Characterization of eye movements and their impact on postural control in Parkinson's disease

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Abstract

More than 75% of individuals with Parkinson's disease (PD) exhibit impaired eye movement functions. The most frequently impacted eye movements are saccades and smooth pursuit. These oculomotor impairments have been directly associated

with postural instability in PD, reflecting a broader association between visual perception deficits and balance. This establishes a direct mechanistic link in which impaired vision exacerbates motor deficits, thereby significantly elevating the risk of falls. Despite the substantial scientific rationale supporting a relationship between eye movement impairments and reduced postural control in pwPD, a comprehensive synthesis of the current evidence on specific oculomotor deficits and their impact on postural control in PD is still lacking. Therefore, this chapter characterizes eye movements and their subsequent impact on postural control in individuals with PD. It begins by outlining the neuroanatomical regions most impacted by the disease and the manner in which these pathologies manifest as visual impairment. Specifically, this chapter discussed three primary oculomotor deficits: saccadic dysfunction, smooth pursuit eye movement, and convergence insufficiency. Subsequently, an exploration of postural control mechanisms and their degradation in PD issues, with particular emphasis on the role of dopaminergic neurodegeneration. The chapter further explored postural impairments, including deficits in static, dynamic, and anticipatory postural adjustments, as well as reactive postural responses. Finally, the critical interplay between visual input and postural control was analyzed, including the complex impact of medication. Crucially, it argues that although visual impairments often exacerbate postural instability, the visual system remains a highly plastic and effective target for rehabilitation to enhance postural control in PD.

Introduction

Parkinson's disease (PD), the second most common neurodegenerative disease, is characterized by motor impairments such as tremor, rigidity, bradykinesia, and postural instability (Magrinelli *et al.*, 2016; Yang *et al.*, 2016). In addition to these motor features, non-motor symptoms such as visual and cognitive impairments have also been reported in this population (Weil *et al.*, 2016; Ekker *et al.*, 2017). Indeed, vision is among the main contributors to quality of life (Leissner *et al.*, 2014). As PD progresses, 78% of individuals with PD (pwPD) report at least one visual symptom, such as difficulty reading, sometimes with double vision, and misjudging objects and distances (Archibald *et al.*, 2011; Urwyler *et al.*, 2014).

Anatomically, the basal ganglia regulate the oculomotor control through the substantia nigra - superior colliculus pathway. This pathway includes the pars compacta and pars reticulata. The GABAergic neurons in the pars reticulata typically inhibit neuronal activity in the superior colliculus and thalamus (DiChiara *et al.*, 1979; Yoshida & Omata, 1979; Chevalier *et al.*, 1981). These neurons act as a gate, allowing collicular burst neurons to signal the brainstem and generate saccadic eye movements (Sparks & Hartwich-Young, 1989; Moschovakis *et al.*, 1996). In the classical model, this gating process is orchestrated by two parallel circuits: the direct and indirect pathways (Calabresi *et al.*, 2014) (Figure 1).

The striatum integrates inputs from cortical regions - including the frontal, supplementary, and parietal eye fields - and modulates basal ganglia output based on dopaminergic signaling. In the direct pathway, D1-receptor neurons provide direct inhibition to the basal ganglia output nuclei, which are the internal globus pallidus (GPi) and the substantia nigra pars reticulata (SNr). Conversely, the indirect pathway involves D2-receptor neurons that modulate

the GPi/SNr via external globus pallidus and subthalamic nucleus (DeLong & Wichmann, 2007). The SNr then inhibits the superior colliculus. In PD, dopaminergic depletion disrupts this balance, resulting in a shift toward indirect pathway dominance. The resulting SNr hyperactivity leads to excessive superior colliculus inhibition, likely driving the oculomotor deficits characteristic of the disease (Terao *et al.*, 2013).

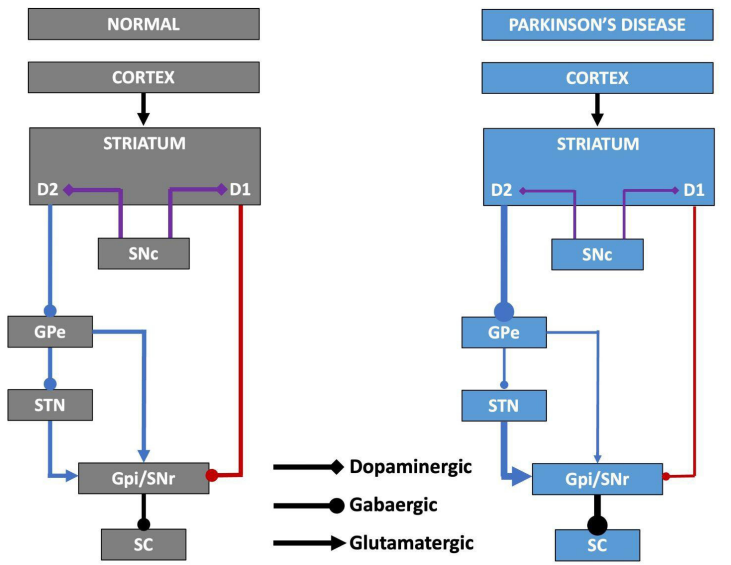

Figure 1. Basal ganglia circuitry in neurologically healthy individuals (gray) and individuals with PD (blue). Cortical inputs converge on the striatum, where the substantia nigra pars compacta dopamine (violet connectors) targets the D1 (direct pathway—red connectors) and D2 (indirect pathway—blue connectors) receptors. The direct pathway has been shown to inhibit the internal globus pallidus/ substantia nigra reticulata, while the indirect pathway modulates it via the external globus and subthalamic nucleus. In PD, dopaminergic depletion triggers a functional imbalance, characterized by reduced inhibition through the direct pathway and increased excitation via the indirect pathway. This imbalance results in excessive substantia nigra reticulata inhibition onto the superior colliculus. SNc: substantia nigra pars compacta; GPe: external globus pallidus, STN: subthalamic nucleus; GPi: internal globus pallidus; SNr: substantia nigra reticulata; SC: superior colliculus.

