BRIEF REPORT

Bradykinesia in Neurodegenerative Disorders: A Blinded Video Analysis of Pathology-Proven Cases

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ABSTRACT: Background: Bradykinesia is a cardinal feature in parkinsonisms. No study has assessed the differential features of bradykinesia in patients with pathology-proven synucleinopathies and tauopathies.

Objective: We examined whether bradykinesia features (speed, amplitude, rhythm, and sequence effect) may differ between pathology-proven synucleinopathies and tauopathies.

Methods: Forty-two cases who underwent autopsy were included and divided into synucleinopathies (Parkinson's disease and dementia with Lewy bodies) and tauopathies (progressive supranuclear palsy). Two raters blinded to the diagnosis retrospectively scored the Movement Disorders Society-Unified Parkinson's Disease Rating Scale Part III and Modified Bradykinesia Rating Scale on standardized videotaped neurological examinations. Bradykinesia scores were compared using the Mann–Whitney test and logistic regression models to adjust for disease duration.

Results: Demographic and clinical parameters were similar between synucleinopathies and tauopathies. There were no differences between speed, amplitude, rhythm, and sequence effect in synucleinopathies and tauopathies in unadjusted comparisons and adjusted models (all $P > 0.05$).

Conclusions: Clinical bradykinesia features do not distinguish the underlying neuropathology in neurodegenerative parkinsonisms. © 2023 The Authors. *Movement Disorders* published by Wiley Periodicals LLC on behalf of International Parkinson and Movement Disorder Society.

Key Words: bradykinesia; neurodegeneration; synucleinopathy; tauopathy; neuropathology

Introduction

Bradykinesia, namely, the slowness of movement, associated with progressive reduction in amplitude and velocity during movement repetition (eg, the so-called decrement/sequence effect),[1-5] is a cardinal feature in parkinsonisms. Semiologically, it may express differently in Parkinson's disease (PD) and other "synucleinopathies," such as dementia with Lewy bodies (DLB) and multiple system atrophy,[5,6] versus such "tauopathies" as progressive supranuclear palsy (PSP) and corticobasal degeneration.[7-10] Recent studies have also documented bradykinesia in traditionally non-parkinsonian neurodegenerative disorders such as Alzheimer's disease (AD) and amyotrophic lateral sclerosis.[10-14]

Neurophysiological studies based on kinematic techniques have shown that bradykinesia features may vary in patients with PD and other neurodegenerative disorders.[10,14] Some studies have suggested that bradykinesia features may differ between early and advanced PD and also between synucleinopathies and tauopathies.[4,8,10,15,16] As it is widely appreciated, a huge phenotypic overlap exists between clinically diagnosed neurodegenerative disorders, and clinical diagnostic criteria have limitations in predicting the underlying pathology of a particular set of features.[6,17]

To date, no study has specifically assessed bradykinesia in patients with pathology-proven neurodegenerative disorders. We performed a retrospective blinded video analysis of bradykinesia features in a cohort of patients with a synucleinopathy or tauopathy diagnosed by pathological examination. We sought to determine whether bradykinesia features (speed, amplitude, rhythm, and sequence effect) can

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Relevant conflicts of interest/financial disclosures: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Received: 10 October 2022; **Revised:** 28 December 2022; **Accepted:** 9 January 2023

Published online in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/mds.29330

distinguish between patients with pathology-proven synucleinopathies versus tauopathies as a group or within a specific pathological diagnosis.

Subjects and Methods

Subjects

Consecutive pathology-proven cases diagnosed with a synucleinopathy or tauopathy between January 2011 and December 2021 at the University of Cincinnati were included in this study. Inclusion criteria were at least one complete videotaped examination of the Unified Parkinson's Disease Rating Scale Part III (UPDRS-III) or Movement Disorder Society-UPDRS Part III (MDS-UPDRS-III) and ability to follow the instructions in the video.

The study protocol was reviewed and approved by the Institutional Review Board (IRB#2019–1312) at the University of Cincinnati.

