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Review Article

Posture and Movement under Influence: Neurophysiological and Neurobiochemical Factors in Neurodegenerative Diseases

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Abstract

Postural instability is a common and tragic symptom of neurodegenerative disorders, markedly elevating the risk of falls and morbidity. Healthcare providers must appropriately evaluate and manage postural instability in individuals with these medical conditions to avert falls and enhance quality of life. Nonetheless, the specific pathophysiological mechanisms underlying these impairments differ significantly across diseases. This study offers an extensive examination of the neurophysiological and neurobiomechanical factors influencing posture in Parkinson's disease (PD), Multiple Sclerosis (MS), Huntington's disease (HD), Alzheimer's disease (AD), and Amyotrophic Lateral Sclerosis (ALS). Dopamine depletion and cholinergic dysfunction in basal ganglia circuits in Parkinson's disease inhibit anticipatory adjustments and sensory reconfiguration, leading to rigidity and distinct sway patterns. Multiple sclerosis is characterised by conduction delays due to demyelination, exhaustion, and stiffness, resulting in increased sway amplitude and velocity. Conversely, HD entails striatal degeneration that produces involuntary choreiform movements, resulting in unpredictable and unexpected shifts in the centre of mass. Alzheimer's disease is characterised by cortical shrinkage and cognitive-motor disruption, revealing significant impairments in sensory integration, especially with diminished visual stimuli. ALS exhibits a distinct profile characterised by the degeneration of upper and lower motor neurones, which undermines trunk control and leads to maladaptive stiffening tactics. This analysis posits that extant generic interventions frequently prove ineffective due to a failure to account for these differing mechanisms. Therefore, we propose a move towards disease-specific evaluation methodologies and customised rehabilitation strategies to effectively reduce falls and improve patient autonomy.

Introduction

Postural control is a crucial component of human motor function, enabling individuals to maintain balance and stability during both static and dynamic movements. The body's centre of mass is maintained within the base of support through the integration of multiple neural systems and biomechanical components in a complex sensorimotor process [1,2]. When this complex system is disrupted by neurodegenerative disorders, the repercussions can be significant, impacting mobility, autonomy, and overall quality of life [3,4].

Neurodegenerative disorders are characterised by the progressive deterioration of neurons in the central nervous system, leading to a range of motor, cognitive, and sensory deficits [5-7]. Among the numerous manifestations, postural instability is a particularly debilitating symptom that substantially increases the risk of falling and reduces functional

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independence. Although many neurodegenerative diseases share a lack of postural control, the specific pathophysiological processes at work cause these symptoms to manifest in quite different ways [8,9].

Parkinson's disease (PD) is a progressive neurodegenerative disorder predominantly marked by the degeneration of dopaminergic neurons in the substantia nigra, manifesting with key motor symptoms such as bradykinesia, stiffness, tremor, and postural instability [10-12]. The postural abnormalities of Parkinson's disease occur in multiple areas, including static stance stability, anticipatory postural modifications, and reactive balance responses. These deficits are particularly alarming as they substantially increase the chance of falls, which are a primary cause of morbidity and mortality in this demographic [13-15].

Posture control is severely impaired in people with multiple sclerosis (MS), a neurological disorder characterised by chronic inflammatory demyelinating damage to the central nervous system and extensive axonal loss in the brain and spinal cord [16-18]. In contrast to Parkinson's disease, which initially presents with more localised pathology, various Sclerosis can simultaneously impact various neurological pathways, resulting in various presentations of postural instability [19-21]. Effective postural control depends on sensory integration, cerebral processing, and motor output, all of which are impacted by the demyelination process, which interferes with neural transmission [22-24].

Huntington's disease (HD), a hereditary neurological condition resulting from a mutation in the HTT gene, is marked by involuntary movements, cognitive deterioration, and behavioural disorders [25-27]. Postural control in Huntington's disease is impaired due to involuntary choreiform movements and degeneration of the basal ganglia, especially the striatum [28-30]. Abnormal body postures and decreased coordination adversely affect balance and mobility, frequently resulting in falls even in the first stages of the condition.

Alzheimer's disease (AD), the predominant cause of dementia globally, predominantly impairs cognitive function while also resulting in considerable abnormalities in postural control, especially as the condition progresses [31,32]. Recent research has shown that Alzheimer's disease patients possess unique postural control traits, particularly under settings of restricted visual input [33]. These balance impairments are associated with the severity of cognitive loss and may indicate the engagement of brain areas that integrate sensory information for postural regulation [34].

Amyotrophic Lateral Sclerosis (ALS) is a progressive neurological disorder that impacts both upper and lower motor neurons, resulting in muscle weakness, atrophy, and ultimately paralysis [35-37]. In the early stages of ALS, postural instability is characterised by abnormal trunk control and altered central control strategies. Research indicates that these postural abnormalities are not merely due to muscle weakness but may signify more intricate alterations in the cerebral processing of balance information [38-40].

Although postural instability is common and clinically significant in various neurodegenerative disorders, the fundamental neurophysiological and neurobiomechanical mechanisms are not completely understood. This information gap affects both assessment and therapy methodologies. Current therapeutic strategies for postural instability in neurodegenerative disorders frequently produce inadequate results, mainly due to a limited comprehension of the specific pathophysiological mechanisms involved.

This study aims to provide an exhaustive examination of the neurophysiological and neurobiomechanical factors influencing posture and movement regulation in prominent neurodegenerative disorders. Our objective is to inform more targeted and effective assessment and treatment approaches by investigating these conditions through both lenses, thereby emphasising the similarities and differences in how these diseases affect postural control.

