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The Impact of Gait Disorders on Executive Functions in Neurological Patients: A Literature Review

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Abstract---*This is a literature review covering the intricate and two-way association between gait disorders and executive functioning deficits in individuals with neurological disorders. The review begins by defining executive functions, followed by a discussion of its fundamental components (inhibition, working memory, cognitive flexibility), and finally, the neural foundation of EF is outlined, focusing on the frontal lobes and related brain networks. A typology of neurological gait disorders (e.g., Parkinsonian, hemiplegic, ataxic) is then described, which is then correlated with their respective etiologies. The cognitive-motor interference model of gait-executive functions interaction and the shared neural resource theories are explored as frameworks explaining the phenomenon, and the dual-task paradigm as a methodological instrument is considered specifically. In the review, the authors examined*

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how this interaction manifests in neurological diseases, including Parkinson's, stroke, and multiple sclerosis, and noted the peculiarities and mechanisms in each instance. Lastly, the clinical implications of this interaction on everyday functioning, risk of falls, and quality of life are addressed, along with what they mean regarding rehabilitation approaches, with a particular focus on integrated motor-cognitive interventions, including dual-task training and novel technologies (e.g., exergaming, virtual reality). The review concludes with a summary that synthesizes major evidence and identifies gaps in the existing literature, as well as outlines future research directions.
Keywords---*Executive Functions, Gait Disorders, Neurological Patients.*

Introduction

Executive function deficits and gait disorders are frequent and frequently co-morbid clinical features of a broad assortment of neurological diseases. Such coexistence is not accidental but implies that there are more profound interactions and, possibly, shared or interacting neuropathological mechanisms. Traditionally, gait was assumed to be a highly automated motor task, which needed little central processing (Sheridan et al., 2003). Modern studies have, however, contradicted this observation, noting that higher cognitive functions, specifically executive functions, play a vital role in gait control and adjustment, especially in challenging environments or when there is neurological damage (Sheridan et al., 2003; Montero-Odasso et al., 2012).

Dementia and gait disorders are common conditions that are independent predictors of falls and have a profound impact on independence, quality of life, and healthcare expenditures (Montero-Odasso et al., 2012). The connection between the two areas is essential since most neurological disorders like Parkinson's disease, stroke, and multiple sclerosis tend to destroy the brain structures and circuits that lead to impaired motor control as well as executive functions. As a result, brain damage may present itself both as a gait disorder and an executive function deficit. Moreover, the difficulty of walking might demand more cognitive resources in compensation, which puts additional pressure on already compromised executive functions. On the other hand, gait performance may also deteriorate due to executive functions impairments, including attention, planning, or inhibition problems, which make gait less safe and efficient (Axer et al., 2010).

This literature review will synthesize and critically examine available literature regarding the effect of gait disorders on executive functions in people with neurological issues. The definitions and neural foundations of executive functions, forms of gait disorders, theoretical frameworks to describe their interplay, their presentation in particular neurological diseases, and their relevance to rehabilitation interventions will be discussed. Characterizing this complex interaction is crucial in developing comprehensive diagnostic and therapeutic strategies that target both realms concurrently, with the ultimate aim of enhancing the functionality and quality of life for neurological patients (Ardila, 2008).

1. Definition, Components, and Neural Basis of Executive Functions

EFs constitute a pillar of human cognitive structure that allows flexible and goal-oriented conduct. It is important that we understand their nature, components, and neural foundation so that we can be able to interpret how neurological diseases affect them.

What are Executive Functions (EFs)?

Executive functions refer to a group of cognitive abilities of a higher level that regulate and coordinate other cognitive abilities and behaviors (UCSF Memory and Aging Center, n.d.). These are cognitive processes that are brought into action every day to resolve issues, develop courses of action, and process emotions (Cleveland Clinic, n.d.-a). They resemble a "chief executive officer" of the brain, overseeing and guiding different mental "departments" in such a way that the organism could successfully proceed towards its aims (UCSF Memory and Aging Center, n.d.). It should be stressed that EFs are conscious activities and are not confused with automatic or instinctual reactions (EBSCO Research Starters, 2024). Our executive system plays a huge role in the way we structure our lives, create a plan of action, and see it through (UCSF Memory and Aging Center, n.d.).

Essential Elements of Executive Functions

EFs do not represent a single entity but rather a complex of cognitive processes that are independent of each other yet closely related. They may be roughly divided into organizational and regulatory skills (UCSF Memory and Aging Center, n.d.). Organizational skills encompass the collection of information and its organization to be evaluated, whereas regulatory skills involve assessing the existing information and molding reactions to the surroundings (UCSF Memory and Aging Center, n.d.).

There are three fundamental elements of EFs that the scientific community has outlined (EBSCO Research Starters, 2024; Carroll & Simpson, 2022).

Inhibition (Inhibitory Control): This element is defined as the capacity of self-control, directing attention to challenging stimuli, at the same time disapproving unnecessary or distracting information (EBSCO Research Starters, 2024). It involves holding back prepotent responses, i.e., the skill of not reacting immediately but thinking consciously about the response one gives (EBSCO Research Starters, 2024; Carroll & Simpson, 2022).

Inhibition plays a key role in disciplined learning, focusing on a task and observing ethical principles (EBSCO Research Starters, 2024).

Lezak enlists response inhibition as four primary components of EFs, which enables the disregard of irrelevant senses and overcomes primary reflexes (Sheridan et al., 2003).

Working Memory: Working memory refers to the capability to temporarily store and at the same time mentally operate on several pieces of information (EBSCO Research Starters, 2024). It is not identical with mere short-term memory because it is not solely a storage; active processing and utilisation of this information are also included (EBSCO Research Starters, 2024). It plays a central role in processing complex concepts, chronological orders (e.g., in a story), sentence comprehension, and carrying out directions (EBSCO Research Starters, 2024).

Cognitive Flexibility (Shifting): This element is about the capacity of adjusting thought and behavior to varying circumstances or requirements (EBSCO Research Starters, 2024). It also involves being able to move between various tasks or frames of mind (task switching), to weigh up various solutions to a problem, and to shift perspective (Carroll & Simpson, 2022). Creativity is allied to cognitive flexibility, which to a large extent involves the inhibition as well as working memory (EBSCO Research Starters, 2024).

