Approach to the elderly patient with gait disturbance

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Summary

The prevalence of gait disturbances and falls increases dramatically with age, but these problems are not universal in the elderly. They should trigger a systematic search for underlying disease states, many of which can be treated medically or surgically, or significantly ameliorated through provision of physical therapy focused on gait training and aids to ambulation, removal of safety hazards in the environment, and the elimination of polypharmacy. While cardiovascular, orthopedic, and rheumatologic diseases account for the majority of gait disturbances in the elderly, the aim here is to outline an approach to the diagnosis and treatment of a broad array of neurologic conditions causing gait disturbance in the elderly.

Accidental injuries are the fifth most common cause of death in the United States (after deaths due to heart disease, cancer, lung disease, and stroke), accounting for 118,000 deaths in 2009. Falls account for two-thirds of the deaths attributed to accidental injuries. More sobering, while only about 13% of the population is 65 or older, three-fourths of the fatal falls in the United States occur in this age group. These numbers suggest that approximately 60,000 elderly Americans die from falls each year, a number nearly twice that of total deaths due to motor vehicle accidents in 2009 (33,808). The majority of falls occur in children and athletes, but the incidence of falls increases dramatically as individuals live beyond age 65. Among community dwelling people older than 65, 4 in 10 will likely fall at least once this year, and the rate goes up with advancing age. Elderly nursing home and hospitalized patients fall at almost 3 times the rate of their age-mates still living in the community.^{1,2}

Neurologic causes of gait disorder leading to falls range from diseases of muscle, neuromuscular junction, and peripheral nerve, to CNS disorders affecting the spinal cord, brainstem, basal ganglia, cerebellum, deep white matter, and cortex. Thus, a systematic approach to the elderly patient with a gait disorder is best understood as an orderly clinical exploration of the entire neuroaxis, relying on key features of the history and examination to clarify the situation.

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Table 1 Drug classes associated with gait disturbance and falls

Drug class	Sedation	Orthostasis	Hallucinations/ psychosis	Extrapyramidal syndrome	Ataxia/ dizziness
Analgesics	X		X		X
Anticholinergics	X	X	X		X
Anticonvulsants			X		X
Antidepressants	X	X			X
Antihistamines	X	X	X		X
Antihypertensives/ antiarrhythmics		X			X
Antiparkinson	X	X	X		
Antipsychotics	X	X		X	
Benzodiazepines	X		X		X

It must also be kept in mind that Occam's razor rarely applies in the geriatric age group, and many gait disorders turn out to be multifactorial. It is often the case that a patient can get by with an impairment in one system (e.g., decreased visual acuity or peripheral neuropathy or cervical myelopathy), but decompensates when 2 or more systems are disrupted (e.g., decreased visual acuity and peripheral neuropathy and cervical myelopathy). The therapeutic implication of this is that, even when the clinician identifies a neurologic condition that cannot be cured, the overall functional capacity of the patient can be enhanced by attention to optimizing those things that can be optimized. For example, in a study of 1,371 adults aged 65 and older monitored for falls over a 2-year period, going barefoot or in stocking feet was associated with a sharply increased risk of falls, even after controlling for health status (adjusted odds ratio = 11.2, 95% confidence interval = 2.4–51.8). Patients need to be counseled to wear good-fitting shoes, preferably canvas sneakers.³

Table 1 emphasizes the potential adverse impact on gait of multiple different classes of medication. In the United States, 40% of older people take more than 5 drugs per day and 12% take more than 10 drugs per day. Polypharmacy (defined as the use of more than 4 different medications) has been shown to increase the risk of falls in the elderly, independent of the number of comorbid conditions. Reducing the number of medications resulted in a 40% reduction in fall rate in a residential care study.⁴ The balkanization of American medicine, with multiple different prescribers for the same patient, likely contributes to the problem of polypharmacy. Do patients a favor and simplify their medication regimen whenever medically appropriate. Drugs can cause gait disturbance in the elderly through many mechanisms, including decreased cognition/confusion, visual blurring, sleepiness, and orthostasis. Take supine, seated, and standing vital signs on every patient presenting with a gait disorder. Reduce offending agents and attempt to improve orthostasis by nonpharmacologic means such as the use of support stockings, increased fluid intake, added salt when appropriate, and elevation of the head of the bed with bed extenders or blocks by 4 inches or so as tolerated. If these approaches fail to alleviate the problem, consider judicious use of fludrocortisone or midodrine in collaboration with the patient's primary care physician or cardiologist.