Fundamentals of postural control

Postural control is a complex sensorimotor mechanism that enables humans to maintain equilibrium in both static and dynamic positions. This essential motor function depends on the complex coordination of various neural networks and biomechanical factors operating in harmony [41-43]. Comprehending the standard mechanisms of postural regulation offers crucial context for analysing the impairments evident in neurodegenerative disorders. The neural network responsible for postural control encompasses various levels of the central nervous system, ranging from the spinal cord to the cerebral cortex [44,45]. Proprioceptive reflexes are the foundation of postural stability, as they provide rapid, automatic responses to perturbations at the spinal level. These reflexes are regulated by descending inputs from supraspinal areas, such as the brainstem, cerebellum, basal ganglia, and cortex [46,47]. The reticular formation and vestibular nuclei in the brainstem play a crucial role in maintaining postural tone and orientation relative to gravity [48]. The cerebellum is essential for coordinating multi-joint movements and adjusting postural responses based on experience. The motor cortex, supplementary motor area, and premotor cortex facilitate anticipatory postural adjustment and the voluntary components of postural control [44,49,50].

A critical node in this network is the basal ganglia, which, through its connections with the thalamus, cortex, and brainstem, is essential for selecting and executing appropriate postural strategies and modulating muscle tone. Subsequent sections will address how pathological changes in these brain circuits, such as dopaminergic depletion in PD or striatal neuronal degeneration in HD, result in apparent and clinically significant postural deficits.

Different neurodegenerative diseases

Appropriate postural regulation also requires sensory integration. Balance is provided by three primary sensory systems: the visual system, which provides information about the environment and body position; the vestibular system,

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which detects head position and movement relative to gravity; and the somatosensory system, which provides information about body position via proprioceptive and tactile inputs. At various levels of the nervous system, these sensory inputs are integrated, with the weighting of each input being dynamically adjusted in response to environmental conditions and task demands. For instance, when standing on an unstable surface, the nervous system may reduce its reliance on unreliable somatosensory information and instead prioritise visual and vestibular cues [51-55] (Figures 1-4).

Each neurodegenerative disease disrupts this sensory integration process through distinct mechanisms—whether by impaired proprioceptive processing (Parkinson's disease, multiple sclerosis, amyotrophic lateral sclerosis), visual dependence (Parkinson's disease, Alzheimer's disease), or disrupted central integration (Huntington's disease, multiple sclerosis).

From a biomechanical standpoint, postural control involves maintaining the body's centre of mass within the base of support. This is accomplished by generating appropriate joint torques through coordinated muscle activations that counteract destabilising forces. Continuous adjustments are necessary to preserve an erect posture, as the human body is an inherently unstable inverted pendulum. These modifications are achieved by distinct postural strategies, encompassing the ankle strategy (for minor perturbations), hip strategy (for bigger or more rapid perturbations), and stepping strategy (when the centre of mass shifts beyond the base of support) [22,24,56,57]. The appropriate strategy is determined by the individual's physical capabilities, the available base of support, and the extent and direction of the perturbation. Postural control is categorised into three main domains: static, anticipatory, and reactive. Static postural control denotes the capacity to sustain equilibrium during a stationary position, frequently evaluated via assessments of postural sway. The process of anticipatory postural control entails the implementation of preparatory adjustments before voluntary movements to mitigate anticipated disturbances to stability. These anticipatory postural adjustments (APAs) are essential for preserving equilibrium during activities such as gait initiation or reaching. Responses to unforeseen disruptions, such as the choice and application of suitable balance recovery techniques, are included in reactive postural control [58-61].

Anticipatory postural adjustments (APAs) are preparatory muscle activations that occur before voluntary movements to counteract predicted perturbations to stability. Reactive postural responses are automatic corrections triggered by unexpected perturbations. These responses involve stereotyped muscle activation patterns (ankle, hip, or stepping strategies) and are modulated by descending cortical and subcortical input. APA and reactive deficits are common across neurodegenerative diseases, but the underlying mechanisms differ substantially.

The remarkable adaptability of human postural control across various environmental conditions and task demands is facilitated by the integration of these neural, sensory, and biomechanical components [62,63]. However, this intricate