In addition to these three main components, other aspects of EFs frequently noted in the literature that are thought to be of importance include planning, i.e., the ability to determine and set the steps needed to reach a goal (Sheridan et al., 2003), problem-solving (UCSF Memory and Aging Center, n.d.), abstract thinking (UCSF Memory and Aging Center, n.d.), and performance/response monitoring, which enables the comparison of ongoing actions with an internal plan and the recognition and repair of errors (Sheridan et al., 2003)

The maturation of these functions also occurs hierarchically, so working memory usually develops first, then inhibitory capacity, and lastly cognitive flexibility, which depends on the former two (EBSCO Research Starters, 2024; Carroll & Simpson, 2022).

Neural Foundation of Executive Functions

The performance of EFs is strictly connected to the work of the frontal lobes of the brain, more specifically the prefrontal cortex (PFC) (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.). The PFC is the evolutionarily most recent and relatively largest region of the human brain compared with other primates, making up approximately 40 percent of the entire brain volume (UCSF Memory and Aging Center, n.d.). It is positioned in the frontal lobes' anterior section, immediately above the orbits. It is the region that is not fully developed until the end of the development of an individual (UCSF Memory and Aging Center, n.d.).

Nevertheless, EFs are not restricted to the prefrontal cortex. An extensive network of neurons that involves communication between the PFC and other cortical areas, including the parietal lobes, and subcortical structures, predominantly the basal ganglia and the thalamus, supports them (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.). It appears that the frontal lobes are more concerned with some aspects of self-regulation (inhibition and self-awareness) in their anterior parts, whereas logical thinking processes are associated with the dorsal parts (Sheridan et al., 2003). The activation of several areas highlights the distributed aspect of EFs, with various aspects being dependent on slightly different, but overlapping neural networks. This network structure provides the basis for why different points of damage within this network can result in various profiles of executive dysfunction (Montalvan et al., 2020).

Executive Functions Disorders

As central and broadly neural structures, EFs are susceptible to a broad variety of neurological and psychiatric disorders. Executive system damage, be it through traumatic brain injury, stroke, neurodegenerative (e.g., frontotemporal dementia, Parkinson -s disease), or developmental disorders, typically causes a complex of impairments and not the loss of a single skill (UCSF Memory and Aging Center, n.d.).
Common manifestations of executive dysfunction include (UCSF Memory and Aging Center, n.d.):

- Difficulty organizing and planning.
- Problems initiating and maintaining activities (problems with initiative).
- Inability to perform multiple tasks simultaneously (multitasking).
- Reduced verbal fluency.
- Difficulty processing, storing, and/or retrieving information.
- Emotional lability and mood swings.
- Reduced interest in activities.
- Socially inappropriate behavior and lack of empathy.
- Inability to learn from the consequences of past actions.
- Difficulty with abstract concepts.
- Lack of awareness or denial of behavioral problems.

The prolonged developmental trajectory of the frontal lobes and EFs makes them particularly vulnerable not only to acquired damage but also to developmental disorders and the degenerative processes associated with aging (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.). Understanding the complexity and vulnerability of EFs is fundamental to appreciating why neurological diseases so often affect these functions and why the consequences can be so pervasive in patients' daily lives.

Table 1
Core Executive Functions, Definitions, and Associated Brain Regions

Executive Function	Brief Definition	Key Involved Brain Regions
Working Memory	Ability to temporarily hold and manipulate information necessary for complex cognitive tasks (EBSCO Research Starters, 2024).	Dorsolateral prefrontal cortex (DLPFC), parietal cortex (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.).
Inhibition/Inhibitory Control	Ability to suppress inappropriate or irrelevant thoughts, impulses, or responses to achieve a goal (EBSCO Research Starters, 2024; Carroll & Simpson, 2022).	Ventromedial prefrontal cortex (vmPFC), anterior cingulate cortex (ACC), right inferior frontal gyrus (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.).
Cognitive Flexibility	Ability to switch between different mental sets, strategies, or tasks, and adapt to new demands (EBSCO Research Starters, 2024; Carroll & Simpson, 2022).	Dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC), parietal cortex, basal ganglia (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.).
Planning	Ability to organize a sequence of actions to achieve a future goal (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.).	Prefrontal cortex (especially dorsolateral and anterior polar regions) (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.).
Performance Monitoring	Ability to monitor behavior, detect errors, and adjust actions accordingly (Sheridan et al., 2003).	Anterior cingulate cortex (ACC), pre-supplementary motor area (pre-SMA) (Sheridan et al., 2003).
Problem Solving	Ability to identify a problem, develop and implement solutions, and evaluate outcomes (UCSF Memory and Aging Center, n.d.; Cleveland Clinic, n.d.-a).	Prefrontal cortex, parietal cortex (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.).

Sources: (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.; Cleveland Clinic, n.d.-a; EBSCO Research Starters, 2024; Carroll & Simpson, 2022)

2. Gait Disorders in Neurological Patients: Typology and Etiology

Gait is an apparent simple daily activity, yet it is an extremely complicated motor activity that involves the perfect collaboration of numerous systems of the human body. This harmonious activity is frequently impaired by neurological diseases, causing different gait disorder patterns.

General Gait Disorders Characteristics

An abnormal gait is called any alteration in the personal, normal walking pattern of an individual (Cleveland Clinic, n.d.-b). As the normal gait requires the complex and coordinated interaction of components of the nervous system (central and peripheral), the musculoskeletal system, and the cardiorespiratory system, any pathology of these structures is a potential cause of a gait disorder (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025).
The prevalence of these disorders is especially high in the geriatric community, where they contribute to a high likelihood of falling and other complications (Loyola Medicine, 2025).

The most typical forms of abnormal gait are limping, toe dragging when walking, shuffling, small-stepped walking, inability to hold body weight, and the overall inability to coordinate movements (Cleveland Clinic, n.d.-b). The changes not only impact mobility but also may contribute to muscle weakness, pain, and a critical decline in the independence of the person (Cleveland Clinic, n.d.-b).

2.2 Neurological Gait Disorders Classification and Description

Many types of gait disorders can be caused by neurological diseases, and each of them has specific kinematic patterns that can serve as a diagnostic hint to the pathology. Close observation and study of the kind of gait disorder may give an idea of the lesion in the nervous system. The most frequent forms of neurological etiology are as follows:

Hemiplegic Gait / Spastic Gait: This is a gait where one side of the body is involved. The patient also normally keeps the upper limb of the involved side in a flexion, adduction, and internal rotation position. There is extension of the lower limb on the same side and plantar flexion of the foot and toes. When walking, the patient scuffs the affected leg in a semicircular way (circumduction) primarily because of the weakness in distal muscles (foot drop) and spasticity (hypertonia) of lower limb extensor muscles (Loyola Medicine, 2025; Stanford Medicine 25, n.d.). In bereavement cases of hemiparesis, the only abnormalities can be the loss of normal arm swing and some circumduction (Stanford Medicine 25, n.d.). Such gait is the most typical of patients that suffered a stroke (Stanford Medicine 25, n.d.), though it may also arise when one has multiple sclerosis or any other illness that leads to hemiplegia (Cleveland Clinic, n.d.-b).

