Human walking and higher-level gait disorders, particularly in the elderly

J.G. Nutt, MD; C.D. Marsden, DSc; and P.D. Thompson, MD

The ability to walk securely without thought is a remarkable human skill. Inability to walk is a major cause of handicap and distress; it is particularly common in the elderly. Although many gait abnormalities in this age group are due to musculoskeletal abnormalities, more frequently they are the result of neurologic disease. Sudarsky and Ronthal analyzed 50 elderly patients with gait disorders presenting to neurologists. Many of the causes of impaired walking that they identified are easily recognized: for example, a spastic paraparesis due to cervical myelopathy, Parkinson’s disease, and ataxia due to cerebellar degeneration or proprioceptive sensory loss. However, the category of gait disorder they termed “frontal gait disorder” or “gait apraxia,” which accounted for 20% of their cases, has caused confusion and debate. Historically, many other terms have been applied to these conditions, including “frontal ataxia,” “marche a petit pas,” “senile gait,” “lower-half parkinsonism,” and “arteriosclerotic parkinsonism.” Our purpose here is to attempt to clarify this murky area and to suggest a nosologic classification of gait disorders that may be of value to clinicians. We will consider (1) the requirements for walking, (2) their neuroanatomic and neurophysiologic bases, (3) a clinical classification of gait disorders, and (4) the voluminous, but confusing, clinical literature and how it relates to our classification of gait disorders.

Requirements for normal walking. What is required to walk? Reduced to its minimum, two abilities are essential to walking: (1) equilibrium, the capacity to assume the upright posture and to maintain balance; and (2) locomotion, the ability to initiate and to maintain rhythmic stepping. These are separate but interrelated components of gait.

Table 1 summarizes the key features.

Equilibrium. Righting reflexes. The human must get from the sitting or lying position to the vertical to walk. This is achieved by righting reflexes that consist of coordinated muscle synergies that bring the head and body into an upright position for stance and locomotion. These reflexes are triggered by vestibular, proprioceptive, tactile, and visual stimuli, any one of which is normally sufficient to allow an animal to right itself.

Supporting reactions. Once vertical, upright posture is maintained by contraction of antigravity muscles, termed supporting reactions. Tone during normal stance is such that slight changes in pattern of muscle contraction alter the position of the body. This is in contrast to decerebrate rigidity in animals in which the marked extensor tone allows the animal to stand but inhibits movement or change in posture.

The vertical posture, with the center of gravity over a narrow base provided by the feet, can be threatened by inherent body sway, by voluntary movement of the limbs and trunk, or by external perturbations. Maintenance of balance is achieved by an interacting sequence of control. The mechanics of the human body provide some resistance to sway through the elasticity of ligaments, tendons, and muscles stabilizing joints, particularly at the ankles, knees, and hips. This alone is inadequate to maintain stance. In addition, balance requires active muscle contraction generated by interacting visual, vestibular, and proprioceptive input, and by experience and foreknowledge.

Anticipatory postural reflexes. Martin was one of the first to emphasize that when the standing human lifts an arm, the trunk shifts to maintain balance. The lifting of the arm is preceded by truncal and leg muscle contractions to shift the body mass to compensate for the altered effect of gravity on the new posture. The presence of such adjustments is revealed by the sensation experienced when lifting an object that is much lighter than anticipated (for example, the “empty suitcase phenomenon”). These anticipatory postural reflexes are of the “feed forward” type; they are programmed in advance of the intended voluntary
movement and are designed to prepare body posture to set the stage for the intended action of the prime mover during that movement. Alternatively, if one pulls on the arm of a standing subject, early muscle responses in distant postural muscles anticipate the resultant shift of the center of gravity. These anticipatory postural reflexes are of the “feedback” type; they can be generated by sensory information from the perturbed limb.

**Reactive postural responses.** When the center of gravity shifts beyond a certain point in the standing subject, a series of sequential reactive postural responses are brought into operation to maintain balance. The earliest responses to body displacement and concomitant muscle stretch are spinal monosynaptic and polysynaptic reflexes. These reflexes are not necessary for the maintenance of upright posture; patients with deafferentation and loss of proprioceptive sensation can stand. “Functional stretch reflexes” appear about 120 msec after an ankle displacement and appear to be responsible for the muscle contractions that preserve balance. These responses, also termed “long loop reflexes” because their latency would allow reflex arcs through brainstem and cerebrum, are most easily seen in the gastrocnemius and tibialis anterior muscles following anteroposterior perturbations of stance when standing, but also involve more proximal leg muscles and trunk muscles. Such responses are complex and have also been referred to as “strategies” for they are influenced by visual and vestibular stimuli, by the nature of the support surface, and by experience and expectation. Reactive postural responses are tested clinically by evaluating the patient’s response to push.

Rescue reactions. If anticipatory postural reflexes and reactive postural responses fail, then rescue reactions involving the arms and legs are brought into play to restore equilibrium. These are involuntary in the sense that they occur automatically, but they also are under voluntary control. They often take the form of a stepping response. The individual whose balance is imperiled takes one or more steps, so as to bring the feet back under the shifted center of gravity. If stepping is inappropriate (for example, when standing on the edge of a cliff), “windmill” arm movements become more appropriate, and the stepping reaction is suppressed. In other words, the nature of these rescue reactions is dictated by the circumstances in which they are called upon.

**Protective reactions.** If the subject is unable to correct his or her posture and restore the displaced center of gravity over a secure base, he or she will fall. Protective reactions such as throwing out the arms then automatically occur to break the fall or protect the face from injury.