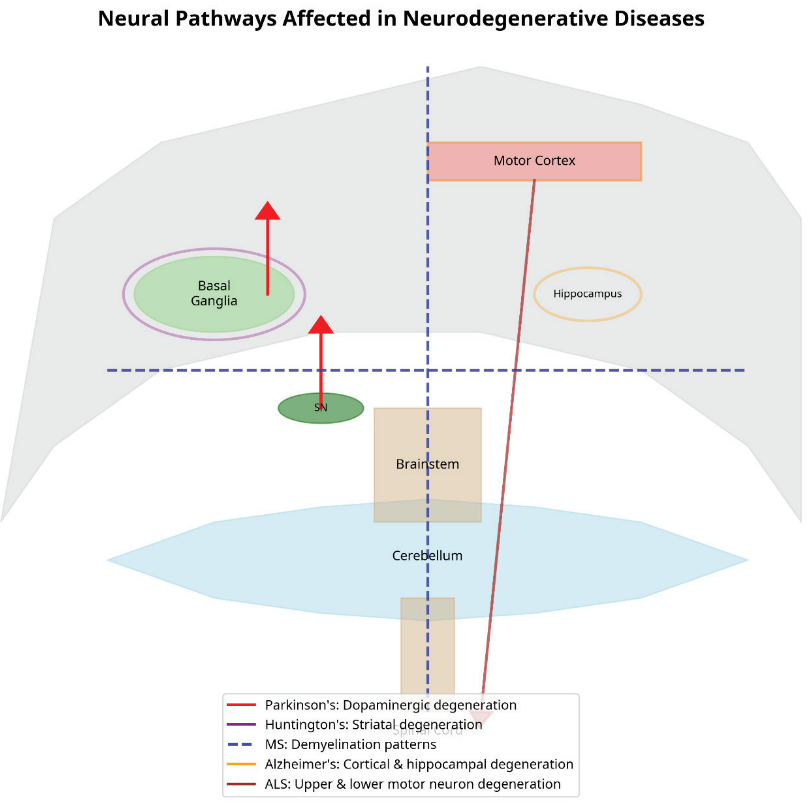

Figure 1: Neural pathways involved in postural control and how they are affected in.

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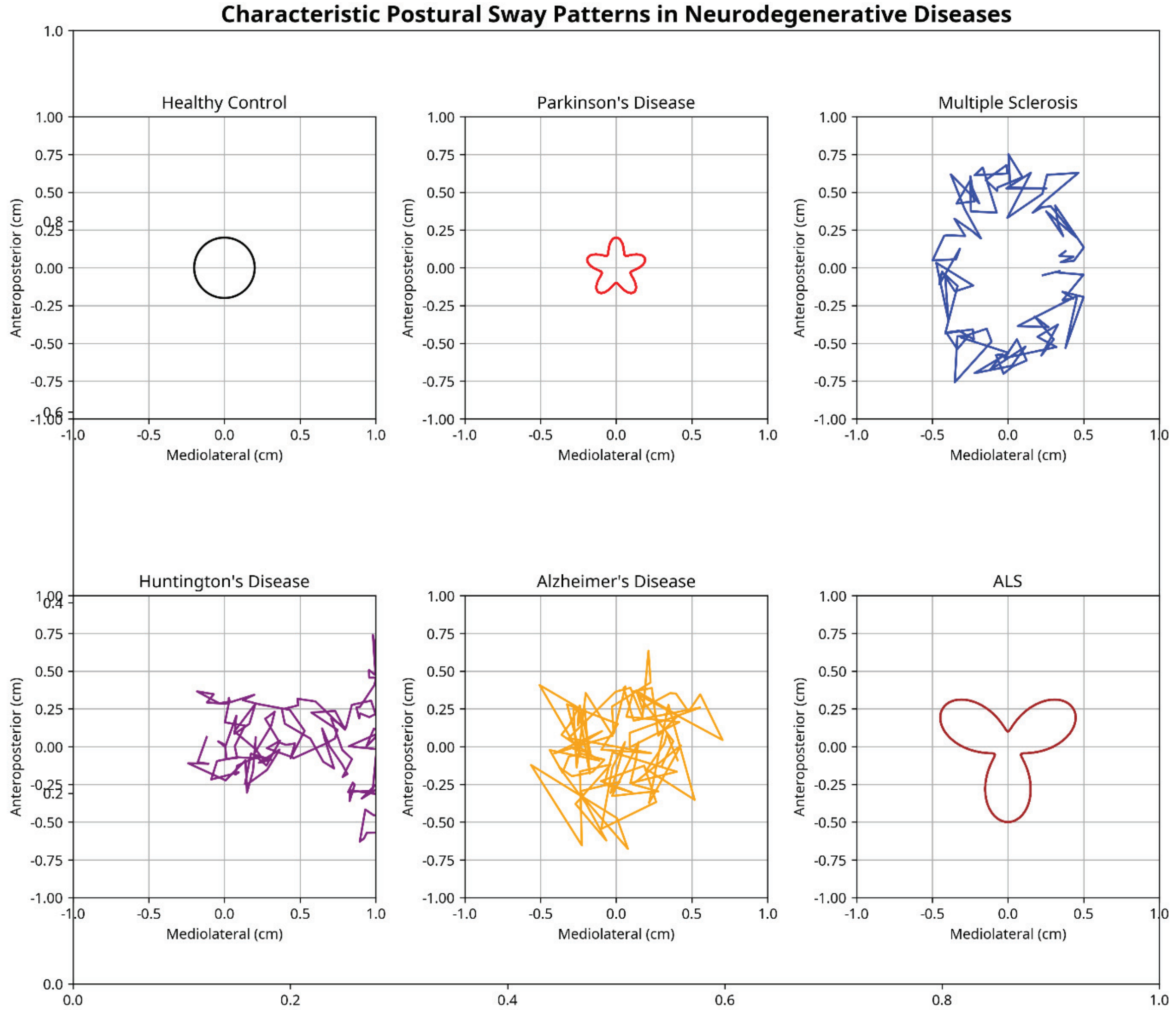

Figure 2: Characteristic postural sway patterns observed in different neurodegenerative diseases compared to healthy controls.

