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Clinical correlates of repetitive speech disorders in Parkinson's disease

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ABSTRACT

Objectives: This study aimed to explore clinical correlates of repetitive speech disorders in patients with Parkinson's disease (PD).

Methods: This study investigated speech function (Assessment of Motor Speech for Dysarthria and Stuttering Severity Instrument-3), motor function (Unified Parkinson's Disease Rating Scale III [UPDRS-III] and UPDRS-IV), cognitive function (Mini-Mental State Examination [MMSE], Montreal Cognitive Assessment [MoCA], Stroop color-word test, verbal fluency, digit span tests, and line orientation), and activities of daily living of 113 PD patients. Comparison between groups (independent *t*-tests, Mann–Whitney *U* tests, or χ^2 test) and linear regression analyses were performed to determine clinical correlates of repetitive speech disorders.

Results: Totally, 65 patients (57.5%) had repetitive speech disorders. Patients with repetitive speech disorders had significantly worse UPDRS-III ($P = .049$), MoCA ($P = .030$), and speech function and higher levodopa equivalent daily dose (LEDD; $P = .031$) than those without repetitive speech disorders. Males were significantly predominant in patients with repetitive speech disorders (64.6%) compared to those without repetitive speech disorders (18.7%; $P < .001$). The univariate and subsequent multiple linear regression analyses revealed that the severity of repetitive speech disorders significantly correlated with gender ($P < .001$), MoCA ($P = .006$), and speech variables (abnormal rate, $P = .007$; imprecise consonants, $P = .043$), independent from disease duration, UPDRS III, and LEDD.

Conclusions: PD patients with repetitive speech disorders had worse motor, cognitive, and speech functions than those without repetitive speech disorders. The most influential factor for repetitive speech disorders might be male gender.

1. Introduction

Speech disorders affect up to 90% of patients with Parkinson's disease (PD) and reduce patients' quality of life [1,2]. Voice deficits (monotonous speech, reduced volume, and harsh voice) appear most frequently and earlier in their disease course; and fluency and articulation deficits tend to become evident later [1]. These speech disorders are collectively called as hypokinetic dysarthria. Other speech disorders frequently observed in patients with PD are repetitive speech disorders, which is characterized by frequent successive repetitions of syllables, words, or phrases [3].

Because there has been no consensus on the define and description of repetitive speech disorders in PD, previous investigators used different terms, including repetitive speech phenomena, stuttering, or palilalia [3–6]. World Health Organization (WHO) defines stuttering as "speech that is characterized by frequent repetition or prolongation of sounds or syllables or words, or by frequent hesitations or pauses that disrupt the rhythmic flow of speech" [7]. In contrast, palilalia is reportedly characterized by the accumulative rapidity and declining loudness besides the repetitive feature of speech [3]. However, the definition of stuttering and palilalia remains the matter of debate [5]; and it has not been determined whether stuttering or palilalia in PD is pathophysiologically equivalent to that in other diseases. Here, the term, "repetitive speech disorders" collectively describe speech disorders characterized by repetition and/or prolongation presumably due to the dysfunction in controlling rhythm and fluency of speech.

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Benke et al. examined 53 patients with PD and found repetitive speech phenomena in 7% of them in the early stage and 54% in the advanced stage (median disease duration, 5 and 11 years, respectively) [3]. In addition, Goberman et al. reported significant speech disfluency in 53% of their 32 patients with PD without data regarding disease duration [8]. Another clinical investigation on 43 patients with PD and 20 controls reported that repetitive speech disorder correlated with freezing of gait (FOG) and advanced disease stages [4]. In a study on 12 patients with a history of childhood stuttering that remitted and subsequently recurred, stuttering recurred 5.9 (range, 0–21) years after the onset of PD; and patients with a worse motor function tended to exhibit more severe stuttering [6]. Tykalova et al. examined 14 patients with PD before and 3–6 years after initiating dopaminergic medication; 6 patients (43%) developed stuttering-like dysfluency, and the authors found a correlation between the increased occurrence of dysfluency and changes in the levodopa equivalent daily dose (LEDD) [9].

Taken together, repetitive speech disorders tended to appear in the advanced stage and possibly correlated with worse motor function, longer disease duration, or higher LEDD. However, pathophysiological mechanisms of repetitive speech disorders in patients with PD remains unclear. The relatively small sample sizes and potential confounding factors in the previous studies rendered the generalization of the results difficult. Thus, the present study aims to investigate demographic and clinical characteristics of a large number of patients with advanced PD to reveal clinical correlates of repetitive speech disorders.

