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False recognition of incidentally learned pictures and words in primary progressive aphasia^{*}

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Abstract

Recognition memory was tested in patients with primary progressive aphasia (PPA), a language based dementia with relative preservation of memory for at least the first 2 years. The goal of the study was two-fold: (1) to compare true and false recognition rates for words versus pictures in patients with PPA and cognitively intact controls and (2) to determine if the semantic relatedness of distracters-to-targets influences recognition memory performance. Overall, performance of PPA patients was worse for words than pictures. PPA patients and healthy elderly controls showed similar recognition rates for studied items. However, the patients had significantly more false alarms than controls, particularly to semantically related items. This suggests that the aphasia in PPA patients contributes to their difficulty in selecting among items within a semantic class.

Keywords

Dementia; Frontotemporal dementia; Recognition memory; Language; Semantic processing

1. Introduction

Primary progressive aphasia (PPA) is typified by isolated and progressive dissolution of language for at least the first 2 years of illness (Mesulam, 1982, 2001, 2003; Mesulam, Grossman, Hillis, Kertesz, & Weintraub, 2003; Weintraub, Rubin, & Mesulam, 1990). Clinically, the most common symptom in the early stages of PPA is anomia (difficulty thinking of words in conversation and/or deficiencies in object naming; Mesulam, 2001). Evidence from functional neuroimaging (e.g. PET, SPECT, and fMRI) and postmortem examinations points to abnormalities in the frontal, perisylvian and temporal cortices, regions normally involved in language function (Chawluk et al., 1986; Kempler et al., 1990; McDaniel, Wagner, & Greenspan, 1991; Mesulam & Weintraub, 1992a; Radanovic et al., 2001; Turner, Kenyon, Trojanowski, Gonatas, & Grossman, 1996).

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The neuroanatomical proclivity of PPA suggests that memory should be intact since the medial temporal areas associated with memory storage are not affected, at least initially. Caregivers and patients corroborate the absence of memory deficits with anecdotal reports of relatively intact episodic memory in patients' activities of daily living. In contrast to these reports, some studies have noted poor memory performance, largely based on tests of verbal recall [e.g. Rey Auditory Verbal Learning Test (RAVLT)] (Zakzanis, 1999). Impaired memory test performance is thought to arise as a secondary consequence of the patients' language disorder (Snowden, Neary, & Mann, 1996), making unclear the degree to which memory is truly compromised in PPA. Therefore, judging the integrity of memory in PPA is largely reliant upon collateral information about the patient's everyday functioning. Since few studies have examined memory in PPA, the extent to which memory deficits are a secondary result of aphasia versus an accompanying deficit of the disorder remains uncertain. The study described in this paper reports on false recognition in patients with PPA.

False recognition, the phenomenon whereby individuals incorrectly claim to have previously encountered a novel stimulus, has been well studied in healthy subjects of all ages (for a review see Roediger, 1996; Schacter, 1996). Healthy populations show elevated false recognition rates when novel test items are semantically related to previously studied stimuli (Roediger & McDermott, 1995). The explanation for the high false recognition rates in this situation is a topic of much debate. One hypothesis is that studied items can implicitly trigger processing of related items. For example if the word *fire* is given in the study phase, this may implicitly activate related words such as *hot*. Thus, when the word *hot* appears in the subsequent test phase, the likelihood of endorsing the word *hot* as being a 'studied' item is high (Roediger & McDermott, 1995). Another explanation is that in the face of weak memory for individual items, subjects respond on the basis of 'gist'. That is, they have difficulty recollecting specific characteristics of the studied items and consequently respond on the basis of more general characteristics of those items. Responding on the basis of gist may also result in the false recognition of the word *hot* in the example given above.

Yet another theory of false recognition, termed source-monitoring, developed by Johnson and colleagues (Johnson, Hashtroudi, & Lindsay, 1993), identifies two distinct factors that contribute to false recognition: (1) the extent to which the qualitative characteristics of true and false memories are similar and (2) the degree to which subjects are able to differentiate between their true and false memories. Still other studies have been designed to look at the relative contribution of recollection versus familiarity in creating false memories with the latter being more sensitive to increased false alarms as a result of the individual relaxing his/her response criteria (for a review see Yonelinas, 2002).

Previous studies have identified that the rate of false recognition is variable depending on the study design (e.g. number of stimuli, degree of similarity between foils and targets, length of presentation of the stimuli, stimulus format, etc.). Elevated false recognition rates have been demonstrated for semantically related pictures as well as words (Koutstaal & Schacter, 1997). Though complete understanding of the false recognition phenomenon has not been achieved, the aforementioned theories provide plausible explanations for elevated false recognition centering around idiosyncrasies in the encoding of target items, which in-turn result in faulty increased memory strength of the new-*related* items.