Functionally, deficits in eye movements have been reported in more than 75% of pwPD (Armstrong, 2015; Frei, 2021). The most frequently impacted oculomotor functions are saccades (rapid eye movements utilized to transition between targets) and smooth pursuit eye movement (SPEM) (movements that maintain a static object's fixation on the retina) (Armstrong, 2015; Frei, 2021). Saccade abnormalities include hypometria and delayed initiation, while SPEM is often slow and saccadic (Armstrong, 2015; Frei, 2021). These oculomotor impairments indicate an underlying dysfunction with the oculomotor frontal-subcortical circuits, which are closely linked to visuospatial processing, working memory, and execution (Ghazi-Saidi, 2020).

These oculomotor impairments have been directly associated with postural instability in PD (Uc *et al.*, 2009; Hamedani *et al.*, 2019), reflecting a broader association between visual perception deficits and balance control (Lee *et al.*, 2025). As pwPD increasingly rely on visual information to compensate for their impaired postural sway control (Rinalduzzi *et al.*, 1995), poor oculomotor coordination has the potential to exacerbate their instability. Specifically, deficits in saccades and SPEM disrupt the stable processing of visual information necessary for balance (Armstrong, 2011; Frei, 2021). This creates a direct mechanistic link where impaired vision compounds motor deficits, significantly elevating the risk of falls (Lord *et al.*, 1994, 1999).

Despite the substantial scientific rationale supporting a relationship between eye movement impairments and reduced postural control in pwPD, a comprehensive synthesis of the current evidence on specific oculomotor deficits and their impact on postural control in PD is still lacking. Therefore, this chapter aims to characterize the main eye movement impairments in pwPD, and to present their relationship with postural control. Therefore, the chapter is divided into three parts. First, a

detailed description of the primary eye movement–related visual symptoms observed in pwPD is provided. Secondly, the definition of postural control is provided, along with an exposition of the deficits associated with PD. Finally, the discussion will address the influence of eye movement impairments on overall postural control in pwPD.

Vision and Parkinson's Disease

Visual dysfunction is a highly prevalent non-motor symptom in PD that directly compromises the visual guidance required for maintaining balance. These deficits manifest in a range of symptoms, including oculomotor impairments (eye movement control) and sensory and perceptual losses. This section will detail these common visual deficits in pwPD (Figure 2).

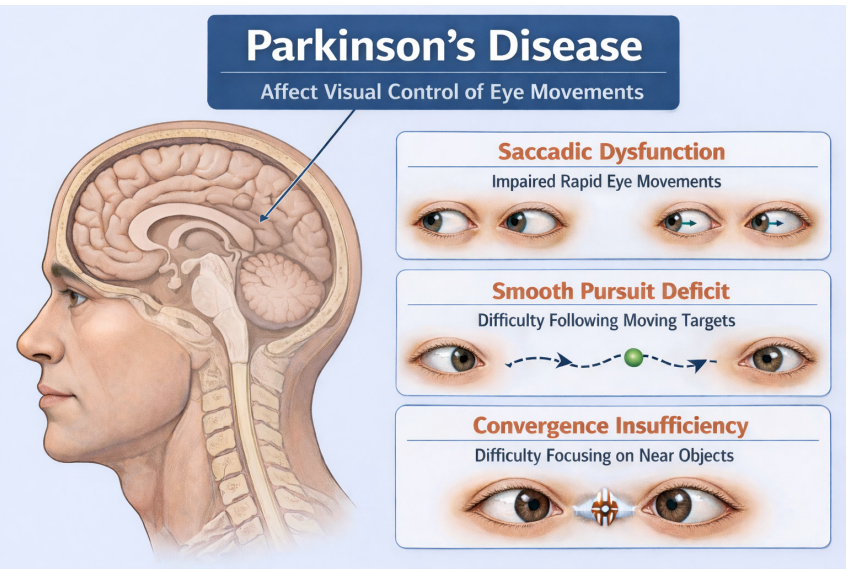

Figure 2. Changes in the brain's basal ganglia can interfere with visual control of the eye movements: saccadic, smooth pursuit, and convergence insufficiency. Adapted from an image generated by OpenAI (2026).

