The Neurological Examination in Aging, Dementia and Cerebrovascular Disease

Part 3: Coordination, Balance and Gait

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Abstract
This four-part series of articles provides an overview of the neurological examination of the elderly patient, particularly as it applies to patients with cognitive impairment, dementia or cerebrovascular disease. The focus is on the method and interpretation of the bedside physical examination, the mental state and cognitive examinations are not covered in this review. Part 1 (featured in the September issue of Geriatrics & Aging) began with an approach to the neurological examination in normal aging and in disease, and reviewed components of the general physical, head and neck, neurovascular and cranial nerve examinations relevant to aging and dementia. Part 2 (featured in the October issue) covered the motor examination with an emphasis on upper motor neuron signs and movement disorders. Part 3, featured here, reviews the assessment of coordination, balance and gait, and Part 4 will discuss the muscle stretch reflexes, pathological and primitive reflexes, sensory examination and concluding remarks. Throughout this series, special emphasis is placed on the evaluation and interpretation of neurological signs in light of findings considered normal in the elderly.

Cerebellar Testing
Examination of cerebellar function involves testing the limbs for coordination and the trunk for balance. Limb ataxia is assessed by the finger-nose-finger and heel-knee-shin tests, and performance of rapid repetitive and alternating movements in the upper and lower extremities. With cerebellar ataxia, there is incoordination characterized by disturbance in the rate, rhythm and force of movements, and there may be overshooting or undershooting of the target (dysmetria). Other features of cerebellar disease include an impaired “checking response” with excessive rebound when attempting to stop an ongoing movement abruptly, a kinetic (intention) tremor, titubation (3–4 Hz) of the head and body, dysarthria, nystagmus and a variety of other oculomotor disorders including ocular dysmetria (over- or under-shooting when moving the eyes from midline to vertical or horizontal extremes). Intention tremor manifests as a contra-axial (horizontal, side-to-side) oscillation as the extremity reaches its target, unlike essential tremor or the tremor of weakness, in which vertical excursions are usually seen. Lesions in the midline of the cerebellum (cerebellar vermis) produce a wide-based stance and gait, gait ataxia and inability to tandem; consequently, such lesions can be missed if the examiner does not check the patient’s ability to stand and walk. Patients who can stand and walk normally are unlikely to have significant normal cerebellar pathology.

Abnormalities of cerebellar function are not a feature of normal aging and are not part of Alzheimer disease (AD). Ischemic stroke is a frequent cause of focal cerebellar dysfunction in the elderly. Several cerebellar stroke syndromes are recognized, and they are often accompanied by brainstem or occipital infarction. Alcoholic cerebellar degeneration produces a “rostral vermis syndrome” with predominantly truncal and gait ataxia. Pancerebellar dysfunction may result from the cerebellar form of multiple system atrophy (olivopontocerebellar atrophy). Although mild dysmetria has been reported to be a feature in some studies of normal aging, the series by Kaye et al. does not support this: rapid hand closure and finger tapping were slower but ataxia on tests of finger-nose, heel-shin and rapid alternating movements did not occur in healthy elderly subjects aged 64–75 and were very uncommon in old age (85+). Tandem gait (walking a straight line heel-to-toe) was impaired in the majority (91%) of the healthiest oldest old (85+) compared to only 12% of people aged 65–74.

Assessment of Gait, Balance and Mobility
Watching a patient walk is often the most informative part of the neurological examination and may reveal disturbances involving motor, sensory, visual, vestibular, cerebellar, cognitive, psychological and musculoskeletal systems. Gait and mobility problems are common in the elderly and increase with aging. Among community-dwelling seniors, abnormal gait is present in 15% of individuals aged 67–74, 30% in those aged 75–84, and nearly 50% in those aged 85 and over. In the Framingham disability study, 15% of individuals aged 74–84 were unable to climb stairs, 23% could not walk half a mile and 7% could not walk across a small room. Poor gait is often associated with falling and 30% of individuals over age 65 experience at least one fall each year.
Familiarity with the common causes of abnormal gait in the elderly is important in helping the clinician to focus the neurological examination.4-13 The most frequent etiologies in 120 elderly outpatients referred to a neurologist for undiagnosed gait disorder6 and in another series of 50 patients admitted to a neurology service with gait impairment14 are shown in Table 1. Up to one-third of patients had a potentially treatable cause for the gait disorder in these series.14,15

A useful framework for describing gait disorders has been proposed by Nutt, Marsden and Thompson, who distinguished three broad categories based on Hughlings Jackson’s hierarchy of sensorimotor levels:

1. The Lowest-level gait disorders arise from arthritis, myopathy, peripheral neuropathy, sensory ataxia, and vestibular and visual impairment.

