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Speech Motor Profiles in Primary Progressive Aphasia

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ABSTRACT

Purpose: Previous research on motor speech disorders (MSDs) in primary progressive aphasia (PPA) has largely focused on patients with the nonfluent/agrammatic variant of PPA (nfvPPA), with few systematic descriptions of MSDs in variants other than nfvPPA. There has also been an emphasis on studying apraxia of speech, whereas less is known about dysarthria or other forms of MSDs. This study aimed to examine the qualitative and quantitative characteristics of MSDs in a prospective sample of individuals with PPA independent of subtype.
Method: We included 38 participants with a root diagnosis of PPA according to current consensus criteria, including one case with primary progressive apraxia of speech. Speech tasks comprised various speech modalities and levels of complexity. Expert raters used a novel protocol for auditory speech analyses covering all major dimensions of speech.
Results: Of the participants, 47.4% presented with some form of MSD. Individual speech motor profiles varied widely with respect to the different speech dimensions. Besides apraxia of speech, we observed different dysarthria syndromes, special forms of MSDs (e.g., neurogenic stuttering), and mixed forms. Degrees of severity ranged from mild to severe. We also observed MSDs in patients whose speech and language profiles were incompatible with nfvPPA.
Conclusions: The results confirm that MSDs are common in PPA and can manifest in different syndromes. The findings emphasize that future studies of MSDs in PPA should be extended to all clinical variants and should take into account the qualitative characteristics of motor speech dysfunction across speech dimensions.
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Primary progressive aphasia (PPA) refers to a class of rare dementia syndromes characterized by progressive language impairment (Mesulam, 2013). According to current consensus criteria, PPA is classified into three main variants, which are defined by distinct patterns of impairment across different linguistic and phonetic domains (Gorno-Tempini et al., 2011): nonfluent/agrammatic variant (nfvPPA), semantic variant (svPPA), and logopenic variant (lvPPA). However, patients with a root diagnosis of PPA often cannot be clearly assigned to one of the clinically defined subtypes because they do not meet all the criteria required for classification (unclassifiable PPA [PPA-U]; Utianski et al., 2019).

The most common cause of nfvPPA and svPPA is neuropathology of frontotemporal lobar degeneration (FTLD), whereas lvPPA is primarily associated with

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Alzheimer's disease (AD) pathology (Montembeault et al., 2018). Clinically and pathologically, there is overlap between PPA and other neurodegenerative syndromes. As the disease progresses, patients with nfvPPA may develop corticobasal syndrome (CBS) or progressive supranuclear palsy syndrome (PSPS; Boeve, 2007; Josephs et al., 2006; Rohrer et al., 2010). Moreover, nfvPPA and svPPA have been described in association with motor neuron disease (MND; Caselli et al., 1993; Catani et al., 2004; Vinceti et al., 2019).

Involvement of the Speech Motor System in PPA

Besides language impairment, PPA can also affect the speech motor control system that is inextricably intertwined with the language system (Duffy et al., 2014). Motor speech disorders (MSDs) associated with PPA comprise apraxia of speech and different dysarthria syndromes as well as mixed forms thereof (Duffy et al., 2014; Poole et al., 2017). Apraxia of speech is ascribed to a dysfunction of speech motor planning processes. Dysarthria syndromes are considered to result from various pathologies afflicting the control and execution of speech movements (e.g., spastic dysarthria following damage to the upper motor neuron system, hypokinetic dysarthria associated with basal ganglia dysfunction). In a recent study, Staiger, Schroeter, Ziegler, et al. (2021) have also observed neurogenic stuttering and impoverished speech drive (adynamic speech) as forms of MSD in PPA.

In primary progressive apraxia of speech (PPAOS), apraxia of speech represents the sole or clearly dominant manifestation of the progressive disease (Duffy et al., 2021). According to the international consensus guidelines (Gorno-Tempini et al., 2011), the condition is generally compatible with a diagnosis of nfvPPA. However, since patients show no or only equivocal aphasia, PPAOS is now commonly distinguished from PPA and recognized as a separate disease entity (Duffy et al., 2021). PPAOS is strongly associated with FTLD-tau pathology and typically also evolves into one of Parkinson-plus syndromes (PSPS, CBS) as the disease progresses (Josephs et al., 2006; Kwon et al., 2022; Seckin et al., 2020).

Impairments of speech motor functions may already manifest at early stages of the disease (Duffy et al., 2014). By affecting intelligibility, speech naturalness, and speech motor efficiency, MSDs can significantly compromise a person's verbal communication skills beyond aphasic impairment. In extreme cases, the impairments can lead to mutism (Caso et al., 2014; Gorno-Tempini et al., 2006). MSDs are also among the common and prominent symptoms of the typical manifestations of PSPS, CBS, and MND (Blake et al., 2003; Clark et al., 2021; Kluin et al., 1993; Tomik & Guiloff, 2010). Thus, MSDs form a key intersection between PPA and the closely related motor syndromes mentioned above. In recent years, studies have shown that the recognition of MSDs in PPA can contribute to the prediction of the underlying neuropathology and disease progression (e.g., Josephs & Duffy, 2008; Santos-Santos et al., 2016). For the above reasons, in-depth knowledge of MSDs in PPA seems essential for advancing the theoretical understanding of the diseases and for implementing appropriate treatment interventions.

Occurrence of MSDs in Subtypes of PPA

MSDs are most commonly associated with the nonfluent subtype. "Effortful, halting speech with inconsistent speech sound errors (apraxia of speech)" is among the core criteria for nfvPPA (Gorno-Tempini et al., 2011). In a comprehensive meta-analysis, Duffy et al. (2014) found prevalence rates for apraxia of speech in nfvPPA ranging from 17% to 100%, with a median prevalence of 78%. Recent studies by Cordella et al. (2019) and Staiger, Schroeter, Ziegler, et al. (2021) have found prevalence rates of 41% and 28%, respectively. For dysarthria, Duffy et al. (2014) reported a median prevalence of 36% in nfvPPA. A comparable proportion (33%) was also observed by Staiger, Schroeter, Ziegler, et al. (2021).

In contrast, MSDs are considered much less common in svPPA and lvPPA. The consensus guidelines list *intact* speech motor function among the (noncore) criteria for the two subtypes, although the presence of an MSD is also not necessarily an exclusion criterion (Gorno-Tempini et al., 2011). In fact, speech motor deficits have been observed in cases of lvPPA (e.g., Croot et al., 2012; Duffy et al., 2014; Duncan et al., 2020) and svPPA (e.g., Agosta et al., 2010; Duncan et al., 2020; Kertesz et al., 2010). In Staiger, Schroeter, Ziegler, et al. (2021), 27% of the individuals with svPPA showed signs of an MSD (dysarthria, neurogenic stuttering, impoverished speech drive). In their lvPPA group, almost 30% of the individuals presented with speech motor deficits (dysarthria, apraxia of speech, neurogenic stuttering, impoverished speech drive). For a discussion of these results, see Staiger, Schroeter, Ziegler, et al. (2021).

Prevalence estimates for MSDs in the different variants of PPA, however, are fraught with the difficulty that motor speech (dys)function is itself among the criteria on which, according to the current gold standard, subtyping of PPA is based (Gorno-Tempini et al., 2011). In certain constellations, the presence of an MSD may preclude a classification as logopenic or semantic type even if all mandatory criteria and most noncore criteria are otherwise met. Mild speech motor symptoms in particular may also be more easily overlooked if they are considered

unlikely given the overall syndrome picture of a PPA (e.g., in cases in which features of a semantic-type PPA predominate). Thus, it cannot be excluded that existing evidence on the occurrence of MSDs in PPA is biased.

Qualitative Manifestations of MSDs in PPA

A number of studies have a priorly included acoustic and/or perceptual parameters indicative of motor speech dysfunction, essentially with the aim of discriminating (statistically) between single PPA subtypes or revealing associations between different aspects of speech production and the distribution of areas of atrophy or underlying pathology. Features that have shown to be strongly associated with nfvPPA include segmental speech errors, particularly phonetic distortions (Ash et al., 2013; Croot et al., 2012; Haley et al., 2021; Leyton et al., 2011; Wilson et al., 2010) and suprasegmental abnormalities such as increased syllable durations, reduced articulation rate, or equal stress (Ballard et al., 2014; Cordella et al., 2019; Croot et al., 2012; Duffy et al., 2017; Garcia et al., 2022; Leyton et al., 2011). However, none of these studies aimed to classify MSD types or describe the overall speech motor profiles of individuals with PPA.

Several studies have been specifically devoted to the clinical description of apraxia of speech, either in association with progressive aphasia or as the sole or clearly dominant manifestation of the progressive disease (PPAOS). In a seminal study, Duffy (2006) conducted a comprehensive retrospective evaluation of medical record findings from 80 patients diagnosed with progressive apraxia of speech. Perceptual characteristics included features of aberrant sound structure (distorted sound substitutions*, distorted sound additions, sound sequencing errors*, more errors with increased utterance length*) and rhythm and fluency (slow rate*, syllable segmentation and equal stress*, sound prolongations, sound repetitions), as well as further features (articulatory groping, effortful orofacial movements during speech, reduced words per breath group during phrase/sentence production relative to maximum vowel duration).[1] These characteristics later informed the development of the Apraxia of Speech Rating Scale (ASRS), a scale used to quantify the presence and severity of apraxia of speech, particularly in neurodegenerative disease (Strand et al., 2014; Utianski, Duffy, Clark, Strand, Boland, et al., 2018).[2] Meanwhile, the articulatory and prosodic feature inventory has been used in many studies to describe apraxia of speech in PPA or PPAOS samples (Botha et al., 2015; Bouvier et al., 2021; Cordella et al., 2019; Duffy et al., 2017; Josephs et al., 2006, 2012; Matias-Guiu et al., 2019; Utianski, Duffy, Clark, Strand, Botha, et al., 2018). Apparent interindividual differences have recently prompted the proposal of different subtypes of progressive apraxia of speech: a phonetic type with predominant speech sound errors, a prosodic type characterized predominantly by slow and segmented speech, and a "mixed" phonetic–prosodic type (Duffy et al., 2021; Josephs et al., 2013; Utianski, Duffy, Clark, Strand, Botha, et al., 2018).

