Flexed Truncal Posture in Parkinson Disease: Measurement Reliability and Relationship With Physical and Cognitive Impairments, Mobility, and Balance

Aimi L. Forsyth, BAppSc (Phty), Serene S. Paul, PhD, BAppSc (Phty)(Hons), Natalie E. Allen, PhD, BAppSc (Phty) (Hons), Catherine Sherrington, PhD, MPH, BAppSc (Phty), Victor S. C. Fung, PhD, MBBS (Hons), and Colleen G. Canning, PhD, MA, BPhty

Background and Purpose: Flexed truncal posture is common in people with Parkinson disease (PD); however, little is known about the mechanisms responsible or its effect on physical performance. This cross-sectional study aimed to establish the reliability of a truncal posture measurement and explore relationships between PD impairments and truncal posture, as well as truncal posture and balance and mobility.

Methods: A total of 82 people with PD participated. Truncal posture was measured in standing as the distance between vertebra C7 and a wall. Univariate and multivariate regression analyses were performed with truncal posture and impairments, including global axial symptoms, tremor, bradykinesia, rigidity, freezing of gait (FOG), reactive stepping and executive function, as well as truncal posture with balance and mobility measures.

Results: The truncal posture measure had excellent test-retest reliability ($ICC_{3,1}$ 0.79, 95% CI 0.60-0.89, $P < 0.001$). Global axial symptoms had the strongest association with truncal posture (adjusted $R^2 = 0.08$, $P = 0.01$), although the majority of the variance remains unexplained. Post hoc analysis revealed that several impairments were associated with truncal posture only in those who did not report FOG. Flexed truncal posture was associated with poorer performance of most balance and mobility tasks after adjustment for age, gender, disease severity, and duration (adjusted $R^2 = 0.24$-0.33, $P < 0.001$-0.03).

Discussion and Conclusions: The C7 to wall measurement is highly reliable in people with PD. Global axial symptoms were independently associated with truncal posture. Greater flexed truncal posture was associated with poorer balance and mobility. Further studies are required to elucidate the mechanisms responsible for flexed truncal posture and the impact on activity.

Video Abstract available for more insights from the authors (see Video, Supplemental Digital Content 1, http://links.lww.com/JNPT/A164).

Key words: *axial symptoms, bradykinesia, human movement system, mobility limitation, postural balance*

(*JNPT* 2017;41: 107–113)

Faculty of Health Sciences, The University of Sydney, Sydney, Australia (A.L.F., S.S.P., N.E.A., C.G.C.); Musculoskeletal Division, The George Institute for Global Health, Sydney Medical School, The University of Sydney, Sydney, Australia (S.S.P., C.S.); Movement Disorders Unit, Department of Neurology, Westmead Hospital, Sydney, Australia (V.S.C.F.); Sydney Medical School, The University of Sydney, Sydney, Australia (V.S.C.F.); and Department of Physical Therapy, University of Utah, Salt Lake City, United States of America (S.S.P.).

Catherine Sherrington's research has been funded by the Australian National Health and Medical Research Council, Parkinson's NSW, Consortium national de formation en santé (Canada), the University of Sydney, Arthritis NSW, Canadian Institute of Health Research, NSW Ministry for Health and her salary is funded by an Australian National Health and Medical Research Council Fellowship and the George Institute for Global Health. She has received travel expenses from the Australian Physiotherapy Association and the Fragility Fracture Network.

Victor Fung is on advisory boards and/or has received travel grants from Abbott, Allergan, Boehringer-Ingelheim, Hospira, Ipsen, Lundbeck, Novartis, Parkinson's KinetiGraph, Solvay and UCB.

Colleen Canning has received travel expenses and honoraria for lectures and educational activities from the Movement Disorders Society and the Postgraduate Allied Health Institute, Singapore.

This research was supported by a seeding grant from the Physiotherapy Research Foundation of Australia (ID: S10-012).

Aimi Forsyth, Serene Paul, and Natalie Allen declare no conflicts of interest.

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.jnpt.org).

Correspondence: Colleen G. Canning, PhD, MA, BPhty, Faculty of Health Sciences, The University of Sydney, PO Box 170, Lidcombe, NSW 1825, Australia (colleen.canning@sydney.edu.au).

ISSN: 1557-0576/17/4102-0107
DOI: 10.1097/NPT.0000000000000171

INTRODUCTION

Flexed truncal posture affects up to 73% of people with Parkinson disease (PD),[1] which is significantly higher than the 20% to 40% prevalence of flexed truncal posture reported in the general older population.[2] In both the general older population and the PD population, flexed truncal posture increases with age.[2] Vertebral fractures, degenerative disc disease, back extensor muscle weakness, and habitual posture are thought to play a role in the development of flexed truncal posture.[2] However, the association between disease severity[3] and increased flexed truncal posture in people with PD suggests that additional mechanisms may play a role in this population. Axial rigidity is a possible cause with one small study reporting an improvement in truncal posture with a reduction in neck rigidity following the administration of apomorphine.[4] In contrast, another study did not find a relationship between axial rigidity and the posture item of the Unified Parkinson Disease Rating Scale (UPDRS).[5] Furthermore, the association of the other cardinal impairments of PD—tremor, bradykinesia, and postural instability—with flexed truncal posture has not been definitively established.