Diplegic Gait / Spastic Gait: Diplegic gait represents a bilateral involvement, and spasticity is more often involved in the lower limbs than in the upper ones. When walking, the patient maintains an unusually small base of support, drag both legs, and scrapes the toes on the floor (Stanford Medicine 25, n.d.). Hips and knees are frequently flexed, and ankles are internally rotated, making both sides of the body swing (Loyola Medicine, 2025).

The most obvious is the terrible spasticity of the adductor muscles of the hips, which may result in crossing of the legs at the midline during gait, a condition called scissors gait (Cleveland Clinic, n.d.-b; Stanford Medicine 25, n.d.). Patients can also seem to be walking on their tiptoes (Stanford Medicine 25, n.d.). The gait pattern may be related to bilateral periventricular lesions, as may be seen in cerebral palsy (Cleveland Clinic, n.d.-b; Stanford Medicine 25, n.d.), or related to stroke or brain injury (Loyola Medicine, 2025).

Neuropathic Gait / Steppage Gait / Equine Gait: This gait pattern is a result of a foot drop i.e. lack of active dorsiflexion of the foot. Not to drag the toes on the ground and fall, the patient involuntarily elevates the leg during the swing phase abnormally high, over-flexing the hip and knee (as a horse walks, hence the name equine gait) (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.). When resting, the foot may look floppy (Cleveland Clinic, n.d.-b). In case of unilateral foot drop, the possible causes are peroneal nerve damage (e.g., pressure, trauma, complication of other conditions such as spinal stenosis or herniated disc) or L5 radiculopathy (Cleveland Clinic, n.d.-b; Stanford Medicine 25, n.d.). In case it is bilateral, it can be caused by diseases like amyotrophic lateral sclerosis (ALS), Charcot-Marie-Tooth, or other peripheral neuropathies (e.g., diabetic neuropathy, alcoholic neuropathy) (Loyola Medicine, 2025; Stanford Medicine 25, n.d.)

Myopathic Gait / Waddling Gait: Myopathic gait is a side-to-side motion of the trunk during walking that is similar to the waddle of a duck (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.). This motion is caused by the weakness of the muscles of the pelvic girdle, specifically the hip abductors (gluteus medius and minimus). Weakness of these muscles during single-leg stance results in a decreased pelvis on the side of the

swinging leg (Trendelenburg sign). In case of bilateral weakness, the pelvis will fall on one side with one step and on the other side with the next step, creating the typical sway (Stanford Medicine 25, n.d.). Such a gait is noticed in patients with primary muscle diseases (myopathies). e.g., different types of muscular dystrophy or with conditions that lead to secondary muscle weakness (e.g., spinal muscular atrophy) (Loyola Medicine, 2025; Stanford Medicine 25, n.d.).

Ataxic Gait: Ataxic gait appears as unstable and may involve stumbling, with irregular and uncoordinated steps, it is hard to walk in a straight line and when attempting to walk with the heel of one foot touching the toes of the other (tandem gait) (Cleveland Clinic, n.d.-b). To enhance stability, the patient tends to broaden his/her base of support (i.e. walks with feet apart) (Loyola Medicine, 2025). When in an upright position, they may experience trunk instability, or titubation (Loyola Medicine, 2025). Another common manifestation of cerebellar dysfunction is ataxic gait; this can be caused by cerebellar degeneration, brain trauma, cerebellar stroke, tumors, and toxic causes (e.g., some antiepileptic drugs, alcohol poisoning) (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.). In case of unilateral cerebellar lesion, the patient will fall towards that side when exhibiting an ataxic gait (Stanford Medicine 25, n.d.).

Parkinsonian Gait / Propulsive Gait: This type of gait is seen in Parkinson disease, but it may also be a medication side effect (of antipsychotics, etc.) (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025). The patient assumes a bent posture, and the trunk, neck, and hips are flexed, and the head and neck are forward (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025). Great difficulty in starting to walk (hesitation, start hesitation). When the movement starts, the steps are usually short (brachybasia), sluggish, and shuffling like the patient is trying to catch his center of gravity (festinating gait) (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; COAST Rehab, n.d.). There is decreased or absent arm swing when walking (COAST Rehab, n.d.). They can also experience freezing of gait, particularly at onset, or when turning or when the patient is negotiating narrow passages or obstacles.

Other described gait disorders are antalgic gait caused by pain and characterized by limping to not load the affected limb (Cleveland Clinic, n.d.-b), scissors gait that has been mentioned as a feature of diplegic gait (Cleveland Clinic, n.d.-b), shuffling gait (no lift of feet) (Cleveland Clinic, n.d.-b) and crouching gait (flexion of ankles, knees, and hips) seen in cerebral palsy (Cleveland Clinic, n.d.-)

Gait Disorders Etiology

In most instances, gait disorders represent the consequence of an underlying pathological condition (Loyola Medicine, 2025). The major types of causes are:

Neurological Diseases: It is among the most widespread causes. They encompass a large spectrum of disorders, including Parkinson disease, stroke, multiple sclerosis, cerebral palsy, myopathies, brain injuries, cerebellar lesions (traitor to ataxia), peripheral neuropathies (capable of causing neuropathic gait), normal pressure hydrocephalus, and degenerative diseases of the spinal cord (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; COAST Rehab, n.d.).

*Musculoskeletal Issues***:** Osteoarthritis, rheumatoid arthritis, joint pain, muscle weakness (non-neurological), joint deformities, fractures, and recent lower-limb surgical procedures may considerably change the gait pattern (Cleveland Clinic, n.d.-b; COAST Rehab, n.d.).

Sensory Disorders: Issues with seeing, proprioception (the sense of body position in space), or the vestibular system (which maintains balance and orientation) may cause wobbly gait and a higher possibility of falls (Cleveland Clinic, n.d.-b; COAST Rehab, n.d.).

Age-Related Decline: As a person ages, muscle strength and flexibility, vision and balance deteriorate, and reaction time slows down, all of which increase the likelihood of developing gait disorders in old age (Cleveland Clinic, n.d.-b; COAST Rehab, n.d.).

Other Health Problems and Contributors: These entail cardiovascular diseases that impact blood flow to lower limbs or brain, metabolic disorders, medication side effects (e.g., sedatives, antipsychotics), and psychological causes like fear of falling.

