eg, related to subtle extrapyramidal signs) and is likely to appear with concurrent, progressive cognitive impairment (eg, Alzheimer disease or vascular dementia).[21]

While the concept of senile gait disorder reflects the multifactorial nature of gait disorder, we feel it is generally not useful in labeling gait disorders in older adults.

APPROACH TO ASSESSMENT

A potentially useful approach to assessing contributors to gait disorder (TABLE 1)[22] categorizes these deficits according to the level of sensorimotor deficit, ie, low, middle, and high.

Low deficits: Peripheral sensory, peripheral motor

Disorders due to deficits at the low sensorimotor level (ie, generally distal to the central nervous system) can be divided into peripheral sensory and peripheral motor dysfunction.

In peripheral sensory impairment, unsteady and tentative gait is commonly caused by vestibular disorders, peripheral neuropathy, posterior column (proprioceptive) deficits, or visual ataxia.

Peripheral motor impairment results from arthritic, myopathic, and neuropathic conditions that result in deformity of the extremities, painful weight-bearing, and focal weakness. The resulting gait disorders are primarily compensatory. Examples include Trendelenburg gait (hip abductor weakness causing weight shift over the weak hip); antalgic gait (avoidance of excessive weight-bearing and shortening of stance on one side due to pain); and "steppage" gait (excessive hip flexion to facilitate foot clearance of the ground, seen in patients with foot drop due to ankle dorsiflexor weakness).

If the gait disorder is limited to this low sensorimotor level and the central nervous system is intact, the patient adapts well to the gait disorder and can compensate with an

TABLE 1

Gait disorders vary according to the level of sensorimotor deficit

LEVEL	DEFICIT/CONDITION	GAIT CHARACTERISTICS
Low	Peripheral sensory ataxia: posterior column, peripheral nerves, vestibular and visual ataxia	Unsteady, uncoordinated (especially without visual input), tentative, "drunken"
	Peripheral motor deficit due to arthritis (antalgic gait, joint deformity)	Avoids weight-bearing on affected side; shorter stance phase Painful hip may produce Trendelenburg gait (trunk shift over affected side) Painful knee is flexed Painful spine produces short, slow steps and decreased lumbar lordosis Non-antalgic features include contractures, deformity-limited motion, buckling with weight-bearing Kyphosis and ankylosing spondylosis produce stooped posture Unequal leg length can produce abnormal trunk and pelvic motion, including Trendelenburg gait
	Peripheral motor deficit due to myopathic and neuropathic conditions (weakness)	Pelvic girdle weakness produces exaggerated lumbar lordosis and lateral trunk flexion (Trendelenburg and "waddling" gait) Proximal motor neuropathy produces waddling and foot slap Distal motor neuropathy produces distal weakness, especially ankle dorsiflexion and "foot drop," which may lead to exaggerated hip flexion, knee extension, foot lifting (steppage gait), and foot slap
Middle	Spasticity from hemiplegia, hemiparesis	Leg swings outward and in semi-circle from hip (circumduction); knee may hyperextend (genu recurvatum); ankle may show excessive plantar flexion and inversion (equinovarus); with less paresis, some may only lose arm swing and only drag or scrape the foot
	Spasticity from paraplegia, paresis	Circumduction of both legs; steps are short, shuffling, and scraping; when severe, hip adducts so that knees cross in front of each other (scissoring)
	Parkinsonism	Small and shuffling steps, hesitation, acceleration (festination), falling forward (propulsion), falling backward (retropulsion), moving the whole body while turning (turning en bloc), no arm swinging
	Cerebellar ataxia	Wide-based gait with increased trunk sway, irregular stepping, staggering (especially on turns)
High	Cautious gait	Fear of falling with appropriate postural responses, normal to widened gait base, shortened stride, slower, turning en bloc
	Frontal-related or white-matter lesions: cerebrovascular lesions, normal-pressure hydrocephalus	Frontal gait disorder: difficulty initiating gait; short, shuffling gait, like parkinsonian, but with wider base, upright posture, arm swing, leg apraxia, and "freezing" when turning or when attention is diverted May also have cognitive, pyramidal, urinary disturbances

assistive device or learn to negotiate the environment safely.

Middle deficits: Spasticity, parkinsonism, ataxia

Deficits at the middle level result from spasticity (due to myelopathy, vitamin B_{12} deficiency, stroke), parkinsonism (idiopathic or drug-induced), and cerebellar ataxia (eg, alcohol-induced). The execution of centrally selected postural and locomotor responses is faulty, and the sensory and motor modulation of gait is disrupted. Initiation of gait may be normal, but stepping patterns are abnormal. Classic

gait patterns appear when spasticity is sufficient to cause leg circumduction and fixed deformities (such as equinovarus), when parkinsonism produces shuffling steps and reduced arm swing, and when cerebellar ataxia increases trunk sway sufficiently to require a broad base of gait support.

