Special Communication

Understanding Gait, Its Neurophysiologic Control and Systematic Localisation of Gait Abnormalities

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Abstract

Gait analysis is a crucial diagnostic tool in neurology and rehabilitation, reflecting the intricate coordination between the central and peripheral nervous systems, musculoskeletal structures, and sensory feedback mechanisms. This article integrates the neurophysiological basis of gait, emphasising the role of central pattern generators in the spinal cord and the mesencephalic locomotor region in regulating initiation, speed, and adaptability (1). Gait disorders result from disruptions in various neural structures, including the cerebellum, basal ganglia, motor and sensory pathways, and the peripheral nervous system (2). Functional gait disorders, characterised by inconsistent or exaggerated patterns, require differentiation from organic conditions (3). This article also highlights diagnostic tools, including motion capture systems, electromyography, and wearable sensors, as well as therapeutic interventions, such as physical therapy, pharmacological treatments, and emerging rehabilitative technologies (4).

Keywords: gait, central nervous system, abnormal gait, gait cycle, gait disorder, neural control

Introduction

Human gait is a complex, coordinated motor activity that integrates multiple neural structures, including the cerebellum, basal ganglia, brainstem, spinal cord, and peripheral nerves (5). Disruptions in these systems result in pathological gait patterns, which provide essential clues for diagnosing neurological or musculoskeletal disorders (6). The gait cycle, divided into stance (60%) and swing (40%) phases, relies on rhythmic motor patterns generated by central pattern generators and modulated by descending supraspinal inputs (7).

Neurophysiology of Gait

Normal Gait Cycle

The human gait cycle consists of two main phases – stance and swing. Each phase is further divided into specific subphases that are essential for efficient locomotion (2).

Stance Phase (60% of Gait Cycle)

i) Initial contact: The heel strikes the ground, stabilising the limb (6).

ii) Loading response: Weight transfers onto the limb, requiring knee flexion control by the quadriceps (4).

iii) Midstance: The body advances over the supporting limb, with the gastrocnemius and soleus stabilising the tibia.

iv) Terminal stance: The heel rises off the ground, propelling forward with assistance from the plantar flexors.

v) Pre-swing: The limb prepares for the swing phase using the rectus femoris and iliopsoas for hip flexion.

Swing Phase (40% of Gait Cycle)

i) Initial swing: The foot lifts off the ground, clearing obstacles via dorsiflexion (1).

ii) Midswing: The thigh advances forward with continued knee and hip flexion.

iii) Terminal swing: The hamstrings decelerate limb movement, preparing for initial contact again.

