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Distinct patterns of imprecise consonant articulation among Parkinson's disease, progressive supranuclear palsy and multiple system atrophy

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ABSTRACT

Distinct speech characteristics that may aid in differentiation between Parkinson's disease (PD), progressive supranuclear palsy (PSP) and multiple system atrophy (MSA) remain tremendously under-explored. Here, the patterns and degree of consonant articulation deficits across voiced and voiceless stop plosives in 16 PD, 16 PSP, 16 MSA and 16 healthy control speakers were evaluated using acoustic and perceptual methods. Imprecise consonant articulation was observed across all Parkinsonian groups. Voice onset time of voiceless plosives was more prolonged in both PSP and MSA compared to PD, presumably due to greater severity of dysarthria and slower articulation rate. Voice onset time of voiced plosives was significantly shorter only in MSA, likely as a consequence of damage to cerebellar structures. In agreement with the reduction of pre-voicing, MSA manifested increased number of voiced plosives misclassified as voiceless at perceptual evaluation. Timing of articulatory movements may provide important clues about the pathophysiology of underlying disease.

1. Introduction

Idiopathic Parkinson's disease (PD) is a common neurological disorder characterized by the progressive loss of dopaminergic neurons in the substantia nigra pars compacta, affecting 1.6% of persons over the age of 65 years (deRijk et al., 1997). Dopamine concentrations have been shown to be significantly reduced before distinct motor deficits become apparent (Hornykiewicz, 1998). The cardinal signs of PD, often referred to as Parkinsonism, include resting tremor, bradykinesia, muscular rigidity and postural instability. Other neurodegenerative diseases that go beyond the signs and symptoms of Parkinsonism are known as atypical Parkinsonian syndromes (APS). Progressive supranuclear palsy (PSP) and multiple system atrophy (MSA) are the most common APS, with an estimated prevalence of 30–40 per 100,000 among persons older than 65 years (Schrag, Ben-Shlomo, & Quinn, 1999). Characteristic clinical features of PSP include supranuclear gaze palsy, frequent falls, bradykinesia, axial rigidity, cognitive decline and communication disorders (Nath, Ben-Shlomo, Thomson, Lees, & Burn, 2003; Steele, Olszewski, & Richardson, 1964), reflecting widespread neurodegeneration involving the midbrain as well as the globus pallidus, striatum, hypothalamic nucleus, pons, superior cerebellar peduncle and cerebellar dentate nucleus (Nath et al., 2003). Conversely, MSA manifests by various combinations of autonomic, cerebellar and Parkinsonian features (Wenning, Colosimo, Geser, & Poewe, 2004), corresponding to degeneration of the cerebellum, middle cerebellar peduncle, striatum, substantia nigra, inferior olivary nucleus and pons (Gilman et al., 2008). APS differ from PD by poor levodopa response and more rapid disease progression resulting in shorter life expectancy (O'Sullivan et al., 2008; Wenning, Litvan, & Tolosa, 2011). Furthermore, the underlying pathophysiology differs as PD and MSA are α-synucleinopathies while PSP is a tauopathy. However, the differentiation between PD and both PSP and MSA can be challenging as the initial signs are frequently nonspecific and overlap those of PD (Osaki et al., 2004; Schrag et al., 1999).

1.1. Speech impairment in PD, PSP and MSA

Dysarthria is a well-recognized clinical manifestation of Parkinsonian disorders, developing in 90–100% of patients with PD, PSP and MSA during the course of the disease (Ho, Iansek, Marigliani, Bradshaw, & Gates, 1998; Kluin, Foster, Berent, & Gilman, 1993;

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Kluin, Gilman, Lohman, & Junck, 1996; Muller et al., 2001; Rusz et al., 2015). Speech impairment is an early and prominent manifestation that can contribute primarily to the diagnosis of PSP (Goetz, Leurgans, Lang, & Litvan, 2003; Kim & McCann, 2015; Wenning et al., 2011), but has also been largely documented in the early stages of PD and MSA (Huh et al., 2015; Kim, Kent, Kent, & Duffy, 2010; Rusz, Cmejla, Ruzickova, & Ruzicka, 2011).

Due to dysfunction of the basal ganglia, the majority of PD patients manifest hypokinetic dysarthria characterized by monopitch, monoloudness, reduced stress, variable rate, imprecise articulation, harsh voice quality, speech dysfluencies and inappropriate silence (Darley, Aronson, & Brown, 1969b; Ho et al., 1998). Conversely, PSP and MSA patients typically evolve mixed dysarthria with a combination of hypokinesia, ataxia and spasticity as a result of more widespread neuronal atrophy (Kluin et al., 1993, 1996; Rusz et al., 2015). Indeed, previous studies (Kluin et al., 1993, 1996) investigating 46 MSA and 44 PSP patients using oral motor and perceptual speech analysis have reported mixed dysarthria with combinations of all hypokinetic, spastic and ataxic components in two-thirds of APS patients. Hypokinetic components followed by ataxic components were predominant in MSA patients, while spastic components were mostly present in PSP patients (Kluin et al., 1993, 1996).

