Acoustical Analysis of Speech in Progressive Supranuclear Palsy

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Summary: Background. Dysarthria often is an early and prominent clinical feature of progressive supranuclear palsy (PSP). Based on perceptual analyses, speech impairment in PSP reportedly consists of prominent hypokinetic and spastic components with occasional ataxic features.

Objective. To measure objectively and quantitatively different speech parameters in PSP as compared with Parkinson's disease (PD) by acoustical analysis and to correlate these parameters with disease duration, global motor, and speech impairment and with the subtype of disease (Richardson's syndrome [RS] vs parkinsonian type of PSP [PSP-P]).

Patients and Methods. Twenty-six patients with clinical diagnosis of PSP (n = 14 classified as RS and n = 12 classified as PSP-P) and 30 age- and gender-matched patients with clinical diagnosis of PD were tested. Speech examination was based on the acoustical analysis of a standardized four-sentence reading task. Several speech variables were measured to assess phonation, intonation variability, speech velocity, and articulatory precision. All participants were tested according to Unified Parkinson's Disease Rating Scale/Motor Score (UPDRS-III) and staged according to Hoehn and Yahr stages. Global speech intelligibility was evaluated on the basis of the UPDRS-III speech item.

Results. In the PSP group, speech velocity, intonation variability, and the fraction of intraword pauses as a measure of articulatory precision were significantly reduced, whereas the percentage of speech pauses was prolonged as compared with the PD group. Only in the male PSP patients, vowel articulation was found to be impaired. Global speech performance was worse in the PSP group in comparison with the PD group and showed a correlation to some distinct speech dimensions. No differences of speech variables were seen between RS and PSP-P patients.

Conclusions. PSP patients feature a mixed type of dysarthria with hypokinetic and spastic components that differ significantly from the speech performance of PD speakers. This probably reflects the widespread neuropathological changes in PSP comprising basal ganglia as well as pontine and further brainstem regions.

Key Words: PSP–Progressive supranuclear palsy–PD–Parkinson's disease–Dysarthria–Speech impairment–Acoustical analysis.

INTRODUCTION

First described by Richardson[1,2] in 1963, progressive supranuclear palsy (PSP) has been clinically defined as a progressive neurodegenerative syndrome of postural instability, axial rigidity, supranuclear gaze palsy, mild dementia, and pseudobulbar palsy with consistent pathological findings defined by an accumulation of tau protein and neuropil threads, mainly in the pallidum, subthalamic nucleus, red nucleus, substantia nigra, pontine tegmentum, striatum, oculomotor nucleus, medulla, and dentate nucleus.[3,4] Although the classical PSP syndrome presents with clear clinical signs in its later stages, several clinical variants have been identified that are less distinctive. Therefore, many patients are initially thought to suffer from Parkinson's disease (PD) or multiple system atrophy.[5] Recently, the classic clinical presentation of PSP has been referred to as "Richardson's syndrome" (RS) to distinguish it from the clinical variants of "PSP-parkinsonism" (PSP-P) and "PSP-pure akinesia with gait freezing." In the latter ones, the underlying tau pathology is less severe and features a more restricted distribution.[6–8] Moreover, there is an overlap to other clinically defined entities, such as PSP-corticobasal syndrome and PSP-progressive nonfluent aphasia, in which the cortical involvement of tau pathology is more severe.[9,10] The heterogeneity of PSP has recently been examined in detail in postmortem series to define pathological surrogate parameters based on the extent and pattern of tau pathology to distinguish RS from PSP-P.[11,12] Differences were most striking in the cerebral cortex, pons, caudate, and some cerebellar regions, whereas the subthalamic nucleus and substantia nigra were affected similarly both in RS and PSP-P.[11,12] The involvement of brainstem and midbrain structures in both subtypes of PSP probably influences the clinical manifestation of dysarthria, which is often an early and prominent symptom of PSP[13] and had already been described by Richardson et al[1] as pseudobulbar palsy. However, there are only few and merely descriptive reports on the pattern and degree of dysarthria in PSP and its subtypes based on qualitative descriptions and perceptual analyses.[14–18] In the largest of these series, the authors had found dysarthria being present in all 44 PSP patients examined with variable degree of spastic, hypokinetic, and ataxic components with further appearance of dysfluencies, such as stuttering or palilalia in a subgroup of patients.[16] The finding of "mixed dysarthria" in PSP patients has been attributed to the widespread neuropathological changes in PSP,[16,17] although patients had not been subclassified as suffering from RS or PSP-P because those phenotypes had not yet been established. In one neuropathological study, the severity of the hypokinetic component of dysarthria was correlated to the degree of neuronal loss and gliosis in the

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TABLE 1.
Clinical Characteristics of PSP Patients (PSP-P and RS)

