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Postural Control in Individuals with Parkinson's Disease

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Abstract

Parkinson's disease is the second most common neurodegenerative disorder in the elderly population. It is a complex, progressive, multisystem disease associated with motor and nonmotor impairments. Postural instability is a crucial component of functional mobility, often overlooked by both clinicians and patients with Parkinson's disease. It is a refractory drug complication for which rehabilitation is the most effective nonpharmacological aid. However, many interventions are based on empirical experience. Improving knowledge on the pathophysiology of postural control disorders is crucial to understand the multifaceted components affected and thus design specific rehabilitation protocols. This chapter intends to offer a comprehensive overview of the current knowledge on this topic starting from the pathophysiology of postural control disorders occurring in various ecological conditions to the most innovative multidisciplinary rehabilitation approaches.

Keywords: postural balance, accidental falls, neurological gait dysfunction, neurological rehabilitation, virtual reality, robotics

1. Introduction

Postural control is a key component for the safety performance of daily activities. Specifically, the control of posture is essential to stabilize the body's center of mass (COM) relative to the base of support and for the proper alignment of the body with the body's center of gravity (COG), the support surface, and the surrounding environment. Moreover, it facilitates performance of a manual task. Postural control disturbances and resulting falls are significant factors determining the quality of life (QOL), morbidity, and mortality [1].

Parkinson's disease (PD) is the second most common neurodegenerative disorder affecting 2–3% of the population over 65 years of age [2]. Neuronal loss in the substantia nigra causes striatal dopamine deficiency and intracellular inclusions containing aggregates of α -synuclein that are the neuropathological hallmarks of PD. Multiple other cell types throughout the central and peripheral and autonomic nervous systems are also involved, probably from the disease onset. The clinical diagnosis relies on the presence of bradykinesia and other cardinal motor features such as rigidity, postural instability, and tremor. However, PD is associated with

motor and nonmotor symptoms that add to overall disability and increase the risk of postural control disturbances.

Postural control disturbances are one of the most common challenging signs in PD and one of the main concerns in rehabilitation. According to a fall retrospective study, falls are a significant problem for older people with PD as well as younger ones with advanced stages of the disease [3]. Up to 68.3% of people with PD fall each year, with around 50% reporting recurrent falls [4]. The most frequent consequences of falling are injuries [5] and fear of falling (FOF), both of which limit physical activity and lower QOL [6, 7]. Several factors have been associated with recurrent falls in PD, including a positive fall history, disease severity and duration, motor disturbances, medications, cognitive impairment, FOF, freezing of gait (FOG), impaired mobility, and reduced physical activity [8].

Rehabilitation has been considered as a crucial adjuvant to pharmacological and surgical treatments in PD to improve postural control, maximize the quality of life, and minimize secondary complications. However, many rehabilitation approaches are based on empirical experience and lack of knowledge of the underlying pathological mechanisms.

This chapter intends to offer a comprehensive overview of the current knowledge on this topic starting from the pathophysiology of postural control disorders occurring in various ecological conditions to the most innovative multidisciplinary rehabilitation approaches.

2. Pathophysiology of postural control

Postural control has two main functional goals: postural orientation and postural equilibrium. The former involves the active control of body alignment and tone to gravity, support surface, the visual environment, and internal references [9]. It is based on the interpretation of convergent sensory information from somatosensory, vestibular, and visual systems [10]. The latter refers to the coordination of sensorimotor strategies to stabilize the body's COM within the limits of stability imposed by the base of support during self-initiated and externally triggered tasks [10]. Body orientation and stabilization are distinct sensorimotor processes, and both are critical factors in preventing falls.

The postural control system consists of many subcomponents that may be grouped into six main domains: biomechanical constraints, movement strategies (i.e., reactive, anticipatory, and voluntary), sensory strategies (i.e., sensory integration and reweighting), orientation in space (i.e., perception, gravity, vision, and verticality), control of dynamics (i.e., gait and proactive), and cognitive processing (i.e., attention and executive functions) (**Figure 1**). A disorder in any of these resources may lead to postural instability and increase the risk of falling.

