

# Disequilibrium and gait disorders in older people

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## Introduction

Disequilibrium is typically described as a feeling of dizziness when standing or walking that is not present while sitting or lying down. Since dizziness is such a common symptom in older people, the symptom may be dismissed as a normal aging phenomenon. On the other hand, the patient may be inappropriately given nonspecific treatment for dizziness such as meclizine or diazepam, which may worsen the condition, due to sedation.

The frequency of dizziness as a presenting complaint increases with age; it is one of the most common complaints in patients over the age of 75. Because dizziness can represent many different abnormal sensations and because the diagnostic evaluation and treatment differ markedly depending on the type of dizziness, the initial task of the examining physician is to determine what the patient means by dizziness. The features of the three most common types of dizziness are summarized in Table 1.<sup>1</sup>

Falls are a well-known source of morbidity and mortality in older people with balance disorders.<sup>2,3</sup> The risk of falls increases with age beyond the age of 60 and it is greater in women than in men.

Most falls in older people result from an accidental slip or trip frequently associated with an unsteady gait.<sup>4</sup> The cause of the disequilibrium can often be traced to decreased sensory input, slowing of motor responses and weakness of support. Medications, musculoskeletal limitations and deconditioning can also contribute to the problem.<sup>5</sup> Falls can be directly traced to an acute attack of dizziness in less than 10% of patients.<sup>4</sup> This low incidence probably can be attributed to the fact that most types of dizziness, including attacks of vertigo, begin slowly enough to allow the patient to sit down or to grab on to a support to avoid falling.

To maintain postural stability when standing and walking, the brain must rapidly process signals from the visual, vestibular and somatosensory systems. Because balance depends on multiple sensory inputs, it can deteriorate when any of these systems fails individually or collectively. Age-related changes have been identified in each of the special senses and in the cortical centres that integrate these signals. Changes in vestibular function with aging have been documented by numerous investigators.<sup>6,7</sup> Degeneration of peripheral

**Table 1.** *Features of different types of dizziness*

Type	Description	Mechanism
Presyncopal Lightheadedness	Impending faint	Diffuse cerebral ischemia
Vertigo	Spinning (environment), motion sickness, tilting	Imbalance of tonic vestibular signals
Disequilibrium	Feeling of being off-balance, unsteady when walking	Abnormal vestibulospinal, proprioceptive, somatosensory, musculoskeletal, cerebellar or motor function

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vestibular structures is known to occur with aging and neuronal loss has been found in the vestibular nuclei and their cortical projections with aging.<sup>8</sup> Impairment of peripheral sensation of the lower limbs correlates with postural instability, and age-dependent proprioceptive loss has been reported.<sup>9</sup> Visual perception is altered with age and poor near-visual acuity also correlates with postural instability.<sup>10</sup> Slowing of central processing of sensory information may also lead to imbalance in older people.<sup>11</sup> There is a loss of cerebellar Purkinje cells with age that can reduce co-ordination and adaptability of visual-vestibular interactions.<sup>12</sup> Cerebral and cerebellar atrophy, ventriculomegaly and subcortical white matter lesions have all been associated with falls in older people.<sup>13,14,15</sup> The combined loss of sensory signals from several systems has been proposed as a common cause of imbalance in older people.<sup>16,17</sup> Cognitive impairment, muscle weakness and the use of certain sedating drugs have been associated with falls in the elderly and may play a role in gait disorders in some older people.<sup>18</sup>

In summary, maintenance of balance and equilibrium is a complex process requiring integration of diverse sensory signals in multiple brain centres. The relative contribution of the proprioceptive, vestibular and visual senses and the cortical, subcortical and cerebellar functions in maintenance of balance is only partly understood. Considering the many possible loci of dysfunction, the examination of a patient complaining of disequilibrium must include a careful assessment of gait, strength, co-ordination, reflexes and sensory function.