False recognition has been studied in several neurological populations, such as patients with amnesia due to acute medial temporal lobe damage (Koutstaal, Schacter, Galluccio, & Stofer, 1999; Koutstaal, Verfaellie, & Schacter, 2001; Schacter, Verfaellie, & Anes, 1997) and patients with Alzheimer's disease (AD) (Budson, Daffner, Desikan, & Schacter, 2000; Budson et al., 2003). A nearly ubiquitous finding in these patients is the presence of both impaired true recognition (correct recognition of a studied item) and lower false recognition rates (fewer

false alarms) to novel items related to the studied items. These recognition memory deficiencies are thought to reflect impairments on two levels: (1) item-specific recollection and (2) the ability to utilize gist information (e.g. Schacter, Verfaellie, & Pradere, 1996). Item-specific recollection refers to the ability to remember distinct characteristics about a presented item so that you can distinguish it from another item. For example, distinguishing between two different types of dogs would be item specific recollection, whereas gist refers to the ability to grasp the general theme presented in the stimuli, such as recognizing that items are from the category *animals* and, more specifically, dogs. Using a test of recognition memory, Koutstaal et al. (2001), found that patients with amnesia have disproportionate deficits in item-specific true recognition compared to gist memory, implying that the two forms of memory may draw on processes supported by different temporal lobe regions. Specifically, they propose that familiarity-based processing (e.g. gist memory) is strongly dependent on medial temporal neocortical regions while successful recognition of one-of-a-kind items may be more reliant on the hippocampus (Koutstaal et al., 2001).

Recently, semantic dementia (SD), a syndrome clinically characterized as having degraded semantic memory (Hodges, Patterson, Oxbury, & Funnell, 1992; Snowden et al., 1996), was used as a model to demonstrate the dissociation of item-specific recollection from gist memory (Simons et al., 2005). Two tests were used to compare recognition memory. First, a recognition test contrasted performance for nameable target objects versus that for semantically related foils. The second test compared recognition for abstract, unnamable target objects versus that for perceptually similar foils. Results from the first study indicated that compared to controls, SD patients had reduced true and false recognition (fewer false positive errors) for semantically grouped exemplars, which was thought to be attributable to impairments in extracting and/or utilizing gist information for semantically related categories of objects. In contrast, the results from the second study of abstract objects showed there were no significant differences in true or false recognition between patients and controls, suggesting that in SD, there is a greater utilization of gist when objects were related perceptually (abstract objects) than when they were related semantically (nameable objects) (Simons et al., 2005). Simons and colleagues (2005) postulated that SD patients' deficits in recognition were a result of the selective degradation of semantic knowledge associated with the syndrome.

The present study investigated true and false recognition in a group of PPA patients and cognitively intact controls. The study was designed to examine the impact of language decline on memory processes in PPA using a recognition memory paradigm. The study deliberately required no verbal output and minimal directions to decrease the confounding effects that aphasia may have on verbal responses and/or auditory language comprehension. The goal of the study was two-fold: (1) determine the effects of PPA on verbal (words) and nonverbal (pictures) recognition memory and (2) determine if the semantic relatedness of distracters-to-targets influences memory performance. Since PPA is a disorder of language, it was hypothesized that word recognition would be inferior to picture recognition.

Recently a semantic priming study showed that in PPA, picture naming reaction time is slower when the picture is preceded by a semantically related prime than by an unrelated prime suggesting that selection among words from the same semantic field is abnormal in this disorder (Vandenberghe et al., 2005). Based on these findings it was predicted that participants in the current study would have higher false recognition rates for semantically related versus unrelated stimuli. Signal detection analyses were used to disentangle the contributions of bias and sensitivity to recognition performance.

2. Method

2.1. Participants

Sixteen cognitively intact elderly normal control subjects (NC) and 16 patients with a clinical diagnosis of primary progressive aphasia (PPA) based on published criteria (Mesulam, 2001, 2003; Table 1), participated in the experiment. All participants were recruited from the Clinical Core registry of subjects at the Northwestern Alzheimer's Disease Center (NADC). Patients were asked to participate in the study at the time of their scheduled medical visit. Control subjects were community-dwelling healthy volunteers enrolled in the NADC Clinical Core registry. Written informed consent was obtained from all subjects and the study was approved by the Institutional Review Board at Northwestern University.