Proper coordination between these phases ensures smooth locomotion, relying on neural input from the cerebellum, basal ganglia, and sensory pathways (5). Figure 1 illustrates the gait cycle divided into stance (60%) and swing (40%) phases. Key lower limb muscles active during each subphase are highlighted, including the gluteus maximus, quadriceps, hamstrings, tibialis anterior, triceps surae, and hip flexors. Arcs indicate periods of double- and single-limb support.
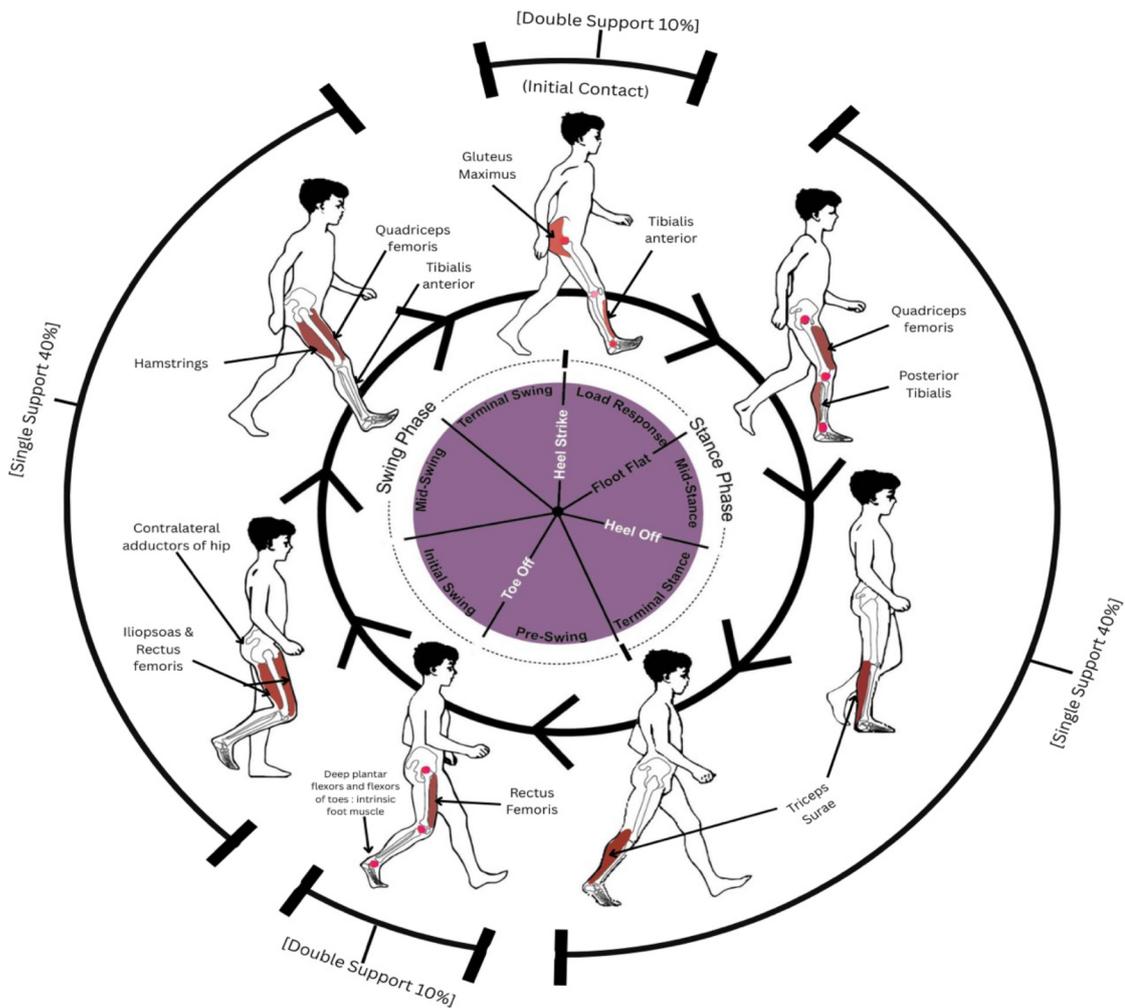

Figure 1. Phases of gait cycle and key muscle activation

Normal Gait Cycle Requirements

Central Pattern Generators (CPGs)

Located in the spinal cord, these neural networks produce rhythmic locomotor patterns essential for walking (4).

Mesencephalic Locomotor Region (MLR)

Regulates gait initiation and speed via reticulospinal pathways (1).

Basal Ganglia and Cerebellum

Modulate movement coordination, amplitude, and postural stability (5).

Sensory Feedback Systems

Proprioceptive, visual, and vestibular inputs ensure real-time adjustments for balance and environmental adaptation (2).

Key neural structures involved in gait control:

i) Higher centres (PFC, SMA, M1) initiate and plan movement.

ii) Basal ganglia modulate automaticity and selection.

iii) The cerebellum ensures coordination and adaptation.

iv) MLR initiates rhythmic patterns via the spinal CPGs, which generate and coordinate locomotor output.

Pathophysiology of Gait Abnormalities and Localisation

Gait abnormalities arise from dysfunction in neural pathways, leading to characteristic movement patterns that provide critical diagnostic insights. Understanding these disruptions enhances the ability to localise the underlying pathology.

Cerebellar Gait Disorders – Ataxic Gait

Caused by lesions in the cerebellum, particularly the spinocerebellum and vestibulocerebellum, this gait is wide-based and unsteady, with irregular step timing (7). Spinocerebellar damage results in dysmetria and impaired limb coordination, while vestibulocerebellar lesions cause significant truncal instability and swaying (8).
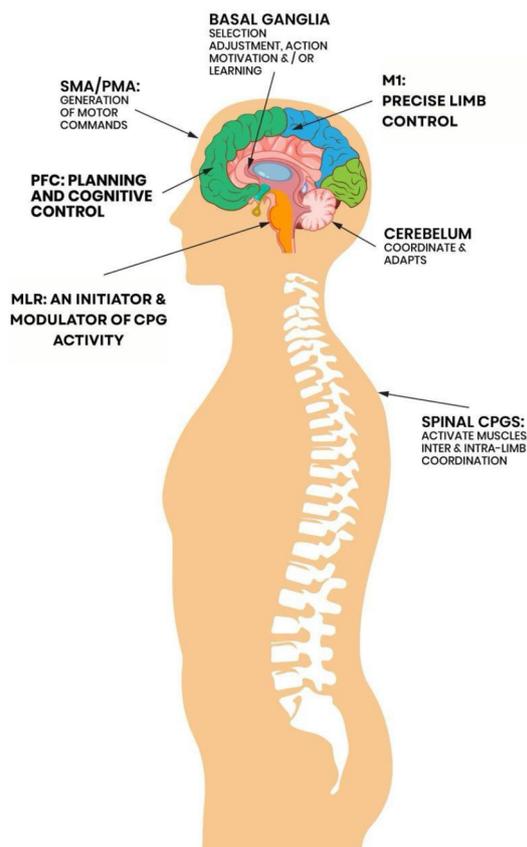

