RESEARCH ARTICLE

When it is not primary progressive aphasia: A scoping review of spoken language impairment in other neurodegenerative dementias

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Abstract

Background: Progressive difficulties with spoken language occur across the spectrum of degenerative dementia. When not a primary presenting and dominant symptom, language difficulties may be overlooked in favor of more prominent cognitive, behavior, or motor deficits. The aim of this scoping review is to examine the extent and nature of the research evidence describing (1) the spoken language impairments found in non-language led dementias, (2) their impact on everyday living, and (3) the reported language interventions.

Methods: We searched PubMed, MEDLINE, OVID-EMBASE, PsycINFO, and Speech-BITE using terms related to spoken language for the following dementia types: Parkinson's disease dementia (PDD), dementia with Lewy bodies (DLB), progressive supranuclear palsy (PSP), cortico-basal syndrome (CBS), behavior variant frontotemporal dementia (bvFTD), early-onset Alzheimer's disease (EOAD), posterior cortical atrophy (PCA), and motor neuron disease associated with FTD (MND+FTD). Risk of bias was assessed with the QualSyst tool.

Results: Seventy-three eligible studies were included. A wide range of spoken language impairments were reported, involving both linguistic (e.g., syntactic processing) and other cognitive (e.g., sustained attention) underlying mechanisms. Although the severity of these deficits was scarcely reported, in some cases they manifested as non-fluent, dynamic, and global aphasias. No papers in the review described either the impact of these language impairments on everyday living or language therapies to treat them.

Discussion: There is a need to understand better the level of disability produced by language impairment in people living with non–language-led dementias. Our findings suggest three calls for action: (1) research studies should assess the clinical relevance of any spoken language deficits examined, (2) both linguistic and cognitive underlying mechanisms should be fully described (to inform the design of effective language

and behavioral interventions), and (3) trials of language therapy should be conducted in those groups of individuals where significant language impairment is proved.

1 | INTRODUCTION

The primary progressive aphasias (PPA) are a syndromic family of disorders that share the presence of language impairment as the first and most salient feature of the clinical picture.[1] Currently, there are three PPA variants identified: a semantic variant characterized by fluent speech with semantic breakdown; a non-fluent/agrammatical variant, characterized by non-fluent speech with agrammatism and/or apraxia of speech; and a logopenic variant, characterized by word-finding pauses and impaired sentence repetition due to phonological working memory deficits.[1] This taxonomy successfully classifies more than half of all PPA cases, but up to 41% of them show overlapping features that do not fit within the three canonical phenotypes.[2] Despite these difficulties in subtype classification, the recognition of progressive aphasias has been an important step in raising awareness of non–memory-related disabilities associated with dementia. This awareness has contributed to the growth of a new area of research focused on the development of interventions to improve expressive and receptive language abilities in people living with PPA (see Cotelli et al., for a review and meta-analysis of the 10 last years of research in the field[3]).

While a significant step forward, it is crucial to recognize that progressive spoken language difficulties also occur in other neurodegenerative dementia, such as early-onset typical Alzheimer's disease (EOAD),[4] Parkinson's disease dementia (PDD),[5] dementia with Lewy bodies (DLB),[6] posterior cortical atrophy (PCA),[7] behavior variant frontotemporal dementia (bvFTD), and frontotemporal dementia with motor neuron disease (FTD+MND),[8] as well as the atypical parkinsonian syndromes of progressive supranuclear palsy (PSP)[9] and cortico-basal syndrome (CBS).[10] There is a current lack of systematic investigation of the language deficits in these patient groups. In this scoping review we examined and mapped the literature concerning progressive spoken language impairments in degenerative dementias wherein language is not the primary and most prominent symptom (i.e., excluding PPA syndromes). In this review we focused on spoken language rather than writing and reading because this is the most clinically salient feature, and the most commonly used in assessment of and communication with people living with dementia. The language profile (spoken and otherwise) of typical, late-onset Alzheimer's disease is characterized by anomia (with well-defined aphasia emerging only in late stages) and has already been well reported in the literature so it was excluded from this review.[11] Studies looking at vascular dementia were also excluded due to the lack of definitional clarity and consequent heterogeneity (due to variation in lesion type and location) within this clinical identity.[12]

Using a scoping review methodology, we aimed to draw a map of the existing evidence regarding three specific questions: (1) What are the currently reported spoken language impairments in non–language-led dementias? (2) What is the clinical significance of these impairments? That is, the impact on quality of life and/or activities of daily living, and (3) What are the reported language-based interventions for people with non–language-led dementias?

2 | METHODS AND DESIGN

A protocol of this scoping review can be found in a previous publication.[13] The protocol followed the Preferred Reporting Items for Systematic Reviews and Meta-analysis Protocols (PRISMA)-ScR guidelines.[14,15] Using the general framework outlined by Arksey and O'Malley,[16] we systematically scoped the literature about language in people with common forms of dementia (excluding PPA): EOAD, PCA, PDD, DLB, bvFTD, FTD+MND (used to denote both FTD-MND and MND-FTD), PSP, and CBS.

2.1 | Selection of studies

Studies that assess and/or treat spoken language difficulties in the target population were included. The following exclusion criteria applied:

- Studies focused on late-onset Alzheimer's disease (AD), vascular dementia, mild cognitive impairment (MCI), or a diagnosis of a non-neurodegenerative disease.
- Studies in which information regarding the nature of the language impairment was not provided or there was insufficient description of the sample, for example, lack of clarity regarding diagnostic category.
- Studies that examined only writing and/or reading.
- Studies that assessed motor difficulties only (i.e., motor speech, voice, prosody, dysarthria, and/or oral apraxia).
- Articles for which the full text was not readily available or was not written in English.
- Studies where no original findings were presented (e.g., reviews/editorials/letters to the editor).
- Non-peer reviewed material.
- Conference abstracts.
- Errata/correction of no significance to required data.
- Gray literature.

2.2 | Information sources, search, and identification of evidence

The search was conducted on PubMed, MEDLINE, OVID-EMBASE, PsycINFO, and SpeechBITE with an unlimited starting date, up until July 2019. These databases were selected as the most relevant to the topic of the review. The literature included was indexed and written in English. Database-specific conventions and use of multiple search fields and filters were customized for individual databases. Reference lists from key articles and reviews were scrutinized and relevant articles included. The search terms were developed in consultation with experienced researchers and librarians and piloted before conducting the actual search.

The specific electronic search strategy can be found in the study protocol published by Savage et al.[13] The search for PubMed comprised the following terms: (Early-onset Alzheimer's disease OR early onset Alzheimer's disease OR young-onset Alzheimer's disease OR young onset Alzheimer's disease OR Parkinson* dementia OR Parkinson* disease dementia OR dementia with Lewy bodies OR Lewy body dementia OR Posterior Cortical Atrophy OR frontotemporal dementia OR Pick's disease OR Progressive Supranuclear Palsy OR Cortico-basal Syndrome OR cortico-basal degeneration OR corticobasal degeneration OR motor neuron disease OR FTD-MND OR MND-FTD OR ALS-FTD OR FTD-ALS) AND (language impairment OR communication disorder OR aphasia).

All references retrieved were exported into EndNote software version X9, with duplicates removed via the "Find duplicates" function and through visual inspection of title and author. The screening and selection of the candidate articles was conducted using a multi-level title-first method.[17] The primary reviewer (ASG) independently inspected all the citations, while reviewers two and three (AC and RG) independently inspected 50% of the citations each, so that every item was considered by at least two independent reviewers. Screening took place first by title, and then by abstract. Any differences in the agreement were discussed. A 10% sample of the full-text articles selected by the primary reviewer were also screened by reviewers two and three to ensure reliability.

2.3 | Data charting and critical appraisal

Information extracted from each article was recorded in a tailored data-charting form by ASG and discussed with SS. Information items included the study design, diagnoses, reported language impairments and their severity (e.g., as measured by standardized tools or clinician's judgment), clinical significance (i.e., measures of quality of life or impact of aphasia on everyday living), and language-based interventions. Risk of bias within the reported studies was evaluated using the Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields (QualSyst)[18] to assist in determining the feasibility of a future systematic review. A score of 75 in QualSyst is

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RESEARCH IN CONTEXT

1. Systematic Review: We reviewed the literature regarding spoken language impairments in eight non-language led dementias (i.e., excluding primary progressive aphasias [PPA]) using PubMed, MEDLINE, OVID-EMBASE, PsycINFO, and SpeechBITE.

2. Interpretation: Results showed a wide range of deficits reported in non-PPA dementias, including difficulties in single-word processing, sentence processing, and narrative building. In some cases, very severe impairments were reported (e.g., global aphasia). Little mention was found, however, regarding the clinical significance of such deficits or of any behavioral or language therapies to treat them.

3. Future Directions: Our findings highlight the need for future studies to examine how the language deficits impact everyday functioning and quality of life. In addition, to guide effective development of interventions, there is a need for improved understanding of the underlying linguistic and cognitive mechanisms. Importantly, trials of language therapy should be initiated in those groups of individuals in which a significant language impairment is present.

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the threshold for a paper to meet quality criteria to be included in a review.[19]

2.4 | Synthesis of results

Studies were grouped by phenotype and summarized in Tables 1 and 2.

