HHS Public Access

Author manuscript

Aphasiology. Author manuscript; available in PMC 2021 January 01.

Published in final edited form as:

Aphasiology. 2020 ; 34(12): 1456–1470. doi:10.1080/02687038.2019.1670330.

Visuomotor Figure Construction and Visual Figure Delayed Recall and Recognition in Primary Progressive Aphasia

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Abstract

Background—Individuals with primary progressive aphasia (PPA) develop visuospatial deficits over time, and those with logopenic variant (lvPPA) are at greatest risk of developing such deficits. However, not all previous studies of visuospatial deficits in PPA have ensured equivalent duration of disease across variants and few have measured deficits longitudinally.

Aims—The aims of our study were to: 1) investigate differences in baseline visuomotor figure construction, visual figure delayed recall, and figure recognition in PPA variants with similar symptom duration at baseline, and 2) explore patterns of decline in these areas.

Methods & Procedures—Ninety-three individuals with PPA [39 lvPPA, 24 nonfluent agrammatic PPA (nfaPPA), and 30 semantic variant PPA (svPPA)] were administered the Benson Complex Figure Copy, Benson Complex Figure Delay (Recall), and Benson Figure Recognition. Thirty individuals completed this testing 3 to 47 months post baseline.

Outcome & Results—Participants with lvPPA and svPPA showed lower mean scores than those with nfaPPA on visual figure delayed recall at baseline, even though there were no differences in estimated time from disease onset or correlation with disease severity as reflected by naming performance, $F(2, 90) = 5.78$, $p < .004$. Those with nfaPPA performed significantly better than those with lvPPA, Tukey HSD $p < .05$, and those with svPPA, Tukey HSD $p < .01$. There were no

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Disclosure Statement

No potential conflict of interest was reported by the authors.

Data Availability Statement

Data will be made available upon request.

differences between variants in rate of decline in visuomotor figure construction, visual figure delayed recall, and figure recognition.

Conclusions—These findings revealed relatively spared visuospatial memory in nfaPPA, which may aid in the differential diagnosis of PPA and contribute to designing therapy or compensatory strategies

Keywords

Primary progressive aphasia; nonfluent agrammatic primary progressive aphasia; semantic variant primary progressive aphasia; logopenic variant primary progressive aphasia; visuospatial memory

Introduction

Visuospatial abilities and visual memory enable our nonverbal understanding of the world. These complex abilities are subserved by the primary visual cortex, posterior parietal cortex, prefrontal cortex, the dorsal and ventral streams of vision processing, and their interconnections. The dual stream model of vision processing is well established: a ventral stream projecting from the primary visual cortex to the inferior and medial temporal areas and limbic system to process object identity (the “what” pathway), and a dorsal stream projecting from the primary visual cortex to the parietal lobe with two important functions: to process object locations relative to the observer and other objects in the environment (the “where” pathway) (Ungerleider & Mishskin, 1982), and to integrate visual input and motor responses (the “how” stream) to facilitate reaching and grasping in space (Milner & Goodale, 1995).

Immediate and delayed figure copying is the current gold standard for visuospatial and visual memory assessment. Different anatomical substrates and cognitive mechanisms contribute to performance on complex figure copying. Complex figure copying is influenced by parietally-mediated abilities (visual spatial perception integration and spatial working memory) via the dorsal stream. It may also be influenced by temporally mediated processes of visual recognition (e.g., recognizing parts of a complex figure as a flag or face) via the ventral stream.

Impairment of visuospatial abilities and visual memory is common in neurodegenerative disease and can compromise function in a broad array of activities of daily living, such as orienting oneself, manipulating objects, judging distances, using a map, and parking and driving a car. Impairment in visuospatial processing and memory using figure copying has been documented in dementia. Possin, Laluz, Alcantar, Miller and Kramer (2011) found that those with Alzheimer’s disease (AD) scored significantly lower on figure copying than those with behavioral variant frontotemporal dementia (bvFTD) and healthy controls, and that there was a trend for those with bvFTD to score lower than healthy controls.

Visuospatial abilities and visual memory also have been studied in primary progressive aphasia (PPA), although to a lesser extent than the language characteristics of the PPA variants (e.g., Gorno-Tempini et al., 2004; Gorno-Tempini et al., 2011; Josephs et al., 2008; Mesulam, Wieneke, Thompson, Rogalski, & Weintraub, 2012; Rogalski et al., 2011; Rohrer

et al., 2013). Individuals with logopenic variant PPA (lvPPA) have been found to perform similarly to individuals with AD on complex figure copy and recall tasks (Foxe, Irish, Hodges, & Piquet, 2013; Foxe et al., 2016). Other research has compared visuospatial abilities in the different variants of PPA. Butts et al. (2015) administered a comprehensive neuropsychological profile to 91 individuals with PPA [51 with lvPPA; 27 with agrammatic PPA (agPPA), 13 with semantic variant PPA (svPPA)] who were not significantly different for age, education, gender, age at symptom onset, aphasia severity, or disease duration, although the method for establishing the latter is not specified. They found group differences in visual learning and memory, as well as in executive and visuospatial function, with the lvPPA group performing more poorly than either the agPPA or svPPA groups on multiple measures. The groups differed on the Rey-Osterrieth Complex Figure Test (Osterrieth, 1944), with the svPPA group scoring higher (M = 11.3, SD = 1.12) than both the lvPPA (M = 6.3, SD = 0.61) and agPPA (M = 7.4, SD = 0.81) groups. Watson et al. (2018) investigated visuospatial cognition across several tasks in 156 individuals with PPA [34 with lvPPA, 48 with nonfluent PPA (nfaPPA), 74 with svPPA] and 79 healthy controls; follow up testing was available for 83 participants with PPA. After adjusting for differences in age, education, and dementia severity, the authors found that those with lvPPA had significantly lower scores on a visuospatial factor and the most impaired composite scores. On the Benson Complex Figure Copy (Kramer et al., 2003; Possin et al., 2011; alz.washington.edu/NONMEMBER/FTLD/FTLD-NpsychInstructions.pdf), those with lvPPA and svPPA scored significantly lower than controls, and those with lvPPA scored significantly lower than those with svPPA. On the Benson Complex Figure Delay (Recall), all participants with PPA scored significantly lower than controls; those with lvPPA and svPPA scored significantly lower than those with nfaPPA. On the Benson Complex Figure Copy, the svPPA group showed a small improvement in performance over time whereas the other two PPA groups had small declines in performance which were significant group differences.

