



Contents lists available at ScienceDirect

Journal of Neurolinguistics

journal homepage: www.elsevier.com/locate/jneuroling

Research paper

Am I looking at a cat or a dog? Gaze in the semantic variant of primary progressive aphasia is subject to excessive taxonomic capture



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ARTICLE INFO

Article history:

Received 7 April 2015

Received in revised form 10 September 2015

Accepted 14 September 2015

Available online 25 September 2015

Keywords:

Primary progressive aphasia

Eye tracking

Visual world paradigm

Taxonomic

Semantic interference

ABSTRACT

Object naming impairments or anomias are the most frequent symptom in aphasia, and can be caused by a variety of underlying neurocognitive mechanisms. Anomia in neurodegenerative or primary progressive aphasias (PPA) often appears to be based on taxonomic blurring of word meaning: words such as “dog” and “cat” are still recognized generically as referring to animals, but are no longer conceptually differentiated from each other, leading to coordinate errors in word-object matching. This blurring is the hallmark symptom of the “semantic variant” of PPA, who invariably show focal atrophy in the left anterior temporal lobe. In this study we used eye tracking to characterize information processing online (in real time) as non-aphasic controls, semantic and non-semantic PPA participants completed a word-to-object matching task. All participants (including controls) showed taxonomic capture of gaze, spending more time viewing foils that were from the same category as the target compared to unrelated foils, but capture was more extreme in the semantic PPA group. The semantic group showed heightened capture even on trials where they ultimately pointed to the correct target, demonstrating the superiority of eye movements over traditional testing methods in detecting subtle processing impairments. Heightened capture was primarily driven by a tendency to direct gaze back and forth, repeatedly, between a set of related foils on each trial, a behavior almost never shown by controls or non-semantic participants. This suggests semantic PPA participants were accumulating and weighing evidence for a probabilistic rather than definitive mapping between the noun and several candidate objects. Neurodegeneration in PPA thus appears to distort lexical concepts prior to extinguishing them altogether, causing uncertainty in recognition and word-object matching.

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1. Introduction

1.1. Word-object linkage

We frequently employ the ability to link words with objects in everyday life. For example, we can utilize nouns (e.g. a shopping list) to sort through objects in visually complex environments (the grocery store), in order to accomplish our goals (preparing dinner). Although seemingly simple, from an information processing standpoint this ability is remarkable: humans are able to recognize a seemingly limitless number of common objects, and to map them onto their respective nouns with great precision. This capacity dwarfs that of non-human species (dogs, parrots, apes, etc), the most gifted of which have an “object vocabulary” in the hundreds to low thousands (Kaminski, Call, & Fischer, 2004; Lyn, 2007; Pepperberg, 2002; Pilley & Reid, 2011). In contrast, even conservative estimates place average human vocabulary in the tens of thousands of words (Zechmeister, Chronis, Cull, D’Anna, & Healy, 1995), with a significant proportion of these being object-referential nouns, particularly in early stages of language acquisition (Gentner & Boroditsky, 2001). Our unique facility with word-object linkage is supported by close coordination between two large-scale neurocognitive networks: the left perisylvian language network and the bilateral inferotemporal object recognition network. Given the sheer amount of neural real estate involved, it is perhaps unsurprising that disrupted word-object linkage is one of the most common consequences of brain injury, often manifest as an inability to name objects aloud (anomia).

Successful word-object linkage requires the crossmodal mapping between the visual form of an object and the auditory or visual form of a word (letters or phonemes). As characterized in cognitive models of word production and object recognition (Bauer, 2006; Dell, Schwartz, Martin, Saffran, & Gagnon, 1997; Ellis & Young, 1988; Farah & McClelland, 1991; Humphreys, Price, & Riddoch, 1999; Levelt, Praamstra, Meyer, Helenius, & Salmelin, 1998), successful linkage is based on the completion of a number of distinct processing stages, including both structural and conceptual stages of processing in the object recognition and language networks.

For example, when attempting to name an object aloud, the structure of the object (form and features) must first be encoded in early stages of the ventral visual stream. Identification of unique, diagnostic features allows the object to be differentiated from other visually-similar objects (Clarke, 2015; Hoffman & Logothetis, 2009; Humphreys et al., 1999). Recognition unlocks conceptual knowledge of the object, which is based on a variety of learned associations, including crossmodal associations with the language network. This crossmodal interface allows the object concept to be connected to a corresponding verbal concept via shared meaning. Crossmodal associations are thought to contact the language lexicon (the theoretical storehouse of word knowledge) as a pattern of spreading activation. A lexical concept that corresponds to the object is chosen once it reaches an activation threshold. According to serial and interactive models of language (Dell & O’Seaghdha, 1991; Levelt, Roelofs, & Meyer, 1999), lexical concepts represent the meaning but not the sound of the word. Thus, in order to name an object aloud, the lexical concept must then be mapped onto a structural (phonological) representation, which in turn is converted into a motor speech program for vocal articulation. Failure at any of these stages (structural or conceptual stages of object or word processing) will result in the common symptom of anomia, requiring careful testing in order to reveal the underlying source of disruption.

1.2. Anomia in primary progressive aphasia

Anomia is the most common symptom of acquired language disorders (aphasias), whether they are caused by stroke (Laine, 2013), tumor resection (Davie, Hutcheson, Barringer, Weinberg, & Lewin, 2009), or neurodegenerative disease (Mesulam, Wieneke, et al., 2009), the latter known as primary progressive aphasias (PPA). Unlike vascular lesions, atrophy in PPA is equally likely to occur in any region of the language network, including areas not typically vulnerable to cerebrovascular incident (Gorno-Tempini et al., 2004; Mesulam, Wieneke, et al., 2009). Consistent with this, individuals with PPA show lesions and corresponding forms of anomia not seen in stroke aphasias.

The semantic variant of PPA (PPA-S) shows a particularly severe anomia apparently based on degradation of word knowledge; they are unable to match nouns with objects or to define those same nouns (Mesulam, Rogalski, et al., 2009; Mesulam et al., 2013). Verbal comprehension deficits take a peculiar form in PPA-S: these individuals no longer differentiate words from the same category such as “cat” and “dog” (taxonomic blurring). Loss of word meaning in PPA-S is gradual rather than absolute: although words can still be assigned to categories, indicating a generic level of recognition is retained, they cannot be differentiated from one another at a more specific level. Behavioral evidence of taxonomic blurring is provided by both superordinate and co-ordinate errors in naming, co-ordinate errors in picture-word matching, and overly-vague word definitions (Mesulam, Rogalski, et al., 2009; Mesulam et al., 2013). Blurring is also evident in electrophysiology: during picture-word matching tasks controls generate lower amplitude N400 event-related potentials in response to objects’ names compared to related words, while PPA-S participants show equivalent responses to both types of words (Hurley, Paller, Rogalski, & Mesulam, 2012). Surprisingly, loss of word meaning in PPA-S is consistently mapped to the anterior temporal lobe (ATL) (Hurley et al., 2012; Rogalski et al., 2011b) rather than the temporoparietal junction (the sometime seat of “Wernicke’s area”) (Bogen & Bogen, 1976; Geschwind, 1965; Mesulam, Thompson, Weintraub, & Rogalski, 2015).

The two other most common presentations of PPA are the agrammatic and logopenic subtypes, characterized by deficits in syntax and verbal repetition, respectively (Gorno-Tempini et al., 2011; Mesulam, Wieneke, et al., 2009). These “non-semantic” variants (PPA-NS) show peak atrophy in posterior and dorsal components of the language network, and are also frequently

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