3 | RESULTS

Of the 4672 records identified, 196 full-text articles were assessed, with 73 of them meeting inclusion criteria (see Figure 1 for PRISMA flowchart of study selection).

3.1 | Characteristics of sources of evidence

An overview of the distribution of studies according to design and quality is shown in Figure 2. Table 1 illustrates the characteristics of the studies, including the critical appraisal score produced by QualSyst with 64% of studies scoring 75 and above (good quality).

TABLE 1 Characteristics of papers included

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Parkinson disease dementia (PDD)								
Cummings et al. (1988)	Case-control	Sixteen people with PDD (compared to 35 PD without dementia)	Reduced phrase length, information content, phrase repetition, comprehension, and naming (compared to PD without dementia)	NR	NR	NR	NO	60
Plat et al. (1999)	Case-control	Twenty people with PDD (compared to 59 healthy control and 57 with PD)	PDD impaired in fluency tasks (particularly action naming)	NR	NR	NR	NO	93
Ash et al. (2017)	Case-control	Three PDD/five DLB (among a group of 23 people with PDD)	Slower speech rate, higher mean of length of utterance and poorer report of content	No relationship found between language function and changes in overall cognitive or motor function	NR	NR	NO	54
Frank et al. (1996)	Case-control	Twelve PDD (part of a group of 42 individuals with dementia due to AD, HD, and PD and age-matched controls)	Decline in confrontation naming in moderate dementia PD group	Naming may be reduced due to reduced access to semantic referents	NR	NR	NO	45
Magdalinou et al. (2018)	Case-control	Eighteen people living with PDD (and seven PSP, four CBS)	Reduced verbal fluency and sentence generation (in the three patient groups)	No associated language deficits: executive function performance appeared to play a role; authors suggest a specific effect on verbal output of dopaminergic stimulation	NR	NR	NO	66
Azuma et al. (1997)	Case-control	Fifteen PDD (also 99 PD and 46 healthy controls)	Reduced semantic fluency; some reduction in letter fluency and fluency for proper names in the PDD group	NR	NR	NR	NO	69
Bayles et al. (1993)	Case-control	Twenty people with PDD (among a group of 88 with PD, 21 AD, and 43 healthy controls)	Reduced semantic and letter fluency in the PDD group (scoring similar to the AD group)	NR	NR	NR	NO	83
Lewis et al. (1998)	Case-control	Eight PDD (among a group of 20 PD)	Reduced naming, word definition, ability to interpret ambiguity and figurative language, sentence construction, and semantic verbal fluency compared to PD and normal score in cognitive functioning	NR	NR	NR	NO	83
Suhr et al. (1998)	Case-control	Twenty-six PDD with mild dementia (compared to 31 AD and 14 HD)	Reduced semantic and verbal fluency; less repetition errors in PDD group	NR	NR	NR	NO	83

(Continues)

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Dementia with Lewy bodies/PDD								
Ash et al. (2012)	Case-control	Fourteen DLB/PDD (out of a group including 21 patients with PD and 21 healthy controls)	Speech rate reduced (wpm) in half compared to seniors and significantly reduced compared to PD. More silent pauses and difficulties in grammatical production	Reduced speech rate correlated with measures of between-utterance pauses, executive function and grammatical comprehension (frontal lobe mediated)	NR	NR	NO	80
Ash et al. (2012)b	Case-control	Twenty-nine DLB/PDD (compared to 26 healthy controls)	Deficit in both narrative comprehension and narrative expression	Deficits partially due to material-neutral deficit in organizational executive resources (correlated with prefrontal cortical atrophy)	NR	NR	NO	79
Ash et al. (2011)	Case-control	Fourteen DLB/PDD (compared to 18 PD)	Significant narrative production deficit: impairment in connecting one scene to the next and poor ability to maintain the theme	Deficit related to impairment on measures of executive function and speech fluency	NR	NR	NO	73
Grossman et al. (2012)	Case-control	Seventeen DLB/PDD (compared to 26 PD and 19 healthy controls)	Sentence processing deficit for syntactic ambiguities	Deficit correlates with impairment in working memory	NR	NR	NO	73
Gross et al. (2012)	Case-control	Eight PDD/nine DLB (compared to 16 PD)	Impaired sentence processing	Increased difficulty processing sentences with increase working memory demands	NR	NR	NO	73
Gross et al. (2013)	Case-control	Twelve DLB/PDD (compared to 30 PD and 12 healthy controls)	Impaired script comprehension	Deficits associated to executive impairment	NR	NR	NO	73
Grossman et al. (2017)	Case-control	Fourteen PDD/12 DLB (compared to 30 PD)	Difficulty appreciating narrative organization	Associated with frontal grey matter atrophy	NR	NR	NO	67
Atypical parkinsonisms PSP and CBS								
Santos-Santos et al. (2016)	Cohort (longitudinal)	Five PSP/nine CBS (and 10 controls)	Both groups showed impairment in verbal fluency, naming and sentence comprehension; fewer words per minute; greater syntactical errors; reduced proportion of words in sentences. CBS but not PSP produced fewer narrative words than controls. Worsening in longitudinal assessment without new differences between groups	Phonetic and syntactic levels affected	NR	NR	NO	100

(Continues)

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Rosser and Hodges (1994)	Case-control	Ten PSP (and 10 with AD, 10 with HD)	Impaired fluency: letter fluency more impaired than category fluency	Poorer performances in the PSP group were associated to initiation and retrieval problems secondary to disruption in frontostriatal circuits	NR	NR	NO	79
Lebrun (1986)	Case study	One PSP	Reduced fluency (apart from voice and articulation deficits)	NR	NR	NR	NO	100
Podoll et al. (1991)	Cross-sectional	Six PSP	High rate of self-correction and errors in object naming (errors referring to visually similar objects)	Visual processing defect/misperception likely related to naming errors as no word-finding difficulty was evident (indicated by word-finding pauses, or semantic paraphasias)	NR	NR	NO	83
Robinson et al. (2006)	Case report	One PSP	Dynamic aphasia. Propositional language impaired (preserved naming, repetition and comprehension)	Impairment interpreted as being underpinned by a deficit in the generation of a fluent sequence of novel thought in discourse generation	NR	NR	NO	70
Barker et al. (2018)	Case-control	Five PSP (compared to a group of 30 controls, three AD and two svFTD)	Four PSP showed initial periods of responding followed by periods of poorer performance for both tasks of verbal fluency and spontaneous speech	Decreased energization, defined as the attentional process of initiating and sustaining a response over time	NR	NR	NO	100
Robinson et al. (2015)	Case report experimental	One PSP	Dynamic aphasia	Aphasia not underpinned by a language-specific deficit in selection or planning, but by a domain-general deficit in fluent sequencing of novel thoughts	NR	NR	NO	100
Boeve et al. (2003)	Case report	One PSP	Initial anomia, paragrammatism, verbal hesitancy and slowly produced speech that evolved to full non-fluent aphasia over time	NR	NR	NR	NO	100
Esmonde et al. (1996)	Case series	Three PSP	Dynamic aphasia	Impairment in planning and initiating language output; impairment in letter and category fluency pointed toward a deficit in initiation and retrieval processes	NR	NR	NO	100
Roher et al. (2010)	Cross-sectional	Four PSP	Non-fluent aphasia characterized by marked reduction in propositional speech, fewer speech errors	NR	NR	NR	NO	75

(Continues)

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Daniele et al. (2013)	Case-control	Ten PSP (and 10 healthy controls)	Poorer verbs/actions compared to nouns/objects in several lexical-semantic tasks: confrontation naming, auditory and visual word-picture matching	Deficits in the comprehension of action-verbs from a specific semantic impairment in the category of actions	NR	NR	NO	100
Josephs et al. (2005)	case series	Four PSP with apraxia of speech	C1–Comprehension: mildly impaired. Expression: poor letter fluency. Agrammatism and semantic errors (non-fluent) C2–Expression: poor letter fluency (only participant with no obvious aphasia) C3–Expression: poor letter fluency. Agrammatism and semantic errors (non-fluent) C4–Expression: poor letter fluency. Paragrammatism (anomic aphasia with paragramatic errors)	NR	NR	NR	NO	100
Burrell et al. (2018)	Case-control	Twenty-two PSP (compared to 29 PNFA and 93 healthy controls)	Language impairment was similar in PSP than in patients with non-fluent progressive aphasia (naming, word comprehension, semantic association and syntactic comprehension)	No significant correlation between performance on the SYDBAT subtests and performance on executive or working memory tasks, suggesting not due to executive dysfunction	SYDBAT: PSP performing similar to PNFA in naming, comprehension and semantic association domains and better in repetition	NR	NO	100
Catricala et al. (2019)	Case-control	Seventeen PSP with movement disorder presentation (compared to 21 PD and 27 healthy controls)	More than 50% showed impairment in picture naming, semantic fluency, and sentence and single word comprehension. More than 40% of PSP showed impaired non-word repetition	Affected linguistic levels identified were phonological, lexico-semantic, and discourse-pragmatic	NR	NR	NO	100
Brito-Marques et al. (2011)	case report	One CBS	Difficulties for verbal and semantic fluency	NR	NR	NR	NO	100
Troiani et al. (2011)	Case-control	Eleven CBS (among 32 with neurodegenerative diseases and 14 healthy controls)	Impaired verbal comprehension of quantifiers	Not attributable to visuospatial processing impairment or a primary language deficit but to a specific quantifier comprehension deficit related to damage of the parietal cortex	NR	NR	NO	73