Oculomotor (eye movement) abnormalities

Saccadic

Saccadic movements, defined as the ballistic eye movements that rapidly redirect the high acuity fovea of the retina toward new visual targets, have been employed as a diagnostic tool in PD. Comparing the saccadic direction, the vertical eye movements are more challenging than the horizontal eye movements, resulting in a delayed gaze response (Waldthaler *et al.*, 2019). Clinically, these movements helped differentiate PD from atypical Parkinsonian syndromes (Termsarasab *et al.*, 2015), where up to 75% of pwPD will exhibit abnormal saccadic and SPEM (Shibasaki *et al.*, 1979). Specifically, pwPD exhibited reduced saccade amplitude, prolonged latencies, and longer time to achieve a specific target (Mosimann *et al.*, 2005; Terao *et al.*, 2013). The impairment of antisaccade latencies has been related to an indirect marker of impaired anticipatory postural adjustments (Ewenczyk *et al.*, 2017). As the disease progressed, both saccade amplitude and latency got worse (Terao *et al.*, 2011). Furthermore, compared with neurologically healthy controls, pwPD presented more directional antisaccade errors (Briand *et al.*, 1999), along with a notable decrease in visually guided saccades, particularly in the upward hypometric direction (Waldthaler *et al.*, 2019). This reduction in control forces pwPD to execute multiple correction saccades to reach a visual target (Kimmig *et al.*, 2002).

Anatomically, saccadic impairments involve the brainstem and the basal ganglia levels (Ewenczyk *et al.*, 2017) that mediate saccade amplitude and latency (Terao *et al.*, 2013), while the cerebellum is involved in saccade accuracy (Beh *et al.*, 2017). Functionally, saccadic eye movements facilitate the accurate shifting of gaze, a process that is critical for visual perception

and motor control (Stoffregen *et al.*, 2006). In PD, the loss of dopaminergic neurons disrupts this circuitry. The resulting depletion of dopamine causes a shift in the basal ganglia's output: increased activity in the indirect pathway (via the subthalamic nucleus) combined with reduced activity in the direct pathway leads to excessive inhibition of SNr/GPi (DeLong & Wichmann, 2007). This over-inhibition is the primary driver of abnormal saccadic performance in PD.

The role of levodopa in the management of this visual impairment remains unclear and controversial. While certain studies have documented prolonged prosaccade latencies (Michell *et al.*, 2006; Hood *et al.*, 2007), other investigations have failed to show any significant effect from dopaminergic medication (Nakamura *et al.*, 1991). Conversely, several studies have demonstrated that medication can enhance saccadic parameters by decreasing error rates in voluntary saccadic tasks and enhancing prosaccade accuracy and amplitude (Gibson *et al.*, 1987; Rascol *et al.*, 1989; Hood *et al.*, 2007).

Smooth Pursuit Eye Movements

SPEM, which enable an individual to maintain a moving object's fixation on the fovea, represent a prevalent and early oculomotor deficit in PD (Armstrong, 2015). These deficits are highly prevalent, affecting up to 67% of pwPD compared to only 20% of neurologically healthy individuals (Shibasaki *et al.*, 1979). This impairment frequently complicates the maintenance of repetitive actions for individuals with PD, resulting in a diminution of magnitude of the eye response and an overall decline as the stimulus is repeated (Armstrong, 2015).

Anatomically, the basal ganglia have been demonstrated to play a role in efficient and automatic SPEM performance (Fukushima *et al.*, 2017). Specifically, the SNr modulates both

saccades and SPEM (Basso *et al.*, 2005). These deficits manifest as impaired movement speed, latency, and accuracy (Frei, 2021). Quantitatively, pwPD demonstrated reduced SPEM gain (eye velocity relative to target velocity) (Frei, 2021), suggesting a form of ocular bradykinesia - or slowness of eye movement (Shibasaki *et al.*, 1979). Furthermore, the initiation of SPEM is often accompanied by a prolonged latency period (Rottach *et al.*, 1996; Ladda *et al.*, 2008; Helmchen *et al.*, 2012), and the correlation between eye movements and target movement (accuracy) is less precise in pwPD (Bareš *et al.*, 2003). As stated previously, SPEM are imperative for stabilizing the visual scene. The resultant oculomotor bradykinesia—which is characterized by reduced gain and accuracy—directly compromises visual input. This, in turn, exacerbates postural instability and increases the risk of falls. Furthermore, as the PD progresses, SPEM velocity generally tends to decrease (Rascol *et al.*, 1989; Lekwuwa *et al.*, 1999).

It is noteworthy that the direction of SPEM in PD is of particular interest, as abnormalities are more pronounced in the vertical direction (up and down) than in the horizontal direction. Abnormalities in the vertical up direction were found in 54% of cases, while 50% exhibited abnormalities in the vertical down direction, compared to 37% in the horizontal direction (Corin *et al.*, 1972). Moreover, deficits in the SPEM are amplified during higher-level tasks, such as tracking a remembered target or relying on visual cues for target selection (Fukushima *et al.*, 2015). With regard to drug treatment, the role of levodopa in SPEM remains inconclusive. Some studies have demonstrated improvement in SPEM with dopaminergic medication (Corin *et al.*, 1972; Bareš *et al.*, 2003; Marino *et al.*, 2010), while others have reported no significant difference (Rascol *et al.*, 1989; Ladda *et al.*, 2008).