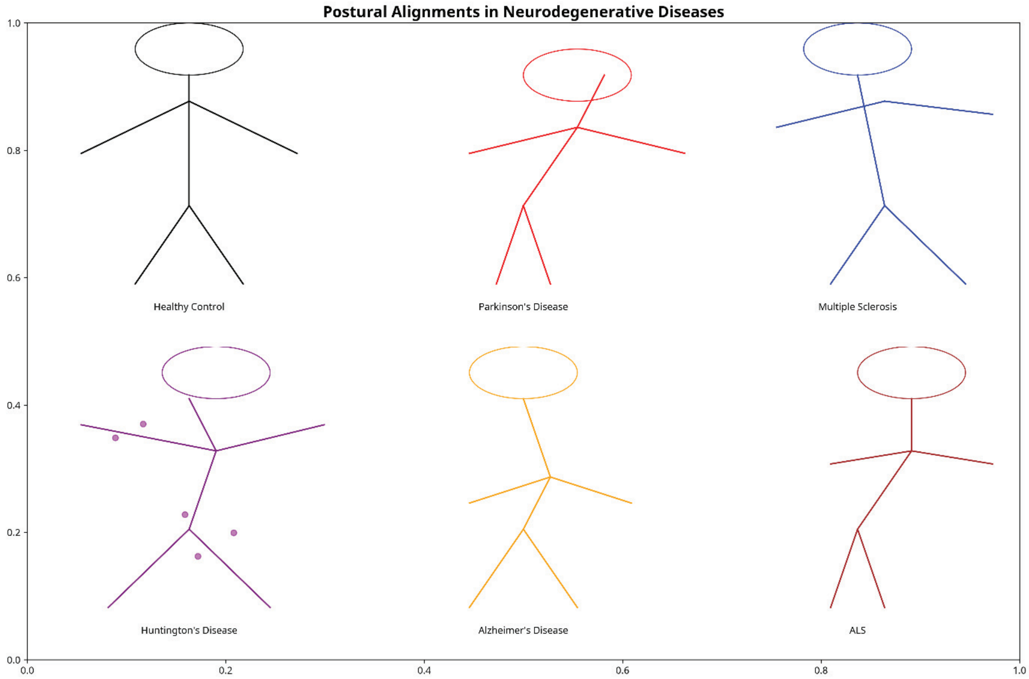

Figure 3: Typical postural alignments and biomechanical adaptations observed in different neurodegenerative diseases.

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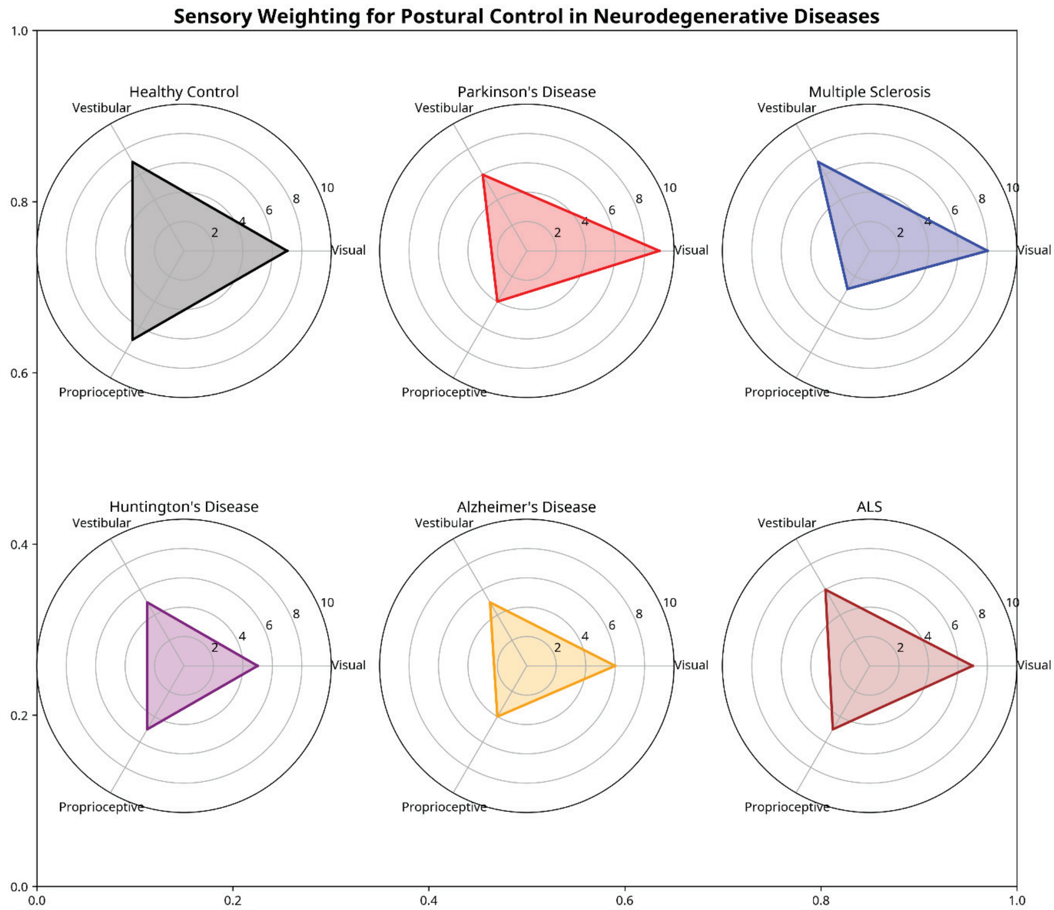

Figure 4: Sensory weighting patterns for postural control in different neurodegenerative diseases.

system is susceptible to disruption by neurodegenerative processes, as demonstrated by the postural instability that is observed in conditions such as Parkinson's disease, Multiple Sclerosis, Huntington's disease, Alzheimer's disease, and Amyotrophic Lateral Sclerosis.