Tables 2 and 3 outline the primary neurologic systems involved in normal ambulation and summarize common symptoms and signs associated with impairment in these systems.^{5,6} In general, dysfunction in supporting structures such as bone, muscle, and ligament give rise to complaints of pain and limited range of motion of the affected limb (or spine). The gait often appears antalgic with decreased time spent weight-bearing on the affected side. Careful attention to the patient's chief complaint often points the clinician in the right direction with respect to musculoskeletal etiologies, and examination supplemented by appropriate imaging confirms the diagnosis. Osteoporosis predisposes to falls and heightens their consequence.

Table 2 Neurologic findings by gait disorder

Gait disorder	Strength	Reflexes	Tone	Cognition	Pain	Numbness	Stance/stride
Non-neurologic	Normal	Normal	Normal	Normal	Common	No	Antalgic/limping
Peripheral nervous system	Decreased	Decreased	Decreased to normal	Normal	Common	Common	Normal/foot drop/waddling
Corticospinal	Decreased	Increased	Spastic	Variable	Variable	Variable	Toe scuffing/circumducted
Cerebellar	Normal	Normal	Hypotonic to normal	Variable	Normal	No	Wide/variable
Extrapyramidal	Normal	Normal	Rigid	Executive dysfunction	Variable	No	Narrow/short/shuffling
Vestibular	Normal	Normal	Normal	Normal	Normal	No	Wide/tilting/deviating
Dysmetric	Normal	Normal	Normal	Variable	Normal	No	Wide/chorea/dystonia
Apraxic	Normal	Normal	Paratonic	Executive dysfunction	Normal	No	Variable/short/shuffling
Psychogenic	Give-way	Normal	Normal	Affective lability	Variable	Variable	Astatic/abasic

The other major supporting systems involved in gait are the cardiovascular and pulmonary systems, with syncope, peripheral edema, palpitations, angina, or dyspnea being compelling clues to trigger directed assessment. Of course, there remains a broad array of other potential medical malfunctions (e.g., anemia, hypoglycemia, electrolyte imbalances) that could contribute to gait impairment. For reasons of scope, this article will focus on neurologic assessment.

Disease of muscle is suggested by the distribution of weakness (typically proximal, but not always) and the absence of focal sensory findings. Inflammatory muscle disease is associated with systemic clinical features (dermatomyositis or polymyositis), with confirmation by laboratory studies, electrophysiology, and muscle biopsy. The rate of underlying malignancy is high in patients with dermatomyositis, and should prompt a thorough workup for occult malignancy in all patients with confirmed diagnosis. Some hereditary dystrophies such as limb-girdle dystrophy can onset in later years causing progressive disability over time, so a review of the family history remains important. The incidence of statin-induced myopathy may be higher than previously thought, and complaints of muscle pain or weakness should prompt consideration of a drug holiday. The upper dose range of simvastatin was lowered from 80 mg to 40 mg by the Food and Drug Administration (FDA) in 2011, and the FDA has recently noted rare postmarketing reports of cognitive impairment associated with statin use, while emphasizing that symptoms are generally mild and reversible.

The neuromuscular junction disorders commonly vary over time in a given individual, and the examiner should keep this possibility in mind when an elderly patient complains of intermittent weakness even though none is observed on the initial examination. Myasthenia gravis (MG) tends to present with ocular manifestations rather than gait disturbance in the elderly; the incidence of MG in patients older than 65 is thought to have doubled in the past 25 years (to ~5.3/100,000/year). As with dermatomyositis, the diagnosis of Eaton-Lambert syndrome should prompt a search for underlying malignancy.

Peripheral neuropathy is the most common neurologic cause of gait disorder in an outpatient neurology practice (18%), while stroke and Parkinson disease (PD) are the most common causes of gait impairment in the inpatient neurology setting.^{7,8} More common causes of peripheral neuropathy include diabetes, alcohol, heredity (Charcot-Marie-Tooth type 2 can present as late the sixth decade), thyroid disease, other toxic/metabolic derangements, and chronic inflammatory demyelinating polyradiculoneuropathy. Infiltrative neuropathy due to paraproteins occurs with a higher incidence in the elderly population and should generally be