Considering individual speech aspects, only the occurrence of stuttering-like behaviour has been reported to be distinctive for PSP as compared to MSA (Kluin et al., 1993, 1996; Rusz et al., 2015). A small number of studies have also focused on an objective description of the dysarthria profile in APS in comparison to PD (Huh et al., 2015; Kim et al., 2010; Rusz et al., 2015; Sachin et al., 2008; Skodda, Visser, & Schlegel, 2011). In general, these studies have shown that the impairment of specific speech dimensions is more pronounced in APS than in PD (Huh et al., 2015; Rusz et al., 2015; Skodda et al., 2011). Dysarthria in PSP has been reported to be characterized by stuttering-like behaviour, reduced speech rate, decreased intonation variability, prolonged pauses, articulation imprecision and poor quality of voice (Rusz et al., 2015; Skodda et al., 2011), whereas MSA patients have been said to manifest with excess pitch fluctuations, excess intensity variations, increased voice pitch, reduced speech rate, prolonged phonemes, vocal tremor, voice perturbations and slow variable alternating motion rates (Huh et al., 2015; Kim et al., 2010; Rusz et al., 2015; Saxena, Behari, Kumaran, Goyal, & Narang, 2014). However, little effort has been made to investigate consonant articulation in APS.

1.2. Consonant articulation in PD, PSP and MSA

The description of disturbed consonant articulation in various diseases has typically been based on perceptual assessment in subgroups of patients defined by dysarthria subtype such as spastic, ataxic or hypokinetic, rather than by disease aetiology (i.e., PD, PSP or MSA; (Chakraborty, Roy, Hazra, Biswas, & Bhattacharya, 2008; Darley, Aronson, & Brown, 1969a; Hartelius, Gustavsson, Astrand, & Holmberg, 2006; Logemann & Fisher, 1981; Weismer, 1984). Furthermore, previous studies were limited primarily to documenting the occurrence of articulation deficits and did not describe specific features characterizing imprecise consonants (Chakraborty et al., 2008; Darley et al., 1969a; Hartelius et al., 2006). In particular, in the classic study by Darley et al. (1969b), imprecise consonant articulation was perceptually found to be one of the most deviant speech dimensions in PD. The presence of imprecise consonant articulation has also been perceptually revealed in a cohort of MSA and PSP patients (Hartelius et al., 2006). Interestingly, although in general speech deviation of greater severity was found in PSP, consonant articulation was more severely affected in MSA (Hartelius et al., 2006).

With regard to acoustic analyses, several measurements can be used for description of consonants including various measures of duration, formant transitions, spectral moments or energy-based measures (Kent & Read, 1992). Among them, voice onset time (VOT) determined for stop plosives is perhaps the most frequently used parameter and a relatively large amount of data has been published on VOT in PD patients. Unfortunately, previous studies have provided rather contradictory findings. While some researchers have reported increased VOT duration (Forrest, Weismer, & Turner, 1989; Novotny, Rusz, Cmejla, & Ruzicka, 2014), others have observed unchanged (Fischer & Goberman, 2010; Ravizza, 2003) or even decreased VOT (Flint, Black, Campbelltaylor, Gailey, & Levinton, 1992) in PD subjects. It has been suggested that these discrepancies may be due to the fact that the measurement of VOT is dependent on speaking rate (Volaitis & Miller, 1992); however, VOT ratio, a rate-independent variation of VOT, did not clarify these ambiguous findings (Fischer & Goberman, 2010; Novotny et al., 2014).

Only one previous study has focused on the acoustic investigation of consonant characteristics for five categories of plosives in PD, PSP, and MSA in comparison to controls (Saxena et al., 2014). However, this study provided rather inconsistent findings across various consonant categories and speaker groups (Saxena et al., 2014). In particular, the authors revealed no significant alterations of VOT duration in dentals across all groups, but observed increased VOT duration of velars in PD, palatals in PSP, bilabials in MSA, PSP and PD, and of retroflexes in PSP and MSA (Saxena et al., 2014). However, a direct comparison of consonant articulation between PD, PSP and MSA has never been performed.

1.3. Aim of the present study

The aim of the current study was therefore to investigate the patterns and degree of consonant articulation deficits across different voiceless and voiced stop plosives in PD, PSP, MSA and healthy speakers using objective acoustic measures to help elucidate distinct speech characteristics that could aid in the differentiation between various forms of Parkinsonism. In addition, perceptual examination of phonetic contrast between voiceless and voiced plosives was performed to determine if consonant imprecision was a notable feature of dysarthria in PD, PSP and MSA. Additionally, the relationships between speech performances and clinical manifestations were explored to provide greater insight into the pathophysiology of speech production in PD, PSP and MSA.

2. Methods

2.1. Participants

From 2011 to 2015, a total of 48 consecutive patients including 16 fulfilling the diagnostic criteria for idiopathic PD (5 men and 11 women), 16 with a diagnosis of probable PSP (11 men and 5 women) and 16 with a diagnosis of probable MSA (5 men and 11 women) were recruited. Among APS, hereafter hypernym for the MSA and PSP subgroups, 13 PSP patients were diagnosed with PSP-Richardson syndrome, 2 with PSP-Parkinsonism and 1 with PSP-pure akinesia with gait freezing, whereas MSA patients were diagnosed with the MSA-Parkinsonian subtype in 14 cases and the MSA-cerebellar subtype in 2 cases. The clinical diagnoses of all patients were established by a specialist in movement disorders (JK) according to the UK Parkinson's Disease Society Bank Criteria for PD (Hughes, Daniel, Kilford, & Lees, 1992), the NINDS-PSP clinical diagnostic criteria for PSP (Litvan et al., 1996) or the consensus diagnostic criteria for MSA (Gilman et al., 2008). At the time of the examination, all patients treated pharmacologically were on stable