Upon enrollment into the NADC registry, a detailed demographic and medical history interview was completed for each subject. During this interview additional information was obtained from the patients about the onset of symptoms (approximate date and description of presenting symptom(s)), the current symptoms as they are evident to others interacting with the patient, and the impact of the disorder on activities of daily living. Each participant was required to have a *study partner*, a relative, or an individual who was well acquainted with the research participant and agreed to serve as an informant for the subject. Both the subject and his/her study partner (caregivers, in the case of patients) were questioned to ensure accuracy of the reported information. Patients underwent neurological examinations and controls had a neurological screening exam. Participants received a neuropsychological test battery to assess overall cognitive functioning. To participate in the study, control subjects must have achieved a score within two standard deviations of age- and education-adjusted normative values on all tests, and have no subjective complaints of memory problems. Study partners corroborated the absence of difficulty in activities of daily living and memory deficits in the control sample. There was no history of neurological, medical, or psychiatric problems in the control group and none were taking psychoactive medications. PPA patients were taking medications as follows: seven psychoactive, typically antidepressants; four cholinomimetic; four memantine. These medications were not anticipated to affect performance on the experimental task. All subjects were required to have a minimum visual acuity of 20/40 (correction permitted) in order to view the stimuli.

A diagnosis of PPA had been made by the consensus of a speech pathologist, a neurologist, and a neuropsychologist at the NADC using the criteria outlined by Mesulam (2001, 2003; Table 1). Clinical neuroimaging (e.g. MRI, PET) was obtained to further assist in the differential diagnosis and to highlight features that are consistent with a diagnosis of PPA such as atrophy of the left perisylvian area and/or metabolic changes in that region (Chawluk et al., 1986; Mesulam & Weintraub, 1992b; Turner et al., 1996).

The level of language comprehension was characterized in the patients quantitatively [using either the “yes/no” comprehension subtest of the western aphasia battery (Kertesz, 1982) or the Complex Ideational Material subtest of the Boston Diagnostic Aphasia Exam (Goodglass & Kaplan, 1983) and qualitatively, based on reports from comprehensive neurological, neuropsychological, and speech-language pathology examinations. Only patients with mild language comprehension deficits were included in this study (complex ideation scores were ≥ 7 ; WAB yes/no scores ≥ 54). Due to the nature of the task, PPA patients who confused “yes” and “no” were excluded from the study. PPA patients who had difficulty with both naming and comprehending the names of objects, what we have called “two-way” naming deficits, were excluded from this study.

Table 2 summarizes demographic information for the groups. There were no differences in gender representation ($\chi^2 = 0.51$, d.f. = 1, $p = 0.48$), age ($t_{(30)} = 0.39$, $p = 0.70$) or education

($t_{(30)} = -0.38, p = 0.71$) between the two groups. Three of the PPA patients and two of the control participants were left-handed.

Mini Mental State Examination (MMSE) score, was used to assess overall level of dementia severity (Folstein, Folstein, & McHugh, 1975). The Activities of Daily Living Questionnaire (ADL-Q) was given as a measure of the PPA patients' functional abilities (Johnson, Barion, Rademaker, Rehkemper, & Weintraub, 2004). Visual confrontation naming was tested with the Boston Naming Test (BNT: Kaplan, Goodglass, & Weintraub, 1983). Lexical fluency (FAS: Benton & Hamsher, 1989) and semantic fluency (animals; Morris et al., 1989) tests were administered. Table 3 summarizes individual test performance, clinical language characterization, PPA subtype as defined by Gorno-Tempini et al. (2004), and imaging results of the PPA patients. The means and standard deviations for the entire control group were reported where applicable.

2.2. Design and materials

There were two phases to the experiment, a study phase followed by a test phase. Stimuli consisted of 120 different concrete items, 60 words and 60 black-and-white line drawings of nameable objects. The names of the objects and the words were matched for frequency and length. Mean word length was 5.7 letters. The mean frequency of the words in print was 18.13 using the Brown Corpus (Kucera & Francis, 1967). Stimuli were matched for frequency and length by format (word/picture) and by presentation (old/new). Words were presented horizontally on a computer monitor in bold lowercase 72 point times new roman font. Equal numbers of words and pictures were presented in both the study and test phases.

Forty of the stimuli (20 words and 20 pictures) were *targets* and were shown in both the study and test phases. Target items were equally grouped into four semantic categories (animals, fruits/vegetables, clothing, and tools/manipulable objects). The remaining 80 stimuli were only displayed in the test phase and were divided into two equal groups by type: *semantically related foils* and *semantically unrelated foils*. The semantically related foils contained exemplars from the same categories as the target stimuli, while the unrelated foils contained an equal number of items from four different categories (body parts, transportation, household items, and musical instruments). Each type of stimulus (targets, semantically related foils, and semantically unrelated foils) contained an equal number of pictures and words.

All stimuli were presented in the center of a computer monitor in black and white using SuperLab Pro version 2.0.3. Each target stimulus was presented for 5 s in the study phase. During the test phase, each item was displayed until the subject made a response up to 5 s. There was a 50 ms interval between the response and the appearance of the next item. All responses were made using labeled buttons on a computer keypad. Stimulus presentation was randomized in both the study and test phases though all subjects received the stimuli in the same random order.