The emergence of cognitive and behavioral deficits over time, beyond the language impairments that are characteristic of PPA (see Gorno-Tempini et al., 2011), is consistent with the spread of atrophy into cortical regions beyond those which typify each of the PPA variants at onset. Atrophy of the left temporoparietal junction is characteristic of lvPPA; atrophy of the left posterior fronto-insular regions [inferior frontal gyrus (IFG), insula, premotor, supplementary motor areas] is typical of nfaPPA; and bilateral atrophy of the ventral and lateral portions of the anterior temporal lobe, left greater than right, is present in svPPA (Gorno-Tempini et al., 2004; Josephs et al., 2006; Wilson et al., 2011). Longitudinal imaging studies reveal that while cortical atrophy remains left hemisphere lateralized over time in PPA, there is progression into the right hemisphere and worsening of clinical deficits (Rogalski et al., 2011; Rohrer et al., 2013). Rogalski et al. (2013) found that atrophy in lvPPA extended over time throughout most of the left perisylvian cortex, and developed in the left dorsolateral prefrontal cortex and right temporoparietal region. In nfaPPA, left hemisphere atrophy progressed to involve more of the dorsal and ventral prefrontal cortex, temporoparietal cortex, and the anterior temporal lobe; right hemisphere atrophy spread to include the IFG, temporoparietal regions, and a larger region of dorsal prefrontal cortex. In svPPA, left hemisphere atrophy extended to include the entire temporal lobe, the

temporoparietal cortex, as well as frontal regions, and right hemisphere atrophy involved more of the temporal lobe.

In sum, prior studies document that, although PPA is a condition with disproportionate impairment of speech and language associated with atrophy of the frontal and temporal regions of the left hemisphere (Gorno-Tempini et al., 2011; Mesulam, 2001; 2013), many individuals develop visuospatial deficits over time, and those with lvPPA are at greatest risk of developing such deficits. This result likely reflects greater atrophy in parietal cortex in lvPPA relative to the other variants, as parietal cortex is particularly important for visuospatial skills and working memory via the dorsal stream. However, equivalency of disease duration (i.e., disease severity) across PPA variants is not established in all previous studies, and longitudinal assessment of deficits is captured in only a limited number of studies; thus the differences between groups could just reflect differences in severity of disease.

The aims of our study were to: 1) investigate differences in baseline visuomotor figure construction, visual figure delayed recall, and figure recognition in the PPA variants with similar symptom duration at baseline, and 2) to explore patterns of decline in these areas. We hypothesized that individuals with lvPPA would demonstrate poorer performance on baseline visual figure recall and figure recognition and greater decline on these tasks than nfaPPA and svPPA because the underlying pattern of atrophy in lvPPA involves the left parietal cortex early in the disease course and involves the right temporoparietal regions with disease progression. Confirmation of this hypothesis would aid in the classification of PPA and the development of recommendations for restriction of activities, such as driving.

Materials and Methods

Participants

Prior to initiation of the study, the data collection, review, and analysis were approved by the Johns Hopkins Medicine Institutional Review Board. All participants or their spouses provided written informed consent and agreed to participate. These individuals were evaluated in one author's (AEH) outpatient cognitive neurology clinic. Ninety-three individuals with PPA (*M* age = 69.15 years, *SD* = 7.61; *M* education = 15.78 years, *SD* = 2.51 for *n* = 86; *M* symptom duration = 44.94 months, *SD* = 23.20; 57% female) completed baseline testing. There were 39 individuals with lvPPA, 24 individuals with nfaPPA, and 30 individuals with svPPA (Table 1). Thirty individuals (14 lvPPA, 10 nfaPPA, 6 svPPA) completed follow up testing (*M* age = 68.07 years, *SD* = 7.40; *M* education = 16.10 years, *SD* = 2.68; *M* symptom duration = 50.07 months, *SD* = 25.09; 60% female) (Table 2). PPA subtype was identified on the basis of history, comprehensive neurological examination, imaging, and a battery of cognitive/language tests. Testing was completed based on participant tolerance and included the following: Phonemic Verbal Fluency; Oral Word Reading (regular and irregular words); Semantic Word Picture Matching (Rogalsky, Love, Driscoll, Anderson, & Hickok, 2011); Semantic Associates Test from the Northwestern Naming Battery, experimental edition (Thompson, Lukic, King, Mesulam, & Weintraub, 2012); JHU Anagram Task; Sentence Repetition Test; short form of the Pyramids and Palm Trees Test (PPTT; Breining, Lala et al., 2015; Howard & Patterson, 1992); Kissing and

Dancing Test (Bak & Hodges, 2003); Noun and Verb Naming Tests (Thompson et al., 2012); Sentence Reading Test; Spelling to Dictation; short form of the Boston Naming Test (BNT) (Mack, Freed, Williams, & Henderson, 1992); Hopkins Assessment of Naming Actions (HANA; Breining, Tippett et al., 2015); Picture Word Verification (Caramazza & Hillis, 1990); and Cookie Theft description from the Boston Diagnostic Aphasia Battery (BDAE) (Goodglass, Kaplan, & Barresi, 2001). This battery (including unpublished subtests) is an expansion of the FTLD Module to the Uniform Data Set, of the National Alzheimer's Coordinating Center (2013; alz.washington.edu) from the National Institute on Aging (NIA, a US Government Health Institute). Some patients were also administered the Apraxia Battery for Adults (Dabul, 2000); in others assessment of speech and limb praxis was done as a part of the comprehensive neurological examination (Table 3). Symptom duration was 24 months or greater (M = 44.94 months, SD = 23.20, median = 36 months, range 24 – 144 months). Patients were classified using consensus criteria for each variant (Gorno-Tempini et al., 2011).

