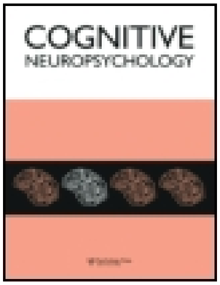

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Phonological facilitation of object naming in agrammatic and logopenic primary progressive aphasia (PPA)

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Phonological processing deficits are characteristic of both the agrammatic and logopenic subtypes of primary progressive aphasia (PPA-G and PPA-L). However, it is an open question which substages of phonological processing (i.e., phonological word form retrieval, phonological encoding) are impaired in these subtypes of PPA, as well as how phonological processing deficits contribute to anomia. In the present study, participants with PPA-G ($n = 7$), participants with PPA-L ($n = 7$), and unimpaired controls ($n = 17$) named objects as interfering written words (phonologically related/unrelated) were presented at different stimulus onset asynchronies (SOAs) of 0, +100, +300, and +500 ms. Phonological facilitation (PF) effects (faster naming times with phonologically related interfering words) were found for the controls and PPA-L group only at SOA = 0 and +100 ms. However, the PPA-G group exhibited protracted PF effects (PF at SOA = 0, +100, and +300 ms). These results may reflect deficits in phonological encoding in PPA-G, but not in PPA-L, supporting the neuropsychological reality of this substage of phonological processing and the distinction between these two PPA subtypes.

Keywords: Primary progressive aphasia; Anomia; Phonological processing; Picture–word interference paradigm.

Primary progressive aphasia (PPA) presents a unique clinical syndrome among neurodegenerative diseases of the brain in that it selectively affects the language network in its early stages, preserving other cognitive capacities such as attention and memory (Mesulam, 1982, 2003). There are three major subtypes of PPA, each associated with different linguistic profiles and distinct patterns of neural atrophy. The agrammatic subtype of PPA (PPA-G) is characterized by effortful speech and impaired processing of morphosyntactic structure, with peak atrophy typically occurring in the left inferior frontal gyrus (Gorno-Tempini et al., 2004, 2011; Mesulam et al., 2009; Mesulam Wieneke, Thompson, Rogalski, & Weintraub, 2012). The logopenic subtype

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(PPA-L) has been linked to deficits in word retrieval and phonological working memory, with atrophy typically focused in the left posterior temporal lobe and temporoparietal junction (Gorno-Tempini et al., 2004, 2008, 2011; Mesulam et al., 2009, 2012; Rohrer et al., 2010). The semantic subtype (PPA-S) is characterized by difficulty in processing lexical−semantic information (i.e., word meaning) in both production and comprehension, with associated neural atrophy in the left anterior temporal lobe (Gorno-Tempini et al., 2004, 2011; Mesulam et al., 2009, 2012). Anomia is a feature common to all three subtypes and is influenced by phonological processing. However, relatively little is known about the contributions of phonological mechanisms to the anomia of PPA and whether these mechanisms are differentially impaired in patients presenting with different subtypes of PPA. In the present study, we test whether PPA-G and PPA-L are associated with impaired phonological processing during object naming.

Phonological processing is one of two major stages of word naming (e.g., Dell, Schwartz, Martin, Saffran, & Gagnon, 1997; Indefrey & Levelt, 2004; Levelt, 1992, 1999; Levelt, Roelofs, & Meyer, 1999; Rapp & Goldrick, 2000). When a person is presented with an object to be named (e.g., a zebra), the image of the object is first processed by the visual system and transformed into a percept that can be linked to its multimodal verbal and nonverbal associations. The verbal association, a lexical progenitor or *lemma*, provides access to stored morphosyntactic and additional verbal information [e.g., that the percept is an "animal" (generic stage of encoding), which is also a "zebra" (specific stage of encoding); see Mesulam et al., 2013]. This first stage, where the percept is transformed into a concept, is known as the *semantic* stage of linguistic processing. The second stage of linguistic processing is the *phonological stage*, which consists of phonological word form retrieval (i.e., retrieval of an abstract phonological representation, e.g., /zibra/) and phonological encoding (i.e., sublexical phonological processes including access to phonological segments and syllabification, e.g., /zi.bra/). This representation then undergoes phonetic encoding—that is, generation of a motor representation for articulation. Disruption at either major level of processing may lead to semantic and/or phonological *paraphasic* production patterns—that is, substitution of semantically related words (e.g., *tiger* for *zebra*) or production of responses that are phonologically related to the target (e.g., *bebra* for *zebra*), respectively. These errors are thought to reflect *spreading activation* both within and across levels of representation. That is, activation spreads to items that are semantically and/or phonologically related to the target (e.g., Caramazza & Hillis, 1990; Dell et al., 1997; Levelt, 1999; Levelt et al., 1999; also see Goldrick & Rapp, 2002; and Rapp & Goldrick, 2000, for discussion). Whether or not the word production system is interactive (i.e., feedback from the phonological processing stage affects lemma selection; Damian & Martin, 1999; Starreveld, 2000; Starreveld & La Heij, 1995, 1996) or serial (i.e., no feedback of this sort; Schriefers, Meyer, & Levelt, 1990) is an unresolved issue.

All speakers occasionally produce paraphasias, but they are particularly common in aphasic speech. Previous research has shown that individuals with PPA-G and PPA-L tend to produce more phonological paraphasias than people with PPA-S, whereas the opposite pattern has been reported for semantic paraphasias (Clark, Charuvastra, Miller, Shapira, & Mendez, 2005; cf. Ash et al., 2010; Mesulam et al., 2012; Wilson et al., 2010). This suggests that phonological processing may be relatively prone to impairment in PPA-G and PPA-L. One recent study reported a higher rate of phonological paraphasias in PPA-L than in PPA-G (Croot, Ballard, Leyton, & Hodges, 2012), whereas other studies have found similar rates of phonological paraphasias across the two PPA subtypes (Mesulam et al., 2012; Wilson et al., 2010). Both PPA-G and PPA-L have also been associated with impairments in other aspects of phonological processing, including phonological working memory (Gorno-Tempini et al., 2004, 2008; Mesulam et al., 2012; Rohrer et al., 2010) and pseudoword reading and spelling (Brambati, Ogar, Neuhaus, Miller, & Gorno-Tempini, 2009; Shim, Hurley, Rogalski, & Mesulam, 2012). The substages of phonological processing (e.g., phonological word form retrieval, phonological encoding) that are selectively impaired during naming in PPA-G and PPA-L have not been characterized. Most previous studies have used offline measures to examine phonological processing difficulty in PPA. However, online studies have the potential to reveal patterns of impairment that are not evident in offline phonological measures. For instance, recent online studies indicate that even nonsemantic variants of PPA (i.e., PPA-G and PPA-L) are associated with semantic processing impairments (Rogalski, Rademaker, Mesulam, & Weintraub, 2008; Thompson, Cho, Price, et al., 2012; Vandenberghe et al., 2005).

Neuroimaging studies have demonstrated that the brain regions supporting phonological processing during naming in healthy participants overlap with the regions that typically undergo atrophy in PPA-G and PPA-L. According to a meta-analysis of word production studies (Indefrey & Levelt, 2004), phonological word form retrieval recruits the left posterior middle and superior temporal gyri and the temporoparietal junction (cf. Graves, Grabowski, Mehta, & Gordon, 2007; Graves, Grabowski, Mehta, & Gupta, 2008; Indefrey, 2011; Wilson, Isenberg, & Hickok, 2009). Indefrey and Levelt (2004) argue that phonological encoding, in contrast, is supported by the left inferior frontal gyrus (IFG), a region that may also support subsequent phonetic encoding and articulatory processes (see also Hickok & Poeppel, 2007; Papoutsi et al., 2009). Given that PPA-L is characterized by atrophy in the left temporoparietal junction, we might predict deficits in phonological word form retrieval for this subtype of PPA. Because PPA-G typically involves atrophy in the left IFG, deficits in phonological encoding are expected. If phonological word form retrieval and phonological encoding are indeed differentially impaired in these subtypes of PPA, this would support previous behavioural and neuroimaging evidence in favour of multiple substages of phonological processing (see Indefrey, 2011; Indefrey & Levelt, 2004, for reviews).