The assessment of postural control is crucial to determine specific deficits in the different subcomponents and to assess fall risk. In particular, instrumental assessment plays a threefold role: (1) understanding the pathophysiology of the postural control system, (2) defining clinical diagnosis, and (3) evaluating treatment efficacy. Among instruments, quantitative posturography is one of the most useful tools [11] because it can be performed during static and dynamic tasks as well as under different sensory conditions [11–13]. Other instruments for quantitative measurements of balance are stereophotogrammetric devices to measure the whole body movements through the detection of retroreflective markers, the pedobarography walkways to acquire spatial-temporal features of gait, and wearable inertial sensors (WIS) incorporating advanced electronic technologies to monitor functional activities (i.e., sensorized insoles to detect plantar pressures) [13].

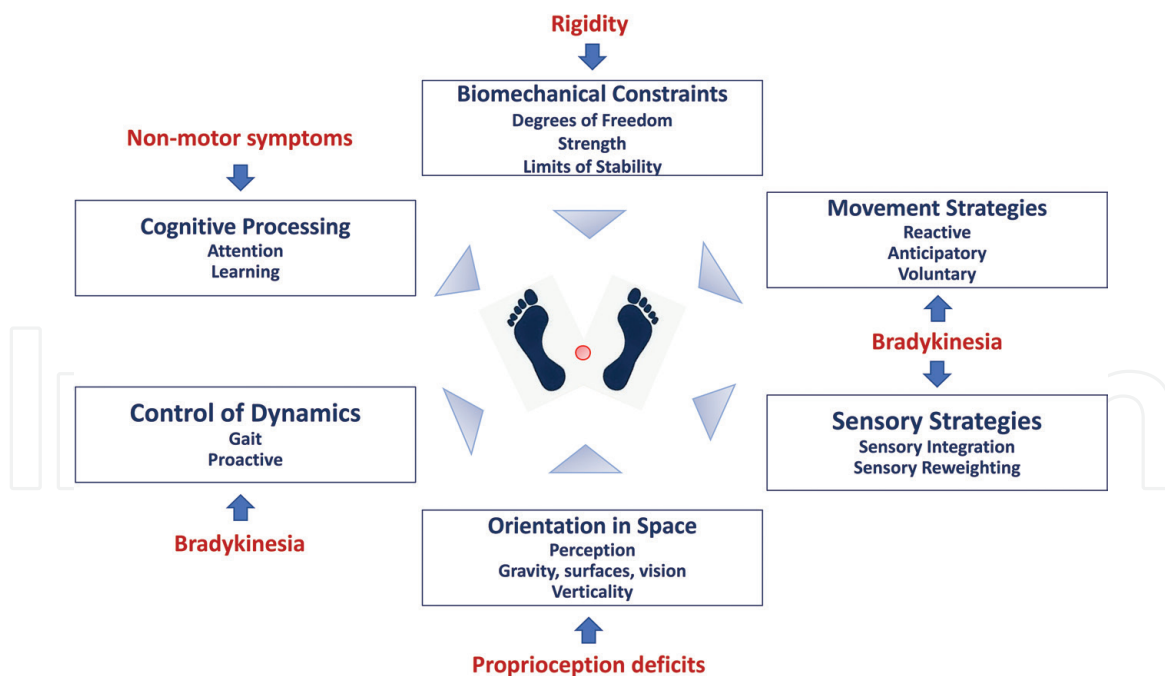


Figure 1.
 Resources required for postural control.

3. Postural control during static tasks

Quiet stance is considered the result of automatic mechanisms working to some extent independently of cerebral cortical control. Postural sway in quiet stance appears to be increased in the elderly [14]. Patients with PD complain of abnormal postural sway even in the early stages of the disease [15–17]. They have increased postural sway, predominantly in the mediolateral direction. Center of pressure (COP) displacement is more significant in the lateral direction and is generally higher in velocity and frequency than in healthy subjects. Increased mediolateral COP displacement has been associated with an increased risk of falling [18, 19].

With eyes closed in the quiet stance, COP displacement is increased in the majority of healthy people, but it is markedly increased in patients with PD [15, 16]. The weaker performance of PD patients suggests that movement strategies and sensory integration deficits are both involved in these automatic tasks that also involve cognitive control and higher order cortical function [20]. For example, PD patients performing a cognitive and motor task in a quiet stance (dual-task condition) show increased sway velocity and area. Dyskinesia might contribute to the increased postural sway in patients with the more advanced stage of the disease, and this may contribute, at least in part, to the higher risk of falling that advanced PD patients experience during ON [21, 22].