Methods

Participants underwent testing on two occasions. We focused on performance on the Benson Complex Figure Copy, Benson Complex Figure Delay (Recall), and Benson Figure Recognition (Kramer et al., 2003; Possin et al., 2011; alz.washington.edu/NONMEMBER/FTLD/FTLD-NpsychInstructions.pdf). Testing was administered by clinical and research staff trained and supervised by an experienced cognitive behavioral neurologist (AEH). Twenty-four of 93 Benson Complex Figure Copy were rescored by one author (DCT) to assess inter-rater reliability. For the Benson Complex Figure Copy, participants were given a pen and paper and were asked to copy a complex geometric figure. There was no limit placed on response time. Participants completed the task using their preferred hand; no one had impairment of their upper extremities which compromised their ability to draw. Performance was scored on a scale from 0 to 17 to capture accuracy and placement of design elements. When participants completed copying the figure, they then viewed the figure for 5 seconds and were told to remember the figure so that they could draw it from memory later in the session. After a 10–15 minute interval of continued testing, participants were given a pen and paper again, and asked to draw the figure from memory. The same scoring guidelines were used on the recall task as for the copying task. After completion of the drawing from recall task, participants were asked to identify the figure from an array of four options. They earned 1 point for a correct response and 0 points for an incorrect response.

The Boston Naming Test (BNT) (short form, Mack et al., 1992) was administered at baseline for comparison with scores on the Benson Complex Figure Copy and Benson Complex Figure Delay (Recall) to investigate whether performance on visuomotor construction and visual figure delayed recall tasks are markers of disease severity rather than distinct characteristics of each PPA variant. On the short form of the BNT, participants were asked to verbally name 30 line drawings of objects (score range 0–30). Objects ranged from high familiarity items, such as "bed," to low frequency items, such as "sphinx." If a participant experienced difficulty naming a pictured object, a phonemic cue was provided; however, these responses after cuing were not included in the total correct.

Data Analysis

We defined *decline* as: percent correct at the final test session minus percent correct at the initial test session. We defined *rate of decline* as: decline divided by the number of months between initial and final test sessions. We defined *rapid decliners* as those with mean decline equal to or less than −1.5 (negative change per month). We defined those with *no decline* as those with mean decline equal to or greater than 0. We used chi square to identify associations between PPA variants and dichotomized scores on Benson Complex Figure Copy, Benson Complex Figure Delay (Recall), and Benson Figure Recognition, and between PPA variants and distributions of decline. We tested differences in mean baseline test scores between PPA variants and differences in mean rate of decline between PPA variants using ANOVA. Pearson product-moment correlation coefficients were computed to assess the relationship between scores on the BNT, and scores on the Benson Complex Figure Copy and Benson Complex Figure Delay (Recall). Percent agreement (within 1 point) and Pearson-product moment correlation coefficients were calculated for inter-rater reliability for Benson Complex Figure Copy.

Results

Tables 1 and 2 show the age, sex, education, and symptom duration for individuals tested as baseline and at follow up. Symptom duration was defined as number of months between participants and/or caregivers first noticing symptoms and the time of assessment. The groups were not significantly different on these characteristics at baseline, age: $F(2, 90) = 3.02$, $p = .054$; education: $F(2, 85) = 2.93$, $p = .059$; symptom duration: $F(2, 90) = 0.62$, $p = .540$; sex: $X^2(2, N = 93) = 0.65$, $p = .722$, or at follow up, age: $F(2, 27) = 0.28$, $p = .758$; education: $F(2, 27) = 0.12$, $p = .887$; symptom duration: $F(2, 27) = 0.22$, $p = .804$; sex: $X^2(2, N = 30) = 3.93$, $p = .140$.

Percent agreement (within 1 point) was 75% (18/24) and Pearson-product moment correlation coefficient was 0.945, $p < .0001$, for the Benson Complex Figure Copy.

The PPA variants were significantly different on performance on the BNT, $F(2, 90) = 15.51$, $p < .0001$. Those with nfaPPA scored significantly higher on the BNT than those with lvPPA, Tukey HSD $p < .01$, and those with svPPA, Tukey HSD $p < .01$. Those with lvPPA scored significantly higher on the BNT than those with svPPA, Tukey HSD $p < .05$, (Table 4).

There were weak positive correlations between the BNT and the Benson Figure Copy for each of the PPA variants and for participants overall, $r(91) = 0.335$, $p = .001$, (Table 5), and between the BNT and the Benson Complex Figure Delay (Recall) for each of the PPA variants and for participants overall, $r(91) = 0.374$, $p = .0002$, (Table 6).

When performance on the Benson Complex Figure Copy was dichotomized into performance equal to or less than 14 of 17 points (greater than −1 *SD* below the mean for healthy controls of similar age and education as participants) and greater than 14 points (within −1 *SD* from the mean for healthy controls of similar age and education as participants) based on data for healthy controls from Watson et al. (2018) (*M* for healthy

controls = 15.72, *SD* = 1.25, *n* = 79, *M* age = 64.60 years, *SD* = 8.08, *M* education = 17.47 years, *SD* = 2.08), there were significant differences between the PPA variants, $X^2(2, N = 93) = 7.77$, $p = .021$. Ninety-two percent of those with nfaPPA scored greater than 14 points whereas 79% of those with lvPPA and 60% of those with svPPA scored greater than 14 points (Table 7). However, there were no significant differences on mean scores between PPA variants on this test, $F(2, 90) = 2.02$, $p = .133$, (Table 8).

When scores on the Benson Complex Figure Delay (Recall) were dichotomized into performance equal to or less than 10 of 17 points (greater than −1 *SD* below the mean for healthy controls of similar age and education as participants) and greater than 10 points (within −1 *SD* from the mean for healthy controls of similar age and education as participants) based on data for healthy controls from Watson et al. (2018) (*M* for healthy controls = 12.68, *SD* = 2.55, *n* = 79, *M* age = 64.60 years, *SD* = 8.08, *M* education = 17.47 years, *SD* = 2.08), there were significant differences between the PPA variants, X^2 (2, $N = 93) = 12.48$, $p = .002$. Fifty-eight percent of those with nfaPPA scored greater than 10 points whereas 18% of those with lvPPA and 23% of those with svPPA scored greater than 10 points (Table 7). There were also significant differences in mean scores on the Benson Complex Figure Delay (Recall), $F(2, 90) = 5.78$, $p < .004$. Those with nfaPPA performed significantly better than those with lvPPA, Tukey HSD $p < .05$, and those with svPPA, Tukey HSD $p < .01$. There were no significant differences on figure delay (recall) between lvPPA and svPPA (Table 8).