In the present study, we sought to test whether PPA-G and PPA-L are associated with deficits in different substages of phonological processing that are observable online as object naming unfolds. The picture–word interference paradigm (PWIP; Rosinski, Michnick-Golinkoff, & Kukish, 1975), which provides an online, automatic, time-constrained measure of the processes that support naming, was employed to study this effect. In the PWIP, adapted from the Stroop task (Stroop, 1935), participants are presented with a picture of an object to be named along with a visually or auditorily presented word, called an *interfering stimulus* (IS), which the participant is instructed to ignore. The dependent measure is naming latency. Generally, naming times are slower in the presence of an IS than they are for pictures in isolation (or for pictures labelled with their correct names), reflecting increased demands on processing resources (Lupker, 1982; Starreveld & La Heij, 1996). However, the interference effect depends on the specific properties of the interfering word as well as differences in time between picture presentation and word presentation, known as stimulus onset asynchrony (SOA). For example, interfering words that are semantically related to the target object (e.g., *horse* for target RABBIT) result in slower naming times than unrelated words (e.g., *turnip* for target RABBIT) when presented in close proximity to the to-be-named object [i.e., SOAs from 300 ms before picture presentation (−300 ms) to 100 ms after picture presentation (+100 ms)], an effect known as *semantic interference* (Glaser & Düngelhoff, 1984; Hashimoto & Thompson, 2010; Lupker, 1979; Rosinski et al., 1975; Schriefers et al., 1990; Starreveld & La Heij, 1995, 1996; Thompson, Cho, Price, et al., 2012). When interfering stimuli are presented outside of this time window, healthy speakers are no longer influenced by them. Interestingly, however, studies with PPA show semantic interference effects at long SOAs. Vandenberghe et al. (2005) found such effects at −750 ms in a group of PPA speakers and, in a recent study examining naming in PPA-G and PPA-L, Thompson, Cho, Price, et al. (2012) found semantic interference effects at −1000-ms

SOA in individuals with both types of PPA, indicating abnormal semantic processing.

In the present study, we focus on the phonological stages of object naming by manipulating the phonological form of the IS. Object naming can be speeded by words that are phonologically related to the target (e.g., *radish* for target RABBIT), an effect called *phonological facilitation* (PF; Bi, Xu, & Caramazza, 2009; Hashimoto & Thompson, 2010; Lupker, 1982; Schriefers et al., 1990; Starreveld, 2000; Starreveld & La Heij, 1995, 1996). Two main explanations of this phenomenon have been proposed. On some accounts, the PF effect occurs because the target word and IS activate some of the same phonological segments, which raises the activation level for the segments in the target word and thus facilitates naming (Meyer & Schriefers, 1991; Roelofs, 1997; Schriefers et al., 1990). Alternatively, the PF effect may result from spreading activation between word forms, with the IS activating a cohort of phonologically related word forms that includes the target (Levelt et al., 1999; Starreveld, 2000; Starreveld & La Heij, 1995, 1996). Spreading activation between orthographically related representations probably also contributes significantly to this effect (Bi et al., 2009; Lupker, 1982). The PF effect is sensitive to the SOA between the target picture and the IS and has been observed at SOAs ranging from 300 ms prior to picture presentation (−300 ms) to 200 ms following picture presentation (+200 ms), with the precise time window varying based on properties of the experimental design (Bi et al., 2009; Damian & Martin, 1999; Hashimoto & Thompson, 2010; Lupker, 1982; Meyer & Schriefers, 1991; Rayner & Springer, 1986; Schriefers et al., 1990; Starreveld, 2000; Starreveld & La Heij, 1995, 1996). To the best of our knowledge, the PF effect has not been observed in healthy speakers outside this range, although Starreveld (2000) found significant PF effects for healthy speakers at +300 ms with part-word IS (e.g., *pa*), but not with full-word IS.

In recent years, the PWIP also has emerged as a tool to study the processing mechanisms underlying naming difficulty in patients with anomia. Two recent studies have sought evidence of abnormal PF effects in aphasic speakers. One study of 11 patients with stroke-induced nonfluent aphasia found larger phonological facilitation effects at SOA = 0 ms (i.e., simultaneous presentation of the picture and interfering word) for people with aphasia than for age-matched controls (Hashimoto & Thompson, 2010). The authors interpret this heightened PF effect as evidence for a phonological processing impairment that led to greater reliance on phonological cues during naming. This hypothesis was supported by the aphasic participants' impaired performance on language tests that targeted phonological processes. In addition, a case study of a patient with stroke-induced anomic aphasia reported a significant PF effect at SOA = 0 ms, while the control group in the study demonstrated a trend towards PF that did not reach significance (Wilshire, Keall, Stuart, & O'Donnell, 2007).

To our knowledge, no previous studies have used the PWIP to investigate phonological processing in patients with PPA. Unlike stroke-induced aphasia, the syndrome of PPA is progressive, and the neuroanatomy of disease is not dictated by vascular territories but rather by principles of neuronal connectivity patterns underlying large-scale networks (Mesulam, 1982, 2007; Seeley, Crawford, Zhou, Miller, & Grecius, 2009). Thus, PPA offers a unique opportunity to study language processing in a network undergoing gradual dissolution. In the present study, we used the PWIP paradigm to test the magnitude and time course of PF effects in people with PPA-G and PPA-L as well as healthy age-matched controls in four SOA conditions. In one condition, target pictures were presented simultaneously with IS (i.e., SOA = 0 ms), which were either phonologically related or unrelated, and in the other three conditions the IS was presented after the target picture: 100 ms (i.e., SOA = +100 ms), 300 ms (i.e., SOA = +300 ms), or 500 ms (i.e., SOA = +500 ms).

On the basis of previous findings indicating impaired phonological processing in PPA-G and PPA-L (e.g., Clark et al., 2005; Gorno-Tempini et al., 2008; Rohrer et al., 2010), we predicted that we would find evidence of abnormal phonological processing in both PPA variants. On the basis of previous studies on stroke-induced aphasia (Hashimoto & Thompson, 2010; Wilshire et al., 2007), we expected that phonological processing impairments would be reflected by larger PF effects in individuals with PPA than in controls. On the basis of neurological evidence (i.e., typical regions of cortical atrophy), we predicted that participants with PPA-L would show abnormal (large) PF effects in earlier stages of naming (SOA 0 and/or +100 ms), reflecting impaired phonological word form retrieval, whereas participants with PPA-G would exhibit abnormal PF effects at later stages of naming (SOA +300 and/or +500 ms), reflecting impaired phonological encoding. Underlying these predictions is the assumption that PF effects may reflect spreading activation at either the lexical (Levelt et al., 1999; Starreveld, 2000; Starreveld & La Heij, 1995, 1996) or segmental (Meyer & Schriefers, 1991; Roelofs, 1997; Schriefers et al., 1990) level of representation.

METHOD

Participants

Participants in this experiment included two groups of patients with PPA, seven with agrammatic PPA (PPA-G) and seven with logopenic PPA (PPA-L), and a group of age- and education-matched healthy controls, consisting of 17 cognitively intact volunteers (see Table 1); age: $\chi^2(2, N = 31) = 4.045$, $p = .132$; education: $\chi^2(2, N = 31) = 1.923$; $p = .382$, Kruskal–Wallis Test. Further, the two patient groups were matched for duration of symptoms ($Z = -0.971$, $p = .383$, Mann–Whitney U test) and reported symptom onsets ranging from 1.5 to 7 years prior to testing. All participants, both patients and healthy controls, were monolingual English speakers, who presented with no prior history of neurological, psychiatric, speech, language, or learning deficits. All passed a pure-tone hearing screening, had normal (or corrected-to-normal) vision, and were right-handed. All participants were recruited through the PPA Research and Clinical Program in the Cognitive Neurology and Alzheimer's Disease Center (CNADC) at Northwestern University (Chicago, IL) and were tested in the Aphasia and Neurolinguistics Research Laboratory at Northwestern University (Evanston, IL). They were paid for their participation, and informed consent was obtained prior to the study. The study was approved by the Institutional Review Board at Northwestern University. Nonparametric statistical tests (Kruskal–Wallis test, Mann–Whitney U test) were used to compare participant groups.