It has also been suggested that flexed truncal posture could be a compensation for some of the motor impairments of PD, particularly for bradykinesia and postural instability.[6,7] Adopting a flexed truncal posture might facilitate step initiation in people with freezing of gait (FOG).[6] Flexed truncal posture may also be a compensatory mechanism for reduced balance by protecting against backward displacement of the center of mass,[7] although evidence for this is inconsistent.[8]

It is likely that flexed truncal posture in people with PD will adversely affect the performance of everyday activities. Flexed truncal posture in both people with PD and the general older population is associated with reduced standing balance,[9,10] altered gait biomechanics,[9,10] and an increased risk of falls.[1,11] Difficulties performing these activities are likely to be due to reductions in the functional limits of stability when standing with a flexed truncal posture.[9] Furthermore, greater flexed truncal posture in the general older population is associated with other negative consequences, including difficulty performing everyday activities,[12] depression,[13] and increased mortality.[14]

Given the adverse consequences associated with greater flexed truncal posture in the general population and the high prevalence of flexed truncal posture in PD, it is important to improve an understanding of both the relationship between PD impairments and truncal posture and the extent to which flexed truncal posture is associated with activity limitations in this population. To explore the relationships outlined earlier, a reliable measure of truncal posture is required. The distance between the seventh cervical vertebra (C7) and a wall is a simple, clinically feasible method of assessing truncal posture and removes the potentially confounding influence of head position. This measure is known to be reliable in the healthy older population,[12] but its reliability in the PD population has not been tested.

The aims of this study were therefore to (i) establish the reliability of the C7 to wall measure of truncal posture in people with PD; (ii) determine whether common PD impairments are associated with flexed truncal posture, after adjusting for other factors known to influence posture; and (iii) investigate the relationship between truncal posture and balance and mobility task performance, after adjusting for other factors known to influence these activities. We hypothesized that PD impairments, particularly axial symptoms (ie, axial rigidity, gait disorder, and postural instability),[15] would be associated with truncal posture, and that balance and mobility tasks would be negatively affected, with tasks not requiring a change of base of support being more affected than tasks requiring a change of base of support.

METHODS

The data used for this cross-sectional study were collected as part of a larger study.[16,17] Methods relevant to this study are described later.

Participants

Community-dwelling participants with idiopathic PD were recruited via advertisement in a PD association newsletter and from our university database of people with PD. Participants 40 years or older who were independently mobile with or without a walking aid were eligible to participate. Participants were excluded if they suffered unstable neurological, orthopedic, or cardiovascular conditions limiting their ability to undergo the assessment procedures or which would affect the interpretation of the results of the previously reported studies[16,17]—those with symptomatic or unhealed vertebral fractures and known disc disease were excluded; participants with osteoporosis were not necessarily excluded. Participants with significant cognitive impairment (Mini-Mental State Examination (MMSE) < 24) were also excluded. Testing was conducted when participants' PD medication was working optimally, typically 1 hour after ingesting the last dose. A battery of tests was administered in a standardized order by 1 of 2 assessors in a university facility. All participants gave written informed consent before testing and approval was gained from the relevant Human Research Ethics Committee.

Outcomes

For the truncal posture measurement, participants were instructed to stand with their usual truncal posture while keeping their buttocks and back against a wall. Feet were placed shoulder width apart, typically 2 to 5 cm in front of the wall. The horizontal distance from C7 to the wall was measured with a tape measure to the nearest 0.1 cm[12] (Figure 1). Test-retest reliability was calculated from a subset of 31 participants who underwent a second test 1 week after the baseline measurement.

A number of PD disease-specific measures were taken including the total motor examination score of the Movement Disorders Society–sponsored UPDRS (MDS-UPDRS),[18] Hoehn and Yahr stage,[19] and time since PD diagnosis. Measures of PD impairment were taken from the motor section of the MDS-UPDRS. The tremor score was the sum of scores for postural tremor (item 3.15), kinetic tremor (item 3.16) plus resting tremor (item 3.17), and tremor constancy (item 3.18) across all body parts. The bradykinesia score was

Figure 1. Truncal posture measurement. Participants stand with their back and buttocks against the wall. The distance between the wall and the seventh cervical vertebra is measured with a tape measure.

the sum of scores for finger tapping (item 3.4), hand movements (item 3.5), supination/pronation (item 3.6), toe tapping (item 3.7), and leg agility (item 3.8). The rigidity score (item 3.3) was the sum of scores for all 4 limbs and the neck. An axial subscore of the motor examination of the MDS-UPDRS was calculated in line with previous work to reflect global axial motor symptoms.[20] The axial subscore is the sum of neck and leg rigidity (item 3.3), leg agility (item 3.8), arising from a chair (item 3.9), gait (item 3.10), postural stability (item 3.12), posture (item 3.13), and lower limb resting tremor (item 3.7). The New Freezing of Gait Questionnaire (NFOGQ)[21] was used to quantify FOG. Measures of reactive stepping and postural sway were taken to reflect postural instability. The Frontal Assessment Battery (FAB)[22] was used to quantify executive function. A short description of these tests and reported reliability are available in the online Appendix (see Supplemental Digital Content 2: Appendix, http://links.lww.com/JNPT/A165).