High deficits: Slowed cognition, fear of falling

At the high level, gait characteristics become more nonspecific, and cognitive dysfunction and slowed cognitive processing become more prominent. Behavioral aspects such as fear of falling play a role, particularly in cautious gait. Dementia and depression contribute to but may not be the sole cause of the gait disorder.

Frontal-related gait disorders often have a cerebrovascular component and are not merely the result of frontal masses and normal-pressure hydrocephalus. The severity of frontal disorders ranges from difficulty initiating gait to frontal disequilibrium, in which patients cannot stand without support. Cerebrovascular insults to the cortex and the basal ganglia and their interconnections may relate to difficulty initiating gait and apraxia.[23,24] Cognitive, pyramidal, and urinary disturbances may also accompany the gait disorder.

Gait disorders in this category have been given a number of overlapping descriptions, including gait apraxia, marche à petits pas, and arteriosclerotic (vascular) parkinsonism. As the severity of the dementia increases, particularly in patients with Alzheimer disease, frontal-related symptoms also worsen.[25]

Deficits at more than one level

In elderly patients with multiple conditions, a gait disorder likely represents deficits at more than one sensorimotor level. One example is a patient with long-standing diabetes with peripheral neuropathy and a recent stroke who is now afraid of falling. Also, certain disorders may actually involve multiple sensorimotor levels: eg, Parkinson disease affects high (cortical) and middle (subcortical) structures. Drugs such as sedatives, tranquilizers, and anticonvulsants may affect more than one level of sensorimotor function: phenothiazines, for example, can cause high-level effects (sedation and associated decreased attention) and mid-level (extrapyramidal) effects.

HISTORY AND PHYSICAL EXAMINATION

A careful medical history and a review of systems help uncover the factors contributing to the gait disorder. A brief systemic evaluation for evidence of acute cardiopulmonary disorders (such as a myocardial infarction) or other acute illness (such as sepsis) is warranted because an acute gait disorder may be the presenting feature of acute systemic decompensation in an older adult. Evaluation for subacute metabolic disease (such as thyroid disorders) is also warranted.

The physical examination should also attempt to identify motion-related factors, such as by provoking both vestibular and orthostatic responses. In the Dix-Hallpike maneuver, while the patient is seated on an examination table, the examiner holds the patient's head, turns the head to one side, and lowers the head usually to 30° below the table level. The patient then sits up, and the maneuver is repeated to the other side. Blood pressure should be measured with the patient both supine and standing to rule out orthostatic hypotension. Patients with dizziness and a sensation of relative motion may be considered for additional vestibular screening utilizing motion of the head to provoke either changes in eye motion (eg, head thrust or head-shaking maneuver)[26] or disruptions in gait (as in the Dynamic Gait Index).[27]

Gait disorders are often due to deficits at more than one sensorimotor level

Vision screening, at least for acuity, is essential.

The neck, spine, extremities, and feet should be evaluated for pain, deformities, and limitations in range of motion, particularly subtle hip and knee contractures. Leg-length discrepancies, such as may occur after placement of a hip prosthesis,[28] can be measured simply as the distance from the anterior superior iliac spine to the medial malleolus, with the patient supine.

A formal neurologic assessment is critical and should include strength and tone, sensation (including proprioception), coordination (including cerebellar function), standing, and gait. The Romberg test screens for simple postural control and for proprioceptive and vestibular system dysfunction when the eyes

are closed. Some investigators have proposed that being unable to stand on one leg for 5 seconds is a risk factor for injurious falls,[29] although even relatively healthy adults age 70 and older may have difficulty standing on one leg.[30] Given the importance of cognition as a risk factor, screening for mental status is also indicated.

LABORATORY TESTING AND IMAGING

Depending on the findings of the history and physical examination, further evaluation with laboratory testing and diagnostic imaging may be warranted. A complete blood cell count, blood chemistry panel, and other metabolic studies may be useful when a systemic disease is suspected of contributing to the gait disorder.

Head or spinal imaging with plain radiography, computed tomography, or magnetic resonance imaging (MRI) is of unclear use, unless the history or physical examination raises the suspicion of a neurologic abnormality either preceding the gait disorder or of recent onset related to the gait disorder.