### Normal aging

The gradual loss of cells in the sensory and motor centres of the brain with aging is usually a very subtle process that parallels similar slight changes in memory and other cognitive functions generally considered the normal aging process. The gait of normal elderly men is characterized by slight ante-rioflexion of the upper torso with flexion of the arms and knees, diminished arm swing, and shorter step lengths<sup>19</sup>; the gait of older women tends to be narrow-based, with a waddling quality.<sup>20</sup> The majority of older people recognize that they must walk slower, turn more carefully and expect their balance to be less steady than it was in young adulthood. These normal older individuals do not generally present to physicians com-

plaining of dizziness and disequilibrium. Only when one or more disease processes occur do overt symptoms develop. We feel that it is inappropriate to dismiss symptoms of dizziness and disequilibrium as due to normal aging or to such nonspecific entities as presbyastasis or multisensory dizziness.

### Quantification of balance and gait disorders in older people

#### *Bedside tests*

Examination of gait and balance should begin by having the patient walk normally in an open area where there is room for a normal stride without fear of bumping into objects. The patient should be asked to walk normally from one end to the other with normal quick turns. One should observe for stride length, base width, overall posture, associated movements and balance on turning. Having the patient walk heel-to-toe narrows the base and can accentuate imbalance associated with central nervous system disorders, particularly cerebellar lesions. However, it is important to keep in mind that normal older people have trouble walking in tandem with eyes open. In our experience, less than 50% of normal subjects over the age of 75 can take 10 steps in tandem without a side step.<sup>21</sup> Romberg<sup>22</sup> first noted that patients with proprioceptive loss from tabes dorsalis were unable to stand with feet together and eyes closed. Bárány<sup>23</sup> later emphasized the importance of vestibular influences in maintaining the Romberg position with eyes closed. Patients with cerebellar lesions often are unable to stand in the Romberg position even with eyes open.

A variety of semi-quantitative measurements of gait and balance have been developed, such as counting the number of steps made in tandem without a side step, timing the subject's ability to stand in the Romberg position or on one foot with eyes open and with eyes closed, and timing the subject's ability to walk through a designed obstacle course. Performance on these measurements tends to deteriorate with age, although there is large individual variability and the sensitivity for identifying balance disorders in older people has yet to be established. Probably the most widely-used semi-quantitative bedside test of gait and balance is the Tinetti Gait and Balance Scale (1). This scale grades such features as gait speed, stride and

symmetry and balance on standing, turning, with nudging and with eye closure. The maximum score is 28 and normal older subjects should score 24 or higher. Performance on the Tinetti scale has been correlated with the incidence of falls and other measures of disequilibrium and imbalance in older people.<sup>14</sup>

### *Posturography*

Body sway is a normal phenomenon that occurs in everyone. Several studies have shown that sway increases in older people<sup>11,24</sup> and that the frequency of falls increases as sway increases.<sup>25-28</sup> Therefore quantitative measurements of sway could be an important clinical tool for identifying older people with balance disorders who are at risk for falling. Since sway tends to be small when subjects stand on a stable platform, moving platforms (dynamic posturography) have been developed in an attempt to increase test sensitivity.<sup>24,29</sup> The platform can either be tilted or linearly displaced and sway can be measured immediately after the movement or during the movement. Furthermore, in an effort to dissect the different sensory contributions to the maintenance of balance, systems have been developed to selectively manipulate somatosensation and vision.<sup>11,30</sup> With these devices, the angle of sway is fed back to a dynamic posture platform or to a movable visual surround so that movement about the ankle joint or movement of the visual surround is sway-referenced.

Sway during both static and dynamic posturography tends to be greater in older subjects compared with younger subjects.<sup>24</sup> We found that measurements of anterior-posterior sway during angular tilt of the platform best distinguished young from older subjects. Similarly, measurements of anterior-posterior sway during dynamic testing best separated normal older subjects from older patients complaining of disequilibrium and imbalance.<sup>31</sup> However, the posturography information provided little data about the cause of the disequilibrium and imbalance. In summary, posturography, particularly dynamic posturography, can provide a quantitative measure of equilibrium and balance in older people but it is not a diagnostic test to help differentiate between the different causes of disequilibrium and imbalance in older people.

