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REVIEW

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Neuroimaging and cognitive correlates of postural control in Parkinson's disease: a systematic review

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Abstract

Parkinson's disease (PD) can cause postural instability, which may result in falls. These issues have been associated with motor and non-motor symptoms (NMS), including cognitive dysfunction. Several techniques have been employed to investigate the underlying neural mechanisms involved in postural control in PD. These include behavioural studies assessing associations between cognition and postural control, functional neuroimaging studies, and resting-state neural correlates. This review provides an overview of these emerging bodies of research. Scopus, PubMed, and ProQuest were searched and detailed the brain-imaging technique, cohort, and postural control measures. A total of 79 studies were identified. Findings supported the notion of cortical involvement in postural control function to compensate for subcortical damage resulting from PD. Future studies should standardise their outcome measures and data analysis to allow comparisons of results across studies and ensure more comprehensive and robust data collection to enhance the reliability and validity of these findings.

Keywords Balance, Brain-imaging, Cognition, Cortical activity, Neural correlates, Parkinson's disease, Postural control

Introduction

Parkinson's disease (PD) is a common neurological disorder characterised by both motor and non-motor symptoms (NMS). Among these, postural control impairment is one of the most debilitating motor symptoms and significantly increases the risk of falls in people with PD, leading to reduced quality of life and increased healthcare costs [3]. Importantly, postural control issues have a limited response to dopaminergic medication, presenting challenges for treatment options [4].

Postural control is a complex skill which requires the concurrent integration and processing of input from multiple sensory systems including: visual, vestibular and proprioceptive systems [5], but also cognitive information [6–8]. The integration of sensorimotor information allows an upright stance to be maintained with the goal of achieving both stability and orientation [9, 10]. This process is facilitated by the Basal Ganglia, which is affected in PD and can lead to postural instability [6]. Performance of postural control depends on the interaction of the individual's capabilities, the task, and the environment.

Poor postural control has been associated with age-related changes, vestibular disorders, musculoskeletal

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issues, visual impairments, and various neurological conditions. Recently, the role of non-motor symptoms (NMS), such as anxiety and cognitive dysfunction, in postural control difficulties in PD has been recognized [6, 11].The associations between cognitive function and postural control impairment have been observed across all stages of disease severity of PD [12–16]. The empirical evidence regarding the effectiveness of dopaminergic medication in the treatment of cognitive dysfunction varies depending on cognitive domains, and between distinct PD phenotypes.

Underlying brain function related to postural control can be measured in several ways. A growing number of behavioural studies have researched the association between cognition and motor skills [17, 18]. Yet, a disproportionate amount of this research focuses on the relationship between cognition and gait, rather than cognition and balance [1].

Cognitive control of postural control involves the dynamic interplay between cognitive function and the neural circuits responsible for maintaining equilibrium in an upright stance. The cognitive decline associated with PD likely exacerbates postural control difficulties and further increases falls risk. Various cognitive domains have been positively associated with balance in PD, such as attention [19], verbal fluency [20], visuospatial function [21], executive function [19, 22], memory [22, 23], and global cognition [19–24].

Dual tasking is the simultaneous performance of two tasks. This paradigm is also often employed to assess the cognitive control of postural control in people with PD (PwPD). By incorporating a second task in addition to one that involves postural control, additional cognitive resources are challenged which can highlight cognitive-motor interactions. When cognitive load exceeds capacity, this can result in reduced/worsened postural control, e.g., larger sway outcome measures [25].

In addition to behavioural associative studies, there exists an emerging body of literature that reports the use of portable brain imaging techniques, such as functional near-infrared spectroscopy (fNIRS) and mobile electroencephalography (EEG), to investigate cortical activity during postural control tasks in PwPD [26]. The advantage of these techniques is that they measure cortical activity in real-time, during task execution, rather than depending on associative analysis of the relationships between postural control and brain activity. Both fNIRS and mobile EEG are relatively inexpensive, non-invasive, portable, and somewhat resilient to movement artefacts, which makes them favourable techniques for the study of cortical activity during tasks that challenge postural control [27, 28]. Traditional imaging studies using techniques such as functional magnetic resonance imaging (fMRI) are constrained by the need for a stationary supine position which creates ecological challenges when researching neural correlates of postural control in PwPD. Therefore, these studies are restricted to associative paradigms or are reliant on tasks to emulate balance performance such as mental imagery or virtual reality (VR). Yet, this research may also provide important insights into the relationship between neural activity and motor functions [29, 30].

Some previous efforts have been made to consolidate findings related to the underlying neural mechanisms involved in motor control in PwPD. For example, one narrative review discussed motor and cognitive factors that contribute to dual-task walking deficits [31]. However, to our knowledge, no such work has been done pertaining to tasks that challenge postural control. Moreover, a structured review from 2018 provides an overview of studies using fNIRS and EEG to examine cortical activity during walking and balance tasks in PwPD and older adults [26]. Few studies were found that involved the assessment of cortical control of postural stability in PD. However, as this is a developing area of research, there have since been a growing number of studies that have focused on this topic, using techniques such as fNIRS and EEG.

The overall aim of this review is to systematically present the current evidence on the neuroimaging/cognitive correlates of postural control in PwPD. This review will encompass several techniques measuring brain function (either through neuropsychological testing or brain imaging) in relation to postural control performance (through objective assessment/measures), to provide a comprehensive overview of this topic. The review includes the following.

(1) Behavioural studies analysing the associations between performance on neurocognitive measures and standardised measures of postural control.
(2) Brain activity recorded in real-time during (postural control-related) task performance with standardised measures of postural control.
(3) Resting-state brain activity associations with standardised measures of postural control, measured by one of the following techniques: functional magnetic resonance imaging (fMRI), positron emission tomography (PET), single-photon emission computed tomography (SPECT), electroencephalography (EEG) or magnetoencephalography (MEG).

This review will provide a summary of the status quo of the field of study that explores the underlying neural mechanisms involved in postural control in PwPD. Enhancing knowledge of the conjunction of brain activity

Neurodegenerative group: Parkinson* OR "Parkinson's disease" OR Parkinsonian

AND

Brain Activity/Cognition/Mobile Imaging: "neural" OR "neuronal" OR "brain" OR "motor" OR "cortical" OR "subcortical" OR "locomotor" OR "corticomotor" OR "cognition" OR "global intelligence" OR "cognitive" OR "memory" OR "attention" OR "executive functioning" OR "executive function" OR "processing speed" OR psychomotor OR visuospatial OR "verbal fluency" OR "reaction time" OR "cognition disorders" OR "neuropsychological tests" OR visuoperception OR visuoconstruction OR EEG OR electroencephalography OR nirs OR "functional near-infrared spectroscopy" OR "near-infrared spectroscopy" OR fNIRS OR "Mobile Imaging" OR "near-infrared" OR "neuroimaging" OR "MRI" OR "diffusion tensor imaging" OR "fMRI" OR "functional MRI" OR "PET" OR "positron emission tomography" OR "single photon" OR "SPECT" OR MEG OR "functional magnetic resonance imaging" OR magnetoencephalography

AND

Real-world task: "postural control" OR "balance control" OR balance OR Standing OR Dual-task* OR "Centre of mass" OR "Postural sway" OR "dual task"

AND NOT
Mouse OR Rat

Fig. 1 Search terms

involved in postural control in PD will inform the development of PD-specific interventions and rehabilitation programmes.

Methods

Search strategy

An initial search was conducted using three databases (Scopus, PubMed & ProQuest) with the search terms shown in Fig. 1**.** The search was limited to full journal articles written or translated into the English language from January 1980 to September 21st, 2023. Once the search was complete, duplicates were deleted, and an initial title and abstract screen were conducted by two independent reviewers (PT and LG). A full article review was completed when it remained unclear from the abstract whether a paper met the inclusion criteria. A meeting was then held by both reviewers to discuss the articles that were not initially mutually agreed upon for inclusion. Any remaining conflicting studies were then evaluated by a third reviewer (RM). This paper adheres to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). This review is registered with PROSPERO. Registration number: CRD42023390771.

Inclusion and exclusion criteria

Articles were included if they assessed individuals with a diagnosis of PD undergoing an objective measure of balance associated with at least one of the following; (i) neurocognitive assessment, (ii) real-time brain imaging, or (iii) resting-state brain activity. Objective measures of balance included rating scales (e.g., UPDRS question 3.12), wearable sensors, force plates, 3D motion capture and timed measures such as the timed up and go (TUG), due to its incorporation of various elements of dynamic balance such as anticipatory postural adjustments (APAs), turning and sit-to-stand/stand-to-sit.). Outcome measures derived from TUG included total time of TUG, but also duration of specific parts of TUG such as sit-to-stand/stand-to-sit and turning. Moreover, TUG tests that were completed with movement sensors were included if they provided temporal and spatial parameters relevant to postural control (e.g., turning angle, gait initiation time). Studies that used a TUG DT paradigm

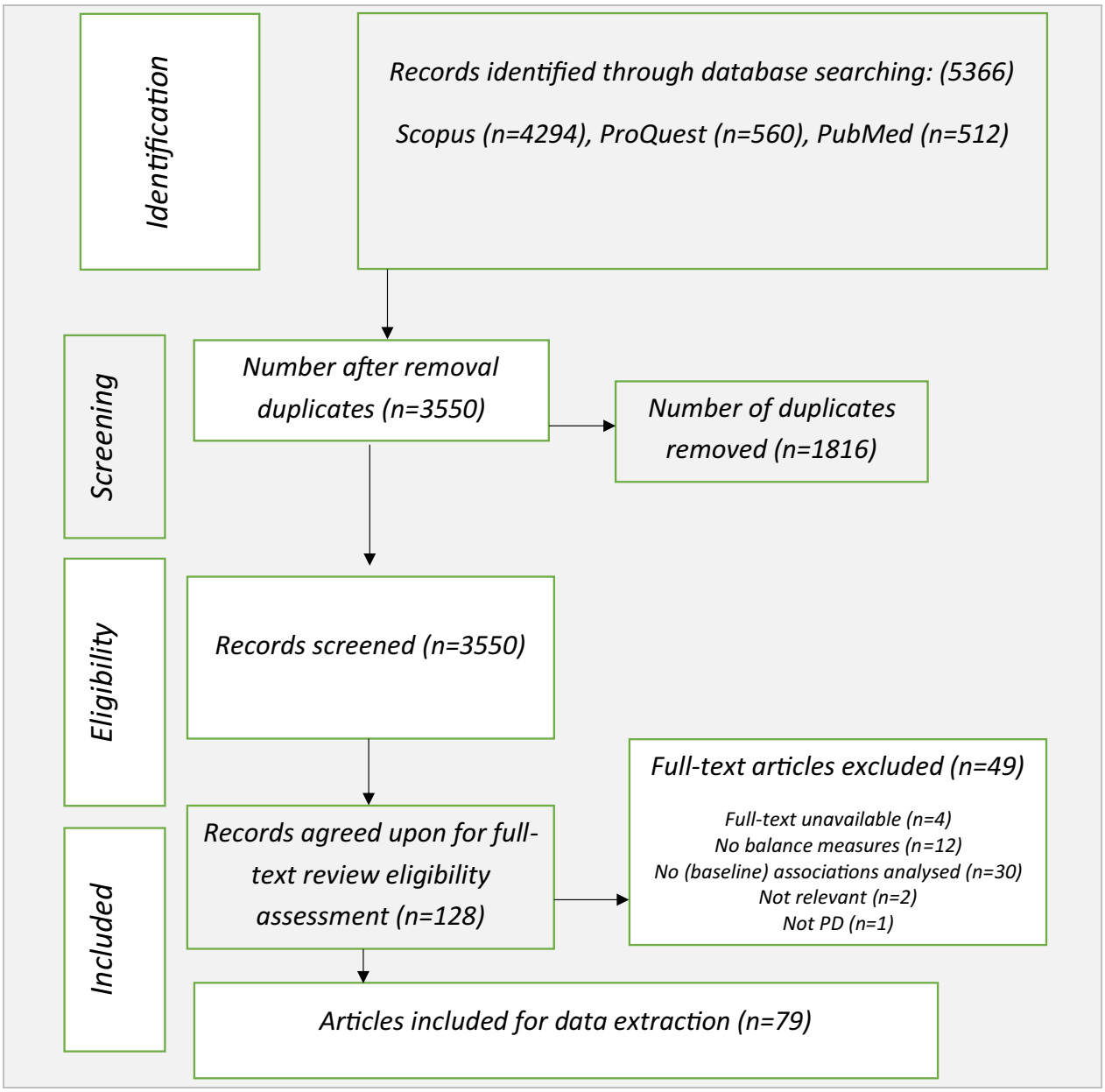

Fig. 2 PRISMA flow chart of the study design. This shows each stage of the review process and the corresponding number of studies

and reported cognitive outcome measures (e.g., DT cost) were also included.

The following domains of postural control were used to classify tests and measures. [10].

1. Static postural control (maintaining equilibrium and stability in unchanging conditions).
2. Dynamic postural control (e.g., postural transitions (sit-to-stand) or maintaining equilibrium whilst moving (turning).
3. Reactive postural control (e.g., adjusting to perturbations).
4. Proactive postural control (e.g., anticipatory postural adjustments (APAs)).

Two additional domains of postural control were used to classify tests and outcome measures that specifically target sensory aspects of postural control and cognitive-motor interference on postural control.

5. Sensory integration of postural control (e.g., manipulating visual, vestibular, and proprioceptive/somatosensory input).
6. Cognitive load on postural control (e.g., cognitive-motor DTs) [32–34].

Several outcome measures can be considered to test multiple domains of postural control. Testing of cognitive function included neuropsychological assessments including; Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), measures of individual cognitive domains, comprehensive cognitive batteries. Studies that employed dual-task paradigms to demonstrate the impact of additional cognitive load on postural control performance were also included.

Brain imaging studies were included if they measured brain activity in relation to balance, either during task performance (real-time) or if they involved resting-state functional connectivity associations with objective measures of postural control. Intervention studies were excluded unless data analysis was performed, and baseline assessment measures were reported. Similarly, longitudinal studies were only included if they did not delineate any type of rehabilitation programme or pharmacological intervention.

Data extraction

Data were extracted and synthesised from full-text articles, into Tables 1 and 2, by the first reviewer (PT) and checked by a second reviewer (LG). Extracted data for the behavioural studies (Table 1) included first author and year of publication, participant characteristics, balance measure(s) and domains, cognitive tests, and key findings and interpretations. For the brain-imaging studies (Table 2), the cognitive domain (tests) section was replaced with "cortical activity measurement device and regions of interest (ROIs)". This process and the corresponding number of studies of each stage of this literature review are displayed in Fig. 2.

Results

Search yield

After removing duplicate articles, the initial search yielded 3550 results (as shown in Figure 2). Two researchers (PT & LG) independently reviewed the titles and abstracts. Following the title and abstract screening, 128 articles were agreed upon for full-text review. This procedure was completed by the first reviewer (PT), after which, 79 articles were identified as relevant and remained for data extraction. Risk of bias was evaluated for all included studies using QUADAS-2 (See appendices). This assessment revealed a low risk of bias was found in 60 studies, moderate risk in 19 studies, and high risk in 1 study. Studies were divided into three separate tables/groups: behavioural studies (n=64), brain-imaging studies during postural control task performance (n=9) and associative brain function with postural control performance (n=7). One study [24] was included in both the behavioural studies, and brain imaging (during task performance) tables as it had a protocol and findings that were pertinent to both sets of inclusion criteria.

The 49 excluded articles that were removed following full-text review and the reasons for exclusion are provided in Supplementary Material Table 1. These were agreed with by the 2nd reviewer. Data from the remaining studies was extracted and synthesised into tables by the 1st author (PT) and confirmed by co-authors (LG, RV, TW, ET, JO, SS, RM).

Behavioural studies (n = 64)

Participants

Data were extracted from the behavioural studies (n = 64) and formatted into Table 1. Within these studies, the average age for the PD groups was 66.31. The majority (n = 34) used an older adult (OA) healthy control group. One study assessed balance and cognitive performance against a healthy younger adult (YA) group aged 24.9 [35] and one [36] included middle-aged adults who had a mean age of 52.8 years. Most studies (n = 46) tested PD patients "ON" Parkinsonian medication. Some studies (n = 12) did test participants in their "OFF" state and one study [37] tested participants ON and OFF medication. Four studies [38–41] did not report medication status. One study [42] tested drug naïve patients i.e., patients who had never taken any dopaminergic medication.

Balance measures/tasks and Dual tasks

Rating scales such as specific items of the UPDRS were used as assessments of balance e.g., MDS-UPDRS item 3.12, (Pull-Test) (n = 5) and Tinetti balance assessment test (n = 3). One study [24] used a combination of MDS-UPDRS items to create a clinical balance score (CBS); the sum of MDS-UPDRS items 3.8 (leg agility), 3.9 (arising from chair), 3.12 (postural stability) and 3.13 (posture).

Several studies employed the use of objective measures of balance (n = 41) e.g., a postuographic force/balance platform (n = 26) or wearable inertial sensors/accelerometers (n = 12). The majority (n = 8) of these were inertial sensors. Two studies used cameras in addition to a force platform.