Parkinson's disease

Parkinson's disease (PD) is characterised by the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to dopamine depletion in the striatum and dysfunction of basal ganglia circuits. Bradykinesia, stiffness, and tremor are the three hallmark motor symptoms of Parkinson's disease (PD). Still, postural instability is one of the most crippling features of the disease, significantly raising the risk of falls and lowering quality of life [64-66].

Neurophysiological factors

The basal ganglia are essential for postural control due to their interactions with the thalamus, cortex, and brainstem components [50]. As detailed in the Fundamentals section, the basal ganglia are essential for selecting and executing postural strategies. In Parkinson's disease, degeneration of nigrostriatal dopaminergic neurons disrupts these circuits, resulting in aberrant basal ganglia output that impairs downstream motor regions [67-69].

Most importantly, postural instability in Parkinson's disease is characterised by dysfunction that extends beyond the dopaminergic system. Cholinergic deficiencies, especially those impacting the pedunculopontine nucleus (PPN) in the brainstem, substantially contribute to balance impairments. The PPN, with broad connections to the basal ganglia, cerebellum, and spinal cord, is important for the regulation of posture and gait. The degeneration of cholinergic neurons in the PPN correlates with falls and postural instability in Parkinson's disease, and these symptoms frequently exhibit inadequate response to dopaminergic treatment, underscoring the role of non-dopaminergic mechanisms [13,68,70-72].

As introduced in the Fundamentals section, effective balance requires dynamic sensory integration. In PD, this process is disrupted by abnormal proprioceptive processing and impaired kinaesthesia [68,73-75]. When visual input is inaccurate, patients' tendency to over-rely on visual information as a compensatory mechanism for reduced proprioceptive signals becomes problematic.

Neurobiochemical factors

The core biochemical deficit in PD is the profound depletion of dopamine in the striatum due to neurodegeneration of the substantia nigra pars compacta. This disrupts the direct and indirect pathways of the basal ganglia, leading to the classic motor symptoms. However, postural instability is particularly

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linked to non-dopaminergic systems. Degeneration of cholinergic neurons in the pedunculopontine nucleus (PPN) correlates strongly with falls and poor response to levodopa. Furthermore, noradrenergic loss from the locus ceruleus may contribute to attention deficits that impair balance under complex conditions. These biochemical failures collectively explain why postural instability in PD is often refractory to standard dopaminergic therapies.

Neurobiomechanical manifestations

Distinct biomechanical changes impact postural control as a result of the neurophysiological anomalies in PD. Rigidity, a primary characteristic of Parkinson's disease, leads to increased axial and limb stiffness, limiting the body's capacity to adjust to disturbances. This increased rigidity modifies the fundamental biomechanics of postural responses, constraining the range and adaptability of movement essential for efficient balance restoration. Bradykinesia, a key characteristic, impairs postural responses, diminishing the capacity to swiftly counteract destabilising forces [76,77].

A specific postural abnormality in Parkinson's disease is the stooping posture, which is characterised by thoracic kyphosis, flexion of the neck, and flexion of the hips and knees. This position advances the centre of mass, constricting the functional base of support and raising the risk of falls, especially in the forward direction. The biomechanical effects of this postural alignment encompass increased energy expenditure during standing and mobility, modified weight distribution, and decreased capacity to employ the ankle approach for balance regulation [78-82].

Reactive postural responses (see Fundamentals) in PD show diminished amplitude, delayed onset, and abnormal muscle sequencing, reflecting impaired basal ganglia modulation of brainstem and spinal circuits [83-85].

Multiple sclerosis

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the central nervous system that causes widespread damage to myelin sheaths and axonal loss in the brain and spine. In contrast to Parkinson's disease, which exhibits a more localised pathology at the beginning, multiple sclerosis can simultaneously impact several brain pathways, resulting in a range of postural instability symptoms [86,87].

Neurophysiological factors

The primary neurophysiological deficit in multiple sclerosis is the demyelination of nerve fibres, which disrupts neural transmission and reduces the velocity and accuracy of information transfer within the central nervous system. The demyelination process can impact any region of the central nervous system, including areas essential for postural control, such as the cerebellum, brainstem, spinal cord, and cortical regions. The resultant conduction impairments impact the integration of sensory information and the coordination of motor outputs essential for optimal balance regulation [88,89].

Sensory integration deficits in MS (see Fundamentals section) arise from demyelination at multiple levels. Dorsal column lesions disrupt proprioceptive transmission, while involvement of vestibular pathways or visual radiations can impair multiple streams of balance-relevant information [90-93]. Unlike the preferential visual dependence in PD, MS patients face multimodal sensory disruption that varies with lesion location.

In MS, fatigue is a distinctive neurophysiological factor that impacts up to 80% of individuals. A recent study indicates that fatigue substantially affects postural control in multiple sclerosis by limiting the central nervous system's capacity to regulate sensory inputs and synchronise motor responses. This decline in postural control, brought on by fatigue, may be especially troublesome when standing or walking for extended periods, thereby increasing the risk of falls throughout the day [94-96].

Neurobiochemical factors

Beyond demyelination, neurobiochemical processes significantly exacerbate postural instability in MS. Chronic inflammation, driven by cytokines such as tumour necrosis factor-alpha (TNF-α) and interleukins (IL-6), can directly impair neuronal and synaptic function, slowing conduction in surviving axons. Oxidative stress and mitochondrial damage contribute to axonal degeneration in critical sensory and motor pathways, including the dorsal columns and corticospinal tract. This biochemical milieu not only slows neural transmission but also increases the metabolic cost of neural activity, contributing to the central fatigue that profoundly degrades postural control later in the day.