### **Causes of disequilibrium and gait disorders in older people (Table 2)**

#### *Sensory distortion syndromes*

Vertigo results from an imbalance in tonic vestibular activity originating in the inner ear and passed on to central visual-vestibular pathways.<sup>1</sup> Disequilibrium is invariably associated with acute vertigo and is often present in between vertigo attacks. By far the most common cause of vertigo in older people is benign paroxysmal positional vertigo (BPPV). Patients with BPPV develop brief episodes of vertigo (less than a minute) after a position change—typically when they turn over in bed, get in and out of bed, bend down and straighten up, or extend the neck to look up (top-shelf vertigo). Most patients will complain of a vague sense of disequilibrium and unsteadiness that can persist throughout the day. The syndrome results from free-floating otoconial debris, usually within the posterior semicircular canal, which causes gravity-dependent movement of the cupula.<sup>32</sup> It can follow any form of injury to the inner ear (for example, trauma, infection or surgery), but most commonly occurs spontaneously, particularly in older people. Results of the physical examination are usually normal, but rapidly moving the patient from the sitting to head-hanging position (the Dix-Hallpike test) induces a torsional vertical nystagmus when the affected ear is down (upper pole of the eye beats toward the ground). The nystagmus appears after a brief latency (5–10 seconds), lasts about 30 seconds, and fatigues with repeated positioning. Menière's syndrome is classically characterized by episodes of vertigo, unilateral hearing loss, roaring tinnitus, and ear fullness. However, in older people, atypical varieties are common and patients can present with sudden fall attacks.<sup>33</sup> With one variety, delayed endolymphatic hydrops, the patient has a long-standing profound unilateral hearing loss so there are no accompanying hearing symptoms with the episodes of vertigo. The sudden dramatic falling spells (otolithic crises) are thought to result from a sudden pressure change across one of the otolith organs of the inner ear. Patients with vestibular neuritis (presumed to be due to an acute viral inflammation of the vestibular ganglion and nerve) veer and fall toward the side of the lesion, particularly within the first several days. Older patients have more difficulty com-

Table 2. Features and common causes of gait disorders in older people

Category	Common disorders	Characteristic features of gait
Sensory distortion (vertigo)	Benign positional vertigo Menière's syndrome Vestibular neuritis Brain stem/cerebellar infarction	Veering to side Sudden falls Lateropulsion
Sensory loss	Peripheral neuropathy Bilateral vestibulopathy Macular degeneration	'Cautious gait', slightly widened base, shortened stride, slow turns
Musculoskeletal	Osteoarthritis Deconditioning Steroid myopathy	Difficulty arising, general slowness, limp, decreased elevation of feet, locking of knees
Cerebellar lesions	Infarction Alcohol cerebellar Degeneration Late onset cerebellar Atrophy	'Atactic gait', markedly widened base, truncal instability, overactive postural reflexes
Basal ganglia Lesions	Parkinson's disease Progressive supranuclear Palsy	Difficulty starting, freezing, rigidity, lack of associated movements, Turns en bloc
Frontal cortex and subcortical White matter lesions	Multi-infarct syndrome Tumors Hydrocephalus	'Apractic gait', small shuffling steps, feet adhere to the floor, poor reflex control