2. The Middle-level gait disorders arise from hemiparesis, paraparesis, cerebellar ataxia, parkinsonism, chorea and dystonia.

3. The Highest-level gait disorders include the cautious gait, subcortical disequilibrium, frontal disequilibrium, isolated gait initiation failure and frontal gait disorder, described further below (see Table 2 for a summary of gait patterns in various neurological syndromes).3,13

The standard neuromuscular examination alone is often not sufficient to detect mobility problems in the elderly.16 Therefore, specific assessments of functional mobility are recommended.16-18 To classify the pattern of gait, the examiner determines whether the problem is primarily one of balance, gait initiation, walking or a combination of these factors. Just as a cardiologist auscultating the heart listens in a systematic fashion to each component of the cardiac cycle in sequence, examination of gait should include careful observation of each of the individual components of walking: rising from a chair (without using the arms), stance, posture, base (wide or narrow), gait initiation (including start hesitation or freezing), walking speed, stride length, step height, foot clearance, continuity, symmetry, trunk sway, path deviation, arm swing, involuntary movements (e.g., tremor, chorea, dystonia), ability to turn, ability to walk on heels and toes and squat, and tandem gait.3,17 It is often necessary to walk in a corridor for a longer distance to observe the features of gait, and to bring out fatigability or claudication (vascular or neurogenic). Where appropriate, stair climbing, timed walking tests and videotape recordings may be used to monitor changes over time. The examination can also point to orthopedic and arthritic problems that may be suggested by an antalgic gait, pelvic tilt, Trendelenburg gait or leg-length discrepancy. Postural instability can be judged by performance on the push/pull tests and is predictive of falls.19 In the pull test, the examiner stands behind the patient and gives a backward pull on the patient’s shoulders. The patient is instructed to resist the pull and maintain balance. To protect the patient from falling, the examiner positions himself/herself in front of a wall and stands behind the patient ready to catch the patient if necessary. Normal attempts to regain postural stability include one or two backward steps. Three or four steps indicate mild impairment and more than four steps or retropulsion indicate definite postural instability. Falling en bloc with no effort to regain balance, as may be seen in progressive supranuclear palsy (PSP), indicates more severe postural instability.4,19,20

The Tinetti mobility evaluation is a bedside assessment that has been used to stratify risk of recurrent falls in elderly individuals.17,21 The characteristics of the gait and performance on the following items are rated: sitting balance, arising from a chair, immediate standing balance, standing with feet together, standing with eyes closed, turning 360 degrees, withstanding a push on the sternum, one-leg standing balance for five seconds, back extension, reaching up for an object, bending down to pick up an object and sitting down.17 Additionally, mobility in the supine position can be assessed by watching the patient change positions from supine to side-lying to supine, moving up toward the head of bed while supine, and moving from supine to sitting over the side of the bed.

Several quick and simple bedside tests have been described that correlate with laboratory measures of gait and balance and predict falls. In the “get-up and go” test (patient arises from a chair, walks 10 feet, turns around, walks back and sits down), a score of three or more out of five points indicates a risk of falling.22 The timed version of this test is a particularly useful screening test of physical mobility that correlates with functional independ-

### Table 1

<table>
<thead>
<tr>
<th>Etiology</th>
<th>% of Patients in Sudarsky et al.6</th>
<th>% of Patients in Fuh et al.14</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory deficits</td>
<td>18</td>
<td>-</td>
</tr>
<tr>
<td>Myelopathy</td>
<td>17</td>
<td>22</td>
</tr>
<tr>
<td>Multiple cerebral infarcts</td>
<td>15</td>
<td>24</td>
</tr>
<tr>
<td>Parkinsonism</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>7</td>
<td>-</td>
</tr>
<tr>
<td>Cerebellar disease</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Brain tumour</td>
<td>-</td>
<td>6</td>
</tr>
<tr>
<td>Binswanger’s disease</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>Alzheimer disease</td>
<td>-</td>
<td>4</td>
</tr>
</tbody>
</table>
Neurological Examination