2. Material and methods

This study was conducted in accordance with the Declaration of Helsinki and the Ethical Guidelines for Medical and Health Research Involving Human Subjects endorsed by the Japanese government. The study protocol was approved by the Ethics Committee of Nagoya University Graduate School of Medicine.

In this retrospective database study, the inclusion criteria were as follows: a) the diagnosis of PD based on the UK Parkinson's Disease Society brain bank criteria [10]; b) the absence of other neurological diseases including stroke; c) no past history of deep brain stimulation (DBS) or ablative stereotactic neurosurgery; and d) the absence of severe cognitive impairment or psychiatric disorders that could hinder the assessment. We enrolled 113 patients who underwent detailed assessments of motor and non-motor functions in order to adjust medication or determine surgical indication. Thus, the participants had relatively long disease duration (52 males; mean age: 64.8 ± 8.7 years; mean disease duration: 12.7 ± 5.9 years). Some of the patients enrolled in this study were those from our previous studies [11–15], and patients were assessed in the on-state under continued medication.

A reading task of the standard passage (The North Wind and the Sun) in Japanese and 1-min conversations were recorded. Then, 3 well-trained and certified speech pathologists independently and blindly rated the speech samples using the Assessment of Motor Speech for Dysarthria (AMSD), which comprises the overall severity of speech disorders (speech intelligibility and speech naturalness) and other specific subscores [16]. These variables are analogous to those developed by Darley et al. [17] Each variable of the AMSD is scored from 0 to 3 (0 = normal, 1 = mild, 2 = moderate, and 3 = severe), except for speech intelligibility and speech naturalness, which are scored from 1 to 5. Higher scores indicate more severe abnormalities. Definitions and interpretations of the variables are summarized in supplementary Table 1. The median value of scores from three raters was used. The inter-rater reliability assessed using Cohen's κ coefficient (R, http://www.r-project.org/) was 0.81, which was considered almost the perfect agreement according to the Landis and Koch classification. Sound repeated score ≥ 1 was considered as significant repetitive speech disorders. Furthermore, Stuttering Severity Instrument-3 (SSI-3) was also used to assess the severity of repetitive speech disorders [18]. Higher scores indicate more severe condition. Of note, behavioral subscores of SSI-3 were excluded because these were not applicable to patients with PD.

In addition, motor function (Unified Parkinson's Disease Rating Scale III [UPDRS-III] and UPDRS-IV), cognitive function (Mini-Mental State Examination [MMSE], Montreal Cognitive Assessment [MoCA], Stroop color-word test, verbal fluency, digit span tests, and line orientation), and activities of daily living (Schwab and England Scale) were assessed. The asymmetry index was calculated as the mean value of the differences between sides (UPDRS III, items 20–26) [19]. Finally, LEDD was calculated as described previously [20].

The demographic and clinical data of patients' groups were compared using independent *t*-tests, Mann–Whitney *U* tests, or χ^2 test, as appropriate. $P < .05$ was considered statistically significant. In addition, univariate and subsequent multiple linear regression analyses were used to assess the correlation between clinical variables and the severity of repetitive speech disorders. Notably, clinical variables with $P < .10$ in the univariate linear regression analysis were included in the subsequent multiple linear regression analysis. Regarding speech variables, we included not speech intelligibility and speech naturalness but the subscores of AMSD to reveal the correlation between stuttering and other aspects of speech disorders in detail. Statistical analyses were performed using the Predictive Analysis Software, version 25 (IBM Inc., Armonk, NY).

3. Results

3.1. Clinical characteristics

Demographic, disease, cognitive, and speech characteristics of the participants are shown in Table 1. Of the 113 patients, 65 patients (57.5%) had repetitive speech disorders. Patients with repetitive speech disorders had significantly worse UPDRS-III ($P = .049$), MoCA ($P = .030$), speech intelligibility ($P < .001$), speech naturalness ($P < .001$), monoloudness ($P = .007$), monopitch ($P = .004$), variable rate ($P = .001$), abnormal rate ($P < .001$), voice tremor ($P = .005$), sound repeated ($P < .001$), hypernasality ($P = .003$), imprecise consonants ($P = .003$), and SSI-3 ($P < .001$) and higher LEDD ($P = .031$) than those without repetitive speech disorders. Remarkably, males were significantly predominant in patients with repetitive speech disorders (64.6%) compared to those without repetitive speech disorders (18.7%; $P < .001$; Fig. 1). No between-group differences were observed in age, years of schooling, handedness, disease duration, asymmetry index, UPDRS IV, S&E scores, MMSE, the Stroop color-word test, verbal fluency (semantic and phonemic), digit span test, and line orientation.