The remarkable capacity of these various postural responses of the human to adapt and compensate for changes in the terrain or the mechanical support systems is illustrated by the ability to walk on stilts, skate on ice, perform acrobatics on a high wire, or even adapt to weightlessness, as seen by the behavior of astronauts.

**Locomotion.** Gait ignition. The following sequence of action is required to start to walk. First, the center of gravity must be shifted laterally onto one foot to allow the other to be raised. Second, the center of gravity must be shifted forward to allow the body to move onto the advancing foot. These shifts of the center of gravity are achieved by redistribution of contraction in posturally active muscles, in the course of which a starter signal must be issued to initiate or ignite locomotion, the rhythmic alternating movements of the legs. Clinical disturbances are manifest as difficulties initiating and maintaining stepping.

**Stepping.** Walking then proceeds by alternating, coordinated movement of legs and trunk. Locomotion is influenced by the bones and joints of legs and trunk, strength of muscles, and central nervous system modification of motor programs. Disease can alter the locomotor pattern without affecting gait ignition.

**Neural structures controlling posture and gait.** Peripheral systems. The peripheral sensory and motor systems required for balance and gait are well recognized. Proprioceptive, vestibular, and visual senses are the critical sensory inputs for equilibrium and gait. These senses are redundant, and only one is sufficient under normal conditions. Muscles and peripheral motor nerves constitute the peripheral motor system. In Hughlings Jackson’s terms of lowest, middle, and highest sensorimotor centers, these would be the lowest centers.

Central systems. Here, the student faces a criti-
tical problem. Experimental studies in four-footed animals give little guidance as to how the human walks on two legs. Clearly, there have been major adaptations of the neural mechanisms in quadruped animals to allow bipedal gait. Even subhuman primates are inadequate examples of what humans can do. Nevertheless, one can use animal neurophysiology to decipher the building blocks upon which two-legged humans may operate. What follows is a description of the contribution of various parts of the CNS to equilibrium and locomotion, largely deduced from animal studies.

**Spinal cord.** The cat with a midthoracic spinal transection is not able to stand but if the hindquarters are supported and the cat is placed on a treadmill, it can walk with appropriate leg movements and even adapt the gait to different speeds of the treadmill. Thus, the isolated spinal cord of the cat cannot generate appropriate postural responses but is capable of generating rhythmic stepping.

Spinal interneurons are organized to function as “locomotor generators” and have been the subject of extensive experimental work in species ranging from locusts to monkeys. Rhythmic pattern generators in the spinal cord allow wings to beat, fins to undulate, and legs to walk. Yet, the higher one moves in the animal phylogenetic scale, the more these mechanisms appear to come under control of higher centers. In monkeys, spinal walking cannot be induced by stimuli that would do so in cats and dogs. Humans with spinal cord transections may generate complex stereotyped movements but can neither balance nor generate rhythmic stepping movements to walk. The paraplegic can be provided with aids and supports to allow locomotion, but this is dependent upon the trunk (above the level of transection) providing stability and imparting movement to the artificial props. The quadriplegic is deprived of trunk balance and drive, and therefore cannot walk. So, while spinal rhythmic generators may well be essential as part of the distributed system producing locomotion in humans, by themselves they are insufficient to allow walking.

**Brainstem.** The brainstem, unlike the spinal cord, generates postural responses. The best known example is decerebrate rigidity, marked tone in extensor muscles that allows the animal to stand independently in what has been termed a caricature of normal posture. Righting reflexes are retained in the decerebrate preparations. Electrical stimulation of dorsal and ventral tegmental regions of the pons induces changes in postural muscle tone in decerebrate cats and in awake, freely moving cats. Stimulating the dorsal tegmental field of the pons causes a standing cat to squat and then lie down; stimulation of the ventral tegmental field induces a lying cat to rise and begin to walk. The crucial role of brainstem structures in balance is illustrated by comparison of the effects of lateral lesions affecting corticospinal (pyramidal) pathways with medial lesions affecting reticulospinal, vestibulospinal, and tectospinal systems in the monkey. Lateral lesions do not affect balance or trunk movement; medial brainstem lesions, in contrast, cause profound disequilibrium. The medial pathways primarily affect proximal limb and trunk muscle synergies. This may be one source of the difficulties the clinician has with disequilibrium and gait disorders; clinical neurologic examination emphasizes distal limb function and largely ignores proximal and trunk motor function.

Decerebrated animals, including monkeys, may be induced to walk by electrical stimulation of the subthalamus. Lateral lesions do not affect balance or trunk movement; medial brainstem lesions, in contrast, cause profound disequilibrium. The medial pathways primarily affect proximal limb and trunk muscle synergies. This may be one source of the difficulties the clinician has with disequilibrium and gait disorders; clinical neurologic examination emphasizes distal limb function and largely ignores proximal and trunk motor function.

Brainstem locomotor regions and postural regions are not independent. Stimulation of the dorsal tegmental field of the pons will cause the cat to stop walking, even if the midbrain locomotor region is simultaneously being stimulated. Stimulation of the ventral tegmental field of the pons will make the cat stand and, if the stimulation is strong enough, walk.

It seems highly probable that brainstem locomotor regions also exist in humans, but perhaps they are more dependent upon cortical or subcortical input to activate the system. Projections from sensorimotor, anterior cingulate, and superior frontal cortex to the brainstem reticular formation may provide the higher input required to initiate and maintain gait.

**Basal ganglia.** Denny-Brown observed that bilateral electrolytic lesions in the globus pallidus of the monkey did not disturb the rhythm of walking but markedly disturbed posture and postural responses. The dopaminergic system has been implicated in gait ignition and postural responses by the effect of MPTP in primates; severely parkinsonian monkeys have a flexed posture, deficient postural responses, and freezing.