(Continues)

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Graham et al. (2003)	Cross-sectional	Ten CBS	Prevalent phonologic and spelling impairments (a minority of patients also showed semantic memory and naming deficits)	NR	NR	NR	NO	100
Ferrer et al. (2003)	Cross-sectional	Three CBS	C2–loss of fluency and anomia and deficits in comprehension of complex orders C3–no description of the characteristics of language disorder C4–no description of the characteristics of language disorder	NR	NR	NR	NO	100
McMillan et al. (2006)	Case-control	Sixteen CBS (also 23 FTD, 25 AD, and 17 healthy controls)	Quantifier comprehension is impaired in CBS, particularly for first order quantifiers (e.g., at least three flowers)	Comprehension impairment at least partially due to number knowledge impairment in CBS (not to language impairment in the verbal representation of the number or object knowledge) and partially due to working memory deficits	NR	NR	NO	70
Morgan et al. (2011)	Case-control	Thirteen CBS, three PCA, 14 bvFTD	People with CBS and PCA showed impairment in comprehension of cardinal quantifiers. bvFTD patients showed impaired in their comprehension of logical quantifiers	CBS and PCA impairments were partially due to deficits in quantity knowledge; bvFTD impairments correlated with deficits in executive measures	NR	NR	NO	84
Levin et al. (2015)	Cross-sectional	Thirty-eight CBS	Sixteen with aphasia	NR	NR	NR	NO	91
Donovan et al. (2007)	Case report	One CBS	Mild aphasia with abnormal social language usage: lack of awareness of the parameters of conversational turn taking, topic cohesion, and listener feedback	The reported frontal executive dysfunction described in CBS is suggested as the underlying cause of this abnormal language usage	WAB: 89 (mild aphasia)	NR	NO	80
McMonagle et al. (2006)	Case-control	Fifty-five CBS: 19 with motor onset and 36 with cognitive/behavioral onset CBS (PPA and AD group control)	Aphasia	NR	WAB: 88 (mild aphasia) in the motor onset group. WAB: 66 (moderate aphasia) in the cognitive onset group	NR	NO	91

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Takao et al. (2006)	Case report	One CBS	Non-fluent aphasia	NR	NR	NR	NO	80
Gross et al. (2010)	Case-control	Twenty CBS (eight healthy controls)	Impaired discourse, with deficits in narrative theme, global connectedness (impaired in 16/20 subjects with CBS) and reductions in local connectedness	Discourse impairment was related to higher-order integration of visual material (the task was based on a picture story) but not basic visuoperceptual, language, or memory functions	NR	NR	NO	80
Tree & Kay (2008)	Case-report longitudinal	One CBS	Non-fluent aphasia	NR	NR	NR	NO	80
Kertesz et al. (2000)	Cohort (longitudinal)	Thirty-five CBS (15 presented with motor symptoms and 20 with cognitive onset)	Twenty-one with aphasia	NR	Measured with the WAB. The aphasia quotient ranged from 28 to 99 in the group presenting with cognitive disorders and 59 to 99 in the group presenting with movement disorders	NR	NO	100
Grossman et al. (2004)	Case-control	Nine CBS	Naming deficits	Confrontation naming deficits correlate with lexical retrieval and also with visual-spatial function	NR	NR	NO	76
Behavioral variant FTD								
Ash et al. (2019)	Case-control	Fourteen people with bvFTD	Baseline: reduced fluency (reduced wpm but nearly free of speech errors); follow-up: impaired dependent clause production and production of well-formed sentences, slower speech that became simplified with disease progression	NR	NR	NR	NO	80
Cotelli et al. (2006)	Case-control	Sixteen bvFTD, 10 PSP, 10 CBS	Action naming more impaired than object naming for all groups. The discrepancy was larger in PSP and CBS	Suggestive of involvement of action knowledge and action representations	NR	NR	NO	69

(Continues)

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Davis et al. (2010)	Case-control	Twenty-two bvFTD (among a group of 20 healthy controls, 144 NPH and 15 PNFA), BvFTD and PNFA groups were analyzed together	bvFTD/PNFA showed impairment in animal and action naming fluency	NR	NR	NR	NO	73
Pakhomov et al. (2010)	Case-control	bvFTD (N not specified; among a total of 38 with PNFA, svFTD, and lvPPA)	Impaired naming and fluency	NR	NR	NR	NO	69
Saxon et al. (2017)	Retrospective case control study	185 people with bvFTD and 56 ALS-FTD	Agrammatism and impaired sentences comprehension, naming, word finding difficulties in conversation, impaired comprehension and repetition, word repetition, presence of phonological errors, agrammatism, reduced speech output, verbal perseveration and echolalia	NR	NR	NR	NO	83
Snowden et al. (2019)	Case-control	Seventy-one bvFTD (compared to 32 svFTD)	Around 50% bvFTD showed impaired naming and 17% impaired word-picture matching	Compared possible semantic deficit versus executive contribution as basis for naming difficulties in bvFTD. Findings support the view that anomia can arise independently of executive impairment	NR	NR	NO	100
Yunusova et al. (2015)	Case-control	Nine bvFTD (among a group of 33 healthy controls, 85 people with ALS and nine PNFA)	Although total pause time and number of pauses were elevated when reading out loud in bvFTD, phrase duration was normal in this group	Reference to pauses being longer because of cognitive-language impairments	NR	NR	NO	69
d'Honincthun & Pillon (2008)	Case-report	One bvFTD	Verb naming impaired when assessed using static depictions of actions but normal when using videotaped actions or verbal stimuli	Authors make a call for caution about stimuli selection for studies	NR	NR	NO	91
Hardy et al. (2015)	Case-control	Twenty-four bvFTD (compared to 14 svFTD and 18 nfvPPA and 24 healthy individuals)	Deficits in noun and verb naming and single word comprehension and diminished propositional speech	Isolated linguistic components, like verbal semantics were identified (authors corrected by nonverbal executive performance)	NR	NR	NO	69
Silvery et al. (2003)	Case-control	Seventeen bvFTD (compared to 42 AD	Impaired action naming	Noun and verb naming and the gap in between showed correlation with executive tasks	NR	NR	NO	92

(Continues)

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Peelle et al. (2008)	Case-control	Thirty-two SOC/EXEC (compared to 28 PNFA and 28 svFTD)	28% of SOC/EXEC impaired in grammatically complex sentence comprehension	Deficits were attributable to decline in executive cognitive resources	NR	NR	NO	62
Grossman et al. (2005)	Case-control	Eight FTD with executive deficits (compared to five PNFA and three svFTD)	Sentence comprehension difficulties	Correlation with measures of working memory, planning, and inhibition control; speculation that a material-neutral executive resources deficit not dedicated to grammatical processing may play a role in the sentence comprehension deficit	NR	NR	NO	62
Peelle et al. (2007)	Case-control	Seven SOC/EXEC (six people with PNFA and 20 healthy controls)	Difficulties processing sentences–partial sensitivity to grammatical errors but insensitivity to thematic violations	Results were consistent with impairment in the formation of a coherent thematic matrix	NR	NR	NO	83
Ash et al. (2006)	Case-control	Twelve people with FTD SOC/EXEC	Profound difficulty organizing narratives: impaired WPM, accuracy of reported information, global connectedness, and maintenance of theme	Correlated with poor performances on measures of executive resources requiring and organized mental speech	NR	NR	NO	77
Cotelli et al. (2007)	Case-control	Nine bvFTD, 15 PSP, 11 CBS (compared to 4 svFTD, 10 AD, and 10 healthy controls)	Syntactic knowledge impairment found in CBS but not PSP or bvFTD	NR	Performed within normal range in Aachener Aphasia Test	NR	NO	75
Early onset AD								
Imamura et la. (1998)	Cross-sectional	N: 150 AD (using age at onset as a continuous variable)	EOAD worse on word comprehension and sequential commands and rapid decline of naming ability	NR	Measured with WAB. The earlier the age at onset the lower the AQ on the WAB	NR	NO	83
Sa et al. (2012)	Case-control	109 EOAD (compared to 171 LOAD)	No language impairment described	NR	NR	NR	NO	72
Borges et l. (2018)	Case-report	One person with EOAD	Marked difficulties in comprehension and sentence repetition that progressed to global aphasia	NR	Global aphasia 3 years after symptoms onset	NR	NO	30
Selnes et la. (1988)	Case-control	Sixty-one EOAD (compared to 72 LOAD)	No language impairment and no differences in language dysfunction between groups found	NR	NR	NR	NO	77

(Continues)