As a complex action, gait is susceptible to the malfunction of many systems. It is sophisticated, relies on the coordinated efforts of neural, musculoskeletal, and sensory processes, and thus is a delicate marker of whole-body health in vulnerable groups, like patients with neurological disease. It is important to determine the exact etiology to manage and treat it.

Table 2
Common Neurological Gait Disorders: Characteristics and Associated Conditions

Type of Gait Disorder	Key Kinematic/Observable Characteristics	Common Associated Neurological Conditions/Causes
Parkinsonian Gait	Stooped posture, bradykinesia, difficulty initiating, small shuffling steps (festination), reduced arm swing, freezing of gait (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; COAST Rehab, n.d.).	Parkinson's disease, drug-induced parkinsonism (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025).
Hemiplegic/Spastic Gait (unilateral)	Semicircular movement of the affected leg (circumduction), flexed and adducted upper limb, spasticity (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).	Stroke, multiple sclerosis, brain injury, brain tumors (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).
Diplegic/Spastic Gait (bilateral)	Narrow base of support, "scissors" movement of the legs (scissoring), walking on tiptoes, flexed hips and knees, spasticity (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).	Cerebral palsy, periventricular lesions, stroke, brain injury, spinal cord injury (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).
Ataxic Gait	Wide base of support, unstable, stumbling, uncoordinated steps, difficulty walking in a straight line, trunk titubation (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).	Cerebellar lesions (degeneration, stroke, tumors), toxicity (alcohol, drugs), multiple sclerosis, proprioceptive disorders (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).
Neuropathic Gait (Steppage)	Excessive hip and knee flexion to lift the foot (high steppage) due to foot drop, foot "slaps" on the ground (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).	Peripheral neuropathies (diabetic, alcoholic, Charcot-Marie-Tooth), peroneal nerve injury, L5 radiculopathy, amyotrophic lateral sclerosis (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).
Myopathic Gait (Waddling)	Side-to-side swaying of the trunk ("duck waddle") due to weakness of pelvic girdle muscles (gluteals) (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.).	Muscular dystrophies, myopathies, spinal muscular atrophy, poliomyelitis (Loyola Medicine, 2025; Stanford Medicine 25, n.d.).

Sources: (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.; COAST Rehab, n.d.)

3. The Bidirectional Relationship between Gait and Executive Functions: Theoretical Frameworks

The historical perception of gait as more of an automatic motor task, not dependent on higher cognitive processes, has been significantly altered. There is abundant research evidence of a tight and two-way connection between gait and executive functions, particularly in patients with neurological problems. Different theoretical models have been developed to make sense of such complex interactions.

Cognitive-Motor Interference (CMI) Model

Among the prevailing theoretical accounts in the learning and processing of inconsistent cognitive and motor tasks is the cognitive-motor interference (CMI) model. CMI is characterized as the fact that a cognitive task and a motor task (walking) performed simultaneously result in worse performance in one or both tasks, compared to when each task is performed alone (in single-task conditions) (Plummer-DAmato et al., 2013; Number Analytics, n.d.).

The theoretical foundation of CMI is founded on the premise that the brain is characterized by scarce processing resources (attentional resources, processing capacity) (Plummer-DAmato et al., 2013; Number Analytics, n.d.). A given percentage of these resources is necessary to execute each task, either cognitive or motor, successfully. When two or more activities are carried out concurrently they rival access to these shared, finite resources. When the total demands of the concurrently executed tasks surpass the total capacity of the brain to process information, then either one or both of the tasks will be inevitably compromised (Plummer-D'Amato et al., 2013).

CMI may take various forms or results when cognitive and motor task are carried out in a dual-task situation compared with the single-task performance (Plummer-D Amato et al., 2013):

1) **No interference:** There is no change in the performance in either of the tasks.
2) **Cognitive-related motor interference:** There is a worsening of motor performance with little or no change in cognitive performance. This implies that the person gives priority to the mental task.
3) **Motor-related cognitive interference:** There is a decline in cognitive performance but no (or little) decline in motor performance. This indicates that the person is motor task prioritized.
4) **Mutual interference:** There is a decline in performance on both tasks at the same time.
5) **Other patterns:** Facilitation (an improvement in one or both tasks) or priority trade-off (an improvement in one task is coupled with a decline in the other) are also possible.

Appreciating the concept of CMI is important because it can explain why, when individuals are walking (a circumstance that is highly frequent in everyday life), the introduction of a modest cognitive task (talking, scanning the environment, and mental calculations) can have a profound destabilizing effect on gait, especially in neurological patients whose processing resources are already limited by the underlying pathology.

Shared Neural Resources and Neural Overlap Theories

Modern research has robustly refuted the traditional perception of gait as a purely automated motor process that needs only a little higher cognitive involvement (Sheridan et al., 2003; Montero-Odasso et al., 2012). Much evidence has accumulated that gait, particularly in complicated or uncertain conditions, or when automation of gait has been perturbed (as in neurological diseases), depends heavily on cognitive processing, principally by executive functions and attention (Sheridan et al., 2003; Montero-Odasso et al., 2012; Allali et al., 2010).

Functional imaging (fMRI, PET) of the brain has demonstrated frontal and parietal cortex regions to be active during walking, which are also important in executive functions (Sheridan et al., 2003). In particular, the prefrontal cortex (especially dorsolateral prefrontal cortex - DLPFC) and anterior cingulate cortex (ACC) have been associated with both the cognitive functioning of EFs as well as gait control, specifically in dual-task conditions (Sheridan et al., 2003). This neural overlap speaks in favor of the notion that gait and executive functions have at least partly common neural substrates and processing resources. This implies that motor control and performance of cognitive tasks can happen concurrently with the use of the same brain structures or networks.

A more general theoretical background on this interaction is given by the theory of Embodied Cognition (Wikipedia contributors, n.d.). What this theory asserts is that cognition is not some abstract structure that is manipulated solely by the brain; rather, it is embodied and directly affected by the condition of the body, the sensory perceptions, and motor capabilities (Wikipedia contributors, n.d.).

In this view, gait is not a mere motor output, but a dynamic process that serves in cognitive processing and environmental interaction. As such, gait disorders are not motor issues that happen to EFs, but rather, walking (or its disordered form) is a constituent of the larger cognitive system (Batara et al., 2022).

Importance of the Dual-Task Paradigm

The dual-task (DT) paradigm is a highly employed methodological instrument as well as a critical approach to examine the interference between gait and cognitive functions, specifically executive functions (Montero-Odasso et al., 2012; Allali et al., 2010). A typical DT experiment involves asking the participant to engage in a primary motor task (e.g., walking on a treadmill or a set distance) and a secondary cognitive task that demand attention and executive control (e.g., counting backward, verbal fluency, mental calculations, keeping track of visual or auditory events) (Allali et al., 2010).