2.3. Procedure

The stimuli were presented in two successive phases (study and test). In the study phase, subjects were instructed to view, one at a time, the 40 *target* stimuli and were not specifically asked to remember them. Instead, they were required to respond 'yes' or 'no' (by pressing the appropriate button on the computer keypad) to the question, 'Does (the item) fit in one hand?' The size judgment made by the subjects was designed to have subjects encode the item without the explicit instruction to remember. Two examples were given to ensure task comprehension and all subjects responded correctly to these before testing was initiated. Auditory instructions accompanied by written directions on the computer screen were given for both the study and

test phases to promote optimal comprehension for the PPA patients. Subjects were asked to decide and respond as quickly as possible.

In the test phase, administered immediately following the study phase, participants were given an 'old-new' recognition test consisting of the 40 target items plus an additional 80 new *foil* items. Here, participants were asked to indicate, as quickly as possible, whether or not each item was one they had seen in the previous phase (yes/no). Subjects had a maximum of 5 s to respond to each item. The surface form (picture/word) of the test phase stimuli never changed (i.e. if they saw the word 'dog' in the study phase then 'dog' would appear as a word again in the test phase).

3. Statistical analysis

Patient data were analyzed as a group since there were no obvious performance differences by PPA subtype (i.e., logopenic, agrammatic). All raw data were converted to proportions of *hits* (items that are correctly identified as being 'old' or studied) and *false alarms* (erroneous identification of a novel item as an 'old' or studied item) for analysis. Proportions were created by dividing the number of hits or false alarms by the total possible number of hits or false alarms. False alarms were further divided and analyzed as 'related' and 'unrelated' false alarms, since one of the interests of this study was whether or not participants made more false alarms to semantically related items. Hits and false alarm responses were also separated and analyzed by their format (word or picture).

Results from four main analyses are described: (1) overall comparisons of 'true' and 'false' recognition rates (Budson et al., 2000), which includes a global comparison of performance followed by a more in-depth investigation of responses by stimulus format and level of relatedness; (2) a false alarm analysis; (3) signal detection analysis to provide estimates of sensitivity (d') and bias (c) (Macmillan & Creelman, 1991); (4) a correlation analysis to determine if there is a relationship between recognition memory performance and the degree of anomia, as measured by the Boston naming test (BNT).

3.1. Overall recognition memory analysis

True and false recognition rates (regardless of item format) were compared by subject group using t -tests. To determine if image format (word/picture) influenced performance, the proportion of correct responses (the sum of the hits and correct rejections) were compared. An ANOVA with one between-subjects factor of diagnosis (PPA, control) and two within-subjects factors: format (word/picture), and response type (hits, semantically related correct rejections, semantically unrelated correct rejections) was used to make the comparisons.

3.2. False alarm analysis

A $2 \times 2 \times 2$ ANOVA using false alarms as the dependent variable was conducted, to determine if semantic relatedness or stimulus format had an impact on false-alarm rates. The within-subjects factors were relatedness (related or unrelated) and the stimulus format (words or pictures), and the between-subjects factor was diagnosis (PPA or control). Post hoc analyses were completed and described as appropriate.

3.3. Signal detection analysis

Signal detection analyses were used to determine whether the main findings of the experiment are attributable to changes in sensitivity or bias (Budson et al., 2000; Macmillan & Creelman, 1991; Westerberg & Marsolek, 2003). Analyses were conducted as recommended by Macmillan and Creelman (1991), using d' as an estimate of sensitivity and c as an estimate of response bias. Data were transformed, as suggested by Snodgrass and Corwin (1988), to

compute corrected hit and false alarm rates using the formula: $p(x) = (x + 0.5)/(n + 1)$ rather than x/n , where x = the number of hits or false alarms and n = the total number of items possible. Sensitivity, the degree to which “old” items can be discriminated from “new” items, was calculated using $d' = z(H) - z(FA)$ (where H = proportion of hits and FA = proportion of false alarms) and was defined as the standard distance between the mean of the target distribution (hits) and the foil distribution (false alarms). When $d' = 0$ subjects were unable to discriminate between targets and foils and as d' increased so did one's ability to discriminate between targets and foils. Bias, the general tendency to produce primarily ‘old’ or ‘new’ responses, was calculated using $c = (-0.5) [z(H) + z(FA)]$. The basic sensitivity and bias formulas were used to examine two types of recollection. The first analysis compares hits versus unrelated false alarms where ‘unrelated’ false alarms were entered into the bias and sensitivity computations instead of all false alarms. The second comparison, hits versus related-foil false alarms, was computed by entering in the ‘related’ false alarms into the bias and sensitivity formulas.

3.4. Correlation analysis

To investigate the relationship between PPA patients' naming ability and recognition memory performance, a Pearson's correlation test was conducted using the patients' Boston Naming Test (BNT) scores and recognition memory test performance scores by response type, relatedness and format.