Clinical Information

One author extracted the following data by chart review, blinded to the pathological diagnosis and to the respective patients' bradykinesia scores: gender, age at symptoms onset, age at death, and the following variables from the day of the videotape evaluation: age, rigidity scores of the UPDRS-III or MDS-UPDRS-III, Hoehn and Yahr (H&Y) score,[18] presence of cognitive impairment (yes/no), concomitant disease (if any), and medications.[19] The levodopa equivalent daily doses (LEDDs) were calculated as reported elsewhere.[20]

Pathological Diagnoses

Neuropathology examination was performed using formalin-fixed paraffin-embedded tissue blocks of several cortical, subcortical, and brainstem regions and the cerebellum (further details of pathological examination are displayed in Supporting Information Data S1).

Retrospective Blinded Analysis of Video Recordings

Two blinded movement disorders–trained assessors (E.A. and N.G.) independently scored the Modified Bradykinesia Rating Scale (MBRS)[21,22] during finger tapping for each hand and then MDS-UPDRS-III.[23] The assessors were instructed to rate the finger tapping at the beginning (without audio on) and then the rest of the MDS-UPDRS-III for each video (both with audio off and audio on), to avoid possible bias. The videos followed a standardized sequence.[24] Any discrepancies were resolved with the intervention of a third rater (L.M.), a neurologist with more than

TABLE 1 *Demographic and clinical characteristics of participants*

Characteristics	Synucleinopathy (n = 25)	Tauopathy (n = 17)	*P* value
Age at symptom onset, y	67 (57–72)	69 (62–72)	0.50[a]
Age at death, y	75 (69–79)	76 (70–82)	0.79[a]
Disease duration at death, y	9 (6–11)	8 (6–10)	0.52[a]
Sex (F/M), n (%)	11/14 (44/64)	9/8 (53/47)	0.75[b]
At videotaped evaluation			
Age, y	71 (64–77)	72.5 (67–78)	0.75[a]
Disease duration at examination, y	5 (3–7)	4 (2–6)	0.25[a]
MDS-UPDRS-III score	45 (36–61)	41 (32–61)	0.69[a]
Hoehn & Yahr scale, n (%)			0.28[a]
1	1 (4)	1 (6)	
2	8 (32)	4 (23.5)	
3	8 (32)	2 (12)	
4	3 (12)	4 (23.5)	
5	5 (20)	6 (35)	
Levodopa equivalent daily dose, mg	1200 (500–1620)	800 (400–1200)	0.35[a]
Cognitive impairment, n (%)	22 (88)	13 (76)	0.25[b]

Data are presented as median (25th–75th percentile) or as noted otherwise.
F, female; M, male; MDS-UPDRS-III, Movement Disorder Society-sponsored Revision of the Unified Parkinson's Disease Rating Scale Part III.
[a]Exact *P* values of the Mann–Whitney *U* test.
[b]Fisher's exact test.

15 years of experience in movement disorders. The MBRS independently scores three components of bradykinesia—amplitude, speed, and rhythmicity—from 0 (normal) to 4 (can barely perform the task). An MBRS total score for each hand is then calculated by summing the three scores. In addition, raters defined whether hands with MDS-UPDRS-III item 3.4 scores between 1 and 3 showed a sequence effect (decrement in amplitude) (yes/no).[23]

For each bradykinesia score (MBRS component and total scores and MDS-UPDRS-III item 3.4), we employed the mean score of both hands, the maximum