Convergence Insufficiency

Convergence insufficiency (CI) and its associated abnormalities are common and well-documented features in PD (Ekker *et al.*, 2017; Pretegiani & Optican, 2017; Savitt & Aouchiche, 2020; Sun *et al.*, 2023). Vergence —defined as the simultaneous movement of the two eyes in opposite directions—is deficient in PD, thereby compromising depth perception and spatial navigation (Sun *et al.*, 2023). Vergence abnormality may result in strabismus and blurred or double vision (diplopia) (Savitt & Aouchiche, 2020; Sun *et al.*, 2023). Vergence abnormalities in pwPD include increased latency (which is longer in vergence of eye movement), decreased velocity, and decreased gain (amplitude/accuracy) (Sun *et al.*, 2023). PwPD appears to affect disparity-driven vergence significantly differently when compared to neurologically healthy individuals (Sun *et al.*, 2023). However, reported that blur-driven vergence is comparable between neurologically healthy individuals and pwPD (Sun *et al.*, 2023). Furthermore, as a compensatory strategy, pwPD tends to utilize saccadic movements to compensate for deficits in disparity-driven vergence (Sun *et al.*, 2023). The decreasing vergence velocity gain in PD has been found to correlate with the propensity for generating these compensatory saccadic eye movements (Sun *et al.*, 2023), which may affect postural control.

Postural Control and Parkinson's disease

Postural control is a complex sensorimotor process that integrates visual, vestibular, and proprioceptive information to maintain balance and body stability (Rinalduzzi *et al.*, 2015; Feller *et al.*, 2019). In PD, this control system is significantly impaired due to neurodegenerative changes that primarily affect the basal ganglia, which are fundamental structures for the automatic

regulation of posture and movement (Rinalduzzi *et al.*, 2015; Takakusaki *et al.*, 2022). The degeneration of dopaminergic neurons in the substantia nigra pars compacta alters striatal neuronal activity and its downstream targets within the thalamo-cortical circuitry of the basal ganglia, leading to the hallmark motor symptoms of the disease (Takakusaki *et al.*, 2022; Bath & Wang, 2024). In addition, the basal ganglia have shown to have downstream GABAergic projections to the thalamus and brainstem, including the pedunculopontine nucleus. These projections have been found to send cholinergic outputs to several motor control centers (Bath & Wang, 2024).

Deficits in postural control in PD manifest across multiple dimensions, including static, dynamic, anticipatory, and reactive control (Rinalduzzi *et al.*, 2015; Bath & Wang, 2024). During static postural control (quiet standing), pwPD exhibit significant variations in postural sway, characterized by augmented displacement in the anterior–posterior and medio-lateral directions when compared with neurologically healthy individuals (Bath & Wang, 2024). These sway metrics have been associated with disease progression and with scores on the MDS-UPDRS (Bath & Wang, 2024). Additional alterations in static postural alignment frequently observed in PD, such as truncal rigidity, stooped posture, and increased co-activation of trunk and lower limb muscles, further contribute to dysfunctional postural responses (Bath & Wang, 2024).

A particularly affected domain in PD involves anticipatory postural adjustments (APAs), which consist of stereotyped and highly regulated muscle activations that shift the center of pressure (CoP) toward the swing leg while stabilizing the body's center of mass over the stance leg in preparation for stepping or turning (Bath & Wang, 2024; Hou *et al.*, 2024; Seuthe *et al.*, 2024). Force plate and inertial sensor assessments of APAs during gait

initiation frequently indicate that pwPD present variable and hypometric APAs with dysfunctional timing (Faria *et al.*, 2023; Bath & Wang, 2024; Hou *et al.*, 2024; Seuthe *et al.*, 2024). This dysfunction is directly linked to the phenomenon of freezing of gait (FOG). PD individuals who experienced FOG demonstrated poorer postural control compared with those who do not experience FOG and with neurologically healthy controls (Hou *et al.*, 2024; Onuma *et al.*, 2024). Evidence further suggests that delayed and reduced-magnitude APAs are particularly evident in individuals with FOG, indicating that the APA ratio may serve as a potential biomarker of postural adjustment capacity (Onuma *et al.*, 2024).

Reactive postural responses are also significantly impaired in PD, as evidenced by weakened and delayed automatic responses to postural perturbations, fragmented muscle activation patterns, and disrupted sequencing of muscle recruitment (Rinalduzzi *et al.*, 2015). When exposed to perturbations, pwPD demonstrate increased amplitude of the medium-latency response, which correlates with disease severity, as well as an earlier onset of the long-latency response (Rinalduzzi *et al.*, 2015). The standard sequence of recruitment, from distal to proximal, is inverted, with the activation of the hip muscles preceding that of the ankle muscles. This reversal has been shown to increase limb rigidity and prevent appropriate corrective movements (Rinalduzzi *et al.*, 2015). Consequently, this altered muscle activation sequence leads to less effective postural responses, exacerbating postural instability and fall risk (Rinalduzzi *et al.*, 2015).

Sensory integration, which is crucial for postural control, is also profoundly altered in PD. PwPD display greater reliance on visual information and are often unable to maintain balance when visual cues are absent, unreliable, or in conflict with vestibular and proprioceptive inputs (Vaugoyeau *et al.*, 2007; Rinalduzzi *et al.*, 2015; Feller *et al.*, 2019). Proprioceptive deficits are particularly

evident, with studies showing that PD is associated with impaired proprioception, which may represent a key factor contributing to postural instability (Vaugoyeau *et al.*, 2007; Bekkers *et al.*, 2014). The reliance on vision observed in pwPD may be understood as an adaptive strategy that partially compensates for impaired proprioception (Vaugoyeau *et al.*, 2007). Specifically, investigations into proprioceptive integration have revealed that pwPD exhibit altered kinesthesia of the upper limbs, head, and trunk. These alterations are characterized by a higher threshold for the minimal detectable range of motion and a reduced ability to perceive movement direction (Rinalduzzi *et al.*, 2015).