Knowledge about dysarthric impairment or other neurogenic speech disorders in PPA or PPAOS remains sparse. As far as the presence of dysarthria is considered in studies, descriptions are largely limited to severity ratings or the specification of the dysarthria syndrome. Studies suggest that in nfvPPA, spastic and hypokinetic or mixed spastic–hypokinetic forms are prevalent (Duffy et al., 2014; Poole et al., 2017). These dysarthria syndromes also commonly occur in PSPS and CBS, syndromes that clinically and pathologically overlap with nfvPPA, as mentioned above (Boeve, 2007). Dysarthrias are rarely described in lvPPA and, if at all, most likely characterized as hypokinetic (Duffy et al., 2014; Staiger, Schroeter, Ziegler, et al., 2021). Very rarely, dysarthria is described in svPPA. Recent studies, however, have shown that patients with svPPA with MND (svPPA-MND) may develop moderate-to-severe dysarthria (Tan et al., 2019; Vinceti et al., 2019). To our knowledge, more detailed descriptions of dysarthria in this population are still pending. However, due to the involvement of the motor neuron system, paretic forms (spastic/flaccid) seem most likely. Occasional cases of paretic dysarthria or hypokinetic dysarthria in speakers with svPPA were also observed by Staiger, Schroeter, Ziegler, et al. (2021).

A few earlier studies provided impressionistic descriptions of speech motor features in individual cases with "progressive language decline" that are strongly suggestive of dysarthric impairment (Chapman et al., 1997; Cohen et al., 1993; Didic et al., 1998; Kempler et al., 1990). However, we identified only three studies in which descriptions of speech motor skills in PPA or PPAOS systematically included dimensions of dysarthria. First, Thompson et al. (1997) provided a systematic perceptual analysis of speech features indicative of dysarthria and apraxia of speech in four participants with PPA. The selection included the variables consonant production, articulatory agility, resonance, vocal quality, pitch, loudness, rate, prolonged pauses, and intelligibility. Only one participant was diagnosed with

[1]Asterisks indicate occurrence in at least 50% of the included patients. Only features observable in natural language contexts (connected speech, word and sentence repetition tasks) are listed here. Further speech tasks considered in Duffy (2006) include alternating motion rates (e.g., rapid repetition of "puhpuhpuh") and sequential motion rates (e.g., rapid repetition of "puhtuhkuh").

[2]Current version: ASRS-3 (Utianski, Duffy, Clark, Strand, Botha, et al., 2018).

mild dysarthria at 4 years after disease onset, which worsened as the disease progressed. The authors described the patient's speech as characterized by decreased articulatory agility and rate as well as, at later stages, prolonged pauses and reduced intelligibility.[3]

Second, Ogar et al. (2007) conducted analyses of speech motor deficits in individuals with nfvPPA based on the Motor Speech Evaluation (Wertz et al., 1984). They selected 10 speech features considered typical of apraxia of speech and spastic or hypokinetic dysarthria and assessed their presence or absence in 16 participants. Of the features particularly characteristic of dysarthria, hypernasality occurred most frequently among participants. Breathiness and hypophonia occurred in two patients each. In one case, voice quality was classified as strained–strangled. However, the features may not cover the entire spectrum of the patient's speech impediments, as the list of features was limited by design.

Third, Bouvier et al. (2021) have recently reported four individual cases of patients with PPAOS, two of the prosodic type and two of the mixed type at baseline. In their in-depth description of speech motor performance based on perceptual and acoustic assessment, the authors also considered clinical signs of dysarthria. Dimensions of impaired speech functioning most reliably attributed to dysarthria included disorders of voice quality, voice stability, speech breathing, and resonance. All subjects showed at least mild phonatory deficits, which increased during the course of the disease. Voice quality was described as strained and breathy in most cases. Other phonatory characteristics included difficulties with voice initiation and control of pitch and loudness, as well as occasional aphonia. Three patients showed deficits in respiratory–phonatory control, which manifested in various symptoms described as, for example, discoordinated respiratory groups, shortness of breath, or use of residual air at the end of speech runs. Two patients had mild hypernasality ascribed to fatigue and task complexity. Other features included word or syllable repetitions at speech onset in one patient and the emergence of vocal tics in another.

The studies cited all deal exclusively with nfvPPA or, as in Bouvier et al. (2021), with PPAOS. None of the previous studies, however, detailed the overall speech motor profiles independent of the subtype of the progressive disease. The aim of this study was to provide an unbiased and comprehensive evaluation of all speech motor domains in a cohort of German-speaking patients with a core diagnosis of PPA (or PPAOS) regardless of subtype.

[3]Note that this list of symptoms alone does not provide clear evidence for the presence of dysarthria, as the combination of symptoms would also largely be consistent with apraxia of speech.

Materials and Method

This study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committees of the medical faculty of the Technische Universität München, Germany, and the medical faculty of the Universität Leipzig, Germany. Informed consent was obtained from all subjects.

Participants

Participants were recruited from the study centers in Munich (Center for Cognitive Disorders, Department of Psychiatry and Psychotherapy, Technische Universität München, Germany) and Leipzig (Clinic for Cognitive Neurology, University Hospital Leipzig, Germany), both of which specialize in patients with PPA. Patients were proposed for participation in the study by their treating neurologists/neuropsychiatrists. Inclusion criteria were as follows: (a) a neurological diagnosis of PPA according to the root criteria proposed by Gorno-Tempini et al. (2011), with the possible inclusion of cases with apraxia of speech as the sole manifestation of the progressive disease (PPAOS; Duffy et al., 2021); (b) German as the native language; (c) intact or corrected vision, hearing, and dentition based on clinical observation and the participants' self-report; and (d) sufficient physical capacity and cognitive ability to participate in the examination. Exclusion criteria were as follows: (a) the presence of further neurological diseases (e.g., stroke), (b) known non-neurologic diseases affecting the oral–pharyngeal–laryngeal apparatus and the respiratory system, and (c) preexisting speech and language disorders. Brain imaging (magnetic resonance imaging; computerized tomography in one case) was used to rule out other structural lesions that might explain aphasia in all but one participant (P31), who declined to undergo brain imaging. The presence of an MSD was not an inclusion criterion.

Thirty-eight individuals met the above criteria (18 women, 20 men). Two participants (P03 and P20) with isolated speech and language impairment in the early stage of their disease had developed CBS as the disease progressed. Their condition had been classified as PPA-CBS at the time of study examination. All participants underwent language assessment, performed within the same 3-month interval as the speech motor examination reported in this study. Sufficient data were collected to examine all consensus-proposed domains using clearly defined criteria and norm data from established assessment tools in German (Staiger, Schroeter, Müller-Sarnowski, et al., 2021), as follows:

- **Aachen Aphasia Test** (AAT; Huber et al., 1983). Subtests of the AAT were used to check the following consensus criteria (Gorno-Tempini et al., 2011): sentence repetition, confrontation naming, and single-word

and sentence comprehension. According to the AAT scoring guidelines for spontaneous speech, statements were made about syntactic structure (agrammatism), semantic structure (word retrieval), and phonological structure (phonemic errors). The reading and writing subtests of the AAT allowed for the identification of written language impairments. However, they did not provide reliable information about the underlying deficit, such as surface dyslexia. In cases of impaired written language performance according to the AAT, patients were presented with (nonstandardized) screening lists containing orthographically irregular words (e.g., parts of LEMO 2.0; Stadie et al., 2013). Note that the detection of surface dyslexia/dysgraphia in German is not as straightforward as that in English due to the relatively strong regularity of the orthography.

- **Nonverbal Semantic Test** (Hogrefe et al., 2022), Semantic Sorting subtest. The subtest involves an odd-one-out paradigm and requires decisions about semantic relationships between pictured objects and/or situations. Participants give their answers by pointing. The subtests were used to check nonverbal semantic knowledge (Gorno-Tempini et al., 2011).

For 34 of the 38 participants, data of amyloid biomarkers associated with AD pathology (cerebrospinal fluid analysis and/or amyloid positron emission tomography) were available. Table 1 summarizes demographic data, AD biomarker results, and selected findings from the language assessment for each participant. Numbers in bold indicate impaired performance (for diagnostic characteristics of nfvPPA, svPPA, and lvPPA according to the 2011 consensus criteria, see Supplemental Material S1).

Clinical diagnostics also included the assessment of speech motor functions, which is the subject of this study. The time interval between the speech and language examinations was less than 1 week in 25 cases, between 1 and 4 weeks in seven cases, and more than 4 weeks in six participants (in the latter cases, the AAT results were already available, and retesting via the AAT was not warranted within the time interval). Depending on the participants' physical capacity and available time resources, the speech and language examinations were each administered in one session (including breaks) or divided into two sessions. In all cases, the testing was performed by certified speech-language pathologists (SLPs; A.S., D.P., and T.R.).

The acquisition and preparation of the speech samples, as well as the analysis procedures, are described below. We purposely refrained from an a priori classification of study participants into PPA subtypes, since motor speech dysfunction (or integrity, respectively) is among the criteria on which, according to the current gold standard, subtyping of PPA is based.

Acquisition and Preparation of Speech Samples for Speech Analyses

Speech samples used for speech analyses were taken from recordings of the Cookie Theft picture description task (Goodglass et al., 2001) and standard clinical assessments for MSDs in German language, as follows:

- **Bogenhausen Dysarthria Scales** (BoDyS; Ziegler et al., 2018). The BoDyS is a clinical tool for the assessment of dysarthria. Administration involves speech tasks in four different modalities (conversation, sentence repetition, text reading, and narration of a picture story) with three parallel versions of each task type.
- **Hierarchical Word Lists**, compact version (Ziegler et al., 2020). Hierarchical Word Lists are a single-word repetition test for apraxia of speech. The test provides a structured list of single words organized according to phonetic complexity criteria.

For this study, a procedure was employed that used selected speech recordings from the aforementioned speech assessment. No recordings of reading tasks were included since the reading ability of several participants was impaired to an extent that would have made reliable judgments of motor speech difficult. In order to provide the broadest possible basis for assessing speech performance, various speech modalities and levels of speech motor complexity were considered.

For each participant, we selected video passages of spontaneous speech (minimum of 2 min) taken from semistructured interviews of the BoDyS battery (e.g., "What do you like to do in your leisure time?"), a video sample of the Cookie Theft picture description task, video samples of 10 sentences taken from the sentence repetition tasks of the BoDyS battery (two sentences of four, six, eight, 10, or 12 syllables each; e.g., "*Keiner kann sagen, ob die Geschichte wahr ist*" ["No one can say if the story is true"]), and audio samples of 28 single-word repetitions from the Hierarchical Word Lists. The samples contained the patients' full responses to each test word, including eventual articulatory groping, false starts, and attempts to self-correct. The selected items were five productions each of one-, two-, three-, and four-syllable words (e.g., 5 × "*Pyramide*" ["pyramid"]) and single-word productions of two 2-syllable, two 3-syllable, and four 4-syllable complex words (e.g., "*Krankenschwester*" ["nurse"]). All video samples were prepared using the annotation tool ELAN (Version 6.3; The Language Archive, 2022). Audio samples were prepared using the phonetic software Praat (Version 6.1.12; Boersma & Weenink, 2020).