Figure 2. Neural control of gait

Basal Ganglia Dysfunction and Parkinsonian Gait

i) Parkinsonian gait: Due to substantia nigra degeneration, this gait is characterised by rigidity, shuffling steps, festination, and freezing episodes, indicative of basal ganglia dysfunction (9). Loss of dopaminergic modulation leads to decreased stride length and difficulty initiating movement.

ii) Hyperkinetic gaits: Disorders such as Huntington's disease and dystonia lead to athetotic, choreic, and dystonic gait patterns, manifesting as involuntary excessive movements disrupting normal walking (10).

Motor and Sensory Tract Lesions

i) Spastic gait: Results from corticospinal tract damage, such as in multiple sclerosis or stroke, causing hypertonia and scissoring of the legs (11). Impaired voluntary control results in stiff, jerky movements with limited knee flexion.

ii) Hemiparetic gait: Typical in stroke patients, this gait involves unilateral weakness, circumduction of the affected limb, and reduced arm swing, indicative of upper motor neuron damage (12).

iii) Sensory ataxia: Due to dorsal column dysfunction, patients rely on visual feedback, leading to a high step gait with heavy heel strikes. A positive Romberg sign confirms proprioceptive deficits (13).

iv) Vestibular ataxia: Vestibular system damage causes veering gait, worsened by eye closure and characterised by imbalance and falling towards the lesion side (14).

Peripheral Neuropathies and Musculoskeletal Gaits

i) Steppage gait: Associated with peroneal nerve palsy or severe neuropathy, causing foot drop. Patients lift the affected leg high to prevent dragging (5).

ii) Waddling gait: Seen in muscular dystrophies, characterised by excessive trunk movement and difficulty stabilising the pelvis due to proximal muscle weakness (15).

Functional Gait Disorders

These disorders present inconsistent patterns and exaggerated movements without a clear neurological basis. Often psychogenic in origin, they include sudden knee buckling, hesitation, and improved gait when distracted (5).

Figure 3 summarises common pathological gait types, detailing the affected gait phases, underlying neurophysiological mechanisms, and typical anatomical localisations. Gait patterns include hemiplegic, scissoring, Parkinsonian, cerebellar and sensory ataxia, steppage, waddling, apraxic, and functional gait, providing a structured framework for clinical localisation.

Management Strategies

Pharmacological Therapy

Dopaminergic medications for Parkinsonian gait and muscle relaxants for spasticity (16).

Physical Therapy and Rehabilitation

Strength, balance, and coordination training (17).

Assistive Devices

Canes, walkers, and orthotics to aid mobility (18).

Emerging Technologies

Robotic-assisted gait training and neuromodulation to improve rehabilitation outcomes (1).

As a supplement to this article, we provide a link to a video of gait examination and several common forms of gait pathology encountered in our practice (duration 18 minutes).

Available at YouTube: https://www.youtube.com/watch?v=jv8lfuAIVhU

Conclusion

A structured understanding of gait pathophysiology aids in diagnosing and managing movement disorders. Advances in diagnostic tools and therapies enhance patient outcomes by providing targeted interventions. A multidisciplinary approach that incorporates neurology, physiotherapy, and rehabilitative technologies optimises functional mobility and quality of life.