Axial rigidity is another significant contributor to postural deficits in PD, interfering particularly with automatic activities typically performed without conscious effort (Rinalduzzi *et al.*, 2015). Within the axial regions, the neck assumes a pivotal role in maintaining balance, mobility, and coordination. A considerable number of falls are attributed to sudden changes in postural orientation, such as turning, and are associated with an inflexible control of axial postural tone (Rinalduzzi *et al.*, 2015). Alterations in ankle muscle strength and rigidity, in conjunction with distorted perceptions of stability limits, further contribute to impaired postural control (Rinalduzzi *et al.*, 2015). Axial rigidity has also been demonstrated to impede the capacity for expeditious adaptation of postural responses to fluctuating environmental conditions, culminating in inflexible postural behavior that is characteristic of numerous pwPD (Rinalduzzi *et al.*, 2015).

The neural mechanisms underlying postural control deficits in PD involve multiple cortical and subcortical networks (Bath & Wang, 2024). The loss of dopaminergic neurons in the substantia nigra pars compacta alters striatal activity and its downstream targets, affecting thalamo-cortical circuits that are essential for automatic motor control (Bath & Wang, 2024). Furthermore, the

disruption of downstream GABAergic connections from the basal ganglia to the thalamus and brainstem, including projections to the pedunculopontine nucleus, are disrupted, leading to dysfunction of brainstem locomotor centers (Bath & Wang, 2024). Functional neuroimaging studies show that pwPD with postural instability exhibit heightened activation in the prefrontal and parietal regions of the brain during anticipatory postural tasks. This finding suggests the presence of compensatory mechanisms and altered connectivity between the frontoparietal and ventral attention networks (Bath & Wang, 2024).

Levodopa remains the gold standard strategy for managing motor symptoms. However, its effects on postural strategies during quiet stance remain to be elucidated. A comparison of the ON and OFF medication states reveals that sway dispersion is higher and more pronounced in the more severe PD group compared to the mild group (Baston *et al.*, 2016). As indicated by the research of Bronte-Stewart (2002) and Bonnet *et al.* (2017), an elevated incidence of body sways has been documented in patients with PD during periods of static standing subsequent to levodopa administration. A body's increased propensity for oscillation has been demonstrated to be a significant predictor of an elevated risk of falling (Revilla *et al.*, 2013). However, an increase in cortical activity has been observed from 60 minutes to 120 minutes after medication intake, which appears to restore the thalamocortical pathway by enhancing basal ganglia function, thereby optimizing postural control (Araújo-Silva *et al.*, 2022). Furthermore, the effects of visual tasks, such as a single gaze shift, on mediolateral postural coordination vary by medication state. During the ON state, patients with Parkinson's disease (PD) rotate their trunk and head less than controls while rotating their eyes more. In contrast, in the OFF state, they tend to track targets

by moving their entire body, while controls primarily turn their head (Anastasopoulos *et al.*, 2011; Bonnet *et al.*, 2015).

These medication-dependent shifts in postural control suggest that the integration of visual cues and motor output is not merely a mechanical issue, but also a disease's underlying pathophysiology. Indeed, the relationship between postural control and the visual system in PD is well established through multiple neural and functional mechanisms, which will be addressed in the following section (Vaugoyeau *et al.*, 2007; Rinalduzzi *et al.*, 2015; Feller *et al.*, 2019). The increased reliance on visual information to compensate for vestibular and proprioceptive deficits, combined with the oculomotor impairments characteristic of PD, creates a multifaceted scenario in which vision concurrently facilitates and restricts effective postural control. This bidirectional interaction between vision and postural regulation is critical for understanding the mechanisms of postural instability and for developing more effective therapeutic strategies in PD. Visual deficits, including hypometric saccades, impaired smooth pursuit, and convergence insufficiency, may impede patients' capacity to utilize visual information as a compensatory strategy, potentially exacerbating postural instability and augmenting the risk of falls.

Interaction between eye movements and postural control in PD

A contemporary overview of the relationship between vision and postural control was provided by means of a comprehensive search of the PubMed/NCBI database. The search was constrained to original, full-length studies published between 2000 and 2025. The inclusion criteria for studies were the publication of English-language articles that assessed

balance-related outcomes. The exclusion criteria encompassed review articles, conference proceedings, abstracts, letters, case series, pilot studies, and studies involving participants without a confirmed PD diagnosis. The search strategy combined three primary thematic clusters: Parkinson, postural control (e.g., body sway, CoP, falls), and visual/oculomotor function (e.g., saccades, vestibulo-ocular reflex, eye tracking).

A comprehensive review of the extant literature yielded a multifaceted interplay between sensory systems. Specifically, the role of eye movements in postural control among pwPD has been widely examined using paradigms that manipulate visual input (i.e., visual information) and assess postural control dynamics (Figure 3, Table 1). Overall, the findings indicate a dual role of vision in postural tasks: under certain conditions, visual information contributes to the reduction of postural instability; in other conditions, deficits in sensory integration caused by PD limit its effectiveness.