There were no significant differences on figure recognition between PPA variants, $X^2(2, N = 93) = 5.78$, p =.055, (Table 8).

There were no differences between PPA variants in the mean rates of decline on figure copy, $F(2, 27) = 0.64$, $p = .535$, figure delay (recall), $F(2, 27) = 0.28$, $p = .758$, or in figure recognition at baseline versus follow up, $X^2(2, N = 30) = 3.86$, $p = .695$, (Table 9).

There were rapid decliners and slow decliners among all three variants on figure copy and figure delay (recall) (Tables 10–11). The distribution among variants did not differ significantly by chi square, figure copy: $X^2(2, N = 30) = 1.84$, $p = .764$; figure delay: $X^2(2, N = 30) = 1.31$, $p = .859$. At least half of the participants in all PPA variants demonstrated stable performance over time on figure copy and figure delay (recall).

Discussion

PPA subtypes were distinguishable by different patterns of performance on visual figure delayed recall at initial testing, when there were no significant differences in estimated time from disease onset. Delayed figure copying was relatively spared in nfaPPA, and significantly more impaired in lvPPA and svPPA. This finding is consistent with the results of the study by Watson et al. (2018). There was also an association between PPA variants and scores on immediate figure copying with a greater percentage of those with nfaPPA scoring within normal limits than those with lvPPA and svPPA.

Our results are consistent with the patterns of atrophy typically associated with the PPA variants. In lvPPA, there is left temporo-parietal atrophy (Gorno-Tempini, et al., 2004;

Josephs et al., 2006; Wilson et al., 2011). Progression of this variant is associated with extension of atrophy into the left temporal, parietal, frontal and caudate areas, and in the right posterior cingulate cortex/precuneus (Rohrer et al., 2013). Semantic variant PPA is associated with atrophy in ventrolateral anterior temporal lobes bilaterally, usually greater atrophy on the left (Gorno-Tempini et al., 2004; Wilson et al., 2011). In our study, higher percentages of those with lvPPA and svPPA scored below normal limits on immediate figure copying than those with nfaPPA. Those with lvPPA may have difficulty on this task because of disruption of the dorsal stream of vision processing, involving the parietal lobe. Poor figure copying has been reported to correlate with right parietal atrophy in AD, which is the most common underlying neuropathology of lvPPA (Possin et al., 2011). Difficulty on this task demonstrated by those with svPPA may be attributable to disruption of the ventral stream of vision processing, involving the temporal lobe. In nfaPPA, these brain regions are relatively spared with neuroimaging abnormalities of the left posterior frontal region (Gorno-Tempini et al., 2004; Josephs et al., 2006; Wilson et al., 2011) as well as atrophy in the insula, premotor, and supplementary motor areas (Gorno-Tempini et al., 2011; Josephs et al., 2008; Wilson et al., 2011). It is possible that impairments of visuomotor figure construction, visual figure delayed recall, and figure recognition may develop with disease progression of nfaPPA as atrophy extends to the dorsolateral prefrontal cortex, inferiorly into the superior temporal cortex, medially into orbital and anterior cingulate regions and posteriorly along the Sylvian fissure into the parietal lobe later in this variant (Grossman, 2010). Similarly, on delayed figure copying, those with nfaPPA in our study demonstrated significantly better performance than those with lvPPA and svPPA. This may be explained because their disease had not progressed sufficiently to involve the posterior parietal cortex as occurs in lvPPA.

In addition to being consistent with patterns of atrophy in PPA, performance on delayed figure copying may aid in the differential diagnosis of PPA variants. Currently, speech and language characteristics form the inclusion criteria for the each of the three phenotypic clinical presentations of PPA. Impaired repetition is a hallmark of lvPPA; agrammatism and apraxia of speech are features of nfaPPA; and impaired semantic knowledge is characteristic of svPPA. Nevertheless, differential diagnosis can be challenging. Sajjadi, Patterson, Arnold, Watson, and Nestor (2012) reported that 19 of 46 participants with PPA (41%) fulfilled the diagnostic criteria for more than one variant or were too impaired to fulfill the criteria for any variant. Wickland et al. (2014) reported that 26 of 84 individuals with PPA (31%) did not meet minimum diagnostic criteria for classification of any variant and hence were labeled as unclassified when applying test data to consensus criteria. Distinguishing the PPA variants is complicated because some speech and language deficits are characteristics of more than one variant. For example, naming is impaired in all three PPA variants, however, there may be different causes of impaired naming. People with lvPPA may have impaired access to the spoken word form; those with svPPA have impaired access to modality-independent semantics; while nfaPPA may have either impaired access to the spoken word form or impaired motor speech (Budd et al., 2010). Another example is repetition impairment which can be seen in lvPPA and nfaPPA. Impaired repetition may be secondary to impaired working memory in lvPPA or to apraxia of speech in nfaPPA, thereby obscuring the distinction between lvPPA versus nfaPPA. Mesulam and Weintraub (2014) point out that

correct classification of PPA is important because the variants are associated with AD versus frontotemporal lobar degeneration (FTLD) pathology, with differing trajectories of behavioral manifestations and consequent management. Although there is heterogeneity in underlying pathologies, lvPPA is typically associated with AD pathology (Giannini et al., 2017; Gorno-Tempini et al., 2008); nfaPPA is most often associated with tau positive pathology (Irwin, Trojanawski, & Grossman, 2013); and svPPA is commonly associated with frontotemporal lobar degeneration-ubiquitin (FTLD-U) (Grossman et al., 2008; Kertesz et al., 2005; Knopman et al., 2005), and its variant, FTLD-TDP-43 (Snowden et al., 2007). Macoir, Lavoie, Laforce, Brambati, and Wilson (2017) advise that it is important to identify behavioral and non-language cognitive changes with disease progression in PPA to aid in clinical care. Thus, performance on visuospatial memory may facilitate appropriate diagnosis of lvPPA versus nfaPPA with relatively spared delayed figure copying helping to identify those with nfaPPA.