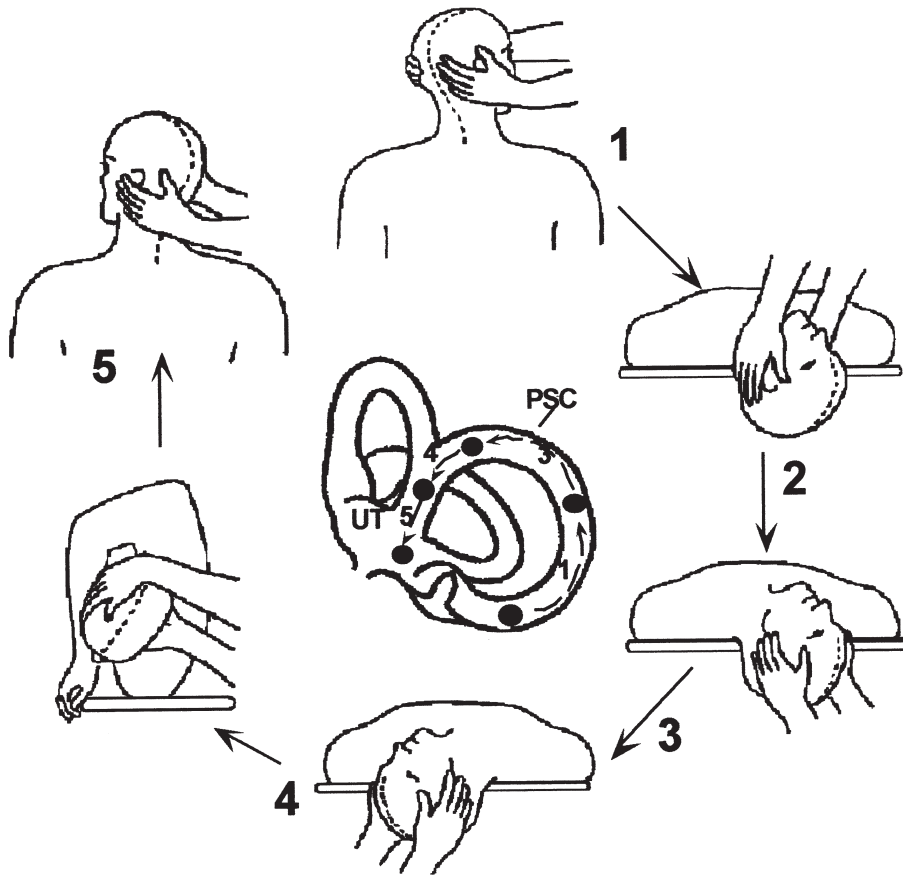
compensating for the acute vestibular loss and the imbalance and unsteadiness can persist for weeks or even months rather than the few days seen in younger patients. Patients with infarction in the lateral brain stem or cerebellum typically will have profound imbalance along with vertigo. Lateral medullary infarction results in lateropulsion, where patients feel a strong force pushing them toward the side of the lesion. Typically, they are unable to stand without assistance.

**Management.** There is now a simple particle repositioning manoeuvre that cures most patients with BPPV (Figure 1). The manoeuvre moves the debris along the posterior semicircular canal and out of the opening of that canal into the utricle.<sup>32</sup> Often older patients with BPPV will not only notice that their positional vertigo disappears after the particle-repositioning manoeuvre, but they will also notice improvement in their gait and balance. Presumably, movement of the debris in the posterior semicircular canal with normal walking is enough to trigger a sense of disequilibrium. Medical man-

agement of Menière's syndrome consists of salt restriction and diuretics. In older patients with a profound unilateral hearing loss, sudden vertigo and fall attacks can be cured by a labyrinthectomy. Surprisingly, we have found that labyrinthectomy is tolerated well in older patients with Menière's syndrome.<sup>33</sup> Currently there is no proven treatment for vestibular neuritis although some recommend a course of steroids and antiviral agents.<sup>34</sup> Compensation for the unilateral vestibular loss can be accelerated with a vestibular rehabilitation programme.<sup>35</sup> Older patients with brain-stem or cerebellar infarction tend to have major permanent balance disorders.<sup>36</sup> Physical therapy can be helpful in maintaining strength and joint mobility but improvement in gait and balance is limited.

#### *Sensory loss syndromes*

Since the somatosensory, vestibular and visual systems provide the main source of information about the position of the head and body in space,



**Figure 1** Treatment manoeuvre for benign positional vertigo affecting the right ear. The procedure can be reversed for treating the left ear.

- 1 The patient is seated upright, with the head facing the examiner, who is standing on the right. The patient should grasp the forearm of the examiner with both hands for stability. The patient is then moved into the supine position, allowing the head to extend just beyond the end of the examining table, with the right ear downward. This position is maintained until the nystagmus ceases.
- 2 The examiner moves to the head of the table, repositioning the hands as shown.
- 3 The head is rotated toward the left, stopping with the right ear upward. This position is maintained for 30 seconds.
- 4 The patient rolls on to the left side, while the examiner rotates the head leftward until the nose is directed toward the floor. This position is then held for 30 seconds.
- 5 The patient is lifted into the sitting position, now facing left. The entire sequence should be repeated until no nystagmus can be elicited. Labyrinth in the center shows the position of the debris before and after each position change as it moves around and out of the posterior semicircular canal (PSC) and into the utricle (UT).

damage to any of these afferent systems can lead to disequilibrium and gait imbalance. As a general rule, the gait disorders associated with loss of sensory input are milder than other gait disorders and are therefore more difficult to identify on examination. Common features include slight widening

of the base, shortening of the stride and slow careful turns. Nutt *et al.*<sup>37</sup> called this a 'cautious gait' which they felt was a nonspecific response to any cause of disequilibrium. Patients with proprioceptive or vestibular loss become 'visual dependent' so that symptoms and signs are much worse in the

dark or with eyes closed (basis of the Romberg test).

