Gait disorders in elderly

By R2 Phatharajit Phatharodom
Introduction

• Gait disorders are common in elderly populations
• Prevalence increases with age
• At the age of 60 years, 85% of people have a normal gait
• At the age of 85 years or older this proportion has dropped to 18%

Introduction

• Gait disorders have devastating consequences → falling → reduction of mobility → loss of independence

• Gait disturbances—even when they present in isolation—can reflect an early, preclinical, underlying cerebrovascular or neurodegenerative disease
Outline

• Pathophysiology of gait
• Anatomical aspects of gait
• Gait and mental function
• Effect of normal ageing on locomotion and gait
• Specific gait disorders
  – Neurological gait disorders
  – Non-neurological gait disorders
Pathophysiology of gait disorders

• Normal gait requires a delicate balance between various interacting neuronal systems
  – **Locomotion** - including initiation and maintenance of rhythmic stepping
  – **Balance**
  – **Ability** to adapt to the environment

Pathophysiology of gait disorders

• The control of gait and posture is multifactorial, and a defect at any level of control can result in a gait disorder
Anatomical aspects of gait

- Poorly understood in humans

- Brainstem locomotor centers → reticulospinal and vestibulospinal projection in ventromedial descending brainstem pathways → conveys signal to interneuron “central pattern generators or spinal locomotor centers” → limb movement in synergy and elaborate walking patterns of muscle activity
Anatomical aspects of gait

• Propriospinal networks link hindlimb, forelimb, and trunk networks to facilitate interlimb coordination

• The cerebral cortex and corticospinal tract are required for precision stepping

• The isolated spinal cord can produce spontaneous movements, but cannot generate rhythmic stepping or maintain truncal balance

• Brainstem and higher cortical connections are necessary for bipedal walking in humans
Anatomical aspects of gait

- **Supraspinal centers** signal when to start walking, when to stop, the speed of locomotion, and the size and direction of stepping.

- **Basal ganglia connections with frontal cortical and basal ganglia** motor circuits influence the initiation of walking and maintain the sequence of rhythmic stepping.
Anatomical aspects of gait

• The **cerebellum** is important in modulating the rate, rhythm, amplitude, and force of voluntary movement and, accordingly, regulates these aspects of stepping.

• **Sensory feedback** about the environment and terrain during the walking cycle also modify motor cortical activity and walking.
Gait and mental function
Gait and mental function

• Normal walking requires strategic planning of the best route, interaction with the environment and with internal factors.

• Failing to understand the significance of an obstacle, choosing an inappropriate route, or misinterpreting one’s own physical abilities can all lead to falls.
Gait and mental function

• Safety and efficacy of normal walking rely on
  – Sensorimotor systems
  – Interaction between the executive control dimension (integration and decision of action) with the cognitive dimension (eg, navigation, visuospatial perception, or attention) and the affective dimension (mood, cautiousness, and risk-taking)
Gait and mental function

- Dual task *(walking and anything else)* paradigm has become the classic way to assess the interaction between cognition and gait.

- In elderly people, this dual task ability deteriorates because *central resources decline*.

- Elderly people slow down or have an increased stride variability (suggesting reduced automaticity) while performing a secondary task during walking.

- Gait becomes less secure and the risk of falling increases.
Gait and mental function

• When **falling** young healthy people → neglect the secondary task and lend more priority to walking safely

• Prudent posture-first strategy is diminished in elderly people and failure to prioritise gait under difficult circumstances

• Research has shown that frontal executive functions are especially important for maintaining walking stability
Gait and mental function

• Dysexecutive functions can be the primary cause of falls in a group of idiopathic elderly fallers

• Explain why falls are so common in patients with dementia and why demented patients are so vulnerable to dual task performance while walking

• Affective disorders are also associated with gait problems in elderly people
Effect of normal ageing on locomotion and gait
Effect of normal ageing on locomotion and gait

• Many older people accept their gait difficulty as being normal for their age and their doctors often support them in this view.

• Senile gait disorder: the slow, shuffling, and cautious walking pattern commonly seen in older age

• Is it true??

• Up to 20% of very old individuals walk normally, hence gait disorders are certainly not an inevitable feature of old age
Effect of normal ageing on locomotion and gait

- The elderly who have gait impairment in fact suffer from underlying disease.

