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Mnemonic anosognosia in Alzheimer's disease: A test of Agnew and Morris (1998)

Eleanor L. Ansell, Romola S. Bucks *

School of Psychology, University of Southampton, Building 44 (Shackleton), Highfield, Southampton SO17 1BJ, United Kingdom

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Abstract

Agnew and Morris [Agnew, S. K. & Morris, R. G. (1998). The heterogeneity of anosognosia for memory impairment in Alzheimer's disease: A review of the literature and a proposed model. *Aging and Mental Health, 2*, 9–15] model of awareness for memory functioning has attempted to account for the variance of anosognosia exhibited within the Alzheimer's disease (AD) population. There has been tentative evidence to suggest that the mnemonic anosognosia sub-type, proposed by this model, is common within the early stages of AD. However, this study is the first directly to test the model. Eighteen older adults with early AD and 18 healthy older adults were recruited. Awareness of memory functioning was monitored using patient-performance measures of "task specific" awareness; a measure of global memory awareness; and a patient-informant measure. The stability of participants' awareness was measured across three word recall lists and after a 20-min delay. Results suggested that, whilst the participants with early AD were less aware of their memory ability than the healthy older adults, they were able to improve their awareness following exposure to a memory task. Furthermore, the improvements in awareness were largely retained after the delay period. These findings are discussed in relation to Agnew and Morris [Agnew, S. K. & Morris, R. G. (1998). The heterogeneity of anosognosia for memory impairment in Alzheimer's disease: A review of the literature and a proposed model. *Aging and Mental Health, 2*, 9–15] model of mnemonic anosognosia and to current thinking about autobiographical memory.

Keywords: Alzheimer's disease; Dementia; Insight; Anosognosia; Memory

Anosognosia is an organically based absence of insight into physical, neurological or cognitive impairments (McGlynn & Schacter, 1989; Schacter, 1990), which is particularly common in Alzheimer's disease (AD; Sevush & Leve, 1993); perhaps more common than in other progressive neurological disorders, such as Parkinson's disease (Danielczyk, 1993) and vascular dementia (Wagner & Crushman, 1994). It may even be predictive of the progression of mild memory impairments to AD (Tabert, Albert, & Borokhova-Milov, 2002). AD is characterised by prominent memory deficits, impairment in self-care abilities and disturbed mood and behaviour. A lack of awareness for these deficits has considerable implications for the clinical management, safety and quality of life of the person with AD (Koltai, Welsh-Bohmer, & Schmechel, 2001) and their caregivers (Seltzer, Vasterling, Yoder, & Thompson, 1997).

Anosognosia in AD has a complex presentation. It varies in degree (Bisiach, Vallar, Perani, Papagno, & Berti, 1986), in its type, e.g., explicit or implicit (De Vreese, Neri, Fioravanti, Belloi, & Zanetti, 2001), in the domain affected (Vasterling, Seltzer, Foss, & Vanderbrook, 1995), in its temporal stability (Neundorfer, 1997) and in its relationship to the progression of AD (McGlynn & Kaszniak, 1991; Migliorelli et al., 1995; Zanetti et al., 1999). Furthermore, although anosognosia has been associated with right hemisphere parietal damage (e.g., Reed, Jagust, & Coulter, 1993), executive dysfunction (e.g., Michon, Deweer, Pillon, Agrid, & Dubois, 1994) and memory impairment (e.g., Starkstein et al., 1995), none of these factors has been consistently associated with it (Clare, Wilson, Carter, Roth, & Hodges, 2004; Migliorelli et al., 1995; Starkstein et al., 1995). It is now believed that the complex presentation of anosognosia in AD represents a number of sub-types, with individuals differing in their vulnerability to specific types, depending on the stage in their disease process and the areas of the brain affected (Agnew & Morris, 1998; Lopez, Becker, Somsak, Dew, & DeKosky, 1994; Zanetti et al., 1999).

* Corresponding author. Tel.: +44 23 8059 3995; fax: +44 23 8059 4597.
E-mail address: romola.bucks@soton.ac.uk (R.S. Bucks).