Table 1. Demographic data, Alzheimer's disease (AD) biomarker results, and selected findings from the language assessment per participant.

ID	Age at exam (y)	Sex (F/M)	Disease durat. (y)	AD pathology	Token Test (age-corrected error)[a]	AAT Word compr. (max. 30)[b]	AAT Sent. compr. (max. 30)[c]	AAT Confr. nam. (max. 30)[d]	AAT Sent. repet. (max. 30)[e]	AAT Read/write (max. 90)[f]	Surface dyslexia/ dysgraphia[g]	AAT Synt. struct. (agramm.)[h]	AAT Sem. struct. (word retr.)[i]	AAT Phonol. struct. (phon. err.)[j]	NVST Obj. knowl. (max. 24)[k]
P01	79	F	4	No[l]	**25**	**16**	**23**	**11**	29	**54**	NE	5	**3**	5	24
P02	67	M	1	—	**19**	**22**	**23**	**21**	**23**	**76**	**P**	4	**3**	**4**	**17**
P03	56	M	2	No[l]	6	30	**23**	**25**	**22**	82		**2**	**4**	**4**	24
P04	56	M	3	No[m]	**38**	**16**	28	**(1)[n]**	(14)[o]	83		5	**4**	5	**19**
P05	77	F	1	Yes[l]	**19**	**24**	**17**	(14)[n]	**(6)[o]**	**54**	—	**2**	**4**	**3**	**18**
P06	76	F	6	No[l]	**14**	27	**26**	30	**27**	86		5	**4**	5	23
P07	77	M	1	—	**17**	**22**	**12**	**2**	**22**	**81**	**P**	4	**3**	5	**21**
P08	74	M	3	No[l]	5	30	**21**	30	30	**81**	NE	4	**3**	**3**	**21**
P09	71	F	5	No[l]	**8**	29	**26**	**24**	**21**	85		**2**	5	**4**	24
P10	68	M	3	No[l]	**35**	**24**	**13**	**21**	**13**	**80**	—	— (due to prevailing speech automatisms)			**8**
P11	76	F	4	No[m]	**12**	**23**	**23**	**28**	**26**	—	—	5	**4**	5	**8**
P12	54	M	2	Yes[m]	**14**	**25**	**18**	**24**	**18**	82		4	**4**	**4**	23
P13	76	M	1	No[m]	**13**	30	27	**27**	28	**76**	NE	5	**3**	**4**	22
P14	58	M	8	Yes[m]	**19**	**20**	**18**	**25**	**17**	**63**	NE	4	**4**	**2**	**15**
P15	68	M	2	Yes[l,m]	**11**	**26**	**16**	**22**	**13**	**45**	NE	4	**3**	**2**	**20**
P16	75	F	3	No[m]	**29**	**19**	**26**	**1**	**15**	**43**	NE	**1**	**3**	**4**	22
P17	65	M	3	No[m]	0	**22**	**26**	**11**	29	89		5	**4**	5	**19**
P18	68	F	4	Yes[l]	**32**	**20**	**14**	**8**	**16**	**37**	NE	4	**3**	**3**	**12**
P19	61	F	3	No[m]	0	27	**26**	30	**26**	87		5	**4**	**3**	23
P20	64	M	2	No[m]	**10**	28	**21**	**26**	**26**	85		5	**4**	5	**19**
P21	76	F	4	No[m]	**12**	**21**	**20**	**16**	**23**	**81**	NE	4	**3**	**4**	24
P22	55	M	2	No[m]	4	27	29	**17**	**26**	**75**	NE	5	**4**	5	24
P23	68	F	3	No[m]	**16**	**21**	**23**	**1**	**23**	**52**	—	4	**3**	5	**18**
P24	74	M	8	Yes[l,m]	**21**	**23**	**14**	**3**	**8**	**52**	**P**	3	**2**	**2**	**21**
P25	70	F	3	No[m]	0	**26**	**25**	30	**21**	88		5	**4**	**3**	22
P26	62	M	5	Yes[m]	**8**	**17**	**13**	**0**	**23**	**70**	**P**	5	**3**	5	**14**
P27	80	M	9	No[m]	0	**21**	**21**	**19**	29	84		5	**4**	5	**21**
P28	61	M	3	Yes[m]	5	28	**26**	**18**	**19**	**76**	NE	5	**4**	**4**	24
P29	55	F	3	No[m]	0	**24**	29	**17**	29	88		5	**4**	5	23
P30	76	M	1	—	**13**	**19**	**23**	**6**	**24**	**81**	**P**	5	**3**	5	**20**

(*table continues*)

Table 1. *(Continued).*

ID	Age at exam (y)	Sex (F/M)	Disease durat. (y)	AD pathology	Token Test (age-corrected error)[a]	AAT Word compr. (max. 30)[b]	AAT Sent. compr. (max. 30)[c]	AAT Confr. nam. (max. 30)[d]	AAT Sent. repet. (max. 30)[e]	AAT Read/write (max. 90)[f]	Surface dyslexia/ dysgraphia[g]	AAT Synt. struct. (agramm.)[h]	AAT Sem. struct. (word retr.)[i]	AAT Phonol. struct. (phon. err.)[j]	NVST Obj. knowl. (max. 24)[k]
P31	68	M	2	—	0	**23**	**21**	**12**	28	**80**	**P**	4	**4**	5	**18**
P32	70	M	3	No[m]	**14**	**25**	**26**	**25**	30	**81**	NE	4	**4**	5	**13**
P33	80	F	1	No[m]	0	**25**	**22**	**18**	**20**	—	—	**2**	5	**2**	22
P34	72	F	3	No[m]	0	30	30	30	**20**	90		5	5	5	24
P35	53	F	3	No[m]	0	29	27	**17**	**23**	**70**	NE	**1**	**3**	**3**	24
P36	69	F	4	Yes[m]	**19**	**23**	**21**	**5**	**8**	**48**	NE	4	**3**	**2**	**20**
P37	70	F	9	No[m]	**30**	**10**	**14**	**1**	**23**	**76**	**P**	5	**3**	5	**6**
P38	55	F	3	Yes[m]	**8**	**21**	**19**	**16**	**19**	**60**	**P**	4	**3**	**4**	**21**

Note. Bold numbers indicate impairment. Em dashes indicate data not available. y = years; F = female; M = male; Disease durat. = disease duration; AD = Alzheimer's disease; AAT = Aachen Aphasia Test (Huber et al., 1983); Word compr. = word comprehension; max. = maximum; Sent. compr. = sentence comprehension; Confr. nam. = confrontation naming; Sent. repet. = sentence repetition; Synt. struct. (agramm.) = syntactic structure (agrammatism); Sem. struct. (word retr.) = semantic structure (word retrieval); Phonol. struct. (phon. err.) = phonological structure (phonemic errors); NVST = Nonverbal Semantic Test (Hogrefe et al., 2022); Obj. knowl. = object knowledge; NE = no evidence; P = probable. AAT subtests and cutoff scores for impairment: [a]Token Test (> six errors, corrected for age), [b]Subtest "Auditives Verständnis für Wörter" (< 27), [c]Subtest "Auditives Verständnis für Sätze" (< 27). [d]Subtest "Benennen Objekte"—Part 3 (< 29), [e]Subtest "Nachsprechen Sätze" (< 28), [f]Subtest "Schriftsprache"—Parts 1–3 (total < 82). Spontaneous speech evaluation according to AAT criteria: [g]Nonstandardized screening lists containing orthographically irregular words (e.g., parts of LEMO 2.0; Stadie et al., 2013), [h]Values 1 and 2 indicate agrammatism, [i]Values 3 and 4 indicate prevailing word retrieval deficits, whereas 2 indicates prevailing semantic paraphasias/neologisms/empty phrases, [j]Values 2, 3, and 4 indicate the occurrence of phonemic errors, [k]NVST cutoff score for impairment: score < 22. [l]Cerebrospinal fluid analysis. [m]Amyloid positron emission tomography. [n]Boston Naming Test (Kaplan et al., 1978, 15-item-version from the German version of the CERAD-NAB; Aebi, 2002; see also Morris et al., 1989), n/correct. [o]Bogenhausen Dysarthria Scales sentence repetition (15 sentences), *n* repetitions free of error.

Auditory Perceptual Evaluation of Speech

Raters

The audio and video samples were evaluated by two raters with extensive experience in the analysis of MSDs (Rater 1 [R1]: A.S. [SLP], Rater 2 [R2]: W.Z. [neurophonetician]) using high-quality headphones. R1 conducted most of the speech and language examinations herself and knew all patients prior to the speech evaluation. She was familiar with each patient's clinical history and aphasia test results. By contrast, R2 did not know any of the patients before conducting the speech rating and had no background information. The only information made available to R2 for speech evaluation was the age and gender of each participant. This was considered important to adequately judge age- and gender-dependent speech motor characteristics (e.g., voice pitch).

Speech Scales and Features Rating Protocol

For the specific purpose of this study, a new protocol was designed to allow for the most complete description of auditory speech motor symptoms possible, without restrictions to specific speech production levels (e.g., articulation, prosody), predefined speech motor syndromes (e.g., apraxia of speech), or degrees of severity. The speech evaluation procedure, which is not yet in use as a clinical tool, was similar to the BoDyS procedure (Ziegler et al., 2018) but differed in several aspects to meet the requirements of this study (e.g., by including a further speech dimension; see below). The rating protocol comprised nine different scales, which were assumed to represent major dimensions of possible speech motor impairment: speech breathing (BREATH), pitch and loudness (PL), voice quality (VOQ), voice stability (VOS), sound production (SP), nasal resonance (RES), articulatory rate (RATE), speech fluency (FLU), and prosodic modulation (MOD). The scales cover the three motor components involved in speaking—respiratory, laryngeal, and supralaryngeal systems—as well as three prosodic dimensions (Ziegler et al., 2017). In order to capture abnormalities in speech behavior that are particularly characteristic of apraxia of speech, we have added a 10th scale labeled speech behavior (BEHAV) to the nine BoDyS scales listed above. Table 2 (left column) provides a list of the 10 speech dimensions (scales). For each participant, the 10 speech scales were scored on a 5-point equally appearing interval scale from 0 to 4, where 4 = *complete absence of any impairment*, 3 = *mild impairment*, 2 = *moderate impairment*, 1 = *severe impairment*, and 0 = *very severe impairment*.