4. Results

4.1. Overall recognition memory results

Table 4 reports the proportion of ‘old’ responses to previously studied words, ‘old’ responses to related foils that are semantic associates of previously studied items, and ‘old’ responses to unrelated foils that are not semantic associates of the previously studied items. Within- and between-group comparisons were conducted and results are detailed below.

PPA patients and the healthy older controls had similar proportions of true recognition, that is the proportion of old items that were so identified (0.83 versus 0.85 respectively, $t_{(30)} = 0.54$, $p = 0.597$). Compared to control subjects, however, PPA patients had higher false recognition rates for both related (mean = 0.21 versus 0.09, respectively, $t_{(21.1)} = -3.56$, $p = 0.002$; Table 4) and unrelated foils (mean = 0.07 versus 0.02, respectively; $t_{(18.5)} = -2.90$, $p = 0.009$; Table 4). Further description of the false recognition analysis is discussed below.

The image format performance comparison revealed two significant main effects irrespective of diagnosis. First, accuracy for pictures was better than words ($F_{(1,30)} = 15.40$, $p \leq 0.001$) and second was an effect of response type, where the greatest accuracy was found for the unrelated items ($F_{(1,30)} = 37.82$, $p \leq 0.001$). Two significant interaction effects were present. First, a format by diagnosis interaction showed that PPA patients had disproportionately reduced accuracy for word stimuli ($F_{(1,30)} = 6.92$, $p = 0.013$; Fig. 1). The second interaction, response type by format, displayed that regardless of diagnosis, participants performed significantly worse on the target words than target pictures ($F_{(1,30)} = 9.26$, $p = 0.001$).

4.2. False alarm analysis results

False alarm analysis results revealed two significant main effects, relatedness and diagnosis, which showed there were more false alarms to related items than unrelated items regardless of diagnosis ($F_{(1,30)} = 55.285$, $p < 0.001$) and higher false alarm rates in the PPA patients versus healthy controls irrespective of stimulus format ($F_{(1,30)} = 14.541$, $p = 0.001$). In addition, there was a significant relatedness by diagnosis interaction, whereby patients had significantly higher false alarm rates to related items than unrelated items ($F_{(1,30)} = 4.317$, $p = 0.046$), a mean

difference which was twice that of controls (Table 4). There were no main effects for stimulus format ($F_{(1,30)} = 0.716, p = 0.404$).

4.3. Signal detection analysis results

Results from the signal detection analysis revealed no differences in bias between the PPA patients and control subjects in item-specific recollection for the related or unrelated foils (Table 5), with both groups showing a tendency to respond new (unrelated: $t = 1.765, p = 0.09$; related: $t = 1.894, p = 0.07$). However, there were significant differences in sensitivity (related: $t = 2.346, p = 0.026$; unrelated: $t = 2.409, p = 0.024$; Table 6). Compared to the controls the PPA patients were less able to discriminate between targets and foils (either related or unrelated).

4.4. Correlation analysis results

Results indicated no significant correlations between BNT score and general recognition memory response type (e.g. hits, false alarms; $p > 0.1$, two-tailed) or degree of relatedness ($p > 0.2$, two-tailed). However, a significant correlation emerged between recognition of picture stimuli and BNT score, whereby, a higher BNT score was associated with a greater number of hits to picture stimuli ($p = 0.004$).

5. Discussion

The present study examined recognition memory performance in patients with primary progressive aphasia. Compared to healthy controls, PPA patients showed similar levels of true recognition. Both patients and controls had more false memories for semantically related versus unrelated items, suggesting that both groups encoded the essence of the semantic categories well. However, the PPA patients had a higher false alarm rate specifically for the related items, suggesting they have difficulty discriminating among items from the same category. Accuracy for word target recognition (hits) was significantly lower than for picture target recognition in both subject groups. However, PPA patients had significantly worse accuracy for all word stimuli compared to the controls. Signal detection analysis indicated PPA patients and controls were similar in their bias or tendency to produce predominantly 'old' or 'new' responses. However, the PPA patients had significantly worse sensitivity scores suggesting that they were less able to discriminate between targets (studied items) and foils (new items). Compared to the PPA patients the controls were better at using item-specific memory to reject the foils. Taken together these results demonstrate that in PPA there is preserved target recognition but an elevated level of false alarms, particularly for the semantically related foils suggesting a deficit in selection among items within a semantic class.

Prior studies suggest that normal subjects' memory is best for target (studied) words, somewhat less accurate for related (nonstudied) words and weakest for unrelated (nonstudied) words (Roediger & McDermott, 1995; Westerberg & Marsolek, 2003). Based on the findings of previous studies, it was expected that healthy controls would show increased false-alarm rates to related-foil items versus unrelated-foil items.