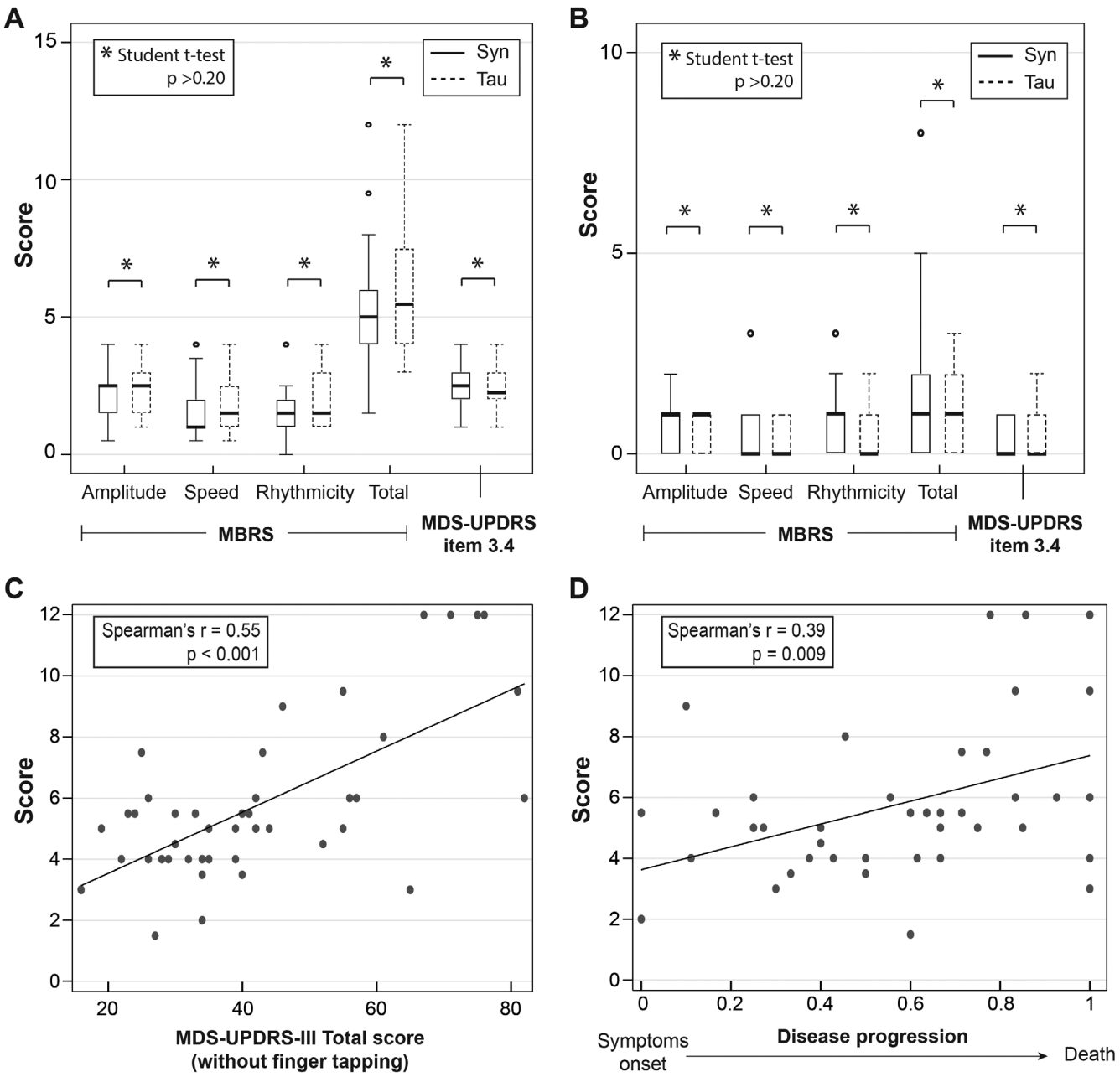

FIG. 1. Mean (**A**; mean Modified Bradykinesia Rating Scale [MBRS] score of both hands) and asymmetry (**B**; simple MBRS score difference between hands) bradykinesia scores. All comparisons between pathology-proven synucleinopathies (Syn; *solid-line boxes*) and tauopathies (Tau; *dashed-line boxes*) yielded $P > 0.20$, including after adjusting by disease duration in a logistic regression analysis. The top, bottom, and the line inside the box represent the first and third quartiles and median, respectively. The whiskers define the extreme value, up to 1.5× the interquartile range. Circumferences beyond the whiskers represent outliers. (**C, D**) Relationship between the bradykinesia score (the average of the MBRS total score of both hands) and Movement Disorder Society–Unified Parkinson's Disease Rating Scale Part III (MDS-UPDRS-III) total score after removing the finger-tapping scores (**C**) and the proportion of disease duration at the examination until death (**D**). Each dot represents the score of each subject. MBRS, Modified Bradykinesia Rating Scale; *r*, Spearman's correlation coefficient.

hand score, and the asymmetry score, defined as the simple score difference between hands, as reported elsewhere.[25]