Interesting results have been obtained with brain stimulation techniques. Pallidotomy and deep brain stimulation (DBS) of the globus pallidus internus may reduce postural sway, whereas conflicting results after subthalamic nucleus DBS have been reported [17, 23].

3.1 Biomechanical constraints

Several biomechanical constraints can affect balance: the base of support, the degree of joint freedom, limits of stability, and muscle strength [10]. The base of support is defined as the horizontal stride width in the double-support condition, with the feet in contact with the floor and the center of gravity (COG) within feet [24]. The limits of stability refer to the maximal excursion of the COM in different

directions without changing the base of support (e.g., taking a step or falling). The central nervous system (CNS) has an internal representation of the limits of stability, which it uses to define the amplitude of movements to maintain balance. Basal ganglia dysfunction in patients with PD results in an abnormal representation of these limits of stability leading to postural control disorders [10]. In quiet stance, PD patients have an altered perception of these limits and display slower movement toward the limits, especially in the forward direction [25]. This slowness of COM movements has been related to the fear of falling (FOF) [7]. As to pharmacological treatments, levodopa therapy has been demonstrated to increase the perception of limits of stability and the velocity of COM displacement [25].

Muscle strength deficits along with muscle imbalance (i.e., muscle cocontraction) are important determinants of postural control disorders in PD. These impairments have rarely been investigated [26–30]. Although the specific causes of muscle weakness in PD are unknown and not explicitly related to tremor or rigidity, central and peripheral mechanisms might be put forward. Finally, whether muscle weakness is intrinsic to the disease or a secondary phenomenon is still under debate [28]. Inkster et al. [27] investigated the maximal concentric isokinetic knee and hip extensor torque in 10 men with mild PD using an isokinetic dynamometer. Data were compared with normative values collected in 10 sex- and age-matched controls. The PD patients were tested in both ON- and OFF-medication state on different days. The data showed that mean hip and knee extensor torques were lower in the PD patients, with more significant deficits at the hip. Greater hip strength was related to better sit-to-stand (STS) ability in the PD patients, whereas greater knee strength was related to better STS ability in the controls.

Durmus et al. [29] measured lower limb isokinetic muscle strength to investigate whether weakness in specific muscle may be associated with clinical severity and falls in 25 patients diagnosed with PD and 24 healthy volunteers.

A significant decrease in isokinetic muscle strength measured using an isokinetic dynamometer was observed in the PD patients, especially in the hip and knee flexors and extensors. Decreased muscle strength was independent of velocity but correlated with clinical severity and falls.

Allen et al. [26] investigated the relationship between leg extensor muscle power (i.e., the product of strength and speed of muscle contraction as a measure of the ability to use muscles quickly) and strength and the association with past falls and walking in 40 PD patients with mild-to-moderate PD. Patients with low muscle power were 6 times more likely to report multiple falls in the past 12 months than those with high muscle power. Although the association between falls and power was no longer significant after adjusting for the Unified Parkinson's Disease Rating Scale (UPDRS) motor score, muscle power was more strongly associated with past falls than muscle strength. This suggests that not only the force of contraction but also the velocity at which it can be generated determines the ability to perform physical activities and to recover from a loss of balance to prevent falls.

The importance of isokinetic muscle strength and power evaluation as a useful tool in the assessment of clinical severity and falls in PD has been emphasized [29–31]. Muscle power, muscle strength, and bradykinesia are potential targets for physical therapy interventions.

3.2 Movement strategies

Three postural strategies can be distinguished: ankle strategies, hip strategies, and stepping strategies. The ankle strategy refers to movements at the ankles in response to backward and forward tilt involving the distal muscles such as the tibialis anterior and the gastrocnemius muscles, respectively. This strategy is generally

implemented to counteract small perturbations leaving feet in place (without the base of support enlargement).