One study only used optoelectrical cameras [37] and one used a mobile device. Other measures included e.g., the BBS (n = 9) and TUG (n = 15), which assess different

Table 1 Extracted data from behavioural studies. (n = 64)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[47]	PD (n=111, m52 / f59) aged 49.41 (6.33) UPDRS III = 13.3 (6.3) H&Y = 2.1 (0.8) LEDD = 321.76 (109.23) ON medication OA: (n=149, m73, f69) aged 50.62 (6.07)	Static postural control (WII Balance Board (Nintendo)) NS EO & EC Task length: 3 x 30s	Global cognition (MMSE) Attention (TMT A/B)	There was a sig. positive correlation between ML/AP COP velocity and TMT A/B. PD performed sig. worse on most balance measures and TMT A/B than OA. No sig. differences were found in AP COP during EC condition. Findings are consistent with previous studies showing a relationship between cognitive dysfunction and balance disturbances in PD and support the notion that attention deficits may have similar underlying neural mechanisms as motor dysfunction.
[15]	PD (n=25, m9 / f18) aged 67.12 (8.83) Disease duration = 6.69 (4.85) ON medication OA (n=18, m11 / f7) aged 69.12 (10.55)	Static postural control (Bertec Balance plate) NS EO & EC Trial duration: 90s	Global cognition (MoCA, NUCOG)	There was a sig. negative correlation between postural sway length and both MoCA and NUCOG scores in the PD group but not for OA. Sway during EC was only sig. increased for the PD group. Findings suggest that increased sway is associated with poorer global cognition and executive functioning.
[102]	PD (n=52, m37 / f15) aged 62.63 ± 9.18 years. Disease duration: 5.81 (3.97) MDS-UPDRS III mean (SD) score: 14.61 (7.32) LEDD: 686.72 (432.16) ON medication	Reactive postural control (MDS-UPDRS 3.12 (Pull Test)) Static postural control (Biodex Balance System (LOS protocol)) 3 trials (2 training) NS	Attention (Computerised SRT)	Regression analysis for LOS% indicates SRT as a sig. predictor of variance. Pull Test was also sig. correlated with SRT. Findings suggest reaction time (SRT) may be as valid as balance measures for evaluation of disease progression in PD.
[20]	PD (n=78, m24 / f16) aged 67.17 (4.30) ON medication OA (n=27, m10 / f17) aged 68.37 (3.70)	Static postural control/Cognive load (Postuographic Balance Platform (AMTI Accusway Plus) Standardised standing EO & EC & 3 x EO DT (Cognitive part: WLG) Trial duration: 60s Static/ Dynamic postural control (BBS) Dynamic/Reactive/Proactive postural control (Mini- BESTest)	Global cognition (MMSE) Verbal fluency (Fluency test)	Both PD and OA had sig. greater postural sway during the DT condition in comparison to both EO and EC trials. These findings suggest that cognition has a sig. impact on postural control and are in conjunction with the literature.PD patients showed higher AP sway on all conditions. PD had higher variation in postural oscillations, suggesting PD patients were less able to adapt to the conditions. DT performance is suggested to be a potential detection for risk of falling in PD
[116]	PD normal cognition (n=71) aged 66.08 (7.16) Disease duration = 6.82 (5.67) UPDRS-III = 39.80 (1.51) LEDD. 39.8 (1.51) H&Y = 2.24 (0.07) OFF medication PD impaired cognition (n=72) aged 70.75 (8.24) Disease duration = 5.69 (4.15) UPDRS-III = 41.26 (1.50) LEDD = 734.57 (1072.42) H&Y = 2.32 (0.07) OFF medication	Static postural control (6 wearable Inertial sensors (OPALS, APDM Precision Motion of Clario)) NS *EO EC EO Foam EC Foam* Trial duration: 30s Dynamic/Reactive/Proactive postural control (Mini- BESTest)	Global cognition/cognitive dysfunction (MoCA)	No differences were found in sway parameters or clinical scale measures between both groups. A cut off, of 26, on the MoCA was used to split participants into normal cognition and impaired cognition groups. It is argued that this was not sensitive enough to observe differences in balance.
[78]	PD (n=162, m95 / 67f) aged 69.49 (8.1) UPDRS-III = 33.21 (11.5) PD + Diabetes Mellitus (n=36, m30 / 6f) aged 69.94 (7.6) UPDRS-III = 37.79 (13.9) OFF medication (n=84) ON medication (n=114) OA (n=170, 30m / 140f) aged 69.1 (9.8)	Static/Dynamic postural control (FAB scale) Static/Dynamic postural control (BBS) Dynamic postural control (Chair stand test) (trial duration: 30s) Static postural control (1 leg stand test) Dynamic postural control (TUG) Dynamic postural control (TUG DT) (counting backwards in 3's)) Dynamic postural control (FSST DT) Dynamic postural control (BPST)	Global cognition (MoCA) Executive function (CWIT) Problem solving (Tower test) Executive function (TMT-A, TMT-B, D- KEFS) Memory (BSMT) Visuospatial working memory (Reversed Corsi blocks) ttention (Serial subtraction in 3's)	PD patients made sig. less subtractions i.e., performed worse on TUG DT compared with OA but no significant differences were found in the FSST DT. These findings support previous literature showing that attention is important for successful dual- tasking on a dynamic balance test.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[61]	PD (n=39): PD FOG + (n=19, m14 / f5) aged 67.9 (7.9) Disease duration = 10.8 (6.6) UPDRS-III = 35.5 (11.9) LEDD = 548 (225.7) H&Y = 2.0 (0.35) PD FOG – (n=14, m8 / f6) aged 65.8 (10.8) Disease duration = 7.5 (6.3) UPDRS-III = 33.9 (13.4) LEDD = 538.8 (317.3) H&Y = 2.0 (0.0) ON medication OA (n=28, m8 / f20) aged75.4 (6.0)	Static postural control/ Sensory integration of postural control Three-Dimensional marker with 7 infrared cameras (Vicon, Oxford metrics) + 2 force places (CAREN) (Motek medical AMTI) NS *EO & EC & EO FOAM & EC FOAM)* Trial duration : 3 x 20s (continuous during surface condition) + all conditions + DT (cognitive part: forward 7-digit span) Dynamic/Reactive/ Proactive postural control (Mini-BESTest)	Global cognition (MMSE, MoCA)	Postural control was sig. worse for PD FOG+ than PD FOG- during DT for ML axis outcomes. Only FOG+ showed higher postural and cognitive DT cost than OA, indicating FOG+ required more attention during DT. This suggests an association between postural control disturbances and FOG. Lower MoCA scores were related to more DTC on postural stability. No correlations were found between cognitive measures and COP/COM within OA. Findings may explain why FOG + generally experience more falls than FOG-.
[43]	PD (n=92, m70 /f22) aged 67.6 (7.4) Disease duration (motor) = 6.0 (4.6) H & Y = 2.5 (0.6) UPDRS III = 35.5 (14.2): PD w/ Romberg 4 failure (n=33, m25 / f6) aged 71.7 (7.5) Disease duration (motor) = 6.4 (3.8) UPDRS III = 37.4 (14.5) LEDD = 675 (331) PD w/o Romberg 4 failure = (n=59, m45 / f14) aged 66.2 (6.3) Disease duration (motor) = 5.8 (4.9) UPDRS III = 33.1 (12.1) LEDD = 629 (430) OFF medication	Sensory integration of postural control (Modified Romberg test: EO + EC + EO on Foam + EC on Foam) Trial duration: 20s	Global cognition (MoCA)	Patients with PD who failed on the Romberg 4 (EC Foam) condition had sig. lower cognitive performance than those who didn't and were sig. more likely to have FOG. Findings suggest that cognitive function plays an important role in postural control.
[62]	PD (n=106, m82 / f24): PD w/ abnormal clinical balance (H&Y 2.5) (n=73 m56 / f17) aged 67.3 (7.6) Disease duration = 6.8 (4.2) UPDRS III = 41.01 (14.1) LEDD = 767.9 (623.2) PD w/ normal clinical balance (H&Y < (n=33, m26 / f7) aged 63.0 (7.05) Disease duration = 4.4 (3.4) UPDRS III = 28.4 (12.1) LEDD = 619.7 (336.2) ON medication OA (n=29, m13 / f16) aged 64.9 (12.3)	Sensory integration of postural control (Equitest posture test platform (Sensory Organisation Test (SOT) (Neurocom)) NS SOT 1 = EO SOT 2 = EC SOT 3 = EO + visual challenge SOT 4 = EO + surface challenge SOT 5 = EC + surface challenge SOT 6 = EO + visual challenge + surface challenge Trial duration: 30s	Global cognition (MoCA)	PD w/ abnormal balance had sig. worse global cognition than both PD w/ normal balance and healthy controls. Binary logistic regression of abnormal balance found MoCA score to be a sig. negative predictor of abnormal balance. Findings may in part reflect a shared pathophysiology between impaired balance and cognitive deficits.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[24]	PD (n=21): PDPI- (n=11, m9 / f2) aged 65.5 ± 10.8 years. Disease duration: 4.2 (3.0) LEDD = 725 (467) mUPDRS = 9.2 (6.3) PDPI+ (n=10, m7 / f3) aged 70.7 ± 7.0 years. Disease duration: 5.0 (3.2) LEDD = 894 (456) mUPDRS = 21.5 (4.0) ON medication OA (n=15, m10 / f5) aged 70.9 ± 8.7 years)	Dynamic postural control/reactive postural control (Clinical Balance Score) Sensory integration of postural control (Triaxial accelerometer (Brain Products) with Standardized standing on foam.	(Executive function) DCCS	Sig. negative correlations were found between DCCS and CBS scores. These findings demonstrate the considerable interrelationship between cognitive function, postural instability, and disease severity.
[71]	PD FOG (n=7, m5 / f2) aged 67.14 ± 8.78 years. MDS-UPDRS III = 28.60 (12.97) PD Non-FOG (n=7, m5 / f2) aged 71.71 ± 5.68 years. MDS-UPDRS III = 21.83 (11.51) ON medication OA (n=7, m5 / f2) aged 63.43 ± 9.79 years.	Dynamic/Reactive/Proactive postural control (Mini- BESTest) Dynamic postural control (TUG) TUG DT (Cognitive part: backwards counting in 3's)	N/A	PD FOG had sig. worse scores in most balance ability scores and the TUG DT and more DTI than both PD Non- FOG and OA. Findings are consistent with the notion that FOG, cognition and postural instability are related.
[14]	PD (n=23, m15 / f8) aged 66.25 (7.60) H&Y = 1.17 (0.18) Disease duration = 1.73 (1.51) UPDRS III = 9.0 (4.34) OFF medication OA (n=23, m16 / f7) aged 64.21 (7.27) UPDRS III = 1.91 (2.02)	Static postural control/Sensory integration of postural control (Wearable accelerometers – OPALS Inertial sensors (APDM) Standardised standing with feet together) EO EC EO DT EC DT (Cognitive part: backward counting in 3's from 100) Trial duration: 30s	Global cognition (MoCA, MMSE)	Early PD patients showed sig. sway acceleration and larger jerkiness values during cognitive task performance but not during single task, in comparison to healthy controls. These findings demonstrate a cognitive DT interference for PD patients. This is potentially a result of the compensatory attentional strategies used by PD patients, to maintain appropriate balance, being challenged.
[63]	PD (n=50, m26 / f14) aged 57.5 (9.6) Disease duration = 7.5 (3.6) UPDRS-III = 21.8 (7.8) LEDD = 1004.1 (424.8) ON medication	Static postural control (1 leg stance - preferred leg and unpreferred leg) Trial duration: 40s	Global cognition (MDRS)	One-leg stance performance decreased in relation to the MDRS score in people with PD. However, MDRS score was not sig. different between patients with a disease duration < 7 years and > 7 years. Yet, disease duration did sig. negatively impact one-leg stance performance on participants unpreferred leg but not on their preferred leg. The sig. association between cognitive function and unpreferred one-leg balance performance is explained by the authors as being an effect of age.
[74]	PD (n=13, m6 / f7) aged 71.7 (6.1) ON medication OA (n=13, m6 / f7) aged 65.8 (4.5)	Static postural control (Force platform (AMTI AccuGait) 100Hz) NS X 3 NS DT (cognitive part: counting backwards by one digit from 30) X 3 Trial duration: 40s	Cognitive dysfunction (MMSE)	There were no sig. differences in COP variables among the groups between ST and DT. However, PwPD committed more mistakes than OA during the standing DT. These findings support the principle of priority of position whereby an individual's attention tends to be allocated to control postural stability in a situation where simultaneous performance of two tasks is required, e.g., maintaining posture and performing a cognitive task.
[72]	PD (n=36, m24 / f12) Mild PD (n=12, m9 / f3) aged 66.75 (8.13) H&Y = 1.0 (n=6), 1.5 (n=6) Moderate PD (n=12, m8 / f4) aged 69.34 (7.75) H&Y = 2.0 (n=2), 2.5 (n=10) Severe PD (n=12, m7 / f5) aged 74.41 (6.22) H&Y = 3.0 (n=4), 4.0 (n=8) ON medication	Dynamic postural control (SST DT X 1) Dynamic/Cognitive load of postural control (TUG DT X 1) (Cognitive part: Verbal fluency, category naming: names, animals, fruits, foods, clothing) Trial duration: 30s	Global cognition (MMSE, MoCA)	Adding a cognitive task negatively affected performance on SST and TUG in PwPD. This effect was most prominent in severe cases of PD, compared with mild. Unexpectedly, performance on VF improved during TUG. It is suggested that prioritisation of the concurrent (cognitive) task occurred.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[11]	PD FOG (n=34, 22m / 12f) aged 66.32 (9.38) Disease duration = 8.21 (4.17) LEDD = 811.51 (264.55) UPDRS-III =51.62 (12.35) PIGD = 8.50 (2.36) H&Y = 3.0 (n=30) ON medication	Static/Reactive/Proactive postural control (Force plate (AMTI OR6-7)) Optoelectronic cameras (Vicon, Model T10) 200 Hz NS NS Foam Trial duration: 30s postural responses: Perturbations displacement range: 7cm Velocity max: 30 cm/s Step initiation: Move right foot on beep X 20	Global cognition (MMSE, MoCA) Inhibitory control/executive function (Stroop-III, TMT-B, DSST) Cognitive flexibility (FAB)	Multiple regression an analysis showed that TMT B explained 23% of APA duration variability. The Stroop test part 3 explained 42 % of quiet standing COP area on an uneven surface (EO Foam). Stroop 3 also showed a sig. correlation with APA duration and are of standing on a rigid surface uneven surface.These findings show an inverse relationship between inhibitory control and reactive balance in PD with FOG. This suggests that inhibition explains the control of automatic postural responses when balance is challenged with an uneven (foam) surface.
[101]	PD (n=56): PD FOG + (n=26, m21 / f 5) aged 69.2 (7.9) Disease duration = 8.3 (5.3) UPDRS III = 43.1 (10.0) PIGD subscore = 6.8 (3.8) LEDD = 875.5 (1320.6) PD FOG – (n=30, m21 / f5) aged 68.6 (8.4) Disease duration = 711.1 (360.8) UPDRS III = 38.7 (9.0) PIGD subscore = 4.5 (2.89) LEDD = 711.1 (360.8) OFF medication	Dynamic/Reactive/Proactive postural control (Mini- BESTest) Static/Dynamic postural control (8 OPAL inertial sensors (APDM) with Stand and Walk test (SAW)) NSNS DT (cognitive part: serial 3's subtraction from 206)Trial duration (standing component) :30s	Global cognition (MoCA)	No sig. differences between FOG + and FOG – were found in global cognition. FOG + also did not show sig. larger DT cost during NS. For postural sway measures during DT, only RMS AP was sig. larger for FOG+. Findings suggest FOG did not have a sig. effect on the ability to perform a cognitive task whilst standing. However, this may be explained by the insignificant differences in global cognition.
[35]	PD (n=15, m6 / f9) aged 67.7 (7.3) UPDRS (motor examination) = 24.5 (10.1) H&Y = 2 OFF medication YA (n=15, m8 /f7) aged 24.9 (4.9)	Static postural control/Sensory integration of postural control (Force platform (AMTI) & (Kistler) + EMG electrodes (Zero-Wires, Aurion Ltd.)) Standing in semi-tandem position EO EC EO-DT EC-DT Cognitive part: subtracting 3's from random number between 1 and 100. Trial duration: 3 x 60s Static/Dynamic postural control (BBS)	Global cognition - Normal cognitive function (MMSE)	No sig. differences were found between NS and NS DT for PwPD. No differences in EMG activity were found across all 4 conditions in either group. Findings are explained as cognitive task potentially providing an external focus, diverting attention from body sways, leading to more automatic postural control. It is also suggested that the cognitive part was not challenging enough.
[105]	PD (n=50, m31 / f19) aged 68.3 (7.3) UPDRS III = 19.1 (7.9) ON medication OA (n=60, m34 / f26) aged 68.9 (10.1)	Static postural control/Sensory integration of postural control (Pressure platform (Emed- AT25 D, Novel Inc)) NS EO EC EO DT EC DT Cognitive part: naming animals/naming words beginning with the letter R. Trial duration: 60s	Global cognition - Normal cognitive function (MMSE) Verbal fluency (SF, PF)	PD did not sig. influence balance during DT compared to OA. No sig. associations were found between COP parameters and VF/PF COP was worse during EC and EC- DT compared to the EO condition, for both groups. Authors suggest the lack of sig. differences may be a result of the cognitive task not having been challenging enough. There was also a limited amount of COP measures.
[22]	PD (n=52, m33 / f19) aged 67.3 ± 8.9 Disease duration: 7.9 (5.5) MDS-UPDRS III mean (SD) score: 18.8 (7.9) ON medication	Dynamic postural control (TUG) Static postural control (Plantar pressure platform) (EMED) NS EO Trial duration: 60s	Global cognition (MMSE) Executive function/Processing speed (RSCardsT, TMT A&B) Attention/Working memory (DST, DST-R)	Cognitive variables explained 23% of variability of anteroposterior displacement in static balance. LRA showed 25% of variability of TUG was explained by cognitive outcomes. TMT A was a sig. predictor of TUG mediolateral displacement and DST was found to be a sig. variable for anteroposterior displacement, suggesting these tests can be used as a predictor of balance control. Visual-spatial orientation, attention and working memory are likely most pertinent domains of cognition related to balance.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[117]	PD without balance issues (n= 29, m12 / f17) aged 69.2 ± 8.8 years. MDS-UPDRS III = 18.6 (5.5) Disease duration = 10.6 ± 5.2 years ON medication OA (n=12, m3 / f9) aged 66.5 ± 6.3	Static postural control (Posturographic force – platform (Pro-Kin 254) EO & EO DT (cognitive part: counting)) Task length: 2x 30s. NS Static/Dynamic postural control (BBS)	N/A	Total SD of body sway during EO DT were sig. higher in PD BBS > 51 compared with OA. Findings suggest that postuographic parameters are highly sensitive to balance control disruption in comparison to BBS values and can be used as an early balance disturbances diagnostic tool.