In addition, each scale was assigned features that provide a more detailed description of the quality of impairment on that particular scale. Table 2 (right column) lists 36 predefined features corresponding to the 10 speech scales (for a definition of each single feature, see Supplemental Material S2). For example, for the VOQ scale, the features "breathy," "strained–strangled," and "harsh" are predefined and can be selected from the feature set. To allow for the documentation of features not listed, we added the open-class category "other" to the lists of features for all scales. If the category "other" was selected, raters gave a free-text description of the observed characteristic. Like the scales, the features were subjected to scaling from 4 (*feature not present*) to 0 (*feature occurs in the most severe form*).

The scales and features are conceptualized as variables representing auditory surface phenomena, initially free of interpretation. For example, a score of less than 4 on the VOQ scale means that voice quality is impaired to some degree. However, this does not necessarily mean that the voice problem is dysarthric in origin. Even many neurologically healthy individuals exhibit voice abnormalities (habitual, functional, or organic in origin; Martins et al., 2016). The interpretation of the findings is finally made against the background of the overall speech motor profile, including the feature level.

The single scales and features cannot be considered independent of each other. Rather, there are many interactions between the functional systems. For example, if patients demonstrate impairments in speech breathing (e.g., reflected by shortened breath groups), this will also affect speech fluency. In such cases, the disturbances were scored on both scales (BREATH and FLU).

Rating Procedure

The speech samples were analyzed auditorily, with the video recordings of the connected speech and sentence repetition tasks additionally conveying visual information about facial movements. Each evaluation included three steps.

Step 1: Using the speech scales and features rating protocol, the raters first scored the BREATH scale and its associated features. The judgments integrated the listener's auditory impression across all speech tasks, that is, the connected speech tasks and the word and sentence repetition tasks. Note that not all speech dimensions can be assessed equally well in all tasks. For example, inconsistently occurring sound errors of phonemic and/or phonetic type can be captured particularly well in (repeated) word productions and sentences. In contrast, many prosodic and respiratory features are more likely to be detected in connected speech than in single-word utterances. The same procedure was followed for each of the other nine scales. Raters were allowed to listen to the speech samples as often as necessary.

Step 2: The raters decided on a dichotomous scale (MSD yes/no) whether the speech characteristics were interpreted as an MSD. If so, they also gave a judgment

Table 2. The 10 speech scales and their associated features.

Scale	Features (predefined)
Speech breathing (BREATH)	- Shortened breath groups (within-word and inappropriate interword inspiration pauses) - Speaking on residual air - Audible/strenuous inspiration
Pitch and loudness (PL)	- Low pitch - High pitch - Low volume - High volume
Voice quality (VOQ)	- Breathy - Strained–strangled - Harsh
Vocal stability (VOS)	- Changes in voice quality - Pitch and loudness changes - Vocal tremor/vocal flutter - Devoicing, vocal decay, vocal stoppage - Involuntary vocalizations
Sound production (SP)	- Sound errors – phonemic type - Sound errors – phonetic type - Open articulation - Closed articulation - Overall reduced articulation - Fluctuating articulatory precision
Nasal resonance (RES)	- Hypernasality - Mixed hypernasality–hyponasality
Articulatory rate (RATE)	- Reduced overall articulation rate - Prolongations of single consonants, vowels, and sound transitions - Increased articulation rate
Speech fluency (FLU)	- Unfilled speech disruptions (pauses) - Filled speech disruptions - Sound and syllable repetitions - Speech blocks - Reduced initiation/maintenance of speech
Modulation (MOD)	- Reduced pitch and loudness modulation - Syllabic (scanning) speech - Blurring of syllable boundaries
Speech behavior (BEHAV)	- Articulatory groping - High articulatory effort

on the overall severity of the speech impairment, with 3 representing *mild impairment* and 0 representing *most severe impairment*. Cases without evidence of an MSD but a suspected linguistic disorder affecting sound production (aphasic–phonological impairment) were noted by the raters. The notes were not systematically evaluated but served the purpose of transparency for subsequent interrater comparisons.

Step 3: If there was evidence of an MSD, raters made a judgment about the type of speech motor syndrome. Classification was based on the individual speech profiles, which allowed for the identification of distinctive feature clusters (see Supplemental Material S3 for typical symptom constellations of the distinct speech motor syndromes). Note, however, that syndromes were identified neither by single mandatory speech features nor by requiring a minimum number of features that must be present.

Raters could choose from the following 13 categories:

- Apraxia of speech

1. prosodic
2. phonetic
3. mixed prosodic–phonetic

- Dysarthria

4. spastic
5. flaccid
6. ataxic
7. hypokinetic
8. hyperkinetic
9. mixed (e.g., spastic–ataxic)

- Further neurogenic speech disorders

10. acquired neurogenic stuttering
11. impoverished speech drive
12. mixed (e.g., prosodic-type apraxia of speech + spastic dysarthria)
13. other (not otherwise specified)

If the raters selected a mixed category, they additionally indicated the respective components of the disorder but without weighting them more precisely. If they selected "other," they gave a more detailed description of the observed speech pattern (free text).

It should be noted that aphasia with phonological impairment shares surface similarities with apraxia of speech at the segmental (phonemic errors) and prosodic (e.g., disfluencies due to successive phonemic approximations) levels. Their differentiation can therefore be difficult at times. A requirement for the classification of apraxia of speech/MSD was that the occurrence of phonemic errors (and disfluencies) was accompanied by additional features characteristic of apraxia of speech, such as phonetic errors and/or syllabic speech, among others (see Supplemental Material S3).

Training Phase

The procedure was tested on a sample of three participants not included in the final evaluation, and methodological adjustments were made where necessary. Once the procedure was established, each rater assessed seven additional cases individually. The results of these assessments were subsequently discussed in two consensus-building sessions and were not included in the reliability analyses. Each of the raters then independently analyzed the remaining 31 data sets. The cases were presented to R1 and R2 in different orders to counteract gradual familiarization and training effects.

Reliability Analyses and Consensus Among Raters

Reliability Analyses

Judgments on the presence or absence of an MSD made by R1 and R2 were compared for 31 participants (38 minus seven training cases). Comparisons of MSD type and MSD severity were restricted to cases classified as having MSD by both raters. We used Krippendorff's alpha for nominal and ordinal data to assess the level of agreement between the two raters. For each of the 10 speech scales and 36 features, percentage of agreement was calculated.

Finding Consensus

In cases where R1 and R2 reached a different judgment about the presence or absence of an MSD or about the type of an MSD, a further highly experienced SLP (Rater 3 [R3]: T.S.) was called in, and consensus was finally reached in a roundtable discussion. R3 went through the same training steps as described above and made her ratings using exactly the same procedures as R1 and R2.

When R1 and R2 differed by 1 point on the MSD severity scale, the speech scale level, or the feature level, a mean was calculated (e.g., BREATH scale: R1 = 2.0, R2 = 3.0; $M = 2.5$). In cases where R1 and R2 differed by more than 1 scale point, R3 rescored the particular scale or feature, and a mean was calculated from all three judgments (e.g., BREATH scale: R1 = 3.0, R2 = 1.0; R3 = 1.0; $M = 1.7$).

Results

Reliability

R1 and R2 agreed on the presence or absence of an MSD in 27 of the 31 cases (87.1%). Krippendorff's α (nominal level, 10,000 bootstraps) was acceptable at .74, 95% CI [0.48, 0.93]. In three of the four cases of mismatch, one of the coders interpreted the symptoms as being caused by *language* impairment. For the participants rated as having an MSD by both raters (11 excluding the training cases), ratings of MSD severity fully matched in six cases. In five cases, the difference was 1 scale point. Krippendorff's α (ordinal level, 10,000 bootstraps) was still acceptable at .69, 95% CI [0.47, 0.91] (with an α greater than or equal to .667, denoting the lowest limit for agreement; Krippendorff, 2004, p. 241). The classifications of speech motor syndrome showed complete agreement in six cases. If the syndromes were evaluated differently, this was expressed primarily in modifiers/additional components, but not in different standard syndromes (e.g., R1: "mixed prosodic–phonetic–type apraxia of speech," R2: "prosodic-type apraxia of speech"). A summary of the nonmatching cases is provided in Appendix A.

Across the speech scales, complete agreement in severity scoring was achieved in 57.3% of all 110 comparisons (11 participants with MSD, 10 speech scales). In 30.9% of the judgments, there was a difference of 1 scale point; in 10%, the difference was 2 scale points; and in two comparisons (1.8%), the raters differed by 3 scale points.

Regarding the severity ratings, deviations of no more than 1 point can be considered tolerable in the context of this study. This was the case in 88.2% of all scale-level ratings. At the feature level, full agreement was achieved in 74.5% of all 396 judgments (11 participants, 36 features); 17.9% of all comparisons showed a difference of 1 scale point, whereas in 6.1%, the difference was 2 scale points. In only six comparisons, there was a difference of 3 scale points (1.5%). Overall, 92.4% of all feature-level ratings differed by no more than 1 scale point.

Speech Motor Syndromes and Severity at the Group Level

By consensus, 18 individuals (47.4%) of the study sample were classified as having some form of MSD. Overall severity was moderate (M = 2.0, SD = 0.7; min = 0.5, max = 3.0). Table 3 lists the distribution of syndromes in the 18 participants with MSDs. The most common MSD type was mixed prosodic–phonetic apraxia of speech (n = 5).

Speech Scales

Figure 1 depicts the results of the evaluations of the 10 speech scales for the 18 participants judged to have some form of MSD. The gray tones correspond to severity levels, with darker shades indicating more severe impairment. As shown by unfilled boxes, none of the speech scales were affected in all speakers, but none of the scales remained unaffected in all either.

SP and FLU were most frequently affected in the speakers (94.4% and 88.9%, respectively), followed by MOD with 72.2%. Impairments on the RATE and VOQ scales were evident in 61.1% and 55.6% of the cases, respectively. PL, BREATH, BEHAV, and VOS were affected in 38.9%–50% of the participants with MSDs. With two in 18 cases (11.1%), abnormalities on the RES scale occurred least frequently.

The severity levels of the single scales varied considerably in the speaker group. Mild and moderate impairments (3.33–1.33) dominated in the various speech dimensions. Only mild impairment was found on the RES scale. Severe impairments (< 1.33) were observed on the SP, FLU, VOQ, and VOS scales, but only in rare cases. None of the scales reflected very severe manifestations, that is, with scores below 1.0.