Balance and mobility tasks were categorized into 2 groups: tasks not requiring a change in the base of support (eg, reaching) and tasks requiring a change in the base of support (eg, walking) (see the online Appendix). Tasks without a change in base of support included functional reach and lateral reach, single leg stand, and coordinated stability. Tasks requiring a change in base of support included the Timed Up and Go (TUG) test performed as a single task and with a dual cognitive task, walking speed, the 5-repetition sit-to-stand (STS), and choice stepping reaction time.

Statistical Analyses

To address aim 1, intraclass correlation coefficients ($ICC_{3,1}$) were used to determine test-retest reliability of the truncal posture measure. Excellent reliability was defined as ICC > 0.75, good 0.60 to 0.74, fair 0.40 to 0.59, and poor less than 0.40.[23]

To address aim 2, relationships between PD impairments (predictor variables) and truncal posture (outcome) were examined with univariate and multivariate linear regression models. The multivariate model included age, axial subscore, tremor, rigidity, bradykinesia, FOG, reactive stepping, and the FAB as predictors; 8 predictors were chosen for entry into the multivariate model to ensure that there were at least 10 outcome cases per predictor variable. A post hoc analysis was performed to explore the association between the NFOGQ score and the axial subscore. An interaction term was created by multiplying the axial subscore with the NFOGQ score and these 3 terms were entered into a multivariate regression model. Separate analyses for participants with and without FOG were then performed; FOG status was categorized on the basis of an answer of yes or no to Question 1 of the NFOGQ (ie, "did you experience freezing episodes in the past month?").

To address aim 3, relationships between truncal posture (as a predictor variable) and balance and mobility task performance (outcomes) were examined with univariate linear regression. For each of the outcomes where truncal posture was a significant predictor ($P < 0.05$) in the univariate analysis, a stepwise multivariate linear regression model incorporating age, gender, disease severity, and duration as covariates was developed. For missing data arising from participants who were physically unable to perform the 5-repetition STS (1% of participants) or choice stepping reaction time (2% of participants), a value of the sample mean minus 3 standard deviations (SDs) was assigned. Data were analyzed using SPSS version 22 (IBM Corp, Armonk, New York).

RESULTS

One hundred thirty-eight volunteers with PD were screened for inclusion in the study. Thirty-six were ineligible, and 20 declined to participate. The remaining 82 people (55 male, 27 female) who participated had a mean time of 7.5 years since PD diagnosis. Participants had slight rigidity and tremor, slight to mild bradykinesia,[18] (Table 1) and mildly abnormal truncal posture (mean distance C7 to wall = 9.1 cm, SD = 2.6, range = 4.2–16.4 cm, compared with the general older population values of a mean distance of 5.2 cm, range = 0-15 cm).[12] They took a mean levodopa equivalent dose of 732 mg/day (SD 430 mg/day). In the subset of 31 participants, test-retest reliability of the C7 to wall truncal posture measurement was excellent ($ICC_{3,1} = 0.79$, 95% CI: 0.60-0.89, $P < 0.001$). The standard error of measurement was 1.1 cm (12.0%).

Association of Impairments With Truncal Posture

Univariate linear regression showed that increased age, higher MDS-UPDRS motor score, and a higher axial subscore were significantly associated with flexed truncal posture (Table 1). Cognition as measured by the FAB, the PD subscores of tremor, rigidity, bradykinesia, and reactive stepping did not demonstrate a significant association with flexed truncal posture. The multivariate linear regression model, including age, axial subscore, tremor, rigidity, bradykinesia, FOG, reactive stepping, and the FAB, explained 14% of the variability in flexed truncal posture (adjusted $R^2 = 0.14$, $P = 0.01$) (Table 2). Higher axial subscores ($\beta = 0.69$, $P = 0.001$) and lower FOG scores ($\beta = -0.26$, $P = 0.04$) were the only variables within the multivariate model that were independently associated with flexed truncal posture ($P < 0.05$).

Further post hoc analyses were performed to assist in understanding the unexpected finding of a nonsignificant relationship between FOG and posture in the univariate model, but a significant, negative relationship in the multivariate analysis. The post hoc analyses suggested that there might be an interaction between the NFOGQ and the axial subscore (interaction variable $P = 0.07$). We therefore investigated separate univariate models for participants with and without FOG (Table 3). Age, higher MDS-UPDRS, higher axial subscore, and male gender were significantly associated with flexed truncal posture in participants without FOG. In participants with FOG, none of the predictors we examined were significantly associated with flexed truncal posture.