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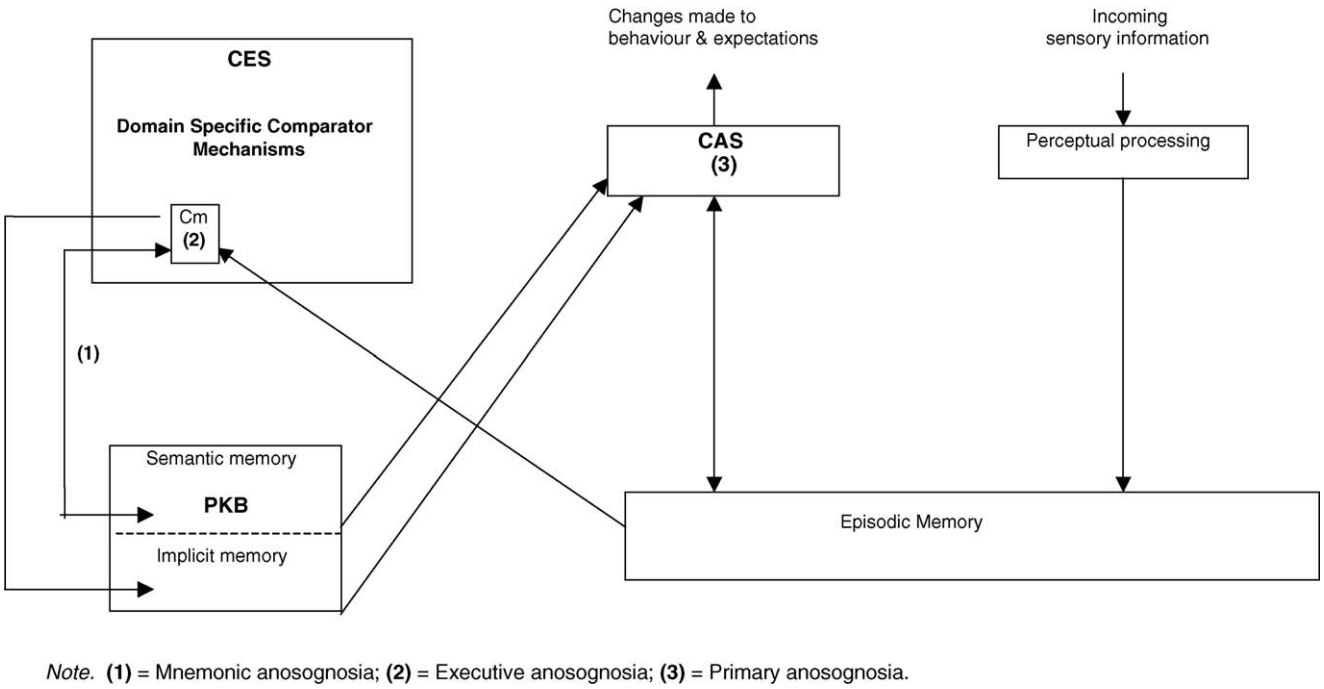

Fig. 1. Agnew and Morris DICE model (adapted from Agnew and Morris, 1998).

Agnew and Morris (1998) proposed a cognitive framework to explain the heterogeneous nature of anosognosia in people with AD (Fig. 1). According to their dissociable interactions and conscious experience model (DICE), incoming information about a recent memory failure first enters episodic memory. The person then consciously experiences the event as a result of the information passing to the conscious awareness system (CAS, Schacter, 1990) based in the parietal lobes. Contemporaneously, this information passes to a mnemonic comparator sub-served by the central executive, where it is compared with existing information about the person's memory ability in comparison to that of others and to past performance. This information is held in the person's semantic personal knowledge base (PKB[1]). If a mismatch arises between the memory event information and the contents of the PKB, then the PKB is updated via inputs from episodic to semantic memory. The person becomes consciously aware of any mismatch in this information through links between the Cm, episodic memory and CAS.

Agnew and Morris (1998) suggested that impairments in this system could result in three types of anosognosia. *Mnemonic anosognosia*: a deficit in the pathway between the Cm and semantic memory results in the person being unable to update their PKB. Thus, they show insight into their memory functioning after completing the task, but are unable to create an enduring awareness of their memory abilities; in effect they forget that they forget. However, due to the intact pathway between the Cm and implicit memory, they may exhibit subconscious alterations in emotional/behavioural reactions, whilst continuing to deny memory impairments. *Executive anosognosia*: a deficit in the Cm results in the person being unable to compare their memory performance with their PKB, leading to a lack of awareness of the deficit. As no information is sent to implicit memory, the person will not alter their behaviour. Although, theoretically, it is possible that the person could still obtain knowledge of their performance via a link between episodic and semantic memory, this knowledge would not have a value judgement component. According to the model, this would not constitute awareness, as they would not be aware that their performance had changed in relation to their own performance or in comparison to others'. *Primary anosognosia*: an impairment of the CAS results in unawareness of the state of functioning in all domains, not just memory. However, implicit learning is retained, which might lead to changes in behaviour, such as social withdrawal, without explicit awareness.

Although, there has been little direct research evaluating this model, there is related evidence that mnemonic anosognosia may be particularly common in people with early AD. Medial temporal lobe and hippocampal pathology are present in early AD and new learning and retrieval are impaired early in the disease course (Baddeley, Bressi, Della Sala, Logie, & Spinner, 1991; Nagy et al., 1999). As the pathology spreads to frontal and parietal structures, executive and primary anosognosia should become more apparent. Mnemonic anosognosia is of clinical interest as it may be possible to improve awareness through exposure to failures, thus facilitating rehabilitation (Clare et al., 2004; Green, Goldstein, Sirockman, & Green, 1993).

Initial evidence for mnemonic anosognosia comes from metacognitive research. Moulin, Perfect, and Jones (2000) and Duke, Seltzer, Seltzer, and Vasterling (2002) explored the ability

[1] We note that there are clear parallels between the PKB and metacognitive knowledge as defined by Flavell (1979; see Kuhn, 2000), although a discussion is beyond the scope of this paper.