[48]	PD PIGD (n=30, 18 / f12) aged 64.65 (7.65) Disease duration = 5.70 (3.68) UPDRS III = 38.74 (10.48) PIGD score = 7.29 (3.10) TD score = 1.53 (0.93) PD TD (n=31, m26 / f5) aged 64.53 (11.77) Disease duration = 5.47 (3.14) UPDRS III = 39.47 (12.53) PIGD score = 1.81 (0.87) TD score = 11.97 (3.57) OFF medication	Static/Dynamic postural control (BBS) iTUG w/ 1 sensor (DynaPort Hybrid, McRoberts) iTUG DT Cognitive part: Subtracting serial 3's	Global cognition (MMSE) Attention/Executive function/working memory/Global cognition (Computerised cognitive battery)	PIGD took sig. longer to complete the iTUG. No sig. group differences were found for cognitive function. Sig. moderate correlations were found between iTUG measures (transition and turn) and cognitive measures (global cognition/executive function. Findings suggest cognitive decline impacts motor function and mobility (dynamic balance).
[21]	PD (n=12, m9 / f3) aged 67.5 (55.0 – 71.5) UPDRS III = 37.5 (21.0 – 47.8) LEDD = 327 (163.7 – 531.3) ON medication OA (n=10, m6 / f4) aged 62.5 (55.0 – 71.5)	Static postural control (Tri- axial activity accelerometer placed on 5th lumbar vertebra) NS Trial duration: 120s	Global cognition (MoCA) Attention (POA, FOA) Visuo-perceptual (VOSP) Visuo-constructive (CLOX 2) Visuo-spatial (JLO, CLOX 1)	Executive function (CLOX1) was sig. correlated with larger ellipsis in PD patients but not for OA. This suggests a non-linear pattern of response. Results may have been skewed due to small sample size and outliers.
[68]	PD (n=12, m8 / f4) aged 64.0 (9.08) ON medication OA (n=12, m8 / f4) aged 62.67(8.11)	Static postural control (Biomechanics platform (OR6-5, Mechanical Technology Inc.)) NS EO X2 EO DT (cognitive part: DST) X2 EO DT (cognitive part: describing familiar place (monologue)) X2 Trial duration: 30s	N/A	PD patients showed sig. less excursion along AP and ML axis and sig. smaller COP pathway during DT (monologue) than controls. Results suggest a sig. effect of cognitive load on postural stability. These findings suggest PD patients are over constraining their postural adjustments during increased cog. load. This effect suggests A maladaptive (relative to cognitive part of DT) posture-first strategy for PD patients during more challenging DT paradigms.
[23]	PD (PIGD) (n=783) aged 67.3 ± 9.7 years. MDS-UPDRS III = 26 (17 - 35) H&Y mean = 2 (2 - 2.5) ON medication	Reactive postural control MDS-UPDRS 3.12 (Pull Test)	Global cognition (MoCA & DRS-2) Executive function (LNS, TMT, SVF & DST) Memory (HVLT-R, BNT & WMS-R) Visuospatial function (JLO) Processing speed (DS)	Poorer performance of global cognition (MoCA, DRS-2), rocessing speed (DS), memory (HVLT-R) and phonemic fluency were associated with more severe postural instability. Findings are consistent with the idea that relationships between specific cognitive domains and distinct aspects of balance are mediated by multiple parallel neural pathways.
[49]	Novo-PD (n=35, m20 / f15) aged 63.63 (9.67) Disease duration = 1.18 (1.44) UPDRS III = 23.79 (9.67) PIGD subscore = 1.79 (1.10) "Drug-naïve patients" (No dopaminergic medication) OA (n=17, m7 / f10) aged 64.41 (9.76)	Dynamic postural control (FSST)	Global cognition (MoCA) Executive function (TMT B, COWAT, CWST, DSC) Language (BNT) Attention (DST) Visuospatial (RCFT)	TMT B and CWST were strongly sig. inversely correlated with FSST in PD. However, no correlations were found between FSST and MoCA, DSC or COWAT. Authors suggest this may be because TMT B and CWST are more visuospatially demanding, much like the FSST. Findings support the notion that visuospatial executive functioning plays an important role in dynamic balance.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[50]	PD (n=121, m76 / 45) aged 64.8 (10.4) H&Y = 2.4 (0.4) Disease duration = 4.4 (3.9) UPDRS III = 24.8 (9.8) LED = 572.15 (468) ON medication	Dynamic postural control (TUG) Dynamic postural control (360 Turn test)	Global cognition (MMSE) Executive control (SCWT) Attention (BTA) Working memory (SSB)	TUG was sig. correlated with attention and global cognition (BTA. MMSE). Findings may reflect a cognitive contribution to global functional performance which includes dynamic balance.
[39]	PD (n=88, m42 / f46) aged 67.3 (10.7) Disease duration = 8.2 (5.1)	Static postural control (Posturography (Gravicorder TM, Anima Corporation)) Romberg position EO	Global cognition (MMSE, MoCA) Executive function (FAB)	A sig. positive correlation was also observed between LNG/ENV and MMSE, MoCA and FAB. Findings demonstrate that cognitive impairment was worse among those with longer CoG and larger ENV.
[51]	PD (n=74) subtypes: PIGD (n=46, m31 / 15f) aged 69.04 ± 8.59 years. TD (n=28, m14 / f14) aged 68.82 ± 8.21 years. OFF medication	Static/Dynamic postural control (FAB Scale) Dynamic postural control (TUG test) Cognitive load of postural control (TUG DT) (cognitive part: counting backwards in 3's)) Dynamic postural control (FSST)	Global cognition (MoCA) Executive functioning (CWIT, ToL, S3sS, TMT, BSMT, CBST & RCBT) Mentation (MDS- UPDRS part I) Motor-cognitive function: (BPST)	PIGD group performed sig. worse on measures of global cognition (MoCA), visuospatial function and executive function (BSMT, CBTS, CWIT, TMT, TOL and BPST) and balance (FAB scale), than the TD group. PIGD deficits in executive function and spatial cognition may be related to impaired balance.
[44]	PD (n=31): PD H&Y 1 (n=10, m5 / f5) aged 67.26 (6.49) UPDRS (motor) = 8.20 (2.35) PD H&Y 2–2.5 (n=21, m10 / f11) aged 68.51(7.75) UPDRS (motor) = 19.88 (5.90) OFF medication OA (n=20, m10 / f10) aged 66.60 (7.80)	Sensory integration of postural control (Computerised dynamic postuography - Dual-force plate system (Smart Equitest, NeuroCom) SOT: Coniditons: 1. EO + fixed surface & visual surround 2.EO + fixed surface 3.EO + fixed surface and sway-referenced visual surround. 4.EO + sway-referenced surface and fixed visual surround 5.EO + sway-referenced surface. 6.EO + sway-referenced surface and visual surround Trial duration: 3x 20s MCT (perturbations): Forward/backward 2.8%/s Forward/backward 6.0%/s Forward/backward 8.0%/s	Global cognition (MMSE) Attention (DST (forward & backward)), Language (BNT) Visuospatial function (RCFT, drawing)	There was a sig. correlation between equilibrium score for condition 4, 5 and 6 and MMSE scores. This suggests postural sway increased with more impaired general cognition. There was also a sig. correlation between MCT for the backward (8.0%/s) condition and MMSE scores, showing motor latency increased with more impaired global cognition. The fact that this correlation only occurred in backward translation could be a result of stooped posture. These results may indicate reduced postural stability related to cognitive decline in PD. Equilibrium scores for conditions 2 and 6 sig. correlated with attention and verbal and visual memory. SOT condition 5 correlated with RCFT. Findings show a close relationship between postural instability and cognitive function in early-stage PD patients.
[69]	PD (n=24, m16 / f8) aged 66.4 (7.9) OA (n=20, m13 /f7) aged 60.9 (7.4) ON medication	Static Postuography force platform (QFP, Systemes, France) Standardised standing: heels together – forefeet open at 30° EO x2 EC x2 EO DT (motor) EC DT (motor) EO DT (cognitive) x 1 EC DT (cognitive) x 1 (Cognitive part: counting backward in 3's 8 trials total Trial duration = 51.2s UPDRS III (item 29) Tinetti test	Cognitive dysfunction/global cognition (MMSE)	During EO and EC single tasks (quiet standing) no sig. differences were found between PD and OA, but COP area was sig. increased for PD patients during motor and cognitive DT. Effect of cognitive DT was more sig. for PD patients that had experienced a fall than PD patients who had not. Findings suggest that balance of PD patients may worsen when their attention is diverted and highlights the importance of attention for postural control in PD.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[38]	PD (n=18, m10 / f8) aged 70.33 (1.85) Disease duration = 6.78 (1.99) H&Y = 3 OA (n=18, m6 / f12) aged 66.64 (1.34)	Sensory integration of postural control (Postuographic force plate) (Dinascan/IBV, Biomechanics institute of Valencia) Standardised standing: heels 3cmapart, toes pointing at 20° angle. EO EC EO + Foam EO DT EC DT EO DT + Foam Cognitive part: subtracting 3's starting at 100. Trial duration = 90s	Cognitive dysfunction (MMSE)	Mean Distance (MD) and Mean Time (MT) values were higher for PD. However Mean Peaks (MP) values were larger for OA. Both OA and PD showed decreased postural control during DT's. Findings suggests that PD patients have different postural control mechanisms, e.g., PD may maintain their balance through anticipatory commands of higher amplitude (higher MD) with lower periodicity (higher MT)
[118]	PD (n=102): PD w/ normal TG (n=34, 20m / 14f) aged 62.0 (10.0) Disease duration = 3.0 (2.6) LED = 266 (269) PD w/ abnormal TG (n=68, m40 / f28) aged 71.0 (9.0) Disease duration = 6.4 (5.4) LED = 430 (415) ON medication	Dynamic postural control (Tandem gait test) Reactive postural control (Pull Test)	Global cognition (MoCA)	Tandem gait abnormality in PD was associated with cognitive impairment (MoCA score < 24). Tandem gait was scored and defined by the relevant sections of the United Huntington's Disease Rating Scale and the Scale for the Assessment and Rating of Ataxia. A score of 1 or more on either scale was defined as abnormal gait.
[52]	PD (n=81) aged 67.4 (7.7) UPDRS III = 25.0 (12.9) ON medication OA (n=69) aged 69.9 (6.6) UPDRS III = 2.2 (3.8)	Static postural control (6 Inertial sensors (OPALS, APMD Inc.) Standardised standing by template NS NS DT (cognitive part: AX- continuous performance task) Trial duration = 60s	Global cognition (MoCA) Language (SF, BNT) Executive function/working memory/attention (PF, Stroop interference/reading, TMT B-A, LNST, TMT A) Visuospatial (JLO) Learning and memory (HVLTi, LMi, HVLTd, LMd,	PD patients showed sig. greater sway across all characteristics and conditions, compared to OA. People with PD also performed sig. worse on certain cognitive tasks e.g., LMi, TMT A, Stroop reading and SF. No sig. differences in DTC of sway were found between PD and OA.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[76]	PD (n=25, 15m / 10f) aged 57.6 (11.5) Disease duration = 4.42 (2.71) UPDRS III = 18.4 (8.69) LED = 751.52 (358.63) ON medication OA (n=20, 10m / 10f) aged 59.1 (13.3)	Static/ cognitive load of postural control (Tri axis force plate (Kistler 9286BA) Standardised standing (feet at 30 degrees angle, hip- width apart) Conditions: Seated DT EO ST EC ST EO DT EC DT (cognitive part: Phoneme monitoring: story listening and counting frequency of pre-established words) Trial duration: 60s	Global cognition/cognitive decline (MMSE)	PD patients demonstrated higher COP displacement, Sway area/ radius AP sway, ML sway, AP range and ML range than OA. Both PD and OA showed a shorter mean sway radius during DT compared to ST. PD patients performed sig. worse on phoneme monitoring task during standing compared to sitting, suggesting a postural control prioritization. OA showed the opposite. Findings are suggested to demonstrate that the attentional demand during standing is greater for PD compared to OA.
[73]	PD (n=30): PD w/ history of falls (n=15, 8m / 7f) aged 67.5 (8.6) Disease duration = 12.3 (4.9) Webster score = 13.3 (4.7) PD w/o history of falls (n=15, 8m / 7f) aged 68.5 (7.2)- Disease duration = 7.5 (5.6) Webster score = 7.9 (4.1) ON medication OA (n=15, 8m / 7f) aged 68.6 (7.7)	Cognitive load of postural control (Modified Webster Scale) Standardised standing: - feet 10 cm apart - feet together - feet 10 cm apart with heel of the front foot in line with toes of back foot. (Stride stance) - Tandem stance X 2 (both feet tested) - Single leg stance with non-weight bearing leg at 45° X 2 (both feet tested Each condition was performed with:NS NS DT (cognitive part: naming days of the week backwards Trial length: 30s Reactive postural control (Pull test)	Cognitive decline/Global cognition (STMS)	Sig. differences between groups were observed during the ST and DT conditions of stride stance, tandem stance and left single limb stance. PD fallers maintained balance for sig. shorter periods of time during DT than PD non fallers and OA. Yet, the differences between PD non- fallers and OA were not sig. Only right tandem chance was sig. worsened in DT condition, compared to ST, for the PD fallers. Steady stance results for OA and PD non-fallers were likely confounded by ceiling effects. PD fallers seemingly attended less to cognitive part of the DT as they stopped reciting words or slowed rate of recital. This may suggest there is a motor control hierarchy (posture/balance first). Postural stability worsened for all participants during DT compared to ST especially for tandem stance and arm raise test.
[54]	PD (n= 332): No GBA variant (n=289, m181 / f108) aged 68.2 ± 8.0 years. MDS-UPDRS III = 23.8 (12.0) With GBA variant (n=43, m28 / f15) aged 66.0 ± 9.6 years. MDS-UPDRS III mean (SD) score = 27.2 (14.7) ON medication	Static postural control (Inertial sensors (OPAL) with standardised standing) Task length: 60 s	Global cognition (MoCA) Executive function (TMT B-A, LNS) Visuospatial (JLO) Memory (HVLT-R, HVLT) Language (VF)	Structural equation Models (SEM) demonstrated sig. indirect relationship between GBA and both sway area (jerk) and sway velocity via cognition, but not for sway frequency. Findings confirmed that cognitive impairment was sig. associated with specific balance deficits and indicates cognitive impairment is likely a contributing factor to balance impairments I people with PD.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[53]	PD (n=198, m125 / 73) aged 67.6 ± 8.2 years. MDS-UPDRS III mean (SD) score = 25.0 (13.1) H&Y = 2.1 (0.5) Disease duration = 8.6 ± 5.4 years. LEDD = 654.0 (467.1) ON medication	Static postural control (Inertial sensors (APDM, Inc.) with standardised (normal) standing.) Trial duration: 60s	Global cognition (MoCA) Attention (LNS, TMT- A, Stroop R) Executive function (SF, TMT B-A, Stoop I, PF) Memory (HVLT-R immediate & delayed, LM I & II) Language (BNT) Visuospatial (JLO)	Only the balance domains of increased sway area and jerkiness were sig. associated with poorer attention and visuospatial function. Findings suggest that static balance is associated with posterior cortical control and highly dependent on vision.
[55]	PD (n=22) aged 69.45 (7.07) Disease duration = 9.70 (4.59) UPDRS (motor) = 28.25 (4.90) PIGD score = 2.8 (1.76) ON medication	Static postural control (Ground forceplate (Bertec Corp) NS X 4 Trial duration: 20s	Global cognition (MMSE) Executive function (DS backward, Stroop colour word) Language (CWA, VF (animals)	Moderate neg. associations were found between COP displacement and DS (backward), Stroop and CWA. No sig. associations were found between COP or UPDRS and MMSE or VF. There was however PIGD score negatively correlated with general cognitive function (MMSE) Findings demonstrate that postural stability was correlated with cognitive performance, in particular relating to frontal executive function performance, in people withearly/mild idiopathic PD.
[46]	PD (n=30, 16m / 14f) aged 67.1 (7.0) Disease duration = 5.4 (3.6) UPDRS III = 11.5 (21) H&Y = 2.1 (1.0) ON medication	TUG TUG DT (cognitive part: Digit span (forwards) TUG DT (cognitive part: Digit span (backwards) TUG DT (cognitive part: repeating 5 words – delayed memory recall) TUG DT (cognitive part: counting backwards the days of the week TUG DT (cognitive part: counting backwards from 20 UG DT (cognitive part: Animal naming test	Global cognition (MMSE) Executive function (Digit span – forward/backwards, animal fluency test, counting backwards from 20, counting days backwards)	MMSE scores were positively correlated with TUG DT (counting down days) and TUG DT (Animal naming). Yet, partial correlation analysis revealed no correlations between clinical, cognitive and TUG scores when MMSE scores were excluded. Positive association of TUG duration and TUG DT (CD days) was explained by two mechanisms: the cognitive deterioration that occur with disease progression but also a consequence of the relationship between cognitive function and dynamic balance. Neural pathways functioning in the TUG DT (CD days) may also constitute critical regions for dynamic balance and gait in PD
[75]	PD (n=28, 15m / 13f) aged 64.5 (7.0) Disease duration = 6.0 (2.7) UPDRS III = 38.0 (9.2) PIGD subscore = 2.7 (1.7) H&Y = 2.3 (0.4) ON medication OA (n=27, 10m / 17f) aged 63.8 (5.7)	Static/Sensory integration of postural control (Mobile device) (Apple iPad) EO double leg stance X2 (-/+ foam) (1&4) EC double leg stance X2 (-/+ foam) (2&5) EO tandem stance X2 (-/+ foam) (3&6) All conditions were also performed as a DT (cognitive part: depressing even numbers presented via headphones with right hand and uneven with left hand) Trial duration: 60s	N/A	Pairwise comparions showed that the PD group had sig. greater dual-task losses during the EC double leg stance + foam DT and EO tandem stance DT + foam condition compared to controls. These findings indicate that during the two conditions where sensory and proprioception were challenged to most, significant deficits in postural control and cognitive errors were demonstrated in the PD group, but not in the OA group. Moreover, cognitive errors significantly increased postural instability, relative to epochs without error in the PD group compared to OA, during condition 5 and 6. These findings demonstrate a time-synchronised phenomenon between cognitive errors and postural instability.