**Cerebellum.** The cerebellum is commonly considered an organ of balance, but its role in postural responses is unclear. Rademaker described acute disequilibrium following cerebellar lesions but noted that the chronic cerebellectomized dog retained righting, rescue, and protective responses. He envisioned the cerebellum as scaling the response but, since postural responses were retained in the absence of the cerebellum, he reasoned that they must arise elsewhere in the nervous system. Flocculonodular lesions in monkeys produced severe but transient disequilibrium with complete recovery in a few weeks so that the animal could again walk on a narrow bar while blindfolded. Force platform studies in patients with cerebellar disorders are consistent with Rade-
Table 2. Classification of gait syndromes

<table>
<thead>
<tr>
<th>Classification of gait syndromes</th>
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<tbody>
<tr>
<td>I. Lowest-level gait disorders</td>
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<tr>
<td>A. Peripheral skeletomuscle problems</td>
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<tr>
<td>Arthritic gait</td>
</tr>
<tr>
<td>Myopathic gait</td>
</tr>
<tr>
<td>Peripheral neuropathic gait</td>
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<tr>
<td>B. Peripheral sensory problems</td>
</tr>
<tr>
<td>Sensory ataxic gait</td>
</tr>
<tr>
<td>Vestibular ataxic gait</td>
</tr>
<tr>
<td>Visual ataxic gait</td>
</tr>
<tr>
<td>II. Middle-level gait disorders</td>
</tr>
<tr>
<td>Hemiplegic gait</td>
</tr>
<tr>
<td>Paraplegic gait</td>
</tr>
<tr>
<td>Cerebellar ataxic gait</td>
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<tr>
<td>Parkinsonian gait</td>
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<tr>
<td>Choreic gait</td>
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<tr>
<td>Dystonic gait</td>
</tr>
<tr>
<td>III. Highest-level gait disorders</td>
</tr>
<tr>
<td>Cautious gait</td>
</tr>
<tr>
<td>Subcortical disequilibrium</td>
</tr>
<tr>
<td>Frontal disequilibrium</td>
</tr>
<tr>
<td>Isolated gait ignition failure</td>
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<tr>
<td>Frontal gait disorder</td>
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</table>

maker’s hypothesis of cerebellar function; the patients show more-or-less normally patterned postural responses but their scaling is inappropriate, generally too large.24,25

Cortex. The cortex does not have a major role in postural responses as judged by the literature on effects of cortical lesions on balance in animals. Spontaneous walking in the cat requires intact thalami and striata, but no cortex.2,26 However, frontal lesions which spare the primary motor cortex produce postural asymmetries and circling in dogs.27 Similarly, unilateral lesions of Brodmann’s area 8 in monkeys initially induced deviation of the head and eyes ipsilaterally, but after disappearance of the deviation the monkeys continued to rotate ipsilaterally when excited.28 The cortex does appear to be important for very precise foot placement such as is required while walking on a narrow beam.29,30 Placing and hopping reflexes that protect balance in monkeys were impaired by lesions of primary motor cortex, leading Woolsey and Bard31 to conclude that, unlike cats, there was a high degree of cortical control of postural reactions in monkeys. This conclusion is consistent with the large body of clinical information implicating frontal lobes in postural control in humans (see Review of the Evolution of Concepts about Highest-Level Gait Disorders, below).

Classification of gait disorders. Lowest-level postural and gait disturbances. We propose considering gait disorders in terms of Hughlings Jackson’s10 hierarchy of lowest, middle, and highest sensorimotor levels (table 2). Disturbances of proprioception, vision, and labyrinthine sense or dysfunction of the musculoskeletal system produce lowest-level gait and postural disturbances, which are easily recognized by clinicians. It is important to note that dysfunction in these lowest-level functions is generally well compensated if the CNS is intact; consider ambulation in people with blindness, loss of proprioception, or artificial limbs. The corollary is that it is unlikely that gait disorders of the elderly can be attributed to isolated alterations in lowest-level functions such as vibration sense or leg weakness.

Middle-level gait disturbances. Middle-level sensorimotor dysfunction causes distortion of appropriate postural and locomotor synergies. That is, the nervous system selects the correct postural and locomotor responses but their execution is faulty. There is no difficulty initiating walking, but the stepping pattern (locomotion) is abnormal. For instance, a person with cerebellar ataxia retains supporting and protective reflexes and can walk, but the postural responses and locomotion are dysmetric. Examples of middle-level dysfunction include spastic, ataxic, dystonic, and choreic gaits. Early parkinsonism may also fall into this category, although more advanced disease produces equilibration and gait ignition difficulties that are characteristic of highest-level dysfunction. Middle-level postural and gait abnormalities are those with which neurologists are most comfortable. Middle-level sensorimotor dysfunction must be severe to preclude walking.

Highest-level equilibrium and locomotor disturbances. The highest sensorimotor systems are responsible for choosing the postural and locomotor responses that are appropriate for the support surface, body position in space, environment, and the intention of the person. The highest systems are the least understood and the cause of much clinical confusion. The proposed classification of highest-level dysfunction (table 2) is based on clinical features and is derived from our general clinical experience and on a review by one of us (J.G.N.) of 43 patients hospitalized on a busy geriatric service in London, UK, who were identified by medical staff as having balance or walking difficulties that could not be explained by musculoskeletal, spastic, cerebellar, or extrapyramidal syndromes. The characteristics of these patients’ gaits are shown in table 3, and the associated signs are shown in table 4.