Somatosensory and visual function are routinely measured as part of the neurological examination. The characteristic stocking-glove distribution sensory loss of a peripheral neuropathy is easily identified. The list of causes of peripheral neuropathy in older people is extensive but often no cause can be found. Common causes of visual loss in older people include macular degeneration, glaucoma and ischaemic retinopathy. Vestibular function can also be assessed at the bedside. With the 'head thrust' test, patients fixate on the examiner's nose while their head is quickly turned back and forth.<sup>38</sup> Normally the eyes move smoothly back and forth in the orbits. Patients with vestibular loss require several quick catch-up saccades to maintain fixation (to one side with unilateral lesions and to both sides with bilateral lesions). Another qualitative bedside test of vestibular function is to have the patient rapidly oscillate the head back and forth (greater than 1 Hz) while reading a standard visual acuity chart.<sup>39</sup> A drop in visual acuity with head-shaking of greater than two lines on the acuity chart suggests bilateral vestibular loss. The most common identifiable cause of bilateral vestibular loss is ototoxic drug exposure. Common ototoxic drugs include aminoglycosides, loop diuretics and chemotherapeutic agents.

**Management** The treatment of patients with disequilibrium due to sensory loss is aimed at improving sensory function when possible and at training the brain to adjust to the sensory loss. Although the majority of causes of peripheral neuropathy are not reversible, some such as those associated with autoantibodies and B<sub>12</sub> deficiency are potentially reversible. Some patients show improvement in vestibular function after ototoxic drugs are discontinued, but often the damage is irreversible. Potentially ototoxic drugs should be used with great caution in older people particularly those with renal impairment. When such drugs are used, patients should be carefully monitored with daily examinations of gait and balance. Some ototoxic drugs such as streptomycin and gentamicin are remarkably selective for the vestibular system, so that monitoring hearing is of little use. Improving vision by treating an underlying disorder such as glaucoma can be helpful regardless of the underlying cause of disequilibrium.

Physical therapy programmes are aimed at gait and balance training.<sup>40</sup> The goal of these programmes is to retrain the brain to use remaining sensory signals to compensate for the areas lost. Patients are taught to understand the nature of their deficits and simple tricks to help overcome them. For example, patients with bilateral vestibular loss cannot see clearly while they are walking so they are taught to stop and hold their head still whenever they want to read a sign or see the face of a passer-by. They are taught about circumstances to avoid and the proper use of aids such as a cane.

### *Musculoskeletal disorders*

Musculoskeletal disorders are ubiquitous in older people.<sup>2</sup> The musculoskeletal system is less pliable and there is a general decrease in strength. Deconditioning due to lack of physical exercise can lead to a classic vicious cycle whereby joint stiffness and decreased strength lead to less activity which in turn leads to more weakness and stiffness. Osteoarthritic changes in the spine, hips and ankles affect all of us to some degree. Involvement of the spine can lead to nerve root and/or spinal cord compression, producing characteristic peripheral nerve and spinal cord signs. Patients with musculoskeletal disorders have difficulty arising from a sitting position and walk in a slow deliberate fashion. They may show a characteristic limp or lock their knees to overcome proximal weakness.

Examination of any patient complaining of disequilibrium should include a careful assessment of strength and joint mobility of the lower extremities. In addition to testing the strength of individual muscles, have the patient arise from a chair without using the arms, walk up and down steps and walk on the toes and heels. Proximal muscle weakness and stiffness are associated with numerous systemic illnesses. One of the more common disorders, polymyalgia rheumatica, is dramatically responsive to steroids. On the other hand, chronic use of steroids is another important cause of proximal myopathy in older people. Spinal stenosis secondary to osteoarthritis gives a characteristic pattern of upper and lower motor neuron signs. Such patients may have sudden falls due to a loss of muscle strength.

*Management* Physical therapy can be very beneficial in patients with musculoskeletal disorders. Joint range of motion and strength can be improved and the vicious cycle associated with deconditioning can be reversed.