Association of Truncal Posture With Gait and Balance Activities

Univariate analyses revealed that flexed truncal posture had a small and significant association with all the tasks not requiring a change in base of support: functional reach, lateral reach, single leg stand, and coordinated stability scores (adjusted $R^2 = 0.05\text{-}0.14$, $P = 0.001\text{-}0.02$), and with some of

Table 1. Univariate Models Examining Associations Between Impairments and Truncal Posture[a]

Explanatory Variables	Mean (SD) or N (%)	Adjusted R^2	P	Unstandardized β (95% CI)
Age, y	66.5 (7.6)	0.04	0.04	0.08 (0.01 to 0.54)
Male gender	55 (67)	0.01	0.16	0.87 (−0.35 to 2.09)
MDS-UPDRS motor (0-132)	31.4 (11.5)	0.04	0.046	0.05 (0.001 to 0.1)
HY stage (1-5)	2.0 (0.7)	− 0.03	0.32	0.41 (−0.41 to 1.23)
Time since PD diagnosis, y	7.5 (5.7)	− 0.01	0.68	0.02 (−0.08 to 0.12)
Bradykinesia[b] (0-40)	15.7 (7.1)	0.01	0.21	0.05 (−0.03 to 0.13)
Tremor[c] (0-40)	5.1 (4.9)	− 0.01	0.93	0.01 (−0.12 to 0.13)
Rigidity[d] (0-20)	4.0 (2.5)	− 0.01	0.61	0.06 (−0.17 to 0.29)
Axial subscore[e] (0-44)	7.4 (4.2)	0.08	0.01	0.19 (0.06 to 0.32)
NFOGQ (0-29)	4.8 (7.3)	− 0.01	0.62	− 0.02 (−0.10 to 0.06)
Sway on foam, eyes closed, mm	287.7 (147.7)	− 0.03	0.73	0.001 (−0.003 to 0.005)
Reactive stepping (0-8)	6.2 (1.8)	− 0.01	0.54	0.10 (−0.22 to 0.42)
FAB[f] (0-18)	14.7 (2.1)	0.02	0.11	− 0.23 (−0.51 to 0.05)

Abbreviations: FAB, Frontal Assessment Battery; HY, Hoehn and Yahr; MDS-UPDRS, Movement Disorders Society–sponsored Unified Parkinson's Disease Rating Scale; NFOGQ, New Freezing of Gait Questionnaire.

[a]Posture measured as distance from the 7th cervical vertebra to the wall in standing.

[b]Bradykinesia is the sum of left and right finger tap, hand movements, supination/pronation, toe tap and leg agility.

[c]Tremor is the sum of resting tremor of each limb and lip/jaw, postural and kinetic tremor, and tremor constancy.

[d]Rigidity is the sum of the 4 limbs and neck rigidity score.

[e]Axial subscore is composed of lower limb resting tremor, neck and lower limb rigidity, leg agility, arising from a chair, postural stability, posture, and gait items of the MDS-UPDRS.[20]

[f]High score = better performance. For all other measures, a high score indicates poorer performance.

Table 2. Multivariate Model Examining Associations Between Explanatory Variables and Truncal Posture (Adjusted $R^2 = 0.14$, $P = 0.01$)

Explanatory Variables	P	Unstandardized β (95% CI)	β
Age	0.38	0.03 (−0.04 to 0.11)	0.10
Bradykinesia (0-40)	0.14	− 0.08 (−0.19 to 0.03)	− 0.22
Tremor (0-40)	0.61	− 0.03 (−0.15 to 0.09)	− 0.06
Rigidity (0-20)	0.12	− 0.22 (−0.50 to 0.06)	− 0.21
Axial subscore (0-44)	0.001	0.43 (0.19 to 0.68)	0.69
NFOGQ (0-29)	0.04	− 0.09 (−0.18 to −0.01)	− 0.26
Reactive stepping (0-8)	0.20	− 0.24 (−0.61 to 0.13)	− 0.17
FAB[a] (0-18)	0.12	− 0.22 (−0.50 to 0.06)	− 0.17

Abbreviations: FAB, Frontal Assessment Battery; NFOGQ, New Freezing of Gait Questionnaire.

[a]High score = better performance. For all other measures, a high score indicates poorer performance.

the tasks requiring a change in base of support: single- and dual-task TUG test, and choice stepping reaction time (adjusted $R^2 = 0.06$-0.142, $P = 0.001$-0.02). Truncal posture was not significantly associated with walking speed or repeated STS (Table 4). The multivariate model containing truncal posture as well as disease severity, disease duration, age, and gender revealed small to moderate associations with the performance of all balance and mobility tasks tested (adjusted $R^2 = 0.24$-0.33, $P = <0.001$-0.03). Truncal posture continued to be independently associated with performance in functional reach, coordinated stability, and single-task TUG test in multivariate models ($\beta = -0.33$ to 0.27, $P = 0.002$-0.04) (Table 5).

DISCUSSION

This study found that the C7 to wall measurement of flexed truncal posture was highly reliable and was independently associated with global axial symptoms in people with PD. In addition, greater flexed truncal posture was associated with poorer performance on balance and mobility tasks over and above the contribution of age, gender, disease severity, and duration.

Reliability of the Truncal Posture Measurement

The simple, clinically feasible C7 to wall measure of truncal posture showed high reliability in people with PD. We elected to measure participants' "usual" truncal posture rather than their "best upright" truncal posture as this has been found to better reflect performance in everyday activities in the general older population.[12] Clinicians should consider using the C7 to wall measurement to assess and monitor flexed truncal posture in people with PD.

Relationships Between Impairments and Flexed Truncal Posture

We found that none of the cardinal motor impairments of PD (ie, bradykinesia, rigidity, tremor, and postural instability) were associated with flexed truncal posture when considered individually. Of the variables we examined, only age, disease severity, and global axial symptoms were significantly associated with flexed truncal posture. Within the multivariate model, global axial symptoms continued to show the strongest association, although FOG also showed a small independent association.