Cautious gait. The cautious gait is characterized by a normal to mildly widened base, shortened stride, slowing of walking, and en bloc turns. There is no hesitancy in initiating gait, no shuffling, and no freezing. Cadence is normal and foot clearance of the support surface is normal. There is mild disequilibrium as judged by the response to a push and the difficulty in balancing on one foot. Most patients with this gait pattern are aware of impaired balance and express the need for caution to avoid falls. Other neurologic symptoms and signs are generally mild and do not form a consistent pattern (tables 3 and 4).

But what is compensatory and what is primary?

February 1993 NEUROLOGY 43 271
Table 3. Final classification of unusual or unexplained gait disorders in 43 elderly patients

<table>
<thead>
<tr>
<th>Gait disorder</th>
<th>No. cases (men/women)</th>
<th>Age range (yr)</th>
<th>Disequilibrium (impaired balance)</th>
<th>Gait ignition failure (start/turn hesitation)</th>
<th>Wide base</th>
<th>Shortened stride</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cautious gait</td>
<td>16 (8/8)</td>
<td>65-98</td>
<td>Mild</td>
<td>Absent</td>
<td>Mild</td>
<td>Mild to moderate</td>
</tr>
<tr>
<td>Subcortical disequilibrium</td>
<td>6 (3/3)</td>
<td>70-95</td>
<td>Severe</td>
<td>Variable</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>Frontal disequilibrium</td>
<td>8 (2/6)</td>
<td>69-85</td>
<td>Severe</td>
<td>Variable</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>Isolated gait ignition failure</td>
<td>5 (5/0)</td>
<td>68-75</td>
<td>None</td>
<td>Absent</td>
<td>Absent*</td>
<td>Variable</td>
</tr>
<tr>
<td>Frontal gait disorder</td>
<td>8 (8/0)</td>
<td>75-88</td>
<td>Moderate</td>
<td>Moderate to severe</td>
<td>Variable</td>
<td>Mild to moderate</td>
</tr>
</tbody>
</table>

* Steps could be of normal size and rhythm once walking was under way.

Table 4. Associated findings in elderly patients with gait disorders

<table>
<thead>
<tr>
<th>Type of gait disorder</th>
<th>Dementia</th>
<th>Apraxia</th>
<th>Frontal release signs†</th>
<th>Parkinsonism‡</th>
<th>Pyramidal signs</th>
<th>Urinary incontinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cautious gait</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Subcortical disequilibrium</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Occasional</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Frontal disequilibrium</td>
<td>Common</td>
<td>Occasional</td>
<td>Common</td>
<td>Occasional</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Isolated gait ignition failure</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Occasional</td>
<td>No</td>
</tr>
<tr>
<td>Frontal gait disorder</td>
<td>Common</td>
<td>Occasional</td>
<td>Common</td>
<td>Occasional</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

* Examined by the ability to perform actions to command or mime gestures with arms or legs.
† Frontal release signs included gegenhalten (a variable increase in muscle tone often accompanied by an inability to relax during passive limb manipulation), grasp reflexes (hand and foot), and rooting responses.
‡ Hypokinesia, difficulty executing rhythmic and repetitive alternating or sequential movements.

Many of the gait phenomena described above may be viewed as a natural response to real or perceived instability. If equilibrium is uncertain, such as walking on ice, we shorten our step, slow our speed of walking, and slightly widen our base. Widening the stance base reduces lateral body sway and, to a lesser degree, also reduces anteroposterior motion.22 Our hypothesis is that if balance is perceived as less secure, the gait pattern is altered accordingly. Thus, the cautious gait pattern is an appropriate response to real or perceived disequilibrium. As such, it is nonspecific and can be a response to arthritic disorders, peripheral sensory and motor abnormalities, and to early, high-level disorders. It is the most common abnormal gait pattern in the elderly; 16 of the 43 subjects exhibited a cautious gait.

The appropriately cautious gait should be distinguished from the incapacitating “fear of falling” producing an inappropriately cautious gait. Elderly people who trip, fall, and fracture their femur may be unable to walk independently after hip surgery, despite adequate strength and neurologic function—the “post-fall syndrome.”23 This gait pattern also has been referred to as stasobasiphobia.34 At one extreme, this may lead to timid locomotion around the periphery of the room, clutching onto furniture or walls, giving rise to the so-called space phobia.35 These gait differ from hysterical gait in that other hysterical or functional symptoms and signs are absent36 and the fear of falling is very prominent. No such patients were identified in our clinical series.

Subcortical disequilibrium. This gait pattern is characterized by prominent disequilibrium with absent or ineffective postural responses. Sometimes the postural responses are inappropriate; for example, the patient may hyperextend the trunk and neck and fall backward. Other patients appear inattentive to their posture and unable to generate postural responses although EMG recordings show that indeed there are postural responses, although ineffective.57 Locomotion is primarily impaired because of the disequilibrium. Associated signs include ocular palsies (vertical gaze palsies and pupillary abnormalities), dysarthria, and extrapyramidal signs. Onset may be sudden, suggestive of vascular disease, or slow, compatible with degenerative disorders such as progressive supranuclear palsy or multiple lacunar infarcts. We encountered six such patients in our survey. All were suspected of having sustained a stroke; patients with obvious extrapyramidal syndromes were excluded from our series.