The PPA participants presented with progressive language deficits with no evidence of other language or neurological deficits. Participants were diagnosed with PPA-G or PPA-L based on criteria presented by Mesulam et al. (2012), with individuals in both groups showing relatively intact single-word comprehension and those with PPA-G, but not PPA-L, showing grammatical sentence production impairments (using a classification template with severity-based cut-offs; see Mesulam et al., 2012, for details). In addition, the classification criteria for PPA-L included impaired repetition. Single-word comprehension was assessed using a 36-item subset of the Peabody Picture Vocabulary Test (PPVT, i.e., moderately difficult items, 157–192; Dunn & Dunn, 2007). No differences between groups were observed ($p > .05$). Participants' grammatical sentence production abilities were assessed with the Sentence Production Priming Test (SPPT) of the Northwestern Assessment of Verbs and Sentences (NAVS; Thompson, 2011; http://northwestern.flintbox.com). Production of noncanonical sentences was more difficult for the PPA-G than for the PPA-L group ($Z = -3.169$, $p < .01$), and the PPA-G group performed more poorly than controls ($Z = -4.592$, $p < .001$) whereas the PPA-L group did not ($Z = -2.74$, $p = .118$). Repetition ability was assessed using a subset of items testing phrase/sentence repetition (10–15; Rep6) from the Repetition subtest of the Western Aphasia Battery–Revised (WAB–R; Kertesz, 2006).

Table 1. *Summary of participant demographic data and scores on classification measures*

Participant	Age	Gender	Education	Handedness	Symptom Duration (years)	PPVT (100%)	NAVS SPPT C (100%)	NAVS SPPT NC (100%)	WAB-R Rep6 (100%)
PPA-G1	62	M	20	R	5	100.0	66.7	6.7	72.7
PPA-G2	59	M	12	R	3.1	94.4	0.0	0.0	66.7
PPA-G3	59	M	14	R	7	97.2	66.7	53.3	65.0
PPA-G4	52	F	18	R	1.5	97.2	80.0	26.7	63.6
PPA-G5	60	M	18	R	2	100.0	86.7	66.7	83.0
PPA-G6	61	F	18	R	5	100.0	100.0	13.3	81.8
PPA-G7	72	M	20	R	5	88.9	80.0	20.0	43.9
PPA-L1	69	M	15	R	2.5	97.2	100.0	100.0	85.0
PPA-L2	58	M	16	R	2	97.2	100.0	86.7	85.0
PPA-L3	65	F	13	R	5.3	83.3	86.7	100.0	81.8
PPA-L4	75	F	16	R	2.5	97.2	100.0	86.7	88.0
PPA-L5	76	F	16	R	2	97.2	100.0	100.0	61.0
PPA-L6	63	M	18	R	2.5	100.0	100.0	100.0	86.0
PPA-L7	64	F	16	R	2.8	N/A	100.0	93.3	84.8
Mean (SD)									
PPA-G	60.71 (5.93)		17.14 (3.02)		4.09 (1.96)	96.83 (4.07)	68.57[C,L] (32.37)	26.67[C,L] (24.65)	68.1[C,L] (13.21)
PPA-L	67.14 (6.57)		15.71 (1.50)		2.8 (1.14)	95.37 (6.0)	98.1 (5.04)	95.24 (6.34)	81.66[C] (9.29)
Control	62.76 (6.44)		15.94 (2.46)		N/A	98.96 (1.72)	100.0 (0.0)	100.0 (0.0)	99.11 (2.09)

Note: PPVT = subset of Peabody Picture Vocabulary Test; NAVS SPPT = Northwestern Assessment of Verbs and Sentences, Sentence Production Priming Test; C = canonical sentences; NC = noncanonical sentences; WAB−R, Rep6 = Western Aphasia Battery−Revised, subset of 6 most difficult items from Repetition subtest; PPA = primary progressive aphasia; PPA-G = agrammatic subtype of PPA; PPA-L = logopenic subtype of PPA. [C]Significantly impaired relative to control group.[L]Significantly impaired relative to PPA-L group ($p < .05$, Mann−Whitney *U* test).

Both PPA groups showed impaired performance on this measure relative to controls (PPA-G vs. controls: $Z = -4.220$, $p < .001$; PPA-L vs. controls: $Z = -4.221$, $p < .001$), and phrase and sentence repetition was more impaired in the PPA-G group than in the PPA-L group ($Z = -2.177$, $p = .026$). See Table 1 for a summary of classification measures.

To examine working memory, visual perception, attention, executive function, and motor speech deficits (see Table 2 for a summary of scores) a battery of neuropsychological tests was administered, which included the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975), the Digit Span subtest (forward and backward spans) from the Wechsler Adult Intelligence Scale−III (Wechsler, 1997), the Facial Recognition Test (Benton, Hamsher, Varney, & Spreen, 1998), the Trail Making Test (Reitan, 1958), and a motor speech screening (i.e., an oral apraxia screen and repetition of one-, two-, and three-syllable words, with a maximum score of 10 for each; after Dabul, 2000, and Wertz, LaPointe, & Rosenbek, 1984). Spontaneous speech samples also were collected (see below) and were evaluated for motor speech ability. The PPA patients performed significantly more poorly than controls on the MMSE (PPA-G vs. control: $Z = -2.713$ $p = .016$; PPA-L vs. control: $Z = -3.444$, $p = .001$), probably reflecting the patients' compromised language ability (see Golper, Rau, Erskine, Langhans, & Houlihan,

Table 2. *Summary of neuropsychological and motor speech measures for PPA participants*

		Motor speech			*WMS–III*			
Participant	*MMSE (30)*	*1 syl (10)*	*2 syl (10)*	*3 syl (10)*	*DSF*	*DSB*	*FR (54)*	*TM Test A*
PPA-G1	28	10	10	9	6	2	48	44
PPA-G2	20	N/A	N/A	N/A	4	2	51	108
PPA-G3	24	10	10	8	3	3	39	87
PPA-G4	30	10	10	8	4	5	45	25
PPA-G5	28	10	10	10	6	6	50	35
PPA-G6	30	10	9	9	6	3	42	64
PPA-G7	28	10	10	7	3	6	52	34
PPA-L1	30	10	10	9	5	4	45	37
PPA-L2	23	10	10	10	5	3	44	49
PPA-L3	19	10	10	10	N/A	N/A	41	N/A
PPA-L4	28	10	10	10	4	5	49	17
PPA-L5	24	10	10	10	4	5	47	25
PPA-L6	27	10	10	10	4	5	48	29
PPA-L7	26	10	10	10	6	4	46	28
Mean (SD)								
PPA-G	26.86[C] (3.63)	10 (0)	9.83 (0.41)	8.57[C,L] (1.04)	4.57[C] (1.40)	3.86[C] (1.77)	46.71 (4.89)	56.71[C] (30.97)
PPA-L	25.29[C] (3.64)	10 (0)	10 (0)	9.86 (0.38)	4.67[C] (0.82)	4.33[C] (.82)	45.71 (2.69)	30.83 (11.0)
Control	29.71 (0.59)	10 (0)	10 (0)	10 (0)	7.41 (1.06)	5.76 (1.35)	46.94 (3.89)	30.35 (8.87)

Note: MMSE = Mini-Mental State Examination; motor speech = single word repetition (1syl = 1 syllable; 2syl = 2 syllables; 3syl = 3 syllables); WMS–III = Wechsler Memory Scale–III; DSF = Digit Span Forward; DSB = Digit Span Backward; FR = Facial Recognition; TM = Trail Making Test; PPA = primary progressive aphasia; PPA-G = agrammatic subtype of PPA; PPA-L = logopenic subtype of PPA. Maximum scores appear in parentheses. Numbers in Trail Making Test A indicate time to complete the test in seconds. [C]Significantly impaired relative to control group. [L]Significantly impaired relative to PPA-L group.

1987; Osher, Wicklund, Rademaker, Johnson, & Weintraub, 2008). In addition, both PPA groups showed impaired performance relative to controls on the Digit Span Forward and Backward tests ($ps < .05$), which may reflect a deficit in phonological working memory (Gorno-Tempini et al., 2008). The PPA-G group also showed impairment relative to controls on the Trail Making Test ($Z = -2.132$, $p = .034$). On single-word repetition, the only significant group difference was that the PPA-G group had impaired performance relative to the controls and PPA-L group on three-syllable words (PPA-G vs. control: $Z = -2.94$, $p = .003$; PPA-G vs. PPA-L: $Z = -2.21$, $p = .027$). These data indicated that the PPA participants showed at most mild motor speech deficits. On spontaneous speech samples, all participants were judged to have good speech intelligibility.