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Posterior cortical atrophy								
Fitzpatrick et al. (2019)	Case report	One PCA	Logopenic aphasia	NR	NR	NR	NO	40
Steeb et al. (2018)	Case-report longitudinal	One PCA	Baseline: impaired fluency for nouns; follow-up: impaired fluency for nouns and verbs	NR	NR	NR	NO	80
Shebani et al. (2017)	Case-control	Ten PCA (compared to one svFTD)	Greater deficits on word processing than for spatial prepositions in PCA (e.g., under, through)	NR	NR	NR	NO	75
Crutch et al. (2013)	Case-control	15 PCA (compared to 7 LPA and 18 healthy individuals)	Impairment across all language domains, more prominent for anomia, reduced fluency and speech rate. PCA performed better than LPA on tasks of comprehension and spontaneous speech	NR	NR	NR	NO	69
Magnin et al. (2012)	Case-control	Nine PCA	8/9 PCA participants showed a logopenic profile, with anomia, reductions in fluency, and a length-dependent deficit	NR	NR	NR	NO	83
Suárez-González et al. (2019)	Case-control	Eight PCA (compared to 21 AD and 18 healthy controls)	Impaired comprehension of measurement units (e.g., grams)	NR	NR	NR	NO	69
Motor neuron disease with FTD								
Bak et al. (2001)	Cross-sectional	2/6 MND-dementia-aphasia	In one case, severe reduction in spontaneous speech, semantic paraphasia, communicating in a "telegraphic" way. The other case shows echolalia and naming inaccuracies.	NR	NR	NR	NO	73

(Continues)

TABLE 1 (Continued)

Author	Design	Sample	Language impairments found	Associated mechanisms	Severity of language impairment	Impact of impairments	Intervention study?	QualSyst (%)
Rakowic & Hodges (1998)	Case-control	Three MND-FTD (compared to two MND with aphasia and 13 MND)	Only 2/3 patients were formally assessed–deficits on verbal fluency, picture naming, word-picture matching, semantics, and grammar were found	NR	NR	NR	NO	79
Bak & Hodges (2004)	Cross-sectional	Seven MND-dementia	Poverty of spontaneous speech, severe impairment on syntactic comprehension, a consistently large impairment noticed for verbs (both naming and comprehension)	NR	described as severe	NR	NO	81
Kamminga et al. (2016)	Case-control	Fifteen FTD-ALS (compared to 20 ALS, 27 PNFA, and 23 controls)	Impaired syntactic comprehension	NR	NR	NR	NO	79

Abbreviations: AD, Alzheimer's disease; ALS, amyotrophic lateral sclerosis; AQ, aphasia quotient; bvFTD, behavioral variant frontotemporal dementia; CBS, corticobasal syndrome; DLB, dementia with Lewy bodies; EOAD, early-onset Alzheimer's disease; FTD, frontotemporal dementia; HD, Huntington's disease; LOAD, late-onset Alzheimer's disease; LPA, logopenic aphasia; lvPPA, language variant primary progressive aphasia; MND, motor neuron disease; nfvPPA, non-fluent variant of primary progressive aphasia; NPH, normal pressure hydrocephalus; NO, xxxxxxxx; NR, no reported; PCA, posterior cortical atrophy; PDD, Parkinson's disease dementia; PNFA, progressive non-fluent aphasia; PPA, primary progressive aphasia; PSP, progressive supranuclear palsy; QualSyst, Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields; SOC/EXEC, disorder of social comportment and executive processing (this is the term used by the authors of the corresponding papers); svFTD, semantic variant frontotemporal dementia; SYDBAT, Sydney Language Battery; WAB, Western Aphasia Battery; WPM, words per minute.

TABLE 2 Summary of language impairments reported on each patient subgroup

Language impairment described	PDD (N = 16)	DLB (N = 8)	PSP (N = 17)	CBS (N = 18)	bvFTD (N = 16)	EOAD (N = 4)	PCA (N = 7)	FTD-MND (N = 5)
Single word processing								
Naming	3 (18%)	–	4 (23%)	7 (38%)	9 (56%)	1 (25%)	3 (42%)	2 (40%)
Verbal fluency	6 (37%)	–	6 (35%)	6 (33%)	1 (6%)	–	4 (57%)	1 (20%)
Verb and/or noun processing	1 (6%)	–	2 (11%)	1 (5%)	4 (25%)	–	1 (14%)	1 (20%)
Semantic processing[a]	2 (12%)	–	–	3 (16%)	1 (6%)	–	3 (42%)	1 (20%)
Sentence level processing and narrative								
Syntactic processing	3 (18%)	2 (25%)	4 (23%)	1 (5%)	3 (18%)	–	–	3 (60%)
Verbal expressive language[b]	13 (81%)	8 (100%)	3 (17%)	1 (5%)	3 (18%)	1 (25%)	3 (42%)	2 (40%)
Auditory comprehension	4 (25%)	5 (62%)	4 (23%)	5 (27%)	6 (37%)	1 (25%)	3 (42%)	1 (20%)
Aphasia syndromes								
Aphasia (as reported by authors)[c]	–	–	3 (17%)	4 (22%)		1 (25%)	–	–
Profile compatible with non-fluent/agrammatic aphasia	–	–	3 (17%)	2 (11%)		–	–	–
Profile compatible with logopenic aphasia	–	–					3 (42%)	–

Abbreviations: bvFTD, behavioral variant frontotemporal dementia; CBS, corticobasal syndrome; DLB, dementia with Lewy bodies; EOAD, early-onset Alzheimer's disease; FTD, frontotemporal dementia; MND, motor neuron disease; N, total number of papers; PCA, posterior cortical atrophy; PDD, Parkinson's disease dementia; PSP, progressive supranuclear palsy.

[a]Concepts mostly related to quantity and/or space.
[b]For example, pauses, phrase length, ability to maintain topic, building narrative, etc.
[c]For example, dynamic, global, or non-specified aphasia.

3.2 | Synthesis of results

3.2.1 | Language impairments reported

A summary of the spoken language impairments reported for every phenotype is available in Table 2.

Parkinson's disease dementia
There were 16 articles describing spoken language impairments in PDD. These included difficulties at both basic and complex levels of language processing. At a single word level, three of them reported difficulties with naming[20,21,22] and six with verbal fluency,[22,23,24,25,26,27] with deficits most commonly found in semantic fluency[22,24] and action fluency.[27]

For phrase- and sentence-level processing, two studies identified reduced words per minute,[28,29] while another study described reduced phrase length and impaired phrase repetition.[20] People with PDD also were found to make more pauses during speech,[29] show a higher mean length of utterance,[28] and have poorer topic maintenance.[30] Reduced sentence generation[23] and construction[22] were also reported. Receptive language difficulties were also found with respect to grammar,[22,29,34] sentence processing,[35] and semantics.[22 34] Three studies reported impaired comprehension of sentences[20,32,33] or scripts.[33]

Nine studies examined different elements of the narrative process in PDD. These included deficits in narrative organization,[31] production,[30] and conveying content.[20,28,32]

Severity of language impairment. None of the PDD studies reported the severity of the language impairments identified.

Dementia with Lewy bodies
We identified eight articles describing language impairments in DLB, which included deficits in both comprehension and expression,[30,31,33,35] with specific reductions in speech rate associated with pausing and grammar difficulties,[29,34] as well as topic maintenance.[30]

Severity of language impairment. None of the DLB studies reported measures of severity for the language impairments found.

Progressive supranuclear palsy
We found 17 studies describing spoken language difficulties in PSP. These included reports of dynamic[36,37,38] and non-fluent aphasias[39,40,41] along with a wide range of discrete deficits in expressive and receptive language.

At a single word level, some of the deficits found included difficulties with comprehension and non-word repetition,[43] reduced verbal fluency,[23,40,43,44,45,58] anomia,[43,46,48,49] and the use of verbs/actions.[47]

For phrase and sentence level, processing difficulties with syntactic knowledge[50] have been reported, as well as the presence of paragrammatism,[46,40] agrammatism,[40] and impaired sentence comprehension.[43] Breakdown in verbal production was also described in studies reporting decreased energization (loss of sustained attention