- Senile gait disorders are an early manifestation of underlying pathology, most notably changes, or subclinical changes.

The study suggest abandoning the term senile gait as a specific gait category.

Specific gait disorders
Recognition of specific gait disorders

• 153 community residents aged 88 years and older

• About 61% reported distinct diseases as a cause of gait impairment
  – Non-neurological disorders were the leading causes of gait impairment, in particular joint pain (52 of 87 people)
  – Many others had multiple causes for their gait impairment
  – Stroke was the most common neurological cause

Recognition of specific gait disorders

• Another study of 120 elderly outpatients showed the most common causes for gait disorders were:
  – Sensory ataxia (18%)
  – Myelopathy (17%)
  – Multiple strokes (15%)
  – Parkinsonism (12%)

Neurological gait disorders

- Hypokinetic-rigid gait disorders
- Spastic gait disorders
- Myopathic gait
- Neurogenic gait
- Cerebellar ataxic gait
- Sensory ataxic gait
- Dyskinetic gait
- Frontal lobe gait disorders
Hypokinetic-rigid gait disorders

- Disease of basal ganglia and frontal lobe
- Most common in *Parkinson’s disease*
- Parkinsonian gait
- The posture is *stooped* with flexion of the shoulders, neck, and trunk
- During walking, there is little associated or synergistic body movement
- *Hesitation and freezing*
Hypokinetic-rigid gait disorders

• **Shuffling** with a reduced step height, often with a reduced stride length, leading to slowness of gait

• **Reduced arm swing**

• To maintain balance when walking and avoid falling forward, the patient may advance with a series of rapid, small steps (**festination**)

• Turning movements become slow and are executed **en bloc**
Hypokinetic-rigid gait disorders
Hypokinetic-rigid gait disorders

• Falls occur in Parkinson’s disease when festinating steps are too small to restore balance

• Tripping or stumbling over rough surfaces because steps are too shallow to clear obstacles and corrective steps are too small also leads to falls
Hypokinetic-rigid gait disorders
anatomy aspect

• Etiology:
  • Basal ganglia and their connection to the frontal cortex, brainstem, or both
  • Lesion in frontal lobe
Hypokinetic-rigid gait disorders

Idiopathic Parkinson disease
Secondary parkinsonism
Parkinsonism plus syndromes
Heredodegenerative parkinsonism
<table>
<thead>
<tr>
<th>Main anatomical substrate</th>
<th>Disease process</th>
<th>Characteristic features</th>
<th>Associated features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parkinson’s disease (PD)</td>
<td>Substantia nigra</td>
<td>Neurodegenerative</td>
<td>Good response to levodopa, Resting tremor hand(s)</td>
</tr>
<tr>
<td>Multiple system atrophy, parkinsonian type</td>
<td>Basal ganglia</td>
<td>Neuropathic</td>
<td></td>
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<tr>
<td></td>
<td>Cerebellum</td>
<td>Early phase like PD gait</td>
<td>Cerebellar ataxia, Autonomic features, Pyramidal signs</td>
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<tr>
<td></td>
<td>Pyramidal tracts</td>
<td></td>
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<td></td>
<td>Autonomic nervous system</td>
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<tr>
<td>Progressive supranuclear palsy</td>
<td>Diffuse brainstem pathology</td>
<td>Wide-based gait, Freezing common, Erect posture, but with retropulsion, Early spontaneous/backward falls, Motor weakness, Frequent and severe injuries</td>
<td>Vertical gaze palsy, Pseudobulbar palsy, Frontal dementia, Apoplexia sign</td>
</tr>
<tr>
<td>Corticobasal degeneration</td>
<td>Basal ganglia</td>
<td>Neuropathic</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cortex</td>
<td>Asymmetrical presentation --- eg. unilaterally, Leg apraxia, dystonia, or myoclonus</td>
<td></td>
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<tr>
<td>Dementia with Lewy bodies</td>
<td>Basal ganglia</td>
<td>Neuropathic</td>
<td></td>
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<tr>
<td></td>
<td>Cortex</td>
<td>Like PD gait, More symmetric</td>
<td></td>
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<tr>
<td>Subcortical arteriosclerotic encephalopathy</td>
<td>Subcortical white matter</td>
<td>Vascular</td>
<td></td>
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<tr>
<td>Vascular parkinsonism</td>
<td>Diffuse white matter</td>
<td>Vascular</td>
<td></td>
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<tr>
<td></td>
<td>Basal ganglia</td>
<td>More wide based, Less stooped, Relatively preserved arm swing</td>
<td></td>
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<tr>
<td>Strategic vascular lesion</td>
<td>Putamen</td>
<td>Vascular</td>
<td></td>
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<td></td>
<td>Globus pallidus</td>
<td>Vascular</td>
<td></td>
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<tr>
<td></td>
<td>Thalamus</td>
<td>Vascular</td>
<td></td>
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<tr>
<td></td>
<td>Dorsal mesencephalon</td>
<td>Vascular</td>
<td></td>
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<tr>
<td>Normal pressure hydrocephalus</td>
<td>Frontostriatal (periventricular)</td>
<td>Vascular</td>
<td></td>
</tr>
<tr>
<td>Drug-induced parkinsonism</td>
<td>Basal ganglia (postsynaptic)</td>
<td>Vascular</td>
<td></td>
</tr>
</tbody>
</table>