In addition to the differences from healthy elderly controls, the PPA patients showed a different pattern of recognition memory performance from previous reports of patients with other forms of dementia. Using a somewhat different paradigm (c.f. Roediger & McDermott, 1995), both AD and SD patients showed reduced false recognition (fewer false alarms) contrasted with impairments in remembering studied items (Budson et al., 2003; Simons et al., 2005, respectively). AD patients' poor performance on tests of recognition is thought to be attributable to their selective impairment of explicit memory as a consequence of the devastation of medial temporal lobe structures (Budson et al., 2003). In contrast, in SD patients,

deficits in recognition memory instead are due to selective degradation of access to semantic knowledge believed to be linked to selective degeneration of the anterior-temporal lobes (Simons et al., 2005). The loss of semantic knowledge, in turn, affects their ability to encode the gist of the objects.

Our data instead suggest that PPA patients' memory may be affected by yet another mechanism. They did not have difficulty accessing semantic category knowledge but could not select the appropriate item from all items within a category. Most of our subjects had agrammatic or logopenic forms of PPA, as defined by Gorno-Tempini and colleagues (2004). These forms of PPA, unlike the semantic form, are associated with atrophy in the anterior inferior frontal gyrus, premotor cortex and anterior insula regions (agrammatic/nonfluent subtype) or atrophy of the posterior temporal and parietal regions (logopenic subtype) rather than the anterior-temporal region (semantic subtype). The relative sparing of the medial temporal cortex and anterior-lateral temporal areas in PPA may therefore explain the relative sparing of true recognition and ability to grasp gist in these patients.

False recognition has not been previously examined in PPA. However, a recent study reported that priming with semantically related words hinders picture naming in patients with PPA, suggesting that selection among words from the same semantic field is abnormal (Vandenberghe et al., 2005). The results from the present study support this finding by demonstrating elevated false recognition of semantically related items in the absence of recognition impairments of studied items. We suggest that the PPA patients encoded the category specific information well but were easily drawn to distracters from the same semantic category as a result of their language deficits. The high rate of false recognition of related items also suggests that PPA patients encode linguistic stimuli at a semantic level despite their other language deficits. Had unnamable objects been included in the stimuli, perhaps the results would have differed.

Previous research in control populations has shown a robust superiority for picture versus word stimuli in recognition and recall memory (Grady, McIntosh, Rajah, & Craik, 1998; Maisto & DE, 1992; Noldy, Stelmack, & Campbell, 1990; Sperber, McCauley, Ragain, & Weil, 1979; Standing, Conezio, & Haber, 1970) with the rationale that pictures are more richly encoded than words (Rajaram, 1996; Schloerscheidt & Rugg, 1997). The present study is in agreement with previous findings by demonstrating that both controls and PPA subjects had significantly better accuracy for pictures than words. Reduced accuracy for word recognition was particularly marked for the target items. The lack of significant format differences for the foil items may be that the task was too easy or that the subject samples were too small to detect significant contributions of format. Alternatively, there may be a hierarchical system where the confusion caused by the semantically related items supercedes the picture superiority effect resulting in comparable performance for both word and picture foil items. In summary, these data suggest that accuracy is highest for unrelated foils, regardless of format, and target pictures; while inaccurate responses are heightened by the presence of target word stimuli and/or semantically related foils.

Compared to the controls, PPA patients had significantly reduced accuracy for word stimuli suggesting a memory deficit for target word stimuli. This is consistent with the fact that the prominent deficit in PPA patients is in the domain of language. Scores from the BNT were positively correlated with recognition of target picture performance, despite the fact that naming was not required for this recognition task. One possible explanation for this correlation is that relatively preserved naming ability enhances encoding of picture stimuli by adding a concrete label to the picture, which in turn, aids in better picture recognition memory performance.

Though many experiments have been conducted, the neuroanatomical correlates of episodic memory and false recognition have not been fully elucidated. In a recent review of the literature Schacter and Slotnick (2004) summarized the progress achieved thus far by showing that false recognition is associated with damage to the medial temporal lobes, while memory monitoring activities are associated with the prefrontal cortex. The results of the present study suggest that damage to the left perisylvian language network can also affect false recognition rates but by a different mechanism. Future neuroimaging studies with a variety of PPA patients will be important in further characterizing the neuroanatomical substrates of this phenomenon.

While the present study presents some unique findings there are limitations to the interpretation. The present study was designed to examine recognition memory in PPA for words and pictures, rather than to test one particular model of false recognition. Since our results did not overtly contradict any of the aforementioned theories, we are unable to explicitly defend or refute any one model. Future studies are encouraged to develop paradigms that directly examine the present theoretical models. For example, using a ‘remember’ versus ‘know’ confidence rating may help explain the elevated false recognition rates for the PPA patients as well as the relative contributions of recollection and familiarity. The current findings may also be extended by contrasting memory for nonverbal abstract (unnamable) objects or letter strings to that of words or nameable objects, which would allow for a juxtaposition of verbal and nonverbal memory abilities in a disintegrating language system.