PRISMA Flow Diagram

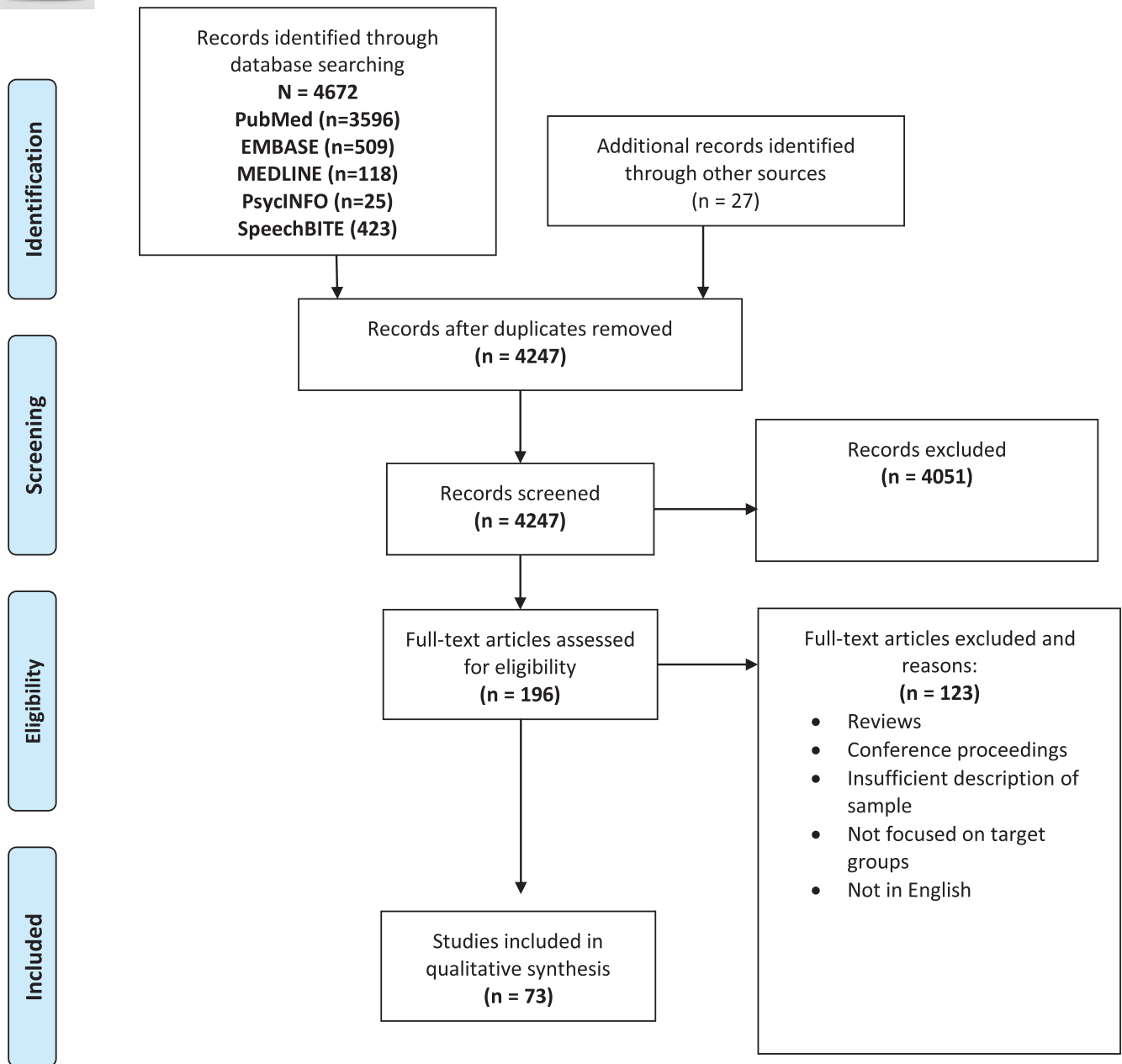**Identification**

Records identified through database searching
N = 4672
PubMed (n=3596)
EMBASE (n=509)
MEDLINE (n=118)
PsycINFO (n=25)
SpeechBITE (423)

Additional records identified through other sources
(n = 27)

Screening

Records after duplicates removed
(n = 4247)

Records screened
(n = 4247)

Records excluded
(n = 4051)

Eligibility

Full-text articles assessed for eligibility
(n = 196)

Full-text articles excluded and reasons:
(n = 123)
- Reviews
- Conference proceedings
- Insufficient description of sample
- Not focused on target groups
- Not in English

Included

Studies included in qualitative synthesis
(n = 73)

FIGURE 1 PRISMA flow diagram

over time that affects verbal communication)[42] and hesitant and slow speech production.[46]

Severity of language impairment. One paper examined the severity of language impairment in a group of 22 people with PSP as compared to a group of people with the non-fluent variant of PPA (nfvPPA)[41] finding that the severity of impairment did not differ between groups (although there was no indication of whether this level was mild, moderate, or severe)

Cortico-basal degeneration

We found 18 studies describing spoken language difficulties in CBS. Four papers reported an aphasia syndrome in CBS without providing further information regarding the details of the aphasia profile,[10,51,52,53,65] with two further studies identifying a language impairment resembling nfvPPA.[54,55] Six other papers reported difficulties with fluency and/or anomia,[23,56–59] with an additional study identifying impairments specifically in action naming.[49] Phonologic impairments,[60] impairment of syntactic knowledge,[50] abnormal

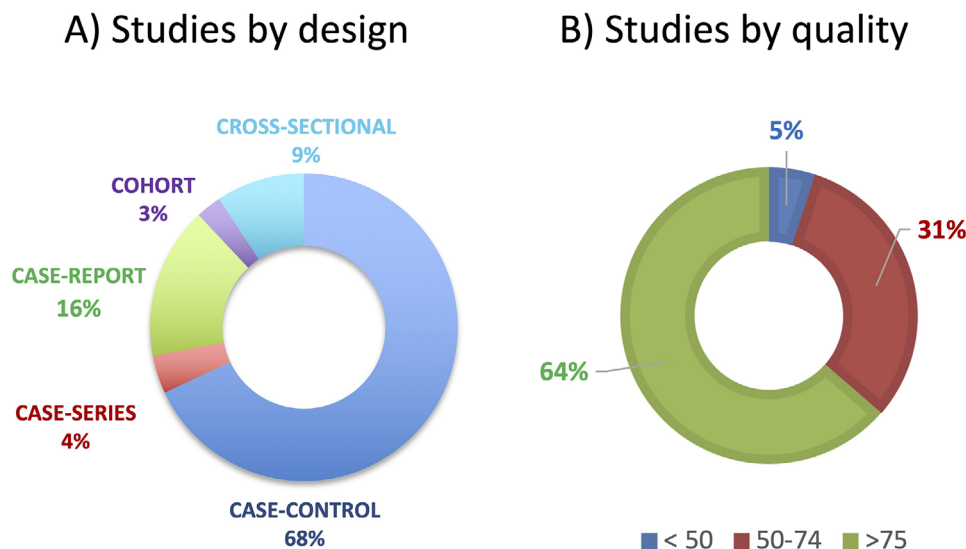## A) Studies by design

B) Studies by quality

FIGURE 2 Overview of the distribution of studies according to design and quality. A, Studies by design. Case-report are considered studies where n = 1; case series are studies in which the paper contains multiple case reports; cohort studies were considered longitudinal studies of a group of patients (a case series followed over time); cross-sectional studies were defined as a group of patients examined at one point in time (or multiple groups of patients at one point in time); case-control studies corresponded to cross-sectional study that compared to a healthy group or other disease group. B, Studies by quality. The definition of quality based on the summary score (SS) obtained on Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields (QualSyst) is strong (SS of >80), good (SS of 71–79), adequate (SS of 50–70) and limited (SS of <50)[19]

use of the pragmatics of language,[52] and deficits in global connectedness of narrative during discourse[61] were shown in different samples.

In addition, difficulties in verbal comprehension were also found in relation to comprehending sentences,[58] complex orders,[56] and quantifiers.[62,63,64]

Severity of language impairment. Three studies reported the aphasia quotient (AQ) in CBS as calculated by the Western Aphasia Battery (WAB), with patients scoring in the mild or moderate range.[52,53,65]

Behavioral variant frontotemporal dementia

Sixteen studies examined language impairment in bvFTD. Nine studies reported impairments in naming[49,66–72] with four of these studies specifically identifying action naming as a deficit.[49,66,71,72] Other impairments included errors in word repetition, phonological errors, reduced speech output, verbal perseverations and echolalia,[69] and difficulties with organizing narratives[73] and with grammar.[69,74]

Several studies also describe impaired comprehension for single words,[66,69] sentences,[69,75,76,77] or logical quantifiers.[64] Total pause time and number of pauses in speech was elevated in the bvFTD group studied by Yunusova et al.[78]

Severity of language impairment. Pakhomov et al.,[70] found that the bvFTD patients in their study were similarly impaired to people with nfvPPA in the category fluency tasks. While this study compared performances between bvFTD and nfvPPA, no studies specifically reported severity levels as measured by standardized tools.

Early-onset Alzheimer's disease

Four studies examined the language deficits in EOAD. Two of them did not find language impairment in EOAD.[79,80] In contrast, another found that people with younger onset showed poorer comprehension of words, sentences, and sequential commands than those with later onset AD.[81] The fourth study described a patient who developed a "full aphasia syndrome" over time.[82]

Severity of language impairment. Although no papers were found describing the severity of language impairments in this group, Imamura et al.[81] suggest that earlier age of onset may be associated with greater severity of aphasia.

Posterior cortical atrophy

Seven studies examined spoken language impairments in people with PCA. A language profile similar to that seen in people with logopenic aphasia was reported in three studies,[83,84,85] but with milder impact on comprehension and spontaneous speech.[84] One paper highlighted impaired fluency for nouns and verbs.[86] The remaining three studies identified additional impairments in comprehending words related to space and quantity.[64,87,88]

Severity of language impairment. No papers were found describing the severity of language impairments in this group.