	PSP-P (n = 12; 7 Male, 5 Female)	RS (n = 14; 10 Male, 4 Female)	Comparison: PSP-P vs RS
Age (yr)	Mean: 71.50, SD: 3.61 Median: 71.5 Range: 65–77	Mean: 72.50, SD: 6.39 Median: 72.5 Range: 59–81	NS
Duration (yr)	Mean: 3.50, SD: 2.50 Median: 3 Range: 1–8	Mean: 2.57, SD: 2.45 Median: 2 Range: 1–5	NS
UPDRS-III	Mean: 30.00, SD: 10.03 Median: 28 Range: 15–50	Mean: 26.29, SD: 11.38 Median: 22.5 Range: 12–45	NS
Item 18/speech	Mean: 2.00, SD: 1.04 Median: 2 Range: 1–4	Mean: 1.93, SD: 0.92 Median: 2 Range: 1–4	NS
Hoehn & Yahr	Mean: 2.79, SD: 0.54 Median: 3 Range: 2–4	Mean: 2.86, SD: 0.72 Median: 2 Range: 1–3	NS

substantia nigra pars compacta.[17] This observation could account for some overlap with the hypokinetic-rigid dysarthria of PD.[18] Based on the perceptual analysis of a large sample of dysarthric speakers, Darley et al[19,20] primarily defined a salient cluster of deviant speech dimensions in Parkinsonian dysarthria, including a harsh breathy voice quality, reduced variability of pitch and loudness, reduced stress, imprecise consonant articulation, and short rushes of speech interrupted by inappropriate periods of silence.

The aim of this study was to carry out a standardized acoustical analysis of speech in PSP patients with the clinical presentation of RS and PSP-P in comparison with PD patients to obtain objective speech parameters and quantitative data, which might be helpful to establish salient patterns of dysarthria in PSP and to further distinguish PSP from PD.

PATIENTS AND METHODS

From 2007 to 2009, 26 consecutive patients (17 male and 9 female) with the clinical diagnosis of probable PSP and 30 patients with idiopathic PD (16 male and 14 female) were recruited for the study. All participants were scored according to Hoehn and Yahr (H & Y) stage; global motor performance was tested according to Unified Parkinson's Disease Rating Scale/Motor Scale (UPDRS-III) before the speech test. Item 18 of the UPDRS-III ("speech") was taken for perceptual description of patients' speech. All participants were tested in the morning 1 hour after the last admission of medication.

At the time of study inclusion, all PSP patients featured falls because of postural instability and supranuclear gaze palsy and/or slowed vertical saccades fulfilling the National Institute of Neurological Disorders and Stroke PSP clinical diagnosis criteria.[21] PSP had been diagnosed from 1 to 8 years before this investigation (mean: 3/median: 2 years). H and Y scores ranged from two to four (mean: 2.83/median: 3). UPDRS-III scores ranged from 12 to 50 points (mean: 28/median: 25 points). Based on their medical history, 12 patients of this series (7 male and 5 female) were to be diagnosed as PSP-P with moderate levodopa response at least in the early stages of the disease, mild irregular jerky tremor in seven of 12 patients, bradykinesia and moderate limb rigidity, and the occurrence of falls and oculomotor abnormalities not before the first 2 years.[4,6] The other 14 patients (10 male and 4 female) were to be diagnosed as RS with the manifestation of falls, gaze palsy, and cognitive decline within the first 2 years.[4,6,22]

In the PSP group, medication comprised varying doses of levodopa alone or in combination with different dopamine agonists and/or amantadine. None of the PSP patients received inhibitors of monoamine oxidase B (MAO-B) or inhibitors of catechol-o-methyltransferase (COMT), anticholinergic drugs, clozapine, or other neuroleptics. PSP patients' characteristics are summarized in Table 1.

Diagnosis of PD was based on UK Parkinson's Disease Society Brain Bank criteria.[23] PD patients' age ranged from 65 to 80 years (mean: 72.3/median: 72 years). PD had been diagnosed from 1 to 8 years before this investigation (mean: 3.27/median: 2.5 years). H and Y score ranged from 1.5 to three (mean: 2.18/median: 2). UPDRS-III ranged from five to 42 points (mean: 20.73/median: 21 points). All PD patients were on stable dopaminergic medication for at least 3 weeks consisting of levodopa and different dopamine agonists; none of the patients received amantadine, MAO-B inhibitors, COMT inhibitors, anticholinergic drugs, clozapine, or other neuroleptics. None of the patients experienced orofacial or abdominothoracic peak-dose dyskinesia or suffered from hypokinetic motor fluctuations. Signed informed consent according to a positive *votum* of the ethic committee of the Ruhr University was obtained from all participants.

Each participant had to perform a speech task consisting of a standard reading passage composed of four complex sentences. Participants were allowed to run through several exercise courses of the reading task before the definite speech recording to eliminate reading difficulties. Speech samples were digitally recorded in a quiet room using a commercial audio software (*Steinberg WaveLab* [Steinberg, Hamburg, Germany]) and