We considered whether impaired performance on visuomotor figure construction and visual figure delayed recall were non-language cognitive changes reflective of disease severity, rather than complementary diagnostic features of the PPA variants. We explored whether there was a correlation between scores on the BNT and scores on the Benson Figure Copy and Benson Figure Delay (Recall). As expected, those with svPPA performed significantly more poorly on the BNT than those with lvPPA and nfaPPA. However, naming abilities and figure copying (both immediate and delayed) were only weakly correlated for the group as a whole and for the PPA variants. Moreover, we found greatest visuospatial deficits in lvPPA, despite their having milder deficits in naming than the svPPA group. These results suggest that performance on visuomotor construction and visual figure delayed recall tasks are not simply markers of disease severity in PPA.

Performance on visuomotor figure construction and visual figure delayed recall may have implications for treatment. In an earlier study, we found that, across language tests, the most precipitous rates of decline occurred in nfaPPA, followed by svPPA, then lvPPA (Sebastian et al., 2018), likely because of apraxia of speech in nfaPPA. In contrast, in this study, many participants in all PPA variants demonstrated stable performance over time on immediate and delayed figure copying. This result may suggest avenues for speech and language treatment, capitalizing on visual modalities, especially for those with nfaPPA as immediate and delayed figure copying were relatively spared in this group. There is growing evidence that speech and language rehabilitation is beneficial in PPA (Tippett, Hillis, & Tsapkini, 2015) and that particular approaches are best suited to specific variants. Henry et al. (2018) reported successful implementation of script training in nfaPPA. Meyer, Tippett, Turner, and Friedman (2018) reported that long term treatment effects were more robust in the orthographic treatment condition and for those with svPPA than other variants. A spaced retrieval treatment approach in which individuals with aphasia view pictured stimuli as part of treatment for anomia (Fridriksson, Holland, Beeson, & Morrow, 2005) may have therapeutic potential for those with PPA, especially for those with nfaPPA. In addition, assessment of immediate and working memory, via the visual modality, may provide insight into individuals' abilities to learn and use new communication strategies, including low and high technology alternative/augmentative modes of communication.

Limitations of the study include the fact that we did not have follow up data for all participants. Some individuals did not return for follow up in the clinic, were not able to complete testing tasks, or declined to complete portions of language testing. This limitation may have undermined our ability to detect significant differences in rate of decline across variants. Another limitation is that we have no validated measure of “disease severity” or “aphasia severity” for PPA. We attempted to capture disease severity by calculating symptom duration defined as the number of months between participants and/or their caregivers first noticing symptoms and the time of assessment. Our PPA participants had similar symptom durations; however, these are estimated subjective values given that symptom duration depends highly on when patients and caregivers first note symptoms and seek out medical care. Also, because most people with PPA have impaired naming, irrespective of variant, naming error rate is often used as a marker of disease severity. Our PPA participants were significantly different on the BNT; however, there was no correlation with immediate or delayed figure copying.

Despite the limitations, this study highlights visuomotor figure construction, visual figure delayed recall, and figure recognition deficits in a condition that is characterized by disproportionate language deficits. It is important for clinicians to be aware of these deficits, as they can affect activities of daily living, such as driving, and may affect the ability to learn or compensate through the visual modality.

Acknowledgement

This work was made possible by NIH grant R01DC011317 from NIDCD. We gratefully acknowledge this support.

References

Bak TH, & Hodges JR (2003). Kissing and dancing—a test to distinguish the lexical and conceptual contributions to noun/verb and action/object dissociation. Preliminary results in patients with frontotemporal dementia. Journal of Neurolinguistics, 16, 169–181. doi:10.1016/S0911-6044(02)00011-8

Breining BL, Lala T, Martínez Cuitiño M., Manes F, Peristeri E, Tsapkini K, … Hillis AE (2015). A brief assessment of object semantics in primary progressive aphasia. Aphasiology, 29, 488–505. doi:10.1080/02687038.2014.973360

Breining BL, Tippett DC, Davis C, Posner J, Sebastian R, Oishie K, … Hillis AE (2015, 5). Assessing dissociations of object and action naming in acute stroke. Paper presented at the Clinical Aphasiology Conference, Monterey, CA.

Butts AM, Machula MM, Duffy JR, Strand EA, Whitwell JL, & Josephs KA (2015). Neuropsychological profiles differ among the three variants of primary progressive aphasia. Journal of the International Neuropsychological Society, 21, 429–435. doi.10.1017/S1355617715000399 [PubMed: 26067425]

Budd MA, Kortte K, Cloutman L, Newhart M, Gottesman RF, Davis C, … Hillis AE (2010). The nature of naming errors in primary progressive aphasia versus acute post-stroke aphasia. Neuropsychology, 24, 581–589. doi.10.1037/0020287 [PubMed: 20804246]

Caramazza A, & Hillis AE (1990). Where do semantic errors come from? Cortex, 26, 95–122.doi:10.1016/S0010-9452(13)800 [PubMed: 2354648]

Dabul B (2000). Apraxia battery for adults – second edition. Austin, TX: Pro-Ed.

Foxe DG, Irish M, Hodges JR, & Piguet O (2013). Verbal and visuospatial span in logopenic progressive aphasia and Alzheimer’s disease. Journal of the International Neuropsychological Society, 19, 247–253. doi.10.1017/S1355617712001269 [PubMed: 23298815]

Foxe D, Leyton CE, Hodges JR, Burrell JR, Irish M, & Piguet O (2016). The neural correlates of auditory and visuospatial span in logopenic progressive aphasia and Alzheimer's disease. Cortex, 83, 39–50. doi.10.1016/j.cortex.2016.07.003 [PubMed: 27474916]

Fridriksson J, Holland AL, Beeson P, & Morrow L (2005). Spaced retrieval treatment of anomia. Aphasiology, 19, 99–109. doi.10.1080/02687030444000660 [PubMed: 16823467]

Giannini LAA, Irwin DJ, McMillan CT, Ash S, Rascovsky K, Wolk DA, & Grossman M (2017). Clinical marker for Alzheimer disease pathology in logopenic primary progressive aphasia. Neurology, 88, 2276–2284. doi.10.1212/WNL.0000000000004034 [PubMed: 28515265]

Goodglass H, Kaplan E, & Barresi B (2001). Boston diagnostic aphasia examination – third edition. Baltimore: Lippincott, Williams, & Wilkins.