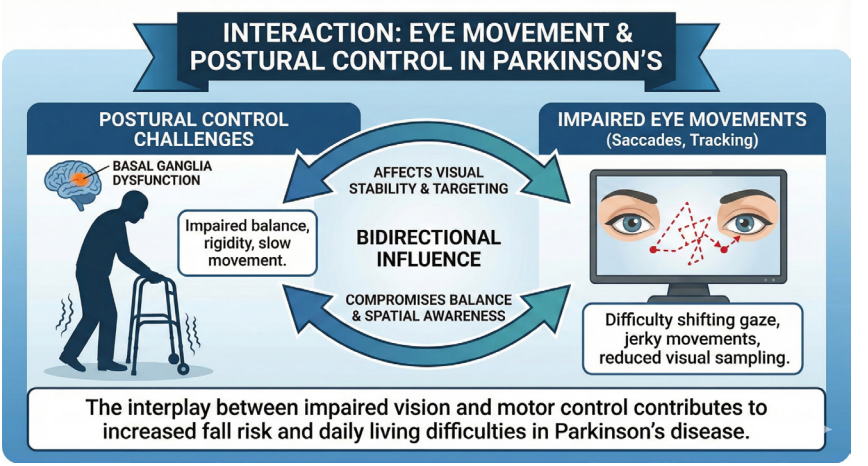

Figure 3. The bidirectional influence of basal ganglia dysfunction on motor and visual systems. This image has been adapted from a model generated by Google's AI model in 2023..

Table 1. Objective and main findings of the studies that investigated the eye movements during postural control tasks in people with PD.

Study	Groups (n)	Objective	Main findings
Smiley-Oyen *et al.* (2002)	PD = 8 CG = 8 YG = 8	To determine the capacity of PD to adapt the postural control when faced with conflicting proprioceptive inputs	- No group x vision interaction effect
Blaszczyk and Orawiec (2011)	PD = 55 CG = 55	To determine the diagnostic utility of the SR in identifying postural instability	- PD - Higher AP and ML sway ratios - EC - Increased SR
Vaugoyeau *et al.* (2011)	PD = 11 CG = 10	To establish the relationship between proprioceptive impairments and postural deficits	- PD - Fewer ability to respond to platform perturbations - PD - With EO the ability to respond to oscillations improved compared to EC
Rabin *et al.* (2013)	PD = 13 CG = 13	To test the efficacy of haptic and proprioception feedback in improving balance control	- PD swayed more than CG in the ML - Visual feedback (eyes open) decreased the ML and AP mean sway amplitude in PD group
Rocchi *et al.* (2014)	PD = 70 CG = 15	To characterize motor subtypes of PD	- EC - CG increased velocity of the sway more than PD in the ML and AP directions
Lahr *et al.* (2015)	PD-D = 9 PD-ND = 9	To assess the interaction between disease lateralization and visual reliance on postural control	- PD-D and PD-ND - Higher total CoP velocity with EC compared to EO - PD-D - Higher total CoP velocity than PD-ND with EC - PD-D and PD-ND - Higher CoP area with EC compared to EO

Study	Groups (n)	Objective	Main findings
Ozinga *et al.* (2015)	PD = 17 CG = 17	To validate the efficacy of tablet-based kinematic data in characterize postural stability	- Double leg stance with EO on a foam surface, and double leg stance with EC on a firm and foam showed great discrimination between PD and CG
Bekkers *et al.* (2018)	PD+FoG = 19 PD-FoG = 14 CG = 28	To compare the impact of dual-tasking on postural control between PD+FoG and PD-FoG	- No group x vision interaction effects during single task - EC - Higher postural DT cost to PD+FoG compared to PD-FoG
Bzdúšková *et al.* (2018)	PD = 13 CG = 13 YG = 13	To clarify the effects of age and PD on postural recovery following muscle vibration	- Larger backward CoP displacement and trunk tilts with EC com to EO (regardless of age and PD) - PD - Shifted their CoP and trunk tilts during vibration with EO - PD - Increased peak-to-peak amplitude in CoP displacement with vibration with EC - PD - Increased peak-to-peak amplitude in upper trunk level with vibration with EC and EO
Cruz *et al.* (2018)	PD = 14 CG = 14	To compare postural control performance and visual information on postural control	- PD - Larger body sway amplitude with stationary room - PD - Less coherence of body sway and visual manipulation compared to CG -PD and CG had similar visual-body sway coupling structure

Study	Groups (n)	Objective	Main findings
Mirahmadi *et al.* (2018)	PD = 17 CG = 17	To evaluate postural stability in early-stage PD with both linear and nonlinear approaches	- PD - Higher path length AP and ML - PD - Higher velocity AP and ML - PD - Higher RMS AP and ML direction - EC - Higher path length AP and ML - EC - Higher velocity AP and ML - No group x vision interaction
Bonnet *et al.* (2020)	PD = 20 CG = 20	To determine PD-related impairment in visual and postural movements during quiet upright stance	- PD - Stronger and weaker correlations between eye and CoP - PD - Used lower and greater attentional resources
Cruz *et al.* (2020)	PD = 21 CG = 21	To evaluate how knowledge and intention influence both postural performance and the visuomotor coupling between visual input and body sway	- PD - Higher MSA to CG with visual manipulation (optic flow manipulated in a moving room)
Delafontaine *et al.* (2020)	PD = 15 CG = 15	To evaluate the impact of visual availability and cognitive load on postural stability	- PD - Longer CoP pathway with EC - PD - Greater ellipse area with EC - PD - Larger RMS AP and ML amplitude with EC
Bonnet *et al.* (2021)	PD = 20 CG = 20	To determine the extent to which PD disrupts the synergistic coupling between the visual and posture systems	- PD - Eye movements and postural movements (lower back, upper back, and head) are correlated in a precise search task