Neurobiomechanical manifestations

Postural control is affected by specific biomechanical changes that are a manifestation of the neurophysiological abnormalities in multiple sclerosis. In comparison to healthy individuals, there is a notable increase in postural sway during quiet stance, as well as a greater sway area, velocity, and variability. These difficulties in regulating lateral stability are frequently more apparent in the mediolateral direction, as evidenced by the increased sway. The parameters of postural sway in multiple sclerosis differ from those in Parkinson's disease, with multiple sclerosis often exhibiting greater amplitude and lower frequency sway patterns [22,97-99].

In MS, spasticity is a prevalent symptom that presents distinctive biomechanical challenges for postural control. Increased muscle tone, particularly in the lower extremities, alters the mechanical characteristics of the musculoskeletal system, affecting the body's ability to generate suitable torques for balance regulation. Spasticity frequently results in an extended postural configuration, unlike the flexed posture observed in Parkinson's disease. This prolonged pattern may enhance stability in certain circumstances, but diminishes adaptability to fluctuating demands of the environment [100-104].

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Asymmetrical weight distribution is another characteristic biomechanical feature of MS, which is indicative of the frequently asymmetric nature of neurological involvement. This asymmetry presents difficulties in sustaining the centre of mass within the base of support, especially during dynamic activities or in response to perturbations. Increasing dependence on assistive devices or extending the range of support are examples of compensatory techniques that may offer stability in the short term but frequently limit mobility and independence in the long run [105-107].

Huntington's disease

Huntington's disease (HD) is a neurodegenerative disorder that is autosomal dominant and is caused by an expanded CAG trinucleotide repeat in the HTT gene. This repeat results in the production of abnormal huntingtin protein. Unlike Parkinson's disease and Multiple Sclerosis, HD has a clear genetic foundation with complete penetrance, which means that people who inherit the defective gene will almost certainly develop the condition if they live long enough. The condition is distinguished by a triad of motor, cognitive, and psychiatric symptoms, with postural instability as a substantial aspect of the motor manifestations [27,108,109].

Neurophysiological factors

The principal neurophysiological anomaly in Huntington's disease is the gradual degradation of striatal neurons, especially the medium spiny neurons within the caudate nucleus and putamen. This striatal disease impairs the functionality of cortico-striatal-thalamic circuits essential for motor control, encompassing the selection and execution of suitable postural strategies. As the disease advances, neurodegeneration expands beyond the striatum to involve the cerebral cortex, especially the frontal and temporal lobes, thereby significantly impairing motor planning and execution [110-112].

While both Parkinson's and Huntington's diseases involve basal ganglia dysfunction (see Fundamentals section), the mechanisms differ fundamentally. PD is characterised by increased inhibitory output from dopamine depletion, whereas HD involves a complex interplay of increased and decreased basal ganglia output due to selective striatal neuronal loss [109,113,114].

Similar to Parkinson's disease and multiple sclerosis, sensorimotor integration is impacted by Huntington's disease; however, the underlying mechanisms are different. Evidence suggests inadequate scaling of postural responses to the amplitude of disturbances, which is consistent with an altered processing of proprioceptive information. The integration of visual and vestibular inputs for balance regulation may be compromised by the cognitive impairments associated with HD, especially defects in attention and executive function that restrict the allocation of cognitive resources to postural tasks [3,115,116].

Neurobiochemical factors

HD is caused by an expanded CAG repeat in the HTT gene, leading to the production of mutant huntingtin protein. This toxic protein causes selective neurodegeneration, primarily in the striatum, through multiple biochemical pathways: mitochondrial dysfunction and energy failure, excitotoxicity from impaired glutamate handling, and transcriptional dysregulation of genes critical for neuronal survival. This striatal degeneration disrupts the fine-tuning of motor output, producing the irregular, chaotic involuntary movements that continuously challenge postural stability.

Neurobiomechanical manifestations

Neurophysiological complications in HD result in specific biomechanical changes that impact postural control. The most distinctive feature is the existence of choreiform movements, which are rapid, irregular, flowing, and involuntary movements that impact different body areas. These movements induce continuous perturbations to the body's centre of mass, necessitating continuous compensatory adjustments to maintain balance. In contrast to the rhythmic tremor associated with Parkinson's disease, the irregular pattern of choreiform movements renders them more difficult to maintain postural control. Abnormal body postures are prevalent in Huntington's disease and substantially contribute to balance deficits. These postures may involve hyperextension of the neck, trunk, and knees, resulting in an unstable alignment that displaces the centre of mass toward the boundaries of the base of support. As the condition advances, dystonic postures may emerge, further compromising biomechanical stability and limiting the spectrum of compensatory movements available for balance restoration [28,117-121].

The parameters of postural sway in Huntington's disease significantly differ from those seen in other neurodegenerative disorders. Research has shown elevated sway amplitude and velocity, characterised by irregular, unexpected patterns that indicate the presence of choreiform movements. In comparison to healthy people, the temporal structure of postural sway is also changed, becoming more random and less complicated. These atypical sway patterns are associated with disease severity and increase the elevated fall risk found in this cohort [117,122,123].

Alzheimer's disease

Alzheimer's disease (AD) is the predominant cause of dementia globally, impacting around 50 million individuals. Although AD is mostly known for its terrible effects on memory and cognition, it also causes serious problems with balance and postural control, especially as the condition progresses. In contrast to conditions like Parkinson's disease or ALS, where motor symptoms frequently manifest initially, postural instability in Alzheimer's disease generally arises later in the progression of the disease, aligning with the deterioration of cognitive function[124-126].