Speech Features

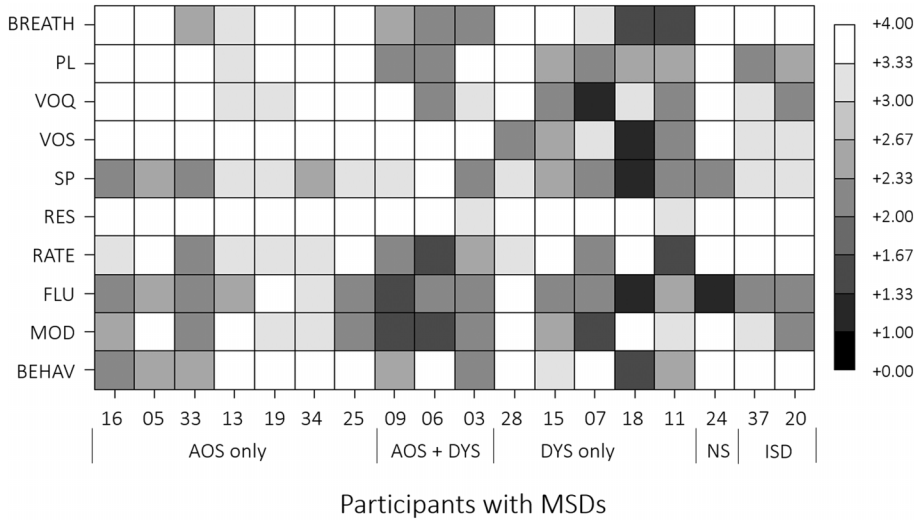
Figure 2 depicts the severity ratings of the speech features related to different dimensions of speech: (a) speech breathing and voice (BREATH, PL, VOQ, VOS), (b) sound production and resonance (SP, RES), and (c) prosody and speech behavior (RATE, FLU, MOD, BEHAV; see also Table 2). The feature patterns differed considerably within the speaker group. With the exception of high volume, each of the 36 features unequivocally occurred in at least one participant diagnosed with MSD. None of the speech features were present in all subjects. The most frequent perceptual speech characteristics, each observed in more than 60% of the participants, were errors of phonemic type, errors of phonetic type, and unfilled speech disruptions (pauses). With frequencies of 44.4% and 38.9%, respectively, the most frequent forms of voice abnormalities were harsh voice quality and devoicing/vocal decay. The prosodic features reduced articulation rate, filled speech disruptions, and syllabic speech occurred with a prevalence between 38.9% and 44.4%. The severity of the single symptoms was predominantly mild to moderate. A severe manifestation (score < 1.33) was observed only for the feature sound and syllable repetitions, and this is only in one case.

Beyond the predefined features, the following "other" characteristics occurred: (a) orofacial dyskinesias; (b) abrupt/involuntary inspirations; (c) hyperkinetic speech arrest; (d) sustained audible expiration beyond end of utterances; (e) abrupt, prosodically/contextually unrelated changes of articulation rate; and (f) facial grimacing. Features (a)–(c) were documented in a single case presenting with hyperkinetic dysarthria. Feature (d) was observed in one participant with prosodic-type apraxia of speech and spastic dysarthria. Feature (e) occurred in two participants

Table 3. Distribution of motor speech disorder (MSD) syndromes in the 18 participants with impaired motor speech.

MSD syndrome	No. of participants
Mixed prosodic–phonetic–type apraxia of speech	5
Prosodic-type apraxia of speech	2
Hypokinetic dysarthria	3
Spastic dysarthria	1
Hyperkinetic dysarthria	1
Mixed (apraxia of speech + spastic dysarthria)	3
Impoverished speech drive	2
Neurogenic stuttering	1

Figure 1. Severity levels of the 10 speech scales among speakers with motor speech disorders (MSDs). A score of 0.00 represents most severe impairment; a severity level of > 3.33 indicates no or equivocal speech impairment. BREATH = speech breathing; PL = pitch and loudness; VOQ = voice quality; VOS = voice stability; SP = sound production; RES = nasal resonance; RATE = articulatory rate; FLU = speech fluency; MOD = prosodic modulation; BEHAV = speech behavior; AOS = apraxia of speech; DYS = dysarthria; NS = neurogenic stuttering; ISD = impoverished speech drive.



diagnosed with hypokinetic dysarthria and in one participant with impoverished speech drive. Feature (f) was observed in an individual with hypokinetic dysarthria.

Individual Speech Motor Profiles

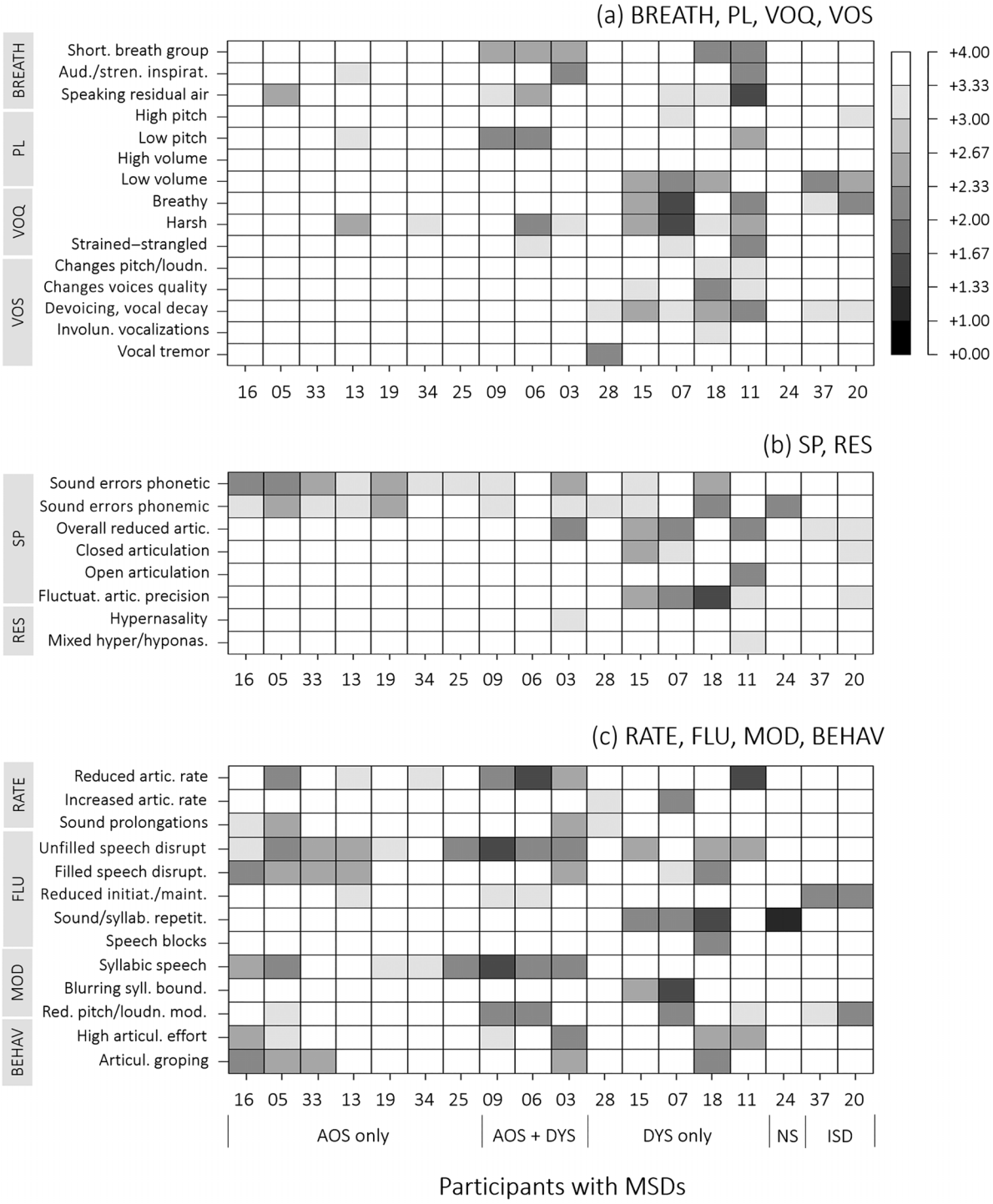
Speech scale and feature profiles of the individual speakers with MSDs can be inferred from Figures 1 and 2 (for profiles of the speakers without MSD, see Supplemental Materials S4 and S5). The ordering of the participants on the *x*-axes was set manually for easier recognition. Descriptions run from left to right. For a summary of all individual cases, see also Appendix B.

Seven participants (leftmost columns) were classified as having apraxia of speech, with five of phonetic–prosodic type (P16, P05, P33, P13, and P19) and two of prosodic type (P34 and P25). The speech motor profiles of these individuals varied, but all reflected some degree of impairment on the SP scale (phonemic errors and/or phonetic errors) and on at least one of the prosodic scales RATE (reduced prolongations of single sounds), FLU (filled/unfilled speech disruptions), and MOD (especially syllabic speech). Three participants (P16, P05, and P33) also showed unequivocal deficits on the BEHAV scale (articulatory effort and/or articulatory groping). P05, P13, and P34 additionally presented with mild deficits on scales not primarily specific to apraxia of speech, such as BREATH (e.g., audible/strenuous inspiration), PL (low pitch), and VOQ (harsh voice). However, based on the overall impression of speech, these characteristics were not interpreted as dysarthric in origin. Although subtle articulation difficulties were present, the prosodic impairments (especially syllabic speech) clearly dominated the auditory impression of the speech deficit in Participants P34 and P25.

Three individuals (P09, P06, and P03) had components of apraxia of speech and dysarthria (mixed-type MSD). In two of them (P09 and P06), apraxic features at the prosody level (syllabic speech) were significantly more pronounced than those at the segmental level (unimpaired in P06) and therefore classified as prosodic in type. Deficits indicative of spastic dysarthria involved the BREATH scale (shortened breath groups and speaking on residual air, interpreted as signs of respiratory insufficiency), the VOQ scale (harsh), and/or the PL scale (low pitch). Presumably, the dysarthric component also contributed to the prosodic deficits in both participants (reduced pitch and loudness modulation). In P03, apraxic features on the SP (errors of phonetic and phonemic types), BEHAV (articulatory effort, articulatory groping), and MOD (syllabic speech) scales were almost equally present, giving the impression of a phonetic–prosodic apraxia of speech. P03 also showed signs of spastic dysarthria on the BREATH (shortened breath groups, audible/strenuous inspiration), VOQ (harsh), and RES (hypernasal) scales. The SP scale was additionally characterized by features indicative of dysarthria (overall reduced articulation).