In our sample, flexed truncal posture was associated with both age and disease severity, findings consistent with that reported in the general older population.[2,24] Our finding that global axial symptoms were the strongest predictor of flexed truncal posture supports the assertion that postural abnormalities in PD are one of a group of axial symptoms present in some forms of the disease.[25] Despite suggestions that flexed truncal posture is at least partly due to increases in axial rigidity,[24] we did not find an association between rigidity and truncal posture. This may be due to the lack of sensitivity of the measurement used or the weighting toward appendicular rigidity. Nevertheless, studies using a more sensitive measurement of axial

Table 3. Univariate Models Examining Associations Between Impairments and Truncal Posture for People With PD Who Do and Do Not Report Freezing of Gait

Explanatory Variables	Participants *Without* Freezing of Gait (n = 52) Mean (SD) or N (%)	Adjusted R^2	P	Unstandardized B (95% CI)	Participants *With* Freezing of Gait (n = 30) Mean (SD) or N (%)	Adjusted R^2	P	Unstandardized B (95% CI)
Posture, cm	9.2 (2.8)				9.0 (2.3)			
Age, y	66.1 (7.9)	0.08	0.02	0.31 (0.02 to 0.21)	67.3 (7.2)	− 0.03	0.75	0.02 (−0.11 to 0.15)
Male gender	33 (63.5)	0.06	0.046	1.59 (0.03 to 3.16)	22 (73.3)	− 0.03	0.59	− 0.53 (−2.54 to 1.15)
MDS-UPDRS motor	29.9 (11.7)	0.07	0.03	0.07 (0.01 to 0.14)	33.8 (10.9)	− 0.03	0.68	0.02 (−0.07 to 0.10)
HY stage (1-5)	1.9 (0.7)	0.02	0.16	0.77 (−0.31 to 1.85)	2.4 (0.6)	− 0.03	0.84	− 0.16 (−1.77 to 1.45)
Time since PD diagnosis, y	5.9 (4.4)	− 0.01	0.57	0.05 (−0.13 to 0.23)	10.3 (6.7)	− 0.03	0.82	0.02 (−0.12 to 0.15)
Bradykinesia[a] (0-40)	14.4 (6.8)	0.03	0.10	0.09 (−0.02 to 0.21)	17.9 (7.1)	− 0.04	0.99	0.001 (−0.13 to 0.13)
Tremor[b] (0-40)	6.2 (5.3)	− 0.02	0.77	− 0.02 (−0.17 to 0.13)	3.1 (3.2)	− 0.01	0.45	0.11 (−0.18 to 0.39)
Rigidity[c] (0-20)	3.8 (2.2)	− 0.01	0.46	0.13 (−0.22 to 0.49)	4.5 (2.9)	− 0.04	0.999	0.00 (−0.31 to 0.31)
Axial subscore[d] (0-44)	6.3 (3.8)	0.20	0.001	0.34 (0.15 to 0.53)	9.4 (4.2)	0.02	0.52	0.07 (−0.15 to 0.28)
NFOGQ (0-29)	0 (0)				13.1 (5.9)	− 0.03	0.60	− 0.04 (−0.19 to 0.11)
Sway on foam, eyes closed (mm)	277.4 (133.6)	− 0.02	0.70	0.001 (−0.005 to 0.007)	306.0 (171.1)	− 0.04	0.98	0.00 (−0.01 to 0.01)
Reactive stepping (0-8)	1.6 (1.5)	0.03	0.10	0.43 (−0.09 to 0.95)	2.2 (2.2)	− 0.02	0.48	− 0.14 (−0.55 to 0.26)
FAB[e] (0-18)	14.9 (2.0)	0.02	0.11	− 0.29 (−0.67 to 0.10)	14.4 (2.1)	− 0.01	0.44	− 0.16 (−0.58 to 0.26)

Abbreviations: FAB, Frontal Assessment Battery; HY, Hoehn and Yahr; MDS-UPDRS, Movement Disorders Society–sponsored Unified Parkinson's Disease Rating Scale; NFOGQ, New Freezing of Gait Questionnaire.
[a]Total bradykinesia is the sum of left and right finger tap, hand movements, sup/pro, toe tap, and leg agility.
[b]Total tremor is the sum of resting tremor of each limb and lip/jaw, postural and kinetic tremor, and tremor constancy.
[c]Total rigidity is the sum of the 4 limbs and neck rigidity score.
[d]Axial subscore is composed of LL resting tremor, neck and LL rigidity, leg agility, STS, postural instability, posture, gait.[19]
[e]High score = better performance. For all other measures, a high score indicates poorer performance.