Neurophysiological factors

The principal neurophysiological anomalies in Alzheimer's disease are the aggregation of beta-amyloid plaques and neurofibrillary tangles formed by hyperphosphorylated tau protein. The pathogenic alterations initially impact the

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entorhinal cortex and hippocampus, regions essential for memory formation, but gradually extend to encompass cortical areas vital for sensory integration and motor planning. Consequently, the neural networks that underlie postural control are disrupted by neuronal dysfunction and mortality [127-129].

Unlike the subcortical sensory integration deficits in PD and MS, AD disrupts sensory integration at the cortical level (see Fundamentals). Atrophy of parietal and prefrontal regions impairs the formation of internal body representations and the allocation of attentional resources to balance tasks [34,130-132]. Unlike the subcortical sensory integration deficits in PD and MS, AD disrupts sensory integration at the cortical level (see Fundamentals). Atrophy of parietal and prefrontal regions impairs the formation of internal body representations and the allocation of attentional resources to balance tasks.

Neurobiochemical factors

The hallmark pathologies of AD—beta-amyloid plaques and tau neurofibrillary tangles—disrupt neural function well before cell death occurs. Soluble forms of these proteins can impair synaptic plasticity and disrupt neural network activity in the parietal and prefrontal cortices. This synaptic failure directly compromises the integration of visual, vestibular, and proprioceptive information required for postural control. Additionally, degeneration of cholinergic neurons in the basal forebrain, a key target in AD, may further impair attention to balance, particularly under dual-task conditions.

Neurobiomechanical manifestations

Neurophysiological anomalies in Alzheimer's disease result in specific biomechanical changes that influence postural control. Recent research has shown that Alzheimer's disease patients display distinct postural control characteristics that differ from those of healthy older persons and individuals with other forms of dementia. These characteristics, which are notably evident in the eyes-closed condition, include an increased postural sway area and velocity. This suggests that the ability to utilise proprioceptive and vestibular information is impaired when visual input is removed [33,133,134].

The temporal pattern of postural sway is modified in Alzheimer's disease, exhibiting less complexity and increased regularity relative to healthy ageing. This reduction in complexity signifies a diminished adaptive capacity that typically enables the postural control system to adjust flexibly to varying environmental demands. As the disease progresses, these anomalies intensify, associated with the degree of cognitive impairment [33,34].

APA deficits in AD (see Fundamentals) reflect cortical dysfunction in motor planning. Unlike the basal ganglia-mediated APA impairments in PD, AD patients struggle with the cognitive and motor planning aspects of transitional movements like sit-to-stand [60,83,135].

Amyotrophic Lateral Sclerosis (ALS)

Amyotrophic Lateral Sclerosis (ALS) is a progressive neurodegenerative disorder marked by the loss of upper and lower motor neurons, resulting in muscle weakness, atrophy, and ultimately paralysis. Although ALS is primarily recognised for its catastrophic impact on bulbar and limb function, postural instability is a substantial but frequently overlooked aspect of the disease that manifests at an early stage and frequently leads to falls [136,137].

Neurophysiological factors

The basic neurophysiological defect in ALS is the gradual degeneration of motor neurons in the primary motor cortex (upper motor neurons) and in the brainstem and spinal cord (lower motor neurons). This combined participation establishes a distinctive pattern of neurological impairment that differentiates ALS from other neurodegenerative disorders. Degeneration of upper motor neurons impairs the corticospinal and corticobulbar pathways, affecting voluntary motor control and leading to spasticity. Loss of lower motor neurons results in muscle fibre denervation, causing weakness, atrophy, and fasciculations [35,138,139].

Postural control in ALS is influenced by failure in both upper and lower motor neurons. Studies indicate that upper motor neuron pathology may be particularly significant in early balance impairments. Research has shown that patients with predominant upper motor neuron involvement experience more postural instability compared to those with primarily lower motor neuron symptoms, even when accounting for overall disease severity. This discovery indicates that the interruption of descending motor pathways from the cortex considerably affects the central processing of balancing information [137,139-141].

In addition to the loss of primary motor neurons, ALS is increasingly acknowledged as a multisystem condition that affects non-motor neural systems. A recent study has shown anomalies in sensory processing and integration, particularly with proprioceptive information, which may contribute to postural instability. In addition, there is evidence of altered central processing of balance information, which implies that postural control deficits in ALS are not solely due to muscle weakness but rather reflect more intricate changes in sensorimotor integration [142-145].

Neurobiochemical factors

ALS is a multisystem disorder driven by complex biochemical cascades. Glutamate excitotoxicity, linked to reduced expression of the astrocytic glutamate transporter EAAT2, leads to motor neuron death. Cytoplasmic mislocalization and aggregation of TDP-43 protein disrupts RNA metabolism and causes toxicity in both upper and lower motor neurons. These processes result in the combined loss of upper motor neurons (disrupting cortical motor commands) and lower motor neurons (causing muscle weakness). The resulting deficit in central motor drive, particularly to axial muscles, is a key biochemical contributor to early postural instability, independent of peripheral muscle weakness.

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Neurobiomechanical manifestations

Neurophysiological complications of ALS result in specific biomechanical changes that impact postural control. A recent study indicates that postural problems in ALS are predominantly influenced by impaired trunk control rather than by limb weakness. Research examining body segment motions during postural tasks has revealed that ALS patients have significantly reduced body excursions in reaction to tilts of the support surface, but comparatively greater upper body excursions than healthy controls. This pattern indicates a modified central control approach rather than merely a result of muscular weakness [144-148].