A number of additional tests also were administered to detail patient language deficit patterns (see Table 3), including the *Western Aphasia Battery–Revised* (WAB–R, Kertesz, 2006), which tests several aspects of language production and comprehension. Both PPA groups demonstrated impaired performance on all WAB subtests, and aphasia quotients for the patients differed significantly from those of control participants (PPA-G vs. controls: $Z = -3.85$, $p < .001$; PPA-L vs. controls: $Z = -3.85$, $p < .001$). No significant differences were noted between PPA groups for naming ability as

Table 3. *Language testing results for PPA patients*

Participant	*WAB* AQ (100)	*WAB* F (10)	*WAB* Comp (10)	*WAB* Rep (10)	*WAB* Nam (10)	*BNT*	*NNB* Noun	*NNB* W Rep (35)	*NNB* NW Rep (10)	*PALPA* Reg (10)	*PALPA* Exc (10)	*PPT* Words	*PPT* Pictures	*Narrative measures* WPM	*Narrative measures* MLU	*Narrative measures* %GS	*Narrative measures* %PE
PPA-G1	82.3	4	9.1	8	10	98.3	93.3	35	10	9	9	100.0	100.0	25.2	4.0	40.0	13.3
PPA-G2	79.9	4	8.85	7.8	9.3	81.7	95.0	35	8	8	10	90.4	94.2	55.4	5.0	66.7	2.8
PPA-G3	90.5	9	9.85	7.4	9	86.7	96.7	34	6	8	8	98.1	100.0	86.0	6.3	85.7	12.8
PPA-G4	78.8	5	8.5	7.6	9.3	76.7	93.3	35	8	10	8	98.1	98.1	110.3	7.6	57.5	4.6
PPA-G5	93.2	9	9.7	8.9	9	98.3	100.0	33	10	10	10	96.2	96.2	58.9	8.6	88.9	2.2
PPA-G6	75.3	4	8.25	8.8	8.6	88.3	98.3	33	7	9	8	88.5	96.2	36.0	5.4	66.7	17.9
PPA-G7	80.6	6	9	5.9	9.4	73.3	85.0	35	3	10	8	98.1	96.2	77.1	11.3	23.6	13.3
PPA-L1	92	9	9.2	9	9.8	98.3	96.7	35	10	10	10	98.1	98.1	104.5	9.5	92.0	5.9
PPA-L2	86.9	6	9.45	9	10	90.0	95.0	35	10	10	10	100.0	100.0	118.7	10.5	81.3	7.5
PPA-L3	78.6	6	7.4	8.8	8.1	83.3	N/A	N/A	N/A	N/A	N/A	78.8	94.2	N/A	5.4	84.6	3.6
PPA-L4	97.2	10	10	9	9.4	88.3	98.3	35	10	10	10	94.2	94.2	157.7	13.9	70.4	4.2
PPA-L5	88.8	8	9.6	7.2	9.6	83.3	98.3	35	10	10	10	100.0	98.1	94.1	10.7	81.3	7.4
PPA-L6	97.1	10	9.85	8.9	9.5	96.7	100.0	35	10	10	10	100.0	96.2	141.1	7.9	88.2	3.5
PPA-L7	93	9	9.2	9	9.3	90.0	83.3	35	10	10	9	N/A	96.2	98.1	7.8	94.7	6.4
Mean (SD)																	
PPA-G	82.94^{C}	5.86^{C}	9.04^{C}	7.77^{C,L}	9.23^{C}	86.19^{C}	94.52^{C}	34.29	7.43^{C,L}	9.14^{C}	8.71^{C}	95.60	97.25	64.13^{C,L}	6.89^{C}	61.30^{C}	9.55^{C}
	(6.49)	(2.27)	(0.58)	(1.00)	(0.43)	(9.80)	(4.88)	(0.95)	(2.44)	(0.9)	(0.95)	(4.40)	(2.18)	(29.41)	(2.51)	(23.46)	(6.24)
PPA-L	90.51^{C}	8.29^{C}	9.24^{C}	8.70^{C}	9.39^{C}	90.00^{C}	95.28^{C}	35	10.0	10.0	9.83	95.19	96.70^{C}	119.02	9.37	84.64^{C}	5.48^{C}
	(6.51)	(1.70)	(0.87)	(0.67)	(0.61)	(5.86)	(6.09)	(0.0)	(0.0)	(0.0)	(0.41)	(8.32)	(2.14)	(25.5)	(2.71)	(8.12)	(1.73)
Control	99.69	10.0	9.97	9.94	9.95	98.24	99.58	35.0	10.0	9.94	9.94	98.08	98.87	131.85	11.15	93.58	0.83
	(0.68)	(0.0)	(0.11)	(0.14)	(0.12)	(2.24)	(1.14)	(0.0)	(0.0)	(0.25)	(0.25)	(1.36)	(1.81)	(19.61)	(2.09)	(4.04)	(1.5)

Note: WAB = Western Aphasia Battery; BNT = Boston Naming Test; NNB = Northwestern Naming Battery; PALPA = Psycholinguistic Assessment of Language Processing in Aphasia; PPT = Pyramids and Palm Trees Test; AQ = Aphasia Quotient; F = Fluency; Comp = Auditory Comprehension; Rep = Repetition; Nam = Naming; Noun = Noun Naming; W Rep = Word Repetition; NW Rep = Nonword Repetition; Reg = Regular Word Reading; Exc = Exception Word Reading; WPM = words per minute; MLU = mean length of utterance; %GS = % grammatical sentences; %PE = % of words with phonological errors. PPA = primary progressive aphasia; PPA-G = agrammatic subtype of PPA; PPA-L = logopenic subtype of PPA. Percentage correct scores for BNT, NNB Noun naming, PPT, %GS, and % PE are shown. Maximum scores for other measures appear in parentheses. ^{C}Significantly impaired relative to control group; ^{L}Significantly impaired relative to PPA-L group.

measured by the *Boston Naming Test* (BNT, Kaplan, Goodglass, & Weintraub, 1983; *p*s > .05) and noun naming on the Confrontation Naming subtest of the *Northwestern Naming Battery* (NNB, Thompson & Weintraub, experimental version; *p*s > .05), though both groups performed more poorly than controls (PPA-G vs. controls, BNT: $Z = -2.98$, $p = .003$, NNB: $Z = -3.46$, $p = .002$; PPA-L vs. controls: BNT: $Z = -3.14$, $p = .001$, NNB: $Z = -3.04$, $p = .01$). All participants demonstrated intact single-word reading, defined as scores of ≥7 on the Regular and Exception Word Reading scales of the Psycholinguistic Assessments of Language Processing in Aphasia (PALPA; Kay, Lesser, & Coltheart, 1992; see Table 3), though the PPA-G group performed more poorly than the controls (*p*s < .05, Mann–Whitney *U* test). Additionally, testing semantic knowledge revealed relatively preserved ability in both PPA participant groups. To evaluate semantic knowledge, both the picture and word versions of the *Pyramids and Palm Trees Test* (PPT; Howard & Patterson, 1992) were administered, with no significant differences between PPA groups for either measure (words: $Z = -.735$, $p = .534$; pictures: $Z = -.464$, $p = .710$).

Narrative language samples were obtained using a wordless picture book of the story of Cinderella using methods described by Thompson and colleagues (Thompson, Ballard, Tait, Weintraub, & Mesulam, 1997; Thompson, Cho, Hsu, et al., 2012; Thompson, Shapiro, Li, & Schendel, 1995). The PPA-G group performed more poorly than controls on measures of fluency, specifically words per minute (WPM; $Z = -3.38$, $p < .001$) and mean length of utterance (MLU; $Z = -2.70$, $p = .005$), and also produced a smaller percentage of grammatically correct sentences than controls ($Z = -3.47$, $p < .001$). The PPA-L group did not differ from controls on measures of fluency but produced a smaller percentage of grammatically correct sentences ($Z = -2.46$, $p = .013$). Further, the PPA-G group, compared to the PPA-L group, produced less fluent speech, measured by WPM ($Z = -2.571$, $p < .01$), and also produced marginally fewer grammatically correct sentences ($Z = -1.985$, $p = .053$).