Case report. A previously healthy 84-year-old woman suddenly felt “queer” and collapsed to the floor. She had a small but reactive left pupil and a mild right hemiparesis. The latter greatly improved over the next week or so. Five weeks after the ictus, she could sit without support but was unable to stand without assistance and exhibited no rescue or protective reactions as she fell. Despite the gross disequilibrium, presumed to be due to left subcortical hemorrhage or infarction, she was alert and inquisitive, without evidence of dysphasia, dyspraxia, cognitive deficit, or cerebellar or parkinsonian signs.
Frontal disequilibrium. This gait pattern also is dominated by disequilibrium, frequently with inappropriate or counterproductive postural and locomotor synergies. Patients cannot stand (or sometimes sit) unsupported and are unable to organize trunk and leg movements to rise; when trying to get up from a chair, they would lean back rather than forward, and they would not bring their legs underneath their center of gravity. Nor could they walk; if they were to step, their feet frequently crossed or moved in a direction that was inappropriate to their center of gravity. Other signs on clinical examination included dementia, frontal lobe release signs such as grasp reflexes, motor perseveration, urinary incontinence, pseudobulbar palsies, exaggerated tendon reflexes, and extensor plantar responses. Eight of the 43 patients in our series exhibited frontal disequilibrium.

Case report. An 83-year-old man developed, subacutely, loss of balance and inability to shift his feet, along with urinary incontinence and cognitive impairment. Muscle strength was normal, the plantar responses were flexor, and there was no limb dyspraxia, but he had prominent gegenhalten of the limbs. He was unable to roll over in bed, trying without success to use his arms to grab bedside furniture for assistance and simultaneously making ineffectual movements of his legs. On attempting to rise from sitting, he straightened his legs in front of him and pushed up on the arms of the seat; this only served to thrust his pelvis in the air. Upon being helped to stand, he hyperextended his trunk so that he fell backward unless supported; given help, he would make stepping movements but continued to thrust his trunk backward so that there was no progression. He was presumed to have had multiple strokes.

An important point is whether frontal disequilibrium is distinguishable from subcortical disequilibrium other than by accompanying signs. Marked disequilibrium and distorted postural responses occur in both, often making locomotion impossible. However, if the patient can take steps, these are relatively appropriate in direction and rhythm in subcortical disequilibrium, in contrast to the breakdown of leg movements in frontal disequilibrium.

Isolated gait ignition failure. This gait pattern is characterized by marked difficulty with initiating gait (start hesitation) and maintaining locomotion (turn hesitation, freezing) in the absence of impairment of equilibrium, cognition, limb praxis, or parkinsonism. Once locomotion is initiated, steps are short and barely clear the ground, giving the gait a shuffling appearance. However, with continued stepping the stride lengthens, foot clearance is normal, and the arms swing normally. Diversion of the patient's attention, negotiating narrow passages, or turning commonly precipitate freezing and resumption of shuffling. Tricks such as pretending to kick the bottom of a cane, stepping over the handle of a cane held upside down, or counting frequently aid gait ignition. Postural responses are normal, stance base is normal, and falls are distinctly rare. Five patients exhibited this gait pattern.

Case report. A 68-year-old man noticed increasing difficulty starting to walk or walking in confined spaces. He had had no falls and continued to climb ladders without fear of falling. He stood from the sitting position without difficulty and stood erect on a narrow base. He could balance on one foot, walk heel-to-toe, and withstand a firm push. Upon attempting to start walking, he would shuffle on the spot. Once mobile he walked briskly with long strides, swinging his arms. He could start the first step by holding a cane upside down and stepping over the handle. He was alert and bright, with no weakness, limb dyspraxia, frontal release signs, ataxia, or parkinsonism. His only other deficit was dysarthria. Tendon reflexes were brisk and the right plantar response was extensor. Brain MRI revealed periventricular high-intensity signals on T2-weighted images, consistent with cerebrovascular disease.

Gait ignition failure is easily differentiated from the cautious gait; the cautious gait does not have start hesitation, freezing, or shuffling, nor does the cautious gait improve greatly when under way as does the ignition failure gait. The normal equilibrium and postural responses differentiate gait ignition failure from frontal and subcortical disequilibrium.

Frontal gait disorder. This gait pattern is characterized by a variable base (narrow to wide), short steps, shuffling, start and turn hesitation, and moderate disequilibrium. Patients with this gait syndrome frequently have cerebrovascular disease with multiple hemisphere lesions often producing cognitive impairment, pseudobulbar palsy with dysarthria, frontal release signs, paratonia, pyramidal signs, and urinary disturbances. Eight patients had this gait pattern.

Case report. An 81-year-old man had a right middle cerebral artery occlusion at age 75, from which he made an almost full recovery. At age 79, he developed progressive difficulty with walking and began to fall. When examined, he was dysarthric and moderately demented. There was no dyspraxia, dysphasias, or incontinence. He exhibited gegenhalten without weakness or spasticity. He stood on a wide base and fell if lightly pushed. When attempting to walk, his feet remained glued to the floor; he swayed his trunk from side to side in an attempt to move his feet, but to no avail. Eventually he might get going with a few shuffling short steps on the spot, and then walk slowly and unsteadily on a wide base with short shuffling steps. Turns were accomplished by pivoting on one foot and making minute shuffling steps with the other foot. He froze spontaneously and with any distraction. Walking with assistance helped him initiate gait and increased the stride length to 6 inches.

Frontal gait disorder differs from the cautious gait by the presence of start hesitation and freezing and more marked impairment of postural responses. The cautious gait pattern, as a nonspecific indicator of perceived instability, may precede the appearance of the frontal gait pattern. Frontal gait
Parkinson's disease than frontal gait disorder. Festination and rigidity syndromes without resorting to accompanying signs, hence the term "lower-half parkinsonism." Other helpful clues to a frontal gait disorder are small stepping, but if the base is wide this gait pattern is unlikely to be due to Parkinson's disease. Other helpful clues to a frontal gait disorder are the upright trunk and leg posture when walking, and the preservation of arm swing. Festination and retropulsion or propulsion are more suggestive of Parkinson’s disease than frontal gait disorder.