Five participants were diagnosed with dysarthria only. The speech profiles of P28, P15, and P07 were most consistent with hypokinetic dysarthria. All three participants showed deficits on at least one of the voice scales

Figure 2. Severity levels of the 36 speech features among speakers with motor speech disorders (MSDs). Data are shown separately with respect to the speech domains: (a) speech breathing (BREATH), pitch and loudness (PL), voice quality (VOQ), and voice stability (VOS); (b) sound production (SP) and nasal resonance (RES); and (c) articulation rate (RATE), speech fluency (FLU), prosodic modulation (MOD), and speech behavior (BEHAV). A score of 0.00 represents most severe impairment; a severity level of > 3.33 indicates no or equivocal speech impairment. AOS = apraxia of speech; DYS = dysarthria; NS = neurogenic stuttering; ISD = impoverished speech drive. Short. = Shortened; Aud./stren. inspirat. = Audible/strenuous inspiration; loudn. = loudness; Involun. = Involuntary; Overall reduced artic. = Overall reduced articulation; Fluctuat. artic. precision = Fluctuating articulatory precision; hyponas. = hyponasality; Reduced artic. rate; Reduced articulation rate; Increased artic. rate = Increased articulation rate; disrupt. = disruptions; initiat. = initiation; maint. = maintenance; syllab. repetit. = syllable repetitions; syll. bound. = syllable boundaries; Red. = Reduced; mod. = modulation; High articul. effort = High articulatory effort; Articul. groping = Articulatory groping.



PL (high pitch, low volume), VOQ (breathy, harsh), and VOS (devoicing/vocal decay). The prominent sign of voice impairment in P28 was vocal tremor. In P15 and P07, deficits on the SP scale seemed to be related to dysarthric impairment (overall reduced articulation, closed articulation), whereas the phonemic errors that occurred in P28 and P15 most probably reflected associated aphasic–phonological disorder. The prosodic patterns differed in the three subjects. In P28 and P07, the RATE scale was abnormal (increased). The FLU (e.g., sound and syllable repetitions) and MOD (blurring of syllable boundaries and/or reduced pitch and loudness modulation) scales were affected in P15 and P07.

One participant (P18) was classified with hyperkinetic dysarthria. The most prominent signs were severe motor instabilities affecting the BREATH (e.g., abrupt/involuntary inspirations), VOS (e.g., changes in pitch and loudness), and SP (e.g., articulatory fluctuations, orofacial dyskinesias) scales. Further affected scales were PL (low volume), BEHAV (e.g., high speech effort), and FLU (e.g., sound and syllable repetitions).

P11 was classified with severe spastic dysarthria, which affected all speech scales. Dysarthric features included, but were not limited to, deficits in BREATH (e.g., shortened breath groups, speaking on residual air), VOQ (breathy, harsh, strained–strangled), SP (overall reduced articulation), RES (mixed hypernasality–hyponasality), RATE (reduced), FLU (unfilled speech disruptions), and MOD (reduced pitch and loudness modulation).

P24 was most remarkable for abnormalities on the FLU scale (multiple, rapid, uniform sound and syllable repetitions), which led to a diagnosis of neurogenic stuttering. The SP scale was characterized by errors of phonemic type, which was considered to reflect associated aphasic–phonological impairment.

The speech abnormalities of two individuals (P37 and P20) were categorized as reflecting impoverished speech drive. The participants were most notable for impaired initiation/maintenance of speech on the FLU scale. Speech production was further characterized by deficits in PL (low volume), VOQ (breathy), VOS (devoicing/vocal decay), SP (overall reduced articulation), and MOD (reduced pitch and loudness modulation).

Distribution of MSDs in Subtypes of PPA

Combining the speech motor findings with the results of the language assessments (see Table 1), eight of the 18 participants with MSD matched the subtype of nfvPPA (P03, P06, P09, P13, P16, P19, P25, and P33; see also Appendix B). In one case with prosodic-type apraxia of speech (P34), there was no clear evidence of aphasia. Therefore, this participant was diagnosed with PPAOS.

We also found MSDs in nine subjects whose language profiles were not compatible with the nonfluent subtype of PPA or with PPAOS: One participant (P28) presenting with dysarthria met the criteria for lvPPA (presence of both core features of lvPPA and three of four supportive diagnostic features). In seven participants, the application of the consensus criteria resulted in a categorization of "unclassifiable" (PPA-U). Although three of them would have been classified as PPA-U irrespective of their speech motor diagnoses (P05, P20, and P24), a violation of the "spared motor speech" criterion ultimately ruled out a standard subtype diagnosis in four cases, thereof two closest to svPPA (P07 and P37) and two closest to lvPPA (P15 and P18).

For one participant, a reliable subtype classification was not possible because the written language parts of the assessment were not feasible, and thus, no judgment could be made regarding the criterion of surface dyslexia (P11). However, since neither agrammatism nor apraxia of speech was present in this participant, a subtype diagnosis of nfvPPA could be ruled out with certainty.

A Mann–Whitney U test was conducted to compare the MSD severity levels in the participants with nonfluent forms (nfvPPA, PPAOS) and those with other forms (lvPPA, PPA-U, undetermined). The median degree of severity was 2.0 in both groups. The distribution in the two groups did not differ significantly (Mann–Whitney $U = 23.00$; $p = .108$).

On the basis of their speech and language profiles, the participants who were not diagnosed with MSD, thus fulfilling the "spared motor speech" criterion (not depicted in Figures 1 and 2), could be classified into the following subtypes: eight with svPPA (P02, P04, P17, P26, P27, P30, P31, and P32), five with lvPPA (P12, P14, P21, P22, and P36), one with nfvPPA (P35), and four with PPA-U (P01, P08, P29, and P38). In one participant (P10), the subtype could not be determined with certainty because the criteria for spontaneous speech were not assessable due to prevailing speech automatisms. In another case (P23), no data were available to estimate the presence of surface dyslexia, which would have been crucial in deciding the subtype.

Discussion

We conducted a prospective investigation of the prevalence and distribution of MSDs in a well-sampled cohort of 38 German-speaking participants with a root diagnosis of PPA independent of subtype, including one case with PPAOS. To our knowledge, this study is the

first that also systematically addressed the qualitative and quantitative auditory manifestations of speech motor dysfunction across all major dimensions of speech, that is, speech breathing, voice, articulation, resonance, prosody, and speech behavior, in a sample of comparable size and composition.

On the basis of consented auditory ratings of three expert raters, 18 of the 38 participants (47.4%) were judged to have an MSD. Besides PPAOS, where apraxia of speech already is the defining feature, the study findings confirm that MSDs are common and thus clinically highly relevant comorbidities in PPA (Staiger, Schroeter, Ziegler, et al., 2021). MSDs also represent a key criterion for subtype classification, making their thorough consideration in clinical assessment and research indispensable.

Subtype Classification of PPA Under Consideration of Speech Motor Performance

On the basis of the results of both the speech and language assessments, one subject was diagnosed with PPAOS. Except for a moderate deficit in sentence repetition, most likely attributable to an underlying speech planning deficit, the participant's language profile did not reveal signs of aphasia. The finding adds to a growing body of evidence supporting the occurrence of apraxia of speech as a primary manifestation of progressive disease (Duffy et al., 2021).

Twenty-four of the remaining 37 participants with PPA (64.9%) could be clearly assigned to one of the standard subtypes (nine with nfvPPA, nine with svPPA, and six with lvPPA). In 13 participants, classification according to the diagnostic scheme was not possible because either not all relevant classification criteria could be reliably determined (three cases) or the participants did not meet the criteria for any variant (PPA-U; 10 cases). The proportion of unclassifiable cases (29.4% of the participants with PPA when the three undeterminable cases are not considered) is well within the range reported in the literature (10%–41%; Utianski et al., 2019). This seems to confirm the overall feasibility and comparability of the classification approach used for subtyping our German patient sample (Staiger, Schroeter, Müller-Sarnowski, et al., 2021). However, the number of unclassifiable cases also suggests that a gradual rather than a categorical classification system may be appropriate given the clinically heterogeneous clinical pictures of PPA (e.g., Ingram et al., 2020). MSDs also appear to play a role in this discussion. In four of the unclassifiable cases with MSDs, it was the presence of a speech deficit that was decisive in ruling out the classification of lvPPA or svPPA when criteria were strictly applied. MSDs, however, have been reported in previous studies of svPPA and lvPPA (Duffy et al., 2014; Poole et al., 2017; Staiger, Schroeter, Ziegler, et al., 2021), and plausible explanations exist for their occurrence (Staiger, Schroeter, Ziegler, et al., 2021). These results challenge the appropriateness of the criterion of "intact motor speech" in the list of characteristics used to define svPPA and lvPPA and should be reconsidered in future modifications of the classification criteria. This will be discussed further below.

Prevalence and Types of MSDs in Different Variants of PPA

nfvPPA and PPAOS

As expected against the background of the existing literature on PPA, MSDs occurred most frequently in the group of speakers with nfvPPA—the only form in which speech motor dysfunction is also among the core (though not mandatory) clinical criteria. Eight of the nine participants with nfvPPA (88.9%) presented with MSDs; only one participant had intact motor speech (but was agrammatic). In our sample, all participants with MSDs in nfvPPA presented with some form of apraxia of speech—either as the sole sign of the MSD or in association with dysarthria. The high prevalence of apraxia of speech in nfvPPA is consistent with findings of previous studies indicating rates of up to 100% (Duffy et al., 2014). However, the prevalence rate reported here is considerably higher than that of a former study by Staiger, Schroeter, Ziegler, et al. (2021), based on retrospective speech data of 160 individuals with a root diagnosis of PPA, thereof 74 with nfvPPA (28% with apraxia of speech). The authors cited methodological reasons for the low prevalence of apraxia of speech in their sample. For example, speech motor evaluations were based exclusively on audio samples of a picture description task, which did not require participants to produce utterances with explicitly high articulatory demands. Such methodological weakness was circumvented in this study by using different tasks with varying degrees of speech motor complexity, including the repetition of multisyllabic complex words (e.g., "*Krankenschwester*, *Strümpfe*, *Elektriker*" ["nurse, socks, electrician"]). This might have increased the sensitivity for detecting speech apraxic symptoms, even in mildly impaired participants.