Table 3 Systems involved in gait and their associated symptoms/findings

System	Symptoms/findings
Peripheral sensory	
Labyrinth/CN VIII	Vertigo/vestibular gait (deviation to one side, improves with eyes open), nystagmus
Eye/CN II	Poor visual acuity or field cuts, stumbling or tentative gait/ocular abnormalities
Peripheral nerve	Staggering, numbness, pain/wide-based ataxia (improves with eyes open), distal sensory loss, hyporeflexia
Peripheral nerve root	Pain/antalgic gait with limping and decreased range of motion, radicular sensory loss, hyporeflexia
Central sensory	
Spinal	Staggering/wide-based ataxia (improves with eyes open), sensory loss (possibly dissociated or with level)
Brainstem	As with spinal/associated brainstem cranial nerve signs
Thalamus	Generally sudden onset (in context of stroke)/hemisensory deficits
Cortex (parietal/occipital/temporal)	Difficulty with visually guided tasks (orienteering)/object or facial recognition errors, homonymous cuts
Peripheral motor	
Muscle	Weakness (generally proximal > distal)/weakness, atrophy, Trendelenburg sign, waddling gait
Neuromuscular junction	Weakness (often episodic)/fatiguing weakness
Peripheral nerve/plexus/root	Weakness, drop foot, stumbling/atrophy and weakness in appropriate distribution, hyporeflexia, hypotonia
Anterior horn cell	Weakness/atrophy, fasciculation, weakness, sensory sparing, hyperreflexia in associated CNS involvement
Central motor	
Spinal cord/brainstem	Weakness, foot dragging, incontinence/spastic gait with unilateral or bilateral circumduction, hyperreflexia
Cerebellum	Staggering/wide-based ataxia (no improvement with eyes open), dysmetria, scanning dysarthria, nystagmus
Basal ganglia/white matter	Stooped posture, slow shuffling gait/bradykinetic-rigid syndrome (spasticity if white matter involvement)
Cortex (frontal)	Poor planning, impulsivity, failure to modulate in response to cues/gait apraxia, dementia, frontal releases
Autonomic	Lightheadedness/syncope, bowel and bladder difficulties/orthostatic hypotension
Non-neurologic/structural	
Bones/joints/ligaments	Poor range of motion, pain, swelling/limited range of motion, tenderness, swelling, possible erythema
Heart/peripheral vascular	Dyspnea, angina, palpitation, claudication, swelling/murmurs, rubs, gallops, poor pulses, pallor, rubor

screened for. The differential diagnostic evaluation of neuropathy is reviewed thoroughly elsewhere and will not be addressed further here, other than to suggest that identification of the underlying cause remains an important first step in management.⁹ Approximately one-third of neuropathy cases remain idiopathic after thorough investigation. Plexopathy remains relatively rare, whereas radicular compromise from degenerative joint disease or metastatic disease (prostate in men, ovarian/breast cancer in women) increases with aging. The diagnosis of radicular disease is

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suggested by radiating pain and appropriate sensory/motor root distribution. Gait disorders associated with disease of the periphery (bone/muscle/ligament/nerve/root) are commonly well managed with a combination of bracing (e.g., ankle foot orthoses, knee stabilizers) and aids to ambulation including regular canes, quad-canes, standard and wheeled walkers, and scooters/electric wheelchairs. Physical therapy can be very useful in these conditions.

Sensory neuropathy due to dorsal root ganglion disease occurs in patients with Sjögren syndrome or as a paraneoplastic phenomenon. The patient demonstrates profound sensory ataxia with wide-based staggering gait. These syndromes have an increased incidence with age.

Progressive weakness with sensory sparing, atrophy, fasciculations, and hyperreflexia implies motor neuron disease (primary cortical and anterior horn cell). Imaging of the cervical spine to rule out a structural lesion near the craniocervical junction is warranted. While bulbar onset is most common in patients older than 75, progressive lower extremity weakness is the presenting symptom of amyotrophic lateral sclerosis in approximately one-fifth of elderly patients diagnosed with the disease.

Chronic cervical myelopathy due to degenerative spine disease is common in the elderly, and can sometimes be obscured by concomitant neuropathy, which robs the patient of the hyperreflexia that would otherwise be apparent. The diagnosis is suggested by the distribution of hyperreflexia (below the level of the foramen magnum), decreased range of motion of the neck, variable radicular symptoms and spastic urinary incontinence with preservation of cognition, eye movements, and the absence of cerebellar or extrapyramidal features. Of course, the presence of such features suggests possible concomitant neurodegenerative or vascular disease of the brain itself, but does not rule out the likelihood of concomitant cervical myelopathy. Because of the invasive nature of the management (neurosurgical decompression), the clinician should have a thorough discussion with the patient (and family as appropriate) prior to obtaining imaging studies. Many times in clinical practice a frail elderly patient will decline to undergo testing because he or she has already decided that surgery is simply not an option to be considered.