Table 5. Comparison of proposed terms used to describe the clinical patterns of gait in the elderly in this paper with those from previous publications

<table>
<thead>
<tr>
<th>Proposed terminology</th>
<th>Previous terms</th>
<th>Lesions</th>
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<tbody>
<tr>
<td>Cautious</td>
<td>Elderly gait</td>
<td>Musculoskeletal</td>
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<tr>
<td></td>
<td>Senile gait</td>
<td>Peripheral nervous</td>
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<tr>
<td></td>
<td></td>
<td>Central nervous</td>
</tr>
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<td>Subcortical disequilibrium</td>
<td>Tottering</td>
<td>Midbrain</td>
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<tr>
<td></td>
<td>Astasia-abasia</td>
<td>Basal ganglia</td>
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<td></td>
<td>Thalamic astasia</td>
<td>Thalamus</td>
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<tr>
<td>Frontal disequilibrium</td>
<td>Gait apraxia</td>
<td>Frontal lobe and white matter</td>
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<td>Frontal ataxia</td>
<td>white matter connections</td>
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<td></td>
<td>Astasia-abasia</td>
<td>connections</td>
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<td>Isolated gait failure</td>
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<td>Frontal gait disorder</td>
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Review of the evolution of concepts about highest-level gait disorders. Our categorization of the highest-level gait disorders does not identify previously unrecognized gait patterns but classifies and relates a variety of gaits described in the literature (table 5). In this section, we review the clinical literature about highest-level gait disorders and relate it to our classification.

Cautious gait. Many "normal" elderly walk cautiously. Murray et al.20 examined the walking patterns of healthy men between 20 and 87 years of age and concluded that the "walking performance of older men gave the impression of a guarded or restrained type of walking in an attempt to obtain maximum stability." In comparison with younger subjects, older normal subjects tend to walk at slightly slower speeds with shorter strides on a widened base, with longer double stance phase and shorter swing phase during each walking cycle, less arm and leg motion but preservation of rhythmic stepping. Indeed, the shorter steps often are accompanied by an increase in the number of steps taken. Eble et al.46 recently emphasized that many of these changes in kinematic measurements during the walking cycle in the elderly were attributable to a reduction in velocity of gait and length of stride. These changes may have many causes; in addition to neurologic factors, musculoskeletal changes with aging and a decline in the level of cardiovascular fitness must also be taken into account.40,41 Murray et al.19 suggested that the most obvious changes, the shorter and slightly broader stride, could provide a more secure base for supporting the body when walking.

The importance of a deterioration in the control of body sway as a factor contributing to the dissolution of gait in the elderly is not a new concept. Anteroposterior trunk sway is increased in the elderly, especially in those who fall.42,43 Several authors have found a correlation between postural sway and the liability to falls and functional mobility.44,45 Deteriorating postural responses therefore appear to be an important factor in the breakdown of gait and posture in the elderly.46 Compensation for this might involve a slight widening of stance, an increase in the double stance phase of each step, and a reduction in stride length, resulting in slowing of the speed of walking, without altering rhythmicity of locomotion.

Why balance control becomes less efficient in old age is not clear. It is most likely multifactorial. One contributor may be a musculoskeletal system that is less pliable and, thus, less rapid in its response to muscular instructions to limit sway. Decreased strength is another variable. Furthermore, the motor plan may be less accurate. This may be due to slight errors in the accuracy of the input from proprioceptors, vestibular organs, or the eyes. Finally, there may be errors in the central evaluation of sensory input, or computation of appropriate motor responses. Although deterioration in balance may be directly related to aging, clinically signifi-
cant disequilibrium is most likely a consequence of superimposed neurosensory disorders.47

The cautious gait also is adopted by those with manifest neurologic disease. Elbbe et al46 studied the kinematics of gait in 10 elderly subjects with a variety of neurologic disorders, including vascular dementia, normal-pressure hydrocephalus, Alzheimer's dementia, and peripheral neuropathy. All walked slower than normal with shortened strides. All kinematic measures (a slight widening of the base, a longer double-limb stance time, a reduction of maximum heel-to-toe displacement, reduced hip rotation and knee flexion) were statistically attributable to the reduction in stride length. There were no characteristics of gait that distinguished the various etiologies. Elbbe et al termed this pattern of walking “the syndrome of senile gait,” but we would argue that a similar pattern also is adopted by younger patients, hence our use of the term “the cautious gait.” Those with neurologic disease may adopt a cautious gait which is superimposed on their underlying basic neurologic deficit. Early in the course of such disorders, the cautious gait may dominate, but with progression of the condition, the characteristics of the underlying gait disorder will emerge.

Subcortical disequilibrium. Subcortical disequilibrium has been described as an acute phenomenon after thalamic, basal ganglia, or midbrain stroke. Masdeu and Gorelick48 reported a series of patients who could not stand after acute vascular lesions of the thalamus, particularly those involving the superior portion of the ventrolateral nucleus. Despite relatively preserved strength and sensation, they fell backward or to the side contralateral to the lesion. This deficit improved over a matter of days or weeks. Labadie et al49 reported a similar phenomenon in patients with infarction or hemorrhage of the putamen (also sometimes involving the globus pallidus). These patients tilted slowly and collapsed toward the side opposite to the lesion “like a falling log,” without appropriate corrective action. A similar clinical picture has been reported to result from a unilateral midbrain lesion.50

Such disequilibrium is similar to that seen early in the course of some progressive degenerative parkinson-plus syndromes, such as progressive supranuclear palsy or multiple system atrophy, but does not occur early in classical Parkinson’s disease. The blurring of the distinction between degenerative, extrapyramidal disorders and subcortical vascular disease is evidenced by the report that the multi-infarct state can produce the clinical picture of progressive supranuclear palsy.51