**Vascular causes**

**Neurodegenerative**

**NPH**

*Lancet Neurol 2007; 6: 63–74*
Spastic gait disorders

- Spasticity of the arm and leg on one side produces the characteristic clinical picture of a **spastic hemiparesis**

- The arm - adducted, internally rotated at the shoulder, and flexed at the elbow, with pronation of the forearm and flexion of the wrist and fingers

- The leg is slightly flexed at the hip and extended at the knee, with plantar flexion and inversion of the foot
Spastic gait disorders

• The swing phase - slight lateral flexion of the trunk toward the unaffected side and hyperextension of the hip on that side to allow slow **circumduction** of the stiffly extended paretic leg as it is swung forward from the hip, dragging the toe or catching it on the ground beneath

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Scuffing toe
Medial side of shoe worn out
Spastic hemiparesis gait
Spastic gait disorders

- **Etiology:**
  - Spastic hemiparesis → determine the site of corticospinal tract that involved
Spastic paraparesis

- Spastic of both legs
- The legs are stiffly extended at the knees, plantar flexed at the ankles, and slightly flexed at the hips
- Both legs circumduct and the toes of the plantar flexed feet catch on the floor with each step
- Scissors gait
- Lesion in spinal cord, ALS
Scissors gait
Myopathic gait

- **Waddling gait or myopathic gait**: Weakness of proximal leg and hip girdle muscles interferes with the stabilization of the pelvis and legs on the trunk during all phases of the gait cycle.
- Failure to stabilize the pelvis produces exaggerated rotation of the pelvis with each step.
- An exaggerated lumbar lordosis occurs.
- *(Trendelenburg sign)*
Waddling gait or myopathic gait
Waddling gait or myopathic gait

• **Etiology**: muscle weakness at limb girdle, proximal muscle weakness
  – Myopathy: hypothyroidism, hyperthyroidism, polymyositis
Neurogenic gait

• Muscle weakness of peripheral nerve origin as in a peripheral neuropathy
• Typically affects distal muscles of the legs and results in a steppage gait
• The patient lifts the leg and foot high above the ground with each step because of weakness of ankle dorsiflexion and footdrop
Steppage gait
Neurogenic gait

- **Etiology**: peripheral nerve origin, as in a peripheral neuropathy eg. Foot drop
Cerebellar ataxic gait
Cerebellar ataxic gait

- Midline cerebellar structures lesions - vermis, and anterior lobe
- Loss of truncal balance, increased body sway, and dysequilibrium

- Wide-based gait, the legs are stiffly extended and the hips slightly flexed to crouch forward and minimize truncal sway
Cerebellar ataxic gait
Cerebellar ataxic gait

• Patients with anterior lobe atrophy develop a 3 Hz anteroposterior sway of the trunk and a rhythmic truncal and head tremor (titubation).

• This combination of truncal gait ataxia and truncal tremor is characteristic of some late-onset cerebellar degenerations affecting the anterior lobe.