B12 deficiency can impact multiple levels of the neuroaxis, including peripheral nerve, posterior columns, and higher cortical function. It should be a routine aspect of the workup for all patients presenting with a gait disorder, neuropathy, myelopathy, or cognitive impairment. Low but nominally normal B12 levels should prompt investigation of methylmalonic acid (MMA) levels, with B12 supplementation if elevated. Similarly, thyroid deficiency (hypothyroidism or hyperthyroidism) should be assessed in those with gait disorder, neuropathy, or cognitive dysfunction.

Brainstem causes of gait disorder in the elderly are generally due to stroke, with diagnosis suggested by the acute onset of weakness or sensory loss, the presence of contralateral long-tract signs, and ipsilateral cranial nerve findings. Initial management unfolds in the inpatient setting, with the stabilized patient typically requiring significant rehabilitation services.

Cerebellar disease from vermian atrophy due to alcohol presents subacutely to chronically. Phenytoin can cause cerebellar atrophy with chronic exposure, or acute ataxia in the setting of acute intoxication. While the acute onset of severe ataxia (especially lateralized) suggests cerebellar stroke or hemorrhage, paraneoplastic cerebellar degeneration can also present acutely to subacutely. Dominantly inherited spinocerebellar ataxias (SCAs) (at last count numbering in

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excess of 30 disorders) generally present indolently well before the age of 65. However, some SCAs can present in later life, with a well-described inverse relationship between the trinucleotide repeat burden and the age at onset in these disorders. Friedreich ataxia is now recognized to have rare variants that can have onset in later years. The grandfathers of young boys with fragile X syndrome can develop progressive ataxia in late life. Multiple system atrophy–cerebellar type (MSA-C) is an important cause of indolently emerging ataxia with peak onset in the late 50s to early 60s, but can occur in older individuals. The diagnosis is suggested by concomitant cerebellar dysfunction and dysautonomia. MSA is not thought to be hereditary, so a positive family history of ataxia should dissuade from the diagnosis. As well, MSA is not associated with dementia, at least early on in the course of illness. More than 100 years after Dejerine and Thomas described sporadic olivo-ponto-cerebellar degeneration, a significant proportion of patients with the insidious onset and indolent progression of cerebellar ataxia in later years have no family history of similar illness, lack dysautonomia or a history of intoxicant, and have no other identifiable cause of their syndrome.

Idiopathic PD (IPD) accounts for approximately 80% of patients with the bradykinetic-rigid (“parkinsonism”) syndrome. Core clinical features include tremor, bradykinesia, rigidity, and postural instability. Not all patients have tremor, and the postural instability in most cases of IPD presents after a “honeymoon” period of about 3–5 years. Secondary (variable) features of parkinsonism include anosmia, hypomimia, hypophonia, dysphagia, drooling, dry eyes, seborrhea skin changes, constipation, urinary urgency, orthostasis, dystonia, depression, anxiety, REM behavior disorder, restless legs syndrome, punding behaviors (compulsive repetition of aimless mechanical tasks), and hallucinations. Robust response of many of the primary and secondary motor features to levodopa challenge helps distinguish IPD from the 20% of patients with a bradykinetic-rigid syndrome due to other causes—the so-called “atypical parkinsonisms” (outlined below). As IPD advances, the majority of patients eventually develop complications of dopaminergic therapy, including motor on/off fluctuations, dyskinesia, unpredictable off periods, and freezing of gait. In addition, the majority of patients with advanced disease have underlying dementia. While this is classically characterized as “subcortical” dementia, a better term would be “striato-frontal” dementia, with significant impairment of executive function and generalized slowing of cognitive responses. Unfortunately, dopaminergic therapy is often dose-limited by worsening hallucinations, drug-induced psychosis, orthostatic symptoms, and troublesome dyskinesia in frail aging patients with IPD. Patients do best with carefully titrated monotherapy carbidopa/levodopa, gradual tapering off of dopamine agonists, and avoidance of anticholinergic agents. Procholinergic therapy with acetylcholinesterase inhibitors has been shown to benefit some patients with Parkinson dementia. Aggressive referral to physical therapy and provision of durable mobility aids remains the most important therapeutic recommendation for patients with IPD with postural instability on retropulsive pull-testing (i.e., those with Hoehn & Yahr stage III disease or higher).