Gorno-Tempini ML, Brambati SM, Ginex V, Ogar J, Dronkers NF, Marcone A, … Miller BL (2008). The logopenic/phonological variant of primary progressive aphasia. Neurology, 71, 1227–1234. doi.10.1212/01.wnl.0000320506.79811.da [PubMed: 18633132]

Gorno-Tempini ML, Dronkers NF, Rankin KP, Ogar JM, Phengrasamy L, Rosen HH, …Miller BL (2004). Cognition and anatomy in three variants of primary progressive aphasia. Annals of Neurology, 55, 335–346. doi.10.1002/ana.10825 [PubMed: 14991811]

Gorno-Tempini ML, Hillis AE, Weintraub S, Kertesz A, Mendez M, Cappa SF, … Grossman M (2011). Classification of primary progressive aphasia and its variant. Neurology, 76, 1006–1014. doi.10.1212/WNL.0b013e31821103e6 [PubMed: 21325651]

Grossman M Primary progressive aphasia: Clinicopathological correlations. (2010). Nature Reviews Neurology, 6, 88–97. doi.10.1038/nrneurol.2009.216 [PubMed: 20139998]

Grossman M, Xie SX, Libon DJ, Wang X, Massimo L, Moore P, …Trojanowski JQ (2008). Longitudinal decline in autopsy-defined frontotemporal lobar degeneration. Neurology, 70, 2036–2045. doi.10.1212/01.wnl.0000303816.25065.bc [PubMed: 18420483]

Henry ML, Hubbard HI, Grasso SM, Mandellia LL, Wilson SM, Sathishkumar MT, …Gorno-Tempini ML (2018). Retraining speech production and fluency in nonfluent/agrammatic primary progressive aphasia. Brain, 141, 1799–1814. doi. 10.1093/brain/awy101. [PubMed: 29718131]

Howard D, & Patterson K (1992). The pyramids and palm trees test: A test of semantic access from words and pictures. Cambridge, UK: Pearson.

Irwin DJ, Trojanowski JQ, & Grossman M (2013). Cerebrospinal fluid biomarkers for differentiation of frontotemporal lobar degeneration from Alzheimer's disease. Frontiers in Aging Neuroscience, 5, 6. doi.10.3389/fnagi.2013.00006 [PubMed: 23440936]

Josephs KA, Duffy JR, Strand EA, Whitwell JL, Layton KF, Parisi JE, …Petersen RC (2006). Clinicopathological and imaging correlates of progressive aphasia and apraxia of speech. Brain, 129, 1385–1398. doi.10.1093/brain/awl078 [PubMed: 16613895]

Josephs K, Whitwell J, Duffy J, Vanvoorst W, Strand E, Hu W, …Petersen R (2008). Progressive aphasia secondary to Alzheimer disease: A clinicopathologic and MRI study. Neurology, 70, 25–34. doi.10.1212/01.wnl.0000287073.12737.35 [PubMed: 18166704]

Kertesz A, McMonagle P, Blair M, Davidson W, & Munoz DG (2005). The evolution and pathology of frontotemporal dementia. Brain, 128, 1996–2005. doi.10.1093/brain/awh598 [PubMed: 16033782]

Knopman DS, Boeve BF, Parisi JE, Dickson DW, Smith GE, Ivnik RJ, …Petersen RC (2005). Antemortem diagnosis of frontotemporal lobar degeneration. Annals of Neurology, 57, 480–488. doi.10.1002/ana.20425 [PubMed: 15786453]

Kramer JH, Jurik J, Sha SJ, Rankin KP, Rosen HJ, Johnson JK, & Miller BL (2003). Distinctive neuropsychological patterns in frontotemporal dementia, semantic dementia, and Alzheimer disease. Cognitive and Behavioral Neurology, 16, 211–218. [PubMed: 14665820]

Mack WJ, Freed DM, Williams BW, & Henderson VW (1992). Boston naming test: Shortened versions for use in Alzheimer's disease. Journal of Gerontology, 47, 154–158.

Macoir J, Lavoie M, Laforce R, Brambati S, & Wilson M (2017). Dysexecutive symptoms in primary progressive aphasia: beyond diagnostic criteria. Journal of Geriatric Psychiatry, 30(3), 151–161. doi.10.1177/0891988717700507

Mesulam M-M (2001). Primary progressive aphasia. Annals of Neurology, 49, 425–432. [PubMed: 11310619]

Mesulam M-M (2013). Primary progressive aphasia and the language network: The 2013 H. Houston Merritt Lecture. Neurology, 81, 456–462. [PubMed: 23897873]

Mesulam M-M, & Weintraub S (2014). Is it time to revisit the classification guidelines for primary progressive aphasia? Neurology, 82, 1108–1109. doi. 10.1212/WNL.0000000000000272 [PubMed: 24598706]

Mesulam M-M, Wieneke C, Thompson C, Rogalski E, & Weintraub S (2012). Quantitative classification of primary progressive aphasia at early and mild impairment stages. Brain, 135, 1537–1553. doi.10.1093/brain/aws080 [PubMed: 22525158]

Meyer AM, Tippett DC, Turner RS, & Friedman RB (2018). Long-term maintenance of anomia treatment effects in primary progressive aphasia. Neuropsychological Rehabilitation, 30, 1–25. doi.10.1080/09602011.2018.1425146 [PubMed: 29526134]

Milner AD, & Goodale MA (1995). The visual brain in action. Oxford: Oxford University Press.

Osterrieth P (1944). Le test de copie d'une figure complex: Contribution a l'etude de la perception et de la memoire. Archives de Psychologie, 30, 286–356.