The reasoning of the DT paradigm is that when two tasks are performed simultaneously, there is bound to be competition regarding the limited cognitive resources of the brain (Montero-Odasso et al., 2012). If gait was a completely automated process that did not demand any cognitive resources, the secondary cognitive task should not

influence the performance of gait and vice versa. Nevertheless, many studies have revealed that gait activity (e.g., speed, stride length, stride variability) tends to decline when a cognitive task is conducted at the same time, a phenomenon referred to as dual-task cost (Plummer-D Amato et al., 2013; Allali et al., 2010). Such interference indicates that gait and the cognitive task are dependent, at least partly, on similar functional subsystems and competent of shared attentional resources (Allali et al., 2010).

The DT paradigm is a cognitive stress test of gait. The dual-task cost can be very small or even absent in healthy young adults whose gait is highly automated, unless the cognitive task is very demanding. But in neurological patients (e.g., Parkinson disease, stroke, MS), gait automation may be perturbed by neural damage or by age-related neural physiological changes in older adults. Due to this, gait becomes more reliant on conscious cognitive control by executive functions. On these tasks, a secondary cognitive task is introduced which leads to an explosive growth in the overall demands placed on cognitive resources that are already limited. Should the resources available be inadequate, then the result will be a marked decline in performance in one gait or the cognitive task (or both) (CMI) (Plummer-D=Amato et al., 2013). The sequence of such deterioration (i.e., which task suffers more) can indicate the strategies of the person in allocating resources (e.g., focusing on fall prevention versus accurate performance on the cognitive task and vice versa) as well as the vulnerability of either the motor or cognitive performance (Plummer-D Amato et al., 2013).

The knowledge of these theoretical frameworks, CMI, shared neural resources, and the use of the DT paradigm, changes the focus of a mere observation of correlation between gait disorders and EF deficits to a more integrative realization of their root dependence. This has profound implications in the way we evaluate and intervene in the neurological patient in support of the need to pursue integrated strategies that do not view cognitive and motor functions as independent and discrete structures, but rather interdependent aspects of a larger system.

4. Manifestation of the Gait-Executive Function Interaction in Specific Neurological Diseases

The reciprocal interaction between gait and executive functions has a specific appearance in several neuropsychological diseases, depending on the neuropathology of the disease, and the structures involved. Studying this interaction in the context of specific diseases gives useful insights into pathophysiological processes and informs the design of specific therapeutic interventions.

Parkinson's Disease (PD)

Parkinson disease (PD) is a progressive nervous system (neurodegenerative) disorder that is predominantly motor-symptom-defined, with the main characteristics being bradykinesia, resting tremor, rigidity, and postural instability, which combine into the characteristic parkinsonian gait pattern (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; COAST Rehab, n.d.). Nevertheless, PD is a complex condition as well, and non-motor symptoms are an important part of it, with cognitive dysfunction having a particularly important role. Mild Cognitive Impairment (MCI) is highly prevalent, with up to 34% of patients developing it at early stages of the disease and its tendency to develop PD-type dementia (Guo et al., 2021). One of the first and most commonly affected cognitive areas in PD is related to executive functions (Allali et al., 2010).

The interplay between gait disorders and EF deficits is most prominent in PD. Gait and posture regulation that usually are dependent on automated mechanisms governed by the basal ganglia are grossly perturbed in PD because of the loss of dopaminergic neurons of the substantia nigra. Such a loss of automation results in gait being more reliant on conscious, executive control by the prefrontal cortex (Guo et al., 2021). But since EFs are also frequently compromised in PD, patients are confronted with a two-pronged problem.

This weakness is especially evident in the conditions of a dual-task (DT). Asking PD patients to do a cognitive task while walking worsens their gait performance notably, more than it does to their healthy peers (Allali et al., 2010; Guo et al., 2021). Indicatively, a relatively simple cognitive task, serial seven subtractions backward, during walking results in a marked decrease in walking speed, stride length, and an increment in gait variability, putting the person at risk of falls (Allali et al., 2010). Such symptom as freezing of gait, which is one of the most disabling symptoms of PD is also likely to be worsened by conditions of DT or in situations where more attention and decision making are needed.

Automation loss in PD implies that patients have to think about the step they are taking, which occupies considerable executive resources. When such resources are also required to support a secondary cognitive task, the already overloaded system breaks down causing a breakdown in gait. This forms a bad loop, where the inability to walk needs more attention, which is constrained by the disease itself and the cognitive load at the same time.

The identification of this intimate association has paved the way to the design of rehabilitation interventions addressing both factors in an integrated manner. Dual-Task Training (DTT) has been announced as an avenue of promise. The research indicated that DTT could enhance the walking speed and stride length during single and dual-task scenarios, walking endurance, balance, and even certain measures of cognitive performance among PD patients (Guo et al., 2021; Stasolla et al., 2016). DTT aims at either re-automating some details of gait or helping patients to better prioritize their limited cognitive resources across the tasks they perform simultaneously.

Stroke

Stroke is a major cause of chronic disability and motor and cognitive outcomes are common. After a stroke, gait disorders, like hemiplegic gait, are prevalent, and so are executive functions deficits, depending on the localization and the size of the brain lesion. Gait and balance are majorly influenced by cognitive-motor interference (CMI) in post-stroke patients (Plummer-D amato et al., 2013).

Research on the dual-task paradigm has revealed that, when a cognitive task is introduced, stroke patients tend to develop a pronounced decline in their motor performance (e.g., slower walking speed, greater stride variability). The paradigm of CMI most often found is a cognitive-related motor interference (in which motor performance declines whereas cognitive performance is comparatively spared) or a mutual interference (in which both performances are disrupted) (Plummer-D'Amato et al., 2013). This implies that post-stroke walking is less automated and needs more cognitive resources.

Especially interesting is the turning of mobility in chronic stroke patients. Turning - it is a complicated motor activity involving dynamic balance, coordination, and quick adjustment of the gait pattern. It was revealed that turn performance is substantially connected to the overall cognitive functioning and several cognitive domains, including visuospatial capacity and language (Hsu et al., 2022). This relationship seems to be mediated by balance and lower limb strength, indicating that cognitive function influences the capability of maintaining balance and producing force, which subsequently is the key to turning success (Hsu et al., 2022). Turning, which is a more cognitively complex task than straight walking, can more readily expose simmering deficits in patients with a low cognitive capacity amid brain injury (Hsu et al., 2022). In imaging research, the activity of the prefrontal cortex has been found to increase at the point when straight walking is changed into turning, suggesting that prefrontal cognitive control can be enlisted to help balance motor deficits (Hsu et al., 2022).