• Midline cerebellar pathologies also include masses, paraneoplastic syndromes, and malnutrition in alcoholism.
Cerebellar ataxic gait

- Lesions of the cerebellar flocculonodular lobe (the vestibulocerebellum) exhibit multidirectional body sway, dysequilibrium, and severe impairment of body and truncal motion.

- Standing and even sitting can be impossible.
- When lying down → the heel-to-shin test normal and upper limb function may be relatively preserved.
Cerebellar ataxic gait

- Cerebellar hemispheres
- Decomposition of normal leg movement
- Steps are irregular and variable in timing (*dyssynergia*), length, and direction (*dysmetria*)
- Dysdiadochokinesia
- Ipsilateral limb ataxia
- Little postural instability or truncal imbalance
Cerebellar ataxic gait

- Exacerbated by the **rapid postural adjustments** needed to change direction, turn a corner, or avoid obstacles and when stopping or starting to walk
- Minor support, such as holding the patient’s arm during walking, and **visual compensation** help the patient with a cerebellar ataxia reduce body sway
- **Eye closure may heighten anxiety about falling** and increase body sway but not to the extent observed in a sensory ataxia
Sensory ataxic gait

• Loss of proprioceptive input from the lower limbs → deprives
  – The position of the legs and feet in space
  – The progress of ongoing movement
  – The state of muscle contraction
  – and finer details of the texture of the surface on which the patient is walking

• Esp. walking on uneven surfaces
Sensory ataxic gait

• Wide-based gait and advance cautiously, taking slow steps under visual guidance
• During walking, the feet are thrust forward with variable direction and height
• The sole of the foot strikes the floor forcibly with a slapping sound (**slapping gait**)
• Romberg’s sign positive
Sensory ataxic gait

• **Etiology:** Lesions at any point in the sensory pathways that interrupt large-diameter proprioceptive afferent fibers

• Peripheral neuropathies, posterior root or dorsal root ganglionopathies, and dorsal column of spinal cord lesions are typical etiologies
Dyskinetic gait

- Dyskinesias includes all involuntary movements or postures—eg, chorea or dystonia

- Dyskinesias can contribute to falls by causing excessive trunk movements
Dyskinetic gait

• Might be absent during clinical examination because of their fluctuating character or because patients suppress them intentionally.

• May be task-specific: For example, patients may have severe gait impairment due to leg dystonia, but can easily walk backwards or even run.

• Misinterpreted as a psychogenic sign.
Dyskinetic gait
Dyskinetic gait
Dyskinetic gait

• **Etiology:** early onset Parkinson’s disease presenting with a foot dystonia while walking

• **Idiopathic torsion dystonia**

• Retrocollis in patients with **progressive supranuclear palsy**

• Antecollis or a Pisa syndrome (severe and persistent lateroflexion of the trunk) in patients with **multiple system atrophy**

• Asymmetrical arm or leg dystonia during walking in patients with **corticobasal degeneration**
Frontal lobe gait disorders

- Interruption of connections between the frontal lobes and other cortical and subcortical structures
- Predominantly wide-based ataxic gait to an akinetic-rigid gait with slow, short steps and a tendency to shuffle
Frontal lobe gait disorders

- Shuffling gait
- Hesitation and freezing (ignition failure)
- Arm swing is normal or even exaggerated, \(\rightarrow\) “military twostep” gait
- Inadequate synergies
- P.E. \(\rightarrow\) normal voluntary upper limb and hand movements and a lively facial expression
Frontal lobe gait disorders

- “lower half parkinsonism” is commonly seen in diffuse cerebrovascular disease
- Misdiagnosed as Parkinson’s disease