The “atypical parkinsonisms” include progressive supranuclear palsy (PSP), multiple system atrophy–parkinson type (MSA-P), and corticobasal-ganglionic degeneration (CBGD). Striatal and subcortical ischemic cerebrovascular disease can give rise to so-called “vascular parkinsonism.” Normal pressure hydrocephalus (NPH) is often a clinical phenocopy of vascular parkinsonism, with both conditions sharing bradykinesia, apraxic shuffling gait, urinary

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incontinence, and dementia with executive dysfunction. Workup with serial high-volume lumbar punctures should be triggered when imaging shows ventricular enlargement out of proportion to cortical atrophy. The relevant clinical question should generally not be “Is this NPH?” but rather “Would this patient benefit from placement of a ventriculoperitoneal shunt?” Only serial drainage of CSF (or short-term placement of a lumbar drain) can adequately answer that very empirical question.¹⁰

All atypical parkinsonisms share in common their relatively refractory response to dopaminergic therapy (though many patients experience at least some benefit from such therapy). Falls within the first year of onset of parkinsonism are the single best predictor of one of these conditions rather than IPD. PSP and CBGD are tauopathies that share considerable overlap both clinically and pathologically. Whereas PSP and MSA typically present symmetrically, hallmarks of CBGD include its asymmetric presentation, prominent lateralized apraxia (at least initially), action induced myoclonus, and cortical deficits including expressive aphasia and abnormalities of higher order sensory integration. Both PSP and CBGD are dementing diseases, whereas MSA (like IPD, an α -synucleinopathy) is nondementing (at least until very late in the disease). Progressive limitation of voluntary eye movement with preservation of oculocephalic reflex-triggered eye movement suggests PSP—it is this phenomenon that lends the disease its name. This finding, however, occurs relatively late in the clinical evolution of the disorder in a significant minority of patients. Look for slowing of saccadic initiation and velocity, typically impacting vertical eye movements earlier than horizontal eye movements, and often discernible well before gross restriction of smooth-pursuit range. With the exception of vascular parkinsonism, the degenerative diseases noted above all unfold more quickly than IPD.

Diffuse Lewy body disease (DLBD) is suspected when parkinsonism and dementia emerge more or less contemporaneously and the clinical picture does not fit well with PSP or CBGD. Patients with this condition typically have more dramatic fluctuations in their functional level for instrumental and basic activities of daily living. In addition, they commonly have prominent visual hallucinations, even in the absence of dopaminergic therapy (and certainly are more vulnerable to such hallucinations when levodopa is used to treat their motor parkinsonism). Because these patients have Lewy bodies in the nigra (in addition to the limbic system and association cortex) they are particularly vulnerable to the adverse motor effects of dopamine blocking agents (typical and atypical neuroleptics, metoclopramide). Management is with ginger amounts of levodopa and an acetylcholinesterase inhibitor as tolerated. Elderly patients with dementia-related psychosis treated with atypical antipsychotic drugs are at an increased risk of death compared to placebo. While these drugs are not approved for the treatment of patients with dementia-related psychosis, clinicians sometimes turn to them under extreme conditions when behavioral agitation or psychosis is obviously terrorizing the patient. It is important that the family be fully informed of potential risks ahead of time and be included in the decision whether or not to use these agents. In general, less is more with these patients. Benzodiazepines in very low dosages can sometimes be tolerated but can also have paradoxical effects (e.g., worsening agitation).

Alzheimer disease (AD) is by far the most common cause of dementia in the United States, affecting approximately 40% of all 80-year-olds. The incidence climbs from 1/200 at age 65 to 1/10 by age 80. Gait difficulty is generally thought of as a later phenomenon in the disease, but research has demonstrated deficits of measured gait parameters even in patients with mild cognitive impairment.¹¹ AD remains the most prevalent cause of falls in elderly patients with

neurodegenerative disease, even though they tend to occur later in the disease progression. Providing a safe environment, including elimination of hazards in the home and provision of progressively closer supervision, remains the most important therapeutic intervention.

Vascular dementia, though accounting for only approximately 10% of dementia overall, is almost invariably associated with gait disturbance—either hemiparetic or bradykinetic/apraxic in nature. The concomitant appearance of gait disturbance and dementia suggests the diagnosis, with atypical parkinsonisms and NPH also a consideration.

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