Possin KL, Laluz VR, Alcantar OZ, Miller BL, & Kramer JH (2011). Distinct neuroanatomical substrates and cognitive mechanisms of figure copy performance in Alzheimer's disease and behavioral variant frontotemporal dementia. Neuropsychologia, 49, 43–48. doi.10.1016/.neuropsychologia.2010.10.026 [PubMed: 21029744]

Rogalski E, Cobia D, Harrison TM, Wieneke C, Thompson CK, Weintraub S, & Mesulam M-M (2011). Anatomy in language impairments in primary progressive aphasia. Journal of Neuroscience, 31, 3344–3350. doi.10.1523/JNEUROSCI.5544-10.2011 [PubMed: 21368046]

Rogalsky C, Love T, Driscoll D, Anderson SW, & Hickok G (2011). Are mirror neurons the basis of speech perception? Evidence from five cases with damage to the purported human mirror system. Neurocase, 7, 178–187. oi:10.1080/13554794.2010.509318

Rohrer JD, Caso F, Mahoney C, Henry M, Rosen HJ, Rabinovici G, …Gorno-Tempini ML (2013). Patterns of longitudinal brain atrophy in the logopenic variant of primary progressive aphasia. Brain and Language, 127, 121–126. doi.10.1016/j.bandl.2012.12.008 [PubMed: 23395096]

Sajjadi SA, Patterson K, Arnold RJ, Watson PC, & Nestor PJ (2012). Primary progressive aphasia: A tale of two syndromes and the rest. Neurology, 78, 1670–1677. doi.10.1212/WNL.0b013e3182574f79 [PubMed: 22573633]

Sebastian R, Thompson CB, Wang N-Y, Wright A, Meyer A, Friedman RB, Hillis AE, & Tippett DC (2018). Patterns of decline in naming and semantic knowledge in primary progressive aphasia. Aphasiology, 32, 1010–1030. doi.10.1080/02687038.2018.1490388 [PubMed: 30613121]

Snowden JS, Bathgate D, Varma A, Blackshaw A, Gibbons ZC, & Neary D (2001). Distinct behavioural profiles in frontotemporal dementia and semantic dementia. Journal of Neurology, Neurosurgery, and Psychiatry, 70, 323–332. doi.10.1136/jnnp.70.3.323

Thompson CK, Lukic S, King MC, Mesulam MM, & Weintraub S (2012). Verb and noun deficits in stroke-induced and primary progressive aphasia: The Northwestern naming battery. Aphasiology, 26, 632–655, DOI: 10.1080/02687038.2012.676852 [PubMed: 23188949]

Tippett DC, Hillis AE, & Tsapkini K (2015). Treatment of primary progressive aphasia. Current Treatment Options Neurology, 17, 362. doi.10.1007/s11940-015-0362-5 [PubMed: 26062526]

Ungerleider LG, & Mishskin M (1982). Two cortical visual systems In Goodale MA & Mansfield RJW (eds.), Analysis of visual behavior (549–596). Cambridge, MA: MIT Press.

Watson CL, Possin K, Allen E, Hubbard I, Meyer M, Welch AE, …Gorno-Tempini ML (2018). Visuospatial functioning in the primary progressive aphasias. Journal of the International Neuropsychological Society, 24, 259–268. doi.10.1017/S1355617717000984 [PubMed: 29039275]

Wicklund MR, Duffy JR, Strand EA, Machulda MM, Whitwell JL, & Josephs KA (2014). Quantitative application of the primary progressive aphasia consensus criteria. Neurology, 82, 1119–1126. doi.10.1212/WNL.0000000000000261 [PubMed: 24598709]

Wilson SM, Galantucci S, Tartaglia MC, Rising K, Patterson DK, Henry ML, … Gorno-Tempini ML (2011). Syntactic processing depends on dorsal language tracts. Neuron, 72, 397–403. doi.10.1016/j.neuron.2011.09.014 [PubMed: 22017996]

Table 1:

Age, Sex, Education, and Symptom Duration for PPA Variants and for Participants Overall

Variant	Age (yrs) (*M, SD*)	Education (yrs) (*M, SD*)	Symptom Duration (mos) (*M, SD*)	Sex (F) *n* (%)
lvPPA (*n* = 39)	70.82 (6.08)	16.32 (2.61)	45.92 (26.36)	23 (59)
nfaPPA (*n* = 24)	69.79 (9.95)	16.00 (2.36)	47.96 (23.02)	12(50)
svPPA (*n* = 30)	66.47 (6.72)	14.85 (2.32)	41.23 (18.81)	18 (60)
Overall (*N* = 93)	69.15 (7.61)	15.78 (2.51)	44.94 (23.20)	53 (57)
p values*	.054	.059	.540	.722

F, female; *M*, mean; *SD*, standard deviation; yrs, years; mos, months; lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

* *p* values were calculated using one-way ANOVA for age, education, and symptom duration and using chi square for sex.

Table 2:

Age, Sex, Education, and Symptom Duration for PPA Variants and for Participants Tested at Follow Up

Variant	Age (yrs) (*M, SD*)	Education (yrs) (*M, SD*)	Symptom Duration (mos) (*M, SD*)	Sex (F) *n* (%)
lvPPA (*n* = 14)	68.86 (7.01)	16.36 (2.87)	52.50 (28.65)	11 (79)
nfaPPA (*n* = 10)	66.60 (8.86)	15.80 (2.90)	45.70 (21.73)	4 (40)
svPPA (*n* = 6)	68.67 (6.47)	16.00 (2.19)	51.67 (24.67)	3 (50)
Overall (*N* = 30)	68.07 (7.40)	16.10 (2.68)	50.07 (25.09)	18 (60)
p values*	.758	.887	.804	.140

F, female; *M*, mean; *SD*, standard deviation; yrs, years; mos, months; lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

* *p* values were calculated using one-way ANOVA for age, education, and symptom duration and using chi square for sex.