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>PARKINSON’S DISEASE</th>
<th>SYMPTOMATIC PARKINSONISM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posture</td>
<td>Stooped (trunk flexion)</td>
<td>Stooped or upright (trunk flexion/extension)</td>
</tr>
<tr>
<td>Stance</td>
<td>Narrow</td>
<td><strong>Often wide-based</strong></td>
</tr>
<tr>
<td>Initiation of walking</td>
<td>Start hesitation</td>
<td>Start hesitation, magnetic feet</td>
</tr>
<tr>
<td>Steps</td>
<td>Small, shuffling</td>
<td>Small, shuffling</td>
</tr>
<tr>
<td>Stride length</td>
<td>Short</td>
<td>Short</td>
</tr>
<tr>
<td>Freezing</td>
<td>Common</td>
<td>Common</td>
</tr>
<tr>
<td>Leg movement</td>
<td>Stiff, rigid</td>
<td>Stiff, rigid</td>
</tr>
<tr>
<td>Speed</td>
<td>Slow</td>
<td>Slow</td>
</tr>
<tr>
<td>Festination</td>
<td><strong>Common</strong></td>
<td>Rare</td>
</tr>
<tr>
<td>Arm swing</td>
<td>Minimal or absent</td>
<td>Reduced or excessive</td>
</tr>
<tr>
<td>Heel-toe walking</td>
<td>Normal</td>
<td>Poor (truncal ataxia)</td>
</tr>
<tr>
<td>Postural reflexes</td>
<td>Preserved in early stages</td>
<td>Absent at early stage</td>
</tr>
<tr>
<td>Falls</td>
<td>Late (forward, tripping)</td>
<td><strong>Early and severe</strong> (backward, tripping or without apparent reason)</td>
</tr>
</tbody>
</table>

TABLE 24-5 Summary of Clinical Features Differentiating Parkinson’s Disease from Symptomatic Parkinsonism in Patients with an Akinetic-Rigid Gait Syndrome
Frontal lobe gait disorders

- When disease progresses → magnetic gait – difficult to initiate a step
- Need sensory cues
- Able to move the legs with greater facility when seated or lying supine than when standing
- Severe balance impairment (no rescue reactions with the pull test; “falling like a log”)
- Impaired truncal mobility in advanced stage
- Paratonic (gegenhalten) rigidity of the arms and legs is common
Frontal lobe gait disorders

- Etiology: bilateral frontal lobe lesion
- Frontal lobe tumors (glioma or meningioma)
- Anterior cerebral artery infarction
- Obstructive or communicating hydrocephalus (especially normal pressure hydrocephalus)
- Diffuse cerebrovascular disease (multiple lacunar infarcts and Binswanger’s disease) all produce a similar disturbance of gait
Non-neurological gait disorders

- Psychogenic gait disorders
- Antalgic gait
- Cautious and careless gaits
Cautious and careless gaits

• Cautious gaits
• Fear of falling
• Loss of confidence when walking
• Move slowly, with a wide base and short strides, with little movement of the trunk, while the knees and elbows are bent
• Improve dramatically when support provided
• Neurological examination is completely normal
Cautious and careless gaits

- **Careless gaits**
- Counterpart of the cautious gait
- Patients seem overly confident and walk inappropriately fast, perhaps because of lack of insight or frontal-lobe disinhibition
- Example
  - PSP
  - Huntington’s disease
  - Delirium
Psychogenic gait disorders

• Suspicious highest in younger patients
• But can occur in elderly
• Not compatible with known gait patterns and they can take unusual forms
• Not to miss underlying organic disease, in particular **frontal-lobe dysfunction**
Psychogenic gait disorders

**Panel: Features suggestive of a psychogenic gait disturbance**

**Suggestive features**
- Incongruous with known gait disorders
- Bizarre presentation
- Variable, inconsistent pattern
- Non-physiological pattern
- Rare falls or injuries*
- Abrupt onset
- Extreme slowness
- Unusual or uneconomic posture
- Exaggerated effort
- Sudden buckling of the knees

**Associated features**
- Incongruous affect (belle indifference)
- Secondary gain
- Prior history of psychiatric disease†

*Striking exemptions with sometimes severe injuries have been described.†Rare, but diagnostic yield is higher with an intensive interview.
Antalgic gait

- Painful gait
- Reduced stance phase on affected limb
- **Limps and gait difficulties** caused by joint disease, bone injury, or soft tissue injury are not usually accompanied by muscle weakness, reflex change, or sensory loss
- **Limitation of the range of movement** at the hip, knee, or ankle joints leads to short steps with a fixed leg posture
Antalgic gait

- Affected limb → shorter stance phase
- Unaffected limb → shorter swing phase
  → ↓ step length
- ↓ walking velocity
Thank you for your attention.