Motor neuron disease combined with frontotemporal dementia

Five studies reported spoken language difficulties in people with a dual diagnosis of FTD and MND. In the largest study to date,[69] involving 56 people with FTD and MND, impairments were identified in:

word finding, confrontational naming, single-word and sentence-level comprehension, with reduced speech production, perseverations, and echolalia. Additional studies have also identified deficits in verbal fluency, confrontational naming, semantic and grammar[89] syntactic comprehension,[90,91] and naming and comprehension of verbs.[91] Two studies reported poverty of spontaneous speech.[91,92]

Severity of language impairment. One paper[91] described the deficits in their patient as severe, evolving to mutism "within a few weeks."

Clinical significance of language impairments: impact on daily activities or quality of life

No papers selected for review reported impact of language impairments on activities of daily living or quality of life.

Reported language-based interventions

No papers selected for review reported language-based interventions.

4 | DISCUSSION

This scoping review shows evidence of a wide range of spoken language impairments within the eight degenerative dementia syndromes examined. The clinical significance of these language deficits was not reported across studies (i.e., their impact on the quality of life or activities of daily living of the patients). Likewise, we did not find studies describing language-based interventions for these patients. The most frequently studied patients in this review were those with the atypical parkinsonism syndromes, PSP and CBS. While the relatively high numbers of studies on PSP and CBS may seem surprising considering the low prevalence of these disorders, the presence of non-fluent aphasia in both people with PSP and CBS has been clearly demonstrated in the literature and incorporated into diagnostic guidelines.[94,95] This knowledge may have contributed to raised awareness about the language impairments in this population, resulting in a larger number of research studies in this group. Although a large volume of language research exists for people who have PD without dementia,[93] the relatively modest number of studies in people with PDD/DLB could be due to a tendency to focus on more prominent cognitive deficits (e.g., executive difficulties) and neuropsychiatric features in these conditions. Likewise, the limited attention given to EOAD may reflect an overshadowing by other cognitive and behavioral symptoms. Small numbers of studies for patients with PCA and MND+FTD are less surprising, given the low prevalence of these phenotypes and difficulty in gathering large samples.

Research question 1: What are the currently reported spoken language impairments in non-language led dementias?

The scoping review revealed a wide range of language deficits across the dementia subtypes examined. Within the PDD/DLB spectrum,

these often included increased pausing, longer latencies of utterances, and reduced phrase length in speech production. Such features appear consistent with the general mental slowing and bradyphrenia observed in parkinsonian syndromes. Poor ability to maintain topic and difficulties in narrative organization and comprehension were also commonly reported and linked with impaired executive function, a cognitive domain known to be impacted in PDD.[96] Other impairments, such as in grammar, appeared purely linguistic, without associations with additional cognitive deficits.

As with previous reviews in PSP and CBS,[94] language impairments consistent with non-fluent aphasia were reported in these two syndromes. Although impairments in verbal fluency, naming, and syntactic processing were also commonly reported in PDD, bvFTD, PCA, and FTD+MND, a full nfvPPA profile was never described in these dementia subtypes. In PSP, additional difficulties with comprehension were reported in association with a loss of sustained attention.[42]

This review also found that people with bvFTD and those with MND+FTD share similar characteristics of language dysfunction with regard to perseverations, echolalia, and difficulties with grammar and comprehension.[69] In addition, people with bvFTD may show poor organization of speech, phonological errors, and impairments in repetition and action naming. As the literature base for MND+FTD is currently small, it is still unclear whether these additional features will also be found in MND+FTD. Given the similarities in their dysexecutive features, one might predict overlapping language impairments. However, the additional life-threatening features and complex care requirements that characterize MND+FTD may limit the perceived relevance of researching these language issues in the context of the broader clinical picture.

Poorer ability to process verbs and actions has been associated with damage of motor cortices, whereas difficulties with phonology and comprehension of quantifiers described in CBS are associated with disruption of posterior cortical regions.[62] Interestingly, impairments in verb processing and action naming were observed in a number of dementia syndromes including bvFTD, PDD, PSP, and CBS. Evidence from both PD and FTD suggest that such deficits are associated with changes in the frontostriatal motor network.[97-98]

We identified only four studies looking at the language profile of EOAD,[79,80,81,82] with mixed reports over the presence of language dysfunction. One study showed impaired comprehension for words, sentences, and commands,[81] while another described a patient who developed full aphasia.[82] The remaining two studies, however, did not identify any language impairments. Given both the paucity of studies and discrepancies found, further research into EOAD is warranted to determine the nature of language impairments and whether such difficulties significantly limit communication.

Atrophy encroaching the posterior portion of the left superior and middle temporal gyri and the inferior parietal lobe is an anatomical marker for logopenic aphasia[1] but it is also present in PCA. This perhaps explains the logopenic language profile (albeit milder than that in logopenic variant PPA) exhibited in PCA.[84,85] In addition, the extended inferior parietal atrophy of PCA may play a pivotal role in

the impairment of quantifiers and magnitude knowledge, similar to that described in people living with CBS.

Research question 2: What is the clinical significance of these language impairments?

Although the extent of the language dysfunction described in certain cases indicates substantial disability for some individuals (e.g., resulting in mutism or global aphasia), no studies directly measured the impact of language difficulties on activities of daily living or quality of life. In part, this may arise due to the lack of suitable tools that can effectively capture or monitor how language impairments affect everyday living and well-being. The lack of development and validation of such questionnaires or inventories may reflect the limited attention received to date within the literature compared to other variables (e.g., descriptions of deficits, classification). Notably, a quality of life and life participation approach is becoming increasingly recognized as a recommended management approach for PPA.[100,101] This might accelerate the creation of new measures aimed to capture the impact of language impairment in daily living and quality of life in PPA. Such progress will likely have the potential to benefit the broader field of progressive language disorders.

Research question 3: What are the reported language-based interventions?

No language-based interventions were reported in any of the eight dementia syndromes examined, including in patients with PSP or CBS. This is surprising considering that not only is non-fluent aphasia common in PSP, but numerous therapeutic studies have been conducted in nfvPPA with positive results.[3,99] Likewise, no investigations were identified to support the language difficulties of PCA, despite its overlap with the logogenic variant of PPA. People living with PCA, PSP, and CBS may therefore likely benefit from the language therapies currently available to treat PPA.[3] It appears that both linguistic and other cognitive causes can be simultaneously involved in the language dysfunction of people with CBS, PSP, PDD, DLB, and bvFTD. If this is true, a close and comprehensive assessment of both cognitive and linguistic functions and their potential involvement in communication outcomes may be needed to develop effective interventions. For instance, it will be unhelpful administering a purely language-based therapy to a patient whose language impairment is predominantly caused by a disruption in working memory.

5 | LIMITATIONS

Following the standard methodology of a scoping review, data were gathered from a wide range of studies and methods to create a map of current evidence. As we were also interested in the quality of avail-

able data to inform a future systematic review, we extended beyond a typical scoping review to also conduct a risk of bias assessment. While the ratings given to the majority of studies suggested satisfactory methodological rigor, it is important to note that approximately one third of studies identified fell below the accepted threshold. This indicates a need for more controlled studies in the future, to confirm and strengthen confidence in the current findings.

6 | CONCLUSIONS

This scoping review describes the volume, extent, and nature of the research evidence regarding spoken language impairment in non–language-led dementias. From this piece of work, three calls for action emerge: (1) research studies should seek to assess the clinical relevance of any spoken language deficits examined; (2) both linguistic and cognitive underlying mechanisms should be explored and sufficiently described (to assist in the design of effective language and behavioral interventions); and (3) trials of language therapies previously found useful in the PPA should be undertaken in patients with PSP, CBS, and PCA.

CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

DATA AVAILABILITY STATEMENT

Data sharing in not applicable to this article as no new data were created or analyzed in this study.

REFERENCES

1. Gorno-Tempini ML, Hillis AE, Weintraub S, et al. Classification of primary progressive aphasia and its variants. *Neurology*. 2011;76(11):1006-1014.
2. Sajjadi SA, Patterson K, Arnold RJ, Watson PC, Nestor PJ. Primary progressive aphasia: a tale of two syndromes and the rest. *Neurology*. 2012;78(21):1670-1677.
3. Cotelli M, Mannenti R, Ferrari C, Gobbi E, Macis A, Cappa SF. Effectiveness of language training and non-invasive brain stimulation on oral and written naming performance in primary progressive aphasia: a meta-analysis and systematic review. *Neurosci Biobehav Rev*. 2020(108):498-525.
4. Imamura T, Takatsuki Y, Fujimori M, et al. Age at onset and language disturbances in Alzheimer's disease. *Neuropsychologia*. 1998;36(9):945-949.
5. Liu L, Luo X-G, Dy C-L, et al. Characteristics of language impairment in Parkinson's disease and its influencing factors. *Transl Neurodegener*. 2015;4(1):2.

6. Teichmann M, Migliaccio R, Kas A, Dubois B. Logopenic progressive aphasia beyond Alzheimer's–An evolution towards dementia with Lewy bodies. *J Neurol Neurosurg Psychiatry*. 2013;84(1):113-114.

7. Crutch SJ, Lehmann M, Warren JD, Rohrer JD. The language profile of posterior cortical atrophy. *J Neurol Neurosurg Psychiatry*. 2013;84(4):460-466.

8. Saxon JA, Thompson JC, Jones M, et al. Examinig the language and behavioural profile in FTD and ALS-FTD. *J Neurol Neurosurg Psychiatry*. 2017;88:675-680.