To assess phonological processing abilities, we obtained measures of word, nonword, and phrase/sentence repetition (Word Repetition and Nonword Repetition subtests of the NNB; Rep6 from the WAB, reported above). No group differences were observed on word repetition (*p*s > .1), but the PPA-G group exhibited impaired performance relative to both controls and the PPA-L group on nonword repetition (PPA-G vs. controls: $Z = -3.802$, $p = .005$; PPA-G vs. PPA-L: $Z = -2.448$, $p = .035$), whereas the PPA-L group did not differ significantly from the controls. In addition, to obtain a measure of phonological processing in narrative speech, we calculated the percentage of nouns and verbs in the narrative sample that contained phonological errors, including phonological paraphasias as well as phonologically related repair sequences (e.g., *pr–prince*). Both PPA groups produced significantly more phonological errors than the controls (PPA-G: $Z = -3.43$, $p < .001$; PPA-L: $Z = -3.43$, $p < .001$); the two PPA groups did not differ significantly.

Experimental stimuli

Fifty nouns and corresponding pictures, all black-and-white line drawings, including 20 living things (fruits/vegetables, birds/mammals; 10 each), 20 nonliving things (tools, clothing; 10 each), and 10 filler items (from various categories) were selected (see Appendix). For each target item, a set of eight IS, consisting of written words (all nouns), were selected. Four were phonologically related words (matched for the onset and rhyme of the first syllable of the target word), and four were phonologically unrelated words. None of the IS were semantically related to their respective targets. For example, for the target item *camel*, the phonologically related IS were *cannon*, *candor*, *cabbage*, and *canvas,* and the unrelated IS were *fashion*, *lagoon*, *detour*, and *wallet*. Phonologically related IS, phonologically unrelated IS, and target words were matched for length in syllables (1–3) and frequency [$M = 372.75$, $M = 458.29$, and $M = 416.85$, respectively; $F(2,357) = 1.161$, $p = .314$; data from the CELEX database (Baayen, Piepenbrock, & van Rijn, 1993)].

Phonologically related and unrelated IS did not differ with respect to imageability, and both were significantly less imageable than target words (ps < .05): Tukey HSD post hoc test; $F(2, 227) = 10.712$, $p < .001$; $M = 533.19$, $M = 559.29$, $M = 593.74$, respectively (data from the MRC Psycholinguistic Database).[1] In addition, 10 healthy volunteers (age 25–46 years), who did not participate in the experiment, rated the phonological relatedness of word pairs, using a 7-point scale (1 = no overlap, 7 = high overlap). Word pairs were included in the phonologically related condition only if their mean relatedness rating was 5.7 or higher and in the phonologically unrelated condition only if their mean relatedness rating was 2.5 or lower.

For each of the four SOAs, one phonologically related and one phonologically unrelated IS were randomly selected and paired with each of the 50 stimulus pictures, for a total of 100 stimulus pairs per SOA [40 related target pairs, 40 unrelated target pairs, and 20 filler pairs (10 related and 10 unrelated)]. Participants were tested on all four SOAs; thus, they were presented with 400 picture–word stimulus pairs in total. The stimulus pairs were pseudorandomly divided into 10 sets of 40 items each, with each set containing stimulus pairs from all four SOAs. Care was taken to ensure that each target item did not occur more than once per set. In addition, items from the same SOA condition were separated by at least three trials on each set. The picture and word stimulus pairs were entered into Superlab (Version 4.0; Cedrus, Phoenix, AZ) for experimental presentation. Three versions of the experiment were created, using identical stimuli, but with different interstimulus intervals (ISIs): 3500 ms, 5000 ms, and 7000 ms. For all healthy participants, the 3500-ms version was used. The version used for the PPA participants depended on their naming ability as observed during administration of the BNT, such that participants with more pronounced naming deficits were given versions with longer ISIs to allow adequate response time. For five participants (4 PPA-G and 1 PPA-L) the 5000-ms version was used, whereas two PPA-L participants were tested with the 7000-ms version. All others were tested with the 3500-ms version.

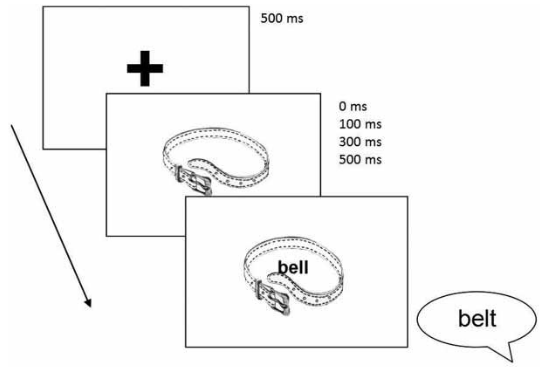

Figure 1. *Example stimulus item. Adapted with permission from Thompson, Cho, Price, et al. (2012).*

Procedure

Seated in front of an iMac computer monitor (20″, OSX 10.4.1), participants were instructed to name pictures as they appeared but to ignore the IS to the extent possible. On each experimental trial a cross was presented for 500 ms, followed by a target picture and IS in one of the four SOA conditions. The acoustic waveform of each response produced was recorded through the computer's internal microphone using Praat 5.0 software (Boersma & Weenink, 2010). A sample trial is illustrated in Figure 1.

Before beginning the experimental trials, participants were pretested on all picture stimuli used in the experiment, in order to ascertain their naming abilities and to familiarize them with the stimuli. First, each picture was presented for naming, and participants were given 5 s to respond with no feedback provided. Pictures were presented a second time when errors occurred. Picture naming performance was at least 69% correct for all participants included in the study. We also pretested participants' ability

[1]Imageability scores were unavailable for 130 stimulus items.

to read the IS by presenting each for them to read aloud, with performance ranging from 92% to 100% correct. Finally, practice trials were presented, which required participants to name target pictures overlaid with written words. These trials used stimuli similar, but not identical, to the experimental items.

Data analysis

Naming responses that matched the target pictures and occurred within the given response time were considered correct. Correct responses preceded by a filler word (e.g., *uh, pencil*) or a minimal grammatical context (e.g., *it's a pen*) were accepted, with naming latency measured from the onset of the target word. Accuracy data were analysed using mixed-effects logistic regression (e.g., Jaeger, 2008) using the languageR package in R (Baayen, 2010). The effects of group, SOA, and relatedness of the IS, and their interactions were evaluated in an additive stepwise procedure, with analysis of variance (ANOVA) tests used to compare models. Random intercepts for participant and item were included; the addition of random slopes did not improve model fit.

Errors were classified into the following types: phonological paraphasias (errors sharing at least 50% of phonemes with the target word, e.g., *punger* for *plunger*), semantic paraphasias (errors semantically related to the target word, e.g., *giraffe* for *camel*), neologistic errors (nonword errors sharing less than 50% of phonemes with the target word; e.g., *azate* for *robot)*, nonrelated responses (real-word errors unrelated to the target word, e.g., *volcano* for *raccoon*), phonological attempts (phonologically related attempts at producing the word followed by a correct production, e.g., *rope–rose*), other self-corrections (e.g., *ka–elephant),* nonresponses (in which the participant failed to respond), and other responses (e.g., *I don't know*). Nonparametric statistical tests (Kruskal–Wallis test, Mann–Whitney *U* test) were used to compare the frequency of each error type across groups.

Correct responses then were analysed for reaction time (RT), measured from picture onset to production of the first phoneme of the target word marked in the acoustic waveform. Thirty percent of the data were rescored for both accuracy and RT by an independent coder for scoring reliability purposes; overall point-to-point agreement between the primary and secondary coders was 98%. Disagreements were resolved by discussions among the experimenters. Extreme outliers (RTs less than 500 or greater than 5000 ms) were excluded (0.06% of correct responses). Following model selection (see below), outlying data points with absolute standardized residuals greater than 2.5 standard deviations were eliminated (2.71% of correct responses), following the procedure in Baayen and Milin (2010).

The reaction time data were analysed using mixed-effects linear regression, employing a stepwise additive procedure to evaluate the effects of group, SOA, relatedness, and their interactions; all models included random intercepts for participant and item, and random slopes for SOA and relatedness were included (for both participant and item) as they significantly improved model fit. Due to significant non-normality in the raw RT data, a log-transformation was applied prior to statistical analysis. These data were further transformed to *z*-scores [(participant trial-specific RT − participant mean RT)/participant *SD* RT] in order to scale the data to account for overall RT differences across groups (see e.g., Schuchard & Thompson, 2013; for a different method of data scaling, see Wilshire et al., 2007).