Study	Groups (n)	Objective	Main findings
Cruz *et al.* (2021)	PD = 21 CG = 21	To examine how the complexity and predictability of visual information constrain postural oscillations during an upright stance	- PD - Larger sway magnitude with complex condition compared to CG - PD - Lagged behind the moving room with simple condition compared to CG
Kahya *et al.* (2021)	PD = 33 CG = 35	To assess pupillary response as a marker of cognitive effort under varying postural demands and visual conditions	- PD - Higher pupillary response with increased postural demand compared to CG (single balance eyes occluded, dual task EO and dual task eyes occluded)
Patel *et al.* (2021)	PD = 10 CG = 17 YG = 25	To determine the extent to which DBS and visual availability modify postural adjustments during quiet standing and balance perturbation	- PD - Increased synchronicity (coupled movement) with EC compared to EO (PD vs. CG; PD vs. YC)
Piras *et al.* (2022)	PD = 11 CG = 10	To characterize the postural response to radial expanding optic flow stimuli during standing	- PD - Lower microsaccade amplitude (t-test baseline and fovea stimuli) - PD - Slower microsaccade peak velocity (t-test baseline and fovea stimuli) - PD - Higher ML oscillation (t-test full and fovea stimuli) - PD - Higher sway area (t-test full, fovea, and periphery stimuli)

Study	Groups (n)	Objective	Main findings
Barbieri *et al.* (2024)	PD = 10 CG = 11	To investigate the effects of saccadic eye movements on body sway in PD in two bases of support positions (side-by-side and tandem stances)	- PD and CG - Lower AP displacement and RMS during horizontal saccadic movement compared to the fixation condition - PD - Higher sway area during vertical compared to horizontal saccadic eye movements
Kechabia *et al.* (2025)	PD = 19 CG = 20	To investigate the impact of PD on the coordination between gaze shift, postural sway and mental workload while performing visual tasks in the standing position	- PD - Higher velocity in gaze shift - PD - Higher velocity of postural sway - PD - Higher SD amplitude of gaze shift and postural sway

Note: AP: anterior-posterior; **CG:** control group of older healthy individuals; **CoP**: center of pressure; **D**: dominant; **DBS**: deep brain stimulation; **DT**: dual-task; **EC**: eyes closed; **EO**: eyes open; **+FoG**: presence of freezing of gait; **-FoG**: absence of freezing of gait; **ML**: medial-lateral; **MSA**: mean sway amplitude; **ND**: non-dominant; **PD**: Parkinson's disease group; **RMS**: root mean square; **SD**: standard deviation; **SR**: sway ratio; **YC**: young control group

Eye movements during postural control

Evidence suggests that eye movements play a pivotal role in postural regulation among pwPD. Gaze behavior is inherently associated with body sway, with eye movements exhibiting a strong correlation with postural adjustments of the head, trunk, and lower back during visual search (Bonnet *et al.*, 2021). However, PD is distinguished by particular oculomotor impairments that have the potential to compromise this coordination. Previous studies show that pwPD exhibit diminished microsaccade amplitude and velocity. This decline

corresponds with an augmentation in postural instability across a range of visual conditions (Piras *et al.*, 2022). This assertion is further substantiated by the observation of heightened variability and velocity in both gaze shifts and body sway (Kechabia *et al.*, 2025), which points to an unstable visuomotor connection. Additionally, an enhanced synchronicity between ocular and postural movements during eyes-closed conditions has been documented (Patel *et al.*, 2021), suggesting a possible maladaptive strategy characterized by over-coupling rather than flexible sensory integration. These findings suggest that altered oculomotor control is not merely indicative of PD-related instability; rather, it is a contributing factor to the observed instability. This underscores the promise of interventions that focus on eye movement training, microsaccade control, and gaze stability as a means of indirectly enhancing balance.

While oculomotor deficits provide a potential explanation for the observed instability, another critical factor that must be considered is the manner in which the central nervous system integrates visual cues. The extant literature suggests that patients with PD exhibit significant impairments in the central processing and integration of visual information necessary for effective postural control. Reduced vision efficiency has been identified as a critical concern. Research findings indicate that pwPD maintain greater sway and CoP velocity compared to neurologically healthy controls, irrespective of whether their eyes were open or closed (Blaszczyk & Orawiec, 2011; Mirahmadi *et al.*, 2018), suggesting a potential reduction in visual integration efficiency. Additionally, older healthy subjects demonstrated a more significant increase in sway velocity during eyes-closed conditions compared to pwPD (Rocchi *et al.*, 2014), suggesting that pwPD may not fully employ visual feedback to adjust their postural responses. This processing deficit becomes especially

apparent under dynamic visual perturbations. When exposed to moving rooms or optic flow manipulations, patients with pwPD exhibit increased body sway, diminished coherence between sway and visual stimuli, and delayed responses to visual perturbations compared to neurologically healthy controls (Cruz *et al.*, 2018, 2020, 2021). These results underscore a compromised visuomotor coupling, thereby impeding the effective utilization of visual cues for balance stabilization.