Table 4. Univariate Analyses of Associations Between Truncal Posture and Balance and Mobility Tasks

Outcome	Mean (SD)	Adjusted R^2	P	Unstandardized B (95% CI)
Tasks *not* requiring a change in base of support				
Functional reach, cm[a]	27.8 (6.9)	0.14	<0.001	− 1.01 (−1.55 to −0.47)
Lateral reach, cm[a]	20.0 (4.8)	0.07	0.01	− 1.05 (−1.84 to −0.27)
SLS, s[a]	16.5 (9.1)	0.05	0.03	− 1.69 (−3.21 to −0.16)
Coordinated stability score	15.9 (16.62)	0.06	0.01	1.74 (0.38 to 3.10)
Tasks requiring a change in base of support				
TUG, s	10.2 (3.1)	0.12	0.001	0.42 (0.18 to 0.67)
TUGcog, s	12.6 (5.0)	0.07	0.01	0.54 (0.13 to 0.95)
Walking speed—preferred, m/s[a]	1.25 (0.23)	0.01	0.17	− 0.01 (−0.03 to 0.01)
Walking speed—fast, m/s[a]	1.73 (0.37)	0.02	0.14	− 0.02 (−0.05 to 0.01)
STS, stand/s[a]	0.50 (0.12)	0.01	0.24	− 0.01 (−0.02 to 0.004)
Choice stepping, step/s[a]	0.35 (0.08)	0.06	0.02	− 0.01 (−0.02 to −0.002)

Abbreviations: SLS, single leg stand; STS, 5 repetition sit-to-stand; TUG, Timed Up and Go; TUGcog, TUG cognitive.
[a] High score = better performance. For all other measures, a high score indicates poorer performance.

Table 5. Multivariate Models Examining Associations With Balance and Mobility Tasks

Adjusted R^2 (P)	Functional Reach,[a] cm 0.24 (<0.001)		Lateral reach,[a] cm 0.31 (<0.001)		Single leg stand,[a] s 0.33 (0.001)		Coordinated Stability Score 0.33 (0.001)		Timed Up and Go, s 0.24 (<0.001)		Timed Up and Go Cognitive, s 0.29 (<0.001)		Choice stepping,[a] step/s 0.35 (<0.001)	
	B	P	B	P	B	P	B	P	B	P	B	P	B	P
Truncal posture	− 0.33	0.002	− 0.17	0.09	− 0.11	0.25	0.20	0.04	0.27	0.01	0.13	0.21	− 0.15	0.13
MDS-UPDRS motor	− 0.26	0.01	− 0.26	0.01	− 0.08	0.39	0.16	0.11	0.09	0.38	0.27	0.01	− 0.39	<0.001
Time since PD diagnosis	− 0.23	0.02	− 0.10	0.31	− 0.42	<0.001	0.37	<0.001	0.05	0.58	0.14	0.14	− 0.11	0.24
Age	− 0.01	0.90	− 0.37	<0.001	− 0.39	<0.001	0.25	0.01	0.37	0.001	0.38	<0.001	− 0.35	<0.001
Male gender	0.11	0.28	0.18	0.06	0.03	0.78	− 0.24	0.01	− 0.1	0.32	0.01	0.90	− 0.05	0.63

Abbreviations: B = standardized B; MDS-UPDRS: Movement Disorders Society–sponsored Unified Parkinson's Disease Rating Scale.
[a]High score = better performance. For all other measures, a high score indicates poorer performance.

rigidity[26] have also reported no association between rigidity and truncal posture.

Freezing of gait is also considered to be an axial feature of PD, so it was expected that worse FOG would be associated with more severe flexed truncal posture. However, in our sample, we found a suggestion of an opposite relationship, with greater flexed truncal posture associated with less FOG. The mechanism behind this finding is unclear. One possible explanation is that the adoption of a flexed truncal posture might assist in facilitating gait initiation[6] as the center of mass is further forward, thereby requiring smaller amplitude anticipatory postural adjustments to initiate gait.[27] It is likely that this compensation would be effective only for a limited time before the severity of the freezing is unable to be overcome by a change in truncal posture.[2,24,26]

Although people with and without FOG had a similar degree of flexed truncal posture, the variables associated with flexed truncal posture in each group differed. In participants without FOG, the pattern of associations was similar to the whole group. In contrast, we did not identify any factors that were significantly associated with flexed truncal posture in participants with FOG, although the small number of participants might have limited our ability to demonstrate significant results.

A large proportion (86%) of the variance in flexed truncal posture in PD remains unexplained. Potential contributors not assessed in this study include spinal proprioception, visuosensory integration, and musculoskeletal problems. Impaired axial proprioception has been identified in people with PD[20] and is an impairment that, like other axial symptoms, responds poorly to levodopa and is thought to be controlled by nondopaminergic central structures.[26] People with PD show an increased reliance on vision to maintain truncal posture,[28] providing further support for a link between flexed truncal posture and impaired spinal proprioception. We excluded participants who had significant musculoskeletal impairments, but some participants may have had undiagnosed vertebral fractures or degenerative disc diseases which are known to be major risk factors for flexed truncal posture in the general older population.[2,29] Other nonmotor impairments of PD, including depression and pain, are also potential contributors to flexed truncal posture, which were not assessed here.

Influence of Truncal Posture on Balance and Mobility Tasks

Participants with greater flexed truncal posture performed worse in a number of balance and mobility tasks, even when other demographic factors were taken into consideration. In univariate analysis there were significant but weak associations between truncal posture and all of the tasks that did not require a change in base of support, in line with our hypothesis. Significant relationships were found with functional reach, lateral reach, and the coordinated stability test, all tasks that require participants to move to their anterposterior and/or mediolateral limits of stability. Flexion of the thoracic spine can lead to an anterior shift of the center of gravity,[30] reducing the stability margin,[8] particularly in the forward direction,[9] and thus impair the postural stability required to achieve tasks that require leaning balance.[30] Recent research in people with chronic stroke has also found negative associations with greater flexed truncal posture and balance.[31] The authors highlighted the influence of pelvic tilt on truncal posture and this is an area that merits further investigation in people with PD.