[16]	PD (n=14, 9m / 5f) aged 61.1 (6.1) Disease duration = 12.7 (6.2) UPDRS (motor) = 21.5 (9.2) PIGD = 2.7 (3.4) H&Y = 2.3 (0.6) ON medication	Six Inertial sensors (Mobility Lab, APDM Inc.) SWAY Standardised standing: hands across chest with feet shoulder width EO EC Hands across chest with feet together EO EC Trial duration = 30s	Global cognition (MDRS) Verbal fluency (COWAT Verbal memory (RAVLT, RAVLT delayed) Processing speed (SDMT) Working memory (ACT) Executive function (TOL)	Mean ML sway velocity correlated with total MDRS scores. UPDRS motor scores were modestly correlated with MDRS scores. This provides some supporting evidence that reduced global cognition is linked to balance impairment.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[64]	PD (n=50, 33m / 17f) aged 69.0 (9.6) UPDRS III = 37.8 (13.0) ON medication OA (n=59, 14m / 16f) aged 72.7 (7.6)	1 tri-axial accelerator (R2015a, Mathworks) NS Trial duration = 120s	Global cognition (MMSE, MoCA)	A weak negative correlation was found, in the PD group, between MMSE/MoCA and sample entropy along AP and vertical axes at 54 months. At 36 months only Sample Entropy at vertical axes was negatively correlated with global cognitive scores. This correlation supports the notion that postural and cognitive function may share common neural pathways that deteriorate over time because of PD.
[65]	PD (n=35, 23m / 12f) aged 66.0 (8.0) Disease duration at baseline = 0.45 (0.33) UPDRS III = 22.5 (9.6) H&Y at baseline = 1.71 (0.52) LEDD = 142.8 (113.1) ON medication	1 tri-axial accelerator (L5) (Axivity AX3) Standardised standing (400mm wide X 600 mm long) EO Trial duration = 120s	Global cognition (MoCA) Working memory (DST)	Jerk was sig. (moderate) negatively correlated with MoCa at 36 months after baseline assessment. At 54 months, there was a weak negative correlation with MoCA. This decline in jerk between 36 and 54 months showed a (moderate) negative correlation with the change in MoCA scores. These findings suggest shared neural pathways between cognition and postural control.
[56]	PD (n=82, 55m / 27f) aged 66.5 (7.6) Disease duration = 7.5 (5.7) UPDRS motor = 31 (12) H&Y = 2.1 (0.7) LED = 728 (430) ON medication	Static postural control (Single-leg-stand) Trial duration = 30s UPDRS motor Reactive postural control (Push and release test) MDS-UPDRS 3.12 (Pull Test)	Global cognition (MMSE) Executive function (FAB)	Executive function is sig. associated with single leg stand and choice stepping. Findings suggest that motor impairments and executive function had task-dependant relationships with balance in people with PD.
[119]	PD (n=21, 12m / 9f) aged 68 (8.5) Disease duration = 7.28 (4.4) UPDRS III = 21.8 (9.6) H&Y = 2.5 (0.5) ON medication OA (n=21, 7m / 14f) aged 68.9 (5.5)	Static/Dynamic postural control (FAB scale)	Visuopatial (computerised test)	Results from the spatial orientation task did not differ sig. with the FAB scale. These findings are evidence against a relationship between spatial perceptual deficits and postural control in PD. This suggests that there are more complex deficits that may affect the integration of spatial or sensori-motor information that may affect balance control in PD patients.
[57]	PD (n=243): PD w/ normal EF (n=87, 49m / 38f) aged 64.4 (9.9) UPDRS III = 22.0 (13.2) PD w/ mild executive dysfunction (n=100, 63m / 27f) aged 69.1 (7.5) UPDRS III = 33.7 (13.5) PD w/ marked executive dysfunction (n=56, 34m / 22f) aged 72.2 (7.8) UPDRS III = 41.8 (15.5) ON medication	Static/Reactive/Sensory integration of postural control (PPA) Static postural control (Coordinated stability test.)	Executive function (FAB)	Both executive dysfunction groups had sig. greater postural sway (PPA), worse leaning balance and higher UPDRS III scores than the normal EF PD group. The marked executive dysfunction group had worse overall balance (increased sway and worse UPDRS III and coordinate stability scores) than the mild executive dysfunction group. The association between EF and balance may be due to underlying common neural pathways as degeneration of the cholinergic system can cause both motor and non- motor impairments in PD.
[79]	PD FOG + (n=15) aged 71.6 (6.0) Disease duration = 11.5 (6.2) H&Y = 2.4 (0.5) PD FOG- (n=17) aged 67.9 (8.9) Disease duration = 5.7 (4.1) H&Y = 1.7 (0.4) ON medication OA (n=24) aged 68.0 (6.9)	Static postural control/Sensory integration of postural control (Force platform (FDM-T) (Zebris medical GmbH, Germany)) 100 Hz NS EO NS EC NS EC DT (cognitive part: counting backwards from 100) Trial duration: 30s	N/A	For PwPD compared with OA, the EC DT sig. increased sway radius and decreased sway path length and normalised sway path. Age and freezing were found to be independent predictors of a higher sway radius with EC. EC DT sig. increased sway path length in patients with FOG but normalised sway path did not show sig. differences. The sway path length did not increase from EC to EC DT for any group. This suggests the cognitive load may not have been high enough as EC condition already sig. increases sway measures.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[77]	PD (n=16) aged 71.4 ± 5.6 years. H&Y mean = 2.3 (2 - 3) MDS-UPDRS III = 41.2 (8.8) ON medication OA (n=14) aged 69.6 ± 7.3 years	Dynamic/Reactive/Proactive postural control (Mini- BESTest) NS DT with 5 backward & 2 forward (Cognitive part = auditory Stroop task) Trial duration = 8 stimuli perturbations caused by treadmill accelerations ST & DT (AST) Full-body Helen Hayes markers (Vicon Corp) EMG electrodes NS DT Trial duration = 11 stimuli	Global cognition (MoCA) Cognitive control (AST)	Both people with mild PD and OA showed a significant prioritization of postural control over cognition during DT. No significant differences between groups. Findings suggest that mild PD did not sig. affect postural or cognitive dual task interference.
[37]	PD-PIGD (n=23, 15m / 8f) aged 65.1 (7.6) Disease duration = 8.1 (3.6) UPDRS III ON = 26.6 (8.9) UPDRS III OFF = 33.3 (11.3) ON & OFF medication OA (n=23, 10m / 13f) aged 63.7 (8.7)	Six-camera optoelectronic SMART-DX7000 system (BTS Bioengineering) Dynamic/Reactive/Proactive postural control (Mini- BESTest) Dynamic postural control (TUG) TUG DT (cognitive part = subtracting 3's)	Global cognition (MMSE) Executive function (PF, SF, TMT B-A, DS backward)	Worse PF, SF and TMT B-A were associated with worse Mini-BESTest scores. Worse balance was also associated with worse TUG DT performance. Findings support the notion that executive dysfunction contributes to balance deficits in PD.
[66]	PD (n=69, 45m / 24f) PD No MCI (n=37, 29m / 8f) aged 65.10 (8.61) Disease duration = 8.27 (9.01) H&Y = 2.13 (0.85) UPDRS = 29.54 (17.38) LEDD = 572.7 (419.1) PD MCI (n=32, 16m / 16f) aged 68.90 (11.03) Disease duration = 7.96 (6.39) H&Y = 2.46 (0.91) UPDRS = 43.18 (21.96) LEDD = 781.7 (528.9) Medication state not reported.	Static/Dynamic postural control (BBS) Dynamic postural control (TBAT)	Global cognition/cognitive dysfunction (MMSE < 24)	The PD MCI group performed sig. worse than the PD No MCI group on the BBS and TBAT. These findings suggest a correlation between cognitive dysfunction and balance issues.
[45]	PD (n=6, 2m / 4f) aged 70.83 (15.89) ON medication OA (n=6, 2m / 4f) aged 70.17 (4.71)	Static postural control/Sensory integration of postural control (Force platform (Bertec 4060-NC)) NS EO X4 EC X4 EO DT X4 EC DT X4 (Cognitive part = Matching 3D object image) Trial duration = 40s - 10s (viewing 3D image) + 30s (sway)	Global cognition/Cognitive dysfunction (MMSE < 26)	The cognitive task did not affect postural sway. This may have been a result of the cognitive part not being tasking enough to produce an effect on postural sway. Moreover, the most challenging part of the DT i.e. the image matching, occurred after sway measures had already been collected.
[70]	PD (n=25, 21m / 4f) aged 65.8 (9.5) Disease duration = 6.5 (4.5) UPDRS (motor) = 10.9 (4.5) ON medication OA (n=16, 4m / 12f) aged 65.9 (9.7)	Static postural control (Force platform (Kistler, Winterthur)) NS (quiet) NS DT (quiet) NS (still) NS DT (still) (instructions to stand as still as possible) (Cognitive part: category naming) Trial duration = 60s	N/A	For the PD group, CoP on the ML axis was sig. larger during the still NS DT compared with the quiet NS DT and the still NS. CoP increased for the OA group in NS DT (quiet). VCoP on ML axis was increased sig. in OA group during NS DT quiet. For PD, both DT conditions increased VCoP ML compared with NS without DT. VCoP in AP direction showed effect of DT in OA but not in PD. Focusing on posture reduced (cognitive) responses and increased ML CoP displacement variability and velocity in DT conditions in PD. This suggests that consciously focusing on balance had a detrimental effect on stability. PD patients may prioritize attentional resources to either motor or cognitive performance, during DT, depending on the task.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[40]	PD (n=232, 153m / 129f) aged 64.4 (7.9) UPDRS III = 33.4 (9.1) Medication state not reported.	Dynamic postural control (TUG)	Global cognition/ cognitive dysfunction (MMSE) Spatial working memory (SWM - CANTAB subtest) Set shifting (CANTAB subtest) Inhibitory control (Stroop) Verbal fluency (WNG) Semantic fluency (naming animals)	Fluency tests and spatial working memory were sig. associated with the TUG test. Semantic fluency was sig. associated with mobility independent of severity of motor signs and age. Results provide evidence that in non-demented patients with PD, certain executive functions are associated with dynamic balance.
[58]	PD (n=107, m79 / f28) aged 61.0 ± 8.2 years. Disease duration: 5.5 (4.1) ON medication	Dynamic/Reactive/Proactive postural control (Mini- BESTest) Dynamic postural control (TUG) TUG DT (Cognitive part: DST)	Working memory (DST) Visuomotor speed/ visuospatial attention (TMT A&B) Global cognition (ACE-III)	Sig. associations were found between Mini- BESTest and cognitive measures (TMT A/B, DST forward, ACE-III) and between TUG and cognitive measures (ACE-III, TMT-A/B, DST forward/backwards) Findings show that balance measures were sig. related to cognition. In particular domains of executive function, and global cognition. Findings demonstrate a critical role of attention, visuospatial ability and mental flexibility in relation to balance.
[13]	PD (n=71, 52m / 19f) aged 63.4 (6.7) Disease duration = 8.7 (5.7) H&Y = 2.5 (0.3) UPDRS (motor) 28.3 (10.8) ON medication	Static postural control/ sensory integration of postural control (Postuography platform) (AMTI AccuSway, AMT INC.) NS EO X3 EC X3 Trial duration: 60s Static/ Dynamic postural control (BBS) Dynamic/Reactive/Proactive postural control (Mini- BESTest) Dynamic postural control (TUG)	Global cognition/cognitive impairment (MMSE) Executive function (TMT A/B, SVF)	Sig. correlations were observed between dynamic balance (step over obstacle) and executive function (TMT). TMT and SVF were correlated to dynamic postural control, but not static postural control. It should be noted that this sample was in the relative early stages of PD.
[67]	De novo PD (n=422) PIGD (n=76, 47m / 29f) aged 61.91 (9.23) Disease duration = 0.52 (0.46) UPDRS III = 21.00 (8.38) TD (n=299, 199m / 100f) aged 61.89 (9.47) Disease duration = 0.56 (0.55) UPDRS III = 21.14 (9.04) ON medication	Reactive postural control (MDS-UPDRS 3.12 (Pull Test))	Global cognition (MoCA) Verbal fluency (SF, HVLT) Visuospatial attention (JLo) Working memory (SDMS, LNS)	After ~ 5 years, 36.3% of PD patients with balance issues at baseline developed cognitive impairment in comparison to 18.3% of those without postural impairment at baseline. Postural instability was a sig. predictor of cognitive impairment. Cognitive decline was however not associated with motor subtype. (PIGD vs TD) Findings suggest that motor subtype does not predict cognitive impairment, at least not in newly diagnosed patients. Postural instability, however, may be an indicator of risk of cognitive decline.
[59]	PD (n=28, 16m / 12f) aged 64.9 (7.1) Disease duration = 6.5 (2.9) H&Y = 2 UPDRS III = 27.3 (12.8) OFF medication OA (n=20, 12m / 8f) aged 66.1 (7.5) UPDRS III = 3.8 (4.5)	Dynamic postural control (iTUG) 1 inertial sensor (triaxial accelerometer + gyroscope) (DynaPort, McRoberts BV, The Netherlands) Trial 1: self-selected fast speed X 2 Trial 2: self-selected convenient speed X 2	Global cognition (MoCA) Executive function (TMT A/B)	None of the iTUG parameters were found to correlate with global cognition. However, turning duration and walk to sit maximum flexion velocity correlated with executive function scores. These findings suggest a connection between dynamic balance and executive function in PwPD.
[60]	PD (n=20, 12m / 8f) aged 70.3 (6.34) Disease duration = 10.65 (5.19) ON medication	Static/Dynamic postural control (BBS) Dynamic postural control (TUG) TUG DT (cognitive part: backward counting)	Global cognition/cognitive dysfunction (MoCA, FAB, MMSE) Executive function (TMT A/B) Memory (MI) Verbal fluency (SVF)	The TUG DT was sig. correlated with the MoCA and TMT B The BBS was sig. associated with the FAB, MoCA and negatively correlated with the TMT B. The TUG was also sig. associated with the MoCA and negatively with the TMT B. Measures of balance and dynamic balance were sig. associated measures of attention, executive functions, and global cognition. These findings support the notion that cognitive function and balance are positively associated in PD patients.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
Vervoort et al. 2015	PD (n=73) PIGD (n=43, 29m /14f) aged 61.4 (10.9) Disease duration 7.3 (4.3) UPDRS III = 30.5 (13.9) PIGD score = 4.3 (2.8) LED = 4.0 (4.1) TD (n=22, 12m / 10f) aged 57.3 (8.8) Disease duration = 4.8 (2.7) UPDRS III = 26.3 (10.9) PIGD score = 1.5 (0.9) LEDD = 286.4 (263.9) OFF medication OA (n=20, 14m / 6f) aged 57.7 (8.8)	Dynamic/Reactive/Proactive postural control (Mini- BESTest)	Global cognition/cognitive dysfunction (MoCA, FAB, MMSE) Executive function/task switching (TMT A/B)	The TD subtypes did not differ significantly with the controls on the Mini-BESTest but the PIGD group did perform sig. worse. PIGD also performed sig worse than controls on the TMT B and FAB, but the TD group did not. Yet, there were no sig. differences in cognitive performance between PD subtypes. Findings are in line with the idea that the PIGD phenotype could be associated with a greater loss of the cholinergic system affecting common neural pathways associated with both cognitive function and postural control.
[120]	PD Non-fallers (n=90, 55m / 35f) aged 67.25 (8.08) Disease duration = 5.3 (4.1) LEDD = 577.79 (337.22) UPDRS III = 4.1 (2.6) PD fallers (n=54, 38m / 16f) aged 70.81 Disease duration = 7.6 (5.9) LEDD = 645.59 (449.11) UPDRS III = 6.5 (3.3) OFF medication	Static postural control/Sensory integration of postural control (8 inertial sensors (OPALS, APDM, Portland OR, USA)) 128 Hz NS EO NS EC NS EO Foam Trial duration: 30s Hands on hips Feet together Dynamic/Reactive/Proactive postural control (Mini- BESTest)	Global cognition (MoCA)	MoCA score did not sig. differ between fallers and non- fallers. Cognition as measured by MoCA scores was not good at predicting fall- status as there were no sig. differences between fallers and non-fallers.
[41]	PD (n=20, 13m / 7f) aged 65.9 (9.4) Disease duration = 6.0 (3.8) UPDRS III = 26.6 (10.8) H&Y = 1.4 (0.9) PIGD = 4.5 (3.6) LED = 622.25 (477.17) Medication state not reported. OA (n=20, 13m / 7f) aged 68.9 (4.8)	Dynamic postural control (TBAT) Static/Dynamic postural control (BBS) Dynamic postural control (TUG) Dynamic postural control (FRT)	Cognitive dysfunction/global cognition (MMSE, ACE) Reaction time (computerised cognitive battery) Executive function (TMT A/B)	PD patients performed sig. worse on all measures of balance. Worse performance on all TMT tests were sig. associated with worse TUG performance in the PD group. Worse TMT A/B was also sig. associated with worse BBS scores. Moreover, TMT A was sig. positively associated with the TUG test in PD. Slower reaction times were sig. correlated with lower BBS scores and negatively associated with TBS scores in PD. There were also sig. correlations between executive function measures and PIGD/UPDRS (motor) scores. No sig. correlations were found between ACE or MMSE and balance for either group and in the OA no correlations between any measures of cognition and balance were found. These findings show that executive dysfunction was associated with worse balance performance across several tasks. Findings support the notion that specifically executive function and attention are important regarding postural control in PD patients, more so than global cognition.
80	PD (n=62, m36 / f26) Early-PD (n=38, m22 / f16) aged 69.0 ± 8.6 years. Disease duration: 4.0 (2.9) H&Y = 1.7 (0.5) LEDD = 373.9 (259.7) Advanced PD (n= 24, m14 / f10) aged 70.1 ± 5.4 years. Disease duration: 8.17 (6.0) H&Y = 3.25 (0.4) LEDD = 699.1 (560.0) ON medication OA (n=40, m15 / f25) aged 67.2 ± 7.7 years.	Static postural control (Postuographic force plate (Pro-Med 2010)) NS NS DT (cognitive part: counting backwards) Trial length: 2 X 32 s	Global cognition (MMSE) Attention/Executive functioning/working memory (CBT)	NS DT reduced postural sway in the advanced PD group and increased postural sway in the HC group compared to NS. No Sig. differences were found between NS and NS DT in early PD. Results are consistent with the notion that PD patients may over- constrain their posture to focus attention during DT. Reduced body sway in advanced PD is explained as cortical resources are directed to cognitive task, resulting in stabilisation of posture beyond normal levels.

