Neuropsychologia 51 (2013) 2016–2025

Contents lists available at ScienceDirect

Neuropsychologia

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

Alzheimer's disease is associated with distinctive semantic feature loss

Kieran J. Flanagan [a], David A. Copland [a,b], Helen J. Chenery [c], Gerard J. Byrne [d], Anthony J. Angwin [a,*]

[a] University of Queensland, School of Health and Rehabilitation Sciences, St Lucia, Queensland 4072, Australia
[b] University of Queensland, Language Neuroscience Laboratory, Centre for Clinical Research, Brisbane, Queensland 4072, Australia
[c] University of Queensland, Centre for Clinical Research, St Lucia, Queensland 4072, Australia
[d] University of Queensland, School of Medicine, Brisbane, Queensland 4072, Australia

ARTICLE INFO

Article history:
Received 31 July 2012
Received in revised form
5 June 2013
Accepted 9 June 2013
Available online 14 June 2013

Keywords:
Alzheimer's disease
Semantic features
Semantic content
Semantic process

ABSTRACT

A central topic of discussion in the exploration of semantic disturbance in Alzheimer's disease (AD) concerns the relative contribution of semantic content (e.g., semantic features) and semantic process. Studies have suggested that semantic dysfunction in AD is the result of deficits to either semantic process, semantic content or both. Studies that have supported the loss of semantic content have been criticised for their use of verbal stimuli and cognitively challenging experimental tasks. The current study used a novel version of the yes–no recognition memory task to compare the processing of distinctive and non-distinctive features in participants with AD whilst controlling the cognitive demands of the task. The task involved five conditions which denoted the relationship between the items in the test and study phase. A 'non-distinctive' and a 'distinctive' condition were included where non-distinctive and distinctive semantic features were manipulated between study and test, respectively. Task accuracy of participants with AD decreased relative to control participants when distinctive features were manipulated between the study and test phase of the experiment. There was no significant difference between groups when non-distinctive features were manipulated. These findings provide evidence to support the loss of semantic content in AD.

1. Introduction

1.1. Semantic disturbance in AD

Language difficulties are very common for people with Alzheimer's disease (AD) (Cummings, Benson, Hill, & Read, 1985). The key clinical language characteristics of people with AD are word finding difficulties, semantic paraphasias and comprehension deficits with the relative sparing of repetition, morphology, syntax, phonology and motor speech skills (Blair, Marczinski, Davis-Faroque, & Kertesz, 2007; Huff, Corkin, & Growdon, 1986). It is widely regarded that the language deficits observed in AD stem from semantic disturbance (Chenery, 1996; Christensen, Kopelman, Stanhope, Lorentz, & Owen, 1998; Garrard & Carroll, 2006; Huff et al., 1986; Martins & Lloyd-Jones, 2006). As a consensus has not been reached in the literature as to the nature of semantic disturbance in AD, this study aims to explore competing accounts.

Alzheimer's disease is associated with multiple cognitive deficits and it is widely accepted that the semantic disturbance of AD involves deficits in semantic control (Reilly, Peelle, Antonucci, & Grossman, 2011). Semantic control is a collective term for the cognitive processes and skills believed to be necessary for completing semantic tasks and includes attention, memory, noise suppression, and enactment (Jefferies, Patterson, & Ralph, 2008). Of controversy in the literature exploring language in AD is the co-occurrence of disturbance to underlying semantic representations (Hornberger, Bell, Graham, & Rogers, 2009). The debate echoes Warrington and Shallice's (1979) access versus storage dichotomy where a division is drawn between semantic deficits caused by either disturbances to the access of semantic information or disturbance to the semantic information itself. However, the discussion of semantic disturbance could be more accurately described as one of access and storage (or process and content) versus access only (Reilly et al., 2011).