In the Timed Up and Go test, > 14 seconds indicates a risk of falling

In some cases, MRI may be helpful. Changes in the cerebral white matter that are often considered to be vascular (leukoaraiosis) have been increasingly associated with nonspecific gait disorders. Periventricular high-signal measurements on MRI and increased ventricular volume, even in apparently healthy older adults,[31] are associated with slowing of gait. White matter hyperintensities on MRI correlate with longitudinal changes in balance and gait,[32] and the periventricular frontal and occipitoparietal regions appear to be most affected.[33] Age-specific guidelines, sensitivity, specificity, and cost-effectiveness of these workups remain to be determined.

PERFORMANCE-BASED ASSESSMENT

Formal kinematic and kinetic analyses have not been widely used in the clinical assessment of balance and gait disorders in the elderly. Yet diminutions in the speed and distance one can walk comfortably are powerful predictors of a number of important outcomes, such as disability, institutionalization, and death.

People tend to walk faster if they are taller, healthier, more active, and less functionally disabled.[3] Recently, it has been suggested that slow (impaired) walking can be defined as slower than 0.6 m/sec, whereas fast (unimpaired) walking is faster than 1.0 m/sec.[34]

A number of timed and semiquantitative balance and gait scales have been proposed as means to detect and quantify abnormalities and direct interventions. For example, higher abnormal gait and balance scale scores may increase the risk of falling.[35]

Perhaps the simplest tool in the clinical setting is the Timed Up and Go (TUG) test,[36] a timed sequence of rising from a chair, walking 3 meters, turning, and returning to the chair. A recent expert panel recommended that patients who report a single fall or any difficulty or unsteadiness during the TUG test require a more extensive evaluation of fall risk factors, many of which overlap with gait disorder risk factors.[37] One study suggests a TUG time of 14 seconds or more as an indicator for fall risk.[38]

Other investigators have found limitations of the TUG test in patients who have cognitive impairment and who have difficulty completing the test due to immobility, safety concerns, or refusal.[39] A more useful clinical approach may be to assess the degree of human assistance required (either manual or verbal cuing) for a patient to walk on different types of surfaces, as well as the distance the patient can walk with or without an assistive device.[40]

INTERVENTIONS TO IMPROVE GAIT DISORDERS

Comorbidity, disease severity, and overall health status tend to strongly influence treatment outcome. In addition, even if a contributing condition is found, many conditions that cause gait disorders are only at best partially treatable,[3] and the patient is often left with some degree of disability. Still, functional outcomes such as reduction in weight-bearing pain, increase in walking distance, or reduction in overall walking limitation justify treatment. Regaining previous, more normal

gait patterns may be unrealistic, but improving gait speed is a reasonable goal, as long as the gait remains safe.

Medical therapy

What evidence do we have that treatment of gait disorders is effective? Many of the older reports dealing with treatment and rehabilitation of gait disorders in older adults are retrospective chart reviews and case studies. Gait disorders presumably secondary to vitamin B_{12} deficiency, folate deficiency, hypothyroidism, hyperthyroidism, knee osteoarthritis, Parkinson disease, and inflammatory polyneuropathy may respond to medical therapy.[3]

Physical therapy

Physical therapy for diseases such as knee osteoarthritis and stroke brings modest improvements. For example, a combined aerobic, strength, and function-based group exercise program increased gait speed approximately 5% in patients with osteoarthritis of the knee.[41] The focus is on strengthening the extensor groups (especially knee and hip) and stretching commonly shortened muscles (such as the hip flexors [42]). A recent review suggested unclear effects of conventional physical therapy in the treatment of gait disorders due to Parkinson disease,[43] but another study found that using audio and visual sensory cueing can improve gait speed.[44]

Task-specific gait training

Recent studies suggest that the gait impairment can be incrementally reduced with the use of a body support and a treadmill to provide task-specific gait training after total hip arthroplasty,[45] in Parkinson disease,[46] and particularly in patients with stroke-related hemiparesis.[47] However, a Cochrane review found no statistically significant effect favoring treadmill training with or without body support over conventional training to improve gait speed or disability in stroke patients.[48] Nevertheless, the Cochrane review did find a small but clinically important trend (an improvement of 0.24 m/sec in the body weight support plus treadmill group) in those who could walk independently.

