

PRIMARY AND SECONDARY ANOSOGNOSIA FOR MEMORY IMPAIRMENT IN PATIENTS WITH ALZHEIMER'S DISEASE

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ABSTRACT

The neuropsychology of anosognosia for memory impairment in Alzheimer's disease (AD) was examined in 92 AD patients and 92 case matched individuals for comparison, using three quantitative methods of assessment: Experimenter Rating Scale (ERS), Objective Judgement Discrepancy (OJD) and Subjective-Rating Discrepancy (SRD). The OJD showed significant domain specific correlations with memory functioning as well as a significant correlation with susceptibility to intrusional errors. Memory or executive dysfunction may affect the immediate ability to judge cognitive performance in a domain specific manner (secondary anosognosia). Longer-term awareness of cognitive deficit appears less influenced by impaired basic cognitive functions, than by the decline of metacognitive function (primary anosognosia).

Key words: primary and secondary anosognosia, neuropsychology, Alzheimer's disease

Anosognosia, or loss of "awareness" concerning neuropsychological impairment is common in Alzheimer's disease (AD). The theoretical issues surrounding anosognosia, however, are complex with the core notion being that there are cognitive mechanisms that facilitate judgement about the current functioning of an individual and that these mechanisms may be selectively impaired. Thus anosognosia may be found for a range of functions, including for example, language, visual perceptual and memory disorders. Anosognosia also varies in degree from a mild lack of awareness into a cognitive disorder to denial of a profound loss of function such as anosognosia for blindsight. In addition, types of awareness may dissociate, for example, those with preserved awareness of hemiparesis have been found to lack awareness of cognitive deficit (Anderson and Tranel, 1989). Although anosognosia in AD may be modulated by psychological and social factors (Clare, 2002; Ownsworth et al., 2006), it is likely to be primarily caused by neurocognitive abnormality. Disruptions of the mechanisms that facilitate awareness at different levels within cognitive processing have been considered, for example, at lower mnemonic or executive or at higher metacognitive levels (Morris and Hannesdottir, 2004).

The specific cause of anosognosia in people with dementia has been a matter for debate (Mullen et al., 1996). Because there is evidence that anosognosia is found in patients who are generally intact neuropsychologically but have specific deficits (e.g., Babinski, 1914; Cutting, 1978) it is less likely explained as being due to generalised brain damage. This is also supported by the fact

that the various studies exploring the relationship between anosognosia and dementia severity appear to show no clear association, some studies show an increase in anosognosia with severity (Lopez et al., 1994; Mangone et al., 1991; Migliorelli et al., 1995; Starkstein et al., 1996; Sultzer et al., 1992; Verhey et al., 1993) but others not (DeBettignies et al., 1990; Feher et al., 1991; Nargeot et al., 1994; Reed et al., 1993). In AD, specifically, there is some indication that anosognosia is linked to frontal lobe dysfunction with neuroimaging studies showing an association with prefrontal cortical hypoperfusion (Reed et al., 1993; Starkstein et al., 1995).

The link between anosognosia and neuropsychological function has also been explored in AD. Executive functioning appears to show the strongest association (e.g., Auchus et al., 1994; Lopez et al., 1994; Mangone et al., 1991; Michon et al., 1994; Starkstein et al., 1993) possibly reflecting frontal lobe disturbance. The role of the frontal lobes has been suggested in the executive functions of self-monitoring, reasoning, problem solving and cognitive flexibility all of which are relevant to the formation of awareness or judgement of cognitive disorder. Moreover, the classical symptoms of executive disorder associated with the frontal lobe syndrome have been associated with unawareness. These include intrusional errors and confabulation, two features frequently observed in AD (e.g., Dalla Barba et al., 1995; Reed et al., 1993). Those studies that have measured anosognosia and executive and attention functioning have generally found associations in AD. For example, Mangone et al. (1991)