The hip strategy consists of quick movements at the hip to maintain uprightiness in more precarious conditions. Finally, the stepping strategy is used when perturbations occur or before reaching the limits of stability to return the COM to within the base of support. This strategy requires taking a step to enlarge the base of support. Together with these movement strategies, upper limb reactions may be employed to increase the limits of stability. In healthy subjects, the latency of the ankle and hip strategies is shorter (100 ms) than the stepping strategy (250 ms) [32]. In a quiet stance, the increase in mediolateral sway reflects decreasing postural control at the hip, whereas control in the anteroposterior direction is mainly related to activation of the ankle strategy [16]. Individuals with higher risk use the hip strategy more than those with a lower risk who use mainly ankle strategies.

Patients with PD have slower postural responses when in the OFF medication state [33]. Slowed postural adjustment induces a delay in returning of COM within the base of support, which exposes the patient to a higher risk of falling. Abnormal muscle activation such as cocontraction and short-duration bursting might further impair postural responses in PD [33, 34]. In the early stage of PD, balance is asymmetrically controlled in some patients with PD with the less affected leg producing more corrective joint torque than the other leg to maintain postural control [35]. Balance performance was evaluated in 20 PD patients and 11 healthy matched controls during 2 independent continuous multiline perturbations in the forward-backward direction. Applying closed-loop system identification techniques, relating the body sway angle to the joint torques of each leg separately, the investigators measured the relative contribution of each ankle and hip joint to the total amount of joint torque. The controls exhibited symmetric balance control. In contrast, the balance contribution of the less affected leg was higher than that of the leg of the more affected side in PD patients. The ratio between the legs helped to preserve a standard motor output at the ankle. These results suggest that PD patients compensate for postural control asymmetries by increasing the relative contribution of the less affected leg. This compensation appears to be successful at the ankle but is accompanied by increased stiffness at the hip [35]. Besides, other components such as intention, experience, and expectation can influence the movement strategy selection and the magnitude of postural responses [36].

Because of the impairment in the stepping strategy, patients with PD are unable to take steps wide enough to keep COM displacement within the base of support and so take additional steps. However, because such protective steps may not be significant enough to arrest movement of the COM, new stepping produces propulsion (if the COM is displaced forward) or retropulsion (if the COM is displaced backward). While healthy individuals take a compensatory step with no or only one anticipatory postural response, PD patients, especially those with freezing of gait (FOG), are often noted to make several anticipatory postural adjustments before stepping [37]. This delayed execution of the step is associated with “trembling of the knees” and increases the risk of falls [38]. Similar to voluntary steps, the size of automatically triggered protective stepping responses is improved by visual cues [39].

Postural abnormalities in PD are drug-refractory complications that affect postural reaction for balance maintenance [10]. The pathological underpinnings of camptocormia, antecollis, Pisa syndrome, and scoliosis have not yet been fully characterized, but either central or peripheral mechanisms might be the reasons for such deformities. Central mechanisms, such as trunk and lower limb muscle dystonia, may play a crucial role. Electromyography (EMG) studies in patients with PD and the Pisa syndrome [40–42] have shown greater activation of the paraspinal and nonparaspinal muscles bilaterally (cocontraction), ipsilateral or contralateral

to trunk leaning or in camptocormia at the abdominal wall muscles than in healthy subjects. Peripheral mechanisms, such as myopathy and degenerative spinal and soft tissue changes, may all lead to muscle imbalance, weakness, and compensatory posture [43].

So far, the presumed effect of posture misalignments in postural control (i.e., Pisa syndrome) has been partially elucidated. Recent data indicate that the Pisa syndrome is associated with abnormal postural responses to maintain postural uprightnes with significantly higher body sway velocity in the anteroposterior and mediolateral directions [44]. It means that a misalignment of posture may negatively influence postural and balance responses in PD patients and may be further comprised in patients unaware of their misalignment.

3.3 Sensory strategies

The ability of the CNS to use vestibular, somatosensory, and visual inputs to create a system of coordinates on which the body's postural control is based [45] is crucial to maintaining uprightnes. In quite stance, healthy subjects rely on somatosensory (70%), vestibular (20%), and visual (10%) system inputs [46].

Sensory reweighting refers to the ability of the nervous system to integrate the different sources of information as well as changes in the surrounding environment. It means that sensory integration deficits may be related not only to dysfunction at the source but also to the ability to quickly change sensory weighting in different conditions (i.e., eyes open, eyes closed, and altered feet contact surface). Patients in the more advanced stage of PD have difficulty in maintaining uprightnes on unstable surfaces with eyes closed [19], which is probably also related to a significant reduction in incoming proprioception information [47].