Table 1 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cognitive domain (tests)	Key (pertinent) findings and conclusions
[36]	PD (n=15, m6 / 9f) aged 73.53 ± 9.72 H&Y stage I (n = 5), II (n = 6), and III (n=4) ON medication MAA (n=10, m4 / 6f) aged 52.8 (5.14) OA (n=14, m5 / 9f) aged 72.36 (9.33)	Static postural control/Sensory integration of postural control (Posturographic force plate (PROMED)) NS EO & EC Trial duration: 32 s	Global cognition (MMSE) Executive funcion (CBT)	No significant differences in stabilographic parameters between OA and PD, except anterior- posterior sway velocity during EO test. CBT and MMSE scores were worse in PD than MAA and OA. COP parameterscorrelated with MMSE scores. Findings suggest there is a relationship between cognition and balance and PD patients experience significant cognitive dysfunction compared to healthy controls.

ACE-III Addenbrooke's cognitive examination-third version, *AP* anterior-posterior, *BBS* Berg Balance scale, *BNT* Boston naming test, *BPST* Body Position Spatial Task, *BSMT* Brooks Spatial Memory Task, *BTA* Brief Test of Attention, *CANTAB* Cambridge Neuropsychological Test Automated Battery, *CB* Corsi Blocks, *CBS* Clinical Balance Score, *CBT* Counting backwards test, *COP* Center of pressure, *COWAT* Controlled Oral Word Association Test, *CWA* Controlled word association, *CWIT* Colour World Interference Test, *DRCFT* Digit span test, *DRCFT-R* Digit span test reversed, *DSC* digit symbol coding, *DRS-2* Mattis Dementia rating scale-2, *DST* Digit span test, *DST-R* Digit span *test reversed, DT* dual task, *EC* eyes closed, *EMG* electromyography, *ENV* envelope area, *EO* eyes open, *FRT* functional reach test, *FAB* Frontal Assessment Battery, *FAB Scale* Fullerton Advanced Balance Scale, *FOG* freezing of gait, *FSST* Four square Step Test, *HVLT-R* Hopkins verbal learning Test-Revised, *HVLTd* Hopkins verbal learning test delayed, *H&Y* Hoehn and Yahr, *iTUG* instrumented timed up and go, *JLO* judgment of Line Orientation, *LEDD* Levodopa equivalent daily dose, *LMI* Logical memory immediate, *LMD* logical memory delayed, *LM* Logical memory, *LNS* Letter-number sequencing, *LOS* limits of stability, *LRA* Linear regression analysis, *LNG* Length of the centre of gravity, *MAA* middle-aged adults, *MBT* Mini-BESTest, *MCT* Motor Control test, *MCI* mild cognitive impairment, *MDRS* Mattis Dementia rating scale, *MI* Memory interference, *ML* mediolateral, *MMSE* Mini-Mental State Examination, *MoCA* Montreal Cognitive Assessment, *MDS- UPDRS III* Movement Disorder Society – Unified Parkinson's Disease Rating scale, *MoCA* Montreal Cognitive Assessment, *N/A* not applicable, *NS* normal standing, *NS DT* normal standing dual task, *NUCOG* Neuropsychiatry Unit Cognitive assessment tool, *PD* Parkinson's disease, *PF* Phonemic fluency, *PKMAS* Photokinetic Movement Analysis Software, *PIGD* postural impairment and gait difficulty, *POA* power of attention, *PPA* physiological profile assessment *PwPD* people with Parkinson's disease, *RAVLT* Rey Auditory Verbal Learning Test, *RCBT* Reverse Corsi Block Task, *RCFT* Rey Complex Figure Test, *RSCardsT* Rule shift cards test, *RULIT* Ruff-Light Trail Learning test, *SF* Semantic fluency, *S3S* Serial 3s Subtraction, *SDMT* Symbol digit modalities test, *SOT* Sensory organization test, *SSB* Spatial span backwards, *Stroop I* Stroop interference, *Stroop R* Stroop reading, *SVL* Semantic verbal fluency, *TBAT* Tinetti Balance assessment tool, *TMT* Trail Making Task, *TUG* Timed up and Go, *UPDRS* Unified Parkinson's Disease Rating Scale, *VCOP* vertical center of pressure, *VF* Verbal fluency, *VOSP* Visual Object and Space Perception Battery, *WLG* World list generator, *WMS-R* Wechsler Memory scale, *YA* younger adults

aspects of balance. Namely, static, and dynamic balance and only dynamic balance, respectively.

Outcome measures from objective assessments of postural control varied across studies and included sway parameters and anticipatory postural adjustments (APAs) derived from digital measuring devices such as inertial measurement units (IMUs) and force platforms. Moreover, clinical rating scales and tests such as the TUG, Mini-BESTest and BBS were also included. The postural control domains that each assessment/measurement device was classified as, for purpose of this review, is shown in the Tables 1, 2 and 3 under "Postural control domain". Which domain each measurement device/assessment tested is considered by the authors of this review to be part of is shown in Fig. 3.

Several studies challenged the visual system during standing, e.g., eyes open/eyes closed (n = 20), and proprioception e.g., with a foam surface (n = 8). Two studies used the Smart Equitest system (Neurocom) to manipulate both vision and proprioception [43, 44]. Manipulating cognitive load during standing trials was often achieved by employing a cognitive dual task (DT) (n = 26). The most common cognitive test used was serial 3 s subtraction (n = 9), followed by counting backwards (n = 5) and category naming (n = 4). Other cognitive parts of dual tasks (DTs) included word list generating (n = 1), digit span forward (n = 3) and backwards (n = 1), counting (n = 1), describing a familiar place, and naming days of the week backwards (n = 2). A small number of studies (n = 3) involved listening to a story and either counting pre-determined words (n = 1) or depressing a button (n = 2) when they heard the pre-established word. One study [45] provided visual stimuli which was a 3D object which patients had to remember and match. Another study [46] used 6 different cognitive tasks during DTs: digit span (forward & backward), repeating 5 words, naming days backwards, counting backwards and animal naming.

Cognitive measures

Global cognition was the most widely tested domain. Studies typically used either the MoCA (n = 27) or MMSE (n = 31). Executive function/attention (n = 31) was the most tested cognitive domain, most commonly by the Trail-Making Test (TMT) (n = 18), followed by and visuospatial function (n = 11), which was most commonly examined with the Judgement of Line Orientation (JLO) (n = 6). Other cognitive assessments included reaction time (n = 2), and verbal/semantic fluency (n = 7).

Outcomes

Cognition and performance on postural control tasks were generally found to be positively associated, i.e., poorer cognition was associated with poorer postural control. Specifically, executive function and attention were among the domains most often positively correlated with Postural control performance (n = 23) [4, 11, 13, 22, 24, 37, 39–41, 47–60], in addition to global cognition (n = 22) [4, 15, 16, 23, 36, 39, 43, 46, 48, 50, 51, 54, 55, 58, 60–67] *(*Fig. 4*.)*

For the studies that incorporated DTs, there was a range of different findings. The majority of results suggest that a cognitive load has a negative impact on postural control (n = 15), e.g., increased sway measures (n = 7) [14, 20, 38, 61, 68–70] and worsened balance task performance (n = 5) [37, 60, 71–73]. For some studies (n = 8), the significant findings pertained to the cognitive part of the DTs, where PwPD showed more errors [61, 74–77], reduced responses [70, 73] and less subtractions [78], compared to healthy controls. Interestingly, a subset of studies (n = 6) demonstrated unexpected outcomes, such as a significant decrease in postural sway measures (i.e. improved performance) and excursion during specific cognitive tasks [68, 76, 77, 79, 80] and improved performance in dynamic balance [72].

Brain-imaging studies (during task performance) (n = 9)

Participants

The average age for PD patients was approximately 66.69 years. Of the nine studies from which data were extracted, eight included the use of healthy control groups, which had a very similar average age of approximately 68.29 years. All studies included both male and female participants. Two studies tested people ON medication and one study tested patients in an "ON" and "OFF" state. The remaining studies (n=6) tested subjects OFF dopaminergic medication or did not provide those details.

Brain imaging devices

As shown in Table 2. Only one study used fNIRS to measure cortical activity in relation to postural control, using a 29-channel array of optodes at 8.36 Hz, measuring PFC activity, during the TUG task [81].

Three studies used fMRI to assess brain activity during balance-related task performance, either with mental imagery balance-related tasks (n = 1) [82] or a built-in force platform enabling stimulation of the feet in a supine position (n = 2) [83, 84].

The remaining studies (n = 5) all used EEG to assess cortical control during a variety of tasks challenging postural control [24, 85–87]. Sampling frequencies ranged from 500 to 4096 Hz with systems ranging from 32 to 64 channels. Three studies used a larger system with 64 channels [24, 85, 88] and one study used a 32-channel system [86]. One other study used a 30-channel EEG

system [89]. All EEG systems differed in type, brand, and model.

Balance measures/tasks and Dual tasks

A force plate was used in three studies [85, 86, 89] to assess postural control performance with sway measures. One study provided visual feedback whilst participants were encouraged to sway mediolaterally [88]. Another [86] used the force plate to administer perturbations, and one [89] had various standing conditions including motor and cognitive DTs. One study used triaxial accelerometers to measure postural sway whilst proprioception was challenged using a foam pad surface [24]. Trial durations ranged from 10 to 100 s.

Regions of interest

The study that used fNIRS to measure cortical activity only examined the PFC [81]. This is likely a result of technological limitations. Hemodynamic differences in the left, right and total frontal cortices were found during dynamic balance performance in PwPD [81]. The EEG studies covered a wider range of cortical ROIs. For example, parietal, frontal, occipital and central or one of the sensorimotor cortices e.g., the primary motor cortex (M1), primary sensory cortex (S1), primary visual cortex (V1), and auditory areas (STS/STG) [24, 88].

Outcomes

In the single fNIRS study found, hemodynamic variations were observed in various frontal cortices during the TUG task in PwPD [81], with left frontal cortex HbO2 levels surpassing those on the right in high-risk fallers. Additionally, frontal cortex HbR concentration negatively correlated with disease duration.

Some studies used fMRI to assess event-related BOLD signal during (balance-related) task performance in a supine position within an MRI-scanner. This included APA's [83], mental imagery [82] and a simulated balance task [84]. PwPD with impaired postural control and a sleep disorder had *decreased functional connectivity between the pedunculopontine nucleus (PPN) and the SMA which also correlated negatively with the duration of APAs i.e., proactive postural control. Furthermore, this group showed* showed a lack of focused brain patterns in the parietal-frontal areas, compared to healthy controls.

During a simulated dynamic balance task, PwPD had increased functional connectivity in regions associated with motor control and parietal areas, compared to OA. They also showed decreased connectivity between the brainstem and subcortical areas, compared to OA. PwPD were also found to have increased connectivity from the SMA and premotor areas to the pre-SMA and the superior parietal cortex to the precuneus. Furthermore, PwPD showed an increase in connectivity from the dorsal premotor area to the pre-SMA.

The remaining EEG (n = 5) studies had a variety of outcome measures [24, 85, 86, 88, 89]. These generally showed dissimilar patterns in neural activity and postural performance between PwPD and OA (n = 5). For example, PwPD with postural instability showed lower theta-band power in the mid-frontal and mid-cerebellar regions during a postural control task, compared to both OA and PD patients without postural issues [24] (Fig. 4). Similarly, another study [85] found absolute power spectral density (PSD) for alpha waves was lower in people with early PD compared to OA, whilst sway was significantly higher in OA. This study also found differences between PRE- and POST-phase PSD. Significant differences in theta band were found between the temporal, frontal, and occipital lobes and alpha band power in the temporal, parietal, and occipital lobes. PwPD also showed a significant increase in normalized alpha modulation in the primary visual cortex and beta modulation in the primary motor cortex during perceptual-motor tasks, compared to healthy older adults [88].

Functional connectivity strength differences, based on the phase-lag index (PLI) of 30 electrode pairs among early-stage PD patients and older adults (OA), were measured using EEG during various standing trials [89]. Both PD patients and older adults successfully reduced postural sway during a trial whereby they were asked to focus on postural control, corresponding to improved postural control performance. PD patients showed higher mean inter-regional connectivity strength during postural single-task and dual task (DT), whilst older adults showed a decrease in inter-regional connectivity, during the DT condition, compared to the single-task.

Contingent negative variation (CNV) in the alpha (8–13 Hz) and beta (13–30 Hz) bands and event-related desynchronization (ERD) were used to analyse cortical activity during perturbations (anticipation) [86]. Among older adults, no associations were found between postural control performance and cortical activity in the primary and supplementary motor areas. However, in PD patients, increased beta ERD was linked with decreased adaptability i.e., worse postural performance.

Brain-imaging studies (resting-state associations with postural control) (n = 7)

Participants

PwPD were an average of 64.64 years old. Most studies (n = 8) used aged-matched controls [82, 84, 90–95], with one study using healthy young adults [83]. Three studies separated participants based on FOG status: freezers vs non-freezers [82, 95, 96].