Group exercise

A few studies of group exercise have shown improvement in gait measures such as speed. Generally, the most consistent effects are seen in programs that include a variety of exercises. A 12-week combined program of leg resistance, standing balance, and flexibility exercises increased usual gait speed 8% in minimally impaired residents of a "life care" community.[49] A similar, varied, 16-week format with more intensive individual support and prompting in select demented older adults (mean Mini-Mental State Examination score 15) resulted in a 23% improvement in gait speed.[50] These studies note improvement in functional, gait-oriented measures (although not strictly measures of gait "disorder"), such as the distance walked in 6 minutes in patients with knee osteoarthritis undergoing either an aerobic or resistance training program.[51]

Behavioral and environmental modifications

Behavioral and environmental modifications can help patients negotiate their environment more safely and include improved lighting (particularly for those with vestibular or sensory impairment) and elimination of pathway hazards such as clutter, wires, and slippery floors. "Furniture surfing" and lightly touching any firm surface like a wall[52] provide feedback and enhance balance.[53]

Exercise programs give best results if they include a variety of exercises

Orthoses and mobility aids

Orthoses and other mobility aids help reduce gait disorders. Shoe lifts (either internal or external) to correct unequal limb length may be used in a conservative, gradually progressive manner,[54] even though we have few data to support their efficacy. Ankle braces, shoe inserts, shoe body and sole modifications, and their subsequent adjustments are part of standard care for foot and ankle weakness, deformities, and pain but are beyond the scope of this review.[55]

In general, well-fitting walking shoes with low heels, relatively thin, firm soles, and, if feasible, high, fixed heel-collar support are recommended to maximize balance and improve gait.[56] Mobility aids such as canes and walkers[57] reduce load on a painful joint and increase stability.

Surgery

Improvement with some residual disability may be seen after surgical treatment for compressive cervical myelopathy, lumbar stenosis, and normal-pressure hydrocephalus. Few controlled prospective studies and even fewer randomized studies compare surgical vs nonsurgical treatment outcomes for these conditions.

A number of problems plague the published studies. Outcomes such as pain and walking disability are not reported separately. The source of the outcome rating is not clearly identified or blinded. The criteria for classifying outcomes differ. The outcomes may be subjective and subject to interpretation. The follow-up intervals are variable. The subjects who are reported in follow-up may be a highly select group. The selection factors for conservative vs surgical treatment between studies differ or are not specified, and there is publication bias (only positive results are published). Many of the surgical series include all ages, although the mean age is usually above 60. A few studies document equivalent surgical outcomes with conservative, nonsurgical treatment.

Weakness, stiffness, and slowed gait are often seen after total knee replacement

Lumbar and cervical stenosis. Many older adults with lumbar stenosis have less pain and can walk farther after laminectomy and lumbar fusion, although they have residual disability. In a somewhat younger cohort (mean age 69) and after an average of 8 years of follow-up after surgery for lumbar stenosis, approximately half reported that they could not walk two blocks, and many attributed their decreased walking ability to their back problem.[58]

Part of the problem in determining long-term surgical outcomes in patients with lumbar stenosis is that comorbid conditions such as cardiovascular or musculoskeletal disease also influence mobility.[59] Nevertheless, improvement can be seen in some patients over age 75 (mean age 78). A recent uncontrolled study found that the 45% of patients with preoperative "severe" limitation of ambulatory ability had either "minimal" or "moderate" limitation postoperatively after an average of 1.5 years of follow-up.[60]

Nonsurgical treatment of lumbar stenosis—eg, oral anti-inflammatory drugs, heat, exercise, mobilization, epidural injections—may also result in modest improvements, such as in walking tolerance.[61]

Recent studies involving gait outcomes in older adults with cervical stenosis are limited. In a case report, a 65-year-old woman underwent surgery for cervical myelopathy and had subsequent improvement in gait speed.[62] Significant improvement in walking speed can be expected in most patients after surgical decompression of cervical myelopathy.[63]

Normal-pressure hydrocephalus. In a recent noncontrolled study of patients who underwent shunting for normal-pressure hydrocephalus (follow-up interval not specified), walking speed increased by over 10% in 75% of the patients and by more than 25% in over 57% of the patients.[64] While there may be initial improvement after shunt placement, long-term results are often disappointing: eg, 65% of patients who undergo shunting have initial improvement in their gait disorder, but only 26% maintain this improvement by 3-year follow-up.[65] The poor long-term outcomes may be related to concurrent cerebrovascular and cardiovascular disease, a frequent cause of death in these cohorts.[66] Gait outcomes after shunting may be better in those in whom the gait disturbance precedes cognitive impairment and in those who respond with gait speed improvement after a trial of cerebrospinal fluid removal.[67,68]

Joint replacement for osteoarthritis. Outcomes for hip and knee replacement for osteoarthritis are better than for surgery for the other conditions discussed above, although these studies have some of the same methodological problems as those mentioned above. Other than pain relief, sizable gains in gait speed and joint motion occur, but there is often residual walking disability due to residual pathology on the treated side and symptoms on the untreated side.

Despite rehabilitation after total knee replacement, patients are often left with some weakness, stiffness, and a slowed or altered gait.[69,70] Simple function may be maintained after knee replacement, such as the ability to safely clear an obstacle, but usually at the expense of additional compensation by the ipsilateral hip and foot.[71]

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