Frontal disequilibrium. Bruns52 is credited with the first use of the term “frontal ataxia” to describe severe disequilibrium due to frontal mass lesions, preventing standing or walking unaided. Among the four patients he described, one exhibited retropulsion and another crossed the legs when attempting to walk, signs that distinguished this gait syndrome from cerebellar ataxia. Bruns considered several etiologies for the disequilibrium, including the possibility that the frontal lobes influenced balance via frontopontocerebellar connections. Gerstmann and Schilder53 reported two patients, one with a frontal glioma and the other with presumed encephalitis, who were unable to lift their feet from the floor when attempting to walk and unable to sit or stand without falling backward. Their limb and body movements were clumsy and stiff and they exhibited perseveration and incontinence. The authors distinguished these gait difficulties from those of cerebellar ataxia and suggested that their problems represented an apraxia of gait, along the lines of Leipmann’s concepts of apraxia for higher-level disorders of arm and hand movements. Van Bogaert and Martin54 described a woman with a frontal abscess whose gait evolved rapidly from an unsteady, weaving walk to an inability to stand. In the course of her illness she exhibited small steps and bizarre, clumsy movements of the legs, which became tangled, causing her to fall. She was unable to make purposeful movements of the legs to command, such as bicycling of the legs when lying in bed, but might do so spontaneously. They interpreted the gait and postural disturbance in terms of an ideomotor apraxia (apraxia de la marche), namely, a disconnection between the idea of walking and the motor programs required to walk. Bell55 in an article on apraxia associated with corpus callosum lesions, described a woman with a right frontal glioma invading the corpus callosum who could not stand upright without falling backward or collapsing in a heap and who made no attempt to walk. A second patient with a small granuloma compressing the corpus callosum accompanied by “marked hydrocephalus” could not stand without assistance. The gait was “bizarre . . . she would twist the body to one side, then raise the opposite leg much higher than was necessary or usual and bring it down as though she was uncertain as to where the floor was or how to make contact with it.”

Petrovici56 interpreted the “apraxia of the gait and trunk” as a disturbance of “postural set.” Of the five patients he reported, three (with hydrocephalus, diffuse cerebrovascular disease, and Pick’s disease) had severe disequilibrium and could only stand with support. They were unable to initiate steps, but two were able to move their legs with greater freedom when lying down. Frontal lobe release signs were present in all three patients. The patient with presumed Pick’s disease exhibited dementia and upper limb apraxia. Petrovici’s cases 4 and 5 (with atherosclerosis and a left frontal astrocytoma, respectively), however, are consistent with our category of frontal gait disorder. They had mild disequilibrium, difficulty rising from the seated position, start hesitation, and shuffling gaits.

All of these clinical descriptions have in common a severe disturbance of equilibrium and inappropriate postural responses. Their gaits were often
bizarre, with crossing of the legs and ineffective propulsion. Some had elements of gait ignition failure (start hesitation). Dementia, incontinence, perseveration, difficulty with repetitive movements, and frontal release signs further point to a disturbance of function in the frontal lobes and their connections. These reports suggest that a variety of structural lesions of the frontal lobes, including tumor, abscess, infarction or hemorrhage, hydrocephalus, or diffuse white matter disease can produce a gait disturbance characterized by profound impairment of postural responses and imbalance (difficulty rising, standing, resisting a push), and variable difficulty with initiating and maintaining gait.

Do these disturbances of gait represent apraxia as suggested by many of the authors above? Several objections to this postulate may be raised. First, to accept “gait apraxia” as an entity, “equilibrium apraxia” must also be accepted because in many of the patients, the distortion of postural responses was the most prominent aspect of their disorder. Second, patients with marked bilateral apraxia or apraxia for sitting may walk normally. Conversely, many patients with gait disturbances described as apraxic do not have limb apraxia. Thus, “gait apraxia” is not routinely associated with other evidence of apraxia. Third, the decerebrate monkey can be made to walk by stimulation of the subthalamic locomotor region, suggesting that cortical areas do not contribute specific programming for gait but rather some facilitatory effect to turn on locomotion.

Isolated gait ignition failure. Petren described five patients who exhibited a gait he termed “trepidant abasia.” This gait was characterized by start hesitation, stepping in place, dragging of the feet, freezing, and turn hesitation. Petren was impressed by the variability in gait, the fact that the patients could at times walk normally, and that the gait was improved by counting aloud or other forms of encouragement. He considered, but dismissed, hysteria as the cause and discussed the possibility that the patients were no longer able to “imagine” the act of walking or that there was a problem with “attention” to the motor task of walking and suppression of distracting sensory information. Review of Petren’s case histories suggests that case 1 may have had relatively isolated gait ignition failure. The staring immobile eyes, bradykinesia, extended neck and trunk posture in case 2 suggest progressive supranuclear palsy; cases 3 to 5 had disequilibrium in addition to locomotor difficulties. In our classification, the latter cases would be considered examples of frontal gait disorder. Von Malaise identified march petit pas to be equivalent to frontal gait disorder with disturbance of balance, ignition, and locomotion, which impair but do not preclude locomotion. At the beginning, frontal gait disorder resembles a short-stepped military gait, with an upright trunk posture and stiff legs. As the underlying condition progresses, start hesitation, shuffling, freezing, and increasing disequilibrium appear. Von Malaise identified march petit pas by the shortness of stride, reduced excursion at the joints of the lower limbs, shallow steps which barely cleared the ground, reduced swing time, and increased double stance time. The gait had a variable asymmetry, the patient leading with one foot and bringing the other foot up to the leading foot, the leading foot changing from day-to-day. He considered this gait to be the result of multiple lacunar infarcts and demonstrated this on pathologic exami-
in one case. Dejerine described the disturbance of equilibrium, rigid trunk posture, and the frequent association with a pseudobulbar palsy. Under the title of “arteriosclerotic parkinsonism,” Critchley described the same shuffling gait, although this review contained only a brief case descriptions. He later emphasized the diagnostic significance of this gait pattern in diffuse cerebral arterial disease and ascribed it to apraxia.66 Thus, this gait has come to be associated with vascular disease, particularly with subcortical arteriosclerotic encephalopathy and the lacunar state (Binswanger’s disease).38