The motor and cognitive deficit patterns arise due to the spatial and extent heterogeneity of stroke lesions. As a result, the gait-EF interaction varies greatly between patients in its nature and severity. These deficits are more likely to emerge during motor tasks that are by nature more complex and necessitate more cognitive processing, turning, obstacle avoidance or walking on irregular surfaces all require constant adaptation, planning and visuospatial analysis.

As far as rehabilitation is concerned, traditional physiotherapy appears to have minor effects regarding the decrease in CMI (Plummer-D Amato et al., 2013). Nevertheless, the interventions that include dual-task training (DTT) and other types of integrated motor-cognitive training, including exergames, are promising (Huber et al., 2025). The interventions are expected to lead to improved capacity of patients to attend to motor and cognitive tasks at the same time, essential to safe and efficient functioning in real life. Guided plasticity facilitation model promotes the hypothesis that physical and cognitive training together can specifically engage neuroplasticity, which is the major to recovery after stroke (Huber et al., 2025).

Multiple Sclerosis (MS)

Multiple sclerosis (MS) is a central nervous system, chronic, inflammatory, demyelinating disease, which results in diverse neurological symptoms, such as motor and cognitive disorders (Kadrnka et al., 2025; Wajda & Sosnoff, 2015). With an impact on up to 75% of PwMS and a significant contribution to disability and poor quality of life, gait and balance disorders are ridiculously widespread (Ghasemi et al., 2024). At the same time, one of the most common and underrecognized symptoms of the disease is cognitive dysfunction, especially in the domains of information processing speed, attention, memory, and executive functions (Ghasemi et al., 2024; Bae et al., 2025).

The interplay between motor and cognitive impairments in MS is especially apparent when assessed in a dual-task condition (Kadrnka et al., 2025; Wajda & Sosnoff, 2015; Ghasemi et al., 2024). Research has revealed that when PwMS are instructed to engage in a cognitive task (e.g., the Paced Auditory Serial Addition Test - PASAT, a test that measures speed of auditory information processing, working memory, and executive attention) during walking, considerable adverse alterations take place in nearly all gait measures. Namely, a decrease in walking speed, stride

length, swing phase percentage, and minimum toe clearance and an increase in stride time and stance phase percentage are noted in comparison to single-task walking (Kadrnka et al., 2025; Wajda & Sosnoff, 2015).

Interestingly, this worsening of gait under DT is greater in PwMS with worse baseline cognitive performance (Kadrnka et al., 2025; Wajda & Sosnoff, 2015). Even PwMS with fairly normal gait during single-task conditions can display considerable impairment when a cognitive load is introduced, indicating that their compensation capabilities of subclinical impairment are depleted in challenging conditions (Kadrnka et al., 2025; Wajda & Sosnoff, 2015). In other instances, the patients can choose to focus on the cognitive task and maintain their performance on that task, however, at the substantial expense of gait performance (Wajda & Sosnoff, 2015).

This interaction seems key to the central importance of the speed of information processing in MS as a result of demyelination and slowed neural conduction. Fluid and efficient walking necessitates a quick and constant combination of sensory data and passing of motor commands. When this process is slowed, it makes the gait more susceptible. With the introduction of a secondary cognitive task, which also consumes processing resources, the already overloaded system is overloaded, and performance suffers.

DTT has been demonstrated to enhance walking velocity, total motor execution, and certain cognitive features in PwMS (Ghasemi et al., 2024). Dual-task assessment is important to uncover "concealed" impairments that may not be observed during single-task assessment and to inform individualized interventions. Objective gait measurement using wearable sensors in different conditions is also becoming popular in MS studies and clinical practice (Sharrack et al., n.d.).

Table 3
Summary of Gait and Executive Function Interaction in Specific Neurological Diseases

Neurological Disease	Typical Gait Disorders	Common Executive/Cognitive Function Deficits	Key Findings from Dual-Task (DT) Studies/ Interaction	Main Sources
Parkinson's Disease (PD)	Parkinsonian gait (bradykinesia, small steps,freezing, reduced arm swing) (Cleveland Clinic, n.d.-b;Loyola Medicine,2025; COAST Rehab, n.d.).	EFdeficits (planning, flexibility, working memory, inhibition), attention, visuospatial functions (Allali et al., 2010; Guo et al., 2021).	Significant deterioration of gait parameters under DT. Loss of automaticity makes gait EF-dependent. DTT improves performance (Allali et al., 2010; Guo et al., 2021; Stasolla et al., 2016).	(Allali et al., 2010; Guo et al., 2021; Stasolla et al., 2016)
Stroke (CVA)	Hemiplegic gait, instability, difficulty turning (Loyola Medicine, 2025; Stanford Medicine 25, n.d.; Hsu et al., 2022).	EF deficits, attention, visuospatial ability, language (depending on lesion location) (Plummer-D'Amato et al., 2013; Hsu et al., 2022).	Cognitive-related motor interference or mutual interference. Turning is particularly sensitive to DT. DTT and exergames show benefits (Plummer-D'Amato et al., 2013; Hsu et al., 2022; Huber et al., 2025).	(Plummer-D'Amato et al., 2013; Hsu et al., 2022; Huber et al., 2025)
Multiple Sclerosis (MS)	Ataxic gait, spastic gait, instability, reduced speed and endurance (COAST Rehab, n.d.; Ghasemi et al., 2024).	Reduced information processing speed, EF deficits, attention, memory (Ghasemi et al., 2024; Bae et al., 2025).	Significant deterioration of all gait parameters under DT, especially in individuals with lower cognitive performance. Possible prioritization of cognitive task. DTT can improve performance (Kadrnka et al., 2025; Wajda & Sosnoff, 2015; Ghasemi et al., 2024).	(Kadrnka et al., 2025; Wajda & Sosnoff, 2015; Ghasemi et al., 2024; Bae et al., 2025)

Sources: As listed in the last column.

5. Clinical Consequences and Implications for Rehabilitation

The interaction between gait disorders and executive function deficits is not merely of academic interest but has profound and direct clinical consequences for neurological patients. These consequences affect their daily functioning, safety, and overall quality of life, making integrated and effective rehabilitation strategies imperative.

Impact on Activities of Daily Living, Fall Risk, and Quality of Life

Gait disorders, by themselves, significantly limit individuals' ability to perform basic activities of daily living (ADLs), such as personal hygiene, dressing, meal preparation, and moving inside and outside the home. When these disorders coexist with executive function deficits, the impact is multiplied.