Three of the participants with apraxia of speech in nfvPPA had concomitant dysarthria of spastic type. There is evidence in the literature for a comorbidity of apraxia of speech and dysarthria, particularly in advanced nfvPPA (Duffy et al., 2014). With 5 and 6 years post onset, two of the affected subjects (P06 and P09) also had the longest disease durations within the speaker sample. Among the forms of dysarthria that occur in nfvPPA, the spastic form is common. Its occurrence is considered to reflect damage

to the (bilateral) upper motor neuron system, indicating increasing involvement of this system by the process of neurodegeneration. One participant with comorbidities of apraxia of speech and spastic dysarthria in nfvPPA had been diagnosed with PPA-CBS. The pattern of his speech motor impairment is fully compatible with the diagnosis, as apraxia of speech and dysarthria (of spastic and/or hypokinetic type) are also considered common signs of CBS (Blake et al., 2003).

lvPPA

Six participants were classified as having lvPPA. Five of them showed no evidence of an MSD. This seems to confirm the assumption that involvement of the speech motor system is rare in the logopenic subtype. However, the argument appears somewhat circular, considering that the presence of an MSD can easily lead to exclusion of the diagnosis (even when other criteria for lvPPA are met; see below). Yet, one participant met the diagnostic criteria for lvPPA despite the presence of an MSD (with the clinical diagnosis being further supported by a biomarker finding indicative of an underlying AD pathology; see Table 1). The type of MSD was classified as mild-to-moderate hypokinetic dysarthria with prominent vocal tremor. The observation fits with a few descriptions of hypokinetic dysarthria in lvPPA (Josephs et al., 2010; Staiger, Schroeter, Ziegler, et al., 2021) and could potentially be explained by Parkinsonism associated with AD pathology (Rowe, 2019).

svPPA

Eight participants of the present PPA sample were classified as being of semantic subtype. None of them presented with an MSD. As with lvPPA, this is consistent with the general assumption of unaffected speech motor functions in this subtype. However, the caveat formulated for lvPPA also applies here. Strict adherence to the consensus criteria can readily lead to exclusion of the diagnosis of svPPA when speech motor impairment is present. Evidence from the literature suggests that MSDs can potentially occur in svPPA, for example, as a sign of an underlying MND (Vinceti et al., 2019; cf. Staiger, Schroeter, Ziegler, et al., 2021). As a very stable finding, however, apraxia of speech does not appear to occur in svPPA.

PPA-U

Seven participants with MSDs could not be assigned to one of the three PPA subtypes when the consensus criteria by Gorno-Tempini et al. (2011) were strictly applied. Patients with PPA-U represent a very heterogeneous group, and no single causative mechanism for deficient speech motor control processes can be reasonably assumed.

One participant with PPA-U presented with apraxia of speech of phonetic–prosodic type. Since the participant also showed agrammatism, the pattern appeared similar to that of nfvPPA. Yet, additional impairments in single-word comprehension and object knowledge precluded a diagnosis of nfvPPA. Brain imaging analyses were beyond the scope of this study. However, it is hypothesized that the participant's pattern of brain atrophy presumably involved areas linked to speech motor planning processes, that is, left anterior perisylvian cortex, superior premotor cortex, and/or supplementary motor area (Duffy et al., 2021; Josephs et al., 2013; Ziegler & Staiger, 2016).

Of the two unclassifiable patients most closely meeting the criteria for the logopenic type (both with positive markers for AD pathology; see Table 1), one presented with hypokinetic dysarthria and concomitant phonological impairment. As discussed above, hypokinetic dysarthria is well reconciled with the presence of lvPPA. Deviation from a further criterion ("spared single-word comprehension and object knowledge"), however, did not allow the diagnosis of lvPPA. The other patient's speech impairment was most consistent with the picture of hyperkinetic dysarthria. To our knowledge, there have been no descriptions of patients with hyperkinetic dysarthria in PPA (of any type) so far. There were also no indications from the participant's medical record that could have explained the speech abnormalities (e.g., medication-induced). However, the dyskinetic speech symptoms (e.g., orofacial dyskinesias, uncontrolled respiration and phonation) strongly suggest basal ganglia involvement (Ziegler & Staiger, 2016).

One participant (P24) showed impairments at almost all linguistic levels and, therefore, could not be classified. Considerable difficulty with sentence repetition, the occurrence of phonological errors (together with a positive AD biomarker finding; see Table 1), nevertheless suggested that the underlying disorder might fall within the logopenic spectrum. Severe iterations of sounds and syllables were the only speech motor symptom present. The disorder was thus classified as acquired neurogenic stuttering. Recent studies in stroke survivors suggest a particular importance of the basal ganglia and their interaction with the frontal cortex in the development of acquired neurogenic stuttering (Theys & De Nil, 2022).

Of the two participants with PPA-U whose clinical profiles most closely matched the svPPA type, one exhibited hypokinetic dysarthria, and the other showed signs of impoverished speech drive. It can only be speculated here whether the findings are related to underlying pathomechanisms of svPPA. At least Staiger, Schroeter, Ziegler, et al. (2021) found evidence for the occurrence of these forms in some patients with semantic-type PPA in their study. Impoverished speech drive was also the main

speech abnormality of a participant with PPA-U who had developed CBS in the course of the disease. According to case reports of patients with PPA-CBS, areas of atrophy can progress from lateral to medial aspects of the frontal lobe (Gorno-Tempini et al., 2004). That the medial parts of the frontal lobe are also assumed to be involved in the initiation and maintenance of speech offers a possible explanation for the observed picture (Ziegler & Ackermann, 2014). It should be noted, however, that CBS is also typically associated with a whole range of features of frontal dysfunction (e.g., slowed information processing, personality changes such as apathy; Mathew et al., 2012) that may be difficult to disentangle from motor speech phenomena.

Scale- and Feature-Level Characteristics of MSDs

Our study showed that all major dimensions of speech, that is, speech breathing, voice, articulation, and resonance, as well as prosody and speech behavior, can be affected in individuals with PPA. A majority of participants demonstrated symptoms of apraxia of speech. Auditory characteristics specifically comprised aspects of impaired sound production (errors of phonetic and perceived phonemic types), articulation rate (decreased rate, single-sound prolongations), prosodic modulation (syllabic speech), and characteristics in speech behavior (articulatory groping, high speech effort), although there were strong interindividual differences in speech patterns. Of the nine individuals with apraxia of speech, five presented with mixed phonetic–prosodic type, and four presented with prosodic type. A comparable classification of subtypes has not yet been established for apraxia of speech after stroke (but see Mailend & Maas, 2021). A purely prosodic type is also rather difficult to reconcile with the traditional definitions of apraxia of speech, in which prosodic and segmental speech errors are among the defining characteristics (Ballard et al., 2015). Except for one participant in our sample, however, all patients classified as "prosodic" showed at least subtle articulatory deficits (mild phonetic distortions, articulatory groping). Yet, their prosodic speech patterns (particularly syllabic speech) clearly dominated the auditory impression of their speech impairment. One may speculate that syllabic speech reflects an implicit strategy to cope with discrete articulation difficulties in mild forms of apraxia of speech. However, it also cannot be ruled out that the different subtypes of progressive apraxia of speech reflect damage at different stages of the speech planning process (Duffy et al., 2021).

Speech disfluencies appeared to be a very common phenomenon, occurring in almost all participants with MSDs to a certain extent. Relatively nonspecific in origin, filled and unfilled pauses occurred most frequently and can be interpreted as a potential consequence of or compensation for impaired respiratory or articulatory functions. Other aspects of impaired speech fluency observed in single cases included sound and syllable repetitions (predominant in an individual diagnosed with neurogenic stuttering), speech blocks (as in one case with hyperkinetic dysarthria), and delayed speech onsets during speech initiation or reduced maintenance of articulatory activity within utterances (considered to reflect impoverished speech drive). The latter symptoms, which have received little attention to date, may even represent the leading symptoms of specific forms of MSDs in PPA (Staiger, Schroeter, Ziegler, et al., 2021) and thus deserve greater consideration in future studies of PPA.

Symptoms reasonably attributable to dysarthria included disturbances in speech breathing (e.g., short breath groups, audible/strenuous inspiration), pitch and loudness (low pitch, low volume), voice quality (e.g., breathy, strained–strangled), and voice stability (e.g., devoicing, vocal tremor), as well as features affecting articulation rather globally (e.g., overall reduced articulation, closed articulation). Their occurrence and combination may inform hypotheses about the underlying pathomechanisms and the neuronal structures involved (e.g., basal ganglia, upper motor neuron system; Ziegler & Staiger, 2016). Rather unexpected was the relative sparing of the resonance scale. Whereas in a study by Ogar et al. (2007), hypernasality was found to be the most common dysarthric feature in their group of speakers with nfvPPA, in this study, only two participants showed mild impairments in nasal resonance (hypernasality, mixed hypernasality–hyponasality) and, thus, in underlying velopharyngeal movement.

Overall, the observed features of motor speech dysfunction may lead to a reduction in intelligibility or may give the impression of unnatural, "bizarre" speech (e.g., caused by abnormalities in voice, speech breathing, or prosody). Furthermore, high speech effort, decreased articulation rate, and/or frequent speech interruptions require a great deal of time and can lead to speaker fatigue. Consequently, the symptoms can have a detrimental effect on communicative interactions in everyday life and may thus aggravate the limitations caused by aphasia. This should always be considered when assessing speech and language in PPA or PPAOS.

Summary, Limitations, and Future Directions

In this study, we examined the motor speech performance of individuals with PPA of all subtypes (one case with PPAOS). Different from previous studies that focused primarily on articulatory and prosodic domains of apraxia

of speech, we examined features across all major dimensions of speech and, thus, also included features of speech breathing, voice, nasal resonance, and speech behavior.

The results demonstrate that MSDs are common in PPA and can manifest in very different patterns. Although our study confirms that speech deficits are primarily anchored in nfvPPA, the findings also show that MSDs can occur in cases whose language characteristics do not correspond to the nonfluent variant as defined in the current consensus guidelines (Gorno-Tempini et al., 2011). It should be noted, however, that the number of patients per PPA subgroup in our study was relatively small, and therefore, only very tentative conclusions can be drawn about the occurrence and manifestations of speech motor syndromes within each subgroup.

Our analyses included consensus-based evaluations by three expert raters on uniform rating scales. However, our procedure did not include phonetic transcriptions or acoustic analyses (e.g., of temporal prosody), as recently accomplished in a study by Haley et al. (2021). Another limitation of our study is that it did not include direct measurements of communication-related parameters such as speech intelligibility and naturalness. To more accurately determine the contribution of MSDs to the overall communication deficit, the inclusion of these parameters is particularly important in future studies (Lehner et al., 2022). A further limitation of our study is that we did not include brain imaging findings so far. Knowledge of associations between areas of atrophy and specific speech motor symptoms, however, may further advance our understanding of MSDs in PPA and may help explore the future potential for predicting underlying diseases and disease progression.