RESULTS

Naming accuracy

Table 4 provides the mean percentage of correct responses for each participant group. Mean accuracy for the control group was near ceiling (98.3% and 98.6% for related and unrelated trials, respectively). Both patient groups also performed quite well, but accuracy was below that of the healthy controls (PPA-G: 89.7% and 88.1% for related and unrelated items, respectively; PPA-L: 91.5% and 88.2% for related and unrelated items, respectively). The best fitting model of the data included

Table 4. *Mean naming accuracy for control, agrammatic PPA, and logopenic PPA groups at each SOA*

Group		*SOA 0 ms*	*+100 ms*	*+300 ms*	*+500 ms*	*Overall accuracy*
Control	Related	97.5 (2.4)	98.3 (1.9)	98.5 (2.0)	98.9 (1.9)	98.3 (1.5)
	Unrelated	98.3 (2.2)	98.5 (2.2)	98.4 (3.3)	99.2 (1.5)	98.6 (1.4)
Agrammatic PPA	Related	89.9 (6.9)	89.5 (8.1)	88.3 (6.6)	90.9 (5.5)	89.7 (5.3)
	Unrelated	85.7 (9.2)	85.8 (11.1)	90.4 (7.8)	90.4 (7.8)	88.1 (7.9)
Logopenic PPA	Related	92.6 (10.4)	89.7 (9.1)	89.3 (11.0)	94.5 (6.9)	91.5 (9.2)
	Unrelated	88.3 (9.7)	86.0 (15.5)	88.8 (11.0)	89.7 (13.2)	88.2 (11.9)

Note: PPA = primary progressive aphasia; SOA = stimulus onset asynchrony. Naming accuracy shown as percentage correct. Standard deviations in parentheses.

predictors for group, SOA, and relatedness, and a Group × Relatedness interaction. While the PPA groups were less accurate than controls (PPA-G: $z = -4.989$, $p < .001$; PPA-L: $z = -3.715$, $p < .001$), accuracy for the two PPA groups did not differ ($z = 1.054$, $p = .292$). Accuracy across groups was higher at SOA +500 ms than at SOA 0 ms ($z = 2.789$, $p = .005$), but no differences were observed between SOA 0 and SOAs +100 and +300 ms ($zs < +/- 1$, $ps > .5$). There was no significant main effect of relatedness ($z = .778$, $p = .437$). The Group × Relatedness interaction was driven by the PPA-L group, who were relatively less accurate on unrelated than on related trials ($z = -2.234$, $p = .026$).

Error analysis

Table 5 summarizes the frequency of each error type (percentage of all responses containing a given error type) across groups. Kruskal–Wallis tests with adjusted p-values (false discovery rate, FDR), revealed significant group differences in the frequency of phonological paraphasias, $\chi^2(2, N = 31) = 18.237$, adjusted $p < .001$, phonological attempts, $\chi^2(2, N = 31) = 18.652$, $p < .001$, self-corrections, $\chi^2(2, N = 31) = 10.563$, $p < .010$, and nonresponses, $\chi^2(2, N = 31) = 12.418$, $p < .005$. In trials with phonologically related IS, 16.5% of phonological errors (paraphasias and attempts) were considered perseverations of the interfering stimulus (17 perseverative errors out of 103 phonological errors). Follow-up pairwise tests (Mann–Whitney U, FDR-adjusted p-values) revealed that both PPA groups exhibited more phonological errors and phonological attempts than the control group ($ps < .05$). In addition, the PPA-G group produced more self-corrections than the control group ($p = .007$), and the PPA-L group produced more nonresponses than the controls ($p = .004$). There were no other significant group differences.

Reaction time analyses

Table 6 shows the mean RT for related and unrelated trials at each SOA for each participant group. The model comparison procedure resulted in a model with significant main effects of group, SOA, relatedness, and all two-way interactions between these factors. There was no significant three-way interaction, and thus this term was excluded from the model. Naming latencies were higher for both PPA groups than for controls (PPA-G: $t = 3.09$, $p = .002$; PPA-L: $t = 4.5$, $p < .001$), but there was no difference between PPA groups ($t = 0.99$, $p = .323$).[2] Faster RTs were observed at later SOAs (+300 ms: $t = -7.407$, $p < .001$; +500 ms: $t = -8.310$, $p < .001$) than at SOA 0, whereas RTs at SOA 0 and +100 ms did not differ ($t = .233$, $p < .816$). In addition, naming latencies were higher on trials with phonologically unrelated than on those with phonologically

[2]Overall group RTs were compared using an otherwise identical model in which the dependent variable was the log-transformed RT (rather than the z-score of the log-transformed RT).

Table 5. *Mean percentage of total responses containing errors of each type, by group*

	Error type							
Group	*Phonological paraphasia*	*Semantic paraphasia*	*Neologism*	*Phonological attempt*	*Self-correction*	*Unrelated*	*Nonresponse*	*Other*
Control	0.1 (0.3)	0.6 (0.8)	0.0 (0.1)	0.2 (0.4)	0.2 (0.5)	0.1 (0.2)	0.3 (0.7)	0.0 (0.0)
PPA-G	2.6 (1.7)	1.5 (1.8)	0.2 (0.3)	3.7 (3.9)	1.7 (1.4)	0.7 (1.0)	0.8 (1.2)	0.2 (0.5)
PPA-L	1.1 (1.4)	0.9 (1.2)	0.3 (0.4)	1.6 (1.5)	0.7 (0.9)	0.4 (0.6)	4.9 (7.1)	0.2 (0.5)

Note: PPA = primary progressive aphasia; PPA-G = agrammatic subtype of PPA; PPA-L = logopenic subtype of PPA. Standard deviations in parentheses.

Table 6. *Mean RTs for phonologically related and unrelated trials for control, agrammatic PPA, and logopenic PPA groups at each SOA*

		SOA			
Group		*0 ms*	*+100 ms*	*+300 ms*	*+500 ms*
Control	Related	1040.0 (117.6)	1041.3 (129.5)	967.4 (105.2)	939.4 (96.3)
N = 17	Unrelated	1094.6 (125.0)	1085.0 (123.4)	953.7 (110.9)	939.2 (93.4)
	PF	54.6**	43.7**	−13.7	−0.2
PPA-G	Related	1257.5 (183.9)	1320.0 (156.2)	1201.1 (188.6)	1124.9 (181.0)
N = 7	Unrelated	1347.0 (180.1)	1436.8 (210.6)	1268.5 (213.3)	1152.0 (194.0)
	PF	89.5*	116.8*	67.4*	27.1
PPA-L	Related	1366.0 (279.9)	1347.0 (227.1)	1161.2 (210.4)	1119.2 (237.5)
N = 7	Unrelated	1474.1 (216.2)	1431.7 (200.1)	1176.6 (217.0)	1127.8 (232.7)
	PF	108.1	84.7*	15.38	8.6

Note: RT = reaction time; PPA = primary progressive aphasia; PPA-G = agrammatic subtype of PPA; PPA-L = logopenic subtype of PPA; SOA = stimulus onset asynchrony; PF = phonological facilitation. Standard deviations in parentheses. *$p < .05$; **$p < .01$ (false discovery rate, FDR, corrected).

related IS ($t = 5.622$, $p < .001$). The Group × SOA interaction was driven by the PPA-G group responding relatively slowly at SOA +100, +300, and +500 ms compared to SOA 0 ms ($t = 2.309$, $p = .021$; $t = 5.018$, $p < .001$; $t = 2.090$, $p = .037$, respectively). The interaction between SOA and relatedness was driven by smaller PF effects at SOA +300 and +500 ms than at SOA 0 ms ($t = -6.403$, $p < .001$; $t = -5.606$, $p < .001$; respectively); SOA 0 and +100 ms did not differ ($t = -1.255$, $p = .21$). Finally, the Group × Relatedness interaction was due to larger PF effects (i.e., RT differences between unrelated and related IS) in the PPA-G group than in controls ($t = 2.893$; $p = .004$); PF effects in the PPA-L group did not differ from those in controls ($t = 1.077$; $p = .281$). To determine which SOAs were associated with larger PF effects in the PPA-G group, we ran the model separately on the data from each SOA. A Group × Relatedness interaction, reflecting larger PF effects in PPA-G participants than in controls, was found at SOA +300 ms ($t = 2.352$; $p = .019$) but not at any other SOA (ts < 1.6 ; ps > .1).