PPA is a relatively heterogeneous syndrome in its course and rate of decline and patients are often divided into subtypes (semantic dementia, progressive nonfluent aphasia, logopenic) based on the nature of their language deficits (Gorno-Tempini et al., 2004). Though the present study lists the disease duration and clinical language profile for each patient, it combined the different subtypes of PPA since inspection of individual performance on the experimental task indicated no clear differences by subtype. However, given the relatively small number of agrammatic patients we cannot definitively guarantee that there are no performance differences by subtype. Future studies with larger sample sizes for each subtype would be useful in deciphering the possible differences in recognition memory performance. Nevertheless, in this sample of patients with progressive language impairment, it was possible to demonstrate relative preservation of true recognition with an exaggerated vulnerability for false memories to semantically related foils.

In summary, these results demonstrate that PPA patients’ memory, while relatively preserved for recognition of target stimuli, is worse for word stimuli and vulnerable to semantic interference. In addition, their ability to name objects affects successful recognition of pictures. This pattern of performance is not only unique from controls but also from that of kindred dementias, namely, AD and SD (Budson et al., 2003; Simons et al., 2005). The findings corroborate a previous priming study, where PPA patients demonstrated difficulty distinguishing among semantically similar items (Vandenberghe et al., 2005). Results do not directly dispute caregivers’ general observations that patients have relatively preserved memory, since only the finer aspects of memory, such as those studied here, are affected.

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Fig. 1.
Format accuracy by diagnosis.

Table 1**Diagnostic criteria for primary progressive aphasia (Mesulam, 2001)**

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- (1) There is an insidious onset and gradual but progressive impairment of word finding, object naming, syntax, or word comprehension manifested during conversation or assessed with the use of standard neuropsychological tests of language
 - (2) All major limitations in activities of daily living can be attributed to the language impairment for at least the first 2 years
 - (3) Premorbid language function (except for developmental dyslexia) is known to be intact
 - (4) Prominent apathy, disinhibition, loss of memory of recent events, visuospatial impairment, visual recognition deficits, and sensory-motor dysfunction are absent during the initial 2 years of illness, as indicated by the history, evaluation of activities of daily living, or neuropsychological testing, so that the patient would not fulfill diagnostic criteria for any other dementia syndrome
 - (5) Acaculia (inability to perform simple mathematical calculations) and ideomotor apraxia (inability to pantomime movement as instructed by an examiner) can be present even in the first 2 years of illness, and deficits in copying simple drawings and preservation may also be noted, but neither visuospatial deficits nor behavioral disinhibition substantially limits activities of daily living
 - (6) Other cognitive functions may be affected after the first 2 years of illness, but language remains the most impaired function throughout the course of the illness and deteriorates faster than other affected functions
 - (7) Specific causes of aphasia, such as stroke or tumor, as ascertained by neuroimaging, are absent
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Table 2

Subject demographics

	Mean (S.D.)		<i>p</i> -value
	PPA (<i>n</i> = 16)	NC (<i>n</i> = 16)	
Gender: M/F	8/8	6/10	0.48
Age	66.8 (7.0)	67.9 (8.3)	0.70
Education	16.8 (2.7)	16.4 (1.9)	0.71
Symptom duration: years	3.5 (1.8)	NA	NA