3.2. Linear regression analyses

Table 2 summarizes the results of the univariate linear regression analysis to assess the correlation between SSI-3 and clinical variables. Gender ($P < .001$), disease duration ($P = .079$), UPDRS-III ($P = .032$), LEDD ($P = .077$), MoCA ($P < .001$), monoloudness ($P = .033$), monopitch ($P = .012$), variable rate ($P < .001$), abnormal rate ($P < .001$), voice tremor ($P = .004$), sound repeated ($P < .001$), short rushes of speech ($P = .031$), hypernasality ($P < .001$), and imprecise consonants ($P < .001$) showed $P < .10$. These variables were included in the subsequent multiple linear regression analysis. Sound repeated was excluded from the multiple linear regression analyses because this variable was intrinsically equivalent to SSI-3. In step-wise multiple linear regression analysis, gender ($P < .001$), MoCA ($P = .006$), abnormal rate ($P = .007$), and imprecise consonants ($P = .043$) were considered significant, independent from disease duration, UPDRS III, and LEDD (Table 3).

4. Discussion

The primary findings of this study that investigated clinical

Table 1
Patient characteristics.

	All patients	PD without repetitive speech disorders	PD with repetitive speech disorders	
	n = 113	*n* = 48	*n* = 65	*P* value
Demographic characteristics				
Age (years)	64.8 ± 8.7	65.8 ± 9.1	64.1 ± 8.4	0.187
Gender (male, %)	46.0	18.7	66.2*	< 0.001
Years of schooling	13.4 ± 2.8	13.1 ± 2.5	13.6 ± 2.9	0.627
Handedness (right/left)	107/6	46/2	61/4	0.569
Disease characteristics				
Disease duration (years)	12.7 ± 5.9	12.1 ± 4.3	13.1 ± 6.8	0.954
UPDRS III	17.5 ± 10.3	15.9 ± 10.8	18.8 ± 9.9*	0.049
Asymmetry index	0.2 ± 0.2	0.2 ± 0.1	0.2 ± 0.2	0.443
UPDRS-IV	6.1 ± 3.3	6.5 ± 3.5	5.8 ± 3.1	0.195
S&E on time	83.7 ± 12.7	82.9 ± 13.8	84.4 ± 11.8	0.734
S&E off time	54.0 ± 21.0	56.0 ± 20.4	52.6 ± 21.6	0.456
LEDD (mg)	941.7 ± 375.7	853.5 ± 329.6	1006.9 ± 396.4*	0.031
Cognitive variables				
MMSE	27.4 ± 2.5	27.5 ± 2.4	27.2 ± 2.6	0.623
MoCA	23.4 ± 4.1	24.5 ± 3.2	22.6 ± 4.5*	0.030
Stroop color-word test	18.8 ± 18.4	18.1 ± 17	19.2 ± 19.5	0.540
Verbal fluency (semantic)	15.7 ± 5.6	16.5 ± 5.7	15.0 ± 5.4	0.159
Verbal fluency (phonemic)	9.8 ± 3.6	10.2 ± 3.4	9.5 ± 3.8	0.345
Digit span test	11.3 ± 3.0	11.1 ± 2.4	11.4 ± 3.4	0.976
Line orientation	16.0 ± 3.0	15.7 ± 2.7	16.3 ± 3.2	0.117
Speech variables				
Speech intelligibility	1.8 ± 0.6	1.4 ± 0.5	2.0 ± 0.6*	< 0.001
Speech naturalness	2.7 ± 0.8	2.4 ± 0.7	3.0 ± 0.8*	< 0.001
Monoloudness	1.1 ± 0.8	0.9 ± 0.8	1.3 ± 0.6*	0.007
Monopitch	1.1 ± 0.8	0.9 ± 0.9	1.3 ± 0.6*	0.004
Low volume	0.9 ± 0.8	0.8 ± 0.8	1.0 ± 0.7	0.218
Variable rate	1.1 ± 0.7	0.8 ± 0.6	1.2 ± 0.6*	0.001
Abnormal rate	0.9 ± 0.8	0.5 ± 0.7	1.2 ± 0.9*	< 0.001
Excess loudness variation	1.0 ± 0.7	0.9 ± 0.6	1.0 ± 0.7	0.505
Voice tremor	0.8 ± 0.7	0.6 ± 0.8	1.0 ± 0.6*	0.005
Sound repeated	0.7 ± 0.8	0.0 ± 0.0	1.3 ± 0.5*	< 0.001
Short rushes of speech	0.5 ± 0.7	0.3 ± 0.5	0.6 ± 0.7	0.011
Hypernasality	0.5 ± 0.6	0.3 ± 0.5	0.7 ± 0.7*	0.003
Abnormal pitch level	0.8 ± 0.6	0.8 ± 0.7	0.8 ± 0.6	0.793
Variable pitch	0.3 ± 0.5	0.3 ± 0.5	0.3 ± 0.5	0.507
Imprecise consonants	0.4 ± 0.6	0.2 ± 0.4	0.6 ± 0.7*	0.003
SSI-3	6.9 ± 6.9	0.6 ± 1.8	11.6 ± 5.3*	< 0.001