Acknowledgements

GAIT TYPE	AFFECTED GAIT PHASE(S)	NEUROPHYSIOLOGICAL BASIS	TYPICAL LOCALIZATION
HEMIPLEGIC SPASTIC	SWING PHASE (INADEQUATE FLEXION); INITIAL CONTACT IS TOE-FIRST	UMN LESION → EXTENSOR HYPERTONIA, FLEXOR WEAKNESS; LOSS OF RECIPROCAL INHIBITION CAUSES STIFF CIRCUMDUCTING LEG	CONTRALATERAL CORTICOSPINAL TRACT (MOTOR CORTEX OR INTERNAL CAPSULE) LESION (E.G. STROKE)
DIPLEGIC SCISSORS	STANCE & SWING (BILATERAL): LEGS ADDUCTED, CROSSING DURING SWING; NARROW BASE STANCE	BILATERAL UMN LESION → ADDUCTOR SPASTICITY AND EXTENSOR HYPERTONIA; IMPAIRED LIMB SEPARATION CAUSES SCISSORING	BILATERAL PERIVENTRICULAR LESIONS (E.G. CEREBRAL PALSY) OR SPINAL CORD (PARAPARESIS FROM MYELOPATHY)
PARKINSONIAN	INITIATION AND STRIDE LENGTH (SHORT SHUFFLING STEPS, REDUCED FOOT LIFT); TURNING DIFFICULTY, POSSIBLE FREEZING	BASAL GANGLIA DYSFUNCTION (DOPAMINE LOSS) → BRADYKINESIA, IMPAIRED GAIT INITIATION; INADEQUATE SCALING OF MOTOR OUTPUT (SMALL STRIDES)	SUBSTANTIA NIGRA / BASAL GANGLIA (E.G. PARKINSON'S DISEASE); FRONTAL-SUBCORTICAL CIRCUITS IN LOWER-BODY PARKINSONISM
CEREBELLAR ATAXIA	ALL PHASES (POORLY COORDINATED): WIDE-BASED STANCE; ERRATIC FOOT PLACEMENT IN SWING, UNSTEADY WEIGHT TRANSFER IN STANCE	CEREBELLAR DYSFUNCTION → DYSMETRIA AND DYSSYNERGIA OF GAIT; INABILITY TO STABILIZE TRUNK AND TIME MOVEMENTS (INCOORDINATION WITHOUT WEAKNESS)	CEREBELLAR VERMIS OR PATHWAYS (E.G. CEREBELLAR DEGENERATION, STROKE OR TUMOR IN CEREBELLUM)
SENSORY ATAXIA	STANCE (UNSTABLE, STOMPING FOOT STRIKE); SWING (HIGH-STEPPING TO ENSURE CLEARANCE)	LOSS OF PROPRIOCEPTIVE FEEDBACK → PATIENT USES VISUAL INPUT AND EXAGGERATED MOVEMENTS (FOOT SLAP ON CONTACT); POSITIVE ROMBERG SIGN	PERIPHERAL LARGE-FIBER NEUROPATHY OR DORSAL COLUMN LESION (E.G. B12 DEFICIENCY, TABES DORSALIS)
STEPPAGE NEUROPATHIC	SWING (FOOT DROP → HIGH HIP/KNEE LIFT); INITIAL CONTACT (FOOT SLAP OR TOE-FIRST LANDING)	PERIPHERAL NERVE LMN LESION → DORSIFLEXOR WEAKNESS (FOOT DROP); NORMAL RHYTHM BUT ABNORMAL AMPLITUDE (EXCESSIVE HIP/KNEE FLEXION TO COMPENSATE)	PERONEAL NERVE PALSY OR L5 RADICULOPATHY; DISTAL POLYNEUROPATHY IF BILATERAL
WADDLING MYOPATHIC	STANCE (TRENDELENBURG SIGN – PELVIS DROPS ON CONTRALATERAL SWING LEG; TRUNK LURCH TOWARD STANCE LEG)	HIP ABDUCTOR (GLUTEUS MEDIUS) WEAKNESS → INABILITY TO STABILIZE PELVIS; PROXIMAL MUSCLE WEAKNESS CAUSES COMPENSATORY TRUNK SWAY AND LORDOSIS	HIP GIRDLE MUSCLES (E.G. MUSCULAR DYSTROPHY, MYOPATHY) OR NEUROMUSCULAR JUNCTION; BILATERAL CONGENITAL HIP ISSUES
FUNCTIONAL APRAXIA	INITIATION (START HESITATION, FREEZING); STRIDE (SHORT, SHUFFLING STEPS WITH BROAD BASE)	IMPAIRED CORTICAL PLANNING AND INITIATION (FRONTAL LOBES) → "GAIT IGNITION FAILURE"; INTACT MOTOR POWER BUT INABILITY TO SEQUENCE MOVEMENT; OFTEN NORMAL ARM SWING	FRONTAL LOBES OR FRONTAL-WHITE MATTER (E.G. NORMAL PRESSURE HYDROCEPHALUS, DIFFUSE SUBCORTICAL ISCHEMIA)

Figure 3. Classification of abnormal gait types by phase, neurophysiology, and localisation

Ethics of Study

None.

Conflict of Interest

None.

Funds

None.

Authors' Contributions

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