Table 3

Clinical subject characterization

Sub no.	Age ^d /sex	Symptom duration: years ^b	MMSE	CDR	ADL Q ^c (%)	Imaging	Language measures				Clinical characterization ^d	
							BNT (60)	Category fluency: animals	Lexical fluency: FAS	WAB yes/no (60)		BDAE complex ideation (12)
PPA 1	74/M	6	18	1	25	CT: normal	5	4	ND	ND	7	Logopenic, phonemic and semantic paraphasic errors, mild comprehension deficits
PPA 2	73/F	4.5	21	0.5	13	MRI: normal	48	14	12	ND	ND	Logopenic, semantic paraphasias, normal comprehension
PPA 3	57/M	2	28	0.5	ND	MRI: mild atrophy particularly in the perisylvian cortex bilaterally	58	14	14	ND	11	Logopenic, occasional phonemic paraphasic errors, normal comprehension
PPA 4	60/M	2.5	24	1	24	MRI: right perisylvian atrophy	52	12	ND	60	12	Logopenic, phonemic and semantic paraphasic errors, normal comprehension
PPA 5	68/M	2	26	1	15	MRI: nonspecific white matter intensities, age-specific atrophy	48	16	35	57	11	Logopenic, phonemic and semantic paraphasic errors, normal comprehension
PPA 6	74/M	2	15	1	15	MRI: prominent atrophy bilaterally in perisylvian and right parietal regions	35	14	6	54	ND	Logopenic, phonemic and semantic paraphasic errors, mild comprehension deficits
PPA 7	65/M	3.5	24	1	32	MRI: left perisylvian atrophy	30	5	3	ND	10	Agrammatic, phonemic paraphasic errors, mild comprehension deficits
PPA 8	63/F	3.5	25	0.5	6	MRI: normal	54	18	22	60	11	Logopenic, phonemic and semantic paraphasic errors, mild comprehension deficits
PPA 9	71/F	2	26	0.5	8	PET: decreased glucose metabolism in frontal and temporal areas	48	8	ND	60	9	Agrammatic, phonemic and semantic paraphasic errors, mild comprehension deficits
PPA 10	74/F	4	21	1	ND	MRI: nonspecific white matter intensities, age-specific atrophy	12	4	15	60	ND	Logopenic, phonemic and semantic paraphasic errors, normal comprehension
PPA 11	70/F	8	Not valid	0.5	10	PET: decreased glucose metabolism left > right	3	2	26	ND	ND	Logopenic, phonemic and semantic paraphasic errors, mild comprehension deficits
PPA 12	75/F	3	25	0.5	16	MRI: severe white matter abnormalities, bilateral perisylvian atrophy left > right	54	12	16	60	ND	Logopenic, no paraphasic errors, mild comprehension deficits
PPA 13	72/F	2.5	ND	1	0	MRI: incidental Falx meningioma; age	54	18	29	60	ND	Agrammatic, no paraphasic errors, normal comprehension

Sub no.	Age ^d /sex	Symptom duration: years ^b	MMSE	CDR	ADL Q ^c (%)	Imaging	Language measures				Clinical characterization ^d	
							BNT (60)	Category fluency: animals	Lexical fluency: FAS	WAB yes/no (60)		BDAE complex ideation (12)
PPA 14	58/M	6	28	0.5	13	appropriate hyperintensities MRI: left anterior- temporal atrophy MRI: left perisylvian and frontal atrophy CT: normal	12	7	29	60	ND	Logopenic, no paraphasic errors, normal comprehension
PPA 15	59/M	2	26	0.5	15		57	23	25	54	ND	Logopenic, no paraphasic errors, mild comprehension deficits
PPA 16	56/F	3	25	0.5	5		51	8	30	60	ND	Logopenic, occasional phonemic and semantic paraphasic errors, normal comprehension
NC Ave	67.9	NA	29.8	0	NA	NA	57.9	26.4	NA	NA	NA	NA

Numbers in parenthesis denote the maximum score. MMSE: Mini-Mental State Examination; CDR: Clinical Dementia Rating Scale; ADLQ: Activities of Daily Living Questionnaire. BNT: Boston Naming Test; WAB: Western Aphasia Battery yes/no comprehension subtest; BDAE: Boston Diagnostic Aphasia Examination Complex Ideational Material subtest; ND: not done; NA: variable not relevant for control participants.

^a Age at time of testing.

^b Reported length of symptom duration at the time of testing.

^c The ADLQ score is a composite of informants' ratings of activities related to self-care, household care, recreation travel, employment, and communication expressed as a percentage reflecting the degree to which activities of daily living are comprised (0–33% = mild, 34–66% = moderate, 67–100% = severe).

^d Patients are described based on their language profiles according to the criteria outlined by Gorno-Tempini et al. (2004).

Table 4

Mean recognition memory performance

	Mean (S.D.)		<i>p</i>-value
	PPA (<i>n</i> = 16)	NC (<i>n</i> = 16)	
True recognition of target items	0.83 (0.11)	0.85 (0.11)	0.597
False recognition of related foils	0.21 (0.11)	0.09 (0.05)	0.002
False recognition of unrelated foils	0.07 (0.07)	0.02 (0.02)	0.009

PPA patients had significantly more false alarms to both related and unrelated foils. There was a significant interaction effect of false alarm type (related, unrelated) by diagnosis.

Table 5Signal detection analyses of bias (c)

	c (S.D.)		p -value
	PPA ($n = 16$)	NC ($n = 16$)	
Item specific recollection (hits vs. unrelated foil false alarms)	0.23 (0.38)	0.44 (0.28)	0.09
Item specific recollection (hits vs. related foil false alarms)	0.13 (0.40)	0.34 (0.21)	0.07

Table 6Signal detection analyses of sensitivity (d)

	d (S.D.)		p -value
	PPA ($n = 16$)	NC ($n = 16$)	
Item specific recollection (hits vs. unrelated foil false alarms)	2.53 (0.64)	2.98 (0.40)	0.02
Item specific recollection (hits vs. related foil false alarms)	2.32 (0.59)	2.79 (0.54)	0.03