9. Burrell JR, Ballard KJ, Halliday GM, Hodges JR. Aphasia in progressive supranuclear palsy: as severe as progressive non-fluent aphasia. *J Alzheimers Dis JAD*. 2018;61(2):705-715.

10. McMonagle P, Blair M, Kertesz A. Corticobasal degeneration and progressive aphasia. *Neurology*. 2006;67(8):1444-1451.

11. Weintraub S, Wicklund AH, Salmon DP. The neuropsychological profile of Alzheimer disease. *Cold Spring Harb Perspect Med*. 2012;2(4):a006171.

12. O'Brien JT, Thomas A. Vascular dementia. *Lancet*. 2015;386(10004):1698-1706.

13. Savage SA, Suárez-González A, Cassani A, Gopalan R, Stott J. Non-primary progressive language impairment in neurodegenerative conditions: protocol for a scoping review. *Syst Rev*. 2021;10(1):32.

14. Moher D, Stewart L, Shekelle P. Implementing PRISMA-P: recommendations for prospective authors. *Syst Rev*. 2016;5(1):15.

15. Tricco AC, Lillie E, Zarin W, et al. PRISMA Extension for Scoping Reviews (PRISMA-ScR): checklist and explanation. *Ann Intern Med*. 2018;169(7):467-473.

16. Arksey H, O'Malley L. Scoping studies: towards a methodological framework. *Int J Soc Res Methodol*. 2005;8(1):19-32.

17. Mateen FJ, Oh J, Tergas AI, Bhayani NH, Kamdar BB. Titles versus titles and abstracts for initial screening of articles for systematic reviews. *Clin Epidemiol*. 2013;5:89-95.

18. Kmet L, Lee R. *Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields*. Heritage Foundation for Medical Research; 2004.

19. Lee L, Packer TL, Tang SH, Girdler S. Self-management education programs for age-related macular degeneration: a systematic review. *Australas J Ageing*. 2008;27(4):170-176.

20. Cummings JL, Darkins A, Mendez M, Hill MA, Benson DF. Alzheimer's disease and Parkinson's disease: comparison of speech and language alterations. *Neurology*. 1988;38(5):680-684.

21. Frank EM, McDade HL, Scott WK. Naming in dementia secondary to Parkinson's Huntington's and Alzheimer's diseases. *J Commun Disord*. 1996;29(3):183-197.

22. Lewis FM, LaPointe LL, Murdoch BE, Chenery HJ. Lewis language impairment in Parkinson's disease. *Aphasiology*. 1998;12(3):193-206.

23. Magdalinou NK, Golden HL, Nicholas JM, et al. Verbal Adynamia in Parkinsonian syndromes: behavioral correlates and neuroanatomical substrate. *Neurocase*. 2018;24(4):204-212.

24. Azuma T, Bayles KA, Cruz RF, et al. Comparing the difficulty of letter, semantic, and name fluency tasks for normal elderly and patients with Parkinson's disease. *Neuropsychology*. 1997;11(4):488-497.

25. Bayles KA, Trosset MW, Tomoeda CK, Montgomery EB, Wilson J. Generative naming in Parkinson disease patients. *J Clin Exp Neuropsychol*. 1993;15(4):547-562.

26. Suhr JA, Jones RD. Letter and semantic fluency in Alzheimer's, Huntington's and Parkinson's dementias. *Arch Clin Neuropsychol*. 1998;13(5):447-454.

27. Piatt AL, Fields JA, Paolo AM, Koller WC, Troster AI. Lexical, semantic, and action verbal fluency in Parkinson's disease with and without dementia. *J Clin Exp Neuropsychol*. 1999;21(4):435-443.

28. Ash S, Jester C, York C, et al. Longitudinal decline in speech production in Parkinson's disease spectrum disorders. *Brain Lang*. 2017;171:42-51.

29. Ash S, McMillan C, Gross RG, et al. Impairments of speech fluency in Lewy body spectrum disorder. *Brain Lang*. 2012;120(3):290-302.

30. Ash S, McMillan C, Gross RG, et al. The organization of narrative discourse in Lewy body spectrum disorder. *Brain Lang*. 2011;119(1):30-41.

31. Grossman M, Irwin DJ, Jester C, et al. Narrative oraganization deficit in Lewy body disorders is related to Alzheimer's pathology. *Front Neurosci*. 2017;11:53.

32. Ash S, Xie SX, Gross RG, et al. The organisation and anatomy of narrative comprehension and expression in Lewy body spectrum disorders. *Neuropsychology*. 2012;26(3):368-384.

33. Gross RG, Camp E, McMillan CT, et al. Impairment of script comprehension in Lewy body spectrum disorders. *Brain Lang*. 2013;125(3):330-343.

34. Grossman M, Gross RG, Moore P, et al. Difficulty processing syntactic ambiguities in Lewy body spectrum disorder. *Brain Lang*. 2012;120(1):52-60.

35. Gross RG, McMillan CT, Chandrasekaran K, et al. Sentence processing in Lewy body spectrum disorder, the role of working memory. *Brain Cogn*. 2012;78(2):85-93.

36. Robinson G, Shallice T, Cipolotti L. Dynamic aphasia in progressive supranuclear palsy: a deficit in generating a fluent sequence of novel thought. *Neuropsychologia*. 2006;44(8):1344-1360.

37. Robinson GA, Spooner D, Harrinson WJ. Frontal dynamic aphasia in progressive supranuclear palsy: distinguishing between generation and fluent sequencing of novel thoughts. *Neuropsychologia*. 2015;77:62-75.

38. Esmonde T, Giles E, Xuereb J, Hodges J. Progressive supranuclear palsy presenting with dynamic aphasia. *J Neurol Neurosurg Psychiatry*. 1996;60(4):403-410.

39. Rohrer JD, Paviour D, Bronstein AM, O'Sullivan SS, Lees A, Warren JD. Progressive supranuclear palsy syndrome presenting as progressive nonfluent aphasia: a neuropsychological and neuroimaging analysis. *Mov Disord*. 2010;25(2):179-188. 30.

40. Josephs KA, Boeve BF, Duffy JR, et al. Atypical progressive supranuclear palsy underlying progressive apraxia of speech and nonfluent aphasia. *Neurocase*. 2005;11(4):283-296.

41. Burrell JR, Ballard KJ, Halliday GM, Hodges JR. Aphasia in progressive supranuclear palsy: as severe as progressive non-fluent aphasia. *J Alzheimers Dis*. 2018;61(2):705-715.

42. Barker MS, Nelson NL, O'Sullivan JD, Adam R, Robinson GA. Energization and spoken language production: evidence from progressive T supranuclear palsy. *Neuropsychologia*. 2018;119:349-362.

43. Catricala E, Boschi V, Cuoco S, et al. The language profile of PSP. *Cortex*. 2019;115:294-308.

44. Rosser A, Hodges JR. Initial letter and semantic category fluency in Alzheimer's disease, Huntington's disease, and progressive supranuclear palsy. *Neurosurg Psychiatry*. 1994;57(11):1389-1394.

45. Lebrun Y, Devreux F, Rousseau JJ. Language and speech in a patient with a clinical diagnosis of progressive supranuclear palsy. *Brain Lang*. 1986;27(2):247-256.

46. Boeve B, Dickson D, Duffy D, et al. Progressive nonfluent aphasia and subsequent aphasic dementia associated with atypical progressive supranuclear palsy pathology. *Eur Neurol*. 2003;49(2):72-78.

47. Daniele A, Barbier A, Di Giuda D, et al. Selective impairment of action-verb naming and comprehension in progressive supranuclear palsy. *Cortex*. 2013;49(4):948-960.

48. Podoll K, Schwarz M, Noth J. Language functions in progressive supranuclear palsy. *Brain*. 1991:1457-1472. Pt 3.

49. Cotelli M, Borroni B, Manenti R, et al. Action and object naming in frontotemporal dementia, progressive supranuclear palsy, and corticobasal degeneration. *Neuropsychology*. 2006;20(5):558-565.

50. Cotelli M, Borroni B, Manenti R, et al. Universal grammar in the frontotemporal dementia spectrum: evidence of a selective disor-

der in the corticobasal degeneration syndrome. *Neuropsychologia*. 2007;45(13):3015-3023.

51. Levin J, Bak TH, Rominger A, et al. The association of aphasia and right-sided motor impairment in corticobasal syndrome. *J Neurol*. 2015;262(10):2241-2246.

52. Donovan NJ, Kendall DL, Moore AB. Why consider impaired social language usage in a case of corticobasal degeneration? *Clin Neuropsychol*. 2007;21(1):190-203.

53. Kertesz A, Martinez-Lage P, Davidson W, Munoz DG. The corticobasal degeneration syndrome overlaps progressive aphasia and frontotemporal dementia. *Neurology*. 2000;55(9):1368-1375.

54. Takao M, Tsuchiya K, Mimura M, et al. Corticobasal degeneration as cause of progressive non-fluent aphasia: clinical, radiological and pathological study of an autopsy case. *Neuropathology*. 2006;26(6):569-578.