EFs are essential for planning and organizing complex ADLs, adapting to unpredictable situations, following instructions, and avoiding risks. Reduced dual-tasking ability, i.e., difficulty performing a motor task (like walking) simultaneously with a cognitive task (like talking or observing the environment), can dramatically limit functional mobility activities and community participation (Plummer-D'Amato et al., 2013).

One of the most serious risks arising from the combination of gait disorders and executive dysfunction is an increased risk of falls (Montero-Odasso et al., 2012; Cleveland Clinic, n.d.-b). Gait in real-world conditions is rarely a single task. It often requires simultaneous processing of environmental stimuli, decision-making (e.g., how to avoid an obstacle), and movement adaptation. If the EFs supporting these processes are impaired, the ability to react to unpredictable events decreases, making falls more likely. Falls in neurological patients can have devastating consequences, leading to injuries (e.g., fractures), hospitalization, fear of new falls, further activity restriction, social isolation, and, in some cases, even death (Montero-Odasso et al., 2012; Allali et al., 2010).

Inevitably, the combination of motor and cognitive deficits negatively affects patients' overall quality of life (Guo et al., 2021; Ghasemi et al., 2024). Loss of independence, fear of falls, difficulty participating in social and recreational activities, and a sense of reduced ability can lead to feelings of frustration, anxiety, and depression.

Rationale and Evidence for Integrated Motor-Cognitive Rehabilitation Strategies

Given the close and bidirectional relationship between gait and executive functions, traditional rehabilitation strategies that individually target either the motor or cognitive component may be insufficient to address the complex problems faced by neurological patients (Guo et al., 2021). There is a growing recognition that interventions must be integrated, simultaneously addressing both aspects of dysfunction.

The theoretical basis for such integrated interventions comes from understanding shared neural substrates and cognitive-motor interference mechanisms. If gait and EFs share processing resources and neural networks, then training that targets both functions simultaneously may be more effective in promoting neuroplasticity and improving functional performance. The guided plasticity facilitation model supports this approach, proposing that the combination of physical and cognitive training can specifically activate the neuroplasticity mechanisms necessary for recovery and adaptation after neurological damage (Huber et al., 2025).

Review of Dual-Task Training (DTT) Interventions

Dual-task training (DTT) has emerged as a primary strategy for addressing combined motor and cognitive deficits. DTT involves the simultaneous performance of two tasks that require cognitive and motor resources, aiming to improve an individual's ability to effectively manage such concurrent demands in daily life (Number Analytics, n.d.). The ultimate goal is often to improve the automation of motor actions (like walking), thereby freeing up cognitive resources for the simultaneous execution of other tasks, or to improve the ability to allocate attention between tasks (Guo et al., 2021).

DTT protocols vary significantly in terms of task type, difficulty, duration, and training frequency. Typically, they include a primary motor task (e.g., walking on a treadmill or overground, balance exercises, cycling) and a secondary cognitive task (e.g., mathematical calculations, verbal fluency tasks, list recall, answering questions, monitoring visual or auditory stimuli) (Number Analytics, n.d.; Guo et al., 2021).

The effectiveness of DTT has been investigated in various neurological populations, with encouraging results.

In Parkinson's Disease (PD): DTT has been shown to improve walking speed and stride length in both single and dual-task conditions, walking endurance (e.g., distance in the 6 Minute Walk Test), and various other gait parameters. Some studies also report improvements in balance and cognitive functions such as attention and

executive function (Stasolla et al., 2016). These benefits may be maintained for some time after the intervention is completed (Stasolla et al., 2016).
In Alzheimer's Disease (AD) and other dementias: DTT has been associated with improvements in stride length in single-task conditions and a reduction in Dual-Task Cost (DTC) for speed and stride length. Improvements in balance (e.g., on the Berg Balance Scale) and some measures of executive functions (e.g., Frontal Assessment Battery) have also been reported (Stasolla et al., 2016).
In Traumatic Brain Injury (TBI) and Stroke: Although data are more limited, some studies and case reports support that DTT can improve walking speed under dual-task conditions and dual-task tolerance (Stasolla et al., 2016). For stroke, as mentioned, conventional rehabilitation has minimal effects on CMI, but dual-task interventions may reduce it (Plummer-D'Amato et al., 2013).
Despite positive findings, heterogeneity in DTT protocols and outcome measures makes it difficult to compare studies and draw definitive conclusions about the optimal "dose" and type of training (Stasolla et al., 2016).

Emerging Therapeutic Approaches

Beyond traditional forms of DTT, new technological approaches are emerging that offer innovative ways for motor-cognitive rehabilitation:
Exergaming (Exercise Games): Exergames are video games that require physical movement to interact with the game. They represent a promising type of combined motor-cognitive training, as their gamified nature can enhance motivation, enjoyment, and adherence to training, factors crucial for rehabilitation success (Huber et al., 2025).
Step-based exergames performed in a standing position appear to be particularly beneficial for simultaneously improving cognitive functions and gait (Huber et al., 2025). A recent randomized controlled trial (PEMOCS) in chronic stroke survivors showed that a personalized exergaming program, added to usual care, helped maintain overall cognitive function and had encouraging results on patients' perception of mobility, as well as on specific cognitive (e.g., alertness, working memory) and motor (e.g., outdoor walking speed, swing width of the unaffected lower limb) parameters (Huber et al., 2025).
Virtual Reality (VR): VR offers the ability to create rich, interactive, and safe environments for training gait and cognitive functions. Patients can practice scenarios that simulate real-life challenges (e.g., crossing a street, avoiding obstacles) without the actual risks. Treadmill training with simultaneous use of VR and cognitive tasks has shown reduced fall risk, improved mobility, and enhanced cognitive function in older adults, individuals with mild cognitive impairment, and PD patients (Guo et al., 2021).
DTT, whether through traditional methods or new technologies, appears to promote neuroplasticity and, potentially, transfer of learning. Findings that DTT improves performance in *single* tasks as well (Stasolla et al., 2016) suggest it may induce more fundamental changes, such as strengthening underlying neural networks or increasing the efficiency of cognitive resources. However, there is a need for personalized interventions, considering the specific needs of each patient, and for greater emphasis on ecological validity, i.e., ensuring that improvements observed in the therapeutic environment translate into meaningful improvement in daily life functionality (Number Analytics, n.d.; Huber et al., 2025).