Table 2 Extracted data from brain-imaging studies measuring brain activity during postural control-related task performance. (n = 9)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cortical activity measurement device and ROI's	Main findings and interpretation
[24]	PD (n=21): PDPI- (n=11, m9 / f2) aged 65.5 ± 10.8 years. Disease duration: 4.2 (3.0) LEDD = 725 (467) mUPDRS = 9.2 (6.3) PDPI+ (n=10, m7 / f3) aged 70.7 ± 7.0 years. Disease duration: 5.0 (3.2) LEDD = 894 (456) mUPDRS = 21.5 (4.0) ON medication OA (n=15, m10 / f5) aged 70.9 ± 8.7 years)	Static postural control/Sensory integration of postural control (Triaxial accelerometers (Brain Products) with standardized standing on foam.) EO seated EO FOAM Trial duration 3–4 minutes	EEG Easycap (500Hz) 64 channels Analysed channels (spectral properties; theta-band (4–7 Hz) & beta-band (13-30Hz)): C3 & C4 (lateral motor cortex), Cz (mid- frontal premotor), Oz (mid-occipital) & Cbz (mid-cerebellar) Reference channel: Pz Ground channel: Fpz	PDPI+ demonstrated lower mid-frontal and mid-cerebellar (Cz & Cbz) theta-band (4–7 Hz) power during postural task compared to PDPI- and HC but no differences between PDPI- & HC. No sig. differences found for beta-band at Cz. This was explained as a result of levodopa. Mid-frontal and mid- cerebellar theta-band power values are associated with distinct measures of postural instability. Decreased mid- cerebellar theta-band power is sig. associated with higher CBS scores. Findings demonstrate a correlation between theta oscillation power and postural control.
[83]	PD (n=8, 5m / 3f) aged 67.50 (4.66) Disease duration = 5.57 (2.22) UPDRS (motor) = 32.25 (7.63) LEDD = 493.75 (324.52) ON medication YA (n=10) aged 26.36 (9.38)	Proactive postural control (Force measurement system (AMTI OR6-6) inside MRI scanner. Task: Raise one leg Two conditions: from the hip with straight knee with or without support.) X 30	fMRI event-related fMRI BOLD signal	Anticipatory postural adjustment inside the MRI scanner was correlated and compatible with gait initiation outside the scanner. Healthy young adults showed brain activation patterns coherent with movement planning. PwPD demonstrated an altered pattern of activation within the motor circuitry. Distribution of functional brain activation for the unsupported condition was sparse in the parietal-frontal areas in the PD group. The lack of any focused brainpattern supports the notion that PD causes the necessity for the involvement of an increasing number of brain areas to complete a balance related motor task.
[82]	PD+FOG (n=12, 8m / 4f) aged 66.7 (5.2) UPDRS-III = 31.25 (6.93) Disease duration = 9.00 (3.72) LEDD = 831.21 (326.92) PD-FOG (n=16, 11m /5f) aged 65.4 (8.1) UPDRS-III = 31.19 (5.69) Disease duration = 7.53 (4.03) LEDD = 654.27 (270.13) OFF medication OA (n=14, 10m 4f) aged 69.8 (8.0)	Dynamic/Reactive/Proactive postural control (Mini- BESTest) WST ML limits of stability MELBA Static postural control/Sensory integration of postural control (Force platform (CAREN-system, MOTEK medical BV, Amsterdam)) ML weight shifts with visual feedback Arms crossed. Diameter 14.0 cm X 10 (5 trials each side) Diameter 29.9 cm X 10 (5 trials each side) FOG-ratio (OPAL inertial sensors APDM Motor imagery medio- lateral weight shifting task X 1 hour. Visual imagery mentally reconstructs images of objects (e.g., a pencil) task X 1 hour.	fMRI (Phillips Achieva ds 3.0 T MRI System) 32-channel head coil BOLD signal ROIs: SMA, anterior/posterior putamen, thalamus, MLR, brainstem vestibular nuclei, - all bilateral. Right anterior cerebellum and cerebellar vermis.	Group differences were found in activations of the right anterior cerebellum, cerebellar vermis and left mesencephalic locomotor region (MLR) during mental imagery of weight shifting. Freezers showed lower relative activations than non- freezers in these ROI's. Non-freezers also showed higher activations than healthy controls in the vermis. No sig. differences in activity were found between freezers and healthy controls. This suggests a compensatory mechanism is recruited by non-freezers, where freezers fail to use these regions effectively during balance control, causing balance issues.

Table 2 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cortical activity measurement device and ROI's	Main findings and interpretation
[87]	PD (n=16, m9 / f7) aged 62.4 (7.5) Disease duration: 4.8 (1.4) PIGD subscore = 3.4 (1.0) OFF medication OA (n=16, m8 / f8) aged 61.9 (8.0)	Static postural control/ Cognitive load on postural control (Force plate (9360AA6, Kistler)) Conditions: Single posture (Standing with feet together) DT with posture-focus instruction DT with motor task focus instruction (motor part: preventing two rings from touching) Trial duration: 6 X 30s UPDRS III	EEG NuAmps (1000 Hz) (NeuroScan Inc.) 30 channels: frontal cortex (Fp1/2, Fz, Fr/4, F7.8, FT7/8, FCz, FC3/4) Central cortex (Cz, C3/4) Parietal cortex (CPz, CP3/4, Pz, P3/4) Temporal cortex (T3/4, T5/6, TP7/8) Occipital cortex) Oz, and O1/2) Ground channel: ahead of Fz	Early-PD patients exhibited higher mean inter-regional connectivity strength in both single and DT postural conditions than OA. Both PD and OA groups had sig. increases in postural sway during DT in comparison to ST. PD patients tended to increase their reliance on cortical control for the concurrent postural task and OA reduced cortical engagement. Findings are interpreted as differential task prioritization effects on DT performance and cortical re- organization between early PD and OA. Overall, findings suggests that appropriate attentional resource allocation and task prioritisation can help minimise exaggerated dual-task interference due to basal ganglia dysfunction in PD.
[85]	Early-PD (n=9, 6m / 3f) age range (56- 76) OA (n=29, 17m / 12f) age range (50- 73)	Sensory integration of postural control/ Reactive postural control (Force plate – 4 sensors under each foot (Vitrualis, Clapiers, France)) VR (BioVRSea) PRE-Phase: NS (Mountain view) (120 s) NS (Sea view – no perturbations) (40 s) Experimental phase: NS (Sea view – 25% wave amplitude) (40s) NS (Sea view – 50% wave amplitude) (40s) NS (Sea view – 75% wave amplitude) (40s) POST-Phase: NS (Sea view – no perturbations (40 s)	EEG EegoTM (4096 Hz down sampled to 1024 Hz) PSD 64 channels: temporal lobe (T7, T8, C6, FT7, FC5, FT8), frontal lobe (AF3), occipital lobe (PO6), parietal lobe (P4),	Sway was found to be greater in OA than early PD. Authors suggest this may be due to reduced mobility in PwPD. When analysing differences between PRE and POST phase, in the theta bands, sig. differences between early-PD and OA were found in the temporal (T7, T8, C6, FT7), frontal (AF3, M1), and occipital lobe (PO6). In the alpha band, sig. differences in PSD included electrodes in the temporal lobe (FC5, T7, T8, FT8), parietal (P4) and occipital lobe (P06). Activity in healthy subjects is greater than in PwPD in both the PRE and POST phases in alpha band. Theta waves: PSD decreases in OA in the parietal areas. For the frontal lobe, theta band showed an increased PSD between PRE and POST phase for the OA group. Alpha waves: Absolute PSD was lower in the PD group compared with OA. This may indicate a higher demand to cope with the balance task in PwPD.

Table 2 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cortical activity measurement device and ROI's	Main findings and interpretation
[84]	PD (n=17) aged 67.6 (1.1) Disease duration = 5.8 (0.9) LEDD = 664.0 (92.5) OA (n=17) aged 68.1 (1.3)	Dynamic/Reactive/Proactive postural control/Sensory integration of postural control Customised balance simulator) - Resting state - Static balancing - Dynamic balancing - Proprioceptive task: simulator moved by experimenter. All tasks performed with EC	fMRI fMRI (3 Tesla scanner) (Philips Achieva 3.0 T R3.2; Philips Medical Systems, The Netherlands) 8-channel head coil BOLD T2* weighted images Repetition time: 2000 ms Echo time: 30 ms 36 axial slices of 3.97 mm thickness	The following areas involve connections that were indicated as being important in predicting discriminating between PD and OA group, during dynamic balance task: - midbrain to the left nucleus accumbens - left anterior cingulate cortex to left caudate - right insular cortex to left insular cortex - left dorsal premotor area to left pre- supplementary motor area - right middle temporal cortex to right lateral orbitofrontal cortex PwPD had increased connectivity associated with motor control and parietal areas during the dynamic/reactive balance task, compared to OA. Moreover, they also had decreased connectivity from brainstem to subcortical areas, suggesting that dynamic balance control in PwPD relies relatively more on cortical motor areas. These results are consistent with the 'posture second strategy' observed in PwPD, whereby a cognitive task results in disproportionate decreases in postural control. PwPD were found to have increased connectivity from the SMA and premotor areas to the pre-SMA, and from the superior parietal cortex to the precuneus. PwPD had an abnormal increase in connectivity from the dorsal premotor area to the pre-SMA compared with OA. Connectivity from the dorsal prefrontal and anterior cingulate cortices to the caudate nucleus were reduced in PwPD, compared to OA, during the dynamic task. PwPD were found to have reduced connectivity from the midbrain to thalamus, cerebellum, nucleus accumbens, and amygdala, during dynamic balancing. These findings support previous work which found that the tendency to shift execution of automatic movements to subcortical areas is less clear in PwPD in relation to OA. The static balance task did not produce significant and effective connectivity networks. This may be due to protocol limitations. Dynamic balancing was more reliant on motor cortical control in PwPD, compared to OA.

Table 2 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cortical activity measurement device and ROI's	Main findings and interpretation
[86]	PD (n=12, 11m / 1f) aged 67.5 Disease duration = 6.42 UPDRS (motor) = 23.0 OFF medication OA (n=12, 6m / 6f) aged 62.9	Reactive postural control (Force plate QNX (QNX Software Systems)) NS X 3 1 trial of large perturbations 1 trial of small perturbations 1 trial of unpredictable mixed perturbations 30 perturbations per trial with visual warnings 2s prior to perturbation.	EEG ANT EEG (512 Hz) Sintered AG-AgCL electrodes 32-channels: Central (C3, C4, CP1, CP2, CP5, CP6, CZ) Frontal-central: (FC1, FC2, FC5, FC6) Frontal parietal: (FP1, FP2, FPZ) Temporal-Parietal: (T7, T8) Frontal: (F3, F7, F4, F8, FZ) Parietal-occipital: P3, P7, P4, P8, POZ, PZ, O1, O2, OZ Reference electrode: Laplacian Ground channel: CZ	Cortical activity in the primary and supplementary motor areas were not associated with postural adjustments madein relation to perturbation difficulty in OA. This suggest that subcortical areas may be responsible for this action in OA. However, in PWPD, increased beta ERD was linked with decreased adaptability. This suggests cortical activity in anticipation of perturbations may have an effect on postural control in PD and suggests a failed compensatory mechanism. This increase in cortical activity may be cause or a consequence of poor postural response performance.
[81]	PD (n=15, 10m / 4f) aged 67.99 (10.83) Disease duration = 6.04 (5.16) H&Y = 1.87 (0.97) UPDRS III = 14.27 (8.21) OA (n=22, 7m / 15f) aged 63.38 (8.53)	Dynamic postural control (TUG) Pre-test – 40s quiet sitting Interval 1: rising from chair and walking 3 m and turning. Interval 2: walking back to chair and sitting. Interval 3: combined interval 1 and 2.	fNIRS (8.36 Hz) fNIRS (NIRSport2 16–16 fNIRS System (NIRx Medizintechnik GmbH, Berlin, Germany) 29-channel array of optodes (10 light sources and 9 detectors): PFC Inter-optode distance: 3 cm Topographic map: (MNI brain template (International Consortium for Brain Mapping. (ICBM-152) – standard template BrainNet view toolbox: NIRS-SPM converted fNIRS channels to MNI coordinates.	For the PD group, there were hemodynamic differences over the front, left, right and total frontal cortices, in different periods of the TUG. Moreover, activity levels (oxygenated hemoglobin) sig. decreased in interval 2 and 3, baseline and interval 1. De- oxygenated levels did not differ in different periods of the TUG. No sig. differences were found between different periods for the OA. There were no sig. differences in Hbo and HbR at baseline, between the PD and OA group, over the four cortices. In interval 1, changes in oxygenated haemoglobin from baseline, over the front, left and total frontal cortices were sig. lower for the PD group, compared to OA. However, for interval 2 the PD group showed sig. higher levels over the left and right frontal cortices. No differences between groups were found in interval 3. HbO2 increase in the left frontal cortex surpassed the right in high-risk fallers. Negative correlations linked PD duration to HbR concentration in their frontal cortices. This suggests that combining frontal cognition and exercise training could effectively boost motor performance in high-risk faller PD patients.

Table 2 (continued)

First author and year of publication	Participant characteristics	Postural control domain (measurement device/tasks)	Cortical activity measurement device and ROI's	Main findings and interpretation
[88]	PD (n=24, 16m / 8f) aged 67.63 (8.74) Disease duration = 8.58 (6.92) UPDRS = 45.00 LEDD = 751.1 (412.0) ON medication OA (n=15, 8m / 7f) aged 66.93 (6.83)	Proactive postural control/Sensory integration of postural control/ (Postuographic force plate (Kiistler 9281B)) NS visual feedback: Voluntary rhythmic swaying (sideways) X3 (Incongruent) Voluntary rhythmic swaying (sideways) X3 (Congruent) Trial duration: 100s	EEG EEG (TMSi) (2048 Hz) Ag/AgCl electrodes 64-channels: Primary motor cortex (M1) Primary sensory cortex (S1) Primary visual cortex (V1) Auditory areas (STS/STG)	PD patients had sig. worse performances on the congruent trials than OA, but this was not reflected in any EEG measures. This may be due to other clinical presentations e.g., rigidity. PD patients did show sig. increased normalized alpha modulation in the V1 region and normalized beta modulation in the M1 compared with OA, during incongruent trials. Yet, no sig. effects were found when analysing normalized mean power. Findings support the notion that beta modulation is a proxy for cortical control of motor performance in PD and shows there is a greater reliance on congruent visual feedback.

AG-AgCL silver-silver chloride, *CBS* Clinical balance scale, *EEG* electroencephalography, *EC* eyes closed, *EMG* electromyography, *EO* eyes open, *fNIRS* functional near-infrared spectroscopy, *Hz* hertz, *H&Y* Hoehn and Yahr, *LEDD* Levodopa equivalent daily dose, *mUPDRS* movement Unified Parkinson's Disease Rating scale, *NS* normal standing, *NS DT* normal standing dual task, *OA* older adults, *PD* Parkinson's disease, *PDPI+* Parkinson's disease with postural impairment, *PDPI-* Parkinson's disease without postural impairment, *PwPD* people with Parkinson's disease, *TUG* Timed up and Go, *UPDRS* Unified Parkinson's Disease Rating scale, *VR* virtual reality

Imaging technique

The majority of studies (n = 4) used fMRI to measure brain activity correlates of postural control using resting-state fMRI (n = 4) [92–95]. The remaining studies included resting-state EEG (n = 1), PET (n = 1) [96] and SPECT scan (n = 2) [90, 97] brain activity correlates of balance.

Outcomes

The BOLD signal was used as a proxy for neural activation in all fMRI studies. However, some studies used resting-state fMRI as a neural correlate of postural control performance outside of the scanner such as task-specific performance on the TUG test [95] or other objective measures of postural control such as inertial sensors [94]. TUG duration was negatively correlated with functional connectivity of the sensory-motor network. Functional connectivity of different brain networks predicted different domains of postural control for PwPD [19].

Both SPECT studies reported 99mTc-TRODAT-1 as an outcome measure reporting dopaminergic uptake [90, 97]. One did not show any significant correlations between dopamine depletion and postural control performance [90]. The other study that used SPECT found that PwPD in H&Y stage 3 had significantly greater depletion of dopamine and worse postural instability (i.e., increased sway displacement) than PwPD in H&Y stage 1 and 2 [97]. The single study that used PET measured C-methyl-4piperdinyl propionate i.e., levels of acetylcholine [96]. This study found an association between postural control performance and the right hemispheric cholinergic network associated related to visual-vestibular integration and self-motion perception. A high anti-cholinergic burden in this region predicted postural control impairment.

Discussion

The objective of this systematic review is to provide a comprehensive overview of the current literature concerning three primary areas of investigation relating to the understanding of the relationship between neuroimaging/cognitive correlates and postural control in PwPD. Firstly, this review encompasses behavioural studies that involve cognitive associations of postural control. Secondly, this review focuses on brain imaging studies that measured brain activity during real-time balance/standing task execution. Additionally, this review considers studies incorporating the use of fMRI, PET, MEG, EEG and SPECT to assess brain function correlates of postural control in PwPD. These include associative relationships between resting-state brain activity and postural control.