Flexed truncal posture was found to be associated with reduced performance of the TUG test, in line with findings from the general older population.[32] However, walking speed and repeated STS were not associated with flexed truncal posture in our study. Studies in the general older population report variable findings regarding the relationship with gait speed, which may be due to differences in the severity of flexed truncal posture of participants.[32,33] Nevertheless, flexed truncal posture is associated with poorer postural control during walking, even when gait speed is maintained.[33] A flexed truncal posture necessitates compensatory changes in the lower limbs, trunk, and upper limbs that may affect the ability to maintain balance while walking.[10,33] In addition, it is plausible that flexed truncal posture negatively affects the ability to turn in the TUG test and contributes to the characteristic "en bloc" movement of the head and trunk observed during turning in people with PD.[34 35]

Overall, our findings suggest that flexed truncal posture has a negative effect on mobility and balance and may consequently have implications for fall risk.[33,36] Flexed truncal posture is a potential target for physical interventions, to either improve or possibly prevent its development. Exploring the efficacy of treating flexed truncal posture in people with PD is an area worthy of further investigation. Future studies should evaluate a broader range of impairments in participants with mild to severe flexed truncal posture to identify other factors that might contribute to flexed truncal posture in people with PD.

This study has a number of limitations. Our group of participants had relatively mild flexed truncal posture[12] and people with severe cardiovascular disease or musculoskeletal or orthopedic conditions were excluded. While we found no association between truncal posture and cognition as measured by the FAB, we cannot preclude that there may be a relationship between truncal posture and cognition. Participants with an MMSE score less than 24 were excluded. All testing was done while participants were "on" medication; thus, we cannot draw conclusions on the associations of PD impairments with flexed truncal posture when "off" medication. Overall, our group of participants had mild to moderate PD, so further studies should evaluate people with more severe PD as well as a greater range of flexed truncal postures.

CONCLUSIONS

Global axial symptoms had the strongest association with flexed truncal posture but nevertheless a multivariate model, including axial symptoms, accounted for only 14% of the variance in truncal posture. Further exploration of the pathogenesis of flexed truncal posture in PD is required. Greater flexed truncal posture is associated with poorer performance in a number of balance and mobility tasks. Further studies are warranted to explore the relationship between flexed truncal posture and function as well as to examine the effect of intervention on truncal posture.

ACKNOWLEDGMENTS

This research was supported by a seeding grant from the Physiotherapy Research Foundation of Australia (ID: S10-012). The authors thank Jooeun Song for her assistance with data collection. We also thank the people with Parkinson disease who participated in this research.