Values are mean ± SD. P value < .05 was considered significant. * Significant difference between two groups. UPDRS = Unified Parkinson's Disease Rating Scale; S&E = Schwab and England scale; MMSE = Mini-Mental State Examination; MoCA = Montreal Cognitive Assessment; SSI-3 = Stuttering Severity Instrument-3; LEDD = levodopa equivalent daily dose.

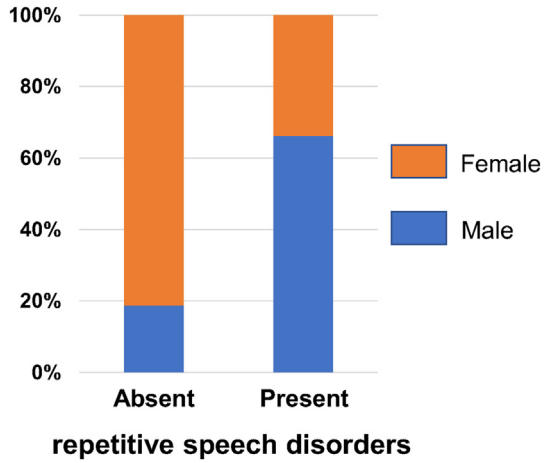

Fig. 1. Percentage of males and females in the patient groups with and without repetitive speech disorders.

correlates of repetitive speech disorders in PD with the largest number of patients to date are as follows: 1) 57.5% of patients with PD had repetitive speech disorders; 2) repetitive speech disorders was significantly associated with male gender, worse MoCA score, and fluency/articulation deficits (abnormal rate and imprecise consonants, respectively). The incidence of repetitive speech disorders corroborated the literature [3,15].

4.1. Gender and repetitive speech disorders in PD

Intriguingly, the multiple linear regression analysis revealed that male gender was the most influential factor for repetitive speech disorders. To the best of our knowledge, no study to date has investigated the gender factor for repetitive speech disorders in patients with PD treated with medication. A retrospective chart-review of 453 patients with PD treated with DBS found that 16 patients (3.5%) developed stuttering after surgery and that 81.3% of the patients who developed stuttering were males [21]. Note that limitations of this study was the retrospective nature and the lack of formal speech assessments. In addition, stuttering after stroke seems to be male-predominant based on a case series and a review of case reports (male/female ratio, 1.90–3.20) [5,22]. Similarly, recent surveys on developmental stuttering have reported male predominance (male/female ratio, 1.34–5.33) [23]. Furthermore, boys with developmental stuttering had lower recovery rates during childhood than girls [23]. Using positron emission tomography (PET), Ingham et al. investigated whether the gender differences in the

Table 2
Univariate analysis of clinical variables associated with repetitive speech disorders.