Finally, PD patients with posture misalignment might rely mostly on visual inputs to control their posture and postural reactions [44].

3.4 Orientation in space

Body alignment is defined as the relationship between body parts to one another and the ability to orient them to gravity, the base of support, and the surrounding environment. The nervous system in healthy humans is automatically able to modify body orientation in space according to the task and context in which a person is involved. In an experimental dark condition, healthy individuals can identify the gravitational vertical within 0.5° [10] according to multiple neural representations of verticality, or uprightnes [48].

A tilted or inaccurate internal representation of verticality may affect postural alignment about gravity and lead to postural disorders. An altered sense of verticality in PD patients has been related to an impairment of the proprioceptive and somatosensory integration system [49], which affects the position of the body's COM over the base of support and makes patients more vulnerable to falls. Whether postural misalignment in PD (i.e., camptocormia, Pisa syndrome, antecollis, and scoliosis) depends on the inaccurate internal representation of verticality is still under investigation [50].

4. Postural control during dynamic tasks

The central nervous system uses compensatory postural adjustments (CPAs) and anticipatory postural adjustments (APAs) to maintain balance during dynamic and goal-oriented tasks [10] (**Figure 2**).

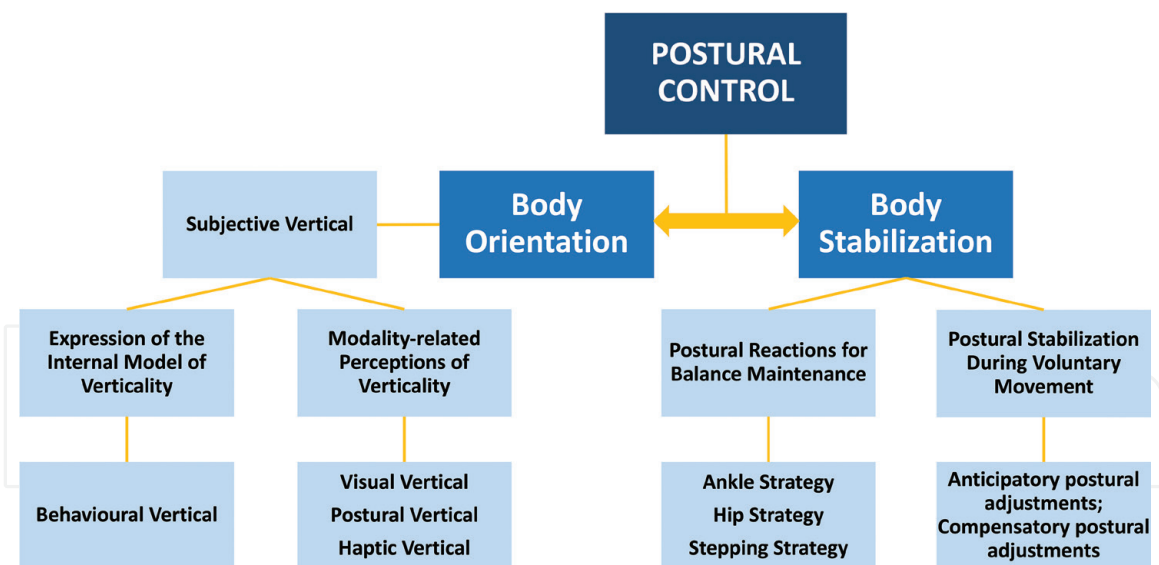


Figure 2.
 Sensorimotor processes involved in postural control.

The former serves to restore the position of COM after a perturbation has occurred [32, 51]. CPAs rely mostly on the somatosensory and vestibular systems to determine the extent and type of perturbation and then to trigger the proper postural strategies. These postural responses are spinal and supraspinal polysynaptic responses and triggered by the difference between the actual and planned ankle joint trajectories [52].

The latter serves to minimize displacement of the COM before a perturbation [51–53]. Usually, APAs are associated with voluntary movements to reduce the effects of forthcoming disturbances by predicting the disturbance onset [53]. Another mode of APA control refers to the capability to detect potential environmental hazards and to implement the right postural adjustment before the risk event. In this context, vision and attention are critical factors in the early detection of potential balance threats, whereas complex sensorimotor integration processes are initiated to modify walking behavior to prevent falls promptly [54].