REFERENCES

1. Latt MD, Lord SR, Morris JG, Fung VS. Clinical and physiological assessments for elucidating falls risk in Parkinson's disease. *Mov Disord*. 2009;24(9):1280-1289.
2. Kado DM, Prenovost K, Crandall C. Narrative review: hyperkyphosis in older persons. *Ann Intern Med*. 2007;147(5):330-338.
3. Kashihara K, Imamura T. Clinical correlates of anterior and lateral flexion of the thoracolumbar spine and dropped head in patients with Parkinson's disease. *Parkinsonism Relat Disord*. 2012;18(3):290-293.
4. Bartolić A, Pirtošek Z, Rozman J, Ribarič S. Postural stability of Parkinson's disease patients is improved by decreasing rigidity. *Eur J Neurol*. 2005;12(2):156-159.
5. Wright W, Gurfinkel V, Nutt J, Horak F, Cordo P. Axial hypertonicity in Parkinson's disease: direct measurements of trunk and hip torque. *Exp Neurol*. 2007;208(1):38-46.
6. Halliday SE, Winter DA, Frank JS, Patla AE, Prince F. The initiation of gait in young, elderly, and Parkinson's disease subjects. *Gait Posture*. 1998;8(1):8-14.
7. Bloem BR, Beckley DJ, van Dijk JG. Are automatic postural responses in patients with Parkinson's disease abnormal due to their stooped posture? *Exp Brain Res*. 1999;124(4):481-488.
8. Jacobs JV, Dimitrova DM, Nutt JG, Horak FB. Can stooped posture explain multidirectional postural instability in patients with Parkinson's disease? *Exp Brain Res*. 2005;166(1):78-88.
9. Mancini M, Rocchi L, Horak FB, Chiari L. Effects of Parkinson's disease and levodopa on functional limits of stability. *Clin Biomech*. 2008;23(4):450-458.
10. Saha D, Gard S, Fatone S. The effect of trunk flexion on able-bodied gait. *Gait Posture*. 2008;27(4):653-660.
11. Kado DM, Huang M-H, Nguyen CB, Barrett-Connor E, Greendale GA. Hyperkyphotic posture and risk of injurious falls in older persons: the Rancho Bernardo Study. *J Gerontol A Biol Sci Med Sci*. 2007;62(6): 652-657.
12. Ryan SD, Fried LP. The impact of kyphosis on daily functioning. *J Am Geriatr Soc*. 1997;45(12):1479-1486.
13. Balzini L, Vannucchi L, Benvenuti F, et al. Clinical characteristics of flexed posture in elderly women. *J Am Geriatr Soc*. 2003;51(10):1419-1426.
14. Kado DM, Browner WS, Palermo L, Nevitt MC, Genant HK, Cummings SR. Vertebral fractures and mortality in older women: a prospective study. *Arch Intern Med*. 1999;159(11):1215.
15. Bloch F, Houeto JL, Tezenas du Montcel S, et al. Parkinson's disease with camptocormia. *J Neurol Neurosurg Psychiatry*. 2006;77(11):1223-1228.
16. Paul S, Canning C, Sherrington C, Fung V. Reduced muscle strength is the major determinant of reduced leg muscle power in Parkinson's disease. *Parkinsonism Relat Disord*. 2012;18(8):974-977.
17. Paul SS, Sherrington C, Fung VS, Canning CG. Motor and cognitive impairments in Parkinson disease relationships with specific balance and mobility tasks. *Neurorehabil Neural Repair*. 2013;27(1):63-71.
18. Goetz CG, Tilley BC, Shaftman SR, et al. Movement Disorder Society–sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS): scale presentation and clinimetric testing results. *Mov Disord*. 2008;23(15):2129-2170.
19. Hoehn MM, Yahr MD. Parkinsonism: onset, progression, and mortality. *Neurology*. 1998;50(2):318-318.
20. Wright W, Gurfinkel V, King L, Nutt J, Cordo P, Horak F. Axial kinesthesia is impaired in Parkinson's disease: effects of levodopa. *Exp Neurol*. 2010;225(1):202-209.
21. Nieuwboer A, Rochester L, Herman T, et al. Reliability of the New Freezing of Gait Questionnaire: agreement between patients with Parkinson's disease and their carers. *Gait Posture*. 2009;30(4):459-463.
22. Dubois B, Slachevsky A, Litvan I, Pillon B. The FAB A Frontal Assessment Battery at bedside. *Neurology*. 2000;55(11):1621-1626.
23. Hallgren KA. Computing inter-rater reliability for observational data: an overview and tutorial. *Tutor Quant Methods Psychol*. 2012;8(1):23.
24. Schenkman M, Butler RB. A model for multisystem evaluation treatment of individuals with Parkinson's disease. *Phys Ther*. 1989;69(11): 932-943.
25. Bejjani B-P, Gervais D, Arnulf I, et al. Axial parkinsonian symptoms can be improved: the role of levodopa and bilateral subthalamic stimulation. *J Neurol Neurosurg Psychiatry*. 2000;68(5):595-600.
26. Franzén E, Paquette C, Gurfinkel VS, Cordo PJ, Nutt JG, Horak FB. Reduced performance in balance, walking and turning tasks is associated with increased neck tone in Parkinson's disease. *Exp Neurol*. 2009;219(2): 430-438.
27. Jacobs JV, Nutt JG, Carlson-Kuhta P, Stephens M, Horak FB. Knee trembling during freezing of gait represents multiple anticipatory postural adjustments. *Exp Neurol*. 2009;215(2):334-341.
28. Vaugoyeau M, Viel S, Assaiante C, Amblard B, Azulay J. Impaired vertical postural control and proprioceptive integration deficits in Parkinson's disease. *Neuroscience*. 2007;146(2):852-863.
29. Schneider DL, von Mühlen D, Barrett-Connor E, Sartoris DJ. Kyphosis does not equal vertebral fractures: the Rancho Bernardo study. *J Rheumatol*. 2004;31(4):747-752.
30. Kasukawa Y, Miyakoshi N, Hongo M, et al. Relationships between falls, spinal curvature, spinal mobility and back extensor strength in elderly people. *J Bone Miner Metab*. 2010;28(1):82-87.
31. Verheyden G, Ruesen C, Gorissen M, et al. Postural alignment is altered in people with chronic stroke and related to motor and functional performance. *J Neurol Phys Ther*. 2014;38(4):239-245.
32. Hirose D, Ishida K, Nagano Y, Takahashi T, Yamamoto H. Posture of the trunk in the sagittal plane is associated with gait in community-dwelling elderly population. *Clin Biomech*. 2004;19(1):57-63.
33. de Groot MH, van der Jagt-Willems HC, van Campen JP, Lems WF, Beijnen JH, Lamoth CJ. A flexed posture in elderly patients is associated with impairments in postural control during walking. *Gait Posture*. 2014;39(2):767-772.
34. Crenna P, Carpinella I, Rabuffetti M, et al. The association between impaired turning and normal straight walking in Parkinson's disease. *Gait Posture*. 2007;26(2):172-178.
35. Schenkman M, Morey M, Kuchibhatla M. Spinal flexibility and balance control among community-dwelling adults with and without Parkinson's disease. *J Gerontol A Biol Sci Med Sci*. 2000;55(8):M441-M445.
36. Katzman WB, Vittinghoff E, Ensrud K, Black DM, Kado DM. Increasing kyphosis predicts worsening mobility in older community-dwelling women: a prospective cohort study. *J Am Geriatr Soc*. 2011;59(1): 96-100.