### *Cerebellar lesions*

The cerebellum is commonly considered the balance centre of the brain, yet how it achieves this function is only partially understood. Cerebellectomized animals retain postural reflexes although their ability to control these reflexes is impaired.<sup>41</sup> Functionally, the cerebellum can be divided into two major components: the midline structures, the vermis and flocculonodular lobes, which are critical for maintaining equilibrium, and the hemispheres which control coordination of the limbs. Lesions that produce truncal imbalance invariably involve the midline structures, particularly the anterior vermis or the flocculonodular region.

The characteristic wide-based atactic gait of cerebellar lesions is usually easily differentiated from other gait disorders. Unlike patients with peripheral neuropathy and bilateral vestibular loss, patients with cerebellar lesions are unable to use vision to stabilize their balance, so that walking is severely impaired even with vision. Some patients with lesions involving the anterior lobe of the cerebellum show a characteristic 3 Hz postural tremor.<sup>42</sup>

The diagnosis of a cerebellar gait disorder is usually readily apparent, based on the severity of the truncal instability and the associated oculomotor and extremity incoordination. Characteristic oculomotor signs include spontaneous vertical nystagmus, gaze-evoked nystagmus, rebound nystagmus, saccadic dysmetria and impaired smooth pursuit. Involvement of the cerebellar hemispheres leads to dysrhythmia, dysmetria and intention tremor of the extremities. The most common cause of an acute cerebellar lesion is infarction within the distribution of the vertebrobasilar circulation.<sup>36,43</sup> Isolated infarction of the cerebellum is not uncommon, particularly embolic infarction, since the cerebellar arteries are supplied by the most distal branches of the vertebrobasilar system. The acute vertigo and imbalance of an embolic cerebellar infarct might be confused with the symptoms of a more benign inner ear disorder but a careful examination documenting the degree of imbalance and associated oculomotor findings

should allow one to distinguish between these two conditions.<sup>36,43</sup> Patients with infarction of the midline cerebellum must be watched carefully for several days since progressive swelling of the cerebellum can lead to delayed brain-stem compression which requires immediate surgical intervention. Chronic alcoholism leads to a selective degeneration of the anterior vermis, producing a profound gait disorder with no or minimal involvement of the extremities. The diagnosis rests on identifying a long history of heavy alcohol intake and localized atrophy of the anterior vermis on magnetic resonance imaging (MRI). So-called late onset cerebellar atrophy typically begins in the 30s or 40s although occasionally the symptoms are delayed into the sixth or seventh decade. The process is slowly progressive and invariably there are associated oculomotor findings (particularly spontaneous downbeat nystagmus). About half of the cases are familial, with spinocerebellar atrophy type 6 (SCA-6) being most common.

*Management* Management of cerebellar gait disorders is usually restricted to helping patients learn to protect themselves from dangerous falls. Gait and balance training often have little effect, since the cerebellum is the key centre for adapting postural reflexes. Patients with alcoholic cerebellar degeneration can stop the progression and even may show some improvement after stopping alcohol.

### *Basal ganglia lesions*

The dopaminergic system of the basal ganglia is critical for initiation of gait and for maintaining postural responses. Monkeys who have had this system lesioned by MPTP exhibit a flexed posture, deficient postural responses and freezing.<sup>44</sup> Not infrequently, patients with basal ganglia disorders, particularly patients with Parkinson's disease, will present with frequent falls due to disturbed postural responses. The characteristic gait is dominated by akinesia and rigidity. There is difficulty starting, freezing, lack of associated movement and turning en bloc. Festination, retropulsion or propulsion all can occur.

The diagnosis of Parkinson's disease is apparent if one observes the typical gait features (described above) along with a 'pill-rolling' tremor, generalized rigidity and a masked facies.

On the other hand, early in the disease process, subtle abnormalities of gait may be the only abnormality. Progressive supranuclear palsy (PSP) is a multi-system disorder that may initially resemble Parkinson's disease, but eventually patients develop oculomotor findings (impaired vertical gaze), pseudobulbar signs and cerebellar signs. An important early differential feature between Parkinson's disease and PSP is the posture when walking. Patients with Parkinson's disease have a stooped-forward posture, while patients with PSP have increased tone in the extensors of the neck so that the head and neck are extended backward. The combination of neck extension and impaired down gaze leads to frequent severe falls in patients with PSP.