CPAs and APAs are thought to be involved in forming the so-called internal model, which is the internal representation of “motor and sensory signals related to a specific motor execution” [55]. An “internal model” exists for both the upper and the lower limbs [56]. In the lower limbs, the model adjusts for postural perturbation and novel dynamic environments during walking. Electrophysiological studies have emphasized that a combination of abnormal CPAs and APAs contributes to postural control disturbances in PD [57].

Different control mechanisms are involved during a static and dynamic task. However, postural control and active tasks are tightly intertwined. Significant insights into this topic have been gained from studies employing a battery of complementary research techniques, including electrophysiological techniques, and detailed postural control clinical evaluations (i.e., posturography) during dynamic tasks.

The sit-to-stand task, for instance, requires mainly APAs to displace the COM over the base of support before planning the sequence of movements to raise the body from a chair [58]. The inability of PD patients to perform this task may depend on altered APAs that do not allow bringing the COM adequately forward over the feet before the liftoff from a chair. In the early stage of the disease, patients show exaggerated movement preparation when performing the sit-to-stand tasks [59], whereas in the more advanced disease stages, reduced APAs have been measured [60].

Locomotion can be seen, in fact, as a four-step repeated sequence of balance challenges: gait initiation and generation of continuous movements to move forward and maintenance of postural equilibrium during progression; adaptability

to any changes in the environment or other simultaneous tasks; gait termination [52]. Locomotion involves an intricate interplay between environmental conditions, goals, biomechanical constraints, sensory integration processes, and cognitive resources [61, 62]. All these aspects (alone or in combination) are impaired in PD patients.

Latash et al. reported that APA deficit might reflect disorders in the fundamental processes of preparation and initiation of a motor act [63]. Bradykinesia, one of the cardinal manifestations of PD, results from a failure of basal ganglia output to reinforce the cortical mechanisms involved in the preparation and execution of motor commands [64]. The resulting cortical deficit primarily involves the midline motor areas and is responsible for the difficulty in performing self-paced movements, for prolonged reaction times, and abnormal premovement electroencephalographic activity [64]. During task performance, the brain tries to compensate to some degree for the basal ganglia deficit by overactivating the lateral premotor areas. When compared with healthy subjects, PD patients have a lower amplitude of premotor potential during self-initiated movements, and underactivation of the supplementary motor area, anterior cingulate, left putamen, left insular cortex, right dorsolateral prefrontal cortex, and right parietal areas [65]. In contrast, no significant differences in early premotor potentials were found between PD patients and healthy subjects when externally triggered movements were performed. These findings confirm that the deficit in self-initiated movements in PD depends on the midline motor areas under activation and explain why movements can be speeded by sensory cues [66, 67].

Gait initiation refers to the capability to activate APAs that are essential to unload the swing leg and create the conditions for progression according to a highly stereotypical preparation muscle pattern activation. Moreover, the APA amplitude and duration are predictive of the subsequent peak step velocity [68]. Gait initiation disturbances in PD consist of the delayed release of APAs and hypokinetic anticipatory (reduced scaling) and bradykinetic APAs (abnormal timing) [68]. The most severe forms of gait initiation are the FOG and other phenomena such as the “knee trembling,” which have been recognized as a sign of impaired APAs in PD patients with FOG [38]. FOG is a debilitating phenomenon during which the subject is suddenly unable to start walking or continue to move forward. It is usually thought to occur in the late stage of PD, but it also occurs in the early stages of idiopathic PD. FOG has been defined as a paroxysmal phenomenon, and little is known about its physiopathology. The influence of emotion, attention, external triggers, and dopaminergic drugs on gait initiation disturbances suggests the existence of multifactorial pathophysiology involving multiple nervous networks.

The generation of continuous movements to move forward and the processing of load-related input by the spinal cord central pattern generators are impaired in PD patients. It is associated with a reduction of leg extensor muscle activation and an enhanced leg flexor activity in the stance phase [52]. This pattern is associated with greater tibialis anterior muscle activity during the swing phase of gait that might reflect the stronger control of stance and gait in these patients by the visual system. Visual information can substitute to some extent reduced proprioception [69] improving walking [62].