6. Conclusions and Future Research Directions

This review has highlighted the complex and clinically significant interaction between gait disorders and executive function deficits in neurological patients. The understanding of this relationship has evolved significantly, moving away from the traditional view of gait as a simple, automated motor act.

Synthesis of Key Evidence

The key evidence emerging from the literature review can be summarized as follows:

1) **Executive functions (EFs)**, a set of higher-level cognitive skills that include inhibition, working memory, and cognitive flexibility, are essential for goal-directed behavior and are supported by an extensive neural network centered on the prefrontal cortex (Sheridan et al., 2003; UCSF Memory and Aging Center, n.d.; Cleveland Clinic, n.d.-a; EBSCO Research Starters, 2024; Carroll & Simpson, 2022).
2) **Gait disorders** in neurological patients are diverse (e.g., parkinsonian, hemiplegic, ataxic) and often reflect the underlying neuropathology, significantly affecting mobility and safety (Cleveland Clinic, n.d.-b; Loyola Medicine, 2025; Stanford Medicine 25, n.d.; COAST Rehab, n.d.).
3) **Gait is not a simple motor act** but a complex behavior requiring significant cognitive processing,

particularly from executive functions, especially when its automation is disrupted or the environment is demanding (Sheridan et al., 2003; Montero-Odasso et al., 2012).

4) **A strong bidirectional relationship exists** between gait and EFs. Neurological diseases often affect common neural substrates or interacting networks, leading to the simultaneous appearance of motor and cognitive deficits (Sheridan et al., 2003; Allali et al., 2010).
5) **The interaction** can be theoretically explained by the cognitive-motor interference (CMI) model and shared neural resources theories (Sheridan et al., 2003; Plummer-D Amato et al., 2013; Number Analytics, n.d.).
6) **The dual-task (DT) paradigm** has become a key methodological instrument to demonstrate and measure the gait-EF interaction, and to identify the deficits that might not be evident during single-task circumstances (Montero-Odasso et al., 2012; Allali et al., 2010).
7) **This interaction in certain neurological pathologies**, including Parkinson diseases, stroke, and multiple sclerosis, is presented in particular manners, involving particular gait parameters and cognitive abilities (Guo et al., 2021; Hsu et al., 2022; Kadrnka et al., 2025; Wajda & Sosnoff, 2015).
8) **This interaction has serious clinical implications** such as the risk of falls, decreased activity in activities of daily living, and compromised quality of life (Montero-Odasso et al., 2012; Cleveland Clinic, n.d.-b; Guo et al., 2021).
9) **Motor-cognitive rehabilitation approaches**, and notably dual-task training (DTT) and novel technologies such as exergames and virtual reality, are promising to enhance gait and cognitive performances (Guo et al., 2021; Stasolla et al., 2016; Huber et al., 2025).

Identification of Current Limitations in the Literature and Unanswered Questions

Despite significant progress, the current literature presents certain limitations and leaves important questions unanswered:

Heterogeneity of Studies: There is significant heterogeneity in published studies regarding patient population characteristics (e.g., type and stage of disease, age, cognitive level), intervention protocols (especially for DTT, where the type, intensity, duration, and frequency of cognitive and motor tasks vary widely), and outcome measures used (Stasolla et al., 2016). This heterogeneity hinders the comparison of findings, the conduct of meta-analyses, and the generalization of conclusions.

Long-Term Follow-up: Most intervention studies have relatively short-term follow-up. There is a lack of data on the long-term maintenance of therapeutic effects and their impact on important indicators such as fall frequency and quality of life over time.

Specificity of Mechanisms: A better understanding is needed of the specific neurobiological and cognitive mechanisms through which different EF components (e.g., inhibition versus working memory versus cognitive flexibility) affect different gait parameters (e.g., speed, rhythm, variability, symmetry) in different neurological diseases.

Influence of Confounding Factors: The role of factors such as fatigue (physical and cognitive), mood (e.g., anxiety, depression), pain, sleep quality, and medications in the gait-EF interaction has not been adequately investigated.

Ecological Validity of Assessments: Many assessments are conducted in controlled laboratory environments, which may not fully reflect the challenges of daily life. There is a need for more sensitive and ecologically valid methods for assessing gait and cognitive function in real-world conditions (Huber et al., 2025).

Responders versus Non-Responders: Not all patients respond equally to motor-cognitive interventions. Understanding the factors that differentiate "responders" from "non-responders" (as mentioned in the PEMOCS study (Huber et al., 2025)) is important for personalizing treatment.

Proposals for Future Research Directions

Based on the above limitations, the following directions for future research are proposed:

1) **Standardization of Protocols and Large-Scale Studies:** Conduct larger, multicenter, randomized controlled trials with well-defined populations, standardized intervention protocols (especially for DTT), and homogeneous, clinically relevant outcome measures. This will strengthen the evidence base and allow for more robust conclusions.
2) **Investigation of Neurobiological Mechanisms:** Use advanced neuroimaging (e.g., fMRI, DTI, fNIRS) and neurophysiology (e.g., EEG, TMS) techniques to investigate the neural changes associated with the gait-EF interaction and the effects of motor-cognitive interventions.

3) **Personalized Rehabilitation:** Develop and validate personalized rehabilitation approaches that consider the patient's specific neurological and cognitive profile, disease stage, comorbidities, motor abilities, and personal goals.
4) **Transfer of Benefits and Ecological Validity:** Emphasize the development of interventions that maximize the transfer of therapeutic benefits to real-life living conditions, improving independence, safety, and community participation. Use wearable sensors and technologies for monitoring activity and performance in the patient's natural environment (Sharrack et al., n.d.).
5) **Utilization of New Technologies:** Further investigate the effectiveness, applicability, accessibility, and cost-effectiveness of new technologies (e.g., VR, exergames, wearable sensors, artificial intelligence) in assessing and treating combined motor and cognitive deficits.
6) **Determinants of Therapeutic Response:** Investigate factors (e.g., cognitive reserve, disease severity, genetic factors, psychosocial factors) that predict response to motor-cognitive interventions, aiming to optimize patient selection and adapt therapeutic strategies.
7) **Investigation of Specific Populations and Aspects:** Focus on less-studied populations or aspects, such as the impact of fatigue or emotional factors, and the development of interventions for rare neurological diseases.

The transition from understanding mechanisms to developing and optimizing personalized and functionally significant interventions is the main challenge for the future. Successfully addressing combined cognitive and motor deficits in neurological patients requires a continuous, iterative process of research and clinical innovation, with the ultimate goal of enhancing patients' independence, safety, and well-being. Interdisciplinary collaboration among neurologists, physical therapists, occupational therapists, neuropsychologists, and engineers is essential to achieve this goal.

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