Findings indicate a positive relationship between cognitive function and postural control in PwPD, although there was a lack of standardisation regarding protocols across studies. Despite differences in techniques, ROIs, measures and domains of postural control, brain-imaging studies typically found dissimilar neural patterns in PwPD compared to older adults, during tasks intended to challenge balance systems. Increased levels of cortical activity among PwPD during these tasks are generally explained to be compensatory for subcortical damage.

Study protocols

One key finding from this review is that study protocols were highly variable, making it challenging to compare and collate findings. Variations among the behavioural studies include factors such as disease duration/severity, postural control tests/measures, trial conditions, participant testing states ("ON" or "OFF"), explored cognitive domains, cognitive performance, chosen tests for assessment, sample size, and clinical characteristics of PD patients (e.g., PIGD vs TD subtype). These factors all play a crucial role in outcomes. Additionally, the difficulty level of the cognitive aspect of a DT is also a significant consideration. These methodological limitations and discrepancies between studies are all likely causes for some of the heterogeneity in results we see in the current literature.

All fMRI studies measuring brain function correlates lack ecological validity. The conventional requirement for participants to assume a supine and motionless position during measurements significantly hampers the real-time assessment of brain activity in relation to postural control, which is a critical limitation. While imaging techniques such as fMRI offer some advantages in terms of superior spatial resolution in comparison to their mobile counterparts like fNIRS and EEG, the associated compromise becomes apparent in the context of assessing dynamic skills like balance. The need for participants to be stationary undermines the authenticity of the evaluation, emphasising the need for methodologies that capture the intricacies of real-world scenarios. However, as mobile imaging techniques are relatively novel, work using conventional techniques like fMRI is important for the current understanding of the underlying neural mechanisms involved in postural control.

The ability to measure brain activity during standing tasks addresses the ecological validity concerns by allowing for real-time assessment of brain function during tasks requiring postural control. Moreover, the superior temporal resolution, for EEG, allows the detection of cortical control of specific events such

Table 3 Extracted data from brain-imaging studies assessing brain function associations with postural control performance (n=7)

First author and year of publication	Participant characteristics	Balance measurement device/tasks	Brain imaging device, (ROI), and indirect neural correlate	Main findings and interpretation
[90]	PD (n=32, 21m / 11f) aged 60.0 (11.0) UPDRS-III = 28.68 (14.21) OFF medication OA (n=39, 24m / 14f) aged 59.0 (11.0)	Force plate (100 Hz) NS EO X 3 Trial duration: 30s Barefoot	SPECT 99mTc-TRODAT-1 SPECT-CT (Siemens, Symbia T2/dual-head 128 x 128 matrix size 360℃ rotation X 60 20s intervals CT: 2.5 mA, 140 kV, 10-mm slices Imaging 4 hours after dose administration (20 mCi pertechnetate) ROIs = Caudate, Anterior Putamen, Posterior Putamen	Sway, in AP direction and total, were sig. increased in PwPD, compared to OA. No sig. correlations between dopamine depletion and postural control (impairment) was found in PwPD. Future studies are recommended to recruit subject with more severe stages of PD and to assess other neurotransmitters like acetylcholine.
[97]	PD (n=42, m24 / f18) aged 59.0 PD H&Y 1 (n=12, m9 / f3) aged 55.0 PD H&Y 2 (n=15, m8 / f7) aged 63.0 PD H&Y 3 (n=15, m7 / f8) aged 60.0 OFF medication	Force platform (100 Hz) NS EO Barefoot Trial duration: 3x 30 s	SPECT 99mTc-TRODAT-1 (SPECT-CT, TRODAT-1) (Siemens, Symbia T2_/dual-head SPECT/CT system)128 x 128 matrix size 360 degree rotation at 3 degree intervals of 20s 60 views per camera head. 50 mCi pertechnetate. CT 2.5 mA, 140 kV and 10-mm slices Roations: 2.6/minute 7–9 minutes Butterworth prefilter 4 OSEM iterations max 8 OSEM subsets.	Results show that 99mTc-TRODAT-1 uptake differed significantly between people with PD in H&Y stage 1 and 3 as did total displacement of sway. These results suggest that dopamine transporter uptake directly affects balance. These findings indicate a correlation between dopaminergic depletion and postural instability in people with mild-to-moderate PD.
[91]	PD (n=10, 6m / 4f) aged 58.5 (6.0) Disease duration = 6.35 (3.3) LEDD = 598 (379.5) H&Y = 2.2 (0.4) ON medication OA (n=10, m4 / f6) aged 53,2 (7.3)	Mini-BESTest TUG DT (cognitive part: mental subtraction	EEG-MS – resting state EEG (actiCHamp, Brain Vision) (500 Hz) 32 channel Ag/AgCl electrodes 10–20 system Global field power Seated EO Seated EC Trial duration: 5 min	No correlations between EEG-MS and balance of people with PD were observed in both EO and EC conditions. Resting state EEG-MS may not capture neural process related tobalance. However, this study has a relatively small sample size. Further research with larger cohorts is needed to comprehensively understand the relationship between EEG-MS and balance in people with PD.

Table 3 (continued)

First author and year of publication	Participant characteristics	Balance measurement device/tasks	Brain imaging device, (ROI), and indirect neural correlate	Main findings and interpretation
[92]	PD (n=14, 10m /4f) aged 57.9 (8.8) LEDD = 550.2 (148.9) UPDRS-III = 10.8 (6.5) PD + RBD (n=22, 17m / 5f) aged 62.4 (6.4) LEDD = 773.4 (264.3) UPDRS-III = 15.7 (7.8) PD + RBD + PI (n=16, 11m / 5f) aged 61.9 (9.4) LEDD = 854.3 (235.7) UPDRS-III = 21.4 (9.4) OFF & ON medication OA (n=25, 13m / 12f) aged59.8 (8.0)	Force platform (AMTI, Inc., Watertown, MA) Anticipatory postural adjustments 5 M walk X15 Pull-test (item 3.12 UPDRS) ON medication	fMRI (3T Siemens TRIO 32-channel TIM system) Resting-state fMRI 12-channel head coil EC Locomotor network ROIs: bilateral PPN, dentate nucleus, centro-medial thalamic nucleus, internal globus pallidus, substantia nigra, subthalamic nucleus, bilateral, M1 and SMA (SMA proper and pre-SMA) Arousal network ROIs: bilateral PPN, mediodorsal nuclei of the thalamus, anterior cingulate cortices, and medial prefrontal cortices.	The PD group with PI and Rem sleep Behaviour disorders (RBD) and the PD group with RBD had worse MDRS scores than the PD only and the OA group. PwPD with impaired postural control and RBD had decreased functional connectivity between pedunculopontine nucleus and SMA (pre and proper), and the dentate nucleus in the locomotor network which correlated negatively with the duration of anticipatory postural adjustments. Worse performance on the pull test was correlated with decreased PPN connectivity with the SMA. This could be related in part with greater severity of motor symptoms in patients with postural instability. These results suggest that PwPD with a defective pull test are less likely to adapt their cortico- subcortical control during a dynamic balance task. PD patients with postural instability also showed specific dysfunction in the arousal PPN networks including the medial PFC.

Table 3 (continued)

First author and year of publication	Participant characteristics	Balance measurement device/tasks	Brain imaging device, (ROI), and indirect neural correlate	Main findings and interpretation
[94]	Training dataset PD (n=40, 15m / 25f) aged 68.5 (6.5) Disease duration = 6.24 (5.3) UPDRS-III = 39.8 (13.6) LEDD = 871 (1338) H&Y = 2.17 (0.5) OA (n=27, 14m / 13f) aged 70.4 (7.6) Test dataset PD (n=25, 15m / 10f) aged 66.4 (10.1) Disease duration = 3.81 (2.9) UPDRS-III = 39.2 (9.5) LEDD = 486 (415) H&Y = 2.32 (0.7) OA (15, 4 / 11f) aged 68.5 (9.3)	Six Inertial sensors (APDM Wearable Technologies, Portland, OR, USA) OFF medication	fMRI Resting state functional connectivity fMRI (Siemens Trio/Prisma) T1 & T2-weighted images. BOLD (5 mins) ROIs: Cortical areas: Auditory, CinguloOpercular, Dorsal attention, Default, FrontoParietal, Language/Ventral attention, OrbitoAffective, Posterior Multiomodal, Somatomotor, Ventral Multimodal, Visual. Sobcortical areas: Basal Ganglia, Cerebellar.	Resting-state functional connectivity of different brain networks predicted each domain of postural control in PwPD: - Frontoparietal and Ventral Attention rsFC for anticipatory postural adjustments). - Cerebellar- Subcortical and Visual rsFC and Auditory and Cerebellar- Subcortical rsFC for anterior postural responses. - Ventral attention and Ventral Multiomodal rsFC for postural sway. In OA, CinguloOpercular and Somatomoro rsFC predicted anterior postural adjustments. These findings suggest cortical networks predict postural control in PD. Moreover, there are clear distinction and little overlap in brain network connectivity that predict the different domains of postural control. Different cortical networks are used in PwPD, for anterior postural adjustments, compared to OA.
[96]	PD FOG + (n=16, 11m / 5f) aged 67.5 (5.2) Disease duration = 5.2 (5.0) UPDRS III = 35.2 (15.2) LEDD = 685.0 (381.6) PD FOG - (n=59, 45m / 14f) aged 67.5 (5.2) Disease duration = 4.0 (5.5) UPDRS III = 25.5 (12.8) LEDD = 450.0 (417.0) OFF medication	Force platform (Equitest posture test platform) (NeuroCom, International, Inc.) NS EO (SOT 1) NS EC (SOT 2) NS EO - visual surround is sway referenced (SOT 3) NS EO - support surface is sway- referenced (SOT 4) NS EC - support surface is sway-referenced (SOT 5) NS EC - visual surround and support surface is sway referenced (SOT 6)	PET C-methyl-4-piperidinyl propionate PET Voxel-wise mass- univariate correlation ROIs: Brodmann areas (Right BA40/22/2,BA44/6/3/1, BA21/37, BA39, BA47/45, BA46, BA10, BA19/7) (Left BA47, BA20)	PwPD with FOG exhibited greater difficulty maintaining balance when proprioception was challenged, compared to non-freezers. This was independent of disease severity. Voxel-based analysis showed an association between postural control and right hemispheric cholinergic network related to visual- vestibular integration and self-motion perception. High anti-cholinergic burden predicted postural control impairment which was dependent on right hemispheric cortical cholinergic integrity. These findings support the notion that cortical cholinergic system plays a role in supporting postural control after nigrostriatal dopaminergic loss resulting from PD. This corroborates the hypothesis that cognitive/cortical areas compensate for the neurological damage in PwPD and provides further evidence for the relationship between balance and cognition.

Table 3 (continued)

First author and year of publication	Participant characteristics	Balance measurement device/tasks	Brain imaging device, (ROI), and indirect neural correlate	Main findings and interpretation
[95]	PD FOG + (n=20, 9m / 11f) aged 64.5 (6.2) Disease duration = 6.3 (5.1) UPDRS III = 21.0 (5.9) H&Y = 2.4 (0.8) LEDD = 487.5 (133.9) OFF medication PD FOG – (n=21, 8m / 13f) aged 60.2 (7.9) Disease duration = 5.5 (2.2) UPDRS III = 18.9 (3.0) H&Y = 2.0 (0.4) LEDD = 470.2 (136.4) OFF medication OA (n=18, 8m / 10f) aged 61.3 (6.7)	TUG	fMRI resting state fMRI functional connectivity GE Signa HDxt 3.0 T scanner (general Electric Medical Systems, Waukesha, WI, USA) channel head coil 3D T1-weighted images ms EC Meditative state (not thinking) 8 ROIs: Dorsal attention, medial visual, lateral visual, default mode, right/left frontal parietal, sensory motor and the working memory network.	TUG duration was negatively correlated with functional connectivity of the sensory-motor network. This association suggests that interventions or therapies targeting enhanced functional connectivity in the sensory-motor network may contribute to improved dynamic balance in individuals with PD.

CT computer tomography, *DT* dual task, *EEG* electroencephalography, *EC* eyes closed, *EO* eyes open, *Hz* hertz, *H&Y* Hoehn and Yahr, *fMRI* functional magnetic resonance imaging, *LEDD* Levodopa equivalent daily dose, *MDS-UPDRS III* movement disorder society – Unified Parkinson's Disease Rating scale, *NS* normal standing, *NS DT* normal standing dual task, *NS EO* normal standing eyes open, *OA* older adults, *PD* Parkinson's Disease, *PD+FOG* Parkinson's disease with Freezing of Gait, *PD-FOG* Parkinson's disease without Freezing of Gait, *PD+RBD* Parkinson's disease with Rapid Eye Movement sleep behaviour disorder, *PD+RBD* Parkinson's disease without Rapid Eye Movement sleep behaviour disorder, *PwPD* people with Parkinson's disease, *SPECT* single-photon emission computerized tomography, *TUG* Ttimed Up and GO, *TUG DT* Timed Up and Go dual task

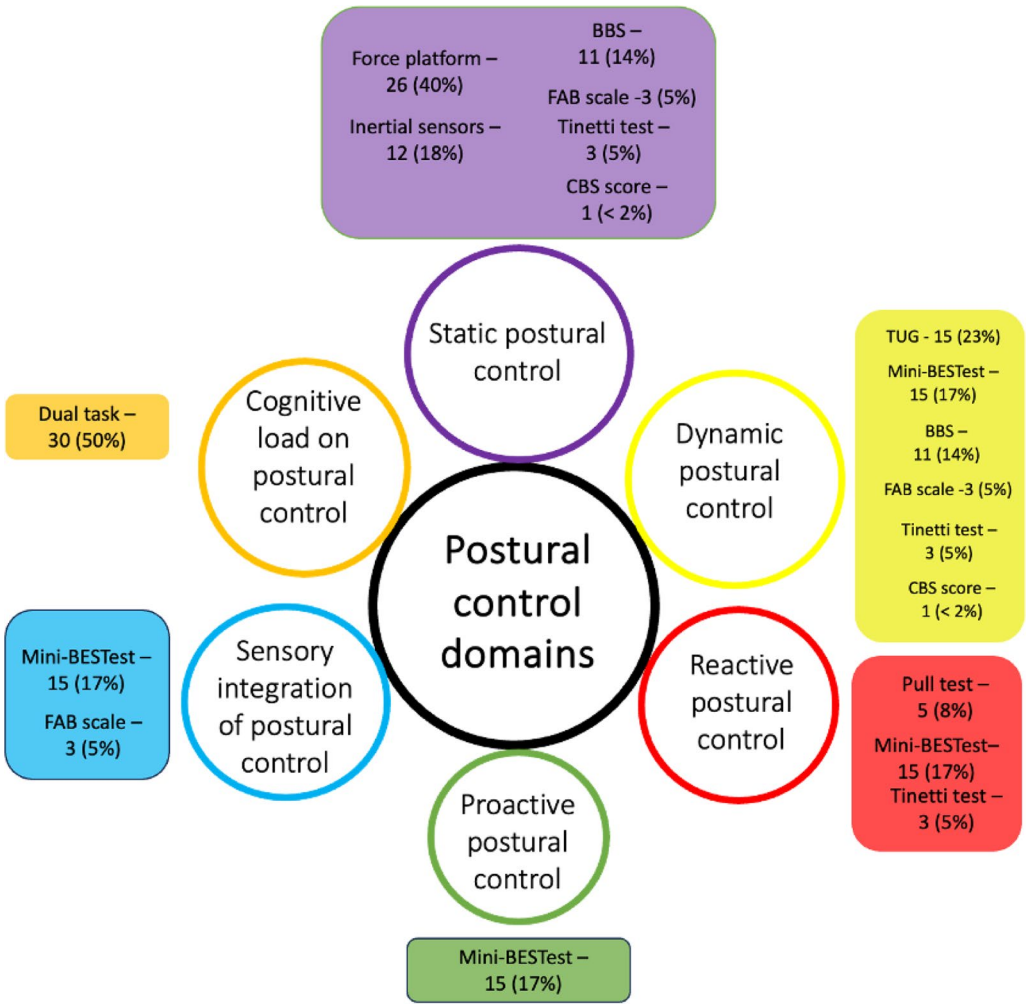

Fig. 3 Six postural control domains that were used to categorise outcome measures. Total (and percentage) of postural control outcome measures/tests used among the cognitive association studies

as perturbations and turns. A limited number of studies were identified as relevant for this review, which reflects the relative recency of this technique. Seven of these studies incorporated the use of EEG and only one employed the use of fNIRS. The fNIRS study had a sampling frequency 8.36 Hz. For the EEG studies, this ranged from 500 to 2048 Hz. The apparent preference for EEG in this research area is likely because of this superior temporal resolution compared to fNIRS.

The evolving landscape in neuroimaging methodologies not only broadens the scope of research possibilities but also underscores the importance of considering the dynamic nature of the skills under investigation. All these variables concerning the protocol are important to consider when testing cortical control and cognitive associations of balance in PD and are crucial to be mindful of when interpreting findings.

Regions of interest

Concerning studies using mobile EEG, there was a lot of variation regarding the use of systems and electrode quantity. There was also very little consensus regarding which electrodes corresponded to what brain regions or how ROIs were reported.