Authors who have supported the loss of semantic content have claimed that the neuropathology of AD compromises structures responsible for storing semantic information, and loss of this information contributes to semantic disturbance (Chertkow & Bub, 1990; Martin, 1992). Authors supporting the loss of semantic process only have suggested that semantic information remains largely intact but access to semantic information is impeded by the cognitive decline associated with the disease (Daum, Riesch, Sartori, & Birbaumer, 1996; Hartman, 1991; Nebes, Brady, & Huff, 1989; Nebes, Martin, & Horn, 1984). Each side of the debate is based upon a different theory of semantic representation and,

* Corresponding author. Tel.: +61 7 3346 7460; fax: +61 7 3365 1877.
E-mail address: a.angwin@uq.edu.au (A.J. Angwin).

0028-3932/$ - see front matter © 2013 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.neuropsychologia.2013.06.008

therefore, the resolution of this argument has implications for our understanding of semantic disturbance in AD as well as our understanding of the structure of the semantic system.

In Warrington and Shallice's (1979) discussion of access versus storage, storage/content disturbances were associated with consistent errors, frequency effects, selective loss of subordinate information, lack of priming and cueing effects. These criteria have been criticised for their lack of empirical or consistent theoretical basis (Rapp & Caramazza, 1993). Many subsequent studies that have supported loss of content in AD have focused upon semantic feature loss (Almor et al., 2009; Garrard, Ralph, Patterson, Pratt, & Hodges, 2005; Laisney et al., 2011). Semantic features can be defined as a description of the properties of a concept and can be classed as distinctive (defining features and therefore common to only one or two concepts) or non-distinctive (non-defining features and therefore common to many concepts) (McRae, Cree, Seidenberg, & McNorgan, 2005).

A number of computational models have been developed to explain semantic disturbance in AD that rely on semantic feature loss. Prominent models include Gonnerman, Andersen, Devlin, Kempler, and Seidenberg (1997), Plaut (1996) and Develin, Gonnerman, Andersen and Seidenberg (1998). Within these models, concepts are generally viewed as an interconnected network of semantic features. As non-distinctive features are common to many items within a category, they are frequently co-active, highly intercorrelated and therefore share numerous, strong links. Distinctive features, on the other hand, share few interconnecting links. With cortical damage characteristic of AD, interconnections between semantic features are expected to be lost. Being highly intercorrelated, non-distinctive features are not affected by the loss of interconnections early in the disease as many other connections are available to compensate. Distinctive features, however, are vulnerable early in the disease process.

Although these connectionist models were originally used to describe category specific deficits in AD, the vulnerability of distinctive semantic features has also been used to explain other patterns of semantic error in AD (Done & Gale, 1997; Garrard et al., 2005; Laisney et al., 2011; Rogers & Friedman, 2008). Early in the disease, people with AD are frequently reported to make category co-ordinate (e.g., calling a 'horse' a 'cow') and superordinate (e.g., calling a 'horse' an 'animal') semantic paraphasias (Bowles, Obler, & Albert, 1987; Hodges & Patterson, 1995). Such errors may be explained by the loss of distinctive semantic features, which make it difficult to distinguish between concepts within a category, yet category level knowledge is retained (Done & Gale, 1997; Garrard et al., 2005; Laisney et al., 2011; Rogers & Friedman, 2008). In the latter stages of the disease, people with AD are reported to frequently make unrelated errors or give 'I don't know' responses on naming tasks (Bowles et al., 1987; Hodges & Patterson, 1995). These errors may be explained by the loss of numerous interconnections later in the disease process, which compromises non-distinctive features and leads to the loss of category level knowledge (Done & Gale, 1997; Garrard et al., 2005; Laisney et al., 2011; Rogers & Friedman, 2008). The loss of semantic features in AD, particularly the loss of distinctive features, has been supported by a number of behavioural studies over the past decade (Done & Gale, 1997; Garrard et al., 2005; Laisney et al., 2011; Rogers & Friedman, 2008).