Table 3:

Tests on the Primary Progressive Aphasia Battery

Phonemic Verbal Fluency
Oral Word Reading
Semantic Word Picture Matching
Semantic Associates
JHU Sentence Anagram Task
Sentence Repetition Test
Pyramids and Palm Trees Test, short form
Kissing and Dancing Test
Noun and Verb Naming Tests
Sentence Reading Test
Spelling to Dictation
Boston Naming Test, short form
Hopkins Assessment of Naming Actions
Picture Word Verification
Cookie Theft Picture Description (BDAE)
Apraxia Battery for Adults

Table 4:

Boston Naming Test Scores for PPA Variants and for Participants Overall

Variant	BNT Score (*M*, *SD*)
lvPPA (*n* = 39)	12.64 (7.52)
nfaPPA (*n* = 24)	19.42 (9.90)
svPPA (*n* = 30)	7.10 (7.11)
Overall (*N* = 93)	12.60 (9.26)
p values*	<.0001

BNT, Boston Naming Test, short form, normal = 27.5 ± 2.4; Mack et al., 2002; *M*, mean; *SD*, standard deviation; lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

*p values were calculated using one-way ANOVA for BNT scores

Table 5:

Pearson Product-Moment Correlation Coefficients (r), Coefficients of Determination (r^2), and Percent Variance between Boston Naming Test Scores and Benson Complex Figure Copy at Baseline for PPA Variants and Participants Overall

Variant	*r*	95% Confidence Interval *r*	r^2	Percent Variance	*p* values
lvPPA (n = 39)	0.370	0.063, 0.614	0.137	13.72	.020
nfaPPA (n = 24)	0.328	−0.086, 0.645	0.108	10.76	.117
svPPA (n = 30)	0.168	−0.204, 0.498	0.028	2.82	.376
Overall (N = 93)	0.335	0.142, 0.504	0.112	11.23	.001

lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

*p values were calculated using Pearson product-moment correlation coefficients

Table 6:

Pearson Product-Moment Correlation Coefficients (r), Coefficients of Determination (r^2), and Percent Variance between Boston Naming Test Scores and Benson Complex Figure Delay (Recall) at Baseline for PPA Variants and Participants Overall

Variant	*r*	95% Confidence Interval *r*	r^2	Percent Variance	*p* values
lvPPA (*n* = 39)	0.399	0.096, 0.634	0.159	15.93	.012
nfaPPA (*n* = 24)	0.323	−0.092, 0.642	0.105	10.45	.124
svPPA (*n* = 30)	0.040	−0.324, 0.394	0.002	1.60	.835
Overall (*N* = 93)	0.374	0.185, 0.536	0.140	13.98	.0002

lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

*
p values were calculated using Pearson product-moment correlation coefficients

Table 7:

Number and Percent with Dichotomized Scores on the Benson Complex Figure Copy and Benson Complex Figure Delay (Recall) for PPA Variants

Variant	Benson Complex Figure Copy Score ≤ 14 (*n*, %)	Benson Complex Figure Copy Score > 14 (*n*, %)	Benson Complex Figure Delay (Recall) ≤ 10 (*n*, %)	Benson Complex Figure Delay (Recall) > 10 (*n*, %)
lvPPA (*n* = 39)	8 (21)	31 (79)	32 (82)	7 (18)
nfaPPA (*n* = 24)	2 (8)	22 (92)	10 (42)	14 (58)
svPPA (*n* = 30)	12 (40)	18 (60)	23 (77)	7 (23)
p value *	.021		.002	

lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

* *p* values were calculated using chi square

Table 8:

Performance on Benson Complex Figure Copy, Benson Complex Figure Delay (Recall), and Benson Figure Recognition for PPA Variants and for Participants Overall

Variant	Benson Complex Figure Copy (*M, SD*)	Benson Complex Figure Delay (Recall) (*M, SD*)	Benson Figure Recognition (#/% 0, #/% 1)	
			0	1
lvPPA (*n* = 39)	14.77 (4.13)	6.41 (4.71)	6 (15)	33 (85)
nfaPPA (*n* = 24)	15.50 (3.60)	10.13 (5.67)	5 (21)	19 (79)
svPPA (*n* = 30)	13.17 (5.25)	5.37 (5.78)	12 (40)	18 (60)
Overall (*N*=93)	14.44 (4.45)	7.03 (5.59)	23 (25)	70 (75)
p values*	.133	.004	.055	

M, mean; *SD*, standard deviation; lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

* *p* values were calculated using one-way ANOVA for figure copy and recall mean scores and using chi square for figure recognition

Table 9:

Mean Rates of Decline on Benson Complex Figure Copy and Benson Complex Figure Delay (Recall), and Numbers of Participants with Correct (1) versus Incorrect (0) Responses on Benson Figure Recognition for PPA Variants and for Participants Overall

Variant	Benson Figure Copy (*M, SD*)	Benson Complex Figure Delay (Recall) (*M, SD*)	Benson Figure Recognition (# 0, # 1)			
			Baseline		Follow Up	
			0	1	0	1
lvPPA (*n* = 14)	−1.48 (2.84)	−0.09 (2.11)	1	13	2	12
nfaPPA (*n* = 10)	−0.59 (1.40)	0.08 (1.67)	1	9	3	7
svPPA (*n* = 6)	−0.53 (1.20)	0.64 (2.33)	2	4	2	4
Overall (*N* = 30)	−0.99 (2.17)	0.11 (1.97)	4	26	7	23
p values*	.535	.758	.695			

M, mean; *SD*, standard deviation; lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

** p* values were calculated using one-way ANOVA for figure copy and recall mean scores and using chi square for figure recognition

Table 10:

Number (Percent) of Scores Segmented for Mean Rate of Decline on Benson Complex Figure Copy for PPA Variants and for Participants Overall

Variant	**≤ −15**	**−1.49 – −1.0**	**≥0**
lvPPA (*n* = 14)	4 (29)	3 (21)	7 (50)
nfaPPA (*n* = 10)	2 (20)	1 (10)	7 (70)
svPPA (*n* = 6)	1 (17)	2 (33)	3 (50)
Overall (*N* = 30)	7 (23)	6 (20)	17 (57)
p value *	.764		

lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

* *p* values were calculated using chi square

Table 11:

Number (Percent) of Scores Segmented for Mean Rate of Decline on Benson Complex Figure Delay (Recall) for PPA Variants and for Participants Overall

Variant	**≤ −1.5**	**−1.49 – −1.0**	**≥0**
lvPPA (*n* = 14)	4 (29)	1 (7)	9 (64)
nfaPPA (*n* = 10)	2 (20)	2 (20)	6 (60)
svPPA (*n* = 6)	1 (17)	0 (0)	5 (83)
Overall (*N* = 30)	7 (23)	3 (10)	20 (67)
p value *	.859		

lvPPA, logopenic primary progressive aphasia; nfaPPA, nonfluent agrammatic primary progressive aphasia; svPPA semantic variant primary progressive aphasia

* *p* values were calculated using chi square