Adaptability and variability in gait are related to the increased risk of falling and the decline in mobility [70]. Patients with PD showed more variability in step length and step time during gait than healthy subjects and freezers have a higher spatial gait variability between freezing episodes [71]. Variability in stride-time intervals is related to dynamic postural control, and it is associated with activation in the medial sensorimotor cortices, as measured using functional near-infrared spectroscopy (fNIRS) system [72]. The variability in stride-time intervals while

walking forward was correlated with maximum activation in the precentral gyrus and supplementary motor area. However, activation in these areas and superior parietal lobule was higher when the subjects walked backward than forward suggesting that backward walking is a challenging task to the nervous system as it controls the stepping pattern [72]. Stride-to-stride variability is markedly increased among PD patients with FOG as compared to those without FOG, indicating that the inability to control cadence may play an essential role in this debilitating phenomenon. The effects of the dopamine-mediated pathways on stride-to-stride regulation and the role of attentional processes are thought to trigger FOG.

Literature on gait termination disorders in PD is scant. PD patients have difficulties in anticipating future target motion, which may play a role for the mechanisms involved in changing direction during gait and gait termination [73]. In contrast, their capacity of building up an internal representation of continuous target motion remains unimpaired. It may explain the clinical effects of medical devices that use visual motion to improve gait initiation (e.g., "PD glasses"). Further studies on the clinical effects of gait termination are needed.

Other relevant tasks concern difficulty turning during gait. Turning is a significant contributor to mobility disability, falls, and reduced quality of life in patients with PD [74]. It requires a complex integration between functionally different control mechanisms characterized by an upweighting of vestibular inputs and anticipatory postural adjustments [75]. Turning is accomplished by a top-down temporal sequence of body segment rotations that begins with an anticipatory redirection of gaze (saccade) toward the new heading direction. Increasing evidence suggests that eye movement is critical for turning control and that when the eyes are constrained, or participants have difficulty making eye movements, steering control is disrupted [76]. It has been demonstrated that APAs can be affected by the turn angle, the pivot foot, and speed and that they occur during the prior step. Healthy subjects usually lean backward and sideward on the previous step in anticipation of the turn, suggesting that the motor system uses central control mechanisms to predict the required anticipatory adjustments and organizes body configuration by the movement goal [75]. Animal studies have demonstrated that the basal ganglia network is involved in the posture-kinetic changes associated with turning and orientating behavior and that even PD patients with mild clinical impairment and no significant abnormalities in linear walking may exhibit turning disturbances [77]. PD patients show increased turn duration, a higher number of steps to turn, and difficulty in changing motor patterns from a straight line to turning [78, 79] even if their ability to scale movement speed and amplitude appears to be preserved. Moreover, altered oculomotor control is responsible for fewer, slower, and smaller preparatory saccades approaching a turn. Visuospatial dysfunction plays a central role in gait disturbance in PD, especially when navigating through complex environments [80].

Specific movement deficits in PD can be classified into perpendicular deficits (taking more steps and shorter steps and an altered turn strategy) and axial deficits (segment rigidity, altered segment coordination and timing, reduced segment rotation, and the effects of altered posture). Furthermore, FOF influences turning metrics in PD patients rather than a positive fall history [81]. Therefore, the specific focus should be made to the assessment and rehabilitation of the axial deficits alongside those of the straight body segments in the design of multimodal treatment strategies to improve turning performance.

The relationship between cognitive function and balance "per se" has rarely been investigated. In contrast, a growing body of evidence indicated the role played by cognition in the control of gait. Gait is considered an activity that requires executive function, visuospatial abilities, memory resources, and attention as well

as the judgment of external and internal cues. Some morphological and functional neuroimaging studies have offered evidence supporting the relationship between gait and cognitive resource functional neuroimaging studies.

5. Rehabilitation approaches

Patients with PD show impairments in many aspects of postural control including rigidity affecting biomechanical constraints, bradykinesia of postural adjustments, impaired sensory integration processes, and bradykinetic gait with freezing. Postural control disorders are disabling and drug-refractory complications in PD. In this context, rehabilitation is crucial to maximize functional ability and minimize secondary complications within the context of education and support. Rehabilitation can ameliorate postural control through practice and enables patients to benefit from exercises that challenge their gait and postural control. Initially, practical experience has driven the rehabilitation of postural control disorders. However, improvements in understanding the mechanisms underlying postural control disorders in PD have ultimately lead us to improve the management strategies.