*Management* Treatment of Parkinson's disease is aimed at replenishing the damaged dopaminergic system of the basal ganglia. The combination of l-dopa plus a peripheral decarboxylase inhibitor is usually the treatment of choice. The gait disorder is often dramatically responsive to l-dopa therapy. Patients with PSP may respond to l-dopa although the benefit is often minimal. Physical therapy aimed at maintaining strength and joint mobility may slow the rate of progression.

#### *Frontal lobe and subcortical white matter lesions*

As noted earlier, minor changes in gait are common with normal aging, but a small number of older people develop a more profound gait disorder, sometimes called 'senile gait'.<sup>13</sup> Their steps shorten and the base widens until their gait is reduced to a shuffle. They turn en bloc rather than with the normal pivot, and upon arising they have great difficulty in initiating the first step. Once they begin, their arms are held rigidly at their sides and they exhibit a characteristic stooped posture. The slow, halting, sliding steps appear as though the feet are adhering to the floor. On examination at this stage, they exhibit cortical release signs and they are unable to relax their limbs voluntarily, a phenomenon called *gegenhalten* or paratonic rigidity. They often attribute their difficulty in walking to a lack of confidence or a fear of falling and, not surprisingly, major falls commonly occur. In late stages, patients cannot walk unassisted and may have difficulty sitting down from a standing position. They land on the edge of the chair and fall off. Ultimately they are confined to bed.

Although many of these features are similar to those seen with basal ganglia disorders, the apraxia along with paratonia and cortical release signs suggest more generalized involvement, particularly frontal lobe involvement.

Periventricular white matter hyperintensities on magnetic resonance imaging (MRI) are common in older people.<sup>13,15</sup> The extensive white matter disease associated with severe hypertension (subcortical arteriosclerotic encephalopathy or Binswanger's disease) causes a characteristic abnormality of gait with elements of both parkinsonism and ataxia.<sup>15</sup> If such extensive white matter lesions cause profound gait abnormalities, earlier stages of white matter lesions probably cause a more subtle gait abnormality. We found that older patients with mild subjective and objective abnormalities of gait and balance have significantly more severe subcortical white matter hyperintensities on MRI than age-matched controls.<sup>15</sup> Interruption of long loop reflexes, particularly those between the thalamus and frontal motor cortices that transverse the periventricular white matter, probably accounts for this spectrum of gait and balance disorders.

The most common cause of the so-called frontal gait disorder is multi-infarct syndrome, a subacute progressive disease commonly associated with hypertension and diabetes mellitus. The MRI identifies multiple infarcts involving both cortex and subcortical white matter on both sides of the brain. Cognitive impairment is usually present although cognitive abnormalities may be relatively mild compared to the profound gait disorder.<sup>45</sup> Primary and secondary tumours involving the subfrontal regions and hydrocephalus are also readily identified on brain imaging studies (MRI or CT). So-called normal pressure hydrocephalus presents with a characteristic clinical triad of apractic gait, cognitive impairment and urinary incontinence, although occasionally the patient will present with only one or two of these features. Although imaging studies can suggest the diagnosis, the most useful diagnostic finding is improvement in the gait disorder after removing 20 or 30 cc of cerebrospinal fluid.

*Management* With the exception of hydrocephalus, which can be dramatically reversed with placement of a shunt, most of the frontal gait disorders seen in older people are not reversible. They can be helped by improving support with



canes or a walker. Tranquilizing medications should be scrupulously avoided inasmuch as they can further impair the central integration of sensory information.

### Summary

Modest impairment of balance and gait occurs with normal aging and most older people recognize that they must be more careful when walking to avoid falls. However, it is usually only when one or more disease processes occurs that patients seek help. It seems inappropriate to dismiss symptoms of dizziness and disequilibrium as due to normal aging or to nonspecific entities such as presbyastasis or multiple sensory deficits. By taking a careful history and performing a detailed neurologic examination, it is usually possible to identify the category of balance and gait disorder and to identify a specific cause (Table 2). There is now a simple bedside cure for the most common cause of vertigo in older people, benign paroxysmal positional vertigo. Furthermore, most older people with gait and balance disorders will benefit from physical therapy aimed at improving joint mobility and strength and at gait and balance training.

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