Meyer and Barron, in an influential review, reported seven patients with frontal lobe disease due to vascular insults, tumor, or atrophy who walked slowly on a wide base, with the feet rooted to the ground, with shuffling short steps and freezing. All had disequilibrium manifest by retropulsion, difficulty rising to a sitting or standing position, and falls. The authors postulated the gait disturbance was a form of apraxia due to a transcortical involvement, difficulty rising to a sitting or standing position, and falls. They defined apraxia of gait in terms of Leipmann’s limb kinetic apraxia as “the loss of the ability to properly use the lower limbs in the act of walking which cannot be accounted for by demonstrable sensory impairment or motor weakness.” They noted that all movements of the legs were abnormal in their patients, with difficulty in performing rapid alternating, bilateral simultaneous or abstract movements with the legs. Motor perseveration, rigidity, hypokinesia, and gegenhalten were present, features that were not seen in patients with cerebellar ataxia.

Similar abnormalities of equilibrium and gait have been described in patients with hydrocephalus.61,65 Estanol, in contrast to Meyer and Barron, noted the ability of his patients to carry out abstract movements with the legs such as kicking an imaginary ball or bicycling in bed, if supported. He suggested that a disinhibition of proprioceptive reflexes might underlie the patients’ difficulties. Fisher attributed the imbalance and short shuffling steps of the “elderly gait” to hydrocephalus, although his criteria for the diagnosis of hydrocephalus are controversial.

Thus, as in the case of frontal disequilibrium, a variety of frontal lobe and subcortical pathologies have been reported to cause the characteristic clinical picture of a frontal gait disorder. Again, some have employed the term “gait apraxia” to describe this condition. As discussed above, there are reasons for rejecting this term. It does not describe what is seen and it presumes an unlikely mechanism to explain these various disorders.

Discussion and conclusions. We have attempted to clarify gait disorders due to highest-level sensorimotor disorders that particularly affect the elderly. Our approach is clinical, namely, to define recognizable gait syndromes. The classification is based on what seem to be separable components of gait: (1) compensatory changes in response to perceived or real disequilibrium (cautious gait), (2) prominent disequilibrium or postural discontrol (subcortical and frontal disequilibrium), (3) difficulty initiating and maintaining locomotion (isolated gait ignition failure), and (4) combinations of the above.

A crucial question is the extent to which these entities are separate or overlap. In pure culture they are quite distinctive, but it must be admitted that the edges are blurred. It may be that gait disorders will be analogous to language disorders; classical Wernicke’s and Broca’s aphasias are easily distinguishable, but in most aphasic patients there are elements of both types of aphasias. Likewise, early dementia may present patterns that suggest predominant frontal, parietal, or subcortical involvement, but with progression, these distinctions are lost and replaced by a picture of global dementia. Similarly, many patients with gait disorders will have mixtures of disequilibrium, locomotor abnormalities, and gait ignition difficulties. In some patients, our classification may resemble stages in the dissolution of gait rather than separate entities; for example, a patient may start with a cautious gait pattern, progress to a frontal gait disorder, and finally develop frontal disequilibrium. Nevertheless, we believe that these entities are useful clinical categories on which further research can be based.

One impression from the review of the clinical literature is that the concepts of highest-level gait disorders are based on a very small number of patients with no longitudinal studies and very few clinicopathologic correlations. Clearly, more patients with well-defined gaits need careful post-mortem examinations to define the neural substrate of the disorders.

We suggest that description of disordered gaits should proceed in three steps: (1) description of the characteristics of balance and locomotion (see below); (2) classification into one of the categories summarized in tables 2 and 3; and (3) definition of the cause, both in terms of pathology and pathophysiology, if possible.

Clinically, description of gait disorders will be facilitated by attention to each of the following key elements: (1) rising from a chair (righting reflex); (2) standing (supporting response); (3) withstanding a push fore and aft and side-to-side (reactive postural responses); (4) initiation of walking, presence of freezing, start hesitation, and turn hesitation; (5) locomotion, with reference to width of the base, stride length, foot clearance, arm swing, and cadence; (6) negotiation of turns; and (7) presence of effective rescue and protective reactions if imbalance spontaneously occurs, or in response to a push. Communication between observers may be improved by such a detailed description, and would be enhanced by videotaping the standard sequence.

We hope that these observations will provide a
future baseline for further study of gait disorders, to be refined and challenged by further observation.

Acknowledgments

We thank Drs. N.L. Leenders, Pippa Tyrrell, and Eric Salmon for assistance in translation of French and German references, and Dr. Angella Middleton for providing us access to the geriatric population at St. Charles Hospital.

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278 NEUROLOGY 43 February 1993

Human walking and higher–level gait disorders, particularly in the elderly
J. G. Nutt, C. D. Marsden and P. D. Thompson
Neurology 1993;43;268
DOI 10.1212/WNL.43.2.268

This information is current as of February 1, 1993

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