Statistical Analysis

We calculated that our sample size would provide an 80% power to detect a mean difference of 0.8 in the MBRS amplitude score (mean score of both hands) at a two-sided 5% significance level, with our calculated mean MBRS amplitude score of 2.2 for synucleinopathy and combined standard deviation of 0.9. Similarly, with our calculated mean MBRS total score of 5 for synucleinopathy and combined standard deviation of 2.4, we could detect a mean difference of 2.0 in the MBRS total score (mean score of both hands) at the same power and significance level. Differences in dichotomous and non-normally distributed continuous demographic variables were assessed by the Fisher's exact test and the exact P values of the Mann–Whitney U test, respectively. Interrater agreement on bradykinesia scores was calculated with the unweighted Cohen's kappa statistic, and the level of agreement was evaluated according to Fleiss et al.[26] The bradykinesia scores (mean, maximum, asymmetry) between synucleinopathies and tauopathies were compared using Student's t tests. We additionally compared patients with PD/DLBD versus patients with PSP, the most common synucleinopathies and tauopathy, respectively. Logistic regressions were used to compare bradykinesia scores after adjusting for disease duration, defined either as the number of years since symptoms onset or the proportion of disease duration until death. Correlation analyses by Spearman's method were used to assess the relationship between bradykinesia severity (mean MBRS total score among hands) and disease severity, considered as the MDS-UPDRS-III total score after removing the finger-tapping scores and the proportion of disease duration until death. All P values were two-sided, and those <0.05 were deemed statistically significant. All analyses were performed in STATA v.17.0 (StataCorp LLC).

Results

Of 71 total autopsies performed, 42 cases (22 males, 52%) met inclusion criteria and were included in the study.

Demographic and Clinicopathological Features

No differences were found between synucleinopathies and tauopathies in terms of age at symptoms onset, age at evaluation, disease duration at evaluation, sex, MDS-UPDRS-III total scores, H&Y scores, presence of cognitive impairment, LEDD, age at death, and disease duration at death (Table 1; all $P > 0.05$). Detailed pathological diagnoses are displayed in Supporting Information Data S1.

Bradykinesia in Synucleinopathies Versus Tauopathies

The interrater agreement for the MBRS scores and MDS-UPDRS item 3.4 scores was excellent (96.5% agreement, 415/430 scores; unweighted kappa = 0.97, $P < 0.001$).

No difference in mean, maximum, and asymmetry bradykinesia scores (MBRS amplitude, speed, rhythmicity, or total scores or MDS-UPDRS-III item 3.4 score) was found between synucleinopathies and tauopathies in bivariate analysis, nor after adjusting for disease duration (all $P > 0.20$; Fig. 1A,B, Supporting Information Table S2). In the subanalysis comparing patients with PD and DLBD with patients with PSP, there were no differences in bradykinesia scores (Supporting Information Table S3). Seventeen (74%) patients showed sequence effect among the 23 synucleinopathy patients with at least one hand with an MDS-UPDRS-III item 3.4 score between 1 and 3, compared with 12 (75%) of the 16 tauopathy patients with an MDS-UPDRS-III item 3.4 score between 1 and 3 ($P > 0.99$).

Bradykinesia Severity and Disease Severity

Mean MBRS total score among hands was correlated with disease severity in terms of the MDS-UPDRS-III total score after removing the finger-tapping scores ($r = 0.55$; $P < 0.001$) and the proportion of disease duration until death ($r = 0.39$; $P = 0.009$; Fig. 1C,D).

Discussion

We retrospectively analyzed the main features of bradykinesia in a blinded video analysis of a cohort of pathology-proven patients with synucleinopathies and tauopathies. We found that bradykinesia features did not differ between the two groups, even when we focused on more homogeneous groups, namely, when comparing only PD/DLBD (among synucleinopathies) versus PSP (among tauopathies). Bradykinesia correlated with disease severity, independently from the pathological diagnosis. The "tauopathy" group showed a trend for higher H&Y scores and lower LEDD compared with synucleinopathies. These nonsignificant findings may be because of the greater underlying disease severity in PSP compared with DLB and PD and the greater dopaminergic sensitivity in the latter.