	B	95% CI		β	*P*-value
Age	−0.059	−0.207	0.090	−0.074	0.437
Gender	−5.491	−7.869	−3.114	−0.398	< 0.001*
Years of schooling	0.217	−0.251	0.685	0.087	0.360
Handedness	2.176	−4.093	8.445	0.065	0.493
Disease duration	0.194	−0.023	0.411	0.166	0.079*
UPDRS-III	0.135	0.012	0.258	0.202	0.032*
Asymmetry index	−2.572	−5.902	0.757	−0.144	0.129
UPDRS-IV	−0.095	−0.503	0.313	−0.047	0.645
S&E on time	0.011	−0.095	0.117	0.021	0.833
S&E off time	−0.026	−0.090	0.037	−0.083	0.416
LEDD	0.003	0.000	0.006	0.167	0.077*
MMSE	−0.386	−0.905	0.133	−0.138	0.144
MoCA	−0.562	−0.863	−0.262	−0.332	< 0.001*
Stroop color-word test	−0.010	−0.081	0.060	−0.028	0.771
Verbal fluency (semantic)	−0.191	−0.422	0.040	−0.155	0.103
Verbal fluency (phonemic)	−0.254	−0.612	0.104	−0.133	0.163
Digit span test	0.024	−0.410	0.457	0.010	0.914
Line orientation	0.061	−0.363	0.486	0.027	0.775
Monoloudness	1.843	0.155	3.531	0.201	0.033*
Monopitch	2.132	0.477	3.786	0.236	0.012*
Low volume	0.549	−1.161	2.259	0.060	0.526
Variable rate	3.780	1.941	5.618	0.361	< 0.001*
Abnormal rate	3.113	1.680	4.546	0.378	< 0.001*
Excess loudness variation	0.495	−1.488	2.478	0.047	0.622
Voice tremor	2.580	0.818	4.343	0.265	0.004*
Sound repeated	7.988	7.145	8.831	0.872	< 0.001*
Short rushes of speech	2.098	0.200	3.996	0.204	0.031*
Hypernasality	4.102	2.235	5.968	0.382	< 0.001*
Abnormal pitch level	0.184	−1.878	2.247	0.017	0.860
Variable pitch	0.462	−2.163	3.086	0.033	0.728
Imprecise consonants	4.576	2.575	6.577	0.395	< 0.001*

UPDRS = Unified Parkinson's Disease Rating Scale; S&E = Schwab and England scale; LEDD = levodopa equivalent daily dose; MMSE = Mini-Mental State Examination; MoCA = Montreal Cognitive Assessment.

Table 3
Multivariate analysis of clinical variables associated with repetitive speech disorders.

	B	95% CI		β	*P*-value
Gender	4.791	2.694	6.889	0.348	< 0.001
MoCA	−0.390	−0.663	−0.117	−0.230	0.006
Abnormal rate	1.906	0.537	3.275	0.232	0.007
Imprecise consonants	2.110	0.073	4.147	0.182	0.043

MoCA = Montreal Cognitive Assessment.

brain metabolism correlated with stuttering; regional correlates of stuttering were typically right-sided in males but bilateral in females [24]. In addition, recent neuroimaging studies on healthy participants have revealed substantial gender differences in functional brain connectivity [25]. Overall, gender differences in the brain network might play a vital role in the development of stuttering. Perhaps, the speech network of males might be susceptible to brain lesions and have weaker compensatory mechanisms than that of females.

4.2. Repetitive speech disorders as a network disorder?

Repetitive speech disorders did not significantly correlate with motor function, disease duration, and LEDD contrary to the findings in the literature [3,6,9]. Because PD patients with long disease duration tend to have worse motor and speech function and higher LEDD, these variables likely have confounding effects on the severity of repetitive speech disorders.

Instead, repetitive speech disorders significantly correlated with the worse global cognitive function (MoCA) and did not significantly corelated with the domain-specific cognitive tests. Similarly, a recent study by Ricciardi et al. showed the relationship between repetitive speech disorders and advanced disease stages independent of disease duration [4]. Most functional imaging studies that have investigated the pathophysiology of repetitive speech disorders enrolled patients with developmental stuttering. Regarding developmental stuttering, the widespread functional and structural brain abnormalities have been primarily reported in the speech-related frontal regions, temporal auditory regions, BG, and their connections [26,27]. To date, only limited consistency has been observed in the description of where or what kind of abnormalities exist. Although this might be partly because of methodological differences, repetitive speech disorders seems to stem from the dysfunction of the BG-cortical speech network rather than dysfunction in one specific brain region; this hypothesis is also supported by the occurrence of stuttering after single unilateral stroke in various brain areas, including the BG, thalamus, white matter, and cortex [5].

4.3. Repetitive speech disorders and hypokinetic dysarthria in PD

The multiple linear regression analysis found significant correlations between repetitive speech disorders and fluency/articulation deficits (abnormal rate and imprecise consonants) and did not find significant correlations between repetitive speech disorders and voice deficits (monoloudness, monopitch, and low volume). These findings suggest that repetitive speech disorders does not develop in parallel with hypokinetic dysarthria and that pathophysiology of repetitive speech disorders is not identical to that of hypokinetic dysarthria.