Nonmotor factors that may be associated with PPA (e.g., impulsivity, distractibility, depression) may also affect speech performance. These factors have not yet been specifically examined in this study. In order to explore the influence of these factors in more detail, their systematic consideration in future studies seems warranted. These limitations aside, we believe that this study contributes to a basic "charting" of speech motor symptomatology in PPA and thus helps raise awareness among researchers and clinicians of the speech motor aspect of these progressive communication disorders.

Data Availability Statement

The conditions of our ethics approval do not permit public archiving of the study data presented in Table 1. Readers seeking access to the data should contact the lead author (Anja Staiger) of the study. Access will be granted to named individuals in accordance with ethical procedures governing the reuse of clinical data upon request and ensuring patients' anonymity, including completion of a formal data-sharing agreement and approval of the local ethics committee.

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Appendix A

Table A1. Comparisons of MSD syndromes by raters R1 and R2 in nonmatching cases.

	R1	R2
1	Prosodic type apraxia of speech	None
2	None (phonological impairment)	Phonetic type apraxia of speech
3	Prosodic–phonetic type apraxia of speech	None (phonological impairment)
4	None (poverty of the *language* aspect of speech production)	Impoverished speech drive
5	Prosodic-phonetic type apraxia of speech	Prosodic type apraxia of speech
6	Prosodic type apraxia of speech	Prosodic-phonetic type apraxia of speech
7	Mixed dysarthria (spastic–ataxic)	Spastic dysarthria
8	Mixed (prosodic type apraxia of speech, spastic dysarthria)	Spastic dysarthria
9	Other (voice tremor)	Hypokinetic dysarthria (with voice tremor)

Note. MSD = motor speech disorders; R1 = Rater 1 (A.S.); R2 = Rater 2 (W. Z.).

Table B1. Summary of PPA subtype, MSD syndrome, MSD severity and specifications of affected speech scales and features in the 18 participants with impaired motor speech.

Id	Subtype	MSD syndrome	MSD severity	MSD scale and feature specifications
P03	nfvPPA/-CBS	Mixed MSD (apraxia of speech of phonetic-prosodic type + spastic dysarthria)	2.0	BREATH: 2.0 (short. breath groups, aud./stren. inspiration); VOQ: 3.0 (harsh); SP: 2 (phonetic errors, phonemic errors, overall reduced art.); RES: 3.3 (hypernasal); RATE: 2.5 (reduced, prolong. single sounds); FLU: 2.0 (filled disrupt., unfilled disrupt.); MOD: 2.0 (syllabic speech); BEHAV: 2.0 (art. effort, art. groping)
P05	PPA-U	Apraxia of speech of phonetic-prosodic type	3.0	SP: 2.5 (phonetic errors, phonemic errors); FLU: 2.5 (filled disrupt., unfilled disrupt.); BEHAV: 2.5 (art. groping)
P06	nfvPPA	Mixed MSD (prosodic type apraxia of speech + spastic dysarthria)	2.0	BREATH: 2.0 (short. breath groups, speaking on residual air); PL: 2.0 (low pitch); VOQ: 2.0 (harsh, strain-strangled); RATE: 1.5 (reduced); FLU: 2.0 (unfilled disrupt., reduced initiation / maintenance of speech); MOD: 1.5 (syllabic speech, reduced pitch/loudness mod.)
P07	PPA-U	Hypokinetic dysarthria	1.5	BREATH: 3.0 (speaking on residual air); PL: 2.0 (high pitch, low volume); VOQ: 1.0 (breathy, harsh); VOS: 3.0 (devoicing/vocal decay); SP: 2.0 (overall reduced art., closed art., art. fluctuations); RATE: 3.0 (increased); FLU: 2.0 (filled disrupt., sound-/syllable repetitions); MOD: 1.5 (blurring of syllable boundaries, red. pitch/loudness mod.)
P09	nfvPPA	Mixed MSD (apraxia of speech of prosodic type + spastic dysarthria)	1.5	BREATH: 2.5 (short. breath groups, speaking on residual air); PL: 2.0 (low pitch); SP: 3.0 (phonetic errors, phonemic errors); RATE: 2.0 (red.); FLU: 1.5 (unfilled disrupt.); MOD: 1.5 (syllabic speech, red. pitch/loudness mod.); BEHAV: 2.7 (art. effort)

(*table continues*)

Table B1. *(Continued).*

Id	Subtype	MSD syndrome	MSD severity	MSD scale and feature specifications
P11	Not determin.	Spastic dysarthria	1.0	BREATH: 1.5 (short. breath groups, aud./stren. inspiration, speaking on residual air); PL: 2.5 (low pitch); VOQ: 2.0 (breathy, harsh, strain-strangled); VOS: 2.0 (devoicing/vocal decay); SP: 2.0 (overall red. art.); RES: 3.0 (mixed hyper-/hyponasality); RATE: 1.5 (red.); FLU: 2.5 (unfilled disrupt.); MOD: 3.0 (red. pitch/loudness mod.); BEHAV: 2.7 (art. effort)
P13	nfvPPA	Apraxia of speech of phonetic-prosodic type	3.0	BREATH: 2.3 (aud./stren. inspiration); PL: 3.0 (low pitch); VOQ: 3.0 (harsh); SP: 3.0 (phonetic errors, phonemic errors); RATE: 3.0 (red.); FLU: 2.5 (unfilled disrupt., filled disrupt.)
P15	PPA-U	Hypokinetic dysarthria / + phonological impairment	2.0	PL: 2.5 (low volume); VOQ: 2.0 (breathy, harsh); VOS: 2.5 (devoicing/vocal decay); SP: 2.5 (overall red. art., phonetic errors, phonemic errors, art. fluctuations); FLU: 2.0 (unfilled disrupt., sound-/syllable repetitions); MOD: 2.5 (blurring of syllable boundaries); BEHAV 3.0 (art. effort, other: facial grimacing)
P16	nfvPPA	Apraxia of speech of phonetic-prosodic type	2.0	SP: 2.0 (phonetic errors, phonemic errors); RATE: 3.0 (reduced, prolong. single sounds); FLU: 2.0 (filled disrupt., unfilled disrupt.); MOD: 2.5 (syllabic speech); BEHAV: 2.0 (art. groping, art. effort)

(*table continues*)

Table B1. *(Continued).*

Id	Subtype	MSD syndrome	MSD severity	MSD scale and feature specifications
P18	PPA-U	Hyperkinetic dysarthria / + phonological impairment	0.5	BREATH: 1.5 (short. breath groups, speaking on residual air, other: abrupt/ involuntary inspirations); PL: 2.7 (low volume); VOQ: 3.0 (harsh); VOS: 1.0 (devoicing/vocal decay, changes in voice quality, changes in pitch/ loudness, involuntary vocalizations); SP: 1.0 (phonetic errors, phonemic errors, art. fluctuations, other: facio-oral dyskinesias); FLU: 1.0 (filled disrupt., unfilled disrupt., sound-/ syllable repetitions, other: dyskinetic speech arrest); BEHAV: 1.7 (art. effort, art. groping)
P19	nfvPPA	Apraxia of speech of phonetic-prosodic type	2.5	SP: 2.5 (phonetic errors, phonemic errors); RATE: 3.0 (reduced, prolong. single sounds); FLU: 3.0 (unfilled disrupt.); MOD: 3.0 (syllabic speech)
P20	PPA-U/-CBS	Impoverished speech drive	2.0	PL: 2.5 (high pitch, low volume); VOQ: 2.0 (breathy); VOS: 3.0 (devoicing/vocal decay); SP: 3.0 (overall reduced art., closed art., art. fluctuations); FLU: 2.0 (red.initiation / maintenance of speech); MOD: 2.0 (red. pitch/ loudness mod.)
P24	PPA-U	Neurogenic stuttering / + phonological impairment	1.5	SP: 2.0 (phonemic errors); FLU: 1.0 (sound-/syllable repetitions)
P25	nfvPPA	Apraxia of speech of prosodic type	2.5	SP: 3.0 (phonetic errors); FLU: 2.0 (unfilled disrupt.); MOD: 2.0 (syllabic speech)
P28	lvPPA	Hypokinetic dysarthria / + phonological impairment	2.5	VOS: 2.0 (vocal tremor); SP: 3.0 (phonemic errors); RATE: 3.0 (increased, prolong. single sounds)
P33	nfvPPA	Apraxia of speech of phonetic-prosodic type	2.0	BREATH: 2.7 (speaking on residual air); SP: 2.0 (phonetic errors, phonemic errors); RATE: 2.0 (red., prolong. single sounds); FLU: 2.0 (filled disrupt., unfilled disrupt.); MOD: 2.0 (syllabic speech, red. pitch/ loudness mod.); BEHAV: 2.5 (art. effort, art. groping)

(*table continues*)

Table B1. *(Continued).*

Id	Subtype	MSD syndrome	MSD severity	MSD scale and feature specifications
P34	PPAOS	Apraxia of speech of prosodic type	3.0	VOQ: 3.0 (harsh); SP: 3.0 (phonetic errors); RATE: 3.0 (red.); MOD: 3.0 (syllabic speech)
P37	PPA-U	Impoverished speech drive	2.0	PL: 2.3 (low volume); VOQ: 3.0 (breathy); VOS: 3.3 (devoicing/ vocal decay); SP: 3.0 (overall red. art.); FLU: 2.0 (red. initiation/ maintenance of speech); MOD: 3.3 (red. pitch/ loudness mod.)

Note. PPA = primary progressive aphasia; MSD = motor speech disorder; BREATH = speech breathing; PL = pitch & loudness; VOQ = voice quality; VOS = voice stability; SP = sound production; RES = nasal resonance; RATE = articulation rate; FLU = speech fluency; MOD = prosodic modulation; BEHAV = speech behavior; short. = shortened; aud./stren. = audible/ strenuous; art. = articulation; prolong. = prolonged; disrupt. = disruptions; mod. = modulation; red. = reduced; nfvPPA = nonfluent variant of primary progressive aphasia; PPAOS = primary progressive apraxia of speech; lvPPA = logopenic variant of primary progressive aphasia; PPA-U = primary progressive apraxia unclassified; CBS = corticobasal syndrome; Not determin. = not determined.