Garrard et al. (2005) requested participants with AD to list as many features as possible for a given object. Compared with controls, participants with AD listed fewer features, with particular difficulty listing distinctive features. The authors also found that the number of features given for an item was correlated with the ability to name that item. Laisney et al. (2011) created a semantic priming experiment where prime-target pairs consisted of concept-attribute pairs of either distinctive (e.g. zebra-stripes) or non-distinctive features (e.g. duck-feathers). Semantic priming was impaired in AD for distinctive attribute-concept pairs but not for non-distinctive attribute-concept pairs, whereas controls showed priming for both conditions. The findings of these studies have led the authors to conclude that semantic disturbance in AD involves the loss of semantic feature information, with distinctive semantic features being more vulnerable to loss than non-distinctive features (Garrard et al., 2005; Laisney et al., 2011). This differential loss of distinctive semantic features in AD is central to claims of semantic content loss as it is used to explain the progressive nature of semantic disturbance in the disease (Done & Gale, 1997; Garrard et al., 2005; Laisney et al., 2011; Rogers & Friedman, 2008).

A number of authors have suggested that semantic disturbance in AD is due to the disturbance of semantic processes only and not a deficit in semantic representations (Daum et al., 1996; Fung, Chertkow, & Templeman, 2000; Nebes et al., 1984; Ober & Shenaut, 1999; Perri et al., 2003). These authors have claimed that because people with AD have impaired cognition, especially attention and executive functioning, the ability to access otherwise spared semantic representations is also impaired. Furthermore, if the cognitive demands of a task are reduced (in a situation where automatic rather than effortful retrieval can occur), then the semantic disturbance observed in AD would be reduced (Daum et al., 1996; Nebes et al., 1984; Perri et al., 2003). Evidence for this account comes from a number of behavioural studies exploring semantic functioning in AD while controlling the cognitive demands of the task.

The first behavioural evidence for the semantic process only view was published by Nebes et al. (1984), who tested semantic memory in participants with AD and healthy controls using tasks with low cognitive demands (semantic priming, approximation to English and approximation to text tasks). The authors reported that participants with AD performed as well as controls on measures of semantic memory. Similarly, Daum et al. (1996) reported that participants with AD showed improved performance on semantic tasks with low cognitive demands such as object decision and preference judgment tasks compared with effortful tasks such as confrontation naming and definitions. These findings led the authors to reason that the semantic deficits of AD were the product of effortful semantic tasks with high cognitive demands preventing access to intact semantic representations (Daum et al., 1996; Nebes et al., 1984). Since these early studies, other authors have also reported that the semantic deficits of AD can be reduced through using non-effortful cognitive tasks (Fung et al., 2000; Perri et al., 2003). Though studies supporting deficits to semantic process only are in the minority, they highlight the importance of minimising and/or accounting for the cognitive demands associated with task performance in AD.

1.2. Theories of semantic representation in relation to AD

The outcome of the debate over loss of content in semantic dysfunction in AD has implications for models of semantic representation. Loss of semantic content, unlike disturbance to semantic process only, is consistent with models of semantic representation that rely on grounded representation. Proponents of grounded models assert that semantic memory is based on sensory representations (Barsalou, 2008). These models are distinct from amodal and hub and spoke models in which semantic memory is based on a central store of amodal symbols (Caramazza, Hillis, Rapp and Romani, 1990; Mahon & Caramazza, 2008; Reilly & Peelle, 2008). Within the context of grounded models, semantic concepts are represented as a distributed network of perceptual symbols/semantic features spread throughout the cerebral cortex, in particular, the somatosensory areas of the brain (Barsalou, Simmons, Barbey, & Wilson, 2003; Coltheart et al. 1998; Dove, 2009; Mayberry, Sage, & Ralph, 2011). Grounded models would predict that because AD involves progressive neural loss in many regions over the cerebral cortex (Perl, 2010), semantic feature loss, or disruptions to