Hypokinetic dysarthria is a highly complex phenomenon arising from dysfunction in multiple processes, such as scaling and maintaining movement amplitude, preparation and execution of movements, sensory processing, and attention to action [28]. Using functional imaging techniques, abnormal activities in the BG and sensorimotor, prefrontal, and auditory cortices have been demonstrated [29,30]. As mentioned earlier, recent studies suggested that stuttering might result from dysfunction of the BG-cortical speech network rather than dysfunction in one specific brain region [26]. Repetitive speech disorders may occur when normal rhythm of speech cannot be produced due to unsatisfactory compensation for dysfunction of the speech network.

4.4. Other clinical features associated with repetitive speech disorders in PD

In our previous cross-sectional study, 24% of 76 patients with PD who were treated with subthalamic nucleus DBS (STN-DBS) had stuttering as the chief characteristic of speech disorder [12]. Furthermore, our prospective study revealed that during the 1-year follow-up, 13% of patients with PD who were treated with STN-DBS and 18% of medically treated patients with PD developed stuttering. Remarkably, 22% of patients with stuttering experienced a significant improvement in stuttering after discontinuing the stimulation [15]. These findings suggest that repetitive speech disorders is primarily caused by PD itself but could be aggravated by STN-DBS. Previously, regional cerebral blood flow was assessed in a patient who experienced severe deterioration of stuttering under effective STN-DBS using PET [31]; the authors anticipated that a release of overinhibited thalamocortical projections by STN-DBS could cause an additional activation of cortical and cerebellar motor projection areas and an increased functional imbalance in brain regions that were involved in the execution and regulation of fluent speech.

Fasano et al. demonstrated that FOG after STN-DBS with “standard” stimulation parameters (i.e., parameters adjusted for hand function) improved by decreasing the stimulation voltage of the less affected side [32]. In addition, a case report described the improvement in acquired stuttering after unilateral STN-DBS in the left side [33]. Collectively, DBS can ameliorate or aggravate repetitive speech disorders or FOG possibly by changing the balance of the activity between the left and right sides of the brain. Thus, the integration of both hemispheres seems to be critical for executing speech and non-speech actions. Although the

present study did not find the correlation between repetitive speech disorders and asymmetry in motor function, it could be because of medical treatment or compensatory mechanisms. Further studies using imaging techniques, such as PET, functional magnetic resonance imaging (fMRI), or diffusion tensor imaging, are warranted to elucidate the pathophysiology of repetitive speech disorders in patients with PD.

4.5. Limitations

First, this retrospective database study did not assessed the time of onset of repetitive speech disorders and the presence or absence of developmental stuttering. Future prospective studies are warranted to illustrate the timing of emergence of repetitive speech disorders and its correlation with clinical and neuroimaging characteristics. Second, the impact of medication on repetitive speech disorders was not assessed because the evaluation was performed only in the on-medication state. No significant differences were observed in repetitive speech disorders between the on- and off-medication states [3]. Finally, environmental and genetic factors were not assessed in the present study.

5. Conclusions

Approximately 50% of the patients with PD had repetitive speech disorders. Note that our cohort mostly comprised patients with advanced PD. This study determined the following clinical correlates of repetitive speech disorders: male gender, worse cognitive function, and fluency/articulation deficits (abnormal rate and imprecise consonants). Amusingly, male gender was the most influential factor for repetitive speech disorders. Repetitive speech disorders may result from dysfunction of the BG-cortical speech network rather than dysfunction in one specific brain region. Perhaps, the speech network of males might be susceptible to brain damage because of gender differences in the speech network. In addition, worse cognitive function may be associated with more severe functional and pathological abnormality in the brain of patients with PD. Thus, inadequate compensation for the disordered speech network might result in the occurrence of repetitive speech disorders. Finally, the imbalance in the activity between both hemispheres could play an important role in the occurrence of repetitive speech disorders.

Various treatment strategies for hypokinetic dysarthria have been reported [2]. Especially, the efficacy of Lee Silverman Voice Treatment (LSVT LOUD) is well established. However, researches on the treatment of repetitive speech disorders in patients with PD are scarce. Treatment strategies for developmental stuttering, such as rehabilitation programs, biofeedback using real-time fMRI, and repetitive transcranial magnetic stimulation, might be efficacious for repetitive speech disorders in patients with PD. [27] However, considering the progressive nature and the individual diverseness of PD, treatment strategies should be individualized. Thus, further studies are warranted to elucidate the pathophysiology of repetitive speech disorders in PD and establish effective treatment strategies. This might contribute to better understanding of the human speech network and generic neural dysfunction in the various types of dysrhythmic phenomena.

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Relevant conflicts of interests

Nothing to report.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jns.2019.04.012.

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