To determine which groups showed significant PF effects at the different SOAs, we performed paired two-tailed t-tests on the related versus unrelated participant mean log RTs for each group, using FDR correction for multiple comparisons. The control group showed significant PF effects at SOA 0 and +100 ms (adjusted ps < .001), but no PF effects at SOA +300 ($p = .061$, a trend towards phonological interference) and +500 ms ($p = .99$). The PPA-G group

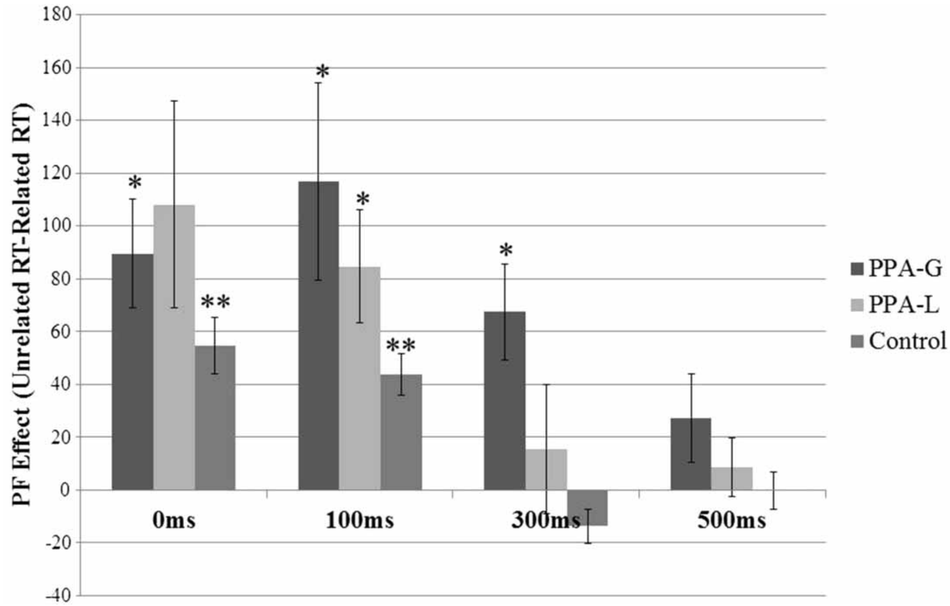

Figure 2. *Phonological facilitation effects at each stimulus onset asynchrony (SOA) for the control, agrammatic primary progressive aphasia (PPA-G), and logopenic primary progressive aphasia (PPA-L) groups. PF = phonological facilitation; RT = reaction time.* *p < *.05;* **p < *.01 (false discovery rate, FDR, corrected).*

exhibited significant PF effects at SOA 0, +100, and +300 ms (ps = .020, .022, .021, respectively) and no PF effect at SOA +500 ms ($p = .259$). The participants with PPA-L showed a marginally significant PF effect at SOA 0 ($p = .064$) and a significant PF effect as SOA +100 ms ($p = .021$) but no effects at SOA +300 and +500 ms (ps > .5). Figure 2 summarizes the observed PF effects across groups and SOAs; for clarity, PF effects are represented as the mean (raw) RT in the related condition subtracted from that in the unrelated condition.

Finally, we tested for correlations between PF effects at SOA +300 ms (the SOA in which abnormal PF effects were found in the PPA-G group) and measures of phonological processing abilities: word and nonword repetition from the NNB, three-syllable word repetition from the motor speech screening, phrase and sentence repetition ability (Rep6; Items 10–15 on the Repetition subset of the WAB), and the rate of phonological errors in both the experimental task and the narrative speech sample. In addition, to determine whether online phonological deficits were related to grammatical impairments (particularly in the PPA-G group), we calculated correlations between PF effects at SOA +300 ms and performance on the noncanonical items on the Sentence Production Priming Task of the NAVS. PF effects were calculated by subtracting the mean (z-score of log-transformed) RT in the related condition from that in the unrelated condition. These correlations were performed separately for each PPA group using Pearson correlations with FDR correction. No statistically significant correlations were found.

DISCUSSION

One of the central goals of research in primary progressive aphasia is to identify the mechanism of the pervasive anomia associated with this disorder. In particular, it is important to determine whether deficits in lexical–semantic processing, phonological processing, or both are at the root of naming impairments in each of the three variants of PPA. Previous online studies have shown that lexical–semantic processing may be impaired even in nonsemantic variants of PPA—that is, PPA-G and PPA-L (Rogalski et al., 2008; Thompson, Cho, Price, et al., 2012; Vandenberghe et al., 2005). In the present study, we investigated the phonological processes that support naming in the agrammatic (PPA-G) and logopenic (PPA-L) variants of PPA. Previous studies have suggested that phonological processing is differentially impaired in these subtypes of PPA (Ash et al., 2010; Clark et al., 2005; Croot et al., 2012; Gorno-Tempini et al., 2004, 2008; Mesulam et al., 2012; Rohrer et al., 2010; Wilson et al., 2010). However, phonological processing has not been studied using online tasks in this patient population, and thus little is known about the nature of phonological processing deficits in PPA-G and PPA-L (i.e., whether phonological word form retrieval and/or phonological encoding is impaired) and how these deficits contribute to anomia. In the present study, we used the picture–word interference paradigm (PWIP) to investigate phonological processing in real time during naming in people with PPA-G and PPA-L as well as in age-matched healthy controls. In doing so, we were able to compare the time course and magnitude of phonological facilitation (PF) effects in both PPA subgroups with that of controls, with the aim of gaining insight into the source of naming deficits in PPA.

Consistent with previous studies (e.g., Damian & Martin, 1999; Hashimoto & Thompson, 2010; Lupker, 1982; Meyer & Schriefers, 1991; Rayner & Springer, 1986; Schriefers et al., 1990; Starreveld, 2000; Starreveld & La Heij, 1995, 1996), the control group performed at ceiling on the task and showed significant PF effects at early SOAs (0 and +100 ms). No significant PF effects at later SOAs (+300 and +500 ms) were found, supporting previous studies using the PWIP. These findings indicate that normal naming involves rapid phonological processing—that is, phonological word form retrieval and phonological encoding.

Participants with PPA-G and PPA-L performed with comparable speed and accuracy on the naming task, with both groups responding more slowly and less accurately than the control group, indicating slowed and impaired processes supporting naming. The two PPA groups also exhibited a similarly high rate of phonological errors, reflecting phonological processing impairments in both groups. However, these offline markers of phonological processing deficits were accompanied by abnormal PF effects only in the PPA-G group. Relative to the control group, the PPA-G, but not the PPA-L, group showed protracted PF effects. Like the controls and the PPA-L group, the PPA-G group exhibited significant PF effects at SOA = 0 ms and +100 ms; however, the PPA-G group also exhibited an abnormal PF effect at +300 ms. This abnormal PF effect, emerging at a late SOA in PPA-G, probably reflects deficits in phonological encoding. Consistent with this interpretation, Laganaro et al. (2009), in an event-related potential (ERP) study using a picture-naming task with participants with phonological encoding deficits resulting from stroke, found that the ERP signal in these participants began to deviate from that of unimpaired controls around 290 ms after picture onset. In addition, these results are consistent with neurological evidence indicating that the left inferior frontal gyrus, a typical site of cortical atrophy in PPA-G (Gorno-Tempini et al., 2004, 2008, 2011; Mesulam et al., 2009, 2012; Rohrer et al., 2010), supports phonological encoding during naming (Indefrey, 2011; Indefrey & Levelt, 2004; Papoutsi et al., 2009). Interestingly, PF effects at SOA +300 ms were not correlated with grammatical ability (noncanonical sentence production), suggesting that phonological and grammatical processing deficits may be independent in PPA-G.