Since the pathophysiology of postural control disorders is multifaceted, the different rehabilitative approaches proposed in literature should be combined in a comprehensive rehabilitation program addressing all the affected domains. Moreover, according to the disease stage, specific rehabilitation approaches should be proposed.

Patients and their family members should be advised on the importance of maintaining adequate motor activity to preserve motor function and prevent the decline in mobility starting soon after the diagnosis. At relatively early stages of the disease, the importance of home exercise routines such as Nordic walking, treadmill training, and balance training consisting of a combination of strengthening and balance exercises may lead to an improvement in self-perceived performance in daily activities and reduce the gradual decline in mobility [83–88]. At this stage, dance therapy and martial arts might improve significantly postural control with a decrease in falls [83] and offer the advantage of social engagement, from an ICF perspective. Exercises aimed at improving sensorimotor integration are crucial, and in the early stage of the disease, they can be carried out using virtual reality scenarios and exergaming. Virtual reality has emerged as a therapeutic tool facilitating motor learning for balance and gait rehabilitation [84] especially in the early stages of the disease as home rehabilitation interventions [85].

With the progression of the disease, home exercise routines need to be progressively supported by specific rehabilitation interventions delivered by health professionals. In the comprehensive management of patients with PD, the presence of limiting factors such as motor fluctuations (on/off phase), dyskinesia, fatigue, and the cognitive status should be taken into account. Previous reviews emphasized the role of exercises that challenge various PD-specific component of postural control including the stability limits, APAs, CPAs, and dynamic stability [82]. In the advanced stage of the disease, the rehabilitation of postural disorders might be enriched using technological devices. Robotic rehabilitation is a rapidly growing field of application. The primary advantage of using robot technologies is the possibility to deliver high-dosage and high-intensity training. A recent systematic review [83] analyzed safety, feasibility, and effectiveness of exergaming for PD and demonstrated that people with PD were able to play exergames, enjoyed playing, and showed some improvements in motor symptoms (balance). Furthermore, MI and AOT demonstrated potential benefit in PD

patients because of improvement in motor skills by enhancing proprioceptive signals normally generated during movements and activation of the mirror neuron system, respectively [83].

Growing evidence has suggested that rehabilitation could induce long-lasting and clinically meaningful benefits, particularly for postural control and gait. These training effects may be associated with exercise-induced neuroplasticity phenomena by increasing synaptic strength and influencing neurotransmission [82].

To date, there is still no consensus about the optimal rehabilitation approach owing to an extensive heterogeneity of interventions (i.e., stretching, muscle strengthening, balance, postural exercises, occupational therapy, cueing, and treadmill training) [83]. Indeed, these interventions bring additional benefits when combined with “conventional rehabilitation” in the context of specific theory-driven protocols. The number of practice variables (i.e., duration, intensity, specificity, and complexity) needs to be tailored to the individual patients’ characteristics. Besides, the interplay between higher-order neural function and postural control has many clinical implications, ranging from integrated assessment tools to possible innovative lines of interventions, including cognitive therapy for fall prevention on the one hand and walking program for reducing cognitive decline risk on the other.

6. Conclusion

The new knowledge on the effects of environment and task on postural control in individuals with Parkinson’s disease is fascinating and emphasized the complicated interplay between cognitive and sensorimotor processes. Improvements in understanding these mechanisms have ultimately improved the management strategies in rehabilitation. To date, specific rehabilitation approaches can be selected according to the stage of the disease and the specific postural control impairment.

Appendices and nomenclature

APAs	anticipatory postural adjustments
CNS	central nervous system
COG	center of gravity
COM	center of mass
COP	center of pressure
CPAs	compensatory postural adjustments
DBS	deep brain stimulation
EMG	electromyography
fNIRS	functional near-infrared spectroscopy
FOF	fear of falling
FOG	freezing of gait
PD	Parkinson’s disease
QOL	quality of life
STS	sit-to-stand

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