Table 2

**Gait Disorders: Some Recognizable Patterns and Syndromes**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Post-stroke hemiparesis</td>
<td>- Extended, circumducting, spastic, paretic leg with foot drop&lt;br&gt;- Upper limb often adducted and held in flexion at the elbow, wrist and fingers</td>
</tr>
<tr>
<td>Cervical spondylitic myelopathy⁹³</td>
<td>- Stiff-legged, spastic or spastic-ataxic gait&lt;br&gt;- Wearing out and scuffing of the shoes over the toes and anterior sole&lt;br&gt;- Examination may reveal bilateral lower limb spasticity, weakness and bilateral extensor plantar responses&lt;br&gt;- Hyperreflexia may be absent if there is coexisting neuropathy&lt;br&gt;- May have accompanying neck pain, limited cervical spine range of motion or signs of cervical radiculopathy in the upper limbs&lt;br&gt;- A common cause of gait disorder in the elderly</td>
</tr>
<tr>
<td>Cerebellar gait ataxia¹³,⁹⁴</td>
<td>- Wide-based stance, unsteady, veering, lurching&lt;br&gt;- Staggering&lt;br&gt;- Irregular stepping direction and distance&lt;br&gt;- Tandem gait is impaired or impossible</td>
</tr>
<tr>
<td>Parkinson's disease (PD)¹³,⁹⁵,⁹⁶</td>
<td>- Slow and shuffling with short steps, stooped posture and reduced or absent arm swing&lt;br&gt;- The base is narrow and tandem gait is performed relatively well&lt;br&gt;- Resting tremor is often evident during walking&lt;br&gt;- With advancing disease there is increasing postural instability with start hesitation, freezing, festination, propulsion, retropulsion and falls</td>
</tr>
<tr>
<td>Progressive supranuclear palsy¹³,⁹⁵,⁹⁷,⁹⁸</td>
<td>- Parkinsonian or stiff-legged gait with upright posture, axial &gt; limb rigidity, occasional neck extension and prominent postural instability early in the disease course&lt;br&gt;- Falls within first year are especially suggestive of this disorder&lt;br&gt;- Failure to make corrective movements on the pull test resulting in en bloc falling</td>
</tr>
<tr>
<td>Normal pressure hydrocephalus⁶³-⁶⁸</td>
<td>- Initially a cautious gait with impaired tandem progressing to a frontal gait disorder (see below), progressing to a slower, unstable gait with shuffling and magnetic foot response (feet glued to the floor)&lt;br&gt;- May have freezing, propulsion or retropulsion. A broad base with outward rotated feet and reduced step height distinguishes from PD&lt;br&gt;- Gait disorder usually precedes cognitive impairment&lt;br&gt;- Stride length and gait speed may improve after CSF removal or shunting</td>
</tr>
<tr>
<td>Cautious gait³</td>
<td>- Slow, careful gait with reduced stride length, slightly wide base, longer double stance phase, reduced arm swing and leg motion, reduced toe clearance, flexed posture, en bloc turns</td>
</tr>
<tr>
<td>Frontal gait disorder³,⁶⁰</td>
<td>- Combined features of a wide-based ataxic gait and a slow parkinsonian shuffling gait&lt;br&gt;- Balance, gait initiation and locomotion are all impaired&lt;br&gt;- Associated with diffuse cerebrovascular disease, normal pressure hydrocephalus and other diseases affecting the frontal lobes</td>
</tr>
</tbody>
</table>
Gait Disorder in Dementia

The ability to stand on one leg declines markedly with age. The one-leg standing balance test (ability to balance for five seconds) has been recommended as a predictor of injurious falls and low functional status in elderly persons. Normal young individuals (under age 45) can stand on one leg for 30 seconds, whereas the average time is 85+. Normal individuals in the seventh decade should be able to stand for at least five seconds on one leg with eyes open. With eyes closed, the average one-leg standing balance time is 29 seconds for healthy persons age 85+. Normal individuals in the seventh decade should be able to stand for at least five seconds on one leg with eyes open.