PF effects at SOA +300 ms also were not correlated with offline measures of phonological processing ability, including phrase and sentence repetition and the rate of phonological errors in the experimental task and narrative speech samples, which were impaired to some extent in both PPA patient groups. This finding is not surprising in that performance on offline measures reflects several components of phonological processing, including phonological working memory, word form retrieval, and encoding. Indeed, online measures using the word interference paradigm are more sensitive for identifying specific processing impairments than offline measures. Specifically, PF effects in the present study probably reflect spreading activation between segments (Meyer & Schriefers, 1991; Roelofs, 1997; Schriefers et al., 1990), rather than between lexical items (Levelt et al., 1999; Starreveld, 2000; Starreveld & La Heij, 1995, 1996), suggesting that such effects reflect phonological encoding (a segmental process), but not phonological word form retrieval (a lexical process). It is plausible, and the present data suggest, that phonological deficits in PPA-L stem from phonological word form retrieval rather than phonological encoding, whereas individuals with PPA-G present with the opposite deficit pattern: impaired phonological encoding. This behavioural pattern coincides with cortical atrophy in PPA. PPA-L is associated with atrophy in the left temporoparietal junction (Gorno-Tempini et al., 2004, 2008, 2011; Mesulam et al., 2009, 2012; Rohrer et al., 2010), which has been argued to support phonological word form retrieval (Graves et al., 2007, 2008; Indefrey, 2011; Indefrey & Levelt, 2004; Wilson et al., 2009), and PPA-G is associated with atrophy in the left IFG, argued to be engaged to support phonological encoding as well as subsequent phonetic encoding and articulatory processes (Indefrey & Levelt, 2004; see also Hickok & Poeppel, 2007; Papoutsi et al., 2009). However, functional magnetic resonance imaging (fMRI) studies using the PWIP have reported decreased activation in left posterior temporal cortex associated with phonological facilitation (de Zubicaray & McMahon, 2009; de Zubicaray, McMahon, Eastburn, & Wilson, 2002, cf. Bles & Jansma, 2008; though see Abel et al., 2009; note that priming effects are typically associated with decreased activation in regions supporting stimulus processing; e.g., Lebreton, Desgranges, Landeau, Baron, & Eustache, 2001). Thus, further research is needed to determine whether atrophy in the temporoparietal junction in PPA-L results in impaired phonological word form retrieval.

CONCLUSION

Previous studies have suggested that the agrammatic and logopenic variants of PPA are associated with phonological processing deficits. The present study used a picture–word interference paradigm to test whether people with PPA-G and PPA-L show evidence of impairments at different substages of phonological processing as naming unfolds. Compared to healthy control participants, participants with PPA-G exhibited protracted PF effects, which may reflect impaired phonological encoding. This processing deficit may be caused by atrophy within the left inferior frontal gyrus, which has been argued to support phonological encoding. Despite phonological deficits evident in offline measures, the PPA-L group exhibited normal online PF effects. These findings indicate that deficits in phonological processing may contribute to anomia in both agrammatic and logopenic variants of PPA, but highlight important differences in the source of these deficits for the two patient types. Importantly, these differences may associate with unique patterns of naming (and other linguistic) declination as well as neural degeneration.

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APPENDIX

Target stimuli and interfering stimuli

Table A1. *Living targets*

		Related IS				*Unrelated IS*			
Item No.	*Target*	*SOA 0*	*SOA +100*	*SOA +300*	*SOA +500*	*SOA 0*	*SOA +100*	*SOA +300*	*SOA +500*
1	beetle	being	beacon	beaker	behalf	towel	guitar	hormone	sofa
2	broccoli	broadcast	bronchitis	bronze	brothel	skeleton	champion	gravity	pharmacy
3	cactus	caddie	candle	camera	campus	magic	fountain	ribbon	pizza
4	camel	cannon	candor	cabbage	canvas	fashion	lagoon	detour	wallet
5	cat	cap	cab	cash	can	gas	boot	hoop	jet
6	chicken	chin	chip	chisel	chimney	whisper	shower	plastic	glacier
7	elephant	element	elegant	elderly	eloquence	umbrella	adventure	insurance	oxygen
8	grape	grade	grace	grain	grate	brick	sleep	plate	shell
9	horse	hoard	horn	horror	horizon	vase	roof	gift	watch
10	lemon	ledger	lesson	lecture	leopard	tower	sewer	cassette	mansion
11	lion	liar	libel	lighter	lightning	curtain	magnet	rocket	tool
12	mushroom	muffler	mustard	muscle	mugger	compass	denim	napkin	congress
13	orange	organ	orbit	order	orphan	iron	album	engine	aspirin
14	pumpkin	punch	puppy	public	publisher	tablet	jelly	harpoon	mountain
15	rabbit	racket	rattle	raft	rally	basket	circle	filter	nickel
16	raccoon	rapture	rampart	ransom	rancher	harbor	motel	lapel	quarter
17	rose	rope	rogue	roach	robe	card	paste	beach	tire
18	sheep	sheet	sheen	sheath	sheaf	phone	chalk	bridge	wheel
19	spider	spice	spike	spine	spiral	photo	thermos	planet	prairie
20	tomato	tobacco	toboggan	touch	tongue	galaxy	jewellery	volcano	cathedral

Note: IS = interfering stimulus; SOA = stimulus onset asynchrony (in ms).

Table A2. *Nonliving targets*

		Related IS				*Unrelated IS*			
Item No.	*Target*	*SOA 0*	*SOA +100*	*SOA +300*	*SOA +500*	*SOA 0*	*SOA +100*	*SOA +300*	*SOA +500*
1	anchor	anger	angle	ancient	ankle	umpire	infant	elbow	orchard
2	balloon	bassoon	banana	barrage	baton	gender	sausage	pirate	nutmeg
3	belt	bet	bell	bend	bench	fig	cook	toe	colt
4	bowl	boat	bone	boa	bolt	run	leaf	jaw	ranch
5	bucket	bubble	budget	buddy	butter	cobra	dentist	tenant	pony
6	crib	crick	cripple	critic	crimson	drug	fruit	knight	shrimp
7	desk	depth	deaf	debt	deck	paint	jazz	fight	beast
8	hammer	habit	hamster	hanger	hamper	turnip	diary	waitress	baboon
9	ladder	lantern	landscape	laughter	lather	fossil	devil	hero	riddle
10	lamp	lab	lamb	latch	lack	yawn	bomb	hook	dove
11	pants	past	pass	patch	path	kiss	mail	hound	tomb
12	pen	pet	pest	pedestal	peasant	rain	germ	cell	joke
13	pencil	petal	pebble	pellet	pedal	turtle	servant	hockey	lobster
14	pillow	pigeon	picnic	pistol	pickle	wizard	hippie	fiber	honey
15	plunger	plug	plum	plus	plunder	shepherd	grammar	rhubarb	knuckle
16	robot	roller	roman	romance	rodent	zero	bachelor	warden	tortoise
17	ruler	ruin	rumor	ruby	rubric	coffee	reptile	lawyer	poison
18	sock	sob	sod	solve	song	king	tea	beak	tooth
19	tank	tab	tack	tap	tag	pork	judge	beard	Hug
20	tie	tide	title	tile	tights	sand	nurse	ear	hen

Note: IS = interfering stimulus; SOA = stimulus onset asynchrony (in ms).

Table A3. *Fillers*

		Related IS				*Unrelated IS*			
Item No.	*Target*	*SOA 0*	*SOA +100*	*SOA +300*	*SOA +500*	*SOA 0*	*SOA +100*	*SOA +300*	*SOA +500*
1	bench	best	beck	belch	bend	noon	gin	valve	disc
2	cannon	cackle	capsule	captor	caption	kernel	soldier	vision	supper
3	clock	cloth	clog	clot	closet	glass	bleach	plant	sleeve
4	donut	dome	donor	domain	docent	valley	digit	neon	cushion
5	leaf	leak	league	lease	leash	toy	wing	golf	dime
6	octopus	octagon	occupant	octave	octane	accountant	universe	idealist	editor
7	rake	race	rage	rate	rave	gold	waist	deal	mess
8	sun	sum	suck	suds	subject	call	dirt	bunk	vote
9	watch	wad	wand	waft	wasp	booth	tar	hole	deed
10	whistle	whimper	whimsy	whip	whisky	princess	grocer	fragrance	chapel

Note: IS = interfering stimulus; SOA = stimulus onset asynchrony (in ms).