The fNIRS study [81, 98] exclusively measured activity in the PFC, most probably due to hardware constraints and precedence set in gait studies in PwPD. Advancements in hardware now allow for the measurement of cortical activity across multiple cortical regions and have already been used to investigate cortical involvement in gait [99] and motor tasks [100] in PwPD. In future studies aimed at exploring the neural underpinnings underlying balance and how pathologies like PD affect these mechanisms, the use of fNIRS to assess activity across multiple cortical regions could significantly enhance our understanding.

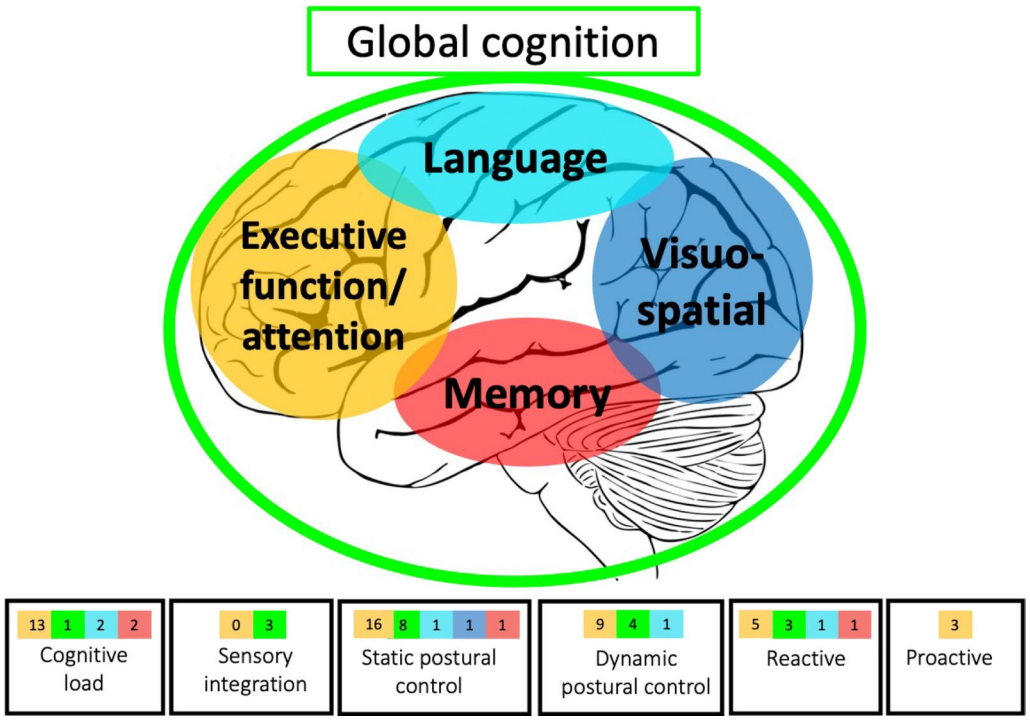

Fig. 4 Visual representation of sig. evidence of associations between cognitive domains and domains of postural control in Table 1. Colours within the postural control domains boxes correspond to each cognitive domain. The numbers represent the total number of study outcomes that found a significant association between postural control and cognitive domain. Some outcomes may correspond to multiple domains. This image is inspired by Fig. 3 in previous work [17] (adapted with permission)

Outcomes

There were many discrepancies in the outcomes being used to measure and report both balance and neural correlates of balance in PwPD within the reviewed studies. Although some studies implemented the use of an objective balance measure, others used more subjective assessments that require the interpretation of the administrator/researcher like the Pull Test or the Mini-BESTest [101, 102]. Although both the Pull Test (i.e., UPDRS item 3.12) and Mini-BESTest are frequently used to assess postural control in PwPD in a clinical setting, for research purposes these types of assessments can introduce variability, reducing accuracy and sensitivity. Using objective, quantitative measures such as a force plate, walkway or accelerometers can improve the accuracy and reliability of balance assessments in research [103, 104]. While these methods may provide valuable insights into postural control performance for clinicians, this diversity in assessments makes it challenging to establish a unified understanding of neural correlates of balance when comparing outcomes.

In parallel, behavioural studies implementing DTs also found divergent outcomes. Three studies showed no significant difference in sway between single task and DTs [35, 45, 105]. Conversely, several studies highlighted significant findings related to the cognitive aspects of DTs such as more errors [61, 74–77], reduced responses [70, 73] and less subtractions [78] than older adults. A subset of studies demonstrated unexpected outcomes, such as a significant decrease in postural sway and excursion during specific cognitive tasks [68, 72, 76, 77, 79, 80]. The lack of significant findings may be indicative of a protocol limitation whereby the cognitive part was not challenging enough and did not demand enough attentional resources to negatively affect sway measures. One explanation for the reduction in sway measures suggests that when the DT becomes too challenging, cognitive load surpasses capacity, leading to a "posture first" strategy. This prioritises postural control resulting in PwPD over constraining and impeding cognitive performance. Evidence for this is shown in one study where dual-tasking reduced postural sway in advanced PD but increased it in older adults [80]

Among the brain imaging studies measuring brain activity during balance-related task performance, one fNIRS study reported both HbO_2 and HbR changes in the PFC during different stages of the TUG in PD but did not change in OA [81].

For the EEG studies, there were more varied outcome measures, including PSD in different bands,

inter-regional and inter-hemispheric connectivity, and ERD. While each of these outcome measures provides its own unique and valuable insight, the lack of consistency poses challenges for generalisation between studies. These methodological differences add to the already difficult challenge of drawing overarching conclusions.

Although the static brain imaging studies lacked ecological validity, several fMRI studies demonstrated increased connectivity that involved cortical areas and decreased subcortical connectivity. Specifically, PwPD showed an altered pattern of activation within the motor circuitry and limited focused brain patterns in the parietal-frontal areas during an anticipatory postural adjustment task in a supine position, compared to healthy younger adults [83].

Resting-state fMRI was used to assess associations between neural activity inside the MRI-scanner, with postural control performance outside the scanner in some studies [94]. For example, decreased resting-state functional connectivity between the pedunculopontine nucleus (PPN) and the pre-SMA and SMA proper was correlated with worse performance on the pull test in PwPD with postural impairment and REM sleep behaviour disorder [92]. Additionally, one study [84] found that PwPD had increased connectivity in frontal and parietal areas associated with motor control during a dynamic balance task in an fMRI scanner, compared to OA. PwPD were also found to have decreased connectivity from the brainstem to subcortical areas, compared to OA. These findings highlight the complex interplay between neural activity and postural control and demonstrate that PD pathology alters these underlying mechanisms.

Both brain imaging outcome measures may provide valuable information on the brain function associations of postural control in PD. However, attempting to directly compare these measures may lead to misinterpretations or oversimplifications. The variations within behavioural, mobile brain-imaging and imaging studies, warrant a cautious interpretation and emphasise the need for future research to address these limitations.

Interpretation of outcomes

Despite heterogeneity in outcome measures and protocols, recurring findings do emerge. Namely, PwPD consistently require increased levels of cortical activity compared to healthy older adults during standing/postural control tasks.

Moreover, cognitive dysfunction is significantly linked to postural control impairments in PwPD. These findings support the hypothesis that degeneration of subcortical dopaminergic neurons in PwPD, which are responsible for automatic postural control, results in a compensatory shift towards cortical/cognitive resources to rely on to maintain appropriate postural control. This would in part explain why cognitive dysfunction is correlated with balance disturbances and increased cortical activity has been found in PwPD.

Until recently, the notion that cognitive function and postural control had shared neural underpinnings was not widely accepted. Motor control and cognitive function were thought to be governed by distinct neural networks. Motor function does indeed rely heavily on subcortical structures such as the basal ganglia and cerebellum [106]. Conversely, higher-order cognitive functions like attention, planning and decision-making are predominantly associated with cortical regions, particularly in the frontal lobes [107, 108].

Yet, experimental methodologies and neuroimaging studies, like those included in this review, have demonstrated that there is a complex interplay between motor and cognitive function. Subcortical structures have increasingly been implicated in cognitive processes. Structural and functional abnormalities in regions like the basal ganglia and thalamus have been linked to various psychiatric disorders including schizophrenia, depression, and autism spectrum disorders [109], demonstrating subcortical involvement in cognitive, social, and affective functions. Cerebellar damage has also been linked to cognitive and emotional impairments [110].

Moreover, individuals with subcortical lesions in the putamen and thalamus show signs of cognitive dysfunctions like aphasia and unilateral spatial neglect [111]. Considering the importance of visuospatial function in balance, as suggested by many study findings included in this review, the bidirectional relationship between cognition and motor control becomes even more apparent. In parallel, behavioural studies have shed light on the importance and involvement of higher-order cognitive capacities, and thus indirectly, cortical regions, in motor function. In conjunction, imaging studies have reinforced this notion by highlighting cortical activity during postural control tasks.

For example, both resting-state and functional connectivity during task performance in some fMRI studies demonstrate a decrease in modularity of cortical regions in PwPD, compared to OA i.e., excessive global integration with less specialisation of brain regions. This suggests a restructuring of brain patterns in individuals with PD. Postural stability requires the integration of sensory, motor, visual, vestibular and cognitive circuits [6]. Damage to any one of these systems, e.g., through pathology like PD, likely leads to changes in the neural circuits involved in maintaining balance. Failure to reorganise these mechanisms effectively may lead to maladaptive

behaviours such as freezing [19] and postural impairment [112].

To date, research on cortical control of, and cognitive associations with, motor function in PwPD has predominantly focused on gait. As a result, supporting evidence for cortical involvement in gait is more extensive and advanced than for balance. However, the limited number of outcomes from the mobile brain-imaging studies do generally demonstrate an increased level of cortical activity during postural control tasks when comparing PD patients and older adults. The only study to use fNIRS showed increased PFC activity over the front, left and total frontal cortices in the PD group was significantly lower than in the control group at baseline, while significantly higher than the control group during the sit-to-stand section of the TUG over the left and right frontal cortices [81].

For the EEG studies, PSD, CNV and increased beta ERD were associated with increased sway. For PwPD, functional inter-regional connectivity was higher during both single and DT conditions compared to OA, whilst OA showed a decrease in connectivity from single to DT. This potentially reflects the neural proficiency in enabling the prioritisation of cognitive processes which is not present in PwPD, even in the early stages of the disease.

The effect of dopaminergic medication on balance performance in people with PD remains unclear. Certain research [113] indicates enhancements in specific facets of postural control, whereas others yield no significant effects [114]. Furthermore, there is also evidence suggesting that dopaminergic medication increases postural sway in individuals with PD [115]. This study's findings demonstrate how dynamic balance performance and cortical activity can change in response to dopaminergic drugs. The cholinergic system also plays a significant part in balance performance. A PET study found an association between postural control and the right hemispheric cholinergic network in freezers and non-freezers [96]. This emphasises the significance of the role of attention and the cholinergic system in maintaining balance for PwPD and corroborates the hypothesis that cognitive systems and cortical areas become increasingly important following nigrostriatal degeneration.

Clinical and future research recommendation

More work is required to enhance the understanding of underlying neural mechanisms of postural control in PD, as it remains poorly understood. Future studies must take several variables into account when designing research

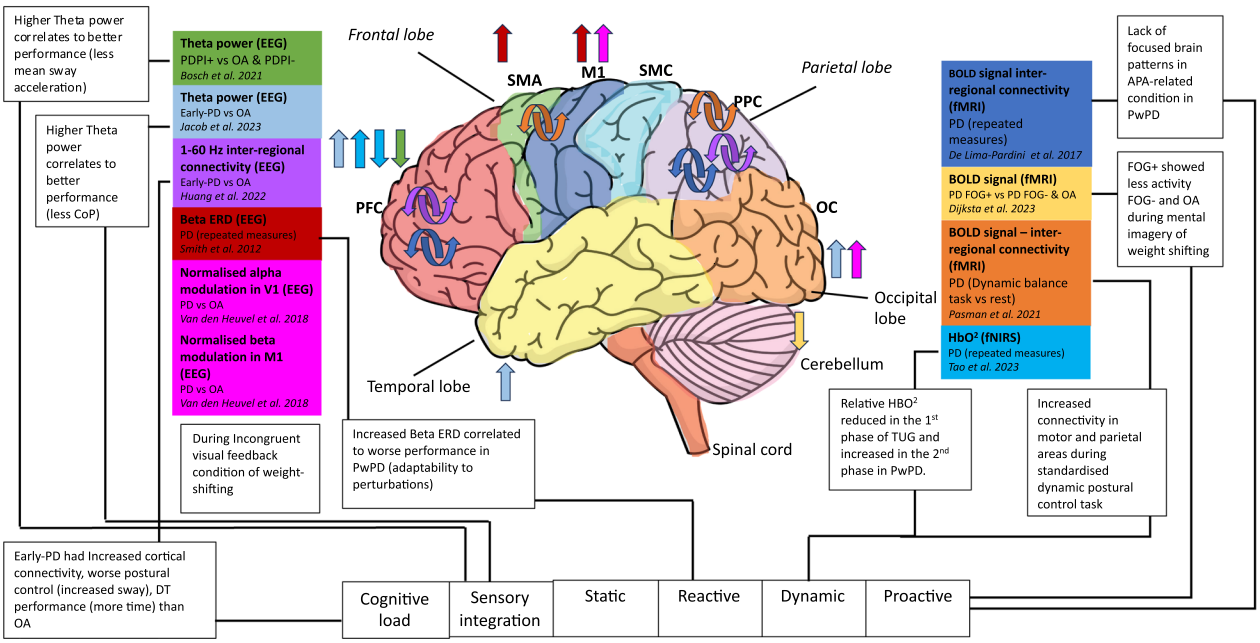

Fig. 5 Overview of evidence of neural activity during postural control tasks in PwPD. (Arrows represent study outcome—upwards arrow indicates increased activity/double curved arrows indicate inter-regional connectivity). This Image is inspired by Fig. 3 in previous work [1] (adapted with permission). *BOLD* blood-oxygen-level-dependant, *CoP* centre of pressure, *EEG* electroencephalography, *ERD* event-related desynchronisation, *fNIRS* functional near-infrared spectroscopy, *FOG+* freezers, *FOG* non-freezers, *HbO²* oxygenated haemoglobin, *M1* motor cortex, *OA* older adults, *OC* occipital cortex, *PDPI+* Parkinson's disease with postural control impairment, *PDPI-* Parkinson's disease without postural control impairment, *PFC* prefrontal cortex, *PPC* posterior parietal cortex, *PwPD* people with Parkinson's disease, *SMA* supplementary motor area, *SMC* supplementary motor cortex

protocols, especially for brain-imaging studies; researchers should attempt to design and utilise standardised procedures and protocols. The single fNIRS study included in this review only measured PFC activity, likely due to technological/hardware limitations. As postural control involves the incorporation of several sensory and motor systems, future projects should aim to assess activity in multiple cortical areas. Moreover, few studies combined cognitive behavioural measures and brain imaging. An integrated approach between cognitive behavioural measures and mobile neuroimaging techniques like fNIRS and mobile EEG will yield deeper insights into the complex relationship between cognitive function and postural control in PD. Regarding literature reviews on this topic, one limitation of this review is that it did not discriminate between subtypes (i.e., PIGD and tremor dominant). As PD is highly heterogenous, future work should aim to focus on classifying subtypes. Moreover, it is important to acknowledge that although cognitive domains have been characterised as independent features for purpose of this review (Fig. 4), there is also a certain level of overlap between these different domains. This does have the potential to complicate results and makes interpretation more challenging (Fig. 5).

In addition, this review included the TUG as an assessment of dynamic postural control. This domain of postural control does overlap significantly with gait. To be able to draw more robust conclusions about neuroimaging/cognitive control correlates of postural control, future projects may focus on specific relationships between different domains.

Increasing the understanding of neural correlates of balance holds the potential to drive the development of more targeted interventions, improving the quality of life for people with PD and reducing the financial burden on healthcare services linked to fall-related injuries.

Conclusions

This review synthesises evidence from 79 studies, indicating that individuals with PD have dissimilar neural activation patterns compared to healthy controls and may rely more on cortical activity to maintain postural control due to subcortical degeneration. Key findings highlight a consistent association between poorer cognitive function and impaired postural control, with increased cortical activity potentially compensating for subcortical deficits. However, evidence is limited, and further work is needed to support this notion. An effort should be made in these studies to (1) standardise measures of postural control, (2) use cortical-imaging devices to assess cortical activity during standing/balance task performance in PwPD, and (3) focus on relationships between neuroimaging/cognitive correlates with specific domains of postural control. The scarcity of cortical brain-imaging studies investigating cortical control of balance in PD and the lack of consensus on ROI's and data analysis procedures are areas that future research projects should consider.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12984-024-01539-y.

Additional file 1

Additional file 2

Author contributions

PT wrote the main manuscript, carried out the searches, screened articles and prepared Figs. 1, 2, 3, 4, 5. LG was the 2nd reviewer and carried out the initial abstract screening of all articles on Rayyan. LG, RV, TW, ET, JO, SS and RM provided feedback, suggestions and revisions on multiple drafts. RM made substantial contributions to the conception and design.

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Availability of data and materials

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Competing interests

The authors declare no competing interests.

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