Oculomotor deficits may further contribute to these impairments. Reduced microsaccade amplitude and slower peak velocity are associated with increased sway magnitude across different visual field conditions (Piras *et al.*, 2022). Additionally, pwPD exhibit abnormal visuospatial responses during proprioceptive and vibratory perturbations, manifesting larger CoP displacements and trunk tilts under both eyes open and eyes closed conditions (Bzdúšková *et al.*, 2018). Furthermore, altered correlations between eye and postural movements indicate that pwPD stabilize posture through both increased and decreased attentional resources to achieve stabilization (Bonnet *et al.*, 2020). Consequently, these findings align with the hypothesis that, while vision provides critical input, individuals with pwPD frequently demonstrate deficiencies in integrating visual and proprioceptive information, resulting in less efficient adaptive postural control.

Positive effects of visual input on postural control

Visual input functions as a compensatory mechanism that mitigates balance impairments in pwPD. Literature has demonstrated that visual feedback significantly reduces sway amplitude in both the anterior-posterior and medial-lateral directions. This effect has been observed to effectively narrow

the balance difference between pwPD and neurologically healthy controls (Rabin *et al.*, 2013). Beyond static visual feedback, specific oculomotor patterns have been shown to significantly modulate postural instability. Barbieri *et al.* (2024) found that horizontal saccades reduced anterior-posterior displacement and root mean squared values compared to fixation, while also producing a smaller sway area than vertical saccades. This stabilization may stem from a shift in postural regulation toward subcortical structures (Stoffregen *et al.*, 1999) - specifically the brainstem and cerebellum - while other lower structures manage oculomotor control. Consequently, pwPD are also able to maintain postural stability comparable to neurologically healthy controls in a quiet stance task. The execution of horizontal saccadic eye movements may serve as a compensatory mechanism, circumventing basal ganglia dysfunction to enhance both gaze shifting and postural control. This stabilizing influence has been observed in dynamic tasks as well, where visual input has been shown to enhance the ability of people with PD to respond to platform oscillations. This suggests that visual input may serve as a partial compensatory mechanism for proprioceptive deficits (Vaugoyeau *et al.*, 2011).

The stabilizing role of vision is reinforced by studies using closed-eye conditions, which reveal a marked deterioration in postural control. In the event of vision obstruction, pwPD demonstrate elevated CoP velocity and augmented sway area, a phenomenon that is particularly pronounced in individuals with dyskinesia (Lahr *et al.*, 2015; Delafontaine *et al.*, 2020). Notably, the presence of vision has been shown to reduce sway magnitude and to facilitate eye–posture coupling. A robust correlation has been demonstrated between eye movements and postural adjustments across the head, upper back, and lower back during visual search, underscoring a functional visuomotor linkage (Bonnet *et al.*, 2021). Furthermore, visual information

appears to engage attentional resources, thereby contributing to the stabilization of posture. In individuals diagnosed with pwPD, elevated postural demands—particularly during dual-task scenarios—have been demonstrated to be associated with a substantially heightened pupillary response (Kahya *et al.*, 2021). This finding suggests that the visual system's demand for cognitive processing may function as a conscious or unconscious strategy to actively support compromised balance (Kahya *et al.*, 2021). Taken together, these findings underscore that vision can serve as an effective compensatory mechanism to mitigate balance impairments in PD, particularly under challenging conditions.

Clinical implications and future directions

The dual role of eye movement in pwPD underscores the necessity for customized rehabilitation strategies that incorporate visual optimization to enhance postural stability. Visual feedback can be leveraged to enhance training through a variety of methods, including augmented reality, optic flow manipulations, and gaze-control exercises aimed at improving visuomotor coupling. Given that postural control in PD is increasingly dependent on a stable visual frame, rehabilitating oculomotor impairments does not merely improve vision in isolation; it directly enhances balance control. However, clinicians must consider that excessive reliance on visual cues may be detrimental in complex or dynamic environments, where multisensory integration is required. Consequently, training programs that integrate oculomotor training within balanced tasks, particularly in dual-tasking contexts, appear to be the most efficacious. These interventions are designed to emulate the multisensory demands of daily life, thereby ensuring that

enhanced ocular motor function translates into a more robust and stable postural system in PD.

Emerging technologies, including wearable eye-trackers, virtual reality systems, and multisensory training platforms, hold considerable potential for enhancing both research and rehabilitation methodologies. These tools may facilitate the elucidation of the causal role of eye movements in stabilizing posture and identify individualized training approaches that fortify visuomotor coupling. Future intervention should aim to not only exploit the compensatory benefits of visual input but also address the sensory integration deficits that limit its stabilizing potential in PD.

Final remarks

This chapter synthesizes the complex dual relationship between oculomotor and postural controls in pwPD. Evidence establishes an anatomical and functional link rooted in basal ganglia circuits in both eye-movement control (saccades, smooth pursuit eye movements, and convergence insufficiency) and axial posture. This relationship is characterized by a critical duality: visual input frequently functions as a compensatory mechanism, helping to attenuate postural instability. However, it can also result in impairment in the processing and integration of visual information, particularly during dynamic visual perturbations. It has been demonstrated that compromised visuomotor coupling results in less efficient adaptive postural control. This is evidenced by greater body sway and delayed response times. Consequently, interventions must be meticulously designed to enhance visuomotor coupling and optimize sensory integration efficiency. This can be achieved by integrating eye movement

training with dynamic balance tasks. However, a significant lacuna in the extant literature pertains to the necessity of establishing unambiguous correlations between particular oculomotor deficits and pivotal postural control metrics. This is imperative for the efficacious management of postural instability and the mitigation of fall risk in pwPD.

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