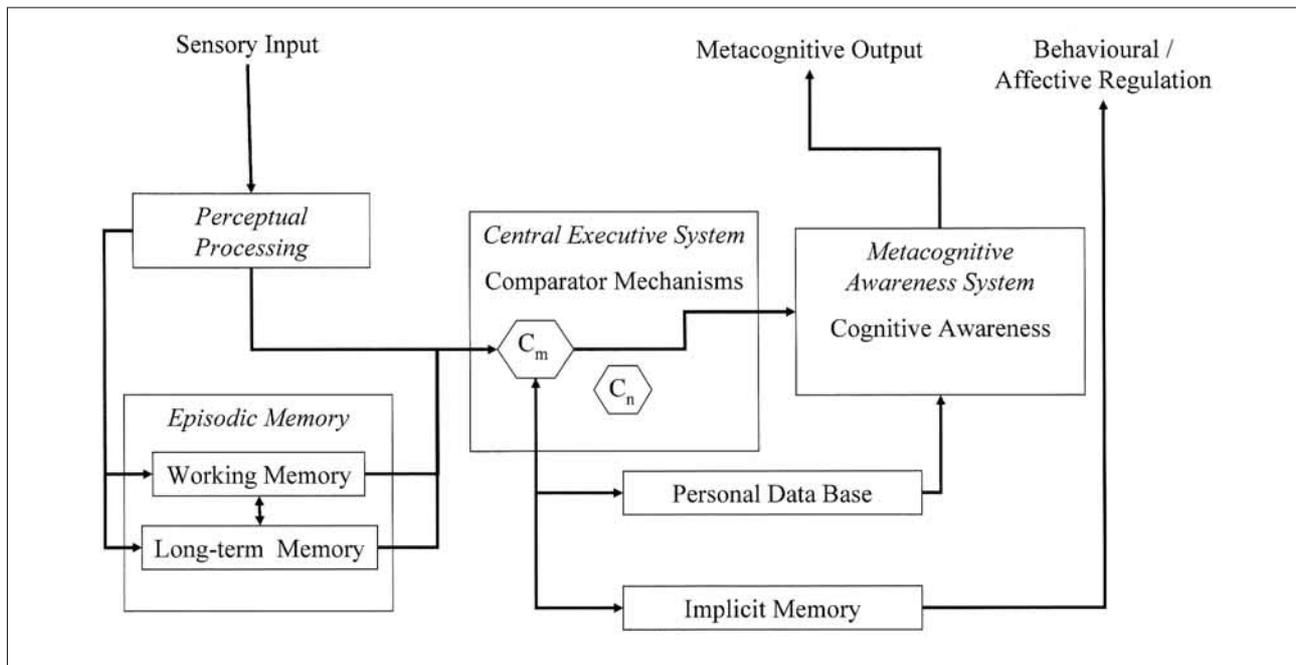


Fig. 1 – A model of anosognosia for memory impairment in Alzheimer's disease (Morris and Hannesdottir, 2004).

demonstrated an association with the Continuous Performance Test and a word sequencing and word generation test. Michon et al. (1994) computed a composite score using the Wisconsin Card Sorting Test, Verbal Fluency and a graphic series task and found a significant correlation with lack of awareness of memory impairment. Lopez et al. (1994) found that performance on a choice reaction time task and the Trail Making Test was associated with lack of awareness. More recently, loss of awareness has been linked to response inhibition impairment as indicated by Stroop test performance (Kashiwa et al., 2005). Nevertheless, associations with executive functioning have not always been found (Derouesné et al., 1999). Loss of memory ability might also be expected to prevent a person from evaluating his/her own functioning over time and hence lead to anosognosia. Thus, one might perhaps expect anosognosia to increase with the severity of memory impairment. However, most studies have not demonstrated such a relationship (Correa et al., 1996; DeBettignies et al., 1990; Derouesné et al., 1999; McGlynn and Kaszniak, 1991; Michon et al., 1994; Nargeot et al., 1994; Reed et al., 1993). This has led to the suggestion that perhaps the impairment of episodic memory may be considered as a maintenance factor in anosognosia rather than a primary cause (Agnew and Morris, 1998). Finally, correlations have been found with visuospatial function, but this might be due to the visuospatial tests having significant executive components; there are also associations with language function, but these may reflect poor understanding of anosognosic questions (Morris and Hannesdottir, 2004).

Cognitive neuropsychological models of loss of awareness have tended to focus the interface

between perception input and monitoring or control mechanisms. An early model put forward by McGlynn and Schacter (1989) is referred to as “dissociable interactions and conscious experience” (DICE; Schacter, 1990). A key feature of this model is the “Conscious Awareness System” (CAS) a specific system that is separate from but interacts with other modular processing, for example language, memory and perception, to form a conscious experience of phenomena. Anosognosia for deficits according to the DICE model could result from disruptions of a CAS or the system of executive function that controls this system. According to McGlynn and Schacter (1989) anosognosia may result from an amalgamation of several lesions of different cerebral areas, varying in degree and resulting perhaps in different types of anosognosia.

Agnew and Morris (1998) proposed an alternative cognitive model of anosognosia for memory impairment in AD. More recently, this has been developed by Morris and Hannesdottir (2004) in the Cognitive Awareness Model (CAM) to explain different features of anosognosia for cognitive impairment and also predict or explain different types of anosognosia in AD (see Figure 1). The underlying principle of the model is that in order for a person to assess their own cognitive functioning, they have to base the evaluation on experience of success or failure regarding a cognitive or behavioural task. This model includes the formation of a “Personal Data Base” (PDB), which maintains information about personal abilities and “comparator mechanisms” operating at various levels, which are used for monitoring performance. For example, problems with memory may be detected by a memory comparator system (C_m) and compared against that stored in the PDB

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