55. Tree JJ, Kay J. Longitudinal assessment of language and memory impairments in pathologically confirmed cortico-basal ganglionic degeneration. *Cortex*. 2008;44(9):1234-1247.

56. Ferrer I, Hernandez I, Boada M, et al. Primary progressive aphasia as the initial manifestation of corticobasal degeneration and unusual tauopathie. *Acta Neuropathol*. 2003;106(5):419-435.

57. Grossman M, McMillan C, Moore P, et al. What's in a name: voxel-based morphometric analyses of MRI and naming difficulty in Alzheimer's disease, frontotemporal dementia and corticobasal degeneration. *Brain*. 2004;127:628-649. Pt 3.

58. Santos-Santos MA, Mandelli ML, Binney RJ, et al. Cross-sectional and longitudinal features of non-fluent/agrammatic primary progressive aphasia with underlying corticobasal degeneration or progressive supranuclear palsy pathology. *JAMA Neurol*. 2016;73(6):733-742.

59. Brito-Marques PR, Vieira-Mello RJ, Montenegro L, et al. Clinico-pathologic analysis of progressive non-fluent aphasia and corticobasal degeneration. *Dement Neuropsychol*. 2011;5(2):135-141.

60. Graham NL, Bak T, Patterson K, Hodges JR. Language function and dysfunction in corticobasal degeneration. *Neurology*. 2003;61(4):493-499.

61. Gross RG, Ash S, McMillan CT, et al. Impaired information integration contributes to communication difficulty in corticobasal syndrome. *Cogn Behav Neurol*. 2010;23(1):1-7.

62. Troiani V, Clark R, Grossman M. Impaired verbal comprehension of quantifiers in corticobasal syndrome. *Neuropsychology*. 2011;25(2):159-165.

63. McMillan CT, Clark R, Moore P, Grossman M. Quantifier comprehension in corticobasal degeneration. *Brain Cogn*. 2006;62(3):250-260.

64. Morgan B, Gross R, Clark R, et al. Some is not enough: quantifier comprehension in corticobasal syndrome and behevioral variant frontotemproal dementia. *Neuropsychologia*. 2011;49(13):3532-3541.

65. McMonagle P, Blair M, Kertesz A. Corticobasal degeneration and progressive aphasia. *Neurology*. 2006;67(8):1444-1451.

66. Hardy CJD, Buckley AH, Downey L, et al. The language profile of behavioural variant frontotemporal dementia. *J Alzheimers Dis*. 2015;50(2):359-371.

67. D'Honincthun P, Pillon A. Verb comprehension and naming in frontotemporal degeneration—The role of the static depiction of actions. *Cortex*. 2008;44(7):834-847.

68. Snowden JS, Harris JM, Saxon JA, et al. Naming and conceptual understanding in frontotemporal dementia. *Cortex*. 2019;120:22-35.

69. Saxon JA, Thompson JC, Jones M, et al. Examining the language and behavioural profile in FTD and ALS-FTD. *J Neurol Neurosurg Psychiatry*. 2017;88(8):675-680.

70. Pakhomov SVS, Smith GE, Chacon D, et al. Computerized analysis of speech and language to identify psycholinguistic correlates of frontotemporal lobar degeneration. *Cogn Behav Neurol*. 2010;23(3):165-177.

71. Davis C, Heidler-Gary J, Gottesman RF, et al. Action versus animal naming fluency in subcortical dementia, frontal dementias and Alzheimer disease. *Neurocase*. 2010;16(3):259-266.

72. Silveri MC, Ivigni BL, Cappa A, et al. Impairment of verb processing in frontal variant frontotemporal dementia: a dysexecutive symptom. *Dement Geriatr Cogn Disord*. 2003;16(4):296-300.

73. Ash S, Moore P, Antani S. Trying to tell a tale: discourse impairments in progressive aphasia and frontotemporal dementia. *Neurology*. 2006;66(9):1405-1413.

74. Ash S, Naomi Nevler N, Jeffrey Phillips J, et al. A longitudinal study of speech production in primary progressive aphasia and behavioral variant frontotemporal dementia. *Brain Lang*. 2019;194:46-57.

75. Peelle JE, Troiani V, Gee J, et al. Sentence comprehension and voxel-based morphometry in progressive nonfluent aphasia, semantic dementia. *J Neurolinguistics*. 2008;21(5):418-432.

76. Peelle JE, Cooke A, Moore P, Vesely L, Grossman M. Syntactic and thematic components of sentence processing in progressive nonfluent aphasia and frontotemporal dementia. *J Neurolinguistics*. 2007;20(6):482-494.

77. Grossman M, Rhee J, Moore P. Sentence processing in frontotemporal dementia. *Cortex*. 2005;41(6):764-777.

78. Yunusova Y, Graham NL, Shellikeri S, et al. Profiling speech and pausing in amyotrophic lateral sclerosis (ALS) and Frontotemporal Dementia (FTD). *PLoS One*. 2016;11(1):e0147573.

79. Sa F, Pinto P, Cunha C, et al. Differences between early and late onset Alzheimer's disease in neuropsychological tests. *Front Neurol*. 2012;3:81.

80. Selnes OA, Carson K, Rovner B, Gordon B. Language dysfunction in early—and late—onset possible Alzheimer's disease. *Neurology*. 1988;38(7):1053-1056.

81. Imamura T, Takatsuki Y, Fujimori M, et al. Age at onset and language disturbances in Alzheimer's disease. *Neuropsychologia*. 1998;36(9):945-949.

82. Borges MK, Lopes TN, Biella MM, Siquieira A, Mauer S, Aprahamian I. Early-onset Alzheimer's disease (EOAD) with Aphasia A case report. *Front Psychiatry*. 2018;9:469.

83. Fitzpatrick D, Blanco-Campal A, Kyne L. A case of overlap posterior cortical atrophy and logopenic variant primary progressive aphasia. *Neurologist*. 2019;24(2):62-65.

84. Crutch SJ, Lehmann M, Warren JD, Rohrer JD. The language profile of posterior cortical atrophy. *J Neurol Neurosurg Psychiatry*. 2013;84(4):460-466.

85. Magnin E, Sylvestre G, Lenoir F, et al. Logopenic syndrome in posterior cortical atrophy. *J Neurol*. 2013;260(2):528-533.

86. Steeb B, Garcia-Cordero I, Huizing M, et al. Progressive compromise of nouns and verbs in posterior cortical atrophy. *Front Psychol*. 2018;9:1345.

87. Shebani Z, Patterson K, Nestor PJ, et al. Semantic word category processing in semantic dementia and posterior cortical atrophy. *Cortex*. 2017;93:92-106.

88. Suárez-González A, Hoffman P, Crutch SJ. Where words meet numbers: comprehension of measurement unit terms in posterior cortical atrophy. *Neuropsychologia*. 2019;131:216-222.

89. Rakowic W, Hodges J. Dementia and aphasia in motor neuron disease: an underrecognised association?. *J Neurol Neurosurg Psychiatry*. 1998;65(6):881-889.

90. Kamminga J, Leslie FVC, Hsieh S, et al. Syntactic comprehension deficits across the FTD-ALS continuum. *Neurobiol Aging*. 2016;41:11-18.

91. Bak TH, Hodges JR. The effects of motor neurone disease on language: further evidence. *Brain Lang*. 2004;89(2):354-361.

92. Bak TH, O'Donovan DG, Huereb JH, Boniface S, Hodges JR. Selective impairment of verb processing associated with pathological changes in Brodmann areas 44 and 45. *Brain*. 2001;124:103-120. Pt 1.

93. Smith KM, Caplan DN. Communication impairment in Parkinson's disease: impact of motor and cognitive symptoms on speech and language. *Brain Lang*. 2018;185:38-46.

94. Peterson KA, Patterson K, Rowe JB. Language impairment in progressive supranuclear palsy and corticobasal syndrome. *J Neurol*. 2019;268(3):796-809.

95. Höglinger GU, Respondek G, Stamelou M, et al. Clinical diagnosis of progressive supranuclear palsy: the movement disorder society criteria. *Mov Disord*. 2017;32(6):853-864.

96. Schapira A, Chaudhuri K, Jenner P. Non-motor features of Parkinson disease. *Nat Rev Neurosci*. 2017;18:435-450.

97. Birba A, Garcia-Cordero I, Kozono G. Losing ground: frontostriatal atrophy disrupts language embodiment in Parkinson's and Huntington's disease. *Neurosci Biobehav Rev*. 2017;80:673-687.

98. Gardini S, Venneri A, McGeown WJ, et al. Brain activation patterns characterizing different phases of motor action: execution, choice and ideation. *Brain Topogr*. 2006;29(5):679-692.

99. Henry LH, Hubbard HI, Grasso SM, et al. Retraining speech production and fluency in non-fluent/agrammatic primary progressive aphasia. *Brain*. 2018;141(6):1799-1814.

100. Rogalski EJ, Khayum B. A life participation approach to primary progressive aphasia intervention. *Semin Speech Lang*. 2018;39(3):284-296.

101. Ruggero L, Nickels L, Croot K. Quality of life in primary progressive aphasia: what do we know and what can we do next?. *Aphasiology*. 2019;33(5):498-519.