The characteristics of spontaneous sway in ALS are distinct from those seen in other neurodegenerative disorders. Patients with ALS generally exhibit greater sway amplitudes and velocities, along with elevated sway frequencies in comparison to healthy controls. These atypical sway patterns indicate both the fundamental neurophysiological alterations and the compensatory mechanisms employed to preserve balance despite advancing motor neuron degeneration [5,98,149].

Reactive postural responses in ALS (see Fundamentals) paradoxically show reduced body excursion. However, this reflects an inability to generate appropriate corrective torques due to upper motor neuron dysfunction, not improved stability [150-153]. This contrasts sharply with the delayed but often excessive responses seen in PD (Table 1).

Conclusion

This review has analysed the neurophysiological and neurobiomechanical determinants affecting posture and movement regulation in prominent neurodegenerative disorders. Although all exhibit postural instability, Parkinson's disease, Multiple Sclerosis, Huntington's disease, Alzheimer's disease, and Amyotrophic Lateral Sclerosis influence balance control via unique pathophysiological mechanisms, leading to disease-specific patterns of postural dysfunction.

For clinical assessment, this understanding mandates a shift from generic balance tests to mechanism-driven evaluations. Instrumented posturography, for instance, can differentiate the high-frequency, anteroposterior sway of PD from the broader, mediolateral instability of MS. In AD and PD, dual-task paradigms (e.g., walking while counting backwards) are essential to unmask the cognitive-motor interference that single-task assessments miss. For ALS, specific trunk control tests, such as the Trunk Impairment Scale, are more sensitive than standard limb strength tests for predicting early fall risk. The Sensory Organisation Test (SOT) can formally quantify the visual dependence seen in PD and AD, guiding environmental modifications.

These mechanistic insights also pave the way for more targeted rehabilitation. In PD, where APAs are deficient, interventions should focus on external cueing and amplitude training (e.g., LSVT BIG) to consciously bypass defective basal ganglia circuits. For MS patients, whose postural control

Table 1: Comparative Synthesis of Postural Control Deficits in Neurodegenerative Diseases.

Disease	Primary Neural Structures Affected	Key Neurobiochemical Factors	Sensory Integration Pattern	Typical Postural Sway Characteristics	Anticipatory vs. Reactive Deficits	Key Clinical & Rehab Implications
Parkinson's Disease	Basal ganglia (SNc), PPN	Dopamine depletion; cholinergic (PPN) loss; noradrenergic loss	Over-reliance on vision; impaired proprioception	Increased sway area; anteroposterior instability; stooped posture	APAs delayed/reduced in amplitude; reactive responses delayed & inflexible	Poor response to levodopa for balance; use external cueing & amplitude training
Multiple Sclerosis	Diffuse WM tracts (cerebellum, dorsal columns, brainstem)	Demyelination; inflammatory cytokines (TNF-α, IL-6); oxidative stress	Impaired multimodal integration; highly sensitive to fatigue	Increased sway velocity & area; mediolateral instability; variable presentation	Deficits depend on lesion location; both APAs and reactive responses can be impaired	Fall risk increases with fatigue; prioritise energy conservation & sensory reweighting training
Huntington's Disease	Striatum (caudate/ putamen)	Mutant huntingtin; mitochondrial dysfunction; excitotoxicity	Irregular scaling of sensory info; cognitive interference	Irregular, chaotic, high-amplitude sway; hyperextended posture	APAs disrupted by involuntary choreiform movements	Unpredictable falls even early on; rhythmic auditory stimulation may help
Alzheimer's Disease	Hippocampus → parietal & prefrontal cortices	Amyloid-β & tau pathology; synaptic failure; cholinergic (basal forebrain) loss	Visual dependence; impaired sensory reweighting	Increased sway with eyes closed; less complex, more regular sway	APAs impaired (motor planning deficits); reactive responses delayed	Dual-task conditions are severely destabilising; ensure adequate lighting
ALS	Upper & lower motor neurons	Glutamate excitotoxicity; TDP-43 pathology	Central proprioceptive processing was altered	Increased sway frequency; reduced perturbation response; "paradoxical" stiffness	Impaired trunk control; altered central strategies, not just weakness	Early trunk assessment is critical; focus on postural supports & stabilization

Legend: SNc: Substantia nigra pars compacta; PPN: Pedunculopontine nucleus; WM: White matter; APAs: Anticipatory postural adjustments; TNF-α: Tumour necrosis factor-alpha; IL-6: Interleukin-6.

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degrades with fatigue, rehabilitation must prioritise energy conservation and sensory integration training on compliant surfaces. In AD, where visual dependence is high, ensuring well-lit environments and avoiding divided-attention tasks is paramount, while in HD, rhythmic auditory stimulation may help regularise chaotic sway patterns. For ALS, early intervention should focus on trunk stabilization and postural supports, even before significant limb weakness is apparent.

Future research must prioritize longitudinal studies to track the progression of these domain-specific postural deficits, the development of disease-specific biomarkers derived from posturography, and, most critically, randomised controlled trials of personalized rehabilitation strategies that target the distinct neurophysiological, neurobiochemical, and biomechanical profiles of each condition.

By enhancing our comprehension of how various neurodegenerative processes disrupt the intricate systems governing postural control, we can move beyond generic fall prevention towards precise, effective interventions that improve balance, mitigate fall risk, and elevate the quality of life for individuals affected by these challenging disorders.

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