Gait Disorder in Dementia With and Without Cerebrovascular Disease

Gait disorders are common in dementia and correlate with disability. Cerebrovascular disease is a leading cause of gait impairment, and focal neurological damage is said to be the major cause of limited mobility in dementia. Gait disorder and poor mobility are more common in patients with vascular dementia (VaD) or mixed dementia than in patients with AD. In fact, early presence of gait disorder or frequent falls are part of the NINDS-AIREN supporting criteria for the diagnosis of probable VaD. Impaired insight and inattention to environmental hazards further increases the risk for falls.

Frontal gait disorder is the gait disturbance most frequently associated with multi-infarct cerebrovascular disease (especially diffuse subcortical small vessel disease [Binswanger’s]), normal pressure hydrocephalus, anterocerebral artery territory infarction, frontal mass lesions, corticobasal degeneration and advanced AD. Referred to previously as Brun’s frontal ataxia, gait apraxia, lower body parkinsonism or arteriosclerotic parkinsonism, frontal gait disorder accounted for 20% of ambulatory difficulty in one series of patients referred to a neurologist for gait disorder. This gait shares features of a wide-based atactic gait and a slow parkinsonian shuffling gait, with impairment of balance, gait initiation and locomotion. There is start and turn hesitation, freezing and disequilibrium. It can be distinguished from Parkinson’s disease (PD) by a normal or increased arm swing, wide base (not seen in PD), upright posture, relatively normal arm function, absence of rest tremor, lack of improvement with visual cues, lack of response to levodopa and presence of upper motor neuron signs. It differs from primary cerebellar disease by the slowness of movement and absence of heel-shin ataxia. Some patients are able to move the lower limbs better when sitting or supine (e.g., able to make stepping and bicycling movements) despite an inability to coordinate the legs in the act of walking. This discrepancy has prompted some to describe this as an apraxia of gait, but this term remains controversial.

Frontal gait disorder is frequently accompanied by dementia, pseudobulbar palsy, dysarthria, paratonic rigidity, incontinence, hyperreflexia and extensor plantar responses. With progression, the gait takes on a frontal disequilibrium pattern with feet glued to the floor (magnetic foot response), severe imbalance and inability to arise, stand, sit or turn in bed. Patients may lean back in the chair on attempting to rise or cross the legs on attempting to walk.

In AD, it is rare for patients to develop difficulty walking until the later stages of the disease. According to the NINDS-ADRDA diagnostic criteria for AD, “gait disturbances at the onset or very early in the course of the illness make the diagnosis of Alzheimer’s disease uncertain or unlikely.” This was confirmed by Ala et al., who reviewed cases of autopsy-proven AD to determine how frequently an abnormal gait was noted at time of presentation. None of the patients with mild dementia had an abnormal gait, and 16% of patients with moderate and 32% with severe dementia had gait abnormalities. The most common gait disturbance in AD is a cautious gait, which occurs more frequently than in age-matched controls; a frontal gait disorder is seen in those with advanced dementia. Early postural instability and falls, which are a hallmark of PSP, are less frequent in PD and would be unusual in typical AD. Patients with dementia with Lewy Bodies (DLB) are also much more likely than AD patients to experience...
multiple falls (37% vs 6%), presumably because of the higher prevalence of parkinsonism.74

In VaD, in contrast to AD, disordered gait is often a prominent and early feature that may precede the development of dementia.75 Periventricular white matter disease correlates with gait impairment, imbalance and falls.75-85 Some studies of dementia patients have found a stronger correlation between periventricular white matter disease and gait/balance measures than with cognitive function.76,84-86 Cerebrovascular disease can sometimes produce a clinical picture that resembles PD (“lower-body parkinsonism”), vascular parkinsonism61,87-90 or PSP.91 Strategically-located strokes affecting the thalamus, basal ganglia or brainstem can produce severe imbalance, inability to stand and falls, despite relatively preserved strength and sensation—a pattern referred to as subcortical disequilibrium.396 It is important to note that patients can present with gait disorder without a known history of stroke.

Part 4, focusing on the muscle stretch reflexes, pathological and primitive reflexes, and sensory examination, will conclude this series in next month’s issue of Geriatrics & Aging.


References

Neurological Examination