Previous studies have clinically and neurophysiologically investigated bradykinesia features only in clinically diagnosed conditions, mainly within the PD spectrum and to a lesser degree in other synucleinopathies and tauopathies.[1-5,7,8,12,27-31] However, no study had previously investigated bradykinesia features by means of

clinical scales in patients with pathology-proven parkinsonisms. Hence in our study, we have confirmed that bradykinesia features significantly overlap among synucleinopathies and tauopathies.[10,14] Second, we have shown that the sequence effect is equally distributed across synucleinopathies and tauopathies and cannot be used as a distinguishing feature for any category, unlike clinical studies suggesting no such feature in PSP.[16,32]

In general, our findings suggest that bradykinesia features are more likely to reflect the underlying disease severity rather than the specific pathology. In this view, similar degrees of bradykinesia may result from widespread degeneration of neural circuitries regardless of associated pathology.[32,33] It has been suggested that bradykinesia should be considered as a network dysfunction of different structures such as basal ganglia, sensorimotor cortical areas, and cerebellum, rather than a consequence of one single system abnormality.[32] Accordingly, we hypothesize that different pathological conditions, in the advanced stages, may share common disruptions in the "bradykinesia network," thus clinically manifesting with similar bradykinetic features. Our study is in line with the clinical diagnostic criteria for PD[5] and DLB,[34] which state that bradykinesia is present (and identical) in the two conditions. In addition, some authors have suggested that PD dementia and DLB should be considered as extremes on a continuum both from the clinical and the pathological standpoint.[35,36] In the category of synucleinopathies, also patients with multiple system atrophy have been shown to harbor the same clinical features of bradykinesia and decrement of patients with PD,[15] thus supporting our patients' classification. Regarding tauopathies, our group was more heterogeneous, with a main subgroup represented by patients with PSP.

This study has several limitations because of its retrospective nature based on previously videotaped clinical assessments and on a cross-sectional design. However, our video dataset was collected and executed in a standardized fashion, as previously reported,[24] and consistently following the items of the MDS-UPDRS-III scale. All videotaped examinations were independently rated by two raters, thus potentially adding a confounder; however, the interrater agreement of the number and type of involved body sites and the movement disorders classification was excellent ($P < 0.001$; $K = 1$). A major limitation is the relatively small sample size with sparse diagnoses such as mixed tauopathies and corticobasal degeneration within the category of tauopathies, precluding comparisons within and between these patients. Unfortunately, we did not have videos of neurological examination in the earlier stages of the disease. Finally, as a result of the study design, we could not correlate the severity and characteristics of bradykinesia to the extent and location of the pathological changes. However, the findings are consistent with the increasing recognition that pathology does not correlate with (or cause) degeneration and may instead represent a consequence of many biological, toxic, or infectious etiologies.[37]

Notably, among the non-AD pathological diagnoses, 59% (22/37) of our cases showed comorbid AD-related pathology. The issue of copathology findings in neurodegenerative diseases is common and has been previously documented as a frequent finding in neurodegenerative diseases.[38] The exact role of copathology in neurodegenerative diseases remains controversial.[38,39] The high frequency of copathology in our series, as reported elsewhere, may well be the rule rather than the exception,[38,39] further complicating the assessment of bradykinesia features across "pure" neurodegenerative disorders.

In conclusion, this study suggests that no clinical features of bradykinesia can reliably distinguish among the various parkinsonisms, possibly reflecting a common disruption of neural circuits shared across all pathological diagnoses.[9] Bradykinesia features do not reflect the specificity of the ongoing pathological processes occurring in neurodegenerative disorders. Larger clinicopathological studies may be warranted to confirm these findings.

Acknowledgments: We acknowledge the contribution of the patients involved in this study.

Data Availability Statement

Luca Marsili had full access to all the data in the study and takes responsibility for the integrity of the data, the accuracy of the data analysis, and the conduct of the research. He has the right to publish all data, separate and apart from the guidance of any sponsor. The study was performed in accordance with the ethical standards of the 1964 Declaration of Helsinki and its later amendments.

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Supporting Data

Additional Supporting Information may be found in the online version of this article at the publisher's web-site.

Graphical abstract

Bradykinesia in Neurodegenerative Disorders: A Blinded Video Analysis of Pathology-Proven Cases

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We examined whether bradykinesia features (speed, amplitude, rhythm, and sequence effect) may differ between patients with pathology-proven synucleinopathies and tauopathies. Forty-two cases who underwent autopsy were included. Demographic, clinical parameters, and bradykinesia scores were similar between synucleinopathies and tauopathies. Clinical bradykinesia features do not distinguish the underlying neuropathology in neurodegenerative parkinsonisms.